Internal Medicine
The Japanese Society of Internal Medicine
doi: 10.2169/internalmedicine.9050-17
Intern Med 56: 3243-3247, 2017
http://internmed.jp

【 CASE REPORT 】

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

Masahiro Okabe[1], Kenji Kasai[2] and Takashi Yokoo[1]

**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*

**(Intern Med 56: 3243-3247, 2017)**
**(DOI: 10.2169/internalmedicine.9050-17)**

## 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℃ (103.1°F) and her blood pressure was 65/48 mmHg, associated with tachycardia (117 beats/min), tachypnea (20/min), and hypoxia ($PaO_2$ 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/$mm^3$, with a marked left shift, and her platelet count was 48,000 cells/$mm^3$. Laboratory data showed elevated levels of

[1]Division of Nephrology and Hypertension, Department of Internal Medicine, Jikei University School of Medicine, Japan and [2]Fuji City General Hospital, Japan
Received: February 15, 2017; Accepted: April 10, 2017; Advance Publication by J-STAGE: October 11, 2017
Correspondence to Dr. Masahiro Okabe, mokabe@jikei.ac.jp

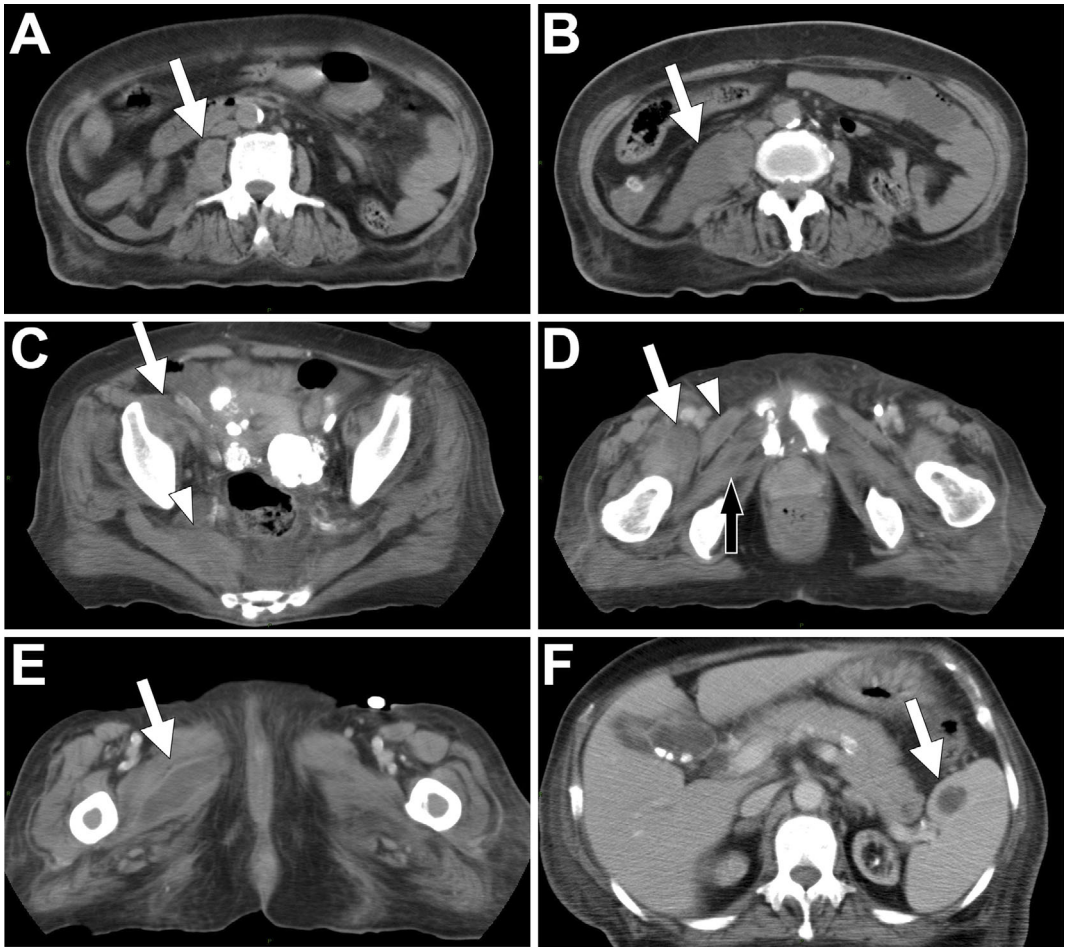


**Figure 1. Computed tomography (CT) findings. Abscesses were observed in the right psoas, from the higher level (A and B, arrow) to the lower level (C and D, white arrow), right piriformis (C, arrowhead), right pectineus (D, arrowhead), right obturator externus (D, black arrow), right adductor muscle (E, arrow), and spleen (F, arrow).**

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

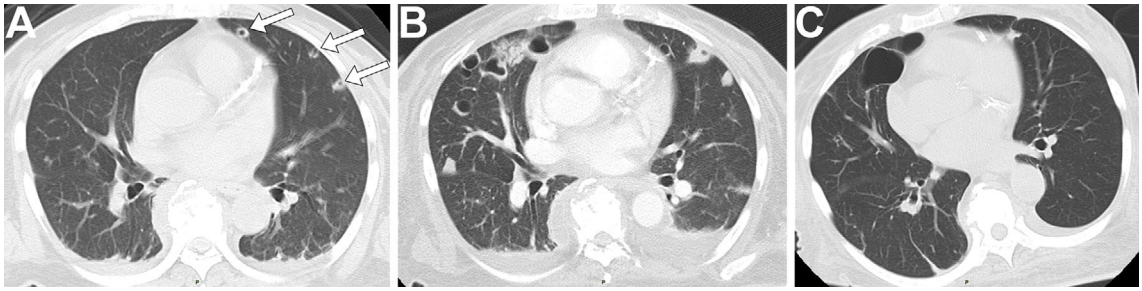


**Figure 2.** **Time-course for the development of pulmonary septic emboli. Small septic emboli were observed on admission (A, arrows). The number and size of septic emboli increased in both lungs on the ninth day of hospitalization (B). The septic emboli resolved and the bullae remained in the right lung after four months of treatment (C).**

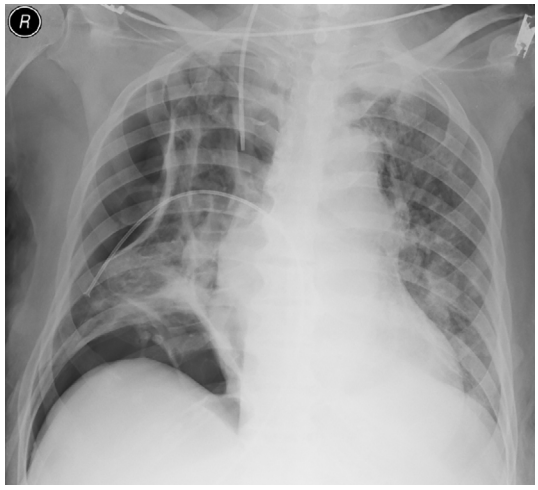


**Figure 3.** **Chest X-ray findings. Right-sided pneumothorax occurred on the tenth day of hospitalization.**

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.

Long-term hemodialysis patients, as in this case, are immunocompromised patients and at a high risk for bloodstream infection. *Staphylococcus aureus*, including MRSA, is the most common causative organism (2). The patient received two nerve block injections before the development of the psoas abscess; therefore, in addition to the possibility of a bloodstream infection, MRSA infection may have resulted from the injections.

Pneumothorax secondary to SPE is rare, and only six cases have been reported within journals in English (7-11). We found two more case reports in Japanese (12, 13). We reviewed all of the previously reported cases, and the characteristics of these as well as our case are summarized in Table. Six of the patients were young intravenous drug abusers, and two were elderly immunocompromised patients. Cases of two immunocompromised patients were reported recently, which is compatible with the etiology of SPE showing an increasing incidence in immunocompromised patients. 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 necrotizing 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.

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

**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 epidermidis* | *Staphylococcus aureus* (MSSA) | *Staphylococcus aureus* (MRSA) | *Staphylococcus aureus* (MSSA) | *Staphylococcus aureus* (MSSA) | *Staphylococcus aureus* (MRSA) |
| Medical history | intravenous drug user | intravenous drug user | intravenous drug user, HIV infection | intravenous drug user, HIV infection | intravenous drug user | rapidly progressive glomerulonephritis (hemodialysis, glucocorticosteroid therapy) | intravenous drug user | type 2 diabetes mellitus | end-stage renal disease due to glomerulonephritis (hemodialysis) |
| 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 | 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.

this case. 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 valvar replacement (16). Splenic abscess is usually managed by a combination of antibiotic therapy and splenectomy, but our patient did not wish to undergo splenectomy.

In conclusion, pneumothorax can occur following SPE in patients on long-term hemodialysis. These patients are immunocompromised hosts at a high risk of bloodstream infec-

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).**

## References


1. Vaziri ND, Pahl MV, Crum A, Norris K. Effect of uremia on structure and function of immune system. J Ren Nutr **22**: 149-156, 2012.
2. Suzuki M, Satoh N, Nakamura M, Horita S, Seki G, Moriya K. Bacteremia in hemodialysis patients. World J Nephrol **5**: 489-496, 2016.
3. Skov Dalgaard L, Nørgaard M, Jespersen B, et al. Risk and prognosis of bloodstream infections among patients on chronic hemodialysis: a population-based cohort study. PLoS One **10**: e0124547, 2015.
4. Ye R, Zhao L, Wang C, Wu X, Yan H. Clinical characteristics of septic pulmonary embolism in adults: a systematic review. Respir Med **108**: 1-8, 2014.
5. Goswami U, Brenes JA, Punjabi GV, LeClaire MM, Williams DN. Associations and outcomes of septic pulmonary embolism. Open Respir Med J **8**: 28-33, 2014.
6. Cook RJ, Ashton RW, Aughenbaugh GL, Ryu JH. Septic pulmonary embolism: presenting features and clinical course of 14 patients. Chest **128**: 162-166, 2005.
7. Aguado JM, Arjona R, Ugarte P. Septic pulmonary emboli. A rare cause of bilateral pneumothorax in drug abusers. Chest **98**: 1302-1304, 1990.
8. Olazabal A, Bartrina J, Perez Andres R. Spontaneous pneumothorax as a complication of septic pulmonary embolism in intravenous drug addicts. Eur J Radiol **10**: 56-58, 2006.
9. Corzo JE, Lozano de León F, Gómez-Mateos J, López-Cortes L, Vázquez R, García-Bragado F. Pneumothorax secondary to septic pulmonary emboli in tricuspid endocarditis. Thorax **47**: 1080-1081, 1992.
10. Sheu C-C, Hwang J-J, Tsai J-R, Wang T-H, Chong I-W, Huang M-S. Spontaneous pneumothorax as a complication of septic pulmonary embolism in an intravenous drug user: a case report. Kaohsiung J Med Sci **22**: 89-93, 2006.
11. Swaminath D, Yaqub Y, Narayanan R, Paone RF, Nugent K, Arvandi A. Isolated pulmonary valve endocarditis complicated with septic emboli to the lung causing pneumothorax, pneumonia, and sepsis in an intravenous drug abuser. J Investig Med high impact case reports **1**: 2324709613514566, 2013.
12. Inamoto S, Hashimoto Y. A case of pneumothorax secondary to septic pulmonary embolism due to central venous catheter infection caused by methicillin-resistant Staphylococcus aureus. Kansenshogaku Zasshi (J Jpn Assoc Infect Dis) **82**: 51-54, 2008 (in Japanese, Abstract in English).
13. Kato K, Irie H, Otake T, Yamashita S, Yonei A. Simultaneous bilateral pneumothorax associated with septic pulmonary embolism: a case report. Nihon Syuchuchiryou Igakkai Zasshi (J Jpn Soc Intensive Care Med) **20**: 421-422, 2013 (in Japanese).
14. Macfarlane JT, Miller AC, Roderick Smith WH, Morris AH, Rose DH. Comparative radiographic features of community acquired Legionnaires' disease, pneumococcal pneumonia, mycoplasma pneumonia, and psittacosis. Thorax **39**: 28-33, 1984.
15. Macfarlane J, Rose D. Radiographic features of staphylococcal pneumonia in adults and children. Thorax **51**: 539-540, 1996.
16. Robinson SL, Saxe JM, Lucas CE, Arbulu A, Ledgerwood AM, Lucas WF. Splenic abscess associated with endocarditis. Surgery **112**: 781-786, 1992.