Cavitory Lung Lesion in a Patient With Congestive Heart Failure*

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(CHEST 1996; 110:276-78)

A 25-year-old white woman was admitted to the coronary ICU because of shortness of breath, orthopnea, fatigue, and bilateral ankle swelling. She also reported experiencing intermittent left-sided chest discomfort that was aggravated by deep breathing. There had been no history of fever, chills, night sweats, productive cough, or hemoptysis. Her medical history was noteworthy for refractory congestive heart failure due to dilated cardiomyopathy that had developed after a term delivery 9 months previously. She was treated with digoxin, furosemide, hydralazine, and nitrates with partial control of her congestive symptoms. She had since required admission to hospital twice for worsening heart failure which was managed with short courses of IV dobutamine and diuresis. There was no history of illicit drug use, seizure disorder, or Mycobacterium tuberculosis exposure. Her family history was remarkable for dilated cardiomyopathy in her mother and sudden death in her aunt.

On examination, her blood pressure was 116/70 mm Hg, pulse 141/min, respiratory rate was 24/min, and her temperature was 38.2°C. Auscultation of the chest revealed basilar crackles on the right and bronchial breath sounds at the left base. Findings on cardiac examination included 12-cm jugular venous distention, right ventricular lift, and S3 gallop. There was 3+ pitting pretibial edema. The remainder of the physical examination was unremarkable.

Chest radiograph demonstrated an enlarged heart, pulmonary vascular congestion, and a left lower lobe infiltrate with a small pleural effusion. A pulmonary artery catheter was inserted which revealed a pulmonary artery pressure of 48/30 mm Hg and a pulmonary capillary wedge pressure of 22 mm Hg.

Pertinent laboratory data were as follows: hemoglobin 9.7 g/dL, WBC count 16,600/mL with normal differential, platelet count 524,000/mL, prothrombin time 14.8 s, and activated partial thromboplastin time 23 s. Routine blood chemistry and cardiac enzymes were within normal limits. Arterial blood gases on 2 L of oxygen were pH 7.52, PaCO2 43 mm Hg, PaO2 122 mm Hg, and oxygen saturation 98%. Electrocardiogram revealed sinus tachycardia with signs of left atrial enlargement.

Induced sputum Gram’s stain showed few polymorphonuclear leukocytes and few Gram-positive cocci, and culture revealed commensal respiratory flora. Repeated blood cultures failed to yield any growth. Fungal and HIV serologic tests were negative. Tuberculin skin tests and anergy panel were nonreactive. An ultrasound-guided thoracentesis disclosed bloody pleural fluid. Gram’s stain and aerobic and anaerobic cultures of the pleural fluid were negative. While she was being treated for acute exacerbation of heart failure with dobutamine, furosemide, nitrates, and hydralazine, the patient was placed on mezlocillin and tobramycin for a presumed left lower lobe pneumonia. Despite the above treatment, her dyspnea, chest pain, and fever persisted.

Follow-up chest radiograph revealed development of hyperlucent areas within the left lower lobe of the

FIGURE 1. Anteroposterior view of the chest showing left lower lobe infiltrate with air-filled cavities and marked cardiac enlargement.

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FIGURE 2. CT of the chest showing air-filled cavities within the consolidated left lower lobe.

lung (Fig 1). The CT study of the chest revealed multiple air-filled cavities in the left lower lobe and a possible clot in the left descending pulmonary artery and left ventricle (Fig 2). Pulmonary arteriogram confirmed the presence of a large clot in the left lower lobe pulmonary artery with absent distal blood flow (Fig 3). An echocardiogram showed markedly diminished left ventricular function, estimated ejection fraction of 20%, left ventricular thrombus, and no pericardial effusion or valvular vegetations.

IV heparin was initiated and antibiotics were stopped shortly thereafter (total 10-day course). Prompt resolution of fever, leukocytosis, and chest pain occurred although radiographic clearing was sluggish. She was discharged home on warfarin and IV dobutamine therapy.

FIGURE 3. Pulmonary arteriography demonstrating complete occlusion of the pulmonary artery branch to the left lower lobe with meniscus sign suggesting acute thromboembolism (arrow). Arterial perfusion is confined to the left upper lobe.
Diagnosis: Pulmonary Embolus With Cavitory Infarction

The occurrence of an infarction following pulmonary thromboembolism is uncommon. Autopsy series revealed that fewer than 10% of pulmonary emboli resulted in infarction.\(^1\) The infrequency of pulmonary infarcts has been attributed to the collateral flow from bronchial arteries and extensive anastomosis in the pulmonary capillary bed.\(^2\) In both animals and humans, it has been demonstrated that pulmonary infarction is more likely to occur if the pulmonary venous pressure is raised.\(^3\)\(^,\)\(^4\) Therefore, while under most circumstances the lung is well protected against ischemic injury from thromboembolism, coexisting cardiopulmonary disease (eg, left ventricular failure with pulmonary congestion) can predispose to the development of infarction.

Pulmonary infarction is typically a coagulative necrosis process that heals with minimal scarring. However, large infarcts may undergo liquefactive necrosis with cavitory formation. Although cavitating pulmonary infarcts (CPI) are usually associated with septic thromboembolism, aseptic liquefaction of pulmonary infarcts has been rarely reported.\(^4\)\(^,\)\(^7\)\(^,\)\(^11\) Levin et al\(^8\) in a study of 550 cases of bland pulmonary infarcts found sterile cavitation in 23 cases (4.2%). Factors that predispose a bland infarct to cavitate have been addressed in several previous reports.\(^7\)\(^,\)\(^11\) Both size and location of the infarct have been suggested to contribute to the development of cavitation. Greico and Ryan\(^7\) noted that CPIs were found only in infarcts greater than 4 cm in diameter. Wilson et al\(^8\) reporting on aseptic cavitation of bland pulmonary infarcts also noted that infarcts larger than 4x4 cm were more likely to undergo cavitation. They noted that aseptic CPIs predominantly involve the upper- and mid-lung zones although 20% of their cases did involve the lower lobes.\(^8\) The contribution of positive pressure ventilation to development of cavitation in the setting of pulmonary infarction has also been demonstrated and ascribed to the effects of barotrauma.\(^9\)\(^,\)\(^10\) In the series by Libby et al\(^11\) of 10 cases of cavitary pulmonary infarction, seven had been treated with mechanical ventilation.

In addition to sterile CPIs just described, large pulmonary infarcts may also cavitate secondary to bacterial super-infection either via hematogenous seeding or tracheobronchial spread. Infected vs sterile CPIs seem to occur with equal frequency; however, distinguishing between the two is often difficult. Although some authors have suggested that presence of fever, leukocytosis, and purulent sputum is a clinical hallmark of infected CPIs, bland pulmonary infarcts too may be attended by fever and leukocytosis. Classification is further hampered by the fact that many of such patients receive empiric antibiotic therapy initially for presumed pneumonia making subsequent retrospective diagnosis of sterile CPI difficult.

The case presented herein demonstrates the challenge of making a diagnosis of pulmonary infarct with cavitation based on presenting clinical and radiographic findings. At initial presentation, considering the patient's low-grade fever and leukocytosis, pneumonia was considered foremost. Later, unremarkable microbiologic work up and lack of clinical response to antibiotics coupled with her high embolic risk in the setting of acute cardiac decompensation raised the suspicion of pulmonary embolism that was confirmed by pulmonary arteriography. The absence of purulent sputum as well as negative blood, sputum, and pleural fluid cultures made the diagnosis of infected CPI less likely. The large infarct size was consistent with the subsequent development of cavitation that occurred.

Pulmonary infarction with cavitation is a rare complication of bland venous thromboembolism which has received little attention in the literature. It tends to occur in patients with limited cardiac reserve who can least tolerate the hemodynamic compromise imposed by unrecognized and untreated pulmonary embolism. In the appropriate clinical setting, regardless of the presence or absence of fever and leukocytosis, cavitation of pulmonary infarct should be included in the differential diagnosis of cavitary lung lesions.

REFERENCES

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