Septic Pulmonary Embolism: A Rare and Cataclysmic Complication of Infective Endocarditis

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Abstract

**Background:** Septic pulmonary embolism (SPE) is an uncommonly reported complication of IE requiring a high index of suspicion. SPE is a rare but potentially fatal complication of infective endocarditis (IE). The exact incidence is not known but in one large study in China, the prevalence of septic pulmonary embolism was 5.7% (20/348) among patients with IE [1] with a mean age of 37.5 to 38.1 years [1,2]. There are no pathognomonic clinical manifestations but it most commonly presents with fever, dyspnea, cough or hemoptysis as seen in our patient. Due to its non-specific presentation, it may be under-recognized without a high index of suspicion of this possible complication [2,3].

**Case:** We report a 21-year-old male, former methamphetamine user, complaining of intermittent fever, cough and dyspnea for 6 months. He had a Graham-Steell murmur, grade 3/6 pansystolic murmur at the left lower sternal border, bipedal edema and petechiae on the trunk and extremities. Ceftriaxone and Gentamicin were given empirically for possible infective endocarditis. 2D echocardiography revealed heavy echogenic densities attached to the pulmonic valve cusps (2.0 × 0.79 cm), floating echogenic densities at the tricuspid valve leaflet tips and spontaneous echo contrast. Anticoagulation with Enoxaparin was started. Streptococcus agalactiae grew on blood culture (3 sites). On the 10th hospital day, he had an episode of hemoptysis (~600 ml) requiring endotracheal intubation. CT angiogram of the pulmonary arteries revealed several filling defects adherent to the pulmonary valve (endocarditic lesion), pulmonary embolism/septic emboli involving the left lateral wall of the main pulmonary artery, anterior segmental arteries of both upper lobes, and bilateral lower lung pulmonary arteries. Multiple small nodules, some exhibiting cavitation and the feeding vessel sign, were seen in both mid to lower lungs. Antibiotics were continued. Repeat blood cultures after 2 weeks no longer showed any growth. He was successfully extubated. Valve surgery was advised but no consent was given. Medical management was maximized. He was discharged improved after 2 months.

**Conclusion:** Patients with RSIE are at risk to develop SPE which is an uncommon and potentially fatal complication of IE which may go unrecognized without a high index of suspicion. Early recognition is therefore important.

Keywords: Embolism; Pulmonary; Echocardiography; Endocarditis; Vasculitis

Introduction

Septic pulmonary embolism (SPE) is an uncommon but potentially fatal complication of infective endocarditis (IE). The exact incidence is not known but in one large study in China, the prevalence of septic pulmonary embolism was 5.7% (20/348) among patients with IE [1] with a mean age of 37.5 to 38.1 years [1,2]. There are no pathognomonic clinical manifestations but it most commonly presents with fever, dyspnea, cough or hemoptysis as seen in our patient. Due to its non-specific presentation, it may be under-recognized without a high index of suspicion of this possible complication [2,3].

This is the first reported case in our institution, highlighting the paucity of this complication.

Case

We report a case of a 21-year-old male who complained of a 6-month history of intermittent fever, cough and exertional dyspnea. No consult was sought. The condition was just tolerated. One month prior to admission, he self-medicated with unrecalled antibiotics for a week without improvement. He developed bipedal edema and dyspnea at rest prompting consult at our institution. He has no known comorbid illnesses but had history of methamphetamine use during his teens. On admission, he had stable vital signs (Blood pressure=120/80 mmHg, heart rate=85 beats per minute, respiratory rate=18 beats per minute, Temperature=36°C, Oxygen saturation of 92% at room air). Pertinent physical examination findings include jugular venous distention, bibasal rales, a dynamic precordium, right ventricular heave, Graham-Steell murmur, grade 3/6 pansystolic murmur at the left lower sternal border, bipedal edema and petechiae on the trunk and extremities. He had leukocytosis (14,400/µL, normal range=5,000 to 10,000/µL), thrombocytopenia (73,000/µL, normal range=200,000 to 400,000/µL) and anemia (730 g/L, normal range=140 to 170 g/L). Blood cultures were taken. Empiric antibiotics for infective endocarditis were started (Ceftriaxone 2 g/24 h and Gentamicin 200 mg/24 h intravenously). Roentgenogram showed a suspicious density on the right mid-lung, prominent main pulmonary artery, left atrial and right ventricular prominence (Figure 1). Electrocardiogram showed sinus rhythm, right ventricular hypertrophy with strain pattern (Figure 2). Two-dimensional echocardiography revealed heavy echogenic densities attached to the pulmonic valve cusps (2.0 × 0.79 cm at its widest diameter) and floating echogenic densities at the tricuspid valve leaflet tips with severe tricuspid regurgitation (Figure 3). Spontaneous...
echo contrast was also noted. This prompted anticoagulation with Enoxaparin to be started. After 15 h of incubation, there was growth of *Streptococcus agalactiae* on blood culture (3 sites) which was sensitive to Ceftriaxone. Patient was clinically stable and was recovering well until the 10th hospital day when he had recurrence of dyspnea and an episode of hemoptysis (~600 ml) requiring endotracheal intubation to protect the airway. Bronchoscopy did not reveal any significant findings other than an edematous mucosa and mucosal injury at the hypopharynx with blood clots. The airway was patent with no active bleeding. Computed tomography angiography of the pulmonary arteries was done showing several filling defects adherent to the pulmonary valve (endocarditic lesion) (Figure 4A), pulmonary embolism/septic emboli involving the left lateral wall of the main pulmonary artery, anterior segmental arteries of the right and left upper lobe, and bilateral lower lung pulmonary arteries (Figure 4B). Multiple small nodules, some exhibiting cavitation that display the feeding vessel sign, were seen in both mid to lower lungs, representing septic emboli (Figure 4C). Antibiotics were continued. Repeat blood cultures after 2 weeks no longer showed any growth. He was successfully extubated. Referral to Surgery for vegetectomy and valve replacement was done but patient did not consent to it. Repeat echocardiography showed a decrease in the size of the vegetations. Repeat computed tomography angiography of the pulmonary arteries showed a decrease in the filling defects and septic emboli. Medical management was maximized. He completed Gentamicin for 2 weeks and Ceftriaxone for 6 weeks. He was discharged improved on the 60th hospital day.

**Discussion**

Studies on SPE from IE are limited. Cardioembolic events were reported in 25 patients (28.7%) with definite IE in one local study and SPE was seen during the course of treatment in only 4 cases (4.5%) [4]. In our patient, the presence of right-sided IE was the main predisposing factor for development of SPE. Involvement of the tricuspid valve endocarditis is more common than the pulmonic valve. Our case featured involvement of both valves with involvement of the latter as the main lesion with its very large size. In one large French study involving 390 patients with infective endocarditis, pulmonic valve involvement was reported in only 2 cases [5]. Our patient most likely started to develop SPE prior to admission based on the 6-month history of cough and dyspnea which are symptoms suggestive of pulmonary embolism.

Staphylococcus spp and *Streptococcus spp* are the most common isolated pathogens. Interestingly, *Streptococcus agalactiae* was isolated in our patient on all 3 blood culture sites. This organism has not been commonly described to cause IE. In one case series, patients with IE due to *S. agalactiae* presented with an acute and aggressive course with major arterial embolism, thus, carry the risk of high morbidity and mortality. This infection has been described to be difficult to control. Delays in the management must therefore be avoided [6]. Since our patient self-medicated with unrecalled antibiotics, we suspect that this could have contributed to the less aggressive course seen in our patient.

**Figure 1:** Chest-X-ray showing signs of pulmonary arterio-venous hypertension and a suspicious density on the right mid lung.

**Figure 2:** Electrocardiogram showing sinus rhythm, right ventricular hypertrophy with strain pattern.

**Figure 3:** Transthoracic echocardiography showing (a) heavy echogenic densities attached to the pulmonic valve cusps (2.0 × 0.79 cm at its widest diameter) and (b) fluttering echogenic densities at the tricuspid valve leaflet tips.

Predisposing factors of septic pulmonary embolism in the setting of IE include intravenous drug use (IVDU), valvular heart disease, congenital heart disease and presence of implanted cardiac devices or placement of central lines [1-3]. In our patient, IVDU was the identified predisposing condition.
Chest X-ray findings are not specific and may suggest an infectious process such as pneumonia. Chest computed tomography scan is an important diagnostic modality in confirming one's clinical suspicion of SPE as what was done in our case. Multiple peripheral nodular opacities with or without the presence of cavitations are the most common finding consistent with SPE [2,3,7,8]. The “feeding vessel” sign is a non-specific finding that may also be seen in pulmonary embolism of septic or non-septic etiology. This refers to the presence of a distinct vessel leading directly to a nodule. However, this is not specific for pulmonary embolism as it may be seen in a number of other conditions such as pulmonary infarction, pulmonary vasculitis, pulmonary arteriovenous malformation, and less commonly, angioinvasive pulmonary aspergillosis [7,8]. This highlights the need to correlate CT findings clinically.

The prognosis of isolated RSIE is favorable in up to 80% of cases and surgery may not be necessary. However, when there are >2 valves involved, morbidity and mortality increases. There are limited data in the management of tricuspid valve (TV) and pulmonic valve (PV) endocarditis and cardiac surgery risk assessment tools are limited only to mitral and/or aortic valve involvement [9,10]. In our case, higher risk for perioperative complications are expected due to involvement of both TV and PV with a large vegetation on the latter. Indications for surgery include persistent infection despite appropriate antibiotics, recurrent pulmonary emboli, refractory heart failure and vegetations >1 cm [11].

In our patient's case, a conservative approach was utilized and improved outcome was observed on discharge after 2 months.

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