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Case report

Spontaneous bacterial empyema in a non cirrhotic end stage renal disease patient with immunosuppression

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A B S T R A C T

Spontaneous Bacterial Empyema (SBEM) denotes infection of the pleural fluid in the absence of pneumonia. Almost all cases of SBEM in literature are described in a background of ascites secondary to cirrhosis. Contiguous spread of the infected ascitic fluid through defects in the diaphragm is the most likely mechanism of SBEM. Most of these cases are transudative in nature and are managed with antibiotics. Literature on SBEM in the absence of cirrhosis or ascites is very limited so far. We describe a 59 year old female with ESRD status post renal transplant, on chronic immunosuppression for renal allograft rejection who was admitted with pleuritic chest pain that turned to be secondary to right sided pleural effusion. Further evaluation revealed *Escherichia coli* in both the blood and pleural fluid. There was no clinical or imaging evidence of pneumonia as well as cirrhosis or ascites. She was managed as a case of SBEM requiring drainage by chest tube. Management of SBEM in non-cirrhotic individuals usually requires drainage with chest tube as against patients with liver cirrhosis with hepatic hydro-thorax in whom chest tube drainage is contraindicated for risk of massive protein and electrolyte depletion and dehydration.

1. Case presentation

A 59 year old Caucasian female was admitted with complaints of sharp right sided chest pain aggravated with deep inspiration, and low grade fever of one day duration. There were no associated symptoms of cough, palpitations or shortness of breath.

Her past medical history was significant for ESRD secondary to chronic glomerulonephritis, status post renal transplant (1995) with chronic allo-graft rejection on immunosuppression for several years with regular intake of mycophenolate mofetil, and prednisone. She has been dialysis independent post-transplant, except for one time emergency dialysis about 6 months prior to this admission when she presented with acute kidney injury secondary to dehydration from diarrhea. At that time she had a temporary dialysis catheter placed into the right internal jugular vein that was removed later. Patient had no history alcohol abuse. Vitals signs were normal apart tachypnea. Oxygen saturation was adequate in room air. Systemic examination revealed dullness over the right lung field from middle zone to base, with decreased breath sounds. Bilateral pitting pedal edema was also seen. Abdomen was soft and non-distended with no shifting dullness. There was no palpable Organomegaly. Chest x ray showed blunting of the Right costo-phrenic angle consistent with effusion. Initial laboratory work showed blood count with leukocyte count of $6900 \cdot ^{9}$/L with 68% neutrophils and 30% lymphocytes. Blood chemistry showed normal electrolytes, BUN 32.85 mmol urea/L, Creatinine 540 μmol/L (baseline Creatinine 257.14–342.85 μmol/L). Liver function test showed AST 13Unit/L, ALT 7Unit/L, ALP 86 Unit/L, Total protein 55 g/L, albumin 29.14 g/L. Serum LDH was 210 Int Unit/L. ESR (Erythrocyte Sedimentation Rate and CRP (C-reactive Protein) levels, initially on admission were elevated at 107 mm/hr and 147.4 mg/dl respectively. Blood cultures were positive for gram negative rods from two different sites, later identified as *Escherichia coli*. Urine analysis and culture was negative for Urinary Tract Infection. The patient was initially started on intravenous meropenem for broad spectrum coverage (as she was allergic to penicillin/fluoro-quinolone), with renal dose adjustment, which was later changed to ertapenem as per microbiological sensitivity pattern of the culture. All immunosuppressive medications were stopped with the exception of prednisone.

She underwent right sided thoracentesis on the same day of admission and 500 cc of cloudy fluid was withdrawn. Pleural fluid analysis was consistent with exudative characteristics (Table 1), and the culture of the exudate was positive for *Escherichia coli*. ESR and CRP levels trended down on day 3 of admission after the initiation of intravenous antibiotics and thoracentesis. ESR decreased from 107 to 93 mm/hr and CRP from 147.4 to 79.6 mg/

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Table 1
Pleural fluid characteristics.

Appearance	Yellow, hazy
PH	7.28
Glucose	4.54 mmol/L
Protein	<20 g/L
WBC	$51{,}450 \cdot ^{9}$/L
Neutrophils	95%
LDH[a]	436 Int Unit/L
Culture	*Escherichia coli*

LDH-Lactate Dehydrogenase.
[a] Meets Light's criteria for exudative pleural effusion (Pleural fluid LDH/Serum LDH-2.07).

dl. However serial ESR/CRP measures were not done as the patient was already showing clinical improvement with appropriate management.

CT scan of the Chest was done after thoracentesis (Fig. 1) for any source of infection, and to assess for chest tube drainage.

CT scan showed bilateral moderate-sized pleural effusion, with right side more than left side. There was no parenchymal disease or features of consolidation on the CT chest. CT scan of the upper abdomen showed normal liver and spleen characteristics with bilateral atrophic kidneys. No ascites was noted on the CT scan of the abdomen. Echocardiogram done 5 months prior to the admission showed normal ejection fraction (>70%), with mild concentric left ventricular hypertrophy. There was no wall motion abnormality.

The patient ultimately had a chest tube drain placed on the right side of the chest to drain the empyema. Chest tube was removed after one week, and intravenous antibiotics were given for a total duration of 8 days. The patient showed clinical improvement, with negative repeat blood cultures. She was transitioned to oral trimethoprim-sulfamethoxazole upon discharge. A permanent catheter was inserted into the Left Internal Jugular Vein for the possible need for long term dialysis. She required 4 sessions of hemodialysis during this admission, and was referred for outpatient hemodialysis upon discharge as she had progressive renal failure.

2. Discussion

Infection of the pleural fluid in the absence of pneumonia is a rare event. It is usually denoted by the term Spontaneous bacterial Empyema (SBEM). SBEM criteria include transudative pleural fluid in the absence of pulmonary source of infection, with polymorphs> 500 cell/mm 3 or positive pleural fluid culture [1]. Several cases of spontaneous bacterial empyema have been described in the past. Almost all cases described in literature are seen in patients with ascites secondary to liver cirrhosis [1–5]. Lower levels of complement C3 (with resultant lower opsonic activity) in the pleural fluid of patients with hepatic hydrothorax and a higher Child-Pugh score are considered to be contributing factors for SBEM [3,5,6]. In a recently published article in CHEST (May, 2015) it was proposed that the term "secondary pleuritis of cirrhosis" be used instead of SBEM as it is not a spontaneous event and is usually due to migration of the infected peritoneal fluid through the defects in the diaphragm [3]. In our case report, we describe a case of spontaneous empyema which occurred in the absence of ascites or liver cirrhosis.

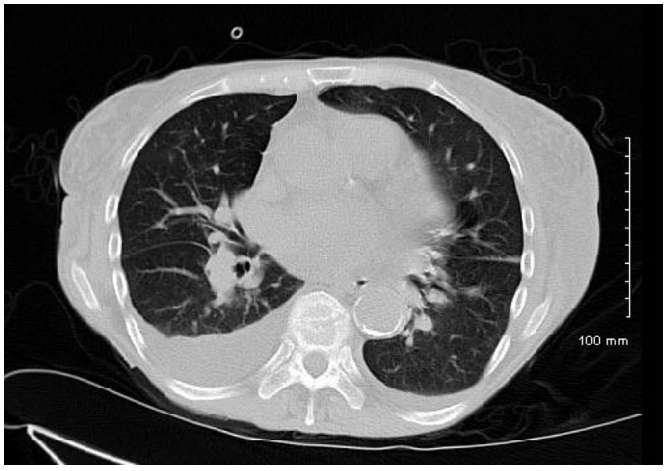

Fig. 1. CT Chest post thoracentesis showing bilateral pleural effusion, right more than left side.

2.1. SBEM in a background of liver cirrhosis

Occurrence of SBEM in cirrhotic patients is well established in literature. In a large prospective study on SBEM which included 120 cirrhotic patients with hydrothorax, about 13% developed SBEM [4]. In majority of the cases (>50%), SBEM was associated with Spontaneous Bacterial Peritonitis (SBP) [1,3,4]. In a retrospective study involving 11 cases of SBEM in cirrhotic patients by Xiol et al., [1] in four out of the six cases with culture positive empyema, the same organism was grown in both ascitic and pleural culture. Most commonly isolated bacteria from SBEM in cirrhotic patients include *Escherichia coli*, enterococcus and klebsiella species, suggesting Gastro Intestinal source [1,2,4]. This again emphasizes the concept of migration of the infected ascitic fluid as a cause of the empyema. However literature on SBEM in non-cirrhotic/ascitic patients is very limited.

2.2. Empyema in chronic kidney disease/end stage renal disease

In a retrospective analysis of empyema cases in 124 patients with either CKD Stage 4 or ESRD, an infective source was identified in over 82% of the cases, with pneumonia and catheter based infections occupying the top 2 causes [7]. In the same study, it was found that empyema culture in patients with ESRD showed a predominance of aerobic gram-positive organisms (54%), while in CKD stage 4 patients, aerobic gram-negative organisms (67%) were the predominant isolates. They described that the predominance of gram positive infection in ESRD patients was secondary to the dialysis catheter. We could not find any source of infection in our patient, and she did not have any dialysis catheter upon presentation to the hospital. The last time she had any central venous catheter was 6 months prior to this admission for emergency dialysis, and it was removed immediately after the dialysis.

Incidence of SBEM in non-cirrhotic individuals is very rare. Upon review of literature, we found only few cases of spontaneous empyema reported in the absence of ascites or liver cirrhosis [8,9] In the case report by Chen et al. [8] *Escherichia coli* and Aeromonas hydrophila were isolated in the pleural fluid, and blood. Hematogenous spread was coming from transient bacteremia from Gastrointestinal tract was thought to be the mechanism of the spontaneous empyema. Nguyen et al. [9] reported a case of spontaneous empyema from streptococcal pneumonia bacteremia in a patient with uncontrolled Diabetes Mellitus and Dialysis dependent renal disease [9]. In both these instances the patients were non cirrhotic and had no ascites. Immuno-compromised state is thought to be contributing factor in both these cases. Both of these patients required management by chest tube thoracostomy.

In our patient, there was no evidence of cirrhosis or ascites or pneumonia based on clinical findings and imaging. The pleural effusion was thought secondary to fluid overload status from

ESRD. The pleural fluid was infected with *Escherichia coli*, and it was exudative in nature based on Light's LDH criteria (pleural LDH: serum LDH (ratio was 2.07). The protein content in the pleural fluid was <2 g/dl and the pleural fluid protein: serum protein (ratio) was <0.36. Her risk factors for infection included immunosuppressive agents for the history of allograft transplantation. The most likely explanation for *Escherichia coli* bacteremia is translocation from Gastrointestinal tract, and the mechanism of infection of pleural fluid is through hematogenous spread. From the above explanation, our patient represents a unique case, where the infection of the pleural fluid is spontaneous (not contiguous with peritoneal fluid infection, absence of pneumonia). We therefore suggest that as stated in the recent article in CHEST [2], the term "secondary pleuritis of cirrhosis" could be used for those cases of non-pneumonia empyema associated with cirrhosis, while the term "spontaneous bacterial empyema" could be used to refer these rare cases of infected pleural fluid, in the absence of pneumonia or liver cirrhosis with ascites.

Management of SBEM has to be individualized on case to case basis. As most of the cases of SBEM associated with liver cirrhosis are transudative in nature [2,4,5], antibiotic treatment is the preferred approach. Also chest tube drainage is contraindicated in hepatic hydrothorax due to concerns of fluid and protein loss and electrolyte imbalance which can be life threatening [10,11]. However, as in our case where SBEM is not secondary to liver cirrhosis with ascites and if consistent with Light's exudative criteria, it has to be managed similar to empyema secondary to pneumonia and a chest tube thoracostomy is warranted.

2.3. Conclusion

We report a case of empyema that occurred spontaneously with no preceding pneumonia or associated liver cirrhosis and ascites. That is considered to be of rare incidence. The presence of co-existent immune-suppression is usually expected in spontaneous bacterial empyema.

Disclosure

No conflict of interest regarding that case report.

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