Indian Journal of Emergency Medicine

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Original Research Article

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Killip Classification and Glucose Levels in Patients with Acute Myocardial Infarction

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Abstract

Aim: Current study is designed to determine the significance of Killip classification and blood glucose levels in risk stratification of patients with Myocardial Infarction. *Method:* 100 patients admitted for the treatment of myocardial infarction during may 2011-2014. These patients were initially given diagnosis of acute MI upon admission to ED. Details regarding 2D echocardiography of the patient is noted. The details of any interventional strategies like CAG, PTCA, CABG were recorded. *Results:* Out of 100 patients 74 (74%) were males and 26 (26%) were females. Their mean age was 50.15 ± 14.04 years. data showing increase in the mean length of stay with increase in killips classification. Ventilator support is needed in killips classes III and IV patients when compared to killips classes Iand II. The need for ionotropic support increases as killips class advances from class II to IV. In IWMI 73.2% of the cases are of killips class I and 9.8% of the cases are of killips class IV. The high percentage of cases i.e 65.9% in killips class I under went coronary angiogram (CAG). Patients in killips class III has higher rates of readmission when compared to other classes i.e 33.3% of cases. Mortality is high in killips class III (42.9%) and class IV (57.1%). patients with initial high blood glucose values on admission have higher readmission rates. patients with initial high blood glucose values have high mortality rate. The mean age group in patients who were discharged home is 49.73 where as the mean age group in patients who were dead is 55.71. *Conclusion:* Patients with higher killip class on initial presentation have longer length of stay in hospital and high mortality rate. Combined together, killip classification & blood glucose levels are better indicators of morbidity and mortality than any one factor alone.

Keywords: Acute Myocardial Infarction; Killip classification; blood glucose levels.

Introduction

Myocardial infarction is one of the most common life threatening diagnoses in emergency hospital admissions. India is undergoing a rapid health transition with rising burden of coronary heart disease (CHD). Among adults over 20 yr of age, the estimated prevalence of CHD is around 3-4 per cent in rural areas and 8-10 per cent in urban areas, representing a two-fold rise in rural areas and a six-fold rise in urban areas [1]. Major risk factors dependent on socioeconomic levels are physical activity, dietary intake, smoking and tobacco use, overweight and obesity, high blood pressure, diabetes, cholesterol levels, the metabolic syndrome and psychosocial stress. In the last 30 years, the prevalence of hypertension and hypercholesterolemia has doubled while that of diabetes has trebled [1].

Myocardial infarction occurs when there is abrupt decrease in coronary blood flow following a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis. Most of the complications occur during the first few hours

while the patients are likely to be in the hospital. Although the mortality rate after admission for myocardial infarction has declined significantly over the last two decades but it still remains high [2]. Survival is markedly influenced by age of the patient, presence of different risk factors and complications that patients develop after myocardial infarction.

When patients with symptoms of MI are at first evaluated, clinicians make decisions based on the history, physical examination and ECG. ECG is generally the first investigation available for making a diagnosis in a patient presenting with acute severe chest pain. Tall T waves and ST elevation are the hallmarks of early presentation within minutes of onset of pain. Conventionally, AMI is diagnosed in the emergency based on ST segment elevation of more than 1.5 mm in 2 or more leads.

Criteria for MI [3] (European Society of Cardiologists), detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:

a) Symptoms of ischaemia. b) New or presumed new significant ST-segment–T wave (ST–T) changes or new left bundle branch block (LBBB). c) Development of pathological Q waves in the ECG. d) Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality. e) Identification of an intracoronary thrombus by angiography or autopsy.

Serum levels of cardiac enzymes and isoenzymes are essential to the diagnosis or exclusion of myocardial damage [4]. Elevated serum levels of biomarkers such as troponin (cTn) or the MB fraction of creatine kinase (CKMB) reflect myocardial injury leading to necrosis of myocardial cells [5].

In patients with acute ST elevation myocardial infarction treated with thrombolysis, the factors like hemodynamics, age, infarct location, history of diabetes, hypertension or angina and time to reperfusion therapy, all have an independent influence on clinical outcome.

The Killip classification system, for clinical assessment of patients with acute myocardial infarction (MI), stratifies individuals according to the severity of their post-MI congestive heart failure. A patient's Killip classification is defined by the following parameters: Killip class I**,** no CHF; Killip class II**,** third heart sound, rales; Killip class III**,** pulmonary edema; and Killip class IV, cardiogenic shock. Early primary angioplasty has contributed to a decrease in mortality in Killip IV patients [6]. Thus, the Killip classification, demonstrated in past decades to be effective in the risk stratification and prognostic evaluation of patients with acute MI, I is still used widely in the era of primary reperfuson, despite the more recent identification of other predictive indices for evaluation of acute MI.

Diabetes Mellitus is another feature that increases the mortality in patients with acute myocardial infarctions. Patient with stress hyperglycemia have more adverse events including heart failure, arrhythmias, heart block, re-infarction and mortality [7]. However, patients with certain risk factors, clinical features and associated conditions are more prone to develop complications and have a higher mortality rate.

While optimal fibrinolysis restores normal coronary flow in 50% to 60% of subjects, PCI is able to achieve restored flow in >90% of subjects. Acute reperfusion therapy using PCI or fibrinolytic therapy in patients with STEMI restores flow in the infarct-related artery, limits infarct size, and translates into early mortality benefit that is sustained over the next decade. PCI also results in a decreased risk of intracranial hemorrhage and stroke, making it the reperfusion strategy of choice in the elderly and those at risk for bleeding complications.

Current study is to determine the significance of Killip classification and blood glucose levels in risk stratification of patients with MI.

Materials and methods

Study Design: In this retrospective cohort study, we studied the records of all cases of myocardial infarction presented to ED from our registry. The protocol was approved by the institutional ethical committee.

Data Collection: All the materials for this study has been taken from 100 patients who got admitted to Narayana medical college hospital for the treatment of myocardial infarction from may 2011-2014. The study course is for a period of 3 yrs i.e from may 2011-2014. Data collected from medical records department & ED register.

Inclusion criteria:

1. Age of patient<85 yrs
2. Window period from onset of chest pain<24 hrs
3. Patients with STEMI & NSTEMI

Exclusion criteria

1. Age <18 yrs
2. Sepsis
3. Lack of drawn blood glucose levels
4. Previous history of CAD
5. Post CABG status
6. Window period>24 hrs
7. CKD

Methods

The studied patients were initially given diagnosis of acute MI upon admission to ED. The standard 12 lead electrocardiography recorded. Initial assessment and management of victim done as per ACLS protocols and vital signs are recorded. Baseline investigations like blood glucose levels, complete blood picture, renal function test, serial estimation of cardiac enzymes, serum lipid profile noted. Thrombolytic therapy is instituted if the patients presence with in the window period of 12 hours and if there are no contraindications. The patients were assigned killips classes based on chest ausculatory findings and blood pressure values. Details regarding 2D echocardiography of the patient is noted. The patients were observed during the course of their stay in the hospital for any recurrence of symptoms. The details of any interventional strategies like CAG, PTCA, CABG were noted. Data collection forms include the above data and date of disposition.

Primary Data Analysis

For categorical variables, percentages were calculated. For continuous variables, the values were represented as Mean ± SD. Chi-Square test and student t test were used to evaluate the association between various categorical and continuous variables. All p values were 2- sided and considered significant at $p<0.05$. All statistical operations were performed using IBM SPSS Ver. 20.0 for Windows (SPSS Inc, Chicago, III).

Results

Out of 100 patients 74 (74%) were males and 26 (26%) were females. Their mean age was 50.15 ± 14.04 years.

The mean age of patients in killips class I (48.90 ± 14.39), class II (50.10 ± 14.36), class III (52.09 ± 14.05, class IV (56.22 ± 10.08) (Table 1).

Out of 74 male patients 48 (64.9%) are in killips class I, 11 (14.9%) in killips class II, (8(10.8%) in killips class III and 7 (9.5%) are in killips class IV. Out of 26 female patients class I are 12 (46.2%), class II are 9 (34.6%), class III are 3 (11.5%) and class IV 2 (7.7%).

Mean length of stay in killips classes

The mean length of stay in killips class 1 (5.73 ± 2.30), class II (7.20 ± 1.99), class III (8.27 ± 3.90) class IV (8.00 ± 5.07). This data showing increase in the mean length of stay with increase in killips classification. Maximum length of stay is in class III.

Mean Blood glucose values values in killips classes

Patients with higher killips classes have higher blood glucose levels when compared to lower Killip classes. Mean blood glucose values of killips class I (126.93 ± 29.73), class II (182.10 ± 65.14) class III (246.18 ± 38.86), class IV (277.22 ± 85.25).

There is a decrease in mean systolic and diastolic blood pressure, as killips class advances from I-IV Mean Systolic blood pressure values in killips class I (126.37 ± 20.7), class II (133.80 ± 32.25), class III (121.64 ± 35.30), class IV (78 ± 6.56) (Table 2).

Table 1: Demographic data – Mean age among killips classes

	Killips					
	I (N=60)	**II (N=20)**	**III (N=11)**	**IV (N=9)**	**F value**	**P value**
Age	48.90 ± 14.39	50.10 ± 14.36	52.09 ± 14.05	56.22 ± 10.88	0.78	0.505

Table 2: SBP, DBP values in killips classes

	Killips					
	I (N=60)	**II (N=20)**	**III (N=11)**	**IV (N=9)**	**F value**	**P value**
SBP	126.37 ± 20.77	133.80 ± 32.25	121.64 ± 35.30	78 ± 6.56	11.73	<0.0001
DBP	82.30 ± 12.11	80.70 ± 14.53	78.73 ± 18.49	51.11 ± 9.28	14.71	<0.0001

Mean diastolic blood pressure class I (82.30 ± 12.11), class II (80.70 ± 14.53), class III (78.73 ± 18.49), class IV (51.11 ± 9.28). killips class IV indicates cardiogenic shock.

Out of patients who needs ventilator support 43.8% are of killips class III and 56.3% of patients are of killips class IV. Ventilator support is needed in killips classes III and IV patients when compared to killips classes Iand II (Table 3).

Out of patients who needs ionotropic support, 5.9% are of killips class II, 41.2% are of killips class III and 52.9% of patients are of killips class IV. The need for ionotropic support increases as killips class advances from class II to IV (Table 4).

From Table 5 we can assess that there is no significant relationship between the incidence of diabetes in patients with MI and Killips classification as the p-value is 0.400.

ECG characteristics in relation with killips classification

From table 7 we can assess that there is no significant relationship between type of MI and Killip classes as P-value is not significant (0.218).

Table 3: Patients on ventilator support in relation with killips class

	Killips					
Vent	**I**	**II**	**III**	**IV**	**Chi-square**	**p-value**
Positive	0 (0%)	0 (0%)	7 (43.8%)	9 (56.3%)	81.061	<0.0001
Negative	60 (71.4%)	20 (23.8%)	4 (4.8%)	0 (0%)		

Table 4: Patients on ionotropic support in killips classes

	Killips					
	I	**II**	**III**	**IV**	**Chi-square**	**p-value**
Ionotropes	0 (0.0%)	1 (5.9%)	7 (41.2%)	9 (52.9%)	75.227	<0.0001
No ionotropes	60 (72.3%)	19 (22.9%)	4 (4.8%)	0 (0.0%)		

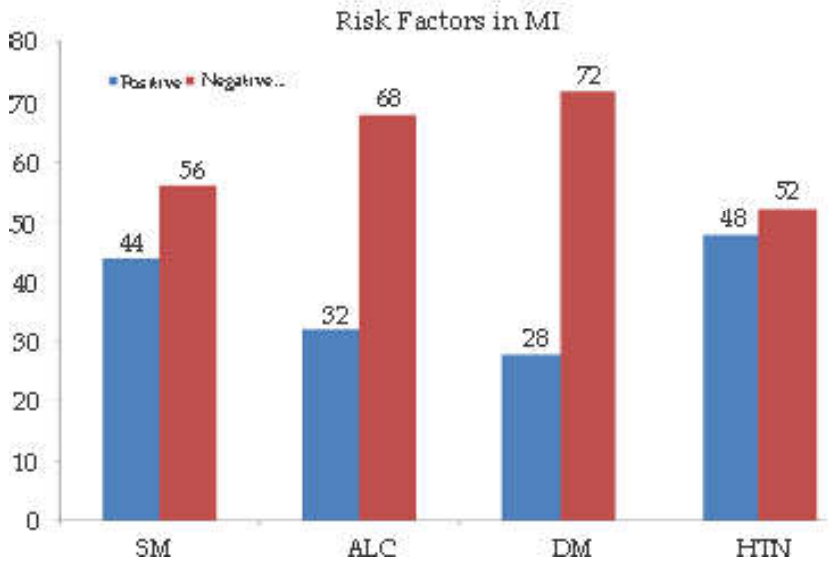

Fig. 1: Incidence of various risk factors

Killips classification in patients with Diabetis as risk factor

Table 5: Incidence of diabetes in killips classes

	Killips					
	I	**II**	**III**	**IV**	**Chi-square**	**p-value**
Diabetic	14 (50.0%)	7 (25.0%)	5 (17.9%)	2 (7.1%)	2.946	0.400
Non Diabetic	46 (63.9%)	13 (18.1%)	6 (8.3%)	7 (9.7%)		

In AWMI 46.2% of the cases are of killips class I and 11.5% of the cases are of killips class IV. In IWMI 73.2% of the cases are of killips class I and 9.8% of the cases are of killips class IV.

Relationship between killips classification and patients who subsequently under went CAG

The high percentage of cases i.e 65.9% in killips class I under went coronary angiogram (CAG). As the P-value is 0.001, the relation is highly significant.

Patients who subsequently underwent CAG in killips class I, 56 (65.9%), class II, 17 (20%), class III 8 (9.4%), class IV 4 (4.7%).

T-test paired sample t-test

Blood glucose values are compared with length of stay, triglyceride values and TIMI (Table 8).

A significant relation had been established between blood glucose values and length of stay.

Readmission in killips classes

There is a significant relationship between initial killips classification of the patient and readmission rates with killips class III as higher readmission rates.

Patients in killips class III has higher rates of readmission when compared to other classes i.e 33.3% of cases.

Relationship between killips classification and mortality

Mortality is high in killips class III (42.9%) and class IV (57.1%). Patients with higher killips classification has higher mortality rate. Maximum mortality is in killips class IV (Table 9).

Killips classification in patients with Hypertension as risk factor

Table 6: Incidence of Hypertension in killips classes

	Killips					
	I	**II**	**III**	**IV**	**Chi-square**	**p-value**
Hypertensive	27 (56.3%)	8 (16.7%)	5 (10.4%)	8 (16.7%)	6.786	0.79
Non Hypertensive	33 (63.5%)	12 (23.1%)	6 (11.5%)	1 (1.9%)		

Table 7: Type of MI and Killips classification

	Killips					
	I	**II**	**III**	**IV**	**Chi-square**	**p-value**
ALMI	6 (66.7%)	2 (22.2%)	0 (0%)	1 (11.1%)	18.914	0.218
ASMI	3 (60%)	1 (20%)	1 (20%)	0 (0%)		
AWMI	12 (46.2%)	6 (23.1%)	5 (19.2%)	3 (11.5%)		
ILMI	0 (0%)	0 (0%)	1 (100%)	0 (0%)		
IWMI	30 (73.2%)	6 (14.6%)	1 (2.4%)	4 (9.8%)		
NSTEMI	9 (50%)	5 (27.8%)	3 (16.7%)	1 (5.6%)		

Table 8: Relation between blood glucose, length of stay and triglycerides

		Mean ± SD	**t-value**	**p-value**
Pair 1	LOS	6.51 ± 2.92	-22.78	<0.0001
	CBG	164.61 ± 69.97		
Pair 2	CBG	164.61 ± 69.97	-1.25	0.215
	TGL	173.57 ± 36.08		

Table 9: Mortality rate in killips

	Killips					
	I	**II**	**III**	**IV**	**CHI-square**	**p-value**
Stable	60 (64.5%)	20 (21.5%)	8 (8.6%)	5 (5.4%)	32.350	< 0.0001
Death	0 (0.0%)	0 (0%)	3 (42.9%)	4 (57.1%)		

Table 10: Readmission with TIMI, TGL, BP, CBG, LOS

	Readmitted	No readmission	t-Value	P-value
TIMI	7.67 ± 2.456	5.33 ± 1.332	4.178	< 0.0001
TGL	182.05 ± 30.26	167.57 ± 33.87	1.763	0.081
SBP	127.43 ± 31.72	125.19 ± 26.03	0.329	0.743
DBP	80.67 ± 18.62	80.06 ± 13.98	0.163	0.871
CBG	199.29 ± 70.15	141 ± 44.9	3.598	0.001
LOS	7.95 ± 3.56	6.31 ± 2.59	1.975	0.059

Table 11: Disposition with TIMI, Age, LOS, BP, CBG, TGL

	Stable (N = 93)	Death (N= 7)	t-value	p-value
TIMI	5.86 ± 1.91	7.86 ± 1.77	-2.681	.009
AGE	49.73 ± 13.979	55.71 ± 14.773	-1.088	0.279
LOS	6.68 ± 2.901	4.29 ± 2.360	2.126	0.036
SBP	125.70 ± 27.249	86 ± 13.753	3.723	<0.0001
DBP	80.19 ± 15.04	60 ± 12.1	3.453	0.001
CBG	154.16 ± 56.8	303.4 ± 84.1	-6.470	<0.0001
TGL	170.84 ± 33.48	209.86 ± 51.2	-1.981	0.92

Table 12: Relation between TIMI risk score & Disposition

	Stable (N = 93)	Death (N= 7)	t-value	p-value
TIMI	5.86 ± 1.91	7.86 ± 1.77	-2.681	.009

The mean value of CBG in patients who were readmitted is 199.29 ± 70.15. Patients who were not readmitted is 141 ± 44.9 with the P value of 0.001. This shows that patients with initial high blood glucose values on admission have higher readmission rates (Table 10).

The mean TIMI in patients who were readmitted during follow period of one year is 7.67 while those who were not readmitted, the mean TIMI value is 5.33. This shows significant relation ship between TIMI and readmission.

The mean TIMI in killips class I is 5.57 ± 1.4, class II 5.90 ± 2, class III 6.64 ± 2, class IV 8.33 ± 2.29.

Out of 100 Patients, the mortality rate is 7%, the mean initial blood glucose values in patients with patients who were discharged home is 154.16 ± 56.8 while it is 303.4 ± 84.1 in patients who were dead. The P value is <0.0001. This shows that patients with initial high blood glucose values have high mortality rate (Table 11).

The mean age group in patients who were discharged home is 49.73 where as the mean age group in patients who were dead is 55.71.

The mean value of TIMI in patients who were discharged home is 5.86 where as in those who were dead is 7.86 (Table 12).

Discussion

Our study is a retrospective cohort study done in 100 patients of myocardial infarction who presented to Emergency Department of Narayana Medical college and Hospital. The patient s were initially stabilized according to ACLS protocols & evaluated.

Patients with acute myocardial infarction have abnormal glucose metabolism. Features adversely affecting in hospital mortality are Killips class on presentation, Age of patient, Initial blood glucose value.

Killip class was the most important variable in predicting death and survival after MI. Killips classification stratifies individuals according to the severity of their post-MI congestive heart failure.

Patients with hyperglycemia develop more adverse cardiovascular events as compared to patients with normal glucose tolerance. Patient s with elevated glucose levels could represent patients with increasing response to stress as seen in patient s with severe incidence of heart failure. We demonstrated positive relationship between initial glucose level & killip classification in acuteMI.

In our study, patients in killips class III and IV are older age group than those in killips class I and II, this is in accordance with the study conducted by Umesh. N. Khot. et al. [8]. In his study the mean age in Killips class I was 63 ± 11 & in killips class III & IV are 69 ± 11. In our study mean age in killips class I was 48 ± 14 and in class IV was 56 ± 10.

The mean age of patients with acute MI in our study is 50.15 ± 14.04 years. Mortality rate and

mean age was higher in killips class III and IV. This is in accordance with study conducted by Tesak. M et al. [9].

Out of 100 cases who suffered acute MI, 74 cases were males and 26 are females. This data indicates that myocardial infarction is more common in male gender.

Incidence of STEMI (82%) is more than NSTEMI (18%). In STEMI, IWMI (41 cases), AWMI (26 cases), ALMI (9 cases), ASMI (5 cases), ILMI (1 case).

Our study shows that out of 100 patients with myocardial infarction 40% of patients had heart failure while 60% of patients did not have heart failure, similar results were reported in the study done by Shahsawar. khan. Matiullah et al. [10]. In his study 37% had heart failure & 63% don`t have heart failure.

Out of patients who needs ventilator support 43.8% are of killips class III and 56.3% of patients are of killips class IV. Ventilator support is needed in killips classes III and IV patients when compared to killips classes Iand II.

Out of patients who needs ionotropic support, 5.9% are of killips class II, 41.2% are of killips class III and 52.9% of patients are of killips class IV. The need for ionotropic support increases as killips class advances from class II to IV.

The mean length of stay in hospital was increased in patients in killips class III (8.27 ± 3.9) & class IV (8.00 ± 5.07) when compared to class I (5.73 ± 2.30) & II (7.20 ± 1.99). In the study done by Hsien - Hung Cheng et al. [7] the mean length of stay in killips class I (7.9 ± 31), class II (7.1 ± 5.9) class III (13.1 ± 11.5) class IV (14.2 ± 10.2).

In our study, Patients with higher killips classification has high initial blood glucose levels i.e the mean blood glucose levels of killips class I (126.93 ± 29.73 mg/dl), Class II (182.10 ± 65.14 mg/dl), class III (246.18 ± 38.86 mg/dl), class IV (277.22 ± 85.28 mg/dl) is proved in the case study done by Hsien - hung Cheng. et al. [7]. In his study mean blood glucose valuesin class I (186.8 ± 82.5), class II (195.9 ± 76.7), class III (216.6 ± 121.9), class IV (236.2 ± 115.5) respectively.

In our study readmission rates are higher in killips class III when compared to class I and II. Killips class I and II showed a favourable prognostic parameter for 1 year survival, this is in accordance with study conducted by Amra-macic-Dzankovic et al. [11].

Patients with high initial blood glucose on admission had high morbidity rates. The mean blood glucose values in patients who are readmitted was 199.29 ± 70.15 mg/dl where as in patients who were not readmitted the mean blood glucose value was 141 ± 44.9 mg/dl.

Patients with high initial blood glucose levels have longer length of hospital stay and high data mortality, this is in accordance with study done by Hsien-hung Cheng et al. [7], Hong.pin Hsu et al. [12], Xue-Lian-Zhang et al. [13]. The similar data was proven in our study.

Patients with high TIMI has higher rates of readmission the mean value of TIMI in cases that were readmitted during 1 yr follow up is 7.67 ± 2.4 & in patients who are not admitted is 5.33 ± 1.3. patients with high TIMI has higher rates of readmission. In the studty done by R. J. Gumina et al. [14]. pt`s with mean value of TIMI >4 has worst long term prognosis than patients with mean TIMI <4.

Patients in higher killips classes have high TIMI risk score. The mean value of TIMI in killips class I (5.57 ± 1.484), II (5.90 ± 2.075), III (6.64 ± 2.461), IV (8.33 ± 2.29).

The mean blood glucose value in patients who are discharged home is 154.16 ± 56.8 mg/dl where as the mean blood glucose values in patients who are expired is 303 ± 84.1 mg/dl, this is in accordance with data collected by Damaris mudespacher et al. [15]. In this study mortality rates are high with initial blood glucose value on admission of 277.2 ± 72 mg/dl.

The relationship between killips classification and mortality was by Eftychio siniorakis et al. [16]. In his study mortality in killips class III was 19.3%, in killips class IV was 61.3%. In our study killips class III has mortality rate of 42.9%, class IV has mortality rate of 57.1%.

As killips classes advances from classes I to IV, the mean blood glucose value also increases. In our study survival rate is more in killips class I & II when compared to classes III & IV. Also 1 yr readmission rate is less in killips class I & II. Readmission rate is high in killips class III Readmission rate is decreased in killips class IV as mortality rate is high in this class (57.1%).

In the study of Ayman-El-Menyar et al. [17] patients with higher killips classification has higher mortality rate. The mortality rate in killips class III (27%). whereas in killips class IV it is 67% In our study also it is proven that mortality rates are high in killips class III (42%) and IV (57.1%).

In the setting of acute MI the patients with high

initial blood glucose levels develop more adverse cardiovascular events as compared to patients with normal glucose tolerance [18,19].

The results of our study indicate that Killip classification of patients at admission for acute MI continues to be a significant tool for early risk stratification and prediction of in-hospital and long-term survival. Patients in higher killips classification had higher morbidity, mortality, readmission rates. Patient s in higher killips classes had high initial blood glucose levels.

Conclusion

Patients with higher killips class on initial presentation have longer length of stay in hospital and high mortality rate. Patients with high initial blood glucose levels have high rates of readmission and mortality. Patients in higher killips classification have high blood glucose levels. Patients with high TIMI risk score are in higher killips classes. Combined together killip classification & blood glucose levels are better indicators of morbidity and mortality than any one factor alone.

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Validation of Emergency Severity Index and its Association with Patients' Vital Signs at Triage: A Prospective Observational Study

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Abstract

Background: To reduce bias in triage validity studies, one should focus on measures that can be obtained directly at triage. *Objectives:* To study the validity of Emergency Severity Index, Associations of the Emergency Severity Index triage categories with patients' vital signs at triage. *Methods and study design:* 260 patients were studied from March 2013 to June 2013.Each patient was triaged by the Researcher on duty and assigned a triage category using standard procedures. Immediately after the Researcher finished the triage assessment and reported the ESI triage category, the researcher registered patients' gender, referrer, main complaint, age and measured vital signs that were measured by the triage nurse. The following vital signs were registered: blood pressure, pulse rate, respiratory rate, oxygen saturation, temperature, the AVPU score (Alert, Voice, Pain and Unconsciousness), and pain, the data was analyzed using statistical designs and significant co relations between the study parameters were interpreted. *Results and Conclusion:* There is a significant correlation (p = 0.00) between the ESI scores and WPSS categories the findings of this study support the validity of the ESI as it showed that patients' vital signs are associated with the ESI triage categories. There were strong associations between the ESI triage categories and patients' WPSS scores at triage. However, no associations were found between pain scores and ESI triage categories, which indicates that ESI is a good triaging tool and reliability on pain scores should be revalidated.

Keywords: Severity Index; AVPU score; ESI scores.

Introduction

Emergency departments everywhere are faced with increasing numbers of patients presenting faster than they can be seen. Triage is the rapid and preliminary assessment of patients identifying those who need to be seen quickly and those who can wait. Additionally, there are patients who will not require major resources for assessment and treatment, and could be seen in a low-intensity (fast-track/minor emergency department) area or by physician extenders. Identifying these patients as they present would permit the emergency department to be decompressed, and allow resources to be invested in the sicker patients at the same time that the less acute and less resource-dependent patients have their needs met.

Internationally, several triage models are used, stratifying patients in categories based on acuity (from urgent to non-urgent). To sort the increasing number of patients presenting to emergency departments (EDs) on the urgency of their complaints, several triage systems have been developed and implemented [1,5,6,8]. Frequently mentioned triage systems in the literature are: the Australian Triage Scale, the Manchester Triage System, the Canadian Triage and Acuity Scale and the Emergency Severity Index (ESI) [7,9,10]. Compared to other triage systems, the ESI is different in that, as well as the level of urgency, it estimates the number of resources that patients need. ESI resources are defined as laboratory tests, radiology, intravenous fluids, specialty consultation, a simple or complex procedure

and intravenous, intramuscular or nebulized medications. Patients can be allocated into five urgency categories. For reasons of patient safety, it is important that ED triage systems are reliable and valid. Previous studies of the reliability of the ESI reported k scores 12 representing moderate to almost perfect reliability [2,3,4,8,10]. The reliability places an upper limit on the validity of triage.

To best of our knowledge, the ESI algorithm has been only validated in Dept. of emergency Netherlands and this study was proposed as we could not locate any study in Indian scenario.

Review of Literature

Evolution of triage systems

The French word "trier", the origin of the word "triage", was originally applied to a process of sorting, probably around 1792, by Baron Dominique Jean Larrey, Surgeon in Chief to Napoleon's Imperial Guard. Larrey was credited with designing a flying ambulance: the Ambulance Volante. Baron Francois Percy also contributed to the organisation of a care system for the ongoing management of casualties. Out of the French Service de Santé, not only emerged the concept of triage, but the organisational structure necessary to handle the growing number of casualties in modern warfare. As the complexity of healthcare systems increases, and as patient expectations rise, triage will be a vital tool in the first steps of patient management. Prioritisation and streaming underpinning triage systems must be developed using a common system across whole health economies.

The various triaging methods in vogue today are *Simple triage, Tags, Advanced triage, Continuous integrated triage, Reverse triage, Secondary (in-hospital) triage.*

Simple triage

Simple triage is usually used in a scene of an accident or "mass-casualty incident" (MCI), in order to sort patients into those who need critical attention and immediate transport to the hospital and those with less serious injuries. This step can be started before transportation becomes available. At its most primitive patients may be simply marked with coloured flagging tape or with marker pens. Pre-printed cards for this purpose are known as a triage tag [11].

Tags

Many triage systems use triage tags with specific formats. Triage tags may take a variety of forms. Some countries use a nationally standardized triage tag, [12] while in other countries commercially available triage tags are used, and these will vary by jurisdictional choice [13]. The most commonly used commercial systems include the Mettag, [14] the Smarttag, [15] E/T Light tm [16] and the Cruciform systems [17]. More advanced tagging systems incorporate special markers to indicate whether or not patients have been contaminated by hazardous materials, and also tear off strips for tracking the movement of patients through the process. Some of these tracking systems are beginning to incorporate the use of handheld computers, and in some cases, bar code scanners.

Typical triaging systems

Emergency Triage (E/T) Lights – particularly useful at night or under adverse conditions

Advanced triage

In advanced triage, doctors may decide that some seriously injured people should not receive advanced care because they are unlikely to survive. It is used to divert scarce resources away from patients with little chance of survival in order to increase the chances of survival of others who are more likely to survive. The use of advanced triage may become necessary when medical professionals decide that the medical resources available are not sufficient to treat all the people who need help. The treatment being prioritized can include the time spent on medical care, or drugs or other limited resources. This has happened in disasters such as volcanic eruptions, thunderstorms, and rail accidents.

Continuous integrated triage

Continuous Integrated Triage is an approach to triage in mass casualty situations which is both efficient and sensitive to psychosocial and disaster behavioral health issues that affect the number of patients seeking care (surge), the manner in which a hospital or healthcare facility deals with that surge (surge capacity) [18] and the overarching medical needs of the event.

Reverse triage

In addition to the standard practices of triage as mentioned above, there are conditions where sometimes the less wounded are treated in preference to the more severely wounded. This may arise in a situation such as war where the military setting may require soldiers be returned to combat as quickly as possible, or disaster situations where medical resources are limited in order to conserve resources for those likely to survive but requiring advanced medical care [19].

Undertriage and overtriage

Undertriage is the underestimating the severity of an illness or injury. An example of this would be categorizing a Priority 1 (Immediate) patient as a Priority 2 (Delayed) or Priority 3 (Minimal). Historically, acceptable undertriage rates have been deemed 5% or less. Overtriage is the overestimating of the severity of an illness or injury. An example of this would be categorizing a Priority 3 (Minimal) patient as a Priority 2 (Delayed) or Priority 1 (Immediate). Acceptable overtriage rates have been typically up to 50% in an effort to avoid undertriage. Some studies suggest that overtriage is less likely to occur when triaging is performed by hospital medical teams, rather than paramedics or EMTs [20].

Secondary (in-hospital) triage

In advanced triage systems, secondary triage is typically implemented by paramedics, battlefield medical personnel or by skilled nurses in the emergency departments of hospitals during disasters, injured people are sorted into five categories [21].

Limitations of current practices

Some of these limitations include:

Lacking the clear goal of maximizing the number of lives saved, as well as the focus, design and objective methodology to accomplish that goal (a protocol of taking the worst Immediate - lowest chances for survival - first can be statistically invalid and dangerous. Using trauma measures that are problematic (e.g. capillary refill) and grouping into broad color-coded categories that are not in accordance with injury severities, medical evidence and needs. Categories do not differentiate differences in injury severities and survival probabilities and are invalid based on categorical definitions and evacuation priorities ordering (prioritization) and allocating resources subjectively within Immediate and Delayed categories, which are neither reproducible nor scalable, with little chance of being optimal.

Evidence-based research indicates there are wide ranges and overlaps of survival probabilities of the Immediate and Delayed categories. Poor

Table 1: Emergency Severity Index

	ESI-1	ESI-2	ESI-3	ESI-4	ESI-5
Vital functions (ABC) and level of Consciousness	Unstable or unresponsive	Threatened or severe pain/distress	Stable	stable	stable
Life threat or organ threat	obvious	Reasonably likely	Unlikely (possible)	No	No
Requires resuscitation	Immediately	Sometimes	seldom	No	No
Expected resource use—x rays, labs, consultations, procedures	Maximum (>2)	High (>2)	Medium (>2)	Low (1)	Low (none)
Response time	Immediate team effect	minutes	Upto 1 hrs.	Can be delayed	Can be delayed

Table 2: WPSS - Worthing Physiological Scoring System Scores and interventions

Physiological Marker	Score 0	Score 1	Score 2	Score 3
Ventilatory Frequency	<19	20-21	>22	
Pulse	<101	>102		
Systolic B.P	>100	<90		
Temperature	>35.3			<35.3
Oxygen saturation in Air	96 to 100	94 to <96	92 to <94	<92
AVPU	Alert			other
Total score			Intervention	
Total score 0-1			Normal	
Total score 2-4			Alert	
Total score >5			Urgent	

assessments, invalid categories, no objective methodology and tools for prioritizing casualties and allocating resources, and a protocol of worst first triage provide some challenges for emergency and disaster preparedness and response.

Study Objectives

Primary

1. To study the validity of Emergency Severity Index.

Secondary

2. Associations of the Emergency Severity Index triage categories with patients' vital signs at triage.

Study Outcome

1. Patients ESI Score in ED.
2. WPSS Score to measure the association of ESI with Vital signs at Triage.

Materials and Methods

Study Design

We will conduct a prospective observational study. The Institutional Scientific And Ethics committees of the study site will approve the protocol, and patients who voluntarily provide written informed consent will be enrolled into the Study. We will perform the study from March 2013 to June 2013 in ED of Max Super Speciality Hospital, Patparganj, Delhi.

Selection of Participants

We will enroll a total of 260 patients from the ED of Max Super Speciality Hospital, Patparganj, Delhi. Patients should meet the eligible inclusion and exclusion criteria;

Inclusion Criteria

1. Age more than 18 years.
2. Presented to the Emergency department for medical ailment.

Exclusion Criteria

1. Patients on Ventilator

Data Collection

The data will be prospectively collected by the researcher on the duty days as per roster. A sample size calculation for regression analysis 20 estimated a minimum required sample size of 260 patients. A dropout rate of 5% due to unforeseen circumstances was taken into account in this calculation. Each patient will be triaged by the Researcher on duty and assigned a triage category using standard procedures. Immediately after the Researcher finished the triage assessment and reported the ESI triage category, the researcher registered patients' gender, referrer, main complaint, age and measured vital signs that will be measured by the triage nurse. The following vital signs will be registered: blood pressure, pulse rate, respiratory rate, oxygen saturation, temperature, the AVPU score (Alert, Voice, Pain and Unconsciousness), and pain. These will be measured by using an automated vital signs monitor, a thermometer and the numerical pain rating scale. The numerical pain rating scale score will be taken by asking patients to allocate a score between 0 and 10, with 0 indicating no pain and 10 the worst pain imaginable. All the data will be registered on to the Study Performa. The Worthing Physiological Scoring System (WPSS) is such a prognostic scoring system (Table 2). The WPSS is based upon identifying physiological markers for mortality at an early stage to undertake timely action. The system has been derived from and prospectively validated in ED patients and is therefore suitable for use in this study. 20 except for pain, the system consists of the vital signs used in the ESI as well as systolic blood pressure

Statistical Plan

Sample Size

On the basis of previous literature the predictive value of ESI score for hospitalization is 84%. For the calculation of sample size this information has been used. So with 5% margin of error and 95% level of significance we will recruit 260 subjects for the study.

- Chi-square and Fisher's exact test (categorical variables), t test (continuous variables).
- Ordinary logistic regression.
- Pearson correlation coefficients to determine the correlations between the ESI Scores and Vital signs.

Results

Male patients were 148 (56.92%) while the number

Table 3: Socio-Demographic profile the study Population

Sex	Freq.	Percent	Cum
Male	148	56.92	56.92
Female	112	43.08	100.00
Total	260	100	

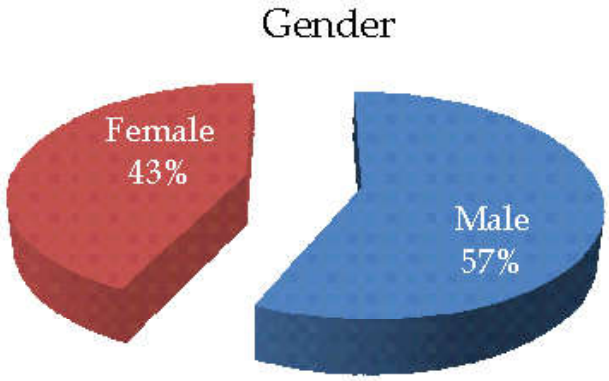

Fig. 3: Socio-Demographic profile the study Population

Table 4: Presenting Complaints

	Freq.	Percent	Cum
Cardiac	37	14.23	14.23
Respiratory	40	15.38	29.62
Gastric	42	16.15	45.77
Neurological	14	5.38	51.15
Poisoning	3	1.15	52.31
Miscellaneous	115	44.23	96.54
Fever	2	0.77	97.31
Renal	7	2.69	100.00
Total	260	100.00	

Table 5: ESI Category

	Freq.	Percent	Cum
ESI 1	9	3.46	3.46
ESI 2	57	21.92	25.38
ESI 3	107	41.15	66.54
ESI 4	84	32.31	98.85
ESI 5	3	1.15	100.00
Total	260	100.00	

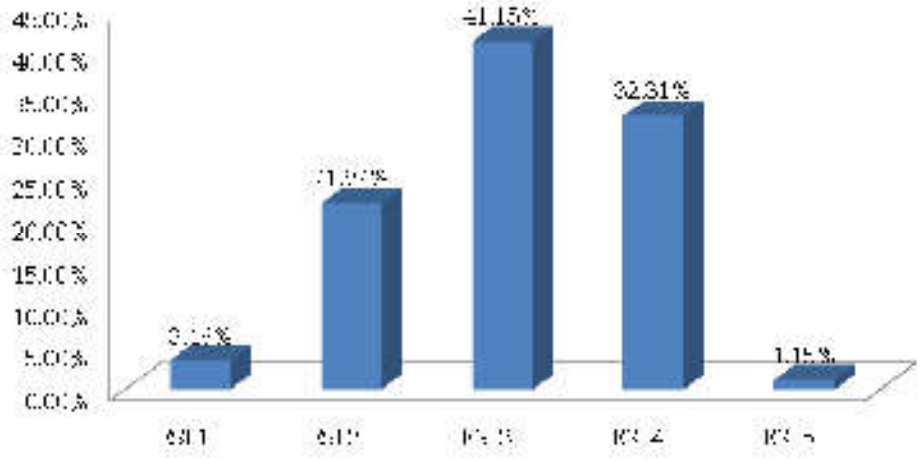

Fig 5: ESI Category

Table 6: Tabulation of WPSS Category

	Freq.	Percent	Cum
Normal	154	59.23	59.23
Alert	63	24.23	83.46
Urgent	43	16.54	100.00

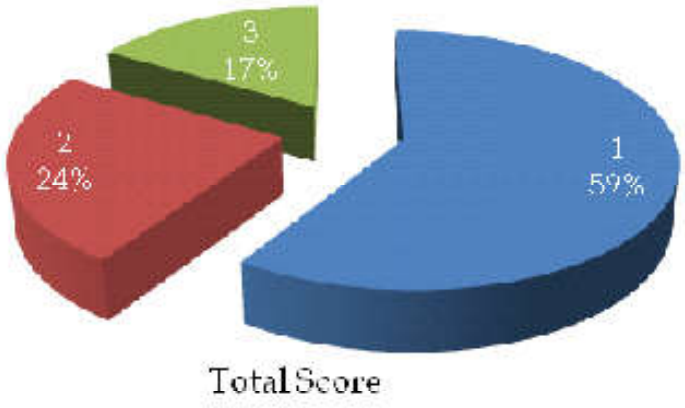

Fig 6: Tabulation of WPSS Category

Table 7: Tabulation of pain Scores

	Freq.	Percent	Cum
0-3	151	58.08	58.08
4-6	83	31.92	90.00
7-10	26	10.00	100.00
Total	260	100.00	

Table 8: Table Correlation of ESI with WPSS category

ESI	Normal	Alert	Urgent	Total
ESI 1	0	0	9	9
ESI 2	15	16	26	57
ESI 3	60	39	8	107
ESI 4	77	7	0	84
ESI 5	2	1	0	3
Total	154	63	43	260

Pearson $chi^2(8) = 135.6130$ Pr = 0.000

Fisher's exact = 0.000

Table 9: Table of ESI Correlation with Pain

ESI	0-3	4-6	7-10	Total
ESI 1	8	1	0	9
ESI 2	32	16	9	57
ESI 3	61	38	8	107
ESI 4	48	27	9	84
ESI 5	2	1	0	3
Total	154	63	43	260

Pearson chi^2 (8) = 7.3360 Pr = 0.501

Fisher's exact = 0.582

of females patients were 112 (43.08%). Most of the patients belong to the Elderly age group 31.92% (83) and middle age groups (from 31-45) 27.31% (71). The patients in the age group 46-60 were third highest to present in the emergency 23.46% (61), while the least patients come to the emergency in the age group 18-60 i.e. 17.31% (45).

Discussion

Most of the patient's falls under the criteria for ESI – 3 category 41.15% (107). The ESI – I category in which resuscitation and immediate attention is needed 9 patients (3.46%) were recorded. Patients in which the need for resuscitation is sometimes; however the risk of life is severe or threaded or in severe distress/pain i.e. ESI-2, are 57 (21.92%).

On correlating the ESI triage category with The WPSS scores it was noticed that the patients with ESI Category 1 were also been rated as Urgent in the WPSS, suggesting that care needed in these individual is urgent and needing immediate team management. In ESI-2 category which needs care in minutes as per the triage category, the WPSS scores were also reflecting the same with exception of One-fourth of patients (15/57) which is rated as WPSS category Normal. However, most, around Three-Fourth (16 plus 26 /57) were labeled as requiring urgent care.

Where as per Triage Category more Resources are utilized and patient can be managed till one hour we have seen that more than half of them were in the Normal category (60/107) of WPSS scoring and only less than 10% (8 /107) were labeled as urgent category. The same features have been noticed in ESI-4 and ESI -5,out of total 79 (77 in ESI-4 and 2 in ESI-5) none was labeled as the URGENT as the urgent intervention as per WPSS, and only 8 out of 89 were labeled as ALERT.

Most of them were in the WPSS category as Normal again reflecting that synchrony in the two scales. Same is also been proven as per results of statistical analysis, which shows the significant correlation between WPSS and ESI Triage Category ($p = 0.00$). Similar results has also been supported by the study by Inekewulp and Rullman 2012. These results are comparable to the findings of the Kim et al. who studied the ESI triage categories and APACHE II scores. the highest mean APACHE II scores were found in the highest triage Categories.

The correlation between the ESI category and the pain scores however does not reflect the same. As none of the patients with severe Pain were categorized in ESI triage category 1. Most of the Patients have been placed in Triage Category ESI-3 & 4.

Half of the patients with the moderate level of pain were placed in the ESI category -3, as these patients needs more of resource utilization and investigation to ascertain the cause of pain, however, they were not requiring the attention and management urgently.

Pain in some patients 9/84 though very disturbing however received ESI triage category 4 in view of stability of the vital signs and other parameters. Thus, supporting that pain alone may not be taken as parameter as escalation of higher ESI category.

Also as per the statistical analysis there is no co-relation between the ESI Triage category and the Pain Scores ($p = 0.582$). The Above finding are in line with study by Inekewulp and Rullman 2012.

A remarkable finding of this study is that pain, a discriminator of the system (ESI category 2), was not associated with urgency. The ESI guidelines state that it is up to the discretion of the triage nurse to indicate whether a patient's pain score is supported by his/her clinical condition and warrants triage in higher ESI categories. However, the guidelines have not specified how to make this decision. This could be an explanation for the lack of association between pain scores and urgency. To increase this association, and consequently the validity of the ESI, a revision of the guidelines is necessary. More specifically, the guidelines should describe symptoms or patient behaviour related to severe pain. Further studies will be needed to measure the effects of such a revision on the reliability and validity of the ESI.

Limitations

The Results of the study has to be interpreted carefully because of several limitations.

Firstly it was an unicentric study, data was collected from one emergency during day hours, so hospital factors or factors associated with data collection shifts could not be accounted.

Secondly, the researcher is present in the triage Area to collect data and direct contact with study objects. Blinding in this study was not a possibility as it may resulted in other major biases.

Conclusions

This study clearly shows the strong association of patients' vital signs with the ESI triage categories. Study findings failed to support any association between pain scores and ESI triage categories, which indicated that apart from a revision of Triage guidelines, pain scoring systems should not be relied upon for triaging patients.

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To Determine Severity and Prognostic Factors of Patients Admitted in Emergency with Community Acquired Pneumonia

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Abstract

Aim: The aim of the study is to determine systematically the performance of existing clinical prediction score (SMART_COP, CURB_65, PSI, ATS/ IDSA, SCAP Score) to risk stratify the Emergency department patients with Community acquired pneumonia. *Method:* This study was conducted on 80 patients presented to the department of emergency medicine, during July 2016 to November 2019. *Results:* Maximum age group of 51-70 with mean age 57.5 ± 14.78 years. Smoking about 75% males have smoking as risk factor and the most common comorbid condition is hypertension. Among 80 patients, 10 have CURB_65 score of 0, 24 patients have score of 1, 30 patients have score of 2, 11 patients have score of 3, 3 patients have score of 4 and 2 patients have score of 5. Among 80 patients, 19 patients has SCAP major criteria and 36 patients had minor criteria. The mortality was seen in about 10 patients. SMART_COP has highest AUC value among different pneumonia severity score for predicting the need of vasopressor support and SMART_COP score > 5 have good accuracy in predicting the need of vasopressor support in patients with CAP. *Conclusion:* Early microbiological diagnosis, early antibiotic administration in patients with SMART_COP score > 4 and PSI class 4 and 5 can decrease the morbidity and mortality in CAP patients.

Keywords: Pneumonia; Vasopressor; SMART_COP.

Introduction

Pneumonia is defined as an acute infection of the lung parenchyma, with symptom onset in the community [1]. Community-acquired pneumonia (CAP), "*Captain of the men of Death*" as described by Sir William Osler in 1982 still remains a major cause for morbidity and mortality despite all highly sophisticated advances in both diagnosis and therapeutic management of community acquired pneumonia [2,3]. Community acquired pneumonia (CAP) is a common disorder with an incidence of about 20% to 30% in developing countries compared to an incidence of 3% to 4% in developed countries [4-6].

Severe CAP is defined as a pneumonia requiring supportive therapy within a critical care environment that is associated with a higher mortality rate. Severe CAP is frequently a multisystem disease and patients will often present with multiple organ failure [7].

Despite their widespread use in clinical practice, traditional markers such as severity of disease estimation by the patient, fever, or white blood cell counts do not reliably assess disease severity and mortality risk [8].

Mortality reduction can be achieved by correct prediction rule that allows physicians to select patients with severe CAP who require ICU treatment early in the course of illness facilitates the appropriate initial management and antibiotic treatment [9]. Number of studies suggest that routine clinical judgment is often not sufficient for assessing the severity of CAP [10].

Clinical judgment alone may underestimate its severity and lead to variations in rates of admission to the hospital and intensive care unit (ICU). In addition, the decision to admit a patient to the ICU based on clinical judgment alone has been found to be sub optimal. In this light, validated clinical prediction rules for CAP management offer a useful adjunct to the art of clinical practice.

In developing countries such as India where half population is low economical status site of care is major burden for the patients. Determination of extent of disease severity in vital in optimizing therapeutic options such as requirement of invasive or non invasive ventilator support, need of vasopressor or inotropic support, path of treatment, whether patient can be discharged home, diagnostic strategy and oral or intravenous antibiotics [11].

The aim of the study is to validate the significance of clinical prediction scores in prognosis of severity of Community Acquired Pneumonia and to determine site of care decisions based on clinical prediction scores.

Materials and methods

Study Design

This is a prospective study done in a tertiary care hospital during July 2016 to November 2018 in Department of Emergency Medicine. A total of 80 patients presented to emergency department wit Community acquired pneumonia are selected for the study.

Inclusion Criteria

1. Signs and symptoms suggestive of Community Acquired Pneumonia.
2. Evaluation variables performed with in 24 hrs.

Exclusion Criteria

1. Age <18 and >80 years.
2. Previously admitted in a hospital for >48.
3. Presence of structural lung disease.
4. Broad-spectrum antibiotic therapy (lasted for at least 7 days in the past month).
5. Corticosteroid therapy with at least 10mg of prednisone per day.
6. Malnutrition.
7. Neutropenic patients.
8. Chemotherapy patients.
9. Patients with HIV related disorders.
10. Transplant recipients.

Results

Age distribution: Among patients studied there were totally 80 patients among which maximum number of patients 42 patients belong to age group between 51-70 (52.5%) followed by 21 patients between age 31-50 (26.3%). 5 patients belong to age < 30 (6.3%) and 12 (15%) patients belong to age >70 as shown in the table. The mean age of the study is 57.5 ± 14.78.

Smoking distribution: 48.75% patients has smoking as risk factor and 51.25% patients do not have smoking as risk factor. Among 55% of male 75% males have smoking as a risk factor.

Comorbidities: The most common comorbid condition is hypertension in 46 patients followed by diabetes mellitus in 36 patients, COPD in 20 patients, Cerebrovascular accident in 8 patients, Coronary artery disease in 5 patients, Tuberculosis and Chronic kidney disease in one patient each.

Distribution of need of vasopressors: In present study among 80 patients 20 (25%) patients required vasopressor support.

Ventilator support: Need for ventilator support studied among the patients studied among 80 patients 52 (65%) patients required ventilator support, which includes both invasive and non-invasive ventilation, and 28 (35%) patients did not require ventilator support.

Distribution of outcome: among 80 patients 70 (87.5%) patients was discharged and mortality was seen in 10 patients (12.5%).

Distribution of patients admitted in ICU and Ward: among 80 patients, 61 (76.3%) patients were admitted in ICU and 19 (23.8) patients were admitted in ward.

Table 1: Distribution of laboratory parameters of patient studied

Laboratory parameters	Number of patients (n=80)	Percentage (%)
Blood urea ≥ 20 mmol/lt		
Yes	75	93.75
No	5	6.25
Sodium ≤ 130		
Yes	18	22.5
No	62	77.5
RBS >250		

Yes	40	50.0
No	40	50.0
HCT ≤ 30%		
Yes	42	52.5
No	38	47.5
Ph ≤ 7.35		
Yes	33	41.25
No	47	58.75
Serum Albumin (< 35)		
Yes	16	20.0
No	64	80.0
Pleural effusion		
Yes	38	47.5
No	42	52.5
Chest X-Ray (ML)		
Yes	39	48.75
No	41	51.25

SMART_COP score: 36 patients belong to low risk, 21 patients to moderate risk, 10 (12.5%) patients to high risk and 13 patients (16.3%) belonged to very high risk need of IRVS.

CURB65 score: among patients studied 10 (12.5%) belonged to Class 0, 24 (30.0) patients belonged to Class 1, 30 patients (37.5%) belonged to class 2, 11 (13.8%) patients belonged to class 3, 3 (3.8%) patients belonged to class 4, 2(2.5%) patients belonged to class 5.

SCAP_MAJOR score: among 80 patients 19 (23.75%) patients were met with SCAP major criteria.

SCAP_MINOR score: among 80 patients 36 (45%) patients were met with SCAP minor criteria.

ATS_MAJOR score: among 80 patients 16 (20%) patients were met with ATS/IDSA major criteria.

ATS/IDSA minor criteria: 34 (42.5%) patients were met with ATS/IDSA minor criteria.

Sputum Culture: 25 patients had shown streptococcus in culture, 18 pts had shown sthaphylococcus, 14 had shown klebsiella, 3 had shown E.coli, pseudomonas is seen in 12 and others in 9 pts.

Table 2: Comparison of various clinical variables with SMART COP

Clinical Variables	SMART_COP_GRP (Mean ± SD)					F Value	p Value
	Low Risk [n = 36]	Moderate Risk [n = 21]	High Risk [n = 10]	Very High Risk [n = 13]	Total [n = 80]		
AGE	59.36 ± 13.55	56.14 ± 15.76	53.3 ± 18.73	57.77 ± 14	57.5 ± 14.78	0.51	0.68
GCS	15 ± 0.0	14.43 ± 2.4	15 ± 0.0	14.38 ± 1.66	14.75 ± 1.4	1.16	0.33
SBP	124.72 ± 22.36	130.95 ± 28.62	104 ± 30.98	90.77 ± 32.26	118.25 ± 30.14	7.71	0.00**
DBP	76.67 ± 13.31	77.62 ± 14.8	63 ± 17.67	55.38 ± 18.98	71.75 ± 17.27	8.35	0.00**
HR	111.28 ± 18.95	109.19 ± 21.46	101.2 ± 23.4	137.54 ± 20.25	113.74 ± 22.84	7.68	0.00**
RR	31.83 ± 9.69	35.33 ± 8.74	33.6 ± 9.16	44.31 ± 4.33	35 ± 9.62	6.60	0.00**
TEMP	99.49 ± 1.29	99.2 ± 1.2	99.08 ± 1.01	100.34 ± 1.3	99.5 ± 1.28	2.79	0.049*
CBG	242.42 ± 102.05	220 ± 110.46	201.8 ± 91.02	242.46 ± 119.8	231.46 ± 105.11	0.52	0.67
HB	11.19 ± 2.03	10.78 ± 2.51	9.97 ± 1.52	10.78 ± 2.67	10.86 ± 2.22	0.81	0.49
TC	15950 ± 5218.95	16023.81 ± 4972.01	11440 ± 4021.66	16669.23 ± 8292.1	15522.5 ± 5752.29	2.05	0.11
PLT	227388.89 ± 85624.86	272519.05 ± 152938.77	275110 ± 104657.37	238823.08 ± 141591.16	247058.75 ± 117992.64	0.86	0.46
UREA	45.69 ± 24.28	56.51 ± 40.76	47.82 ± 28.95	67.5 ± 43.46	52.34 ± 33.61	1.55	0.21
S_CR	1.36 ± 0.61	1.55 ± 0.92	1.63 ± 1.02	2.18 ± 1.58	1.58 ± 0.98	2.38	0.08
NA	133.14 ± 8.21	135.95 ± 9.16	137.1 ± 4.68	135.38 ± 6.69	134.74 ± 7.92	0.98	0.41
K	4 ± 0.65	3.87 ± 0.9	4.35 ± 0.47	4.38 ± 0.53	4.07 ± 0.71	2.16	0.10
CL	96.86 ± 3.85	94.86 ± 20.12	97.4 ± 3.63	99.15 ± 4.52	96.78 ± 10.75	0.44	0.73
HCT	31.14 ± 4	30.33 ± 4.76	29.2 ± 2.62	31.92 ± 5.27	30.81 ± 4.3	0.91	0.44
S_ALB	40.01 ± 6.12	40.24 ± 3.16	36.5 ± 5.97	32.54 ± 3.91	38.42 ± 5.8	8.14	0.00**
Ph	7.4 ± 0.06	7.35 ± 0.1	7.31 ± 0.12	7.26 ± 0.12	7.35 ± 0.1	7.16	0.00**
pCO_2	33.32 ± 7.18	34.17 ± 13.57	37.54 ± 13.25	38.02 ± 18.73	34.84 ± 12.1	0.67	0.58
pO_2	163.73 ± 63.94	105.91 ± 54.36	88.29 ± 25.49	108.15 ± 43.64	130.09 ± 62.4	8.42	0.00**
HCO_3	18.91 ± 3.89	19.83 ± 6.17	19.38 ± 4.93	18.65 ± 7.77	19.17 ± 5.33	0.18	0.91
SaO_2	98.41 ± 3.34	95.64 ± 4.18	95.06 ± 5.26	93.41 ± 5.41	96.45 ± 4.55	5.52	0.00**
LAC	1.54 ± 0.94	2.07 ± 0.79	2.19 ± 1.37	4.91 ± 1.44	2.31 ± 1.57	32.83	0.00**
LOS	5.19 ± 1.65	5.62 ± 1.16	6.6 ± 1.35	6.31 ± 3.2	5.66 ± 1.9	2.14	0.10

p < 0.05 - Significant, **p < 0.0001 - Very High Significant

Table 3: Comparison of various clinical variables with CURB 65

Clinical Variables	CURB_65 [Mean ± SD]							F Value	p Value
	0.0 [n = 10]	1.0 [n = 24]	2.0 [n = 30]	3.0 [n = 11]	4.0 [n = 3]	5.0 [n = 2]	Total [n = 80]		
AGE	54.7 ± 10.92	52.5 ± 15.15	57.17 ± 15.66	67.36 ± 9.41	73.33 ± 10.41	58.5 ± 13.44	57.5 ± 14.78	2.52	0.04*
GCS	15 ± 0	14.5 ± 2.25	14.97 ± 0.18	14.45 ± 1.81	15 ± 0	14.5 ± 0.71	14.75 ± 1.4	0.47	0.80
SBP	116 ± 10.75	132.08 ± 27.5	111.67 ± 28.9	126.36 ± 37.76	76.67 ± 5.77	80 ± 0	118.25 ± 30.14	3.84	0.00**
DBP	74 ± 12.65	78.33 ± 14.04	68.33 ± 17.24	74.55 ± 22.07	50 ± 0	50 ± 14.14	71.75 ± 17.27	2.93	0.02*
HR	98.7 ± 18.1	108.13 ± 20.11	114.73 ± 18.52	133.18 ± 26.74	129.67 ± 31.79	110.5 ± 43.13	113.74 ± 22.84	3.56	0.01*
RR	26.7 ± 6.04	34.25 ± 9.74	34.87 ± 10.14	40.09 ± 4.41	43 ± 7.55	47.5 ± 3.54	35 ± 9.62	3.80	0.00**
TEMP	99.56 ± 1.33	99.31 ± 1.28	99.27 ± 1.15	100.09 ± 1.54	100.2 ± 1.39	100.5 ± 0.71	99.5 ± 1.28	1.20	0.32
CBG	228.5 ± 97.22	225.33 ± 113.18	236.07 ± 110.5	234.82 ± 95.34	214.33 ± 78.36	258 ± 178.19	231.46 ± 105.11	0.07	1.00
HB	10.86 ± 1.78	11.3 ± 2.08	10.25 ± 2.12	11 ± 2.89	11.4 ± 3.14	13.3 ± 0	10.86 ± 2.22	1.17	0.33
TC	14320 ± 3651.73	15150 ± 4815.64	14203.33 ± 6423.1	19636.36 ± 5529.24	21366.67 ± 6619.92	14400 ± 3111.27	15522.5 ± 5752.29	2.37	0.047*
PLT	220400 ± 83764.48	271754.17 ± 140662.83	240130 ± 110470.64	245336.36 ± 134294.25	239666.67 ± 102163.27	208500 ± 12020.82	247058.75 ± 117992.64	0.36	0.87
UREA	34.41 ± 18.91	38.07 ± 28.92	52.97 ± 28.19	90.72 ± 36.28	82.33 ± 33.71	47.75 ± 35	52.34 ± 33.61	6.44	0.00**
S_CR	1.07 ± 0.84	1.08 ± 0.44	1.78 ± 0.84	2.28 ± 1.55	2.67 ± 0.58	1.55 ± 1.06	1.58 ± 0.98	4.86	0.00**
NA	131.2 ± 6.18	136.08 ± 10.76	133.87 ± 6.66	136.09 ± 5.65	135 ± 4.58	141.5 ± 0.71	134.74 ± 7.92	0.96	0.45
K	3.92 ± 0.52	3.99 ± 0.86	4.11 ± 0.73	4.11 ± 0.44	4.57 ± 0.8	4.25 ± 0.07	4.07 ± 0.71	0.48	0.79
CL	96.4 ± 4.2	94.5 ± 18.78	97.53 ± 3.95	98.36 ± 3.8	99.33 ± 3.06	102 ± 1.41	96.78 ± 10.75	0.41	0.84
HCT	29.6 ± 3.89	31.75 ± 4.75	29.77 ± 3.86	30.91 ± 3.59	32.33 ± 4.93	38.5 ± 0.71	30.81 ± 4.3	2.27	0.06
S_ALB	41.4 ± 1.84	40.33 ± 2.58	37.15 ± 7.52	38.36 ± 5.28	30.67 ± 3.06	31.5 ± 6.36	38.42 ± 5.8	3.44	0.01*
Ph	7.38 ± 0.06	7.39 ± 0.1	7.35 ± 0.11	7.29 ± 0.09	7.26 ± 0.13	7.33 ± 0.03	7.35 ± 0.1	2.19	0.07
pCO_2	35.35 ± 9.26	34 ± 12.35	32.88 ± 10.67	44.66 ± 14.76	25.8 ± 12.17	31.1 ± 7.21	34.84 ± 12.1	2.15	0.07
pO_2	152.51 ± 40.28	128.8 ± 69.22	125.25 ± 61.63	133.08 ± 73.31	136.2 ± 61.37	80.5 ± 21.92	130.09 ± 62.4	0.54	0.74
HCO_3	20.5 ± 4.25	20.16 ± 5.57	18.09 ± 4.2	21.03 ± 7.08	13.33 ± 7.57	15.3 ± 0.42	19.17 ± 5.33	1.82	0.12
SaO_2	99.5 ± 1.27	96.68 ± 3.56	96.67 ± 4.01	93.03 ± 7.47	96 ± 5.29	94.8 ± 3.96	96.45 ± 4.55	2.44	0.04*
LAC	1.24 ± 0.49	1.66 ± 0.75	2.38 ± 1.44	3.83 ± 2.03	4.33 ± 2.89	3 ± 1.41	2.31 ± 1.57	6.59	0.00**
LOS	5 ± 1.89	5.25 ± 1.54	5.73 ± 1.36	5.82 ± 1.99	6.33 ± 3.06	11 ± 4.24	5.66 ± 1.9	4.59	0.00**

*$p < 0.05$ - Significant, **$p < 0.0001$ - Very High Significant

Table 4: Comparison of various clinical variables with PSI GRP

Clinical Variables	PSI_GRP (Mean ± SD)					F Value	p Value
	Class-II [n=4]	Class-III [n=5]	Class-IV [n=53]	Class-V [n=18]	Total [n=80]		
AGE	66.25 ± 10.9	61.8 ± 4.32	53.32 ± 15.23	66.67 ± 10.64	57.5 ± 14.78	4.99	0.00**
GCS	15 ± 0	15 ± 0	14.74 ± 1.52	14.67 ± 1.41	14.75 ± 1.4	0.12	0.95
SBP	130 ± 34.64	128 ± 25.88	117.55 ± 28.75	115 ± 35.36	118.25 ± 30.14	0.45	0.72
DBP	75 ± 23.8	78 ± 10.95	71.89 ± 15.82	68.89 ± 21.66	71.75 ± 17.27	0.42	0.74
HR	98.75 ± 15.39	106.4 ± 16.02	108.77 ± 19.91	133.72 ± 23.27	113.74 ± 22.84	7.76	0.00**
RR	21.75 ± 3.95	26.2 ± 5.97	34.09 ± 9.08	43.06 ± 5.57	35 ± 9.62	11.65	0.00**
TEMP	99.15 ± 1.24	99.16 ± 0.77	99.39 ± 1.27	99.99 ± 1.39	99.5 ± 1.28	1.23	0.31
CBG	131.5 ± 11.36	197.2 ± 64.92	222.55 ± 102.15	289.44 ± 109.11	231.46 ± 105.11	3.68	0.02*
HB	11.23 ± 1.61	12.18 ± 0.86	10.84 ± 2.13	10.48 ± 2.77	10.86 ± 2.22	0.80	0.50
TC	15250 ± 3570.71	11900 ± 2952.96	15162.26 ± 5512.16	17650 ± 6888.46	15522.5 ± 5752.29	1.59	0.20
PLT	241500 ± 87857.84	231800 ± 85106.99	254301.89 ± 122261.65	231205.56 ± 124107.53	247058.75 ± 117992.64	0.20	0.90
UREA	37.8 ± 9.38	37.46 ± 19.66	47.48 ± 29.81	74.03 ± 41.67	52.34 ± 33.61	3.81	0.01*
S_CR	1.25 ± 0.5	1.17 ± 0.26	1.51 ± 0.86	1.98 ± 1.36	1.58 ± 0.98	1.58	0.20
NA	143 ± 4.9	135.2 ± 7.79	134.49 ± 8.4	133.5 ± 6.25	134.74 ± 7.92	1.66	0.18
K	4.18 ± 0.69	3.42 ± 0.29	4.15 ± 0.79	4.01 ± 0.43	4.07 ± 0.71	1.76	0.16
CL	98.75 ± 4.57	97.6 ± 2.97	96.02 ± 12.89	98.33 ± 4.2	96.78 ± 10.75	0.26	0.85

HCT	31.75 ± 4.65	34 ± 3.39	30.7 ± 4.26	30.06 ± 4.45	30.81 ± 4.3	1.19	0.32
S_ALB	39.75 ± 4.27	41.8 ± 2.05	38.44 ± 6.35	37.11 ± 4.85	38.42 ± 5.8	0.94	0.43
Ph	7.41 ± 0.04	7.43 ± 0.04	7.36 ± 0.1	7.3 ± 0.11	7.35 ± 0.1	3.36	0.02*
pCO_2	35.58 ± 6.79	32.26 ± 6.48	32.07 ± 10.91	43.53 ± 13.89	34.84 ± 12.1	4.67	0.01*
pO_2	124.33 ± 97.21	140.16 ± 62.69	125.87 ± 59.01	140.99 ± 68.06	130.09 ± 62.4	0.31	0.82
HCO_3	20.75 ± 1.58	21.04 ± 4.75	18.71 ± 5.38	19.65 ± 5.96	19.17 ± 5.33	0.49	0.69
SaO_2	97 ± 2.58	98.2 ± 2.68	97.06 ± 3.51	94.05 ± 6.87	96.45 ± 4.55	2.38	0.08
LAC	1.03 ± 0.45	0.9 ± 0.64	2 ± 1.16	3.91 ± 1.81	2.31 ± 1.57	13.39	0.00**
LOS	3.75 ± 2.75	4.6 ± 0.55	5.98 ± 1.77	5.44 ± 2.04	5.66 ± 1.9	2.61	0.06

*p < 0.05 - Significant, **p < 0.0001 - Very High Significant

Table 5: Comparison of various clinical variables with SCAP MAJOT

Clinical Variables	SCAP_MAJOR [Mean + SD]			t Value	P Value
	No [n=61]	Yes [n=19]	Total [n=80]		
AGE	58.25 ± 15.02	55.11 ± 14.1	57.5 ± 14.78	0.81	0.42
GCS	14.98 ± 0.13	14 ± 2.79	14.75 ± 1.4	1.54	0.14
SBP	126.39 ± 23.6	92.11 ± 34.41	118.25 ± 30.14	4.93	0.00**
DBP	76.72 ± 13.87	55.79 ± 17.74	71.75 ± 17.27	5.36	0.00**
HR	109.59 ± 20.25	127.05 ± 26.04	113.74 ± 22.84	-3.06	0.00**
RR	32.82 ± 9.09	42 ± 7.9	35 ± 9.62	-3.96	0.00**
TEMP	99.31 ± 1.23	100.12 ± 1.28	99.5 ± 1.28	-2.43	0.02*
CBG	229.62 ± 98.81	237.37 ± 126.07	231.46 ± 105.11	-0.28	0.78
HB	10.86 ± 2.07	10.89 ± 2.71	10.86 ± 2.22	-0.05	0.96
TC	15455.74 ± 5187.28	15736.84 ± 7447.61	15522.5 ± 5752.29	-0.19	0.85
PLT	242672.13 ± 114775.12	261142.11 ± 130067.7	247058.75 ± 117992.64	-0.59	0.56
UREA	50.37 ± 31.8	58.66 ± 39.12	52.34 ± 33.61	-0.94	0.35
S_CR	1.45 ± 0.77	1.99 ± 1.41	1.58 ± 0.98	-1.60	0.12
NA	134.36 ± 8.47	135.95 ± 5.84	134.74 ± 7.92	-0.76	0.45
K	3.99 ± 0.73	4.34 ± 0.58	4.07 ± 0.71	-1.90	0.06
CL	96.18 ± 12.08	98.68 ± 3.96	96.78 ± 10.75	-0.89	0.38
HCT	30.64 ± 3.84	31.37 ± 5.6	30.81 ± 4.3	-0.53	0.60
S_ALB	40.24 ± 5.06	32.58 ± 3.92	38.42 ± 5.8	6.05	0.00**
Ph	7.38 ± 0.08	7.26 ± 0.12	7.35 ± 0.1	3.96	0.00**
pCO_2	33.8 ± 9.33	38.16 ± 18.37	34.84 ± 12.1	-1.00	0.33
pO_2	137.42 ± 66.8	106.56 ± 38.03	130.09 ± 62.4	2.53	0.01*
HCO_3	19.69 ± 4.71	17.51 ± 6.86	19.17 ± 5.33	1.57	0.12
SaO_2	97.26 ± 3.97	93.86 ± 5.38	96.45 ± 4.55	2.98	0.00**
LAC	1.75 ± 0.95	4.1 ± 1.84	2.31 ± 1.57	-5.35	0.00**
LOS	5.52 ± 1.42	6.11 ± 2.96	5.66 ± 1.9	-0.83	0.42

*p < 0.05 - Significant, **p < 0.0001 - Very High Significant

In the present study among 80 patients 36 patients had ATS/IDSA minor criteria and 44 patients did not have minor criteria. The mean age for patients who had minor criteria was 56.47 ± 14.08. The mean SBP for patients who had minor criteria was 110. ± 37.25. The mean DBP for patients who had minor criteria was 67.35 ± 20.5. The mean HR for patients who had minor criteria was 123.5 ± 23.8. The mean RR for patients who had major criteria was 39.88 ± 7.46. The mean temperature for patients who had minor criteria was 99.73 ± 1.29. The mean serum creatinine for patients who had minor criteria was 2.02 ± 1.22. The mean serum albumin for patients who had minor criteria was 35.85 ± 5.34. The mean serum blood urea nitrogen for patients who had minor criteria was 68.65 ± 39.34. The mean pH for patients who had minor criteria was 7.3 ± 0.12. The mean partial pressure of oxygen for patients who had minor criteria was 108.82 ± 51.68. The mean saturation of oxygen for patients who had minor criteria was 94.48 ± 5.49. The mean lactate for patients who had minor criteria was 3.17 ± 1.88.

Table 6: Area under the ROC curve (AUC) for Vasopressor with SMART COP, CURB 65, PSI, SCAP (Major, Minor) and ATS (Major, Minor).

Test Result Variable(s)	Area	Std. Error	P Value	95% Confidence Interval	
				Lower Bound	Upper Bound
SMART_COP	0.885	0.053	<0.0001	0.780	0.989
CURB_65	0.815	0.051	<0.0001	0.715	0.914
PSI	0.829	0.049	<0.0001	0.733	0.926
SCAP_MAJOR	0.867	0.060	<0.0001	0.750	0.983
SCAP_MINOR	0.800	0.054	<0.0001	0.694	0.906
ATS_MAJOR	0.833	0.064	<0.0001	0.708	0.959
ATS_MINOR	0.817	0.053	<0.0001	0.713	0.920

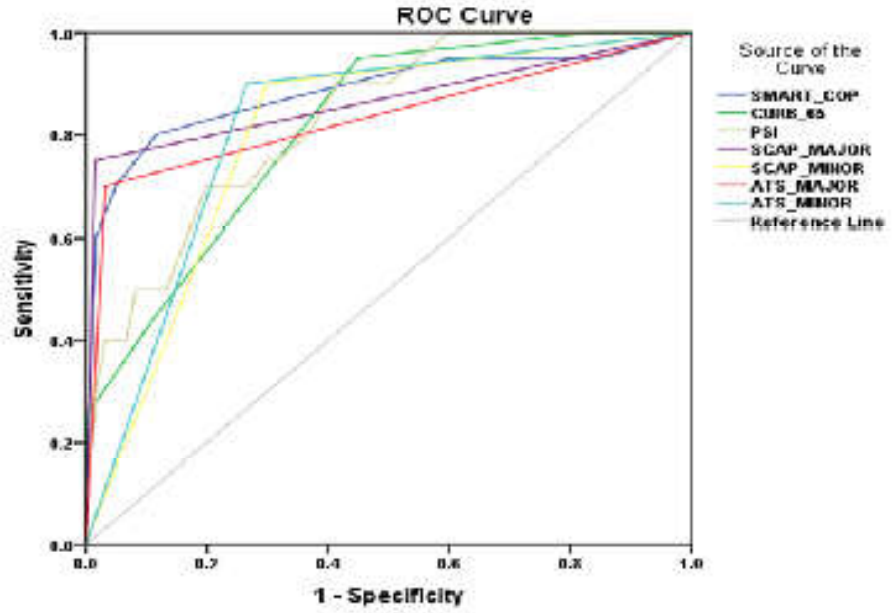

Fig. 1: Receiver operating characteristic (ROC) curves for Vasopressor with SMART COP, CURB 65, PSI, SCAP (Major, Minor) and ATS (Major, Minor)

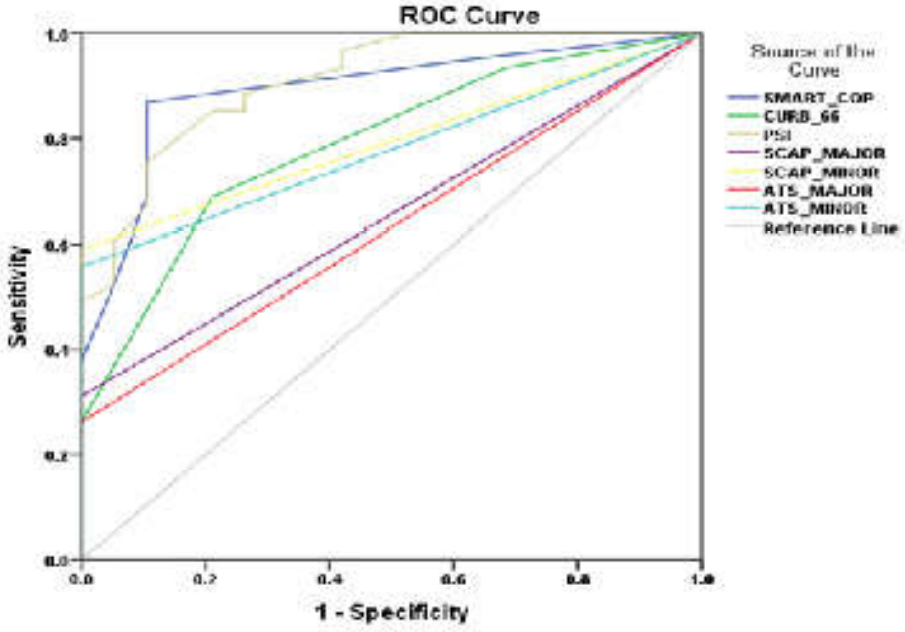

Fig. 2: Receiver operating characteristic (ROC) curves for Admit_in (ICU) with SMART COP, CURB 65, PSI, SCAP (Major, Minor) and ATS (Major, Minor).

ROC curve to predict ICU admission among different score studied. PSI was sensitive to predict ICU admission when compared to other pneumonia severity scores. Area under curve for PSI is 0.91 where as SMART COP is 0.89, CURB65 is 0.79, SCAP major criteria is 0.65, minor criteria is 0.79, ATS/IDSA minor criteria was 0.77, major criteria was 0.63. p value of SMART_COP, CURB65,PSI, SCAP minor and ATS/IDSA minor criteria was very high significant i.e, <0.0001 and p value of SCAP major criteria 0.041 and ATS major criteria was 0.086.

ROC curve to predict mortality among different score studied. PSI was sensitive to predict mortality when compared to other pneumonia severity scores. Area under curve for PSI is 0.936 where as SMART COP is 0.89, CURB65 is 0.828, SCAP major criteria is 0.92, minor criteria is 081, ATS/IDSA minor criteria was 0.82, major criteria was 0.84. P value of SMART_COP, CURB65, PSI, SCAP major/ minor and ATS/IDSA major/minor criteria was very high significant i.e, p<0.0001 (Table 7).

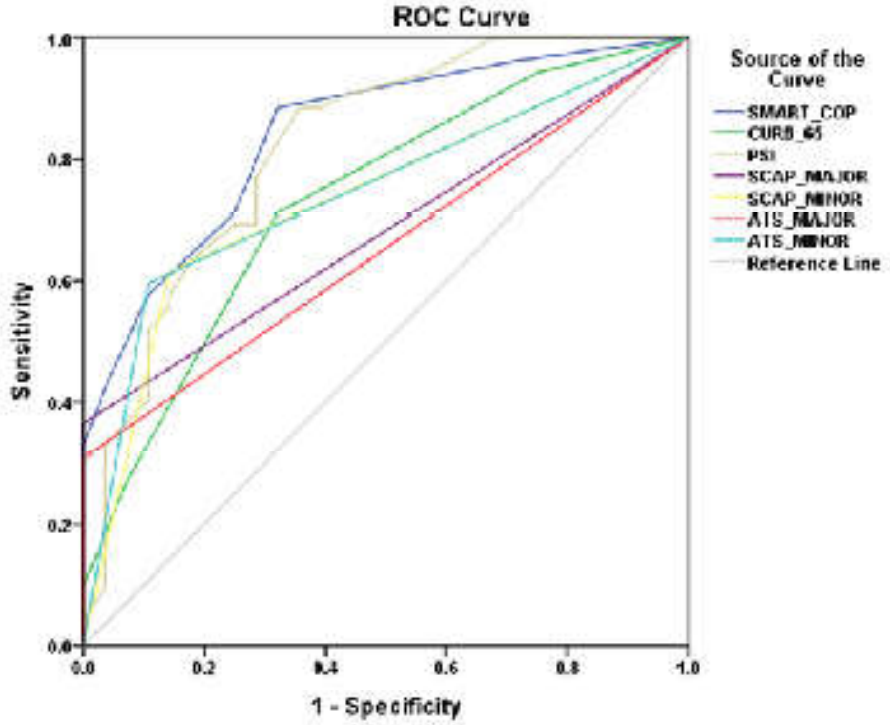

Fig. 3: Receiver operating characteristic (ROC) curves for Ventilator support with SMART COP, CURB 65, PSI, SCAP and ATS.

Table 7: Area under the ROC curve (AUC) for outcome with SMART COP, CURB 65, PSI, SCAP (Major, Minor) and ATS (Major, Minor).

Test Result Variable(s)	Area	Std. Error	p Value	95% Confidence Interval	
				Lower Bound	Upper Bound
SMART_COP	0.891	0.088	<0.0001 VHS	0.719	10.000
CURB_65	0.828	0.056	0.001 SIG	0.718	0.938
PSI	0.936	0.027	<0.0001 VHS	0.883	0.988
SCAP_MAJOR	0.926	0.036	<0.0001 VHS	0.855	0.996
SCAP_MINOR	0.814	0.050	0.001 SIG	0.716	0.912
ATS_MAJOR	0.843	0.077	<0.0001 VHS	0.692	0.994
ATS_MINOR	0.829	0.048	0.001 SIG	0.735	0.922

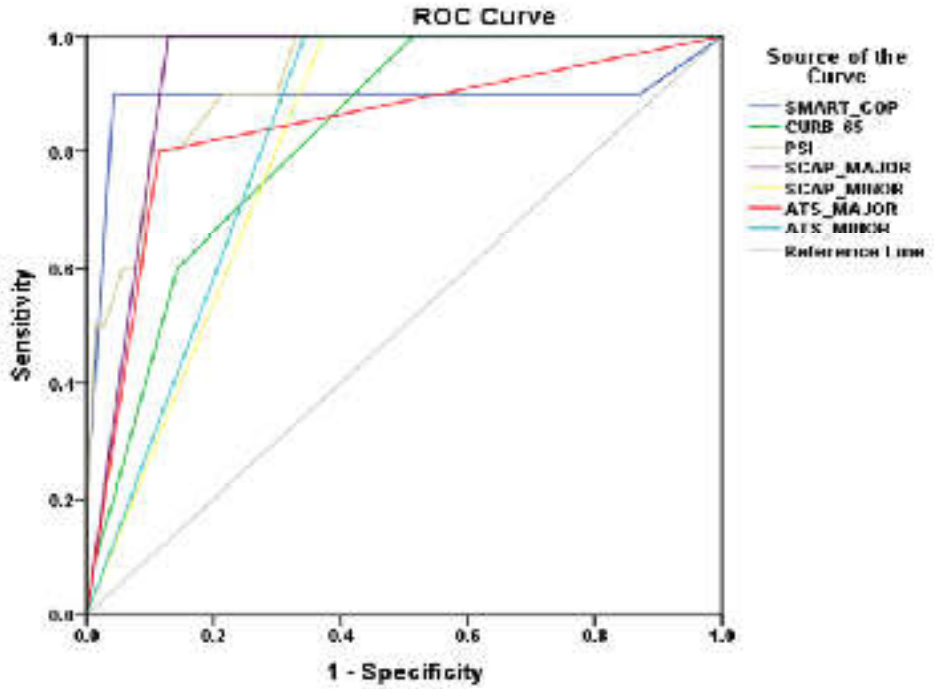

Fig. 4: Receiver operating characteristic (ROC) curves for outcome with SMART COP, CURB 65, PSI, SCAP and ATS.

Discussion

In present study (n=80), 5 (6.3%) patients were <30 years, 21 patients (26.3%) were between 31-50 years, 42 (52.5%) patients are between 51-70 years, 12 (15%) patients are greater than 70 years of age. In this study group majority of patients were between 51-70 years.

In a study by Vidyasagar et al. [12] majority of subjects (70%) were less than 60 yrs of age with 30% in the age group > 60 years. 25% were in 51 to 60 years. In the present study, 55% were males, and 45% were females.

The most common comorbidity of patients associated with CAP was hypertension in 46 patients. In a study conducted by Babu et al. (2017) [13] the major comorbidities of patients associated with CAP were chronic renal failure (40%), congestive heart failure (30%), and chronic liver failure (25%).

Among 80 patients, 25 patients had shown streptococcus in culture, 18 pts had shown staphylococcus, 14 had shown Klebsiella, 3 had shown E.coli, Pseudomonas is seen in 12 and others in 9 patients respectively.

In a study by Vidyasagar et al. (2015) [12] out of 95 patients, 34 subjects (42.5%) were admitted in ICU, 32.5% of subjects were put on ventilator support.

In present study, 61 (76.3%) patients were admitted in ICU and 19 (23.8) patients were admitted in ward. Study done by Man et al. (2007) [2] demonstrated that prospective comparison of three predictive rules for assessing severity of community-acquired pneumonia The ICU admission rate of low-risk groups was 2.7% in PSI, 2.3% in CURB65.

In a study done Shah et al. (2009) [1] to validate Pneumonia Severity Index and CURB-65 Severity Scoring Systems in Community Acquired Pneumonia in an Indian Setting out of 130 patients 35 patients required ICU admission.

Out of 80 patients, mortality is seen in 10 patients (12.5%). In a study by Eldaboosy et al. (2015) [14], the mortality rate in this study was 10% and mortality was higher in the elderly and patients with comorbidities.

In a study done by Shah et al. (2009) [1] to Validate Pneumonia Severity Index and CURB-65 Severity Scoring Systems in Community Acquired Pneumonia in an Indian Setting out of 150 patients mortality was seen in 16 patients.

In present study, 20 patients required Vasopressor support. In a study done by Chalmers et al. (2008) [15] in Predicting the Need for Inotropic Support for Young Adults Admitted to the Hospital with Community-Acquired Pneumonia.

AUC value to predict the need of vasopressor among different group studied SMART_COP score has highest AUC value to predict the need of vasopressor support i.e. 0.88. AUC value for CURB_65 is 0.81, PSI is 0.82, SCAP major /minor 0.86/0.80, ATS/IDSA major and minor criteria is 0.83/0.81.

In Australian CAP Charles et al. (2008) [16] studies A SMART-COP has highest AUC value (0.87) to predict the need of vasopressor support. Whereas the AUC value for PSI is 0.69 and CURB_65 is 0.67. The AUC values correlates with present study.(31).

Marti et al. (2012) [17], performed a meta-analysis comparing different scoring systems in pneumonia prognosis. They concluded that new severity scores for CAP in predicting the need for IRVS or ICU admission (ATS/IDSA 2007 minor criteria, SCAP score and SMART- COP), had better discriminative performance in comparison to the previous ones (PSI and CURB-65).

In present study, 61 patients required ICU admission. 36 patients belong to low risk group and among them 19 patients required ICU admission.

Among 80, 16 patients had major criteria and all patients required ICU admission. 34 patients had minor criteria and 34 patients required ICU admission.

Among different scores used to predict ventilator support in patients admitting to ER PSI has more AUC to predict ICU admission.

In a study by Splinder et al. (2006) [18] to evaluate the accuracy of score systems. The need for ICU treatment was significantly higher (P<0.0001) in high-risk than in low-risk patients for two severity scores: 19 out of 53 (35.8%) versus one out of 61 (1.6%) for PSI; 12 out of 22 (54.5%) versus eight out of 92 (8.7%) for CURB-65.

In a study done by Pereira et al (2012) [8] to assess severity of patients with community acquired of pneumonia, a ROC value for predicting ICU admission in patients with CAP PSI was 0.86, curb 65 was 0.79 ATS/IDSA was 0.82, SMARTCOP 0.83, SCAP 0.75. The value of the study correlates with present study.

In a study done by Singanayagam et al. (2009) [19] in severity assessment of SCAP AUC value to predict ICU admission are 0.87, 0.77, 0.80 for SMART_COP, CURB_65, PSI respectively.

In present study among 80 patients 52 patients required ventilator support. In present study among 80 patients mortality was seen in 10 patients and 70 patients were discharged home.

In distribution of patients in SMART_COP, 36 patients belong to low risk group and 21 patients belong to moderate risk group, 10 patients belong to high risk group, only 1 patient of low risk group had mortality 13 patients belong to very high risk group and mortality was seen in 9 patients.

In distribution of patients in CURB_65, among 80 patients 10 patients has score 0 and, 24 patients has score 1 and no mortality was seen in both scores, 30 patients has score 2 and mortality was seen in 4 patients, 11 patients has score 3 and mortality was seen in 4 patients, 3 patients has score 4 and mortality was seen in 1 patient, 2 patients has score 5 and mortality was seen in 1 patient.

In distribution of patients in PSI, among 80 patients no patient belonged to class 1, 4 patients belonged to class 2, 5 patients belonged to class 3 and no mortality was seen in first 3 classes, 53 patients belonged to class 4 and 2 patients had mortality, 18 patients belonged to class 5 and 8 patients had mortality.

In distribution of patients in ATS/IDSA, among 80 16 patients had major criteria and mortality was seen in 8 patients. 34 patients had minor criteria and mortality was seen in 10 patients.

In distribution of patients in SCAP, among 80 19 patients had major criteria and mortality was seen in 10 patients. 36 patients had minor criteria and mortality was seen in 10 patients. Among different scores used to predict ventilator support in patients admitting to Emergency department PSI has more AUC value to predict outcome.PSI has AUC of 0.93 where as CURB65 has 0.82 SMART_COP has 0.89, major criteria of SCAP has 0.92 minor criteria of SCAP has 0.81, major criteria of ATS/IDSA has 0.84, minor criteria of ATS/IDSA has 0.82. P value for SMART_ COP, PSI, SCAP major criteria, SCAP minor criteria, ATS/IDSA major and minor criteria have very high significant P value <0.0001, whereas CURB 65 have P value of 0.13.

In a study done by Pereira et al. (2012) [8] to assess severity of patients with community acquired of pneumonia, AUC value for predicting mortality in patients with CAP PSI was 0.89,CURB_ 65 was 0.87, ATS/IDSA was 0.67, SMART_COP and SCAP were not assessed.

Conclusion

SMART_COP and PSI can be used to determine the severity and prognosis of the patients presenting to Emergency Department with Community acquired pneumonia. Early microbiological diagnosis, early

antibiotic administration in patients with SMART_ COP score > 4 and PSI class 4 and 5 can decrease the morbidity and mortality in CAP patients.

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Dehydration as a Poor Prognostic Factor in Acute Ischemic Stroke: An Observational Study in a Tertiary Care Hospital in India

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Abstract

Background: Stroke is a leading cause of mortality among patients presenting to the emergency department with Acute Ischemic Stroke being a significant contributor. Clinical guidelines emphasize the importance of adequate hydration in management of stroke. Dehydration may impair cerebral oxygen delivery and worsen clinical outcome in patients with acute ischemic stroke (AIS). Elevated blood urea nitrogen to creatinine ratio (BUN/Cr) as a marker of dehydration has been associated with poor clinical outcome in emergency department (ED) patients presenting with AIS. Dehydration also attributes to the increased rate of infections and length of hospital stay in patients with AIS. *Objectives:* To study the association of dehydration markers BUN/Creatinine ratio, plasma osmolality, haematocrit and Caval Index with the outcome in patients with Acute Ischemic Stroke in terms of Mortality and Morbidity (Length of Stay in Hospital) and to correlate the hydration status with the disease progression by using NIHSS score and Modifies Rankin score. *Methods and study design:* The study was conducted from January 2017 till January 2018 over a period of 1 year. It was a Prospective observational cohort study. Patient included in the study were followed up at 3 and 30 days as per the questionnaire designed for the study after the approval by scientific and ethics committee of the institute. For the purpose of statistical significance we will recruit 64 patients as per the inclusion & exclusion criteria of the study. Data collection was done as per the approved data collection forms for the purpose of study. Patient's initial, BUN, Creatinine, Urea, Sodium, blood glucose, Caval index (optional) and clinical status was be collected as per NIHSS and Modified rankin scale. The patient was followed up during their duration of stay in the hospital on 1st, 2nd and 3rd day. During the follow up period patients was followed for progression and outcome based on clinical parameters and the mentioned scores. *Results and Conclusion:* In conclusion, though we were able to associate plasma osmolality and BUN/Creatinine ratio with mortality, length of stay and with poorer outcome in terms of early neurological deterioration; hematocrit and Caval Index failed to associate to significant level to prove as a prognostic marker. Initial risk stratification using the plasma osmolality and BUN/Creatinine ratio may assist with prognostication and help prevent deterioration of the patients with Acute Ischemic Stroke.

Keywords: Dehydration; Acute Ischemic Stroke; Dehydration.

Introduction

Stroke is a leading cause of mortality among patients presenting to the emergency department with Acute Ischemic Stroke being a significant contributor. Several predictors of outcome have been studied so far and clinical outcome, survival and residual neurological impairment have been

attributed to a variety of clinical and biochemical parameters [1].

Clinical guidelines emphasize the importance of adequate hydration in management of stroke. Dehydration may impair cerebral oxygen delivery and worsen clinical outcome in patients with acute ischemic stroke (AIS) [3]. Elevated blood urea nitrogen to creatinine ratio (BUN/Cr) as a marker of dehydration has been associated with poor clinical outcome in emergency department (ED) patients presenting with AIS [4]. Dehydration also attributes to the increased rate of infections and length of hospital stay in patients with AIS.

Dehydration can be measured using any of the 4 parameters-

1. Plasma Osmolality
2. BUN/creatinine Ratio
3. Haematocrit
4. Caval Index (optional)

Intensive review of literature shows no direct studies have been conducted on the association of these four parameters with poor prognosis of AIS. All the study parameters form a part of routine initial investigations and have individually been correlated with the outcome of AIS.

The objective of this study is to find the association of dehydration measured using, haematocrit, BUN/Creatinine ratio, plasma osmolality and Caval Index with the clinical outcome in patients in terms of NIHSS scores and Modified Rankin Score, these scores being the standardized measure of monitoring disease progression among stroke patients.

The Modified Rankin Scale (mRS) is used to measure the degree of disability in patients who have had a stroke, as follows [20, 21, 22].

0	No symptoms at all
1	Significant disability despite symptoms; able to carry out all usual duties/activities
2	Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance
3	Moderate disability; requiring some help, but able to walk without assistance
4	Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability; bedridden, incontinent and requiring constant nursing care and attention
6	Dead

The National Institutes of Health Stroke Scale (NIHSS)

1a LOC	0=Alert, 1=Not alert but arouse, 2=Not alert, 3=Unresponsive
1b LOC questions	0=Answers both, 1=Answers one, 2=Answers neither question
1c LOC commends	0=Perform both, 1=Perform one, 2=Perform neither task
2 Best gaze	0=Normal, 1=Partial gaze palsy, 2=Forced deviation
3 Visual fields	0=No visual loss, 1=Partial, 2=Complete, 3=Bilateral hemianopsia
4 Facial weakness	0=Normal, 1=Minor, 2=Partial, 3=Complete paralysis
5a Motor left arm	0= No drift, 1=Drift before 10 seconds, 2= Fall before 10 seconds, 3= No effect against gravity, 4= No movement
5b Motor right arm	0= No drift, 1=Drift before 10 seconds, 2= Fall before 10 seconds, 3= No effect against gravity, 4= No movement
6a Motor left leg	0= No drift, 1=Drift before 5 seconds, 2= Fall before 5 seconds, 3= No effect against gravity, 4= No movement
6b Motor right leg	0= No drift, 1=Drift before 5 seconds, 2= Fall before 5 seconds, 3= No effect against gravity, 4= No movement
7 Ataxia	0=Absent, 1=Present in one limb, 2=Present in two limb
8 Sensory	0=Normal, 1= Mild to moderate loss, 2=Severe to total loss
9 Best language	0=Normal, 1=Mild to moderate aphasia, 2=Severe aphasia, 3=mute, global aphasia
10 Dysarthria	0=Normal, 1=Mild to moderate dysarthria, 2=Severe dysarthria
11 Extinction	0=Normal, 1=Mild, 2=Severe

Literature Review

LOC: Loss of Consciousness

Previous studies have evaluated various factors defining dehydration and correlating it with the progression of disease in patients with Acute Ischemic stroke.

"Predictors of early clinical deterioration after acute ischemic stroke" a study by Lin LC et al in Am J Emerg Med. 2011 Jul;29(6):577-81, did a prospective study on 196 patients with Acute Ischemic Stroke and identified risk factors which lead to early deterioration. The patients with early deterioration in National Institutes of Health Stroke Scale scores (increase ≥ 3 points within 3 days) were defined as stroke-in-evolution (SIE). BUN/Creatinine higher than 15 was an independent predictor of Stroke in evolution. These patients were 3.4 folds more likely to have Stroke in evolution ($p = 0.08$) [5]. This concluded at BUN/creatinine higher than 15 was associated with early clinical deterioration.

"Dehydration is an independent predictor of discharge outcome and admission cost in acute ischaemic stroke" a study by C.H. Liu et al. in Eur J Neurol. 2014 Sep. Studied predictive value of initial hydration status on clinical outcome and admission cost in acute ischaemic stroke and highlights the importance of monitoring BUN/Cr in the acute stage. Discharge outcome were monitored using modified Rankin scale and Barthel Index [7]. Acute ischaemic stroke with admission dehydration had higher infection rates ($p = 0.006$), worse discharge BI ($p < 0.001$), worse mRS ($p < 0.001$) and higher admission costs ($p<0.001$)

"Influence of raised plasma osmolality on clinical outcome after acute stroke" a study by Ajay Bhalla et al. in Stroke. 2000 Sep, concluded that a plasma osmolality of >296 mOsm/kg on admission showed a significant association with stroke mortality at 3 months independent of age, sex, stroke severity, Barthel Index before stroke and stroke subtype [9].

"Predictors of early neurological deterioration in patients with acute ischaemic stroke with special reference to blood urea nitrogen (BUN)/creatinine ratio & urine specific gravity" a study by K. Bhatia et al. in Indian J Med Res. 2015 Mar studied 114 patients of Acute Ischemic Stroke (AIS) in an Emergency Department setting. They defined Early neurological deterioration (END) as worsening of neurological condition as indicated by an increase of three or more points in the NIHSS score or death not attributed to other cause, within the first three days. They concluded that BUN/creatinine >15 was found as an independent factor predictive of END [4].

"Elevated blood urea nitrogen/creatinine ratio is associated with poor outcome in patients with ischemic stroke" a study by Schrock JW et al. in ClinNeurolNeurosurg. 2012 Sep studied 324 patients with AIS and concluded an elevated BUN/Cr ratio of ≥ 15 OR 2.2 (1.2-4.0) is associated with poor outcome at 30 days [14].

Ultrasound has become an important tool for evaluating a variety of acute conditions in ED, because it is not invasive and relatively easy to perform with adequate training, portable and with good effectiveness. An observational study Identification of the hydration state in emergency patients: correlation between caval index and BUN/creatinine ratio done by A. Riccardi et al. in Eur Rev Med Pharmacol Sci. 2013 Jul; found a good correlation between Caval index and BUN/Cr Ratio (Pearson Index 0.76, $p < 0.001$, with significant t-Student test ($p < 0.01$, IC 99%) [8].

Inspiratory inferior vena cava and expiratory inferior vena cava diameters were measured, 2 cm from the right atrial border in a long-axis/subxiphoid view with a 3.5 MHZ curvilinear probe. Measurements were taken during a normal respiratory cycle, and the CIn was recorded (CIn is the difference between end-expiratory and end-inspiratory IVC diameter divided by the end-expiratory diameter). Bedside ultrasonographic measurement of caval index greater than or equal to 50% is strongly associated with a low central venous pressure.

Elevated hematocrit (Hct) contributes to blood viscosity and has an adverse effect in acute stroke. *"Elevated hematocrit is associated with reduced reperfusion and tissue survival in acute stroke"* Allport LE et al. in Neurology November 08, 2005 studied 64 patients and found that an increasing Hct was a significant predictor of infarct growth (OR = 1.26, 95% CI = 1.00 to 1.59) [16].

"Use of the Barthel index and modified Rankin scale in acute stroke trials" study by Sulter G et al. in Stroke journal 1999 Aug investigated the commonly used scales BI and mRS that measure disability or dependence in activities of daily living in stroke victims. Favorable outcome on the MRS was defined as either </=1 or </=2.

Study Questions

1. How are the markers of dehydration correlating with the outcome in patients with Acute Ischemic stroke?
2. What is the impact of the hydration status on the progression of Acute Ischemic stroke?

Study Objective

1. Primary Objective

To study the association of dehydration markers BUN/Creatinine ratio, plasma osmolality, haematocrit and Caval Index with the outcome in patients with Acute Ischemic Stroke in terms of Mortality and Morbidity (Length of Stay in Hospital).

2. Secondary Objective

To correlate the hydration status with the disease progression by using NIHSS score and Modifies Rankin score.

Study Endpoint/ Outcome

The results of the study would indicate the trends of dehydration with early neurological deterioration and poor prognosis in patients with Acute Ischemic Stroke.

An association of the clinical parameters, BUN/ Creatinine ratio, plasma osmolality, hematocrit and Caval Index with the prognosis of patients with Acute Ischemic Stroke.

Study Design:	Prospective observational study.
Study Duration:	12 Months.
Data Selection:	Based on Inclusion and Exclusion Criteria.

Study Duration and Timeline

Data would be collected from the site of study from January 2017 to January 2018.

Materials and Methods

Study Area

The study was conducted in the Emergency Department of Max Hospital, Shalimar Bagh, New Delhi.

Inclusion criteria

1. Patients both males and females with age more than 18 years old and above.
2. All diagnosed cases of acute ischemic stroke diagnosed by the clinical presentations and brain imaging.
3. Venous strokes.
4. Has a measurable neurologic deficit according to the National Institutes of Health Stroke Scale (NIHSS) or Modified Rankin Scale (mRS).
5. Patients presenting at the ED with a history of onset of neurological symptoms within the last 24 hours.

Exclusion criteria

1. Uncontrolled Hyperglycemia (> 400 mg/dl)
2. Active Infections.
3. Initial NIHSS of > 22.
4. Pre-existing Chronic Kidney Disease or Liver Disease.

Study design

It was a Prospective observational cohort study. Patient included in the study were followed up at 3 and 30 days as per the questionnaire designed for the study after the approval by scientific and ethics committee of the institute.

For the purpose of statistical significance we will recruit 64 patients as per the inclusion & exclusion criteria of the study.

Data collection method

- Patient's history was taken from the patient and his/her attendants.
- CPRS (Computerized Patient Record System) and investigations reports were be used for collection of following data:

Data Collection

The study was conducted in the Emergency department (ED) of Max Super Specialty Hospital, Shalimar Bagh, which is a tertiary care referral hospital with an average of 9000 visits per annum.

Study subjects was followed prospectively from the time of presentation to the ED. Data collection was collected from patient reports and case files with no intervention on behalf of the investigator. Patient safety and care was in no way be compromised as the study is based on data which is yielded by routinely monitored parameters.

Data collection was done as per the approved data collection forms for the purpose of study. Patient's initial, BUN, Creatinine, Urea, Sodium, blood glucose, Caval index (optional) and clinical status was be collected as per NIHSS and Modified rankin scale. The patient was followed up during their duration of stay in the hospital on 1st, 2nd and 3rd day. During the follow up period patients was followed for progression and outcome based on clinical parameters and the mentioned scores.

Early neurological deterioration (END) is defined as worsening of neurological condition as indicated by an increase of three or more points in the NIHSS score or death not attributed to other cause, within the first three days. An increase of three or more points in the NIHSS score was used to diagnose END. Patients were divided into two groups. The first group included patients with END and the second group included those without END. Patients were followed up on day 30th for mRS scoring. AnmRS Scoring of 3 or more was taken as an unfavorable outcome.

Dehydration was defined by any one of the following:

1. Plasma Osmolality > 295, or
2. BUN/creatinine Ratio ≥ 15, or

3. Hematocrit > 43%, or
4. Caval Index > 50%

At the end of the study period the data collection was utilized to derive an association of dehydration and progression of AIS in terms of END and mRS.

All data mentioned above and required for the study was entered into a data collection form. (Annexure 1)

Sample Size and Statistical Analysis

Based on the results of previous studies to derive a statistically significant co relation we recruited a sample size of 64 patients. Correlation between intravascular volume depletion markers such as BUN/ Creatinine ratio, Plasma osmolality, Hematocrit, Caval Index, NIHSS score, Modified rankin score and, outcome and survival was obtained by pearson correlation coefficient and its statistical significance was tested by Z test after fisher transformation. The same procedure was also be used to compare the correlation between two groups under study. For comparing average in two groups student t test was used assuming Gaussian distribution. In case the distribution was far from Gaussian wilcoxon-Mann whitney test was used.

Statistical Plan

Statistical Analysis

Summarized data was presented using Tables and Graphs. Data was normally distributed as tested using the Shaperio-Wilk W test (p-value was less than 0.05). Level of statistical significance was set at p-value less than 0.05. Spearmans and pearson correlation coefficient was used to calculate correlation. Chi square test was used to find association between categorical variables and Mann Whitney U test to compare mean between two independent groups.

Results

Demographic details

Table 1: Distribution of study population according to age group

Age (years)	Frequency	Percent
30-40	6	9.4
41-50	4	6.3
51-60	12	18.8
61-70	18	28.1
71-80	18	28.1
81-90	6	9.4
Total	64	100.0

Table 2: Distribution of study population according to Plasma Osmolality levels

Plasma Osmolality	Frequency	Percent
Greater than 295	25	39.1
Less than or equal to 295	39	60.9
Total	64	100.

Table 3: Distribution of study population according to BUN/ Creatnine ratio

BUN/Creatnine ratio	Frequency	Percent
Greater than or equal 15	44	68.8
Less than 15	20	31.3
Total	64	100.0

Table 4: Distribution of study population according to Hematocrit

Hematocrit	Frequency	Percent
Greater than 43%	15	23.4
Less than or equal to 43%	49	76.6
Total	64	100.0

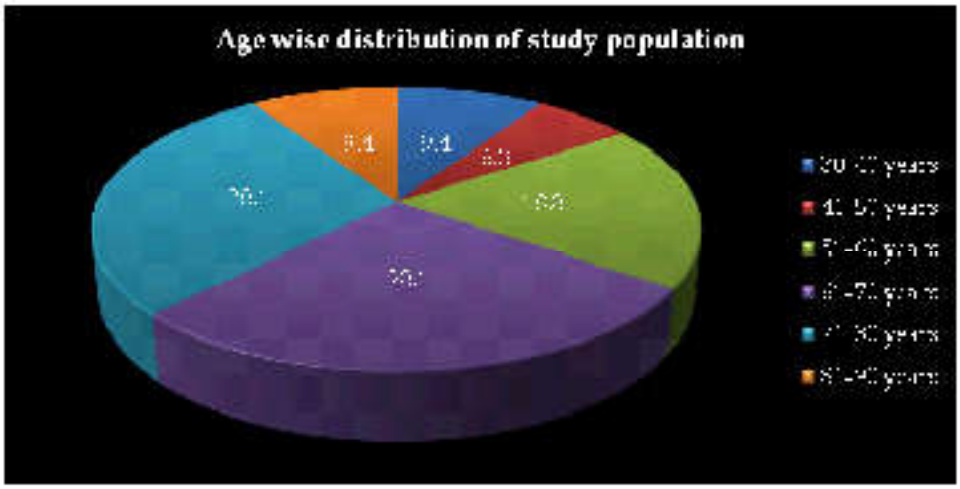

Fig. 1: Distribution of study population according to age group

Table 5: Distribution of a sub group among the study population in whom the caval index was calculated

Plasma Osmolality	Frequency	Percent
Less than 50%	14	70.0
More than or equal to 50%	6	30.0
Total	20	100.0

Table 6: Comparison of early neurological deterioration and dehydrated patients among the study population

		Early neurological deterioration		Total
		Present	Absent	
Dehydrated	N	18	33	51
	%	35.3%	64.7%	100.0%
Non Dehydrated	N	2	11	13
	%	15.4%	84.6%	100.0%
Total	N	20	44	64
	%	31.3%	68.8%	100.0%
Chi Square Value			1.911	
p Value			0.147	

Table 7: Comparison of modified Rankin Score and dehydrated patients among the study population

		Modified Rankin Score (mRS score)			Total
		2	3-5	6	
Dehydrated	N	20	25	6	51
	%	39.2%	49.0%	11.8%	100.0%
Non Dehydrated	N	8	5	0	13
	%	61.5%	38.5%	0.0%	100.0%
Total	N	28	30	6	64
	%	43.8%	46.9%	9.4%	100.0%
Chi Square Value			2.956		
p Value			0.228		

Table 8: Mean ± SD of Length of stay among study population based on dehydration

Length of stay	Mean	Std. Deviation
Dehydrated	7.49	6.76
Non Dehydrated	10.6	11.1

Mann whitney u test. Level of significance set at 0.05 Z value: -101, p value: 0.919

Table 9: Distribution of NIHSS among the study population

NIHSS	Frequency	Percent
Minor Stroke (1-4)	14	21.9
Moderate Stroke (5-15)	36	56.3
Moderate to Severe Stroke (16-20)	14	21.9
Severe Stroke (21-42)	0	0
Total	64	100.0

Table 10: Comparison of plasma osmolality with NIHSS

Minor Stroke		NIHSS			Total
		Moderate Stroke	Moderate to Severe Stroke		
Greater than 295	N	11	6	8	25
	%	44.0%	24.0%	32.0%	100.0%
Less than or equal to 295	N	3	30	6	39
	%	7.7%	76.9%	15.4%	100.0%
Total	N	14	36	14	64
	%	21.9%	56.3%	21.9%	100.0%
Chi Square Value			18.6889		
P Value			0.001*		

Chi Square Test, Level of Significance at $p < 0.05$

Table 11: Comparison of BUN/Creatinine ratio with NIHSS

Minor Stroke		NIHSS			Total
		Moderate Stroke	Moderate to Severe Stroke		
Greater than or equal 15	N	7	24	13	44
	%	15.9%	54.5%	29.5%	100.0%
Less than 15	N	7	12	1	20
	%	35.0%	60.0%	5.0%	100.0%
Total	N	14	36	14	64
	%	21.9%	56.3%	21.9%	100.0%
Chi Square Value			6.151		
P Value			0.046*		

Chi Square Test, Level of Significance at p< 0.05

Table 12: Comparison of hematocrit with NIHSS

Minor Stroke		NIHSS			Total
		Moderate Stroke	Moderate to Severe Stroke		
Greater than 43%	N	4	6	5	15
	%	26.7%	40.0%	33.3%	100.0%
Less than or equal to 43%	N	10	30	9	49
	%	20.4%	61.2%	18.4%	100.0%
Total	N	14	36	14	64
	%	21.9%	56.3%	21.9%	100.0%
Chi Square Value			2.301		
P Value			0.316		

Chi Square Test, Level of Significance at p < 0.05

Table 13: Comparison of Caval Index with NIHSS

Minor Stroke		NIHSS			Total
		Moderate Stroke	Moderate to Severe Stroke		
Less than 50%	N	5	6	3	14
	%	35.7%	42.9%	21.4%	100.0%
Greater than or equal to 50%	N	2	2	2	6
	%	33.3%	33.3%	33.3%	100.0%
Total	N	7	8	5	20
	%	35.0%	40.0%	25.0%	100.0%
Chi Square Value			0.844		
p Value			0.340		

Chi Square Test, Level of Significance at p< 0.05

Table 14: Comparison of plasma osmolality with mortality

		Mortality		Total
		Died	Survived	
Greater than 295	N	6	19	25
	%	24.0%	76.0%	100.0%
Less than or equal to 295	N	0	39	39
	%	0.0%	100.0%	100.0%
Total	N	6	58	64
	%	9.4%	90.6%	100.0%
Chi Square Value			10.382	
p value			0.002*	

Chi Square Test, Level of Significance at p< 0.05

Table 15: Comparison of BUN/Creatinine ratio with mortality

		Mortality		Total
		Died	Survived	
Greater than or equal 15	N	6	38	44
	%	13.6%	86.4%	100.0%
Less than 15	N	0	20	20
	%	0.0%	100.0%	100.0%
Total	N	6	58	64
	%	9.4%	90.6%	100.0%
Chi Square Value			3.009	
p Value			0.094	

Chi Square Test, Level of Significance at $p < 0.05$

Table 16: Comparison of hematocrit with mortality

		Mortality		Total
		Died	Survived	
Greater than 43%	N	2	13	15
	%	13.3%	86.7%	100.0%
Less than or equal to 43%	N	4	45	49
	%	8.2%	91.8%	100.0%
Total	N	6	58	64
	%	9.4%	90.6%	100.0%
Chi Square Value			0.361	
p Value			0.432	

Chi Square Test, Level of Significance at $p < 0.05$

The comparison of hematocrit with mortality among the study population was done using chi square test. It failed to reach the level of significance.

Table 17: Comparison of caval index with mortality

		Mortality		Total
		Died	Survived	
Less than 50%	N	1	13	14
	%	7.1%	92.9%	100.0%
Greater than or equal to 50%	N	1	5	6
	%	16.7%	83.3%	100.0%
Total	N	2	18	20
	%	10.0%	90.0%	100.0%
Chi Square Value			0.521	
P Value			0.423	

Chi Square Test, Level of Significance at $p < 0.05$

The caval index was dichotomized in two groups based on median value 50% to calculate association.

Discussion

Acute Ischemic stroke has been one of the significantly known contributors of mortality among the patient present to the emergency room with stroke. As an emergency physician, apart from the duty of diagnosing, resuscitating and managing Acute Ischemic Stroke patients there also lies a role of recognizing dehydration and correcting it to prevent further deterioration of these patients. In this study, we had aimed to associate the parameters i.e Plasma Osmolality, BUN/Creatinine ratio, Hematocrit and Caval Index (optional) with the mortality and morbidity of these patients and also with the progression of the disease.

In a general perspective, any patient fulfilling the criteria for dehydration as per even one of the variable was considered dehydrated. The

comparison of mRS score and dehydration among the study population, the group with dehydration had about 11% subjects with a mRS of 6 i.e death, while majority of dehydrated patients i.e 49% reported to have a mRS score between 3-5 i.e they had unfavorable outcome. Most patients with early neurological deterioration were found to be dehydrated i.e 18 patients in comparison to the non dehydrated i.e 2 patients Surprisingly, the length of stay in the hospital among the survivors was found to be higher in the mean of 10.6 (standard deviation 11.1) days against 7.49 (standard deviation of 6.76).

On considering the individual markers of dehydration; plasma osmolality of more than 295 in comparison with NIHSS, 32% had moderate to severe stroke as per NIHSS (p = 0.001). In comparison with mortality, subjects who died showed an plasma osmolality more than 295 while none of the subjects with normal osmolality had died (p = 0.002). In terms of length of stay in the hospital, about 75% of patients with plasma osmolality stayed in the hospital for at least more than 6 days.

With BUN/Creatinine ratio, among those with a ratio of more than 15, about 30% were found to have moderate to severe stroke as per the NIHSS. In terms of mortality, the patients who died (mRS score of 6) showed a BUN/Creatinine ratio of 15 or more.

Hematocrit on the other hand, showed no correlation with mortality, length of stay, early neurological deterioration to reach a statistical level of significance.

Caval Index using IVC diameters were assessed in a small sub group of the patient of the study population. Its comparison with NIHSS, mRS, length of stay failed to form any sort of association. In terms of Early neurological deterioration, in about 90% of those with Caval index of more than 50% had been seen to have early neurological deterioration (p = 0.09). But this could not be considered significant statistically as the sub group consisted of only 20 patients.

Our study had several limitations.

- It was a single-center study with relatively small numbers of patients. Hence the results cannot be applied to all centers across the India.
- Our sample size was inadequate to provide results which were statistically significant.
- Although we had taken into consideration the degree of hydration and its association with mortality during the time of admission; once admitted in the ICUs, the course of treatment and interventions done based on the treating physician's preferences could have independently varied the outcome within the study population, which could not be accounted for.

Conclusion

In conclusion, though we were able to associate plasma osmolality and BUN/Creatinine ratio with mortality, length of stay and with poorer outcome in terms of early neurological deterioration; hematocrit and Caval Index failed to associate to significant level to prove as a prognostic marker.

Initial risk stratification using the plasma osmolality and BUN/Creatinine ratio may assist with prognostication and help prevent deterioration of the patients with Acute Ischemic Stroke.

Future directions for consideration include a multicentric trial with a larger sample to validate the predictive value of these markers of dehydration in prognosticating Acute Ischemic Stroke.

Annexures I

Protocol no- IVD Stroke 01/2016

Data Collection Form

SSN No. Age : Sex : ☐ M ☐ F

Date : Time of Arrival : Onset of Symptoms:

Diagnosis –

Vitals on arrival : SBP- DBP- PR- Temp- SpO_2-

Radiological imaging impression-

TOAST Classification:

Large Artery Atherosclerosis-

Small Artery Occlusion-

Cardioembolism-

Caval Index-

Inspiratory IVC Diameter

Expiratory IVC Diameter-

LABS	Day 1	Day 2	Day 3
Hb.			
HCt			
TLC			
S.Urea			
S.Creat			
BUN			
BUN/Creat			
S.Na			
S.K			
Blood Glucose			
Plasma Osmolarity			

Severity	Day 1	Day 2	Day 3	Day 30 mRS
GCS NIHSS mRS				

Length of stay in Hospital :

Complications during stay :

Co-Morbidities- ☐ DM ☐ HTN ☐ CKD ☐ CLD ☐ AF

Signature of ER Resident

Signature of ER Consultant

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Age – Can it be a Navigational Factor in Dengue: A Study

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Abstract

Background: Dengue infections can occur as epidemics in India causing high morbidity and mortality. The awareness of risk factors can help in early recognising and treatment of severe forms and reduction in mortality. Age is one such intrinsic risk factor which can affect the outcome to a significant extent. *Objective*: To analyse the haematological variables affected by different age groups and its impact by comparison with other similar or clinical studies on complications of dengue. *Materials and Methods*: A total of 132 serologically proven Dengue positive cases were analysed along with relevant haematology data after tabulation against patients unique hospital identification number, age and sex. The data was collected over a one month period in November 2016 in Haematology Department of KIMS Hospital and Research Centre, Bengaluru. *Results*: Our study showed an age range between 5 months to 65 years with most patients in the 12-25 years group and an average of 32 years. We had 30% paediatric cases (≤12 years) and 70% non-paediatric cases (>12 years). The male to female ratio was 1.2:1 with a slight male predominance. The analysis of haematocrit patterns in association with age showed 70% paediatric cases with rise in haematocrit above reference range in comparison to 43% in adults. The highest haematocrit was 59.6 in adults as against to 47% in paediatric cases. Total white cell count patterns were uniform in both groups. The lowest count was noted in adults (1100 cells/cumm) as against paediatric (1900 cells/cumm) cases. The differential count showed marginal increase in lymphocytosis in non-paediatric (70%) as against paediatric (63%) cases, whereas neutrophilia (23% vs 50%) in paediatric and non- paediatric cases was more significant. Neutropenia was seen in a higher proportion of non- paediatric (38%) vs paediatric cases (18%). Platelet counts less than 1.0 lakhs/cumm was noted in 98% in non-paediatric as against 90% of paediatric cases. There was a significantly higher proportion of NS1 Antigen positivity noted in non-paediatric (34%) as against paediatric (15%). Paediatric cases had a higher association (85%) with antibody pattern as against non-paediatric (65%) cases. *Conclusion*: Age has a significant impact as a prognosticator in dengue and can play an important and decisive role in early diagnosis and management of severe cases.

Keywords: Dengue fever; Prognosticators; Age; Paediatric; Blood counts; Serology.

Introduction

Dengue, an arboviral infection presents as undifferentiated fever, dengue fever, dengue haemorrhagic fever and dengue shock syndrome [1]. It has been classified as non severe dengue without and with warning signs and severe dengue [2]. Most cases are self limited but complicated cases have a high morbidity and mortality [3].

Certain risk factors impact progression to severe dengue includes age, gender, race, nutritional status and blood group. These aid in early management and a reduction in morbidity and mortality [4,5].

Several studies have analysed the effect of age on dengue and claimed increased risk of severe dengue in children [6,7,8,9]. This is due to increased base line vascular permeability in children with increased plasma leakage leading to shock [9,10,11]. Some of these studies claim children < 5 years have a higher risk and evolve faster into severe dengue [11,12].

Other studies have suggested that adult have a higher risk for severity especially for internal haemorrhage [10,11] possibly due to increased risk of severe thrombocytopenia [13]. A few studies show elderly tend to have severe dengue (due to comorbidities and weaning immunity) [10].

Our study explores the haematological impact of age and its utility in predicting severity in dengue.

Aims and Obectives

The aim of the this study is to analyse the variations in haematological parameter seen in paediatric and non-paediatric group and to compare with similar clinical studies, to assess the risk group in dengue.

Materials and Methods

This is a prospective study conducted on 132 patients with dengue positive serology in KIMS Hospital, Bengaluru over a one month period in November 2016.

All patients with dengue positive serology (NS1, IgM, IgG or all) by rapid card method (standard diagnostic-Bioline alera) with results of relevant haematology tests – haematocrit, blood counts (obtained by automated haematology analyser – Syemex 1800e) with differentiated counts obtained from leishman stained peripheral smear (done as per hospital protocol to verify platelet counts) with age and sex details were included in the study.

The result of these tests were tabulated and analysed.

Patients with concomitant infection like Malaria, Typhoid along with dengue, those with normal, high platelet count and those without age details were excluded from the study.

Ethical Committee Clearance

This study consists of analysing data against the patients unique hospital identification number with age and sex details only. The anonymity of patients was maintained. The study was approved by Ethical committee of the hospital.

Results

Our study showed patients aged between 5 months to 65 years, most were in the 12-25 years age group and the average age was 32 years (Table 1).

There was a slight male predominance (M:F-1.2:1)

Non-pediactric group includes adolecents and adults and for simplicity sake will be categorised as adults in the study. The haematological parameters analysed included haematocrit and blood counts

Haematocrit: There was an increased proportion of paediatric cases showing a rise in haematocrit above reference range for age and sex, however rise in haematocrit over 20% for age and sex was seen more in adults (12%) than paediatric group (8%). The highest haematocrit in adult was 59.6% vs 47%

Table 1: Age and sex distribution of patients

Age Group	Number	Percent	Gender	Nember	Percent
Pediactric	40	30	Male	73	55
Non Pediatric	92	70	Female	59	45
Total	132	100		132	100

Table 2: Comparison of haematocrit (rise above reference range for ages)

Age Group	Normal		Rise in Haematocrit		Total (n)
	Number	Percent	Number	Percent	
≤12 years Pediactric	12	30	28	28	40
≥12 years Non Pediactric	52	57	40	40	92
Total	64	100	68	100	132

in children (Table 2).

Total WBC counts: There was no significant variation in the impact on WBC counts in the different age groups. Leucocytosis was marginally higher in non-paediatric group. The highest total count in paediatric group was 11350 cells/cumm, the lowest was 1000 cells/cumm whereas in non-paediactric it was 33410 cells/cumm and 1100 cells/cumm respectively (Table 3).

In both categories the percent of leucopenia was significant (40%)

Differential counts

There was a significant variation in the proportion of neutrophilia with 23% noted in paediatric group as against 5% in non-paediatric.

Differential counts cut off values were the standard reference ranges for particular age groups (Polymorphs-adults: 40-75%, children ≤ 12 yrs: 20-40%; Lymphocytes-adults: 20-45%, children: 40-60%) (Table 4).

Lymphocyte count: The highest lymphocyte count in non-paediatric group was 90% as against 80% in paediatric group lymphocytosis was marginally higher in adults

The number of significant atypical lymphocytosis (≥20%) was seen in a higher portion of paediatric cases as against to non-paediatric (57% vs 50%) significant variation was noted in the proportion of neutropenia cases which was higher in non-paediatric cases, 38% as against 8% in paediatric cases. The lowest neutrophil count in adults was 10%, in paediatric age 12% (Table 5).

Platelet Counts: An analysis of platelet counts showed lowest platelet count in paediatric group, was 15000 cells/cumm as against 8000 cells/cumm in non-paediatric group (Table 6).

There was a higher proportion of adults with

Table 3: Comparison of total WBC counts

Age Group	≤4000 cells/cumm		4000-11000 cells/cumm		≥11000 cells/cumm	
	Number	Percent	Number	Percent	Number	Percent
≤12 years Pediactric n=40	16	40	22	55	02	05
≥12 years Non Pediactric n=92	36	39	47	51	09	10
Total	52		69		11	

Table 4: Comparison of differential counts.

Age Group	Lymphocytosis		Neutrophilia		Normal pattern	
	Number	Percent	Number	Percent	Number	Percent
≤12 years Pediactric n=40	25	63	09	23	06	14
≥12 years Non Pediactric n=92	64	70	05	05	23	25
Total	89		14		29	

Table 5: Comparison of atypical lymphocyte counts

Age Group	<20%		≥20%	
	Number	Percent	Number	Percent
≤12 years Pediactric n=40	17	43	23	57
≥12 years Non Pediactric n=92	46	50	46	50
Total	63		69	

Table 6: Comparison of platelet counts.

Age Group	≤0.5 Lakhs/cumm		≤1.0 Lakhs/cumm		<1.5 Lakhs/cumm	
	Number	Percent	Number	Percent	Number	Percent
≤12 years Pediactric n=40	22	55	14	35	04	10
≥12 years Non Pediactric n=92	53	58	38	41	01	01
Total	75		14		05	

Table 7: Comparison of serology patterns

Age Group	NS1 Antigen pattern		NS1 +Ab pattern		Ab pattern	
	Number	Percent	Number	Percent	Number	Percent
≤12 years Pediactric n=40	06	15	18	35	16	40
≥12 years Non Pediactric n=92	31	34	26	41	35	38
Total	37		44		51	

thrombocytopenia 99% (≤1.0 lakh) as against to 90% in paediatric group.

Serology pattern: The serology pattern showed a higher proportion of non-paediatric cases in association with NS1 antigen (34%) and a higher proportion of paediatric cases in association with Ab pattern (85%) as against 65% in non-paediatric cases (Table 7).

Discussion

Our study showed most cases in in 12-25 years group, range 5 months -85 years (average age 32 years) in accordane with few studies [13] with slight male predominance.

There was higher proportion of cases with rise in haemocrit in paediatric group [14] where as 20% rise and highest haemocrit was more in non-paediatric cases. Few studies claim higher risk of Dengue shock syndrome in children [10,11] and others showed increased risk was more in adults [15].

The total WBC count showed no significant variations in our study in contrast to few studies which showed higher proportion of leukopenia in paediatric group [14,16].

Lymphocytosis was noted in a higher proportion of cases in paediatric category [16,17] but these included those ≤ 15 years unlike our study which included those ≤ 12 years.

There was significant atypical lymphocytosis in a higher proportion in children than adults, one study didn't find any difference between the two [15] in another it was more in non-paediatric group [14].

Neutropenia was noted in a higher proportion of cases in adults than children in accordance with few studies [14].

Few studies noted that leucopenia and lymphocytosis were prominent in adults (non paediatric) than paediatric group [13,16]. Our study noted that lowest total count and highest lymphocyte count were noted in non-paediatric group.

Platelet count showed that ≤ 1.0 lakhs/ cummwas seen in a higher proportion of non-paediatric as against paediatric group (99% vs 90%) in accordance with few studies [13,15,16]. But others claim higher proportion in paediatric group [11,17,18]. Our study showed higher proportion of severe thrombocytopenia ≤0.5 Lakhs/cumm in non-paediatric group [13]. The severity of thrombocytopenia was noted in non-paediatric group as the lowest platelet count was observed in this category [13]. Few studies noted that haemorrhagic tendencies were more in non-paediatric group [10,11,13].

The increased haemocrit was due to haemo concentration secondary to plasma leakage. An increase in plasma leakage is noted in children due to increased microvascular permeability [9,10,14].

Leucopenia is attributed to bone marrow suppression with lymphocytosis in acute phase [19]. Atypical (plasmacytoid) lymphocytes represent an augmented immune response to control the spread of the virus and neutropenia is due to marked degeneration of mature neutrophils with shift to left during febrile phase [20]. Thrombocytopenia is due to direct bone marrow suppression by virus and antibody mediated platelet destruction etc [21].

Leucopenia is an early severity predictor in dengue [4]. Atypical lymphocytosis was associated with thrombocytopenia and could be a marker of disease severity. Haemocrit and thrombocytopenia are criteria for diagnosis of Dengue haemorrhagic fever and thus prognosticators [4].

The serology pattern showed significant association of non-pediatric group with NS1 antigen (34%) as against the paediatric group (15%) whereas the Ab pattern (isolated and in combination with NS1 antigen) showed significant association (85%) with pediatric group than non-paediatric group (65%). NS1 antigen positivity correlates with increased viremia and is found in severe cases. Ab to NS1 antigen are cross reactive with platelets and endothelial cells and may cause haemorrhagic manifestations [18].

The take home points of the study could be

summarised as the risk of dengue shock syndrome is more in children than in adults, however in adults it could be more severe. This is in support of few studies claiming higher mortality in adults with dengue shock syndrome than children [15].

Neutropenia and thrombocytopenia were seen more in non-paediatric group suggestive of higher incidence of bone marrow suppression, contributing to severe disease with increased haemorrhagic manifestation in non-paediatric group, in accordance with few studies [10,11,13,15].

Limitation of study included small sample size, limited data to confirm findings and lack of clinical correlation.

Conclusion

Dengue can occur in epidemics in India. Early management is essential to limit the mortality rate. Age is an important factor which can influence the outcome in dengue infections as certain complications have an affinity to selective age groups. Awareness of this can go a long way in reducing morbidity and mortality in dengue.

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Acute Illicit Alcohol Poisoning: A Systematic Approach to Diagnosis and Management

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Abstract

Introduction: Illicit alcohol is spiked with various substances e.g. methanol, solvents, pesticides, sedatives and plant extracts. Patient with poisoning due to illicit alcohol are usually brought to the hospital in inebriated state and diagnosis in these patients becomes a challenge for the emergency physicians. We report our successful management of four patients who presented to us with illicit alcohol poisoning. We also tabulate the differential diagnosis, management and systematic approach to these patients. *Material and Method:* In July 2018, four patients were admitted in emergency department after consuming illicit alcohol and were managed expectantly. Retrospective review of the hospital record was done to retrieve the emergency department and ICU data. *Results*: Out of four patients, two were admitted in unconscious state on ventilator while other two were conscious and sober. Apart from methanol, other commonly associated poisoning with illicit alcohol were ruled out. Urine toxicity screen was negative. Two patients on ventilator were managed conservatively in the absence of definitive diagnosis. Both patients gradually improved and recovered uneventfully. On personal communication with bootleggers, it was observed that illicit alcohol is usually spiked with Nitrazepam to enhance the sedative effect. Nitrazepam is usually not detected by routine urine toxicology screen. *Conclusion:* Apart from methanol, illicit alcohol is spiked with several known and unknown substances. Emergency department physician should be aware of poisoning due to these substances and an approach to an unconscious patient due to consumption of illicit alcohol.

Keywords: Illicit alcohol; Methanol, Solvents, Pesticides, Sedatives.

Introduction

Alcohol is highest consumed beverage across the globe and this includes both legally and illegally produced alcohol [1]. Illegal alcohol usually made from locally available ingredients such as molasses, sugar, fruits, vegetables, plants or residues of wine production [2]. In developing countries including India, there is huge demand for illegal alcohol in lower socio-economic strata, especially in the urban areas; because it is much cheaper [3-5].

To fulfill the demand and to reduce the cost, substances like methanol and other solvents are added to illicit alcohol. Other substances added are aluminum nitrate and aluminum sulphate to enhance the fermentation and pesticides, sedatives and plant extracts to enhance the inebriating effect of alcohol. Studies have established a higher mortality risk for people who consume illicit alcohol. Increased mortality is mostly attributed to higher levels of alcohol consumption in people who consume illicit alcohol rather than due to its toxic ingredients. However, issue is more complicated than simply increased consumptionof illicit alcohol [1,6].

Methanol poisoning is the most published health consequence of consumption of illicit alcohols due to ingredients other than ethanol [1]. However, majority patients who present to the hospitals with toxicity after drinking illicit alcohol; their symptoms are not attributable to methanolpoisoning. These patients present a new challenge to the emergency physicians as patients are usually inebriated, constitution of illegal alcohol is unknown and alcohol is not available for analysis. Therefore, treatment is mostly empiric.

In this article, we present our experience of management of four patients who presented to our hospital due to poisoning with illicit liquor and present outline to suspect these poisoning and treatment.

Material and methods

Four patients, plumber by occupation consumed illicit alcohol in varying quantity from 150 ml to 600 ml on 3rd July 2018 at 10:30 pm. On 4th July, at 6:00 am, all four patients were admitted to the local hospital with variable complaints. All the patients were managed for nearly 12 hours at local hospital and were later referred to our hospital at 8:45 pm on 4th July 2018.

Results

First patient, 30 years male presented with one episode of vomiting associated with giddiness and blurring of vision followed by loss of consciousness. On examination, patient was hemodynamically stable. Breathing was slow and shallow hence patient was intubated and mechanically ventilated. Blood investigation were normal. Patients was given nasogastric infusion of diluted absolute alcohol. Over next 12 hours, patient remained hemodynamically stable but, unconscious; hence, he was referred to our hospital.

At presentation in our hospital, patients was unconscious, intubated, afebrile, and not responding to deep painful stimulus. There was no spontaneous respiration. Cardiorespiratory examination and vitals were normal. Oxygen saturation (SpO_2) by pulse oximetry 99% on ventilator. Deep tendon reflexes (DTR) were absent. Bilateral pupils were dilated and not reacting to light due to effect of topical mydriatics and fundus examination was normal. In view of history of illicit alcohol drinking, possibility of solvent poisoning or Wernicke's encephalopathy were considered. Patient was managed with intravenous fluids including 5% dextrose, inj. thiamine and inj. folinic acid. Patient was loaded intravenously with diluted absolute alcohol at dose 600 mg/kg for 1 hour followed by maintenance dose at 100 mg/kg/hour. As shown in Table 1, blood and urine investigations including serum and urine osmolality were normal. Blood methanol level was 0.004 mg/dl. Urine toxicology screen was negative. Blood lead level was normal. Serum and red blood cell (RBC) cholinesterase level and total creatinine phosphokinase (CPK) level were normal (Table 1).

In view of normal methanol levels, absence of acidosis in arterial blood gas analysis (ABG), normal serum and urine osmolality, absence of crystals in urine, and negative toxicology screen; absolute alcohol infusion was stopped and supportive management was continued. Patient regained consciousness after 6 hours with return of spontaneous respiration but had quadriplegia, ptosis and ophthalmoplegia and DTR were absent. On day 2, patient started to respond to verbal commands. From day 2 to day 4, muscle power gradually improved to 5/5 in all four limbs, ophthalmoplegia and ptosis also recovered completely and DTR became normal. On day 4, patient developed adequate respiratory effort hence was extubated. By 7th day, patients was able to carry out his daily activities without assistance and was discharged on 10th day. At discharge, patient was conscious, oriented and was able to walk without support.

Second patient, 30 years male was admitted with complains of giddiness and blurring of vision started at 4:00 am followed by rapidly progressive quadriparesis beginning distally first in toes and fingers culminating into quadriplegia followed by loss of consciousness in next two hours. At presentation, patient was unconscious, respiration was slow and shallow. Patient was intubated and mechanically ventilated. Same management as in patient 1 was started. However, as there was no improvement in the sensorium of the patient; hence patient was referred to our hospital along with 1st patient.

At presentation in our hospital, patient was on mechanical ventilation. Hemodynamics were stable and SpO_2 was 99%. Cardio-respiratory examination was normal. Neurologically, patient was unconscious, no spontaneous respiration, not responding to deep pain stimulus with absent DTR. Pupils were dilated due to effect of topical mydriatics. Fundus examination showed

Table 1: Blood and urine investigations of the patients

	Patient-1			Patient-2			Patient 3		Patient 4	
	4th July	**6th July**	**10th July**	**4th July**	**6th July**	**10th July**	**4th July**	**6th July**	**4th July**	**6th July**
pH	7.78	7.49	7.4	7.51	7.49	7.4	7.58	7.49	7.49	7.49
HCO_3^- (meq/l)	36.9	15.5	18.8	29.6	15.5	18.8	24	15.5	28	19.5
pO_2 (mmHg)	154	210	99.5	147	210	99.5	97	210	97	210
pCO_2 (mmHg)	30.6	21	28.1	33.6	21	28.1	28.8	21	30	38
Hb (g/dl)	14.2	13.7	12.5	13.8	13.7	12.5	14.7	13.7	10.6	11.7
Urea (mg/dl)	12.3	26.5	36	22.4	26.5	36	19.9	12.2	60.9	13.6
Cr (mg/dl)	0.93	1.3	1.1	0.73	1.3	1.1	0.79	0.97	2.83	0.98
Na^+ (meq/l)	137	136	138	135	136	138	140	139.4	145.5	137
K^+ (meq/l)	4.66	3.68	4	4.3	3.68	4	4.16	3.92	3.97	4
RBS (mg/dl)	98			95			88		90	
Bil (mg/dl)	0.86	0.57	0.56	1.0	0.57	0.56	0.63	0.72	0.6	1.08
Serum Osmolality (mosm/kg)	269			274						
Urine Osmolality (mosm/kg)	525			637						
Urine Examination	N			N						
Lead (µg/dl)	1.28			1.28						
Total CPK (U/l)	85.4			110						
Plasma CE (U/l)	5780			6200						
RBC CE (U/l)	2225			2834						
	Urine toxicology screen									
BDZ	Neg			Neg						
Barbiturates	Neg			Neg						
Cocaine	Neg			Neg						
Amphetamines	Neg			Neg						
Morphine	Neg			Neg						
THC	Neg			Neg						

HCO_3^-- Bicarbonate, pO_2- Partial pressure of oxygen, pCO_2- Partial pressure of carbondioxide, Hb- Hemoglobin, Cr- Creatinine, Na^+- Sodium, K^+- Potassium, RBS- Random blood sugar, Bil- Bilirubin, N- Normal, CPK- Creatine phosphokinase, CE- Cholinesterase, BDZ- Benzodiazepines, THC- Tetrahydrocanabinoids, Neg- Negative.

mild peri-papillary hyperemia in both the eyes. Management similar to patient 1 was begun. As in patient 1, blood and urine investigations were non-contributory (Table 1). Patient didn't have improvement in neurological status in 6 hours after admission. Therefore, absolute alcohol infusion was stopped and supportive treatment was continued. Possibility of an atypical poisoning was considered and two cycles of hemodialysis were performed 12 hours apart. However, patient continued to be unconscious with no spontaneous respiratory effort. On day 3, patient regained spontaneous respiration. From day 4 to 10, patient become conscious with response to verbal commands; quadriparesis, ptosis and opthalmoplegia gradually improved to 4/5 power on 7th day and 5/5 power on 10th day. On day 7, patient had adequate respiratory effort and was extubated. Repeat fundus examination on day 7 was normal. Electromyelogram and nerve conduction studies done on 9th day and magnetic resonance imaging (MRI) brain done on 10th day were normal. At discharge on day 15, patient was conscious, oriented and was able to walk without support.

Other two patients, a 24 years male complained of burning sensation over back and bilateral upper limb and a 32 years male without any complaints were also referred for observation along with previous two patients. Both the patients were conscious and had normal cardio-respiratory and neuro-muscular examination. Blood and urine investigations were also normal (Table 1). Blood methanol levelwas normal. Both the patients were kept under observation and were discharged on day 3.

Discussion

In India, in states with alcohol ban including

Gujarat, there is huge demand for illicit alcohol [7]. Often, illicit alcohol is spiked with methanol, other solvents, pesticides, sedatives, plant extracts or chemicals to hike its potency. The health hazards due to these toxic ingredients is difficult to suspect and even more difficult to detect [1,6-8].

Patients with toxicity due to illicit alcohol present to the emergency department hours after consumption with varying symptoms. Initial symptoms are usually drowsiness, unsteadiness, and disinhibited behavior which may later culminate into headache, vomiting, abdominal pain, vertigo, visual disturbance and neurological symptoms. If left untreated, patient may develop coma, convulsions, and death from respiratory arrest. However, based on presenting symptoms, it is almost impossible to differentiate ethanol toxicity from methanol, solvent or any other toxin. Symptoms vary from patient to patient and also depends upon dose of alcohol or toxin consumed. In our series, all four patients had variable presentation varying from minimal symptoms in 3rd and 4th patient to muscle paralysis and coma in 1st and 2nd patient.

In our series, two patients who were in coma, our initial suspicion was methanol or solvent poisoning based on history of illicit alcohol consumption and managed them with infusion of diluted absolute alcohol. However, normal blood methanol level, absence of increased anion gap metabolic acidosisin ABG, normal serum and urine osmolality and absence of crystals in urine ruled out the methanol or other solvent poisoning. Further, lack of improvement in the sensorium after absolute alcohol infusion further ruled out the possibility of methanol or solvent poisoning. Other suspicion was pesticide poisoning as illegal alcohol is usually spiked with pesticides and both 1st and 2nd patient presented with acute onset muscle paralysis. However, normal blood and RBC cholinesterase levels and lack of response to inj. Neostigmine eliminated the possibility of pesticide poisoning. As ethanol, solvent and pesticide poisoning was ruled out, other etiologies for acute onset muscle paralysis with coma were sought. Negative urine toxicology screen ruled out the poisoning due to commonly detected benzodiazepines, barbiturates, cocaine, cannabinoids, amphetamine and morphine.

After ruling out the poisoning due to commonly known toxins, we continued supportive management in first two patients. First patient had spontaneous improvement in sensorium after 6 hours while second patient continued to be in coma despite two cycles of hemodialysis and regained spontaneous respiration after 36 hours and consciousness after 60 hours of admission. It is difficult to ascertain whether improvement in sensorium in both the patients was because of decrease in blood levels of unknown toxin or reduction in blood ethanol level or combined effect of both. Complete recovery of sensorium and muscle power with-in 5-7 days clearly demonstrates that toxin had acute, severe and reversible CNS depressant effect with muscle paralysis without involvement of the cranial or peripheral nerves. Our assumption was further supported by normal nerve conduction study and MRI brain performed in second patient before discharge.

On personal interrogation with bootleggers, it was learned that illicit alcohol is commonly spiked with different benzodiazepines like alprazolam, diazepam, and lorazepam and nitrazepam. We believe that illicit alcohol that our patients consumed was spiked with nitrazepam. Nitrazepam is a hypnotic and anticonvulsant with strong sedative and skeletal muscle relaxant properties. It has high abuse potential and potentiates the CNS depressant effects of alcohol. Nitrazepam if consumed along with ethanol may lead to respiratory depression, muscle paralysis and coma as seen in our first two patients. However, we could not ascertain why other two patients were asymptomatic. Nitrazepam is usually difficult to detect by routine urine toxicology screen. Our diagnosis is still speculative due to absence of symptoms in other two patients and lack of laboratory evidence of toxicity.

Suggested management algorithm for patient with acute illicit alcohol toxicity: Patients presenting with acute illicit alcohol intoxication are usually inebriated and history is either not available or is unreliable. Therefore, toxicity due to all the ingredients mixed in illicit liquor should be considered.

Initial management of unconscious patient with illicit alcohol poisoning in emergency is management of airway, breathing and circulation. In hemodynamically unstable patient, inotropes along with fluids should be started. Initial management should include intravenous dextrose infusion along with inj. Thiamine and inj. Folinic acid to prevent precipitation of Wernicke's encephalopathy [9]. After stabilization, complete examination including assessment for injuries, eye examination for size and reaction of pupils to light and fundus examinationand complete neuro-muscular assessment should be performed.

Table 2: Battery of investigation to be performed in patients with suspected illicit alcohol intoxication and their clinical importance

Renal function test	Renal function,
Blood urea nitrogen	Renal failure
Creatinine	calculate the osmolality and osmolal gap
Complete blood count	Assess the presence of a macrocytic anemia (high mean corpuscular volume, low
Hemoglobin	hemoglobin, low hematocrit)
Hematocrit	Assess for hemolysis
RBC count	Toxic metabolic process (elevated WBC count)
WBC count	
Platelet count	
Electrolytes	Electrolyte disturbances
Sodium	Calculate the anion and osmolal gaps
Potassium	
Chloride	
Glucose	
Osmolality	
Blood toxicology screen	Presence of ethanol, methanol, ethylene alcohol and isopropyl alcohol.
Ethanol	Monitor ethanol levels as a therapeutic regimen after toxic alcohol ingestion
Methanol	Organophosphorus poisoning
Ethylene glycol	Lead poisoning
Ketone	Myonecrosis and rhabdomyolysis
Serum and RBC cholinesterase	
Serum Lead	
Creatine phosphokinase (CPK)	
Random blood sugar	Presence of hypoglycemia
	calculate the osmolality and osmolal gap
Lactic acid	Determine other causes of metabolic acidosis
Osmolality (measured)	Calculate the osmolal gap
Arterial blood gas	Assess ventilatory status
pH	Confirm the presence of metabolic acidosis
pO_2	High anion gap or normal anion gap metabolic acidosis
pCO_2	
HCO_3--	
Lactate	
Anion Gap	
Urinalysis	Detect the presence of ketones (ketoacidosis)
Ketones	Oxalate crystals (ethylene glycol)
Oxalate crystals	Myonecrosis and rhabdomyolysis
Myoglobin	
Urine toxicology screen	Toxicity due to drugs of abuse
Benzodiazepines	
Barbiturates	
Cocaine	
Cannabinoids	
Amphetamine	
Morphine	
Liver function test	Acute or chronic liver failure
Bilirubin	Hepatic encephalopathy
SGPT	
SGOT	
Alkaline phosphatase	
Total Protein	
Albumin	
Serum osmolality	2 (Na+ + K+) + Glucose/18 + Blood Urea Nitrogen /2.8
Anion gap	$Na+ - (Cl^{-} + HCO_3^{-})$

RBC: Red Blood Cells, WBC: White Blood Cells, SGOT: Serum Glutamate Oxaloacetate Transaminase, SGPT: Serum Glutamate Pyruvate Transaminase, Na: Sodium, K: Potassium, Cl: Chloride.

Laboratory investigations as shown in Table 2 should be sent and serum osmolality and anion gap should be calculated [10].

As shown in Table 3 and 4, clinical examination findings at presentation and associated change in serum and urine osmolality, metabolic acidosis and urinary crystals can help in differentiating various alcohols and alcohols from other substances [11-13]. In patient with high suspicion of solvent poisoning, supportive management is begun along with empiric

Table 3: Clinical features and lab investigations to differentiate different poisoning associated with illicit alcohol.

	Clinical feature and lab investigations							Metabolic Acidosis with increased anion gap	Increased osmolal gap	Serum acetone	Urine Ketone	Urine oxalate crystal
	CNS	**Cranial and peripheral nerves**	**Eye**	**Muscle paralysis**	**Acute kidney failure**	**Other**	**Muscle necrosis**					
Ethanol	Inebriation coma	Wernicke's Encephalopathy		No	No		+/-	-/+	+	-	-	-
Methanol	Inebriation Acute onset parkinsonism	Delayed	Pupil dilated	No	No	Blurred vision	-	+	+	-	-	-
Isopropyl Alcohol	Inebriation Stupor coma	-	Optic disc erythmatous Atrophy later	No	No	Acitonemia acitonuria	-	-	+	+	+	-
Ethylene Glycol	Inebriation	Delayed	Pupil pin pointed	No	Yes	Oxalate crystalluria	-	+	+	-	-	+
Diethylene Glycol	Headache	Delayed		No	Yes	Pancreatitis	-	-/+	+	-	-	-
OPC	Anxiety restlessness insomnia coma seizures	No		Yes	No		-	-	-	-	-	-
Sedative and Hypnotics	Stupor Coma	No	Pupil pin pointed	Yes	No		-/+	-	-	-	-	-
Plant Extract	Stupor Coma	No	Pupil pin pointed	Yes	No		-/+	-	-	-	-	-
Lead	Encephalopathy Seizures	Yes		No	Tubular defects	Anaemia, colic	-	-	-	-	-	-
Mixed	Inebriation Stupor Coma	+/-		+/-	+/-		+/-	+/-	+/-	+/-	+/-	+/-

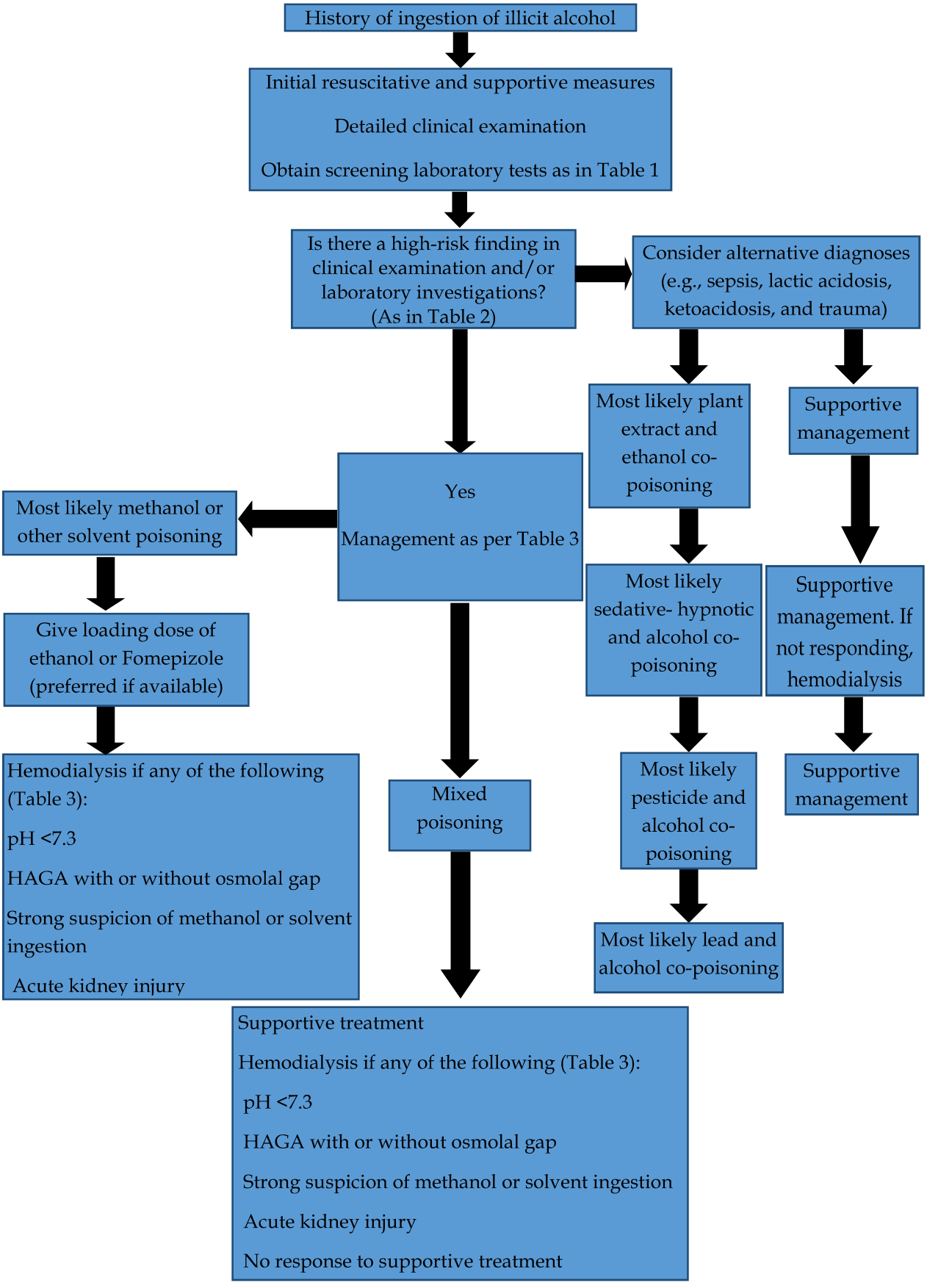
CNS: central nervous system, OPC- Organophosphorus,

Table 4: Common diagnostic tests, prognostic factors and management of poisoning associated with illicit alcohol.

	Diagnostic clue	Management	Poor prognostic factors	Indication of hemodialysis	Discontinuation of hemodialysis	Additional treatment
Ethanol	HAGA, trace positive or negative nitroprusside reaction with increase with H2O2; hypoglycemia; Increased osmolal gap	Administer intravenous fluids including dextrose and NaCl	Blood pH <7.0; Severe comorbid conditions	Rarely needed if blood ethanol level >500mg/dl, respiratory failure, shock, lactic acidosis	-	Dextrose and NaCl Thiamine
Isopropyl Alcohol	Increased osmolal gap without HAGA	Supportive	Severe LA; Hypotension; Serum isopropanol level ≥200 to 400 mg/dl	serum level 200 to 400 mg/dl or in presence of marked hypotension or coma		
Methanol	Increased osmolal gap with HAGA Visual difficulties with optic papilliti	Gastric lavage, induced emesis, or use of activated charcoal to remove alcohol from gastrointestinal tract needs to be initiated within 30 to 60 min after ingestion of alcohol. Administration of ethanol or fomepizole to delay or prevent generation of toxic metabolites needs to be initiated while sufficient alcohol remains unmetabolized	Blood pH 7.1; LA; severe coma; severe hypotension; serum methanol 50 to 100 mg/dl	Methanol >10 mg/dl. HAGA with or without osmolal gap Strong suspicion of methanol ingestion.	pH normalized and methanol levels <10-15 mg/dl. If measurement of methanol not available use return of blood pH and serum osmolality to normal as goals of therapy	Folinic or folic acid. HCO3- for severe acidosis
Ethylene Glycol	Increased osmolal gap with HAGA ARF with increased osmolal gap Calcium oxalate crystals in urine		Blood pH <7.1; glycolate level >8 to 10 mmol/L; ARF requiring HD; diagnosis >10 h after ingestion; serum ethylene glycol >50 to 100 mg/dl	Ethylene glycol >10 mg/dl. HAGA with or without osmolal gap Strong suspicion of ethylene glycol ingestion.	pH normalized and ethylene glycol levels <10-15 mg/dl. If measurement of ethylene glycol not available use return of blood pH and serum osmolality to normal as goals of therapy	Thiamine and pyridoxine HCO3- for severe acidosis
Diethylene Glycol	Increased Osmolal gap with HAGA increased osmolal gap with ARF increased osmolal gap with coma		Blood pH <7.1; ARF requiring HD; severe coma; ingestion of >1.34 mg/kg body weight	Increased osmolal gap, HAGA, ARF or high suspicion of ingestion	recovery of renal function, normalization of acid-base parameters and osmolal gap	-
Organophosphorus	Decreased serum and RBC Cholinesterase level	Gastric lavage. Inj. Atropine. Iv Fluids and supportive	Pupil pin pointed	-	-	Inj. Pralidoxime.
Sedative and Hypnotics	Acute onset Muscle paralysis with normal investigations	Supportive management	Pupil pin pointed	-	-	Flumazanil for Benzodiazepines
Plant Extract	Acute onset Muscle paralysis with increased CPK or normal investigations	Supportive management	-	Not required	-	-
Lead	Encephalopathy, motor neuropathy					
Mixed	Combination of any of above investigations	Supportive management	Depends on type of poisoning	If any of above criteria fulfilled Low threshold for hemodialysis if no response to supportive treatment even in absence of above criteria	Same as above Patient's neurological status improves	

HAGA: high anion gap acidosis, LA: Lactic Acidosis,

Chart 1: Flow chart for systematic approach to patient with illicit alcohol poisoning



intravenous absolute alcohol or Fomepizole while awaiting the results of investigations. If the results of laboratory investigations are negative for solvent poisoning, absolute alcohol or fomepizole infusion can be discontinued safely. Gastric lavage should be reserved for selected patients who present within 30-60 minutes of ingestion; otherwise gastric lavage may result in more harm than benefit. In majority patients, empiric treatment would suffice. However, all the patients should be evaluated for poor prognostic signs at time of presentation or during treatment. Most important poor prognostic sign is severe metabolic acidosis at presentation. In patients with solvent poisoning, hemodialysis is reserved for patients with acute renal failure, severe metabolic acidosis or high blood levels of solvent at time of presentation. In patients with suspected or proven severe co-poisoning due to barbiturates, organophosphorus, lead or mixed poisoning, hemodialysis may be helpful [14-16]. Hemodialysis is also not harmful if does not benefit, in patients with benzodiazepine and plant extract poisoning [17,18]. Therefore, patient with illicit alcohol poisoning with ominous signs at presentation or patient not responding to the treatment, there should be low threshold for hemodialysis. Hemodialysis should be discontinued once blood pH improves, blood level of solvent decreases or clinical condition of the patient improves.

Chart 1 shows flow chart for systematic management algorithm for patient presenting with ingestion of illicit alcohol. Use of a systematic approach in the poisoned patients with illicit alcohol can reduce the morbidity and mortality of these patients.

Conclusion

Illicit alcohol is usually spiked with methanol, other solvents, pesticides, sedatives, plant extracts and chemicals to hike its potency. High index of suspicion, detailed physical examination and laboratory investigation are invaluable for diagnosis. Systematic approach and prompt treatment are key to improved clinical outcome. In patients with undiagnosed toxicity, there should be low threshold for hemodialysis.

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Case Report

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Delayed Presentation of Rectum and Sigmoid Colon Injury Following a Blunt Abdominal Trauma

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Abstract

Introduction: The delayed presentation of Rectal Injuries / perforations following a blunt trauma to the abdomen adds significantly to the morbidity, mortality and increased hospital stay and pain to the patient. *Case Report:* A 42 yr old male patient has been admitted in the emergency department with history of blunt abdominal trauma after getting struck between the steering wheel and the drivers seat for a long time in a RTA. His vitals were normal. He was discharged home after the CT scan (both plain and contrast) showed no organ injury as reported by the radiologist. Later after 12 hrs he was re-admitted in the emergency with severe abdominal pain and guarding. X- ray erect abdomen showed air under diaphragm and it was later diagnosed as rectal perforation in CT and also taken to OT for surgical repair. *Conclusion:* Rectal injury or perforation following a blunt abdominal trauma is such a trivial thing in the beginning to find even on CT scans. It takes time to evolve into fully blown acute abdomen with peritonitis. So there is a need for re-evaluation in all patients of blunt abdominal trauma who appears to be normal on investigations out of proportion to the injury that they sustained.

Keywords: Rectal perforation; Blunt abdominal trauma; CT scan.

Introduction

The rare occurence of colonic injuries following blunt abdominal trauma and lack of definitive diagnostic modality can lead to delay in diagnosis and treatment in the initial stage which can ultimately result in high morbidity and mortality.

Case Presentation

A 42 year old man involved in a Motor Vehicle Accident (MVA) had been brought to our emergency department.

Mechanism: patient was driving lorry and had head on collision with another lorry and he was stuck between the steering wheel and the driver's seat.

Clinical Examination

Primary survey and vital signs: ABCDE – Normal. BP – 152/102 mm Hg, Pulse – 94 bpm.

Secondary survey: tenderness in left iliac fossa.

Initial ER Investigations:

E Fast - Negative.

Erect Chest X Ray: Normal Study (Fig. 1).

The initial leucocyte count was 12040.

CECT Abdomen and pelvis: No solid organ injury or free fluid in the abdomen (Fig. 2 and 3).

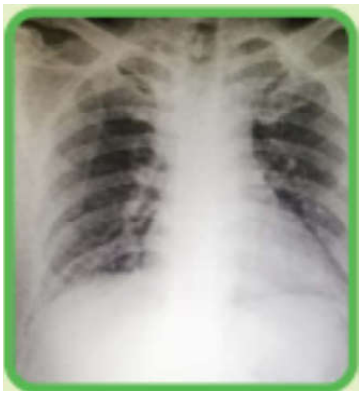

Fig. 1: Initial X ray

Table 1: CBC

Hemoglobin	13.8	g/dl
Total count	12040	Cells/mm^2
Neutrophil	86.0	%
Lymphocyte	9.2	%
Platelet count	199000	Cells/mm^2
PCV	39.7	%

Course in the hospital:

Surgical consultation was obtained and patient was admitted to observation unit. During initial 6 hours of observation patient remained vitally stable with no signs and symptoms suggestive of intra abdominal injury. The patient was discharged on request after detail advice regarding warning signs and symptoms and to return SOS.

Return To ER:

6 hrs after the discharge patient returned to the emergency department c/o severe pain abdomen and abdominal distension. On examination abdomen was distended with diffuse tenderness and guarding. Repeat chest X ray was obtained (Fig. 4).

Management:

Immediately taken to OR for explorative laparotomy. Primary closure of sigmoid colon perforation and loop ileostomy was done and he was discharged after complete recovery a week later (Fig. 5).

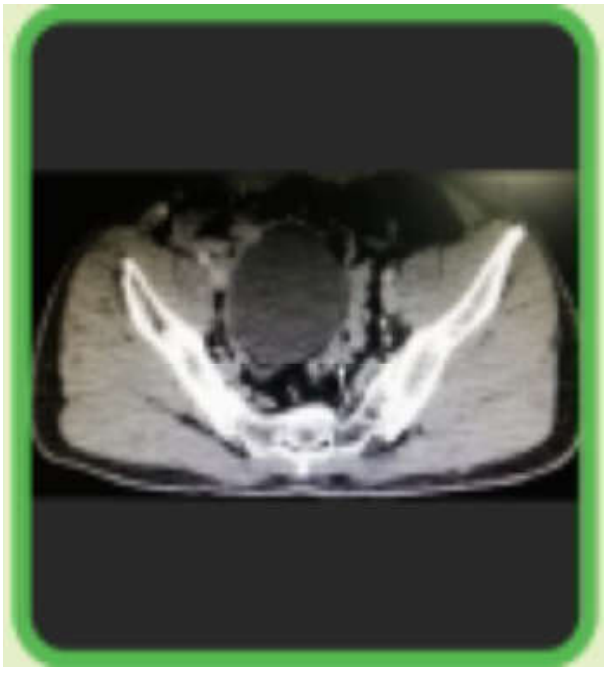

Fig. 2: Initial CT scan white arrow - Sigmoid colon

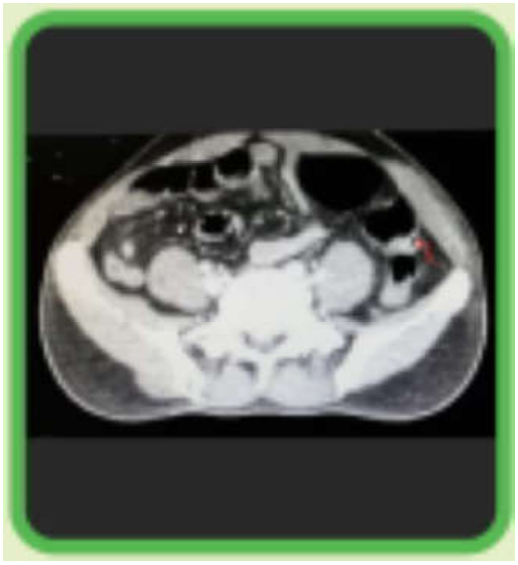

Fig. 3: Initial CT scan Red arrow - Descending colon

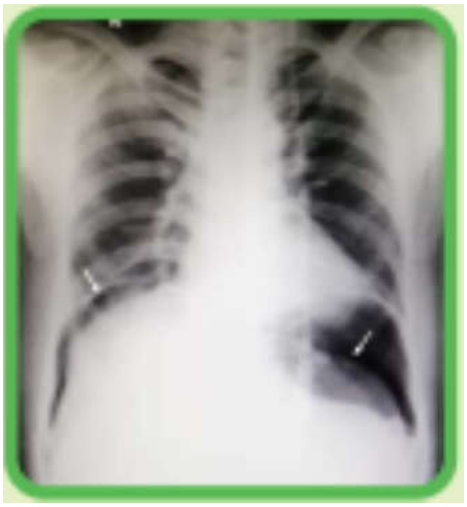

Fig. 4 Chest X ray after re-admission

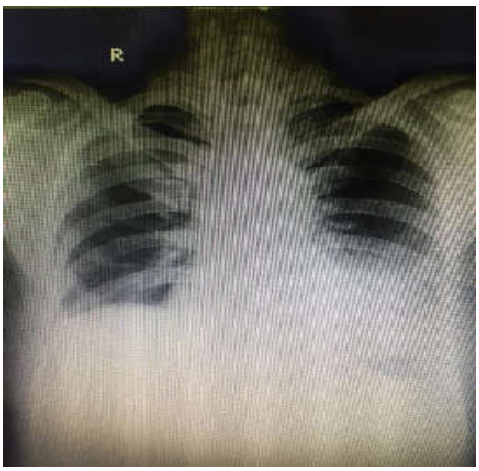

Fig. 5: X ray at the time of discharge

Discussuion

Traumatic colonic injuries

1. Mechanism – Penetrating abdominal injury is much more common as compared to blunt abdominal injury (with incidence as low as 1.1% and most common reason being motor vehicle accident) [1,2].
2. Anatomy – Transverse colon is most vulnerable and sigmoid colon is least vulnerable for injuries [1].
3. Imaging – No imging modality has 100% sensitivity and specificity for diagnosing colonic injuries in initial stages. Diagnostic value of contrast CT scan in initial stages remains controversial [1].
4. Diagnosis – Dangerous mechanism of injury + Serial physical examination + Serial imaging if required + High index of suspicion.
5. Prognosis – prognosis depends upon the time between arrival to the emergency department and surgery. Rate of complications is higher if duration is more than 24 hrs [2].

Conclusion

Colonic injury secondary to blunt abdominal trauma is rare and management has the potential to get delayed due to difficulties in establishing a conclusive diagnosis. Clinicians should maintain a high index of suspicion when evaluating patients with dangerous mechanism of injury and initial normal ER examination. Patients should be examined repeatedly and should undergo serial imaging if indicated to reach definitive diagnosis.

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Case Report

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Intussusception in Adults: A Case Report

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Abstract

Abdominal pain is one of most frequent presenting complain in ED among adult population. Intussusceptions although rare, can be consider as a cause of bowel obstruction in adults. Its occurrence in adult is not as common as in children; also difficulty to diagnose as patient usually present with non-specific abdominal pain or features of obstruction. Most sensitive diagnosis is computed tomography and adult intussusceptions rarely resolved without surgical interventions. Delay in diagnosis and treatment may leads to dangerous consequences. Here we have reported a case of 20 year old female with long standing pain abdomen found to have intussusceptions.

Keywords: Intussusceptions; Current jelly; Telescoping.

Introduction

Intussusception occurs when a portion of the alimentary tract telescopes into another segment, commonly ileum invaginates into the upper colon, bringing the mesentery with it (ileocolic) [1]. This leads to contriction of mesentery and bowel ischaemia. Children mostly present with classical triad of intussusception; abdominal pain, current-jelly stool and palpable tender abdominal mass where as in adults predominance of nausea, vomiting, abdominal distension/pain or any other non-specific symptoms distorted the clinical diagnosis [2,3].

Adult intussusceptions represent about 5% of all cases of intussusceptions and account a minor percentage (1%-5%) of intestinal obstruction [4]. As per their locations it is classified into four categories: enteric, ileocolic, ileocaecal and colonic [5]. Etiology of intussusceptions in children usually primary and benign where as in adults Mackel's diverticulum, carcinoma, polyps and malignancy bears a major risks.

Case Report

20 year old female was presented to emergency with complain of severe epigastric pain, progressive in nature 10-12 days associated with bilious vomitus 2-3 episodes since 1 day. She had no history of any commorbity previously and not on any regular medication. She passed flatus normally. She denied for any history of chest pain, fever, weight loss, burning micturation, any vaginal discharge and trauma.

On Examination

Airway Assessment : Patent

Breathing Assessment

Respiration (RR/min) : 18/Min

Laboured : No

SpO_2 : 98% on Room Air

Circulation

Pulse : 90/Min

BP : 100/70 MM HG

Peripheral Pulses : Yes

Temperature : 98 F

Cardiac Monitor : Sinus rhythm

GRBS : 104 mg/dl

LMP : Regular

Systemetic Examination

HEENT :	No Pallor/Icterus/Cyanosis/ Dehydration.
CHEST :	B/L Air entry equal
CVS :	S1S2 Heard
ABD :	Soft, Mild epigastric tenderness with distension, no organomegaly, BS +
EXT :	Warm, No Pedal Edema
Neuro :	Conscious, oriented, no focal neurological deficit
	Reflexes were normal
	Plantars : B/L downgoing

Ample History:

Allergies : No Known Drug Allergy

Medications : No past medical/Surgical history

Provisional Diagnosis was made as SAIO with Dyselectrolytemia, Cholelithiasis, Pancreatitis.

She was evaluated in emergency and investigated for her symptoms. Her Ultrasonography of abdomen s/o small bowel obstruction (? Intussusceptions). She was planned for CT whole abdomen, features s/o Jejunum-jejunal Intussusception with mucosal thickening of multiple small bowel loops with enlarged mesenteris lymph node? infective enteritis. General Surgery and gastroenterology consultation was done, Intravenous fluid, IV Antibiotics, Ryles' tube started and admitted for further intervention. As per Surgery consultation, pt was taken to operating room for Laproscopy and reduction of intussusceptions. Intra-operative findings revels small bowel intussusceptions at two places with jejunal congestion and hyperaemic but apperantly healthy tissue. Post operative period was uneventful and patient's condition improved in subsequent hospital stay.

Lab Reports

Blood gas report :

PH : 7.3, PO2 : 100, PCO2 : 33, HCO3 :19

Na^+ : 133, K^+ : 3.3, CL : 110, GLU : 148, LAC :0.8

Complete blood counts

WBC – 6.6, RBC – 4.2, Hb – 12.5, Plt-1.76 L

Differential counts : N-75 M-5 L-18 E-2

Electrolytes

Na^+ – 132, K^+ – 3.7

Renal Function Test, Liver function tests, Coagulation profile are within normal limits.

CXR-WNL

Abdomen ERC/Supine – Apparently normal.

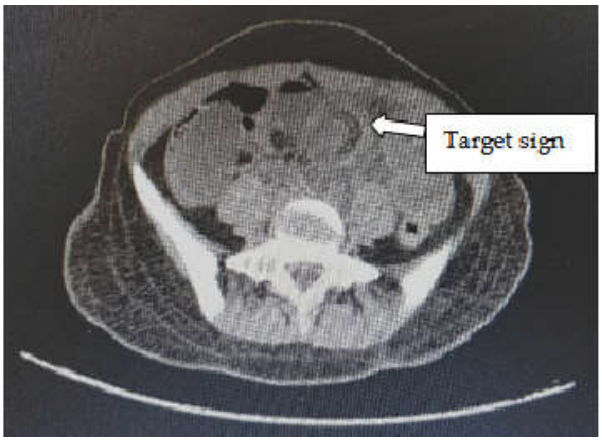

Fig. 1: CT Abdomen s/o Target Sign

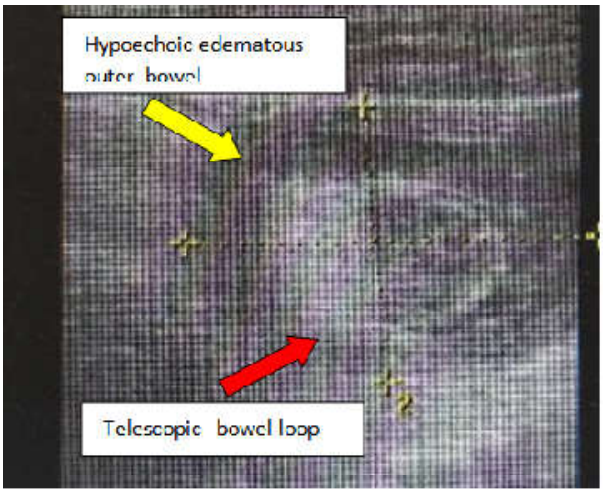

Fig. 2: USG Whole Abdomen

Discussion

Intussusception as an infrequent cause of intestinal obstruction was first reported in 1674 by Barbette of Amsterdam [6]. Clinical features of adult intussusceptions varies as mostly present with features of obstruction eg, nausea, vomiting, abdominal pain and are often long standing. we have reported a young female having long standing abdominal pain presented with features of obstruction.

Abdominal plain radiograph were consided as

initial diagnostic tool to evaluate any features of obstruction. Abdominal x-ray with multiple air – fluid level (usually more than 3) rise suspicion of an obstruction. Ultrasonography gives more clue about it as it may shows classical signs ("target sign" or "doughtnut sign" in transverse view Figure 2) or pseudo-kidney sign in longitudinal view.

Contrast enhanced abdominal CT considered as a gold standard with accuracy 58%-100%. Typically it suggest a soft tissue mas "target" or "sausage-shaped" mass consisting an outer intussuscipiens and inner intussusceptum. Abdominal CT scan came out as a best modality to comment on the location, length and diameter of intussusceptum, possibility of strangulation and relationship with surrounding tissue that guides the operative surgeons. It is also used to see if there any proximal bowel obstruction or not (lead point) and staging in case of malignant mass [7,8]. Adenocarcinoma is the most common malignant lead point in the colon, whereas metastasis is the most common malignant lead point in the small intestine [9,10].

Symptomatic intussusceptions in adult requires operative intervention mainly because of underlying structural abnormality. It involves exploratory laparotomy followed by resection of ischaemic bowel segment. Reduction of intussusceptions preoperatively by barium or air generally not recommended due to increased risks of perforation of bowel leads to spread of infection. Manual reduction can be considered with consultation of surgeons if diagnosis of benign lesion has been established [11,12].

Intussusception was previously believed as a disease of pediatric population, but due to radiological advances and use of abdominal CT scans asymptomatic or idiopathic intussusception is being seen more commonly. Early diagnosis and timely treatment is helpful to overcome adverse outcomes.

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A Rare Presentation of Delusion of Pregnancy and it's Response to Modified Electroconvulsive Therapy

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Abstract

Delusion of pregnancy is a special form of hypochondriacal / somatic delusion. High potency antipsychotics such as pimozide are indicated in the treatment of delusional disorder. However, the use of Modified Electroconvulsive Therapy (MECT) in delusional disorders has been reported infrequently. We report a case of a 38 years old femalewho presented to the department of Emergency Medicine and then later referred to the Department of Psychiatry. History and mental status examination in the Emergency Department revealed pre-morbid paranoid traits, bad obstetric history and significant interpersonal stressors with her only child and a delusion of pregnancy. A diagnosis of persistent delusional disorder (ICD10) was made. In view of significant hostility and acting out behaviour MECTS were initiated along with antipsychotic Pimozide. Serial evaluation of symptoms was done. The case highlights the successful encapsulation of a delusional belief with MECT and also discusses the psycho-social factors that might have contributed to the evolution of this delusion.

Keywords: Pseudocyesis; Modified Electroconvulsive Therapy (MECT); Delusion of pregnancy; Delusion Rating Scale (DRS); Brief Psychiatry Rating Scale (BPRS); ICD10.

Introduction

Delusion of pregnancy is a Delusional Disorder of Somatic Type. Somatic types of delusions include delusional belief that the subject has some general medical condition. Delusion of pregnancy has been reported in various psychotic conditions. It is mostly reported in cases of Schizophrenia and in mental disorders of organic etiologies; and recently in association with antipsychotic induced Metabolic Syndrome [1-3]. It is seen more commonly in male patients [3] (SIMS, 1988). The efficacy of MECT in treatment of these cases had not been studied. We report the first case of mono-symptomatic delusion of pregnancy that was successfully managed using one course of MECT along with antipsychotic drug.

Case Study

A 38-year old, married female, with pre-morbid paranoid personality traits, bad obstetric history (G5P2L1A3), with tubectomy done 14 years back, presented to the Emergency Department of our hospital saying that she was pregnant and was demanding for conduction of labor.

She had believed for a year now that she was pregnant with a male child and had consulted multiple doctors. Patient considered her body aches and abdominal distension as a proof of full-term pregnancy. She also believed her tubectomy was re-canalized 4 years back when in fact she had been operated for hernia. She refused to believe the negative ultra-sonography (USG) and urine pregnancy test (UPT) reports. She broke

into arguments her relatives in the emergency department when confronted after being told about the reports. Patient was unable to explain the duration of her belief being more than a year and disregarded her regular menstrual cycles as spotting.

Personal history revealed a strained interpersonal relationship with her husband and only child (daughter). She had expressed desires of having a male child for a long time and had reported feeling lonely and insecure.

Physical examination, neurological examination and baseline investigations (inclusive of CBC, blood sugar, LFT, KFT and thyroid functions) were within normal limits. Her BMI was 33 which suggested obesity. A diagnosis of persistent delusional disorder (ICD10) was made. Evaluation of symptoms was done using Brief Psychiatric Rating Scale (BPRS) and Delusion Rating Scale (DRS). Baseline scores on BPRS and DRS were 50 and 21 respectively.

Antipsychotic Pimozide was titrated to 6 mg/day. She continued to be irritable, demanding conduction of labor and showed aggressive behavior towards her husband and the treating team.

The patient's attendants wanted to take a second opinion and they took the patient to another hospital. In view of the significant hostility and acting out behavior, alternate day Modified Electro Convulsive Therapy (MECT) was initiated there. Serial evaluation of symptoms using BPRS and DRS was repeated after the 3rd, 6th, 9th and 11th MECT. Psycho-education and psychosocial intervention was carried out.

Table 1: Delusion Rating Scale (DRS)

	Baseline	6th ECT	11th ECT
Amount of Preoccupation	3	1	1
Duration of Preoccupation	3	2	2
Conviction at the Time of Interview	5	2	0
Amount of Distress	4	2	1
Intensity of Distress	3	2	0
Disruption of Life	3	2	2

Table 2: Areas that Showed Significant Change in Brief Psychiatric Rating Scale (BPRS)

	Base Line	6th ECT	11th ECT
Somatic Concern	4	3	1
Anxiety	5	3	2
Hostility	6	4	2
Suspiciousness	5	2	2
Total Scores	50	30	24

The patient showed significant response after the initial 6 MECTs and thereafter response showed a gradual plateau. At the end of 11th ECT there was a substantial reduction in her BPRS and DRS ratings and she did not spontaneously report the delusion and did not demand for conduction of labor.

Discussion

Pseudocyesis and pregnancy delusions develop more commonly in individuals who conceive procreation as a symbol of feminine fulfilment, who are embroiled into insecure relationships that are mostly characterized by dissatisfaction and an intense wish to be appreciated [4]. Patient under discussion seems to have many vulnerability factors, considering the acute stress of interpersonal conflicts laid over the ground of paranoid personality traits. Psychotherapy targeting family interventions, empathic attitude towards psychological conflicts, and production of evidence discarding the false belief in a graded manner is cornerstone of successful therapy in such cases. Therapy is more person-centred [5].

Historically MECT has not been considered an effective modality for treatment of delusional disorders and when used, a longer course of MECT is considered essential [6]. In 2012, a case of oral cenesthopathy (also a somatic delusion) successfully treated with MECT was reported, and altered regional cerebral blood flow was demonstrated before and after the treatment by single-photon emission computed tomography [7]. This hints at the probable mechanism of effectiveness of MECT in treatment of delusional disorders, and calls for further research in this direction [8]. So far, response of other delusions to MECT has not been reported. The response in this case of delusional pregnancy within 6 MECTs shows that MECT can prove to be therapeutically very effective in treatment of mono-symptomatic delusion of pregnancy and possibly other delusional disorders without a need to re-course to a prolonged treatment [8].

Conclusion

The response in this case of delusional pregnancy within 6 MECTs shows that MECT can prove to be therapeutically very effective in treatment of mono-symptomatic delusion of pregnancy and possibly other delusional disorders without a need to re-course to a prolonged treatment. A multi-team approach involving Emergency physicians, Obstetricians and Psychiatrists might be needed as proven in this case.

Obstetricians and Emergency physicians should keep an open mind and seek expert psychiatric help whenever a definitive diagnosis can not be made.

Source(s) of support: Oxford Textbook of Psychiatry, Google search engine, Institute's Library.

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Case Report

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Reactive Hypoglycaemia of Pre-Diabetes: A Diagnostic Delima

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Abstract

Reactive hypoglycemia, postprandial hypoglycemia, or sugar crash is characterized by recurrent episodes of symptomatic hypoglycemia occurring within 4 hour after a high carbohydrate meal. The frequent sampling of postprandial blood glucose levels will frequently lead to the value below 50 mg/dl, with no hypothalamic-pituitary-adrenal (HPA) stress to the low blood sugar and do not manifest adrenergic symptoms. HbA1C levels between 5.7%-6.4% are suggestive of pre-diabetes. According to the Centers for Disease Control and Prevention (CDC), about 15-30% people with pre-diabetes symptoms progress to type 2 diabetes mellitus within 5 years if they fail to make appropriate lifestyle changes.

Keywords: Reactive Hypoglycaemia; Diabetes mellitus; hypothalamic-pituitary-adrenal (HPA).

Introduction

Reactive hypoglycemia, postprandial hypoglycemia, or sugar crash is characterized by recurrent episodes of symptomatic hypoglycemia occurring within 4 hour after a high carbohydrate meal. The alleged mechanism for the feeling of a crash is correlated with an abnormally rapid rise in blood glucose after eating. This normally leads to insulin secretion (known as an insulin spike), which in turn initiates rapid glucose uptake by tissues either accumulating it as glycogen or utilizing it for energy production. The consequent fall in blood glucose is indicated as the reason for the "sugar crash". In pre-diabetes, the cells are resistant to the effect of insulin and the pancreas can't produce enough to overcome this resistance. As a result, glucose levels build up in the blood.

It is manifested as double vision or blurry vision, unclear thinking, insomnia, palpitations, fatigue, dizziness, light-headedness, sweating, headaches, nervousness, muscle twitches, irritability, tremors, disorientation, flushing, the need to sleep or 'crash', coma can be a result in severe untreated episodes.

According to the U.S. National Institute of Health (NIH), a blood glucose level below 70 mg/dL at the time of symptoms followed by relief after eating confirms a diagnosis for reactive hypoglycemia.

The majority of people with pre-diabetes have no symptoms and it remains undiagnosed until routine blood investigations reveal an elevated blood glucose levels. Some, however, will experience symptoms characteristic of diabetes such as increased thirst, frequent urination, fatigue, or blurred vision.

Pre-diabetes and type 2 diabetes mellitus are diagnosed based on the following test results:

Pre-diabetes-

- Fasting glucose test: 100–125 milligrams per deciliter (mg/dl)
- Oral glucose tolerance test: 140–199 mg/dl
- HbA1c: 5.7–6.4%

Type 2 diabetes mellitus-

- Fasting glucose test: 126 mg/dl or higher

- Oral glucose tolerance test: 200 mg/dl or higher
- HbA1c: ≥ 6.5%

The treatment of reactive hypoglycemia of pre-diabetes focuses on preventing further attacks & relieving acute symptoms.

- Low carbohydrate diet and/or frequent small split meals is the first & most important treatment of this condition.
- Avoiding or limiting sugar intake, exercising regularly, eating a variety of foods including meat, poultry, fish or nonmeat sources of protein, foods such as whole-grains, fruits, nuts, vegetables, and dairy products, choosing high-fiber foods.
- Avoiding eating meals or snacks composed entirely of carbohydrates; simultaneously ingest fats and proteins, which have slower rates of absorption.
- Consistently choosing longer lasting, complex carbohydrates to prevent rapid blood-sugar dips in the event that one does consume a disproportionately large amount of carbohydrates with a meal.

Case Report

38 yr old female patient, with no pre-existing co-morbidities, had visited to OPD with C/o sudden onset palpitations & sinking sensation since 1-2 days, not associated with h/o vomiting/ blurring of vision/ headache/ chest pain/ syncope/ shortness of breath/ sweating. Patient had h/o similar complaints 10 days back. RBS-63 MG/DL. She immediately consumed a packet of 5g of sugar but her symptoms didn't relieve. A couple of minutes later, she fainted. Code RRT was announced and patient was therefore brought to the emergency department. There was no family h/o diabetes mellitus.

On Examination-

Airway Assessment: Patent, Protected, Talking

Breathing Assessment:

Respiraory Rate-12/min

Laboured-No

SpO_2-98% on room air

Circulation

Pulse-90/min

BP-130/70 mmhg

Peripheral Pulse-Palpable

Temperature-98.4 degree F

Disability

GRBS-56 mg/dl

GCS-E4V5M6

Pupils-B/L Reactive

Exposure:

Afebrile

No exantehmatous/petechial rashes noted, no injuries/ scars/ venous engorgements noted.

AMPLE History:

Allergies-None

Medications-None

Past Medical History- nothing significant

Last Meal- 1 hour back

LMP-17/6/18 (10 days back from the date of case reporting)

ECG- Sinus Tachycardia

Care Plan:

- ✓ IV Fluids 100 ml 25% Dextrose NS STAT
- ✓ IV Fluids 0.9% NS @100 ml/Hr
- ✓ 2 hourly RBS monitoring
- ✓ Immediate Physician reference was taken in view of recurrent hypoglycemia
- ✓ Room admission was planned
- ✓ Routine Investigations were sent- CBC, LFT, KFT, Serum Electrolytes, HbA1c, C-peptide levels.
- ✓ USG Whole Abdomen was immediately ordered for diagnosis of insulinoma.
- ✓ Patient was shifted to the room & reports were chased.

Differential Diagnosis of Hypoglycemia:

- ✓ Insulinoma
- ✓ Addison's Disease
- ✓ Hypopituitarism
- ✓ Anxiety disorders

Investigations

- ✓ CBC: Haemoglobin-12.7, Platelet Count-2,67,000, TLC-10,600
- ✓ LFT: Total bilirubin-0.21, Direct-0.03, SGOT/SGPT-WNL
- ✓ KFT: S.Creatinine-0.46, Urea-13, S.Sodium-140
- ✓ Thyroid Profile: FT3-2.66, FT4-0.92, TSH-2.04
- ✓ HbA1c: 5.7
- ✓ C-peptide: 19.4 (1.1-4.4)
- ✓ Pro-insulin: 28.90 pmol/L (<18.8)
- ✓ Fasting Insulin: 36.75 uIU/ml (1.9-23.0)
- ✓ Prolactin: 14.52 ng/mL (3.34-26.74)
- ✓ S. Cotisol: 12.26 ug/dl (6.7-22.6)
- ✓ IGF-1 (Somatomedin-C): 212 (109-284)

Course In The Hospital:

2 hrs after being shifted to the ICU, patient's RBS raised to 213 mg/dL but on subsequent RBS monitoring, readings dropped down to 42 mg/dl which raised to 58 mg/Dl after 100 ml 25% Dextrose infusion. 10% DNS was started at 100 ml/hr. CECT abdomen & MRI Brain with Contrast was done which showed no significant abnormality. Gastroenterology & Endocrinology reference were obtained for the same.

Discharge Advice

Low carbohydrate diet with small frequent meals

Diagnosis

Reactive Hypoglycemia of Pre-diabetes

Conclusion

This above case report shows how confusing the clinical presentation can be. The recurrent hypoglycemia was initially thought to be due to Insulinoma and in view of that USG whole abdomen was also done. But inconclusive labs & radiological investigations changed the entire diagnosis.

Although the patient improved but such patients can return back with similar events in future. Therefore, it is necessary to thoroughly counsel the patient regarding the condition & necessary actions must be taken. The best treatment would be intake of small frequent low carbohydrate meals with regular RBS monitoring. From the emergency medicine perspective, in such cases we should keep our mind open to variety of differential diagnosis.

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Case Report

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Rare Presentation of a Traumatic B/L Carotid and Vertebral Arteries Dissection; Diagnostic Challenges in Emergency Department

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Abstract

A 46 years old previously healthy female was brought to emergency department (ED) with alleged history of road traffic accident (RTA) while driving a car one hours back. She was initially assymptomatic for about one hours post accident but later deteriorated both hemodynamically and mentation. After aggressive resuscitation as per ATLS protocol, she underwent multiple radiological imaging studies which revealed bilateral carotid and vertebral arteries dissection and severe traumatic brain injury with bilateral peripheral infarction. She was managed conservatively with mechanical ventilation in the intensive care unit.

Keywords: Road traffic accident; Trauma; Traumatic brain injury; Bilateral carotid artery dissection; Vertebral artery dissection; Multiple brain infarction.

Introduction

Cervical artery consist of four main arteries that supply blood flow to the brain, two carotid arteries and two vertebral arteries. The carotid arteries can be felt on each side of the lower neck, and the vertebral arteries are located in the back of the neck near the spine and cannot be felt on physical examination.

Carotid artery forms the main arterial blood supply to the brain. The "Circle of Willis" begins to form when the right and left internal carotid artery (ICA) enters the cranial cavity and each one divides into two main branches: the Anterior Cerebral Artery (ACA) and Middle Cerebral Artery (MCA). The anterior cerebral arteries are then united and blood can cross flow by the anterior communicating (ACOM) artery The ACAs supply most midline portions of the frontal lobes and superior medial parietal lobes. The MCAs supply most of the lateral surface of the hemisphere, except the superior portion of the parietal lobe (via ACA) and the inferior portion of the temporal lobe and occipital lobe. The ACAs, ACOM, and MCAs form the anterior half, or better known as the anterior circulation of the circle of Willis. Posteriorly, the Basilar Artery (BA), formed by the left and right vertebral arteries, branches into a left and right Posterior Cerebral Artery (PCA), forming the posterior circulation as soon in fig below.

Carotid artery dissection or Vertebral artery dissection is the tear in the arterial wall, forcing blood between the layers of the wall by splitting the layers. They have similar aetiology with carotid artery dissection being twice as common as vertebral artery dissection. It can be caused by major or minor trauma, hyperextension of the neck. And it can also be spontaneous, in which case, genetic, familial, or heritable disorders are likely etiologies [1,2]. It can be unilateral or bilateral. Dissection

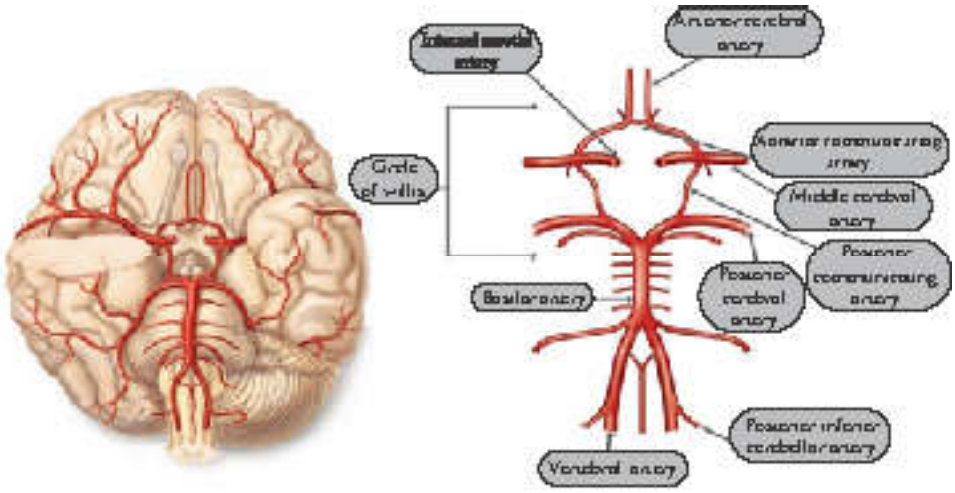

Fig. 1: Carotid artery (Google Courtesy)

of the carotid arteries is rare with recent data suggesting rates in patients with blunt head trauma mainly (high-speed motor vehicle accidents) ranges from less than 1% to 3% [1,3]. Carotid artery and vertebral artery dissection is an important and leading cause of ischemic brain stroke in patients under 45 years of age and the condition is likely to be underdiagnosed.

We are reporting a case of an unusually delayed presentation of bilateral carotid artery and bilateral vertebral arteries dissection with bilateral brain infarct after a Road traffic Accident.

Case Study

A previously healthy 46 years old female was brought to the emergency department with alleged history of road traffic accident (RTA) while driving a car with her child in the front passenger seat. She was initially taken to another hospital for initial management. As per her transfer documents, She did not have vomiting, seizures, loss of consciousness nor ENT bleed. She had complaints of dizziness but was conscious and oriented with time, place and person. After some medications there, she came to our emergency department for further management as her dizziness did not subside.

On arrival in our ED 4 hours post-accident, she was conscious and oriented but had light-headedness and generalized fatiguability. Her pulse was 90/min, BP 140/80 mmHg, SpO_2 94% at RA, RBS 99 mg%.

While being examined, she had a seizure (GTCS) which was managed appropriately with benzodiazepines.

She was immediately assessed thoroughly as per ATLS protocol.

She was intubated and put on mechanical ventilator.

She was started on antiepileptics, IV fluids, IV antibiotics and IV sedation and paralytic agents.

NCCT head was done which showed normal findings. (Fig. 1).

X ray C-spine did not reveal any significant abnormality.

It was therefore thought initially that the seizures were due to concussion injury to the brain and was managed conservatively.

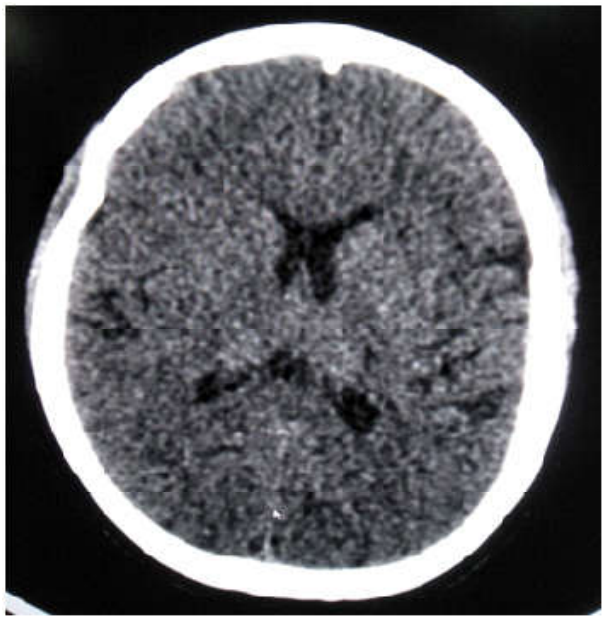

Fig. 1: NCCT Head (normal)

After few hours of elective ventilation, sedation and paralytics were stopped to see response (GCS). After stopping sedation her response was not as good as expected (E1 V1 M2, pupils bilateral sluggishly reacting); In view of prolonged poor GCS, Neurology opinion was sought in this case of trauma. MRI brain showed right MCA, ACA, PCA infarction and left ACA, part of MCA and PCA infarction. (Fig. 2).

After the MRI report of the massive infarct, CT angio brain and neck vessels was done which revealed dissection of bilateral carotid arteries and bilateral vertebral arteries (Fig. 3).

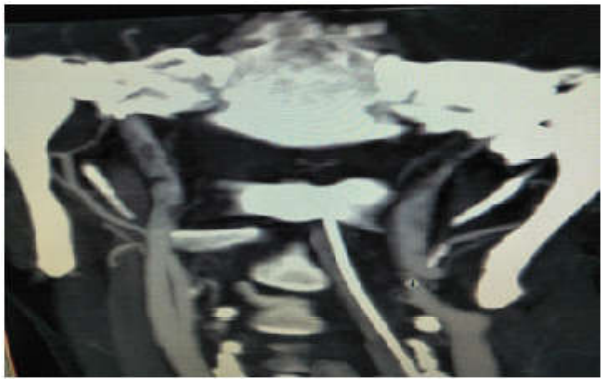

Fig. 3: CT Angio Neck

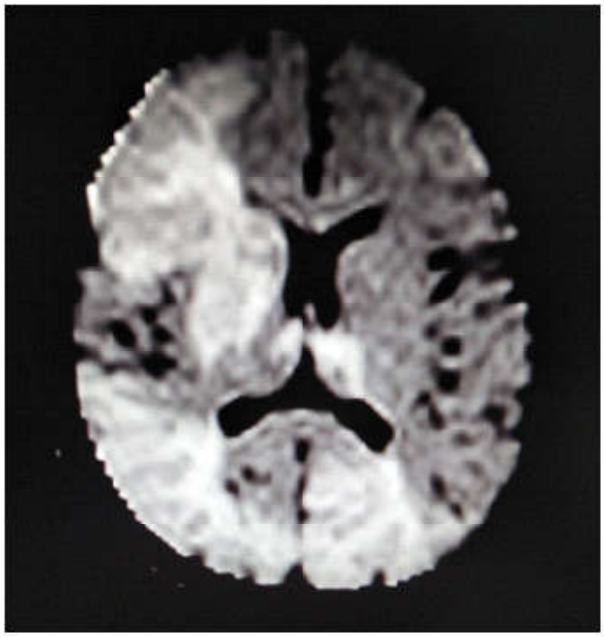

Fig. 2: MRI Brain showing right MCA, ACA, PCA and left ACA, part of ACA and PCA infarction.

All other radiological imaging studies were unremarkable.

She started deteriorating hemodynamically, therefore a multi-speciality approach was undertaken, including Neurosurgery, Neurology, and the Surgical trauma team.

Further considering the massive volume of infarction bilaterally, the chances of haemorrhagic conversion was very high if anticoagulation was started; so it was decided to continue elective ventilation and conservative management.

She remained more or less stable for about 3 days in the ICU but started deteriorating from 4th day onwards. She was then taken to another hospital by her family members for further management.

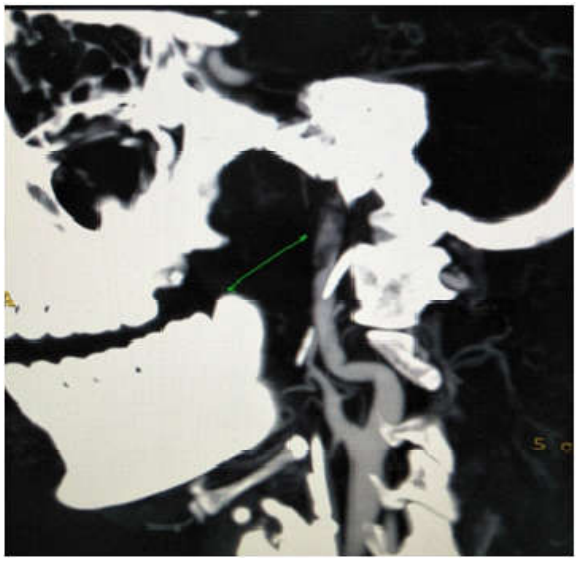

Fig. 4: CT Angio Right side

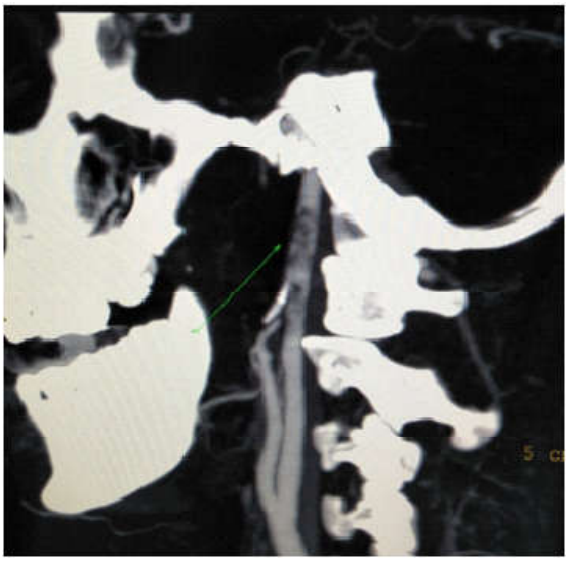

Fig. 5: CT Angio Left side

Discussion

This case report summarizes a very unique case where the patient had severe internal vascular injuries but she had very vague and mild clinical features on examination. It was only after the MRI brain and CT angiography findings that the actual injuries were known.

Normal CT head findings and the delayed presentation of the seizures post-injury also mislead the ER physician; the initial impression was that the patient was in post-ictal state. However the repeated GCS monitoring showed prolonged poor GCS inspite of withholding sedatives and paralytics which made us suspicious of another cause of her altered mental status. This was when Neurological opinion and MRI brain were undertaken.

The use of Glasgow Coma Scale (GCS) is very important for monitoring any patient involved in trauma. The GCS provides an objective, reliable way in quantifying consciousness and is particularly valuable when it is used for continuous evaluation [1]. Despite a normal CT brain, MRA brain showed right MCA, ACA, PCA infarction and left ACA, part of MCA and PCA infarction. Futher imaging was obtained with CT angio brain and neck which is suggestive of bilateral corotid artery dissection and thrombosis.

Although the cause of internal carotid artery dissection remains elusive, it can be due to mechanical forces (eg, trauma, blunt injury, and stretching) and underlying arteriopathies (eg, Ehlers-Danlos syndrome IV and other connective tissue disorders and aberrations). The probable mechanism of injury for most internal carotid injuries is rapid deceleration, hyperextension and rotation of the neck, which stretches the internal carotid artery over the upper cervical vertebrae, leading an tear in the tunica intima or directly within the tunica media with blood flow into the arterial wall, creating an intramural hematoma that leads a thrombus [2,3,4].

Carotid artery dissection is always very serious and life threathening condition. Post-traumatic carotid dissection is rather rare and can be asymptomatic at the initial stage and is usually diagnosed late. It can cause cerebral hypoperfusion and embolism which can lead to stroke [5]. And there are no specific guidelines or screening protocol for detection of carotid injury in the absence of symptoms after Road Traffic Accidents. Hence the diagnosis of carotid artery injury before the development of neurological symptoms is a significant challenge in Emergency Department [6].

The incidence of carotid artery dissection as a result of blunt injuries (mainly high-speed motor vehicle accidents) ranges from less than 1% to 3% and it is a common cause of ischemic stroke in patients younger than 45 years and accounts for as many as 25% of ischemic strokes in young and middle-aged patients. In a review of the literature, only 16 cases of documented blunt traumatic bilateral internal carotid artery dissections were found, one of these was associated with bilateral traumatic disruptions of the vertebral arteries [7,8,9].

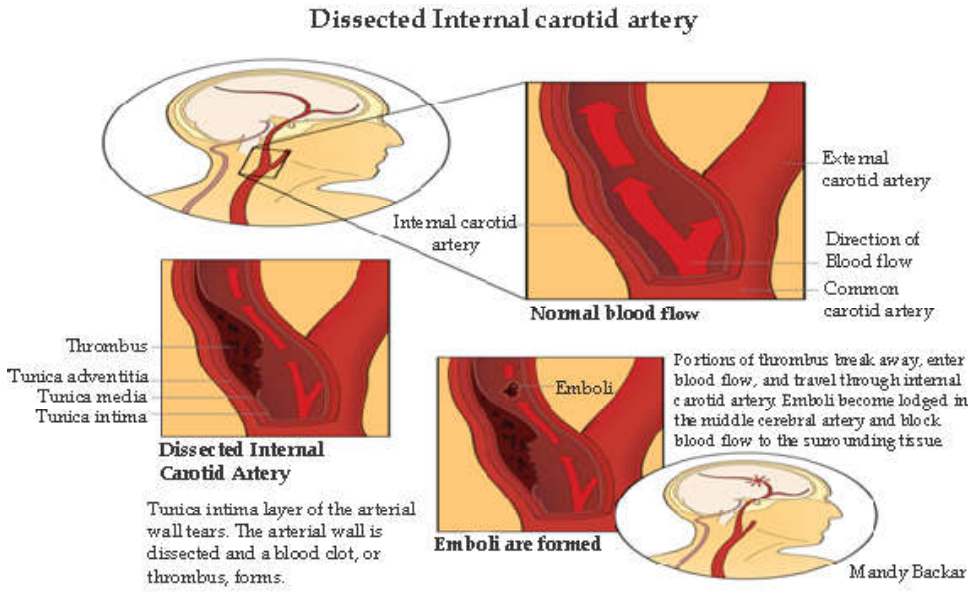

Fig. 6: Internal Carotid Artery Dissection (Google Courtesy)

The classical presentation are Localised headache, mostly around one of the eyes, neck pain, decreased pupil size with drooping of the upper eyelid (Horner syndrome). Patients detected early with mild neurological deficits recovered well with treatment, while those with profound neurological deficits and delayed diagnoses had poor prognosis [10].

The diagnosis is quite challenging and it is made because of awareness of its clinical manisfestations and of advances in non-invasive imaging like color duplex ultrasonography, CT angiography, MRI and MR angiography, and conventional catheter angiography.

Initial computed tomography (CT) of the head is usually warranted, depending on the patient's presentation as we also performed in our case but was found to be normal. The primary indication for carotid angiography is any neurologic deficit that cannot be explained by head CT findings.

Early carotid and intracranial angiography should be performed when there is high suspection of carotid artery lesion involvement and it is more appropriate as initial screening modality in cases of severe trauma. Although, the golden standard for identifying a possible dissection is Digital Subtraction Angiography (DSA) as it offers a very high diangostic performances of 97% for vascular injuries. However, DSA is an invasive method and not readily available in all institutions, hence CT scan combined with CT angiography would be more appropriate in identifying carotid dissection patient with severe trauma. "String signs", also known as "angiographic string sign" or "carotid string sign" indicating constrictions of the lumen, creates a string like appearance found in CT angiography indicating carotid artery dissection, ICA thrombosis and pre-occlusive atherosclerosis at the carotid bifurcation.

MRI with MRA could be an alternative for early detection of traumatic carotid artery detection specially due to blunt trauma and also it provides additional information about concomitant injuries such as infarction, brain injury or skull fractures as in our case also MRI report showed right MCA, ACA, PCA and left ACA, part of MCA and PCA infraction. The diagnostic quality of CT angiography and MRI has been demonstrated in this case report with several excellent images obtained. Hence both modalities can be used in side by side to aid management in carotid artery dissection [8,11,12].

Treatment of carotid artery dissection depends on the patient's symptoms. In general, asymptomatic patients with low grade dissections are typically treated conservatively with medical management and close imaging observation.

Anticoagulation remains the mainstay of medical treatment as studies have shown that ischemic event can be avoided in most cases if early diagnosis and treatment with anticoagulants is implemented but do not initiate anticoagulation in trauma patients without first ruling out intracranial hemorrhage (ICH) and extracranial sources of hemorrhage. Anticoagulation with antiplatelet (intravenous heparin followed by warfarin) has generally been accepted as adequate medical management for preventing thromboembolic complications [12,13].

Conclusion

Bilateral carotid artery dissection is rare but it can cause serious life threatening condition after high-speed motor vehicle accidents. CT angiography and MRI are the gold standard for early diagnosis. The initial clinical and radiological picture may be normal or there may be a delay in symptom presentation on first arrival; so it is crucial for emergency physicians to aggressively search for this injury in the presence of blunt cranial trauma. It is therefore challenging for emergency physicians to diagnose carotid dissection early on time, potentially delaying definitive medical management.

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Erratum

Screening for Haemoglobin E in the Ethnic Groups of East Sikkim a Hospital based Study

Dey Debomita, Khandelwal Bidita

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[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ20.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.

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