Doi: 10.5152/TJAR.2019.39924

Case Report
Intensive Care

Turkish Journal of Anaesthesiology & Reanimation

Septic Pulmonary Embolism Associated with Klebsiella spp. Infection in a Patient with Previous ASD Operation

Meltem Genez
Clinic of Anaesthesiology and Reanimation, Gebze Konak Hospital, Kocaeli, Turkey

Cite this article as: Genez M. Septic Pulmonary Embolism Associated with Klebsiella spp. Infection in a Patient with Previous ASD Operation. Turk J Anaesthesiol Reanim 2019; 47(6): 511-4.

Abstract

Septic pulmonary embolism (SPE) is an uncommon disease caused by thromboembolism associated with microorganisms. A 57-year old woman patient who was monitored for a prediagnosis of subileus suffered from sudden respiration problems in the hospital ward. The patient's history included atrial fibrillation and atrioseptal defect (ASD) operation. The patient was admitted to the intensive care unit (ICU) in order to apply non-invasive mechanical ventilation and keep her under observation because of low saturation and loss of consciousness. In the ICU, the patient's temperature was 38.8°C, which led to the diagnosis of sepsis. The chest X-ray showed bilateral and multiple patchy infiltrates. The thorax CT showed right lower pulmonary consolidation and left pulmonary pleuroparenchymal density with nodular shapes. The electrocardiogram showed mild pulmonary hypertension and third-degree tricuspid failure. These features indicated SPE because of the ASD operation in the past. The blood culture showed the presence of *Klebsiella* spp., which is uncommon. A characteristic CT sign is very important for the early diagnosis and treatment of SPE.

Keywords: Embolism, sepsis, septic pulmonary embolism

Introduction

Septic pulmonary embolism (SPE) is an uncommon diagnosis caused by thrombus associated with microorganisms, leading to mechanical obstruction and inflammation of the pulmonary vessels (1, 2). Typical findings on the thorax CT reveal the presence of peripheral nodules with or without cavitation and wedge-shaped peripheral lesions abutting the pleura (2, 3). Most patients with SPE have diagnosis with characteristic CT findings and a primary infection source (2, 4). Histopathological findings do not aid the diagnosis in clinical practice (5). The clinical findings of SPE are non-specific, such as mild respiratory failure and septic shock (6). As a result, a differential diagnosis is important (6). Patients with SPE require intensive care unit (ICU) admission, but it is not reported to be life-threatening (5). However, patients with SPE should be carefully evaluated, and pulmonary embolism should be considered. Therefore, the aim of this study is to elucidate the clinical spectrum, causative pathogens, and radiological screening.

Case Presentation

A 57-year-old woman with subileus had planned an operation because of recurrent abdominal operations. The patient had a past medical history of atrioseptal defect (ASD) operation five years earlier as well as atrial fibrillation (AF). Medical treatment included the administration of warfarin 5 mg (1×1), digoxin 0.25 mg (2×1), Beloc 50 mg (1×1), and Aldactazide 25 mg (1×1).

In the hospital ward, the patient suddenly developed respiratory problems and required ICU admission. In the ICU, the patient's temperature increased to 38°C on the first day, leading to the diagnosis of sepsis. The saturation was 88 with 6 L min^{-1}, and the patient was confused. The electrocardiogram (ECG) showed AF and no ischemic changes.

Corresponding Author: Meltem Genez E-mail: smyrna_mel@yahoo.com
©Copyright 2019 by Turkish Society of Anaesthesiology and Reanimation - Available online at www.jtaics.org
Received: 05.12.2018 Accepted: 13.01.2019

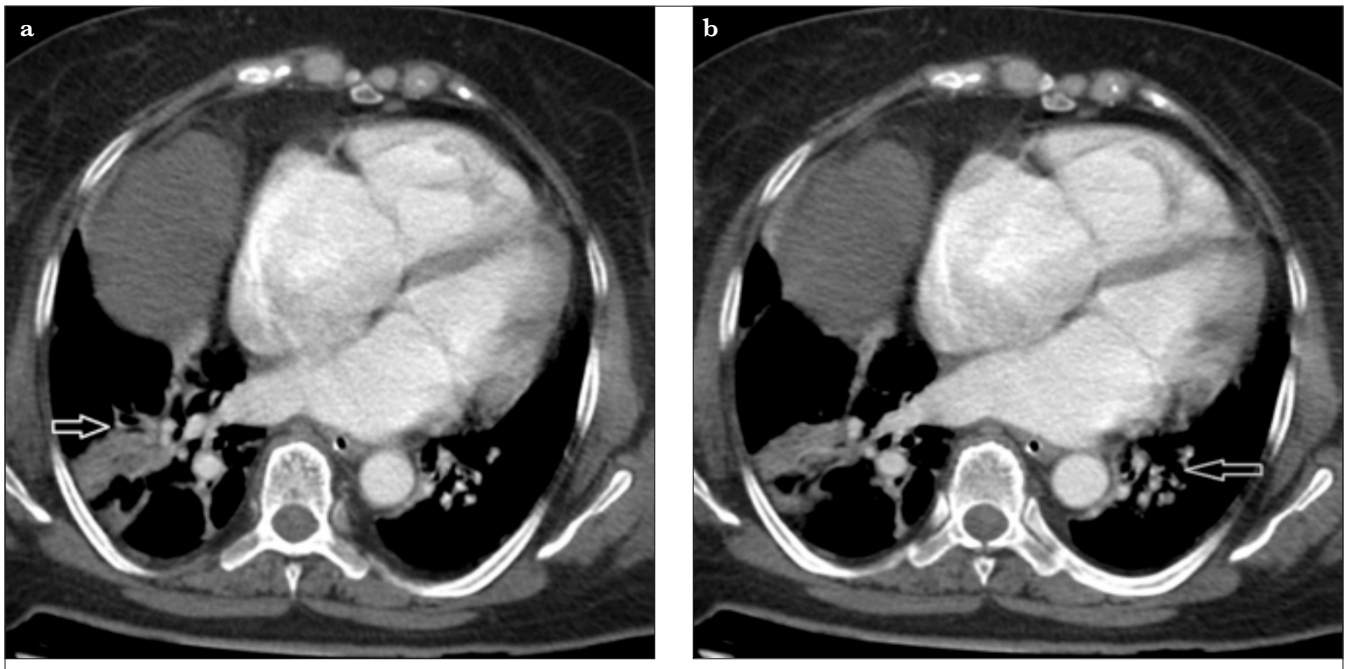

Figure 1. a, b. diffuse fibroatelectatic appearance and consolidation at the base of the pleura of the right lung (a). Nodular-shaped pleuroparenchymal density in the left lung (b)

The pulse was 185/min; blood pressure was 194/91 mm Hg. After then hypotension occurred and norepinephrine infusion was initiated. Arterial blood had a pH of 7.34, pCO_2 of 36.0 mm Hg, and pO_2 of 57.4 mm Hg; the lactate and glucose levels were 4.9 mEq L^{-1} and 65 mg dL^{-1}, respectively. Non-invasive mechanical ventilation (NIMV) was applied because the patient's respiration rate was 33/min. Controlled arterial blood sample showed a pH of 7.50, pCO_2 of 30.2 mmHg, and pO_2 of 66.7 mm Hg; the lactate level was 3.9 mEq L^{-1}.

The chest X-ray showed bilateral patched infiltrations with multiple localisations. The contrast-enhanced chest CT showed diffuse fibroatelectatic appearance and consolidation at the base of the pleura of the right lung and nodular-shaped pleuroparenchymal density in the left lung apex (Figure 1a, b).

The increased pulmonary truncus and main pulmonary arterial measuring were determined in favour of pulmonary hypertension. The treatment continued with the SPE diagnosis algorithm. Echocardiography revealed mild pulmonary hypertension accompanied by third-degree tricuspid failure. The transition leading to the consideration of a possible defect in the atrial septum was observed. This situation is thought to have developed secondary to the ASD operation.

On the 2nd day in the ICU, the patient was intubated due to inadequate oxygenation with NIMV and ventilated with 70% FiO_2 on SIMV mode. Initially, the white blood cell and c-reactive protein values were normal; however, during monitoring, it was observed that both these were increased. The antibiotic therapy of meropenem 1 g (3×2) was planned. The SPE diagnosis algorithm heparin infusion (25000 IU/1000 mL PS) was initiated. With the aim of maintaining the active partial thromboplastin time values in the interval from 75 to 90 s, the heparin infusion dose was set accordingly. Steradine support continued at a dose of 4 mcg kg^{-1} h^{-1}. On the 5th day of observation, the patient was extubated and intermittent NIMV support was ensured. The subileus with planned surgery threated with medications. No fever peak was observed. From the 7th day, NIMV was not applied. On the 9th day, heparin and norepinephrine infusions were stopped. For SPE treatment, enoxaparin 0.8 mL (2×1) was initiated, and the patient was discharged to the ward after the completion of a 14-day meropenem treatment.

The study was completed when the consent form was signed by the first-degree relatives of the patient due to the loss of consciousness in the patient.

Discussion

The symptoms of SPE are not specific and involve respiratory symptoms such as dyspnoea, chest pain, and cough, accompanied by bacteraemia. Fever linked to bacteraemia was identified in 85% patients (6).

For the diagnosis, the findings on the CT and direct radiography were similar to those from the CT superior to the radiog-

raphy, revealing the presence of difficult-to-see lesions in the feeder veins (6). Cavitation is a significant finding, revealing that infarctus has an infectious source (7). In addition, with or without cavitations on the CT, the presence of peripheral nodular infiltrations aids the diagnosis (2). With regard to the tomography, it might get confused with pneumonia due to the presence of multiple nodules in the cavity and a frosted-glass appearance. One study observed opacity in the feeder vein in 90% patients and the formation of a peripheral patch in 75% patients. In 80% patients, there may be a nodular appearance without cavity, with 65% having an identified cavity (4). In our patient, a cavity was not observed on the CT, but a nodular appearance was present (Figure 1b).

Studies in the recent years have found that *S. aureus* is the most commonly isolated vector in the SPE aetiology of tricuspid valve infective endocarditis and pneumonia (4). Another study by Chou et al. (5) identified the most commonly isolated vectors as *K. pneumoniae* (50%), *S. aureus* (35%), and *P. aeruginosa* (5%). A study described the isolation of methicillin-susceptible staphylococcus infection (MSSA) in 18.9% cases, with methicillin-resistant staphylococcus infection (MRSA) found in 8.09% cases (6). There are SPE cases without any proliferation in the blood cultures (7). For our patient, the microorganism proliferating in the blood culture was identified as to be *Klebsiella spp*.

Liver abscess linked to the *K. pneumoniae* infection is the most common (40%), with pneumonia and tricuspid valve infective endocarditis (2), as well as the rarer Leiner's syndrome (8) and periodontal diseases (9), identified to cause SPE. Chou et al. (4) identified the causes of SPE as liver abscess (40%), pneumonia (25%), tricuspid valve infective endocarditis (15%), renal abscess (10%), and soft tissue abscess (5%). A review reported vegetation in 48.6% cases, with 86% cases having tricuspid valve vegetation (6). A study by Park et al. (10) explained an SPE case developing because of VSD defects. Musci et al. (11) reported that a prognosis was better for patients with isolated right-sided endocarditis and that it responded to a simple antibiotic treatment. Our case had a history of ASD operation in the past. The ECG found third-degree tricuspid valve failure along with a mild degree of pulmonary hypertension, reducing the chances of septic embolism being linked to the tricuspid valve infective endocarditis. The patient's ileus tableau was caused by *Klebsiella* bacteraemia along with septic embolism in circulation that formed after the ASD operation, passing partial fenestration, and considered to cause the pulmonary embolism tableau.

Considering the high mortality rates for cases with septic shock, it is appropriate to begin the antibiotic treatment at the earliest possible time for such patients. For this, the most commonly used antibiotic groups are penicillin and cephalosporins (6). Antibiotic treatment durations for patients are between 4 and 9 weeks. For MSSA treatment, vancomycin was not found to be superior to nafcillin or oxacillin (12). Patients in which MRSA was isolated in the blood cultures should first undergo the removal of the infected tissue along with an effective antibiotic treatment. For appropriate antibiotherapy, 4–6 weeks of use of vancomycin or daptomycin is recommended; the addition of gentamicin to vancomycin is not recommended (13). Due to the identification of the *Klebsiella* infection in the blood culture from our patient, after 14 days of meropenem treatment, the patient began treatments with tazocin and amikacin under ward surveillance.

It is important to evaluate specific thorax CT findings for a differential diagnosis in SPE cases. Septic embolism may develop subsequent to liver abscess, tricuspid endocarditis, and pneumonia, with mortality rates known to be high among cases with a pneumonia source. SPE should be considered for patients with this diagnosis. It is noteworthy that antibiotic treatment in the early period might be lifesaving.

Informed Consent: Written informed consent was obtained from patients' parents who participated in this case.

Peer-review: Externally peer-reviewed.

Conflict of Interest: The author have no conflicts of interest to declare.

Financial Disclosure: The author declared that this study has received no financial support.

References

1. Bach AG, Restrepo CS, Abbas J, Villanueva A, Lorenzo Dus MJ, Schöpf R, et al. Imaging of nonthrombotic pulmonary embolism: biological materials, nonbiological materials, and foreign bodies. Eur J Radiol 2013; 82: 120-41. **[CrossRef]**
2. Cook RJ, Ashton RW, Aughenbaugh GL, Ryu JH. Septic pulmonary embolism: presenting features and clinical course of 14 patients. Chest 2005; 128: 162-6. **[CrossRef]**
3. Iwasaki Y, Nagata K, Nakanishi M, Natuhara A, Harada H, Kubota Y, et al. Spiral CT findings in septic pulmonary emboli. Eur J Radiol 2001; 37: 190-4. **[CrossRef]**
4. Chou DW, Wu SL, Chung KM, Han SC, Cheung BM. Septic pulmonary embolism requiring critical care: clinicaradiological spectrum, causative pathogens and outcomes. Clinics 2016; 71: 562-9. **[CrossRef]**
5. Chou DW, Wu SL, Chung KM, Han SC. Septic pulmonary embolism caused by a Klebsiella pneumoniae liver abscess: clinical characteristics, imaging findings, and clinical courses. Clinics 2015; 70: 400-7. **[CrossRef]**
6. Ye R, Zhao Li, Wang C, Wu X, Yan H. Clinical characteristics of septic pulmonary embolism in adults: A systematic rewiew. Resp Med 2014; 108: 1-8. **[CrossRef]**

7. Bozkus F, Dikmen N, Atilla N, Arpağ H, Kahraman H. Septic pulmonary embolism,a case report. J Contemp Med 2016; 6: 76-9.
8. Riordan T, Wilson M. Lemierre's syndrome: more than a historical curiosa. Postgrad Med J 2004; 80: 328-34. **[CrossRef]**
9. Shiota Y, Arikita H, Horita N, Hiyama J, Ono T, Ohkawa S, et al. Septic pulmonary embolism associated with periodontal disease. Chest 2002; 121: 652-4. **[CrossRef]**
10. Park HE, Cho GY, Kim HK, Kim YJ, Sohn DW. Pulmonary valve endocarditis with septic pulmonary thromboembolism in a patient with ventricular septal defect. J Cardiovasc Ultrasound 2009; 17: 138-40. **[CrossRef]**
11. Musci M, Siniawski H, Pasic M, Grauhan O, Weng Y, Meyer R, et al. Surgical treatment of right-sided active infective endocarditis with or without involvement of left heart; 20-year single center experience. Eur J Cardiothorac Surg 2007; 32: 118-25. **[CrossRef]**
12. Zuo LE, Guo S. Septic pulmonary embolism in intravenous drug users. Chin J Tuberc Respir Dis 2007; 30: 569-72.
13. Wang WY, Zeng YM. Septic pulmonary embolism: report of three cases and literature review. Int J Respir 2010; 30: 592-6.