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CASE REPORT | LIVER

Decompensated Liver Cirrhosis Presenting as a Spontaneous Left-Sided Bacterial Empyema

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Abstract

Decompensation of cirrhosis presents with ascites, encephalopathy, variceal bleeding, or spontaneous bacterial peritonitis. Infrequently, decompensation can result from spontaneous bacterial empyema. A 38-year-old man presented with fevers, chills, and dyspnea. Labs were significant for leukocytosis, transaminitis, and coagulopathy. Imaging showed liver cirrhosis with ascites and a left pleural effusion. Treatment of the effusion consisted of chest tube drainage and antibiotics. Spontaneous bacterial empyema was diagnosed after pleural fluid cultures were positive for *Escherichia coli*. Our case demonstrates that spontaneous bacterial empyemas can be left-sided, and the first sign of decompensation.

Introduction

Spontaneous bacterial empyema (SBE) is infection of a pre-existing hepatic hydrothorax in the absence of pneumonia.[1-6] This condition is rare and associated with significant morbidity and mortality. SBE is typically right sided and is diagnosed by thoracentesis. Chest imaging is often helpful to exclude an underlying pneumonia as the cause.

Case Report

A 38-year-old man with a history of alcohol abuse presented with fever and dyspnea. The patient reported that he had been drinking approximately 12 beers and 1 liter of vodka per day for 10 years. His physical exam was significant for fever at 38.9°C, sinus tachycardia at 115 bpm, and hypotension at 95/65 mm Hg. He had scleral icterus, diminished breath sounds and rhonchi in the left lower lobe, mild abdominal distension, trace peripheral edema, and spider angiomata. Laboratory evaluation was notable for anemia with hemoglobin 12.3 g/dL and hematocrit 36.4%, thrombocytopenia 98,000/mm^3, leukocytosis 19,000/mm^3, total bilirubin 8.8 mg/dL, ALT 141 U/L, AST 67 U/L, alkaline phosphatase 155 U/L, PT 26.9 s, and INR 2.4. Further evaluation revealed a negative viral hepatitis panel, autoimmune panel, CMV, EBV, negative acetaminophen and salicylate levels, and ethanol 100 mg/dL. Initial infectious work-up with blood and urine cultures was negative. Chest radiography was remarkable for diffuse opacification of the left hemithorax due to a large pleural effusion (Figure 1). Abdominal ultrasound showed a cirrhotic liver with patent hepatic vessels and moderate ascites (Figure 2).

Given the high likelihood that this patient's infection was from his large left-sided pleural effusion, a bedside thoracentesis was performed. Pleural fluid demonstrated frank purulence consistent with empyema, so a pigtail catheter was placed for continued drainage. The pleural fluid analysis showed leukocytosis (9,920/mm^3; 80% neutrophils) and positive culture for *Escherichia coli*. The post-catheter chest CT and radiographs did not demonstrate pneumonic densities or evolving airspace densities compatible with pneumonia (Figure 3 and Figure 4). Repeat abdominal ultrasound to evaluate for ascites amenable to paracentesis again did not show an easily accessible ascitic fluid pocket (Figure 5). He was diagnosed with SBE and treated with ceftriaxone and continued chest tube drainage.

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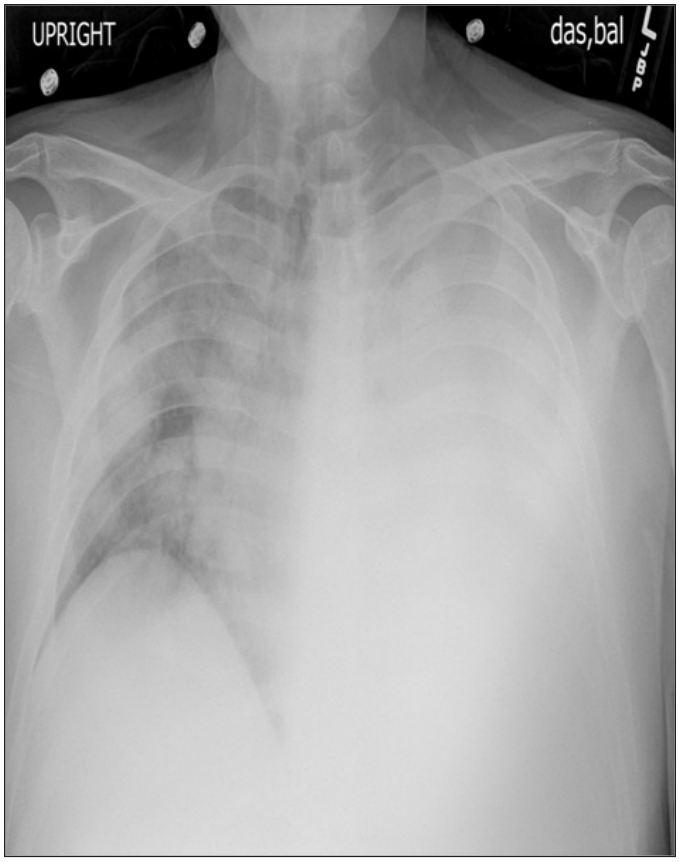

Figure 1. Chest radiograph on admission with opacification of the left lung due to a large pleural effusion prior to pigtail catheter insertion.

Over the course of the patient's 6-day hospitalization, his overall condition improved and he was discharged on ciprofloxacin with close outpatient hepatology and pulmonology follow-up. He is currently prescribed prophylactic dosing of ciprofloxacin and has not had a recurrence of SBE or any other signs of decompensated cirrhosis.

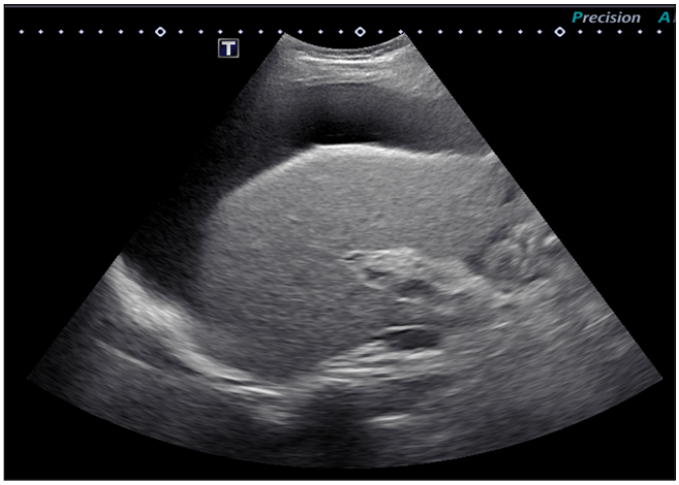

Figure 2. Abdominal ultrasound with a cirrhotic nodular appearing liver and moderate ascites.

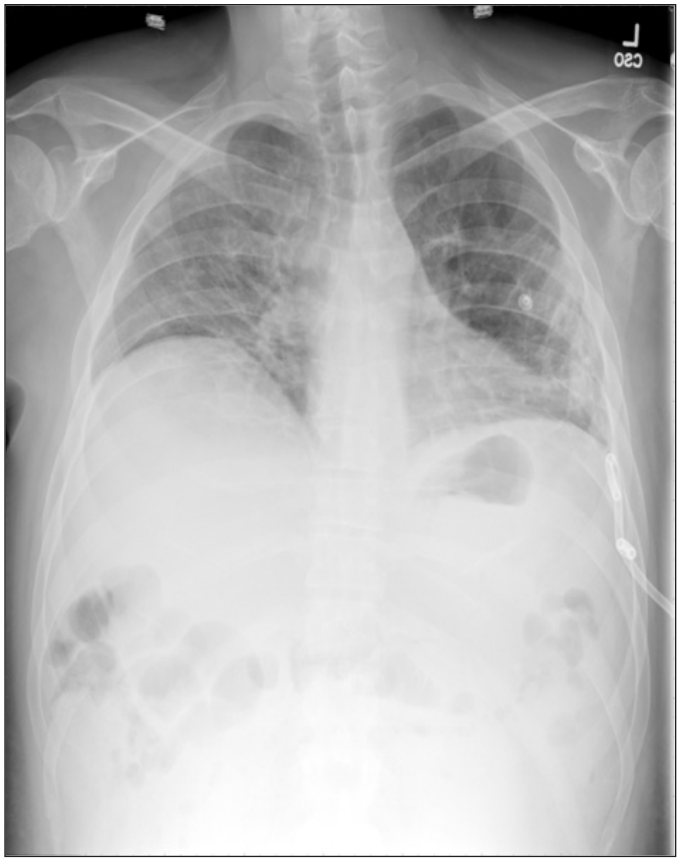

Figure 3. Chest radiograph showing improvement of the left pleural effusion after pigtail catheter insertion.

Discussion

SBE is a rare and underdiagnosed complication of decompensated cirrhosis.[7] The diagnosis of SBE is made by pleural fluid analysis that is culture positive with more than 250 neutrophils/mm^3 or negative with more than 500 neutrophils/mm^3 in the absence of pneumonia.[1,2,3,8]

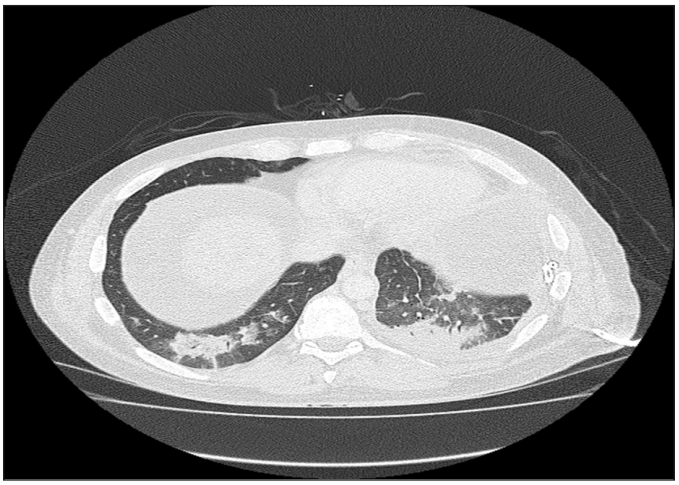

Figure 4. Thoracic CT scan after thoracentesis showing mild residual left pleural effusion after chest tube insertion.

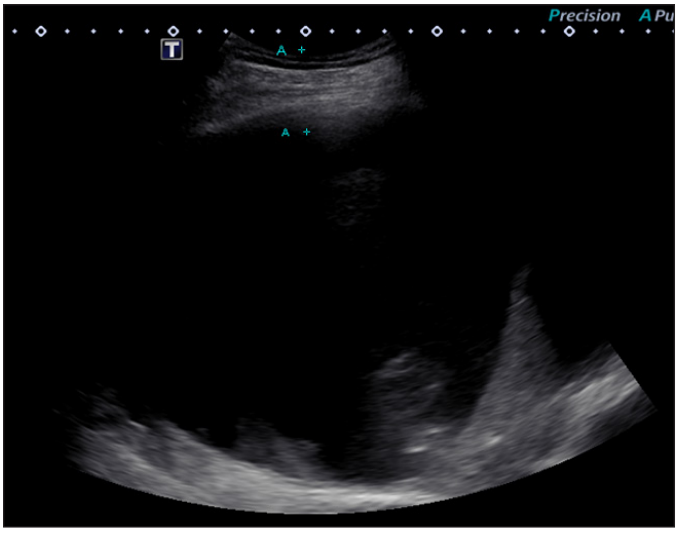

Figure 5. Thoracic ultrasound showing residual left pleural effusion.

The most common cause of SBE is gram-negative bacteria, for which ceftriaxone is the treatment of choice and is typically given for 7-10 days.[2,8,9] Chest tubes are not typically utilized, as continued drainage can lead to protein loss, fluid depletion, coagulopathy, and electrolyte imbalances that can be life-threatening.[4,8,9] However, Chen et al treated 35% of patients with a pigtail catheter due to bacteria isolated in pleural fluid and frank purulence.[3,4]

The estimated mortality of SBE can be as high as 28%. Risk factors associated with the development of SBE include high Child Pugh score, decreased pleural protein, low C3 levels in pleural fluid, low serum albumin, and concomitant spontaneous bacterial peritonitis (SBP). Independent factors associated with poor outcome include high MELD, initial ICU admission, and initial antibiotic failure.[1,4] Long-term survival of SBE is poor, and thus liver transplant should be considered independent of SBP. As with SBP, prophylaxis should be initiated in survivors of SBE.[3]

Like hepatic hydrothorax, SBE is usually right-sided, with left-sided SBE being reported in only small subsets of patients.[4] To our knowledge, this is the first case report of left-sided SBE as the initial presentation of decompensated cirrhosis. Thus, it is important to consider SBE as a diagnosis even if a left-sided effusion is present in a patient without a previous history of cirrhosis.

Disclosures

Author contributions: Both authors contributed equally to study concept and design, analysis and interpretation of data, and writing and critical revision of the manuscript. J. Chertoff is the article guarantor.

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