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CASE REPORT

Glomerulonephritis associated with tuberculosis: A case report and literature review

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Abstract Rapidly progressive glomerulonephritis caused mycobacterium tuberculosis is rare; however, three case have been reported to date. Crescentic glomerulonephritis is a life-threatening disease and together with the presence of tuberculous infection is associated with a poor outcome if treatment is inadequate and delayed. We describe the case of a 31-year-old female patient with nephrotic syndrome and progressive renal failure secondary to pulmonary tuberculosis. Renal biopsy showed crescent formation in 14 out of 27 glomeruli, and there was diffuse linear staining of immunoglobulin G deposits. Treatment included corticosteroids in combination with antituberculosis drugs for 2 months, and resulted in a significant improvement in renal function, the disappearance of proteinuria and pulmonary symptoms. We also present a review of the pertinent literature and discuss the pathophysiology of tuberculosis-related acute postinfectious glomerulonephritis.

Introduction

Historically, various pathogens have been reported to cause postinfectious glomerulonephritis, including bacteria, viruses, fungi and parasites. [1]. Tuberculosis may be seen in the urinary system in 4–5% of all cases of extrapulmonary tuberculosis, and renal involvement may occur by direct infection of the kidney and/or through secondary amyloidosis [2]. The disease usually develops 5–25 years after the primary tuberculosis infection [3]. Glomerulonephritis secondary to tuberculosis is uncommon in the group of post-infectious nephritides [4]. Although several cases of

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tuberculosis associated with various forms of glomerulonephritis have been reported, only three reports of well-documented crescentic glomerulonephritis associated with tuberculosis have been published [5–7].

Crescentic glomerulonephritis is potentially life-threatening and is frequently associated with a rapid clinical deterioration and poor outcome [8]. If treatment is delayed, patients will develop renal failure within days or weeks of diagnosis [9,10]. The presence of tuberculosis associated with progressive glomerulonephritis complicates the choice and sequence of therapeutic options of immunosuppressive agents and/or anti-tuberculosis drugs. It is very important to make an early diagnosis of nephropathy associated with tuberculosis and to start appropriate treatment. We report on a patient with crescentic glomerulonephritis secondary to pulmonary tuberculosis in which antituberculosis treatment ameliorated renal function. We also discuss the related cases reported in the literature.

Case report

A 31-year-old female patient presenting with uremia, fever, fatigue, and productive cough was admitted into the nephrology ward with suspicion of contrast-induced nephropathy. Two weeks before admission, the patient had undergone appendectomy for perforated appendicitis, and after the operation abdominal computed tomography (CT) was performed in order to search for an intra-abdominal abscess. Initially the patient had normal kidney function and she had no prior history of renal or pulmonary disease. She said that her mother had a history of tuberculosis. There was no palpable lymphadenopathy and pretibial edema. On pulmonary auscultation, there were rhonchi in the middle zone of the left lung. Blood pressure was 130/80 mmHg and examination of the cardiovascular, gastrointestinal and urogenital systems were unremarkable. Urine output was 3600 mL/24 h on the first day of admission. Laboratory examination showed hemoglobin 10.7 g/dL, hematocrit 37.2%, white blood cell count 11,000/mm^3, platelets 298 $\times$ 10^3/μL, erythrocyte sedimentation rate 41 mm/h, C-reactive protein 68.5 mg/dL, blood urea nitrogen (BUN) 81 mg/dL, serum creatinine 5.7 mg/dL (glomerular filtration rate 9.2 mL/min by modification of diet in renal disease formula), potassium 6.0 mEq/L, serum albumin 2.8 g/dL, total protein 6.17 mg/dL, and activated partial thromboplastin time 26.8 seconds. Urine analysis yielded 3+ erythrocytes on strip and proteinuria 75 mg/dL. Twenty-four-hour urine collection showed protein excretion 9.8 g/24 h, urine microalbumin 1980 mg/24 h, urine creatinine 1204 mg/24 h and creatinine clearance of 17.4 mL/min. Renal ultrasonography showed bilateral kidneys with normal size and shape, but increased parenchymal echogenicity. Markers for immune-associated nephrites [antinuclear antibodies (ANA), antineutrophil cytoplasmic antibodies (ANCA), anti-dsDNA, antiglomerular basement membrane (GBM) antibody] were negative. Renal biopsy showed crescent formation in 14 out of 27 glomeruli and there was diffuse linear staining of immunoglobulin G deposits (Fig. 1). There were no granulomas and Langhans' giant cells. The sputum and urine culture produced a negative result for mycobacterium tuberculosis. A chest X-ray image showed bilateral upper lobe opacities compatible with pneumonic consolidations, which was more prominent in the left lung (Fig. 2A). Chest CT confirmed this pneumonic consolidation (Fig. 3). We diagnosed pulmonary tuberculosis based on the findings of chest CT and examination of sputum, which was positive for acido-resistant bacilli (ARB). Considering the presence of nephrotic syndrome and crescent formation, we started intravenous methylprednisolone pulses of 500 mg/day for 3 days, and then methylprednisolone was continued orally at a dose of 64 mg/day. Four-drug antituberculosis treatment was also administered after having seen ARB positivity. After 2 months of immunosuppressive and antituberculosis therapy, renal function repaired, plasma creatinine level decreased from 5.7 mg/dL to 1.4 mg/dL and the pulmonary symptoms also responded to the treatment (Fig. 2B). The clinical course of the patient during her hospitalization is illustrated in Fig. 4.

Discussion

Acute postinfectious glomerulonephritis (APIG) is uncommon in adults and its incidence continues to decline thanks to early and more effective therapies of infections. In a recent series by Nasr et al. [11], the most common

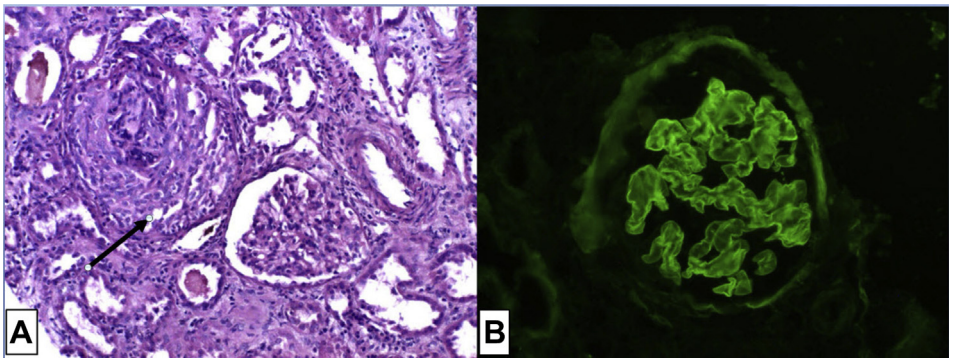

Figure 1. (A) Light microscopy of the renal biopsy shows a cellular crescent (arrow) in one of the glomeruli (hematoxylin and eosin staining, magnification $\times$100). (B) Direct immunofluorescence image shows diffuse linear staining of immunoglobulin G deposits (magnification $\times$200).