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CASE REPORT

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A challenging case of spontaneous bacterial empyema in a cirrhotic patient

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KEYWORDS
Cirrhosis; spontaneous bacterial empyema; infection; thoracentesis; spontaneous bacterial peritonitis

1. Introduction

Spontaneous Bacterial Empyema (SBEM) is distinguished as a primary infection of pre-existing hepatic hydrothorax in the absence of cardiopulmonary or pleural disease. It is an under-recognized complication of cirrhosis and is associated with high morbidity and mortality. The diagnosis requires chest imaging to rule out underlying pneumonia as an etiology; a diagnostic thoracentesis is crucial to make the diagnosis and should be obtained in any patient with pleural effusion and evidence of infection. Expeditious administration of antibiotics is essential to ameliorate the outcome. We present an interesting case of spontaneous bacterial empyema in a cirrhotic patient with hepatic hydrothorax.

2. Case presentation

A 55-year-old male with non-alcoholic steatohepatitis (NASH) cirrhosis presented with four days of shortness of breath, fever, and chills. He denied any urinary symptoms, diarrhea, nausea, or vomiting. He has a history of bleeding esophageal varices that required repeat banding, hepatic encephalopathy, and ascites. He did not require therapeutic paracentesis in the past and never had spontaneous bacterial peritonitis. He was undergoing a liver transplant evaluation but was not yet listed due to abnormal cardiac testing. Before his current presentation, he was hospitalized at an outside hospital for fever up to 103.4. Sepsis work-up revealed negative blood and urine cultures. Chest x-ray was done, and it showed a sizeable right sided pleural effusion, but inadvertently, a diagnostic thoracentesis was not performed. The source of fever was not identified, and he was treated empirically with intravenous antibiotics and was discharged on ten days of oral Levofloxacin.

Physical exam was significant for fever of 102.2, mild tachypnea, and icteric sclera. Chest examination revealed decreased breath sounds on the right lung base with no crackles or rhonchi. The abdomen was mildly distended with mild right upper quadrant tenderness. He had trace bilateral lower extremity edema. Labs were significant for leukocytosis 11.55 k/ul with 74% neutrophils, hemoglobin 10.5 g/dl, thrombocytopenia 57 k/ul, and INR 1.6. Blood cultures and urinalysis were negative for infection. MELD-Na was 23. The Child-Pugh score was 10; class C. Chest X-ray showed a large right pleural effusion with no consolidation (Figure 1). Also, chest computed tomography (CT) was done and showed only large right side pleural effusion with no evolving airspace disease or consolidation (Figure 2).

Abdominal ultrasound showed a small amount of ascites not amenable to paracentesis. Thoracentesis was performed, and 1100 ml of blood-tinged exudative fluid was removed. The fluid analysis was significant for RBC 16000, WBC 2643, with 70% neutrophils. Serum/pleural fluid albumin gradient >1.1 g/dL (Table 1). Fluid gram stain showed moderate polymorphonuclear white blood cells, and fluid culture was negative. Post-thoracentesis chest X-ray showed improvement in the right sided pleural effusion with no evidence of airspace disease (Figure 3). Based on the pleural fluid studies and absence of pneumonia on chest radiography, a diagnosis of spontaneous bacterial empyema was made, and the patient was treated with intravenous ceftriaxone 2 gram every 24 hours for seven days, following which he was discharged in a stable condition on ciprofloxacin prophylaxis with scheduled hepatology outpatient follow-up.

3. Discussion

With a compromised immune system, patients with liver cirrhosis are more susceptible to a bacterial infection such as pneumonia, urinary tract infections, and spontaneous bacterial peritonitis (SBP) which can lead to significant morbidity and mortality [1,2]. Spontaneous bacterial empyema (SBEM) is often less-recognized, which occurs in 2% of cirrhotic patients without hydrothorax and up to 13% of cirrhotic patients with hydrothorax [3,4].

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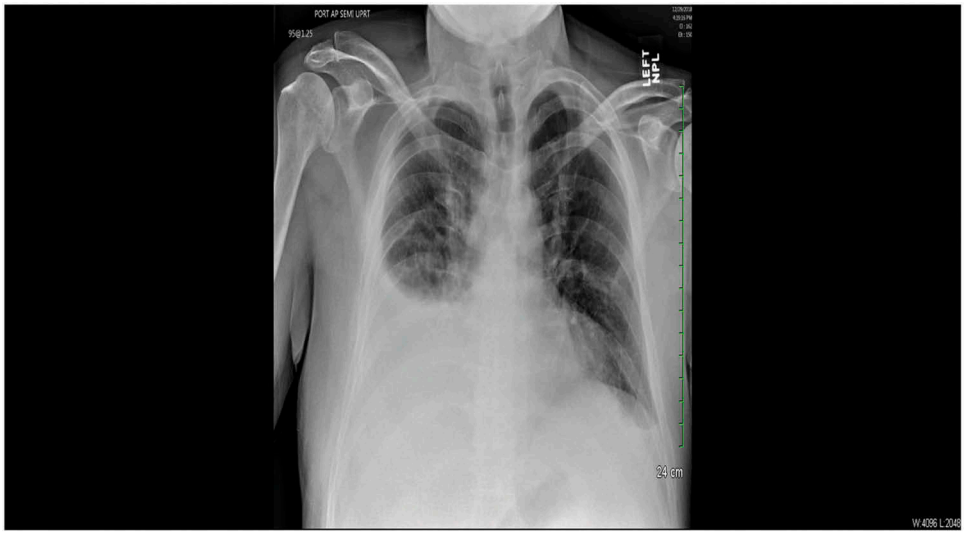

Figure 1. Chest X ray before thoracentesis. It shows large right-sided pleural effusion.

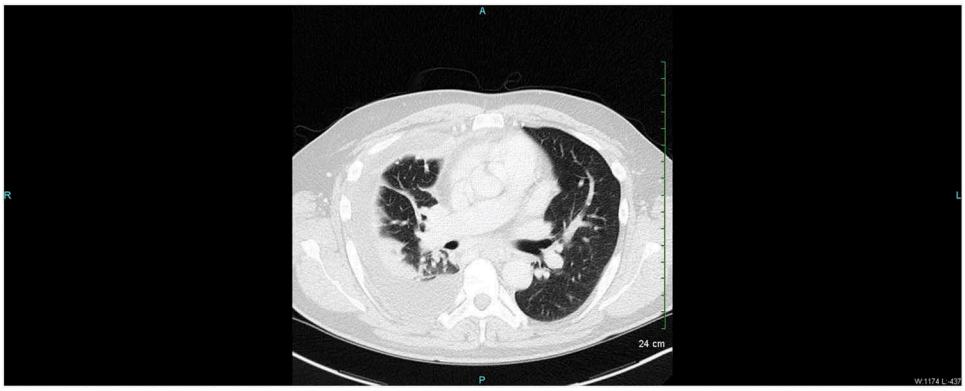

Figure 2. CT scan chest before thoracentesis. It shows large right-sided pleural effusion, but no evidence of consolidation.

Table 1. Pleural fluid analysis and light's criteria.

	Pleural fluid	Serum	Light's criteria
Color	Yellow but slightly blood-tinged	NA	
PH	7.28	NA	
LDH	590 U/L	276 U/L	2.13
Protein	3.8 g/dl	5.7 g/dl	0.66

Usually right sided in location, SBEM is thought to be secondary to a direct seepage of infected ascitic fluid through diaphragmatic defects [4,5]. However, up to 40% of cases occur without underlying SBP or the presence of ascites [3,4]. Thus, an alternative potential explanation of its pathogenesis is the direct infection of the pleural space via transient bacteremia [5,6]. A low pleural fluid opsonic activity, C3, and total protein levels coupled with a high Child-Pugh score are risk factors for bacterial translocation and infection of the pleural space in these patients [7].

While patients may manifest with pulmonary symptoms such as chest pain or dyspnea, other presenting signs and symptoms are often less specific.

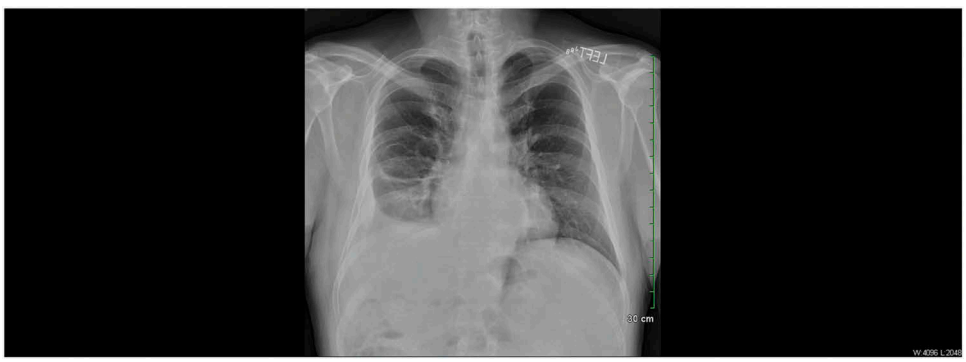

Figure 3. Post thoracentesis chest X-ray. It shows improvement in the right-sided pleural effusion, and absence of consolidation.

Patients may present with fever, malaise, acute encephalopathy, renal insufficiency, or general clinical deterioration [6,8]. Therefore, a high index of suspicion is required for diagnosis [6].

As a significant proportion of SBEM cases occur in the absence of ascites or SBP, diagnostic thoracentesis should not be held or delayed in cirrhotic patients without these complications. Established diagnostic criteria of SBEM are: positive pleural fluid culture and a pleural fluid neutrophil count greater than 250 cells/mm3 or a negative culture study with a pleural fluid neutrophil count greater than 500 cells/mm3 with no evidence of pneumonia on chest imaging [6,8,9]. Reagent test strips of leukocyte esterase originally designed for urine testing are sensitive and specific in providing rapid diagnosis of SBEM [10].

Like most other bacterial infections in these patients, gram-negative bacteria, especially Escherichia coli and Klebsiella pneumonia are the principal causative agents in SBEM [4,6].

There are no clear guidelines regarding management, but the European Association for the Study of the Liver (EASL) recommends to manage SBEM similarly to SBP [11].

The antibiotic of choice is a third-generation cephalosporin such as ceftriaxone or cefotaxime, which should be initiated promptly. Except in the rare event of frank pus in the pleural space, chest tube drainage is not recommended as it predisposes to prolonged fluid and protein loss, electrolyte abnormalities, renal failure, and secondary infection [12,13].

Due to its high rate of recurrence, antibiotic prophylaxis with oral antibiotics should be offered to survivors of SBEM [6]. In contrast to SBP, the utility of albumin infusion in SBEM has not been explored.

SBEM is associated with poor long-term survival and high mortality rates between 20%-38% [5,8,14]. Independent predictors of mortality are initial intensive care unit (ICU) admission, high MELD-Na score, and initial antibiotic treatment failure [14]. Consequently, hepatic hydrothorax and SBEM are considered indications for liver transplantation with similar post-transplantation long-term outcomes between patients with refractory hydrothorax or SBEM and those with noncomplicated hepatic hydrothorax [6,15].

4. Conclusion

SBEM is an under-recognized but important marker of decompensation in liver cirrhosis. We hope to increase physicians awareness of this clinical entity as it may occur independently of SBP and portends a poor prognosis. With a mortality rate comparable to those of the more imminent bacterial complications of liver cirrhosis, prompt recognition and institution of diagnostic and therapeutic interventions are imperative.

Disclosure statement

No potential conflict of interest was reported by the authors.

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