

Pleural effusion in a patient with end-stage renal disease

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Pleural disease is a common problem in patients with chronic renal insufficiency. In 1836, Bright reported that only 29% of patients with albuminous urine had healthy pleura at autopsy.¹ There are several reasons why pleural disease may be common in patients with renal failure. These include congestive heart failure, an increased risk of infection, the presence of diseases associated with renal and pleural manifestations (eg, systemic lupus erythematosus), uremic pericarditis, an increased risk for certain malignancies and pulmonary embolism. Uremic pleurisy results from an unknown putative agent, and therefore uremic pleuritis is a diagnosis of exclusion.

Case

A 45-year-old Saudi male with end stage renal disease (ESRD) for more than 16 years presented with shortness of breath and right-sided pleuritic chest pain for more than 3 days and cough with whitish sputum for more than 1 month, without fever. He also reported weight loss of more than 10 kg in 4 months. The patient was on hemodialysis 3 times per week, had hepatitis C virus disease, and was recently found to have hyperthy-

roidism. He was also a smoker. The patient was taking atenolol 50 mg orally once daily, methimazole 10 mg orally once daily, erythropoietin 2000 units subcutaneously per week, calcitriol 0.25 mg orally once daily, and aluminum hydroxide 400 orally three times per day.

Initial laboratory tests are shown in Table 1. Pleural fluid cultures for bacteria and mycobacteria were negative. Chest radiography and computed topography showed bilateral pleural effusion right more than left, no pulmonary infiltrates, and no evidence of infection or infarction. The patient underwent thoroscopic pleural biopsy (Figure 1), which showed a layer of fibrin with underlying reactive mesothelial cells and granulation tissue, which was consistent with fibrinous pleuritis. Subsequently the patient's hemodialysis regimen was intensified, with gradual improvement in symptoms and complete resolution of his pleural effusions. There was no recurrence on follow up over the following year.

Table 1: Laboratory investigations.

Serum	
White blood cells	14.7 x 10 ⁹ /L
Hemoglobin	99 x 10 ¹² /L
Protein	82 g/L
Lactic dehydrogenase	462 g/L
Urea	19.5 µmol/L
Creatinine	587 mmol/L
CO ₂	29 mmol/L
Pleural	
White blood cells	673 x 10 ⁶ /L
Red blood cells	960 x 10 ⁶ /L
Histocytes	58%
Polymorphs	20%
Lymphocytes	12%
Eosinophils	11%
Lactic dehydrogenase	315 u/L
Protein	39 g/L
Glucose	5.9 mmol/L
LDH ratio	0.68

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Accepted for publication

May 2005

Ann Saudi Med 2006;26(2):145-146

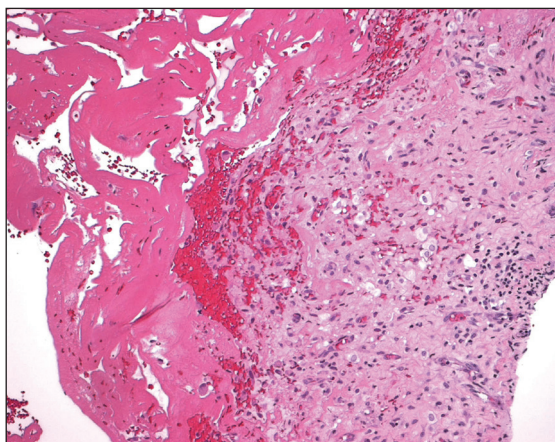


Figure 1. Thorascopic pleural biopsy (intermediate magnification).

Discussion

Berger and coworkers² reported a 4% (14 of 436) incidence of uremic pleural effusions in chronic renal failure. However, the incidence could have been underestimated because patients who had little effusion or who resolved rapidly after detection were not included. Jarratt and Sahn³ reported a 16% (16 of 100) incidence of pleural effusion secondary to uremia.

The typical patient with uremic pleural effusion has been undergoing hemodialysis or peritoneal dialysis for one or two years, with a range of a few months, to greater than four years. Patients usually but not always have symptoms at the onset of effusion, with fever, cough, or chest pain. Dyspnea is an unusual presenting manifestation. Pericardial and pleural rub are heard in 30% to 50% of patients. The pleural rub usually persists for two or three days. Nidus et al⁴ found that the incidence of pleural rub is about 15% in patients with severe renal insufficiency. Only one rub was observed to at least 18 days, but others were of brief duration.

The chest radiograph typically shows a unilateral moderate-to-large pleural effusion without accompanying pulmonary infiltrates. Thoracentesis most

often reveals a serosanguinous to bloody exudate. Berger et al² found serosanguinous or hemorrhagic fluid in 10 of 14 patients. The nucleated cell count ranges from 80 to 3700/mm³ with lymphocyte predominance (averaging 70% of the cells). Only a few cases of pleural fluid eosinophilia have been reported. Pleural eosinophilia rates of 86% and 10%⁴ have been noted previously. The erythrocyte count of pleural fluid was an average of 146 140/mm³ (range, 0.6-0.12 x10⁶). Pleural fluid glucose approximates serum glucose and pleural fluid acidosis has not been observed. The creatinine concentration is high, reflecting the blood creatinine but the pleural fluid to serum fluid is less than 1.0. Berger et al² found that all patients with uremic pleural effusion who had pleural biopsy or autopsy evidence of fibrinous pleuritis had high values for pleural protein and LDH.

Pleural effusion generally resolves with continued dialysis over several weeks, although some may later recur. Some patients will progress to fibrothorax, resulting in severe pulmonary restriction. The occurrence of fibrous pleuritis in uremic effusion may have several explanations. The pleural fluid is hemorrhagic and this in itself may produce fibrothorax.⁵ Uremic toxins theoretically can be implicated since there may be exudation of protein and extensive fibrin deposition on serous membranes. Systemic heparinization during dialysis may contribute to the development of hemorrhagic effusion.⁶

Despite an abnormal bleeding tendency that exists in patients with renal insufficiency, decortication of the chest wall and the lung can be carried out safely with minimal bleeding and restoration of pulmonary function.⁷ The question of whether one can halt the progression of this process remains unanswered. One group of investigators has suggested using regional heparinization during dialysis to decrease bleeding into the pleural space.⁸ Installation of non-absorbable corticosteroids into the pleural space might be beneficial but has not been tried.⁹ Surgery seems to be the ultimate recourse if severe incarceration occurs.

References

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