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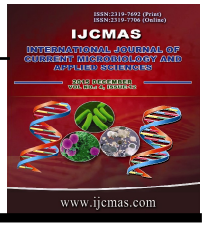

Case Study

Thrombotic Microangiopathy secondary to Malignant Hypertension presenting as acute Kidney injury-A Case Report

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Keywords

Malignant hypertension, Acute Kidney Injury

A B S T R A C T

Thrombotic microangiopathy (TMA) is a rare cause of Acute kidney injury. Malignant hypertension is one of the causes of TMA. We report a case of 26 year old male who presented with complains of swelling over body and decreased urine output. He was diagnosed as a case of thrombotic microangiopathy secondary to malignant hypertension.

Introduction

Thrombotic microangiopathy (TMA) is a rare cause of Acute kidney injury. Malignant hypertension is one of the causes of TMA. Reports in this regard are scant. We report one such case.

Case Report

A 26-year-old male patient was presented to our emergency with complains of swelling over body and decreased urine output along with headache and blurring of vision for past 10 days. Swelling was insidious in onset gradually progressive and first appeared on face. He reported of passing 400ml of black tea colored urine in 24 hrs. Headache was frontal and associated with occasional episode of vomiting. On examination, His face was slightly pale with edema of the eyelids and pitting pedal edema was present.

There were no focal neurological signs. He was found to have a blood pressure of 210/110 mm Hg, pulse rate 90/min regular and all peripheral pulses was palpable and no audible bruits were detected. Respiratory rate was 20/min, chest was clear and he was afebrile. ECG shows left ventricular enlargement. X ray chest PA view shows normal lung field with slightly increased cardio thoracic ratio. Fundus examination revealed bilateral disc edema, dilated tortuous vessels with flame shaped hemorrhages, obscured disc margin multiple cotton-wool spots and hard exudates in bilateral retina. He had a history of

hypertension for 1 year which was diagnosed by some community physician but he was not on any regular treatment for the same.

A clinical diagnosis of malignant hypertension was made and patient was shifted to CCU where he was put on Inj. furosemide and nitroglycerine infusion, later he received oral antihypertensive in form of tab Amlodipine, tab furosemide, and tab clonidine.

Investigations showed hemoglobin of 8 g/dl, total leukocyte count of 9000/mm ^{3} with a differential count of 76 neutrophils, 33lymphocytes, and 1 eosinophils. Platelet count was 60000/mm^3 . Blood urea was 100 mg/dl and serum creatinine was 6.4 mg/dl. Serum sodium was 135 mEq/l and potassium was 5.4 mEq/l. Random blood glucose was 145 mg/dl. Liver function tests showed serum bilirubin to be 1mg/dl, aspartate aminotransferase was 30 IU/l, alanine aminotransferase was 20 IU/l and alkaline phosphatase of 15 IU/l. Total serum protein was 5.5 g/dl, serum albumin was 3.7 g/dl and prothrombin time was 17.7 s with an INR of 1.47.

Human immunodeficiency virus, hepatitis B surface antigen and anti-hepatitis C virus serology, Serum ANA, serum Scl 70 were also negative. Total serum calcium value was 8.9 mg/dl, serum phosphorous was 3.0 mg/dl and serum parathyroid hormone was 15 IU/l which was in normal range (normal range 12-95 IU/l). Vitamin D level (25-hydroxylation [OH] vitamin D) was 25 ng/ml, suggestive of vitamin D insufficiency. (normal value 30-100 ng/ml). His arterial blood gas (ABG) analysis was suggestive of compensated metabolic acidosis (pH 7.2, pCO_2 18 mm Hg, actual bicarbonate 15 mEq/l, and standard bicarbonate 21 mEq/l.

Urinalysis examination showed 3+ albumin, 2+ hematuria no glucose and 2-3 pus cells/high power fields on microscopy. 24 h total urinary protein (TUP) was 1 g.

On the basis of de-arranged renal function test patient was transferred to nephrology unit. He complained of further decrease in urine output with nausea and vomiting. Renal function test was repeated on next day which showed *Blood urea 180 mg/dl* and *serum creatinine 10.2 mg/dl.* TUP showed 3gm protein in 24 hrs on next day

General blood picture was characterized by presence of marked anisopoikilocytosis with presence of schistocytes, tear drop cells, bite cells with normocytic and normocromic appearance. Platelet count was reduced by smear.

USG Abdomen with renal Doppler revealed bilateral bulky kidneys with kidney size being Right kidney 103x40 mm and Left kidney 100x38 mm with normal echotexture and corticomedullary differentiation. No evidence of renal artery stenosis was found on renal Doppler.

With all this investigative background renal biopsy was performed which showed-

1. Features of severe accelerated hypertension with evidence of thrombotic microangiopathy.

2. Acute ischemic alterations with features of thrombotic microangiopathy in glomeruli and tuft sclerosis in 3 (20%) of sampled glomeruli.

Moderately advanced tubulointerstitial chronicity in sampled cortical parenchyma with cortical interstitial inflammation and features of patchy acute injury in viable tubules [Figure 1,2].

A diagnosis of Thrombotic Microangiopathy with Acute Kidney Injury secondary to Malignant Hypertension was made and patient was put on inj. furosemide 20 mg QID, inj. sodabicarbonate and inj. Calcium gluconate10%, but his urine output did not improve and he developed anuria along with signs and symptoms of uremia. Owing to detoriating condition of patient and decreasing urine output patient was subjected to haemodialysis, 5 cycles of which were performed uneventfully. Following haemodialysis, urine output improved and he was switched to Tab. Amlodipine 10mg OD, Tab. Furosemide 40mg BD, Tab. Clonidine 0.2mg TDS

His general condition improved and he was discharged on Tab. Amlodipine 10 mg OD, Tab. clonidine 0.2 mg TDS, Tab. furosemide 40 mg BD, Tab. Iron and folic acid 100 mg BD, Tab. calcium carbonate 500 mg BD. He was asked to come for follow-up after two weeks. On follow-up after a period of 8 weeks, the patient reported subjective improvement in swelling over body and urine output .

Results and Discussion

The term Thrombotic microangiopathy (TMA), first introduced by *Symmers* in 1952 [1] defines a lesion of vessel wall thickening (mainly-arterioles or capillaries) with swelling or detachment of the endothelial cell from the basement membrane, accumulation of fluffy material in the subendothelial space, intraluminal platelet thrombosis, and partial or complete obstruction of the vessel lumina [2]. Laboratory features of thrombocytopenia and hemolytic anemia are almost invariably present in patients with TMA lesions, and reflect consumption and disruption of platelets and erythrocytes in the microvasculature. Additional clinical signs depend on the diverse distribution of the microvascular lesions and the consequent organ dysfunction depending on whether renal or brain vessels are involved. Diseases that cause TMA includes Thrombotic Thrombocytopenic Purpura (TTP)[3], Hemolytic Uremic Syndrome (HUS)[4], Malignant *Hypertension [5], Atypical TTP/HUS, Disseminated Intravascular Coagulation (DIC), Syndrome of Hemolysis, Elevated Liver Enzymes, and Low Platelets, Scleroderma Renal Crisis.*

Patients may present with acute renal failure and/or cerebral dysfunction, although cardiac, gastrointestinal and other organ involvement can also occur. Malignant hypertension may be either a cause of TMA or a manifestation of renal involvement from an underlying disorder such as aHUS. It is postulated that the endothelial dysfunction caused by activation of the renin–angiotensin–aldosterone system (RAAS) has a central role in the pathogenesis of malignant hypertension [6, 7].

In the setting of severe hypertension associated with activation of vasoconstricting RAAS, the endothelial cells secrete vasodilating substances such as nitric oxide and adrenomedullin or prostacyclin to compensate for the change in vascular resistance. If hypertension is severe enough and/or prolonged, this compensatory mechanism fails and a vasoconstricting substance such as angiotensin II promotes expression of proinflammatory cytokines. These series of events subsequently damage the endothelium further and activate a coagulation cascade, leading to fibrinoid necrosis, edema of the arterioles and local platelet aggregation [8,9]. We report an educational case of malignant hypertension leading to thrombotic microangiopathy (TMA).

Table.1

Diseases that cause TMA
1. Thrombotic Thrombocytopenic Purpura
2. Hemolytic Uremic Syndrome
3. Atypical TTP/HUS
4. Malignant Hypertension
5. Disseminated Intravascular Coagulation
7. Scleroderma Renal Crisis
6. Syndrome of Hemolysis, Elevated Liver Enzymes, and Low Platelets

Figure.1 Histopathology Image of Renal Biopsy Showing Changes of Thrombotic Microangiopathy

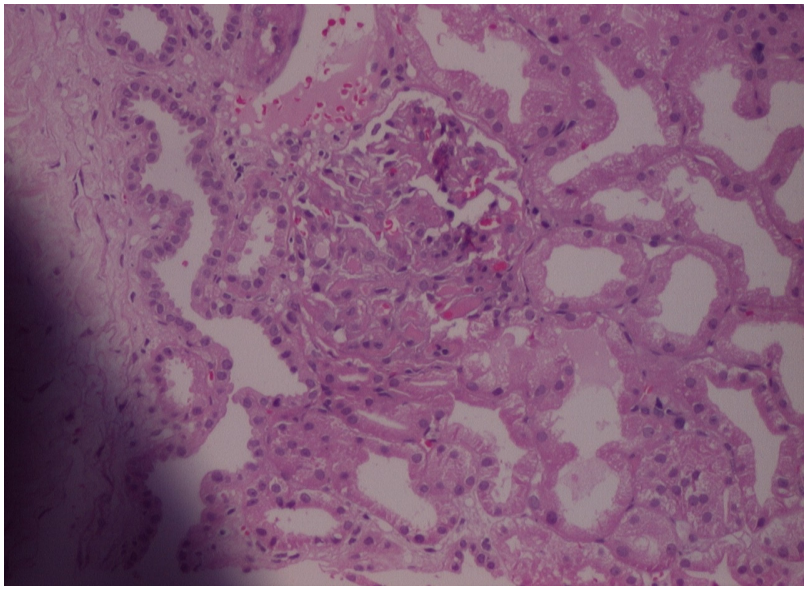
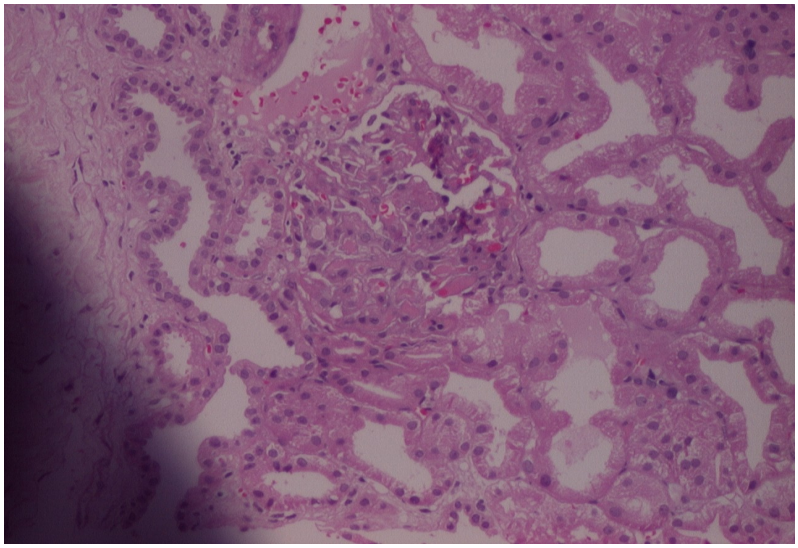


Figure.2 Histopathology Image of Renal Biopsy Showing Changes of Thrombotic Microangiopathy



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