Multiple Myeloma Masquerading Falciparum Malaria
- Presenting as Acute Renal Failure

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Introduction:

Herewith we report a case of acute renal failure precipitated by falciparum malaria in an elderly male which was not recovering for 6 weeks and hence, needed renal biopsy. Renal biopsy showed features suggestive of multiple myeloma. Bone marrow examination and serum protein electrophoresis showed 40% plasma cells and M. band respectively; thus confirming the diagnosis of multiple myeloma.

CASE REPORT

Sixty-five year old male, labourer by occupation was transferred to our hospital for evaluation of acute renal failure (ARF). He was in good health till 10th May, 1997 when he had high grade fever with chills which was followed by altered sensorium and unconsciousness. His laboratory evaluation showed severe anemia and azotemia (BUN 135 mg/dl, serum creatinine 20 mg/dl) peripheral smear showed ring and trophozoite form of plasmodium falciparum. He was diagnosed to have cerebral malaria with ARF. He was treated with blood transfusion and intravenous quinine. Patient received hemodialysis on alternate day through right internal jugular double lumen catheter. Since patient did not show signs of recovery, he was referred to our hospital on 23.06.97 for further evaluation.

He gave history of nausea, anorexia, vomiting, generalized weakness and breathlessness on exertion. He was non-hypertensive and nonasthmatic. He had not received any nephrotoxic drugs prior to fever. He did not complain of joint and bone pains. He was non oliguric and his output varied between 1000-1500cc for 24 hours. Physical examination revealed well built elderly male with anemia. He was normotensive and euvoletic with no bony tenderness. Cardiovascular, respiratory, alimentary and neurological systems were unremarkable.

Laboratory evaluation revealed haemoglobin of 7.4gm/dl with MCV 81fl, Total WBC count 6500 per cubic mm with P-66%, L-23%, E-7%, M-4% and ESR 100mm/hr. Urine examination revealed pH-8, protein +3 and no sugar was detected. Leucocytes and erythrocytes were occasionally present in the urine. Twenty-four hours excretion of protein was 3.9gm/day. Renal profile showed blood urea 189mg/dl, Serum creatinine 13.7mg/dl, Serum Na. 133 mmol/lit, K-4.4mmol/lit. S. Ca-8.5 mg/dl, S. Phosphorus 7.8mg/dl with normal Serum alkaline and acid phosphatase. Liver function test revealed total protein of 7.00gm/dl with albumin 3.1gm/dl and globulin 3.9 gms/dl, ALT/AST were normal. Bleeding and coagulation profile revealed platelet count of 1,30,000/cm2 with normal bleeding, clotting and prothrombin time. His fasting blood glucose was 109mg/dl. Serology was negative for HBsAg, HIV and HCV antibodies. X-ray chest showed cardiomegaly. X-ray skull, spine, and pelvis were unremarkable. USG-abdomen showed Right Kidney 9.6 x 5.9 cms, Left Kidney 9.9 x 5.6 cms with increased echogenicity and no dilatation of pelvi-calycetal system. Liver, spleen, gall bladder were normal and no retroperitoneal lymph nodes were noted.

Patient was continued on thrice weekly hemodialysis. He received 2 units of blood transfusions. After stabilization, renal biopsy was done. It showed nine glomeruli with no evidence of crescent formation or increased cellularity. Interstitium showed diffuse cell infiltrate consisting of lymphocytes, polymorphs and eosinophils with interstitial oedema. Tubules showed several fractured cast, dilatation and patchy necrosis (Fig.1). As tubules were showing fractured cast, patient was evaluated for multiple myeloma. Protein electrophoresis showed M band; (Fig.2). Bone marrow revealed monocellular, late normoblastic hyperplasia with 40% plasma cells with blasts in its various maturation stages and many mitosis of plasma cells; (Fig.3). Urinary Bence Jones Protein

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were positive, confirming the diagnosis of multiple myeloma. Patient received intensive dialytic therapy and chemotherapy with melphalan and prednisolone. His creatinine stabilized and he became dialysis free.

**DISCUSSION**

This case illustrates the importance of timely renal biopsy in a case of ARF. Occasionally despite a logical and sequential approach, the cause of ARF remains unclear. Under such circumstances, renal biopsy should be considered. The renal biopsy is often considered in setting of ARF when (1) no obvious cause of ARF is detected, (2) there is extra-renal clinical evidence or history of systemic disease, (3) there is history of heavy proteinuria & persistent haematuria, (4) there is marked hypertension in absence of volume expansion, (5) there is prolonged (more than 2-3 weeks) oliguria, (6) anuria is present in absence of obstructive uropathy. This patient had renal failure for more than 6 weeks with normal sized kidneys and nephrotic range proteinuria. Though the patient presented with features of ARF following falciparum malaria: when out of critical oliguria, urinary protein excretion was 3.9 gms/24 hrs.

His recovery was not satisfactory, which prompted us to do renal biopsy as to rule out rapidly progressive glomerulonephritis (RPGN). Surprisingly, it showed features suggestive of multiple myeloma. Various renal manifestations of multiple myeloma include light chain proteinuria, myeloma cast nephropathy (myeloma kidney), light chain nephropathy, (nodular-gomulerosclerosis), pyelonephritis, nephrotic syndrome, amyloidosis hypercalcaemic nephropathy with tubular dysfunction, uric acid nephropathy and obstructive uropathy, acute and chronic renal failure, pseudohyponatremia, low anion gap metabolic acidosis and hyperkalemia. Our patient had ARF precipitated by falciparum malaria. Usually in falciparum malaria ARF resolves within 2-4 weeks. As the recovery was not as predicted; renal biopsy was done, which revealed features suggestive of multiple myeloma. Renal failure is a common manifestation of myeloma and it is present more than half of patient at some point in the course of their illness. Hypercalcemia, dehydration and infection are three major contributing factors where as radiocontrast uric acid nephropathy and hyperviscosity are less frequent causes of precipitation of ARF. In our patient precipitating factor for renal failure was falciparum malaria infection.

Myeloma cast nephropathy is characterized by presence of tubular cast formation, giant cell reaction and tubular atrophy. Myeloma cast nephropathy is a pathologic entity that can present as asymptomatic light chain proteinuria, acute renal failure or progressive renal failure. Histopathologically casts are found mainly in the distal convoluted tubules and collecting ducts. Casts have a typical appearance and are bright eosinophilic, homogenous and frequently bear multiple fracture lines. Our patient showed this characteristic feature. Hence, it is emphasis that in cases of ARF where the cause is either not readily apparent or recovery is delayed, renal biopsy is a reasonable consideration.

**REFERENCES**