Case Report

Indian Journal of Emergency Medicine
Volume 3 Number 2, July - December 2017
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.3217.19

Renal Thrombotic Microangiopathy Due to Malignant Hypertension

Harini Agnes[1], Venugopal A.V.[2]

Author's Affiliation:
[1]Resident, Department of Emergency Medicine, [2]Consultant, Department of Nephrology, Care Hospitals, Visakhapatnam, Andhra Pradesh 530002, India.

Corresponding Author:
Venugopal A.V.,
Consultant, Department of Nephrology, Care Hospitals, Visakhapatnam, Andhra Pradesh 530002, India.
E-mail:
drvenuvenkat@yahoo.com

Received on 01.09.2017,
Accepted on 14.09.2017

Abstract

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

Keywrods: Malignant Hypertension; Mmicroangiopathic Hemolysis; Renal Failure.

Introduction

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

Renal Thrombotic Microangiopathy (TMA) occurring as a result of malignant hypertension is known in the literature but very few case reports from India. The renal TMA due to malignant hypertension may closely resemble Thrombotic Thrombocytopenic Purpura (TTP) but differentiating these two entities is very important because of variable therapeutic implications. Plasmapheresis is beneficial in TTP but of no benefit in TMA associated with malignant hypertension [2]. Renal TMA is characterized by features of intra vascular hemolysis, small vessel thrombosis, thrombocytopenia, indirect hyperbilirubinemia and elevated Lactate Dehydrogenase (LDH) levels. Acute Kidney Injury (AKI) associated with this entity is usually reversible after variable period of renal replacement therapy. So,

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

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

Case Report

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

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

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

Color Doppler renal vessels showed no evidence of renal artery stenosis. 24 hour urinary metanephrin levels were within normal range. His PRA activity was significantly high (>8ng/ml/hr). His renal biopsy showed diffuse arteriolar thrombosis and fibrinoid necrosis of arterioles. He was continued on dialysis for 2 weeks after which he showed good clinical signs of improvement in the form of increased urine output, no signs of fluid overload and improvement in renal function tests. He is being followed up closely in nephrology outpatient department. His last serum creatinine is 2.3mg/dl. All anti-hypertensives he was using till now have been withdrawn and was started on Telmisartan 40mg/day and achieved adequate blood pressure control.

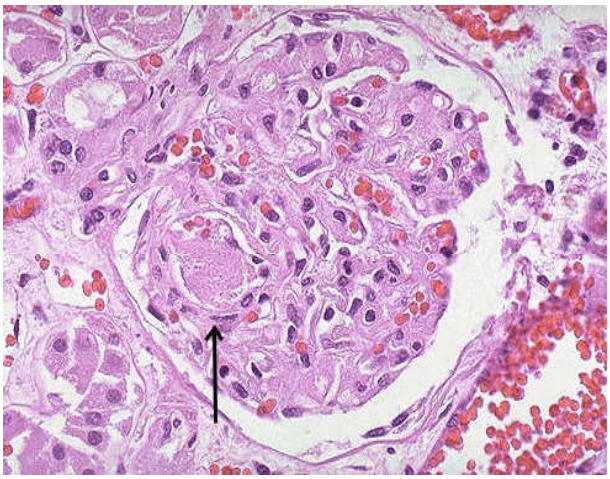

Fig. 1: Histopathology–intra-glomerular capillary thrombi

Discussion

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

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

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

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

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

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

Conclusions

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

References

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