SPONTANEOUS BACTERIAL PERITONITIS DUE TO STREPTOCOCCUS PNEUMONIAE – CASE REPORT

ANDRZEJ LITARSKI, DARIUSZ JANCZAK, JAN CIANCIARA, MARCIN MERENDA

Department of Surgery of 4th Military Clinical Hospital in Wroclaw
Kierownik: dr n. med. J. Cianciara
Department of Vascular, General and Transplantological Surgery, Medical University in Wroclaw
Kierownik: prof. dr hab. P. Szyber

Spontaneous bacterial peritonitis is caused by infection of ascitic fluid without any apparent intra-abdominal source of infection. The disease most commonly occurs in patients with cirrhosis and 70% of cases of infections are caused by pathogens from gastrointestinal tract. The article presents the case of 38-year-old patient with spontaneous peritonitis who was treated surgically. The primary nature of the disease was confirmed by laparotomy and bacteriological examination results (Streptococcus pneumonia) of ascitic fluid. After 54 days of hospitalisation and undergoing re-laparotomy, he was discharged in good condition.

Key words: spontaneous bacterial peritonitis, Streptococcus pneumoniae, ascites
inal ultrasound showed the presence of large amounts of free fluid in the peritoneum. Next, peritoneal puncture was performed, resulting in evacuation of turbid, milky fluid. Biochemistry tests performed upon admission revealed elevated glucose level (141 mg%), leukocytosis (12 G/l), creatinine level of 3.58 mg%, urea of 215 mg%, ALT of 41 U/l, AST of 77, CRP of 375 mg/l, extension of PT to 13.8 sec and lowered haemoglobin to 10.5 mg% and haematocrit to 33%.

In view of the above, the patient was qualified for surgery. The exploratory laparotomy confirmed the presence of large amount of turbid, milky and odourless fluid within the peritoneum. The perioperative examination did not reveal any pathologies in abdominal cavity organs. After evacuating the fluid and multiple drainage of the peritoneal cavity, the patient was transferred to the Anaesthesiology and Intensive Care Department due to intensifying symptoms of cardiorespiratory failure. The culture from the abdominal fluid produced bacterium: Streptococcus pneumoniae sensitive to ceftriaxone, ciprofloxacin, co-trimoxazole, erythromycin and penicillin. Blood and urine cultures did not reveal the presence of any pathogens. Chest X-rays, performed several times, did not show focal lesions in lungs.

In the initial phase of the disease, the treatment included: ceftriaxone, co-trimoxazole, meropenem and fluconazole, while after obtaining the culture results: ceftriaxone with metronidazole. Due to persisting inflammatory markers (CRP 170 mg/l, PCT 4.23 ng/ml) and abdominal pain, CT scan of the abdomen was performed on day 10 post surgery. The imaging examination revealed a fluid sac of 12 cm x 5 cm x 4.5 cm on the side of the greater curvature of the stomach, with the sac being enhanced after the administration of contrast agent. Laparotomy was performed again, during which the abscess from the region of omental bursa was drained. Culture from the abscess produced Acinetobacter baumannii sensitive to imipenem and meropenem – treatment as per the antibiogram was initiated. During the subsequent days of hospitalisation, due to the intensifying renal failure, dialysis was initiated and continued for 25 days. Next, the patient was transferred again to the Surgery Department where his condition was systematically improving. The patient was discharged home in a good general condition, after 54 days of hospitalisation.

**DISCUSSION**

Spontaneous bacterial peritonitis is caused by the infection of ascitic fluid of the abdomen and most commonly occurs in the course of uncompensated liver cirrhosis. Currently, it is estimated that SBP in the course of cirrhosis and ascites occurs in approx. 15% of patients (4). In over 90% of cases, the infection of the ascitic fluid is caused by one bacterial strain only, of which 50-70% are due to gastrointestinal pathogens such as Escherichia coli, Enterococcus faecalis, Enterobacter, Proteus spp. Non-gastrointestinal pathogens account for 25-30% of cases and these are usually due to Streptococcus pneumoniae, while only rarely (3-5%) due to anaerobic bacteria (3, 5, 6). SBP pathogenesis has not been fully elucidated, but it is assumed that the infection is caused by bacteria penetrating from the gastrointestinal tract lumen into the mesenteric lymph nodes, and from here into the portal circulation. The translocation is facilitated by oedema and increased permeability of intestinal mucosa, as well as excess proliferation of bacteria colonising the intestinal lumen, caused by disrupted gastrointestinal tract motility. In addition, there also occurs impairment of the phagocytic activity of the reticuloendothelial system and the antibacterial one of the ascitic fluid. Other sources of infection may be inflammatory foci within the respiratory and urinary system (3, 7).

In the case presented here, in the course of toxic liver damage, there occurred an infection of the ascitic fluid with a single bacterial strain, i.e. Streptococcus pneumoniae. In the history, the patient reported pneumonia suffered 8 years previously, which might have some bearing on his current condition. SBP caused by Streptococcus pneumoniae in patients with uncompensated liver cirrhosis is usually associated with respiratory tract infection. In such cases, the infection of ascitic fluid is by blood, from the focus localised in the respiratory system (7, 8). Of impact on the current patient’s condition might have been endoscopic examinations performed in the past due to duodenal peptic ulcer, as well as long-term use of agents lowering the secretion of gastric juice (omeprazole).
Invasive diagnostic and therapeutic procedures in patients with ascites carry a risk of SBP. Due to the impaired defence mechanisms, such as reduced secretion of bile and gastric acids as well as compromised immune system in the intestinal mucosa, there occurs colonisation and translocation of bacteria within the gastrointestinal tract (5, 7, 9). Although Streptococcus pneumoniae is absent in the physiological environment of the gastrointestinal tract, if the above-described situation occurs, this pathogen may proliferate (7). Such a mechanism of ascitic fluid infection seems most likely in the case presented here, since the peripheral blood cultures performed several times did not reveal any presence of Streptococcus pneumoniae. The clinical picture of SBP is not very characteristic or distinct. The typical symptoms of peritonitis occur rarely and are often difficult to detect due to the existing ascites. The most common disease symptoms are fever and spread abdominal pain. Peritoneal symptoms and abolished peristalsis occur in half of the patients (3). Sometimes, the only symptom of SBP is fever and unexplained encephalopathy. In 10% of cases, the infection is completely asymptomatic (3, 5, 10), and some of them suffer septic shock at the time of diagnosis. In the case presented here, the patient manifested typical symptoms of peritonitis in the form of fever, spread abdominal pain, evident guarding and inaudible peristaltic sounds.

In available publications indicate that mortality in asymptomatic SBP stands at 27%, while that of symptomatic SBP reaches even 55%. This is why proper diagnostic and therapeutic approach is of crucial importance (4). The diagnosis is dependent on finding neutrophilia >250/mm$^3$ in the ascitic fluid, without detectable source of infection within the abdominal cavity, and positive culture. However, in 20-40% of patients, despite inflammation features, cultures from the ascitic fluid are negative. In such cases, the diagnosis is dependent on the ascitic fluid neutrophil count of >500/mm$^3$ with no symptoms, or of 250/mm$^3$ with peritonitis symptoms present (3, 6). Paracentesis was performed in the patient described here, which confirmed the presence of free fluid found earlier in abdominal ultrasound. In the face of peritoneal symptoms and deteriorating general condition of the patient, it was decided to perform surgery. In addition, positive history of duodenal peptic ulcer suggested the possibility of peritonitis in the course of gastrointestinal perforation. Only the performed exploratory laparotomy and absence of detectable infection source in the abdominal cavity indicated the spontaneous nature of the disease. The obtained result of culture from the ascitic fluid, revealing the presence of Streptococcus pneumoniae, confirmed the diagnosis.

Of crucial importance is differentiation SBP from secondary peritonitis in patients with ascites, e.g. that resulting from gastrointestinal perforation. It has been reported that 5% to 15% of patients with infected ascitic fluid have intra-abdominal source of infection (6). Proper differentiation is crucial also as regards the selection of therapeutic approach for these two disease processes. Secondary peritonitis definitely requires surgical treatment, while the majority of SBP cases is treated conservatively with the use of antibiotics. Third generation cephalosporins are the drugs of choice in the empirical treatment until the time of obtaining the culture results for the ascitic fluid. The antibiotic therapy may also include piperacillin with tizobactam, ampicillin with sulbactam, and ticarcillin with clavulanic (5, 6, 10). This is why SBP diagnosis is of crucial importance, although it is not an easy task due to not very specific symptoms of peritonitis and distinct clinical picture. For a surgeon who does not often encounter cases as described here, SBP may pose a real diagnostic and therapeutic problem. In due time, a correct choice has to be made between conservative and surgical treatment. In the SBP case presented here, laparotomy and peritoneal drainage seem to be a correct decision. The diseases in patient’s history, the presence of free fluid in the peritoneal cavity, and the clinical symptoms, all indicated the need for emergency surgical intervention.
REFERENCES


Received: 3.01.2011 r.
Adress correspondence: 50-980 Wrocław, ul. Rudolfa Weigla 5