Recurrent Septic Pulmonary Embolism Related to an Implanted Central Venous Access Port Device

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To the Editor: Septic pulmonary embolism (SPE) is a rare type of nonthrombotic pulmonary embolism, in which, emboli-containing pathogens and purulent material admixed with fibrin thrombus embolize to the pulmonary artery, causing pulmonary embolism with focal lung infection. Embolization from this complex results in stenosis and/or obstruction of small pulmonary vessels, which frequently causes pulmonary infection. SPE is usually associated with the use of infected central venous catheters and with endocarditis, septic thrombophlebitis (including Lemierre's syndrome), and oropharyngeal infection.[1] The use of central venous access port devices (CVAPDs) has increased rapidly worldwide because of their convenience and safety for patients who need repeated blood sampling or long-term continuous intravenous therapy such as chemotherapy, transfusion of blood products, fluid hydration, or parenteral nutrition. Due to the increasing use of intravascular devices and catheters, more catheter-related and device-related SPEs have been reported. Here, we report a case, in which, a CVAPD was infected with *Staphylococcus epidermidis,* causing recurrent SPE.

A 38-year-old female was admitted to our hospital with fever and chill that had begun 3 days earlier. She had a previous history of breast cancer and SPE. Her first episode of SPE (described below) had been treated at our hospital 6 months before the current admission. Her breast cancer was treated with total mastectomy followed by adjuvant chemotherapy (adriamycin and docetaxel). To facilitate adjuvant chemotherapy, a CVAPD had been place implanted in her left subclavian vein 10 months before this admission.

The first episode of SPE – 6 months before the current admission, she presented to our hospital with fever and dyspnea. Her vital signs included blood pressure of 110/69 mmHg, body temperature of 38.2°C, heart rate of 120 beats/min, and respiratory rate of 26 breaths/min. Arterial blood gas analysis (ABGA) showed pH 7.40, pCO_2 32.5 mmHg, PaO_2 68.0 mmHg, HCO_3 21.3 mmol/L, and SpO_2 90% with oxygen supplied through a nasal prong at a flow rate of 2 L/min. The complete blood count results were as follows: leukocytes, 2240/mm^3 (segmented neutrophils, 44.3%; lymphocytes, 33.5%); hemoglobin, 86 g/L; hematocrit, 26.4%; and platelets, 126,000/mm^3. The blood chemistry results were blood urea nitrogen (BUN), 80 mg/L; creatinine, 5.9 mg/L; aspartate transaminase, 16 U/L; alanine transaminase, 19 U/L; total bilirubin, 3.5 mg/L; lactate dehydrogenase, 182 U/L; C-reactive protein, 75.2 mg/L; and procalcitonin, 16.59 ng/ml. Chest radiography revealed focal nodular opacity in the right middle lung zone. The CVAPD was visible in its implanted state [Figure 1a]. Chest computed tomography (CT) revealed multiple nodular and cavitary consolidations of varying size in both lungs particularly on the right lower lobe (RLL) [Figure 1b–1d]. Flexible bronchoscopy with bronchoalveolar lavage (BAL) for microbial evaluation was performed in the laterobasal segmental bronchus of the RLL. Empirical intravenous antibiotic therapy was initiated with piperacillin/tazobactam and levofloxacin. On the 3rd hospital day, her BAL fluid and blood culture (through both the peripheral vessel and the CVAPD) were positive for methicillin-resistant *S. epidermidis*. She was diagnosed with SPE related to sepsis caused by CVAPD infection. The CVAPD was removed promptly, and empirical antibiotic therapy was stopped. She was started on intravenous vancomycin. Transthoracic and transesophageal echocardiography (TTE and TEE, respectively) showed no vegetation and normal left ventricular systolic function with a normal heart valve. After 3 weeks of the intravenous vancomycin therapy, the negative conversion was achieved in her blood culture. Improvement was detected on clinical and laboratory tests and on radiologic images [Figure 1e]; thus, the patient was discharged. At 1 month after discharge, a CVAPD was reinserted because venous access was difficult.

Second episode of SPE after 6 months – on the day of admission, her vital signs included blood pressure of 80/50 mmHg, body temperature of 39.1°C, heart rate of 144 beats/min, and respiratory rate of 22 breaths/min. Chest auscultation revealed crackles in the right middle and lower lung fields. The ABGA showed pH 7.40, pCO_2 32.0 mmHg, PaO_2 70.4 mmHg, HCO_3 20.8 mmol/L, and

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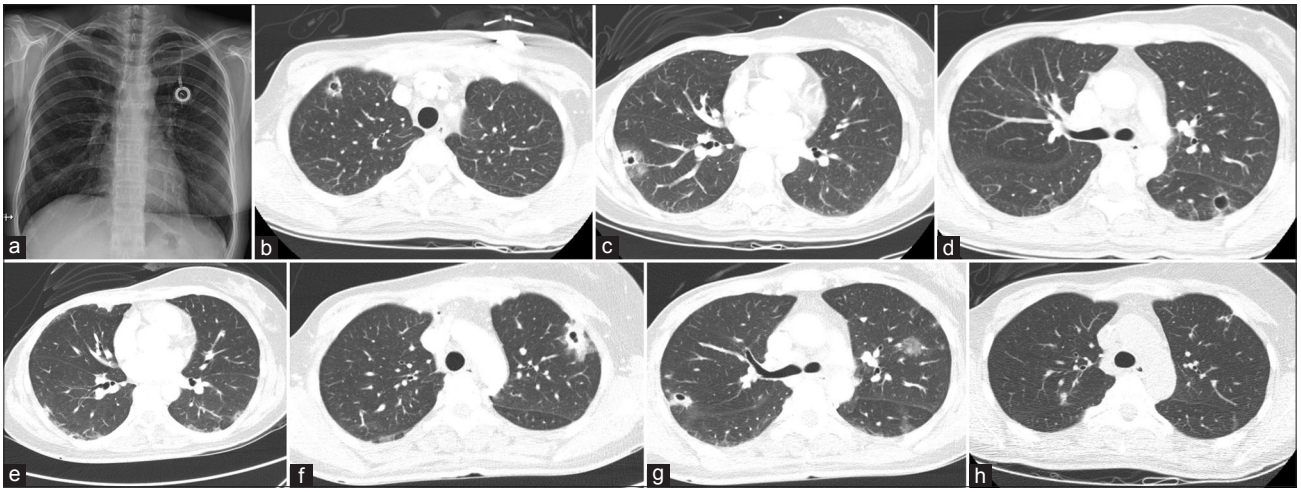

Figure 1: Chest radiography of this patient. (a) Initial chest radiography for the first episode of SPE. The chest radiograph revealed multifocal patchy opacities in both lungs. The CVAPD insertion site is also visible. (b-d) Initial chest CT of the first episode of SPE revealed multiple nodules associated with cavitary changes in both lungs particularly on the right upper lobe, right lower lobe, and left lower lobe. (e) On hospital day 21 of the first episode of SPE, chest CT showed the improvement in multiple nodules and cavitary formations in both lungs. (f and g) Initial chest CT of the second episode of SPE. Chest CT revealed multifocal variable-sized ill-defined nodular consolidations, some with cavitary formations, in both lungs. (h) On hospital day 21 of the second episode of SPE, chest CT showed improvement of the multifocal nodular consolidations, including those with cavity formations, in both lungs. SPE: Septic pulmonary embolism; CVAPD: Central venous access port device; CT: Computed tomography.

SpO_2 96% on room air. The complete blood count results were as follows: leukocytes, 3620/mm^3 (segmented neutrophils, 61.3% and lymphocytes, 37.3%); hemoglobin 105 g/L; hematocrit, 31.4%; and platelets, 151,000/mm^3. The blood chemistry results were BUN, 90 mg/L; creatinine, 7.9 mg/L; aspartate transaminase, 22 U/L; alanine transaminase, 15 U/L; total bilirubin, 3.1 mg/L; lactate dehydrogenase, 213 U/L; creatine phosphokinase, 49 U/L; C-reactive protein, 59.4 mg/L; and procalcitonin, 23.62 ng/ml. The prothrombin time (PT), international normalized ratio, and activated partial thromboplastin time (aPTT) were 19.0 s, 1.66, and 27.0 s, respectively. Chest CT revealed multifocal variable-sized ill-defined nodular consolidations, some with cavity formations, in both lungs, but particularly on the left upper lobe [Figure 1f and 1g]. Empirical intravenous antibiotic therapy was initiated with piperacillin/tazobactam and vancomycin. On the fourth hospital day, her blood cultures, through both the peripheral vessel and the CVAPD, were positive for methicillin-susceptible *S. epidermidis*. We suspected SPE related to the CVAPD; therefore, the CVAPD was removed promptly, and antibiotic therapy was switched to ampicillin/sulbactam. After 3 weeks of intravenous ampicillin/sulbactam administration, her symptoms improved, as did the cavitary lesions initially observed on chest CT [Figure 1h]. Her blood culture was no longer positive for methicillin-susceptible *S. epidermidis*. The patient was discharged on hospital day 22 without complications. She continued oral antibiotics (amoxicillin/clavulanic acid) for 7 additional days.

An implanted CVAPD is commonly used to maintain long-term central venous access. For patients in whom peripheral venous access is difficult such as breast cancer, obese, and elderly patients, CVAPD should be considered at an early stage of treatment. CVAPD implantation minimizes the need for frequent vascular access, improving the patient's quality of life. In cancer patients who exhibit poor venous access, refusal to undergo port implantation can result in the discontinuation of chemotherapy and, in the worst-case scenarios, can lead to peripheral vasculitis or the extravasation of anticancer drugs. A CVAPD needs no external dressing, and allows the patient to continue with their normal activities; this has resulted in increased clinical demand for CVAPDs. Nonetheless, CVAPDs are not free from complications. Complications, such as infection, thrombosis, and catheter breakage associated with CVAPD use, have been reported in various diseases. Pocket infection, port-related bacteremia, and thrombotic complications are critical problems associated with the long-term use of CVAPDs. However, the incidence rates of port-related infections are generally low.[2] Biffi *et al*. conducted two prospective observational studies on patients with CVAPD.[3,4] In these studies, port pocket infection occurred at a rate of 0.53% (0.01 episodes/1000 days of use) to 1.4% (0.05 episodes/1000 days of use), and port-related bacteremia occurred at a rate of 0.8% (0.016/1000 days of use) to 1.4% (0.05 episodes/1000 days of use).

SPE is a relatively rare disease caused by infective emboli arising from either septicemia or a generalized focus of infection, often resulting in occlusion of the pulmonary artery. Its clinical features are usually nonspecific, such that the diagnosis is typically difficult to establish.[1] The diagnosis of SPE is made based on clinical manifestations, culture results, and radiographic evidence of pulmonary embolism. Bilateral multiple nodular opacity, a patchy shadow, and small cavitary lesions are often observed on chest radiography; however, these observations do not constitute a basis for definitive diagnosis. For a detailed evaluation of SPE, chest CT is effective. In 30% of cases, the diagnosis of SPE is first suggested by CT findings.[5] A study including 168 cases of SPE by Ye *et al*. showed that multiple nodular opacities (66%) were the most commonly observed radiographic sign, followed by cavitary lesions (56%), local infiltrations (36%), pleural effusions (30%), feeding vessel sign (28%), and peripheral wedge-shaped lesions (17%).[1] As the chest CT findings are often distinctive, recognition thereof early in the disease course should permit the diagnosis and prompt institution of therapy.[6]

The probable source of SPE, in this case, was bloodstream infection related to the CVAPD. During both episodes of SPE, *S. epidermidis* was identified in blood culture through the

peripheral vessel and the CVAPD. Furthermore, when an SPE lesion develops into a microabscess or a focal abscess, we can identify the responsible pathogen from BAL fluid collected from the abscessed lesion. In this case, we obtained the BAL fluid from the lobe that contained the embolic lesion. Early diagnosis and prompt administration of appropriate antibiotics are important factors in the successful treatment of patients with SPE because sepsis carries a high risk of death and a high complication rate.[1,6] When a CVAPD-related infection occurs, device removal or a conservative approach should be performed. In conclusion, here, we report a very rare case of recurrent SPE related to an implanted CVAPD, which was successfully treated with antibiotics. Early diagnosis, appropriate antibiotic therapy, and control of the infection source are essential for successful treatment and improved prognosis of SPE. Physicians should be aware of the possibility of SPE related to CVAPD infection in CVAPD-inserted patients. In these unusual cases of SPE, physicians should make a conscious effort to identify the pathogen responsible such as by collecting BAL fluid samples and performing appropriate blood cultures through the peripheral vessel and indwelling catheter.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initial will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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