

Soft-tissue infection in an adult presenting as septic pulmonary embolism: A case report

Sir,

Pulmonary thromboembolism has received considerable attention in the recent past owing to increased awareness and utilization of computed tomography (CT) pulmonary angiography. A sizable proportion of patients who present with symptoms of pulmonary embolism have nonthrombotic material of varied etiologies occluding the pulmonary arteries. Septic pulmonary embolism (SPE) results from occlusion of pulmonary circulation by emboli-containing microorganism that arises from an extrapulmonary focus of infection. Typically, SPE in adults is associated with infective endocarditis, intravascular catheter devices, or intravenous drug abuse.^[1,2] Here, we present a case of SPE arising from soft-tissue infection in the left lower limb.

A 56-year-old man presented with breathlessness on exertion for the last 1 month. He had acute onset of chest pain and dyspnea necessitating hospitalization 1 month ago. After preliminary evaluation, he was diagnosed with acute coronary syndrome and new diabetes mellitus and received antiplatelets, beta blocker, and oral glucose lowering agents and was discharged after a few days. However, he continued to have dyspnea and developed new onset of left calf pain requiring re-hospitalization after 1 week. This time, deep vein thrombosis and pulmonary thromboembolism were suspected and initiated on empirical unfractionated heparin. After 2 days, the patient developed scanty hemoptysis; heparin was discontinued and he was referred to a coronary care unit. There, echocardiography was normal and Doppler ultrasound of the lower limbs failed to demonstrate the presence of intravascular thrombus; however, there was evidence of multiple collaterals in the left calf. Chest CT scan was reported to be suspicious of lung cancer with metastases and he was referred to our hospital for tissue diagnosis.

At presentation he had no fever, cough, chest pain, or hemoptysis and his vitals were as follows: Pulse rate 90 beats/min, blood pressure 130/80 mmHg, respiratory rate 24 breaths/min, and peripheral capillary oxygen saturation (SpO₂) 94% in room air. He was a smoker and worked as an office assistant. Systemic examination was essentially normal except for a mildly tender left calf. Repeat transthoracic echocardiogram was normal. Blood metabolic panel and hemogram were normal other than mild elevation of serum creatinine (1.4 mg/dL). Urine microscopic examination was normal. Blood culture

was sterile. Sputum cytology and microbiology were noncontributory. Ultrasonography of the lower limbs revealed an organized abscess (8.5 × 3 cm) in the inferomedial aspect of the left calf that was dry to aspiration. Fiberoptic bronchoscopy showed normal tracheobronchial tree. Bronchoalveolar lavage cytology was negative for malignancy but culture-grown *Klebsiella spp.* was sensitive to ciprofloxacin. Antinuclear antibody test and antineutrophilic cytoplasmic antibody test were negative. Chest radiograph showed bilateral peripheral haziness [Figure 1]. A review of the patient's previous CT scan showed bilateral, multiple lung nodules of variable sizes; some with cavitations and feeding vessel and pleural-based wedge-shaped opacities [Figure 2a]. Considering the clinical presentation, radiographic findings, and a negative workup for malignancy and vasculitis, SPE was diagnosed. He received oral ciprofloxacin and coamoxiclav for 2 weeks. The follow-up CT scans showed the gradual disappearance of lung nodules reaffirming the diagnosis of SPE [Figures 2b and c].

Pulmonary embolism is a potentially catastrophic medical emergency. The majority of pulmonary emboli are thrombotic in nature resulting from migration of deep vein thrombosis to the pulmonary circulation. Nonthrombotic pulmonary embolism may be due to embolization of cells including tumor cells, amniotic fluid, bacteria, fungi, fat, foreign material, and gas into pulmonary circulation. Traditionally, SPE has been associated with right-sided infective endocarditis, suppurative infections of the head and neck, and septic thrombophlebitis. Recently, with the increasing use of indwelling intravenous catheters, prosthetic vascular devices, increased intravenous drug abusers, and immunocompromised patients, the incidence of SPE is rising.^[1,2]

The causative organisms for SPE are diverse and depend on the site of infection. *Staphylococcus aureus* is the predominant organism for SPE in patients with intravenous drug abuse and prosthetic intravascular devices whereas Streptococcus, Enterobacteriaceae, and anaerobes are common in pelvic and ovarian vein thrombophlebitis though cultures are often negative.^[3,4] Lemierre syndrome is an anaerobic thrombophlebitis of the internal jugular vein with metastatic infection. This mostly affects adolescents and young adults with tonsillopharyngitis, odontogenic infection, mastoiditis, or sinusitis. The usual anaerobes implicated are *Fusobacterium necrophorum* and *Fusobacterium*



Figure 1: Chest radiograph showing bilateral ill-defined peripheral consolidation (arrows)

nucleatum and *Bacteroides* and *Streptococcus* species. Pulmonary involvement in Lemierre's syndrome has been reported in up to 97% of the patients with lung abscesses, pleural effusion, and empyema.^[2,3] SPE related to deep tissue infections such as osteomyelitis, septic arthritis, cellulitis, and pyomyositis caused by *Staphylococcus aureus* is well-described in pediatric patients^[4] and of late is increasingly being recognized in adults.^[5,6] Recently, *Klebsiella pneumoniae* has been found to be the second most common organism in skin and soft-tissue infections. It is more prevalent in males and among those with liver cirrhosis, malignancy, and alcoholism.^[6] The pathogenesis of SPE in our patient may be due to local septic thrombophlebitis from the calf abscess with an infected embolus gaining access into circulation. The usual clinical features of SPE are fever, dyspnea, pleuritic chest pain, cough, and hemoptysis. Almost always, there is a predisposing factor or an obvious source of infection. Patients may present with symptoms related to the site of extrapulmonary infection alone or in association with respiratory symptoms.^[3,4] The diagnosis of SPE is often delayed due to nonspecific clinical and radiological features unless a high index of clinical suspicion is maintained. Our case visited two hospitals before receiving the correct diagnosis after 1 month of initial presentation. This highlights the need for increased awareness about SPE among clinicians. We feel that the diagnosis of SPE should not be of great difficulty in a patient with compatible clinical history and radiological findings, provided this is considered as a possibility. It is of paramount importance to differentiate SPE from thromboembolism as their management and clinical outcomes are widely different. Though pulmonary thromboembolism and SPE share similar clinical features, the presence of fever, a defined source of infection, characteristic CT scan appearance, response to antibiotics and lack of hemodynamic instability, right ventricular dysfunction, or thrombus in the main arteries usually differentiate SPE from thromboembolism.

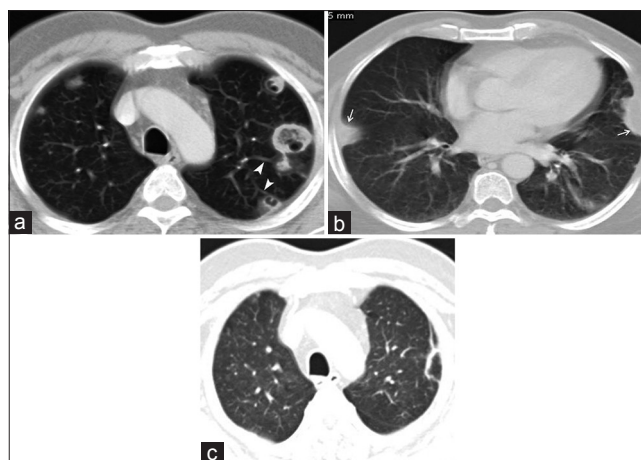


Figure 2: CT of the chest at admission showing bilateral peripheral nodules of variable sizes, cavitations and "feeding vessel sign" (arrowheads in a), and wedged-shaped opacities representing pulmonary infarctions (arrows in b); CT of the chest after 3 months showing complete disappearance of the nodules (c)

Chest radiographic findings of SPE are nonspecific and may be misleading. This includes peripheral infiltrates, haziness, or nodules of varying sizes. Day-to-day variability may be observed in the number, size, or cavitations of the nodules. Pleural effusion is uncommon. SPE is rarely suspected in the initial interpretation of chest radiograph. Chest CT scan is often helpful in characterizing the lesion; it delineates the extent of the disease and potential complications such as extension into the pleural space. The usual CT manifestations of SPE are multiple pulmonary nodules of varying sizes that are located peripherally with or without cavitations. Feeding vessel sign (vessel supplying directly to a nodule) may be found in 60–70% of the patients and heterogeneous subpleural wedge-shaped opacities representing pulmonary infarctions are seen in 70–75% of the patients. Pulmonary infarction and cavitations are more common in occlusion of distal pulmonary arteries.^[1-3]

Treatment of SPE is usually done with appropriate antibiotics. If an indwelling catheter is the source of the infection, it should be removed. If there is no prompt response to this regimen, surgical isolation of the septic vein, if present, should be considered. The role of systemic anticoagulation remains uncertain.^[1-3] In the modern era, clinical outcome of SPE is excellent with use of appropriate antibiotics and timely surgical intervention. Soft-tissue infection should be considered as a potential source of SPE in adults.

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Conflicts of interest

There are no conflicts of interest.

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