CASE REPORT

Glomerulonephritis with Acute Renal Failure Related to Osteomyelitis

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We report a case of acute renal failure in which renal biopsy showed immune complex-mediated proliferative glomerulonephritis. The patient had been afflicted with progressive lower back pain for 2 months. No fever was noted. Magnetic resonance imaging of the lumbar spine demonstrated characteristic findings of vertebral osteomyelitis and bilateral psoas abscesses. Culture of the computed tomography-guided needle aspirated specimens grew *Staphylococcus aureus*. The patient was treated with prolonged antibiotic therapy. The lower back pain improved gradually. Furthermore, significant improvement in renal function occurred approximately 2 weeks after the beginning of antibiotic treatment. The pathologic findings and clinical causal relationship between the bacterial osteomyelitis and the glomerulonephritis suggests a close association of the 2 conditions. Although glomerulonephritis is well documented to be related to a variety of infectious diseases, its association with osteomyelitis has rarely been reported. We review the clinicopathologic characteristics of another 3 cases previously reported in the literature. [*J Chin Med Assoc* 2008;71(6):315–317]

Key Words: glomerulonephritis, kidney failure, osteomyelitis

Introduction

Infection has been well documented to cause glomerulonephritis, with *Streptococcus* being the most common pathogen and predominantly affecting children. However, the pattern of the disease has changed over recent decades. Not only *Streptococcus* but also other bacterial, viral, fungal and parasitic infections can trigger the disease in both children and adults. Furthermore, the responsible bacteria now comprise a majority of *Staphylococcus* and Gram-negative strains, in contrast to *Streptococcus*, which predominated 3 decades ago.[1] Glomerulonephritis secondary to osteomyelitis has rarely been reported. To the best of our knowledge, there have only been 3 documented cases of glomerulonephritis with acute renal failure in association with osteomyelitis previously reported in the literature.[2–4] We describe an episode of biopsy-proven immune complex-mediated diffuse proliferative glomerulonephritis with acute renal failure occurring concurrently with vertebral osteomyelitis caused by *Staphylococcus aureus*. Spontaneous recovery of renal function occurring after eradication of the infection suggested a causal relationship between the osteomyelitis and the glomerulonephritis.

Case Report

A 70-year-old woman was admitted with a 1-week history of decreased urine output and generalized edema. She had also been afflicted with lower back pain for 2 months prior to admission. No fever had been noted during that period. Her past medical history included type 2 diabetes mellitus which had been diagnosed approximately 5 years previously and that was controlled with oral antidiabetic agents.

Physical examination showed a body temperature of 36.8°C, regular pulse rate of 95 beats/min, respiratory rate of 22 breaths/min, and blood pressure of 160/95 mmHg. Fundus examination revealed background diabetic retinopathy. Examinations of the lungs, heart and abdomen were unremarkable. There was a 2 × 2-cm shallow ulcer on the left foot.

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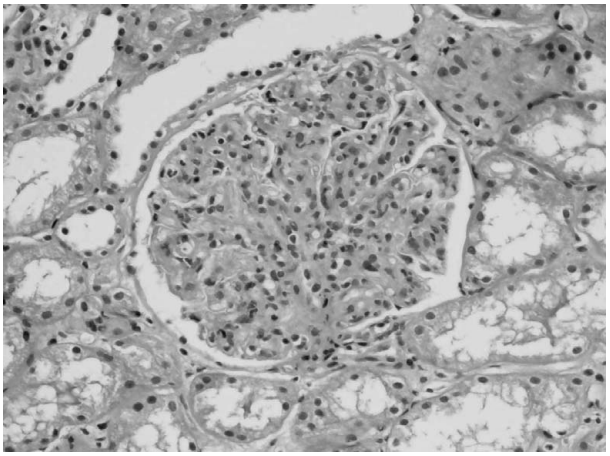

Figure 1. Kidney biopsy shows glomerular endocapillary proliferation and considerable increase in mesangial cell count which contributed to glomerular lobularity (hematoxylin & eosin, 400×).

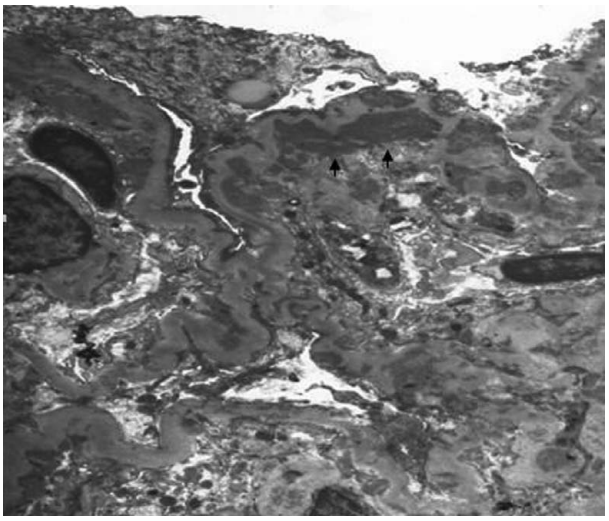

Figure 2. Electron microscopy shows electron-dense deposits in the subendothelial regions (arrows).

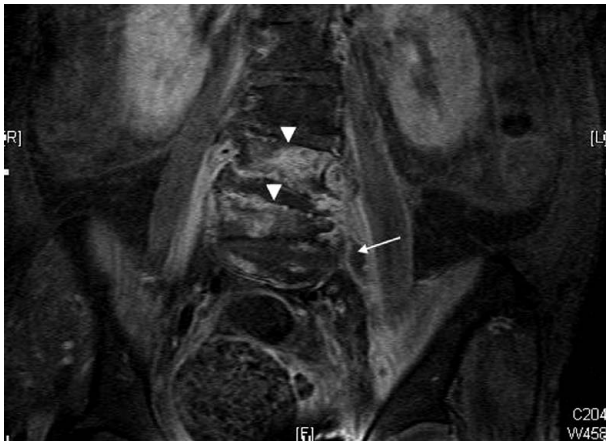

Figure 3. Magnetic resonance imaging of the lumbar spine demonstrates disc space narrowing with increased signal intensity in the vertebral bodies of L4 and L5 on T2-weighted imaging (arrowheads) and left paravertebral abscess (arrow).

Complete blood count showed a white blood cell count of 16,300/μL, with 95% segmented neutrophils, 2% lymphocytes, and 3% bands, hemoglobin of 10.5 g/dL, and platelet count of 467,000/μL. Blood chemistry showed urea nitrogen of 52.5 mg/dL, creatinine of 3.0 mg/dL, sodium of 118 mmol/L, potassium of 5.8 mmol/L, glucose of 230 mg/dL, HbA1c of 10%, albumin of 2.6 g/dL, and globulin of 2.6 g/dL. Urinalysis revealed occult blood (++++) and protein (++), with numerous red blood cells and 11–15 white blood cells/high-powered field in the urinary sediment. Daily urinary protein excretion was 1.2 g. Blood and urine bacterial cultures were negative.

Renal ultrasonography showed normal-sized kidneys. Serological studies showed negative findings for antinuclear antibody and antineutrophil cytoplasmic antibody. Serum complement C3 level was 72 mg/dL (normal range, 90–180 mg/dL), and C4 level was 28.3 mg/dL (normal range, 10–40 mg/dL). Kidney biopsy showed diffuse proliferative glomerulonephritis (Figure 1). Immunofluorescence studies showed strong granular deposits of immunoglobulin (Ig) G and C3 within the glomerular mesangium and along the capillary walls. IgM and C1q were likewise identified in a similar but less intense pattern. On electron microscopy, subendothelial and mesangial electron-dense deposits were noted (Figure 2).

Progressive lower back pain had led to the patient's disability. Magnetic resonance imaging of the lumbar spine demonstrated vertebral osteomyelitis, bilateral paravertebral and psoas abscesses (Figure 3). Computed tomography-guided needle aspiration of the left side psoas abscess was performed, and culture of the specimens grew *Staphylococcus aureus*. The patient was treated with intravenous antibiotic oxacillin. The peak serum creatinine levels had been 4.5 mg/dL. Approximately 2 weeks after the beginning of antibiotic treatment, there was a dramatic improvement in renal function. After a 3-month course of antibiotic therapy, the patient's lower back pain and spinal instability markedly improved. At this time, serum creatinine was 1.8 mg/dL. Urinalysis was negative for protein, but microscopic hematuria was still noted. Follow-up serum complements returned to normal.

Discussion

Today, infectious glomerulonephritis is increasingly recognized worldwide. The most common infections

associated with the development of glomerulonephritis are bacterial endocarditis, infective ventriculoatrial shunt, and visceral abscesses. The onset is frequently insidious and the course usually protracted. The reduction in serum levels of complement components, which may be a valuable clue in classical postinfectious glomerulonephritis, is not always present.[5] By light microscopy, the glomerular lesions of infectious glomerulonephritis are of varying severity. The most common histologic lesion described is diffuse endocapillary proliferative glomerulonephritis, sometimes with membranoproliferative features.[4] Extracapillary proliferation may occur.[6] Glomerular deposits consist mainly of IgG and C3. If IgA is present, it is neither the sole nor predominant deposition.[5,7] By electron microscopy, immune-complex deposits have been demonstrated in subendothelial, intramembranous, and subepithelial locations.[4]

Osteomyelitis has only rarely been associated with glomerular diseases. To the best of our knowledge, there have been 7 cases previously reported in the English language literature. Boonshaft et al[8] reported 4 cases of nephrotic proliferative glomerulonephritis occurring in the course of chronic osteomyelitis. Immunofluorescence and electron microscopy studies were not mentioned in these cases. Osteomyelitis was not eradicated in any of these cases. Two patients died of renal failure, while the other 2 showed no evidence of progression of their renal disease. Because of the long latent period and the observation that antibiotic therapy did not alter the course of the renal disease, it is uncertain that osteomyelitis was etiologically related to the renal disease. Spital et al[2] described 1 patient with diabetes mellitus who developed acute renal failure associated with mesangiocapillary glomerulonephritis type I that was apparently related to osteomyelitis. Marked improvement in renal function occurred after successful treatment of the osteomyelitis. Tevlin et al[3] described a case of IgA nephropathy with acute renal failure in which resolution of renal disease was associated with eradication of the underlying osteomyelitis. Griffin et al[4] described a patient who developed acute renal failure associated with immune complex-mediated diffuse proliferative glomerulonephritis after coronary bypass surgery that had been complicated by sternal osteomyelitis. Renal function significantly improved after successful treatment of the osteomyelitis.

In conclusion, glomerulonephritis with acute renal failure can be caused by osteomyelitis. Renal recovery is dependent on successful treatment of the underlying infection. Furthermore, this case underlines the value of kidney biopsy in the work-up of acute renal failure, even in patients with known infectious diseases.

References

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