Case Reports

Fulminant Group A Streptococcal Infections

Report of Two Cases

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Summary. We describe two female patients presenting with spontaneous peritonitis and fulminant Streptococcus pyogenes (Strep. pyogenes) septicemia and shock. Both patients recovered completely upon immediate antibiotic therapy, initially with broad range combination therapy effective against Strep. pyogenes, which was switched to penicillin G when culture results became available. The isolated strain in case 1 was M-type 28, which is the M-type most often isolated from vaginal swabs (as commensal) and from blood from patients with puerperal sepsis. Patient 1 had signs and symptoms of a toxic shock-like syndrome, including rapid onset of fever and shock, skin rash, desquamation of palms and soles, and multisystem involvement with vomiting, diarrhea, myalgia, renal failure, and severe disorientation without focal neurological deficits.

Key words: Streptococcus pyogenes – Group A beta-hemolytic streptococci – Streptococcal pyrogenic toxins – M-Typing – Peritonitis – Septicemia – Toxic shock syndrome

Streptococcus pyogenes, the group A beta-hemolytic streptococcus, is a common pathogen that causes pharyngitis, impetigo, erysipelas, necrotizing fasciitis, myositis, puerperal fever, bacteremia, and the nonsuppurative sequelae of rheumatic fever, glomerulonephritis, and erythema nodosum. While most infections caused by this organism are relatively benign, the pathogen has the capacity for explosive and life-threatening disease [1, 3–6, 8, 10, 12, 13, 23, 25]. Furthermore, a toxic shock-like syndrome has been associated with Strep. pyogenes infections [3, 5, 12, 23, 25]. The pyrogenic streptococcal type A, B, and C toxins are common extracellular products, found in approximately 90% of streptococcal isolates [19], and have been implicated in the pathogenesis of the streptococcal toxic shock-like syndrome [3, 5, 12, 13].

We describe two patients presenting with peritonitis, fulminant streptococcal septicemia, and shock. In addition, case 1 presented signs and symptoms which fulfilled accepted criteria of the toxic shock syndrome [11].

Case 1

On April 6, 1988, a 50-year-old woman was admitted to another hospital with the clinical signs of peritonitis and hypotensive shock (blood pressure 50/20 mmHg). The day before admission there was a sudden onset of nausea, vomiting, progressive diarrhea, and fever up to 39.2°C. The clinical diagnosis was acute gastroenterocolitis and puerperitis, and an emergency laparotomy was performed 2 days after admission. About 1 l of serous ascites was drained. The peritoneum was hyperemic, but without fibrin deposits or adhesive bands. The large and small bowel were dilated and their wall was edematous.

Beta-hemolytic group A streptococci, susceptible to all antibiotics tested, including ampicillin, erythromycin, sulfamethoxazol-trimethoprim, and vancomycin, grew in blood cultures. Gram-positive cocci arranged in chains of varying length were found in the peritoneal fluid. Unfortunately, the peritoneal fluid samples were not further cultured. Antibiotic treatment with cefamandol and tobra-
mycin was begun immediately and subsequent cultures remained negative. After initial improvement the patient subsequently developed myalgias (the creatine phosphokinase remained within the normal range), persistent diarrhea, vomiting, relapsing fever, hypotension, and progressive renal failure with a rise of the serum creatinine up to 460 μmol/l and transient hypocalcemia (lowest recorded value 1.97 mmol/l). Severe disorientation without focal symptoms of the central nervous system was further noted. On continuing antistreptococcal therapy with high dose penicillin-G she progressively improved, but later developed erythema and severe desquamation of the fingers and toes.

Further serotyping of the isolated group A streptococcal strain, based on the antigenic property of the M-protein, revealed an M-type 28. Blood samples for serological examination were drawn on day 6 and 35 after onset of fever and gastrointestinal symptoms. Antistreptolysin O and antideoxyribonuclease serum levels fell from 800 U/ml to 640 U/ml and from 1920 U/ml to 1440 U/ml, respectively; while antihyaluronidase serum levels remained unchanged (> 4096 U/ml).

Case 2
In June 1988, a 58-year-old woman was hospitalized because of exacerbation of rheumatoid arthritis. The diagnosis of rheumatoid arthritis was made 31 years before, in 1957. At the time of admission to the hospital, rheumatoid arthritis was being treated with sodium-proxen 500 mg q.i.d. and regular gold injections. On admission antiinflammatory therapy was intensified by adding prednisone 20 mg per day and methotrexate 2.5 mg per day. One the 29th day of the hospital stay there was a sudden onset of abdominal pain with signs of paralytic ileus and fever up to 39°C. Five hours later hypotensive shock set in. Septic toxic shock was diagnosed and empiric antibiotic therapy with flucloxacillin, netilmicin, and ornidazol was begun immediately. Since normal blood pressure could not be restored by the use of large volumes of intravenous crystalloids and vasopressors and signs of paralytic ileus progressed, emergency laparotomy was performed 12 h after the onset of abdominal pain. About 1500 ml of fibrinoid exudate within the abdominal cavity, multiple microabscesses along the mesenterial root of the ileum and colon, as well as a right-sided pyosalpinx were found. Multiple specimens of intraabdominal microabscesses were taken for culture and right-sided salpingectomy and oophorectomy was performed. Postoperatively the patient was still hypotensive and extubation was possible only after 12 h. There was persistent oliguria due to prerenal failure and the serum creatinine and urea nitrogen peaked at 163 μmol/l and 11.7 mmol/l, respectively. Furthermore, thrombocytopenia (lowest recorded value 43000/μl) and mild and reversible hepatic dysfunction with a rise of the alkaline phosphatase to 416 U/l (60–170 U/l) and of the gamma-glutamyl transpeptidase to 183 U/l (4 to 28 U/l) were noted. The blood cultures drawn immediately after the onset of fever grew group A beta hemolytic streptococci; the strain was susceptible to all antibiotics tested, including penicillin, doxycycline, ampicillin, cefuroxim, ceftriaxone, and netilmicin. Therapy was switched to penicillin G 4000000 units six times per day, and was continued for another 2 weeks. Cultures of peritoneal fluid, mesenterial microabscesses, and the resected pyosalpinx remained negative. Postoperatively gastrointestinal mobility normalized promptly, and the patient's condition improved rapidly without persisting sequelae of peritonitis.

Discussion
We describe two patients with fulminant Strep. pyogenes septicemia and shock. The clinical courses of the two patients was strikingly similar: both female patients with Strep. pyogenes septicaemia and shock initially presented with peritonitis without preexisting abdominal disease, i.e., as spontaneous bacterial peritonitis. Emergency laparotomy revealed large amounts of ascites in both cases and right-sided pyosalpinx with multiple microabscesses along the mesenterical root in case 2. Both patients recovered completely upon immediate antibiotic therapy with initially broad range combination therapy, and subsequently, after obtaining culture results, high-dose penicillin G therapy.

These two cases corroborate several recent studies suggesting that the frequency of severe group A streptococcal infection may have increased [3, 5, 8, 9, 12, 13, 23]. Two recent large surveys demonstrate the dominante role of the skin as a portal of entry for Strep. pyogenes bacteremia [8, 13]. Most patients included in these series presented with cellulitis or necrotizing fasciitis at sites of a recent trauma or preexisting ulcer [8]. Less frequently bacteremia originates from the respiratory tract [8, 13]. Strep. pyogenes bacteremia originating from the peritoneal cavity has also been described [6, 8, 10, 13]. Of 23 patients with Strep. pyogenes sepsis who initially had no evident primary site of sepsis, seven were later shown to have