Acute Pyelonephritis Associated with Transudative Pleural Effusion in a Middle-Aged Woman without Urinary Tract Obstruction

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Acute pyelonephritis · Pleural effusion · Bacteremia

Abstract
Objective: To describe a case of acute pyelonephritis associated with pleural effusion. Clinical Presentation and Intervention: A 39-year-old female non-smoker who had Escherichia coli bacteremia due to acute pyelonephritis, developed bilateral transudative pleural effusions during hospitalization. She was successfully treated with intravenous antibiotic therapy. Follow-up chest radiographs revealed complete resolution of the bilateral pleural effusions. Conclusion: Though quite rare, pleural effusion is a potential complication of acute pyelonephritis. The exact pathogenesis of transudative pleural effusion is unknown, but the effusion may resolve spontaneously when infection is adequately controlled.

Introduction
The pleural space lies between the lung and chest wall and normally contains a very thin layer of fluid, which serves as a coupling system. A pleural effusion is present when there is an excess quantity of fluid in the pleural space. Many diseases or disorders may lead to the development of pleural effusion. The first step of diagnostic procedure for a pleural effusion is to determine whether the effusion is a transudate or an exudate. An exudative pleural effusion occurs when local factors that influence the formation and absorption of pleural fluid are altered. The leading causes of exudative pleural effusions are bacterial pneumonia, malignancy, tuberculosis, and pulmonary embolism. A transudative pleural effusion occurs when systemic factors that influence the formation and absorption of pleural fluid are altered. The most common causes of transudative pleural effusions are congestive heart failure, liver cirrhosis with ascites, and nephrotic syndrome [1]. A variety of intra-abdominal or pelvic disorders, such as renal and/or perirenal abscesses [2] and urinary tract obstruction [3–7] are associated with pleural effusion. Pleural effusion associated with acute pyelonephritis, however, is uncommon, with only a few cases, mostly in pregnant women, being reported [8–10]. Only two pleural effusion specimens from patients with this association were ever studied; one transudative in a pregnant woman and the other exudative in a non-pregnant woman [9, 10]. Because of the rarity of this association, we reported a 39-year-old female who contracted bacteremia of Escherichia coli due to right-sided acute pyelonephritis without urinary tract obstruction and developed bilateral moderate amounts of transudative pleural effusion.
Case Report

A 39-year-old married female (gravida 2, para 2) was admitted to our hospital with a chief complaint of fever for 3 days. Three days before admission, she began experiencing right flank pain, dysuria, fever, chills, nausea, and vomiting. Her last menstrual period was 4 days before admission. She had a history of gallbladder polyps without surgical treatment. Frequent cystitis (2–3 episodes annually) had been noted for 1 year prior to admission. No other past medical history was remarkable. On admission, body temperature was 39.2 °C, pulse rate 116/min, respiratory rate 20/min, and blood pressure 129/85 mm Hg. Breathing sound was clear, and no abnormal dullness was evident by percussion. There was marked tenderness over the right costovertebral area.

Laboratory examination revealed a white blood cell (WBC) count of 11,600/mm³ (normal range 3,500–11,000/mm³), with a differential count of 84% segments, 1% bands, 22% lymphocytes, 2% atypical lymphocytes, 11% monocytes, 3% eosinophils, 1% metamyelocytes, and 1% myelocytes. Hemoglobin was 11.3 g/dl (normal range 12.0–16.0 g/dl), mean cell volume 86.5 μm³, mean cell hemoglobin 28.3 pg/cell, and mean cell hemoglobin concentration 32.8 g/dl. Urinalysis was negative for proteinuria, but revealed 33–35 WBCs and 8–10 red blood cells (RBCs) per high-power field. Serum creatinine was 0.9 mg/dl (normal range 0.4–1.4 mg/dl), and alanine aminotransferase 11 IU/l (normal range 0–36 IU/l). A chest radiograph taken 1 month previously and a current x-ray of KUB were unremarkable. Renal sonography showed a normal renal size without parenchymal change.

Intravenous antibiotic therapy with parenteral 1 g of cefazolin and 60 mg of gentamicin every 8 h/day was initiated immediately upon admission. By the 3rd day of hospitalization, blood cultures and urine culture yielded *E. coli* resistant to gentamicin, therefore, the antibiotic regimen was changed to parenteral 1 g of cefazolin every 8 h/day and 300 mg of amikacin every 12 h/day. During the 5th day of hospitalization, the patient began to suffer shortness of breath and chest tightness. Physical examination revealed no sign of fluid overload and decreased breathing sound in the bilateral lower lung fields. Chest radiograph (fig. 1) and sonography revealed bilateral pleural effusions. Thoracentesis yielded a light yellow, clear, non-viscous, odorless fluid. Effusion analysis revealed: glucose 109 mg/dl, total protein 2.6 g/dl, lactate dehydrogenase (LDH) 17 U/l, RBC 1,900/mm³, and WBC 250/mm³ with 86% lymphocytes and 14% neutrophils. Serum total protein was 6.2 g/dl (normal range 6.3–8.0 g/dl), albumin 3.5 g/dl (normal range 3.5–5.5 g/dl), and LDH 88 IU/l (normal range 47–140 IU/l). The fever subsided on the 8th day of hospitalization. The dyspnea and symptoms of chest discomfort improved. The patient was discharged on the 11th day of hospitalization. Follow-up of chest radiograph on the 7th day after discharge revealed the complete resolution of bilateral pleural effusions (fig. 2).

Discussion

Transudative pleural effusions are usually caused by an alteration in the hydrostatic and oncotic pressure distribution across the pleura, and are characterized by low cell and protein contents. Congestive heart failure is the most common cause of transudative effusion. The fluid that accumulates in hepatic hydrothorax, urinothorax during peritoneal dialysis and in many patients with nephrotic syndrome may also have the characteristics of a transudate. The development of a transudative effusion...
indicates that the pleural membranes per se are intact; therefore, if the underlying problem can be resolved, the effusion will be reabsorbed. Transudative pleural effusion may also develop in cases of hypothyroidism or pulmonary embolism, probably because of the vascular permeability abnormality. A variety of other intra-abdominal diseases may also be associated with pleural effusion, and the pathogenesis is complex, varying according to the particular disease examined. An intra-abdominal abscess close to the diaphragm, for example, may cause inflammation of the diaphragm and increased capillary permeability and fluid leakage into the pleural cavity.

Pulmonary complications occur in up to 20% of renal and/or perirenal abscesses, and associated findings of chest radiographs often include an elevated or fixed hemidiaphragm, pleural effusion, empyema, lung abscess, lower lobe infiltrates, or atelectasis [2]. Furthermore, nephrobronchial fistula is an uncommon complication of perirenal abscesses [11]. In these cases, however, the effusion should have exudate characteristics.

The association of urinary tract obstruction and pleural effusion has been shown in many reports [3–7]. Most published reports attributed the source of fluid collection in the thorax to retroperitoneal collections of urine (urinoma), developing from extravasation of urine from the urinary tract obstruction. Possible mechanisms for retroperitoneal urinomas extending into the thorax include lymphatic drainage of the extravasated urine [5], dissection through the retroperitoneum, and rupturing into the pleural space [6, 7]. An elevated pleural fluid creatinine concentration (which is higher than serum creatinine) is specific for the diagnosis of urinotorax [3, 4, 7]. The fluid of urinotorax may be transudative in nature [3]. Few reported cases of pyelonephritis associated with pleural effusion have been reported [8–10], mostly occurring in pregnant women. There is evidence that pregnancy enhances organ system damage caused by endotoxemia. For example, the generation of Schwartzman reaction evoked by endotoxin does not require a preparatory dose during pregnancy [12]. There is only one case report of acute pyelonephritis associated with exudative pleural effusion in a non-pregnant patient [10], however, urinotorax rather than pyelonephritis may in fact be the correct diagnosis [13].

Our patient with acute pyelonephritis and E. coli bacteremia developed bilateral transudative pleural effusions without evidence of pulmonary parenchymal disease or urinary tract obstruction. Her menstrual cycle was regular and normal, and her last menstrual period was only 4 days before hospitalization. Although the creatinine concentration of pleural fluid was not measured, urinotorax is unlikely because of lack of urinary tract obstruction. The exact pathogenesis of transudative pleural effusion associated with simple pyelonephritis in this case is yet uncertain. Probable causes may include endotoxemia resulting in pulmonary injury, and/or cytokines released in inflammatory response leading to an increase in the permeability of vessels.

### Conclusion

Though quite rare, pleural effusion is a potential complication of acute pyelonephritis, but the effusion may resolve spontaneously when infection is adequately controlled.

### References