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Not an ordinary UTI: A case of multiple myeloma stage III, with no manifestation of bone pain, hypercalcemia or osteolytic lesions

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A 63-year-old AAM was admitted for dysuria and brown colored urine of 2 weeks' duration. He had no fever but had chills on and off. He reported a 23-lb weight loss and a decreased appetite. Work up on admission revealed anemia with hemoglobin of 7.5 g/dL and renal insufficiency with creatinine as 1.67. He was treated with intravenous ceftriaxone which relieved his dysuria and discolored urine. However, his renal insufficiency persisted despite hydration and antibiotics. During his hospitalization, he was found to have worsening anemia, renal failure and weight loss. Due to his anemia and renal insufficiency, further work up was initiated. With the constellation of renal insufficiency stage III along with macrocytic anemia, multiple myeloma was suspected by the medicine team. Serum protein electrophoresis showed M protein of 4.4 g/dl with IgG kappa and free lambda on serum immunofixation. IgG was 6911 mg/dL. Kappa light chains were 622.3 mg/L with kappa/lambda ratio of 3.66. Subsequently, bone marrow biopsy showed 90% cellularity with 70-80% plasma cells that were kappa restricted. The following cytogenetics by FISH was reported: CCND1-IGH fusion, extra signal for chromosome 9 and loss of one copy of 13q14. Interestingly, the patient denied bone pain and had no lytic lesions on skeletal survey or MRI of the spine. He also did not have hypercalcemia, instead he actually has hypocalcaemia with latest calcium level as 7.7 mg/dl. The patient was diagnosed with IgG kappa multiple myeloma, International Staging System Stage III, as his B2-microgobulin level is 10.3 mg/L.

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Atypical hemolytic uremic syndrome, diagnosis and treatment dilemma: A case report and literature review

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Background: Atypical hemolytic uremic syndrome (aHUS) is a rare disorder defined by the concurrent characteristic triad of microangiopathic hemolytic anemia, thrombocytopenia and acute kidney injury. Primary causes of aHUS are most likely due to complement gene mutations and in a very small percentage of cases from antibodies to complement proteins. Despite aHUS having a distinct pathophysiology, it is still difficult to differentiate from other thrombotic microangiopathies.

Case Report: A 71 year old male patient presented with left scapular pain, anuric acute renal failure, hypertensive emergency, hemolysis and thrombocytopenia. Initially he was found to have non ST-segment elevation myocardial infarction (NSTEMI), but further intervention was withheld as the evaluation resulted in another life threatening condition: aHUS versus thrombotic thrombocytopenic purpura (TTP). The patient immediately started with plasmapheresis, as eculizumab was not readily available. Hemodialysis was initiated for anuric acute renal failure. ADAMTS 13 activity study was within normal range. Evaluation of therapeutic response was promising as the patient showed significant improvement within a few days.

Conclusions: Understanding the genetics and epidemiology of aHUS is very limited and the overlapping clinical features with other thrombotic microangiopathy (TMA) syndromes often delay diagnosis and initiation of appropriate treatment of this rare disease.

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