# Disseminated Staphylococcal Infection in an Immunocompetent Adult: A Case Report

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**ABSTRACT**

Septic pulmonary embolism is an uncommon disorder in which septic thrombi are mobilized from an infectious nidus and transported in the vascular system of the lungs. We report a case of a 52-year-old immunocompetent female who suffered from septic pulmonary embolism associated with polymyositis, deep venous thrombosis and pericardial effusion. Oxacillin-sensitive staphylococcus aureus (MSSA) was isolated from her sputum. Clinical presentation improved after incision of the muscle abscess and vancomycin treatment.

*Key Words: septic pulmonary embolism, pyomyositis, septic thrombophlebitis, Staphylococcus aureus*

## Introduction

Septic pulmonary embolism (SPE) is a rare but life-threatening disease if not recognized and treated promptly. It is characterized by embolization of infected thrombi from a primary infectious site to the venous circulation with implantation in the pulmonary vasculature resulting in parenchymal infection.[1] It is usually associated with tricuspid valve vegetation in IV drug users, infected venous catheters, pelvic thrombophlebitis, Lemierre's syndrome (postanginal septicemia) and odontogenic infections.[2-5] Recently, there has been an increasing number of reports of SPE among adults in the setting of septic thrombophlebitis adjacent to a primary infectious source, such as contiguous deep soft tissue infection or osteomyelitis of the extremities.[5] Clinical and radiologic features at presentation are usually non-specific and the diagnosis is frequently delayed. We present an unusual cause of septic pulmonary embolism from pyomyositis associated with deep venous thrombosis and pericardial effusion.

## Case Presentation

A previously healthy 52-year-old married Filipino woman was admitted to our hospital due to right thigh pain. The patient was in good condition until seven days earlier, when right thigh pain and swelling developed. The patient consulted a primary care physician who prescribed pain relievers providing temporary relief of symptoms. The pain worsened gradually until she had difficulty of walking. She consulted at our institution and was subsequently admitted.

The patient was a mother and was working as a government employee. She was not under medication for any illness. She was a non-smoker and non-alcoholic beverage drinker. She mentioned a history of skin infection on her back which resolved after self-medication of unrecalled oral antibiotics four months prior to admission.

On admission, the patient was afebrile with heart rate of 88/bpm and respiratory rate of 21/minute. Physical examination revealed tender, erythematous and edematous anterior and lateral aspects of the right thigh. Auscultation revealed clear breath sounds. Initial laboratory investigation showed white blood cell count of 7,100 u/L with predominance of neutrophils. The rest of the chemistry results were normal. Patient was started on empiric antibiotic therapy (Piperacillin-Tazobactam) for the soft tissue infection of the right thigh.

On the second hospital stay, the patient complained of sudden new-onset chest pain with dyspnea and hemoptysis. Physical examination revealed coarse crackles and diffuse rhonchi. Differential diagnoses included septic pulmonary emboli or acute respiratory distress syndrome (ARDS). Arterial blood gas (ABG) revealed hypoxemia. Chest radiograph revealed moderate patchy opacities scattered in both lungs (Figure 1A). Antibiotic was shifted to Oxacillin and Clindamycin. The patient was brought to the intensive care unit (ICU) due to acute respiratory failure requiring non-invasive ventilation (NIV). Repeat complete blood count showed leukocytosis with WBC of 23,000 u/L. The patient also developed septic shock. She was given intravenous fluid challenge and vasopressor. Heavy growth of *Oxacillin-sensitive Staphylococcus aureus* was isolated from the sputum. On the 4th hospital stay, repeat chest radiograph showed progression of confluent densities on both lungs (Figure 1B). Transthoracic echocardiography did not demonstrate any vegetation.

Computed tomography (CT scan) of the chest showed several patchy areas of consolidation scattered in both lobes. Rounded consolidation was noted in the right lower lobe and the superior basal segment of the left lower lobe at the

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subpleural aspect. Cavitary formations are seen in both upper lobes. Ground glass hazy densities are also seen in both lobes, most evident in the upper lobes as well as in the periphery (Figures 2A and 2B).

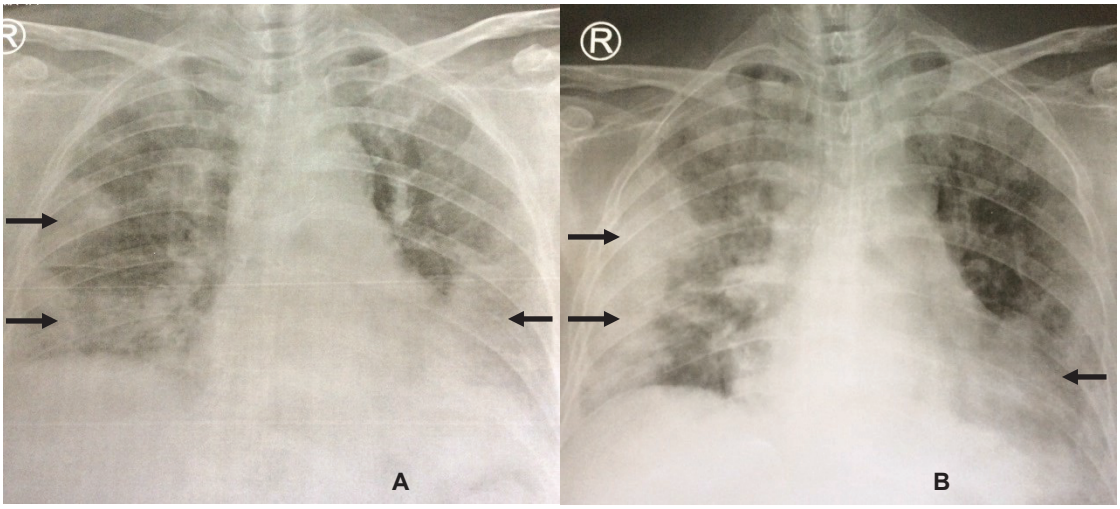


**Figure 1.** Chest radiographs. A. Bilateral peripherally located opacities in both lungs [black arrows]. B. Progression of bilateral consolidations/opacities [black arrows].

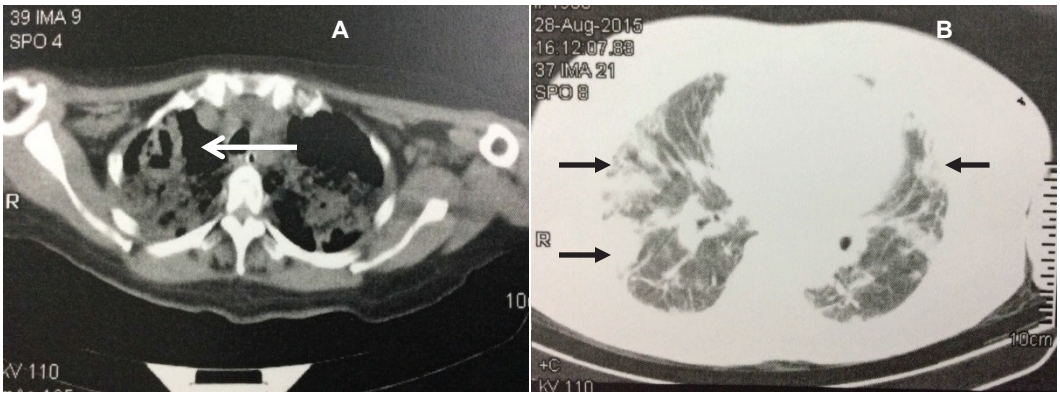


**Figure 2.** Chest CT Scan. A. Ovoid thick-walled lucencies at the right upper lobe of the lung which may represent cavity formation [white arrow]. B. Patchy areas of consolidation scattered in both lungs as well as in the periphery [black arrows].

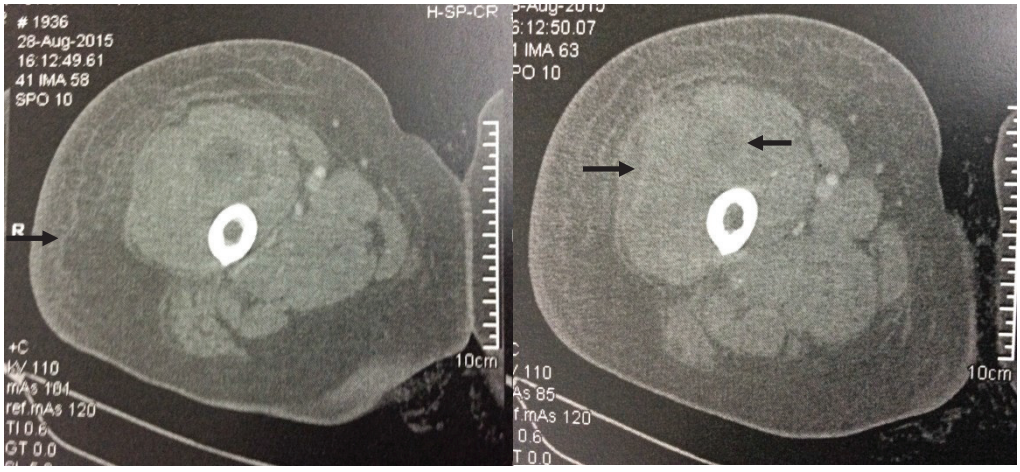


**Figure 3.** CT Scan of Right Thigh. A. CT of right thigh showing marked subcutaneous soft tissue swelling [black arrow]. B. Intramuscular abscesses in vastus medialis and lateralis [black arrows].

CT of the right thigh revealed intramuscular abscess formations, one centered in the vastus medialis muscle, and the other centered in the vastus lateralis muscle extending to the iliotibial tract. Moderate to marked subcutaneous soft tissue swelling involving the thigh, most significant at its lateral and anterior aspect (Figures 3A and 3B). Venous duplex scan of both lower extremities showed deep vein thrombosis, probably acute to subacute, in the right distal external iliac vein, common femoral vein and femoral vein (proximal). Antibiotics were shifted to Vancomycin. Anti-coagulation with Enoxaparine was started. Patient's blood pressure stabilized. Her condition gradually improved and was weaned off from the NIV.

Repeat venous duplex scan of both lower extremities after 10 days showed complete resolution of the deep vein thrombosis. Repeat blood culture (obtained during Oxacillin and Clindamycin therapy) showed no growth. Repeat 2D-echocardiography showed interval appearance of moderate to large pericardial effusion with signs of tamponade. Patient refused pericardiocentesis.

Surgical incision and drainage of the abscess was performed. Repeat blood culture (obtained during vancomycin therapy) was negative. Repeat chest radiograph prior to discharge showed significant interval regression of the confluent opacities on both lung fields. Patient completed a 3-week course of Vancomycin. Repeat 2D-echocardiography revealed significant decrease in the amount of pericardial effusion. Patient had complete resolution of infection prior to discharge.

**Discussion**

Septic pulmonary embolism (SPE) is an uncommon condition in which a thrombus containing microorganisms embedded in fibrin is mobilized from an infectious nidus and implanted in the pulmonary arteries, inciting metastatic abscess and leading to infarction.[2,6-7] Septic pulmonary emboli reach the lung from many sources, including infected heart valves, thrombophlebitis and infected catheters or pacemaker wires. Historically, intravenous drug abuse, odontogenic, postanginal (Lemierre's syndrome), soft tissue and pelvic infections were commonly reported as causes of SPE.[2-4]

Recently, septic pulmonary emboli arising from primary deep tissue infections such as pyomyositis, cellulitis, osteomyelitis, prostatic and periproctal abscess have been described in the adult population.[8-13] A recent systematic review of 76 articles consisting of a total 168 cases by Ye et al[14] in 2014 showed that major risk factors for SPE were intravenous drug use, intravascular indwelling catheter and skin or soft tissue purulent infection. Seventeen cases died and 101 cases were cured.

Goswami et al[1] have shown that the epidemiology of SPE has broadened over the past decade with an increase in identified extrapulmonary, non-cardiac sources. In their review[1] of 41 cases of SPE, sources of infection include skin and soft tissue (44%); infective endocarditis (27%) and infected peripheral DVT (17%).

A high index of suspicion for SPE should be considered in a febrile patient with an identified extrapulmonary source of infection who develops secondary pulmonary symptoms such as pleuritic chest pain, dyspnea, cough and hemoptysis. All these symptoms were seen in our patient. Although chest radiographs are usually obtained in these patients, findings are non-specific. Chest CT scan play an important role in the diagnosis of septic pulmonary embolism. In a review by Huang et al[15] of 15 patients with clinically documented septic pulmonary emboli, CT features include peripheral nodules with clearly identifiable feeding vessels associated with metastatic lung abscesses (67%) and subpleural wedge-shaped densities with and without necrosis caused by septic infarcts (73%). Cavitary parenchymal nodules may be caused by septic occlusion of small peripheral PA branches.[15] Systematic review by Ye et al[14] in 168 cases, chest CT scan showed multiple peripheral nodules in both lungs, cavitation, focal or wedge-shaped infiltrates and pleural effusion. In our patient, pulmonary symptoms and radiologic features, including bilateral peripherally located multiple consolidations and cavitary nodules, rapid progression of cavities and opacities, were compatible with septic embolization.

Brenes and colleagues[5] postulated that an extrapulmonary site of infection can allow extravasation or translocation of an organism, most commonly bacterial, into the systemic venous circulation. Once in the bloodstream, the pathogen can produce damage directly through toxins and indirectly via inflammatory mediators, which may occasionally promote local thrombosis, serving as an additional nidus for proliferation of the bacteria. Embolization of these thrombi into the pulmonary circulation allows for metastatic parenchymal infection of the lungs, even in the absence of cardiac valvular involvement.[5]

*Staphylococcus aureus*, the leading causative agent, has a tendency to promote venous thrombosis. It produces an extracellular, heat stable Panton-Valentine Leukocidin (PVL) that exhibits thrombogenic effects, through indirect inflammatory mechanisms including the formation of reactive oxygen species and release of secondary inflammatory mediators from dying granulocytes, causing intense endothelial dysfunction. S. aureus also produces coagulase, which specifically interacts with fibrinogen and causes coagulation.[5] Wang CF et al,[17] reported a case of disseminated staphylococcal disease (multiple staphylococcal arthritis, deep vein thrombosis, pulmonary embolism, pericardial effusion and occlusion of the right middle cerebral artery) in a 10-year-old boy.

As postulated in previous studies,[1,5] deep tissue infections may be associated with local venous, and

presumably septic thrombophlebitis. It is therefore plausible that our patient's pyomyositis was complicated by local septic thrombophlebitis causing the SPE.

In patients presenting with a locally invasive soft tissue infection, Staphylococcus aureus should be highly suspected. Staphylococcus aureus and MSSA in particular, remains the most likely pathogen as evidence in the series of Goswami et al[1] and in the systematic review by Ye, et al[14] in patients with SPE. Blood cultures grew MRSA (n=27) and MSSA (n=52).

Eradication of infection is the cornerstone in the management of SPE. This includes prompt administration of appropriate antibiotics and removal of purulent collections. Our patient was treated with a 3-week course of Vancomycin followed by drainage of the abscesses in the right thigh, which led to significant clinical improvement.

The role of anticoagulation in the treatment of SPE associated with septic thrombophlebitis remains controversial because of the risk of bleeding, especially in the CNS. However, recent systematic reviews have demonstrated the safety of IV heparin in the treatment of septic thrombophlebitis.[16]

To the best of our knowledge, this is the first reported local case of septic pulmonary embolism with pyomyositis, deep venous thrombosis and pericardial effusion in an immunocompetent adult.

## Conclusion

The epidemiology of SPE has changed over time with an increase in identified extrapulmonary sources related to contiguous infection. Clinicians should consider pyomyositis in patients with severe muscle tenderness and also recognize S. aureus as the possible cause of the infection. Early diagnosis of SPE and prompt antimicrobial therapy with appropriate surgical removal or drainage of the extrapulmonary source of infection will lead to improve patient outcome.

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