Presentation of a case with Salmonella glomerulonephritis

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Salmonella infection is frequently encountered in childhood, but it is rarely associated with glomerulonephritis. We present in this report a case with Salmonella glomerulonephritis, which is infrequent in children. His general condition was found moderate, and temperature and blood pressure were 38 °C and 150/90 mmHg, respectively. The whole blood counts were as follows: white blood cell count 3,800/mm^3, hemoglobin 6.3 g/dl and platelet count 240,000/mm^3. Serum urea was 140 mg/dl, albumin 2.5 g/dl and complement (C_3) 23.6 mg/dl. Both Salmonella type O and Salmonella typhi H were detected positive (1/800 titer). In view of these findings, the case was considered as Salmonella glomerulonephritis; his clinical and laboratory recovery were achieved by supportive therapy.

Key words: children, typhoid fever, glomerulonephritis, salmonella.

Many etiologic agents have been implicated in acute post-infectious glomerulonephritis. The identification of an etiologic agent requires the temporal association with an illness in which a specific agent has been isolated, a serologic response to that agent that can be documented by rising antibody titers, and/or identification of the antigen or its antibody in the glomerulus[1,2]. Typhoid fever is relatively common but is rarely associated with glomerulonephritis[2]. Glomerular involvement has been described as being either in mesangial proliferative glomerulonephritis or diffuse proliferative glomerulonephritis form[3,4]. In this report, a nine-year-old male patient was presented who was diagnosed as Salmonella glomerulonephritis.

Case Report

A nine-year-old boy was admitted to our clinic because of macroscopic hematuria, oliguria, edema, hypertension and high fever persisting for two weeks. On physical examination he was found in moderate condition, with pallor, periorbital and pretibial edema and hepatosplenomegaly. His body temperature, pulse rate and blood pressure were 38°C, 108 beat/min and 150/90 mmHg, respectively. The whole blood counts were as follows: white blood cells 3,800/mm^3, hemoglobin (Hb) 6.3 g/dl and platelets 240,000/mm^3. Urine analysis revealed severe proteinuria with a specific gravity of 1020. Urine protein loss was 52 mg/m^2 per hour. Serum urea was 140 mg/dl, creatinine 1.8 mg/dl, aspartate aminotransferase 66 U/L, alanine aminotransferase 59 U/L and serum complement (C_3) 23.6 mg/dl. His serum total protein was 5 g/dl and albumin 2.5 g/dl, and serum immune globulins were normal. Renal biopsy was performed because of heavy proteinuria. Irregular, coarse granular mesangial accumulation of C_3 (+++) and IgG (+) was detected by immuno-fluorescence microscopy. Histopathological findings were in agreement with diffuse proliferative glomerulonephritis. Agglutination tests were performed due to persisting high fever. Both Salmonella typhi O and Salmonella typhi H were detected positive (1/800 titer). ANA and Anti ds-DNA were found negative. According to these findings, Salmonella glomerulonephritis was considered in this case. Ceftriaxone treatment along with supportive therapy were given. The clinical findings improved and urine protein became negative following the treatment. Serum complement level also returned to normal within eight weeks.

Discussion

Salmonella infections can be diagnosed easily when presenting with classical symptoms. In contrast, in those cases presenting with atypical

symptoms, difficulty in diagnosis might occur[3]. Renal involvement in typhoid fever might manifest as cystitis or pyelonephritis[2,5]. Acute tubulointerstitial nephritis was also reported as a rare cause of acute renal dysfunction during Salmonella typhimurium infections[6,7]. In addition, glomerular involvement has been described as a rare complication of typhoid fever[2,3]. In patients with glomerular involvement, microscopic and occasionally macroscopic hematuria, associated with moderate proteinuria and normal or slightly diminished renal function, could be found at the first stage of the disease. Clinical findings of Salmonella glomerulonephritis are different from those of poststreptococcal glomerulonephritis (PSGN)[5]. Although the cause has not been accurately explained, edema may last more than four weeks in some cases with Salmonella glomerulonephritis[2,3]. Persisting high fever, increased transminases and splenomegaly are also found in these cases. In addition, serum C_3 level decreases to a lesser degree than in cases with PSGN[2,3,5].

Our case was admitted to the clinic because of high fever lasting for two weeks, hematuria, oliguria, edema and hypertension. His physical findings revealed high fever, hypertension, edema, pallor, oliguria, splenomegaly and hepatomegaly. Laboratory results showed elevated serum urea, creatinine, and transaminase and decreased C_3 levels. In addition, hematuria and proteinuria were present. Glomerulonephritis was considered on the basis of these findings. Renal biopsy was performed due to continuous severe proteinuria, and diffuse proliferative glomerulonephritis was detected. It was reported that biopsy results of glomerular involvement in typhoid glomerulonephritis might be found in such forms as diffuse mesangial proliferation, IgA nephropathy or acute diffuse proliferative glomerulonephritis[1-5]. Sitprija et al.[4] detected Salmonella Vi antigen in the renal biopsy of patients with typhoid glomerulonephritis. Diagnostic difficulties can be experienced in Salmonella glomerulonephritis since it is not seen as frequently as PSGN and because the clinical symptoms of Salmonella glomerulonephritis are not well described. In this case, Salmonella glomerulonephritis was diagnosed based on both clinical and renal biopsy findings. Edema, macroscopic hematuria, oliguria, and hypertension were the striking symptoms along with positive group agglutination test and diminished serum C_3 level. However, serum complement levels returned to normal following the antibiotic treatment. This also supported the diagnosis of Salmonella glomerulonephritis. Our patient is now in follow-up, and his clinical and laboratory findings have improved.

In conclusion, immune-mediated glomerulonephritis must be considered as a rare cause of oliguria, hypertension, hematuria and decreased serum complement (C_3) during Salmonella typhimurium infection.

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