

## Septic pulmonary embolism with deep vein thrombosis caused by physical immobility

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### Abstract

**Introduction:** Septic pulmonary embolism is a rare disease. It occurs when a pathogen-containing thrombus embolizes to the lung. We experienced a patient who had deep vein thrombosis due to physical immobility, and sequentially had septic pulmonary embolisms. Computed tomography showed a thrombus in the pulmonary artery and multiple nodules with surrounding blood vessel conversion. We noticed that the patient had periodontitis, and we used phosphomycin, which was quite effective. In conclusion, computed tomography is useful for diagnosing a septic pulmonary embolism. Antibiotics are key drugs in controlling this disease.

**Keywords:** periodontitis, phosphomycin, septic pulmonary embolism

### Introduction

Septic pulmonary embolism is a relatively uncommon syndrome characterized by the embolization of pathogen-containing thrombi admixed with fibrin from an infected site into the venous circulation; the emboli then become implanted in the vascular system of the lungs, leading to a secondary infection.<sup>1-3</sup> Infective endocarditis<sup>4</sup> and infectious phlebitis<sup>5</sup> are two major causes of septic pulmonary embolism. The pediatric literature has recently described the following triad: (1) an active extrapulmonary source of infection; (2) an adjacent venous thrombosis; and (3) septic pulmonary embolism.<sup>6</sup> We experienced a case of massive deep vein thrombosis due to physical immobility. The patient sequentially developed a septic pulmonary embolism. The infectious cause was believed to be periodontitis (although this was not adjacent to the thrombus formation); the septicemia was believed to be secondary to an infected deep vein thrombus.

### Case Presentation

An 80-year old female was found in a rest room after 12 hours of immobility. She had fallen between a

toilet stool and was unable to escape by herself. She was taken to another hospital by an ambulance. Thrombosis was suspected, and she was transferred to our hospital. On the patient's arrival, edema and pain of the bilateral lower extremities, and mild Homan's sign were noted. Laboratory data showed a white blood cell count of 19800/ $\mu$ L; C-reactive protein level of 8.0 mg/dL; aspartate amino transferase, 168 IU/L; alanine aminotranferase, 75 IU/L; lactate dehydrogenase, 590 IU/L; creatine kinase, 15295 IU/L; fibrin degradation product, 452  $\mu$ g/mL; brain natriuretic peptide, 254 pg/ $\mu$ L; blood urea nitrogen, 37 mg/dL; and creatinine, 1.04 mg/dL. Thrombosis, dehydration, heart failure, and crush syndrome were suspected. Electrocardiography showed regular sinus rhythm but no right heart overload, left heart dysfunction, or infective endocarditis. Echocardiography of the lower extremities revealed soleal vein thrombosis.

We started anticoagulation therapy by using heparin and warfarin. Her inflammatory response improved on the fourth day of her illness. Her C-reactive protein level was 3.0 mg/dL, white blood cell count was 7900/ $\mu$ L, and body temperature was 36.7°C.

However, a chest X-ray image obtained on the fourth day of illness showed a nodular lesion that was not noticed in the lung field on admission. She developed a fever. On chest X-ray image, a nodule in the lung field was apparent on the sixth day of illness (Figure 1). On the same day, enhanced computed tomography revealed a pulmonary embolus.

Figure 1. Chest X-ray image on the sixth day of illness reveals nodules (arrows) in the right lung field.



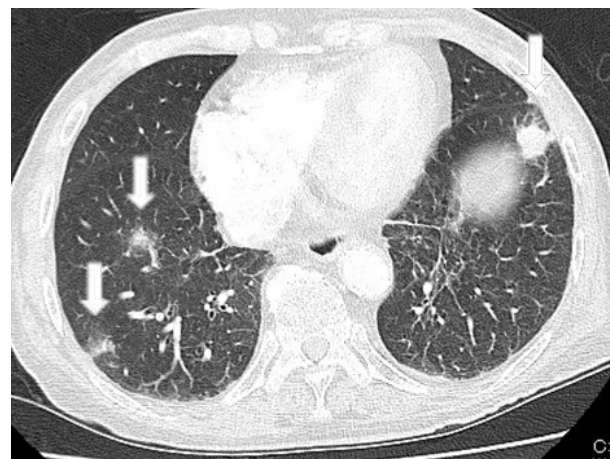
It was visible at the peripheral branch of the right pulmonary artery (Figure 2). In right segments 2, 5, and 8, and in left segment 10, there were also multiple nodular lesions (up to 13 mm) with a convergence of surrounding vessels.

Figure 2. Computed tomography on the sixth day of illness shows a pulmonary embolus (arrow) at the peripheral branch of the right pulmonary artery.



This was considered the “feeding vessel sign” of septic pulmonary embolism (Figure 3). A blood culture was performed.

Figure 3. Computed tomography reveals multiple nodular lesions (arrows) (up to 13 mm) with a convergence of surrounding vessels. This is the feeding vessel sign.



Levofloxacin was empirically started as the antibiotic therapy, but her inflammatory response worsened. The body temperature was elevated to 38.8°C on the 10th day of illness, and the C-reactive protein level was elevated to 16.9 mg/dL on the 11th day of illness. A blood culture was negative.

We could not find the traumatic source of the bacterial invasion. After a resident noticed a bad odor in her mouth, we were then aware she had severe periodontitis. After the failure of a broad spectrum new quinolone antibiotic, we chose phosphomycin because of its performance in treating oral infections.<sup>7</sup> After the administration of phosphomycin, her body temperature normalized; the C-reactive protein level decreased to 6.4 mg/dL; nodular lesions began to disappear; and the pain of the lower extremities improved. The fibrinogen degradation product level reduced to 8 µg/mL and warfarin control was achieved. She was discharged from the hospital on the 17th day of illness.

## Discussion

Septic pulmonary embolism is a rare disease that develops after a fever or respiratory symptoms; its diagnosis is often delayed.<sup>1</sup> Infective endocarditis<sup>4</sup> and infectious thrombophlebitis<sup>5</sup> are two major causes. Other causes include dental focus,<sup>8</sup> malignancy,<sup>9</sup> pharyngitis, tonsillitis,<sup>5</sup> and more recently infection related to devices such as a vein catheter or pacemaker.<sup>1,10,12</sup> A triad consisting of the following features has been reported: (1) an active extrapulmonary infection; (2) an adjacent

venous thrombosis; and (3) septic pulmonary embolism.<sup>6</sup> In our patient, deep vein thrombus and pulmonary embolism were present. The infectious invasion source was believed to be periodontitis. The origin of the infection was therefore not adjacent to the thrombus formation. Septicemia due to periodontitis can cause thrombus infection.

Clinical signs of septic pulmonary embolism include pain in the chest, breathing disorders such as dyspnea, hemoptysis, hydrothorax, and a thoracic empyema.<sup>13</sup> The clinical signs in our patient was only fever and the local signs of deep vein thrombosis and thrombophlebitis.

Chest X-ray images often show round nodules, wedge-shaped scattered lesions, and cavity formation.<sup>5</sup> Multiple nodules were noted in our patient. Clinical symptoms and chest X-ray findings are not very specific for diagnosing septic pulmonary embolism. By contrast, computed tomography provides rather specific features. They include multiple nodular shadows (0.5–3 cm) in the bilateral peripheral regions; cavity formation by tissue necrosis (i.e., target sign); convergence of vessels to the region, which may be a nourishment blood vessel (i.e., the feeding vessel sign); and a collection of pleural effusion.<sup>14,15</sup>

Treatment for septic pulmonary embolism includes the prompt empiric administration of intravenous antibiotics and the removal of any potentially infected devices.<sup>3</sup>

In our patient, the first empiric choice of a new quinolone antibiotic was unsuccessful. However, after focusing on periodontitis as the infectious source, the choice of phosphomycin was a successful treatment. We believe that determining the source of infection and choosing an effective antibiotic is crucial. Anticoagulation is an important treatment for deep venous thrombosis and is also beneficial to reduce an infected thrombus for which there may be a concern about the potential increased risk of septic embolism and hemorrhage.

## Conclusion

We experienced a case of deep vein thrombosis caused by physical immobility, followed by septic pulmonary embolism. The infectious source seemed to be periodontitis, which was treated successfully by phosphomycin. Computed tomography is useful for diagnosing a septic pulmonary embolism. Controlling the infectious organism is crucial for controlling septic pulmonary embolism. Anticoagulation is also beneficial.

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