A Rare Diagnosis of Sarcoidosis in Workup for Possible Multiple Myeloma

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ABSTRACT

A 78 year-old gentleman with acute on chronic renal failure (serum Cr increased from 1.6 to 4 mg/dL), anemia 11 g/dL, proteinuria (1 g/dL), hypercalcemia (11 mg/dL), and a positive serum protein electrophoresis (SPEP). The patient was worked up with a bone marrow biopsy (BM) to rule out Multiple Myeloma. There was no evidence of Multiple Myeloma, but an incidental finding of non caseating granulomas, without evidence of fungal or acid fast organisms, established diagnosis of sarcoidosis.

INTRODUCTION

Sarcoid disease is a chronic systemic multi-organ disease of unknown etiology that presents with non caseating granulomas. It is three times more common in blacks than whites. Traditional sarcoid disease presentation involves bilateral hilar lymphadenopathy, pulmonary infiltrates, and skin lesions. Lesions in the liver, muscles, heart, salivary gland, nervous system, kidneys are less frequent, but have been reported in the literature. Sarcoid can result in hypercalcemia, nephrocalcinosis, nephritis, and reversible dysfunction due to hypovolemia. Sarcoid disease can cause granulomatous interstitial nephritis which is a rare presentation.

PAST MEDICAL HISTORY

Diabetes mellitus II, hypertension, benign prostatic hyperplasia, chronic obstructive pulmonary disease, skin cancer, and nephrolithiasis. The patient was admitted with complaints of a mild back pain and with acute on chronic renal failure with serum Cr of 4 mg/dL, with baseline serum creatinine of 2 mg/dL.

Three and six months earlier, the patient had two admissions for apparent COPD exacerbation, acute renal injury (AKI), and hypercalcemia. In both occasions, the patient’s renal failure and hypercalcemia were attributed to dehydration. Steroids was given and hypercalcemia and AKI were partially resolved.

RESULTS/LABS:

Anemia (Hgb10 g/dl)

Hypercalcemia

Suppressed PTH

Positive SPEP with both a kappa and lambda monoclonal spikes. The kappa/lambda ratio was within normal limits and there was no evidence of a monoclonal peak or Bence Jones proteins in UPEP. Kidney sonogram showed normal size kidneys with no hydronephrosis. CT of the abdomen showed non obstructing renal pelvic calculi. Antinuclear antibody, rheumatoid factor, hepatitis B and C panel, and serological tests for syphilis were negative.

DISCUSSION:

In retrospect manner, the patient’s history of recurrent AKI and hypercalcemia which resolved partially with steroid were likely resembling flares of unrecognized sarcoidosis. In addition, during the admission which took place six months prior, a CT of the thorax which had been ordered to further elucidate on his COPD, revealed small bilateral mediastinal lymphadenopathy which was not pursued at this time. Probable absence of typical sarcoid renal pathologic findings was attributed to prior use of prednisone for two weeks so pathologic findings were unfortunately altered by the anti-inflammatory effects of prednisone. Otherwise, moderate or severe acute interstitial nephritis would have been found. In addition, the patient may have been also suffering from renal failure secondary to hypercalcemia. The patient’s response to prednisone was significant with resolution of hypercalcemia and return of serum Cr to baseline levels.

CONCLUSION:

The link between sarcoid disease and MM has been reported in the literature in several small case studies. The dysregulation of the immune system in sarcoid disease may be related to the development of MM. It is hinted that sarcoidosis may actually expedite that development of MM. It has not been shown which one precedes the other in time. In closing, clinicians should always be aware of the possible relationships between sarcoid and MM in order to obtain the best outcomes for their patients.