

A Dramatic Presentation of Pulmonary Edema Due to Renal Failure

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INTRODUCTION

Pulmonary edema is a common cause of dyspnea and a leading reason for inpatient admission. Pulmonary edema results from accumulation of fluid or proteins in the alveoli, resulting in a ventilation perfusion mismatch, hence resulting in shortness of breath.¹ The principal mechanisms underlying formation of pulmonary edema are transudation of protein poor fluid into the pulmonary interstitial and alveolar spaces, as happens in cardiogenic pulmonary edema; and increased permeability of the pulmonary endothelium as happens in non-cardiogenic causes such as acute respiratory distress syndrome (ARDS).¹⁻³

Pulmonary edema etiology can be divided into cardiogenic and non-cardiogenic classes. By far, heart diseases are the leading cause of pulmonary edema in inpatient services.² Non-cardiogenic pulmonary edema commonly is associated with acute respiratory distress syndrome and pulmonary embolism.⁴ Distant organ dysfunction such as renal failure is emerging as a cause of pulmonary edema, especially with widespread access to hemodialysis services and enhanced survival.⁵ Noncompliance with this lifesaving procedure, as is common with many medical prescriptions, is the leading reason for this association. This case report detailed a dramatic presentation of pulmonary edema due to noncompliance with hemodialysis.

CASE REPORT

A 50-year-old, African American male with a past medical history of end stage renal disease (ERSD) on hemodialysis presented to the emergency department with shortness of breath, exertional dyspnea (New York Heart Association Class IV), weight gain, bilateral leg edema, and new-onset hemoptysis. He reported to have missed a hemodialysis session, making his last session about seven days prior to presentation. He denied fever and recent flu-like illness. He had no previous history of heart disease or congestive heart failure. On presentation, he was sickly, but with a normal temperature and heart rate. He had tachypnea with a respiratory rate of 22 breaths per minute and elevated blood pressure of 197/93 mmHg. He had pulse oximetry reading of 64% on room air. He was placed on 15 L of oxygen on a non-rebreather mask. Physical examination revealed edema, jugular venous distention, and an S3 gallop on cardiac auscultation. Pulmonary auscultation revealed crackles in the lower lung fields bilaterally, decreased aeration in the upper lung fields bilaterally, and diffuse expiratory wheezes. The patient had anasarca and bilateral lower extremity pitting edema. Significant blood was seen on examination of the patient's sputum.

Noteworthy laboratory findings included a white blood cell count of $17.2 \times 10^3/L$, hemoglobin 14.6 g/dL, blood urea nitrogen 61 mg/dL,

serum creatinine 10.9 mg/dL, procalcitonin 5.26 ng/mL, brain natriuretic peptide (BNP) 480 pg/mL, and lactic acid 4.3 mmol/L. The admission chest x-ray (Figure 1) and a computerized tomography (CT) angiogram (Figure 2) ruled out pulmonary emboli but revealed multi-lobar airspace opacities. A 2-D transthoracic echocardiogram revealed an ejection fraction of 65%, grade 2 diastolic dysfunction, and pulmonary artery pressure was estimated at 90 mmHg. There were no valve regurgitations or stenosis noted on Doppler echo.

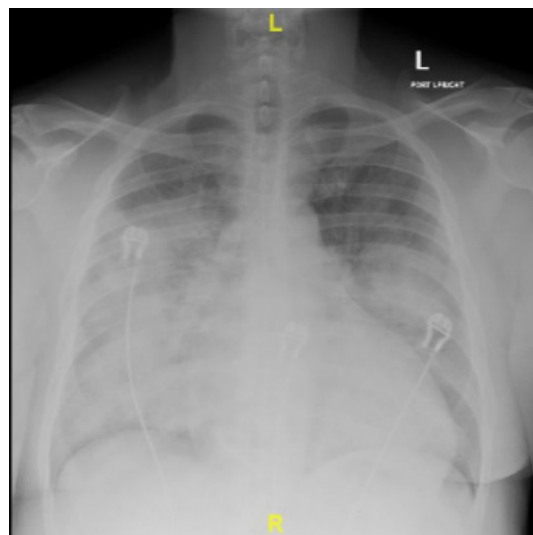


Figure 1. Admission chest x-ray showed cardiomegaly and bilateral pulmonary alveolar infiltrates.

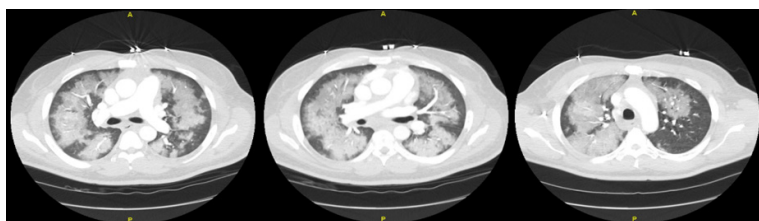


Figure 2. Admission CT angiogram showed diffuse multi-lobar airspace opacities.

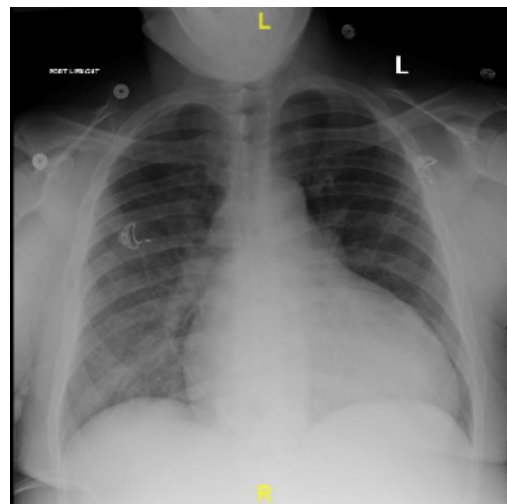


Figure 3. Day 3 chest x-ray showed stable cardiomegaly and improvement of pulmonary infiltrates after two days of hemodialysis.

The patient was started on antibiotics for possible health-care-associated pneumonia and received hemodialysis for three consecutive days. His hemoptysis resolved after the first session of hemodialysis and, after two days of dialysis treatment, his physical exam was improved with resolved edema, jugular venous distension, and crackles on lung auscultation. A repeat chest x-ray was done on Day 3 of admission (Figure 3), which showed significant improvement in the pulmonary infiltrates.

DISCUSSION

Pulmonary edema results from fluid or protein accumulation in the alveoli, largely from processes that alter the Starling forces, changing the net flow of liquids across a membrane.¹⁻³ The distinction between cardiogenic and non-cardiogenic pulmonary edema often is difficult. History and examination are often similar and measurement of pulmonary artery pressure of less than 25 mmHg on echocardiography and right heart catheterization measurement of the pulmonary capillary wedge pressure level less than 18 mmHg predicts non-cardiogenic causes.

Non-cardiogenic pulmonary edema is often secondary to acute respiratory distress syndrome, high altitude, opioid overdose, pulmonary embolism, and in our case, renal failure.^{5,6} Accumulation of fluid in the lungs among renal failure patients results from down regulation of the epithelial salt-water transporters such as ENaC, sodium-potassium ATPase and aquaporin-5 in the lung. These transporters are responsible for sodium absorption from the alveolar cavity into the alveolar epithelium cells, with water following passively.⁵ Additionally, accumulation of inflammatory cytokines (such as interleukins IL-6, IL-8, IL-1 β), tumor necrosis factor α , macrophage inflammatory protein 2, nuclear factor- κ B, chemokines, and activated innate immune cells in acute and chronic kidney injury have been theorized to provoke and initiate pathological cascades that leads to acute lung injury and ARDS.⁷

Patients with pulmonary edema generally present with dyspnea, bilateral lower-extremity edema, and chest radiographs showing bilateral alveolar filling patterns.⁴ Hemoptysis, as in the above case, is an uncommon presenting symptom. The management of pulmonary edema involves treatment of the underlying disease and supportive measures, such as mechanical ventilation, maintenance of adequate nutrition, and lowering the pulmonary artery wedge pressure with diuretics, ultrafiltration (during hemodialysis), and fluid restriction.

Since our patient had severe hypertension and normal ejection fraction, it is possible diastolic dysfunction contributed at least in part to the pulmonary edema. Moreover, our patient had no mitral stenosis or regurgitation. However, the dramatic improvement in his symptoms following hemodialysis implied, at least to some degree, that the pulmonary edema was volume dependent in the setting of hemodialysis dependent end-stage renal failure.

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Keywords: pulmonary edema, hemoptysis, renal failure