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Renal Sarcoidosis Presenting as Acute Kidney Injury with Granulomatous Interstitial Nephritis

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Abstract **Case Report**

Sarcoidosis is a nonnecrotizing granulomatous disease of unknown origin that can affect lungs, eyes, kidneys and skin. Although kidney involvement is usually clinically silent, granulomatous interstitial nephritis - the hallmark of renal sarcoidosis – can lead to functional impairment and organ failure. We report a case of renal sarcoidosis revealed by acute kidney injury with biopsy finding of a granulomatous interstitial nephritis. Outcome was favorable after the institution of high dose corticosteroids.

Keywords: Sarcoidosis, interstitial nephritis, noncaseating granuloma.

INTRODUCTION

Sarcoidosis is a systemic disease of middle-aged patients, with a prevalence of about 4.7-64 in 100000, and an incidence of 1.0-35.5 in 100000 per year [1].This inflammatory disorder is characterized by noncaseating epithelioid cell granulomas commonly involving the lung and reticuloendothelial system [2].

Kidney disease develops in up to a third of patients, and may present as acute kidney injury related to hypercalcemia, or glomerular filtration rate (GFR) decline and low level proteinuria due to granulomatous parenchymal injury. Reported patterns of kidney injury are interstitial nephritis or glomerular disease [3]. We report a case of renal sarcoidosis revealed by acute kidney injury.

CASE REPORT

A 58-year-old morocan female presented with acute kidney injury. The patient's medical history included productive cough, dyspnea and headache a month before. However, She was a teacher and reported no smoking, alcohol dependance or illicit drug use.

Clinical examination found a bood pressure of 160/8. Urine studies showed dipstick proteinuria (1+), no hematuria. Neurologic examination was normal.She had a erythema nodosom .Finding of the examination the heart, lungs, and abdomen were unremarkable.

The initial laboratory tests were notable for the following values: serum creatinine (SCr), 415 µmol/L; serum calcium, 131 mmol/L, serum albumin, 33 g/L ; and hemoglobin, 9.8 g/dL. Proteinuria was 0.7 g/day with no hematuria. Liver function tests were normal. Antineutrophil cytoplasmic antibody serologic test results were negative. The work up was negative for hypocomplementemia, monoclonal gammapathy, and viral serology. .Angiotensin-convertiong enzyme level was elevated in serum 199 UI.

Ultrasound showed normal kidney size and echogenicity with no evidence of obstruction or nephrolithiasis.A chest radiopraph showed mild diffuse interstitial disease.

Computed tomography showed 2 nodules and calcifications in the lung. Pulmonary function testing was normal. Angiotensin-convertiong enzyme level was elevated in serum. ANCA serologic test results were negative. Magnetic resonance imaging of the brain was normal. A kidney biopsy was performed to determine the cause of the AKI.

Light microscopic evaluation of the kidney biopsy specimen was performed with hematoxylin and eosin. There were 2 nonglobally sclerosed glomeruli, several of which showed focal ischemic tuft retraction. Approximately 30% of the renal cortex showed interstitial fibrosis, tubular atrophy, and interstitial lymphocytic and granulomatous inflammation (Figure A). There were multiple interstitial noncaseating

granulomas comprised of lymphocytes and monocyts (Figure B). Routine immunofluorescence was negative for all immunoglobulins.

The patient was strarted on treatment with methylprednisolone, 500 mg, intravenously daily for 3 days, followed by oral prednisone, 0.5 mg/kg/day mg. Durant the next month, her SCr level decreased to 123 µmol/L. After six months, she continues on maintenance prednisone therapy at 10 mg daily, with the improvement of kidney function (serum creatinine: 123 µmo/L).

DISCUSSION

Sarcoidosis was recognised more than 120 years ago, and it remains a confusing disease with many grey areas (4). The cause of sarcoidosis is not known and diagnosis can be difficult and delayed from the diverse, nonspecific presentations [1].

The notion of environmental or transmissible agents having a causal role in sarcoidosis is lent support by temporal and space–time clusters. Exposure to musty odours, insecticides, or to metal-processing industries is disease risk factors [5, 2]. Sarcoidosis is usually sporadic, but is familial in 3·6–9·6% of cases [6].

The disease can occur in both men and women, with 70% of patients aged 25–45 years; however, in Europe and Japan, a second peak of incidence occurs in women older than 50 years of age [7,8].

The exact cause of sarcoidosis is still not known. Many studies suggest that the genitic susceptibility and environmental factors contribute to disease development [9, 2, 10].

We present a case of a patient who had an AKI with a Lofgren syndrome suggestive of sarcoidosis. The kidney biopsy findings of granulomatous inflammation .Such clinical presentation and the exclusion of other possible causes of granulomatous disease established a clinicalpathologic diagnosis of renal sarcoidosis.

Granulomatous inflammation can be observed in various conditions such us infections, lymphoma, or granulomatous with polyangiitis [11].

Granulomas characteristically consist of epitheloid cells and multinucleated giant cells with CD4 sponse. Over time, fibroblasts and mast cells lay down collagen and proteoglycans, causing fibrosis. At a single center, the histopathologic findings of interstitial nephritis with non-necrotizing granulomas were attributed to drugs (44%), sarcoidosis (29%), or granulomatosis with polyangiitis (5%) [3, 12].

Corticosteroid therapy is the treatment of choice in sarcoid-associated GIN, often resulting in significant improvement in kidney function [13]. Steroid therapy cessation or withdrawal frequently is associated with relapse of kidney injury that is reversed on reinstitution of the therapy [14]. Long-term lowdose steroid treatment (5-7.5 mg/d of prednisolone) may preserve kidney function, but this regimen needs to be balanced with its adverse side effects. We report in our case the improvement in kidney function with glucocorticoids and immuno-suppressive therapy which corroborate our diagnosis.

Mycophenolate and azathioprine are acceptable alternatives that have been shown to be effective in case series [15]. Biologic agents such as infliximab have been used in relapsing or steroid-dependent cases [16].

CONCLUSION

The presence of AKI with kidney biopsy findings of GIN generates a broad differential diagnosis, among which renal sarcoidosis is a diagnosis of careful exclusion. Our case report recognizes granulomatous inflammation as one of the rare renal manifestations of sarcoidosis and of AKI.

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