Spontaneous Pneumothorax as a Complication of Septic Pulmonary Embolism in an Intravenous Drug User: A Case Report

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Infective endocarditis has been the major cause of morbidity and mortality among intravenous drug users (IDUs) with infections, mostly involving the tricuspid valve and presenting multiple septic pulmonary embolisms. Numerous pulmonary complications of septic pulmonary embolism have been described, but only a few have reported spontaneous pneumothorax. Our patient, a 23-year-old heroin addict, was hospitalized for tricuspid endocarditis and septic pulmonary embolism. Acute onset of respiratory distress occurred on his seventh hospital day and rapidly resulted in hypoxemia. Immediate bedside chest radiograph demonstrated left pneumothorax. It was thought to be a spontaneous pneumothorax, because he had not undergone any invasive procedure before the occurrence of pneumothorax. His clinical condition improved after the insertion of an intercostal chest tube. He later underwent surgery to replace the tricuspid valve as a result of the large size of the vegetation and poor control of infection. He ultimately survived. Pneumothorax is a possible lethal complication of septic pulmonary embolism in IDUs with right-sided endocarditis and should be considered in such patients when respiratory distress occurs acutely during their hospitalization.

Key Words: endocarditis, intravenous drug user, pneumothorax, pulmonary embolism, *Staphylococcus aureus*

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Infections are the leading cause of morbidity and hospitalization in intravenous drug users (IDUs), accounting for 31% of their admissions [1]. Infective endocarditis (IE) is well known to be the major cause of morbidity and mortality among IDUs with infections. The majority of IE in IDUs is right-sided, mostly involving the tricuspid valve and causing multiple septic pulmonary embolisms. The pulmonary complications of septic pulmonary embolism have been described, including pulmonary infarction, pulmonary abscess, pleural effusion, empyema, and fatal pulmonary hemorrhage caused by rupture of mycotic aneurysms of the pulmonary arteries.

To the best of our knowledge, there have been only three cases in two articles reporting pneumothorax as a complication of septic pulmonary embolism secondary to tricuspid endocarditis in IDUs [2,3]. We present here another case and review the literature.

Case Presentation

A 23-year-old man who had been a heroin addict for the past 2 years, entered our hospital with fever, productive cough, and shortness of breath. His temperature was

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38.6°C (101.7°F) with a respiratory rate of 30 breaths/min and a pulse rate of 136 beats/min. His blood pressure was 77/46 mmHg. On physical examination, wheezing and crackles were noted in both lungs. His white blood cell count was 61,750 cells/μL (normal 4,500–11,000 cells/μL) with a marked left shift. His platelet count was 21,000 cells/μL (normal 150,000–400,000 cells/μL). He had acute renal failure with a blood urea nitrogen level of 215 mg/dL (normal 11–23 mg/dL) and a creatinine level of 6.0 mg/dL (normal 0.6–1.2 mg/dL). Chest radiograph and computed tomography (CT) scan demonstrated bilateral nodular infiltrates with thin-walled cavities (Figure 1). Echocardiography showed a large vegetation 2.1 × 2.6 cm in size in the tricuspid valve with moderate tricuspid regurgitation. A diagnosis of tricuspid endocarditis with septic pulmonary embolism and septic shock was made. He received hemodialysis immediately. The blood tests produced a negative result for human immunodeficiency virus and a positive result for hepatitis C virus. Piperacillin/tazobactam and teicoplanin were administered as the initial antibiotics. Later, three sets of blood culture all revealed methicillin-sensitive *Staphylococcus aureus*. Antibiotic treatment was changed to oxacillin plus gentamicin.

On the third hospital day, his shock and acute renal failure resolved. The white blood cell count was 16,190 cells/μL; however, the patient remained febrile. On the seventh hospital day, he had acute onset of dyspnea with rapid deterioration of oxygenation. Breathing sounds in his left lung diminished. A bedside chest radiograph (Figure 2) confirmed the diagnosis of pneumothorax. He denied any attempt to inject drugs through the central venous access. The central line had been placed in the left subclavian vein on the day of admission, and chest radiographs taken after the procedure did not reveal pneumothorax. Therefore, the pneumothorax was thought to be spontaneous in nature. His respiratory distress improved dramatically after insertion of an intercostal chest tube. The pleural exudate was sterile. Because of the large size of the vegetation and poor control of infection, he underwent surgery to replace the tricuspid valve on the eighth hospital day. Nosocomial *Pseudomonas* spp. infection complicated the postoperative period. He was finally discharged after 85 days of hospitalization.

Discussion

Pneumothorax is a common complication in IDUs. Most cases occur secondary to self-injection of drugs through central venous access [4]. It is also reported to be a complication of *Pneumocystis carinii* pneumonia, miliary tuberculosis, and drug-related bullous lung disease in IDUs. In 1990, Aguado et al first reported a case of bilateral pneumothorax as a complication of septic pulmonary

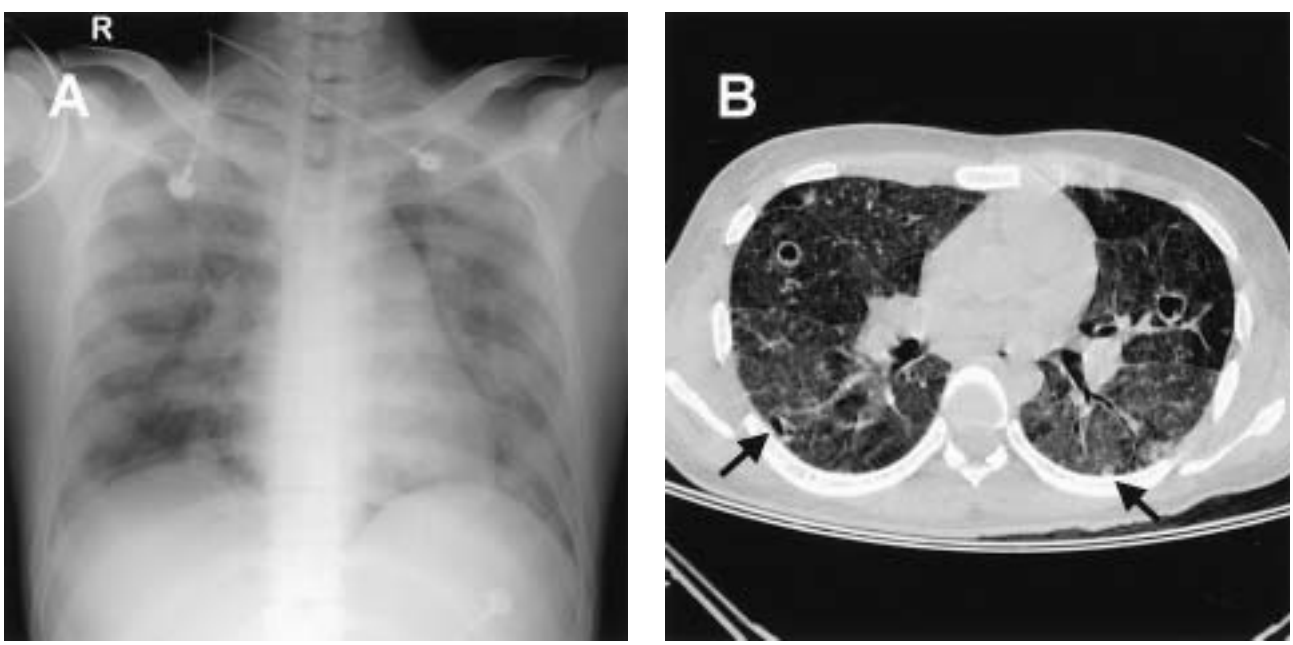

Figure 1. *Image studies on the day of admission. (A) Chest radiograph shows bilateral nodular infiltrations. (B) CT scan reveals multiple thin-walled cavities. Some of them are located subpleurally (arrows).*

embolism in an IDU [2]. After that, only two other cases were reported by Corzo et al [3]. We reviewed all four cases reported, including our patient, and summarized the findings in the Table.

All four cases were young males aged 19–26 years old. They were all addicted to heroin. Pneumothorax was located bilaterally in two cases, right-sided in one case, and left-sided in our case. It occurred 1–15 days after hospitalization (mean, 7 days). The responsible pathogen in three of the four patients was *S. aureus*. The pleural exudate was sterile in two cases and purulent in two cases. All patients had chest tubes inserted,

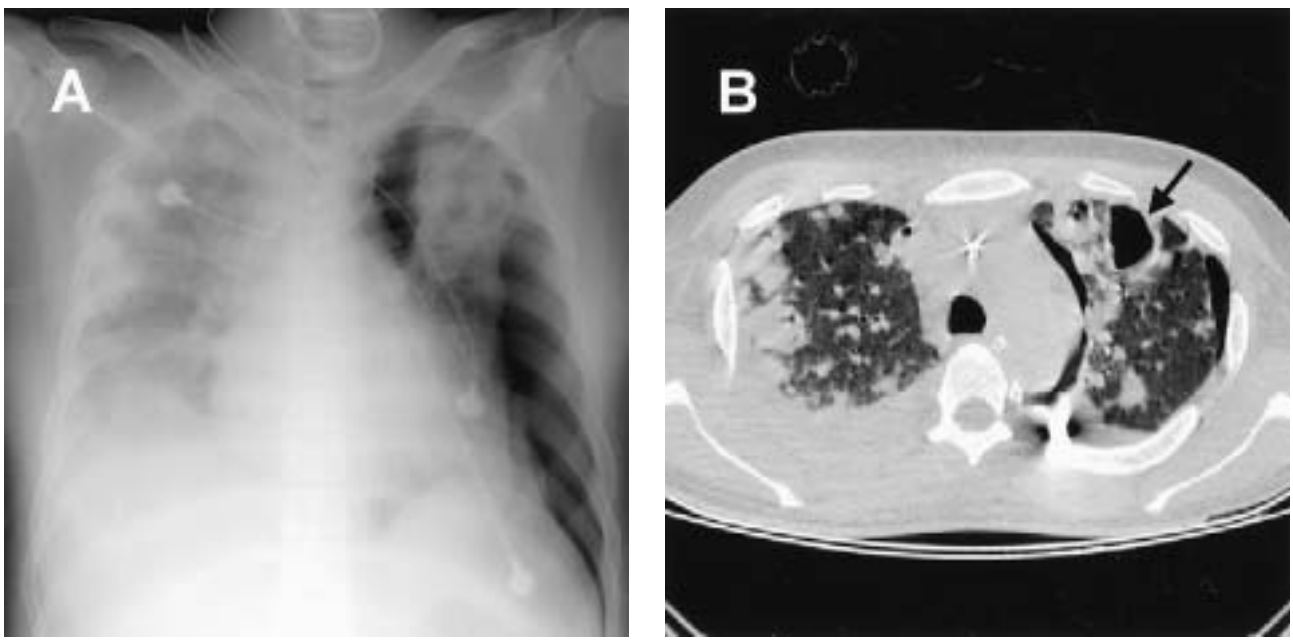

Figure 2. *Image studies on the day of spontaneous pneumothorax. (A) Chest radiograph taken immediately after the onset of respiratory distress demonstrated left pneumothorax. (B) CT scan performed after chest tube insertion showed incomplete lung expansion as well as progression of bilateral nodular infiltrations and subpleural cavities (arrow).*

Table. Summary of four reported intravenous drug users presenting with pneumothorax as a complication of septic pulmonary embolism secondary to tricuspid endocarditis

Author	Aguado et al[2]	Corzo (case 1) et al[3]	Corzo (case 2) et al[3]	Sheu et al
Year published	1990	1992	1992	2005
Patient age	19	23	26	23
Location of pneumothorax	bilateral	bilateral	right	left
Day of onset*	Day 6	Day 15	Day 1	Day 7
Causative agent†	*Staphylococcus aureus*	*S. aureus*	*S. epidermidis*	*S. aureus*
Pleural exudate‡	Sterile	Purulent	Purulent	Sterile
Treatment	Chest tube drainage	Chest tube drainage	Chest tube drainage	Chest tube drainage
Outcome	Survival	Survival	Survival	Survival

*The day of hospitalization that spontaneous pneumothorax occurred. †The causative agent was confirmed by the microorganisms isolated from blood cultures. ‡The microorganisms isolated from purulent pleural exudates were identical to those isolated from blood cultures.

and their outcomes were excellent. The pneumothorax was thought to be due to rupture of the subpleural lesions.

Olazabal et al reported a similar case of spontaneous pneumothorax as a complication of septic pulmonary embolism not secondary to IE (the echocardiogram was normal) in a 24-year-old male with intravenous drug addiction [5]. The pneumothorax occurred on the fifth hospital day and was successfully treated with surgical drainage. The pleural exudate was sterile. The causative agent was *S. aureus*.

S. aureus is the most relevant bacterial pathogen in IDUs, not only in cases of endocarditis but also in thrombophlebitis and soft tissue infections [6,7]. Secondary pulmonary staphylococcal infections (e.g. septic pulmonary embolism) frequently develop in these cases, with a notably higher rate in patients with right-sided endocarditis. Pneumothorax has been reported by many authors to be a common complication of staphylococcal pneumonia, including seven of 34 patients reported by Macfarlane and Rose [8], and three of 44 patients reported by Singla et al [9]. Therefore, we believe there is under-reporting of pneumothorax as an important pulmonary complication when IDUs have IE and septic pulmonary embolism.

In conclusion, spontaneous pneumothorax could be a complication of septic pulmonary embolism in IDUs, particularly when the causative agent is *S. aureus*. Clinicians caring for IDUs with right-sided endocarditis and septic pulmonary embolism should rule out pneumothorax immediately when acute onset of respiratory distress occurs during their hospitalization.

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藥癮患者因敗血性肺栓塞症引起的自發性氣胸

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感染性心內膜炎為藥癮患者併發感染時最為嚴重之病症，主要侵犯心臟之三尖瓣並以多發性敗血性肺栓塞來表現。已有許多敗血性肺栓塞症之肺部併發症被提出，但極少報告有自發性氣胸。我們報告的案例為一位海洛因成癮之二十三歲男性，因右心三尖瓣感染性心內膜炎及敗血性肺栓塞入院治療。病患在住院的第七天突發急性呼吸窘迫，並快速導致低血氧。胸部 X 光檢查顯示有左側氣胸。由於在此之前病患並未接受任何侵入性處置，顯然此為自發性之氣胸。病患之病況在插入胸管治療後立即穩定下來。其後，病患因瓣膜之贅生物（vegetation）過大且感染無法以藥物控制而接受了瓣膜置換手術。最後病患存活下來並已出院。自發性氣胸為藥癮患者併發右心心內膜炎與敗血性肺栓塞時可能發生之一嚴重併發症，當此類病患於住院期間發生急性呼吸窘迫時，應立即排除自發性氣胸之可能性。

關鍵詞：心內膜炎，靜脈注射藥癮患者，氣胸，肺栓塞，金黃色葡萄球菌

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