Pneumothorax Secondary to Septic Pulmonary Emboli in a Long-term Hemodialysis Patient with Psoas Abscess

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Abstract:
Pneumothorax secondary to septic pulmonary embolism (SPE) is rare but life-threatening. We herein report a long-term hemodialysis patient with psoas abscess caused by methicillin-resistant *Staphylococcus aureus*, associated with other muscle and splenic abscesses and SPE. Intravenous vancomycin treatment and percutaneous drainage of the psoas abscess rapidly improved her condition. However, the SPE lesions continued to increase, and right-sided pneumothorax occurred 10 days after treatment. The pneumothorax resolved after two months and SPE and all abscesses after four months of treatment. Since late-onset pneumothorax caused by SPE can occur despite successful treatment of the primary infection, care should be taken with such patients.

Key words: septic pulmonary emboli, pneumothorax, hemodialysis, psoas abscess, methicillin-resistant *Staphylococcus aureus*

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Introduction

Patients with end-stage renal disease have a compromised immune system (1). Infection is a common complication and is the second leading cause of death in hemodialysis patients (2). Indeed, the risk of bacteremia in hemodialysis patients is 26-fold higher than in the general population (3).

Septic pulmonary embolism (SPE) is an uncommon disorder in which infected thrombi from a primary infectious site lead to infarctions in the pulmonary vasculature as well as focal abscesses. SPE has a mortality rate of 10-20% (4, 5). Historically, SPE was most commonly associated with right-sided infective endocarditis in intravenous drug users, or Lemierre’s syndrome, and pelvic thrombophlebitis. However, recently, the incidence of SPE has been increasing in immunocompromised patients and patients using vascular catheters and implantable devices (5, 6).

Pneumothorax is a rare but life-threatening complication of SPE, as it sometimes occurs bilaterally. No cases of pneumothorax secondary to SPE have been reported in a long-term hemodialysis patient. We herein report a hemodialysis patient with psoas abscess caused by methicillin-resistant *Staphylococcus aureus* (MRSA) in whom pneumothorax occurred secondary to SPE.

Case Report

A 62-year-old woman with end-stage renal disease due to glomerulonephritis had been undergoing hemodialysis for 13 years. She had received two nerve block injections for back pain caused by lumbar canal stenosis at one and two weeks (one injection each) before admission. As she presented with a fever and recent-onset confusion, she was transported by an ambulance to our hospital. Her body temperature was 39.5°C (103.1°F) and her blood pressure was 65/48 mmHg, associated with tachycardia (117 beats/min), tachypnea (20/min), and hypoxia (PaO₂ 80.1 mmHg on 2 L/min of oxygen).

A physical examination revealed that the patient’s knees were swollen. An arteriovenous dialysis graft infection was not obvious. Her white blood cell count was 19,000 cells/mm³, with a marked left shift, and her platelet count was 48,000 cells/mm³. Laboratory data showed elevated levels of
C-reactive protein, fibrinogen, fibrinogen-degradation products, and D-dimer (46.32 mg/dL, 876 mg/dL, 53.9 μg/mL, and >30 μg/mL, respectively). The activated partial thromboplastin time was prolonged (50.8 s), but the prothrombin time was not prolonged.

Computed tomography (CT) revealed a right psoas abscess in addition to right piriformis, pectineus, obturator externus, adductor muscle, and splenic abscesses (Fig. 1). Multiple septic pulmonary emboli in both lungs were also found on chest X-ray and chest CT (Fig. 2A). There were no findings of brain abscesses on brain CT. Vegetation was also not observed on the heart valves by an echocardiograph. A diagnosis of psoas abscess with multiple abscesses, SPE, and septic shock was therefore made.

Meropenem was administered as the initial antibiotic treatment, and percutaneous drainage of the psoas abscess was performed. Later, cultures of blood, abscess content, and knee synovial fluid showed MRSA growth. The antibiotic treatment was changed to intravenous vancomycin on the third day of hospitalization. Her condition rapidly improved, and she recovered from septic shock. The psoas abscess markedly reduced, and blood cultures performed on the sixth day of hospitalization were negative. However, the multiple SPE lesions continued to increase and enlarge (Fig. 2B), and right-sided pneumothorax occurred on the tenth day of hospitalization (Fig. 3).

A chest tube was inserted for drainage of air, and the intravenous vancomycin treatment was continued. The pneumothorax resolved after 8 weeks, with all abscesses except for the splenic abscess and SPE resolving after 11 weeks of treatment (Fig. 2C). However, the SPE lesions and splenic abscess showed a decreasing trend. As the patient refused the offered treatment of splenectomy, intravenous vancomycin treatment was continued. The SPE lesions disappeared 14 weeks after the commencement of treatment, and the splenic abscess resolved 2 weeks later. Intravenous vancomycin treatment was discontinued the following week. Neither the abscesses nor the SPE relapsed, and she was transferred to a rehabilitation hospital.

**Discussion**

We described a case of a long-term hemodialysis patient with MRSA-related psoas abscess with multiple abscesses in...
Other muscles and in the spleen, septic arthritis of the knees, and SPE. Pneumothorax occurred at a late stage as a consequence of the progression of SPE, even though the MRSA-related psoas abscess and septicemia had responded to the treatment with vancomycin and percutaneous drainage. However, in the present case, several SPE lesions had progressed subpleurally, and subpleural pulmonary bullae were observed in the right lung after treatment. Coalescence of necrotic infarcts caused by MRSA might form large abscesses and involve the pleura, causing pneumothorax. Previous studies have reported infectious pleural effusion (9, 12), which may indicate that SPE lesions involve the pleura. However, pleural fluid culture was not performed in the present case.

In 6 previous cases (1-10, 12, 13), pneumothorax secondary to SPE occurred 5-15 days after hospitalization, despite appropriate treatment for the infection. Similarly, in the present case, although the septic shock and bacteremia were resolved by intravenous vancomycin treatment and percutaneous drainage of the psoas abscess, pneumothorax occurred 10 days after treatment. This time lag may be explained by the fact that infective thrombi were lodged in the lung capillaries. Therefore, vancomycin could not be delivered to the peripheral lung abscesses, which may have progressed, leading to the rupture of the pleura.

The treatment period of the present case was longer than in previous cases. This may be due to the treatment of the splenic abscess, which continued for the entire duration of hospitalization. One elderly patient received hemodialysis therapy for rapidly progressive glomerulonephritis, but this was for a short period until the onset of SPE following the use of a central venous catheter. The present report is the first of pneumothorax secondary to SPE in a long-term hemodialysis patient with an arteriovenous fistula or graft.

Staphylococci were the infectious pathogens in all reported cases of secondary pneumothorax due to SPE, and eight of the nine cases were caused by Staphylococcus aureus. Methicillin-sensitive Staphylococcus aureus (MSSA) was identified in 5 cases and MRSA in 2 cases. Staphylococcus aureus, including MRSA, is also a predominant cause of SPE (4, 5). Pulmonary cavitation is a well-known manifestation in Staphylococcus aureus pneumonia (14), and pneumothorax is also a common complication of staphylococcal pneumonia (15). Community-associated-MRSA is often associated with severe necroizing pneumonia, which is characterized by pulmonary inflammation with consolidation, peripheral necrosis, and multiple small cavities. In the present case, several SPE lesions had progressed subpleurally, and subpleural pulmonary bullae were observed in the right lung after treatment. Coalescence of necrotic infarcts caused by MRSA might form large abscesses and involve the pleura, causing pneumothorax. Previous studies have reported infectious pleural effusion (9, 12), which may indicate that SPE lesions involve the pleura. However, pleural fluid culture was not performed in the present case.

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Figure 2. Time-course for the development of pulmonary septic emboli. Small septic emboli were observed on admission (A, arrows). The number and size of septic emboli increased in both lungs on the ninth day of hospitalization (B). The septic emboli resolved and the bullae remained in the right lung after four months of treatment (C).
Splenic abscess is an uncommon infection with a high mortality rate. Robinson et al. reported that all infective endocarditis and splenic abscess patients who did not undergo splenectomy died, and half of these had undergone splenectomy. In conclusion, pneumothorax can occur following SPE in our patient did not wish to undergo splenectomy.

### Table. Summary of Cases with Pneumothorax as a Complication of Septic Pulmonary Embolism.

| Reference | (7) | (8) | (9) case 1 | (9) case 2 | (10) | (12) | (11) | (13) | This case |
|-----------|-----|-----|------------|------------|------|------|------|------|-----------|
| Patient age/sex | 19/male | 24/male | 23/male | 26/male | 23/male | 72/female | 25/male | 66/male | 62/female |
| Pathogen | Staphylococcus aureus (MSSA) | Staphylococcus aureus | Staphylococcus aureus (MSSA) | Staphylococcus aureus | Staphylococcus aureus (MSSA) | Staphylococcus aureus (MSSA) | Staphylococcus aureus (MSSA) | Staphylococcus aureus (MSSA) |
| Medical history | intravenous drug user | intravenous drug user | intravenous drug user, HIV infection | intravenous drug user | intravenous drug user | intravenous drug user | intravenous drug user | intravenous drug user |
| Source of infection | tricuspid valve endocarditis | tricuspid valve endocarditis | tricuspid valve endocarditis | tricuspid valve endocarditis | tricuspid valve endocarditis | central venous catheter-related infections | pulmonary valve endocarditis | prostatic abscess | psoas abscess |
| Time to onset after treatment* | 6 days | 5 days | 15 days | within a day | 7 days | 13 days | same time | 13 days | 10 days |
| Location of pneumothorax | bilateral | right | bilateral | right | left | right | left | bilateral | right |
| Treatment period | 6 weeks | N/A | 8 weeks | 4 weeks | 12 weeks | 7 weeks | N/A | 8 weeks | 17 weeks |
| Outcome | survival | survival | survival | survival | survival | survival | survival | survival | dead | survival |

MSSA: Methicillin-sensitive Staphylococcus aureus, MRSA: Methicillin-resistant Staphylococcus aureus

*Time to the onset of pneumothorax after the commencement of treatment for the primary infection.
tion, usually caused by *Staphylococcus aureus*. Furthermore, careful attention should be paid to the progression of SPE and the potential for late-onset pneumothorax, even in cases in which the primary infection is controlled.

The authors state that they have no Conflict of Interest (COI).

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