

# Indian Journal of Emergency Medicine

## Editor-in-Chief

**Indraneel Dasgupta,**  
Peerless Hospital & B.K. Roy Research Center, Kolkata

---

## Associate Editor

**Sajid Nomani,** AMRI Hospital, Odisha

## National Editorial Board

**Anoop Chakrapani,** Trivandrum

**Bidita Khandelwal,** Sikkim

**C.L. Nawal,** Jaipur,

**Habib Md. Rezaul Karim,** Port Blair

**Indranil Mitra,** Kolkata

**Jigar kumar Gosai,** Ahmedabad

**Ketan Patel,** Ahmedabad

**Kishalay Datta,** New Delhi

**P.K. Sasidharan,** Calicut

**S. P. Patel,** Lucknow

**S.K. Sharma,** New Delhi

**Saptarshi Saha,** Kolkata

**Sayan Misra,** Kolkata

**Sudip Chakraborty,** Kolkata

**Sujoy Das Thakur,** Kolkata

**Suman Kumar Kotwal,** Jammu

## International Editorial Board

**Jeffrey Smith,** Director, Ronald Reagan Institute of Emergency Medicine, U.S.A.

**George P. Abraham,** President, Indian Institute of Emergency Medical Services

## Managing Editor

A. Lal


## Publication Editor

Manoj Kumar Singh

---

**Indian Journal of Emergency Medicine (IJEM)** (pISSN: 2395-311X, eISSN: 2455-8370) is an international peer review journal covering pre-hospital and hospital emergency medicine, and critical care. The journal publishes original research, reviews and evidence based articles on resuscitation, major trauma, minor injuries, acute cardiology, acute paediatrics, toxicology, toxinology, disasters, medical imaging, audit, teaching and reflections on clinical practice. The journal is aimed at doctors, nurses, paramedics and ambulance staff.

→ Secure | <https://journals.indexcopernicus.com/search/form?search=Indian%20Journal%20of%20Emergency%20Medicine>



**INDEX COPERNICUS**  
INTERNATIONAL


**ICI World of Journals**

ICI Journals Master List    Contact

Login/ Register

---

**Search Results**



**Journal title:** Indian Journal of Emergency Medicine  
**ISSN:** 2455-8370, 2395-311X  
**GICID:** n/d  
**Country / Language:** IN / EN  
**Publisher:** Red Flower Publication Private Limited

<b>Citation:</b>	N/A
<b>MNISW 2016:</b>	N/D
<b>ICV 2016:</b>	77.03
<b>ICV 2015:</b>	66.10

# Indian Journal of Emergency Medicine

Vol. 3 No. 2, July - December 2017

## CONTENTS

---

---

### *Original Research Articles*

- Study of Serum Sodium and Potassium Levels in Patients of Acute Myocardial Infarction** 183  
Amith Kumar, Sathyanarayan T.B., Virupakshappa V.
- Achieving Sustainable Door-To-Balloon Time of 90 Minutes in a Tertiary Centre Hospital for St-Segment Elevation Myocardial Infarction** 188  
Gulati V., Datta K.
- Rational Use of Anti-Snake Venom: Trial of Various Use Regimes in Hemotoxic Snake Envenomation** 190  
Aravinda C.L., Nagabhushana S., Ranganatha M., Virupakshappa V.
- Management and outcome of Acute Kidney Injury at a Tertiary Care Hospital** 197  
Cijo John, Selin Abraham
- Prognosis of Haemodynamically Unstable Patients Secondary to Trauma Based on Lactate Clearance** 203  
Khan Khader Ali, Begum Naheeda Shaik, Shabbir Mohammed, Samir Mohammed
- Spectrum of Acute Febrile Illness in Children Presenting in Emergency of a Tertiary Care Hospital and Its Clinico - Laboratorial Correlation** 211  
Kishalay Datta, Rigenjyoti Kalita
- Non-Invasive Ventilation: First Line Therapy in the Acute Exacerbations of COPD in Emergency Department** 217  
Mohammed Ismail Nizami, Narendra Kumar N., Ashima Sharma, G. Vishwa Reddy, S. Raghavendra Goud
- The Study of the Clinical Profile and Laboratory Parameters of Acute Neonicotinoid Compound Poisoning at a Rural Tertiary Care Public Hospital in Central India** 223  
Mundhe Sanjay A., Birajdar Siddheshwar V., Chavan Sheshrao S., Kendre Vitthal M.
- Role of Intravenous Magnesium Sulphate in Predicting Outcomes of ICU in Acute Organophosphate Poisoning** 231  
Sri Harsha J., Srinivas Prabhu N.C., Ronak M. Raheja, O.R. Ranjan
- Patient Expectations in the Emergency Department of a Super-Speciality Hospital** 236  
Sudip Chakraborty, Saptarshi Saha, Indraneel Dasgupta

<b>Clinical Presentation of Renal Injury at a Tertiary Care Hospital</b>	<b>244</b>
Cijo John, Selin Abraham	
<b>'Tetpro Score' for Evaluation of Progression in a Case of Tetanus</b>	<b>249</b>
Vinay Swamy P.M., Bopanna C.A.	
<i>Review Article</i>	
<b>Myotonic Dystrophy: A Rare Autosomal Dominant Disorder</b>	<b>252</b>
Vikram Shah, Kishalay Datta, Sarat Naidu, Balasubramanyam E.V., Sonal Singh, Jitesh K. Bhandarkar	
<i>Case Reports</i>	
<b>A Rare Serious Ocular Side Effect of Topiramate: Bilateral Acute Angle Closure Glaucoma</b>	<b>255</b>
Dhruvkumar M. Patel, Mukundkumar V. Patel, Ajay V. Garg	
<b>A Case Report on Stroke in Young</b>	<b>258</b>
Aisvarya Girotra, Balasubramanyam E.V., Hilal Ahmad Yattoo, Kishalay Datta	
<b>A Case Report on Acute Myocardial Infarction in Young: Atypical ECG Changes Vs. Angiographic Correlation</b>	<b>261</b>
Aisvarya Girotra, Kishalay Datta, Rigenjyoti Kalita	
<b>One and Half Syndrome in Acute Pontine Infarct: A Rare Entity</b>	<b>264</b>
Anita Rawat, Kishalay Datta, Vaibhav Gulati	
<b>Carcinoma Prostate with Metastasis to Vertebral Column and Right Cerebellum Causing Sol and Hydrocephalus</b>	<b>266</b>
E.V. Balasubramanyam, Sonal Singh, Indranil Das, Kishalay Datta	
<b>Renal Thrombotic Microangiopathy Due to Malignant Hypertension</b>	<b>269</b>
Harini Agnes, Venugopal A.V.	
<b>A Case of Infant with Factor VII Deficiency Presenting as ICH</b>	<b>272</b>
Hilal Ahmad Yattoo, Vaibhav Gulati, Kishalay Datta, Rupinder Kahlon	
<b>An Unusual Presentation of Recurrent Hypoglycemia</b>	<b>275</b>
Singh A., Datta K., Das I., Kalita R., Govil P., Patel M.	
<b>Cerebral Venous Thrombosis and Hyperhomocysteinemia, How Important is the Co-Relation?-A Review of 3 Cases</b>	<b>278</b>
Lipoktemsu Jamir, Dina J. Shah	
<b>Asymmetrical and Late Onset of Pulmonary Edema Post Scorpion Sting: Case Report of Rare Manifestation</b>	<b>282</b>
Susmeet Mishra, Gouri Kumar Rath, Sajid Nomani	
<b>Acute Isolated Posterior Myocardial Infarction; Challenges in Recognition and Management in the Emergency Department</b>	<b>286</b>
Sarat Kumar Naidu, Ankur Pandey, Kishalay Datta	

<b>Petrol Ingestion Causing Methaemoglobinaemia in Glucose 6-Phosphate Dehydrogenase (G6PD) Deficiency Patient</b>	<b>290</b>
Nasir Shakilli, Mohammad Kamal, Bakshi Surrinder Kumar, Madhusudhanan M., Moosa Al Abri, Almur Abdullah Alabri	
<b>Early Diagnosis and Treatment not Always a Key to Favorable Outcome: A Case Report of ADEM Correctly Diagnosed and Treated Still Surviving for Better Life</b>	<b>293</b>
Aakansha Singh, Vaibhav Gulati, Kishalay Datta, Hilal Ahmad Yatoo	
<b>Traumatic Cardiac Tamponade: Relearning Old Lesions to Avoid Delay in Diagnosis and Management of a Life-Threatening Thoracic Injury</b>	<b>296</b>
Sarat Kumar Naidu, Vikram Shah, Gurjit Kaur, Kishalay Datta	
<b>Torsion of Non-Gravid Uterus with Myoma Presenting to Emergency with Shock</b>	<b>301</b>
Muhammad Aamir Mir, Kritika Nanda, Kamal Preet Palta, Kishalay Datta	
<b>A Rare Case of Complicated Neuroleptic Malignant Syndrome with Rhabdomyolysis and Acute Kidney Injury</b>	<b>304</b>
Sarat Kumar Naidu, Gurjit Kaur, Vikram Shah, Kishalay Datta	
<b>Life Threatening Rhabdomyolysis, A Rare and Unusual Presentation with Rosuvastatin Ingestion</b>	<b>308</b>
Umran Rafeeq Sheikh, Kishalay Datta, Priya Govil, Deepika Mittal	
<b>Beyond ACLS Protocol: A Rare Case of Refractory Supraventricular Tachycardia Responding Only to a Much Higher Dose of Adenosine</b>	<b>311</b>
Sarat Kumar Naidu, Hilal Yatoo, Kishalay Datta	
<b>An Unusual Presentation of Fat Embolism Syndrome as Cerebral Fat Embolism in Trauma: A Rare Clinical Entity</b>	<b>316</b>
Nitish Dhand, Kishalay Datta, Vaibhav Gulati, Indranil Das, E.V. Balasubramanyam, Vikram Shah	
<b>Bilateral Acute Lower Limb Arterial Occlusion after Long Term Tranexamic Acid Usage</b>	<b>319</b>
P. Anvesh, A.V. Venugopal, Harini Agnes, Siddardh	
<b>Glossopharyngeal Neuralgia Leading to Sinus Pause: A Rare Entity</b>	<b>322</b>
Vaibhav Gulati, Kishalay Datta, Naveen Bhamri	
<b>G6PD Deficiency as a Precipitant of Haemolysis in Hepatitis E Patients</b>	<b>325</b>
Umran R. Sheikh, Kishalay Datta, Shahid Mustafa Khan, Indranil Das, Deepika Mittal	
<b>Guidelines for Authors</b>	<b>329</b>
<b>Subject Index</b>	<b>333</b>
<b>Author Index</b>	<b>335</b>

**Subscription Information****Institutional** (1 year) INR12000/USD857**Here is payment instruction for your reference.****Check:**

Please send the US dollar check from outside India and INR check from India made:  
Payable to 'Red Flower Publication Private Limited'.  
Drawn on Delhi branch

**PayPal Instructions for the payment (only for transfer from outside India):**

Payments can be made through our PayPal account at <https://www.paypal.com>.  
Our PayPal recipient email address is [redflowerppl@gmail.com](mailto:redflowerppl@gmail.com).

**Credit Card:**

We accept Visa or MasterCard.

**Wire transfer:**

Complete Bank Account No. 604320110000467  
Beneficiary Name: Red Flower Publication Pvt. Ltd.  
Bank & Branch Name: Bank of India; Mayur Vihar  
MICR Code: 110013045  
Branch Code: 6043  
IFSC Code: BKID0006043 (used for RTGS and NEFT transactions)  
Swift Code: BKIDINBBDOS

**\*\*Please kindly add bank charge at your side if you pay by check or wire transfer.**

**Payment, orders and all correspondences should be sent to;**

Red Flower Publication Pvt. Ltd.  
48/41-42, DSIDC, Pocket-II  
Mayur Vihar Phase-I  
Delhi - 110 091(India)

## Study of Serum Sodium and Potassium Levels in Patients of Acute Myocardial Infarction

Amith Kumar<sup>1</sup>, Sathyanarayan T.B.<sup>2</sup>, Virupakshappa V.<sup>3</sup>

### Author's Affiliation:

<sup>1</sup>Assistant Professor <sup>2</sup>Associate Professor <sup>3</sup>Professor and HOD,  
Dept. of Medicine, Shimoga  
Institute of Medical Sciences,  
Shivamogga, Karnataka 577201,  
India.

### Corresponding Author:

**Sathyanarayan T.B.,**  
Associate Professor,  
Department of Medicine,  
Shimoga Institute of Medical  
Sciences, Shivamogga, Karnataka  
577201, India.  
E-mail:  
medicalsuptd@rediffmail.com

Received on 23.09.2017,

Accepted on 06.10.2017

### Abstract

*Background:* Cardiovascular disease is one of the leading causes of morbidity and mortality across the world. World Health Organization (WHO) has declared cardiovascular disease as a modern epidemic. Acute Myocardial Infarction is one of the manifestations of coronary heart disease leading to morbidity and mortality. Arrhythmias and hemodynamic abnormalities in left ventricular dysfunction are the major causes of mortality along with acute myocardial infarction. In majority of the patients with acute myocardial infarction, one of the commonest cause of death is life threatening arrhythmias. Many inorganic salts especially of alkaline elements including sodium and potassium. *Material and Methods:* Prospective study carried out in Medicine department, SIMS, Shimoga for 6 months from Jan 2017 to June 2017. 50 patients of acute myocardial infarction admitted to intensive coronary care unit, of Shivamogga institute of medical sciences, Shivamogga, irrespective of site of infarction and irrespective of type of arrhythmia were included in the study. Detailed history of each patient was obtained. Thorough physical and systemic examination was done in all the patients. Routine blood and urine examination was sent. First electrocardiogram was taken at the time of admission. Serial electrocardiograms were taken till patient remained till the time of discharge or death. Serum sodium and potassium was estimated in the manner like i. At the time of admission to ICCU. ii. At the time of development of arrhythmia or after 24 hours of admission if arrhythmias were not present. *Result:* 15 patients (30%) of all MI patients found to be hyponatraemic. 10 patients (20%) found to be hypokalaemic. Patients with hyponatremia were not found to have any rhythm disturbance, 3 patients with hypokalemia had frequent ventricular ectopics, 1 patient had atrial fibrillation and 2 patients had ventricular tachycardia. *Conclusion:* there was no increase in rhythm disturbances in hyponatremic patients, however there was definite correlation with arrhythmias in patient with hypokalemia..

**Keywords:** Myocardial Infarction; Hyponatremia; Hypokalemia; Arrhythmia.

### Introduction

Acute myocardial infarction in the age group 31-70 years is increased from 270 cases per 100,000 person years in 1999 to a peak of 294 cases per lakh person years in 2000, and then reduced every year

thereafter, to 208 cases per lakh person years in 2008 [1]. The prevalence of ischemic heart disease in Indian population was estimated at 96.7 per 1000 individuals in the urban and 27.1 percent in rural areas [2]. Cardiovascular disease is one of the leading causes of morbidity and mortality across the world. World Health Organization (WHO) has declared

cardiovascular disease as a modern epidemic [3]. Acute Myocardial Infarction is one of the manifestations of coronary heart disease leading to morbidity and mortality. Arrhythmias and hemodynamic abnormalities in left ventricular dysfunction are the major causes of mortality along with acute myocardial infarction. The arrhythmias predisposing factors are: autonomic nervous system dysfunction, electrolyte disorders, left ventricular dysfunction, myocardial ischemia and medications [4]. Different electrolytes such as potassium and sodium play an important role in the cell metabolism, electrical conduction and membrane excitability. Abnormalities of these electrolytes due to different causes can lead to a significant cardiac life threatening events [5].

### Material and Methods

50 patients of acute myocardial infarction admitted to intensive coronary care unit, of Shivamogga institute of medical sciences, Shivamogga, irrespective of site of infarction and irrespective of type of arrhythmia were included in the study.

Detailed history of each patients was obtained. Thorough physical and systemic examination was done in all the patients. Routine blood and urine examinations were completed. First electrocardiogram was taken at the time of admission. Serial electrocardiograms were taken till time of discharge or death.

Serum sodium and potassium will estimated in the following manner.

- i. At the time of admission to ICCU
- ii. At the time of development of arrhythmia or after 24 hours of admission if arrhythmias were not present

### Inclusion Criteria

Patients with acute ST segment elevation myocardial infarction.

### Exclusion Criteria

- i. Patients with unstable angina
- ii. Patients with nonST segment elevation myocardial infarction
- iii. Anaemia, significant hepatic, renal and pulmonary disease, diabetes mellitus, patient on drugs which can interfere with serum Na and K, like ace inhibitors and diuretics were excluded from the study

All the patients of the STEMI were grouped according to Serum

Serum sodium (mmol/l) < 136 mmol/l, 136 to 145 mmol/l, > 145 mmol/l

Serum potassium mmol/l < 3.5 mmol/l, 3.5 to 5 mmol/l, > 5mol/l

Observed outcome was presence of arrhythmias and their correlation with serum electrolytes. The observed clinical outcome was analysed by Chi square test . P value of less than 0.05 was taken as statistically significant.

### Results

In our study population, 70 % (n=35) were male and 30% (n=15) were female. Out of these 12% (n=6) patients died during first seven days. Amongst the patients died 8 % (n=4) were male while 4% (n=2) were female. Majority of study subjects (70%) had normal serum sodium of 136-145 mmol/l. Study subjects with serum sodium level < 136 mmol/L were (30 %) . there was no association of arrhythmias in patients with low sodium, among 6 patients died, all had normal sodium levels between 136 -145 mmol/l.

In our study, 20% of patients (N = 10) were found to have hypokalemia, out of which 14% (N=7) were males, and 6% (N=3) were females. Among these 3 patients developed frequent ventricular ectopics, f1 had atrial fibrillation and 2 patients developed ventricular tachycardia which was statistically significant.

**Table 1:** Distribution of cases according to serum sodium concentration

Serum Sodium	Total Number of Patients	Male	Female
< 135 mmol/L	15 (30%)	12 (24%)	5(10%)
136-145 mmol/L	35 ( 70%)	23 (46%)	10(20%)

**Table 2:** Distribution of cases according to potassium concentration

Serum Potassium	Total Number of Patients	Male	Female
< 3.5 mmol/L	10 (20%)	7 (14%)	3(6%)
3.5 - 5.0 mmol/L	40 (80%)	30 (60%)	10(20%)



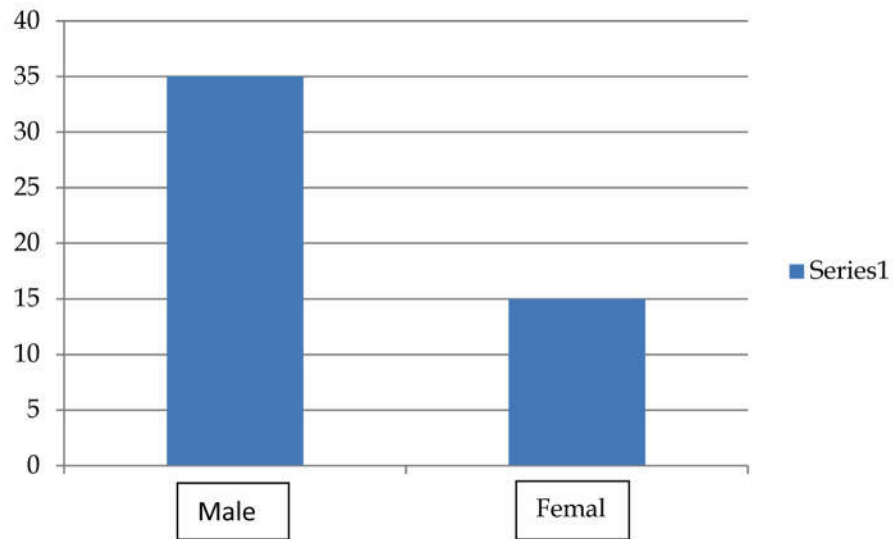


Fig. 1: Bar graph showing sex distribution of cases

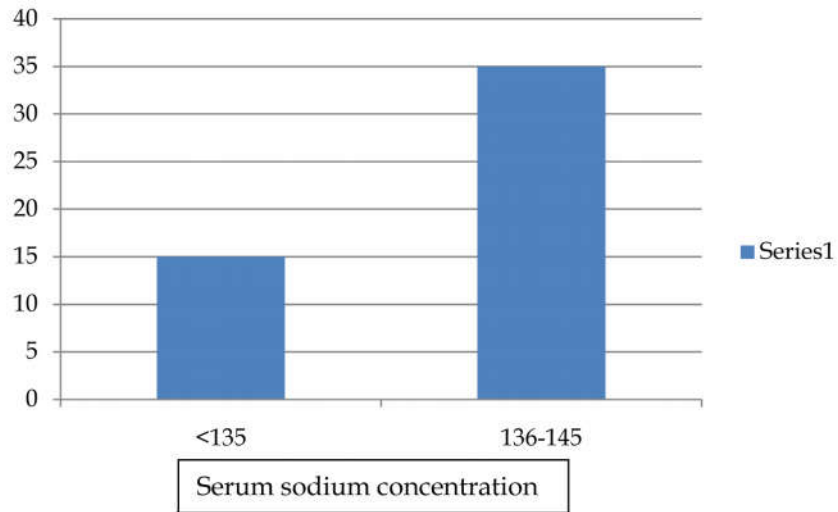


Fig. 2: Bar graph depicting serum sodium levels in patients

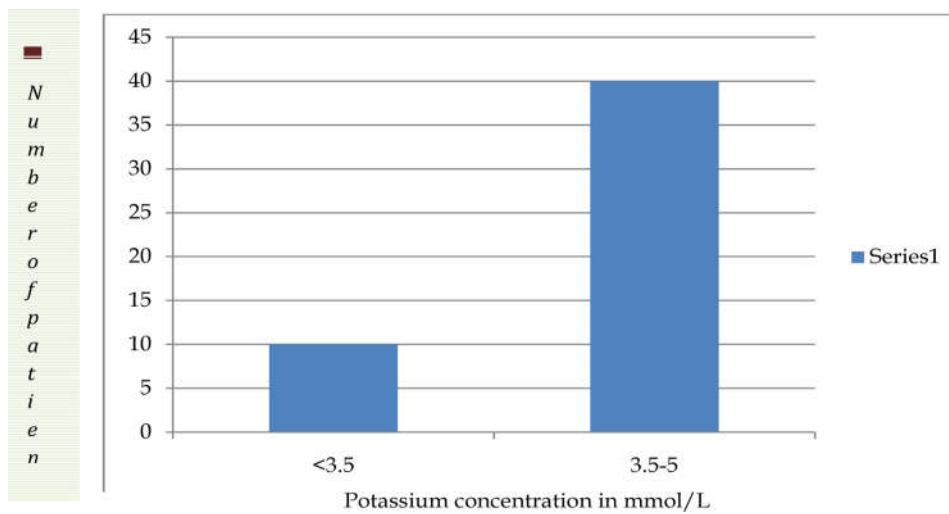


Fig. 3:

Table 3:

Arrhythmia	VPCS	Atrial Fibrillation	Ventricular Tachycardia
No of patients	3	1	2

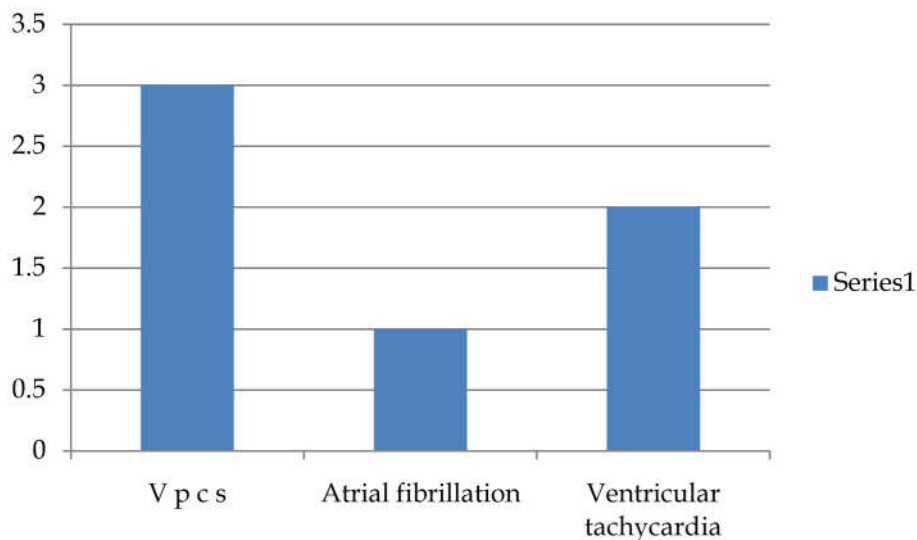


Fig. 4: Graph showing incidence of arrhythmia in hypokalemia

## Discussion

Dyselectrolyemia is often present in the acute MI. When measured on admission, low sodium levels were found to be reduced in 30% of acute myocardial infarction patients in our study and 10% of our patients had low potassium levels.

Hyponatremia often associated with increased morbidity and mortality in MI patients. MI patients with hyponatremia also found to have features of heart failure. A study conducted by Flear et al showed 45% of infarcted patients had hyponatremia and were associated with increased mortality [6]. In this study 30% of MI patients were found to have hyponatremia. Hyponatremia could probably be due to non-osmotic secretion of vasopressin thereby reducing the water removal causing dilutional hyponatraemia. Sodium is freely filtered by the glomerulus, majority of it is reabsorbed in the proximal tubule, 20-25% in the loop of Henle and remaining 5-10% in distal tubules [7]. Flear et al had hypothesized that the hypoxia and cardiac ischemia increased the cell membrane permeability to sodium ions, activation of sympathetic nervous system and rennin-angiotensin system [5]. None of the patients were found to be hypernatraemic in our study.

Hypokalaemia is associated with an increased risk of ventricular arrhythmias like ventricular

tachycardia and ventricular fibrillation [8]. Skeletal muscle is an important reservoir pool for potassium maintaining potassium in vital organs such as the heart and brain [9,10]. In a study done by Goyal et al, it was found the hospital mortality in MI patients to be the least in patients with normal potassium levels (3.5-4.5mmol/l) [11]. In our study 20% of cases were hypokalaemic which was significant. There was no patient in our study who had hyperkalemia. Patients who had hypokalemia 3 patients had ventricular ectopics, 1 had atrial fibrillation and another 2 had ventricular tachycardia. The association of hyponatremia and hypokalaemia with early presentation in acute MI may alert the clinician about the acuteness and severity of patient's illness.

## Conclusion

Dyselectrolyemia is fairly common in patients with acute myocardial infarction. Hyponatremia was present in about 30% of our patients however there was no correlation of arrhythmias in patients with low sodium. Hyponatremia was a fairly common finding among acute MI patients, probably attributed to the non-osmotic secretion of vasopressin. Hypokalaemia was present in 20% of patients in acute myocardial infarction, mostly due to the catecholamine response in such patients. It has been

associated with ventricular arrhythmias and increased mortality in post MI patients. The clinicians are advised to closely monitor these electrolyte changes and correct them as they seem to have adverse effects on the disease outcome and prognosis.

## References

1. Robert WY, Stephen S, Malini C, Michael S, Joseph VS, Alan SG. Population trends in the incidence and outcomes of acute myocardial infarction. *N Engl J Med* 2010;362(23):2155-65.
2. Vamadevan SA & Dorairaj P. Coronary heart disease in Indians: Implications of the interheart study. *Indian J Med Res* 2010;132:561-566.
3. K Park. *Park's Textbook of Preventive and Social Medicine*, 22nd ed. Jabalpur: Bhanot Publishers; 2013.p.338.
4. Vera Z, Janzen D, Desai J. Acute hypokalemia and inducibility of ventricular tachyarrhythmia in a nonischemic canine model. *Chest*. 1991;100(5): 1414-20.
5. Tada Y, Nakamura T, Funayama H, Sugawara Y, Ako J, Ishikawa S, Momomura S. Early development of hyponatremia implicates short and long term outcomes in ST elevation acute myocardial infarction. *Circ J* 2011;75:1927-1933.
6. Flear C T G, Hilton P. Hyponatremia and severity and outcome of myocardial infarction. *BMJ*. 1979;1:1242-1246.
7. Singla I, Zahid M, Good C B, Macioce A, Sonel A F. Effect of hyponatremia on outcome of patients in non ST elevation acute coronary syndrome. *Am J Cardiol*. 2007;100:406-408.
8. Solomon R J, Cole A G. Importance of Potassium in Patients With Acute Myocardial Infarction. *Acta Medica Scandinavica*. 1981;209:87-93.
9. Madias JE, Shah B, Chintalapally G, Chalavarya G, Madias NE. Admission serum potassium in patients with acute myocardial J *Clin Biomed Sci* 2012;2:173. EshaMati et al Source of Support: Nil Conflict of Interest: Nil infarction: its correlates and value as a determinant of in hospital outcome. *Chest*. 2000;118:904-913.
10. Kaltofen A, Lindner KH, Ensinger H, Ahnefeld FW. The modification of the potassium concentration in blood by catecholamines. A literature review. *Anasth Intensivther Notfallmed*. 1990;2:405-410.
11. Xianghua F, Peng Q, Yanbo W, Shigiang L, Weize F, Yunfa J. The relationship between hypokalemia at the early stage of acute myocardial infarction and malignant ventricular arrhythmia. *Heart*. 2010;96:196.

# Achieving Sustainable Door-To-Balloon Time of 90 Minutes in a Tertiary Centre Hospital for ST-Segment Elevation Myocardial Infarction

Gulati V.<sup>1</sup>, Datta K.<sup>2</sup>

**Author's Affiliation:**  
<sup>1</sup>PGY-III, MEM <sup>2</sup>HOD and Associate Director, Department of Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

**Corresponding Author:**  
**Vaibhav Gulati**, PGY-III, MEM, Emergency Department, Max Super Speciality Hospital, Shalimar Bagh, Delhi.  
E-mail: [dr.vaibhavgulati@gmail.com](mailto:dr.vaibhavgulati@gmail.com)

Received on 25.10.2017,  
Accepted on 08.11.2017

## Abstract

Chest pain is one of the most common presentation in emergency department and India has the highest burden of acute coronary syndrome in the world. Increasing awareness regarding it has led to increased diagnosed cases and hence people getting early definitive treatment. Various studies have shown that door to balloon time within 90 min increases the survival rate. In this study, we analyse the door to balloon time of 90 min in a tertiary centre hospital for ST-elevation myocardial infarction.

**Keywords:** Infarction; ST-Segment Elevation; ECG.

## Objectives

To achieve door-to-balloon times for PCI within 90 min in a STEMI patient.

## Background

- India has the highest burden of ACS in the world. The CREATE registry has provided contemporary data on 20,468 patients from 89 centers from 10 regions and 50 cities in India.
- The median time from symptoms to hospital was 360 min (several times higher than in the US and other high income countries). However from hospital to thrombolysis was only 50 minutes.
- 59% of patients with STEMI received thrombolytics (96% streptokinase). Coronary angioplasty was given to 8% of STEMI and 7% of non-STEMI; coronary bypass surgery was given to 2% of STEMI and 4% of NSTEMI/UA. The 30-day outcomes for patients with STEMI were: death 9%; reinfarction 2%; and stroke 0.7%.

## Methods

Retrospective study for a period of 1 year from July, 2016 to June, 2017 was done. All STEMI patients

regardless of comorbidities, transfer from other hospital were included in the study.

## Inclusion Criteria

- Any age
- All STEMI patients
- Any co-morbidities
- Transfer from other hospitals.

## Parameters Studied

- Average door to cath lab time (1),
- average cath lab to balloon time (2)
- average door to balloon time(1+2) was calculated.
- Data was collected from the STEMI-form attached with patients admission sheet which were filled by the ED doctor and doctor doing the procedure.

## Results

- A total of 271 STEMI patients presented with STEMI during the study period. All the patients were taken up for Coronary angioplasty(100%).
- The average door to cath lab time was 15.45min.
- The average cath lab to balloon time was 28.4min.

## Data

Month	No. of patients	No. of deaths	Door to Cath lab time (min)	Cath lab to balloon time (min)	Door to Balloon time (min)
July,16	15	1	11	22	33
August,16	15	1	10	19	29
September,16	23	1	11.34	29.7	41.04
October,16	21	2	15.52	25.71	41.24
November,16	26	3	19.19	28.38	47.58
December,16	38	4	20.5	26.07	46.57
January,17	28	2	18.3	20	38.3
February,17	21	3	15.9	27	42.9
March,17	19	1	15.16	26.16	41.32
April,17	22	2	12	26.9	38.9
May,17	28	1	16.5	45.25	61.75
June,17	15	2	20	44.67	64.67
<b>TOTAL</b>	<b>271</b>	<b>23</b>	<b>15.45</b>	<b>28.4</b>	<b>43.85</b>

- The average door to balloon time calculated was 43.85min

## Conclusions

- These results demonstrated that these timings are much less than the International and Indian standard protocol (<90Min).
- This reduced door to balloon time has decreased the length of stay in hospital and mortality in STEMI patients remarkably.

## Abbreviation

STEMI- ST Elevation Myocardial Infarction

ACS - Acute Coronary Syndrome

PCI - Percutaneous Coronary Intervention

## References

1. Robert L. McNamara, MD, MHS, Yongfei Wang, MS, Jeph Herrin, PHD, Jephtha P. Curtis, MD, Elizabeth H. Bradley, PHD, David J. Magid, MD, MPH, Eric D. Peterson, MD, MPH, Martha Blaney, PHARM.D., Paul D. Frederick, PHD, Harlan M. Krumholz, MD, SM. Effect of Door to Balloon time on mortality in patients with ST-segment elevation Myocardial Infarction. J Am Coll Cardiol. 2006 Jun 6;47(11):2180-6. Epub 2006 May 15.
2. Daniel S. Menees, M.D., Eric D. Peterson, M.D., Yongfei Wang, M.S., Jephtha P. Curtis, M.D., John C. Messenger, M.D., John S. Rumsfeld, M.D., Ph.D., and Hitinder S. Gurm, M.B., B.S. Door-to-Balloon Time

and Mortality among Patients Undergoing Primary PCI. N Engl J Med 2013;369:901-909 September 5, 2013 DOI: 10.1056/NEJMoa1208200.

3. Katsufumi Nishida, Sean K Hirota, Todd B Seto, Daniel C Smith, Cathy Young, MBAC, Wanda Muranaka, Suzanne Beauvallet, David J Fergusson. Quality Measure Study: Progress in Reducing the Door-to-Balloon Time in Patients with ST-segment Elevation Myocardial Infarction. Hawaii Med J. 2010 Oct;69(10):242-6.
4. David J Fergusson, Christian Spies, Robert A Hong, Catherine Young, Suzanne Rinn Beauvallet. Door-to-Balloon time in Acute ST Segment Elevation Myocardial Infarction - Further Experience. Hawaii J Med Public Health. 2012 Nov;71(11):320-323.
5. Dr Brahmajee K Nallamothu, Prof Sharon-Lise T Normand, Yongfei Wang, Prof Timothy P Hofer, Prof John E Brush Jr, John C Messenger, Prof Elizabeth H Bradley, Prof John S Rumsfeld, Prof Harlan M Krumholz. Relation between door-to-balloon times and mortality after primary percutaneous coronary intervention over time: a retrospective study. The Lancet. 2015 March 21;385(9973):1114-1122.
6. Saif S Rathore, Jephtha P Curtis, Jersey Chen, Yongfei Wang, Brahmajee K Nallamothu, Andrew J Epstein. Association of door-to-balloon time and mortality in patients admitted to hospital with ST elevation myocardial infarction: national cohort study. BMJ 2009;338 doi: <https://doi.org/10.1136/bmj.b1807> (Published 19 May 2009).
7. Harlan M. Krumholz, Jeph Herrin, Lauren E. Miller, Elizabeth E. Drye, Shari M. Ling, Lein F. Han, Michael T. Rapp, Elizabeth H. Bradley, Brahmajee K. Nallamothu, Wato Nsa, Dale W. Bratzler, Jephtha P. Curtis. Improvements in Door-to-Balloon Time in the United States, 2005 to 2010. Circulation. 2011 Aug 30;124(9):1038-45.

## Rational Use of Anti-Snake Venom: Trial of Various Use Regimes in Hemotoxic Snake Envenomation

Aravinda C.L.<sup>1</sup>, Nagabhushana S.<sup>1</sup>, Ranganatha M.<sup>1</sup>, Virupakshappa V.<sup>2</sup>

### Author's Affiliation:

<sup>1</sup>Associate Professor, <sup>2</sup>Professor and HOD, Dept of Medicine, Shimoga Institute of Medical Sciences, Shimoga, Karnataka 577201, India.

### Corresponding Author:

**Nagabhushana Seetharama**, Associate Professor, Dept of Medicine, Shimoga Institute of Medical Sciences, Shimoga, Karnataka 577201, India.  
E-mail: [sn\\_bhushan@yahoo.co.in](mailto:sn_bhushan@yahoo.co.in)

Received on 06.10.2017,  
Accepted on 23.10.2017

### Abstract

*Background:* India is estimated to have the highest snakebite mortality in the world. The hitch with determining the optimum ASV dose is that the quantity of venom injected at a bite is very variable. There are very few studies to determine the effective dose of ASV. But recent studies have found that low dose ASV is as good as or even better (lesser complications) than high dose ASV. Despite evidence for smaller doses from evidence-based medicine, most centers are still using large doses. There is a need for investigating in this area to know the effective dose of ASV in management of snake bite patients resulted in taking up of present study. *Material and Methods:* this study carried out in Medicine department, SIMS, Shimoga for 6 months from January 2017 to June 2017. 100 snakebite patients with haemostatic abnormality admitted to Mc Gann Hospital. A detailed history was taken in all the patients and a thorough physical examination was done. CBC, RFT, LFT, BT,CT, PT, INR, ECG, is done. The two study groups are 50 consecutive patients formed Group I (Conventional high dose regime (100ml) group). 50 consecutive patients formed Group II (Low dose regimen (30ml) group). *Results:* The mean age was 37.67 ( $\pm 4.56$ ) years. With male to female ratio being 1.3:1. The mean Snakebite to ASV given time was 14.5 hours. Average CT (at presentation), Group 1- 22.6 $\pm$ 7.59 mins. Group 2- 29.47  $\pm$ 5.59 mins. ASV dose required, Group 1- 325  $\pm$ 183 ml. Group 2-175.75  $\pm$ 87.4 ml. Time lapse for CT normalization, Group 1- 24.97 $\pm$ 5.58 hrs. Group 2- 14.93 $\pm$ 4.49 hrs. About 20-25% of patients developed acute renal failure [11 (22%) and 5 (10%) patients in groups I and II respectively]. Number of patients died in Group 1-5 (10%). Group 2-4 (8%). *Conclusion:* The observation that very low dose of ASV is adequate to save lives of victims of poisonous snake bites with early hospitalization and good supportive management. This will definitely decrease economic burden on the society.

**Keywords:** Venom; Snakebite.

### Introduction

India is a country known to the western population as a country of snake charmers. India is estimated to have the highest snakebite mortality in the world.

Snakebite is a major problem in rural India with more than 2 lakh snakebites being reported in India annually of which 35,000-50,000 die [1-2]. A nationally representative study of 123,000 deaths from 6,671

randomly selected areas in 2001-03 conducted by Mohapatra B et. al. revealed an annual age-standardized rate of 4.1/100,000. This proportion represents about 45,900 annual snakebite deaths nationally (99% CI 40,900 to 50,900) [3].

The estimated death in India is 50,000/yr, an underestimate because of lack of proper registration of snake bite. Most of the fatalities are due to the victim not reaching the hospital in time where definite treatment can be administered. In addition community

is also not well informed about the occupational risks and simple measures which can prevent the bite. It continues to adopt harmful first aid practices such as tourniquets, cutting and suction, etc. Studies reveal that primary care doctors do not treat snakebite patients mainly due to lack of confidence [4]. At the secondary and tertiary care level, multiple protocols are being followed for polyvalent anti-snake venom (ASV) administration, predominantly based on western textbooks.

The hitch with determining the optimum ASV dose is that the quantity of venom injected at a bite is very variable, depending on the species and size of the snake, the mechanical efficiency of the bite, whether one or two fangs penetrated the skin and whether there were repeated strikes. A proportion of bites by venomous snakes do not result in the injection of sufficient venom to cause clinical effects [5]. About 50% of bites by Malayan pit vipers and Russell's vipers, 30% of bites by cobras and 5-10% of bites by saw-scaled vipers do not result in any symptoms or signs of envenoming [6]. Also, neutralization by antivenom must occur almost immediately after venom enters the circulation to significantly impact on recovery time of the coagulopathy due to envenomation [7].

ASV used in India is polyvalent and contains antivenin against cobra, Russell's viper, krait, saw scaled viper. Each vial of ASV containing 10 ml of antivenin costs about 500 rupees. To the rural poor patients from agricultural background who are the most common victims of snake bite it is a huge burden. Another problem with ASV is that, it being an animal serum product some patients develop hypersensitivity reactions to it.

The infrastructure of the medical profession in India is mal-distributed in such a manner that it is very difficult to protect this poor rural population against the snake bite. Scientifically and ethically we, the doctors can not treat the patients of snake bite properly.

In response, Government of India, Health and Family Welfare Department has prepared a National Snakebite Management Protocol [8] to provide doctors and lay people with the best possible, evidence-based approach to deal with this problem in country.

There are very few studies to determine the effective dose of ASV. Previously many tens of vials of ASV were used in the treatment of snake bite- sometimes being given direct IV. But recent studies have found that low dose ASV is as good as or even better (lesser complications) than high dose ASV [9-12]. Despite evidence for smaller doses from evidence-based

medicine, most centers are still using large doses.

There is a need for investigating in this area to know the effective dose of ASV in management of snake bite patients resulted in taking up of present study.

## Materials and Methods

This study was carried out in Mc Gann Hospital, Shimoga. The material of study consisted of 100 consecutive patients of snakebite patients with haemostatic abnormality admitted to Mc Gann Hospital from January 2017 to June 2017 over 6 months.

A Prospective study consisting of 100 snakebite patients with haemostatic abnormality was undertaken to study the efficacy of low dose anti snake venom over conventional regimen in the treatment of patients with poisonous snake bites.

### Inclusion Criteria

A total of 100 snakebite patients with haemostatic abnormality presented to our hospital between January 2017 and June 2017, of patients who were aged  $\geq 15$  yrs with history of snakebite within the previous 24 hrs and had signs and symptoms of systemic envenomation which included hemostatic abnormalities in the form of spontaneous GI bleeding, uncontrolled bleeding from external wounds, prolonged CT ( $>10$  min), PT (INR $>1.5$ ), aPTT ( $>2x$  control), shock (requiring ionotropic support), cardiac arrhythmia, abnormal ECG, Acute renal failure evidenced by oliguria, anuria, rising creatinine ( $>1.5$  mg/dl), albuminuria, hemoglobinuria / myoglobinuria, dark brown urine were found eligible for the study

*Patient allocation:* There are four medical units in our hospital. Two Units A and B were chosen for trial of high and low-dose regimes. The two study groups, as follows, were formed.

50 consecutive patients formed Group I (Conventional high dose regime group).

50 consecutive patients formed Group II (Low dose regimen group).

ASV was administered as mentioned in Table 3. Groups I and II received regimens I and II respectively.

Patients with ARF were managed with fluid challenge and hemodialysis, wherever indicated.

The study was approved by the Institute Ethics Committee and informed consent was obtained from each patient.

*Exclusion Criteria*

1. No signs of envenomation
2. No signs of haemostatic abnormality
3. Known cardiac, hepatic and renal disorder
4. Presentation after 24hrs

A detailed history was taken in all the patients and a thorough physical examination was done as per the proforma.

*Investigations are as Follows*

- Blood routine (Hemoglobin percentage, Total count, differential count, Erythrocyte sedimentation rate).
- Bleeding time, clotting time repeated at intervals
- PT, APTT and INR
- Random blood sugar (Fasting blood sugar/Post prandial blood sugar was done whenever necessary), blood urea, serum creatinine.
- Urine routine analysis (Sugar, Albumin and microscopy)
- ECG

*Special Investigations*

- a. Chest X-ray / screening (whenever required)
- b. Serum electrolytes (whenever required)

**Statistical Analysis***Observations*

100 consecutive patients of snake bite with haemostatic abnormality admitted to Mc Gann Hospital, Shimoga from January 2017 to June 2017 were studied. They were given treatment according to Regimen I-50 patients, Regimen II-50 Patients. The following are the observations made from this study.

*Age Distribution*

The mean age of the studied patients was  $36 \pm 5$  years and  $39 \pm 6$  years in groups I and II respectively. Most of the patients were males and were agricultural laborers. All our patients were from rural areas. Approximately 40% had the bite on one of the lower limbs, 30% had bite in upper limbs.

**Table 1:** Showing age distribution

Age group (in years)	Regimen I (50) No. of patients (Percentage)	Regimen II (50) No. of patients (Percentage)
Less than 30	4(8)	3(6)
31-40	20(40)	18(36)
41-50	12(24)	13(26)
51-60	5(10)	4(8)
61-70	4(8)	4(8)
71-80	3(6)	5(10)
More than 81	2(4)	3(6)
Total	50(100)	50(100)

**Table 2:** Showing Sex distribution

Sex	Regimen I (50) No. of patients (Percentage)	Regimen II (50) No. of patients (Percentage)
Male	33(66)	35(70)
Female	17(34)	15(30)

**Table 3:** Different regimens of ASV used in the study

Regimens	Loading Dose	Followed By	End-Point
Regimen I (Conventional High Dose Regimen)	100 ml	50 ml Q 6 Hours Till CT normalizes	Till CT normalizes
Regimen II (Low Dose Regimen)	30 ml	30 ml infusion over 6 hours process repeated till CT normal, followed by 30 ml over 24 hours	Till 24 hours after CT normalizes

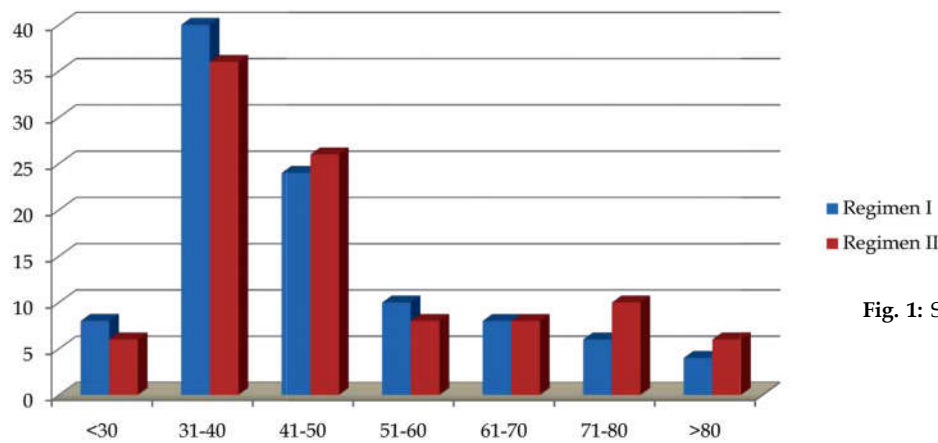


**Table 4:** ASV Therapy

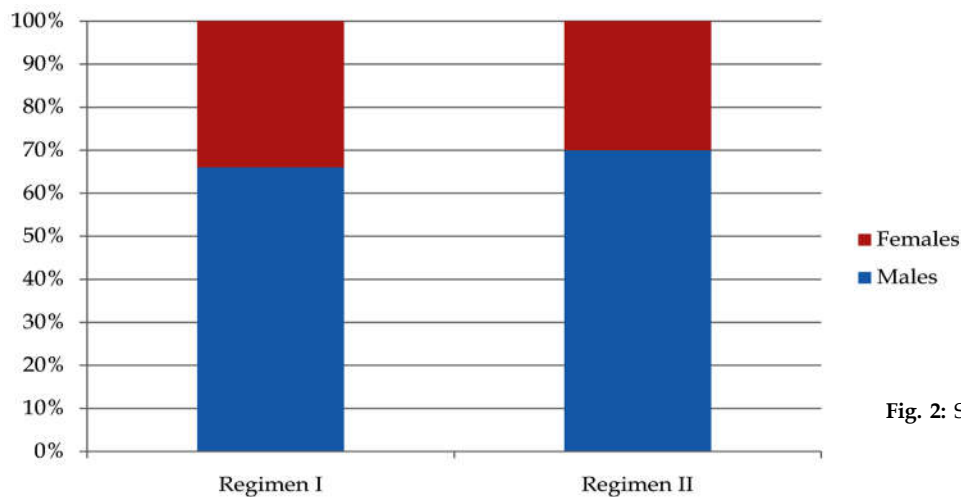
Risk Factors	Regime I		Regime II	
Average CT (at presentation)	22.6 ±7.59mins		29.47 ±5.59mins.	
ASV dose required	325 ± 183 ml		175.75 ± 87.4 ml	
Adverse reaction	12(24%)		15 (30%)	
Time taken for CT normalization	24.97 ±5.58 hrs		14.93 ± 4.49 hrs	
Recurrence	8 (16%)		6 (12%)	
Outcome	Cured 45(90%)	Death 5(10%)	Cured 46 (92%)	Death 4 (8%)

**Table 5:** Characteristics of patients with mild envenomation (clotting time 11-20 min) and severe envenomation (clotting time >20 min)

	Mild envenomation		Severe envenomation	
	Regimen I	Regimen II	Regimen I	Regimen II
No. of patients	30	32	20	18
Snake Bite to ASV given time (hours)	13.2±12.2 hours	14.9±13.44 hours	10.55±13.67 hours	13.5±11.46 hours
Mean CT (min)	15.48 ± 5.3	17.33 ± 3.2	24±5.56	27±5.47
Average dose of ASV (ml)	267±65.5 ml	154±74.8 ml	394±58.9 ml	235±94.9 ml
Time taken to CT normalization(hours)	14.56±5.5	11.76±2.4	22.76±5.7	16.67±5.8
Relapse of bleeding after treatment	7	2	5	3
No. with ARF	4	1	7	4
No. with DIC	5	4	8	7
Duration of stay (days)	5	4	8	7



**Fig. 1:** Showing age distribution



**Fig. 2:** Showing Sex distribution

The male to female ratio was 1.3:1. More number of males are affected by snake bite in our study compared to females.

The average requirement of ASV, time lapse for CT normalization, incidence of adverse reaction and recurrence of coagulation dysfunction in various groups are shown in below table.

Most of patients had local swelling (swelling at the site of bite). 55% patients had presented with signs of mild envenomation, whereas 45% patients presented with signs of severe envenomation (incoagulable blood).

Adverse ASV reactions were mainly in form of itching, urticaria, and erythema; and responded to antihistaminics and hydrocortisone. Ten patients, however, developed hypotension and required adrenaline.

Characteristics of patients with mild and severe envenomation are shown elaborately in Table

The mean Snake bite to ASV given time was 14.5 hours; only one-third of patients presented within six hours of bite. The mean bite to needle time was 13.2 hours and 14.9 hours in groups I and II respectively. 30% and 36% of patients in groups I and II respectively reached the hospital after 24 hours of snakebite.

About 20-25% of patients developed acute renal failure [11 (22%) and 5 (10%) patients in groups I and II respectively].

Of the total of 100 patients enrolled in the study, 9 (18%) patients succumbed to various complications. causes contributing to death were DIC, ARF and septicemia.

## Discussion

The study is aimed at knowing usefulness of lower dose regimen over conventional regimen of ASV.

The observations made in 100 case of snake bite with haemostatic abnormality admitted to the Mc Gann Hospital Shimoga from January 2017 to June 2017 are discussed here and the results have been compared with other studies.

### Age

The age of patients in this study ranged from 25 years to 89 years with maximum number of patients in the age group 31 to 40 years (38%). Mean age 37.67 ( $\pm$  4.56) years. This is consistent with findings of

AM Cherian et al [13] where Mean age was 35.72  $\pm$  14.42 years.

In most of the Indian studies commonly affected patients are rural agricultural labourers it is consistent with our study (AM Cherian et al [13], J Srimannarayana et al [10]).

### Sex

There were 68 males (68%) and 32 females (32%) in the present study. The male to female ratio was 1.3:1. This findings is consistent with that of AM Cherian et al [13] - males 70%, females (30%); V Paul et al [9] - 75% male, 25% female.

The mean bite to needle time of our patients was 14.5 hours; only 38% of patients presented within six hours after bite. This was in contrast to the studies by Thomas et al [14] and Tariang et al [15]. Where majority of patients reached hospital within six hours. This explains higher requirement of ASV in the current study; experimentally delay in administering antivenom results in steep increase of median effective neutralizing dose [16]. Further, none of the patients in Tariang's study [15] groups had incoagulable blood; whereas almost 60% of our patients had incoagulable blood at presentation, thus requiring more of ASV.

In a study by Paul V et al [9], authors found no additional advantage of giving fixed 12 vials (120 ml) of ASV over six vials (60 ml) of ASV. However all the cases included in that study were those who arrived within 24 hours of bite, whereas 36 (66%) of our patients arrived after 24 hours of bite. However, ours being a tertiary referral center, we had a higher load of critically ill patients and thus had higher mean requirement of ASV.

However, the average dose of ASV required in Regimens II in our study was significantly lower than that required in Regimen I. The lower requirement in regimens II was probably due to the delivery of ASV by continuous infusion and thus more accurate titration of dose, as opposed to delivery by multiple bolus doses in Regimen I.

Repeated high doses of ASV to restore the clotting time to normal do not seem to be necessary to reduce the mortality and a smaller dose sufficient to bring down the clotting time seems to be adequate. As evidenced in this study. The body's detoxifying system will bring down the clotting time eventually though it may take a slightly longer time.

In patients with *mild envenomation*, Regimen II was found as effective as the other regimens and at the

**Table 6:** Average dose of ASV and modes of administration in various studies

Study	Protocol	ASV Required
Our Study	Regimen I (Conventional High Dose Regimen)	325 ± 183 ml
Vijeth et al (2000), Pondicherry <sup>17</sup>	Regimen II (Low Dose Regimen)	175.75 ± 87.4 ml
	Intermittent bolus doses: Initial - 100 ml	179.2 ml
Thomas and Jacob (1985), Kerala <sup>14</sup>	Repeat - 50 ml q 6 hr till CT corrects to normal	
	Traditional schedule: 40 ml in 1st hour, 40 ml in next 2 hrs, 40 ml in next 3 hrs, 30 ml every 3 hours.	153 ml
Tariang et al(1999), Vellore <sup>15</sup>	Modified Schedule: 20 ml in 1st hour, 20 ml over 2 hrs, 20 ml every 3 hrs till CT normalizes. (After CT normalizes, 20 ml in 5% dextrose over 24 hours).	79ml
	Continuous iv infusion: High dose: 20 ml in 100 ml 5% dextrose over 1 hr, followed by 20 ml in 100 ml 5% dextrose over 4 hrs, till CT normalizes, and then, 2 vials over 24 hours	89 ml
J Srimannarayana et al <sup>10</sup>	Low dose: 20 ml over 1 hour, followed by 10 ml in 100 ml of 5% dextrose over 4 hours till CT normalizes, then 10 ml in 100 ml 5% dextrose over 24 hours.	47ml
	Conventional High Dose Regimen) 100 ml (Low Dose Regimen) 30ml	376 ± 205.83 ml
Paul V et al <sup>9</sup>	High dose group	197.67 ± 76.4 ml
	Low dose group	120 ml 60 ml

same time it had comparatively lesser requirement of ASV at 154±74.8 ml.

In patients with *severe envenomation*, Regimen II with requirement of ASV at 235±94.9 ml, where as Regimen I which required 394±58.9 ml. Regimen II appear to be significantly economical regimens as compared to Regimen I (standard regimen).

Following these new regimens, the amount of ASV saved with Regimen II in our study was as much as 100 ml to 200 ml in mild and severe envenomation. Further, giving extra dose of ASV after CT normalization reduced recurrence of coagulation dysfunction. In the low-dose group there were five deaths giving a mortality rate of 8%, which is consistent with study by V Paul et al [9] which showed mortality of 10%.

Following the prescribed regimes suggested in this study, the requirement of ASV will become automatically low in mild and severe envenomation, even though the mean requirement of ASV may be high due to more number of severe envenomation cases, as in this study. May be due to late presentation or referral of cases to our tertiary hospital.

The mean dose requirement in mild and severe cases with the prescribed regimes concluded from this study was found to be not much different from that required by Bhat RN et al [18] study, Slightly higher mean dose requirement in mild and moderate envenomation in our study was due to extra dose of ASV given after

correction of CT. this is recommend to prevent relapse of coagulation dysfunction. Since there are several studies reporting the recurrence of coagulation defect as a significant problem [17,19,20].

Since more than 8 years, there has been a growing scarcity of ASV due to various reasons (including animal rights protests and introduction of Drug price control by Govt of India) and there are periods when ASV is not available at all in the market. In the government sector, there are often logistic difficulties in procuring ASV due to stringent tender and quotation rules or shortage of funds.

However, because of the high cost and limited availability of ASV and reports of patients with severe envenomation recovering without its use, there was a change in dosage protocols from high to low. The antivenin is effective only if given early enough to neutralize the venom in the circulation, Therefore, the use of large doses late in the course is unlikely to be effective [21].

## Conclusion

The observation that very low dose of ASV adequate to save lives of victims of poisonous snake bites with early hospitalization and good supportive management. It is of very much importance in developing countries like India. While there was no

additional advantage in following a high-dose regime for snake bite cases, there was considerable financial gain by following the low-dose regime. It is a win-win situation for both patients and the institution/nation.

### Acknowledgement

I greatly appreciate the support given by Medical Research Unit (MRU) of Shimoga Institute of Medical sciences. Shimoga.

### References

1. Bawaskar H.S., Snake venoms and antivenoms: critical supply issues. *J Assoc Phys India* 2004;52: 11-13.
2. Malhotra P. et al. Fatal acute disseminated encephalomyelitis following treated snake bite in India. *EMJ* 2005;22:308-309.
3. Mohapatra B, Warrell DA, Suraweera W, Bhatia P, Dhingra N, et al. Snakebite Mortality in India: A Nationally Representative Mortality Survey. *PLoS Negl Trop Dis* 2011;5(4):e1018. doi:10.1371/journal.pntd.0001018.
4. Simpson ID. A study of current knowledge base in treating snake bite among doctors in high risk countries of India and Pakistan: does snake bite treatment training reflect local requirements? *Trans R Soc Trop Med Hyg.* 2008;102:1108-14.
5. B. Kalyan Kumar et al. Antisnake venom serum. *International Journal on Pharmaceutical and Biomedical Research (IJPBR)* 2010;1:76-89.
6. Shashi Kiran, Senthilnathan TA. Management of snake envenimation. *Update in Anaesthesia* 2003:16.
7. GK Isbister et al. Failure of antivenom to improve recovery in Australian snakebite coagulopathy. *QJ Med* 2009;102:563-568.
8. National snakebite management protocol, India. (2008). [online] Available at [www://mohfw.nic.in](http://www.mohfw.nic.in) (Directorate General of Health and Family Welfare, Ministry of Health and Family Welfare, India).
9. Paul V, Pratibha S, Prahlad KA, Earali J, Francis S, Lewis F. High dose anti-snake venom versus low dose anti snake venom in the treatment of poisonous snake bites- a critical study. *J Assoc Phys India* 2004; 52:14-17.
10. Srimannarayana J, Dutta TK, Sahai A, Badrinath S., Rational use of anti-snake venom: Trial of various regimens in Hemotoxic Snake envenomation. *J Assoc Phys India* 2004;52:788-793.
11. Tariang DD, Philip PJ, Alexander G, Macaden S, Jeyaseelan L., Peter JV, Cherian AM. Randomised control trial on the effective dose of anti-snake venom in case snakebite with systemic envenomation. *J Assoc Phys India* 1999;47:369-371.
12. Agarwal R, Aggarwal AN et al. Low dose of snake anti venom is as effective as high dose in patients with severe neurotoxic snake envenoming. *EMJ* 2005;22:397-399.
13. AM Cherian et al. High or Low- A Trial of Low Dose Anti Snake Venom in the Treatment of Poisonous Snakebites. *Journal of the association of physicians of India.* June 2013;61:387-396.
14. Thomas PP, Jacob J. Randomized trial of antivenom in snake envenomation with prolonged clotting time. *Brit Med J* 1985;291:177-78.
15. Tariang DD, Philip PJ, Alexander G, Macaden S, Jeyaseelan L, Peter JV, Cherian AM. Randomized controlled trial on the effective dose of antisnake venom in cases of snakebite with systemic envenomation. *J Assoc Phys India* 1999;47:369-71.
16. Progress in the characterization of venoms and standardization of antivenoms. WHO Offset Publication. 1981:58.
17. Vijeth SR, Dutta TK, Shahapurkar J, Sahai A. Dose and frequency of antisnake venom injection in the treatment of *Echis carinatus* (saw-scaled viper) bite. *J Assoc Phys India* 2000;48:187-91.
18. Bhat RN. Viperine snakebite poisoning in Jammu. *J Indian Med Assoc* 1974;63:383-92.
19. Ho M. Clinical significance of venom antigen levels in patients envenomed by the Malayan pit viper (*Calloselosma rhodostoma*). *Am J Trop Med Hyg* 1986;35:579-87.
20. Reid HA, Chan KE, Thean PC. Prolonged coagulation defect (defibrination syndrome) in Malayan pit viper bite. *Lancet* 1963;1:621-6.
21. Agarwal R, Aggarwal AN, Gupta D, Behera D, Jindal SK. Short report on "Low dose of snake antivenom is as effective as high dose in patients with severe neurotoxic snake envenoming. *Emerg Med J.* 2005;22:397-9.

## Management and outcome of Acute Kidney Injury at a Tertiary Care Hospital

Cijo John<sup>1</sup>, Selin Abraham<sup>1</sup>

---

### Author's Affiliation:

<sup>1</sup>Assistant Professor, Department of Medicine, Mount Zion Medical College, Chayalode, Adoor Pathanamthitta, Enadimangalam, Kerala 691556, India.

### Corresponding Author:

**Selin Abraham**  
Assistant Professor,  
Department of Medicine,  
Mount Zion Medical College,  
Chayalode, Adoor  
Pathanamthitta,  
Enadimangalam,  
Kerala 691556, India.  
E-mail:  
sowmyatshassan@gmail.com

Received on 10.06.2017,

Accepted on 20.06.2017

### Abstract

*Introduction:* The most frequent causes of postrenal AKI in the elderly include benign prostatic hypertrophy (BPH) or prostate cancer, retroperitoneal adenopathy or malignancies, pelvic neoplasms, and neurogenic bladder. Although BPH and prostate cancer are common in older men, they cause obstruction in only a minority of cases. In elderly women, pelvic and retroperitoneal malignancies are the most frequent causes of postrenal AKI. *Methodology:* This study was conducted on 200 admitted patients who presented with Acute Kidney Injury or developed Acute Kidney Injury during the hospital stay in the Department of Medicine. The symptoms, signs and basic lab data like Routine Blood Examination for Hb, TC, DC, ESR & Platelet count; Renal function tests, Liver function tests, Serum Electrolytes & Routine Urine examination, was noted at the time of admission, during the course of hospital stay and at the date of discharge. Specific investigations like USG Abdomen, Renal Biopsy, Arterial Blood Gas analysis was done accordingly to analyze the etiology. *Results:* Pre renal conditions predominate as the cause for AKI. Post renal causes account for only 2.5% of the total. People above the age group of 50yrs was at an increased risk for the development of AKI. *Conclusion:* Septic AKI was the commonest cause of increased mortality followed by leptospirosis.

**Keywords:** AKI; Outcome; Leptospirosis.

---

### Introduction

AKI can also develop from acute or rapidly progressive glomerulonephritis. Timely diagnosis and treatment of these conditions is critical to preserve renal function and avoid life-threatening complications. Diffuse proliferative forms of glomerulonephritis can be associated with infections and generally carry a good prognosis in the elderly and in the young [1,2]. Rapidly progressive (crescentic) glomerulonephritis is a fulminant presentation of glomerular disease that will lead to renal failure over days to weeks if left untreated. Evidence suggests that rapidly progressive glomerulonephritis may be more common among the elderly and carries a poorer prognosis [3]. Clinically, patients often present with AKI, hypertension,

hematuria, and proteinuria. Characteristically, the urinary sediment demonstrates dysmorphic red blood cells and red blood cell casts. Serologic studies including complement levels, antinuclear antibodies (ANA), antineutrophil cytoplasmic antibodies (ANCA), antiglomerular basement membrane antibodies, cryoglobulin levels, and hepatitis B and C antibodies can be useful in suggesting the cause, although kidney biopsy is nearly universally required for specific diagnosis. Treatment, including high-dose glucocorticoids, immuno-suppressive therapy and plasmapheresis, will be dependent on the specific cause. Despite the potential for treatment associated toxicities, case series have demonstrated that elderly patients with limited comorbidities may tolerate and respond well to therapy [4].

Postrenal or obstructive AKI is more common in the aged than in the young, accounting for 9% to 30%

of cases [5]. Postrenal AKI can be categorized as affecting either the upper urinary tract (proximal to the bladder) or lower urinary tract (obstruction occurring at the bladder outlet or urethra). Obstruction of the lower tract will affect both kidneys and diminish renal function. In contrast, unilateral upper tract obstructing processes may cause renal colic and unilateral hydronephrosis, but will not cause deterioration in renal function if the contralateral kidney can compensate. However, if the obstruction is bilateral, is of a unilateral functioning kidney, or if there is significant underlying chronic kidney disease, upper tract obstruction can also cause AKI.

The most frequent causes of postrenal AKI in the elderly include benign prostatic hypertrophy (BPH) or prostate cancer, retroperitoneal adenopathy or malignancies, pelvic neoplasms, and neurogenic bladder. Although BPH and prostate cancer are common in older men, they cause obstruction in only a minority of cases. In elderly women, pelvic and retroperitoneal malignancies are the most frequent causes of postrenal AKI.

Postrenal AKI may present with either complete or partial obstruction. Complete obstruction is characterized by anuria. The patient may also report flank and abdominal pain or suprapubic fullness. In contrast, the patient with partial obstruction may remain completely asymptomatic or may report similar pain symptoms, as well as voiding complaints including frequency, urgency, hesitancy, hematuria, and nocturia. Urine output can be variable, ranging from oliguria to polyuria, or fluctuating between the two [6].

Due to its increased incidence in the elderly and varying presentation, the clinician must maintain a high index of suspicion for postrenal AKI. The diagnosis should especially be considered in patients with BPH or lower urinary tract symptoms, diabetes, kidney stones, abdominal or pelvic malignancies, surgeries or radiation, retroperitoneal adenopathy or neoplasms, and medication use associated with urinary retention. Lower tract obstruction is diagnosed by confirmation of urinary retention using ultrasonographic bladder scans or placement of a bladder catheter. An elevated residual bladder volume (>100–150 mL) after voiding is highly suggestive of postrenal AKI, although, some elderly patients may suffer from chronic urinary retention with elevation in the postvoid residual bladder volume in the absence of kidney dysfunction [7]. Radiographic workup for upper tract obstruction usually begins with ultrasound imaging, which is sensitive and specific in detecting obstruction [8,9]. However,

ultrasonography may appear normal in patients presenting with early obstruction or with retroperitoneal processes encasing the kidneys and ureters, preventing ureteral dilation. CT can be valuable in determining the cause and level of obstruction if ultrasound fails to identify the lesion. Together, ultrasound, abdominal plain films, and CT scanning are diagnostic in most cases.

Intravenous pyelography has been supplanted by CT imaging and is now only rarely required. Antegrade or retrograde pyelography, however, can be valuable in identifying the site and cause of obstruction, and provides an opportunity for therapeutic intervention. Laboratory findings are nonspecific in postrenal AKI often mimicking prerenal AKI in the early phase and intrinsic AKI later.

Treatment of postrenal AKI consists of the rapid detection and relief of obstruction. This can be accomplished by placement of a bladder catheter in lower tract disease or ureteral stents or percutaneous nephrostomy tubes for upper tract disease. A brisk postobstructive diuresis frequently ensues due to water and sodium reabsorptive deficits as well as an osmotic diuresis attributable to previously retained solutes including urea. Careful monitoring of the patient's volume status and electrolytes is essential to avoid the development of volume depletion or serious electrolyte disturbances. Although use of intravenous fluids may be required, it is important to avoid overly aggressive fluid replacement that can drive further diuresis. If the obstruction has been quickly diagnosed and reversed, renal function will improve. However, in patients with a longer duration and higher grade of obstruction, renal functional recovery may be delayed, incomplete, or absent. Brisk urine output following correction of the obstruction does not always correlate with renal recovery and hence close laboratory monitoring remains necessary.

## Methodology

### *Definition of the Study*

This study has utilized the classifications called the RIFLE and AKIN. The following definitions have been utilized for the study.

*Oliguria:* Refers to a 24hr urine output <400ml.

*Anuria:* Complete absence of urine formation (<100ml/d).

*Nonoliguria:* Refers to urine output >400ml/d in patients with acute or chronic azotemia.

Calculation of **GFR** by Cockcroft-Gault formula

$\text{CrCl (ml/min)} = (140 - \text{age (years)} \times \text{weight (kg)} \times (0.85 \text{ if female}) / 72 \times \text{S.Cr (mg/dL)}$ .

*Add:* Acute diarrheal disease including Acute gastroenteritis.

*Diaki:* Drug induced Acute Kidney Injury including Aminoglycosides, Cisplatin, amphotericin B, vancomycin and others excluding NSAID's.

*CIN:* Contrast induced nephropathy following iodinated contrast agents.

*MM/AKI:* Multiple myeloma associated acute kidney injury.

*CVA/AKI:* Cerebro vascular accidents leading to poor intake and pre-renal failure.

*NSAID/AKI:* Non-steroidal anti inflammatory drug induced acute kidney injury.

*HUS/TTP:* Hemolytic uremic syndrome/ thrombotic thrombocytopenic purpura-characterized by history of recent GI infection or use of calcineurin inhibitors with the presence of schistocytes on peripheral blood smear, elevated LDH, anemia and thrombocytopenia.

#### Study Population

This study was conducted on 200 admitted patients who presented with Acute Kidney Injury or developed Acute Kidney Injury during the hospital stay in the Department of Medicine.

*Study Period:* One year.

*Study Design:* Prospective observational study.

*Data Collection Tool:* Structured interview schedule.

#### Study Details

Each case was individually seen and data was collected according to the prepared performa, after obtaining informed consent for participation in the study.

The symptoms, signs and basic lab data like Routine Blood Examination for Hb, TC, DC, ESR & Platelet count; Renal function tests, Liver function tests, Serum Electrolytes & Routine Urine examination, was noted at the time of admission, during the course of hospital stay and at the date of discharge. Specific investigations like USG Abdomen, Renal Biopsy, Arterial Blood Gas analysis was done accordingly to analyse the etiology

Conservative management in the form of removal of precipitating factors for prerenal failure, fluid restriction and use of renoprotective drugs like ACE inhibitor and interventional treatment in the form of Haemo-dialysis or Peritoneal-dialysis was instituted as needed.

Complications if any like sepsis and worsening of renal reserve was studied according to clinical, radiological and biochemical evidences. Patients was followed up at 3 weeks, 3 months and 6 months after discharge with S. Creatinine, B.Urea, Urine examination results.

## Results

This study consisted of 112 males and 88 females. Males contributing 56% compared to 44% of females.

**Table 1:** Gender distribution

Gender	Frequency	Percent
Male	112	56
Female	88	44
Total	200	100

**Table 2:** USG Abdomen

USG Abdomen	Frequency	Percent
Normal	171	85.5
Bladder Stone	1	0.5
BPH	3	1.5
Cystitis	5	2.5
HM+	18	9
Pyelonephritis	1	0.5
Ure. Stone	1	0.5
Total	200	100

**Table 3:** Diagnosis and conservative treatment

Diagnosis	Treatment: Conservative		Total
	No	Yes	
ADD AKI	3 6.30%	31 20.40%	34 17.00%
AGN AKI	-	19 12.50%	19 9.50%
CIN	2 4.20%	10 6.60%	12 6.00%
CVA AKI	-	9 5.90%	9 4.50%
DIAKI	2 4.20%	10 6.60%	12 6.00%
HUS/TTP	-	9 5.90%	9 4.50%
Lepto/AKI	12 25.00%	22 14.50%	34 17.00%
LVF AKI	-	10 6.60%	10 5.00%
MM AKI	-	5 3.30%	5 2.50%
NSAID AKI	6 12.50%	19 12.50%	25 12.50%
Obst. AKI	2 4.20%	3 2.00%	5 2.50%
Sepsis AKI	17 35.40%	5 3.30%	22 11.00%
Viper Bite	4 8.30%		4 2.00%
Total	48	152	200

Chi Square: 71.389; P &lt; 0.001

**Table 4:** Diagnosis and hemodialysis

Diagnosis	Treatment: HD		Total
	No	Yes	
ADD AKI	31 19.70%	3 7.00%	34 17.00%
AGN AKI	19 12.10%		19 9.50%
CIN	10 6.40%	2 4.70%	12 6.00%
CVA AKI	9 5.70%		9 4.50%
DIAKI	10 6.40%	2 4.70%	12 6.00%
HUS/TTP	9 5.70%		9 4.50%
Lepto/AKI	22 14.00%	12 27.90%	34 17.00%
LVF AKI	10 6.40%		10 5.00%
MM AKI	5 3.20%		5 2.50%
NSAID AKI	19 12.10%	6 14.00%	25 12.50%
Obst. AKI	3 1.90%	2 4.70%	5 2.50%
Sepsis AKI	9 5.70%	13 30.20%	22 11.00%
Viper Bite	1 0.60%	3 7.00%	4 2.00%
Total	157	43	200

Chi Square: 47.954; P &lt; 0.001



**Table 5:** Outcome and Diagnosis

Diagnosis	Cured	Outcome Relieved	Dead	Total
ADD AKI	34 22.50%	-	-	34 17.00%
AGN AKI	9 6.00%	10 28.60%	-	19 9.50%
CIN	10 6.60%	2 5.70%	-	12 6.00%
CVA AKI	9 6.00%	-	-	9 4.50%
DIAKI	11 7.30%	1 2.90%	-	12 6.00%
HUS/TTP	-	9 25.70%	-	9 4.50%
Lepto/AKI	27 17.90%	4 11.40%	3 21.40%	34 17.00%
LVF AKI	10 6.60%	-	-	10 5.00%
MM AKI	-	5 14.30%	-	5 2.50%
NSAID AKI	23 15.20%	2 5.70%	-	25 12.50%
Obst. AKI	3 2.00%	2 5.70%	-	5 2.50%
Sepsis AKI	12 7.90%	-	10 71.40%	22 11.00%
Viper Bite	3 2.00%	-	1 7.10%	4 2.00%
Total	151	35	14	200

Chi Square: 163.616; P < 0.001

## Discussion

Out of the 200 patients studied 152 were treated conservatively by fluid management and antibiotics. Of the 152 patients managed conservatively, 20.40% were in the acute diarrheal group, 14.5% were leptospirosis induced AKI, 12.5% were NSAID induced, 12.5% were due to acute glomerulonephritis (P=<0.001). The higher incidence of conservative management in ADD/AKI was that the patients were identified early and appropriate treatment was instituted and that resulted in the reversal of AKI. Similarly patients with history and clinical features suggestive of leptospirosis were aggressively managed resulting in the regression of the disease. Only patients presenting late were at an increased risk of progression of the disease.

NSAID intake was more common in the elderly and those that were managed conservatively were largely devoid of confounding factors that lead to a rapid progression of symptoms. NSAID intake led to dialysis in patients whose renal function was already compromised. Almost all cases of acute post streptococcal glomerulonephritis were managed

conservatively and improved. Out of the 200 patients 48 were subjected to Renal replacement therapy (RRT). Most patients subjected for hemodialysis were having septic AKI (30.20%), lepto/AKI (27.90%) and viper bite (7%). (P=<0.001).

Outcome profile was studied according to age group <50yrs and >50yrs, gender and to the mode of treatment given to the patient. Of the 200 patients 151 were cured (75.50%). 35 (17.50%) were relieved of there symptoms but had to undergo more than one hemodialysis sessions. 14 (7%) had died during the study period due to complications of AKI.

The major cause of death in this study was sepsis induced AKI. 10 patients (71.40%) out of the the 14 died (P=<0.001). Of the 22 patients studied with sepsis induced AKI, 13 patients underwent hemodialysis (63.6%)(P=<0.05). This finding was in accordance with the study done by the BEST investigators where they showed a mortality rate of 70.2% in hospitals. The cause of such a high rate of mortality was due to ischaemia-reperfusion injury, direct inflammation injury, coagulation, endothelial dysfunction and apoptosis [10]. Sepsis induced AKI did not respect gender nor age. Mortality is certainly higher among

people more than 50 yrs of age due to preexisting conditions like diabetes, low serum albumin, atherosclerosis which are more common in the elderly.

Another cause of increased mortality in this study is leptospirosis induced AKI. 34 patients presented with leptospirosis and 3 (21.40%) died ( $P < 0.001$ ). 12 patients required dialysis (27.90%) ( $P < 0.05$ ). Leptospirosis is endemic to Alappuzha and is a major cause of mortality in young as well as older persons. Early recognition and timely action usually saves the person.

Out of the 43 persons requiring dialysis, 3(7%) were bitten by Russels viper, which is a common poisonous snake found in close proximity to human dwelling and in farm lands. One person died of complications (9.10%) ( $P < 0.05$ ). There were 5 patients of multiple myeloma (20%) who developed AKI and none of them were subjected to dialysis. There were a total of 9 patients of Hemolytic Uremic syndrome who developed AKI and none of them required hemodialysis.

### Conclusion

Septic AKI was the most common cause for hemodialysis in patients with AKI, followed by leptospirosis.

### References

1. Moorthy AV, Zimmerman SW. Renal disease in the elderly: clinicopathologic analysis of renal disease

in 115 elderly patients. *Clin Nephrol* 1980;14(5): 223-9.

2. Watts RA, Lane SE, Bentham G, et al. Epidemiology of systemic vasculitis: a tenyear study in the United Kingdom. *Arthritis Rheum* 2000;43(2):414-9.
3. Booth AD, Almond MK, Burns A, et al. Outcome of ANCA-associated renal vasculitis: a 5-year retrospective study. *Am J Kidney Dis* 2003;41(4):776-84.
4. Higgins RM, Goldsmith DJ, Connolly J, et al. Vasculitis and rapidly progressive glomerulonephritis in the elderly. *Postgrad Med J* 1996;72(843): 41-4.
5. Feest TG, Round A, Hamad S. Incidence of severe acute renal failure in adults: results of a community based study. *BMJ* 1993;306(6876):481-3.
6. Arora P, Kher V, Kohli HS, et al. Acute renal failure in the elderly: experience from a single centre in India. *Nephrol Dial Transplant* 1993;8(9):827-30.
7. Kaplan SA, Wein AJ, Staskin DR, et al. Urinary retention and post-void residual urine in men: separating truth from tradition. *J Urol* 2008;180(1): 47-54.
8. Kolman C, Girman CJ, Jacobsen SJ, et al. Distribution of post-void residual urine volume in randomly selected men. *J Urol* 1999;161(1):122-7.
9. Cronan JJ. Contemporary concepts in imaging urinary tract obstruction. *Radiol Clin North Am* 1991;29(3):527-42.
10. Naidich JB, Rackson ME, Mossey RT, et al. Nondilated obstructive uropathy: percutaneous nephrostomy performed to reverse renal failure. *Radiology* 1986;160(3):653-7.

## Prognosis of Haemodynamically Unstable Patients Secondary to Trauma Based on Lactate Clearance

Khan Khader Ali<sup>1</sup>, Begum Naheeda Shaik<sup>1</sup>, Shabbir Mohammed<sup>2</sup>,  
Samir Mohammed<sup>3</sup>

---

### Author's Affiliation:

<sup>1</sup>Consultant, Dept of Emergency Medicine, P.E.S. Institute of Medical Sciences and Research, Kuppam, Andhra Pradesh 517425, India.

<sup>2</sup>Consultant & Head, Department of Emergency Medicine & Critical Care, Sparsh Hospital, Bommasandra, Bengaluru, Karnataka 560099, India.

<sup>3</sup>Senior Consultant, Dept of Emergency Medicine, BGS Global Hospital, Bengaluru, Karnataka 560060, India.

### Corresponding Author: Begum Naheeda Shaik,

Consultant, Dept. of Emergency Medicine P.E.S. Institute of Medical Sciences and Research, Kuppam, Andhra Pradesh 517425, India.

E-mail: [rdccgtl@gmail.com](mailto:rdccgtl@gmail.com)

Received on 05.04.2017,

Accepted on 24.04.2017

### Abstract

TRAUMA is the third overall cause of death and the first cause of death before 40 yr of age [1]. Early recognition of haemorrhage and shock in traumatic patients can prevent death, most deaths (80%) occurring within 48 hrs, haemorrhage being the leading cause of death [1]. Estimation of lactate clearance can be used as a diagnostic and prognostic biomarker in case of trauma patients [1]. *Aims:* To establish the prognostic significance of the lactate clearance in unstable trauma patients. *Settings and Design:* This is a "prospective observational study" among the patients who presented to the Emergency Department of BGS Global Hospital. *Methods and Material:* The patients will undergo ABG or VBG, as feasible along with blood lactate estimation which shall be repeated after 4 hours and the lactate clearance is calculated for 4 hrs. *Statistical Analysis Used:* All quantitative data are analysed using mean, median and standard deviation. All qualitative data are analysed using the chi-square test. *Results:* According to the lactate clearance values, the death rate was high at higher lactate clearance values and the discharge rate was high with lower lactate clearance values. According to the initial lactate values, the discharge rates are high with low initial lactate levels than at higher initial lactate levels. *Conclusion:* This study concludes that all the trauma patients with lower Lactate clearance and lower initial lactate levels had better outcome when compared with those who had high lactate clearance and high initial lactate levels at the time of admission.

**Keywords:** Lactate Clearance; Trauma; Haemorrhage; Shock; Initial Lactate Values.

---

### Introduction

TRAUMA is the third overall cause of death and the first cause of death before 40 yr of age, causing many handicaps and high cost burden to the patients [1].

Early recognition of haemorrhage and shock in traumatic patients can prevent death, most deaths (80%) occurring within 48 hrs, haemorrhage being the leading cause of death [1].

The three main principles of trauma patient care are

1. To recognize and treat the haemorrhage early [1].
2. To limit the consequences of shock, and [1]
3. To diagnose traumatic lesions [1].

Haemorrhage and shock is responsible for inadequate oxygen delivery, that results in tissue hypoxia, anaerobic metabolism, and lactate production [1]. Hence estimation of lactate clearance can be used as a diagnostic and prognostic biomarker in case of trauma patients [1].

Lactic acidosis may persist despite control of the haemorrhage, reflecting flow-demand mismatch or

loss of appropriate capillary density as a consequence of shock, vasoconstriction, or other dysfunctional responses [1].

Lactate clearance (LC) has recently emerged as an important concept in septic shock, as part of quantitative resuscitation that aims to reach the predefined physiological goals to be achieved within the first hours of trauma or sepsis [1].

Anaerobic glycolysis sharply increases production of cellular lactate, which diffuses into blood stream during pro-longed cell ischemia [2]. Elevated circulating lactate concentration thus often indicates the widespread inadequate tissue oxygenation due to inadequate oxygen delivery and/or consumption [2].

However, besides these anaerobic processes, the aerobic (metabolic) mechanisms that affect the host's efficiency of energy transfer also contribute to lactate production [2]. Cytokine-mediated glucose uptake and catecholamine-stimulated Na-K pump overactivity both can result in increased pyruvate production that will eventually overwhelm the catalytic capacity of pyruvate dehydrogenase and result in increased

lactate either due to mass effect, or due to sepsis-induced pyruvate dehydrogenase dysfunction, or due to both [2].

In addition, reduced lactate clearance may reflect globally impaired metabolic function by liver and kidney, both of which normally contribute to systemic lactate disposal through anaplerosis, a mechanism that carboxylates lactate and delivers it to the tricarboxylic acid cycle, independent of the action of pyruvate dehydrogenase [2].

Thus, lactate clearance biologically reflects more of the general homeostasis of the host and thus provides more meaningful data about the overall adequacy of the resuscitative processes [2]. Lactate clearance (LC) has recently emerged as an important concept in septic shock, as part of the quantitative resuscitation concept that aims to reach predefined physiological goals to be achieved within the first hours [1]. The lactate clearance was defined by the equation [1]:

Therefore this study is intended to study the prognostic effect of Lactate Clearance on the outcome of haemodynamically unstable patients secondary to trauma.

$$\text{Lactate clearance} = \left\{ \frac{[\text{Lactate (initial)} - \text{Lactate (delayed)}]}{\text{Lactate (initial)}} \times 100 \times \text{Delay}^{-1} \right\}$$

(expressed as %/h) [1]

#### *Objectives of the Study*

1. To determine whether the early lactate clearance (0 to 4 hr) is predictive of inhospital mortality of the haemodynamically unstable trauma patients.
2. To establish the prognostic significance of the lactate clearance in unstable trauma patients in the Emergency Department.

#### *Inclusion Criteria*

1. Presenting history of trauma.
2. Age more than 15 yrs.
3. GCS less than 10.
4. Haemodynamically unstable patients with tachycardia, systolic blood pressure of less than 90 and saturation less than 90% on room air with PaO<sub>2</sub> less than 60.

#### *Exclusion Criteria*

1. History or evidence multi organ failure.
2. No history of trauma.
3. Patient in sepsis.

4. Age below 15 yrs.
5. History of diabetes mellitus on treatment with metformin.
6. Patient treated outside.

#### *Other Variables That Are Recorded Are*

1. Age
2. Gender
3. Brief history of presenting symptoms
4. Mechanism of trauma
5. Coexisting diseases
6. Medications patient is receiving
7. Nystagmus

#### **Materials and Methods**

##### *Source of Data*

All patients who presented to the Emergency Department of BGS Global Hospitals during the period between July 2015 and June 2016 with history of trauma and who are haemodynamically unstable.

*Method of Collection of Data*

After attaining the required permissions from the Ethical Committee, all patients satisfying the inclusion criteria have been enrolled in the study. They will initially go through history taking and examination as per standard proforma.

The patients will undergo ABG or VBG, as feasible along with blood lactate estimation. The ABG or VBG shall be repeated after 4 hrs to estimate the blood lactate and the lactate clearance is calculated 4 hrs.

*Type of Study*

This is a “prospective observational study” among the patients who presented to the Emergency Department of BGS Global Hospital and have satisfactorily been included in the study after having satisfied the inclusion criteria.

*Primary End Point:* Death.

*Secondary End Point*

Shifting to Operating Room.

*Adequacy of Resuscitation*

Until the peripheral pulse is of good volume and the systolic blood pressure is more than 100 mg and there is adequate urine output.

*Duration of Resuscitation*

As long as the patient stays in Emergency Department.

*Statistical analysis*

All quantitative data are analysed using mean, median and standard deviation. All qualitative data are analysed using the chi-square test.

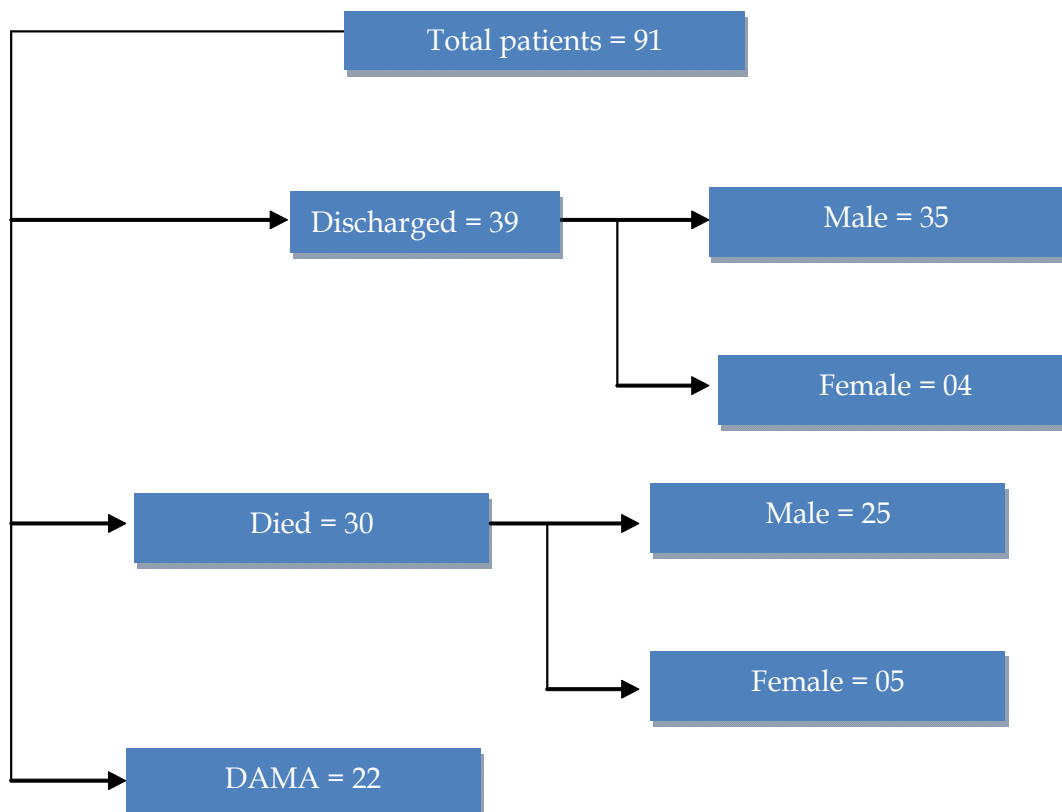
*Investigations Needed during the Study*

ABG or VBG depending on the feasibility.

*Ethical Committee*

Clearance has been obtained from the Ethical Committee of the institution before the study has started.

**Flow Chart of the Study:**



## Results

Total number of patients evaluated were 91 out of which 22 patients have left against medical advice. 39 patients have been discharged in neurologically intact state and 30 patients have died during the course of their stay in the hospital. No bias had occurred while recruiting the patients into the study. Out of 39 patients discharged in neurologically intact state 35 patients were male and 4 patients were female. And out of 30 patients died during the course of their stay in the hospital, 25 patients were male and 5

patients were female. According to the lactate clearance values, the death rate was high at higher lactate clearance values with 16 people dying within the range of 0 to 50. The discharge rate was high with lactate clearance values between 0 to 8 than at higher values of lactate clearance.

According to the initial lactate values, the discharge rates are high with low near to normal initial lactate levels than at higher initial lactate levels. The death rates were high between the initial lactate levels between 4 to 10 than at higher initial levels.

**Table 1:** Patient distribution

Total number of patients	91
Number of patients discharged	39
Number of patients died	30
Number of patients DAMA	22

**Table 2:** Death and Discharge distribution among male and female

Total number of patients = 91			
Death = 30		Discharge = 39	
Male	25	Male	35
Female	5	Female	4

**Table 3:** Death and discharge distribution as per lactate clearance

Lactate Clearance	Number of Deaths	Number of Discharges
-150 to -100	1	0
-99.9 to -50	2	0
-49.9 to 0	11	1
0.1 to 5	1	9
5.001 to 10	2	23
10.001 to 15	10	2
15.001 to 20	3	4

**Table 4:** Death and discharge distribution as per initial lactate

Initial Lactate Levels	Number of Deaths	Number of Discharges
0 to 2.2	6	9
2.2 to 4	4	9
4 to 6	8	7
6 to 8	3	8
8 to 10	5	3
10 to 15	3	3
>15	1	0

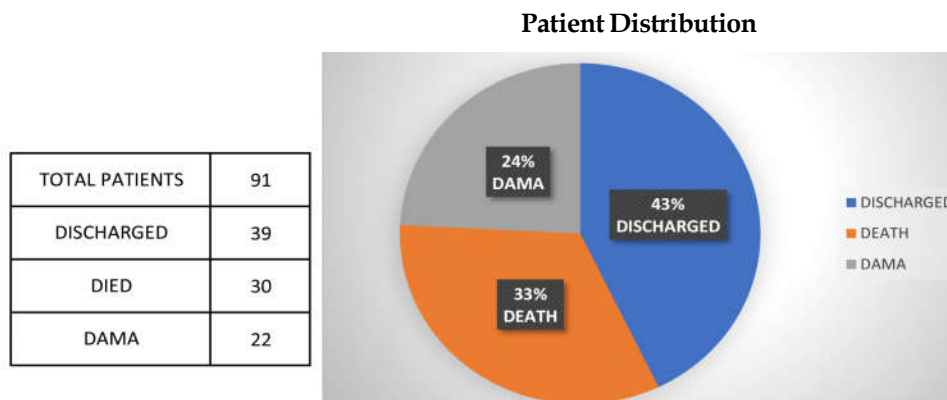
**Table 5:** Age distribution of discharge and death

Age	Death	Discharge
16-25	14	5
25-35	6	8
35-45	10	8
45-55	8	5
55-65	1	0
65-75	0	4

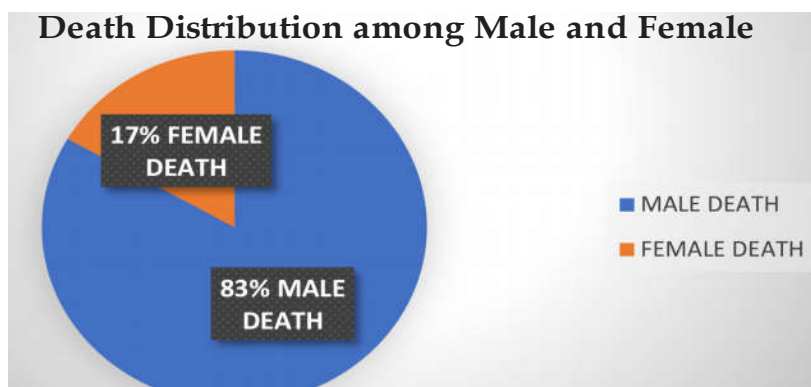
**Table 6:** Calculation of p value

	Death	Discharge	Marginal Row Totals
Lactate clearance < 10	14	33	47
Lactate clearance >10	16	6	22
Marginal column totals	30	39	69 ( Grand total )

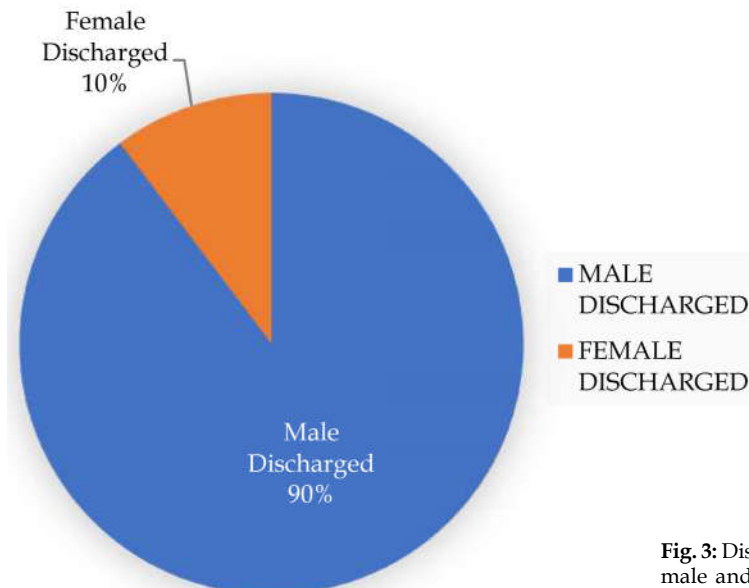
The chi square statistic is 11.2437. The p value is 0.000799. The result is significant at  $p < 0.05$ .



**Fig. 1:** Patient distribution



**Fig. 2:** Death distribution among male and female



**Fig. 3:** Discharge distribution among male and female

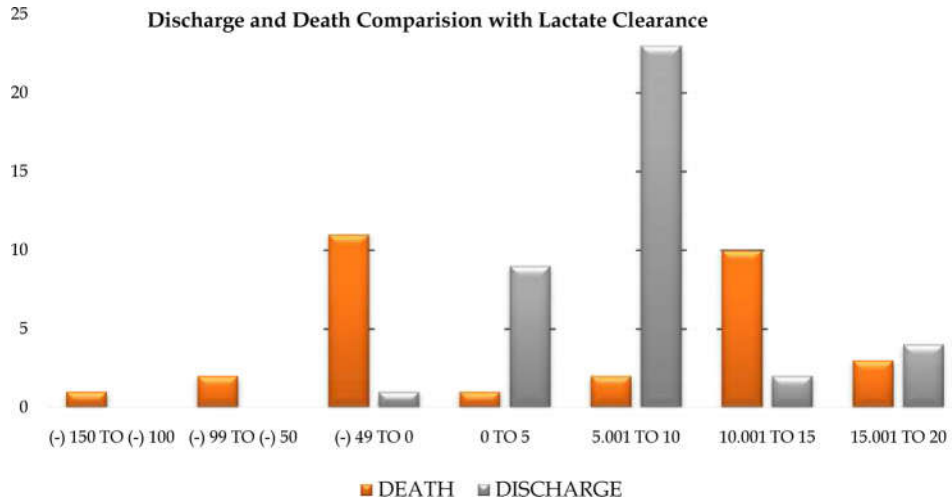


Fig. 4: Death and discharge comparison as per Lactate clearance

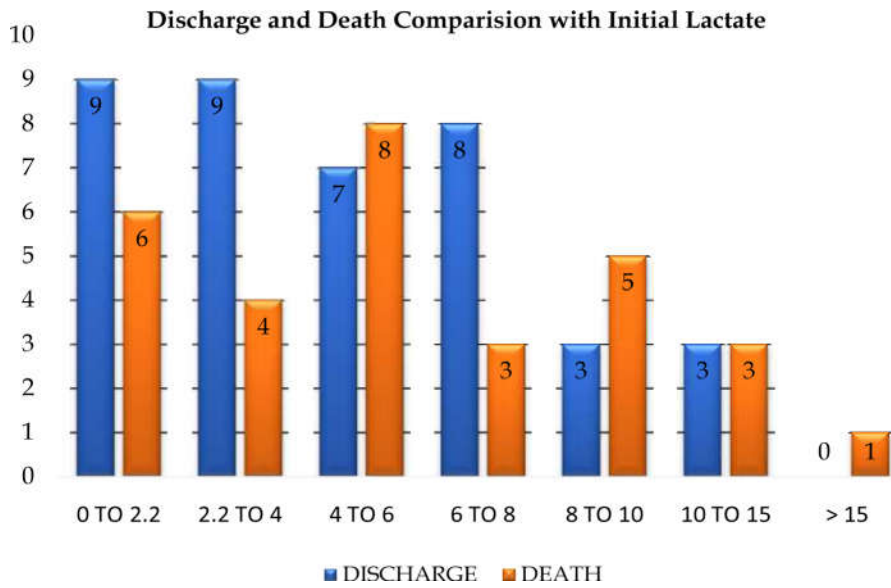


Fig. 5: Discharge and Death comparison as per initial lactate levels

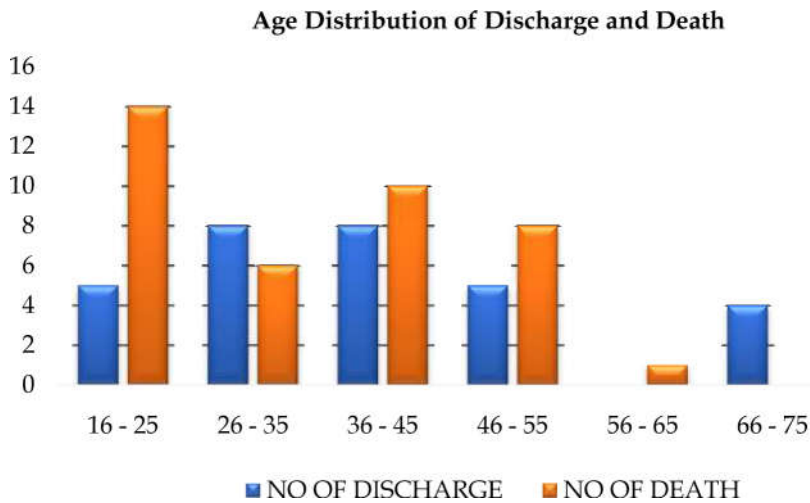


Fig. 6: Age distribution of discharge and death



## Discussion of the Study

Our study was done in a sample of 91 trauma patients admitted in BGS Global Hospital, Bengaluru.

Various studies have proved the predictive value of lactate clearance levels and initial lactate levels in predicting the outcome in trauma patients.

In his study, "Prognostic Significance of Blood Lactate and Lactate clearance in Trauma patients" Regnier et al. has shown that the mortality is high in case of high lactate clearance levels (~28%) and high initial lactate levels (~80%) in comparison to low lactate clearance (~18%) and low initial lactate levels (~30%). Our study also has shown that the lactate clearance calculated in the initial 4 hrs of admission has shown that in higher lactate clearance levels the mortality is high (72.72%) and when the lactate clearance is less than 10 the admission to discharge rate was high (mortality is low). In case of initial lactate levels also the mortality is high when the initial lactate is > 4 mmol/L (66.66%) when compared to high lactate levels (33.33%).

In the systematic review "Blood lactate as a predictor for in-hospital mortality in patients admitted acutely to hospital" Kruse et al., reviewed the usefulness of a single blood lactate measurement obtained at the time of admission in predicting the adverse outcomes in trauma patients and also the dose-response relationship that states the higher the lactate levels, the higher the mortality rates ( $p < 0.001$ ). Our study also has demonstrated that the mortality rate is higher when the initial lactate levels are higher (66.66%).

In his research article "Serum lactate as a predictor of early outcomes among trauma patients in Uganda", Okello et al., has shown that the initial lactate measurement of > 4 mmol/L has been associated with high admission rates (37%) and a 72 hr non discharge from hospital (44%). Our study has shown that there is high mortality associated with trauma patients with initial lactate levels of > 4 mmol/L (66.66%).

In his systematic review "Do lactate levels in the Emergency Department predict outcome in adult trauma patients", Baxter et al., has concluded that there is increase in the mortality with increasing lactate levels ( $p < 0.001$  - significant). Our study has also shown that there is increasing mortality with higher lactate levels of > 4 mmol/L (66.66%).

Henceforth the study had similarities with other studies in stating that higher lactate clearance levels and higher initial lactate levels have been associated with high mortality rates. Therefore it can be stated

again that the lactate clearance levels and the initial lactate levels either singly or both combined prove to be an effective tool in the prediction of outcome in trauma patients.

## Conclusion

This study concludes that all the trauma patients with lower Lactate clearance and lower initial lactate levels at the time of admission had better outcome when compared with those trauma patients who had high lactate clearance and high initial lactate levels at the time of admission.

## Limitations

1. The group studied is small.
2. Conducted in a single centre.
3. There is no geographical representation.
4. This study was not conducted in different races.

## References

1. Marie-Alix Régnier, Mathieu Raux, Yannick Le Manach, Yves Asencio. Prognostic Significance of Blood Lactate and Lactate Clearance in Trauma Patients. *Anaesthesiology* 2012;117(6):1276-88.
2. Alan E. Jones, MD., Department of Emergency Medicine University of Mississippi Medical Center Jackson, MS. Lactate Clearance in the Acutely Traumatized Patient. *Anesthesiology*. 2012 December;117(6):1162-1164.
3. Andra L. Blomkalns, MD Assistant Professor; Vice Chairman-Education; Residency Program Director, Department of Emergency Medicine, University of Cincinnati College of Medicine, Cincinnati, OH, Director of CME and Enduring Materials, EMCREG-International. Lactate - A Marker For Sepsis AND Trauma Copyright EMCREG-International, 2007;43-49. [www.emcreg.org](http://www.emcreg.org).
4. Ole Kruse, Niels Grunnet and Charlotte Barfod. Blood lactate as a predictor for in-hospital mortality in patients admitted acutely to hospital: a systematic review. *Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine* 2011;19:74.
5. Zhenjiang Bai, Xueping Zhu, Mengxia Li, Jun Hua, Ying Li, Jian Pan, Jian Wang and Yanhong Li. Effectiveness of predicting in-hospital mortality in critically ill children by assessing blood lactate levels at admission. *BMC Pediatrics* 2014;14:83. <http://www.biomedcentral.com/1471-2431/14/83>.

6. Michael Okello, Patson Makobore, Robert Wangoda, Alex Upoki and Moses Galukande. Serum lactate as a predictor of early outcomes among trauma patients in Uganda. *International Journal of Emergency Medicine* 2014;7:20 <http://www.intjem.com/content/7/1/20>.
  7. Jay D. Pal, MD, PhD, Gregory P. Victorino, MD, Patrick Twomey, MD, Terrence H. Liu, MD, M. Kelley Bullard, MD, and Alden H. Harken, MD. Admission Serum Lactate Levels Do Not Predict Mortality In The Acutely Injured Patient. *The Journal of TRAUMA, Injury, Infection and Critical Care*; 2006;60(3);583-589.
  8. Zachary D.W. Dezman, Angela C. Comer, Mayur Narayan, Thomas M. Scalea, Jon Mark Hirshon, Gordon S. Smith. Alcohol Consumption Decreases Lactate Clearance In Acutely Injured Patients. *Injury, Int. J. Care Injured* 2016;47:1908-1912. <http://dx.doi.org/10.1016/j.injury.2016.03.007>.
  9. Tim C. Jansen, MD; Jasper van Bommel, MD, PhD; Roger Woodward, MD; Paul G. H. Mulder, PhD; Jan Bakker, MD, PhD. Association between blood lactate levels, Sequential Organ Failure Assessment subscores, and 28-day mortality during early and late intensive care unit stay: A retrospective observational study. *Crit Care Med* 2009;37(8); 2369-2374.
-

## Spectrum of Acute Febrile Illness in Children Presenting in Emergency of a Tertiary Care Hospital and its Clinico - Laboratorial Correlation

Kishalay Datta<sup>1</sup>, Rigenjyoti Kalita<sup>2</sup>

---

### Author's Affiliation:

<sup>1</sup>Associate Director and HOD  
<sup>2</sup>Attending Consultant, Dept. of  
Emergency Medicine, Max  
Healthcare, Saket, New Delhi,  
Delhi 110017, India.

### Corresponding Author:

**Kishalay Datta,**  
Associate Director and HOD,  
Dept. of Emergency Medicine,  
Max Healthcare, Saket, New  
Delhi, Delhi 110017, India.  
E-mail: [dr.kishalay@gmail.com](mailto:dr.kishalay@gmail.com)

**Received on** 18.07.2017,  
**Accepted on** 16.08.2017

### Abstract

In a clinical setting, Fever is the most common sign of illness in infants and children and accounts for as many as 20% of pediatric emergency department (ED) visits. Clinical evaluation of febrile illness is guided by history and physical examination, along with judiciously selected screening test. Most of the studies have been done at ambulatory care setting with lack of proper follow up of the patients and accurate data regarding bacterial and viral aetiologies because of the difficulty in making microbiological diagnosis in ambulatory care settings. In this study all children more than 3 months and under the age of 15 years presenting to ED with fever and warranting hospitalization were included and analyzed as regards the spectrum of febrile illness and its correlation with clinical findings at presentation and laboratory investigations at and during the entire hospital stay. *Objectives:* To study the spectrum of acute febrile illnesses and develop a Clinical and laboratorial correlation in children more than 3 months and under the age of 15 years warranting ER visit and subsequent hospitalization. *Methods and Material:* It was a Prospective observational unicentric study done at Max Super Speciality Hospital, Shalimarbagh, New Delhi . Data was collected over a period September 2016 to May 2017 from 580 pediatric patients visited to ED and got admitted, as per data collection sheet after the approval by scientific and ethics committee of the institute. *Statistical Analysis Used:* The collected data was entered in the Excel spreadsheet using Microsoft Excel Software and transferred to Statistics Package for Social Sciences (SPSS) version 20, IBM Inc. for analysis. It was subjected to descriptive statistics for calculation of mean, standard deviation, frequencies and percentages. Summarized data was presented using Tables and Graphs. Shapiro Wilk test was used to check which all variables were following normal distribution. Chi-square test was used for comparison between categorical variables. Pearson's correlation coefficient (ordinal data) was calculated to measure the strength of a relationship between provisional diagnosis and final diagnosis. Level of statistical significance was set at p-value less than 0.05. *Results:* This study was done at a tertiary care hospital where yearly about 14200 patients visited to Emergency, 40% of all attendance is pediatric and among them 60% were prompt by fever. This data was also supported by the previous studies. The majority of the children who presented with fever and got admitted fall under the age group of 4-15 years which accounts or 76% of the total study population. Male outnumbered females. Male accounts for 67% (n=389) and Female accounts for 33% (n=191) among all total pediatrics hospital admissions. Out of 580 study population in presenting symptoms respiratory predominance was seen (29.1%) which was followed by fever with rash (26.8%). Most common diagnosis documented in our pediatric patients with acute febrile illness were URTI, majority of them presumably viral

rhinopharyngitis (26.4%). This was followed by Dengue and dengue like illness and chikungunya each accounts for 13.4% and 21.5% respectively. Enteric fever was the most common diagnosis documented (24.7%) in among all specific bacterial diseases. Electrolyte disturbances specially hyponatremia was observed in 58% of pediatrics patients and was invariably associated with dehydration. Dehydration accounts for 5.7% of total study population. *Conclusions:* In conclusion, Emergency services are an integrable part of any healthcare infrastructure with almost 40% of being pediatrics attendance. Fever constituted 60% (almost 2/3 rd patients) of febrile illness. Infectious diseases still accounts for the majority of ER attendance. The standard protocol of diagnosis and management if applied well, confirms the diagnosis with accuracy resulting in a favorable outcome.

**Keywords:** Fever Without Source (FWS); Serious Bacterial Infection (SBI); Urinary Tract Infection (UTI); Febrile Seizure (FS); Emergency Department (ED).

---

## Introduction

Fever is the most common sign of illness in infants and children which accounts for as many as 20% of paediatric emergency department (ED) visits and the underlying conditions may range from mild self limiting illness to the most serious of bacterial and viral illness. Fever is defined as a documented temperature of 38 degree or higher per rectum. A rectal equivalent temperature is calculated by adding 0.5 degree C to the oral temperature and 0.8 degree C to the axillary temperature. A careful history and thorough physical examination is essential in the evaluation of the febrile child. Child's demographic information including Vital signs, length and weight with percentiles, nutritional status, level of physical activity, and level of arousal should be a part of evaluation. Physical examination findings that suggest serious bacterial infections in febrile children (aged 3-36 mo) include ill appearance, fever, vomiting, tachypnea with retractions, and delayed capillary refill time. Hence challenge of a emergency physician is to focus on the etiology of fever and to identify the infant or child who is at risk for serious infection.

## Materials and Methods

This study was done at Max Super Speciality Hospital, Shalimarbagh, New Delhi where yearly about 14200 patients visited to Emergency. The main objective of the study was to study the spectrum of acute febrile illnesses and develop a Clinical and laboratorial correlation in children more than 3 months and under the age of 15 years warranting ER visit and subsequent hospitalization. We also aimed to study Prevalence of different types of febrile illness.

All the patients presenting to ED of Max Hospital Shalimar Bagh and meeting the Inclusion and Exclusion Criteria as mentioned below were enrolled in the study.

### *Inclusion Criteria*

1. Fever being recorded more than 38°C
2. Fever of less than 7 days of occurrence, presented to ED and warrant subsequent hospitalization.
3. Children more than 3 months and under the age of 15 years.

### *Exclusion Criteria*

1. Children with co-morbidities like pre-existing cardiac disease, respiratory, metabolic, gastrointestinal, neurological, immune compromised states, malignancies etc
2. Patients not willing for hospitalization.

All patients who meet the inclusion criteria and none of the exclusion criteria were enrolled in the study after taking their voluntary consent for participating in the study. Any medicines taken prior to ED visit shall also be recorded. All patients with febrile illness were evaluated by the ER physician and validated by the pediatrician on duty. Patient's Demographical information, pertinent historical and physical findings were recorded and a provisional diagnosis was made. Appropriate laboratorial investigations were ordered including complete blood cell count (CBC), CRP, urinalysis, relevant cultures of blood, cerebrospinal fluid, urine or other body fluids, and imaging modalities as advised by the attending pediatrician as per the SOP (standard operating procedure). The patient was dully followed during the entire course of hospital stay and final diagnosis

were recorded which is utilized for analysis.

Acute febrile illness is defined as a patient with fever of 38 degree or higher at presentation to ED or history of fever that persisted for 2-7 days with no localizing source. An invasive bacterial illness is defined as bacterial growth of a known pathogen in cultures of blood (bacteraemia), spinal fluid

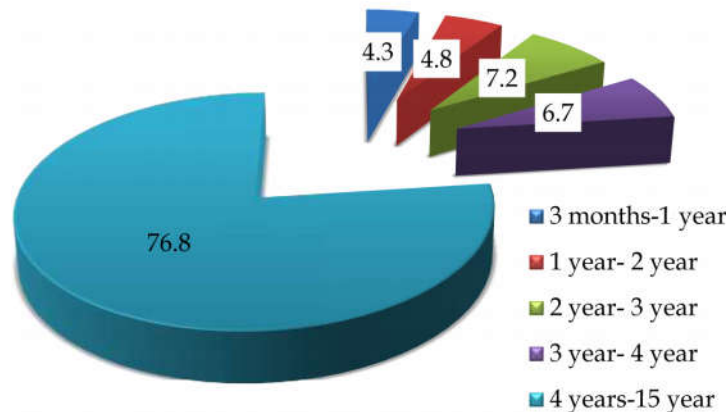
(meningitis), joint fluid (septic arthritis) or urine (urinary tract infection) with the relevant clinical signs and symptoms. Pneumonia was confirmed radiographically as per standard protocol. Viral illness were documented as diagnosis of exclusion when no focus of infection on the physical examination and cultures were sterile.

**Results**

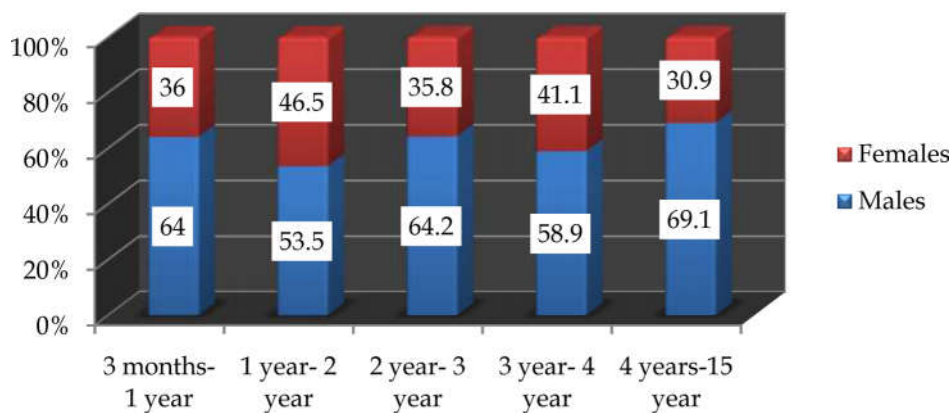
**Table 1:** Distribution of study population according to age group and gender

	Male		Female		Total	
	N	%	N	%	N	%
3 months-1 years	16	64	9	36	25	4.3
1 year- 2 year	15	53.57	13	46.4	28	4.8
2 year- 3 year	27	64.2	15	35.7	42	7.2
3 year- 4 year	23	58.9	16	41.0	39	6.7
4 years-15 years	308	69.0	138	30.9	446	76.8
Total	389	67.0	191	32.9	580	100

**Age wise distribution of study population**



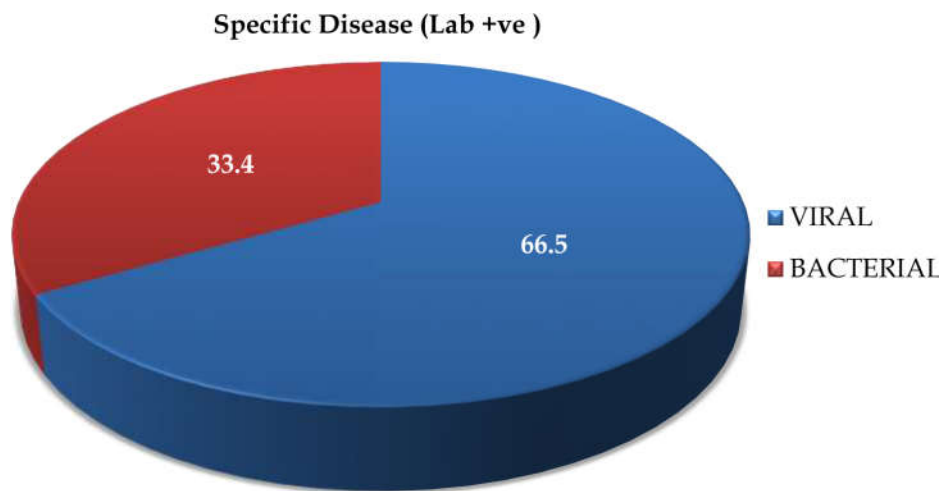
**Distribution of study population**



**Fig. 1:** Study population reflect Male outnumbered Female (67% vs 33%)

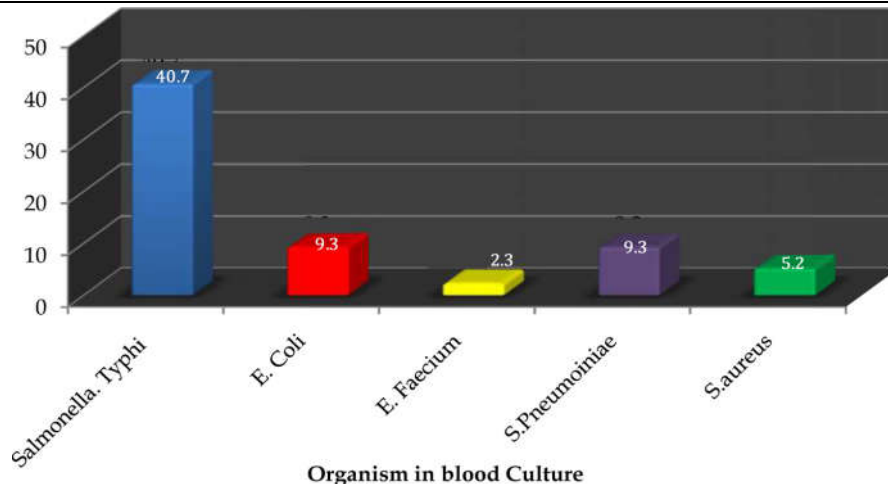
**Table 2:** Distribution of specific disease (viral and bacterial)

	N (514)	%
<b>Viral (n=342), 66.5%</b>		
Dengue And Dli	69	13.4
Chikungunya	111	21.5
Viral Rhinopharyngitis	136	26.4
Viral Gastroenteritis	22	4.2
Influenza And Fli	4	0.7
<b>Bacterial (n= 172), 33.4%</b>		
Sepsis	5	0.9
UTI	8	1.5
LRTI	21	4.0
Enteric Fever	127	24.7
Gastroenteritis	7	1.3
Ssti /Osteomyelitis	4	0.7



**Table 3:** Distribution of organism identified in blood cultures among the study population

	N	%
Salmonella. Typhi	70	40.7
E. Coli	16	9.3
E. Faecium	4	2.3
S.Pneumoniae	16	9.3
S.aureus	9	5.2



**Fig. 3:** Distribution of organism identified in blood cultures among the study population

## Discussion

This study was done at a tertiary care hospital where yearly about 14200 patients visited to Emergency, 40% of all attendance is pediatric patients. Among them 60% encounters were prompt by fever. The majority of the children who present with fever and got admitted fall under the age group of 4-15 years which accounts for 76% of the total study population. Children less than one year of age has low frequency (4.7%) who got hospitalized. Male outnumbered females. Male accounts for 67% (n=389) and Female accounts for 33% (n=191) among all total pediatric hospital admissions. Out of 580 study population respiratory symptoms predominant (29.1%) which was followed by fever with rash (26.8%) probably can be explained by children exposed to Dengue/Chikungunya outbreak in the months of July to October. Vomiting and Gastrointestinal symptoms accounts for 21% and 5.3% respectively of total presentations. Seizure was described in only miniscule number of patients. Specific disease compromises 88.6% of total study population whereas Clinically diagnosed and miscellaneous cases accounts for 7.4% and 3.9% respectively. As regards of diagnosis of specific disease Male and Female febrile patients does not show any significant correlation (90% vs 85%). In the study population average number of days of fever was 2.49(SD 1.05) in specific disease group and 3 (SD1.24) in miscellaneous group. As per total population concern average length of stay in hospital was 3 days. Most common diagnosis documented in our pediatric patients with acute febrile illness were URTI, majority of them presumably viral rhinopharyngitis (26.4%). This was followed by Dengue and dengue like illness and chikungunya each accounts for 13.4% and 21.5% respectively. Enteric fever was the most common diagnosis documented (24.7%) in among all specific bacterial diseases. Blood culture for salmonella typhi was found to be positive in 55% cases and Typhidot came to be positive in 44% of cases. Lower respiratory tract infection, particularly pneumonia and gastroenteritis accounts for 12.2% and 4.1% respectively. Urinary tract infection (UTI) had a occurrence of 4.1% and SSTI/Osteomyelitis found to be positive in 2.3% patients. Apart from S.typhi Other isolated organisms in blood culture were E. coli (9.3%), E.facecium (2.3%). S.pneumoniae (9.3%) and S.aureus (5.2%). In laboratorial analysis TLC did not show any correlation in diagnosing a specific disease as 85.3% of specific diagnosis had normal TLC value. Raised Hepatic transaminases (SGOT/SGPT) were observed

in 50% febrile patients explainable by the fact that having dengue, chikungunya, enteric causes. Electrolyte disturbances specially hyponatremia was observed in 58% of pediatric patients and was invariably associated with dehydration. Dehydration accounts for 5.7% of total study population.

Our study had several limitations.

1. Enrollment of febrile patients in the ongoing outbreak situation (Dengue/Chikungunya) can skewed the data.
2. Short duration of the study unable to reflect the seasonal variability of diseases.
3. To derive a meaningful conclusion on the spectrum of illness a large database is required.

## Conclusion

In conclusion, Emergency services are an integrable part of any healthcare infrastructure with almost 40% of being pediatric attendance. Fever constituted 60% (almost 2/3 rd patients) of febrile illness. Infectious diseases still accounts for the majority of ER attendance and the standard protocol of diagnosing and management if applied well, confirm the diagnosis with accuracy resulting in better results. Thus the recommendation would be integrate pediatric ER/triage services in any health infrastructure and standard operating procedures (SOPs) should be strictly adhere to for a favorable outcome.

Future directions for consideration include;

- A multicentric study with a long study period including adult population is recommended to document the seasonal variability of diseases and to derive a meaningful conclusion on the spectrum of disease.

## References

1. Alpern ER, Henretig FM. Fever. Fleisher GR, Ludwig S, Henretig FM, eds. Textbook of Pediatric Emergency Medicine. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.p.295-306.
2. Al-Eissa YA, Ghazal SS, Al-Zamil FA et al. Pattern of pediatric illness in children seen at a pediatric ambulatory care setting. J Family Community Med. 2000 May;7(2):61-5.

3. Simon AE, Lukacs SL, Mendola P. Emergency department laboratory evaluations of fever without source in children aged 3 to 36 months. *Pediatrics* 2011;128:e1368.
  4. McGowan JE Jr, Bratton L, Klein JO, Finland M. Bacteremia in febrile children seen in a "walk-in" pediatric clinic. *N Engl J Med*. 1973;288:1309-12.
  5. Teele DW, Pelton SI, Grant MJ, Herskowitz J, Rosen DJ, Allen CE, et al. Bacteremia in febrile children under 2 years of age: results of cultures of blood of 600 consecutive febrile children seen in a "walk-in" pediatric clinic. *J Pediatr*. 1975;87:227-30.
  6. Baraff LJ, Bass JW, Fleisher GR, Klein JO, McCracken GH, Powell KR, et al. Practice guideline for the management of infants and children with fever without source 0-36 months of age. *Pediatrics*. 1993;92:1-12.
  7. Baraff LJ, Lee SI. Fever without source: management of children 3 to 36 months of age. *Pediatr Infect Dis J*. 1992;11:146-51.
  8. Fleisher GR, Rosenberg N, Vinci R, Steinberg J, Powell K, Christy C, et al. Intramuscular versus oral antibiotic therapy for the prevention of meningitis and other bacterial sequelae in young, febrile children at risk for occult bacteremia. *J Pediatr*. 1994;24:504-12.
  9. Shaw KN, Gorelick M, McGowan KL, et al. Prevalence of urinary tract infection in febrile young children in the emergency department. *Pediatrics* 1998;102:e16.
  10. Bulloch B. Fever without focus in the older infant. In: Moyer V, Elliot EJ, Davis RL, editors. *Evidence Based Pediatrics and Child Health*. Boston: Blackwell Publishing; 2004.p.169-77.
-



# Non-Invasive Ventilation: First Line Therapy in the Acute Exacerbations of COPD in Emergency Department

Mohammed Ismail Nizami<sup>1</sup>, Narendra Kumar N.<sup>2</sup>, Ashima Sharma<sup>3</sup>, G. Vishwa Reddy<sup>1</sup>, S. Raghavendra Goud<sup>1</sup>

## Author's Affiliation:

<sup>1</sup>Assistant Professor <sup>3</sup>Professor and Head, Department of Emergency Medicine, <sup>2</sup>Associate Professor, Department of Pulmonary Medicine, Nizam's Institute of Medical Sciences Punjagutta, Hyderabad, Telangana 500082, India.

## Corresponding Author:

**Mohammed Ismail Nizami**  
Assistant Professor, Department of Emergency Medicine, Nizam's Institute of Medical Sciences Punjagutta, Hyderabad, Telangana 500082, India.  
E-mail: ismail\_nizami@rediffmail.com

Received on 26.05.2017,  
Accepted on 13.06.2017

## Abstract

Non-invasive ventilation has been a major advancement in the management of acute exacerbations of chronic obstructive pulmonary disease. It reduces the need for endotracheal intubation, thereby reducing associated complications and hospital cost. The aim of our study is to assess the efficacy of non-invasive ventilation in acute exacerbations of chronic obstructive pulmonary disease with respiratory failure. A total of 86 patients presenting with acute respiratory distress at our emergency room were initially included and evaluated. Non invasive ventilation was initiated in addition to standard medical treatment in all cases. Response to therapy in terms of improvement in patients' vitals and ABG were sequentially recorded and analyzed. Overall 72.5% (n=29) of the patients improved, whereas 27.5% (n=11) did not improve with NIV among whom 63.63% (n=7) had to be mechanically ventilated. 62.5% (n=25) showed a good ABG response with improvement in pH and decrease in PaCO<sub>2</sub> levels. Therefore, NIV should be considered to be the first line of management in acute exacerbations of COPD with respiratory failure.

**Keywords:** Acute Exacerbation; Chronic Obstructive Pulmonary Disease; Respiratory Failure; Non-Invasive Ventilation; Endotracheal Intubation, Arterial Blood Gas.

## Introduction

Chronic obstructive pulmonary disease (COPD) is a major health problem and leading cause of morbidity and mortality worldwide. The disease burden is expected to rise in the years to come. World Health Organization has predicted that by 2020, COPD will be the 5th most prevalent disease worldwide and will be among the three leading causes of death. Acute exacerbations of COPD (AECOPD) are largely responsible for the morbidity and mortality associated with the disease. The frequency of hypercapnic respiratory failure in patients with AECOPD varies from 16-35% with overall mortality of 35-43%.

Non-invasive ventilation (NIV) is effective in the treatment of patients with acute respiratory failure (ARF) as shown by a number of controlled trials and meta-analyses. However, evidence for the use of NIV

remains strongest in patients with hypercapnic ARF due to exacerbations of chronic obstructive pulmonary disease (COPD) and cardiogenic pulmonary edema. NIV is proved to reduce the need of endotracheal intubation (ETI), to prevent ETI-associated pneumonia and to decrease incidence of mortality compared to ventilated patients. The use of NIV has been continuously increasing over the last decade and has been substantiated by enough clinical evidence. The current study was planned to determine the safety and efficacy of NIV in the subgroup of patients with respiratory failure due to AECOPD presenting to the emergency room of our hospital.

## Material and Methods

This was an institution based prospective study carried out in the emergency room and MICU of our

hospital over a period of twelve months. It was approved by the institutional ethics committee and an informed written consent was obtained from all patients or the next of kin before enrolment into the study. A total of 86 patients presenting with acute respiratory distress were initially evaluated. Among them, 40 patients were enrolled in the study after confirmation of the episode as acute exacerbation of COPD based on history, clinical examination, lab investigations and chest X-ray. Cases with mild to moderate respiratory acidosis (pH between 7.25-7.35) were included. Exclusion criteria included dyspnea due to other causes, metabolic acidosis, life-threatening refractory hypoxemia, impaired mental status, excessive secretions, hemodynamic instability or life-threatening arrhythmias, uncooperative or agitated patients and inability to use mask because of trauma or surgery.

The baseline clinical parameters were recorded and an ABG was obtained from all patients at the time of presentation (Table 1). All patients were started on standard medical therapy including supplemental oxygen, intra-venous steroid, antibiotics and nebulised bronchodilators (Levo-salbutamol and/or Ipratropium bromide). A portable Non-invasive ventilator with monitor (BIPAP, VIVO-30 from BREAS) was used in the spontaneous mode using full face mask. Patients were asked to lie supine with head end elevated by about 45°. After explaining the procedure and reassurance, a correct sized interface was placed. To start with, low pressures were given to acclimatize the patient. The initial trial parameters (in spontaneous mode) were set to 8 cmH<sub>2</sub>O of IPAP and 4 cmH<sub>2</sub>O of EPAP with oxygen flow rate of 1-2 L/minute in patients with hypoxemia. EPAP was increased by 1-2 cms H<sub>2</sub>O till the patient triggers the ventilator. IPAP and EPAP parameters were titrated to optimize patient's comfort. The difference between IPAP and EPAP was always maintained at not less than 4 cms H<sub>2</sub>O.

Each patient was closely monitored for mental status, signs of air leak around the mask and vital parameters. ABG was obtained in all patients one hour after starting of NIV. If satisfactory degree of patient comfort, ventilation and oxygenation were not achieved, BIPAP was discontinued and the patient was excluded from the study. Criteria for non-compliance included irritability and restlessness, worsening dyspnea, falling oxygen saturations and abdominal distention. However, if adequate response was achieved, NIV was continued for up to 6 hours and again an ABG was taken to assess improvement. The response of the patient was sequentially recorded.

- Subjective response: dyspnea quantified by MMRC, use of accessory muscles of respiration, degree of comfort and mental alertness.
- *Objective Response*: respiratory rate, oxygen saturation, blood pressure, heart rate and improvement in ABG.

In our study ABG response is defined as:

1. Corrected: pH increased more than or equal to 7.35.
2. Improved: increase in pH by 0.05 - 0.1
3. Not improved: increase in pH by less than 0.05, by comparing the ABG's taken at 0, 1 and 6 hrs.

The patients were divided into responders (ABG corrected or improved) and non-responders (ABG not improved). Data was entered into Microsoft Excel spreadsheet 2007 and the statistical analysis was performed by using Graphpad Prism® version 4 USA®. The data was described as mean ±SD for continuous variables and frequencies/percentages for category variables. Between group analysis was performed by using Oneway ANNOVA followed by BONFERRONI multiple comparison test. A 2 tail p-value of less than 0.05 was considered statistically significant.

## Results

During the study period, a total of 86 subjects were evaluated for acute respiratory distress and out of them 40 were enrolled into the study after acute exacerbation of COPD with respiratory failure was confirmed. Those subjects who met the inclusion and exclusion criteria were started on NIV. There were 37 male and 3 female patients with a mean age of 57.5 (SD±8.2) years. 40% of the study group was in the age group of 61-70 whereas 32.5% and 27.5% were in the ages between 51-60 and 40-50 respectively. The serial clinical and arterial blood gas parameters are shown in Table 2. There was significant improvement in the clinical (respiratory rate, pulse rate and blood pressures) and ABG (pH, PaCO<sub>2</sub>) parameters in patients successfully responding to NIV. However the PaO<sub>2</sub> values and the SPO<sub>2</sub> tend to decline and all of the patients required supplemental oxygen which was delivered through a port available at the facial interface.

The biochemical response and clinical outcome is shown in Tables 3 & 4 respectively. Positive biochemical response (improvement in pH and reduction in PaCO<sub>2</sub>) was achieved in 27 of the patients in 1<sup>st</sup> hour of NIV. However 3 (7.5%) patients showed

a delayed improvement by the end of 6<sup>th</sup> hour. There were 2 (5%) patients whose ABG improved in the first hour but worsened by the end of 6<sup>th</sup> hour. 32 (80%) patients improved clinically within 1 hour of the initiation of NIV; however 3 of them deteriorated and did not tolerate NIV.

Overall 29 (72.5%) patients improved, whereas 11 (27.5%) did not improve with NIV and 7 (63.63%) among them had to be intubated. Among the study group 25 (62.5%) patients showed a good ABG response with improvement in pH and decrease in PaCO<sub>2</sub> levels. Even among the Non responder group, 5 (12.5%) patients showed clinical improvement. ABG

response could not be assessed in the remaining 5 (12.5%) patients. All patients were followed up till discharge. There were no deaths within the study period. The patients who did not show any clinical improvement by the end of 1<sup>st</sup> hour did not improve subsequently thereby proving that the 1<sup>st</sup> hour response is important in the outcome of NIV. The most frequent complication for which the NIV had to be discontinued was the worsening dyspnea and decreasing oxygen saturation. 2 (5%) of the patients developed altered sensorium and 2 (5%) others complained of abdominal distention. Only 1 (2.5%) had dryness of mouth as shown in Table 5.

**Table 1:** Demographic and physiological baseline characteristics

Number of Subjects (N)	40
Age in years	57.5±8.2
Males	37 (92.5%)
Females	3 (7.5%)
RR (per min.)	33.0±4.7
PR (per min.)	95.0±14.0
SBP (mmHg)	149.0±15.0
DBP (mmHg)	93.0±11.0
pH	7.30±0.1
PaO <sub>2</sub> (mmHg)	53.2±6.0
PaCO <sub>2</sub> (mmHg)	70.0±15.0
SPO <sub>2</sub> %	85.0±5.7

RR-Respiratory rate, PR-Pulse rate, SBP-Systolic blood pressure, DBP-Diastolic blood pressure

**Table 2:** Hemodynamic and biochemical variables

Variables	0 hrs	1hr	6 hrs	P value		
				0 vs.1	1 vs.6	0 vs.6
RR (per min.)	33.0±4.7	31.0±4.9	27.0±4.1	P < 0.05	P < 0.05	P < 0.001
PR (per min.)	95.0±14.0	93.0±20.0	86.0±12.0	P > 0.05	P > 0.05	P < 0.05
SBP (mmHg)	149.0±15.0	147.0±19.0	139.0±12.0	P > 0.05	P > 0.05	P < 0.05
DBP (mmHg)	93.0±11.0	89.0±12.0	84.0±6.7	P > 0.05	P > 0.05	P < 0.01
pH	7.3±0.1	7.3±0.1	7.4±0.1	P > 0.05	P < 0.01	P < 0.001
PaO <sub>2</sub> (mmHg)	53.2±6.0	65.1±10.4	69.0±18.0	P > 0.05	P > 0.05	P > 0.05
PaCO <sub>2</sub> (mmHg)	70.0±15.0	64.0±15.0	61.0±11.0	P > 0.05	P > 0.05	P < 0.05
SPO <sub>2</sub> (%)	85.0±5.7	92.0±7.1	95.0±4.7	P > 0.05	P > 0.05	P < 0.05

Respiratory rate -RR, Pulse rate -PR, Systolic blood pressure-SBP, Diastolic blood pressure-DBP

**Table 3:** Biochemical response

Responders	1 hr.	27 (67.5%)
	6 hrs.	25 (62.5%)
Non responders	With clinical improvement	5 (12.5%)
	With no clinical improvement	5 (12.5%)
Response could not be assessed		5 (12.5%)

**Table 4:** Clinical outcome

Improved	1 hr.	32 (80%)
	6 hrs.	29 (72.5%)
Not improved	MV	7 (17.5%)
	DNR	2 (5%)
	AMA	2 (5%)

MV-Mechanical Ventilation, DNR-Do not resuscitate, AMA-Against medical advice

**Table 5:** Complications

Complications	N	(%)
Worsening SOB & falling saturation	7	17.5%
Diminished level of consciousness	2	5%
Abdominal distension	2	5%
Dryness of mouth	1	2.5%

**Table 6:** Baseline characteristics in various studies

Study	pH	PaO <sub>2</sub>	PaCO <sub>2</sub>
Our study	7.30±0.1	53.2±6.0	70.0±15.0
Agarwal et al	7.27±0.07	55.6±16.1	73.1±24.3
S.P. Rai et al	7.26	61.0	75.2
R. Prasad et al	7.307±0.03	41.11±10.74	79.11±14.17

**Table 7:** Comparison of 2<sup>nd</sup> sample of ABG

Variables	Present Study (1 hour)	Agarwal et al (1 hour)	S P Rai et al (2 hours)	R.PRASAD et al (2 hours)
pH	7.30±0.1 (>0.05)	7.33± 0.07	7.30 (> 0.05)	7.36±0.034 (0.0002)
PaO <sub>2</sub>	65.1±10.4	61.5±9.7	63.08 (> 0.05)	65.0±18.83
PaCO <sub>2</sub>	64.0±15	56.4±16.5	68.20 (< 0.05)	74.88±17.11

**Table 8:** Comparison of 3<sup>rd</sup> sample of ABG

Variables	Our study (6 hours)	Agarwal et al (4 hours)	S P Rai et al (24 hours)	R.PRASAD et al (24 hours)
pH	7.40 ± 0.1 (<0.01)	7.37 ± 0.07	7.34 (<0.05)	7.48±0.07 (0.0007)
PaO <sub>2</sub>	69.0±18.0 (>0.05)	64.2±7.9	57.17 (<0.05)	72.17±18.59
PaCO <sub>2</sub>	61.0±11.0 (<0.05)	53.6±14.9	59.40 (<0.05)	61.19±10.73

## Discussion

The role of NIV has been studied in various acute respiratory conditions but was found to be more useful as an effective therapeutic modality along with standard treatment in the management of acute exacerbations of COPD. NIV is a cost effective, readily available technique and can be used safely outside the ICU [1]. The advantages of NIV include patient's comfort, preservation of airway defenses like cough, ability to eat and speak. The complications of endotracheal intubation such as nosocomial pneumonias, injury to airways, aspiration and post-intubation laryngeal stenosis can be avoided.

The baseline physiological characteristics of our patients at the onset of NIV were comparable to earlier Indian studies by Agarwal [2] et al, Rai [3] et al & Prasad [4] et al. In our study, the 2<sup>nd</sup> sample of ABG was taken 1 hour after the institution of NIV, where as in the studies by Rai et al and Prasad et al, the 2<sup>nd</sup> sampling of ABG was done at the end of 2 hours. Successful treatment with NIV is associated with an improvement in pH, PaO<sub>2</sub> and PaCO<sub>2</sub> within 1 hour of treatment. If the ABG parameters do not improve,

invasive ventilation should be considered. There was a concern that delay in starting mechanical ventilation in severely ill patients may be harmful. But Conti [5] et al, in their prospective randomized controlled study of NIV versus immediate MV in patients with exacerbation of COPD showed that positive response to early use of NIV in a sicker group of patients is comparable to MV. Results of other studies [6] in more severely ill patients in outpatient setting were not as good as those seen in the ICU studies, suggesting that a NIV trial may be preferable in sick patients admitted in a higher dependency setting where a patient can be immediately switched over to MV, in case NIV fails. Retrospective analyses, uncontrolled studies, and some randomized controlled trials (RCTs) indicate that NPPV can be successfully initiated in the emergency department (ED) [7,8].

In our study, NIV was found to be successful in 72.5% cases causing rapid and sustained improvement in gas exchange in patients with respiratory failure. The overall success rate was similar to that described elsewhere, both from India [2,3,4] and the European-American countries [9,10].

In a prospective randomized placebo controlled trial by Thys et al in 2002 [11], it was found that clinical outcome was better with use of NIV support than with the conventional medical treatment alone. The application of NIV led to a true physiological improvement which could not be explained by placebo effect. Many recent studies have established the role of NIV in decreasing the morbidity and mortality in patients hospitalised for acute exacerbations of COPD [12,13,14].

In general, the factors predicting success of NIV in hypercapnic respiratory failure include pH at admission, pH after one hour of NIV trial and the severity of underlying illness. Short term application of NIV was well studied but very few studies evaluating the long term effectiveness of NIV in COPD with chronic respiratory failure are available.

The improvement in pH and the partial pressures of oxygen and carbon dioxide values of our study is comparable with that of the other studies as shown in Tables 6, 7 and 8. In our study as the 3<sup>rd</sup> sample of ABG was taken 6 hours later, whereas in the study by Agarwal et al the 3<sup>rd</sup> ABG sampling was done at the end of 4 hours. In the studies by Rai et al and R.Prasad et al, the 3<sup>rd</sup> ABG sample was delayed and taken after 24 hours of institution of NIV. Our study shows that 12.5% (n=5) of the patients who did not show initial biochemical response improved clinically proving that in chronic respiratory failure, ABG may take longer time to show improvement. Hence outcome assessment is based on the clinical improvement of the patient irrespective of the biochemical response. The patients who did not show any clinical improvement by the end of 1<sup>st</sup> hour did not improve subsequently thereby indicating that the 1<sup>st</sup> hour of NIV is important in predicting the outcome in COPD patients with respiratory failure. The disadvantages of NIV include slow improvement of blood gases, the need for a conscious and cooperative patient and decreased ability to clear bronchial secretions due to application of facemask. Ventilators specifically designed for NIV with a full face mask as an interface are recommended [15]. There are no absolute contraindications to NIV although a number of them have been suggested. Nebulised bronchodilator therapy should be administered through the ventilator tubing if the patient is feared to go into respiratory distress during breaks of NIV [16]. Agitation and distress are commonly seen in patients with hypercapnic respiratory failures. Few recent studies have shown the effectiveness of mild anxiolytic drugs while on NIV with a caution for respiratory depression [17]. Ventilator-patient asynchrony is commonly encountered which causes increased discomfort and

work of breathing ultimately leading to NIV failure. Increasing the trigger sensitivity and pressure support under continuous monitoring and assurance to the patients are key to successful outcome [18,19]. The optimum duration of NIV have not been extensively studied and normalisation of pH and pCO<sub>2</sub> are usually considered as a guide to weaning. Further studies are required to evaluate the effect of NIV on reducing recurrence and severity of exacerbations of COPD.

Our study had limitations both technical as well as statistic. These include lack of an objective indicator as to when NIV should be discontinued. The relatively small number of patients and lack of a control group had an impact on the statistical analysis of group differences. The patients in the study group were monitored till discharge but the good initial response, however cannot predict long term outcome.

## Conclusions

1. This study provides a strong evidence for the use of NIV (BIPAP) as a first line intervention in patients of acute exacerbations of COPD with respiratory failure. Continuous and efficient monitoring of patient's clinical and ABG status after NIV administration improves the outcome.
2. First hour clinical and biochemical response is a very important factor in the overall outcome. Supplemental oxygen therapy helps in maintaining the oxygen saturation as well as the PaO<sub>2</sub>.
3. Early ABG sampling within one hour after initiating NIV does impact the clinical decision to streamline those who are successful in therapy and can be continued with NIV. Those who do not improve should be immediately considered for invasive ventilation, so that any adverse outcome due to delay in ventilatory support can be averted.
4. NIV can be safely administered in an emergency room with monitoring facilities and trained nursing staff.

## References

1. Elliott MW, Confalonieri M, Nava S. Where to perform NIV? *Eur Respir J* 2002;19:1159-66.
2. Agarwal R, Gupta R, Aggarwal AN and Gupta D. Non-invasive positive pressure ventilation in acute respiratory failure due to COPD vs other causes:

- Effectiveness and predictors of failure in a respiratory ICU in North India. *Int J COPD* 2008;3:737-43.
3. Rai SP, Panda BN, Upadhyay KK. Non invasive positive pressure ventilation in patients with acute respiratory failure. *MJAFI* 2004;60:224-6.
  4. Prasad R, Rout A, Garg R, Kushwaha RAS and Ahuja RC. An open randomized controlled trial of Non-invasive positive pressure ventilation in patients of acute on chronic hypercapnic respiratory failure in a general respiratory ward setting. *Lung India* 2007; 24:132-8.
  5. Conti G, Antonelli M and Navalesi P. Noninvasive vs. conventional mechanical ventilation in patients with chronic obstructive pulmonary disease after failure of medical treatment in the ward: a randomized trial. *Intensive Care Med* 2002;28:1701-7.
  6. Plant PK, Owen JL and Elliott MW. Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease in general respiratory wards: a multicentre randomised controlled trial. *Lancet* 2000;355:1931-5.
  7. Wysocki M, Tric L, Wolff MA, Millet H, Herman B. Noninvasive pressure support ventilation in patients with acute respiratory failure: a randomized comparison with conventional therapy. *Chest* 1995;107:761-768.
  8. Kramer N, Meyer TJ, Meharg J, Cece RD, Hill NS. Randomized, prospective trial of noninvasive positive pressure ventilation in acute respiratory failure. *Am J Respir Crit Care Med* 1995;151:1799-1806.
  9. Brochard L, Mancebo J, Wysocki M, Lofaso F, Conti G, Conway J, et al. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. *N Engl J Med* 1995;333:817-22.
  10. Ambrosino N, Foglio K, Rubini F, Clini E, Nava S and Vitacca M. Non-invasive mechanical ventilation in acute respiratory failure due to chronic obstructive pulmonary disease: correlates for success. *Thorax* 1995;50:755-7.
  11. F Thys, J. Roeseler and M. Reynaert. NIV for acute respiratory failure *ERJ* 2002;20:545-55.
  12. Elliot MW, Nava S. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease: "Don't think twice, it's alright!". *Am J Respir Crit Care Med* 2012;185(2):121-3.
  13. Chandra D, Stamm JA, Taylor B, et al. Outcomes of noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease in the United States, 1998-2008. *Am J Respir Crit Care Med* 2012; 185(2):152-9.
  14. Lindenauer PK, Stefan MS, Shieh MS, Pekow PS, Rothberg MB, Hill NS. Outcomes associated with invasive and noninvasive ventilation among patients hospitalized with exacerbations of chronic obstructive pulmonary disease. *JAMA Intern Med* 2014;174(12):1982-93.
  15. Davidson AC, Banham S, Elliott M, et al. BTS/ICS guideline for the ventilatory management of acute hypercapnic respiratory failure in adults. *Thorax* 2016;71:ii1-ii35.
  16. Abdelrahim ME, Plant P, Chrystyn H. In-vitro characterisation of the nebulised dose during non-invasive ventilation. *J Pharm Pharmacol* 2010; 62:966-72.
  17. Senoglu N, Oksuz H, Dogan Z, et al. Sedation during noninvasive mechanical ventilation with dexmedetomidine or midazolam: a randomized, double-blind, prospective study. *Curr Ther Res Clin Exp* 2010;71:141-53.
  18. Epstein SK. How often does patient-ventilator asynchrony occur and what are the consequences? *Respir Care* 2011;56:25-38.
  19. de Wit M, Miller KB, Green DA, et al. Ineffective triggering predicts increased duration of mechanical ventilation. *Crit Care Med* 2009;37:2740-5.
-

## The Study of the Clinical Profile and Laboratory Parameters of Acute Neonicotinoid Compound Poisoning at a Rural Tertiary Care Public Hospital in Central India

Mundhe Sanjay A.<sup>1</sup>, Birajdar Siddheshwar V.<sup>2</sup>, Chavan Sheshrao S.<sup>3</sup>, Kendre Vitthal M.<sup>4</sup>

### Author's Affiliation:

<sup>1</sup>Assistant Professor <sup>2</sup>Professor  
<sup>3</sup>Associate Professor, Department of Medicine, Swami Ramanand Tirth Rural Government, Medical College, Ambajogai, Maharashtra 431517, India. <sup>4</sup>Resident Doctor, Department of Pathology, GS Medical College and KEM Hospital, Mumbai, Maharashtra 400012, India.

### Corresponding Author:

**Birajdar Siddheshwar V.**,  
Professor, Department of Medicine, Swami Ramanand Tirth Rural Government Medical College, Ambajogai, Maharashtra, India, 431517.  
E-mail: drsanjaymundhe@gmail.com  
**Received on** 09.10.2017,  
**Accepted on** 23.10.2017

### Abstract

*Context:* Pesticide exposures are common health issues in India. Traditionally used pesticides like organophosphates are associated with higher morbidity and mortality. Neonicotinoids are newer class of effective and safer insecticides. However, literature of human exposures is very limited. *Aims:* To study clinical profile, laboratory features and factors associated with mortality after acute human neonicotinoid exposures. *Settings and Design:* This retrospective observational study was performed at department of general medicine in rural tertiary care public hospital. *Methods and Material:* Necessary data of admitted eligible cases of acute neonicotinoid poisoning during five year period of January 2012 to December 2016 were retrieved from medical record section and were analysed. *Statistical Analysis:* Statistical analyses were performed by using Graph pad prism 5. The incidence of Clinical findings, 95% confidence interval, relative risk, and baseline characteristics of patients were calculated by Wilcoxon rank sum test and chi square test. Statistical significance was established at  $p < 0.05$  and RR values were considered statistically significant if 95% of CI excluded 1%. *Results:* A total of 141 cases were analyzed. Most exposures involved oral intentional consumptions of Imidacloprid. Clinical manifestations of acute neonicotinoids exposures involved variety of body systems. Severe/fatal cases had higher proportion of respiratory, neurological and cardiovascular manifestations and variety of laboratory and ECG finding. Although most exposures were asymptomatic or non-severe poisoning, 26 cases had severe poisoning with five deaths. *Conclusions:* Even though considered as relatively safer insecticides, large intentional consumption can lead to severe poisoning and even death. Supportive treatment is usually sufficient and severe poisoning needs intensive case.

**Keywords:** Imidacloprid; Insecticide; Neonicotinoid; Poisoning.

### Introduction

Acute pesticide poisonings are among common healthcare issues in India, particularly in settings of low education and poor regulatory frameworks. Among pesticides, highly toxic organophosphates are commonly used and are associated with high morbidity and preventable mortality [1]. Neonicotinoids are newer insecticides that are effective

for crop protection, flea control in agricultural and domestic settings [2]. They act on postsynaptic nicotinic acetylcholine receptors (nAChRs) by displacing acetylcholine [2]. Because of relative specificity for target insects, lower risk for non-target organisms, versatility in application and no cross-resistance to other insecticides, they are becoming popular in recent years [2-4]. These are classified as "moderately hazardous" (Class II WHO; toxicity category II EPA) [5,6]. There are reports which describe

cardiac, neurological, pulmonary, renal, multiorgan failure and death as their exposures [7-11]. Despite increasing use, literature about acute human poisonings is limited to few studies & case reports [12-14]. So, we planned study with objective to study different clinical features, laboratory changes and factors associated with mortality with these neonicotinoids. We hope, this information will help in risk assessment and clinical management of acute neonicotinoids exposures and also help concerned regulatory agencies to decide policies regarding their safe use.

## Subjects and Methods

This retrospective observational study was carried out at Rural Tertiary Care Public Hospital in Marathwada region of Maharashtra, India. All patients of neonicotinoid poisonings, who were admitted to our hospital during period of January 2012 to December 2016, were identified from hospital records and were considered for study. Study was approved by institutional ethics committee of our hospital. Patient who had history of exposure to neonicotinoids like Imidacloprid, Acetamiprid, Clothianidin, Thiacloprid, Dinotefuran, Nitenpyram or Thiamethoxam and who was admitted to hospital was defined as neonicotinoid poisoning. Cases that consumed other insecticide, discharged against medical advice, age less than 12 yrs and with incomplete records were excluded from study. The records of all patients of neonicotinoid poisonings admitted during study period were obtained from records section of our hospital. Cases which fulfilled inclusion & exclusion criteria were selected and data regarding demographic profile, clinical features, details of compound exposed, elapsed time, laboratory parameters, complications, treatment received and outcomes were recorded. Clinical features were grouped according to various organ systems. Gastrointestinal effects were defined by symptoms like nausea, vomiting, abdominal pain, gastroesophageal bleeding & odynophagia, central nervous system effects were dizziness, drowsiness, seizures, mydriasis and unconsciousness. Cardiovascular effects included palpitations, chest pain & hypotension. Respiratory effects were sore throat, breathlessness, respiratory failure & aspiration pneumonia. Clinical presentations were classified as "non-severe" and "severe" as per American Association of Poison Control Center data collection system [14]. Patients with manifestations that were potentially life-threatening or caused death (e.g.

seizures, respiratory failure, ventricular tachycardia, hypotension, cardiac or respiratory arrest, haematemesis) were categorized as severe. All other presentations were categorized as non-severe. Data collected from medical records were compiled using excel sheet and analysed with Graph pad prism 5. Descriptive statistical method was used to describe frequencies and percentages for categorical data. Statistical analysis was performed to evaluate distribution of baseline characteristics and clinical features between male and female cases. To assess parameters associated with severity, we compared demographic, clinical and laboratory findings between severe/fatal and non-severe cases by Wilcoxon rank sum test for continuous variables and either chi-square test or Fisher's exact test for categorical variables. Death rates for various insecticides were evaluated for statistical significance by calculating ratio of rate for neonicotinoids to rate for other insecticides (rate ratio, RR) and 95% confidence interval (CI) by Newcombe-Wilson method without continuity correction. RRs were considered statistically significant if 95% confidence interval excluded 1.00. Elsewhere, p-value of less than 0.05 was considered statistically significant.

## Results

Total 141 cases of acute neonicotinoid exposures, which qualified inclusion and exclusion criteria, were studied. Among the cases, males were 105 (74.46%) and females were 36 (25.54%) (Table 1). During year 2012-2013, there were ten and 18 cases respectively. Number of cases increased after 2014 and there were 31, 34 and 48 cases for these respective years. Median age of cases was 41 years for males (13-64 year) and 29 years for females (12-77 year). Exposure involved oral ingestion in 89 (63.12%) cases, 14 (9.93%) had inhalational contact, nine (6.38%) had dermal and 29 (20.57%) had mixed exposures (Table 1). Reason for exposure was intentional consumption in 83 (58.87%) cases while remaining 58 (41.13%) had accidental exposures and 46 (43.81%) had alcohol co-ingestion, all males. Out of 141 cases, 53 (37.59%) were asymptomatic, 62 (43.97%) had symptomatic & non-severe poisoning while 26 (18.44%) had severe/fatal poisoning with five (3.55%) deaths. There was no significant difference in male and females for year of exposure, route of exposure, reason for exposure and severity of poisoning. However, males had significantly higher age and number of alcohol co-ingestion than females. Most commonly observed neonicotinoid compound was Imidacloprid, reported



**Table 1:** Distribution of baseline characteristics of the cases with acute neonicotinoid exposures in the study

Characteristics	Males N (%) 105 (74.46)	Females N (%) 36 (25.54)	P-value
<b>Calendar year of poisoning</b>			
2012	7 (6.66)	3 (8.33)	0.854
2013	15 (14.28)	3 (8.33)	
2014	24 (22.85)	7 (19.44)	
2015	25 (23.81)	9 (25.00)	
2016	34 (32.38)	14 (38.89)	
<b>Age (median and range, years)</b>	41 (13-64)	29 (12-77)	0.013
<b>Reason of exposure</b>			
Intentional	62 (59.04)	21 (58.33)	0.940
Accidental	43 (40.95)	15 (41.67)	
<b>Route of exposure</b>			
Oral	67 (63.81)	22 (61.11)	0.975
Inhalational	10 (9.52)	4 (11.11)	
Dermal	7 (6.67)	2 (5.55)	
Non-oral Mixed	21 (20.00)	8 (22.22)	
<b>Simultaneous alcohol intake.</b>	46 (43.81)	0 (0)	
<b>Severity</b>			
Asymptomatic	34 (32.38)	19 (52.78)	0.189
Symptomatic & non-severe	49 (46.67)	13 (36.11)	
Symptomatic & severe	18 (17.14)	3 (8.33)	
Death	4 (3.81)	1 (2.78)	

**Table 2:** Distribution of the individual compounds and various clinical features among all acute neonicotinoid exposures in the study

Neonicotinoid compound	Number (n)	Percentage (%)
Imidacloprid	108	76.60
Acetamiprid	9	6.38
Thiamethoxam	9	6.38
Clothianidin	6	4.26
Dinotefuran	4	2.83
Nitenpyram	3	2.12
Thiacloprid	2	1.42
total	141	100
Clinical features	Number (n)	Percentage (%)
Nausea	77	54.61
Vomiting	59	41.84
Sore throat	42	29.79
Abdominal pain	42	29.79
Chest pain	24	17.02
Dizziness	23	16.31
Odynophagia	22	15.60
Dermal irritation	19	13.48
Ocular irritation	17	12.06
Breathlessness	17	12.06
Drowsiness	15	10.64
Respiratory failure	15	10.64
Palpitations	13	9.22
Unconsciousness	9	6.38
Gastroesophageal bleeding	9	6.38
Hypotension	8	5.67
Aspiration pneumonia	6	4.25
Seizures	3	2.13
Mydriasis	2	1.42
Rhabdomyolysis	1	0.71

in 108 (76.60%) cases, followed by Acetamiprid & Thiamethoxam, each nine cases (6.38%) and exposures with Clothianidin (4.26%), Dinotefuran (2.83%), Nitenpyram (2.12%) & Thiacloprid (1.42%) were less commonly reported (Table 2). Among cases, variety of clinical features involving gastrointestinal, cardiovascular, respiratory, nervous, renal system and other local effects involving eyes and skin were observed (Table 2). To find out various factors associated with development of severe/fatal poisoning, we classified 141 cases into severe/fatal poisoning and non-severe poisoning (Table 3). All of 26 cases with severe/fatal poisoning had exposure to imidacloprid. Out of these 26 cases, 25 had oral ingestion (96.15%) while one had mixed inhalational & dermal exposure (55.65%) and this difference was significant. Other parameters like older age (39 vs 32), delay for treatment (2.5 vs 1.5), systemic manifestations of Gastrointestinal system

(88.46% vs 35.06%), Cardiovascular system (76.92% vs 7.83%), nervous system (73.08 % vs 15.65%), Respiratory system (69.23% vs 28.70%), abnormal laboratory findings (50.00% vs 0%) and alcohol use (76.92% vs 22.61%) were significantly more common in severe/fatal poisoning (Table 3). Varieties of ECG findings were noted in study cases. (Table 4). The ECG was abnormal in 21 (80.77%) cases of severe/fatal poisoning while three (2.60%) cases of non-severe exposures had abnormal ECG and this difference was significant. Table 5 compares fatal outcome rate of Neonicotinoids to other insecticides poisonings reported to our hospital. Majority of patients (96.45%) recovered and were discharged; however, five cases died. The proportion of deaths for neonicotinoids was 3.55%, which is significantly lower than Organophosphates (15.56%), Organochlorides/Carbamates (17.18%) and Herbicides (10.16%), which are commonly used

**Table 3:** Comparison of demographic and clinical characteristics between patients with severe/fatal and non-severe neonicotinoid insecticide exposures in the study

Characteristics	Severe/fatal poisoning n=26 (%)	Non-severe poisoning n=115 (%)	p-value
<b>Mean Age (years)</b>	39 (19-54)	32 (12-77)	0.049
<b>Delay for medical treatment (h)</b>	2.5 (0.5-9)	1.5 (0.25-9.5)	0.034
<b>Gastrointestinal effects</b>	23 (88.46)	65 (35.06)	0.0002
Nausea	21 (80.77)	56 (48.70)	0.0002
Vomiting	20 (76.92)	39 (33.91)	0.0001
Abdominal pain	10 (38.46)	32 (27.82)	0.284
Gastroesophageal bleeding	9 (34.61)	0 (0)	0.0001
Odynophagia	5 (19.23)	17 (14.78)	0.575
<b>Cardiovascular effects</b>	20 (76.92)	9 (7.83)	0.0001
Palpitations	9 (34.61)	4 (3.48)	0.0001
Chest pain	17 (65.38)	7 (6.09)	0.0001
hypotension	8 (30.77)	0 (0)	0.0001
<b>Respiratory tract effects</b>	18 (69.23)	33 (28.70)	0.0001
Sore throat	9 (34.62)	33 (28.70)	0.548
Breathlessness	16 (61.54)	1 (0.87)	0.0001
Respiratory Failure (type 1 & 2)	15 (57.69)	0 (0)	0.0001
Aspiration/ ventilator associated pneumonia	6 (23.07)	0 (0)	0.0001
<b>Central nervous system effects</b>	19 (73.08)	18 (15.65)	0.0001
Dizziness	5 (19.23)	18 (15.65)	0.652
Drowsiness	15 (57.69)	0 (0)	0.0001
Seizures	3 (11.54)	0 (0)	0.0002
Unconsciousness	9 (34.61)	0 (0)	0.0001
Mydriasis	2 (7.69)	0 (0)	0.0027
<b>Other effects</b>	6 (23.07)	23 (20.00)	0.726
Ocular irritation	3 (11.54)	14 (12.17)	0.928
Dermal irritation	4 (15.38)	15 (13.04)	0.748
<b>Abnormal ECG findings</b>	21 (80.77)	3 (2.60)	0.0001
<b>Laboratory findings</b>	13 (50.00)	0 (0)	
Hypokalemia	8 (30.77)	0 (0)	0.0001
Renal failure	2 (7.69)	0 (0)	0.0027
Abnormal liver enzymes	5 (19.23)	0 (0)	0.0001
Metabolic acidosis	3 (11.54)	0 (0)	0.0002
<b>Alcohol intake</b>	20 (76.92)	26 (22.61)	0.0001
<b>Route of exposure</b>			
oral	25 (96.15)	64 (55.65)	0.0001
Non-oral	1 (3.85)	51 (44.35)	0.0001

**Table 4:** Various ECG findings noted and Treatment modality used among the patients with acute neonicotinoid exposures in the study

ECG finding	Number (n)	Percentage (%)
Normal ECG	86	60.99
Sinus tachycardia	31	21.99
ST-T Changes	9	6.38
Prolonged QTc interval	5	3.55
Atrial fibrillation	4	2.84
Sinus Bradycardia	3	2.13
Ventricular tachycardia	1	0.71
Ventricular ectopic	2	1.42

Treatment modality used	Number (n)	Percentage (%)
Decontamination	129	91.49
H2 Antihistamines Or Proton Pump Inhibitors Or Antiemetics	65	46.10
IV Fluids	49	34.75
Bronchodilators	21	14.89
Oxygen	19	13.48
Antibiotics	14	9.93
Ventilatory Support	14	9.93
Anti-Convulsant/ Sedative Drugs	10	7.09
Potassium Chloride	8	5.67
Inotropes	6	4.26
Atropine & Pralidoxime	5	3.55
Anti-Arrhythmic Drugs	4	2.84
Blood Transfusion	3	2.13
DC Shock	3	2.13
No treatment	12	8.51

**Table 5:** Death rates for neonicotinoid and other insecticides exposures during the study period

Insecticide	Total	Death (N)	Death rate (%)	RR	95% CI
Neonicotinoids	141	5	3.55	-	-
Organophosphates	842	131	15.56	0.25	0.10-0.61
organochlorines/ carbamates	390	67	17.18	0.26	0.11-0.61
Pyrethroids	423	6	1.42	1.8	0.93-3.52
Herbicides	305	31	10.16	0.43	0.19-0.98

insecticides in our region. Treatments modalities used were recorded and treatment received was all symptomatic and supportive (Table 5).

## Discussion

In this study, we studied cases of acute neonicotinoid poisoning for period of January 2012 to December 2016. There was gradual increase in number of cases from 2012 to 2016 with most cases occurring in year 2016. This observation suggests that these compounds are becoming popular and are being used increasingly in recent years and number of acute human exposures might increase in future [2,4]. This observation is similar to earlier studies by Phua et al and Forrester who also described increasing trend

number of cases [5,12]. Imidacloprid (76.60%) was most commonly reported, followed by Acetamiprid & Thiamethoxam (each 6.38%) while Thiocloprid (1.42%) was least common. Retrospective analyses of poison control center data by Forrester (76.5%) & Phua et al (90%) and prospective observational cohort study by Mohamed et al, reported similar observation regarding Imidacloprid to be the most commonly exposed neonicotinoid [5,6,12]. Lin et al, in review concluded that Imidacloprid was major poison among Neonicotinoids, which constituted 94% of intoxication events [15]. We conclude that widespread use and easy availability were reasons that most patients were exposed to Imidacloprid [16]. We noted more number of intentional oral consumptions than accidental inhalational and/or dermal exposures. This is in contrast to study by Forrester where majority of exposures were unintentional and below 2% were

intentional [12]. However, study by Phua et al reported 69% cases of suicides and study by Mohamed et al reported 89.71% cases of intentional poisoning [5,6]. Nearly all reported case studies with both fatal and favourable outcomes, reported intentional consumptions as cause of exposure [15]. These differences might be due to different study criteria, poison center operations, or types of reported exposures. In our study, majority of cases were males (74.46%) while rest were females. These observations were similar to Taiwan study where males were majority while UK study had evenly distributed male and females [5,17]. However, in study by Forrester, female were more than males [12]. These differences can be due to differences in cases reported to poison centres and various socio-demographic background. Majority of our cases (62.41%) were symptomatic while 37.59% cases were asymptomatic. Neonicotinoids have agonistic action at nAChRs; their toxic effects, therefore, may be similar to nicotine. Activation of nicotinic receptors by nicotine classically shows a biphasic clinical pattern with initial stimulation followed by inhibition [2,5,18]. The most commonly reported clinical features in our study were gastrointestinal with variable degree of respiratory, neurological, cardiovascular, ocular, dermal & other symptoms and were consistent with available literature of acute exposures [5-12,15,19-21]. In our study, severe/fatal poisoning were observed in 26 cases (18.44%) with five (3.55%) deaths. In study by Forrester, having 1,142 exposures with more than 98% unintentional exposures, only 32 (2.9%) resulted in serious outcomes with no deaths [12]. Another study by Phua et al with total 46 exposures, reported ten cases (21.74%) of severe poisoning with two (4.35%) death [5]. Mohammad et al studied 68 cases of intentional Imidacloprid poisoning and reported severe features requiring intensive care in two cases (2.94%) and no deaths [6]. Double-blind crossover study of 19 planters by Elfman reported no adverse effects with Imidacloprid [22]. Lin et al observed severe manifestations in 22 cases and six deaths from total 66 cases having detailed clinical records [15]. The differences in proportion of cases having severe/fatal outcome in these studies could be due to differences in study design, types of exposures reported, differences in exposure assessment methods and different definitions used to define severe poisoning. The definition used in our study and by Phua et al was similar and proportion of severe cases (18.44% vs 21.74%) and fatal outcome (3.55% vs 4.35%) were almost similar [5]. Moreover, fatal outcome rate for neonicotinoids (3.55%) was significantly lower than carbamate/organochlorines (17.18%), organophos-

phates (15.56%) and herbicides (10.16%), however, it was marginally but not significantly more than pyrethroids (1.42%). This is consistent with other three poison center investigations. Study by Adams et al observed that Neonicotinoids have less serious medical outcomes than pyrethroids and carbamates [17]. In study by Phua et al, mortality for Neonicotinoids was lower than organophosphates and carbamates but was similar to Pyrethroids [5]. In study by Forrester, serious outcome rate for neonicotinoid insecticides was substantially lower than carbamates/chlorinated hydrocarbon/organophosphates and pyrethroids [12]. Therefore, it can be proposed that acute exposures of neonicotinoids are relatively safer than other insecticides. This finding can be explained by their selective action at insect nAChRs and high water solubility reducing ability to penetrate mammalian blood-brain barrier rendering them less toxic to CNS [2,23,24]. However, it must be remembered that severe toxic effects and even death have occurred following acute neonicotinoid exposures, especially following large ingestions [5,7-8,10,11,13,15,21,25]. All cases with major severity or death in our study were exposed to Imidacloprid alone. This may be related to fact that Imidacloprid was most frequently encountered neonicotinoid in study and it is expected to be more toxic than other Neonicotinoids because of higher selectivity of other neonicotinoids [2]. However, it is worth to note that Neonicotinoids like Acetamiprid and Thiacloprid can cause severe poisoning and even death [9,13,15,21,26]. Average age of severe/fatal poisoning group was significantly higher than that of non-severe group. Phua et al and Lin et al also noted similar observations with older patients having more severe poisoning [5,14,15]. Inhalational and dermal exposures were significantly associated with non-severe poisonings and oral exposures with severe/fatal poisonings, a finding consistent with studies from Sri Lanka, Taiwan and review by Lin et al [5,6,15]. We noted higher proportion of severe/fatal outcomes in males than females but this difference was not significant. This finding might be due to fact that study had higher proportion of males having oral ingestions. Patients with co-ingestion of alcohol had significantly higher proportion of severe poisoning and four of five cases with fatal outcome had consumed alcohol. In study by Mohamed et al, prolonged sedation and respiratory depression was noted in two patients who had co-ingestion of ethanol [6]. There are case reports of severe/fatal poisoning with co-ingestion of alcohol and Neonicotinoids. Yeh et al reported case of ingestion of alcohol with Imidacloprid and manifestations included disorientation, bradycardia,

ventricular arrhythmia, and cardiopulmonary arrest [7]. However, we could not find any study assessing role of alcohol co-ingestion in severity of neonicotinoid poisoning and thus, warrants further research. We found that different symptoms like abdominal pain, odynophagia, sore throat, dizziness, eye and skin irritation occurred equally in both severe/fatal and non-severe groups and there was no significant difference. Conversely, majority of respiratory, cardiovascular and neurological symptoms occurred more commonly in severe/fatal group and these differences were significant. Study by Phua et al & Mohamed et al noted similar observations and proposed that coma, respiratory depressions, respiratory muscle weakness, cardiac arrhythmia and aspiration pneumonia are associated with severe/fatal cases [5,6]. Lin et al in a review noted that respiratory, cardiovascular and some neurological symptoms occurred more commonly in severely intoxicated patients and meticulous observation is indicated in neonicotinoid-poisoned patients presenting with these warning signs [15]. We could study different ECG findings in cases of acute neonicotinoid exposures and noted that ECG was either normal or had sinus tachycardia in majority of cases. We observed abnormal ECG findings like ST-T changes, prolonged QTc, atrial fibrillation, sinus bradycardia, ventricular ectopic, and ventricular tachycardia in order of frequency of occurrence. Except for fatal ventricular tachycardia in one patient and atrial fibrillation in other, most of ECG changes were reversible. We could not find any literature which studied different ECG findings in acute neonicotinoid poisoning. Few case reports have noted abnormal ECG findings. Huang et al reported case of fatal ventricular fibrillation following ingestion of Imidacloprid compound which was refractory to DC shock and IV anti-arrhythmics [8]. Yeh et al reported case of fatal ventricular tachycardia following ingestion of imidacloprid and alcohol [7]. Case report by Todani et al reported atrial fibrillation lasting for 11 hours with Acetamiprid poisoning [26]. Here, we can conclude that life threatening arrhythmias do occur with neonicotinoid poisoning and can be fatal. The cause of arrhythmias can be multifactorial including activation of autonomic system with resultant coronary spasm & cardiac ischemia, hypoxia, electrolyte imbalance, direct toxic effects on myocardium and alcohol co-ingestion. There is no specific antidote for neonicotinoid poisoning in humans [2]. Treatment given to cases in our study was mainly supportive, that involved decontamination, administration of H2 antihistamines/proton pump inhibitors/antiemetic drugs, fluids, antibiotics,

oxygen, bronchodilators, DC shock, anti-arrhythmics, potassium chloride, ventilatory support, blood transfusion, atropine & pralidoxime, anti-convulsants/sedatives and inotropic agents. Review of available literature demonstrated similar findings and treatment given was mainly supportive [5,6,12,13,15].

We noted use of atropine and pralidoxime in few cases where clinical features were similar to organophosphate poisoning and were misdiagnosed initially in unavailability of compound details on presentation, which later turned out to be Imidacloprid poisoning. Similarly, there are descriptions of Imidacloprid poisoning getting misdiagnosed as organophosphate poisoning due to similar manifestations and were given treatment with atropine and pralidoxime [5,9,27]. Oximes in absence of organophosphate poisoning have inhibitory effect on acetylcholinesterase activity and therefore, might increase nicotinic effects [6]. Thus, treatment with oxime in neonicotinoid poisoning might be ineffective and may be contraindicated. Mohamed et al noticed that two most seriously poisoned cases received treatment with pralidoxime [6]. Therefore, it can be said that poisoning with Neonicotinoids should be considered in differential diagnosis of patients having features suggestive of organophosphate poisoning and use of pralidoxime should be avoided in these cases.

#### *Limitations*

Being a hospital based retrospective study of admitted cases of only neonicotinoids, out of hospital deaths, combinations with other insecticides and cases not admitted, were likely to be missed. Although, we accessed key data of most patients, accurate information on exact timing, elapsed time before treatment and minor clinical information may be incomplete. We could not measure exposed quantity, solvent present in preparations and blood levels of insecticides. In our study, majority of exposures were due to imidacloprid, so evaluation may miss differences in clinical presentations for other neonicotinoids due to their limited number.

#### **Acknowledgement**

Authors are thankful to Mr. Nitin A. Mundhe for his assistance in statistical analysis of data and incharge and staff of medical record section of our hospital for their co-operations.

### Key Messages

Neonicotinoids, being used increasingly, their human exposures tend to increase in future. Though, they have specific mode of action on insects and considered less toxic to humans, can cause death, especially after intentional Imidacloprid consumptions. Treatment is Supportive and severe poisoning with respiratory, nervous & cardiovascular manifestations needs intensive case.

### References

1. Eddleston M, Phillips MR. Self poisoning with pesticides. *BMJ* 2004;328:42-44.
2. Tomizawa M, Casida JE. Neonicotinoid insecticide toxicology mechanisms of selective action. *Annu Rev Pharmacol Toxicol*. 2005;45:247-268.
3. Nauen R, Denholm I. Resistance of insect pests to neonicotinoid insecticides: current status and future prospects. *Arch Insect Biochem Physiol* 2005;58: 200-15.
4. Elbert A, Haas M, Springer B, Thielert W, Nauen R. Applied aspects of neonicotinoid uses in crop protection. *Pest Manag Sci* 2008;64:1099-105.
5. Phua DH, Lin CC, Wu ML, Deng JF, Yang CC. Neonicotinoid insecticides: an emerging cause of acute pesticide poisoning. *Clin Toxicol (Phila)* 2009; 47:336-41.
6. Mohamed F, Gawarammana I, Robertson TA, Roberts MS, Palangasinghe C, Zawahir S, et al. Acute human self poisoning with imidacloprid compound: a neonicotinoid insecticide. *PLoS One* 2009;4:e5127.
7. Yeh IJ, Lin TJ, Hwang DY. Acute multiple organ failure with imidacloprid and alcohol ingestion. *Am J Emerg Med* 2010;28:255.e1-3.
8. Huang NC, Lin SL, Chou CH, Hung YM, Chung HM, Huang ST. Fatal ventricular fibrillation in a patient with acute imidacloprid poisoning. *Am J Emerg Med*. 2006;24:883-885.
9. Imamura T, Yanagawa Y, Nishikawa K, Matsumoto N, Sakamoto T. Two cases of acute poisoning with acetamiprid in humans. *Clin Toxicol (Phila)* 2010; 48:851-3.
10. Iyyadurai R, George IA, Peter JV. Imidacloprid poisoning—newer insecticide and fatal toxicity. *J Med Toxicol* 2010;6:77-8.
11. Shadnia S, Moghaddam HH. Fatal intoxication with Imidacloprid insecticide. *Am J Emerg Med*. 2008;26:634.
12. Forrester MB. Neonicotinoid insecticide exposures reported to six poison centers in Texas. *Hum Exp Toxicol* 2014;33:568-573.
13. Cimino AM, Boyles AL, Thayer KA, Perry MJ. Effects of Neonicotinoid Pesticide Exposure on Human Health: A Systematic Review. *Environ Health Perspect* 2016.
14. Yang CC, Wu JF, Ong HC, Hung SC, Kuo YP, Sa CH, et al. Taiwan National Poison Center: epidemiologic data 1985-1993. *J Toxicol Clin Toxicol* 1996;34:651-63.
15. Lin PC, Lin HJ, Liao YY, Guo HR, Chen KT. Acute Poisoning with Neonicotinoid Insecticides: A Case Report and Literature Review. *Basic Clin Pharmacol Toxicol* 2013;112:282-6.
16. Jaschke P, Nauen R, Schindler M et al. Overview of the status and global strategy for neonicotinoids. *J Agric Food Chem* 2011;59:2897-908.
17. Adams RD, Perry L, Bennett A. The NPIS pesticide surveillance project neonicotinoids: comparison of toxicity against other insecticide classes. *Clin Toxicol* 2013;51:353.
18. David D, George IA, Peter JV. Toxicology of the newer neonicotinoid insecticides: imidacloprid poisoning in a human. *Clin Toxicol (Phila)* 2007; 45:485-486.
19. Kumar A, Verma A, Kumar A. Accidental human poisoning with a neonicotinoid insecticide, imidacloprid: a rare case report from rural India with brief review of literature. *Egyptian J Forensic Sci* 2013; 3:123-126.
20. Agarwal R, Srinivas R. Severe neuropsychiatric manifestations and rhabdomyolysis in a patient with imidacloprid poisoning. *Am J Emerg Med* 2007;25:844-5.
21. Vinod KV, Srikant S, Thiruvikramaprakash G, Dutta TK. A fatal case of thiacloprid poisoning. *Am J Emerg Med*. 2015;33:310.
22. Elfman L, Hogstedt C, Engvall K, Lampa E, Lindh CH. Acute Health Effects on Planters of Conifer Seedlings Treated with Insecticides. *Ann Occup Hyg* 2009;53:383-390.
23. Wu IW, Lin JL, Cheng ET. Acute poisoning with the neonicotinoid insecticide imidacloprid in *N*-methyl pyrrolidone. *J Toxicol Clin Toxicol* 2001;39:617-621
24. Vale JA. Poisoning due to neonicotinoid insecticides. *Clin Toxicol* 2008;46:404-405.
25. Proenca P, Teixeira H, Castanheira F, Pinheiro J, Monsanto PV, Marques EP, et al. Two fatal intoxication cases with imidacloprid: LC/MS analysis. *Forensic Sci Int* 2005;153:75-80.
26. Todani M, Kaneko T, Hayashida H, Kaneda K, Tsuruta R, Kasaoka S, et al. Acute poisoning with neonicotinoid insecticide acetamiprid. *Chudoku Kenkyu*. 2008;21:387-90.
27. Hung YM, Lin SL, Chou KJ, Chung HM. Imidacloprid-*n*-methyl pyrrolidone insecticides poisoning mimicking cholinergic syndrome. *Clin Toxicol (Phila)* 2006;44:771-772.

# Role of Intravenous Magnesium Sulphate in Predicting Outcomes of ICU in Acute Organophosphate Poisoning

Sri Harsha J.<sup>1</sup>, Srinivas Prabhu N.C.<sup>2</sup>, Ronak M. Raheja<sup>3</sup>, O.R. Ranjan<sup>1</sup>

## Author's Affiliation:

<sup>1</sup>Post Graduate <sup>2</sup>Professor and HOD <sup>3</sup>Under Graduate, Department of Emergency Medicine, Kempegowda Institute of Medical Sciences, Bengaluru, Karnataka 560004, India.

## Corresponding Author:

Sri Harsha J., Post Graduate, Department of Emergency Medicine, Kempegowda Institute of Medical Sciences, Bengaluru, Karnataka 560004, India.  
E-mail: [drnakraheja@gmail.com](mailto:drnakraheja@gmail.com)

Received on 10.06.2017,

Accepted on 28.06.2017

## Abstract

Organophosphorus chemicals (OPs) are the pesticides most often involved in serious human poisoning in developing countries like India. Treatment of intoxication with OPs conventionally involves atropine for reduction of muscarinic signs and oximes that increase the rate of hydrolysis of the phosphorylated enzyme acetylcholinesterase (AChE). Although oximes (pralidoxime or obidoxime) are traditionally considered specific antidotes and used in the management of such poisoning, their efficacy remains a major issue of debate. Thus, the goal of this clinical study was to elaborate the value of magnesium sulfate (MgSO<sub>4</sub>) in the management and outcome of acute OP insecticide poisoning. This unicenter, randomized trial study was conducted on patients who were acutely poisoned with OPs and admitted to Kempegowda Institute of Medical Sciences & Hospital. In this study patients were randomly divided into 2 groups (25 patients each). Control group and test group. Control group received conventional management with injection atropine and injection PAM while the test group in addition to above received intravenous Magnesium sulphate. Magnesium sulphate was administered at dose of 4 g/day intravenous infusion over 4 hours within first 24 hours after ingestion. There was a significant decrease in number of days of ventilation ( $z=-2.1$ ,  $p=0.04$ ) and days of ICU stay ( $z=-4.1$ ,  $p<0.001$ ) on independent Mann Whitney Tests in patients who received MgSO<sub>4</sub> than those who had not received MgSO<sub>4</sub>. The mortality rate, total atropine required and hospitalization days of patients who received MgSO<sub>4</sub> treatment were significantly lower than those who had not received MgSO<sub>4</sub> ( $P=<0.05$ ). It is concluded that administration of MgSO<sub>4</sub>, in a dose of 4 g/day concurrent to conventional therapy, in OP acute human poisoning is beneficial by reducing the hospitalization days and rate of mortality.

**Keywords:** Human; Magnesium Sulphate; MgSO<sub>4</sub>; Organophosphate Poisoning; OP; Treatment; Mg<sup>2+</sup>.

## Introduction

Organophosphorus poisoning (OP) is the most common poisoning in India because of its easy availability. Organophosphorus pesticides are used widely for agriculture, vector control, and domestic purposes. Despite the apparent benefits of these uses acute organophosphorus pesticide poisoning is an increasing worldwide problem, particularly in rural

areas. Organophosphorus pesticides are the most important cause of severe toxicity and death from acute poisoning worldwide, with more than 2,00,000 deaths each year in developing countries. Unintentional and intentional OP poisonings continue to be a significant cause of morbidity and mortality in India [1]. The farmers are the most hard working and underpaid socioeconomic group in India. They work for hours in the fields without sufficient equipment and machines and despite their

vigorous efforts, they fail to meet their financial requirements. Many of the people consuming organophosphorus poison, that were appearing in the emergency department were suffering from financial insufficiency, and were usually un-insured. Thereby this study was taken up to decrease the cost involved in the treatment of these kind of poisonings. This study was performed in Kempegowda Institute of Medical Sciences a reputed tertiary care hospital, owned by the Vokkaliga Sangha (which means association of Vokkaliga group). The majority of the native farmers endogenous to Karnataka belong to the Vokkaliga (Gowda) group, which personally looks at Kempegowda Institute as its primary health access site, in case of any medical emergency, which explains the appropriateness of the Location chosen to do this study. Intravenous magnesium sulphate has been used in few smaller studies that has shown promising results which motivated us to start this study [4,5,6].

#### *Aims and Objectives of the Study*

- To assess the usefulness of MgSO<sub>4</sub> in acute OP poisoning in terms of decreasing duration of hospitalization, days of stay required in intensive care unit to return to a stable condition, and directly decreasing the cost and severity of symptoms of organophosphorus poisoning, without compromising the quality and efficiency of care.
- To assess and compare the use of MgSO<sub>4</sub> along with conventional standard therapy versus conventional standard therapy alone.

#### **Materials and Methods**

All patients with history of organophosphorous poisoning only were included in the study.

- All patients were decontaminated, treated with gastric lavage and the standard treatment based on severity of symptoms in accordance to standard treatment dose of iv atropine. Also iv pralidoxime was included in both treatment and control arms of the study.
- Patients who fulfil the inclusion criteria were divided into 2 groups.
- Two groups (25 each) - one group received intravenous magnesium sulphate 4gms along with iv atropine and iv pralidoxime (TEST). While second group received only iv atropine and iv pralidoxime (Control).

- Usually when a patient comes to the emergency department a bolus of 5 mg of atropine is given, and then the required dose of atropine is titrated in accordance to severity of presenting cholinergic symptoms.

#### *Source of Data*

Data was collected from all In-patients who fulfilled the inclusion and exclusion criteria. Patients with a history of OP poisoning in the time period from November 2013 to September 2015 were received in the emergency department of Kempegowda institute of medical sciences Hospital, decontaminated, given a gastric lavage and given bolus 5mg atropine dose and then admitted in the Intensive care unit for further management.

#### *Inclusion Criteria*

Patients admitted with history of OP compound poisoning within 24 hours of consumption.

Patients/attenders who were willing to give written informed consent.

Ingestion of poison by oral route only.

Patient survived the episode of poisoning and did not die.

#### *Exclusion Criteria*

Patients with Renal dysfunction.

Organophosphorous compound mixed with other compounds.

Any medical Contraindications for MgSO<sub>4</sub> therapy.

Death of the patient irrespective.

#### *Type of Study*

Comparative Interventional study.

#### *Analysis of Outcome Measures*

Data were analyzed using SPSS version 17 for windows. Frequency distribution of category variables were compared between intervention (MgSO<sub>4</sub>) and control groups using Chi-square test for proportions. The means were compared between the groups at baseline using ANOVA. The dose of atropine, PAM, ICU stay and ventilation were compared between groups using non parametric Independent Mann Whitney tests as they differed from a normal distribution. P value of less than 0.05 was



considered significant. Extreme care and appropriate steps like matching were taken under the guidance of statistical experts to prevent confounding and other statistical errors.

## Discussion

The present study was undertaken in the department of emergency medicine of KIMS Hospital to assess the efficacy of intravenous magnesium sulphate in the management of acute organophosphorous poisoning. Traditional treatment of this form of poisoning includes injection atropine to manage the muscarinic symptoms and injection PAM as specific antidote to salvage the enzyme acetyl cholinesterase with or without mechanical ventilator support on a need basis.

In recent times the use of PAM in acute OP compound poisoning has become a subject of debate, as mentioned previously the requirement for alternative drug/therapeutic modality which could decrease the mortality and hospital stay with better outcomes was needed. A couple of small studies have appeared in medicine literature regarding the benefits of intravenous magnesium sulphate in acute OP poisoning [4,5,6]. After reviewing sufficient literature the study was started.

50 patients who met the eligibility criteria were included in the study and they were divided in two groups. The first group referred to as control group received conventional standard management of OP compound poisoning in the form of GI decontamination, injection atropine for muscarinic symptoms control and injection pralidoxime chloride as specific antidote.

The other group referred to as test in addition to the above treatment received a one time only intravenous magnesium sulphate 4 gram dose as an infusion over 4 hours. A total number of 1200 poisoning cases were admitted and managed during the study period between December 2013 and August 2015 in Kempegowda Institute of Medical Sciences. Out of which 200 Patients were organophosphorus compound poisoning.

A total number of 8 organophosphorus poisoning cases died during the study period that was not included in the study because they failed to meet the inclusion criteria. Poisoning cases admitted and managed by the Department of Emergency Medicine of which, 50 subjects full filling the inclusion/exclusion criteria were included the study.

## Sex

The present study there were 33 males and 17 females ratio of 2:1, this male domination has also been noticed in similar studies by other authors. This sex difference could probably be attributed to the male subjects going out to purchase the easily accessible insecticide and consuming outdoors.

## Age

In the present study majority of the subjects were in the age group of 21-30 years this is collaborated by similar study done by various other authors in the country. The younger age group seem to be much more vulnerable to emotion upheavals and impulsive decision making. Since all cases included in the study were oral consumption with suicidal intention. There were no cases of accidental exposure we encountered.

## Occupation

Among the study subjects enrolled majority were students (42%) compared to other occupational groups. This explains the younger age group vulnerability. Followed by farmer (14%) and house wives (14%). Other studies done in the country shows the farmers among the occupation group being more vulnerable to organophosphorus poisoning. Since OP insecticides are used in their profession. Many of the students who consumed this organophosphorus poison, had their families in the agricultural background.

## Place of Consumption

The present study was done in a tertiary care teaching hospital located in city. Hence majority of cases were from urban area (76%). While other similar studies were done at suburban or rural population which were catering to rural population. This also explains the majority of population being from rural background with agricultural workers dominance in their studies.

## Clinical Observations

The common clinical features and presenting symptoms in the present study among the subjects were of gastrointestinal manifestation in form of vomiting and diarrhea. Oral ingestion was the only route of poisoning. Patients presented to the emergency room with parasympathetic, muscarinic symptoms in form of excessive secretions i.e. sweating, lacrimation etc. the similar presentations were noticed by other authors in their studies.

*Ingestion to Admission Interval*

A majority of the study population presented to the emergency department quite early after ingestion. This being an urban setup with easy access to transportation from place of ingestion to medical facility. Hence time interval between ingestion to admission was shorter and of milder severity. Similar reporting has been done by other studies. They were 12 cases of severe poisoning as determined by modified driesbach score included in the study requiring ventilator support for respiratory failure. Out of which 9 were in control group and 3 were in test group.

*Regarding the Type of OP Compound*

Dichlorvos (22%) was the most commonly encountered OP compound followed by dimethionate (14%). Methyl parathion (12%) and propenfos (12%). Majority of the OP compounds were methoxy organophosphates (20%). And other (20%). Hence early aging of acetyl cholinesterase is to be expected in our study.

Since methylated OP compounds age much faster and earlier than ethylated group. Propenfos and phorate cause aging very rapidly as compared to methylated OP compounds. Other studies shown similar grouping of OP compound poisoning depending on the geographical area which determines the type of OP compound. Since these insecticides are need based on the agricultural produce of that area. All patients were treated initial atropine bolus to overcome the muscarinic symptoms of OP compound poisoning and later titrated through slow atropine infusion to alleviate these symptoms based on severity.

Injection pralidoxime chloride was used as the specific antidote in both the groups. But at doses which could be considered sub optimal compared to WHO guidelines (30mg/kg bolus followed by 8-10mg/kg/hour). In this study only 20% of poisoning were due to ethoxy OP compound which age slowly hence treatment with oximes would be useful. The other 20% were due to phorate, propenfos which age rapidly the use of oximes is of less proven efficacy. Other 60% were methoxy OP compound which age relatively early compared to ethoxy OP hence efficacy of oximes as antidote in this group is debatable.

*Mortality*

There was no mortality in the present study however other studies home reported mortality of

13.3-18% This nil mortality in present study could be due the selection. Wherein patients who had consumed OP compound poisoning < 24 hours were included in the study. A vast majority of them presented earlier and received medical attention within 4 hours of consumptions. Urban population constituted 3/4<sup>th</sup> of study population (76%) who had probably consumed less concentrated OP compounds used for domestic purposes as compared to more toxic and lethal field poisons used in rural area. Prompt mechanical ventilator support was given to cases with severe poisoning is another factor reducing mortality. In present study, The study population was divided into 2 arms with one group receiving conventional therapy as described earlier. The other group in addition receiving intravenous magnesium sulphate.

In present study there was significantly decrease in the total number of days patients needed to be on ventilator (0.8days) and the total duration of ICU stay (5 days) in TEST group as compared to Total number of days needed to be on ventilation (4.28 days) and ICU stay (10.68 days) in the control group. In study done by pajamound et al [4] mean ICU stay in the test group (2.90±0.60 days) was significantly less as compared to the number of days as needed in the control group (5±0.82days). In study done by Basher et al [68]. Mean atropine required in control group was 127 mg and in test was 159mg.

Since there was a near significant difference in distribution of severity across control and test groups, We performed an analysis of co-variance (ANCOVA) for outcome measures in ICU stay and days of ventilation required between the groups using severity, age and other statistical significant matching processes. There was a significant effect of severity on ICU stay  $F(1,47) = 20.9, P < 0.001$ . Nevertheless intervention also reduced the ICU stay irrespective of the severity  $F(1,47) = 6.8, P = 0.01$ . There was a significant effect of severity on number of days of ventilation  $F(1,47) = 30.8, P < 0.001$ , but interventional effects were non-significant for days of ventilation.

**Conclusion**

- Male sex and younger age were predominant in the study population which is reflected in other similar studies done across the country.
- Ingestion with suicidal intention was the only route of poisoning there were no accidental or homicidal incidents.
- Methylated OP compounds with other class like propenfos dominated as the type of compound

ingested which age faster and the efficacy of using injection PAM in these cases remains questionable.

- In spite of the above there was no mortality in the study population.
- Majority of the subjects in both the groups presented with mild degree of severity as per Driesbach's severity score.
- All subjects presented with GI manifestations predominantly since oral route of ingestion was the only modality of poisoning.
- Intravenous magnesium sulphate 4 grams administered in the test group did not have any form of adverse events like hypotension, hyporeflexia.
- The test group which received intravenous magnesium sulphate had better outcomes in terms of lesser number of days in ICU, lesser number days on ventilator and lesser amount of total atropine required.
- The study is small with differences in distribution of severity between the two groups, further studies including larger number of cases and inclusion of more severe cases in test group would be adding more credence to the future of intravenous magnesium therapy in acute OP poisoning.

### Summary

Organophosphorus compounds are commonly used agents for suicidal purpose because of their easy availability. Male sex and younger age were predominant in the study population Among these compounds the most common compound present in our study is dichlorvos (22%). Intravenous magnesium sulphate 4 grams administered in the test group did not have any form of adverse events like hypotension, hyporeflexia or respiratory depression. The test group which received intravenous magnesium sulphate had better outcomes in terms of lesser number of days in ICU, lesser number days on ventilator and lesser amount of total atropine required. The study is small with differences in distribution of severity between the two groups, further studies including larger number of cases and inclusion of more severe cases in test group would be adding more credence to the future of intravenous magnesium therapy in acute OP poisoning.

### List of Abbreviations

Ach - Acetylcholine

CVS - Cardiovascular system  
 ECG -Electrocardiography  
 GIT - Gastrointestinal system  
 IMV - Intermittent Mandatory Ventilation  
 IMS - Intermediate Syndrome  
 IPPV - Intermittent positive-pressure ventilation  
 MS - Musculoskeletal system  
 NMJ -Neuromuscular Junction  
 OPP - Organophosphate poisoning  
 PEEP - Positive end expiratory pressure  
 RS - Respiratory system  
 RBC -Red blood cells  
 SIMV - Synchronous intermittent mandatory ventilation  
 AchE - Acetylcholinesterase  
 CNS - Central Nervous System  
 DPN - Delayed Polyneuropathy.  
 OP - Organophosphorus  
 OPC - Organophosphorus compound  
 PAM - Pralidoxime chloride  
 Mg<sup>2+</sup>- Magnesium  
 MgSO<sub>4</sub> - Magnesium sulphate  
 mg- milligrams

### References

1. Roberts Darren M, Aaron Cynthia K. Management of acute organophosphorus pesticide poisoning. *BMJ* 2007;334:629-634.
2. Brunton Laurence L. Principles of toxicology and treatment of poisoning. In: Klaassen Curtis D, editor. Goodman and Gilman's the pharmacological basis of therapeutics. New York (NY): McGraw-Hill; 2006. 1156-1159.
3. Peter G Blain ,Organophosphorus poisoning (acute). clinical evidence, Search date April 2010.
4. Pajoumand A, Shadnia S, Rezaie A, Abdi M, Abdollahi M. Benefits of magnesium sulphate in the management of acute human poisoning by organophosphorus insecticides. *Hum Exp Toxicol*. 2004 Dec.23(12):5659.
5. A.Basher, S. H. Rahman, A. Ghose, S. M. Arif, M. A. Faiz, and A. H. Dawson. a pilot trail in Dhaka medical college, Bangladesh. Accessed google.com 2013-11-26.
6. Basher A, Rahman SH, Ghose A, Arif SM, Faiz MA, Dawson AH. Phase II study of magnesium sulphate in acute organophosphate pesticide poisoning. *Clin Toxicol (Phila)*. 2013 Jan;51(1):3540.

# Patient Expectations in the Emergency Department of a Super-Speciality Hospital

Sudip Chakraborty<sup>1</sup>, Saptarshi Saha<sup>2</sup>, Indraneel Dasgupta<sup>3</sup>

## Author's Affiliation:

<sup>1</sup>MEM final Year PGT <sup>2</sup>Associate Consultant <sup>3</sup>Clinical Director, Department of Emergency Medicine, The Institute of Emergency and Trauma care, Peerless Hospital and B.K. Roy Research Center, Kolkata, West Bengal 700094, India.

## Corresponding Author:

**Saptarshi Saha,**  
Associate Consultant,  
Department of Emergency Medicine, The Institute of Emergency and Trauma care, Peerless Hospital and B.K. Roy Research Center, Kolkata, West Bengal 700094, India.

Received on 25.10.2017,

Accepted on 07.11.2017

## Abstract

*Introduction:* As the patients are the consumers of a Hospital. There is huge importance of evaluating patient services from consumer's perspective. If we compare what people expected about a health care service with their real experiences, it has been found to influence their overall satisfaction. *Aim & Objective:* of this study was to find out in which parameters the expectations are high and what are the unmet expectations with respect to the Emergency Department. So that Hospital can find out ways of improvement of the same. *Material & Method:* Individuals were given pre and post ED visit 12 preformed questionnaires & individual perception scores of questions have statistically analyzed. *Results & Discussion:* It was found that out of 12 only in 3 questions the post visit experience superseded the previsit expectations. *Conclusion:* This study gives a scope of discussion for further improvement in quality of healthcare provided in the tertiary healthcare center & paves a path for further studies to occur and help to make policies to give better patient care.

**Keywords:** Healthcare; Emergency; Intensive Care.

## Background

A lot of studies have been conducted regarding this topic all over the world but none such can be found in Kolkata.

Previously the hospitals were regarded only as a place where patients would be treated and a small emergency room used to exist where the most junior people without much knowledge of true emergency medicine were posted [1].

Emergency department took no role in resuscitation. They just triaged the patient and guided whether patients require intensive care or ward [2].

But with time the people's expectations about hospitals have changed. They need treatment as soon they arrive. Their knowledge has increased with internet accessibility. This change in attitude and expectation has come due to media, commercialization and improvement in the facilities [3].

Emergency medicine is the face of Hospital and the mostly first contact with the patient to a hospital.

Patients construct their first opinion about the hospital from the services they receive in the Emergency Department. These opinions are carried over from Emergency to the in-patient unit and will influence their actual response to care [4].

So this study is very important from the viewpoint of the patient about the hospital as the first impression is the best impression.

If we review the complaints of the patients in the emergency department those will give us a clue about the targets for quality improvement. But it is better to assess expectations using thorough study methods. Hence expectation studies like this should be encouraged. This study paves the way for further studies to take place [5]. As for example we can include the questionnaire involving the healthcare professionals also.

## Objective

1. To improve the quality of patient care in Emergency Department, to assist in policy making decisions

of the emergency department and to develop the staff education programme to meet patient expectations.

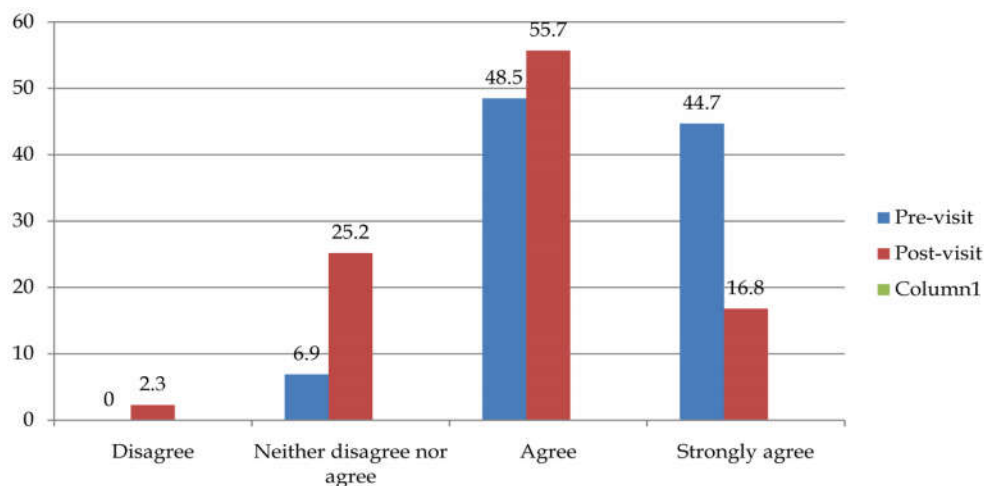
**Material and Methods**

This study was conducted between August 2014 and October 2014 at the department of emergency medicine in a tertiary care hospital where every month around 1000 patients visit the emergency department. The individual perception scores of questions have been given in the statistical analysis portion of the dissertation. Data was collected through random sampling in morning, evening and night shift. Pre-visit questionnaire was given immediately after entering the E.D before contact with any doctor, nurse

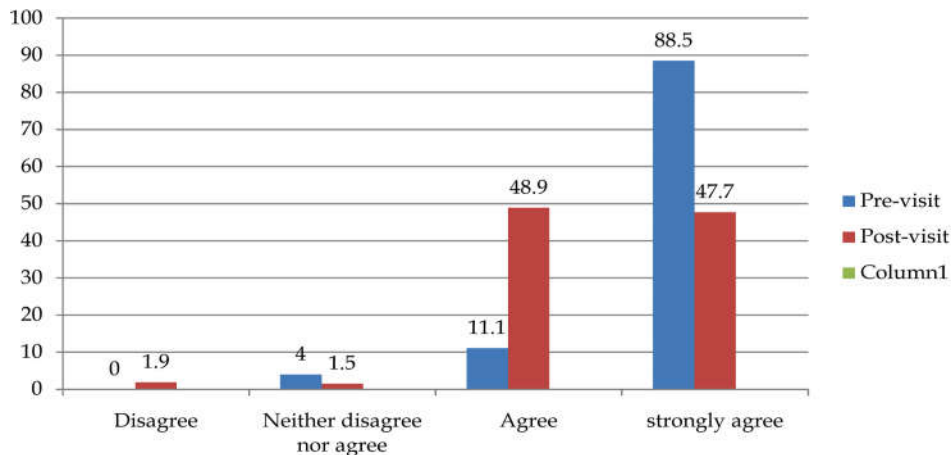
and other staff. Post-visit questionnaire was given before shifting the patient to the wards or intensive care. The inclusion criteria consisted of: Conscious, oriented stable patient whose age was more than 18yrs.

A pre-structured, pre-tested questionnaire was given to the patients. Every question was graded in 5 point Likert scale. Patients were requested to mark those scales. An informed consent was taken before that. The data so obtained was scrutinized, tabulated, analyzed by biostatistician where sample size was calculated to be 261 and the data was validated through logical checks and analyzed by statistical Software package (SPSS ver. 19.0) and statistical analyses were done. For categorical data, Chi-square test has been applied for the pre-visit expectation with post visit met expectation.

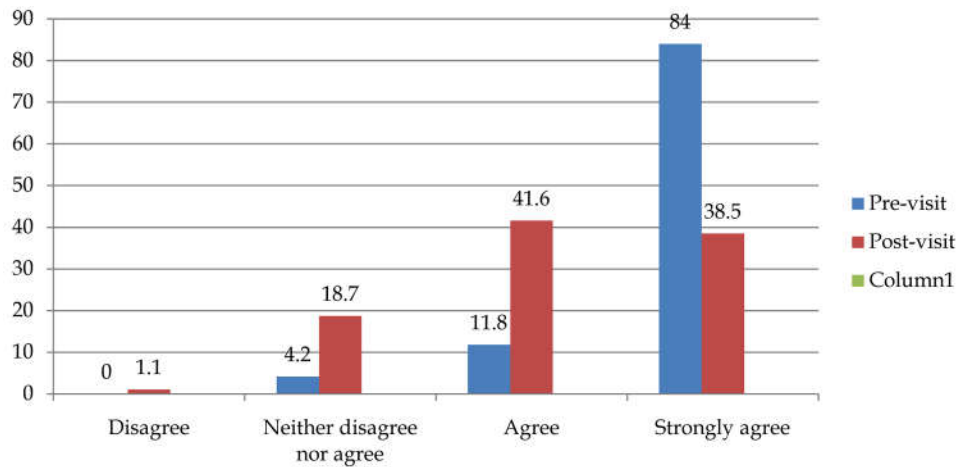
**Results and Analysis**



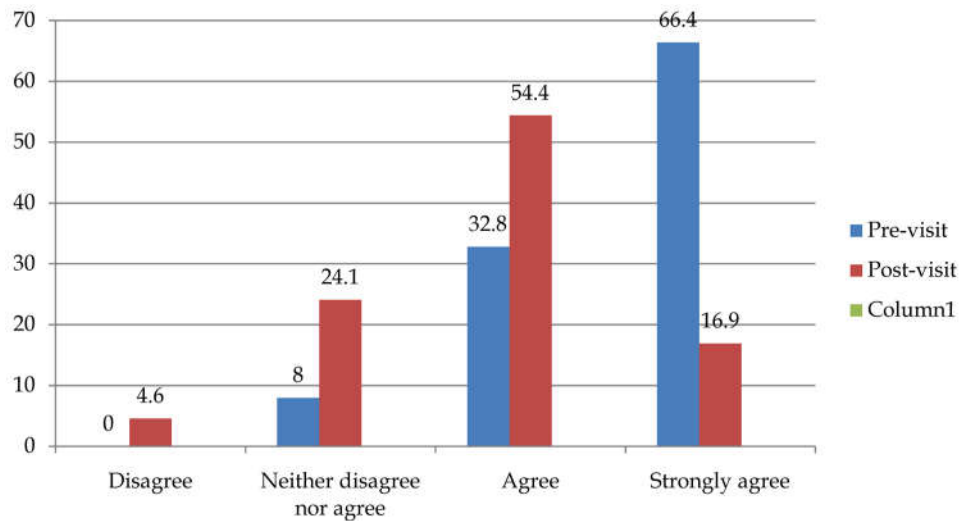
**Question 1:** Was concerning the cleanliness of the inside of the building where it was found that the pre visit expectation was more than post visit opinion



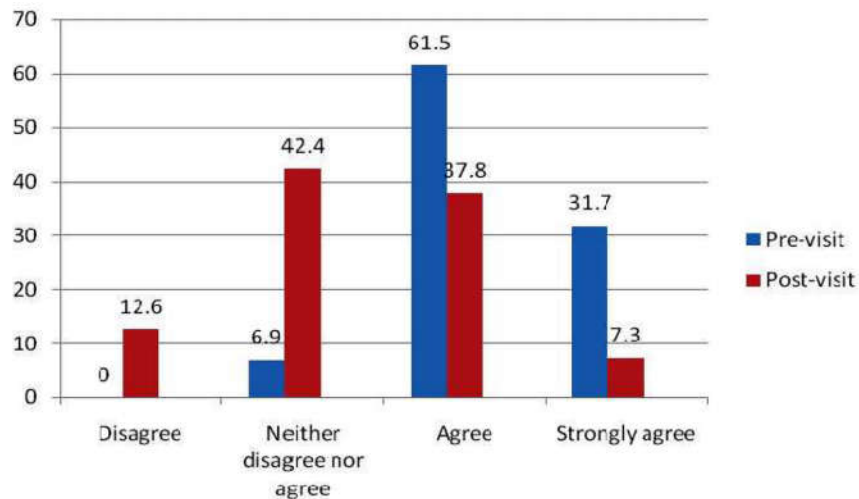
**Question 2:** Was concerning the timing of attending by medical professional where it was found that the pre visit expectation was more than post visit opinion



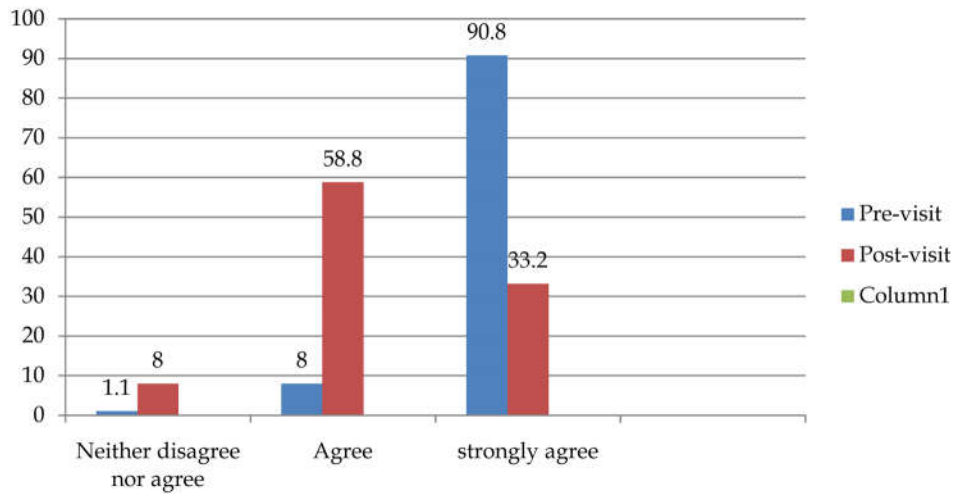
**Question 3:** Was regarding a choice of doctors to be given to patient where it was found that the pre visit expectation was more than post visit opinion



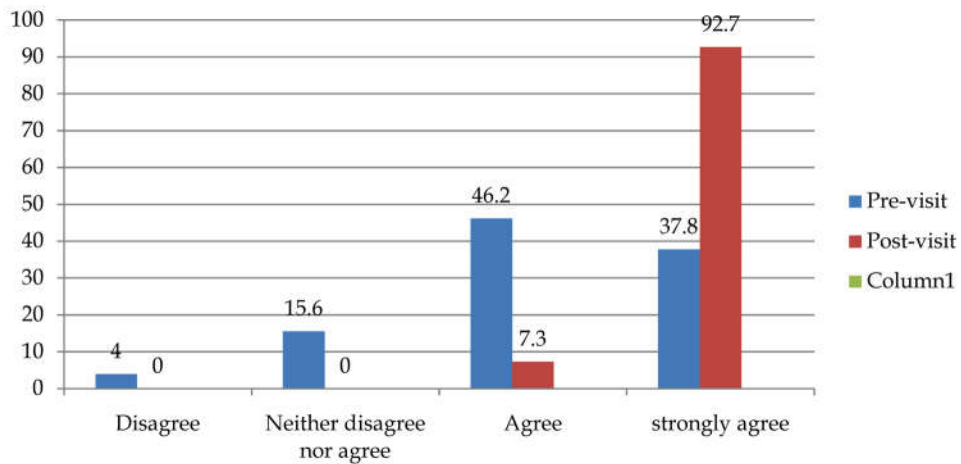
**Question 4:** Was regarding the behaviour of nursing where it was found that the pre visit expectation was more than post visit opinion



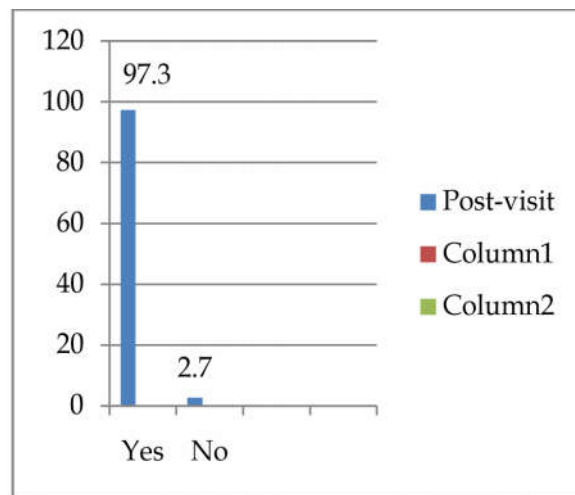
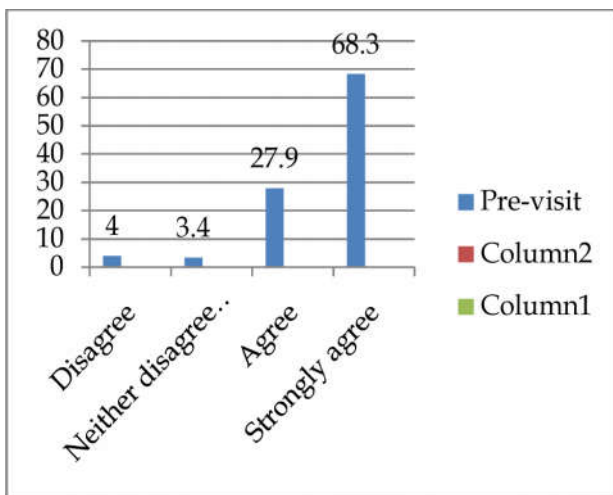
**Question 5:** Was regarding the helpful attitude of the reception/admission staff where it was found that the pre visit expectation was more than post visit opinion



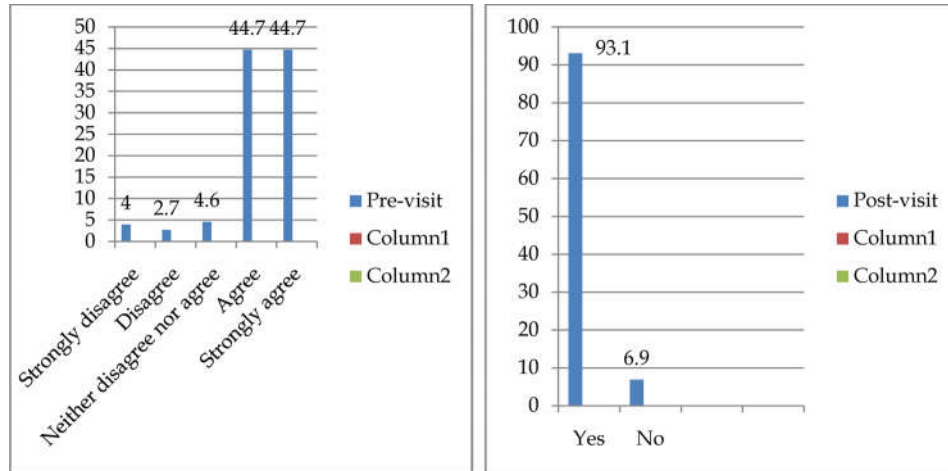
**Question 6:** Was regarding the expectations of patient about the dignity and respectfulness of the doctor towards the patient it was found that the pre visit expectation was more than post visit opinion.



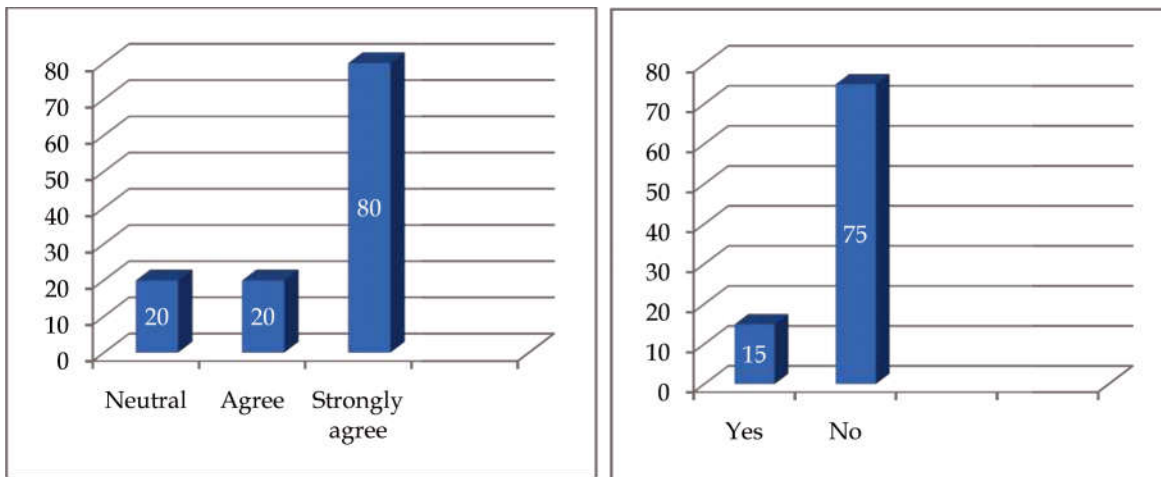
**Question 7:** Was regarding the knowledge and understanding of the health of patient by the doctor it was found that the pre visit expectation was less than post visit opinion



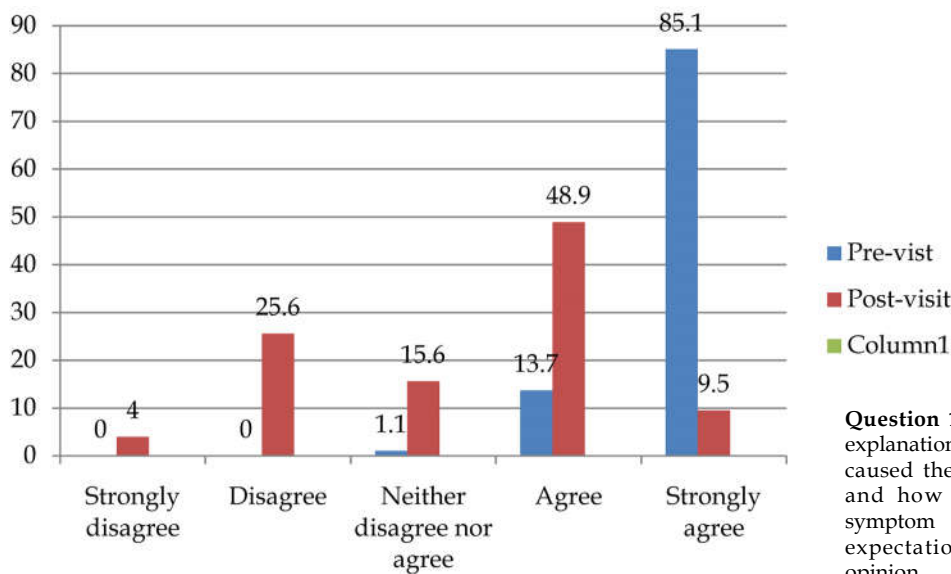
**Question 8:** Was regarding the physical examination of the patient where it was found that the pre visit expectation was less than post visit opinion



**Question 9:** Was regarding the investigations given to the patient where it was found that the pre visit expectation was less than post visit opinion

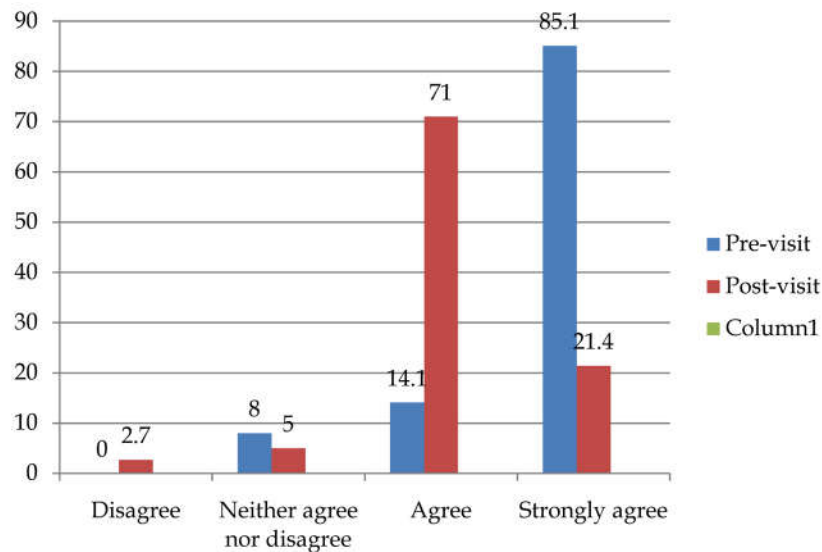


**Question 10:** Was regarding the expectation about a definitive diagnosis to be provided in the emergency where it was found that the pre visit expectation was more than post visit opinion.



**Question 11:** Was regarding given full explanation in clear language about what caused the patients condition/problem and how to manage the condition/symptom it was found that the pre visit expectation was more than post visit opinion





**Question12:** Was regarding overall quality of treatment where it was found that pre visit expectations were more than the post visit opinion

**Table 1:** Comparison of individual score between post visit met expectation and pre visit expectation

Variables	Pre-visit (Mean Score ± SD)	Post-visit (Mean Score ± SD)	Difference of Post-pre	Paired-t-value	p-value
Q1	4.4±0.6	3.9±0.7	-0.5±0.9	8.8	0.001
Q2	4.9±0.3	4.4±0.6	-0.5±0.7	10.8	0.001
Q3	4.8±0.5	4.2±0.8	-0.6±0.9	11.3	0.001
Q4	4.7±0.5	3.8±0.7	-0.8±0.8	15.7	0.001
Q5	4.2±0.6	3.4±0.8	-0.8±0.9	15.0	0.001
Q6	4.9±0.3	4.2±0.6	-0.6±0.7	15.3	0.001
Q7	4.2±0.7	4.9±0.3	0.7±0.7	15.7	0.001
Q8	4.6±0.6	4.9±0.6	-0.2±0.8	5.0	0.001
Q9	4.4±0.7	4.7±1.0	0.4±1.2	4.8	0.001
Q10	4.6±0.6	3.1±2.0	-1.4±2.0	11.7	0.001
Q11	4.8±0.4	3.4±1.0	-1.4±1.1	21.2	0.001
Q12	4.8±0.3	4.1±0.6	-0.7±0.6	16.3	0.001

Demography of the patient population showed patient from middle and older age group are more in number than other age group. The male female ratio is almost equal. More data was collected in the morning shift. The Hindus were more in number than other communities. Maximum patients were either graduates or high-school passed. Income of the average population is between Rs.30,000-50,000. Health insurance is present in almost 50% of population.

**Discussion**

It is important to have these types of surveys from time to time where we can try to fathom the expectation of patients before they enter into a hospital and compare it with their opinion post visit to the hospital [6].

In the previous study The measurement of patients' expectations for health care: a review and psychometric testing of a measure of patients' expectations A Bowling, G Rowe, N Lambert, M Waddington, KR Mahtani, C Kenten, A Howe and SA Francis:

In that study it was found that:

1. The post visit opinion about cleanliness was less than the pre-visit expectation which matched the finding of this study as well.
2. The post visit opinion about whether the patient was seen in time was less than the pre-visit expectation which matched the finding of this study as well.
3. The post visit opinion about whether a choice of doctors would be given to them was less than the

pre-visit expectation which matched the finding of this study as well.

4. The post visit opinion about reception staff was lower than the pre-visit expectation regarding the same. This finding was same as we have found in our study.
5. The post visit opinion about the helpful, respectful and dignified behaviour expected from the doctors were not met as per the post visit opinion which matched the post visit opinion of our study.
6. The post visit opinion about knowledge and understanding of health problems of the doctor was less than expected but in our study it was found that the post visit opinion about the same parameters were more than what was expected.

Comparing with above mention study, it was found that in the patients in both the studies, the pre visit expectation about physical examination and investigations were less than in the post visit opinion

About the point where the patients pre-visit expectation about being given a diagnosis both the studies showed that the post visit opinion was less.

In question number 11, pre visit expectation about full explanation in clear language about what caused the patient's condition/problem and how to manage the condition/symptom was found to be more than post visit opinion.

About the overall quality of treatment, the previsit expectation of the patient was found to be more than the post visit expectation.

Emergency Department being one of the main portal of entry of the patients in the hospital is perhaps the most important place of such surveys<sup>7</sup>. But at the same time the outdoor departments and the laboratory and radiology departments also are the important departments where such surveys need to be conducted. The strength of this survey lies in the facts that it was done in the department of emergency medicine where perhaps the expectation of a patient is the most and this survey was done prospectively [8]. But the limitations of the survey lies in the fact that the age group was above 18 and patients with poor Glasgow coma scale was excluded. It can be suggested that the survey could have included the relatives of such patients e.g. paediatric patients who are probably the most sensitive patients visiting the emergency department.

At the same time it can be pointed out that as it contains only the perspective of the patient, and the healthcare personnel were not questioned so it is difficult to judge about the extent of practicability of

the expectations of the patients which at many points of time can be unrealistic as well. It may be that as the results showed that the post visit expectations were more in only question number 7,8 and 9 which were regarding the knowledge and understanding of the health of patient by the doctor, regarding the physical examination of the patient and was regarding the investigations given to the patient where it was found that the pre visit expectation was less than post visit opinion. But we need to understand that regarding question number 7, the patient actually can not have any medical knowledge at all, so how can they judge the depth of medical knowledge and understanding of the physician. Regarding question number 8, how can a patient who is supposedly a non-medical person know about what physical examinations are the healthcare providers going to do for any particular illness. Regarding question number 9, the number of investigations to be sent for a particular illness can vary depending on the illness and the corresponding physical findings and the bedside investigations. So it is quite impractical for a patient to judge his or her own illness and decide on the number of investigations to be sent for the illness or complaints with which the patient has reported to the emergency department [8]. In this context it can be mentioned that if the healthcare givers point of view was considered and compared with the expectations of the patients then it could have been more justified and a more practical approach could have been made to get near the expectations of the patient. Another limitation of the study lies in the fact that it was done in a single centre. If it would have been a multicentric study, then we have got a broader perspective about the expectations of the patient and the degree of their satisfaction. Considering the patients to be customer of a hospital it is always or mostly that the customer will demand more from an institution where he or she is seeking service from but the constrains of the institute delivering the service should be kept in view. As for example the simple triaging system of an emergency department which dictates that the most serious patient should be dealt with first [10] will obviously increase the waiting time of a walk-in patient who may feel neglected but at the same time good counselling can increase the post visit rating of the same patient.

## Conclusion

At the end it can be concluded that it is a well built study and the questionnaires are also validated so this study can be the framework and base of many

other similar studies as for example a study which will compare the point of view of patients regarding the expectations with the point of view of the patients. It gives us an insight into the expectations from the end of the patient which can be worked on further by various discussions and/or change of existing practices amongst the hospital staff about to extent the expectations can be met from practical point of view.

### References

1. Henry D. McIntos: The Evolution of Today's Hospital Emergency Departments - Current Problems and Challenges, *Clin. Cardiol.* 1996;19(11):846-856.
2. McIntosh HD: Personal recollection( s) and/or observation(s). Peabody FW: The care of the patient. *JAnz Med A.ssoc* 1927;88:x77-8x2.
3. Lola Butcher: Is Your Hospital Ready to Answer Consumer Demands? *Hospital and health networks.* Feb10, 2015.
4. Accident and Emergency care- CHKS, [www.chks.co.uk/icompare/userfiles/files/What-makes-a-top-hospital-A&E.pdf](http://www.chks.co.uk/icompare/userfiles/files/What-makes-a-top-hospital-A&E.pdf).
5. Lena Burström, Bengt Starrin et al: Waiting management at the emergency department – a grounded theory study *BMCHealthServ Res.* Published online 2013 Mar 12. doi: 10.1186/1472-6963-13-95.
6. Desmond, J. Managing patient expectations. *Healthcare Collaborator.* 2003;3:9-16.
7. Waris Qidwai, Syed Sohail Ali, Muhammad Baqir, Semi Ayub. Patient expectations from an emergency medical service. *J Ayub Med Coll Abbottabad.* 2005 Jul-Sep;17(3):3-6.
8. Robert John Adams. Improving health outcomes with better patient understanding and education. *Risk Manag Healthc Policy.* 2010;3:61-72. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3270921/>
9. Anderson WG, Cimino JW, Ernecoff NC, Ungar A, Shotsberger KJ, Pollice LA, Buddadhumaruk P, Carson SS, Curtis JR, Hough CL, Lo B, Matthay MA, Peterson MW, Steingrub JS, White DB. A multicenter study of key stakeholders' perspectives on communicating with surrogates about prognosis in intensive care units. *Ann Am Thorac Soc.* 2015 Feb;12(2):142-52.
10. Ramesh P Aacharya, Chris Gastmans, and Yvonne Denier. Emergency department triage: an ethical analysis. *BMC Emerg Med.* 2011;11:16. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3199257/>

## Clinical Presentation of Renal Injury at a Tertiary Care Hospital

Cijo John<sup>1</sup>, Selin Abraham<sup>1</sup>

### Author's Affiliation:

<sup>1</sup>Assistant Professor, Department of Medicine, Mount Zion Medical College, Chayalode, door Pathanamthitta, Enadimangalam, Kerala 691556, India.

### Corresponding Author:

**Selin Abraham**,  
Assistant Professor,  
Department of Medicine,  
Mount Zion Medical College,  
Chayalode, Adoor  
Pathanamthitta,  
Enadimangalam,  
Kerala 691556, India.  
E-mail:  
sowmyatshassan@gmail.com

Received on 10.06.2017,

Accepted on 20.07.2017

### Abstract

*Introduction:* AKI occurs predominantly in urban intensive care units and is associated with multiorgan failure and sepsis, high mortality, and occurrence in older populations. While cases of AKI in urban areas of the developing world have similar characteristics to those in the developed world, AKI in rural regions commonly develops in response to a single disease and specific conditions (e.g. gastroenteritis) or infections (e.g. severe malaria, leptospirosis, or hemolytic-uremic syndrome) and in younger otherwise healthy individuals. *Methodology:* Acute Kidney Injury, the major inclusion and exclusion criteria were identified. Data regarding etiology, clinical features, outcome to treatment were collected over a period of one year from Jan 2011 to Jan 2012 in total of 200 admitted patients. The outcome of the study was analyzed and documented. *Results:* The youngest person enrolled was 20 yrs and oldest was 86 yrs of age. Amongst the pre renal conditions Acute diarrheal diseases are the commonest. Oliguria dominate as the most common presenting symptom in patients with AKI. *Conclusion:* Acute kidney injury is commonly seen in men than in women below the age group of 50 yrs.

**Keywords:** Acute Renal Failure; Glomerulonephritis; AKI.

### Introduction

The evolution of the term 'acute renal failure' dates back to 1802, when William Heberden first described it as *Ischuria Renalis*. Since then there are over 35 official definitions of the term; these include: *Acute Bright's disease, war nephritis and crush syndrome*. It wasn't until 1951 that Homer W. Smith introduced the term 'Acute Renal Failure' [1].

Today, Acute Kidney Injury (AKI) is considered the correct nomenclature for the clinical disorder formerly termed 'Acute Renal Failure' (ARF). AKI, is a protean syndrome of varied severity. It is characterized by a rapid (hours to days) decline in the glomerular filtration rate (GFR) and retention of nitrogenous waste products such as blood urea nitrogen (BUN) and creatinine. Acute kidney injury (AKI) has become increasingly prevalent in both developed and developing countries, and is associated with severe morbidity and mortality [2].

In developed countries, AKI occurs predominantly in urban intensive care units and is associated with multiorgan failure and sepsis, high mortality, and occurrence in older populations. While cases of AKI in urban areas of the developing world have similar characteristics to those in the developed world, AKI in rural regions commonly develops in response to a single disease and specific conditions (e.g. gastroenteritis) or infections (e.g. severe malaria, leptospirosis, or hemolytic-uremic syndrome) and in younger otherwise healthy individuals. Many causes of AKI in rural settings, such as diarrhea, poisoning, malaria, or septic abortion, can be prevented by interventions at the individual, community, and regional levels. Treatment with dialysis is often unavailable or too costly in developing regions, so there must be community-wide efforts to eradicate causes of AKI, expedite diagnosis, and aggressively manage prerenal conditions and specific infections [3].

Despite several advances in our treatment and understanding of the pathogenesis of acute kidney

injury (AKI), many aspects in this field remain subject to controversy, confusion, and lack of consensus. One of these important aspects is the definition of AKI. To make consensus-based recommendations and delineate key questions for future studies, the Acute Dialysis Quality Initiative (ADQI) workgroup identified a definition/classification system for AKI [4].

Accordingly, a multilevel classification system was proposed, in which the complete spectrum of acute renal dysfunction could be included, such as Risk of renal dysfunction, Injury to the kidney, Failure or Loss of kidney function, and End-stage kidney disease; these criteria are identified by the acronym RIFLE. The RIFLE criteria were later modified and referred to as the acute kidney injury network (AKIN) definition. For all practical purposes, RIFLE and AKIN criteria are the same. The aim of this study is to summarize the clinical profile of AKI as defined by the RIFLE/AKIN criteria but limited by the inability to define the baseline creatine and GFR levels [5].

## Methodology

This study was conducted on admitted patients in the Department of Medicine, and was aimed at

## Results

The youngest person enrolled was 20yrs and oldest was 86yrs of age.

identifying the more common causes, clinical features and outcome of treatment of these patients admitted with Acute Kidney Injury above 18 yrs of age. Approval from ethical committee and written consent from patients or his/her relatives were obtained.

Acute Kidney Injury, the major inclusion and exclusion criteria were identified. Data regarding etiology, clinical features, outcome to treatment were collected over a period of one year from Jan 2011 to Jan 2012 in total of 200 admitted patients. The outcome of the study was analyzed and documented.

### *Inclusion Criteria*

1. Patients admitted in the Department of Medicine, T.D Medical College, Alappuzha.
2. Patients above 18yrs of age.
3. Both sex included.
4. Clinical and Lab values suggestive of Acute Kidney Injury.

### *Exclusion Criteria*

1. Patients below 18yrs of age.
2. Already diagnosed cases of Chronic kidney disease.

**Table 1:** Sex and Age distribution

Age	Gender		Total
	Male	Female	
18 - 27	9 8.00%	5 5.70%	14 7.00%
28 - 37	24 21.40%	14 15.90%	38 19.00%
38 - 47	23 20.50%	13 14.80%	36 18.00%
48 - 57	27 24.10%	34 38.60%	61 30.50%
58 - 67	21 18.80%	17 19.30%	38 19.00%
68 - 77	5 4.50%	5 5.70%	10 5.00%
78 - 87	3 2.70%		3 1.50%
Total	112	88	200

Chi Square: 8.012; P > 0.05

**Table 2:** Gender distribution between Age groups

Age	Gender		Total
	Male	Female	
< 50 yrs	62 55.40%	44 50.00%	106 53.00%
>= 50 yrs	50 44.60%	44 50.00%	94 47.00%
Total	112	88	200

Chi Square: 0.568; P &gt; 0.05

Most patients presented with oliguria as the main symptom.

**Table 3:** Presenting complaints

Presenting Complaints	Frequency	Percent
None	68	34.0
Oliguria	99	49.5
Anuria	33	16.5
Total	200	100

**Table 4:** Age wise distribution of presenting complaint

Presenting Complaints	Age		Total
	< 50 yrs	>= 50 yrs	
None	45 42.50%	23 24.50%	68 34.00%
Oliguria	46 43.40%	53 56.40%	99 49.50%
Anuria	15 14.20%	18 19.10%	33 16.50%
Total	106	94	200

Chi Square: 7.191; P &lt; 0.05

**Table 5:** Etiology and Frequency

Diagnosis	Frequency	Percent
ADD AKI	34	17.0
AGN AKI	19	9.5
CIN	12	6.0
CVA AKI	9	4.5
DI AKI	12	6.0
HUS/TTP	9	4.5
Lepto/AKI	34	17.0
LVF AKI	10	5.0
MM AKI	5	2.5
NSAID AKI	25	12.5
Obst. AKI	5	2.5
Sepsis AKI	22	11.0
Viper Bite	4	2.0
Total	200	100

## Discussion

The predominant symptom with which the patients presented was Oliguria (49.5%). 16.5% patients had Anuria as their presenting symptom and 34% of patients did not have either of these symptoms. Oliguria is defined as a urine output that is less than

1 mL/kg/h in infants, less than 0.5 mL/kg/h for six consecutive hours in children and adults, or <400ml/d. The beginning and ending supportive therapy (BEST) kidney investigators highlighted the fact that oliguria was more common in septic AKI and viper bite induced AKI.

It is important to acknowledge, however, that at

least half of all cases of AKI are nonoliguric. This was highlighted by Liano, F, Pascual. M et al in their study on the epidemiology of acute renal failure, in a community based study in Spain. Thus, healthy urine output does not ensure normal renal function. Rarely, ARF comes to the attention of the clinician because of symptoms of uremia (eg, anorexia, nausea, vomiting, confusion, pruritus) or laboratory findings compatible with renal failure (metabolic acidosis, hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, hypermagnesemia, anemia). This finding is also in accordance with the above studies.

It was also observed that oliguria was the predominant symptom in age group above 50 yrs. 56.40% of patients above the age of 50 counted oliguria as their predominant symptom. This finding was statistically significant  $P < 0.05$ . This finding might be due to the fact that kidneys give up early as a fall in GFR as age advances.

The most common cause of AKI in the study was acute diarrheal disease (17%) and leptospirosis induced AKI (17%). NSAID induced AKI was seen in 12.5% and 11% in septic AKI. Combining drug induced AKI and contrast induced AKI accounted for 12%. Acute left ventricular failure induced AKI was seen in 5% of cases. Post renal failure accounted for only 2.5% of the cases of AKI. 59% of the cases could be considered as due to prerenal causes as compared to 38% of AKI due to intrinsic causes. This finding is in accordance with the studies conducted by The Madrid Acute Renal Failure Study Group in 1998.

The male gender incidence in diarrhea associated AKI was (16.1%) and in leptospirosis it was 15.20%, in NSAID and septic AKI, it was similar (10.7%). The females also showed similar incidence (18.2%, 19.3%, 14.8% and 11.4%). The major difference was noted in the incidence of Contrast induced nephropathy, males accounting for 8% as compared to 3% in females. This difference can be attributed to the fact that males are more prone to respiratory and cardiovascular diseases due to various reasons, than females and therefore the need of diagnosis in them with the use of contrast agents.

The incidence of CIN has been reported to range from less than 1% to greater than 30%. This wide variation in incidence is attributed to factors that include wide variability in CM doses, variation in the completeness of timing of patient follow-up, and a likely variation in the patient's hydration state [6].

Patients above the age group of 50 had higher incidence of pre renal failure like ADD associated AKI, CVA causing AKI, NSAID induced and LVF

associated AKI (112 patients out of 200) (56%). Septic AKI had an equal distribution among age and gender. Post renal failure was more in the age group more than 50 yrs (100%). CIN was also seen in increased incidence in age group more than 50 yrs (11 cases out of 12) (91%). Elderly patients may be at increased risk for true volume depletion due to changes in body composition with aging, leading to decreased total body water as a fraction of body weight, and from an increased burden of comorbid disease [7]. Non steroidal anti inflammatory drugs (NSAIDs), which are used by approximately 10% to 25% of the elderly [8], inhibit production of vasodilatory prostaglandins. NSAID use has been associated with a threefold higher risk of AKI in the general population, (Huerta et al, 2005), and an absolute risk of prerenal AKI of 13% in a nursing home cohort (mean age 87 years) (French study group on acute renal failure).

Postrenal or obstructive AKI is more common in the aged than in the young [9] accounting for 9% to 30% of cases. The most frequent causes of postrenal AKI in the elderly include benign prostatic hypertrophy (BPH) or prostate cancer, retroperitoneal adenopathy or malignancies, pelvic neoplasms, and neurogenic bladder. Although BPH and prostate cancer are common in older men, they cause obstruction in only a minority of cases. In elderly women, pelvic and retroperitoneal malignancies are the most frequent causes of postrenal AKI [9].

## Conclusion

Acute kidney Injury is more commonly seen in the age group 48-57 yrs and it is increased in incidence in the 38-47 and 58-67 yrs age group.

## References

1. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P: Acute renal failure – Definition, outcome measures, animal models, fluid therapy and information technology needs: The Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care* 2004;8:R204–R212.
2. Hou SH, Bushinsky DA, Wish JB, Cohen JJ, Harrington JT. Hospital-acquired renal insufficiency: a prospective study. *Am J Med.* 1983;74(2):243-248.
3. Nash K, Hafeez A, Hou S. Hospital-acquired renal insufficiency. *Am J Kidney Dis.* 2002;39(5):930-936.

4. Liano F, Pascual J. Epidemiology of acute renal failure: a prospective, multicenter, community-based study. Madrid Acute Renal Failure Study Group. *Kidney Int.* 1996;50(3):811-818.
  5. Brivet FG, Kleinknecht DJ, Loirat P, Landais PJ. Acute renal failure in intensive care units – causes, outcome, and prognostic factors of hospital mortality; a prospective, multicenter study. French Study Group on Acute Renal Failure. *Crit Care Med.* 1996;24(2):
  6. Mehta RL, Pascual MT, Soroko S, et al; Program to Improve Care in Acute Renal Disease. Spectrum of acute renal failure in the intensive care unit: the PICARD experience. *Kidney Int.* 2004;66(4):1613-1621.
  7. Uchino S, Kellum JA, Bellomo R, et al. Acute renal failure in critically ill patients: a multinational, multicenter study. *JAMA.* 2005;294(7):813-818.
  8. Xue JL, Daniels F, Star RA, Kimmel PL, Eggers PW, Molitoris BA, Himmelfarb J, Collins AJ: Incidence and mortality of acute renal failure in Medicare beneficiaries, 1992 to 2001. *J Am SocNephrol* 2006;17: 1135-1142.
  9. Waikar SS, Curhan GC, Wald R, McCarthy EP, Chertow GM: Declining mortality in patients with acute renal failure, 1988 to 2002. *J Am SocNephrol* 2006;17:1143-1150, 2006.
-



## 'Tetpro Score' for Evaluation of Progression in a Case of Tetanus

Vinay Swamy P.M.<sup>1</sup>, Bopanna C.A.<sup>2</sup>

### Author's Affiliation:

<sup>1</sup>Professor <sup>2</sup>Post Graduate Resident, Department of Emergency Medicine, J.J.M Medical College, Davangere, Karnataka 577004, India.

### Corresponding Author:

**Bopanna C.A.**,  
Post Graduate Resident,  
Department of Emergency  
Medicine, J.J.M Medical College,  
Davangere, Karnataka 577004,  
India, Karnataka.  
E-mail:  
drcabopanna@gmail.com

Received on 10.05.2017,

Accepted on 23.05.2017

### Abstract

Tetanus is an acute disease manifested by skeletal muscle spasm and autonomic system disturbance. A case of tetanus is a medical and social concern due to its high prevalence in developing countries. Tetanus as a disease is very distressing for the caretakers of the patient due to its painful and distressing presentation. A scoring system to monitor the progression or deterioration in the course of the disease was a felt need of the hour. 'TETPRO' scoring system was devised for the same. The scoring involved 10 parameters involving assessment of motor and autonomic symptoms. Using this it was very comfortable to monitor the progression of the disease and also for daily counselling the family members regarding the response of the patient and possible outcome.

**Keywords:** Tetanus; Progression; Deterioration.

### Introduction

Tetanus is an acute disease manifested by skeletal muscle spasm and autonomic system disturbance. Tetanus is caused by powerful neurotoxin produced by *clostridiumtetani* bacteria. The disease continues to have a substantial health impact in developing countries. The worldwide incidence of tetanus is approximately 1 million cases per year, with a mortality rate of 20% to 30% [1].

### Aim

Most cases of tetanus occur in incompletely vaccinated or unvaccinated individuals. Prognostication and monitoring the disease has been a handicap in the department of Emergency medicine and Critical care. Scoring system for clinical case of tetanus was devised for this purpose.

### Discussion

The Centers for Disease Control and Prevention defines tetanus as a syndrome of acute onset of

hypertonia and/or painful muscular contractions (usually of the muscles of the jaw and neck) and generalized muscle spasms without other apparent medical cause as reported by a health professional [2].

We in the department of Emergency Medicine, JJMMC, Davangere encountered five cases of Tetanus diagnosed based on history and clinical presentation over a period of one year in 2016 - 2017. Four patients had a history of trivial trauma and one had a history of dental extraction prior to admission in emergency department During the course of treatment patient were kept in isolation and were administered with Tetanus toxoid and tetanus immunoglobulin based on their vaccination status. Metranidazole 400mg and symptomatic treatment was given as per protocol [3].

During the course of treatment we encountered inconvenience regarding prognostication and monitoring the progression/deterioration of the disease process. It was also felt that objective assessment of the disease process would help in counselling the patient's caretakers regarding the status of the disease and possible outcome which forms the important part of patient-doctor interaction

During the study of literature we found that notable contribution has been done in this regard. Patel and Joag's [4] scoring system classified tetanus into mild moderate and severe. This scoring system had limited parameters and parameters were felt not discrete. The scoring system recommended by Singh et al [5] and 8 point scoring system which was devised by S SSidhartha et al [6] were also reviewed. It was felt that scoring pattern involved parameters that were more subjective in nature and chance of inter-observer variation in critical analysis of subjective symptoms will be a possible pit hole.

We decided to devise a scoring system for monitoring a case of tetanus keeping into account subjective and objective analysis in a case of tetanus. The parameters were selected that were discrete and a scoring pattern with no much inter observer variation possible and with no ambiguity in recognising the parameters involved.

The scoring involved 10 parameters involving assessment of motor and autonomic symptoms of

Tetanus keeping in mind the varied presentation of the disease process. Each parameter if found positive is given a score of 1 or 2 as described in Table 1. Any parameter that is normal is marked zero. Total score range from '0' to '15'. Higher score indicates deterioration in the course and is a predictor of poor prognosis/outcome. Lesser the score better the prognosis.

Our first patient in whom we used this scoring system (Table 2); patient recorded a score of 11/15 on the day 1 of admission. On day 3 the score was 6. Subsequently 9<sup>th</sup> and 10<sup>th</sup> day the score was 1 and 0 respectively. Our second patient recorded a score of 10/15 on the day of admission, on day 6 the score was 5/15 and on day 9 the score was '0'. Patients were shifted to general ward/ general medicine and subsequently discharged home.

The limitation during the process of devising this score was that only a small number of cases were taken into account to devise the 'Tetpro' scoring system. Keeping in acceptance of this fact and also the rarity

**Table 1:** Tetpro scoring chart

Department of Emergency Medicine, JJMMC Tetpro Scoring										
	Day	-	-	-	-	-	-	-	-	-
1	<b>Lock Jaw</b>	-	-	-	-	-	-	-	-	-
	<i>Inability to insert finger in oral opening</i>	-	-	-	-	-	-	-	-	-
	Normal-0									
	<2Finger-1									
	<1Finger-2									
2	<b>Flexion difficulty of Neck</b>	-	-	-	-	-	-	-	-	-
	Yes-1	-	-	-	-	-	-	-	-	-
	No-0									
3	<b>Difficulty swallowing</b>	-	-	-	-	-	-	-	-	-
	Yes-1	-	-	-	-	-	-	-	-	-
	No-0									
4	<b>Upper limb rigidity</b>	-	-	-	-	-	-	-	-	-
	<i>Difficulty in flexion from anatomical position</i>	-	-	-	-	-	-	-	-	-
	0-Normal									
	1->30 degree									
	2-<30 degree									
	<b>Lower limb rigidity</b>	-	-	-	-	-	-	-	-	-
	<i>Difficulty in flexion from anatomical position</i>	-	-	-	-	-	-	-	-	-
	0-Normal									
	1->30 degree									
	2-<30 degree									
5	<b>Painful Spasms</b>	-	-	-	-	-	-	-	-	-
	Yes-1	-	-	-	-	-	-	-	-	-
	No-0									
6	<b>Abdominal Guarding</b>	-	-	-	-	-	-	-	-	-
	Yes-1	-	-	-	-	-	-	-	-	-
	No-0									
7	<b>Able to sit from supine position</b>	-	-	-	-	-	-	-	-	-
	0-Normal	-	-	-	-	-	-	-	-	-
	1-Sit with help									
	2-Inability to sit with help									
8	<b>Tachycardia/Bradycardia</b>	-	-	-	-	-	-	-	-	-
	Yes-1	-	-	-	-	-	-	-	-	-
	No-0									
9	<b>Hypertension/Hypotension</b>	-	-	-	-	-	-	-	-	-
	Yes-1	-	-	-	-	-	-	-	-	-
	No-0									
10	<b>Sweating(Unexplained)</b>	-	-	-	-	-	-	-	-	-
	Yes-1	-	-	-	-	-	-	-	-	-
	No-0									
11	<b>Total Score</b>	-	-	-	-	-	-	-	-	-

**Table 2:** Illustration of Tetpro scoring in a patient

Tetpro Scoring		-	-	-	-	-	-	-	-	-	
	Day	1	2	3	4	5	6	7	8	9	10
1	Lock Jaw	2	2	1	1	1	1	0	0	0	0
	Inability to insert finger in oral opening	-	-	-	-	-	-	-	-	-	-
	Normal-o	-	-	-	-	-	-	-	-	-	-
	<2Finger-1	-	-	-	-	-	-	-	-	-	-
	<1Finger-2	-	-	-	-	-	-	-	-	-	-
2	Flexion difficulty of Neck	1	0	0	0	0	0	0	0	0	0
	Yes-1	-	-	-	-	-	-	-	-	-	-
	No-0	-	-	-	-	-	-	-	-	-	-
3	Difficulty swallowing	1	0	0	0	0	0	0	0	0	0
	Yes-1	-	-	-	-	-	-	-	-	-	-
	No-0	-	-	-	-	-	-	-	-	-	-
4	Upper limb rigidity	0	0	0	0	0	0	0	0	0	0
	Difficulty in flexion from anatomical position	-	-	-	-	-	-	-	-	-	-
	0-Normal	-	-	-	-	-	-	-	-	-	-
	1->30 degree	-	-	-	-	-	-	-	-	-	-
	2-<30 degree	-	-	-	-	-	-	-	-	-	-
	Lower limb rigidity	2	2	1	1	1	1	0	0	0	0
	Difficulty in flexion from anatomical position	-	-	-	-	-	-	-	-	-	-
	0-Normal	-	-	-	-	-	-	-	-	-	-
	1->30 degree	-	-	-	-	-	-	-	-	-	-
	2-<30 degree	-	-	-	-	-	-	-	-	-	-
5	Painful Spasms	1	1	1	1	1	1	1	0	0	0
	Yes-1	-	-	-	-	-	-	-	-	-	-
	No-0	-	-	-	-	-	-	-	-	-	-
6	Abdominal Guarding	0	0	0	0	0	0	0	0	0	0
	Yes-1	-	-	-	-	-	-	-	-	-	-
	No-0	-	-	-	-	-	-	-	-	-	-
7	Able to sit from supine position	2	2	2	2	2	2	2	1	1	0
	0-Normal	-	-	-	-	-	-	-	-	-	-
	1-Sit with help	-	-	-	-	-	-	-	-	-	-
	2-Inability to sit with help	-	-	-	-	-	-	-	-	-	-
8	Tachycardia/Bradycardia	1	1	1	1	1	1	1	1	1	0
	Yes-1	-	-	-	-	-	-	-	-	-	-
	No-0	-	-	-	-	-	-	-	-	-	-
9	Hypertension/Hypotension	0	0	0	0	0	0	0	0	0	0
	Yes-1	-	-	-	-	-	-	-	-	-	-
	No-0	-	-	-	-	-	-	-	-	-	-
10	Sweating(Unexplained)	1	0	0	0	0	0	0	0	0	0
	Yes-1	-	-	-	-	-	-	-	-	-	-
	No-0	-	-	-	-	-	-	-	-	-	-
	Total Score	11	8	6	6	6	5	5	2	2	0

of a case of tetanus presenting to Emergency department in daily practise the 'Tetpro' scoring system is open for further validation and discussion

**Conclusion**

In our experience with the patient and the scoring system we used, we found it very comfortable to monitor the progression of the disease and also for the daily counselling of the anxious family members regarding the response of the patient and possible outcome. In a developing country like India, recognising and treating a case of tetanus is a medical challenge and the scoring system we devised will help in its own way in this direction. The subject is open for discussion/acceptance and further validation.

**References**

1. Thwaites CL, Farrar JJ: Preventing and treating tetanus. *BMJ* 2003;326:117. [PMID: 12531822].
2. Centers for Disease Control and Prevention: Tetanus surveillance – United States, 2001–2008. *MMWR Morb Mortal Wkly Rep* 2011;60:365. [PMID: 21451446].
3. Tetanus, Joel Moll, Donna carden: *Tintinallis emergency medicine, A comprehensive study guide* 8<sup>th</sup> edition, chapter 156, page 1062.
4. Patel JC, Joag GG. Grading of tetanus to evaluate prognosis. *Indian J Med Sci* 1959;13:834-40.
5. Singh GP, Sikka PK, Gupta MM. Tetanus - a method of scoring to determine the prognosis. *Indian J Med Sci* 1986;40:124-8.
6. Sidhartha S S, Peter J V, Subhash H S, Cherian M, Jeyaseelan L, Cherian A M. A proposed new scoring system for tetanus. *Indian J Crit Care Med* 2004;8: 168-72.

## Myotonic Dystrophy: A Rare Autosomal Dominant Disorder

Vikram Shah<sup>1</sup>, Kishalay Datta<sup>2</sup>, Sarat Naidu<sup>3</sup>, Balasubramanyam E.V<sup>4</sup>,  
Sonal Singh<sup>4</sup>, Jitesh K. Bhandarkar<sup>3</sup>

### Author's Affiliation:

<sup>1</sup>Secondary DNB-PGY-02  
<sup>2</sup>Associate Director and HOD  
<sup>3</sup>DNB-PGY-03 <sup>4</sup>DNB-PGY-02,  
Emergency Medicine, Max Super  
Specialty Hospital, Shalimar  
Bagh, New Delhi,  
Delhi 110088, India.

### Corresponding Author:

**Vikram Shah**, Secondary DNB-  
PGY-02, Emergency Medicine,  
Max Super specialty Hospital,  
Shalimar Bagh, New Delhi, Delhi  
110088, India.  
E-mail  
drshah\_vikram@hotmail.com

Received on 03.10.2017,

Accepted on 23.10.2017

### Abstract

Myotonic Muscular Dystrophy is inherited form of an autosomal disease which may include cataract, low I.Q. , and heart conduction problems. In men their may be early balding and an inability to have children and gastric tract problems are common. It is a form of muscular dystrophy that affects muscles and many other organs in the body. Myotonia means an inability to relax muscles at will, which makes it difficult to relax the fingers after a firm hand grip. Muscular Dystrophy means progressive muscle degeneration leading to weakness and shrinkage of muscle tissues. It is caused by CTG triplet repeat expansion in non coding region of DMPK gene on chromosome 19q13.3, encoding myotonin. Myotonin is required for inter cellular conduction.

**Keywords:** Myotonic Dystrophy; Myotonia; Autosomal Dominant Disorder; Dmpk Gene; Myotonin.

### Introduction

Clinical presentation of myotonic dystrophy is extremely variable, even in families. It can vary from severe respiratory insufficiency in infancy to cataract alone in adulthood. Molecular DNA analysis and electromyogram (EMG) is routinely available for myotonic dystrophy, including pre-natal diagnosis.

### Clinical Presentation

A 51 year old male patient was brought to our E.D. at 2.00 pm with C/O slurring of speech, distension of abdomen and mild breathing difficulty since 2 days with B/L Upper and Lower Limb weakness since 9-10 months with B/L drooping of eyelids and diminision of vision since 4-5 years. No h/o fever, vomiting and change in bowel habits.

#### Primary Survey:

Airway Assesment: Patent

#### Breathing Assesment:

Respiratory rate - 16 CYCLES /MIN

Laboured breathing present

SPO2 at room air - 92%

SPO2 with oxygen-100% @ 3L/MIN O2 VIA nasal prongs.

#### Peripheral Pulsations

all peripheral pulsations present

Temperature : 98.2 F

Cardiac Monitor: Shows ST Depression.

Pupils: B/L Cataract Noted.

#### Secondary Survey:

#### Sample History

Physical Signs and Symptoms:- slurring of speech with laboured breathing with abdominal distension with B/L upper and lower limb weakness.

No Drug Allergy Known

Medications- not taken any treatment in the past

Past History: No H/O Dm, HTN, COPD, Weight

## Loss in the Past.

### *Investigations and Management in E.D.:*

12 Lead ECG Done Shows Sinus Rhythm @63 B/Min with Minimal ST Elevation in I and AVL with Deep T inversion in Antero-Lateral Leads.

2D ECHO: NO LV RWMA, EF = 60%

Troponin I – NEGATIVE

SOB Profile: CPKMB – 3.0 ng/ml

MYO – 220 ng/ml

TNI – < .05 ng/ml

BNP – 63.1 pg/ml

DDIM – 102 ng/ml

*Nerve Conduction Study:* Normal nerve conduction study in B/L upper and lower limbs.

*NCCT Head:* shows normal study.

*EMG Study:* Shows myopathic pattern/ muscular atrophy – Using Concentric Needle EMG Done In APB, FDI, ADM, Biceps, Tibialis Anterior, Vastus Lateralis, And L5 Paraspinal Muscles.

Reduced MUP'S and Incomplete IP'S are Recorded With Sign of Muscular Atrophy.

*Management in E.D.:* Patient was managed conservatively with NIV support and other supportive medications and supportive care as advised by Neurophysician and Cardiologist.

## Discussion

### *What is Already Known on this Topic*

Myotonic dystrophy type 1 is the most common adult onset of muscular dystrophy, presenting as a multi systemic disorder with extremely variable clinical manifestation, from asymptomatic adults to severely affected neonates.

### *Commonly Seen Complications*

Myopathy, Lens opacities, heart conduction defects, gastrointestinal dysfunction, obstructive sleep apnea and daytime hyperinsomnience, higher incidences of miscarriage in pregnancy are commonly seen.

### *Life Expectancy*

Mean age of Death is 60 years.

Mortality is most commonly due to pneumonia and

cardiac dysarrhythmias.

### *How this Might Change the Clinical Practice*

High level of clinical suspicion by ER Physician is needed for diagnosis.

Bed side general history, past history and drug history must be taken.

Prompt intervention with NIV to assist labored breathing is needed to reduce the work of breathing.

Genetic counseling is recommended to discuss the implications including the psychosocial and offspring risk reduction.

All survivors should undergo Annual Check-up for ECG, Urine Dipstick for Glucose and Ophthalmologist.

## Conclusion and Take Home Message

Myotonic Dystrophy is the most common heritable autosomal neuromuscular disorder.

As a ER Physician we should keep in mind regarding the typical presentation of such patients including the physical signs like early frontal balding, Ptosis, Lens opacities, inability to frown, clench teeth, smile and limb weakness. We should elicit the signs of Myotonia by asking the patient to rapidly relaxing the clenched fist or by tapping the thenar eminence and last but not least look for the ability to swallow and the pattern of breathing and gait of the patient will give us a good clue for early diagnosis and prompt treatment in highly suspected cases.

## References

1. Gibson G J, Pride N B, Davis J N. *et al* Pulmonary mechanics in patients with respiratory muscle weakness. *Am Rev Respir Dis* 1977;11(5):389-395 [PubMed].
2. Davis J, Goldman M, Loh L. *et al* Diaphragm function and alveolar hypoventilation. *Q J Med* 1976;45:87-100. [PubMed].
3. Griggs RC, Donohoe KM, Utell M J. *et al* Evaluation of pulmonary function in neuromuscular disease. *Arch Neurol* 1981;12:389-12. [PubMed].
4. Ward NS, Hill NS. Pulmonary function testing in neuromuscular disease. *Clin Chest Med* 2001;22:769-781. [PubMed].
5. ATS/ERS Statement on respiratory muscle testing. *Am J Respir Crit Care Med* 2002;16(6):518-624. [PubMed].

6. Stefanutti D, Benoist M R, Scheinmann P. et al Usefulness of sniff nasal pressure in patients with neuromuscular or skeletal disorders. *Am J Respir Crit Care Med* 2000;162(Pt 1):1507-1511. [PubMed].
7. Hart N, Polkey M I, Sharshar T. et al Limitations of sniff nasal pressure in patients with severe neuromuscular weakness. *J Neurol Neurosurg Psychiatry* 2003;74:1685-1687. [PMC free article] [PubMed].
8. Chaudri M B, Liu C, Watson L. et al Sniff nasal inspiratory pressure as a marker of respiratory function in motor neuron disease. *Eur Respir J* 2000;15:539-542. [PubMed].
9. Suarez A A, Pessolano F A, Monteiro S G. et al Peak flow and peak cough flow in the evaluation of expiratory muscle weakness and bulbar impairment in patients with neuromuscular disease. *Am J Phys Med Rehabil* 2002;81:506-511. [PubMed].
10. Bach J R, Saporito L R. Criteria for extubation and tracheostomy tube removal for patients with ventilatory failure. A different approach to weaning. *Chest* 1996;110:1566-1571. [PubMed].

# REDKART.NET

(A product of RF Library Services (P) Limited)  
(Publications available: Journals, Books, Articles and Single issues)  
(Date range: 1967 to till date)

**T**he Red Kart is an e-commerce and is a product of RF Library Services (P) Ltd. It covers a broad range of journals, Books, Articles, Single issues (print & Online). The publications are available in print and online (PDF) form and the all are in Hindi and English languages. All these publications are in stock for immediate shipping and online access in case of online.

## **Benefits of shopping online are better than conventional way of buying.**

1. Convenience.
2. Better prices.
3. More variety.
4. Fewer expenses.
5. No crowds.
6. Less compulsive shopping.
7. Buying old or unused items at lower prices.
8. Discreet purchases are easier.

URL; [www.redkart.net](http://www.redkart.net)

## A Rare Serious Ocular Side Effect of Topiramate: Bilateral Acute Angle Closure Glaucoma

Dhruvkumar M. Patel<sup>1</sup>, Mukundkumar V. Patel<sup>2</sup>, Ajay V. Garg<sup>3</sup>

---

### Author's Affiliation:

<sup>1</sup>3rd Year Student, Smt NHL Municipal Medical College, Ellis Bridge, Ahmedabad, Gujarat 380006, India. <sup>2</sup>Consultant Physician, Dhruv Healthcare Multi-Speciality Hospital, 59, Rajeshwari Society, CTM Crossroad, Amraiwadi, Ahmedabad, Gujarat 380026, India. <sup>3</sup>Consultant, Ophthalmologist, Shivam Eye Hospital, Krishna Complex, Nr.Wonder Point, Amraiwadi, Gujarat 380026, India.

### Corresponding Author:

**Mukundkumar V. Patel,**  
Consultant Physician, Dhruv Healthcare Multi-Speciality Hospital, 59, Rajeshwari Society, CTM Crossroad, Amraiwadi, Ahmedabad, Gujarat 380026, India.  
E-mail:  
[drmkundvpatel123@yahoo.co.in](mailto:drmkundvpatel123@yahoo.co.in)

Received on 25.09.2017,  
Accepted on 13.10.2017

### Abstract

Topiramate is an anticonvulsant drug which is also used for migraine prophylaxis. It has many neurological and psychiatric side effects in addition to diarrhoea and weight loss. It can cause serious ocular side effects like sudden dimness of vision secondary to acute narrow angle glaucoma and or myopia. These side-effects usually occur at more than 200 mg per day dose and after 4 to 6 weeks of starting treatment with the drug. We report a case of 20-years old female who developed sudden dimness of vision in both eyes after migraine prophylaxis with topiramate 25 mg daily for seven days. Her cause of this vision problem was secondary acute angle glaucoma and myopia because of topiramate. After stopping the drug her vision became normal within seven days. Clinicians should explain ocular side effects of topiramate and if he/she develops such visual problems, he/she should stop the drug and consult clinician immediately.

**Keywords:** Rare Ocular Side Effect; Topiramate.

---

### Introduction

Topiramate is an antiepileptic drug which is also used for migraine prophylaxis, bipolar disorder and neuralgia [1]. Topiramate's common side-effects are diarrhoea, weight loss, excessive sleepiness, dizziness, cognitive and behavioural problems, suicidal thoughts, high grade fever with anhidrosis. 1-2 out of one hundred patients receiving topiramate have renal stone side effect and it is manageable with medical treatment. It rarely causes ocular side effects like acute myopia, secondary acute angle closure glaucoma (AACG), uveitis, scleritis, choroid effusion and visual field defects [2] and if not diagnosed early, it may lead to permanent Vision loss [1]. Ciliochoroidal effusion (Idiosyncratic adverse reaction of drug) leading to anterior shifting of lens iris diaphragm make anterior chamber shallow and this cause AACG. The side-effects usually occur at higher dose of 200 mg per day and after 4 to 6 weeks of treatment with drug [3-6].

### Case Report

A 20-year-old female was having history of chronic recurrent unilateral throbbing headache 6 to 8 times in a month associated with nausea and photophobia since last two years. Her mother also had history of headache suggestive of migraine. She had consulted ophthalmologist before 7 days as she thought refractive error as a cause of headache but her ophthalmic examination was normal at that time. On examination her vitals and neurological examination were normal. Her headache was diagnosed as migraine headache. She was prescribed tablet Topiramate 25mg once daily for 7 days and then to step up twice daily after 7 days for prophylaxis of migraine along with tablet naproxen 500mg SOS for headache relief. After 7 days of starting Topiramate, she developed sudden dimness and blurring of vision of both eyes not associated with headache or other neurological symptoms. She consulted ophthalmologist again and he diagnosed glaucoma

as a cause for vision symptoms. He referred her to glaucoma clinic and was diagnosed uveal effusion causing myopic shift and acute angle closure glaucoma of both eyes likely because of Topiramate. Topiramate was stopped and cholinergic eye drops was prescribed for 7 days. Her vision became completely normal after 7 days and she became asymptomatic.

## Discussion

Our patient developed acute angle closure glaucoma (AACG) after taking topiramate 25mg OD dose for seven days which resolved spontaneously after stopping it. Ocular side effects of topiramate are not mentioned in standard pharmacological

textbooks. By reviewing literature, it is found that Topiramate can cause serious ocular side-effects like acute angle closure glaucoma, acute myopia, suprachoroidal effusion, periorbital oedema, scleritis, oculogyric crisis. But these side effects usually occur when topiramate is given in dose of 200 mg per day or higher or after 4 to 6 weeks of treatment. In our case ocular side effect occurred at dose of 25 mg per day for seven days which is quite unusual [1-6].

### Mechanism

Topiramate causes Ciliary body oedema or cilio-choroidal detachment which leads to forward rotation of ciliary body and displacement of the iris. It closes the anterior chamber angle precipitating an attack of AACG. Swelling of lens may also contribute to the shallow anterior chamber [3-6]. (Figure 1).

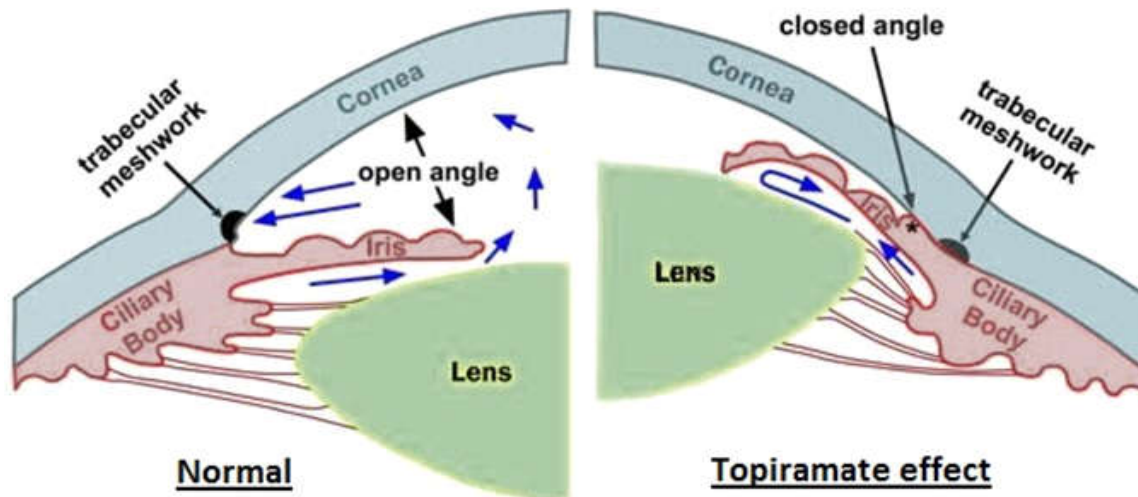


Fig. 1: Schematic Topiramate Ocular side Effects Mechanism

## Conclusion

Topiramate is frequently used for migraine and epilepsy and it may cause rare ocular side effects like dimness of vision even with low dose of 25mg per day and short duration of seven days' treatment. Our patient's cause of sudden decrease in vision was likely due to topiramate induced myopia and secondary acute angle glaucoma. So clinician should counsel the patient regarding possible ocular side-effects of topiramate and should contact him immediately for any ocular symptoms. If patient is not instructed for possible ocular side effect of Topiramate patient may have to undergo extensive costly investigations for vision problem. If drug is not discontinued timely, permanent vision damage may occur.

## References

1. Shorvon, S. D. Safety of Topiramate: Adverse Events and Relationships to Dosing. *Epilepsia*, 1996;37:S18-S22. doi: 10.1111/j.1528-1157.1996.tb06029.x.
2. Fraunfelder FW, Fraunfelder FT, Keates EU. Topiramate-associated acute, bilateral, secondary angle-closure glaucoma. *Ophthalmology* 2004; 111(1):109-11.
3. Thambi L, Kapcala LP, Chambers W, Nourjah P, Beitz J, Chen M, et al. Topiramate-associated secondary angle-closure glaucoma: a case series. *Arch Ophthalmol* 2002;120(8):1108.
4. Rhee DJ, Goldberg MJ, Parrish RK. Bilateral angle-closure glaucoma and ciliary body swelling from topiramate. *Arch Ophthalmol* 2001;119(11):1721-3.



5. Chen TC, Chao CW, Sorkin JA. Topiramate induced myopic shift and angle closure glaucoma. *Br J Ophthalmol* 2003;87(5):648-9.
  6. Medeiros FA, Zhang XY, Bernd AS, Weinreb RN. Angle-closure glaucoma associated with ciliary body detachment in patients using topiramate. *Arch Ophthalmol* 2003;121(2):282-5.
-

## A Case Report on Stroke in Young

Aisvarya Girotra<sup>1</sup>, Balasubramanyam E.V.<sup>2</sup>, Hilal Ahmad Yatoo<sup>3</sup>,  
Kishalay Datta<sup>4</sup>

---

### Author's Affiliation:

<sup>1</sup>MEM Resident <sup>2</sup>DNB Resident  
<sup>3</sup>Attending Consultant <sup>4</sup>Associate  
Director & HOD, Emergency  
Medicine, Max Hospital, Shalimar  
Bagh, New Delhi, Delhi 110088,  
India.

### Corresponding Author:

**Aisvarya Girotra**  
MEM Resident, Department of  
Emergency Medicine, Max  
Hospital, Shalimar Bagh, New  
Delhi, Delhi 110088, India.  
E-mail:  
aisvarya.girotra93@yahoo.in

Received on 21.09.2017,

Accepted on 13.10.2017

### Abstract

Stroke is often considered as a disease of elder population, being uncommon in young has a large socio-economic impact to the families by leaving the victim disable before their most productive years. Of all stroke cases, 10% are seen in patients younger than 45 years of age. The risk factors for stroke in young adult patients can be traditional vascular risk factors but rare risk factors are not uncommon. Stroke in young patients; though considered to have a better prognosis than stroke in the older population; can cause significant limitations in quality of life of these patients, with them being at higher risk of cardiovascular events as well as higher death rate. Such patients also have a higher five year risk of recurrent stroke especially if associated with age > 40 years, type 1 DM, history of TIA, Hypertension. Here we present a case report of an Acute left basal ganglia ischemic stroke in a young previously diagnosed hypertensive male, non compliant to anti-hypertensive medications.

**Keywords:** Ischemic Stroke; Stroke in Young; Thrombolysis; Vascular Risk Factor; TEE-Trans Oesophageal Echocardiography.

---

### Introduction

Stroke is a major cause of disability and death worldwide. Young stroke applies to an age group of >18 to < 45 years (excluding pediatric stroke <18 years). Acute stroke is defined as sudden onset of focal neurological deficits, presumably of vascular origin, lasting more than 24 hours or leading to death. Cerebral infarction in younger age groups may be due to a variety of local, systematic diseases. Full evaluation of the young patient will reveal an underlying cause, many of which are treatable. The management of young stroke requires a modified approach, prompt and focussed investigations and treatment, as well as advice on prognosis.

### Case Report

A 28 year old male presented to ER with complaints of sudden onset right upper limb weakness associated

with deviation of mouth to left side 10 minutes prior to presentation. The patient gave no history of slurring of speech, seizure, headache, vomiting, LOC, trauma, chest pain, fever. Past medical history revealed Hypertension (non compliant to anti-hypertensive medications).

#### On Examination

Working diagnosis - CVA with Right UMN facial paralysis, right hemiparesis, dysarthria-Young stroke (? Cause).

Pt was thrombolysed in ED with Inj Actilyse 50 mg total dose, 5 mg as bolus and 45 mg as infusion over 60 min with continued BP, GCS monitoring. Post thrombolysis NCCT HEAD was normal and admitted in ICU.

Further investigations revealed:

CBC and KFT were Normal

Homocysteine- 17.5umol/L

Cholesterol- 193 mg/dL  
 Triglyceride-333 mg/dL  
 HDL- 40.6 mg/dL,  
 LDL- 128 mg/dL  
 TSH- 0.2uIL/ml  
 APTT- 34.3, PT- 11, INR- 0.93  
 ECHO- borderline concentric LVH, no LV RWA,

LVEF 60%, valves normal, no clot, veg, PE. TRACE TR  
 RVSP- 24 mmHg.

Carotid doppler- normal study.

Patient showed gradual neurological recovery; managed conservatively with T. Aspirin, Enoxaparin, Furosemide and was discharged after 4 days on anti-platelet and anti-hypertensive medication, planned for TEE, Holter, ANA and vasculitis panel.

### On Examination

Primary Survey	Secondary Survey
AIRWAY- Patent	HEENT- no pallor, icterus, cyanosis; tongue moist
BREATHING- Respiration(RR/min)- 18 Laboured- No SpO2- 100% on room air	CHEST- B/L air entry equal, no add sounds CVS- S1 S2 +, no murmur ABD- soft, non tender, BS + EXT- warm, no pedal edema, no dilated veins, all peripheral pulses palpable CNS- Conscious, oriented to time,place and person
CIRCULATION- Pulse- 98/min Blood pressure- 150/90 mmHg Peripheral pulses – Y Temperature- 98.4 F	Power- RT U/L- 0/5 RT L/L- 4/5 LT U/L and L/L – 5/5 hand grip- Rt absent, Lt 100%
DISABILITY- GCS- 15/15 Pupils- B/L 2mm normal reacting to light GRBS- 126 mg/dl	Plantars- Right extensor, left flexor Speech- Mild dysarthria +, no aphasia Cranial nerves- right UMN facial paralysis+, Deviation of mouth to left side present Tone- decreased in right UL and LL, normal in left UL and LL. Sensory – normal No cerebellar signs No signs of meningeal irritation No slurring of speech NIHSS 7 Weight- 60kg

### Discussion

Presentation of young patients with stroke is similar to stroke in the elderly but mis/delayed diagnosis of stroke in young is a common occurrence because it is still considered a disease of the elderly and the patient may not have any comorbidities leading to low suspicion for stroke.

Apart from the typical presentation, atypical presentations of stroke such as Neuropsychiatric symptoms (delirium, depressed level of consciousness), abnormal movements (chorea, hemiballismus, dystonia, hemifacial spasm etc), cranial neuropathies (acute vestibular syndrome, acute hearing loss, Horner syndrome, third/seventh

nerve palsies) and Isolated symptoms (isolated dysarthria, isolated facial paresis, isolated dystonia, isolated visual loss, monoparesis, isolated headache) need to be considered while making a diagnosis of stroke.

Treatment includes Urgent Thrombolysis (if indicated), Aspirin and treatment of underlying cause (if known).

Prognosis in young stroke depends upon the underlying cause and extent of initial neurological damage. The greater collateral reserve in the young adult brain limits the initial size of infarction and there is greater scope for functional recovery than in the elderly. Initial mortality is 2-7% and risk of recurrent stroke is 1-3% per annum.

Patients with premature atherosclerosis as a cause of stroke have a higher chance of future morbidity. Physiotherapy, occupational therapy, speech therapy and psychiatric interventions are especially important in young stroke cases.

### Conclusion

Recent advances in imaging modalities, hematology, immunology and genetics have enabled early and accurate diagnosis of stroke in patients. We report on this patient because ischaemic stroke in a 28 year old patient and his subsequent neurological recovery over the course of hospitalisation is a classic case of progression and recovery from the disease. A complete but quick neurological examination is essential in the ED. Distinguishing acute stroke from other disorders that mimic stroke (hypo/hyperglycemia, hyponatremia, seizures, migraines, multiple sclerosis, intracranial infection/tumours etc.) is vital in the ED to ensure prompt and appropriate management. Early and accurate diagnosis enable us to intervene early in the progression of the disease which significantly affects the patients ultimate outcome.

### References

1. Nedeltchev K, der Maur TA, Georgiadis D, et al. Ischaemic stroke in young adults: predictors of outcome and recurrence. *J Neurol Neurosurg Psychiatry*. 2005;76:191-195.
2. Varona JF, Guerra JM, Bermejo F, Molina JA, de la Camara Gomez. Causes of ischemic stroke in young adults, and evolution of the etiological diagnosis over the long term. *Eur Neurol*. 2007;57:212-2.
3. Marini C, Russo T, Felzani G. Incidence of stroke in young adults: a review. *Stroke Res Treat*. 2011;2011:535672.
4. Smajlovic DŽ, Salihovic D, Ibrahimagic OC, Sinanovic O. Characteristics of stroke in young adults in Tuzla Canton, Bosnia and Herzegovina. *Coll Antropol*. 2013;37:515-519.
5. Groppo E, De Gennaro R, Granieri G, et al. Incidence and prognosis of stroke in young adults: a population-based study in Ferrara, Italy. *Neurol Sci*. 2012;33:53-58.
6. Jacobs BS, Boden-Albala B, Lin IF, Sacco RL. Stroke in the young in the Northern Manhattan Stroke Study. *Stroke*. 2002;33:2789-2793.
7. Rasura M, Spalloni A, Ferrari M, et al. A case series of young stroke in Rome. *Eur J Neurol*. 2006;13:146-152.
8. Putaala J, Yesilot N, Waje-Andreassen U, et al. Demographic and geographic vascular risk factor differences in European young adults with ischemic stroke: the 15 Cities Young Stroke Study. *Stroke*. 2012;43:2624-2630.
9. Yesilot Barlas N, Putaala J, Waje-Andreassen U, et al. Etiology of first ever ischaemic stroke in European young adults: the 15 Cities Young Stroke Study. *Eur J Neurol*. 2013;20:1431-1439.
10. Mackey J. Evaluation and management of stroke in young adults. *Continuum (Minneapolis)* 2014;20:352-369.

## A Case Report on Acute Myocardial Infarction in Young: Atypical ECG Changes Vs. Angiographic Correlation

Aisvarya Girotra<sup>1</sup>, Kishalay Datta<sup>2</sup>, Rigenjyoti Kalita<sup>3</sup>

---

### Author's Affiliation:

<sup>1</sup>MEM Resident <sup>2</sup>Associate Director & HOD <sup>3</sup>Attending Consultant, Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

### Corresponding Author:

**Aisvarya Girotra**  
MEM Resident, Department of Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.  
E-mail:  
[aisvarya.girotra93@yahoo.in](mailto:aisvarya.girotra93@yahoo.in)

Received on 21.09.2017,

Accepted on 13.10.2017

### Abstract

Acute myocardial infarction (AMI) among young is relatively uncommon. Coronary artery disease (CAD) mostly occurs in persons older than 45 years of age. In recent times, with the advent of sedentary lifestyles, smoking, drug abuse and obesity; among other traditional risk factors; incidence of young patients suffering from acute coronary syndrome in particular acute MI, is on the rise. Atypical presentations and the reluctance to seek medical attention are other contributory factors in young adults. The disease carries significant morbidity, psychological as well and financial effects on the patient and his close ones. Here we have reported a case of a 28 year old male with no known co morbidities presenting to the ED with ongoing chest pain since an hour and h/o diaphoresis. Patient was evaluated in ED, ECG suggestive of progressively increasing ST segment elevation in inferior leads. The patient was evaluated, Coronary angiography was done and found to have an uncommon Apical Left Anterior Descending artery (Type III or "wraparound" LAD) occlusion leading to an inferior wall MI.

**Keywords:** Acute MI; Thrombolysis; Coronary Angiography; Troponin I; Angioplasty; AMI- Anterior Myocardial Infraction.

---

### Introduction

Chest pain in young adults has a diagnostic challenge in Emergency. They are more prone to misdiagnose due to lack of established risk factors. Acute MI is defined as a clinical or pathological event caused by myocardial ischemia in which there is evidence of myocardial injury or necrosis. Acute MI in young is usually defined as MI in ages < 45 years. In recent times, with the advent of sedentary lifestyles, smoking, drug abuse and obesity; among other traditional risk factors; incidence of young patients suffering from acute coronary syndrome in particular acute MI, is on the rise.

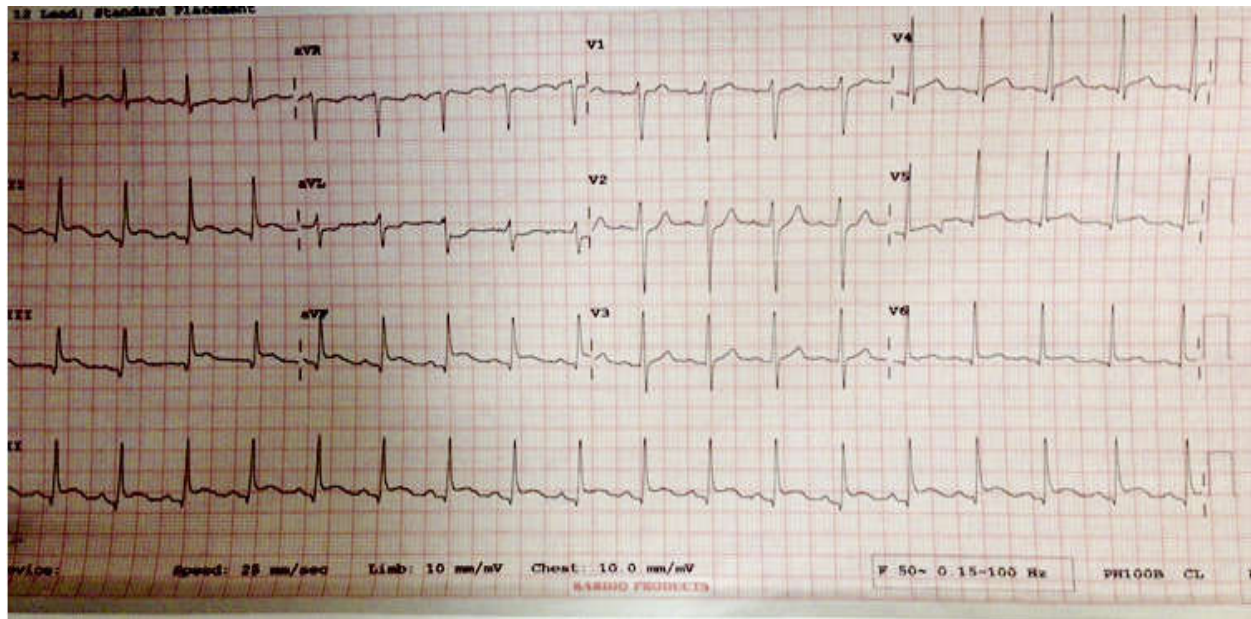
In Global Registry of Acute Coronary Events (GRACE) study, the prevalence of young acute coronary syndrome (ACS) was 6.3% [1]. Atypical presentations and the reluctance to seek medical attention are other contributory factors in young

adults. The disease carries significant morbidity, psychological as well and financial effects on the patient and his close ones.

### Case Presentation

A 28 year old male presented to ER with complaints of chest pain since an hour associated with radiation of pain to right arm and diaphoresis. The patient gave no history of breathlessness, fever, nausea, vomiting, palpitations.

Patient's past medical history was not significant. Patient was a smoker for 3-4 years and had family h/o ACS. He was evaluated in ED; Vitals were stable with pulse-102/min and BP on higher side (BP-150/100 mm hg). Rest systemic examination did not show any abnormality. ECG - ST elevation in inferior leads and Troponin I- positive.



Working diagnosis – STEMI- Acute Inferior Wall MI – Young MI (? Cause)

Patient was loaded with Tab. Disprine, Tab Clopitab and Tab . Atorva and shifted to cath lab for Coronary intervention .Angiography revealed 99% occlusion of proximal LAD and 100 % occlusion of apical LAD (Type III or “wraparound” LAD). PCI to LAD (Thrombosuction) was performed. Thrombus burden was reduced but residual thrombus was present so stent was not implanted. Check angiography after 2 days revealed no residual thrombus or stenosis of proximal LAD; distal LAD after turning at apex was 100% occluded. No further intervention to proximal LAD was planned.

Patient showed prompt recovery post procedure and was managed conservatively with Ecosprin, Enoxaparin, Atorvastatin, Ivabradine, Ticagrelor, Metoprolol, Nicorandil and Analgesia. Patient’s 2D-Echo revealed basal and mid inferior wall hypokinesia with LVEF 55% and other bio-chemical tests were normal.

### Conclusion

Of all the patients of coronary artery disease, 3% of the cases occur in young adults less than 45 years of age. Risk factors like smoking, obesity, lack of physical activity and abuse of recreational drugs (cocaine) has increased the incidence to AMI in young adults. In this report, we shall be discussing a patient who is obese, is a smoker, leads a life with lack of exercise and family history of ACS.

Causes of MI in a young adult can be divided into 4 groups

1. Atheromatous CAD- cigarette smoking, positive family history of CAD, obesity, Dyslipidemias, hyperhomocystenemia
2. Non atheromatous CAD- Congenital coronary artery anomalies, carotid dissection, infective endocarditis, myocardial bridging, IV drug users
3. Hypercoagulable states- Antiphospholipid syndrome, Nephrotic syndrome, Factor V Leiden mutation, oral contraceptive use.
4. Recreational drug use- cocaine, amphetamines, marijuana, binge alcohol drinking

Presentation of young patients with AMI is very different to that of AMI in the elderly. In young patients, the first onset of angina rapidly progresses to MI unlike the elderly where worsening angina over a period of time progresses to MI. An ECG should be performed ideally within 10 minutes of presentation to the ED. Treatment includes concomitant use of oxygen, analgesics, Antiplatelets, Antithrombins, Fibrinolytics and other anti-ischemic agents. A check angiography may be indicated in cases where residual thrombus is found.

Recent advances in imaging modalities and access to catheterization labs have enabled early and accurate diagnosis and management of MI in patients. Inferior wall MI is most commonly associated with a Right Coronary Artery occlusion or even a Left Circumflex Artery occlusion. We report on this patient because it is an uncommonly seen case of acute inferior

wall MI due to apical Left Anterior Descending artery (LAD III) occlusion in a 28 year old male known to have no co morbidities. The case also demonstrates the prompt relief of symptoms post procedure as well as timely discharge from the hospital. Distinguishing acute MI from other disorders that might present with similar complaints (gastritis, pancreatitis, GERD, spontaneous pneumothorax, aortic dissection) is essential to significantly improve the patients ultimate outcome.

### References

1. Avezum A, Makdisse M, Spencer F, Gore JM, Fox KA, Montalescot G, et al. Impact of age on management and outcome of acute coronary syndrome: Observations from the Global Registry of Acute Coronary Events (GRACE) *Am Heart J.* 2005;149:67-73.
2. Padler FA, Comad AR. Myocardial infarction with normal coronary artery: A case report and review of literature. *Am J Med Sci.* 1997;314:342-5.
4. Hamsten A, Norberg R, Björkholm M, de Faire U, Holm G. Antibodies to cardiolipin in young survivors of myocardial infarction: An association with recurrent cardiovascular events. *Lancet.* 1986;1:113-6.
5. Xie CB, Chan MY, Teo SG, Low AF, Tan HC, Lee CH. Acute myocardial infarction in young Asian women: A comparative study on Chinese, Malay and Indian ethnic groups. *Singapore Med J.* 2011;52:835-9.
6. Milonig G, Malcolm GT, Wick G. Early inflammatory and immunological lesions in juvenile atherosclerosis from the pathological determinants of atherosclerosis in youth (PDAY) study. *Atherosclerosis.* 2002;160:444-8.
7. Morillas P, Bertomeu V, Pabón P, Ancillo P, Bermejo J, Fernández C, et al. Characteristics and outcome of acute myocardial infarction in young patients. The PRIAMHO II study. *Cardiology.* 2007;107:217-25.

# One and Half Syndrome in Acute Pontine Infarct: A Rare Entity

Anita Rawat<sup>1</sup>, Kishalay Datta<sup>2</sup>, Vaibhav Gulati<sup>3</sup>

## Author's Affiliation:

<sup>1</sup>Associate Consultant <sup>2</sup>HOD and Associate Director <sup>3</sup>PGY-3, MEM {GWU-USA}, Dept of Emergency Medicine, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

## Corresponding Author:

**Vaibhav Gulati**, PGY-3, MEM {GWU-USA} Dept of Emergency Medicine, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.  
E-mail: [dr.vaibhavgulati@gmail.com](mailto:dr.vaibhavgulati@gmail.com)

Received on 25.10.2017,

Accepted on 07.11.2017

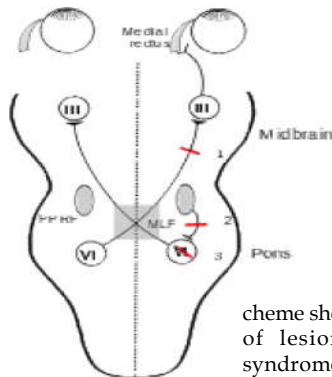
## Abstract

One-and-a-half syndrome is a clinical disorder characterized by an ipsilateral conjugate horizontal gaze palsy and an ipsilateral internuclear ophthalmoplegia. The main causes of this rare syndrome are stroke and multiple sclerosis. Other causes include tumors, AV malformations, basilar artery aneurysms and rarely, vasculitis, brainstem tuberculoma and neurocysticercosis. Here we present a case of 69 year old male patient who presented to emergency with only blurring of vision and was diagnosed to have acute left sided/paramedian acute dorsal pontine infarct, one and a half syndrome.

**Keywords:** One and Half Syndrome; Infarct; Pons.

## Introduction

One-and-a-half syndrome is a clinical disorder characterized by an ipsilateral conjugate horizontal gaze palsy (the "one") and an ipsilateral internuclear ophthalmoplegia (the "half"). The most common manifestation of this unusual syndrome is limitation of horizontal eye movement to abduction (moving away from the midline) of one eye (e.g. right eye in the diagram) with no horizontal movement of the other eye (e.g. left eye in the diagram). Nystagmus is also present when the eye on the opposite side of the lesion is abducted. Convergence is classically spared as cranial nerve III (oculomotor nerve) and its nucleus is spared bilaterally.



cheme showing anatomical location of lesions in one and a half syndrome.

The syndrome usually results from single unilateral lesion of the paramedian pontine reticular formation and the ipsilateral medial longitudinal fasciculus. An alternative anatomical cause is a lesion of the abducens nucleus (VI) on one side (resulting in a failure of abduction of the ipsilateral eye and adduction of the contralateral eye = conjugate gaze palsy towards affected side), with interruption of the ipsilateral medial longitudinal fasciculus after it has crossed the midline from its site of origin in the contralateral abducens (VI) nucleus (resulting in a failure of adduction of the ipsilateral eye). The main causes of this rare syndrome are stroke and multiple sclerosis. Other causes include tumors, AV malformations, basilar artery aneurysms and rarely, vasculitis, brainstem tuberculoma and neurocysticercosis.

## Case Report

Sixty nine year old male brought to emergency department with history of blurring of vision from one day. There was no loss of consciousness, headache, trauma, fever, cough, vomiting, weakness of any part of the body, paresthesias or numbness of limbs or face, urinary incontinence, deafness, tinnitus or any slurring of speech.



*On presentation:*

**Primary Survey**

*Airway:* Patent

*Breathing:* Respiratory rate- 20/min

SpO<sub>2</sub> – 99% on room air

*Circulation:* Heart rate- 100 bpm

*Blood Pressure:* 130/70 mm of Hg

*Peripheral Pulses:* palpable, good volume , rhythmic.

*Disability:* GCS- E4V5M6

*Pupils:*

*Right Eye:* NSNRL, lateral gaze along with ptosis present

*Left Eye:* NSNRL.

*Exposure:* T-98 F

GRBS: 125mg/dl

ECG : 1st ECG: NSR

**Secondary Survey**

HEENT: No external head/neck/face injury.  
No Cervical tenderness present.

EYE: Rt- abducted, vision - 6/6  
Lt - fixed at the midline, vision - 6/6.

RS: Trachea midline, No distended  
neck veins.  
B/L air entry equal, no added sounds.

CVS: S1,S2 heart sounds normally heard.

P/A: No visible bruise, abdomen soft, Non  
tender, bowel sounds normally heard.  
No external genitalia injury.

CNS: Conscious, oriented. Power- B/L UL/  
LL-5/5, sensations-intact, tone-normal,  
DTR-normal.

Ample

Allergies: No known allergies

Medication: on OHA, regular medications

Past medical history: K/C/O DM from 20 Yrs , On  
OHA

Events leading to incident: As described above.

Investigations

MRI brain shows focal acute infarct in left dorsal pons.

Care Plan- patient was admitted under neurology  
department in ICU and treatment started accordingly.

**Discussion**

Pontine lesion boundaries there were five main clinical patterns that depended on the constant territories of intrinsic pontine arteries: (1) Anteromedial pontine syndrome who present with motor deficit with dysarthria, ataxia, and mild tegmental signs in one third of patients; (2) Anterolateral pontine syndrome developed with motor and sensory deficits in half of the patients, and were associated with tegmental signs more frequently than the anteromedial infarct syndrome; (3) Tegmental pontine syndrome presented with mild motor deficits and associated with sensory syndromes, eye movement disorders and vestibular system symptoms including vertigo, dizziness and ataxia; (4) Bilateral pontine syndrome consisted with transient consciousness loss, tetraparesis and acute pseudobulbar palsy; (5) Unilateral multiple pontine infarcts were rarely observed, and were always associated with severe sensory-motor deficits and tegmental signs. The clinical pattern is according to the area and subsequent nucleus involvement. There can be some variation in the clinical patterns and these can be overlapping as well.

**References**

1. Maqbool Wani, Asrar Ahmed, Rouf Asmi, Mushatq Wani, Amit Sharma, Saima Nazir. One-and-a-half syndrome in Pontine hemorrhage- JK-Practitioner 2007;14(1):38-40
2. Merck. The Merck Manual Home Health Handbook. Wiley, 2011.
3. Ophthalmology and Visual Sciences (<http://medicine.uiowa.edu/eye>).
4. Dan Longo, Anthony Fauci, Dennis Kasper, Stephen Hauser, J. Jameson, Joseph Loscalzo. Harrison's Principles of Internal Medicine, 18th Edition, McGraw Hill Professional, 2011.
5. Michael Wall, MD and Shirley H. Wray. The one and a half syndrome A unilateral disorder of the pontine tegmentum. A study of 20 cases and review of the literature. Neurology August 1983;33(8):971
6. Patricio S. Espinosa. Teaching NeuroImage: One-and-a-half syndrome. Neurology January 29, 2008;70(5):e20
7. C N Martyn and D Kean. The one-and-a-half syndrome. Clinical correlation with a pontine lesion demonstrated by nuclear magnetic resonance imaging in a case of multiple sclerosis. Br J Ophthalmol. 1988 Jul;72(7). PMC1041515.

## Carcinoma Prostate with Metastasis to Vertebral Column and Right Cerebellum Causing Sol and Hydrocephalus

**E.V. Balasubramanyam<sup>1</sup>, Sonal Singh<sup>1</sup>, Indranil Das<sup>1</sup>, Kishalay Datta<sup>2</sup>**

---

### Author's Affiliation:

<sup>1</sup>DNB Resident <sup>2</sup>Associate Director and HOD,  
Dept. of Emergency Medicine, Max Super  
Specialty Hospital, Shalimar Bagh, New Delhi,  
Delhi 110088, India.

### Corresponding Author:

**E.V. Balasubramanyam**  
DNB Resident, Dept. of Emergency Medicine,  
Max Super Specialty Hospital, Shalimar Bagh,  
New Delhi, Delhi 110088, India.  
E-mail: [subramanyamev@gmail.com](mailto:subramanyamev@gmail.com)

Received on 05.08.2017,

Accepted on 01.09.2017

### Abstract

A patient Mr. Satya Narain Chauhan, 67 yrs old male, diagnosed case of DM, Ca prostate presented with complaints of urinary retention and slurring of speech. On examination Mr. Chauhan is having left lower limb weakness-not able to stand/walk, midline lumbosacral tenderness present. So, Mr. Chauhan was evaluated for spinal cord compression, and to rule out CVA. Patient had sclerotic metastasis in lumbosacral spine, and mass lesion in cerebellum.

**Keywords:** Carcinoma Prostate; Cerebellum; Hydrocephalus.

---

### Introduction

Although vertebral and epidural metastasis are common in adenocarcinoma of prostate, intra cerebral, cerebellar and intramedullary metastasis occur in rare.

This is a case of adenocarcinoma prostate on chemotherapy with eisenmengers syndrome, which developed vertebral metastasis along with right cerebellar metastasis.

Patient was given palliative treatment comprising chemotherapy, radiation therapy, physiotherapy and planned for VP shunting in view of SOL in Right cerebellum with hydrocephalus.

### Case Presentation

Presenting complaints of patient are abdominal discomfort and not able to pass urine, with H/o weakness of left lower limb.

H/o slurring of speech present.

No H/o chest pain, SOB, cough, fever, loose motions, hematuria, burning micturition, increased frequency of micturition.

On Examination

#### Primary Survey

AIRWAY: Patent

#### Breathing

Respiration(RR/min): 20/MIN

Laboured: No

SpO<sub>2</sub>: 100% on Room Air

#### Circulation

Pulse: 72/MIN

BP: 130/90 MMHG

Peripheral Pulses: Yes

Temperature: 98.4 F

#### Disability

GRBS: 139mg/dl

Pupils:

Right eye: NSNR

Left eye: NSNR

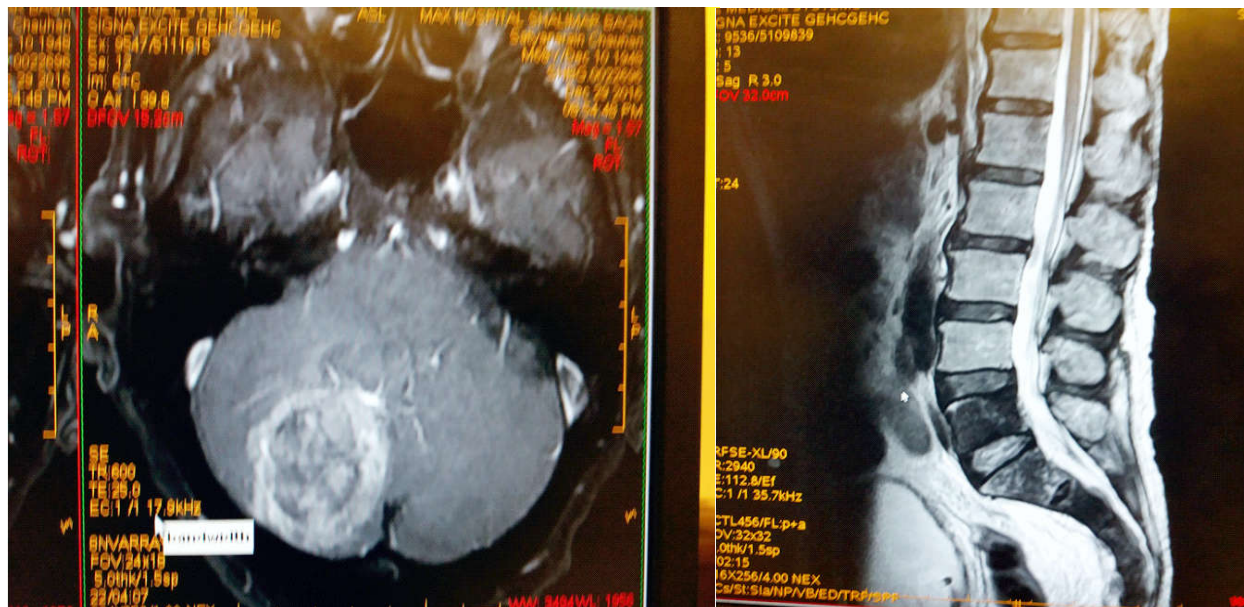
#### Secondary Survey

Review of Systems

HEENT : Pallor +, No Icterus, Cyanosis, Tongue Moist.

CHEST: B/L AE +, no added sounds  
CVS: S1S2 +, no added sounds  
ABD: Soft, swelling in lower abdomen suggesting bladder distention+, BS +,  
EXT: Warm, No Pedal Edema, No Dilated Veins  
Neuro: Conscious, Coherent, Oriented  
RT UL-TONE-N, POWER-5/5,

LEFT UL-TONE-N, POWER-5/5,  
RT LL-TONE-N, POWER-5/5,  
LEFT LL-TONE-N, POWER-4/5,  
ALL FOUR LIMBS-NO Sensory Deficit, All Reflexes Mute, B/L Plantar Reflexes-Mute, Left Sided Dysdinokinesia +  
Past History: Known case of prostate cancer, DM, large OSD-ASD with Eisenmenger's syndrome



A MRI spine screening and MRI brain plain was done which showed sclerotic vertebral metastasis in lumbosacral spine and Right cerebellum

## Diagnosis

This is a clear case of carcinoma prostate with vertebral metastasis and right cerebellar metastasis causing a space occupying lesion with hydrocephalus.

## Treatment

Patient admitted to ICU and seen by oncology and neurosurgery team, in view of other comorbidities like DM, large OSD-ASD with Eisenmenger syndrome, planned for symptomatic, palliative treatment including chemotherapy, radiation therapy along with physiotherapy and VP shunting. Patient was feeling better after palliative and physiotherapy.

## References

1. J. McLoughlin, J.C. Gingell, G. Harper' and A. Hinchliffe, Cerebellar manifestations of prostatic carcinoma, Postgrad Med J 1992;68:584- 586

2. Andrew Lawton, B.A.,1 Gary Sudakoff, M.D.,2 Lisa C. Dezelan, PA-C,3 and Nancy Davis, M.D., Presentation, Treatment, and Outcomes of Dural Metastases in Men with Metastatic Castrate-Resistant Prostate Cancer: A Case Series, J Palliat Med. 2010 Sep; 13(9): 1125-1129.
3. McMurtry CT1, McMurtry JM., Metastatic prostate cancer: complications and treatment, J Am Geriatr Soc. 2003 Aug;51(8):1136-42.
4. Clark PE1, Torti FM. Prostate cancer and bone metastases: medical treatment, Clin Orthop Relat Res. 2003 Oct;(415 Suppl):S148-57.
5. Mario A. Eisenberger, Leonard Michael Glode, Case Reports on Prostate Cancer, Rev Urol. 2004;6(Suppl 7): S39-S45.
6. Nagata M1, Ueda T, Komiya A, Suzuki H, Akakura K, Ishihara M, Tobe T, Ichikawa T, Igarashi T, Ito H, Treatment and prognosis of patients with paraplegia or quadriplegia because of metastatic spinal cord compression in prostate cancer, Prostate Cancer Prostatic Dis. 2003;6(2):169-73.
7. Smith EM1, Hampel N, Ruff RL, Bodner DR, Resnick ML, Spinal cord compression secondary to prostate

- carcinoma: treatment and prognosis., J Urol. 1993 Feb;149(2):330-3.
8. Schmidt MH, Klimo P Jr, Vrionis FD., Metastatic spinal cord compression. J Natl Compr Canc Netw. 2005 Sep;3(5):711-9.
  9. Zengbao Wu, Siyi Xu, Chunlong Zhong, Yang Gao, Qiang Liu, Yan Zheng, Yang Guo, Yong Wang, Qizhong Luo, And Jiyao Jiang. Intramedullary conus medullaris metastasis from prostate carcinoma: A case report and review of the literature Oncol Lett. 2014 Mar;7(3):717-720. Published online 2014 Jan 16. doi: 10.3892/ol.2014.1808.
  10. Moul JW, Davis R, Vaccaro JA, Sihelnik SA, Belville WD, McLeod DG., Acute urinary retention associated with prostatic carcinoma, J Urol. 1989 Jun;141(6): 1375-7.
-

## Renal Thrombotic Microangiopathy Due to Malignant Hypertension

Harini Agnes<sup>1</sup>, Venugopal A.V.<sup>2</sup>

---

### Author's Affiliation:

<sup>1</sup>Resident, Department of Emergency Medicine, <sup>2</sup>Consultant, Department of Nephrology, Care Hospitals, Visakhapatnam, Andhra Pradesh 530002, India.

### Corresponding Author:

**Venugopal A.V.**,  
Consultant, Department of Nephrology, Care Hospitals, Visakhapatnam, Andhra Pradesh 530002, India.

E-mail:

[drvenuvkat@yahoo.com](mailto:drvenuvkat@yahoo.com)

Received on 01.09.2017,

Accepted on 14.09.2017

### Abstract

Malignant hypertension (MHTN) is a hypertensive emergency with end organ dysfunction and MHTN presenting as renal thrombotic microangiopathy is rare. It is characterized by microangiopathic hemolysis, anemia, thrombocytopenia, indirect hyperbilirubinemia and variable degrees of renal failure apart from papilledema and acutely elevated blood pressure. The degree of renal failure may vary depending on the extent of endothelial damage and stimulation of renin angiotensin aldosterone system. Herein, we report a rare case of malignant hypertension in a young boy with renal TMA. He has bilateral papilledema and initial blood pressure of 210/100 mm of Hg. He has features of microangiopathic hemolysis and severe oliguric renal failure. His blood pressure was managed in Emergency Department with IV nitroglycerin and IV labetalol continuous infusion. He was instituted on hemodialysis through right Internal Jugular access and was continued on dialysis for the next two weeks. His blood pressure was managed with oral anti-hypertensives (Metoprolol (100mg/day), Nifedipine (60mg/day), Hydralazine (100mg/day), Torsemide 40mg/day). He showed good signs of improvement with adequately controlled blood pressure (140/80) and a stable renal function (Ser. Creat of 2.3 mg/dl, during last followup). Unlike Thrombotic thrombocytopenic purpura/hemolytic uremic syndrome complex, renal TMA associated with malignant hypertension will not respond to plasmapheresis and adequate emergent management of blood pressure in emergency department will limit the extent of renal damage. The level of LDH, platelet count and hemoglobin can be used as markers of microangiopathic hemolysis. Renal recovery can vary from complete to partial recovery.

**Keywords:** Malignant Hypertension; Microangiopathic Hemolysis; Renal Failure.

---

### Introduction

Malignant hypertension is hypertensive emergency resulting in target organ damage with papilledema [1].

Renal Thrombotic Microangiopathy (TMA) occurring as a result of malignant hypertension is known in the literature but very few case reports from India. The renal TMA due to malignant hypertension may closely resemble Thrombotic Thrombocytopenic

Purpura (TTP) but differentiating these two entities is very important because of variable therapeutic implications. Plasmapheresis is beneficial in TTP but of no benefit in TMA associated with malignant hypertension [2]. Renal TMA is characterized by features of intra vascular hemolysis, small vessel thrombosis, thrombocytopenia, indirect hyperbilirubinemia and elevated Lactate Dehydrogenase (LDH) levels. Acute Kidney Injury (AKI) associated with this entity is usually reversible after variable period of renal replacement therapy. So,

it is prudent to wait for prolonged period for complete renal recovery to occur in these patients.

Herein, we report a rare case of malignant hypertension with renal failure (biopsy proven renal TMA) who showed good recovery with effective blood pressure control in Emergency Department and timely initiation of hemodialysis.

### Case Report

A 28 year old patient was admitted to hospital with headache, nausea, blurring of vision and an initial blood pressure of 210/100 mmHg. He is not a known hypertensive or diabetic. Physical examination revealed Grade IV hypertensive retinopathy, there is no abdominal bruit and all his peripheral pulses are well felt. There is significant peripheral edema and bilateral basal crackles. At presentation his serum creatinine levels was 8mg/dl, hemoglobin 6gm/dl and platelet count of 50,000. His LDH was 5,500 and peripheral smear showing schistocytes. His initial MRI brain showed posterior reversible leuco-encephalopathy changes.

Emergency department management of hypertension included IV labetalol (10 mg bolus followed by 4 mg/hr for 12 hours. Target BP (140/80) achieved in 12 hours. AKI was managed with emergency hemodialysis through right internal jugular access.

Over the next one week, his blood pressure was controlled with Metoprolol (100mg/day), Nifedipine (60mg/day), Hydralazine (100mg/day), Torsemide 40mg/day. His direct and indirect comb's tests were negative. Abdominal ultrasound showed normal sized kidneys. His serological tests like HIV, Hepatitis-B, Hepatitis-C and Antinuclear Antibodies and Anti Scl-70 were negative. Urine analysis showed microscopic hematuria and nephrotic proteinuria [4].

Color Doppler renal vessels showed no evidence of renal artery stenosis. 24 hour urinary metanephrin levels were within normal range. His PRA activity was significantly high (>8ng/ml/hr). His renal biopsy showed diffuse arteriolar thrombosis and fibrinoid necrosis of arterioles. He was continued on dialysis for 2 weeks after which he showed good clinical signs of improvement in the form of increased urine output, no signs of fluid overload and improvement in renal function tests. He is being followed up closely in nephrology outpatient department. His last serum creatinine is 2.3mg/dl. All anti-hypertensives he was using till now have

been withdrawn and was started on Telmisartan 40mg/day and achieved adequate blood pressure control.

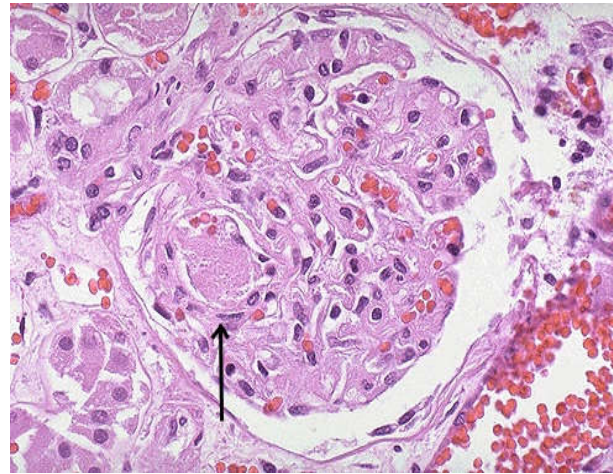


Fig. 1: Histopathology–intra-glomerular capillary thrombi

### Discussion

TMA is a constellation of thrombosis microangiopathic hemolysis and end organ damage. In our patient, renal failure and hypertensive retinopathy were major concerns. So far, only 11 case reports of this combination of malignant hypertension of renal TMA has been reported [3].

The presence of the combination of TMA in malignant hypertension as reported by Akimoto et al was around 44%. Our patient has been followed up for 4 months so far. Repeat kidney biopsy has not been done but there is significant resolution in lab parameters like LDH, creatinine and platelet count. ADAMTS 13 (*a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13*) activity has not been done due to non-availability of the test.

The pathogenesis of TMA due to malignant hypertension could possibly be due to activation of renin angiotensin system as evidenced in our case by elevated Plasma Renin Activity (PRA). Elevated LDH and PRA could represent micro infarcts in kidney. In malignant hypertension PRA highly correlates with LDH and also with elevated serum creatinine. Combined PRA and aldosterone levels were good markers in malignant hypertension. The strong correlation with PRA, Renal dysfunction, aldosterone and micro angiopathic markers suggest renin mediated pathogenesis in malignant hypertension [5].

The ADAMTS 13 activity will be low in either acquired or congenital TTP whereas it is normal in



renal TMA due to malignant hypertension. In recent reports this activity can be used as a guide in plasmapheresis dosing [6].

The recovery of renal function in these cases would be variable and it can vary from complete recovery to total non-recovery progressing to chronic kidney disease.

### Conclusions

Malignant hypertension as a cause of renal failure and renal TMA should always be considered in Emergency department and effective control of Blood pressure in ED will lead to good renal recovery and plasmapheresis is of no use in renal TMA associated with malignant hypertension.

---

### References

1. Rodriguez MA, Kumarsk, De caro M. Hypertensive crisis, *cardiol rev.* 2010 March-April;18(2):102-7.
2. Boctor FN, Prichard JW. Kidney involvement in thrombotic thrombocytopenic purpura and malignant hypertension. *Transfusion.* 2009;49:1783-1784.
3. ChikeNzerue, Kemi Oluwole, Marquetta Faulkner. *Clinical kidney journal*, 2014. Dec;7(6):586-89.
4. Gowda M, Nainani N, Lohr J et al, *Am J kidney Dis.* 2014;63:A23-A25.
5. Bert-Jan H.van den born, Richard p. Koopmans and Gert A. Van montfrans *AJH* 2007;20:900-906.
6. Remuzzi G. *J Thromb haemost.* 2003;1:632-634.

## A Case of Infant with Factor VII Deficiency Presenting as ICH

Hilal Ahmad Yatoo<sup>1</sup>, Vaibhav Gulati<sup>2</sup>, Kishalay Datta<sup>3</sup>, Rupinder Kahlon<sup>4</sup>

### Author's Affiliation:

<sup>1</sup>Attending Consultant <sup>2</sup>PGY-3, MEM {GWU-USA} <sup>3</sup>HOD and Associate Director <sup>4</sup>Attending Consultant, Dept of Emergency Medicine, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

### Corresponding Author:

**Hilal Ahmad Yatoo**, Attending Consultant, Dept of Emergency Medicine, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

E-mail: [calldrhilal@gmail.com](mailto:calldrhilal@gmail.com)

Received on 19.01.2017,

Accepted on 13.10.2017

### Abstract

ICH can be spontaneous or traumatic. the most common cause of ICH in adults is trauma (road traffic accident or fall from height) and CVA. In neonates and infants ICH is caused by trauma associated with labor and delivery. Factor VII deficiency presenting as ICH on an infant is a rare entity. Here we present a case of 40 day old male child presenting as seizure which was later diagnosed to have ICH due to severe factor VII deficiency.

**Keywords:** Intracranial Haemorrhage; Factor VII Deficiency; Seizure; Prothrombin Time; Haemorrhage.

### Introduction

The most common cause of ICH in adults is trauma and CVA.

Whereas the causes of ICH in neonates and infants include:

- Trauma associated with labor and vaginal delivery
- Acidaemia
- Hypoxia
- Hypercarbia
- Immaturity of the coagulation system, hereditary disorders/syndromes.

The majority of neonates with intracranial haemorrhage have no clinical symptoms, including some with moderate to severe haemorrhages. Term newborns with intracranial haemorrhage may manifest with a neonatal seizure, decreased level of consciousness, or both.

Bleeding/clotting disorders are among the rare causes of ICH. Among these, Factor VII deficiency is the most common among rare inherited Autosomal recessive bleeding disorders. In spite being the most common, prevalence is estimated to be 1 case per 500,000 persons in the general population.

Factor VIIa can be detected in plasma by a sensitive assay using a recombinant soluble form of tissue factor. The mean plasma concentration is 3.6 ng/mL in healthy individuals. The half-life of factor VIIa is relatively long (2.5 h) compared with other activated coagulation factors.

Factor VII deficiency is an autosomal recessive disease, unlike haemophilia (X-linked recessive). Only homozygote or compound heterozygote patients with factor VII deficiency are symptomatic. Heterozygote who have partial factor VII deficiency may not exhibit hemorrhagic manifestations, even following trauma. In symptomatic patients, clinical phenotypes vary from mild to severe and do not necessarily correlate with factor VII levels. A multicenter European study of patients who are congenitally factor VII deficient showed that clinical symptoms did not vary with the frequency of functional polymorphisms and that homozygote with the same mutation presented with striking differences in severity of bleeding.

The most frequently reported bleeding symptoms among "platelet-like" FVII deficiency are-

- Epistaxis (60%),
- Gum bleeding (34%),
- Easy bruising (36%),
- Menorrhagia (69% of females).



Bleeding Risk	Factor VII (%)	Personal History	Family History
High risk	<2	CNS bleed, umbilical stump bleed, hemarthrosis, GI bleed	Life-threatening bleeding, death for hemorrhage in first degree relatives
Low risk	>20	Negative for spontaneous bleed	Negative for spontaneous bleeding

Among the severe forms-

- Recurrent hemarthrosis (19%)
- Gastrointestinal bleeding (15%)
- Central nervous system bleeding (2.5%)

### Case Report

Forty days male child presented to ER with complains of (Historian-mother) abnormal movement of the body from 1 day. There was no history of trauma/fall/fever/cold/cough/loss of consciousness. The patient was admitted in another hospital for 1 day where NCCT head was done which was suggestive of large hyperdense hemorrhage in right fronto-accipital region with perifocal edema and mass effect on right lateral ventricular and midline shift to left side. The patient was managed conservatively.

On arrival, the child was conscious, playful, and all vitals were within normal range according to age. The systemic examination was unremarkable except increased tone and brisk deep tendon reflexes. There was history of prolonged umbilical bleed after birth. Patient has a positive family history of death of elder brother at 6 months of age with history of patchal spots all over the body.

MRI brain with contrast was done which was suggestive of Intraventricular hemorrhage in left lateral ventricle and fourth ventricle, Supra and infratentorial subdural and subarachnoid hemorrhages. Neurosurgery consult was taken and patient was admitted in PICU after starting antiepileptics, measures to decrease ICP and Inj Vit K. Routine investigations were sent which included complete hemogram, liver function test, renal profile, coagulation profile. Investigations revealed Hemoglobin of 9.8gm/dL, Prothrombin time >1min. peripheral smear for type of anaemia was suggestive of normocytic normochromic anaemia.

The initial investigation was suggestive of anaemia and prolonged PT. Accordingly, factor VII assay was sent and plan to replace factor VII was made. Lab values showed factor VII to be <1%, Hematology consult was taken and so accordingly factor VII was transfused. The patient was discharged 21 days after

admission in a stable condition with no new bleed. A follow-up CT of the brain at 1 month showed a resolving ICH.

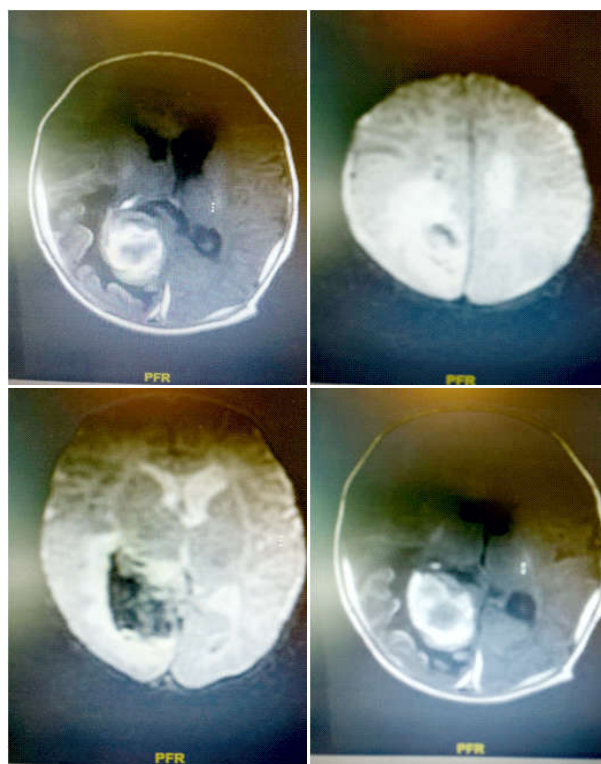


Fig. 1:

### References

1. Mariani G, Colce A. Congenital factor VII deficiency. In: Lee CA, Berntorp EE, Hoots WK, editors. Textbook of Hemophilia. 2nd ed. Oxford: Wiley-Blackwell; 2010.pp.341-347.
2. Perry DJ. Factor VII Deficiency. Br J Haematol. 2002;118:689-700. [PubMed].
3. Lapecorella M, Mariani G International Registry on Congenital Factor VII Deficiency. Factor VII deficiency: defining the clinical picture and optimizing therapeutic options. Haemophilia. 2008;14:1170-1175. [PubMed].
4. Mariani G, Konkle BA, Ingerslev J. Congenital factor VII deficiency: therapy with recombinant activated factor VII - a critical appraisal. Haemophilia. 2006;12:19-27. [PubMed].

5. Mariani G, Dolce A, Marchetti G, Bernardi F. Clinical picture and management of congenital factor VII deficiency. *Haemophilia*. 2004;10(Suppl 4):180-183. [PubMed].
  6. Mannucci PM, Duga S, Peyvandi F. Recessively inherited coagulation disorders. *Blood*. 2004;104:1243-1252. [PubMed].
  7. Korea hemophilia foundation. 2009 Annual report. Seoul: KHF; 2010.pp.30-34.
  8. Hoffman M, Monroe DM., 3rd A cell-based model of hemostasis. *Thromb Haemost*. 2001;85:958-965. [PubMed].
  9. Van den Berg HM, Fischer K. Phenotypic-genotypic relationship. In: Lee CA, Berntorp EE, Hoots WK, editors. *Textbook of hemophilia*. 2nd ed. Oxford: Wiley-Blackwell; 2010.pp.33-37.
  10. Mariani G, Herrmann FH, Dolce A, Batorova A, Etro D, Peyvandi F, et al. International Factor VII Deficiency Study Group. Clinical phenotypes and factor VII genotype in congenital factor VII deficiency. *Thromb Haemost*. 2005;93:481-487.
-

## An Unusual Presentation of Recurrent Hypoglycemia

Singh A.<sup>1</sup>, Datta K.<sup>2</sup>, Das I.<sup>3</sup>, Kalita R.<sup>3</sup>, Govil P.<sup>3</sup>, Patel M.<sup>4</sup>

---

### Author's Affiliation:

<sup>1</sup>MEM, PGY-3, <sup>2</sup>Associate Director and HOD <sup>3</sup>Attending Consultant <sup>4</sup>DNB, PGY-3, Department of Emergency Medicine, Max Hospital, Shalimarbagh, New Delhi, Delhi 110088, India.

### Corresponding Author:

Aakansha Singh, MEM, PGY-3, Department of Emergency Medicine, Max Hospital, Shalimarbagh, New Delhi, Delhi 110088, India.

E-mail:

[dr.aakansha23@gmail.com](mailto:dr.aakansha23@gmail.com)

Received on 24.05.2017,

Accepted on 13.06.2017

### Abstract

Hypoglycemia is defined as random blood sugar equal to or lower than 60mg/dl. The most common cause is medications such as sulfonylurea, biguanides and insulin. Other causes include liver disease, certain tumors, kidney disease, severe infections and starvation. It can be a very common presentation in elderly patients with altered mental status who are on polypharmacy. Investigating recurrent hypoglycemia can be a challenge. Whilst the obvious focus is to rule out an underlying endocrine etiology, a thorough history and recognition of factitious cause is important and worth bearing in mind. This can be difficult to diagnose and often, can only be ruled out by extensive investigations and exclusion of other causes. Patients with clinical hypoglycemia unawareness are at high risk of severe hypoglycemia that requires third-party assistance. Hypoglycemia is less frequent in type 2 diabetes than it is in type 1. Population-based data indicate that the overall event rate for severe hypoglycemia (requiring the assistance of another individual) in insulin-treated type 2 diabetes is approximately 30 percent of that in type 1 diabetes (35 versus 115 episodes per 100 patient-years). In this case a young non diabetic female presented to ED in a state of altered mental status with recurrent hypoglycemia, the cause for which was thought to be sepsis and ultimately diagnosed as fulminant hepatic failure. In fulminant hepatic failure there is altered mental status with coagulopathy in setting of acute liver disease. Neurotoxins like ammonia and glutamine with cytokines produce cytogenic and vasogenic effects which leads to cerebral oedema and thus altered sensorium. Patient presents in a state of hepato cellular dysfunction, encephalopathy and cerebral oedema, infections or multi organ failure. The case emphasizes the importance of appropriate history taking and correct differential diagnosis establishment in order to achieve good outcome of a patient with fulminant hepatic failure.

**Keywords:** Hypoglycemia; Diabetes; Hepatic Failure; Altered Sensorium.

---

### Introduction

Hypoglycemia is defined as random blood sugar equal to or lower than 60mg/dl. The most common cause is medications such as sulfonylurea, biguanides and insulin. Other causes include liver disease, certain tumors, kidney disease, severe infections and

starvation. It can be a very common presentation in elderly patients with altered mental status who are on polypharmacy. Investigating recurrent hypoglycemia can be a challenge. Whilst the obvious focus is to rule out an underlying endocrine etiology, a thorough history and recognition of factitious cause is important and worth bearing in mind. This can be difficult to diagnose and often, can only be ruled out

by extensive investigations and exclusion of other causes. In a setting of endogenous insulin deficiency (type 1 and advanced type 2 diabetes), one episode of hypoglycaemia reduces both counterregulatory hormone responses to and subjective awareness of subsequent hypoglycaemia, thus impairing physiological defences against hypoglycaemia. This phenomenon may lead to a vicious cycle of recurrent hypoglycaemia and glucose counterregulatory failure, of which hypoglycaemia unawareness (i.e. the inability to perceive symptoms of hypoglycaemia) is the clinical representative.

The underlying mechanism of hypoglycaemia-induced counterregulatory failure has not yet been disclosed. Patients with clinical hypoglycaemia unawareness are at high risk of severe hypoglycaemia that requires third-party assistance. Hypoglycemia is less frequent in type 2 diabetes than it is in type 1. Population-based data indicate that the overall event rate for severe hypoglycemia (requiring the assistance of another individual) in insulin-treated type 2 diabetes is approximately 30 percent of that in type 1 diabetes (35 versus 115 episodes per 100 patient-years) and that event rates for hypoglycemia requiring professional emergency medical treatment range from 40 to 100 percent of those in type 1 diabetes.

In this case a young non diabetic female presented to ED in a state of altered mental status with recurrent hypoglycemia, the cause for which was thought to be sepsis caused by fulminant hepatic failure. In fulminant hepatic failure there is altered mental status with coagulopathy in setting of acute liver disease. Hepatic encephalopathy occurring within 8 weeks of onset of illness defines fulminant hepatic failure. The common cause is either viral hepatitis or toxin mediated. Neurotoxins like ammonia and glutamine with cytokines produce cytogenic and vasogenic effects which leads to cerebral oedema and thus altered sensorium.

Patient presents in a state of hepato cellular dysfunction, encephalopathy and cerebral oedema, infections or multi organ failure. Altered mental status with coagulopathy in setting of acute liver disease. Hepatic encephalopathy occurring within 8 weeks of onset of illness defines fhf.

Laboratory studies show higher levels of transaminase (>1000), with mixed hyperbilirubinemia, elevated ammonia with prolonged pt, aptt, metabolic acidosis and increased lactates. Many therapies for management like insulin and glucagon to stimulate regeneration, prostaglandin-E, corticosteroids, hemofiltration, charcoal hemo-

perfusion, plasma exchange have been tried but the best results are achieved by liver transplantation.

The case emphasises the importance of appropriate history taking and correct differential diagnosis establishment in order to achieve good outcome of a patient with fulminant hepatic failure.

### Case History

36 year old female presented to ED with c/o decreased responsiveness since 1 day associated with 2-3 episodes of vomiting since morning following which she became drowsy.

The airway was maintainable by using a nasopharyngeal device, breathing labored with a respiratory rate of 32/m, saturating at 100% on room air. She had a heart rate of 77 beats per minute and blood pressure of 110/70mmhg.

Her Glasgow coma scale reading was E4V1M5, pupil bilaterally reactive, RBS of 44 mg/dl.

50% of dextrose given i.v bolus.

POC done include ECG and ABG.

On further history taking she was known to be a case of psychosis, was taking medications 4 months ago along with some pain killers.

Icterus was noted on HEENT examination, chest was bilateral clear, CVS-S1, 2 heard with no murmur, abdomen was soft, tenderness was noted over right hypochondrium with hepatomegaly, bowel sounds heard, CNS examination revealed decreased left side body movement and her plantars were bilateral extensors.

Her LMP-9/12/14 (5d/28d), last delivery-9yrs ago, Copper t - in situ.

On repeat vitals, her heart rate, blood pressure, saturation, respiratory rate were all similar except her blood sugar level which was noticed to be 450mg/dl after 50% dextrose.

Her ECG showed normal sinus rhythm.

ABG shows :- ph- 7.4, pco<sub>2</sub>- 24.6, po<sub>2</sub>- 112 on 4l of o<sub>2</sub>, Na- 117 meq/l, k- 5.5 meq/l, hco<sub>3</sub>- 15.3, LAC- 5.9.

In view of above investigations differentials of CVA, Sepsis due to hepatic cause, Isulinoma and drug overdose were made for which ct brain plain and ct abdomen along with complete blood count, renal and liver profile, viral markers were sent.

The ct brain and abdomen revealed a normal study.

Before shifting the patient to ICU her vitals were

rechecked this again revealed similar parameters except rbs of 95mg/dl.

Patient was started on i.v. antibiotics and maintainence fluids.

In the ICU she was managed symptomatically.

Her CBC revealed hb-10.7m/dl, platelets-421 ,rbc- 3.13, mcv- 101.7

Mch- 34.3, TLC-39.6\*10<sup>9</sup>/l, neutropils-79%, eosinophils- 1%,lympocytes- 6% .

Liver function test were bilirubin- total-7.6mg/dl (direct- 3mg/dl, indirect- 4mg/dl), total proteins- 5.5 mg/dl, albumin-2.7 mg/dl, globulin-2.8 mg/dl, sgot- 452 iu/l, sgpt- 1025 iu/l, alk phosphate- 230 iu/l.

Renal function test na- 124.8 mmol/l, k- 4.4 mmol/l, cl- 101.3 mmol/l

S.urea- 12mg/dl, s.creat- 36mg/dl.

Urine routine- normal, urine for tox- not sinificant, s. markers- hbsag- negative, hiv- negative, hcv- negative, Hep E- positive, S.Ammonia – 183microgm, APTT- 86.7.

*Final Diagnosis:*

Severe sepsis

Hepatitis E

Hepatic encephalopathy

Fulminant hepatic failure

## Discussion

In ED if a patient presents with recurrent hypoglycemia apart from ruling out the other causes of altered mental status e should also think about the underlying liver pathology and detailed liver profile should be sent.

## References

1. Hughes S. Pain Med Linked to Hypoglycemia. Medscape Medical News. Dec 11 2014.
2. Fournier JP, Azoulay L, Yin H, et al. Tramadol Use and the Risk of Hospitalization for Hypoglycemia in Patients With Noncancer Pain. JAMA Intern Med. 2014 Dec 8.
3. Pugh SK, Doherty DA, Magann EF, et al. Does hypoglycemia following a glucose challenge test identify a high risk pregnancy?. Reprod Health. 2009 Jul 14;6:10.
4. Hill NR, Thompson B, Bruce J, et al. Glycaemic risk assessment in children and young people with Type 1 diabetes mellitus. Diabet Med. 2009 Jul;26(7): 740-3.
5. Turnbull FM, Abraira C, Anderson RJ, et al. Intensive glucose control and macrovascular outcomes in type 2 diabetes. Diabetologia. 2009 Aug 5.
6. Prolonged Nocturnal Hypoglycemia Is Common During 12 Months Of Continuous Glucose Monitoring In Children And Adults With Type 1 Diabetes. Diabetes Care. 2010 Mar 3.
7. Swinnen SG, Dain MP, Aronson R, et al. A 24-week, randomized, treat-to-target trial comparing initiation of insulin glargine once-daily with insulin detemir twice-daily in patients with type 2 diabetes inadequately controlled on oral glucose-lowering drugs. Diabetes Care. 2010 Mar 3.
8. Ito T, Otsuki M, Igarashi H, et al. Epidemiological Study of Pancreatic Diabetes in Japan in 2005: A Nationwide Study. Pancreas. 2010 Feb 22.
9. Chen L. A literature review of intensive insulin therapy and mortality in critically ill patients. Clin Nurse Spec. 2010 Mar-Apr;24(2):80-6.
10. Garza H. Minimizing the risk of hypoglycemia in older adults: a focus on long-term care. Consult Pharm. 2009 Jun;24(Suppl B):18-24.

# Cerebral Venous Thrombosis and Hyperhomocysteinemia, How Important is the Co-Relation?:

## A Review of 3 Cases

Lipoktemsus Jamir<sup>1</sup>, Dina J. Shah<sup>2</sup>

### Author's Affiliation:

<sup>1</sup>Attending Consultant, Department of Emergency and Trauma care, Medanta The Medicity, Gurugram, Haryana 122018, India. <sup>2</sup>Director Emergency Medical Services, Delhi-NCR.

### Corresponding Author:

**Dina J. Shah**, Director Emergency Medical Services, Fortis Hospital, Noida, Uttar Pradesh 201301, India.  
E-mail: [dina.shah@fortishealthcare.com](mailto:dina.shah@fortishealthcare.com)

Received on 15.10.2017,

Accepted on 23.10.2017

### Abstract

Thrombosis of the cranial venous sinuses and the cerebral cortical veins can lead to a distinct cerebrovascular disorder, which unlike arterial stroke, most often affects even young adults and children. Symptoms and clinical courses are highly variable, etiological factors are even more heterogeneous making cerebral cortical vein thrombosis (CVT) a unique clinical entity. We report three cases in which the initial presentation in our Emergency Department (ED) led to suspicion of CVT, had it diagnosed and recognised hyperhomocysteinemia.

**Keywords:** Cerebral Venous Thrombosis; Hyperhomocysteinemia; Headache.

### Introduction

Thrombosis of the cranial venous sinuses and the cerebral cortical veins can lead to a distinct cerebrovascular disorder, which unlike arterial stroke, most often affects even young adults and children. Symptoms and clinical courses are highly variable, etiological factors are even more heterogeneous making cerebral cortical vein thrombosis (CVT) a unique clinical entity. Common presenting symptoms in the International Study on Cerebral Venous and Dural Sinuses Thrombosis (ISCVT) were headache (89%); seizures (39.3%); unilateral or bilateral weakness (37.2%); papilledema (28.3%); and mental status changes (22%)[6]. Unusual presentations that can present with CVT include acute subdural haematoma[16], cerebellar ataxia and cortical blindness[13], subarachnoid haemorrhage[12], Paroxysmal Nocturnal Hemoglobinuria (PNH)[19] and Homocystinuria [14] among others.

Because of the heterogeneity in the clinical presentation and etiology, the diagnosis of CVT is often missed, and even if a diagnosis is made the contributory factors which are often subclinical are also missed or overlooked [15]. It can present to various specialists apart from emergency physicians such as general physicians, obstetricians and neurologists.

Diagnosis is often missed unless clinicians maintain a high index of suspicion and be aware of the varied clinical presentations to be able to recognize and manage by prompt and proper application of clinical skill, rather than depending heavily on investigations alone for effective management of these patients.

After making a diagnosis of CVT the clinician should apply clinical skill and common sense with which it is possible to arrive at one or more completely correctable common etiological factors contributing to the development of CVT, even if there is an underlying inherited disorder which cannot be corrected, and thus can avoid recurrences in future. Once the diagnosis of CVT is made it is easily managed if we know all the contributory factors and almost always has a good prognosis compared to other cerebrovascular accidents [14]. It is also an observation that in many patients with the so called idiopathic CVT, nutritional deficiencies and life style issues are more important basic etiological factors in pathogenesis, at least in some epidemiological settings as strict vegetarians and those who consume an unbalanced diet. Research by observation and studying the patients for their diet, lifestyle and environment might give the answer to the several etiological factors in cerebral cortical vein thrombosis, as in all other clinical problems, rather than depending on the costly laboratory investigations alone[1].

The main progress in CVT study has been focused on identification of thrombophilic factors. Epidemiological studies have suggested that even mild Hyperhomocysteinemia (hyper-Hcy) is associated with occlusive arterial vascular disease and venous thromboembolism. Little information about the role of homocysteine in CVT is available. A systematic study on CVT and hyper-Hcy has been published in which Martinelli et al found that hyper-Hcy increases the risk of CVT by approximately 4-fold [5]. Vitamin supplementation with folic acid, pyridoxine, and cobalamin lowers the plasma levels of total homocysteine (tHcy) in most cases. Therefore, if hyperhomocysteinemia is associated with cerebral vein thrombosis, vitamin therapy has the potential to decrease the risk of recurrence.

We report three cases in which the initial presentation in our Emergency Department (ED) led to suspicion of CVT, had it diagnosed and recognized hyperhomocysteinemia.

## Case Reports

### Patient 1

A 24-year-old male came to the Emergency Department (ED) with complaint of headache and vomiting (projectile in nature) for the past 2 days. He had no co-morbidities. His neurological examination was normal. NCCT head was done because of the unexplained headache, which was suggestive of CVT (left transverse, left sigmoid and straight sinuses with dense clot sign as seen in Fig.1). He was assessed by neurology and admitted in Intensive Care Unit (ICU). MRI venography was advised, which confirmed the diagnosis. He was treated with injection Heparin with 6 hourly APTT monitoring. His thrombocheck panel was normal except for raised S Hcy level (>50 micro mol/l). His vitamin B12 and D3 levels were also in the lower range (130.7pg/ml and 4.4 ng/ml respectively). His condition improved and on discharge he was put on tablet acenocoumarol 1 mg/day prophylactically along with oral Vitamin B12 and vitamin D3 and to consume a diet low in vitamin K. He was advised for follow up and explained about the morbidity and mortality of the condition

### Patient 2

The second patient, a 38-year-old male patient presented to our ER with complaint of headache followed by diplopia and blurred vision for the past 3 days. He had history of fever a week back which was

of moderate grade and intermittent in nature. It had subsided on self-medication. NCCT head was done which was suggestive of CVT in the right transverse, right sigmoid and superior sagittal sinuses (Fig. 2). He then underwent MRI venography, which showed hypointense-filling defects superior sagittal, bilateral transverse and sigmoid sinuses which confirmed the diagnosis. He was also admitted in Intensive Care Unit (ICU) under neurology team and put on intravenous heparin with regular APTT monitoring. His lipid profile, Complete Blood Count (CBC), Lupus Anticoagulant, Antiphospholipid Antibody (APLA) was normal except for elevated Serum Homocysteine (28.84 micro mol/l). He was discharged after a week without any complication with improved vision. He was also started on tablet acenocoumarol 3 mg/day, vitamins and advised for regular follow up.



Fig. 1: Left transverse sinus CVT with dense clot sign



Fig. 2: Image showing infarction in the area of the vein of Labbe

### Patient 3

The third patient, a 21-year-old male patient, had chief complaint of headache, neck pain with recurrent vomiting for the past 4 days. It was associated with bilateral lower limb weakness. NCCT head was done which was suggestive of CVT in the sagittal sinus. He then underwent MRI venography, which showed hypointense-filling defect in the transverse, right sigmoid and posterior part of superior sagittal sinuses. In the Intensive Care Unit (ICU) he put on intravenous heparin with regular APTT monitoring. His serum homocysteine level was high (28.18 micro mol/l) and his vitamin B12 and D3 were on the lower side. He was discharged with the same advice as the above 2 patients with tablet acenocoumarol in a dose of 4 mg/day.

### Discussion

Headache is one of the most frequent presentations in our emergency department. Etiology of headache varies and generally it is due to meningitis, cerebral tumors, hydrocephalus, intoxications, overwork-stress related, stroke or just migraine. The varied presentations in our patients led to the inclusion of CVT in our differentials. The symptom and clinical course of CVT are highly variable and can range from isolated headache and visual or auditory problems, to serious symptoms such as hemiparesis and coma. Its incidence is reported as 0.5 of 100,000 annually, more frequently diagnosed in women, accounting for 0.5% to 1% of all strokes [10]. Young age group with varied causes ranging from taking hormones to recreational drugs and chronic alcohol abuse are at risk for CVT. As this is a potentially life-threatening condition with high mortality rate in untreated patients, early diagnosis and treatment are important. None of our patients had any risk factors ranging from APLA, Lupus Anticoagulant except for raised Serum Homocysteine. All the three patients in our study were discharged with similar diagnosis of CVT with hyperhomocysteinemia with hypovitaminosis B12 and D3. All of them were non-smoker, non-alcoholic and had no co-morbidities.

Hyperhomocysteinemia can lead to vascular events like acute coronary syndromes, recurrent coronary events, stroke and venous thrombosis. It can be familial or acquired due to vitamin deficiencies. Homocysteine has primary atherogenic and prothrombotic properties. Histopathologic hallmarks of homocysteine-induced vascular injury include intimal thickening, elastic lamina disruption, smooth

muscle hypertrophy, marked platelet accumulation, and the formation of platelet-enriched occlusive thrombi [15]. Vitamin B12, folate and pyridoxine deficiency contributes to development of hyperhomocysteinemia.

To date, thrombophilia screening, including coagulation factor abnormalities such as factor V Leiden, prothrombin mutation, deficiencies of antithrombin, protein C, and protein S, and the presence of antiphospholipid antibodies, is recommended in the diagnostic work up in patients with cerebral vein thrombosis. The cases that we have taken up further support the evidence that measurements of plasma tHcy are an important entity in thrombophilia screening. At variance with other types of thrombophilia, hyperhomocysteinemia can be easily and safely treated with vitamin supplementation as stated above. Hyper-Hcy has proved to be a strong and independent factor associated with ischemic stroke. The probable causal link is also observed in young patients and children, suggesting a thrombogenic rather than an atherogenic effect in these young subjects. The findings of Carlos Cantu et al were consistent with the hypothesis that high blood concentrations of tHcy are associated with increased risk of CVT [7]. Furthermore, low plasma folate levels were also associated highly with an increased risk for CVT in this population in which low socioeconomic conditions and deficient nutritional status may contribute to its relatively high incidence.

Spence et al [9] found that in the era of folate fortification, B12 plays a key role in vitamin therapy for total Hcy. Higher doses of B12, and other treatments to lower total Hcy may be needed for some patients. Thus in the western world, effective vitamin intervention has shifted from folate to vitamin B12 in post fortification era unlike what was seen in 2002 where intervention with folate reduced the incidence of stroke, cardiovascular disease and venous thrombosis effectively. That B12 and folate deficiency can lead to hyper-homocysteinemia and venous thrombosis has been well documented [2,4,8] and its role cannot be ignored.

### Conclusion

CVT should be considered in any young patient who presents with an unexplained headache. Patients should be started on treatment as soon as the diagnosis is made to improve the outcome and thereby decrease morbidity and mortality. Stress is made once again



on the importance of measurements of plasma tHcy and its role in development of CVT. Its role in CVT diagnosis and prognosis cannot be overlooked.

## References

1. M. G. Bousser, J. Chiras, J. Bories, and P. Castaigne. Cerebral venous thrombosis—a review of 38 cases. *Stroke*, 1985;16(2):199–213.
2. J.B. Ubbink, W.J. Vermaak, M.A. Vander, et al. Vitamin B12, Vitamin B6 and folate nutritional status in men with hyperhomocysteinemia. *Am. J. Clin. Nutr.* 1993;57:47–53.
3. M. Cataneo. Hyperhomocysteinemia: a risk factor for arterial and venous thrombosis. *Int J Clin Lab Res* 1997;27:1139–1144.
4. M.A. Mansoor, O. Kristen Sen, T. Hervig, et al. Total plasma homocysteine response to oral doses of folic acid and pyridoxine hydrochloride (vitamin B6) in healthy individuals: oral doses of vitamin B6 reduce concentrations of serum folate. *Scand. J. Clin. Lab. Invest.* 1999;59:139–146.
5. Martinelli I, Battaglioli T, Pedotti P. Hyperhomocysteinemia in cerebral vein thrombosis. *Blood* 2003;102:1363–6.
6. Ferro JM, Canhao P, Stam J, et al, ISCVT Investigators. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke* 2004;35(3):664–70.
7. Carlos Cantu, Elisa Alonso, Aurelio Jara Leticia Martı́nez, Camilo Rı́os, Marı́a de los Angeles FernándeZ, Irma Garcia, Fernando Barinagarre menteria. Hyperhomocysteinemia, Low Folate and Vitamin B12 Concentrations, and Methylene Tetrahydrofolate Reductase Mutation in Cerebral Venous Thrombosis. *Stroke*, 2004;35:1790–1794.
8. C. Fernandez-Miranda, M. Yebra Yebra, C. Ribera Casado, T. Toledo Urgarte, M. Martin Mola, P. Gomez Gonzalez. Venous thromboembolism and hyperhomocysteinemia as first manifestation of pernicious anemia. *Rev. Clin. Esp.* 2005 Oct;205 (10):489–492.
9. J.D. Spence, H. Bang, L.E. Chambless, M.J. Stampfer. Vitamin Intervention For Stroke Prevention trial: an efficacy analysis. *Stroke* 2005 Nov;36(11):2404–2409.
10. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol* 2007;6:162–70.
11. P. K. Sasidharan and A. Mohammed. Cortical vein thrombosis due to acquired hyperhomocysteinemia. *The National Medical Journal of India*, 2009;22(6).
12. Yves Benabu, Levental Mark, Suissa Daniel, Rafael Glikstein. Cerebral venous thrombosis presenting with subarachnoid hemorrhage Case report and review. *American Journal of Emergency Medicine* 2009;27:96–106.
13. Samia Ben Sassi, Habiba Mizouni, Fatma Nabli, Lamia Kallel, Mounir Kefi, and Fayc ‘al Hentati. Cerebral Venous Thrombosis Presenting With Cerebellar Ataxia and Cortical Blindness. *Journal of Stroke and Cerebrovascular Diseases*, 2010 Nov-Dec;19(6): 507–509.
14. Parveen Bhardwaj, Ravi Sharma, and Mino Sharma. Homocystinuria: A rare condition presenting as stroke and megaloblastic anemia. *J Pediatr Neurosci.* 2010 Jul-Dec;5(2):129–131.
15. P. K. Sasidharan. Cerebral Vein Thrombosis misdiagnosed and mismanaged. *Thrombosis*, 2012; 1–11.
16. Satoshi Takahashi, Jun Shinoda and Takuro Hayashi. Cerebral Venous Sinus Thrombosis in an Adult Patient Presenting as Headache and Acute Subdural Hematoma. *Journal of Stroke and Cerebrovascular Diseases*, 2012 May;21(4):338–340.
17. M. Sayadnasiri, A.A. Taheraghdam, M. Talebi. Cerebral venous thrombosis presenting as subarachnoid hemorrhage: Report of two cases. *Clinical Neurology and Neurosurgery* 2012;114:1099–1101.
18. Piazza G. Cerebral venous thrombosis. *Circulation* 2012;125:1704–9.
19. H. Sumbul, O. Taktakoglu, M. Buyuksimsek, B. Guven. Paroxysmal Nocturnal haemoglobinuria presenting as cerebral venous sinus thrombosis. *Leukemia Research* 2014;38(S1): S1–S65.
20. Sofia E. Thorell, Adrian R. Parry-Jones, Martin Punter, Robert Hurford, Jecko Thachil. Cerebral venous thrombosis – A primer for the haematologist. *Blood Reviews*. 2015;29:45–50.

# Asymmetrical and Late Onset of Pulmonary Edema Post Scorpion Sting: Case Report of Rare Manifestation

Susmeet Mishra<sup>1</sup>, Gouri Kumar Rath<sup>2</sup>, Sajid Nomani<sup>3</sup>

## Author's Affiliation:

<sup>1</sup>Registrar <sup>2</sup>PGT <sup>3</sup>Consultant, Dept  
of Emergency Medicine, AMRI  
Hospitals, Khandagiri, Odisha  
751030, India.

## Corresponding Author:

**Sajid Nomani**, Consultant,  
Dept. of Emergency Medicine,  
AMRI Hospitals, Khandagiri,  
Odisha 751030, India.  
E-mail: [dr.sajidn@gmail.com](mailto:dr.sajidn@gmail.com)

Received on 24.05.2017,

Accepted on 13.06.2017

## Abstract

Scorpion bites are common in India and an important public health hazard in tropical and subtropical regions of India. Though generally bites are harmless, sometimes they can lead to serious sequelae including death. Herein we present a rare case of scorpion sting presenting as myocardial infarction manifesting in the form of asymmetric pulmonary edema after 24 hours of sting along with congestive cardiac failure, successfully treated with non invasive ventilation and inotropes. The etiology of the cardiovascular manifestations in scorpion sting is related to the venom effects on the sympathetic nervous system and the adrenal secretion of the catecholamines as well as to the toxic effects of the venom on the myocardium.

**Keywords:** Scorpion Sting; Pulmonary Edema; Congestive Cardiac Failure.

## Introduction

Out of the 1000 scorpion species known worldwide only few are toxic to humans. Among the 86 species of scorpion present in India, *Mesobuthus tumulus* (Indian red scorpion) and *Palmanus gravimanus* (black scorpion) are of medical importance [1]. Though local symptoms including severe pain and burning sensation at the site of sting are the most common manifestations, systemic complications can ensue [2]. Cardiovascular manifestations are particularly prominent following stings by Indian red scorpion [3]. Such bites infrequently have serious clinical sequelae including myocardial infarction, acute pulmonary oedema and even death. We present here in a case report with the clinical manifestations following scorpion bite mimicking acute myocardial infarction.

## Case Presentation

A 40 year old lady presented to the Emergency room with complaints of shortness of breath associated with profuse sweating since 1 hour prior to arrival. Her attendants gave alleged history of her being bitten

by a scorpion in her right leg 2 days ago following which she had pain and swelling around the site of sting. She was taken to a local hospital for treatment where she was given intravenous fluids, hydrocortisone, and tablet prazosin but after two days she developed breathing difficulty, head reeling and sweating for which she was referred to this hospital for further management.

Her past history was not significant and she had no predisposing cardiac risk factors. Her initial blood pressure was 70/40 mm hg, heart rate 117 bpm, regular, oxygen saturation by probe 56% and respiratory rate 41 cpm. On auscultation of chest bilateral diffuse inspiratory basal crepitations were found, more on the left side than right side. Jugular venous pressure of the patient was raised.

An immediate Arterial blood gas revealed severe hypoxia and increased lactate (Fig.1).

On further investigations serum cardiac enzymes and total leukocyte count were grossly raised and the level of CPK MB was 25 U/l and that of Troponin T was 0.36ng/ml. Chest X-ray revealed features suggestive of asymmetric pulmonary edema (Fig. 2).

Electrocardiograph revealed sinus tachycardia with secondary ST-T changes (Fig. 3). Echocardiogram

demonstrated dilatation of all 4 chambers with hypokinesia of interventricular septum and inferior posterior wall, moderate MR and TR with severe Left ventricular dysfunction (LVEF 23%). She was initially started with oxygen through a high flow oxygen mask but due to persisting low saturation level patient was put on non-invasive ventilation with high PEEP. Intravenous fluids could not be given as patient was assumed to be in fluid overload status. Inotropes noradrenaline and dobutamine were started along with diuretics infusion at a slower rate.

Total fluid intake of the patient was restricted. Her admission course was smooth and she was weaned of non invasive ventilation on the second day of admission and weaned of inotropes on the 3rd day of admission. She was shifted to ward on the 4th day and subsequent xray showed resolution of pulmonary edema and echocardiogram showed improved left ventricular ejection fraction. She was discharged on the 5th day of admission and is due for follow up one month later.

Results: Gases+			
pH	7.407		
pCO2	34.7	mmHg	Low
pO2	36.8	mmHg	Low
cHCO3-	21.9	mmol/L	
BE(ecf)	-2.8	mmol/L	Low
cSO2	71.3	%	Low

*an 10L O2 High*

Results: Chem+			
Na+	144	mmol/L	
K+	3.7	mmol/L	
Ca++	1.11	mmol/L	Low
cTCO2	22.9	mmol/L	
Hct	29	%	Low
cHgb	9.7	g/dL	Low
BE(b)	-2.4	mmol/L	Low

Results: Meta+			
Glu	120	mg/dL	High
Lac	2.60	mmol/L	High

Reference Ranges			
pCO2	35.0 - 48.0	mmHg	
pO2	83.0 - 108.0	mmHg	
BE(ecf)	-2.0 - 3.0	mmol/L	
cSO2	94.0 - 98.0	%	
Ca++	1.15 - 1.33	mmol/L	
Hct	38 - 51	%	
cHgb	12.0 - 17.0	g/dL	
BE(b)	-2.0 - 3.0	mmol/L	
Glu	74 - 100	mg/dL	
Lac	0.56 - 1.39	mmol/L	

Fig. 1: Arterial blood gas showing severe hypoxia with increased lactate

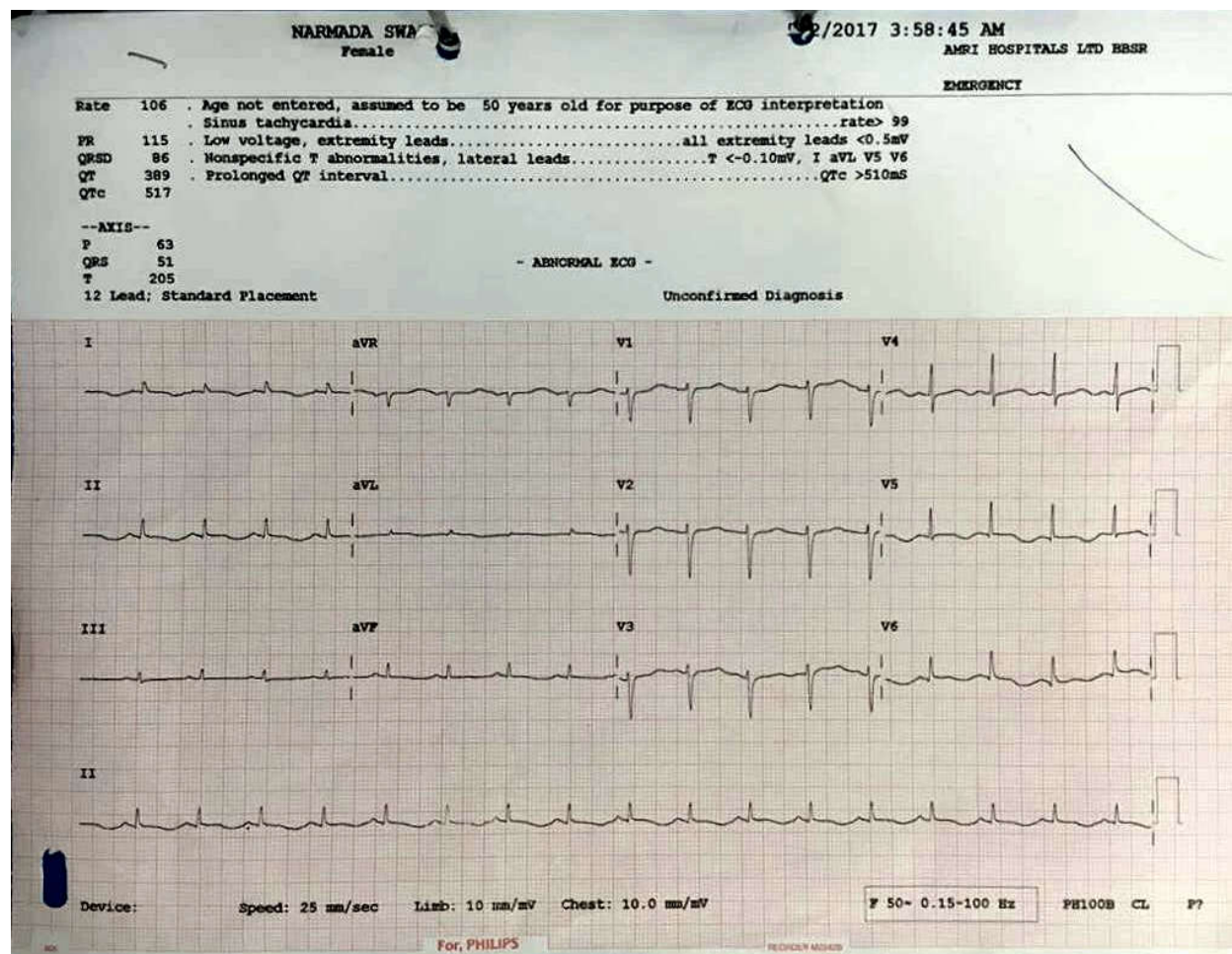


Fig. 2: Electrocardiograph showing sinus tachycardia with T inversions in inferior and lateral leads(I, II, III, aVL, aVF, V4, V5, V6)



**Fig. 3:** Chest X-ray showing diffuse opacities in bilateral lung fields (left more than right) suggestive of asymmetric pulmonary edema

### Discussion

The scorpion venom is a water soluble antigenic complex mixture of neurotoxin, cardiac-toxin, nephrotoxin, haemolysin, phosphodiesterases, phospholipase, hyaluronidases, histamine and other chemicals. These toxins are responsible for intense and persistent depolarization of autonomic nerves with massive release of endogenous catecholamines, an autonomic storm. The primary target of scorpion venom is voltage dependent ion channels. The venom produces both local as well as systemic reactions. Local reactions consist of itching, edema, and ecchymoses with burning pain [4]. The cardiovascular manifestations comprise successively of giddiness, bradycardia, a fall of body temperature; restlessness and tachycardia; and finally pulmonary edema [5].

Scorpion venom can cause myocardial damage by realising vasoactive, inflammatory and thrombogenic peptides and amine constituents (histamine, serotonin, bradykinin, leukotriens).

Which acts on the coronary vasculature and induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis [6].

Direct cardiotoxic effect of the venom causes toxic myocarditis by reduction of Na-K<sup>+</sup> ATPase and adrenergic myocarditis by releasing adrenaline and nor adrenaline from neurons, ganglia and adrenals,

thereby increasing myocardial oxygen demand by direct inotropic and chronotropic effect on already compromised myocardial blood supply [7].

Release of allergenic proteins causes anaphylactic shock leading to hypotension with vasodilatation and decreased intravascular volume with reduced myocardial perfusion [8].

Scorpion venom inhibits angiotensin converting enzyme (ACE) resulting in accumulation of bradykinin which is implicated in the development of pulmonary oedema.

### Conclusion

Pulmonary edema is a common manifestation in scorpion bite but asymmetric pulmonary edema is quite rare. The mechanism of pulmonary edema induced by scorpion bite, though not completely understood, could be due to cardiogenic or non cardiogenic causes. The occurrence of pulmonary edema in our patient could be due to the reasons mentioned in discussion or severe left ventricular dysfunction as evidenced by Doppler echocardiography. What was striking was that pulmonary edema was asymmetrical and it developed more than 24 hours after the scorpion sting despite the use of prazosin early in the course. Patient also had hypotension, tachycardia and warm extremities. Though warm extremities could be due to prazosin therapy, the possibility of warm shock cannot be ruled out especially when the patient had breathlessness, tachycardia and hypotension.

Not only does this case emphasize the occurrence of asymmetrical pulmonary edema in scorpion sting, it also emphasizes that pulmonary oedema can occur late after sting and close monitoring of patients of scorpion sting is required beyond 24 hours as well by the Emergency physician and Critical care doctors.

### References

1. Erfati P. Epidemiology, symptomatology and treatment of buthinae stings. In: Bettini S, editor. *Arthropod Venoms: Hand Book of Experimental Pharmacology*. New York: Springer Verlag; 1978. pp. 312-5.
2. Bawaskar HS, Bawaskar PH. Scorpion sting. *J Assoc Physicians India*. 1998;46:388-92. [PubMed].
3. Bawaskar HS, Bawaskar PH. Indian red scorpion envenoming. *Indian J Pediatr*. 1998;65:383-91. [PubMed].

4. Wallace JF. Disorders caused by venoms, bites and stings. In: Isselbacher KJ, Adams RD, Braunwald E, Petersdorf RG, Wilson JD, editors. *Harrison's Principles of Internal Medicine*. 9th ed. Johannesburg, London, Tokyo, etc: McGraw-Hill International Book Co; 1980.pp.924-5.
  5. Mundle PM. Pulmonary edema following scorpion stings. *Br Med J*. 1961;1:1042.
  6. Yang HP, Chen FC, Chen CC, Shen TY, Wu SP, Tseng YZ. Manifestations mimicking acute myocardial infarction after honeybee sting. *Acta Cardiol Sin*. 2009;25:31-5.
  7. Rahav G, Weiss AT. Scorpion sting-induced pulmonary edema. Scintigraphic evidence of cardiac dysfunction. *Chest*. 1990;97:1478-80. [Pubmed].
  8. Bawaskar HS, Bawaskar PH. Management of scorpion sting. *Heart*. 1999;82:253-4. [PMC free article] [PubMed].
-

## Acute Isolated Posterior Myocardial Infarction; Challenges in Recognition and Management in the Emergency Department

Sarat Kumar Naidu<sup>1</sup>, Ankur Pandey<sup>1</sup>, Kishalay Datta<sup>2</sup>

---

### Author's Affiliation:

<sup>1</sup>DNB Resident <sup>2</sup>HOD and Associate Director, Dept. of Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

### Corresponding Author:

**Sarat Kumar Naidu**,  
DNB Resident, Department of Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.  
E-mail: [saratnaidu@gmail.com](mailto:saratnaidu@gmail.com)

Received on 16.10.2017,

Accepted on 23.10.2017

### Abstract

Posterior wall myocardial infarction (PWMI) accounts for about 15-20% of all STEMIs and is usually seen in the context of inferior and/or lateral wall MI [2]. Isolated posterior wall MI are much less common, of about only 3.3% of all myocardial infarcts [1].

The clinical presentation of PWMI may not be very specific and is confusing even for a cardiologist. Moreover the lack of ST elevation in a standard 12-lead ECG leads to missed or delayed diagnosis of a true PWMI. We are reporting a case of isolated PWMI in a 65 years old, previously healthy male patient, who presented with only gradual onset shortness of breath, who was later found to have 100% LCx stenosis. We have tried to emphasize some facts that may make the clinicians aware of a possible PWMI.

**Keywords:** ST Elevation Myocardial Infarction (STEMI); LCx; PWMI; Posterior ECG Leads V<sub>7</sub>, V<sub>8</sub>, V<sub>9</sub>; Right Coronary Artery (RCA); Left Anterior Descending Artery (LAD); ST Depression; Dominant R Wave; Flip Test; Coronary Angiography (CAG); Troponin I; Percutaneous; Coronary Intervention (PCI); Stenting.

---

### Introduction

PWMI is caused by necrosis of dorsal and infra-atrial part of left ventricle located beneath the atrioventricular sulcus [1].

The majority of PWMI are associated with occlusion of left circumflex artery (LCx) [3-5] but they sometimes may also be associated with right coronary artery (RCA) occlusion.

LCx is the dominant vessel in 10% population and is the least commonly infarcted coronary artery.

PWMI is usually associated with either inferior MI or with lateral wall MI or both where ST elevation can be seen in the respective leads in ECG but when this occurs in isolation ECG diagnosis becomes very difficult. When PWMI is associated with inferior or lateral MI, the area of infarction is very extensive and is associated with high mortality [11,12].

The risk factors for PWMI are same as that of other myocardial infarctions like diabetes, hypertension, hyperlipidemia, smoking etc.

True PWMI is difficult to recognise because the leads of the standard 12-lead electrocardiogram are not a direct representation of the area involved. Only with indirect changes in the precordial leads as such the diagnosis can be suspected.

As the posterior myocardium is not directly visualized in a standard 12-lead ECG, reciprocal changes are seen in the anteroseptal leads V<sub>1</sub>-V<sub>3</sub> [2].

The ECG changes [2] of a true PWMI in a standard 12-lead ECG as seen in leads V<sub>1</sub>-V<sub>3</sub> are as follows:

- Horizontal ST depression (more consistent finding)
- Tall and slightly broad R waves (30ms)
- Upright T waves
- Dominant R wave in V<sub>2</sub> (R/S>1).



However all of these changes may not be present and that makes the diagnosis even more difficult based on ECG alone.

Any patient with ischemic symptoms and horizontal ST depression in anteroseptal leads must be suspected to have a PWMI.

The anteroseptal leads are directed from the anterior precordium towards the internal surface of the posterior myocardial wall. Because posterior electrical activity is recorded from the anterior side of the heart, the typical injury pattern of ST elevation and Q waves becomes inverted; therefore the following changes occur [2].

- ST elevation becomes ST depression
- Q waves become R waves
- Terminal T-wave inversion becomes an upright T wave.

The addition of posterior leads V<sub>7</sub> to V<sub>9</sub> significantly increases the ability to detect posterior MI compared with the standard 12-lead ECG [6,7].

Posterior leads are placed at the following landmarks as shown below (figure 3).

Lead V<sub>7</sub> - at the level of lead V<sub>6</sub> at the posterior axillary line.

Lead V<sub>8</sub> - on the left side of the back at the tip of the scapula.

Lead V<sub>9</sub> - halfway between lead V<sub>8</sub> and the left paraspinal muscles.

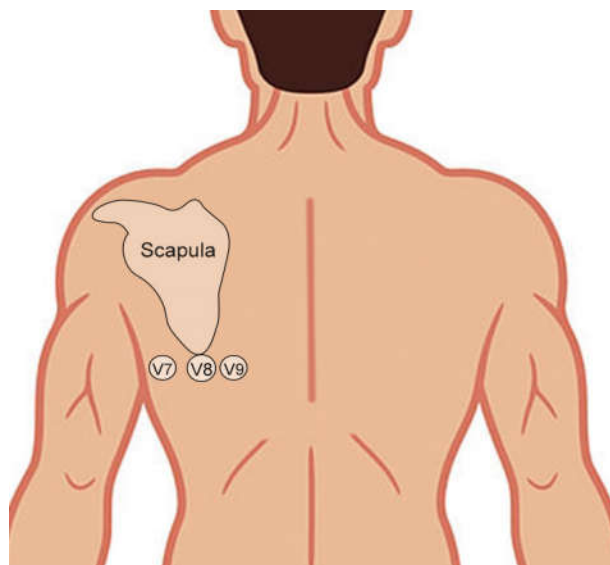


Fig. 1: Placement of posterior leads

When using posterior leads to diagnose PWMI, ST-segment elevation in leads V<sub>7</sub> through V<sub>9</sub> is defined as elevation of at least 0.5 mm in 2 or more of the leads

on the basis of the increased distance between the posterior chest wall and the heart. Posterior ECG leads significantly improve sensitivity and specificity when identifying patients with isolated PWMI [7,8].

Many a Times A “Flip Test” [9] is Performed before doing the Posterior Leads ECG using the Following Steps

1. Get a standard 12 lead ECG
2. Turn it over 180 degrees to look at the back of the upside-down paper.
3. Aim the paper at a bright light source to enable seeing the “flipped” tracings.
4. ST elevation in these leads V1 – V3 with Q waves is consistent with posterior STEMI.

Other supporting investigations like cardiac markers and echocardiography can help in the diagnosis similar to any other types of myocardial infarction.

### Case Study

A 65 years old male patient presented to ED at around 6am with c/o shortness of breath on and off since 2 weeks which got severely aggravated since 3am that woke him up from sleep.

There was no h/o chest pain, cough, nausea, vomiting, palpitations, syncope.

He did not give any h/o chronic illnesses nor was he on any regular medications. He was however an old chronic smoker.

He was taken to the monitored bed and initial evaluation done.

He was conscious, oriented but was tachycardic with PR = 108/min regular and tachypneic with RR = 26/min.

His oxygen saturation was 58% at room air which improved to 90% with oxygen supplementation @8LPM via face mask.

His BP was 150/90 mmHg and random blood sugar level was 263 mg/dl.

He did not have any pallor, cyanosis, icterus, jugular venous distension nor any peripheral edema.

Cardiac monitor showed sinus rhythm and the 12-lead ECG showed sinus rhythm with horizontal ST depression in V<sub>1</sub> to V<sub>5</sub>.

Initial ECG of the patient is shown below (Figure 2).

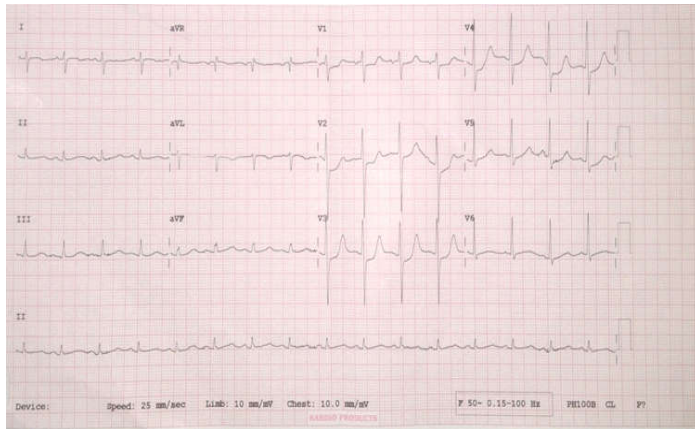


Fig. 2:



Fig. 3:

A large IV canula was inserted in left cubital vein and samples were taken for ABG, cardiac markers, D-dimers, and BNP.

Patient's ABG showed  $\text{pH} = 7.17$ ,  $\text{pO}_2 = 75.2\text{mmHg}$ ,  $\text{Pco}_2 = 50\text{mmHg}$ ,  $\text{HCO}_3 = 17.6\text{mmol/L}$ ,  $\text{Na}^+ = 134\text{meq/L}$ ,  $\text{K}^+ = 4.5\text{meq/L}$ ,  $\text{Ca}^{2+} = 1.16\text{mmol/L}$

Chest X ray showed increased bronchovascular markings B/L. His systemic examination revealed minimal wheeze and basal crepitations on auscultation of lungs b/l and nothing else was significant.

Pt was initially evaluated by a junior Doctor in the ED and was treated in lines of acute exacerbation of COPD and the ECG was initially misinterpreted as either anterior wall ischemia or strain pattern of LVH.

He was given oxygen supplementation with BIPAP support, IV deriphylline, IV Hydrocortisone 200mg and IV Piperacillin +Tazobactam but his symptoms did not improve.

He was then reviewed by a senior ED doctor who after seeing the 1<sup>st</sup> ECG ordered a posterior leads ECG which is shown Above (Figure 3).

This ECG showed mild ST elevation in leads  $V_7$ ,  $V_8$ ,  $V_9$  of  $>1\text{mm}$  and Q waves  $>2\text{mm}$  which strongly suggests posterior wall MI.

By then other laboratory tests showed CKMB 15.9 IU/L, raised myoglobin of 419ng/ml, raised troponin-I of 17ng/ml, raised BNP of 1050pg/ml, and normal D-Dimer and normal urine ketone levels. 2-D echocardiography was done urgently in the ED which showed mild LVH with hypokinetic LCx territory and LVEF of 45% with moderate MR. A diagnosis of posterior wall myocardial infarction with LVF was made and he was given loading dose of Ecosprin 325, Ticagrelor 180 mg, Atorvastatin 80 mg and also was started on Furosemide infusion @ 5 mg/hour and NTG infusion @ 10mcg/min. He was then prepared and sent to cathlab for coronary angiography.

### Course in the Hospital and Outcome

Coronary angiography revealed 100% occlusion in LCx and minimal blockage in RCA and LAD and a stent was placed in LCx after thrombosuction and tyrofiban injection resulting in good TIMI III flow.

He was kept in CCU for observation and was started on post-stenting medications.

His initial creatinine report was 1.2mg/dl but after the angiography it increased to

2.8mg/dl possible due to the contrast.

Nephrology consultation was requested and drug modification was done along with controlled fluid management and his creatinine came down to 1.1mg/dl on 5<sup>th</sup> day of hospitalization. His LVF also revolved after about 5 days of hospitalization.

He was then discharged in a stable condition after 1 week of hospitalization with Ecosprin 75mg HS, Clopidogrel 75mg BD, Rosuvastatin 40mg OD, Metoprolol 25mg BD, Nicorandil 5mg TDS, Furosemide 20mg BD, Ceftum 500mg BD, Alprazolam 0.25mg HS, Pantoprazole 40mg OD.

He was followed up in the cardiology OPD after 4 days and was found to be stable and symptom-free.

### Discussion and Therapeutic Considerations

This case report illustrates a 65 years old male who had isolated PWMI. Coronary angiography (CAG) showed 100% LCx stenosis which was opened and a stent was inserted in the cathlab.



High suspicion by the ED doctor for a PWMI led to timely diagnosis and appropriate interventions to save the life of the patient. The patient's 1<sup>st</sup> ECG showed horizontal ST depression in V<sub>1</sub> to V<sub>5</sub> with tall R waves in V<sub>2</sub>-V<sub>6</sub> with upright T waves in V<sub>1</sub>-V<sub>4</sub> without any ST elevation in inferior or lateral leads.

On high suspicion for a PWMI, a posterior leads ECG was taken which showed ST elevation in V<sub>7</sub>-V<sub>9</sub>, that suggested PWMI. Troponin I was very high and echocardiography showed hypokinetic LCx territory which all confirmed high possibility of PWMI.

Lung crepitations, high BNP and low LVEF suggested left ventricular failure (LVF). Finally PWMI was confirmed in CAG and appropriate management was done with PCI (Percutaneous coronary intervention).

When PWMI is associated with either inferior or lateral wall MI, management is straightforward by giving anti-ischemic therapies and thrombolysis or PCI [10]. However the management of isolated PWMI is somewhat controversial [10]. One school of thought suggests the use of an approach similar to that used for NSTEMI; anti-ischemic, anti-platelet, anti-coagulation and then the patient is taken for CAG with or without PCI [10].

Others are of the opinion that isolated PWMI is an acute infarction and so the patient should undergo urgent PCI similar to management of STEMI; but there is not enough data to support this more aggressive management [10]. However the concept of opening the closed arteries as soon as possible thereby restoring perfusion to the damaged myocardium is likely the better option [10].

In our case it was a right decision to take the patient for urgent CAG+/-PCI; the procedure went uneventful and the patient recovered eventually.

## Conclusion

Why should an emergency physician be aware of the challenges in recognition of an acute posterior wall MI!

This is because this is a STEMI and this requires urgent reperfusion of the myocardium but the diagnosis is often missed or delayed due to lack of typical symptoms and lack of the usual ST elevation of a standard 12-lead ECG.

High degree of suspicion and proper ECG knowledge of a PWMI and appropriate investigations are required for timely diagnosis and management for such a patient.

If there is unnecessary delay in identifying a PWMI due to lack of proper knowledge, there is high risk of ventricular dysfunction and death.

This report will highlight the electrocardiographic fine-tuned diagnosis of PWMI by using the posterior leads V<sub>7</sub> - V<sub>9</sub>, leading to easier and faster recognition with consequences for treatment and improved prognosis.

## References

- 1 <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1847720/>.
- 2 <https://lifeinthefastlane.com/ecg-library/pmi/>.
- 3 Perloff J. The recognition of strictly posterior myocardial infarction by conventional scalar electrocardiography. *Circulation* 1964;30:706-18. [PubMed].
- 4 Agarwal J, Khaw K, Aurignac F, et al. Importance of posterior chest leads in patients with suspected myocardial infarction, but nondiagnostic, routine 12-lead electrocardiogram. *Am J Cardiol* 1999;83:323-6.[PubMed].
- 5 Bough E, Korr K. Prevalence and severity of circumflex coronary artery disease in electrocardiographic posterior myocardial infarction. *J Am Coll Cardiol* 1986;7:990-6. [PubMed].
- 6 Rich MW, Imburgia M, King TR, Fischer KC, Kovach KL. Electrocardiographic diagnosis of remote posterior wall myocardial infarction using unipolar posterior lead V9. *Chest*. 1989 Sep;96(3):489-93. DOI: <http://dx.doi.org/10.1378/chest.96.3.489>. [PubMed].
- 7 Matetzky S, Freimark D, Feinberg MS, et al. Acute myocardial infarction with isolated ST-segment elevation in posterior chest leads V7-9: "hidden" ST-segment elevations revealing acute posterior infarction. *J Am Coll Cardiol*. 1999 Sep;34(3):748-53. DOI: [http://dx.doi.org/10.1016/S0735-1097\(99\)00249-1](http://dx.doi.org/10.1016/S0735-1097(99)00249-1). [PubMed].
- 8 <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5293126/#b7-ecg>
- 9 <https://www.aliem.com/2013/08/posterior-myocardial-infarction-how-accurate-is-the-flipped-ecg-trick/>.
- 10 <http://www.patientcareonline.com/cardiovascular-diseases/isolated-acute-posterior-myocardial-infarction-middle-aged-man>.
- 11 Oraili S, Maleki M, Abbas Tavakolian A, et al. Prevalence and outcome of ST-segment elevation in posterior electrocardiographic leads during acute myocardial infarction. *J Electrocardiol* 1999;32:275-8. [PubMed].
- 12 Matetzky S, Freimark D, Chouraqui P, et al. Significance of ST segment elevations in posterior chest leads (V<sub>7</sub> to V<sub>9</sub>) in patients with acute inferior myocardial infarction: application for thrombolytic therapy. *J Am Coll Cardiol* 1998;31:506-11. [PubMed].

## Petrol Ingestion Causing Methaemoglobinaemia in Glucose 6-Phosphate Dehydrogenase (G6PD) Deficiency Patient

Nasir Shakilli<sup>1</sup>, Mohammad Kamal<sup>2</sup>, Bakshi Surrinder Kumar<sup>3</sup>, Madhusudhanan M.<sup>4</sup>, Moosa Al Abri<sup>5</sup>, Almur Abdullah Alabri<sup>2</sup>

### Author's Affiliation:

<sup>1</sup>Specialist <sup>2</sup>Senior Specialist  
<sup>5</sup>Medical Officer, Department of  
Medicine, <sup>4</sup>Specialist, Department  
of Pathology, Rustaq Hospital,  
Rustaq, Sultanate of Oman.  
<sup>3</sup>Associate Professor, Department  
of Medicine, Oman Medical  
College, Rustaq/Sohar, Sultanate  
of Oman.

### Corresponding Author:

**Surrinder Kumar Bakshi**,  
Associate Professor, Department  
of Medicine, Oman Medical  
College, Rustaq/Sohar, Sultanate  
of Oman.  
E-mail: bakshisk@omc.edu.om

Received on 09.06.2017,  
Accepted on 28.06.2017

### Abstract

A 31-year-old male found unconscious in his parked car on road side was admitted for evaluation and management in the hospital. The patient presented with central and peripheral cyanosis and arterial blood gas (ABG) report revealed 54.4% oxyhaemoglobin, 44.5% methaemoglobin and 1.1% carboxyhaemoglobin. The patient was put on 100% oxygen. Keeping in view the ABG report, the patient was treated as methaemoglobinaemia and started on 1% methylene blue solution intravenously. The patient, on regaining consciousness, told that he had accidentally ingested petrol a few hours back. Later on, further investigations revealed that the patient had bite cells in peripheral blood smear suggestive of hemolytic anaemia. The patient informed that he is a known case of Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency. The patient was managed as a case of petrol ingestion methaemoglobinaemia with G6PD deficiency and discharged from hospital.

**Keywords:** G6PD Deficiency; Methaemoglobinaemia; Methylene Blue; Petrol Ingestion.

### Case Report

A 31-year-old male was found unconscious in his parked car on road side. He was brought to A & E department and admitted in the hospital for evaluation and management. The patient presented with central and peripheral cyanosis, bilateral crackles at the bases of lungs with spleen 3 cm below costal margin. Arterial blood gas (ABG) report revealed 54.4% oxyhaemoglobin, 44.5% methaemoglobin and 1.1% carboxyhaemoglobin. While the blood samples were being drawn, the sister-in-charge noticed the chocolate-brown colour of blood and informed the treating physician. The G6PD level estimation and High Performance Liquid Chromatography (HPLC) could not be done as these facilities were not available in the hospital. The laboratory parameters on admission were: Haemoglobin 115 g/L, Haematocrit 33%, C-reactive protein (CRP) 72 mg/L (Normal 0-5 mg/L), direct Coomb's test (DCT) and sickling tests were negative.

The patient developed jaundice and his serum lactic dehydrogenase (LDH) levels increased which returned to normal after a few days. The reports of laboratory tests/ABG are tabulated (Table 1). The urine of the patient was dark in colour. Renal function tests were normal. Chest X-ray showed bilateral haziness at bases and CT chest and abdomen showed bilateral lower lobe consolidation (probably due to aspiration) and spleen 15 cm long in long axis. Electrocardiography (ECG) was within normal limits. Keeping in view the ABG report and his oxygen saturation levels and non-availability of G6PD level estimation and HPLC chromatography, the patient was treated as a case of methaemoglobinaemia and was started on IV 1% methylene blue solution and put on 100% oxygen. Methylene blue (1 mg/kg body weight) was given in the dose of 50 mg IV slowly over 5 minutes and after 30 minutes each two more IV doses of methylene blue 50 mg IV were given (Total 3 doses of methylene blue 50 mg each were administered). The patient was also given Tazobactam/piperacillin 4.5 g thrice a day for five days and initially Inj.

Omeprazole 40 mg twice a day IV and the shifted on oral omeprazole besides IV dextrose saline. The patient, on regaining consciousness, told that he had accidentally ingested petrol a few hours back and he is a case of G6PD deficiency. Peripheral blood smear revealed mild anisocytosis, normocytic normochromic polychromasia with occasional nucleated RBC and presence of bite cells suggestive of hemolytic anaemia due to G6PD deficiency (Figure1).

Levels of benzene were not measured in blood and urine due to non-availability of facilities. The patient received five units of blood and fresh frozen plasma during hospitalization. The patient was managed as a case of methaemoglobinaemia with underlying haemolytic anaemia due to G6PD deficiency and discharged from hospital.

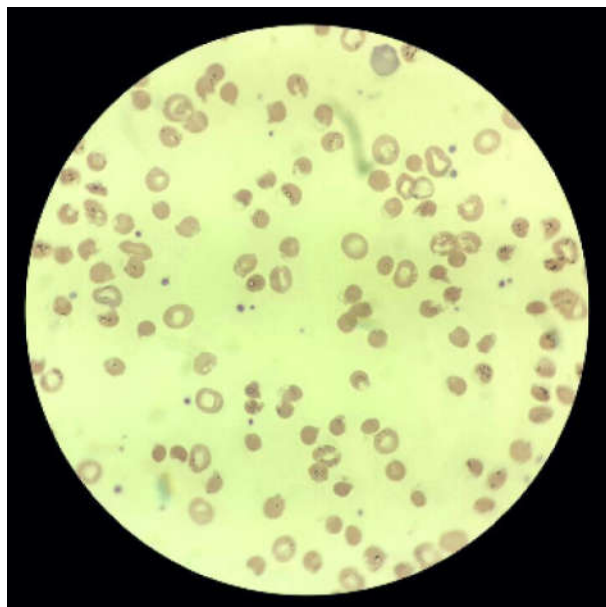


Fig. 1: Peripheral blood smear showing mild anisocytosis, normocytic normochromic polychromasia, occasional nucleated RBCs and bite cells (Wright Stain, 10x100 Magnification)

## Discussion

Petrol is a life-line for any developed or developing country. Countries like USA and Europe have their petrol (gasoline) marketed with 1-5% benzene [1,2]. Sultanate of Oman, one of the middle East countries is also marketing petrol (gasoline) with less than 5% benzene content [3].

Benzene is acutely toxic by inhalation, causing mucous membrane irritation, neurological and other symptoms due to respiratory failure. Chronic exposure has been reported to result in bone marrow depression, aplasia and leukaemia, cardiac abnormalities, heart attack and other cancers of lung, brain and stomach. Following inhalation, benzene vapour is rapidly absorbed into the blood and distributed throughout the body. One of the effects of benzene in the body is the production of methaemoglobin (MetHb) which contains iron in ferric state ( $Fe^{3+}$ ) [4].

Methaemoglobinaemia is a rare condition characterised by increased quantities of haemoglobin in which the iron of haem is oxidised to the ferric ( $Fe^{3+}$ ) form. Clinically the condition presents with cyanosis and low oxygen saturations on pulse oximetry but normal oxygen saturation on arterial blood gas analysis. Most cases are acquired and are frequently drug-related.

Udonwa NE et al [5] studied the exposure of petrol station attendants and auto-mechanics to premium motor spirit fumes in Nigeria and suggested increased exposure to petrol fumes among automobile mechanics, petrol station attendants and MetHb as a useful biomarker in determining the level of exposure to benzene in petrol vapour.

Our patient had ingested petrol by accident and as petrol is volatile, some of the petrol may have gone into the respiratory tract causing bilateral consolidation and chemical pneumonitis.

Table 1: Relevant ABG/ blood parameters of the patient on Day 1-3 in the hospital

Relevant Blood/ ABG Parameters	Normal Range in our Hospital	Day 1 1346 hrs	Day 1 1949 hrs	Day1 2134 hrs	Day 1 2344 hrs	Day 2 0705 hrs	Day 2 1847 hrs	Day 3 1448 hrs
Met Hb	(0.0-1.5%)	44.5%	30.0%	26.7%	25.6%	15.0%	9.8%	9.7%
O <sub>2</sub> Hb	(94.0-98.0%)	54.4%	68.0%	71.7%	72.4%	83.0%	87.3%	82.5%
CO Hb	(0.0-3.0%)	1.1%	1.3%	1.5%	1.8%	2.0%	2.9%	5.1%
SO <sub>2</sub>	(94.0-98.0%)	100%	99.1%	99.8%	99.7%	100%	100%	96.9%
Hb	(11.5-17.8g/ dL	12.7g/ dL	11.6g/ dL	11.1g/ dL	10.3g/ dL	8.1g/ dL	8.1g/ dL	7.3g/ dL
Hct	(36.0-53.0%)	41.8%	38.5%	37.5%	34.5%	29.6%	29.2%	27.4%

**Table 2:** Signs and symptoms associated with different levels of methaemoglobin in blood [6]

Methaemoglobin levels	3-15%	15-25%	25-50%	50-70%	Above 70%
Signs and symptoms	Pale, gray or blue discoloration of the skin may be present	Mild cyanosis otherwise relatively asymptomatic	Headache, dyspnoea, lightheadedness, syncope, weakness, confusion, palpitations, chest pain	Cardiovascular- Abnormal cardiac rhythms CNS-Altered mental status; delirium, seizures, coma Metabolic- Profound acidosis	Death

Acute methemoglobinemia can be life-threatening and usually is acquired as a consequence of exposure to toxins or drugs. Therefore, obtaining a detailed history of exposure to methemoglobinemia-inducing substances is important. Such history may not always be forthcoming, but it should always be sought actively since long-term or repeated exposure may occur. Consultation with a toxicologist may be necessary, especially with exposure to a new medication, because the list of medications known to cause methemoglobinemia changes constantly. Symptoms are proportional to the fraction of methemoglobin. A normal methemoglobin fraction is about 1% (Range 0-3%). Symptoms associated with various levels of methaemoglobin are shown (Table 2)[6].

G6PD deficiency, the most common human enzymopathy, affects 10% of the world's population, causing haemolysis due to intake of various drugs and other conditions [7]. G6PD deficiency is common in Oman with the G6PD Mediterranean mutation accounting for most cases [8].

Clarification regarding known family history of methemoglobinemia or glucose-6-phosphate dehydrogenase (G6PD) deficiency is important. Even patients who are heterozygous for methemoglobin reductase enzyme deficiencies are susceptible to low doses of oxidant drugs with resultant methemoglobinemia. In our case report, we were unable to ask the history at the time of admission to rule out G6PD deficiency as he was found in an unresponsive state. Since the facilities were not available at the hospital to assess the level of percentage of G6PD deficiency and HPLC chromatograph, it was decided to treat the patient with IV 1% methylene blue solution.

### Conclusion

The case report is unique because of accidental ingestion of petrol by the patient and lying unconscious at roadside in his car. The patient's

health status was further complicated by his being a G6PD deficiency patient which was unknown till he regained consciousness. Patients of G6PD deficiency should be encouraged to carry an identity card or bracelet which may be life-saving and help them getting the best treatment in emergency situations.

### Conflict of Interest and Funding

The authors reported no conflict of interest and no funding was received for this work.

*Disclosure:* Nil

### References

- Wallace L. Environmental exposure to benzene: an update. *Environmental Health Perspectives* 1996;104 (Suppl.6):1129-1136.
- Owen K and Coley T. *Automotive Fuels Reference Book*, Society of Automotive Engineers, Warrendale, PA, USA, 2<sup>nd</sup> Edition, 1995.
- Unleaded Gasoline. Material Safety Data Sheet (MSDS). Oman Refinery Company LLC, Muscat, Oman. <http://www.guidechem.com/cas-862/86290.81.5.html>. Website accessed on 27 May 2017.
- Travis CC, Quillen JL and Arms AD. Pharmacokinetics of benzene. *Toxicology and Applied Pharmacology* 1990;102:400-420.
- Udonwa NE, Uko EK, Ikpeme BM, Ibanga IA, Okon BO. Exposure of petrol station attendants and automechanics to premium motor spirit fumes in Calabar, Nigeria. *Journal of Environmental and Public Health* 2009 June: Article ID 281876: 5 pages.
- Www. Medscape.com. Acquired methaemoglobinemia. Accessed on 02 June 2017.
- Davidson's Principles and Practice of Medicine 2014. 22<sup>nd</sup> Edition. Edited by Walker BR et al. Published by Churchill Livingstone Elsevier. pg 1029.
- Daar S, Vulliamy TJ, Kaeda J, Mason PJ, Luzzatto L. Molecular characterization of G6PD deficiency in Oman. *Hum Hered* 1996;46(3):172-176.

## Early Diagnosis and Treatment not Always a Key to Favorable Outcome: A Case Report of ADEM Correctly Diagnosed and Treated Still Surviving for Better Life

Aakansha Singh<sup>1</sup>, Vaibhav Gulati<sup>1</sup>, Kishalay Datta<sup>2</sup>, Hilal Ahmad Yatoo<sup>3</sup>

### Author's Affiliation:

<sup>1</sup>MEM, PGY-3,  
<sup>2</sup>HOD and Associate Director  
<sup>3</sup>Attending Consultant, Dept. of  
Emergency Medicine, Max Super  
Specialty Hospital,  
Shalimar Bagh, New Delhi, Delhi  
110088, India.

### Corresponding Author:

**Vaibhav Gulati**, PGY-3, MEM  
{GWU-USA},  
Dept. of Emergency Medicine,  
Max Super Specialty Hospital,  
Shalimar Bagh, New Delhi, Delhi  
110088, India.

E-mail:  
[dr.vaibhavgulati@gmail.com](mailto:dr.vaibhavgulati@gmail.com)

Received on 21.08.2017,

Accepted on 14.09.2017

### Abstract

Acute disseminated encephalomyelitis (ADEM) is a rare disease of central nervous system with a spectrum of presentation. It is a diagnosis of exclusion and relies on neuroimaging which may be normal at the onset. It is a diagnostic challenge at its first attack. The disease is although more common in children it can invariably be present in adults. Here we present a case report of ADEM in a 30 year old female who presented to ER with history of multiple episodes of vomiting followed by sudden onset of dysphasia and other neurological complaints. The patient had a history of recent travel to a pilgrimage where she had enteric fever around 15 days ago. It was our neurology team which correctly recognised and treated it as ADEM. The patient responded well to the treatment and discharged in stable condition after 5 days. Sadly the disease had a relapse which now showed no response to iv immunoglobulins, steroids or plasmapheresis. The patient was in the hospital for symptomatic management and is still surviving in the hope of a normal well being.

**Keywords:** Acute Disseminated Encephalomyelitis; Central Nervous System; Neuroimaging.

### Introduction

Acute disseminated encephalomyelitis (ADEM) is an inflammatory demyelinating disease of the central nervous system. Its onset is acute and often rapidly progressive. It is traditionally mono phasic but some patients may have recurrences.

ADEM typically presents with multifocal neurological signs, including motor, sensory, cranial nerve, brainstem deficits as well as nonspecific symptoms such as headache, malaise and altered mental status.

The diagnosis is supported by the presence of one or more supratentorial or infratentorial demyelinating lesions in the brain on magnetic resonance imaging (MRI) and the absence of destructive black hole lesions on T1-weighted MRI. Abnormal cerebrospinal fluid findings such as mild lymphocytic pleocytosis and slightly elevated protein level are suggestive of ADEM.

More than half of patients have an illness, usually an infection, two to four weeks before developing ADEM. Most of these illnesses are viral or bacterial. In children with ADEM, prolonged and severe headaches occur. In addition the patient develops fevers during the ADEM course.

Along with this pattern, the patients usually get neurological symptoms which may include:

- Confusion, drowsiness and even coma
- Unsteadiness and falling
- Visual blurring or double vision
- Trouble swallowing
- Weakness of the arms and legs

In adults with ADEM, motor (movement) and sensory (tingling, numbness) symptoms tend to be more common. Overall what triggers a diagnosis of ADEM is a rapidly developing illness with neurological symptoms often with fever and headache

usually following an upper respiratory tract infection and which has significant MRI and spinal fluid findings consistent with ADEM.

### Case Report

30 year old female brought by attendants with history of multiple episodes of vomiting followed by sudden onset of dysphasia.

On examination in emergency her vitals were HR-88/m, BP-130/80mm Hg, RR-18/m, T-99 F, RBS-140mg/dl with patent airway and bilateral equal air entry.

Secondary examination was all normal except CNS which revealed GCS E4V1M6, planters bilateral mute, right sided neck dystonia and reflexes all limbs 2+.

The attendants gave a history of recent travel to some pilgrimage around 15 days ago where patient had complaints of loose watery stools and vomiting. She was diagnosed as enteric fever and managed symptomatically.

All routine investigations were sent from the ER and MRI brain planned. The blood reports revealed elevated TLC levels. MRI brain showed multiple demyelinating lesions in bilateral cerebral hemisphere. CSF was acellular with high protein. A diagnosis of ADEM was made.

Patient was admitted under Neurology team and treatment was started accordingly.

Patient received high dose of steroids, immunoglobulins, iv fluids, iv antibiotics. She gradually became better and discharged home in a stable conditions with advise for gradual ambulation.

After about one month, patient represented with complaints of mild remitting fever since 10 days, history of twisting of the tongue around 6 days ago. Weakness of right side of body since 1 day with decreased responsiveness since the day of readmission.

Again the vitals were normal, secondary examination was all normal except CNS which revealed GCS-E4V1M5, plantars bilateral extensor, hyper reflexia, power grade- Left side -5/5, Right side-1/5. Bilateral pupils mid dilated with sluggishly reaction. Repeat MRI revealed similar changes of severe ADEM with brain stem involvement.

Patient was again admitted under Neurology unit and was restarted on steroids, anti epileptics. Plasmapheresis was done but the patient's clinical condition gradually detoriated. The patient had decerebrate rigidity with severe hyperthermia for which she had been treated accordingly. The patient was sent home in the same state and advised symptomatic management.

It has been found that the family is still making all possible efforts but no response is noticed.

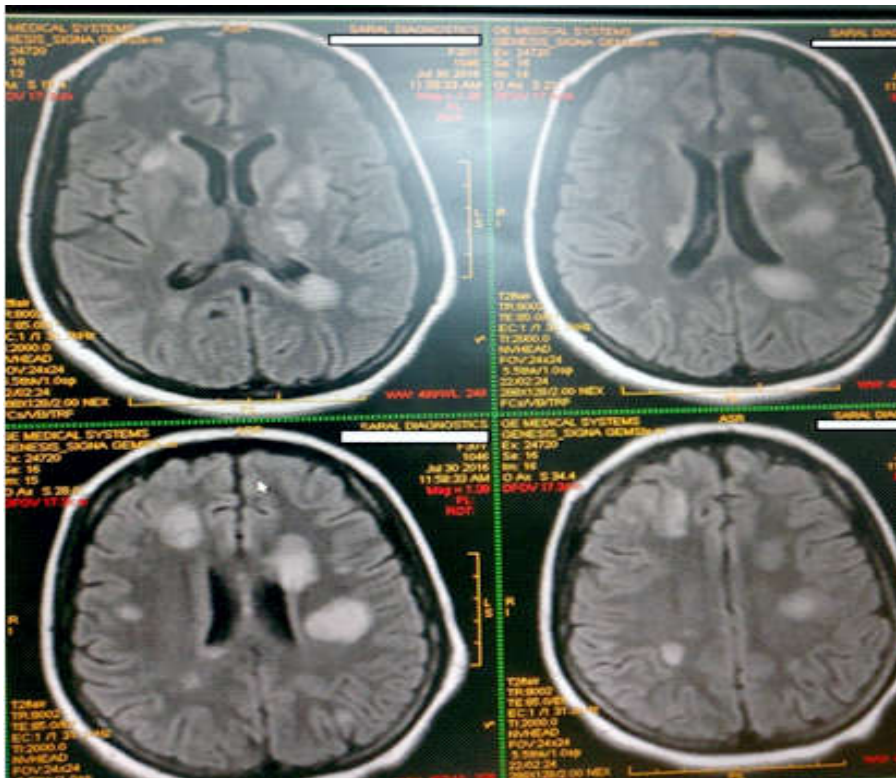


Fig. 1:



## Discussion

Early diagnosis and management is definitely a key to every disease but the response it has on every individual is not unanimous. Here we had a 30 year old female who was correctly diagnosed and treated for ADEM. The results were favourable initially but the relapse showed no response to the appropriate management of the disease. It has been more than 8 months now that the patient is in a debilitated stage though every attempt is continued to make her live better.

## References

1. Poser CM, Brinar VV. Disseminated encephalomyelitis and multiple sclerosis: two different diseases-a critical review. *Acta Neurol Scand.* 2007;116:201-206. [PubMed].
  2. Mikaeloff Y, Caridade G, Husson B, Suissa S, Tardieu M. Neuropediatric KIDSEP Study Group of the French Neuropediatric Society. Acute disseminated encephalomyelitis cohort study: Prognostic factors for relapse. *Eur J Paediatr Neurol.* 2007;11:90-5. [PubMed].
  3. Tenenbaum S, Chamoles N, Fejerman N. Acute disseminated encephalomyelitis: A long-term follow-up study of 84 pediatric patients. *Neurology.* 2002;59:1224-31. [PubMed].
  4. Apak RAKose GAnlar BTuranli GTopaloglu HOzdirim E Acute disseminated encephalomyelitis in childhood: report of 10 cases. *J Child Neurol* 1999; 14:198-201.
  5. Murthy SNFaden HSCohen MEBakshi R Acute disseminated encephalomyelitis in children. *Pediatrics* 2002;110e21.
  6. Hart MN, Earle KM. Haemorrhagic and perivenous encephalitis: a clinical-pathological review of 38 cases. *J Neurol Neurosurg Psychiatry* 1975;38(6):585-591.
  7. Mikaeloff Y, Caridade G, Assi S, Suissa S, Tardieu M. Prognostic factors for early severity in a childhood multiple sclerosis cohort. *Pediatrics* 2006;118(3): 1133-1139.
-

## Traumatic Cardiac Tamponade - Relearning Old Lesions to Avoid Delay in Diagnosis and Management of a Life-Threatening Thoracic Injury

Sarat Kumar Naidu<sup>1</sup>, Vikram Shah<sup>1</sup>, Gurjit Kaur<sup>2</sup>, Kishalay Datta<sup>3</sup>

### Author's Affiliation:

<sup>1</sup>DNB Resident <sup>2</sup>MEM Resident  
<sup>3</sup>HOD and Associate Director,  
Department of Emergency  
Medicine; Max Hospital,  
Shalimar Bagh, New Delhi, Delhi  
110088, India.  
E-mail: saratnaidu@gmail.com

### Corresponding Author:

**Sarat Kumar Naidu**  
DNB Resident, Department of  
Emergency Medicine, Max  
Hospital, Shalimar Bagh, New  
Delhi, Delhi 110088, India.  
E-mail: saratnaidu@gmail.com

Received on 16.10.2017,

Accepted on 30.10.2017

### Abstract

Cardiac tamponade is a life-threatening condition due to abnormal collection of fluid in the pericardial sac causing hemodynamic instability. In trauma it is blood that gets collected in the sac, most commonly due to penetrating chest injuries or less commonly, blunt chest trauma. If this is not diagnosed and intervened timely, this can be rapidly fatal. We are reporting a case of 35 years old male who was involved in a road traffic accident (RTA). He sustained steering wheel injury on his chest and was taken to multiple hospitals where he was managed only conservatively due to missed diagnosis, before presenting to our ED (Emergency Department). He was here diagnosed with cardiac tamponade with obstructive shock and was urgently taken to operation theatre (OT) for pericardial decompression and was saved.

**Keywords:** Cardiac Tamponade; Thoracic Injury; Hypotension; Obstructive Shock; Beck's Triad; Muffled Heart Sounds; Pulsus Paradoxus; Electrical Alternans; Kussmaul Sign; Road Traffic Accident (RTA); Controlled Fluid Resuscitation; Thoracotomy; Sternotomy; Pericardiectomy; Pericardiotomy; FAST Scan.

### Introduction

Traumatic cardiac tamponade most commonly occurs in penetrating thoracic injuries, more specifically penetrating cardiac injuries. However blunt injuries can also produce tamponade commonly due to cardiac rupture, injuries of great vessels or pericardial vessels.

In atraumatic tamponade, fluid gets collected gradually in the pericardial sac over a period of weeks to months depending upon the cause and the body's compensatory mechanism keeps the hemodynamics stable for a longer period of time. This may be referred to as chronic tamponade and may collect as high as 1litre fluid [1].

However in trauma, blood gets collected in a short span of time usually minutes to hours causing severe rapid hemodynamic instability. This is acute tamponade or may be referred as surgical tamponade; as little as 150ml blood can be lethal [1].

Tamponade is defined as the decompensated phase of cardiac compression resulting from increased intrapericardial pressure [1]. This causes decreased venous return, decreased cardiac output, hypotension, obstructive shock, hypoperfusion, metabolic acidosis and multi-organ dysfunction syndrome (MODS).

Figure 1 shows how fluid or blood gets collected in the pericardial sac in tamponade.

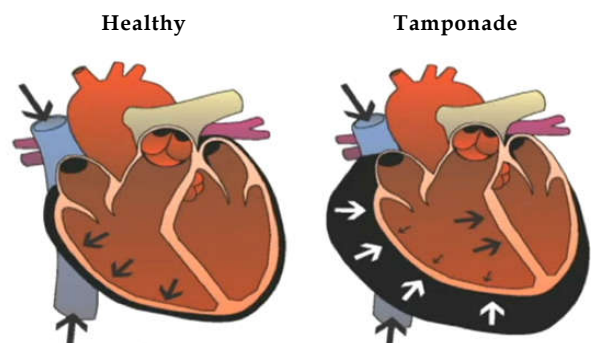
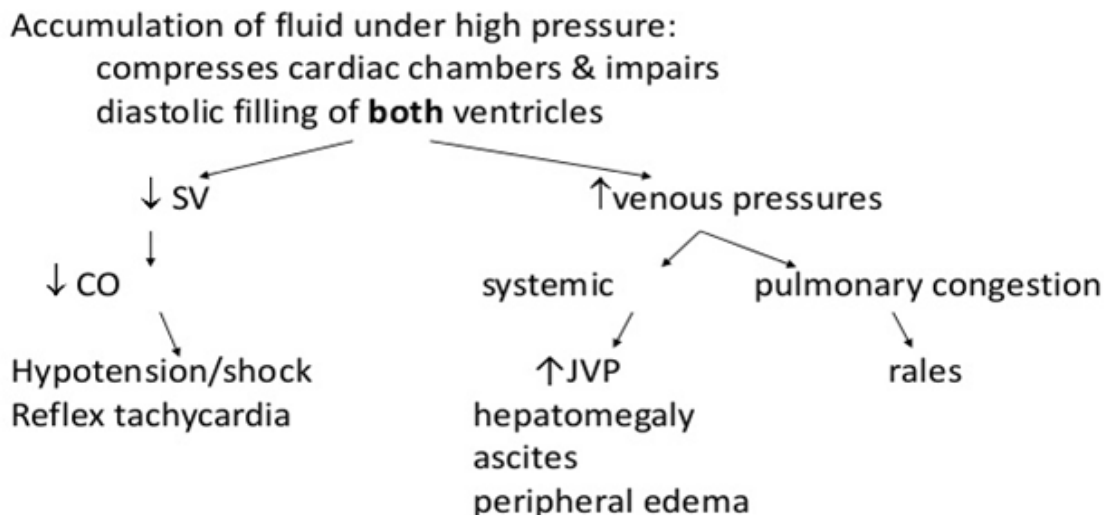


Fig. 1:



## Cardiac Tamponade -- Pathophysiology



The pathophysiology of tamponade can be demonstrated pictorially as Above (Figure 2)

The underlying process for the development of tamponade is a marked reduction in diastolic filling, which results when transmural distending pressures become insufficient to overcome increased intrapericardial pressures [4]. Tachycardia is the initial cardiac response to these changes to maintain the cardiac output [4]. The amount of pericardial fluid needed to impair diastolic filling of the heart depends on the rate of fluid accumulation and the compliance of the pericardium. Rapid accumulation of as little as 150mL of fluid can result in a marked increase in pericardial pressure and can severely impede cardiac output, whereas 1000 mL of fluid may accumulate over a longer period without any significant effect on diastolic filling of the heart [4].

The typical features of tamponade popularly called Beck's triad are

- Hypotension
- Raised JVP or CVP
- Muffled heart sounds

Raised JVP may not be present many a times due to presence of severe hypotension.

Beck's triad is found only in 10% of patients with tamponade [2].

Some other clinical features [3] of cardiac tamponade are chest tightness, tachypnea,

tachycardia, confusion/altered mental status, oliguria/anuria, cold clammy extremities, pulsus paradoxus (drop in systolic BP >10mmHg during inspiration) etc.

Kussmaul sign (Paradoxical increase in jugular venous pressure during inspiration) is also sometimes seen.

Tamponade is a medical emergency, the complications of which include pulmonary edema, shock, renal failure and death [4].

The overall risk of mortality depends on the speed of diagnosis, the treatment provided, and the underlying cause of the tamponade. If left untreated, the condition is rapidly and universally fatal [4].

### Case Study

A 35 years young male was brought to our ED at around 12 midnight with an alleged h/o RTA 4 hours back. He was driving a car when his car hit head-on with another vehicle coming from opposite direction. There was apparently no loss of consciousness (LOC), seizures, vomiting, ENT bleed. The patient was then taken to 2 different nearby hospitals by the paramedics before being brought to our ED.

As per the notes of previous two hospitals, he was diagnosed as blunt chest injury and was managed conservatively after doing chest X ray and FAST scan

which were reported normal then. However the patient was deteriorating in terms of consciousness and this is when his attendants brought him to our hospital for further management.

He was immediately taken to a monitored bed and initial trauma evaluation done.

He was drowsy and not responding to verbal commands.

Airway was patent with C-collar in situ; no secretions or blood in oral cavity; trachea was in midline and the neck veins were not distended.

Breathing rate was rapid with RR 28/min and oxygen saturation was only 76% at room air which improved to 80% with high flow oxygen via facemask. Air entry were equal and clear bilaterally with minimal bony crepitus over midsternal region on palpation; however there was no external sign of injury on inspection.

In terms of circulation, his pulse rate was 125/min with feeble central pulses and non-palpable peripheral pulses; BP was not recordable; Capillary refill time (CRT) was more than 4 seconds, heart sounds were difficult to be appreciated in the noisy ED.

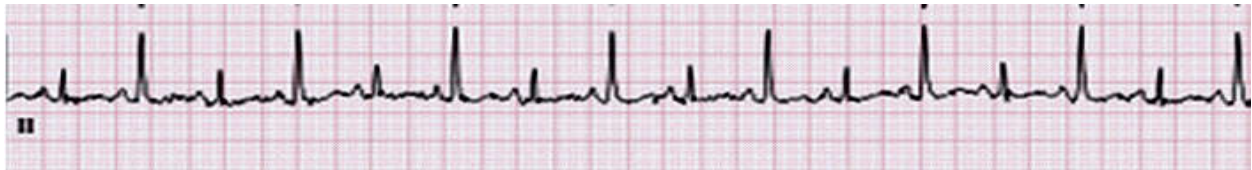


Fig. 3:

CXR showed mildly increased cardiac shadow and FAST scan revealed fluid in pericardial sac around 500-700ml causing tamponade effect.

Other radiology imaging were not done in the ED due to hemodynamic instability.

Patient's VBG showed  $\text{pH} = 7.206$ ,  $\text{pO}_2 = 14.7$  mmHg,  $\text{PCO}_2 = 48$  mmHg,  $\text{HCO}_3 = 18.5$  mmol/L,  $\text{Na}^+ = 146$  meq/L,  $\text{K}^+ = 3.2$  meq/L,  $\text{Ca}^{2+} = 1.01$  mmol/L, Lactate = 3.9; he was in metabolic acidosis.

A provisional diagnosis of Cardiac tamponade with obstructive shock was made and the Cardiothoracic surgeon was informed immediately who after evaluation advised to shift the patient immediately to operation theatre (OT).

Controlled fluid resuscitation was given in order to avoid further worsening of the condition and just to get a palpable peripheral pulse.

In terms of disability, his GCS score was E2V3M5 = 10/15; random blood sugars (RBS) was 114mg/dl; pupils were bilaterally equal and normally reacting to light and there were no lateralizing signs.

On exposure, peripheries were cold and clammy; swelling on mid-forehead of 2x2 cms; there was no other external sign of injury.

Ryle's tube was in situ; no bleeding seen.

Foley's catheter was in situ; only 30ml urine since last 3 hrs.

Trauma code was activated and 2 large bore IV canulae were inserted in cubital veins and samples taken for VBG, Blood grouping Crossmatching, complete blood count, kidney function tests and liver function tests.

Radiological imaging studies were ordered; CXR, NCCT Head, NCCT C-Spine, Pelvic Xray, and FAST scan.

On secondary survey, the only significant finding was mid-sternal deformity with bony crepitus.

ECG rhythm strip showed electrical alternans as shown below (Figure 3) and 12 lead ECG also showed only sinus tachycardia with electrical alternans.

BP came up to 70mmHg systolic but he was still in altered mental status.

Pt was taken for urgent thoracotomy/sternotomy.

### Course in the Hospital and Outcome

Patient was electively intubated in the OT and was put on mechanical ventilator and general anaesthesia induced.

Intraoperatively, there was a complete fracture of mid-sternal region; sternotomy was done followed by pericardiectomy; 700ml blood clot was removed from the pericardial sac; diffuse bleeding found in the SVC region which was controlled and wound closed with 3 drains. Following pericardial decompression his pulse and blood pressure started settling down.

He was shifted to ICU early morning for observation with stable vitals with pulse of 90/min and BP of 90/60 mmHg.

His 1<sup>st</sup> set of laboratory reports showed urea of 25mg/dl and creatinine of 0.97mg/dl and samples taken just after the surgery showed increased creatinine of 1.48 mg/dl, meaning that he developed acute kidney injury (AKI).

When hemodynamically stabilized, he was sent for other radiological imaging studies including CT head, C-spine and thorax and multiple X rays which did not reveal anything significant.

On 1<sup>st</sup> post-op day, he was extubated and was conscious and oriented and vitals were stable without any inotropic support with good urine output.

Repeat echocardiography showed no pericardial fluid collection.

His renal function tests also improved when his hemodynamics got stabilized.

He was eventually discharged after 5 days of hospital stay in a stable condition; OPD follow up after 3 days was also satisfactory.

### Discussion and Therapeutic Considerations

This case report illustrates a 65 years old male who sustained blunt chest trauma and presented with altered mental status and hypotension. He was misdiagnosed in previous two hospitals where he presented first and was then brought to our ED.

He was later diagnosed to have cardiac tamponade with obstructive shock and acute kidney injury.

He was immediately taken to OT for cardiac decompression after which he improved.

Cardiac injuries are most commonly overlooked injuries in patients who die from trauma.

The case we describe here is unusual in 4 counts. First, around 700ml blood was removed from the pericardial sac without any evidence of cardiac rupture. Secondly, once the blood and blood clots were removed and the SVC laceration repaired, he improved quickly without any re-effusion later. Thirdly, although it was a high speed RTA, he had only isolated cardiac tamponade without any other injury. Fourthly, the AKI which developed due to hypotension improved quickly once the tamponade was relieved.

The initial CXR did not show any sign of tamponade and the initial FAST scan also was normal

which means that the bleeding was more gradual over a period of 3-4 hours to cause the tamponade effect and hemodynamic instability.

Moreover the initial aggressive fluid resuscitation given to stabilize the BP might have worsened the condition of bleeding vessel (s). This emphasizes the importance of balanced resuscitation in trauma when there is hemodynamic instability and the source of bleeding is not yet identified and controlled.

At some point, it was thought that altered mental status could be due to head injury as there was a forehead hematoma which created suspicion and the hypotension could be due to spinal shock but with high degree of suspicion cardiac tamponade was correctly diagnosed and appropriate treatment was given and the patient was saved without any morbidities.

In 2009, Rastogi, described a case of a 50 years old man who was hit by a motorbike who was conscious and oriented with stable vitals but had only mild breathing difficulty without any external signs of injury; he was discharged after giving first aid. The man died after 7-8 hrs and his postmortem report revealed cardiac tamponade [5]. This report also clearly shows that any significant trauma to chest should be evaluated completely and monitored eventhough initial examination seem normal.

### Conclusion

Diagnosis of cardiac tamponade is not always very easy.

Cardiac tamponade may take several hours to develop and to cause circulatory failure; therefore any chest trauma must be properly and completely evaluated before coming to any conclusion.

The physical findings of cardiac tamponade are not always apparent despite life-threatening acute cardiac tamponade after blunt trauma.

Focus should always be given to entire vital organs like the heart and the possibility of tamponade must be kept in mind.

Pericardiotomy or pericardiectomy via a thoracotomy or sternotomy is mandatory for life saving cardiac decompression in acute traumatic cardiac tamponade.

A prompt diagnosis using FAST scan and appropriate treatment are lifesaving.

**References**

1. [https://www.med.mun.ca/getdoc/0ff79126-232e-411c-a03c-03b5f52b97b3/Traumatic Cardiac Tamponade.aspx](https://www.med.mun.ca/getdoc/0ff79126-232e-411c-a03c-03b5f52b97b3/Traumatic%20Cardiac%20Tamponade.aspx).
  2. <https://academic.oup.com/bja/article/87/2/309/263254/Delayed-diagnosis-of-cardiac-tamponade>.
  3. <https://emedicine.medscape.com/article/152083-overview>.
  4. <https://emedicine.medscape.com/article/152083-overview#a3>.
  5. <http://medind.nic.in/jbc/t09/i1/jbct09i1p27.pdf>.
- 

**Special Note!**

Please note that our all Customers, Advertisers, Authors, Editorial Board Members and Editor-in-chief are advised to pay any type of charges against Article Processing, Editorial Board Membership Fees, Postage & Handling Charges of author copy, Purchase of Subscription, Single issue Purchase and Advertisement in any Journal directly to Red Flower Publication Pvt. Ltd.

Nobody is authorized to collect the payment on behalf of Red Flower Publication Pvt. Ltd. and company is not responsible of respective services ordered for.

## Torsion of Non-Gravid Uterus with Myoma Presenting to Emergency with Shock

Muhammad Aamir Mir<sup>1</sup>, Kritika Nanda<sup>2</sup>, Kamal Preet Palta<sup>3</sup>, Kishalay Datta<sup>4</sup>

---

### Author's Affiliation:

<sup>1</sup>MEM Resident <sup>2</sup>Attending Consultant <sup>3</sup>Consultant and HOD <sup>4</sup>Associate Director & HOD, Emergency Medicine, Max Smart Super Speciality Hospital, Saket, New Delhi, Delhi 110017, India. <sup>4</sup>Associate Director & HOD, Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

### Corresponding Author:

**Muhammad Aamir Mir**, MEM Resident, Emergency Medicine, Max Smart Super Speciality Hospital, Saket, New Delhi, Delhi 110017, India  
E-mail: [mirammir88@gmail.com](mailto:mirammir88@gmail.com)

Received on 03.10.2017,  
Accepted on 23.10.2017

### Abstract

Abdominal pain is one of most frequently encountered complain in the emergency; poses a diagnostic challenge for the emergency physician as differential diagnosis ranges from benign to life threatening conditions. History, vital signs and physical findings may not point a specific diagnosis and laboratory testing is often not helpful. Especially in females difficulty in physical examination and non-specific clinical picture may lead to delay in diagnosis. Sometimes patients' hemodynamic instability limits radiological intervention. Uterine torsion is a rare condition in the non gravid uterus may cause irreversible ischemic damage to the uterus, leading to rapid clinical deterioration, firstly reported by The Times in 1861 [1]. Here we report a case of a *young non-gravid woman* presenting with acute *abdominal pain* with hemodynamic instability and upon investigation, she was found to have uterine torsion of uterus due to uterine fibroid.

**Keywords:** Torsion; Non Gravid Uterus; Ligaments; Fornix.

---

### Introduction

Uterine torsion is a rare condition in non-gravid uterus. Early diagnosis and high clinical suspicion are keys to prompt identification and definitive surgical treatment of this diagnostic dilemma. Torsion is mainly due to the weakness of supporting ligaments of the uterus, sometimes associated with an intra-abdominal mass diagnosed intraoperatively.

### Case Report

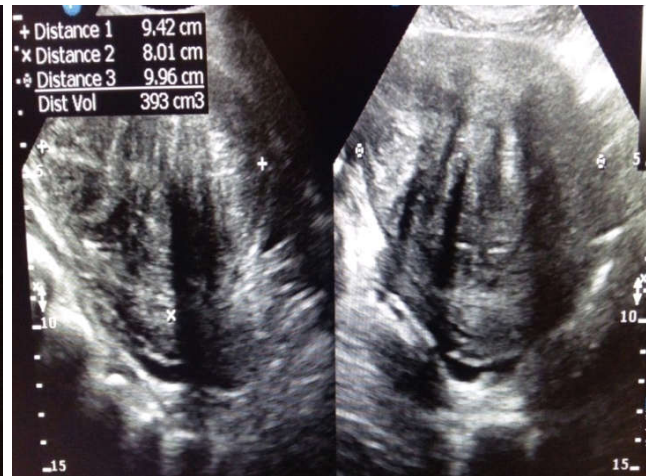
A 27 yr old female presented to emergency with severe abdominal pain, progressive in nature associated with shortness of breath and decreased urine output since 2 days. Patient was conscious, oriented and in severe pain. She was tachycardic, hypotensive (P-102/min, BP- 70/50 mm hg) and maintaining oxygen saturation in room air. Patient denied any history of fever, chest pain, bleeding per

vagina or per rectum, previous surgeries, and any vaginal discharge. During clinical examination of abdomen she had tenderness in lower abdomen with guarding and rigidity, no palpable mass, bowel sound present and on auscultation of chest B/L decreased breath sounds with crepitations. Per vaginum examination was done showed bulky uterus, decreased mobility and tenderness of anterior fornix.

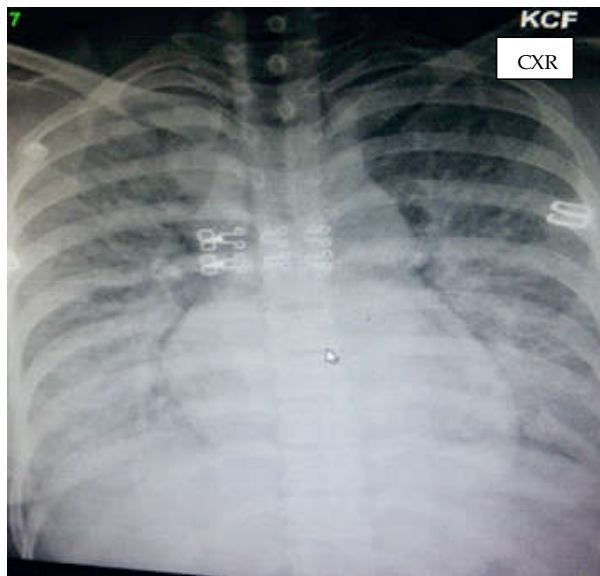
Rest systemic examination were normal. Patient was resuscitated in emergency and ionotropic support started. Her UPT was negative and other laboratory investigations were sent. Abdominal ultrasound and TVS was done which revealed a mass in the right side of tubo-ovarian complex which wasn't clearly delineated.

Patient responded well to the initial treatment and after ensuring hemodynamic stability CT-Scan of abdomen was done showed mild ascites, bulky uterus, B/L plueral effusion and no signs of perforation. The origin of large mass couldn't be delineated.

**USG Abdomen-Pelvis:** Uterus was not clearly visualized. A large mass measuring  $9.7\text{cm} \times 9.8\text{cm}$  anterior to uterus in right adnexal region with specs of vascularity minimal ascites with B/L pleural effusion.



**USG Abdomen-Pelvis:** Uterus was not clearly visualized. A large mass measuring  $9.7\text{cm} \times 9.8\text{cm}$  anterior to uterus in right adnexal region with specs of vascularity minimal ascites with B/L pleural effusion.



**Fig. 2:** Differential diagnoses at this point were Ruptured ectopic pregnancy, Torsion of uterus with mass, Torsion of Ovarian cyst, ARDS, Meig's Syndrome

Patient was managed with I.V fluids, Inotropic support and high end of Antibiotics, Gynaecology and Internal Medicine references were given and shifted to ICU for further intervention. Her CBC, LFT, KFT were normal, B-HCG negative and pleural fluid was negative for malignant cells. Patient's hemodynamic condition improved with support, but continued to have pain. So, decision of Emergency diagnostic laparoscopy was taken which revealed bulky uterus with a large fibroid on anterior surface

## CXR

Differential diagnoses at this point were Ruptured ectopic pregnancy, Torsion of uterus with mass, Torsion of Ovarian cyst, ARDS, Meig's Syndrome.

leading to torsion of the uterus. Tubes and ovaries were normal. Decision of Laparotomy was taken. Detorsion of uterus was done followed by myomectomy. Base of fibroid sutured. Left round ligament plication done to prevent recurrent torsion. Diagnosis of leiomyoma with red degeneration was confirmed by histopathology. Post-operative patient was shifted to ICU, and patient made quick recovery in subsequent days.

## Discussion

Uterine torsion is mainly due to loss of stability of the supported ligaments of uterus, namely broad ligament and the uterosacral ligament by an abdominal mass in most cases. Uterine rotation on its long axis by more than  $45^\circ$  leads to torsion. In our case, the cause of torsion was myoma on one side and the degree of torsion was  $170^\circ$ . This was enough to cause severe pain and ischemic necrosis in short time. Previously uterine torsion in a non-pregnant woman is difficult to diagnose pre-operatively. Nowadays with advancement of radio diagnosis provisional diagnosis can be expected early. Severe abdominal pain with hemodynamic instability prompted for the decision to do a laparotomy. our patient was in reproductive age group and there was no signs of necrosis, so decision of myomectomy was taken. Uterine torsion should be considered as a differential diagnosis in women presenting with acute

abdominal pain and Emergency physician should have high degree of suspicion in all patients with acute pain abdomen to prevent fatal outcome.

### References

1. Omurtag K, Session D, Brahma P, Matlack A, Roberts C. Horizontal uterine torsion in the setting of complete cervical and partial vaginal agenesis: a case report. *FertilSteril* 2009;91(5):1957.e13-1957.e15.
  2. Grover S, Sharma Y, Mittal S. Uterine torsion: a missed diagnosis in young girls? *J PediatrAdolesc Gynecol* 2009;22(1):e5-e8.
  3. Jeong YY, Kang HK, Park JG, Choi HS. CT features of uterine torsion. *EurRadiol* 2003;13(Suppl 6):L249-L250.
  4. Hawes CH. Acute axial torsion of the uterus. *Ann Surg* 1935;102(1):37-40.
  5. Nicholson WK, Coulson CC, McCoy MC, Semelka RC. Pelvic magnetic resonance imaging in the evaluation of uterine torsion. *ObstetGynecol* 1995;85(5 pt 2):888-890.
  6. P. Gule, R. Adjobi, E. Nguessan et al., "Uterine torsion with maternal death: our experience and literature review," *Clinical and Experimental Obstetrics and Gynecology*, 2005;32(4):245-246.
-



# A Rare Case of Complicated Neuroleptic Malignant Syndrome with Rhabdomyolysis and Acute Kidney Injury

Sarat Kumar Naidu<sup>1</sup>, Gurjit Kaur<sup>2</sup>, Vikram Shah<sup>1</sup>, Kishalay Datta<sup>3</sup>

## Author's Affiliation:

<sup>1</sup>DNB Resident <sup>2</sup>MEM Resident  
<sup>3</sup>HOD and Associate Director,  
Department of Emergency  
Medicine, Max Hospital, Shalimar  
Bagh,  
New Delhi, Delhi 110088, India.

## Corresponding Author:

**Sarat Kumar Naidu**  
DNB Resident, Department of  
Emergency Medicine  
Max Hospital, Shalimar Bagh,  
New Delhi, Delhi 110088, India.  
E-mail: [saratnaidu@gmail.com](mailto:saratnaidu@gmail.com)

Received on 09.10.2017,

Accepted on 23.10.2017

## Abstract

A 38 years old female, a known case of MDP/Schizophrenia, was brought to ED with 5 days h/o high fever, tightness of whole body, altered mental status, reduced urine output, inability to eat and speak, following an intake of an atypical antipsychotic Amisulpiride 100mg over period of 2-3 days prior to symptoms. With the history, physical examination and investigations, a diagnosis of neuroleptic malignant syndrome (NMS) with rhabdomyolysis and acute kidney injury (AKI) was made and supportive treatment started with hydration, dopamine agonism, anticholinergic drugs and urine alkalinization. She started improving after 1 week of aggressive treatment and was discharged in stable condition after 3 weeks.

**Keywords:** Neuroleptic Malignant Syndrome; Manic Depressive Psychosis; MDP; Schizophrenia; Muscle Rigidity; Rhabdomyolysis; Acute Kidney Injury; Kidney Failure; Amisulpiride; Antipsychotic; Neuroleptic; Idiosyncratic; Dopamine; Dopaminergic; Prolonged QTc; Urine Alkalinization; Creatinine Phosphor Kinase; CPK.

## Introduction

Neuroleptic Malignant Syndrome is a life-threatening idiosyncratic reaction to neuroleptic antipsychotic drugs [2] like typical antipsychotics such as chlorpromazine, haloperidol and atypical antipsychotics such as olanzapine, risperidone, aripiprazole, amisulpiride.

The reported incidence of NMS is around 0.02-3.0% in patients taking antipsychotic medications [4].

This is characterized by high fever, altered mental status, muscle rigidity, autonomic instability which typically occurs shortly after starting of neuroleptic drugs or alteration of these medications.

There is more risk with typical antipsychotics than with atypical antipsychotics.

This can also develop when dopaminergic drugs like levodopa is abruptly reduced or stopped [3].

Drugs with anti-dopaminergic activity like metoclopramide can also induce NMS.

In short, NMS occurs with reduced dopaminergic activity, either from withdrawal of dopaminergic drugs or from blockade of dopaminergic receptors.

Neuroleptic drugs or antipsychotic drugs are commonly used for schizophrenia and Manic Depressive Psychosis (MDP).

Dopamine, a neurotransmitter responsible for mood cycling, is found to be high during manic episode of MDP and psychosis.

The neuroleptic drugs act by blocking dopaminergic D2 receptors in hypothalamus, nigrostriatal pathways, spinal cord.

If the D2 receptor antagonism is in excess, as compared to dopamine activity, NMS can develop.

Hypothalamic D2 receptor antagonism results in elevated temperature set point which leads to hyperthermia and alteration of heat-dissipating mechanisms like sweating, cutaneous vasodilatation [8].

Nigrostriatal D2 receptor blockade results in muscular rigidity.



Spinal cord D2 receptor antagonism leads to muscle rigidity and tremors via extrapyramidal pathways.

The usual onset of symptoms of NMS is after 4-14 days, majority of cases occur within 10 days after initiation of the neuroleptic drugs. However NMS may occur even after months of the therapy.

Once symptoms start, they progress very rapidly and reaches its peak as early as 3-4 days [1].

In severe cases, NMS can be complicated by rhabdomyolysis, hyperkalemia, kidney failure and seizures [2] after which prognosis becomes very poor.

No single test is confirmatory for NMS.

This is diagnosed clinically which requires high degree of suspicion.

Treatment is mainly supportive and to prevent complications like rhabdomyolysis and renal failure.

Once complications develop, there is higher risks of mortality.

That is why it is of utmost importance to diagnose it early before the complications develop.

### Case Study

A 38 years old female who was a known case of schizophrenia and MDP presented to ED with high fever, tightness of whole body, altered mental status, reduced urine output, inability to eat and speak since 5 days with progressively worsening symptoms.

Her attendants gave a h/o new drug intake called Amisulpiride since 2-3 days for her MDP.

She did not have cough, vomiting, altered bowel movement, abdominal pain, seizures, LOC.

She did not have h/o any other drug intake.

Physical examination, revealed she was drowsy and occasionally responding to verbal commands with vacant stare.

She was immediately taken to monitored bed and vitals taken.

Her pulse rate was 132/min, regular; her BP was 100/60 mmHg and was tachypneic with RR 30/min and her body temperature was 103 degree F.

Her oxygen saturation was 80% at room air and random blood sugar was 220 mg/dl.

She was started on oxygen @10LPM via facemask after which saturation improved to 96%.

Her ECG showed sinus tachycardia with prolonged QTc.

Her neurological examination revealed that she was stuporous, very occasionally responding to verbal commands, increased muscle tone, brisk DTR, occasionally responding to painful stimulus and B/L plantars flexors.

Her respiratory, cardiovascular and per abdominal systemic examinations were within normal limits.

Arterial blood gas analysis showed pH = 7.40, PO<sub>2</sub> = 56mmHg, PCO<sub>2</sub> = 27.5 mmHg, Lactate = 2.8mmol/L, Na = 162mmol/L, K = 3.5mmol/L, Ca = 0.97.

Chest X-ray showed right lower lobe consolidation.

She was given IV paracetamol 1gm, IV normal saline 2L, IV Rabeprazole 20mg, IV Ondansetron 8mg.

Foley's catheter was inserted for urine output monitoring and urine was found to be very dark in colour and her urine dipstick showed blood +++, protein ++, specific gravity 1.030.

In view of above findings, IV fluids were started with Dextrose 10% 500ml +Sodabcarb 8.4% 200ml @ 150ml/hr to alkalinize the urine.

Ryle's tube was inserted and oral medications were given through RT. She was also started on Bromocriptine 5mg IV stat and 2.5mg PO TDS and Trihexyphenidyl 2mg PO TDS.

She was also started on antibiotics Tazact 1.125gm (piperacillin+tazobactam) in view of her pneumonia and later clarithromycin.

Neurology, Psychiatry, Pulmonology and Nephrology consultations were requested and the patient was shifted to ICU after 3 hrs of aggressive management in the ER.

### Course in the Hospital and Outcome

The diagnosis of complicated Neuroleptic Malignant Syndrome with rhabdomyolysis and acute kidney injury was made.

As per Hynes and Vickar [4] scoring system, she could be classified severe NMS.

With aggressive treatment with IV fluids, urine alkalization, paracetamol and trihexyphenidyl, bromocriptine to restore the dopaminergic tone and other supportive treatment, patient started improving after 24 hrs though gradually.

Her MRI brain plain showed no significant abnormality.

Her blood reports showed very high CPK levels of 11099 U/L, urea = 92, creatinine = 2.47, mildly raised liver enzymes.

Her kidney functions improved gradually and she started responding to verbal commands after 7-10 days.

Her body temperature and muscle rigidity improved gradually after 24-48 hrs of treatment.

Her CPK levels reduced from 11099 to 6700 to 3790 to 1345 to 941 to 279 over a period of 1 week since admission.

She was started on high protein diet parenterally and on active and passive physiotherapy.

Her sensorium started improving gradually after 10 days of admission.

Subsequently she was shifted to ward after 10 days of ICU stay and was discharged in stable condition after 2 weeks of hospitalization with advice to take Tab Amantadine 100mg BDX1 week, Tab Trihexyphenidyl 2mg OD X 3 days, Tab Valproate 200mg BD, Tab Cefixime 200mg BDX5 days.

She was followed up after 1 week of discharge and was found to be stable with normal mentation.

### Discussion and Therapeutic Considerations

This case report illustrates 38 yrs old female with complicated NMS induced by Amisulpiride, an atypical antipsychotic medication.

As discussed above, NMS is more common with traditional antipsychotics and much less common with newer atypical antipsychotics like Amisulpiride.

Amisulpiride [5] acts by reducing signaling via dopamine D2 receptors by blocking the pre-synaptic D2 receptors. These presynaptic receptors regulate the release of dopamine into the synapse; so by blocking them, amisulpiride increases the dopamine concentration in the synapse. The increased dopamine in the synapse then acts on D1 receptors to control the depressive symptoms and the negative symptoms of schizophrenia.

However in some patients, reduced dopamine activity can lead to NMS as seen in our patient.

The mainstay of treatment is to stop the offending drug.

Bromocriptine [6] is a potent agonist at D2 receptors which counteracts the action of antipsychotic Amisulpiride.

When Bromocriptine and other supportive measures were started in our patient, she showed good and gradual improvement.

Trihexyphenidyl [7] is a synthetic antispasmodic which exerts direct inhibitory effect on parasympathetic nervous system and also exert relaxing effect on smooth muscles.

It was already late when she presented to our ED as she already had developed complications like rhabdomyolysis and kidney failure.

However with aggressive treatment, she improved and was discharged in a stable condition.

Diagnosis requires a high degree of suspicion with proper history and examination and correlating with laboratory parameters.

### Conclusion

NMS when sets in, progresses very rapidly and reaches its peak in 2-3 days. Complications can develop within 1 week if not treated aggressively.

It is therefore very important to diagnose it early and reverse the disease process and to prevent its complications.

Although the usual onset of NMS is between 4-14 days but it can occur within 2-3 days of the initiation of neuroleptic medications as seen in our case.

Moreover small doses of neuroleptics can also cause NMS as in our case who ingested only around 100mg of amisulpiride over 2-3 days period.

In spite of lower risk with atypical antipsychotics, life-threatening NMS can still develop and therefore patient education is of utmost importance to those who are taking antipsychotic medications. Emergency physicians and General physicians where the patient usually presents, must be made aware of signs and symptoms and the management of NMS.

### References

1. Strawn JR, Keck PE, Caroff SN. "Neuroleptic malignant syndrome". *The American Journal of Psychiatry*. 2007;164(6):8706. PMID 17541044. doi:10.1176/ajp.2007.164.6.870.
2. Berman, BD. "Neuroleptic malignant syndrome: a review for neurohospitalists." *The Neurohospitalist*. 2011 Jan;1(1):417. PMID 23983836. doi:10.1177/1941875210386491.
3. Keyser DL, Rodnitzky RL. "Neuroleptic malignant syndrome in Parkinson's disease after withdrawal or alteration of dopaminergic therapy". *Archives of Internal Medicine*. 1991;151(4):794-6. PMID 1672810. doi:10.1001/archinte.151.4.794.

4. Daniel Molina. Aripiprazole as the Causative Agent of Neuroleptic Malignant Syndrome: A Case Report. Prim Care Companion J Clin Psychiatry. 2007;9(2): 148-150.
  5. <https://en.wikipedia.org/wiki/Amisulpride>
  6. <https://en.wikipedia.org/wiki/Bromocriptine>
  7. <https://en.wikipedia.org/wiki/Trihexyphenidyl>
  8. <http://emedicine.medscape.com/article/816018-treatment>
-

## Life Threatening Rhabdomyolysis, A Rare and Unusual Presentation with Rosuvastatin Ingestion

Umran Rafeeq Sheikh<sup>1</sup>, Kishalay Datta<sup>2</sup>, Priya Govil<sup>3</sup>, Deepika Mittal<sup>1</sup>

### Author's Affiliation:

<sup>1</sup>Masters in Emergency Medicine, PGY-3, <sup>3</sup>Associate Consultant, Department of Emergency Medicine, Max Healthcare, Saket, New Delhi, Delhi 110017, India.

<sup>2</sup>HOD, Associate Director, Department of Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

### Corresponding Author:

**Umran Rafeeq Sheikh**, Masters in Emergency Medicine, PGY-3, Max Healthcare, Saket, New Delhi, Delhi 110017, India.

E-mail: [documran@gmail.com](mailto:documran@gmail.com)

Received on 25.05.2017,

Accepted on 13.06.2017

### Abstract

Rhabdomyolysis is the breakdown of skeletal muscle which is found commonly associated with crush injuries, compartment syndromes, strenuous exercise and drug abuse but rarely found due to consumption of medications like statins. Here we present a case of a 62 year old male who had presented to the emergency room with paraplegic, myalgia and hyperkalemia after about a month of being started on statins. Further clinical and laboratory evaluation were suggestive of a diagnosis of statin induced rhabdomyolysis causing acute renal failure and hyperkalemia. Awareness about the adverse effects of individual statins may help develop a clinical suspicion of rhabdomyolysis among the Emergency physician and also help other physicians make better decisions in the choice of statin use and promote regular monitoring of CPK levels in preventing incidences of rhabdomyolysis.

**Keywords:** Rhabdomyolysis; Statins; Acute Renal Failure; Hyperkalemia; Paraplegia; Myalgia.

### Introduction

Rhabdomyolysis associated with the use of statins has been demonstrated to be a rare but potentially life-threatening adverse effect of statins. The incidence of rhabdomyolysis has been 1.6 per 100,000 person-years [1]; the US FDA Adverse Event Reporting System database has reported the rates of statin-induced rhabdomyolysis of 0.3–13.5 cases per 1,000,000 statin prescriptions [2]. Among the patients with rhabdomyolysis, 10–40% have been estimated to develop ARF [3]. Here, we report a rare case of rhabdomyolysis in a patient who had started using rosuvastatin and developed acute renal failure (ARF) and hyperkalemia which necessitated the initiation of dialysis.

### Case Report

A 62-year-old male, brought with history of progressive bilateral lower limb weakness with muscle pain since 4 days along with burning

micturation since 3 days and decreased urine output. He had no history suggestive of trauma, fever, immobilisation, seizures.

On primary survey; his Airway was patent; Breathing, the respiratory rate was 16/min with a saturation of 98% on room air; Circulation, heart rate was 98/min with a blood pressure reading of 130/70mmHg, Peripheral pulses felt regular and bilaterally equal and a capillary refill time of less than 3 seconds. Disability, the patient was drowsy but responding to verbal commands, moving all four limbs with a GRBS of 220mg/dL. Icterus was seen.

On secondary survey; there were features suggestive of Pallor, Icterus, Cyanosis, or dehydration. Chest had equal air entry bilaterally with no adventitious sounds, heart sounds S1S2 heard with no murmurs and a normal JVP; Abdomen was soft, non-tender with no organomegaly.

Central nervous system examination, he was Conscious and Oriented. But Motor examination revealed a power of 4/5 in both upper limbs and 2/5 in both the lower limbs. Weakness more marked in proximal muscles. No sensory deficit could be

elicited. Deep tendon reflexes were normal and plantars were flexor. Bilateral Pedal oedema was seen.

He was a known diabetic and coronary artery disease had undergone percutaneous coronary angioplasty about a month prior to presentation. His medication history revealed he had been on oral hypoglycemic agents from a long time and that he had been recently since a month been started on Aspirin 75mg and rosuvastatin 40mg once a day.

Among the Point of Care Investigations Done in the Emergency; ECG was suggestive of Global Broad Complex QRS with tented tall T waves. Arterial blood gas revealed partially compensated severe metabolic acidosis, Serum Lactate of 0.8mmol/L, Serum Sodium of 119mmol/L and Serum Potassium of 7.8mmol/L. Urine dipstick done revealed blood +++, proteins +.

Following this he was managed with appropriate anti-hyperkalemic measures and shifted for urgent haemodialysis.

Haemogram - Haemoglobin was 12g/dL, TLC of 10,400/mm<sup>3</sup>, Platelets 200,000/mm<sup>3</sup>; Renal profile - S.Urea 270mg/dL, S.Creatinine 7.24mg/dL, S. Sodium 134mEq/L, S. Potassium 4.4mEq/L S. Chloride 94mEq/L

Liver function tests - S.Albumin 3.4g/dL, S. Globulin 2g/dL, Total bilirubin 0.7mg/dL, unconjugated bilirubin 0.3mg/dL, Alkaline phosphatase 120U/L, SGOT 31 IU/L and SGPT 40IU/L Serum LDH of 2040 U/L and a S. CPK of 74, 500 U/L.

A collaboration of clinical and lab findings lead us to a diagnosis of statin induced rhabdomyolysis leading to acute renal failure and hyperkalemia. Immediate hemodialysis and withdrawal from statins, was the last resort to provide relief in clinical symptoms and decrease CPK levels.

## Discussion

Statins have been used for the prevention and treatment of cardiovascular disease. The treatment is quite safe but not free of side effects. Adverse effects on muscles occur in approximately 5 to 10% of patients taking statins which are usually mild and disappear upon discontinuation of the medication [4].

Rarely, the creatine phosphokinase (CPK) enzyme level may increase to exceptional values (10 times the upper normal level) and rhabdomyolysis is extremely rare. A few of the factors that may increase the risk of myopathy among statin users are; Elderly, Female sex, Multi-systemic diseases, Frailty, small

body frame, Multiple medications, Perioperative period, Concomitant use of drugs (such as Fibrate, Nicotinic acid/ Cyclosporine, Azole antifungal, Macrolide antibiotic, Erythromycin and Clarithromycin, HIV protease inhibitors, Verapamil, Warfarin, Digoxin, Alcohol).

Rhabdomyolysis has been seen to present with myalgias, weakness, fatigue, and dark coloured urine, which usually develop within a few days of starting the treatment [5]. It is common to see muscular and renal adverse effects in association with statins as seen in our patient. Among these; muscular adverse effects like myopathy, rhabdomyolysis and increase in CPK levels have been more strongly associated with rosuvastatin use and; acute renal failure seen to be more strongly associated with atorvastatin use [6].

For patients being managed solely with statin drugs, the incidence of muscular adverse effects has been reported as 0.1% to 0.2% [7]. However, the incidence increases to 1% to 7% for patients taking multiple medications and those with multiple risk factors for developing adverse events [7]. With the growing number of drug permutations and combinations, great deal of suspicion and awareness is required among the ER physicians. Current recommendation are to obtain a prior baseline CK level of patients with increased risk of musculoskeletal disorders and routine monitoring only for those who experience muscle pain or weakness [8].

Knowledge about adverse effects of individual statin may lead to change in choice of statin use and regular monitoring of CPK levels at the primary stage of initiation.

## Conclusion

The clinical manifestations of rhabdomyolysis associated with statin use are varied and Rhabdomyolysis associated with rosuvastatin monotherapy is extremely rare and may result in potentially fatal myoglobinuria with acute renal failure. In similar ED presentations, diagnosis of statin induced rhabdomyolysis by ER physician would require vigilance to help improve the outcome. Diagnosis requires a high degree of clinical suspicion.

A large number of patients developing such adverse effects are unaware and go undiagnosed and untreated. Therefore, further research needs to be

directed as to what drug levels would guide the dosing, frequency and stopping & changing over to different drug; how frequently should the drug levels be monitored and as to what drug dosage & duration of treatment would cause these derangement. Although statins provide medical benefits, they should always be prescribed with caution and attention directed towards appropriate dosage adjustments with minimal side effects.

### References

1. Law M et al. Statin safety: a systematic review. *Am J Cardiol.* 2006 Apr 17;97(8A):52C-60C.
  2. Davidson MH et al. Statin safety: an appraisal from the adverse event reporting system. *Am J Cardiol.* 2006 Apr 17;97(8A):32C-43C.
  3. Kasaoka S et al. Peak value of blood myoglobin predicts acute renal failure induced by rhabdomyolysis. *J Crit Care.* 2010 Dec;25(4):601-4.
  4. Pohjola SS et al. Muscle-related adverse effects of statins. *Duodecim.* 2014;130:1622-7
  5. Torres PA et al. Rhabdomyolysis: Pathogenesis, Diagnosis, and Treatment. *The Ochsner Journal*, 2015; 15(1):58-69.
  6. Sakaeda T et al. Statin-Associated Muscular and Renal Adverse Events: Data Mining of the Public Version of the FDA Adverse Event Reporting System. *PLoS ONE* 2011;6(12):e28124.
  7. Tomlinson SS et al. Potential adverse effects of statins on muscle. *Phys Ther.* 2005;85:459.
  8. Mlodinow SG et al. Statin adverse effects: Sorting out the evidence. *The Journal of Family Practice.* 2014 Sep;63(9):497.
  9. Kato K et al. Pravastatin-induced rhabdomyolysis and purpura fulminans in a patient with chronic renal failure. *International Journal of Surgery Case Reports* 2015;8:84.
  10. Stasi S L D et al. Effects of Statins on Skeletal Muscle: A Perspective for Physical Therapists. *Phys Ther.* 2010 Oct;90(10):1530.
-

## Beyond ACLS Protocol – A Rare Case of Refractory Supraventricular Tachycardia Responding Only to a Much Higher Dose of Adenosine

Sarat Kumar Naidu<sup>1</sup>, Hilal Yatoo<sup>2</sup>, Kishalay Datta<sup>3</sup>

**Author's Affiliation:**  
<sup>1</sup>DNB Resident <sup>2</sup>Attending Consultant <sup>3</sup>HOD and Associate Director, Department of Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

**Corresponding Author:**  
**Sarat Kumar Naidu**  
DNB Resident, Department of Emergency Medicine  
Max Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.  
E-mail: [saratnaidu@gmail.com](mailto:saratnaidu@gmail.com)

**Received on** 10.10.2017,  
**Accepted on** 30.10.2017

### Abstract

Symptomatic supraventricular tachycardia (SVT) is a common presentation in the Emergency Department which can be a life-threatening condition and this requires immediate intervention. Stable SVTs are commonly treated with Adenosine but adenosine is more effective in AV node-dependant SVTs as it causes transient AV block [1] and causes normalization of the cardiac rhythm. American Heart Association AHA's ACLS protocol recommends 6mg then 12mg (total 18mg) dose for SVTs [2]. However there is limited data as to how much maximum dose can be given for those SVTs that fail to terminate with standard dosing schedule [1]. This case report describes a 30 years old female with symptomatic SVT which failed to revert with normal dosing of adenosine neither with electrical cardioversion nor with multiple antiarrhythmic drugs but responded only to a much higher dose of adenosine.

**Keywords:** Supraventricular Tachycardia; AVRT (Atrioventricular Re-Entry Tachycardia); AVNRT (Atrioventricular Reentrant Tachycardia); Adenosine; Cardioversion; ACLS (Advanced Cardiac Life Support); AV (Atrioventricular) Block; AHA (American Heart Association); ED (Emergency Department); Refractory; Ursodeoxycholic Acid; WPW Syndrome (Wolf Parkinson White).

### Introduction

Supraventricular tachycardias (SVTs) are tachyarrhythmias arising from above the level of Bundle of His. It may be from atria or from AV node and may be regular or irregular.

It is caused by re-entry phenomenon causing increased heart rate and is less likely to be caused by

structurally abnormal heart. The heart rate in SVT is usually around 150-250 beats/min and regular in rhythm. Symptoms of SVT include palpitations, chest discomfort or pain, shortness of breath, lightheadedness, dizziness, nausea and vomiting.

Increased heart rate is frightening to the patient if persisting or recurrent and may cause significant morbidity.

*SVTs May be Broadly Classified as Follows*

Site of Origin or Propagation	Regular	Irregular
<b>Atria</b>	Atrial tachycardia Atrial flutter	Atrial fibrillation Multifocal atrial tachycardia
<b>AV node</b>	Sinus node reentrant tachycardia AVNRT AVRT	Atrial flutter with variable AV block

*Common Types of SVTs can be Classified as Follows*

Types	Features
AVNRT <sup>(5)</sup>	Most common type of SVT about 50-60% <sup>(4)</sup> Seen in young females Reentry caused by nodal pathways or tracts HR is 118-264/min <sup>(5)</sup> . Narrow complex (QRS<120sec) 2 types: Typical (slow/fast) 90% of all AVNRTs RP interval <PR interval Pseudo R wave in V <sub>1</sub> Pseudo S wave in I, II, aVf Atypical (fast/slow) 10%
AVRT <sup>(5)</sup>	Second most common SVT about 30% cases Seen in younger women and children Reentry caused by accessory pathways HR is 124 – 256/min <sup>(5)</sup> . Narrow complex (QRS<120sec) 2 types: Orthodromic – antegrade conduction through AV node Antidromic – retrograde conduction through AV node Most commonly associated with WPW syndrome Delta waves in ECG
<b>Atrial tachycardia Or Multifocal atrial tachycardia (MAT)</b>	3 <sup>rd</sup> most common SVT about 10% cases Seen in middle aged Associated with heart failure or COPD In MAT, 3 successive P waves have different morphology

The commonest cause of palpitations in a normal structured heart is AVNRT [3].

Diagnosis is often delayed or misdiagnosed as panic disorder or anxiety disorder.

About 25% of SVTs get reverted with vagal maneuvers like valsalva maneuver or carotid sinus massage [2]. The remainder may require adenosine or electrical cardioversion.

Adenosine [6] is a naturally occurring purine nucleoside and is a short acting antiarrhythmic drug with onset of action 10 seconds and duration of action 10 seconds.

It causes depression of SA node and AV nodal activity and antagonizes c-AMP-mediated catecholamine stimulation of ventricular muscle thereby causing negative chronotropy and negative dromotropy [6].

Adenosine is contraindicated in 2<sup>nd</sup> and 3<sup>rd</sup> degree AV blocks and WPW syndrome (Wolf Parkinson White) and must be used with care in asthma and COPD.

Higher dose may be required in caffeine and theophylline users and lower dose (3mg) may be required in carbamazepine and dipyrindamole users [2].

**Case Study**

A 30 years old female presented to ED with c/o palpitations since 4 hours associated with nausea and light headedness. She did not have any chest pain, vomiting, syncope, near syncope, loss of consciousness, or cough.

She had similar episode 1 year back when her local physician gave tablet verapamil 120mg which relieved her symptoms and did not recur again. She did not undergo any further evaluation during that episode but has been on verapamil 120mg OD since then.

She took additional dose of verapamil 120mg before coming to the ED but symptoms did not subside.

She was taken to the monitored bed and initial evaluation done.

She was conscious oriented but was tachycardic with PR = 208/min regular and tachypneic with RR = 24/min.

Her oxygen saturation was 98% at room air.

Her BP was 120/80 mmHg and random blood sugar level was 94mg/dl.

She did not have any pallor, cyanosis, icterus,

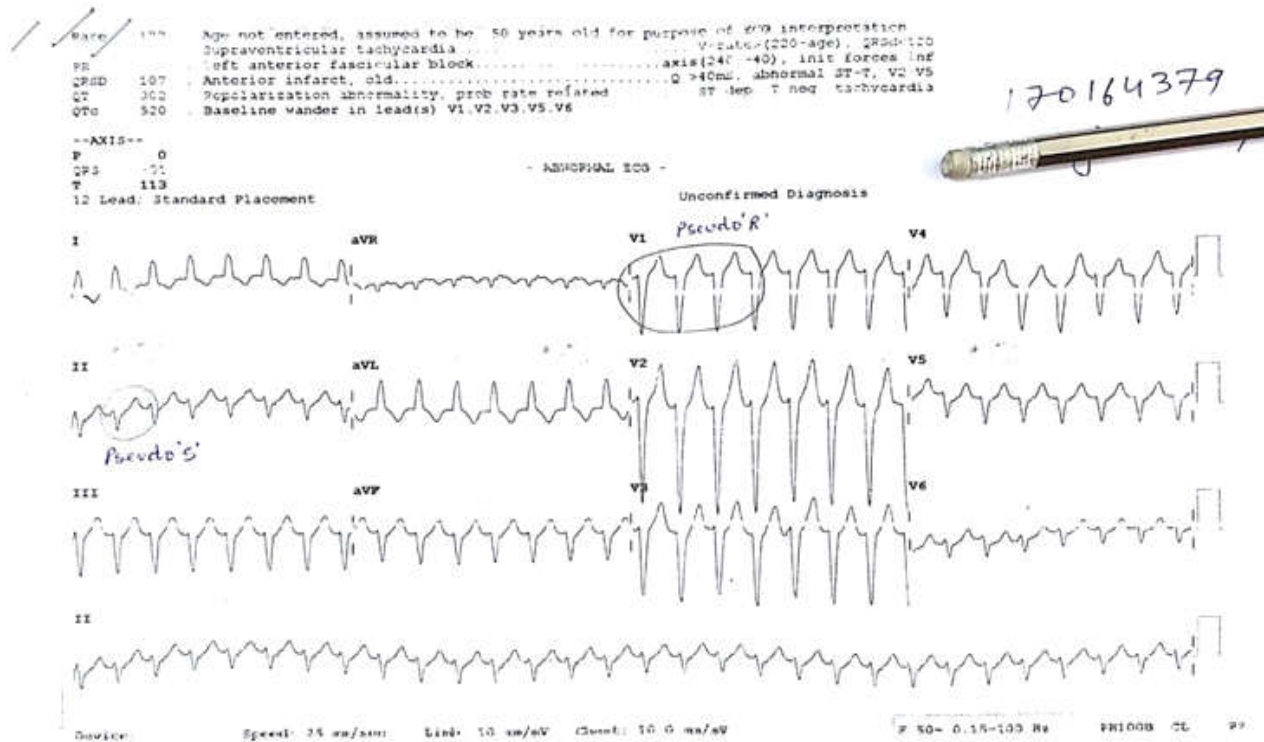


jugular venous distension nor peripheral edema.

Cardiac monitor showed narrow complex tachycardia and 12-lead ECG showed SVT with

pseudo R in V<sub>1</sub> and pseudo S in lead II which most likely was atrioventricular nodal reentrant tachycardia (AVNRT).

ECG of the Patient is Shown below.



A large IV canula was inserted in left cubital vein and samples taken for ABG, CBC, KFT, LFT, thyroid profile, serum calcium and magnesium.

Her systemic examination did not reveal anything significant.

After checking for carotid bruit, vagal manoeuvre was done on right side but there was no change in the ECG rhythm.

She was immediately given adenosine 6mg IV stat followed by 20ml NS flush. This did not revert the SVT. Another 12mg of adenosine was pushed through the cubital vein after 3 minutes of the first dose but this also did not revert the SVT.

Cardiologist on-call was immediately called who advised inj diltiazem 15+15 = 30mg IV which also did not revert the SVT.

After the above medications, the patient started feeling dizzy and BP was found to be 60 mmHg systolic.

Since she became unstable with hypotension, electrical cardioversion was done with 50J then with

100J but that too did not revert the SVT.

Senior cardiologist was called-in and inj Amiodarone 150mg given IV over 10 mins but that did not revert the SVT either.

Eventually she regained normal sinus rhythm with another dose of adenosine of 18mg IV after which she remained in normal sinus rhythm and her blood pressure increased to 130/70 mmHg.

Her symptoms of palpitations and lightheadedness also subsided. She did not have any side effects of adenosine like bronchospasm or flushing.

Her VBG report showed pH = 7.312, pO<sub>2</sub> = 32.5, Pco<sub>2</sub> = 52.3, HCO<sub>3</sub> = 25.7, Na<sup>+</sup> = 144, K<sup>+</sup> = 4, Ca<sup>+</sup> = 1.17, lactates = 2.6. She was started on infusion of Amiodarone @ 60mg/hr and was then shifted to CCU (Cardiac Care Unit) for further management.

#### Course in the Hospital and Outcome

A diagnosis of refractory SVT – AVNRT was made and amiodarone infusion was initiated @ 60mg/hour in the ED.

Echocardiography showed EF 64% and no RWMA. CXR showed no abnormality.

She was started on Diltiazem 30mg PO BD, Amiodarone 200mg BD then OD, Ursodeoxycholic acid 300mg TDS.

Her blood investigations showed normal Complete blood count, normal electrolytes, normal kidney function tests, normal liver function tests, normal thyroid function tests.

The patient later underwent radiofrequency ablation of an accessory pathway.

She was discharged in a stable condition after 4 days of hospital stay.

During the entire stay in hospital she did not have any further symptoms nor did she have any arrhythmia on cardiac monitoring.

On further follow up in cardiology OPD after 1 week she was found to be stable.

### Discussion and Therapeutic Considerations

This case report illustrates a 30 years old female who presented with refractory SVT which was not reverting with normal dosing of adenosine (6mg, 12mg) nor with multiple antiarrhythmic drugs like amiodarone, diltiazem nor with electrical cardioversion with maximum energy but her SVT reverted only to a subsequent higher dose of adenosine (18mg), total 36mg.

As discussed above, higher dose may be required in caffeine and theophylline users and lower dose (3mg) may be required in carbamazepine and dipyridamole users [2].

In our case, when asked retrospectively, she mentioned that she had consumed 3-4 cups of coffee that day and was under some stress due to her office work.

This caffeine intake and stress could explain the triggering of SVT and requirement of high dosage of adenosine.

As per the ACLS guidelines [2], when the tachycardia with a pulse is unstable, meaning the presence of any of 1) hypotension SBP<90mmHg, 2) altered mental status, 3) signs of shock, 4) ischemic chest discomfort, 5) acute heart failure, the treatment of choice is electrical cardioversion. Therefore we tried to electrically cardiovert the patient when the BP fell down to 60mmHg but still there was no response.

Antiarrhythmic drugs like Amiodarone and Diltiazem were started to keep her heart rate under control.

Ursodeoxycholic acid was also started in consultation with the Cardiologist as it has shown some anti-arrhythmic properties via preventing ICP-associated cardiac conduction slowing and development of reentrant arrhythmias, although the cellular mechanism is still not clearly known.

### Conclusion and Limitations

About 75% of SVTs can be terminated with pharmacological cardioversion, that is by adenosine and usually it gets reverted by the standard dosing of 6mg and then 12mg as per the ACLS protocol 2015 CPR/ECC guidelines of American Heart Association.

However in some refractory cases, additional doses may be required as seen in our case. Some known causes for this refractoriness is caffeine intake and some drugs like theophylline as discussed above.

Why should an emergency physician be aware of this deviation from the standard dosing of adenosine? This is because there is no confirmed data as to how much dose of adenosine can be given for refractory SVTs and very few cases do respond to a higher dose. This is also evidenced by one study<sup>(1)</sup> by Bailey AM whose study was published in Journal of Emergency Medicine in 2016 where the patient responded to a very high dose of adenosine.

Prior to 2015 ACLS guidelines, the standard dose was 6mg, 12mg, 12mg but in 2015 guidelines, the 3<sup>rd</sup> 12mg dose was removed from SVT management.

Our intention is only to make the emergency physicians aware of situations where the standard dosing of adenosine may not be sufficient for SVT termination. However a bigger study is required to come to any conclusion.

### References

1. Bailey AM, Baum RA, Rose J, Humphries RL. High-Dose Adenosine for Treatment of Refractory Supraventricular Tachycardia in an Emergency Department of an Academic Medical Center: A Case Report and Literature Review. *J Emerg Med.* 2016 Mar;50(3):477-81. doi: 10.1016/j.jemermed.2015.11.012. Epub 2016 Jan 2
2. American Heart Association's ACLS manual 2015 guidelines.

3. <https://lifeinthefastlane.com/ecg-library/svt/>.
  4. Porter MJ, Morton JB, Denman R, et al. Influence of age and gender on the mechanism of supraventricular tachycardia. *Heart Rhythm*. 2004;1(4):393-396.
  5. <http://www.aafp.org/afp/2010/1015/p942.html>.
  6. <https://lifeinthefastlane.com/ccc/adenosine/>.
- 

## Instructions to Authors

Submission to the journal must comply with the Guidelines for Authors.  
Non-compliant submission will be returned to the author for correction.

To access the online submission system and for the most up-to-date version of the Guide for Authors please visit:

<http://www.rfppl.co.in>

Technical problems or general questions on publishing with IJEM are supported by Red Flower Publication Pvt. Ltd's Author Support team ([http://rfppl.co.in/article\\_submission\\_system.php?mid=5#](http://rfppl.co.in/article_submission_system.php?mid=5#))

Alternatively, please contact the Journal's Editorial Office for further assistance.

Editorial Manager  
Red Flower Publication Pvt. Ltd.  
48/41-42, DSIDC, Pocket-II  
Mayur Vihar Phase-I  
Delhi - 110 091(India)  
Phone: 91-11-22754205, 45796900, 22756995, Fax: 91-11-22754205  
E-mail: [author@rfppl.co.in](mailto:author@rfppl.co.in)

# An Unusual Presentation of Fat Embolism Syndrome as Cerebral Fat Embolism in Trauma: A Rare Clinical Entity

Nitish Dhand<sup>1</sup>, Kishalay Datta<sup>2</sup>, Vaibhav Gulati<sup>3</sup>, Indranil Das<sup>4</sup>, E.V. Balasubramanyam<sup>5</sup>, Vikram Shah<sup>6</sup>

---

## Author's Affiliation:

<sup>1</sup>PGY-3, DNB <sup>2</sup>HOD and Associate Director <sup>3</sup>PGY-3, MEM <sup>4</sup>Attending Consultant <sup>5</sup>PGY-2, DNB <sup>6</sup>PGY-2, Secondary DNB, Emergency Medicine, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

## Corresponding Author:

**Vaibhav Gulati,**  
PGY-3, MEM{GWU-USA} Dept of Emergency Medicine, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.  
E-mail:

[dr.vaibhavgulati@gmail.com](mailto:dr.vaibhavgulati@gmail.com)

Received on 11.10.2017,

Accepted on 30.10.2017

## Abstract

Fat embolism syndrome is a rare clinical complication of fat embolism which occurs in almost 90% of long bone fractures. Incidence of FES is around 0.2 to 2.5 % in overall cases of fat embolism. Its diagnosis is mainly clinical characterized by triad of respiratory, dermatological and neurological manifestations. We are presenting a case of 20 year young male who suffered traumatic left femoral shaft fracture in RTA. After uneventful 24 hours patient suddenly developed altered sensorium in absence of any respiratory or dermatological manifestation. He was confirmed to have CFE after series of brain imaging and was then managed conservatively for the same to which he responded well.

**Keywords:** Cerebral Fat Embolism; Fat Embolism Syndrome; Major Trauma; Traumatic Brain Injury.

---

## Introduction

Fat embolism occurs very commonly in patients who have sustained major injuries. Although, its incidence is as high as 90% in such cases but most of them are subclinical. However, rarely it can lead to life threatening complication as "fat embolism syndrome".

FES is characterized by systemic inflammatory cascade affecting multiple organ systems.

Its diagnosis is mainly clinical indicated by development of respiratory distress, petechiae and cognitive dysfunction in first few days following trauma, long bone fractures or medullary surgery.

FES is believed to occur due to a sequence of biochemical reactions resulting from injury sustained in major trauma. Release of fat emboli leads to occlusion of microcirculation, leading to an inflammatory response that is clinically presented by dermatological, pulmonary and neurological dysfunction. Usually initial clinical presentation of every case of FES is pulmonary with symptoms as observed in ARDS typically appearing within 24 hours after the initial injury.

1 out of 5 cases of FES can present with other features along with pulmonary symptoms particularly involving brain and kidney. As a result of cerebral microcirculation occlusion, patient can have gross encephalopathy, localized cerebral edema and white matter changes.

In our case, the patient presented with isolated neurological features making the clinical suspicion of diagnosis of "cerebral fat embolism" less likely at first place.

## Case Report

A 20 years old patient was presented to emergency department after alleged history of road traffic accident at about 2:30 pm in Chandigarh. As per attendants, patient while driving two wheeler was hit by an unknown vehicle from behind. He was wearing helmet at time of injury.

There was no history of loss of consciousness, seizure, ENT bleed, vomiting.

Patient was admitted in government hospital, Chandigarh.

Initial NCCT head was normal, X-ray left thigh showed fracture shaft femur. Initial systemic examination was unremarkable and patient was conscious, oriented with GCS- E4V5M6.

Almost 24 hours after injury patient became irritable and there was deterioration of GCS for which NCCT head was repeated which was again normal.

In view of worsening condition patient was referred for Max Hospital, Shalimar Bagh.

Patient was transported by ambulance with Thomas splint in situ for immobilisation of left femur.

*On Presentation:*

*Primary Survey*

Airway- Patent

Breathing - Respiratory rate- 20/min

Spo2 - 99% on room air

Circulation - Heart rate- 100 bpm

Blood pressure- 130/70 mm of Hg

Peripheral pulses- palpable, good volume, rhythmic.

Disability - GCS- E3V3M6

B/L pupils - Mid dilated with sluggish reaction to light

Exposure- Left thigh swelling present

Left Thomas splint in situ

Pelvis compression - Negative

Log roll - No step deformity, No back and perineal injury.

PR examination -WNL

*Secondary Survey*

HEENT: No external head/neck/face injury. No Cervical tenderness present.

RS: Trachea midline, No distended neck veins.

B/L air entry equal, no added sounds.

No palpable crepitus.

CVS: S1,S2 heart sounds normally heard.

P/A: No visible bruise, abdomen soft, Non tender, bowel sounds normally heard.

No external genitalia injury.

CNS - Irritable, confused, bilateral plantar extensors.

Extremities Multiple abrasions in lateral aspect of left thigh.

Multiple linear abrasions in right flank region.

A 2 cm linear abrasion over right foot

A 3x3 cm bruise over medial aspect of left thigh.

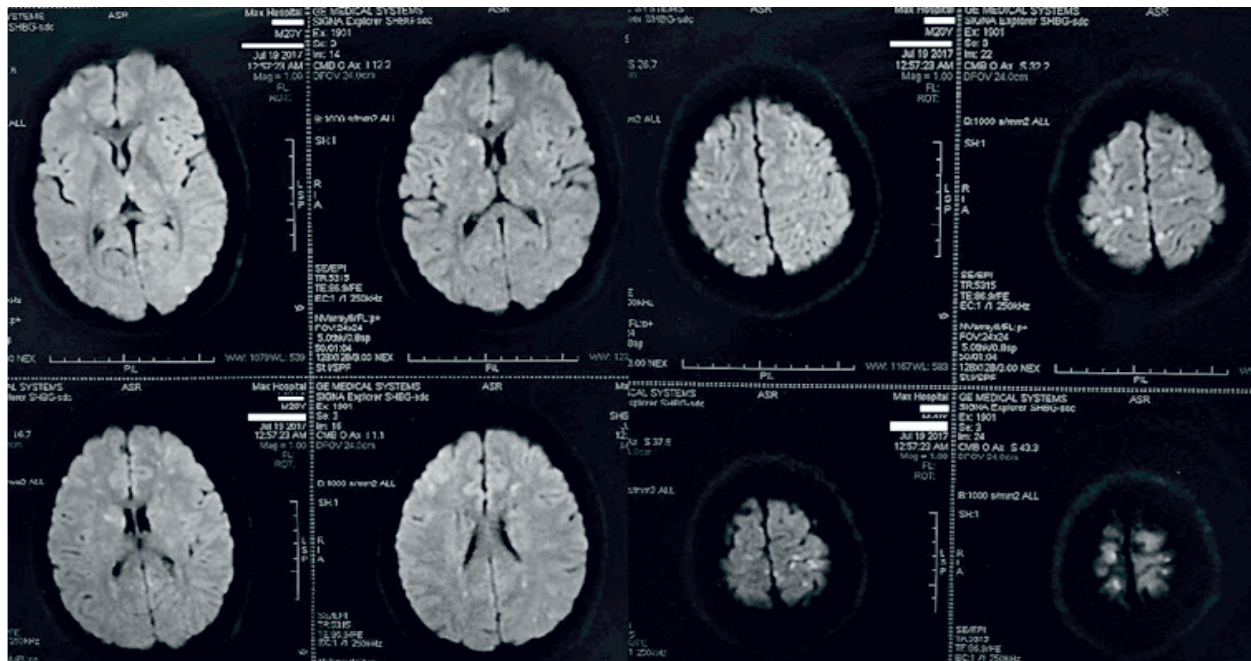


Fig. 1:

*AMPLE*

Allergies: No known allergies.

Medication: Not on any medications.

Past medical history: No significant past medical history.

Events leading to incident: As described above.

After examination, primary treatment was done and in suspicion of any intracranial pathology patient was immediately shifted to radiology department for brain imaging.

*On Investigations*

MRI brain revealed multiple small dot like lesion in cortico-subcortical junction and B/L basal ganglia suggestive of cerebral fat embolism.

In view of above findings immediate neurology consultation was taken and patient was admitted in ICU under combined care of neurology, orthopaedics and cardiology team. Patient was started on conservative treatment for cerebral fat embolism to which patient responding well.

**Conclusion**

Fat embolism syndrome is a very rare complication of fat embolism which can even present with isolated neurological finding as cerebral fat embolism in absence of any classical pulmonary and dermatological findings.

So we as emergency physicians should have high suspicion to diagnose it at earliest with help of clinical

features supported by investigations and to initiate appropriate therapy at earliest because in case of delayed diagnosis it can lead to poor prognosis and even death.

**References**

1. Gupta B, Kaur M, D'souza N, Dey CK, Shende S, Kumar A, Gamangatti S. Cerebral fat embolism: A diagnostic challenge. *Saudi J Anaesth* 2011;5:348-52.
2. Rohit Sharma and Dr Yuranga Weerakkody et al. Cerebral fat embolism. <https://radiopaedia.org/articles/cerebral-fat-embolism>.
3. Yihua Zhou, Ying Yuan, Chahua Huang, Lihua Hu, Xiaoshu Cheng. Pathogenesis, diagnosis and treatment of cerebral fat embolism. *Chinese Journal of Traumatology* 2015 April;18(2):120-123.
4. Christina Mijalski, Alexandra Lovett, Rahul Mahajan, Sophia Sundararajan, Scott Silverman, Steven Feske. Cerebral Fat Embolism-A Case of Rapid Onset Coma. *Stroke*. 2015; STROKEAHA.115.011440.
5. Naila Goenka, and Allan H. Ropper. Cerebral Fat Embolism. *N Engl J Med* 2012;367:1045.
6. Leonidas Gregorakos, Katerina Sakayianni, Despina Hroni, Victoria Harizopoulou, Nikos Markou, Fani Georgiadou, Maria Adamidou. Prolonged coma due to cerebral fat embolism: report of two cases. *emj* 2017(2).
7. Andrew D. Simon, John L. Ulmer and James M. Strottmann. Contrast-Enhanced MR Imaging of Cerebral Fat Embolism: Case Report and Review of the Literature. *American Journal of Neuroradiology* January 2003;24(1):97-101.

## Bilateral Acute Lower Limb Arterial Occlusion after Long Term Tranexamic Acid Usage

P. Anvesh<sup>1</sup>, A.V. Venugopal<sup>2</sup>, Harini Agnes<sup>1</sup>, Siddardh<sup>3</sup>

---

### Author's Affiliation:

<sup>1</sup>Resident, Department of Emergency Medicine <sup>2</sup>Consultant Department of Nephrology <sup>3</sup>Consultant, Department of Plastic Surgery, Care Hospitals, Ramnagar, Visakhapatnam, Andhra Pradesh 530002, India

### Corresponding Author:

**Venugopal Anumanchipalli**, Consultant, Department of Nephrology, Care Hospitals, Ramnagar, Visakhapatnam, Ram Nagar, Visakhapatnam, Andhra Pradesh 530002, India.

E-mail:

[drvenuvenkat@yahoo.com](mailto:drvenuvenkat@yahoo.com)

Received on 23.09.2017,

Accepted on 13.10.2017

### Abstract

Tranexamic acid is widely used as an anti-fibrinolytic agent in different conditions including menstrual bleeding, trauma, dental procedures etc. though considered safe, its use on a long term basis in an abnormal dose is not without adverse effects. Both arterial and venous thrombosis in different vascular beds has been described with its use. Here in, we report a rare case of bilateral acute lower limb arterial thrombosis in a young female who had used tranexamic acid inadvertently for a long period of time. Our patient presented with sudden onset of weakness of both lower limbs and progressively worsening blackish discoloration ascending from foot to mid leg. Her evaluation revealed bilateral common femoral and superficial femoral arterial occlusion on CT angiogram, moderate renal insufficiency, and evidence of rhabdomyolysis. Her pro-coagulant screening and connective tissue disease profile were negative. She has been managed with anti-coagulation as per hospital protocol and also been given anti-platelets. She progressed to bilateral lower limb gangrene for which fore quarter amputation was done. It is advisable to exercise caution in using long term tranexamic acid usage especially in people who have thrombophilic tendencies.

**Keywords:** Tranexamic Acid; Arterial Thrombosis.

---

### Introduction

Tranexamic acid is an anti-fibrinolytic agent that reversibly binds with lysine receptor sites on plasminogen and prevents its conversion to plasmin, thereby preventing plasmin from binding and degrading fibrin [1]. This preserves the framework of fibrin matrix. Therefore, tranexamic acid is a competitive inhibitor of plasminogen activation and at much higher concentrations, a non-competitive inhibitor of plasmin. Tranexamic acid is ten times more potent than any other anti-fibrinolytic agent. It is mainly excreted through glomerular filtration and has a half-life of four hours. Usually tranexamic acid is used to prevent and treat blood loss in variety of situations like dental procedures in hemophiliacs, heavy menstrual bleeding and in major trauma [2]. The long-term usage of tranexamic acid very rarely can lead to deep vein thrombosis, pulmonary embolism and visual disturbances.

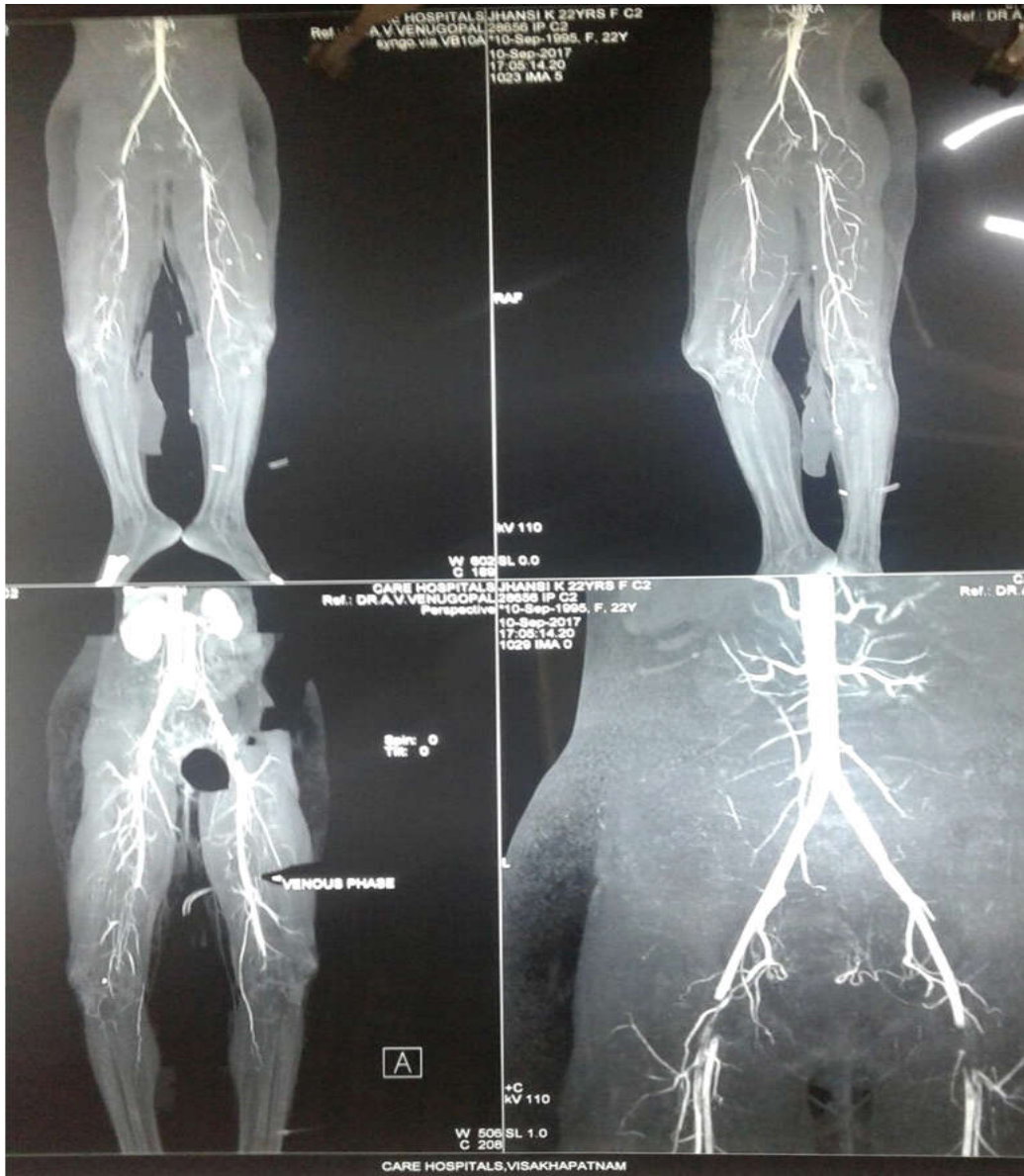
### Case Report

A 22 year old female was admitted to emergency department with history of pain and weakness of both lower limbs for the last 10 days. Claudication distance reported was around 50 feet. There is progressively ascending blackish discoloration of both lower limbs starting from toes. She also complains of vague ill health, reduced urine output and shortness of breath. She has been using oral tranexamic acid at a dosage of 500mg twice a day in the last 4 weeks for menorrhagia. No other significant past medical history except she has been using oral contraceptive pills for the last two years. On physical examination, her vitals are stable. There are no distal pulses felt in both lower limbs. Dry gangrenous patches noted on both feet extending up to ankles. Her Hb% was 12.4gm/dl, Total leucocyte count 11,400, Lactate dehydrogenase 900 IU/ml, Creatinine phosphokinase was 1,89,800 IU/lit, platelet count 1.3lacs/mm<sup>3</sup> and



serum total bilirubin 1.8 mg/dl. Her urine examination revealed 1+ protein and plenty of RBCs. Urine myoglobin was positive. Renal function showed eGFR of 40ml/min/1.73m<sup>2</sup>. After adequate hydration

CT angiogram of lower limb vessels was performed. It revealed total occlusion of right common femoral, superficial femoral, popliteal artery and left superficial femoral artery. Pro-coagulant factor screening (protein



C, protein S and anti-thrombin III) was negative. Her antinuclear antibody and anti-phosphate antibody were negative. Her ultrasound abdomen was within normal limits. She was given anti-coagulation (UFH 5000units/hr for 48 hours), anti-platelet agents and three sessions of hemodialysis through right internal jugular catheter over the next one week. Initially fasciotomy was attempted to try and salvage the limbs. As she developed frank gangrene bilateral lower limb fore quarter amputation was done. Her renal function improved over the next one week. LDH and CPK became normal.

## Discussion

Tranexamic acid widely used in bleeding tendencies though generally safe is not without any major side effects. It has the potential to cause major arterial thrombosis and is contraindicated in patients with thrombophilic tendencies and also in patients with active thrombotic or embolic disorders. In our patient inadvertent long term high dose tranexamic acid has resulted in bilateral acute arterial occlusion. Renal failure in our patient could be attributed to



Rhabdomyolysis. In the literature this agent causing venous thrombosis has been reported. There are 56 reports of deep vein thrombosis, pulmonary embolism or both and these include reports of cerebral and retinal vein thrombosis in the World Health Organization's international drug monitoring database. But, there are only few reports of arterial thrombosis so far [3]. Two reports of arterial thrombosis have been reported in literature, both of whom were on oral TA for menorrhagia and developed cerebral arterial thrombosis [4]. Additionally, there are 22 reports of cerebral embolism and 9 of arterial thrombosis [5]. In our patient bilateral lower limb arterial thrombosis was developed after the usage of high dose tranexamic acid for one month. She has no additional risk factors for development of thrombosis except for she has been using oral contraception for the last 2 years.

### Conclusion

Early recognition of this rare entity could salvage vital organs. The extent of arterial or venous thrombosis depends on the dosage and duration of

tranexamic acid therapy in susceptible patients. The tendency to develop arterial or venous thrombosis in high risk population should be kept in my mind before prescribing long term tranexamic acid.

### References

1. The CRASH-2 Collaborators. "Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant hemorrhage (CRASH-2): a randomized, placebo-controlled trial". *Lancet*. 2010;376(9734):23-32.
2. Takada A et al., Interaction of plasmin with tranexamic acid and alpha 2 plasmin inhibitor in the plasma and clot. *Thromb Hemostat*, 1980 Feb 29;43(1):20-3.
3. S P Upadhyay, Piyush N Mallik, Manish Jagia, R K Singh., "Acute Arterial Thrombosis associated with inadvertent high dose of tranexamic acid". *Indian J. Crit. Care. Med.* 2013 Jul-Aug;17(4):237-239.
4. Letter: Tranexamic acid and intracranial thrombosis. Rydin E, Lundberg PO., *Lancet*: 1976, July 3;2(7975): 49.
5. Ruth Salvage: thrombosis with tranexamic acid for menorrhagia: prescriber update: 2003;24:26-7.

## Glossopharyngeal Neuralgia Leading to Sinus Pause: A Rare Entity

Vaibhav Gulati<sup>1</sup>, Kishalay Datta<sup>2</sup>, Naveen Bhamri<sup>3</sup>

---

### Author's Affiliation:

<sup>1</sup>PGY-3, MEM {GWU-USA} <sup>2</sup>HOD and Associate Director, Dept of Emergency Medicine, <sup>3</sup>HOD and Director, Dept of Cardiology, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.

### Corresponding Author:

**Vaibhav Gulati,**  
PGY-3, MEM {GWU-USA} Dept of Emergency Medicine, Max Super Specialty Hospital, Shalimar Bagh, New Delhi, Delhi 110088, India.  
E-mail: [dr.vaibhavgulati@gmail.com](mailto:dr.vaibhavgulati@gmail.com)

Received on 25.10.2017,

Accepted on 09.11.2017

### Abstract

Glossopharyngeal neuralgia is in itself a rare entity and often remains undiagnosed. Asystole, convulsions, and syncope are associated with glossopharyngeal neuralgia in many patients described in the literature, and this condition is called vagoglossopharyngeal neuralgia. These reactions occur due to the complex anatomical relationship between the intermedius, vagus, and glossopharyngeal nerves leading to difficulties during neurosurgical assessment. Here we report a case of 66 year old male, known case of glossopharyngeal neuralgia, presenting with seizure followed by syncope and later on diagnosed to have prolonged sinus pause.

**Keywords:** Glossopharyngeal Neuralgia; Sinus Pause.

---

### Introduction

Glossopharyngeal neuralgia is a rare facial pain syndrome, accounting for 0.2–1.3% of facial pain syndromes. Approximately 10% of patient are misdiagnosed as trigeminal neuralgia because both syndromes are manifested with facial pain. However in case of Glossopharyngeal neuralgia is located unilateral and extends to the ear and throat.

The first description of severe pain in the distribution of the glossopharyngeal nerve is credited to Weisenberg, in 1910, in a patient with cerebellopontine angle tumor. The term glossopharyngeal neuralgia was coined in 1926 to describe this rare condition characterized by paroxysms of excruciating pain located laterally at the back of the tongue, soft palate, throat, and lateral and posterior pharynx, radiating to the ear. Swallowing, coughing, yawning or chewing may trigger pain, which usually lasts from seconds to minutes.

The association between glossopharyngeal neuralgia and syncope is very rare, being identified by brief episodes of bradycardia, asystole, and hypotension. Such an association, with this same

pathophysiology, was first described by Riley et al in 1942.

Onset is sudden and is usually characterized by severe, unilateral, paroxysmal pain along the glossopharyngeal nerve course. Syncope in Glossopharyngeal neuralgia related to neuralgic pain is most likely caused by activation of the dorsal motor nucleus of the vagus nerve by abnormally enhanced input from afferent or ischemic lesions of the glossopharyngeal nerve. The reflex arrhythmia could be explained from the fact that afferent nerve impulses from the glossopharyngeal nerve may reach the tractus solitarius of the brainstem and via collateral fibers reach the dorsal motor nucleus of the vagus nerve. One afferent branch of the glossopharyngeal nerve supplies the somatosensory information to the nucleus ambiguus, while another afferent branch of the glossopharyngeal nerve, the carotid sinus nerve (Hering nerve), conducts impulses from the body of the carotid sinus to the nucleus dorsalis of the vagal nerve. It has been hypothesized that by artificial synapses in the glossopharyngeal nerve the impulses from the somatosensory branches stimulate the carotid sinus nerve and thereby the nucleus dorsalis. Activation of this abnormal loop during severe neuralgic pain would be responsible for bradycardia/

asystole, with cerebral hypoperfusion, slowing of electro-encephalographic activity, syncope, and convulsions in proportion to the duration of asystole. Individual differences in the susceptibility of the dorsal motor nucleus to the pain impulse may explain why not all cases are associated with syncope.

### Case Report

66 year old male, known case of glossopharyngeal neuralgia, recently diagnosed as seizure disorder, on anti epileptics, K/C/O hypertension presented to ER with 1 episode of seizure followed by one episode of vomiting after which the patient developed respiratory distress and eventually drowsy. There was no history of fever, cough, urinary/bowel disturbance, chest pain, palpitations.

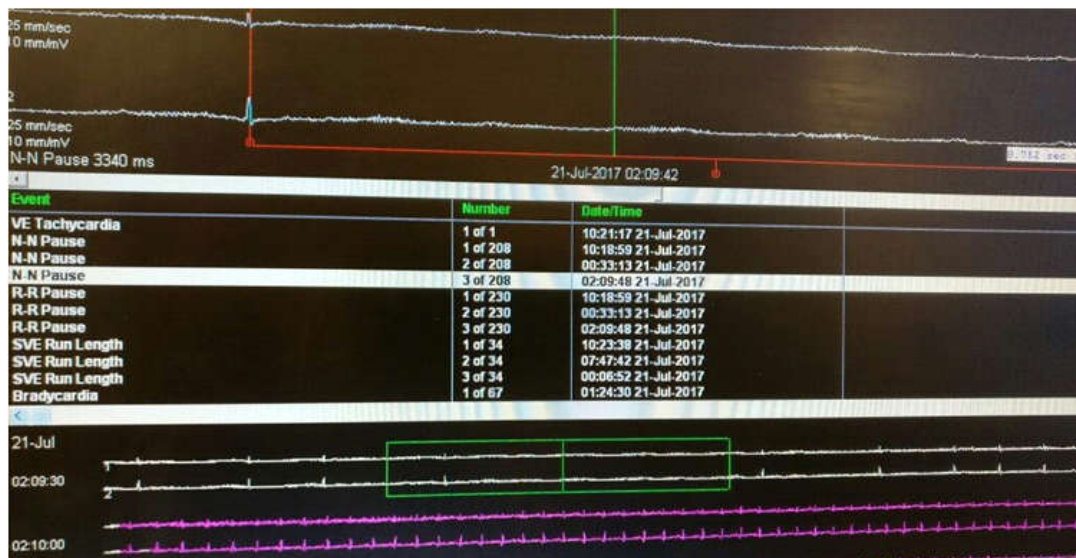
On examination, patients airway was compromised and low GCS, in view of which patient was intubated and ventilated in order to protect the airway. Vitals-BP-160/110mm Hg, HR-74/min, SpO<sub>2</sub>- 99% on ventilator, RBS-112mg/dl. Systemic examination was unremarkable except decreased air entry on right side. An initial differential diagnosis of ?Breakthrough seizure, ?CVA with aspiration with type-2 respiratory

failure was made. Patient was started with anti-epileptics, antibiotics, nebulization, other supportive management and admitted in ICU under neurology department.

MRI brain was suggestive of right parietal small subacute infarct. Patients investigation were suggestive of hypocalcemia and hypomagnesemia and was managed accordingly. Patient responded well to the treatment, was extubated after one day, improved symptomatically and was shifted to HDU after three days.

In HDU, patient had 1 episode of seizure which was managed accordingly. On the same night, patient developed bradycardia and eventually asystole, hypotension and became drowsy. Inj atropine 0.6mg iv stat followed by fluid bolus was given after which patient become responsive and vitals stabilized. Patient was again shifted to ICU, Holter monitoring was planned.

Patient was advised lidocaine spray for his pain and later USG guided glossopharyngeal nerve block was done. Holter monitoring showed intermittent prolonged sinus pause of 13.5 seconds. Following this, patient was taken up for PPI. The patient remained free from syncope after placement of PPI in the hospital as well as on follow up.



### Holter Monitoring

#### Final Interpretation

- Base rhythm is sinus
- Normal Heart rate variability
- Episode of prolonged sinus pauses seen (Maximum 13.5 sec)
- No tachyarrhythmia (AF/PSVT/VT).
- Occasional Supraventricular ectopics.
- Occasional/Frequent VPCs

## Discussion

As far as treatment is concerned, the medical literature supports the use of carbamazepine in the management of idiopathic neuralgia. Temporary pacemaker implantation to treat the reflex cardiac syncope until therapeutic levels of carbamazepine are reached was first described by Khero in 1971.

However permanent pacemaker implantation, the available literature is quite controversial but our patient responded well to the permanent pacemaker and remained free from symptoms.

## References

1. Helio Korkes, Eduardo Mesquita de Oliveira, Luigi Brollo, Denise Tessariol Hachul, Jose Carlos da Silva Andrade, Mario Fernando Prieto Peres, Victor Schubsky. Cardiac syncope induced glossopharyngeal neuralgia: a rare presentation. *Arq. Bras. Cardiol.* 2006 Nov;87(5).
2. Antonios Krasoudakis, Dimitrios Anyfantakis, Athanasios Hadjipetrou, Miltiades Kastanakis, Emmanouil K. Symvoulakis, Stavros Marathianos. Glossopharyngeal neuralgia associated with cardiac syncope: Two case reports and literature review. *Int J Surg Case Rep.* 2015;12:4-6.
3. Bronson S. Ray, Harold J. Stewart. Glossopharyngeal neuralgia: A cause of cardiac arrest. *AHJ March* 1948; 35(3):458-462.
4. Alexander W. den Hartog, Evelien Jansen, Jasper E. Kal, Debby Duyndam, Jeldican Visser, Pepijn van den Munckhof, Jonas S.S.G. de Jong, Krischan D. Sjauw. Recurrent syncope due to glossopharyngeal neuralgia. *HeartRhythm Case Rep.* 2017 Jan;3(1): 73-77.
5. Jawed H. Siddiqui. Sick Sinus Syndrome : A case Study. *Pakistan Heart Journal* 1984;17(2).
6. Hye Yun Kim, Do Young Yoon, Dong Sun Kim, Ji Sun Kwon, Hyun Jeong Han. A case of Sick Sinus Syndrome presenting as Exploding Head Syndrome. *J Korean Sleep Res Soc.* 2012;9(2):61-63.
7. Aditya Prasad, Jay Tiongson, Tasneem Z Naqvi. Asystole during sleep in a 44 year old male athlete. <http://www.mdmag.com/journals/cardiology-review-online/2007/june2007/june-2007-prasad>.

## G6PD Deficiency as a Precipitant of Haemolysis in Hepatitis E Patients

Umran R. Sheikh<sup>1</sup>, Kishalay Datta<sup>2</sup>, Shahid Mustafa Khan<sup>1</sup>, Indranil Das<sup>3</sup>  
Deepika Mittal<sup>1</sup>

---

### Author's Affiliation:

<sup>1</sup>MEM, PGY-3 <sup>2</sup>HOD <sup>3</sup>Attending  
Consultant, Department of  
Emergency Medicine, Max Super  
Speciality Hospital, Shalimar  
Bagh, New Delhi, Delhi 110088,  
India.

### Corresponding Author:

Umran R. Sheikh, MEM, PGY-3,  
Department of Emergency  
Medicine, Max Super Speciality  
Hospital, Shalimar Bagh,  
New Delhi, Delhi 110088, India.  
E-mail: [drindradas@gmail.com](mailto:drindradas@gmail.com)

Received on 24.05.2017,

Accepted on 13.06.2017

### Abstract

Hepatitis E is one of the common forms of Acute viral hepatitis in epidemic proportions in India. It has been seen to cause severe Haemolysis when associated with G6PD deficiency which is rarely seen in the northern India. This case report is of a 35 year old male with Hepatitis E who presented to the Emergency Room pale and icteric and on evaluation was found to have G6PD deficiency as the cause of severe haemolysis. Therefore, in patients with acute viral hepatitis and severe anaemia with unconjugated hyperbilirubinemia, it becomes a necessity to rule out G6PD deficiency as a cause of the intravascular haemolysis.

**Keywords:** G6PD Deficiency; Hepatitis E; Viral Hepatitis; Intravascular Haemolysis; Unconjugated Hyperbilirubinemia; Anaemia.

---

### Introduction

Hepatitis E, is one of the most common forms of acute viral hepatitis in India [1], it is potentially fatal in pregnant females and is a concerning cause of epidemic proportions of viral hepatitis in India. In patients with G6PD deficiency, it has been known to cause complications such as severe anaemia, haemolysis, hepatic, renal impairment or even death [2,3]. Since G6PD deficiency is of very low occurrence in the Indian population, reported between 2.2-14% in northern India [4].

We present the case report of a 35 year old male who presented to the ED with Hepatitis E with icterus, anaemia and was later on evaluation discovered to have G6PD deficiency.

### Case Report

A 35 year old male had presented in the ED with a history of fever since past 10 days associated with nausea, vomiting and diarrhoea. Patient also complained of yellowish discolouration of eyes and dark coloured urine since past 2-3 days along with

excessive drowsiness. No history of constipation, loose stools, malena, hematemesis, trauma.

On primary survey; his Airway was patent; Breathing, the respiratory rate was 16/min with a saturation of 85% on room air which improved to 89% despite supplementing with high flow oxygen; Circulation, heart rate was 98/min with a blood pressure reading of 130/70 mmHg, Peripheral pulses felt equally and a capillary refill time of less than 3 seconds. The patient was drowsy but responding to verbal commands, moving all four limbs with a GRBS of 220mg/dl.

On secondary survey; conjunctival pallor, icterus was seen, oral mucosa was dry, there were no distended neck veins, chest had equal air entry bilaterally with no adventitious sounds, heart sounds S1S2 heard with no murmurs and a normal JVP; Abdomen was soft, non-tender with mild hepatomegaly, no splenomegaly, shifting dullness present and bowel sounds heard. Central nervous system examination, the patient was drowsy but arousable, moving all four limbs, no sensory or motor deficit, Deep tendon reflexes were normal in all four limbs, the plantar reflexes were flexors bilaterally and flapping tremors were absent. Extremities showed no rashes, deformities or oedema.

He was a known case of Diabetes Mellitus, Bipolar Mood Disorder and Hypertension for which he was on oral hypoglycaemics, Lithium and Amlodipine.

Among the point of contact tests done in the Emergency, his ECG and Chest X ray were within normal limits. Arterial blood Gas was within normal limit with no hypoxaemia seen and S. Lactate was 1.4mmol/L.

His Lab Investigations revealed as follows:

Haemogram - Haemoglobin was 6.3 g/dl, TLC of 6,400/mm<sup>3</sup>, Platelets 200,000/mm<sup>3</sup>;

Renal profile - S.Urea 24 mg/dL, S.Creatinine 0.7mg/dL, S. Sodium 122.5mEq/L, S. Potassium 4.4mEq/L S. Chloride 94mEq/L

Liver function tests - S.Albumin 3.4g/dL, S. Globulin 2g/dL, Total bilirubin 50.3mg/dL, unconjugated bilirubin 19.7 mg/dL, Alkaline phosphatase 422 U/L, SGOT 310 IU/L and SGPT 640 IU/L

Coagulation profile - PT 12.6 S, INR 1.11, APTT 24.6

Abdominal sonography was suggestive of Hepatomegaly, a thickened oedematous Gall Bladder with minimal ascites.

He was admitted with a working diagnosis of Viral Hepatitis with Hepatic Encephalopathy (Grade 1). Investigation results revealed Serum Ammonia 233 mcg/dl and Serum LDH 2244 U/L was seen.

Hepatitis A, Hepatitis B and Hepatitis C were tested negative. Hepatitis E virus was positive. Reticulocyte counts were elevated and G6PD enzyme was found to be 4.1 (low).

No evidence of Malaria, Typhoid, Dengue on investigation.

Coomb's test (Direct/Indirect) was Negative.

Patient was transfused 2 units of PRBCs. Patient was managed conservatively, avoiding all oxidant, hepatotoxic and nephrotoxic drugs, while maintaining an adequate urine output following which, on the fourth day, his lab parameters had improved with haemogram showing Hb of 10 g/dl.

After five days of hospital stay he was discharged in a stable condition with normal vital parameters, diagnosed as Acute Hepatitis E with Haemolytic anaemia due to G6PD deficiency.

## Discussion

Viral Hepatitis has been known to cause mild haemolysis which rarely becomes evident clinically

[5]. Severe haemolysis has been known in patients with G6PD deficiency on exposure to certain drugs [5,7,9]. But as in our case, viral hepatitis has been known to cause haemolysis in the absence of any such drugs. The patient described above in this case, had a fall in Haemoglobin, reticulocytosis, unconjugated hyperbilirubinemia along with low levels of G6PD which suggested severe intravascular haemolysis due to G6PD deficiency. The presence of severe hyperbilirubinemia in patients with viral hepatitis and G6PD deficiency has been reported previously [8-10]. The mechanism is believed to be through decreased levels of glutathione in RBCs as a result of accumulation of oxidants due to hepatic dysfunction, thus causing haemolysis in presence of G6PD deficiency [6].

Prognosis in these patient is associated with the degree of hepatic injury. Severe haemolysis could lead to increase in free haematin and bilirubin, thus leading to obstruction of renal tubules and acute renal impairment. Renal failure in these patients might be non-oliguric. Hence, renal function monitoring should be done with blood tests and urine osmolality and sodium.

Tests for G6PD deficiency might be negative during or after a haemolytic episode because the old red cells deficient in G6PD have undergone haemolysis and the newer red blood cells with higher content of G6PD might lead to false normal levels.

Hence, a repeat test needs to be done 8 to 10 weeks after the disease resolves. All G6PD-deficient individuals should be vaccinated against Hepatitis A and B.

## Conclusion

In patients presenting with acute viral hepatitis and an unexplained severe anaemia with unconjugated hyperbilirubinemia, the possibility of intravascular haemolysis should be considered and evaluated with due consideration to rule out G6PD deficiency.

## References

1. Das K, Agarwal A, Andrew R, Frosner GG, Kar P. Role of hepatitis E and other hepatotropic virus in etiology of sporadic acute viral hepatitis: A hospital based study from urban Delhi. *Eur J Epidemiol* 2000;16:937-40.
2. Agarwal RK, Moudgil A, Kishore K, Srivastava RN, Tandon RK. Acute viral hepatitis, intravascular hemolysis, severe hyperbilirubinemia and renal

- failure in glucose-6-phosphate dehydrogenase deficient patients. *Postgrad Med J* 1985;61:971-5.
3. Jolly JG, Sarup BM, Bhatnagar DP, et al. Glucose-6-phosphate dehydrogenase deficiency in India. *JIMA* 1972; 58:196.
  4. Choudhury VP, Bagga A, Desai N. Increased morbidity of viral hepatitis in patients with G6PD deficiency. *J Trop Paed* 1992;38:139-40.
  5. Abid S, Khan AH. Severe hemolysis and renal failure in glucose-6-phosphate dehydrogenase deficient patients with hepatitis E. *Am J Gastroenterol* 2002;97:1544-7.
  6. Pitcher CS, Williams R. Reduced red cell survival in jaundice and its relation to abnormal glutathione metabolism. *Clin Sci* 1963;24:239.
  7. Kattamis CA, Tuortjatou F. The hemolytic process of viral hepatitis in children with normal or deficient glucose-6-phosphate dehydrogenase activity. *J Pediatr* 1970;77:422-30.
  8. Clearfield HR, Brody JI, Tumen HJ. Acute viral hepatitis, glucose-6-phosphate dehydrogenase deficiency and hemolytic anemia. *Arch Int Med* 1969;123:689.
  9. Salen G, Goldstein F, Hanrani F, et al. Acute hemolytic anemia complicating acute viral hepatitis in patients with glucose-6-phosphate dehydrogenase deficiency. *Ann Int Med* 1966;65:1210.
  10. Morrow RH, Smetana HF, Sai FT, et al. Unusual features of viral hepatitis in Accra, Ghana. *Ann Int Med* 1968; 68:1250.
-

**Revised Rates for 2018 (Institutional)**

<b>Title</b>	<b>Frequency</b>	<b>Rate (Rs): India</b>		<b>Rate (\$):ROW</b>	
Community and Public Health Nursing	3	5500	5000	430	391
Dermatology International	2	5500	5000	430	391
Gastroenterology International	2	6000	5500	469	430
Indian Journal of Agriculture Business	2	5500	5000	413	375
Indian Journal of Anatomy	4	8500	8000	664	625
Indian Journal of Ancient Medicine and Yoga	4	8000	7500	625	586
Indian Journal of Anesthesia and Analgesia	4	7500	7000	586	547
Indian Journal of Biology	2	5500	5000	430	391
Indian Journal of Cancer Education and Research	2	9000	8500	703	664
Indian Journal of Communicable Diseases	2	8500	8000	664	625
Indian Journal of Dental Education	4	5500	5000	430	391
Indian Journal of Emergency Medicine	2	12500	12000	977	938
Indian Journal of Forensic Odontology	2	5500	5000	430	391
Indian Journal of Hospital Administration	2	7000	6500	547	508
Indian Journal of Hospital Infection	2	12500	12000	938	901
Indian Journal of Law and Human Behavior	2	6000	5500	469	430
Indian Journal of Library and Information Science	3	9500	9000	742	703
Indian Journal of Maternal-Fetal & Neonatal Medicine	2	9500	9000	742	703
Indian Journal of Medical & Health Sciences	2	7000	6500	547	508
Indian Journal of Obstetrics and Gynecology	4	9500	9000	742	703
Indian Journal of Pathology: Research and Practice	4	12000	11500	938	898
Indian Journal of Plant and Soil	2	65500	65000	5117	5078
Indian Journal of Preventive Medicine	2	7000	6500	547	508
Indian Journal of Research in Anthropology	2	12500	12000	977	938
Indian Journal of Surgical Nursing	3	5500	5000	430	391
Indian Journal of Trauma & Emergency Pediatrics	4	9500	9000	742	703
Indian Journal of Waste Management	2	9500	8500	742	664
International Journal of Food, Nutrition & Dietetics	3	5500	5000	430	391
International Journal of Neurology and Neurosurgery	2	10500	10000	820	781
International Journal of Pediatric Nursing	3	5500	5000	430	391
International Journal of Political Science	2	6000	5500	450	413
International Journal of Practical Nursing	3	5500	5000	430	391
International Physiology	2	7500	7000	586	547
Journal of Animal Feed Science and Technology	2	78500	78000	6133	6094
Journal of Cardiovascular Medicine and Surgery	2	10000	9500	781	742
Journal of Forensic Chemistry and Toxicology	2	9500	9000	742	703
Journal of Geriatric Nursing	2	5500	5000	430	391
Journal of Microbiology and Related Research	2	8500	8000	664	625
Journal of Nurse Midwifery and Maternal Health	3	5500	5000	430	391
Journal of Organ Transplantation	2	26400	25900	2063	2023
Journal of Orthopaedic Education	2	5500	5000	430	391
Journal of Pharmaceutical and Medicinal Chemistry	2	16500	16000	1289	1250
Journal of Practical Biochemistry and Biophysics	2	7000	6500	547	508
Journal of Psychiatric Nursing	3	5500	5000	430	391
Journal of Social Welfare and Management	3	7500	7000	586	547
New Indian Journal of Surgery	4	8000	7500	625	586
Ophthalmology and Allied Sciences	2	6000	5500	469	430
Otolaryngology International	2	5500	5000	430	391
Pediatric Education and Research	3	7500	7000	586	547
Physiotherapy and Occupational Therapy Journal	4	9000	8500	703	664
Psychiatry and Mental Health	2	8000	7500	625	586
Urology, Nephrology and Andrology International	2	7500	7000	586	547

**Terms of Supply:**

1. Agency discount 10%. Issues will be sent directly to the end user, otherwise foreign rates will be charged.
2. All back volumes of all journals are available at current rates.
3. All Journals are available free online with print order within the subscription period.
4. All legal disputes subject to Delhi jurisdiction.
5. Cancellations are not accepted orders once processed.
6. Demand draft / cheque should be issued in favour of "Red Flower Publication Pvt. Ltd." payable at Delhi
7. Full pre-payment is required. It can be done through online (<http://rfppl.co.in/subscribe.php?mid=7>).
8. No claims will be entertained if not reported within 6 months of the publishing date.
9. Orders and payments are to be sent to our office address as given above.
10. Postage & Handling is included in the subscription rates.
11. Subscription period is accepted on calendar year basis (i.e. Jan to Dec). However orders may be placed any time throughout the year.

**Order from**

**Red Flower Publication Pvt. Ltd.**, 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091 (India), Tel: 91-11-22754205, 45796900, Fax: 91-11-22754205. E-mail: [sales@rfppl.co.in](mailto:sales@rfppl.co.in), Website: [www.rfppl.co.in](http://www.rfppl.co.in)



Manuscripts must be prepared in accordance with "Uniform requirements for Manuscripts submitted to Biomedical Journal" developed by international committee of medical Journal Editors.

## Types of Manuscripts and Limits

Original articles: Up to 3000 words excluding references and abstract and up to 10 references.

Review articles: Up to 2500 words excluding references and abstract and up to 10 references.

Case reports: Up to 1000 words excluding references and abstract and up to 10 references.

## Online Submission of the Manuscripts

Articles can also be submitted online from [http://rfppl.co.in/customer\\_index.php](http://rfppl.co.in/customer_index.php).

1) First Page File: Prepare the title page, covering letter, acknowledgement, etc. using a word processor program. All information which can reveal your identity should be here. use text/rtf/doc/PDF files. Do not zip the files.

2) Article file: The main text of the article, beginning from Abstract till References (including tables) should be in this file. Do not include any information (such as acknowledgement, your name in page headers, etc.) in this file. Use text/rtf/doc/PDF files. Do not zip the files. Limit the file size to 400 Kb. Do not incorporate images in the file. If file size is large, graphs can be submitted as images separately without incorporating them in the article file to reduce the size of the file.

3) Images: Submit good quality color images. Each image should be less than 100 Kb in size. Size of the image can be reduced by decreasing the actual height and width of the images (keep up to 400 pixels or 3 inches). All image formats (jpeg, tiff, gif, bmp, png, eps etc.) are acceptable; jpeg is most suitable.

Legends: Legends for the figures/images should be included at the end of the article file.

If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks from submission. Hard copies of the images (3 sets), for articles submitted online, should be sent to the journal office at the time of submission of a revised manuscript. Editorial office: Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091, India, Phone: 91-11-22754205, 45796900, 22756995. E-mail:

[author@rfppl.co.in](mailto:author@rfppl.co.in). Submission page: [http://rfppl.co.in/article\\_submission\\_system.php?mid=5](http://rfppl.co.in/article_submission_system.php?mid=5).

## Preparation of the Manuscript

The text of observational and experimental articles should be divided into sections with the headings: Introduction, Methods, Results, Discussion, References, Tables, Figures, Figure legends, and Acknowledgment. Do not make subheadings in these sections.

## Title Page

The title page should carry

- 1) Type of manuscript (e.g. Original article, Review article, Case Report)
- 2) The title of the article, should be concise and informative;
- 3) Running title or short title not more than 50 characters;
- 4) The name by which each contributor is known (Last name, First name and initials of middle name), with his or her highest academic degree(s) and institutional affiliation;
- 5) The name of the department(s) and institution(s) to which the work should be attributed;
- 6) The name, address, phone numbers, facsimile numbers and e-mail address of the contributor responsible for correspondence about the manuscript; should be mentioned.
- 7) The total number of pages, total number of photographs and word counts separately for abstract and for the text (excluding the references and abstract);
- 8) Source(s) of support in the form of grants, equipment, drugs, or all of these;
- 9) Acknowledgement, if any; and
- 10) If the manuscript was presented as part at a meeting, the organization, place, and exact date on which it was read.

## Abstract Page

The second page should carry the full title of the manuscript and an abstract (of no more than 150 words for case reports, brief reports and 250 words for original articles). The abstract should be structured and state the Context (Background), Aims, Settings and Design, Methods and Materials, Statistical analysis used, Results and Conclusions. Below the abstract should provide 3 to 10 keywords.

## Introduction

State the background of the study and purpose of the study and summarize the rationale for the study or observation.

## Methods

The methods section should include only information that was available at the time the plan or protocol for the study was written such as study approach, design, type of sample, sample size, sampling technique, setting of the study, description of data collection tools and methods; all information obtained during the conduct of the study belongs in the Results section.

Reports of randomized clinical trials should be based on the CONSORT Statement (<http://www.consort-statement.org>). When reporting experiments on human subjects, indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000 (available at [http://www.wma.net/e/policy/17-c\\_e.html](http://www.wma.net/e/policy/17-c_e.html)).

## Results

Present your results in logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations. Extra or supplementary materials and technical details can be placed in an appendix where it will be accessible but will not interrupt the flow of the text; alternatively, it can be published only in the electronic version of the journal.

## Discussion

Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying

mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

## References

List references in alphabetical order. Each listed reference should be cited in text (not in alphabetic order), and each text citation should be listed in the References section. Identify references in text, tables, and legends by Arabic numerals in square bracket (e.g. [10]). Please refer to ICMJE Guidelines ([http://www.nlm.nih.gov/bsd/uniform\\_requirements.html](http://www.nlm.nih.gov/bsd/uniform_requirements.html)) for more examples.

### Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. *J Oral Pathol Med* 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. *Acta Odontol Scand* 2003; 61: 347-55.

### Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antiseptics. State of the art. *Dermatology* 1997; 195 Suppl 2: 3-9.

### Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. *J Periodontol* 2000; 71: 1792-801.

### Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. *Dent Mater* 2006.

### Personal author(s)

[6] Hosmer D, Lemeshow S. Applied logistic regression, 2<sup>nd</sup> edn. New York: Wiley-Interscience; 2000.

### Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p.7-27.

### No author given

[8] World Health Organization. Oral health surveys - basic methods, 4<sup>th</sup> edn. Geneva: World Health Organization; 1997.

### Reference from electronic media

[9] National Statistics Online – Trends in suicide by method in England and Wales, 1979-2001. [www.statistics.gov.uk/downloads/theme\\_health/HSQ\\_20.pdf](http://www.statistics.gov.uk/downloads/theme_health/HSQ_20.pdf) (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

More information about other reference types is available at [www.nlm.nih.gov/bsd/uniform\\_requirements.html](http://www.nlm.nih.gov/bsd/uniform_requirements.html), but observes some minor deviations (no full stop after journal title, no issue or date after volume, etc).

### Tables

Tables should be self-explanatory and should not duplicate textual material.

Tables with more than 10 columns and 25 rows are not acceptable.

Table numbers should be in Arabic numerals, consecutively in the order of their first citation in the text and supply a brief title for each.

Explain in footnotes all non-standard abbreviations that are used in each table.

For footnotes use the following symbols, in this sequence: \*, ¶, †, ‡.

### Illustrations (Figures)

Graphics files are welcome if supplied as Tiff, EPS, or PowerPoint files of minimum 1200x1600 pixel size. The minimum line weight for line art is 0.5 point for optimal printing.

When possible, please place symbol legends below the figure instead of to the side.

Original color figures can be printed in color at the editor's and publisher's discretion provided the author agrees to pay.

Type or print out legends (maximum 40 words, excluding the credit line) for illustrations using double spacing, with Arabic numerals corresponding to the illustrations.

### Sending a revised manuscript

While submitting a revised manuscript, contributors are requested to include, along with single copy of the final revised manuscript, a photocopy of the revised manuscript with the changes underlined in red and copy of the comments with the point to point clarification to each comment. The manuscript number should be written on each of these documents. If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks of submission. Hard copies of images should be sent to the office of the journal. There is no need to send printed manuscript for articles submitted online.

### Reprints

Journal provides no free printed reprints, however a author copy is sent to the main author and additional copies are available on payment (ask to the journal office).

### Copyrights

The whole of the literary matter in the journal is copyright and cannot be reproduced without the written permission.

### Declaration

A declaration should be submitted stating that the manuscript represents valid work and that neither this manuscript nor one with substantially similar content under the present authorship has been published or is being considered for publication elsewhere and the authorship of this article will not be contested by any one whose name (s) is/are not listed here, and that the order of authorship as placed in the manuscript is final and accepted by the co-authors. Declarations should be signed by all the authors in the order in which they are mentioned in the original manuscript. Matters appearing in the Journal are covered by copyright but no objection will be made to their reproduction provided permission is obtained from the Editor prior to publication and due acknowledgment of the source is made.

but no objection will be made to their reproduction provided permission is obtained from the Editor prior to publication and due acknowledgment of the source is made.

### Abbreviations

Standard abbreviations should be used and be spelt out when first used in the text. Abbreviations should not be used in the title or abstract.

### Checklist

- Manuscript Title
- Covering letter: Signed by all contributors
- Previous publication/ presentations mentioned, Source of funding mentioned
- Conflicts of interest disclosed

### Authors

- Middle name initials provided.
- Author for correspondence, with e-mail address provided.
- Number of contributors restricted as per the instructions.
- Identity not revealed in paper except title page (e.g. name of the institute in Methods, citing previous study as 'our study')

### Presentation and Format

- Double spacing
- Margins 2.5 cm from all four sides
- Title page contains all the desired information. Running title provided (not more than 50 characters)
- Abstract page contains the full title of the manuscript
- Abstract provided: Structured abstract provided for an original article.
- Key words provided (three or more)
- Introduction of 75-100 words
- Headings in title case (not ALL CAPITALS). References cited in square brackets
- References according to the journal's instructions

### Language and grammar

- Uniformly American English
- Abbreviations spelt out in full for the first time. Numerals from 1 to 10 spelt out
- Numerals at the beginning of the sentence spelt out

### Tables and figures

- No repetition of data in tables and graphs and in text.
- Actual numbers from which graphs drawn, provided.
- Figures necessary and of good quality (color)
- Table and figure numbers in Arabic letters (not Roman).
- Labels pasted on back of the photographs (no names written)
- Figure legends provided (not more than 40 words)
- Patients' privacy maintained, (if not permission taken)
- Credit note for borrowed figures/tables provided
- Manuscript provided on a CDROM (with double spacing)

### Submitting the Manuscript

- Is the journal editor's contact information current?
- Is the cover letter included with the manuscript? Does the letter:
  1. Include the author's postal address, e-mail address, telephone number, and fax number for future correspondence?
  2. State that the manuscript is original, not previously published, and not under concurrent consideration elsewhere?
  3. Inform the journal editor of the existence of any similar published manuscripts written by the author?
  4. Mention any supplemental material you are submitting for the online version of your article. Contributors' Form (to be modified as applicable and one signed copy attached with the manuscript)

## Subject Index

Title	Page No
'Tetpro Score' for Evaluation of Progression in a Case of Tetanus	249
A Case Report on Stroke in Young	258
A Case of Infant with Factor VII Deficiency Presenting as ICH	272
A Case Report on Acute Myocardial Infarction in Young: Atypical ECG Changes Vs. Angiographic Correlation	261
A Classic Presentation of Guillian Barre Syndrome	155
A Comparative Study to Evaluate the Degree of Correlation between Emergency Department Admission Diagnosis and Hospital Discharge Diagnosis	32
A Rare Case of Complicated Neuroleptic Malignant Syndrome with Rhabdomyolysis and Acute Kidney Injury	304
A Rare Serious Ocular Side Effect of Topiramate: Bilateral Acute Angle Closure Glaucoma	255
A Study of Pulmonary Manifestations in Rheumatoid Arthritis and Its Correlation with Disease Activity	56
A Study on Management and outcome of Acute Myocardial Infarction	107
Achieving Sustainable Door-To-Balloon Time of 90 Minutes in a Tertiary Centre Hospital for ST-Segment Elevation Myocardial Infarction	188
Acute Isolated Posterior Myocardial Infarction; Challenges in Recognition and Management in the Emergency Department	286
Amitraz (Acaricide) Poisoning : A Study of this Unusual Emerging Poison	27
An Unusual Presentation of Recurrent Hypoglycemia	275
An Unusual Presentation of Fat Embolism Syndrome as Cerebral Fat Embolism in Trauma: A Rare Clinical Entity	316
Asymmetrical and Late Onset of Pulmonary Edema Post Scorpion Sting: Case Report of Rare Manifestation	282
Beyond ACLS Protocol – A Rare Case of Refractory Supraventricular Tachycardia Responding Only to a Much Higher Dose of Adenosine	311
Bilateral Acute Lower Limb Arterial Occlusion after Long Term Tranexamic Acid Usage	319
Carcinoma Prostate with Metastasis to Vertebral Column and Right Cerebellum Causing Sol and Hydrocephalus	266
Case of Takayasu Arteritis in The Indian Population: Probable Association with Tuberculosis	161
Cerebral Venous Thrombosis and Hyperhomocysteinemia, How Important is the Co-Relation?-A Review of 3 Cases	278
Clinical Presentation of Renal Injury at a Tertiary Care Hospital	244
Clinical Profile of Patients with Rheumatoid Arthritis Attending Tertiary Care Hospital	102
Comparative Study of Blood Sugars, Lipid Profile, in Type 2 Diabetes Mellitus and its Correlation to Ischemic Heart Disease	74
Comparative Study of Clinical Profile of Acute Myocardial Infarction between Elder Age Groups and Nonelder (Younger) Age Group	51
Comparison of Simultaneously Obtained Central Venous Blood Gas and Arterial Blood Gas Analysis for pH, pCO <sub>2</sub> , BE and K <sup>+</sup> In Patients Presenting to Emergency Medicine and Critical Care Unit	7
Detection of Endotracheal Intubation by Insertion Depth of Endotracheal Tube, Bilateral Chest Auscultation, and Observation of Bilateral Chest Movement during Emergency Intubation: Prospective Observational Study	112
Diagnostic Dilemma in a Case of Ischaemic CVA	126
Does Henna Affect Pulse Oximetry Reading? – A Study	37
Early Diagnosis and Treatment not Always a Key to Favorable Outcome: A Case Report of ADEM Correctly Diagnosed and Treated Still Surviving for Better Life	293
Emphysematous Pyelonephritis	130

Epidemics of Acute Encephalitis in Young Children : Lychee the Sweet Hypoglycemic or a Silent Killer	121
G6PD Deficiency as a Precipitant of Haemolysis in Hepatitis E Patients	325
Glossopharyngeal Neuralgia Leading to Sinus Pause: A Rare Entity	322
Hypertension-Induced Posterior Reversible Encephalopathy Syndrome as the Presentation of Progressive Bilateral Renal Artery Stenosis	134
Hypokalemic Periodic Paralysis Mimicking as CVA	158
Integrative Weaning Index: Can it be Used Routinely as a Predictor of Weaning Success?	23
Left Ventricular Rupture: Where is the Key in Management?	149
Life Threatening Rhabdomyolysis, A Rare and Unusual Presentation with Rosuvastatin Ingestion	308
Management and outcome of Acute Kidney Injury at a Tertiary Care Hospital	197
Metronidazole Induced Encephalopathy	152
Myotonic Dystrophy: A Rare Autosomal Dominant Disorder	252
Non-Invasive Ventilation – First Line Therapy in the Acute Exacerbations of COPD in Emergency Department	217
One and Half Syndrome in Acute Pontine Infarct: A Rare Entity	264
Partial Empty Sella Syndrome Presenting to Emergency as A Case of Recurrent Hyponatremia: A Rare Presentation	137
Patient Expectations in the Emergency Department of a Super-Speciality Hospital	236
Petrol Ingestion Causing Methaemoglobinaemia in Glucose 6-Phosphate Dehydrogenase (G6PD) Deficiency Patient	290
Prevalence and Characterization of Chronic Kidney Disease Associated Pruritus in Patients Undergoing Maintenance Hemodialysis	89
Primary Hypothyroidism with Facial Paralysis: A Case Report	164
Prognosis of Haemodynamically Unstable Patients Secondary to Trauma Based on Lactate Clearance	203
Rational Use of Anti-Snake Venom: Trial of Various Use Regimes in Hemotoxic Snake Envenomation	190
Renal Thrombotic Microangiopathy Due to Malignant Hypertension Reperfusion: for Better or for Worse	141
Role of Intravenous Magnesium Sulphate in Predicting Outcomes of ICU in Acute Organophosphate Poisoning	231
Spectrum of Acute Febrile Illness in Children More than 3 Months and Under the Age of 15 Years Presenting in Emergency of a Tertiary Care Hospital and Its Clinico-Laboratorial Correlation	211
Study of Hematological Profile and Effect of Antitubercular Medications on the Hematological Derangements in Patients Suffering from Tuberculosis	45
Study of Acute Myocardial Infarction in Post Menopausal Women with Special Reference to Dyslipidemia	97
Study of Hyperglycaemic States and Its Outcome	79
Study of Hypertension and Anthropometrics Measurement in Type 2 Diabetes Mellitus	84
Study of Serum Sodium and Potassium Levels in Patients of Acute Myocardial Infarction	183
Supraventricular Tachycardia in a Woman with Third Trimester Pregnancy and Atrial Septal Defect: A Case Report	167
Tetanus Prevention Knowledge and Practices in Doctors of a Tertiary Care Centre of India	16
The Study of the Clinical Profile and Laboratory Parameters of Acute Neonicotinoid Compound Poisoning at a Rural Tertiary Care Public Hospital in Central India	223
To Evaluate Prophylactic Use of Antiemetic (Ondansetron) with Opioid Analgesics (Tramadol) for Acute Pain in Emergency Department	62
Torsion of Non-Gravid Uterus with Myoma Presenting to Emergency with Shock	301
Traumatic Cardiac Tamponade – Relearning Old Lesions to Avoid Delay in Diagnosis and Management of a Life-Threatening Thoracic Injury	296

## Author Index

Name	Page No	Name	Page No
A.V. Venugopal	319	Datta K.	322
Abdul Hai Gojwari	62	Deepak Garg	130
Abhinav Gupta	89	Deepika Mittal	308
Adithya Udupa K.	149	Deepika Mittal	325
Aisvarya Girotra	258	Deepika Mittal	121
Ajay V. Garg	255	Dhand N.	316
Akash Gandotra	164	Dhruvkumar M. Patel	255
Almur Abdullah Alabri	290	Dina J. Shah	278
Amit Kumar Yadav	32	E.V. Balasubramanyam	266
Amith Kumar	183	Forhad A. Zaman	45
Anand Chavan	107	G. Vishwa Reddy	217
Anand Chavan	51	G. Vishwa Reddy	7
Anita Rawat	134	Girotra A.	261
Anita Rawat	137	Gouri Kumar Rath	282
Anjali Patel	141	Govil P.	275
Ankur Pandey	286	Gulati V.	188
Annil Mahajan	164	Gulati V.	264
Aravinda C.L.	190	Gulati V.	272
Arun Prasad	167	Gulati V.	293
Arunil Gupta	130	Gulati V.	316
Ashima Sharma	217	Gulati V.	322
Bakshi Surrinder Kumar	290	Gurjit Kaur	296
Balasubramanyam E.V.	158	Harini Agnes	269
Balasubramanyam E.V.	252	Harini Agnes	319
Balasubramanyam E.V.	258	Hilal Ahmad Yatoo	134
Balasubramanyam E.V.	316	Hilal Ahmad Yatoo	161
Begum Naheeda Shaik	203	Hilal Ahmad Yatoo	258
Bhambri N.	322	Hilal Yatoo	152
Bharath Angadi	23	Hilal Yatoo	311
Bharath Angadi	27	Indraneel Dasgupta	112
Bharath Angadi	79	Indraneel Dasgupta	126
Bidita Khandelwal	45	Indraneel Dasgupta	16
Birajdar Siddheshwar V.	223	Indraneel Dasgupta	236
Bopanna C.A.	249	Indraneel Dasgupta	32
Chaitali Kundu	16	Indraneel Dasgupta	37
Chandrakala	97	Indranil Das	325
Chandrashekar S.	23	Indranil Das	121
Chandru Lamani	102	Indranil Das	137
Chandru Lamani	107	Indranil Das	152
Chandru Lamani	51	Indranil Das	155
Chavan Sheshrao S.	223	Indranil Das	161
Chethan B.H.	102	Indranil Das	266
Chethan B.H.	56	Indranil Das	62
Cijo John	197	Indranil Mitra	16
Cijo John	244	Indranil Mitra	32
Das I.	275	Indranil Mitra	37
Das I.	316	Jitesh Bhandarkar	134
Das Indranil	158	Jitesh Bhandarkar	152
Datta K.	158	Jitesh K. Bhandarkar	252
Datta K.	188	K. Datta	266
Datta K.	261	Kahlon R.	272
Datta K.	264	Kalavathi G.P.	74
Datta K.	272	Kalavathi G.P.	84
Datta K.	275	Kalita R.	261
Datta K.	293	Kalita R.	275
Datta K.	304	Kallesh Shamanur	23
Datta K.	316	Kallesh Shamanur	27

Kallesh Shamanur	79	Rigenjyoti Kalita	211
Kalyan Dutt	89	Rignesh Patel	141
Kamal Preet Palta	301	Rohan Gupta	164
Kaur G.	304	Ronak M. Raheja	231
Kendre Vitthal M.	223	Rudraneel Kumar	126
Ketan Patel	141	Rupinder Khalon	155
Khan Khader Ali	203	S. Raghavendra Goud	217
Kiran Puli	56	Sajid Nomani	282
Kishalay Datta	121	Samir Mohammed	203
Kishalay Datta	325	Sanjeev Kumar	167
Kishalay Datta	134	Santhosh Kumar D.	74
Kishalay Datta	137	Santhosh Kumar D.	84
Kishalay Datta	152	Saptarshi Saha	236
Kishalay Datta	155	Sarat Kumar Naidu	286
Kishalay Datta	161	Sarat Kumar Naidu	296
Kishalay Datta	211	Sarat Kumar Naidu	311
Kishalay Datta	252	Sarat Naidu	252
Kishalay Datta	258	Sathyanarayan T.B.	183
Kishalay Datta	286	Satyanarayana	56
Kishalay Datta	296	Sayani Banerjee	112
Kishalay Datta	301	Sayani Banerjee	126
Kishalay Datta	308	Selin Abraham	197
Kishalay Datta	311	Selin Abraham	244
Kondle Raghu	7	Shabbir Mohammed D.A.	203
Kritika Nanda	301	Shah V.	304
Lipoktemsü Jamir	278	Shah V.	316
Madhusudhanan M.	290	Shahid Khan	161
Manjunatha B.H.	23	Shahid Mustafa Khan	121
Manjunatha B.H.	27	Shahid Mustafa Khan	325
Manjunatha B.H.	79	Shyama	167
Mayank Kumar	130	Siddardh	319
Mohammad Kamal	290	Singh A.	275
Mohammed Ismail Nizami	217	Singh A.	293
Monil Patel	152	Singh Sonal	158
Monilkumar Patel	134	Sonal Singh	252
Moosa Al Abri	290	Sonal Singh	266
Muhammad Aamir Mir	301	Sri Harsha J.	231
Mukundkumar V. Patel	255	Srinivas Prabhu N.C.	231
Mundhe Sanjay A.	223	Sudip Chakraborty	236
Nagabhushana S.	190	Sujoy Das Thakur	112
Naidu S.K.	304	Sujoy Das Thakur	37
Narendra Kumar N.	217	Suman Kumar Kotwal	164
Nasir Shakilli	290	Surendra E.M.	27
Nikhilesh Das	16	Susmeet Mishra	282
O.R. Ranjan	231	Tamorish Kole	62
P. Anvesh	319	Umran Rafeeq Sheikh	308
P.V. Sai Satyanarayana	7	Umran R. Sheikh	325
Pankaj Jhaldiyal	130	V.G.R. Shastry	62
Patel M.	275	Varsha Koul	164
Prakash Kumar Koirala	45	Venkatesh Desai	97
Priya Govil	308	Venugopal A.V.	269
Puneeta Gupta	89	Vikram Shah	252
Rajesh Gupta	89	Vikram Shah	296
Ramya Kumari	7	Vinay Swamy P.M.	249
Ranganatha M.	190	Vinaya Swamy P.M.	79
Ravi Kirti	167	Virupakshappa V.	183
Rawat A.	264	Virupakshappa V.	190
Reeta Sood	89	Vivek Tirlapur	102
Rigenjyoti Kalita	134	Writuparna Ray	37
Rigenjyoti Kalita	137	Yatoo H.A.	272
		Yatoo H.A.	293