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Acute Renal Failure Due to IgA Nephropathy in Sarcoidosis

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Sarcoidosis is a systemic disorder affecting multiple organs. We presented a 56-year old woman with renal impairment who was diagnosed with sarcoidosis accompanied by IgA nephropathy. Treatment with methylprednisolone was started for the patient. After treatment, the patient was discharged with good general condition and resolved proteinuria.

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Case Report

INTRODUCTION

Sarcoidosis is a systemic disorder affecting multiple organs. Renal impairment is usually due to an overproduction of 1,25-dihydroxyvitamin D, leading to hypercalciuria with or without hypercalcemia.[1] Common form of renal involvement is noncaseating epithelioid granulomas and its uncommon forms are various types of glomerulonephritis including membranous glomerulonephritis, focal and segmental glomerulosclerosis, diffuse mesangial proliferative glomerulonephritis, mesangiocapillary glomerulonephritis, IgA nephropathy, and crescentic glomerulonephritis.[2]

CASE REPORT

A 56-year old woman admitted to Ayatollah Rohani Hospital of Babol with 5 months history of hematuria and 6 kg weight loss without any other symptom. In physical examination, normal body temperature and pulse rate were obtained. Blood pressure was 130/80 mmHg. Pulmonary, cardiac, abdominal, neurological, and skin examinations and electrocardiogram were normal. She was suffering from hypertension and hyperlipidemia; receiving losartan 50 mg and, amlodipine 5 mg every 12 hours, and atorvastatin 20 mg daily.

Urinalysis showed blood (4+) and many RBCs/HPF with normal morphology and 24-hour urine protein was 77.5 mg/d. The creatinine level was 0.9 mg/dL, which increased gradually up to 3 mg/dL within last 5 months. Erythrocyte sedimentation rate (ESR) was 115 mm/h.

Laboratory investigations revealed, hemoglobin level of 10.3 g/dL, white blood cell count of 3.4×10^9 /L, platelet count of 183×10^9 /L, total protein of 8 g/dL, albumin of 4 g/dL, and calcium of 10 mg/dL (8.6-10.3). Secondary work up revealed negative results for antinuclear antibodies (ANA), anti-double stranded DNA (Anti-dsDNA), anti-neutrophil cytoplasm antibodies (ANCA), HBsAg, anti-HCV antibody, anti-HIV and Bence Jones protein tests. Serum complement C3 and C4 levels were within normal ranges. In Renal ultrasonography, right kidney size was 93×36 mm and left kidney size was 102×38 mm without any other abnormality.

She had serum IgA level of 699 mg/dL (70-400) and serum IgG level of 2287 mg/dL (700-1600). Protein electrophoresis showed polyclonal gammopathy. The Venereal Disease Research Laboratory test (VDRL), wright, coombs wright and 2-mercaptoethanol (2ME) brucella agglutination tests were negative.

Renal biopsy was performed due to unknown azotemia and hematuria and the result was consistent with noncaseating granulomatous interstitial nephritis and immunofluorescence

showed IgA deposition (Figure 1).

The patient was worked up for Sarcoidosis and Tuberculosis (TB) clinically and laboratory tests were performed. Purified protein derivative (PPD) test, sputum smear and culture, and urine culture for evaluation of TB were negative. Chest X ray showed diffuse reticulonodular pattern and thorax CT scan revealed paratracheal and subcarina lymphadenopathy, diffuse round nodules and pleural thickening (Figure 2).

Pulmonary consultation was requested. Bronchoalveolar lavage (BAL) was done and it was negative for acid-resistant bacillus (ARBs), fungal or bacterial growth, and cytology. BAL cultures remained negative for tuberculosis. Sampling of cells had lymphocyte predominance. CD4/CD8 ratio was high as 3.72 (0.8-2) in the BAL. ACE level was 154 U/L (18-114). Transbronchial lung biopsy (TBLB) was done and noncaseating granulomatous reaction reported (Figure 3).

Ophthalmic evaluation did not show any abnormality.

Finally, the patient was diagnosed with sarcoidosis accompanied by lung and renal involvement in addition to IgA nephropathy. Treatment with 40 mg/d prednisolone was started. After treatment, the patient was discharged with good general condition and resolved proteinuria.

DISCUSSION

We presented a case of sarcoidosis associated with granulomatousis interstitial nephritis and IgA nephropathy. There is limited data about this association. This relationship is controversial. Both systemic disorders have unknown causes. There are reports of familial clustering of both IgA nephropathy and sarcoidosis and more than one gene are responsible for it.[3,4]

Kidney involvement in sarcoidosis is rare and commonly manifests as nephrocalcinosis secondary to hypercalcemia and non-caseating granulomatous interstitial nephritis.[5] Our patient had no history of nephrocalcinosis or urinary lithiasis.

According to several causes of granulomatosis,

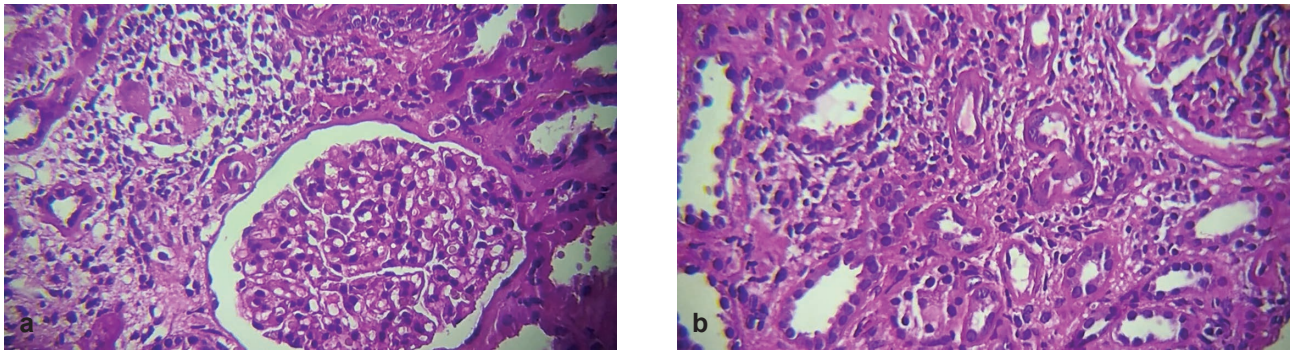


Figure 1. Pathology of renal biopsy revealed non-caseating granuloma.

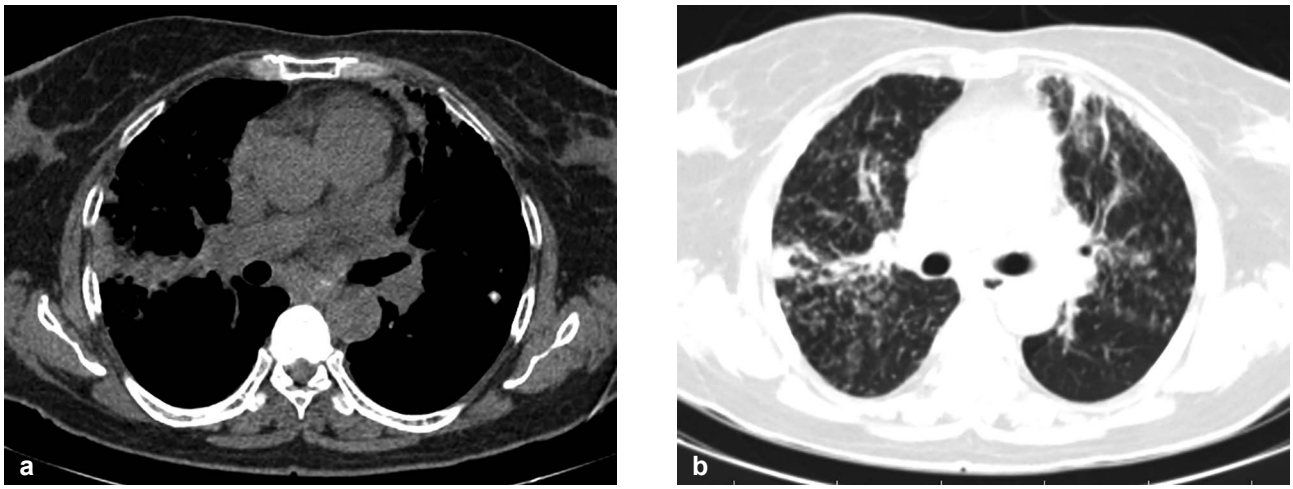
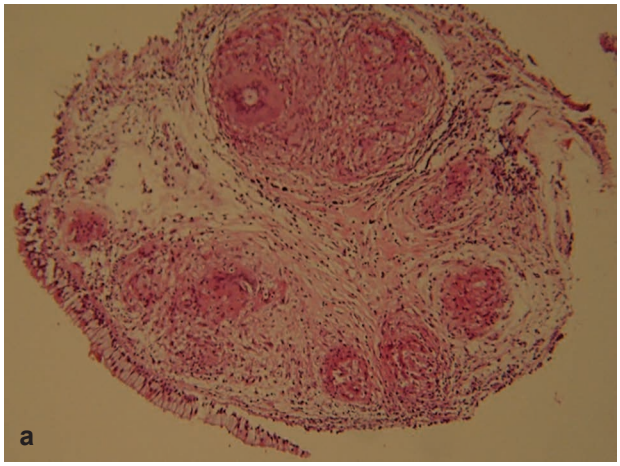


Figure 2. It showed thorax CT scan.



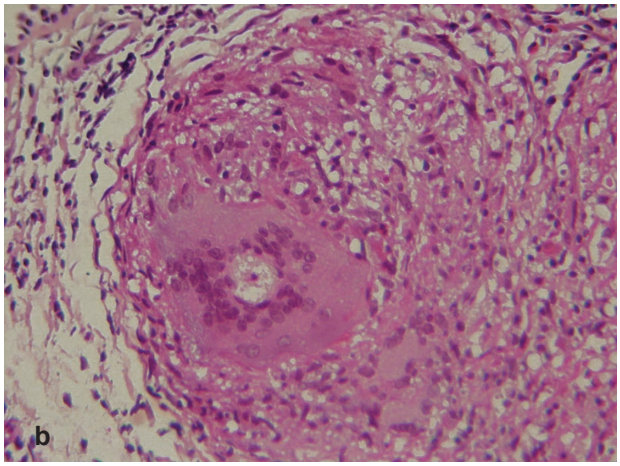


Figure 3. TBLB revealed noncaseating granulomatous reaction.

other diagnoses should be considered. In endemic countries, tuberculosis must be excluded and mycobacterium tuberculosis was negative in our patient. On the other hand, because of anergy in sarcoidosis to tuberculin we ruled out TB by BAL and smear.

Mahfoudhi *et al.* reported a case of sarcoidosis with ocular, ganglionnar and salivary involvement associated to IgA nephropathy.[6] Our patient had no ocular involvement in ophthalmologic evaluation. Vanhille *et al.* reported a case of sarcoidosis complicated with acute renal failure due to rapidly progressive IgA glomerulonephritis.[7] Murray *et al.* described a sarcoidosis and IgA nephropathy diagnosed simultaneously in a 23-year old man.[8]

Our case had renal and pulmonary complications of sarcoidosis and according to literature, there is a physiopathological link between sarcoidosis and IgA nephropathy. Treatment of both sarcoidosis and IgA nephropathy is corticosteroids.

Nishiki *et al.* treated their case by oral administration of prednisone. The follow-up was marked by the subsidence of cough and edema, regression of lymphadenopathy, pulmonary infiltrates and proteinuria, the normalization of serum angiotensin-converting enzyme and IgA levels.[9]

To best of our knowledge IgA nephropathy can physiologically related to sarcoidosis. The rarity of IgA nephropathy caused by sarcoidosis, necessitates the early diagnosis and treatment to avoid renal insufficiency.

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