Spontaneous Bacterial Peritonitis in an Adult Patient with Nephrotic Syndrome

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Most cases of spontaneous bacterial peritonitis (SBP) in association with nephrotic syndrome are children. The complication of SBP in adults with nephrotic syndrome is extremely rare. Herein, we report a 25-year-old man with nephrotic syndrome and chronic renal failure who suffered from SBP. Citrobacter freundii was isolated from ascites. Irreversible deterioration of renal function followed the development of SBP, though the peritonitis was cured with antibiotic treatment. This case suggests that SBP is a rare, but serious complication of adult nephrotic syndrome with ascites. (Internal Medicine 32: 719-721, 1993)

Key words: peritonitis, adult, nephrotic syndrome

Introduction

Spontaneous bacterial peritonitis (SBP) is one of the important complications of childhood nephrotic syndrome which occurs not infrequently (1-4). The complication of SBP in adults with nephrotic syndrome is, however, extremely rare and only two cases have been reported to our knowledge (5, 6). We herein report a case of SBP in an adult nephrotic patient with chronic renal failure.

Case Report

A 25-year-old Japanese-Brazilian man was admitted to our hospital for the treatment of massive ascites due to nephrotic syndrome on November 11, 1992. The diagnosis of nephrotic syndrome was first made when he was 3 years old, and from then he had suffered frequent relapses of nephrotic syndrome. Histological diagnosis of focal glomerulosclerosis was made when he was 10 years old. He was treated with prednisolone and cyclophosphamide, but the renal function gradually deteriorated. These treatments had been discontinued in May 1989. On admission, physical examination revealed massive ascites with peripheral edema. Blood pressure was 140/90 and pulse rate was 92/min. Laboratory findings were the following: serum protein, 4.9 g/dl; serum albumin, 1.8 g/dl; serum IgG, 671 mg/dl; serum IgA, 135 mg/dl; serum IgM, 286 mg/dl; serum cholesterol, 313 mg/dl; serum creatinine, 6.0 mg/dl; serum urea nitrogen, 53.5 mg/dl; serum C₃, 94 mg/dl; serum C₄, 49 mg/dl; serum CH₅₀, 51 U/ml; urine volume, 900 ml/day; urinary protein excretion, 14.9 g/day. Echographic findings revealed atrophic kidneys and massive ascites. He was treated with furosemide and water restriction. On December 28, he suddenly complained of abdominal pain and watery diarrhea. His temperature was 38.7°C. The abdomen was distended and diffusely tender with rebound tenderness. The white blood cell count was 6800/mm³ with 71% segmented neutrophils and 10% band forms, and the serum CRP was 30.7 mg/dl. The aspirated ascites was cloudy yellow, and contained 7,200 white cells/mm³. The organism isolated from ascites was Citrobacter freundii. Blood culture for bacteria was negative. Abdominal X-ray and echographic studies showed no evidence of gastrointestinal perforation, appendicitis, diverticulitis or cholangitis, which cause secondary peritonitis. Following treatment with intravenous imipenem/cilastatin and intraperitoneal tobramycin for eight days, the abdominal pain and fever disappeared. However, his renal function worsened and he complained of appetite loss and general malaise. Following the treatment with hemodialysis, these symptoms disappeared, but the renal function did not improve (Fig. 1).

Discussion

Spontaneous bacterial peritonitis is a relatively common problem in adult patients with liver cirrhosis and occurs in approximately 10% of cirrhotic patients (7). The complication of SBP in adults with nephrotic patients is, however, extremely
rare (Table 1). To our knowledge, only two cases have been reported in the literature (5, 6). Rusthoven and Kabins (5) first reported a 41-year-old woman with nephrotic syndrome for 2 years due to focal glomerular sclerosis. She suffered from SBP when she was not on corticosteroid therapy. Cameron et al (6) also reported a case of nephrotic syndrome complicated with pneumococcal peritonitis.

Recent studies demonstrated that Streptococcus pneumoniae is the most common pathogen causing SBP in childhood nephrotic patients (1, 2). Gram-negative organisms, which are the major bacterial pathogen in adult cirrhotic patients, account for only 10–20% of organisms isolated from ascites (1, 2). Citrobacter freundii isolated in this case has not been previously reported as a cause of spontaneous peritonitis in patients with nephrotic syndrome. The predisposition to pneumococcus infection in nephrotic patients might reflect the fact that the most common pathogen causing bacteremia and/or sepsis is pneumococci in childhood but it is gram-negative organisms in cirrhotic patients.

In the present case SBP occurred 22 years after the onset of nephrotic syndrome. The complication of peritonitis usually occurs in the first 2 years of nephrotic syndrome (1). This case demonstrated that it may occur at any time of the course of the nephrotic syndrome. The prognosis of SBP in nephrotic syndrome is poor and a mortality rate of 9% has been reported (2). In this case, the peritonitis induced the deterioration of renal function and the development of uremic symptoms, though the peritonitis was cured by treatment with antibiotics.

The reason why the complication of SBP in nephrotic patients is extremely rare in adults when compared with the cases in children is unclear. Low IgG level, impairment of Factor B level and leukocyte dysfunction are implicated as causes of altered defense mechanisms in children with nephrotic syndrome (6). Akalin et al (8) demonstrated that bactericidal activity and the concentrations of complement and immunoglobulins in peritoneal fluid are also significantly lower in adult patients with nephrotic syndrome when compared with normal subjects. Actually, the serum IgG level in this case was decreased to 671 mg/dl.

The increased susceptibility to infection during immunosuppressive therapy is well known in patients with nephrotic syndrome. Corticosteroid dosage per lean body mass for treatment of nephrotic syndrome is generally higher in children than in adults. The possibility remains that corticosteroid-induced immunological abnormalities might predispose to the development of infection in children with nephrotic syndrome. However, Feinstein et al (2) reviewed that in only about 50% of the nephrotic children the spontaneous peritonitis occurred while receiving corticosteroid therapy. It is unlikely, therefore, that immunosuppressive therapy is the primary factor in the development of peritonitis in nephrotic children. On the other hand, it is known that ascites in nephrotic syndrome is observed more frequently in children than in adults. The existence of ascitic fluid may contribute to the development of SBP (6).

In summary, an adult case of SBP in nephrotic syndrome with chronic renal failure was reported. The peritonitis induced deterioration of the renal function and development of uremic syndrome, though the peritonitis was cured by treatment with antibiotics. This case suggests that SBP is a rare, but a serious complication of adult nephrotic syndrome with ascites.

References

5) Rusthoven J, Kabins SA. Hemophilus influenzae fcellulitis with bacteremia, peritonitis, and pleuritis in an adult with nephrotic syndrome.

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Table 1. Spontaneous Bacterial Peritonitis in Adult Patients with Nephrotic Syndrome

<table>
<thead>
<tr>
<th>Age/sex</th>
<th>Histology</th>
<th>Organism</th>
<th>Steroid therapy</th>
<th>Outcome</th>
<th>Ref.</th>
</tr>
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<tbody>
<tr>
<td>1. 41/F</td>
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<td><em>H. influenzae</em> f</td>
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<tr>
<td>2. 20/M</td>
<td>MC</td>
<td><em>S.pneumoniae</em></td>
<td>ND</td>
<td>ND</td>
<td>6</td>
</tr>
<tr>
<td>3. 25/M</td>
<td>FGS</td>
<td><em>C.freundii</em></td>
<td>(–)</td>
<td>Deterioration of</td>
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