



Bloody Pleural Effusion In Septic Pulmonary Emboli :A presentation of Right-Eided Endocarditis: A Report of Two Cases

Zohreh Aminzadeh^{1,2*}, Hamid Reza Behzad¹

¹ Infectious Diseases Research Centre, Shahid Beheshti Medical University, Tehran, IR Iran

² The University of Queensland, UQ Centre for Clinical Research, Herston, Brisbane, Australia

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ABSTRACT

Pulmonary embolism is the fourth leading cause of pleural effusion. Pleural effusion that results from a pulmonary embolus is usually small and unilateral. A pulmonary embolus is the most common cause of pleuritic chest pain and pleural effusion in patients aged under 40 years. The incidence, characteristics, and pathogenesis of pleural effusions in patients with right-sided endocarditis (RSE) are poorly defined. Possible mechanisms of pleural fluid formation in RSE include parapneumonic effusion, septic pulmonary emboli with or without infarction, and empyema. We report 2 cases of bloody pleural effusion, combined with septic pulmonary emboli and dyspnea, as the initial manifestation of right-sided endocarditis.

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► Implication for health policy/practice/research/medical education:

Septic pulmonary emboli, pleural effusion and right-sided endocarditis should be considered as a possible cause of any pulmonary symptoms in drug users. Paraembolic effusions usually begin to resolve within a few days after institution of anticoagulant therapy, although those that are associated with parenchymal infiltrates may resolve more slowly.

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1. Introduction

Pulmonary embolism is the fourth leading cause of pleural effusion. Possible reasons for pleural effusion that is caused by a pulmonary embolus are increased interstitial fluid in the lungs due to ischemia and the release of vasoactive cytokines. The most common cause of pleuritic chest pain and pleural effusion in patients aged under 40 years is a pulmonary embolus. Approximately 75 % of patients with pulmonary emboli and pleural effusion have pleuritic chest pain. Dyspnea is frequently disproportional to the size of the pleural effusion. Pleural fluid that is caused by pulmonary emboli is usually exudative

but is occasionally transudative (1). Pleural effusion that results from a pulmonary embolus is usually small (less than one-third of the hemithorax in 90 %) and unilateral (85 %), but occasionally, they reach more than half of the hemithorax (2, 3). It appears soon after the symptoms of thromboembolism begin and tend to reach maximum size very early in the course of the disorder (2, 3). Septic pulmonary emboli present with variable and often non-specific clinical and radiographic features (4). Occasional or regular use of illicit drugs can lead, not exceptionally, to severe respiratory complications that require rapid management (5). Although paraembolic effusions have classically been considered to be bloody exudates with a predominance of polymorphonuclear leukocytes, many such effusions have none of these characteristics (3, 6). Up to 25 % may be transudates, and the RBC count exceeds 100,000 per mm³ in fewer than 20 % of such effusions. The WBC may range from less than 100 to more than 50,000

* Corresponding author: Zohreh Aminzadeh, Infectious Diseases Research Centre, Loghman Hakim Hospital, Tehran, IR Iran. Tel: +9821 55411717, Tel: +9821 55411717, E-mail: zohrehaminzadeh@yahoo.com

cells per mm³. Characteristics of these effusions vary so widely that no diagnostic patterns are apparent (6). In addition to using illicit drugs, there are other possible predisposing factors, including immobilization, surgery, deep vein thrombosis, malignancy, and pulmonary embolism (7). In this report, we describe 2 drug users with a pulmonary embolus and bloody pleural effusion.

2. Cases History

2.1. Case I

A 33 years-old-man complaining of chest pain, dyspnea, and swelling of the lower extremities was admitted to our ward. He experienced a sudden onset of chest pain and dyspnea. Upon admission, the patient developed hemoptysis, and his dyspnea later aggravated. He had been an IV drug user for the past 5 years. He had no history of trauma, recent travel, or any recent illness. There was no history of coagulopathy or other heart diseases in his family. On physical examination, he was aware of his surroundings but was in respiratory distress at rest. His vital signs were as follows: oral temperature 37°C, pulse rate 104 beats per min, respiratory rate 20 per min, and blood pressure 110/70 mm Hg. Coarse crackle was heard at the base of his lungs. There was no appreciated murmur on the cardiac examination. Multiple subcutaneous abscesses were also detected in his extremities.

Chest x-rays showed bilateral patchy infiltration and bilateral pleural effusion but no cardiomegally. The CT scan showed multiple ill-defined nodules, with excavation in both lungs. Pleural effusion was also prominent in the right lung. In addition, segmental collapse of the right inferior lobe was observed. By transthoracic echocardiography, there was a vegetation (15 mm in diameter) in the tricuspid valve, whereas transthoracic echocardiography showed no evidence of vegetation. A pleural tap analysis showed the following: red color, RBC 400,000/dL, WBC 4500/dL containing 75 % PMNs and 25 % Lym, protein 3.2 gr/dL, LDH 1449 mg/dL, glucose 59 mg/dL, pH 7.29, and negative culture. The blood culture revealed methicillin-resistant *Staphylococcus aureus*.

2.2. Case II

A 28 years-old-man complaining of fever, chills, weakness, and productive cough for 20 days and exacerbation of the cough for the past 3 days was admitted to our ward. He had been using an inhaled form of crack but was not an intravenous drug user. On physical examination, he was aware of his surroundings and appeared ill and intoxicated. His vital signs were as follows: oral temperature 40.3°C, pulse 110 beats per min, respiratory rate 36 per min, and blood pressure 100/60 mm Hg. He showed poor oral hygiene. Respiratory sounds in the bases of both lungs were not heard clearly. No heart murmur was detected. Chest x-rays showed bilateral patchy infil-

tration, which was prominent at the periphery of both hemithoraces. The right costophrenic angle was blunted, reflecting the possibility of pleural effusion. A CT scan was not performed. Transthoracic echocardiography showed a vegetation (18 x 18 mm) in the tricuspid valve. Blood culture revealed methicillin-resistant *S. aureus*. The pleural tap analysis showed the following: red color, RBC 260,000/dL, WBC 1300/dL containing 30 % PMNs and 70 % Lym, protein 2.83 gr/dL, LDH 740 mg/dL, and negative culture. Serum CRP and circulatory immunocomplexes were above the upper limits of normal. The patients were commenced on vancomycin and heparin and continued with warfarin.

3. Discussion

Pleural effusion was reported in 7 (5 %), 8 (23 %), and 2 (50 %) radiographic findings of pulmonary emboli. Pulmonary embolization should be considered a possible cause of pleural effusion of unknown etiology. This disorder may most commonly be overlooked in the workup of patients with pleural effusions (6). An exudative, sterile, serosanguinous, or bloody effusion is common in RSE (8). If diagnosis of PE is delayed, the pleural effusion tends to become loculated (3). Measurement of D-dimers is useful in the diagnosis. If the D-dimer test is negative, the diagnosis is virtually excluded. If the D-dimer test is positive or if there is a high clinical probability of a pulmonary embolism, the best method of diagnosing pulmonary embolism is likely computed tomographic angiography (9).

Septic pulmonary emboli, pleural effusion, and right-sided endocarditis should be considered as possible causes of any pulmonary symptoms in drug users (10). The diagnosis is usually suggested by the presence of a predisposing factor, fever, and radiographic findings (11). The detailed CT characteristics of peripheral nodules in pulmonary septic emboli may differentiate the causative microorganisms and provide additional information regarding treatment plans in patients with sepsis (12). Although the pathogens of septic pulmonary emboli may differ depending on the primary foci of infection, early diagnosis and prompt antimicrobial therapy with radiological or surgical intervention can lead to a successful outcome (13). With early diagnosis, appropriate antimicrobial therapy, and control of the infectious source, resolution of the illness can be expected for most patients (11). Respiratory diseases in drug abusers can manifest as a wide range of complex presentations. Knowledge of the principal complications and the appropriate diagnostic procedures is essential (5). Due to its marked cardiovascular effects, cocaine is also a major cause of coronary syndromes and myocardial infarction. Recognition of these acute complications is urgent, and treatment must be based on an understanding of the likely underlying problem as well as the basic principles of supportive

care (14). A wide variety of pulmonary complications, including interstitial pneumonitis, fibrosis, pulmonary hypertension, alveolar hemorrhage, asthma exacerbation, barotrauma, thermal airway injury, hilar lymphadenopathies, and bullous emphysema, may be associated with the inhalation of crack cocaine or its associated substances, such as talc, silica, and lactose (15). Clinical, imaging, and laboratory findings can help physicians reach a diagnosis of “crack lung” (16).

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