

Hematuria – Look beyond the Urinary Tract – A Rare Case Report

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ABSTRACT

The major causes of hematuria in a middle-aged female include infections, renal calculi, or structural abnormalities of the urinary tract. Most patients are investigated and treated on an outpatient basis. While a possibility of malignancy of the bladder or kidneys may also be considered, hematological malignancy is an uncommon cause of hematuria. Detailed evaluation is important to rule out malignancy in new-onset hematuria. We report a case of a 49-year-old female who presented with gross hematuria, later diagnosed to have multiple myeloma (MM), despite the absence of its classical symptoms. This unusual presentation reaffirms its status as “The Great Masquerader.” The case is worth highlighting because gross hematuria as the first presentation of MM is uncommon. The author wishes to stress that the clinician should also suspect blood dyscrasias with associated coagulation abnormalities in the workup of gross hematuria.

KEYWORDS: Hematuria, multiple myeloma, plasma cell dyscrasia, coagulopathy

INTRODUCTION

Multiple myeloma (MM) is caused by clonal expansion of plasma cells in bone marrow which secrete a monoclonal immunoglobulin's light chains that get deposited as amyloid fibrils in the tissue. The disease causes clinical symptoms which present in varied forms such as tumor mass effects (cord compression), cytokine production (anemia), bone destruction (pain), protein deposition in visceral organs (kidney, heart), and immunosuppression (infection). While coagulation system abnormalities are an important complication of plasma cell disorders, the incidence of bleeding tendencies is low. Here, we report a case of MM presenting with gross hematuria, which is a rare first symptom of this “great masquerader.”

CASE REPORT

A 48-year-old postmenopausal lady, with no prior comorbidities, presented to the hospital with complaints of easy fatigability for 15 days. She also complained of gross hematuria, burning micturition, decreased urine output, and multiple episodes of vomiting for 4 days. Her temperature was 100°F for 2 days, which was not associated with chills and rigors. There was no history of breathlessness or swelling of limbs. There was no history

of loss of appetite or weight. There was no history of renal colic, analgesic abuse, or rash. There was no other bleeding manifestation. Severe pallor was present on examination. Her blood pressure was 160/80 mm Hg and pulse rate 94/min. Fundus examination was normal. Rest of the systemic examination was normal.

Routine blood investigations revealed a hemoglobin of 4.4 mg/dL, total leukocyte count-13500/mm³, platelet count-1.37/mm³. Peripheral smear showed normocytic normochromic anemia with rouleaux formation. Erythrocyte sedimentation rate was 82 mm/h. Liver and kidney function showed total serum proteins 10 mg%, albumin 2.5 mg%, globulin 7.5 mg%, alkaline phosphatase, alanine transaminase and aspartate aminotransferase were normal, blood urea 324 mg/dL, serum creatinine 23 mg/dL, Na + 135 mEq/L, and K + 5.1 mEq/L. Her corrected serum calcium level was 8.6 mg/dL and phosphorus level was 5.0 mg/dL. Prothrombin time (PT) was prolonged by 22s (14s) with INR of 1.6, bleeding time was normal. Platelet function tests were done, which were normal. Urine routine

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microscopy showed albumin 2+, 3–4 pus cells/hpf, numerous fresh RBCs/hpf, with no casts.

The patient was further worked up for severe anemia with deranged kidney function and reversed albumin:globulin ratio. Urine cultures were negative. ANA, p-ANCA, c-ANCA, and anti-GBM antibody were negative, and levels of C3 complement were within the normal range. Ultrasound abdomen showed right kidney - 11 cm × 4 cm, left Kidney - 11.5 cm × 5 cm; both kidneys showed increased cortical echogenicity with preserved corticomedullary differentiation suggestive of bilateral medical renal disease. Serum electrophoresis revealed markedly raised gamma globulins - 9.25 g% with M-spike of 8.79 g/dl. Renal biopsy was not done in view of coagulopathy. Urine for Bence Jones proteins was negative. Cystoscopy was normal. X-rays of the chest, spine, hip, pelvis, and skull were normal. Bone marrow aspiration showed plasma cells increased to 12% of marrow cells. Beta-2 microglobulin level was markedly increased-31.2 mg/L. Durie-Salmon criteria diagnosed the patient to have MM. According to the International Staging System, the patient was in Stage III of the disease.

Fresh frozen plasma and red cell concentrates were transfused and four cycles of hemodialysis were done for deranged kidney function. Her blood biochemistry showed improvement and coagulopathy resolved. She was started on medical management with lenalidomide in consultation with a medical oncologist.

DISCUSSION

The feature that stands out in our case is hematuria as the initial presenting symptom. Most of the symptoms associated with MM are a result of the end organ damage caused by either MM tumor cell infiltration and/or the associated paraprotein. The patients generally present with bone pain (60%), fatigue (30%), weight loss (25%), paresthesia (5%), or recurrent infections.^[1]

A variety of factors affect the coagulation system and increase the risk of bleeding and thrombotic complications in patients with MM. These include: (a) interference of paraproteins with the normal function of coagulation factors (b) enhanced clearance of coagulation factors by the reticuloendothelial system; (c) anticoagulant activity of paraproteins; (d) abnormal platelet function; (e) excessive fibrinolysis; and (f) hyperviscosity *per se*.^[2,3] In addition, a series of case reports demonstrated that acquired hemophilia A and acquired von Willebrand syndrome can cause severe bleeding in MM patients.^[4] In our case, the coagulopathy was probably multifactorial.

Yin *et al.* described a case of a 64-year-old female with a history of gross hematuria and renal dysfunction. She was diagnosed to have MM. Her Serum C3 levels were low, and renal biopsy revealed histological findings of C3 glomerulonephritis. The authors have not highlighted the coagulation profile of the patient. She was treated with thalidomide and dexamethasone. However, no improvement was seen with respect to deranged kidney function or hematuria following treatment.^[5] In our patient, the C3 levels were normal, and the coagulation profile was deranged. Renal biopsy could not be done due to the same. The hematuria resolved following transfusion of fresh frozen plasma.

Salahuddin *et al.* highlighted that while renal involvement may be the presenting feature in MM, the pattern of renal involvement is proteinuria, microscopic hematuria, renal impairment, or urinary tract infection.^[6] Our patient presented with gross hematuria as the first symptom.

In a study done by Hinterleitner *et al.*, one hundred and sixty-four patients diagnosed with MM were analyzed for coagulation abnormalities and bleeding complications. Fifty-six patients reported spontaneous bleeding events.^[7] These included skin hematomas, epistaxis, gastrointestinal bleeding, and menorrhagia. The most common hemostatic abnormality was a prolonged closure time followed by qualitative defects of von Willebrand factor. Prolonged PT or aPTT, or reduced FVIII activity, was also seen in some patients. Our patient with spontaneous bleeding in the form of gross hematuria had PT prolongation by 22s (14s) with INR of 1.6.

In summary, MM is not a uniform disease but rather a group of clinical syndromes that are characterized by the special properties of their proliferating plasma cell clones. MM coming to light due to a seemingly benign hematuria is rare, and physicians must be vigilant to look for this diagnosis during workup.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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