CASE REPORT

Severe Pulmonic Valve Endocarditis with Septic Pulmonary Embolism in a Teenager: Case Report

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A 14-year-old boy with an uneventful past history suffered from one week of high-grade fever and progressive dyspnea. The patient developed necrotizing pneumonia with massive loculated pleural effusion and septic shock. Echocardiogram revealed a small perimembranous VSD with multiple vegetations at pulmonic valve and both pulmonary arteries. Intravenous antibiotic therapy, mechanical ventilatory support, chest tube placement, and intrapleural fibrinolytic therapy were performed. He was clinically improved after eight weeks of antibiotic therapy.

Keywords: Septic pulmonary embolism; Pulmonic valve endocarditis; Infective endocarditis

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Septic pulmonary embolism (SPE) is a rare disorder in which a pathogen-containing thrombus from a primary infection site embolizes into the pulmonary artery and causes parenchymal infection[1,2]. The common causes include right-sided infective endocarditis in intravenous (IV) drug users[3], infected vascular catheters, and implantable devices[2]. It remains a difficult diagnosis because of non-specific clinical presentations and radiographic features. Early diagnosis and proper treatment provide good outcome[2]. The authors described a case with an unknown small ventricular septal defect (VSD) that presented as necrotizing pneumonia with septic shock and SPE secondary to severe pulmonic valve (PV) endocarditis.

Case Report

A 14-year-old boy presented with fever and dyspnea for one week. He denied having a history of intravenous drug use and history of comorbidities. His vital signs revealed temperature of 42°C, pulse rate of 140 per minute, respiratory rate of 40 per minute, and SpO_2 of 90% in room air. The physical examination showed left subconjunctival hemorrhage, multiple Osler nodes at knuckle of fingers and systolic heart murmur grade IV/VI at left parasternal border. Chest auscultation revealed crackles with decreased breath sounds on both lungs. The patient developed acute respiratory failure and septic shock. Therefore, the patient was urgently transferred to pediatric intensive care unit after endotracheal intubation. Fluid resuscitation, inotropic support, and intravenous antibiotics using meropenem, cloxacillin, and clindamycin were given. The chest radiograph (Figure 1A) revealed cardiomegaly, consolidation with cavities at both lungs and bilateral pleural effusion. In addition, the chest ultrasound demonstrated loculated bilateral pleural effusion. Therefore, to prevent the progression of parapneumonic pleural effusion, thoracostomy tube placement and intrapleural instillation of fibrinolytic drug were performed. Although pleural fluid culture was negative, the profiles showed exudative effusion. Nonetheless, his blood and tracheal suction cultures were positive for methicillin-susceptible *Staphylococcus aureus*. The echocardiogram (Figure 1C) revealed dilation of right heart chambers, restrictive at 5 mm on left to right shunt perimembranous ventricular septal defect (PmVSD), moderate tricuspid regurgitation (TR), thick PV leaflets with moderate pulmonary regurgitation, multiple large mobile hyperechoic

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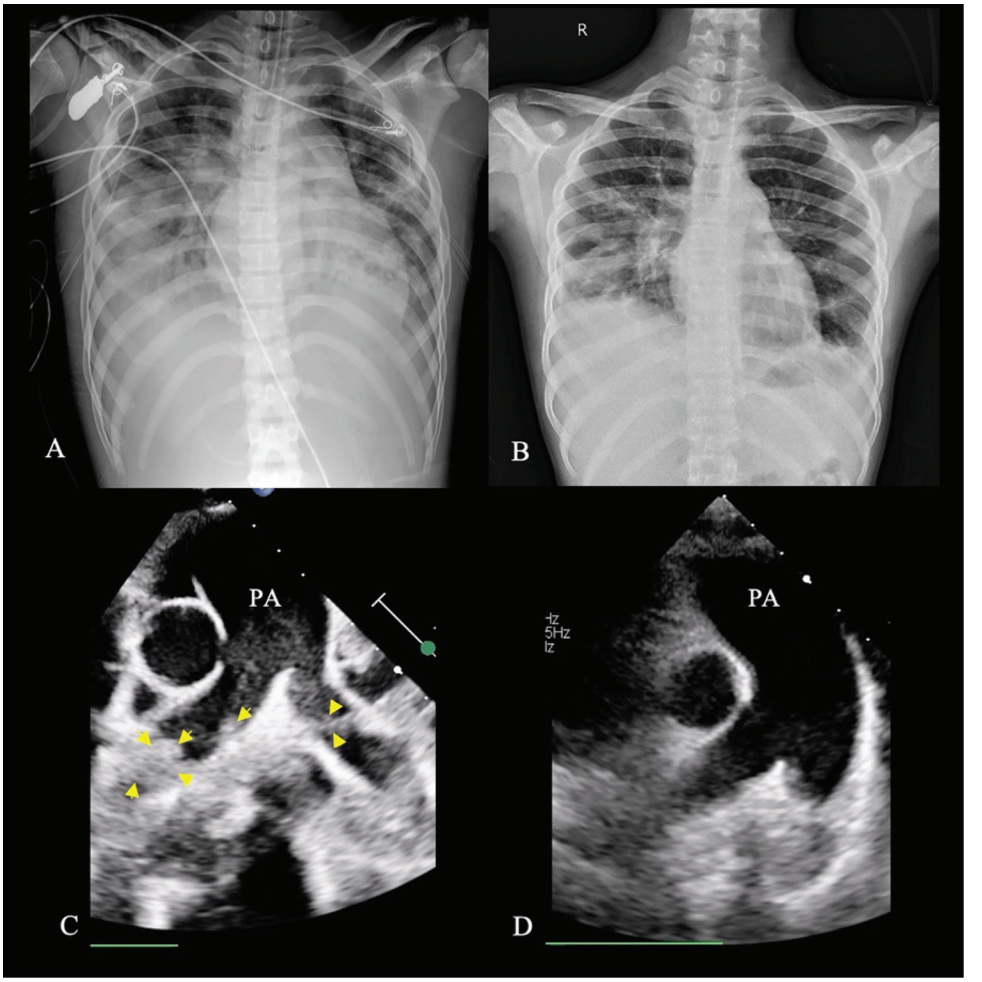

Figure 1. Chest radiograph of the patient on admission (A) demonstrated consolidation with cavitations prominent at both lower lungs, blunt costophrenic angles, and cardiomegaly. Chest radiograph on the discharge day (B) demonstrated cavities at right lower lobe. Echocardiogram in parasternal short axis view of the patient on admission (C) demonstrated multiple hyperechoic masses (arrow heads) on the right and left pulmonary artery. This finding was completely resolved on follow-up echocardiogram before discharge (D).

masses at the right and left pulmonary arteries, left ventricular ejection fraction of 67%, and estimated pulmonary arterial pressure of 75 mmHg from TR pressure gradient. After stabilization, chest computed tomography and angiography (CCTA) was performed. It demonstrated (Figure 2) multiple filling defects at the main pulmonary artery adhered to PV and distal pulmonary arteries, dilated pulmonary trunk, multifocal pulmonary infarction, and necrotizing pneumonitis with non-cavitate nodules in both lungs, and mild pleural effusion. The patient could be extubated within two weeks and received augmented respiratory support with heated humidified high flow nasal cannula (HHHFNC) for an additional five weeks. After six weeks of antibiotics therapy, follow-up chest radiograph (Figure 1B), echocardiogram (Figure 1D), and CCTA showed complete resolution of the vegetations and pulmonary abnormalities. His clinical condition gradually improved. The patient was discharged eight weeks after treatment with cloxacillin. The patient was asymptomatic at 12-weeks of follow-up and planned for transcatheter PmVSD closure in the next four to six months.

Discussion

SPE is an uncommon disorder but is associated with morbidity and mortality. An infected organism from an extrapulmonary site translocate into the systemic venous circulation. The pathogen produces toxin and induces inflammatory mediators, which

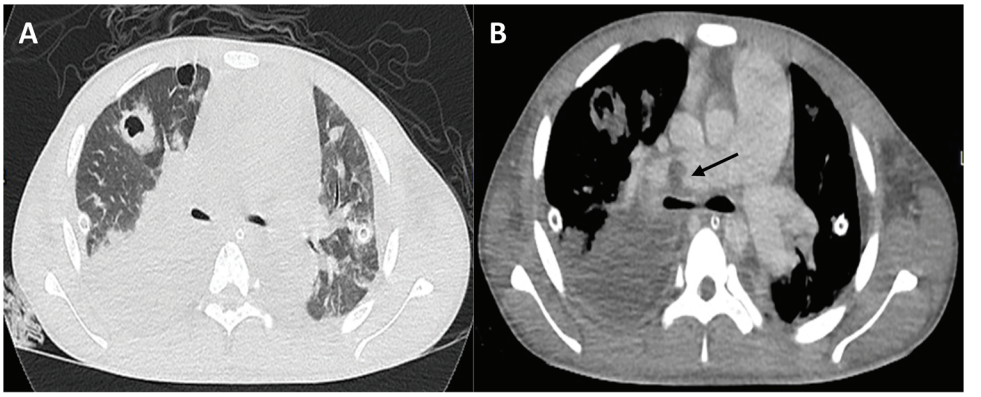

Figure 2. Necrotizing pneumonia with septic pulmonary embolism. (A) A lung window of chest CT shows consolidation right lung, bilateral nodules with cavities. (B) A mediastinal window of chest CT shows filling defect at right main pulmonary artery suggesting pulmonary embolism (arrow).

may promote thrombosis. These infected thrombi embolize into pulmonary circulation resulting in lung parenchymal infection[4]. Right-sided infective endocarditis secondary to intravenous drug use is one of the common primary sites of infection[3]. However, right-sided infective endocarditis is rare, especially PV[5]. There are several predisposing factors for PV infective endocarditis such as central venous catheters, alcoholism, chronic hemodialysis, hepatic transplantation, celiac disease, and systemic infection with bacterial agents[6,7]. Vegetation length greater than 20 mm, fungal origin, and age older than 45 years were associated with high mortality[8,9]. A previous adult case presented with chest pain, cough, and history of PV disease. Echocardiogram revealed pulmonary valve stenosis and vegetation at PV. Blood culture was positive for *Streptococcus* spp. Chest CT demonstrated multiple ill-defined pulmonary nodules with peripheral ground-glass halo and cavitary changes at peripheral zone of both lungs. Surgery was performed after one week of intravenous antibiotic therapy for removal of the vegetation. The patient was treated with antibiotics for six weeks. The pulmonary symptoms gradually improved. The patient was asymptomatic at 3-months follow-up[10]. In the present case, a previously healthy adolescent patient presented with respiratory distress and septic shock. The echocardiogram showed multiple vegetations on the right and left pulmonary artery and small PmVSD. CCTA was compatible with necrotizing pneumonia and septic pulmonary emboli with pleural effusion. In this case, PV endocarditis may be caused by turbulent flow created by untreated small PmVSD, then spread to PV. The patient was treated with intravenous antibiotics, mechanical ventilatory support, chest tube placement, and intrapleural fibrinolytic therapy. His clinical condition improved, and follow-up echocardiogram showed complete resolution of the vegetations without surgery.

The diagnosis of SPE should be considered in bacteremic patients with extrapulmonary source of infection, who developed respiratory symptoms. Chest CT findings include consolidation, ground glass opacities and multiple nodules, often periphery with or without cavitation[4,11]. In addition, tachypnea, low-level oxygen saturation, altered mental status, and segmental or lobar consolidation on CT were found to be independent predictors of in-hospital death[12,13]. On the contrary, the number of lesions and the extent of disease did not differ between the death and survival groups[12].

Empirical antibiotic therapy should be initially started. Then antibiotics can be modified depending on culture results. Thoracentesis and chest tube placement with or without installation of fibrinolytic drug should be considered. Enhanced chest CT examination should be performed to identify the emboli such as blood culture, liver ultrasonography, and echocardiography. Other examinations should be performed to detect the source of the emboli. Early removal of infected lesions, indwelling catheters, and cardiac devices are highly recommended. Some cases may need surgical interventions including removal of infected tissue, valve replacement, and drainage of purulent collection. Surgery for removal of the vegetation should be considered in patients with progressive heart failure, uncontrollable infection, bacterial infection resistant to antibiotics, or vegetation greater than 10 mm[10]. Anticoagulant therapy is controversial because it increases the risk

of bleeding[4,11].

SPE can cause a high mortality rate of 10% to 12%[12,14]. Successful treatment and improve rates were 60% and 28%, respectively[13]. Death is associated with septic shock and multiple organ failure[14]. Therefore, physicians should pay attention not only to the local infection but also to end-organ function to detect and correct shock as early as possible and thereby improve treatment outcome.

Conclusion

The SPE with right-sided infective endocarditis involving PV is rare and difficult to diagnose. SPE should be suspected in patients who have fever with respiratory symptoms and when the chest imaging shows multiple peripheral nodules or cavities with or without pleural effusion[14]. Chest CT helps to identify pulmonary embolism. Investigating the infection source including echocardiogram and blood culture are important for appropriate treatment. Additionally, although antibiotic prophylaxis for bacterial endocarditis in small VSD is not recommended by The American Heart Association[15], detrimental SPE in the present patient urged physician for close follow-up and consider of closing small PmVSD, which can be safely performed percutaneously nowadays[16,17].

What is already known on this topic?

Clinical presentation of SPE is non-specific symptoms and right-sided infective endocarditis in intravenous drug users is one of the common causes of this condition.

What does this study add?

This case report shows that PV endocarditis may be caused by VSD, therefore, physicians should be mindful of SPE when a patient with VSD develops bacteremia and respiratory symptoms.

Ethical approval

The present case report was approved by the Institutional Review Board (REC.020/2565).

Conflicts of interest

The authors declare no conflict of interest.

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