Spontaneous Bacterial Empyema Caused by *Streptococcus pneumoniae*

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Spontaneous bacterial empyema, a rare and an under-recognized complication of liver cirrhosis, is an infection of a preexisting hydrothorax, which is defined by the presence of > 500 neutrophils/mm^3 in pleural fluid or a positive culture of pleural fluid with > 250 neutrophils/mm^3, after the exclusion of parapneumonic infection by chest X-ray or computed tomography scan [1].

Previously reported cases of SBEM were mostly caused by Enterobacteriacae, Aeromonas spp, and Streptococcus spp (*Streptococcus bovis*, *mitis*, *sanguis*, *agalactiae*, and *intermidius*), which are part of the gastrointestinal flora. We report here the first case of SBEM caused by *Streptococcus pneumoniae*.

PATIENT DESCRIPTION

An 84 year old man presented with dyspnea and palpitations of a few days duration. His past medical history was remarkable for chronic obstructive pulmonary disease, hypertension, smoking and heavy alcohol consumption. His medications included allopurinol, folic acid, alfuzosin and atenolol. His blood pressure was 140/66 mmHg, pulse rate regular at 108/min, oral temperature 37°C, respiratory rate 32/min, and hemoglobin oxygen saturation 85% while breathing room air. Physical examination revealed stigmata of chronic liver disease, ascites, mildly increased jugular venous pressure, and decreased breath sounds with dullness on percussion of both lung bases. Although the patient was not known to suffer from liver cirrhosis, the stigmata of liver disease and ascites on physical examination in addition to the consumption of large amounts of alcohol strongly suggested liver cirrhosis.

Laboratory examination revealed white blood cells 12.15 x 10^9/L (95% neutrophils), macrocytic anemia (hemoglobin 9.4 g/dl and mean corpuscular volume 110 fl), platelets 114 x 10^9/L, aspartate aminotransferase 54 IU/L, alanine aminotransferase 30 IU/L, alkaline phosphatase 97 IU/L, albumin 2.28 g/dl and sodium 132 mEq/L. Prothrombin time was 64% (normal > 70) and erythrocyte sedimentation rate was 116 mm in the first hour. A chest X-ray demonstrated bilateral pleural effusion, larger on the left side, and a CT of the chest demonstrated bilateral pleural effusion with atelactasis of the left lower lobe with no evidence of pneumonia. Pleurocentesis demonstrated clear pleural fluid, with low levels of protein 2.2 g/dl (serum protein 5.6 g/dl), glucose 171 mg/dl, pH 7.4, elevated lactate dehydrogenase level of 691 IU/dl, and white blood cell count of 2.34 x 10^9/L (67% neutrophils); cytology was negative for tumor cells. An echocardiography showed normal left ventricular function and increased pulmonary artery pressure, 60 mmHg.

Considering that the patient suffered from alcoholic liver cirrhosis with ascites and hydrothorax, treatment with diuretics and a low salt diet was initiated, but there was no improvement.

Streptococcus pneumoniae, sensitive to all antibiotics, grew in culture of pleural fluid. Since the patient remained afebrile and there was no other clinical or laboratory evidence of empyema, a repeat pleurocentesis was performed, which again revealed similar parameters and growth of the same bacterium. Treatment with ampicillin was initiated and a repeat CT scan of the chest was performed, again showing bilateral pleural effusion with no sign of an inflammatory infiltrate. A third pleurocentesis was performed, finally demonstrating parameters consistent with empyema: lactate dehydrogenase 992 U/dl, glucose 1 mg/dl, pH 6.94, and protein 3 g/dl (serum protein 5.8 g/dl). Blood cultures that were obtained before starting ampicillin were negative.

A chest tube was inserted with subsequent drainage of 1500 ml of foul-smelling serous fluid. The chest tube was removed a few days later and antibiotic treatment was continued for a total duration of 2 weeks. The patient recovered uneventfully.

COMMENT

Hepatic hydrothorax is defined as a pleural effusion, usually larger than 500 ml, in a patient with cirrhosis without a primary cardiac, pulmonary or pleural disease [2]. It is present in 4%–10% of patients with advanced cirrhosis and is caused by fluid

SBEM = spontaneous bacterial empyema

retention and passage of ascites from the peritoneal to the pleural cavity, through small diaphragmatic defects located in the tendinous portion of the diaphragm [2].

Cirrhosis of liver is known to be associated with increased incidence and severity of infections, with infection-attributed death occurring in 7%–40% of these patients. This is due to neutrophilic dysfunction, complement deficiency, deficient reticuloendothelial phagocytic action, increased tumor necrosis factor-alpha activity, and impaired antibody-mediated bactericidal activity found in cirrhosis [3].

Whereas spontaneous bacterial peritonitis is a well-known entity, SBEM is a rare infective complication of a preexisting hydrothorax in cirrhotic patients. It is probably caused by two different mechanisms: passage of infected ascitic fluid into the pleural cavity or bacteremic seeding of a preexisting hydrothorax [1]. Pleural fluid from patients with hepatic hydrothorax was found to have lower opsonic activity than found in the pleural fluid of patients with other causes, which might be a predisposing factor for developing SBEM [4].

In a prospective study of 120 cirrhotic patients with pleural effusion on admission who underwent a diagnostic pleurocentesis, 13% had 24 episodes of SBEM; all of them with advanced cirrhosis, and only 57% had associated SBP [1].

Our patient, like most previously reported cases, had a pleural fluid with normal protein and glucose levels, and only LDH was elevated. In many of the previously reported episodes of SBEM the pleural fluid properties mimic those of transudative pleural effusion [1]. We suggest that continuous passage of transudative ascitic fluid to the pleural cavity may cause dilution of its protein contents, making it similar to those of transudative effusion. After a few days our patient developed full-blown empyema requiring chest tube insertion. Despite its name, empyema is a rare complication of SBEM. Antibiotic treatment alone is sufficient in most patients with SBEM, and chest tube insertion should be limited to those with empyema [1,5]. Chest tube insertion in cirrhotic patients with hydrothorax can be harmful as it may cause massive electrolyte and protein depletion [5].

The microorganisms isolated from pleural fluid/blood cultures of patients with SBEM in decreasing order of prevalence are *Escherichia coli*, Streptococcus spp, Enterococcus, and *Klebsiella pneumoniae* [1]. To the best of our knowledge, this is the first published case of SBEM caused by *Streptococcus pneumoniae*.

Since the clinical manifestations and pleural fluid findings in SBEM are not as pronounced as in parapneumonic empyema, there should be a low clinical threshold of suspicion in a cirrhotic patient with hydrothorax who is hospitalized because of any clinical deterioration. A diagnostic pleurocentesis should be performed in these patients to rule out SBEM.

SBP = spontaneous bacterial peritonitis
LDH = lactate dehydrogenase

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