

Re: Sarcoidosis Presenting with Inguinal Lymphadenopathy and Treated with a Combination of Corticosteroids and Azathioprine

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We read with great interest the article entitled "Sarcoidosis Presenting with Inguinal Lymphadenopathy and Treated with a Combination of Corticosteroids and Azathioprine", by Isik¹ in the esteemed Turkish Journal of Rheumatology in which the author, described a 50-year-old female diabetic patient who had complained of inguinal pain and a mass for three months. Isik finally diagnosed the sarcoidosis, and treated the patient successfully. I would like to point out a few things about renal involvement associated with sarcoidosis.

Previously we had a similar case in which a 68-year-old woman was referred to our clinic with complaints of decreased appetite, nausea, polydipsia, nocturia, and intense constipation. A physical examination revealed that the only positive finding was three lymph nodes situated close together in the submental zone, and a subsequent submental lymphadenectomy detected multiple foci of a non-caseating granulomatous reaction along with multinucleated giant cells with some asteroid bodies, which were mostly consistent with sarcoidosis.² Similar to the case of Isik, there was a dramatic response to corticosteroid therapy, and the patient's renal function returned to normal.

Recently, we also had a 57-year-old woman who presented with hematuria. The primary evaluations revealed glomerular hematuria of 290 mg/day, proteinuria, and a serum calcium level of 12 mg/dl

along with renal failure and a serum creatinine level of 1.6 mg/dl. On the physical examination, the only positive finding was mild splenomegaly, and a secondary work-up consisting of collagen vascular tests, especially for systemic lupus erythematosus, and virus markers was negative. Furthermore, the chest radiography results only had a few nonspecific infiltrations, and an abdominal computed tomography scan was normal. Moreover, the serum parathormone and vitamin D levels were also normal, and bone marrow aspiration and a biopsy yielded nothing abnormal. The erythrocyte sedimentation rate was 86 mm/hr and further evaluations showed a serum immunoglobulin A (IgA) level of 492 mg/dl (70-400) and a high serum level of angiotensin-converting enzyme at 82 Iu/l (8-52). This was subsequently rechecked, and it continued to be high. Meanwhile, we also conducted a kidney biopsy which showed the presence of mesangial and endocapillary proliferation, segmental sclerosis of the glomeruli, and mild interstitial fibrosis/tubular atrophy. Furthermore, immunofluorescence microscopy detected prominent mesangial IgA deposits with negative C_{1q} deposition, and light microscopy identified mesangial proliferation with widening, which was mostly consistent with IgA nephropathy. There was also mesangial immune complex deposition in the mesangial region and endocapillary area as well as mild interstitial fibrosis, but there was no evidence of granulomatous inflammation. Additionally, focal interstitial lymphocytic infiltration was also present. The status of the kidney biopsy according to the Oxford classification for IgA nephropathy³⁻⁶ was M₁E₁S₁T₁. After one month of treatment with prednisolone 1 mg/kg/day, the serum creatinine and calcium levels regressed to 1 mg/dl and 8.5 mg/dl, respectively. In addition, the serum angiotensin-converting enzyme level also decreased to a normal value, and the patient continues to make progress in her recovery.

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Our reason for reporting these cases is that we wanted to focus further attention on atypical cases of sarcoidosis, such as the one described by Isik. This article also provided us with a good opportunity to explain the rare association between sarcoidosis and IgA nephropathy.⁷⁻¹⁰ This relationship is intriguing but remains controversial; therefore, further kidney studies involving documented cases of sarcoidosis are needed.

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