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CASE REPORT

CLINICAL CASE

Localized Pulmonary Edema Secondary to Pulmonary Embolism

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ABSTRACT

A 47-year-old man had localized pulmonary edema (LPE) and a massive pulmonary embolism. The cause of LPE was believed to be a high blood supply to the spared pulmonary artery territories without a thrombus. The patient was successfully treated with unfractionated heparin and thrombolytic agents. (J Am Coll Cardiol Case Rep 2024;29:102332)

HISTORY OF PRESENTATION

A 47-year-old man presented to the emergency department with worsening dyspnea on exertion for 3 weeks, chest pain, and pinkish sputum. On the day of the visit, the degree of his dyspnea worsened suddenly, and it became noticeable at rest. On arrival at the emergency department, his chest pain had resolved. However, he presented with severe respiratory failure. His blood pressure was 159/119 mm Hg, his pulse was 126 beats/min, his body temperature was 37.7 °C, his respiratory rate was 36 breaths/min, his oxygen saturation (Spo_2) was 81% using a reservoir mask at 15 L/min, and arterial blood gas values suggested respiratory alkalosis and a widened alveolar-arterial oxygen gradient (Supplemental Table 1). Crackles were auscultated in the left upper lung field.

LEARNING OBJECTIVES

- To be able to make a differential diagnosis of LPE, considering the patient's medical history.
- To understand the mechanism and management of LPE secondary to pulmonary embolism.

PAST MEDICAL HISTORY

The patient had a history of hypertension and bronchial asthma. He was receiving amlodipine, montelukast, and fluticasone furoate. In addition, he had left rib fractures resulting from a fall 1 month before his presentation and that had been managed conservatively. The patient reported a recent decrease in his activity because of the fractures, and he was seated indoors for the whole day, except when sleeping.

DIFFERENTIAL DIAGNOSIS

On the basis of the patient's dyspnea, pinkish sputum, respiratory failure, prolonged inactivity, mild fever, and history of trauma, the differential diagnoses included pulmonary embolism, pneumonia, pulmonary alveolar hemorrhage, and lung contusion. Mitral regurgitation should also be considered in patients with localized pulmonary edema (LPE) if regurgitated blood flow is blown into

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ABBREVIATIONS AND ACRONYMS

CECT = contrast-enhanced computed tomography

ECG = electrocardiogram

LPE = localized pulmonary edema

LVEDD = left ventricular end-diastolic diameter

RVEDD = right ventricular end-diastolic diameter

t-PA = tissue-type plasminogen activator

Spo_2 = oxygen saturation

TRPG = tricuspid regurgitation pressure gradient

TTE = transthoracic echocardiography

the pulmonary vein, thus causing localized pulmonary consolidation associated with the pulmonary vein territory.

INVESTIGATIONS

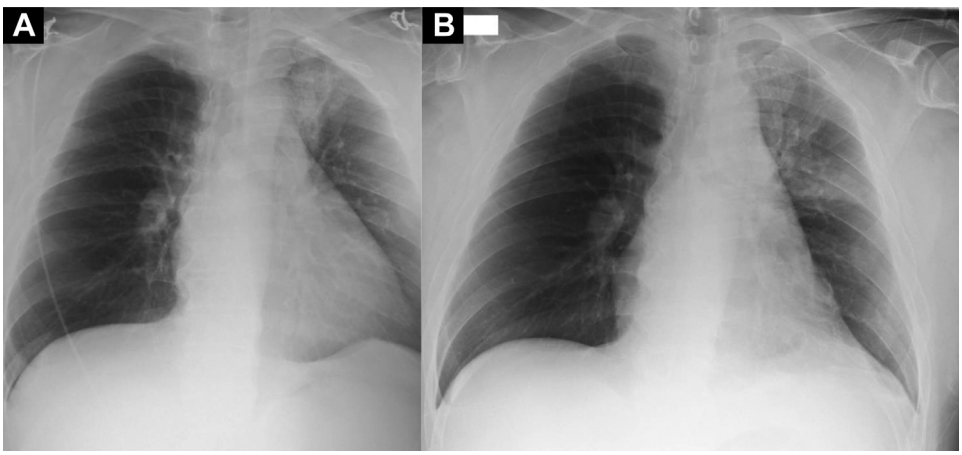
Blood test results revealed lactic acidosis with elevated levels of troponin I, N-terminal pro-B-type natriuretic peptide, and C-reactive protein (0.039 ng/mL, 5,200 pg/mL, and 3.95 mg/dL, respectively). We did not measure procalcitonin. Chest radiography revealed increased right-sided translucency and consolidation in the left upper lung field (**Figures 1A and 1B**). The electrocardiogram (ECG) showed sinus tachycardia, T-wave inversions in the right precordial leads (V_1-V_4) and inferior leads (II, III, and aVF), a deep S-wave in lead I, a Q-wave in lead III, and clockwise rotation. The QRS interval transition zone was V_5 (Supplemental Figure 1A). Transthoracic echocardiography (TTE) revealed normal left ventricular function and no mitral regurgitation, although a dilated right ventricle, an elevated tricuspid regurgitation pressure gradient (TRPG) (84 mm Hg), an elevated right ventricular end-diastolic diameter-to-left ventricular end-diastolic diameter ratio (RVEDD/LVEDD ratio, 1.42; RVEDD, 47 mm), and a McConnell sign were observed, suggesting significant right-sided heart overload (Video 1). Contrast-enhanced computed tomography (CECT) detected regional infiltrative shadows in the left upper lobe. Although the left upper pulmonary artery remained patent, thrombotic occlusions were noted in the other pulmonary artery branches (**Figures 2A to 2F**, Videos 2A and 2B). CECT revealed deep vein thrombosis in the left popliteal vein. LPE as a consequence of massive pulmonary embolism was diagnosed.

MANAGEMENT

The patient received oxygen through a high-flow nasal cannula and a continuous unfractionated heparin infusion to maintain an activated partial thromboplastin time within 2 to 3 times the upper normal limit. Antibiotics were also administered because the possibility of aspiration or bacterial pneumonia could not be excluded. The following day, the patient continued to experience respiratory distress, and the oxygenation levels or A-a DO_2 remained insufficient (Supplemental Table 1). On chest radiography, the left upper lung consolidation had not changed (**Figure 1B**). No signs of bleeding complications, such as alveolar hemorrhage, were observed, and the patient became afebrile. Tissue-type plasminogen activator (t-PA) was administered (13,750 mg/kg; 1,375,000 IU). The day after t-PA administration, the patient's oxygenation had significantly improved, and the respiratory distress resolved (respiratory rate, 18 breaths/min; arterial blood gas on 6 L/min oxygen through a facial mask: pH, 7.44; Pao_2, 95 mm Hg; $Paco_2$, 31 mm Hg; bicarbonate, 21 mmol/L; base excess, −1.3 mmol/L; Spo_2, 97%).

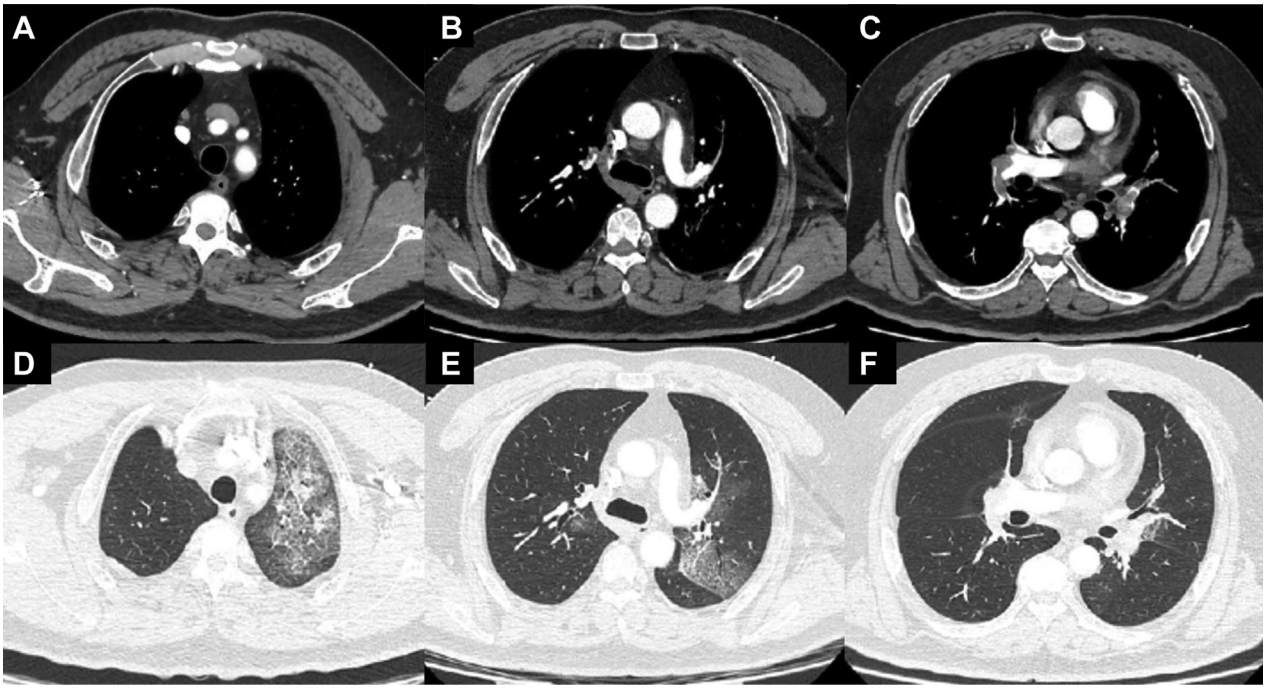
On the fourth day after admission, oxygen supplementation was no longer necessary, and the antithrombotic regimen was switched from

FIGURE 1 Chest Radiography



(A) Chest radiography on admission. (B) One day after admission.

FIGURE 2 Contrast-Enhanced Computed Tomography on Admission



(A) The mediastinal window, upper lung. (B) Mediastinal window, pulmonary artery trunk level. (C) Mediastinal window, middle pulmonary artery trunk level. (D) Lung window, upper lung. (E) Lung window, pulmonary artery trunk level. (F) Lung window, middle pulmonary artery trunk level.

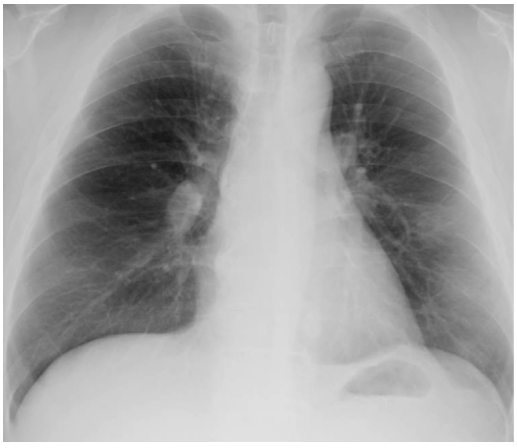
continuous heparin infusion to oral rivaroxaban (30 mg/day). Chest radiography showed gradual normalization of the increased right-sided translucency and consolidations in the upper left lung field (**Figure 3**). The ECG showed slight resolution of the T-wave inversion in the inferior leads (Supplemental Figure 2). On follow-up CECT before discharge, improved blood flow was observed in the embolized pulmonary arteries, and the pulmonary edema in the left upper lung had disappeared (**Figures 4A to 4F**, Videos 3A and 3B). TTE before discharge showed improvement in the right ventricular enlargement, resolution of the McConnell sign, and alleviation of right-sided heart overload (RVEDD/LVEDD ratio, 0.84; RVEDD, 37 mm), although the TRPG could not be measured (Video 4). The patient was discharged on the ninth day of hospitalization.

DISCUSSION

This patient had LPE secondary to massive pulmonary embolism that was successfully treated using intravenous anticoagulant agents and thrombolytic therapy.

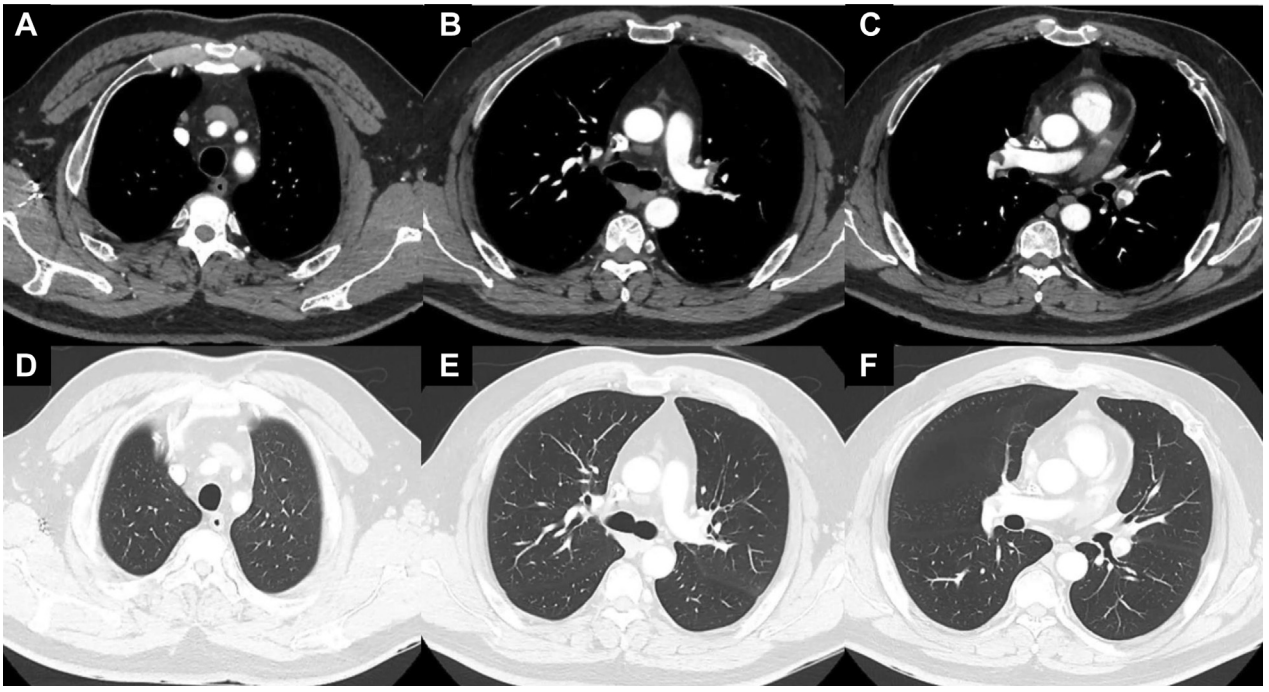
LPE accounts for 2.1% of cases of cardiogenic pulmonary edema.[1] Although several factors can trigger LPE, particularly in the right upper lobe, severe mitral regurgitation is the most prevalent cause.[2] However,

FIGURE 3 Chest Radiography Before Discharge



Chest radiography obtained 8 days after admission.

FIGURE 4 Contrast-Enhanced Computed Tomography Before Discharge (Day 8)



(A) The mediastinal window, upper lung. (B) Mediastinal window, pulmonary artery trunk level. (C) Mediastinal window, middle pulmonary artery trunk level. (D) Lung window, upper lung. (E) Lung window, pulmonary artery trunk level. (F) Lung window, middle pulmonary artery trunk level.

LPE can result from pulmonary embolism (termed overflow pulmonary edema).[3] This mechanism is thought to involve the concentration of blood flow into nonthrombosed arteries, thus leading to an increase in the hydrostatic pressure in that segment of the capillary bed.[2] The radiologic changes and therapeutic reactions support the diagnosis of LPE in the current patient. The clinical implication of LPE secondary to pulmonary embolism may be as follows: 1) the area affected by the embolism occupies most of the lung and decreases the efficiency of gas exchange in the embolized lung territories; 2) the concentration of blood flow to the remaining healthy lung fields results in pulmonary edema, further reducing the efficiency of gas exchange and increasing pulmonary hydrostatic pressure; and 3) decreased gas exchange and obstructive shock cause acidemia and lead to progressive circulatory failure.

This patient experienced rapid symptom improvement after thrombolysis. The unclear date of onset, a lack of objective data before the visit, unilateral consolidation of the lung, a history of trauma, and pinkish sputum suggestive of alveolar hemorrhage made the decision to administer thrombolytic agents difficult despite the patient's pulmonary embolism with severe respiratory failure. We thought that the dyspnea that occurred 3 weeks earlier was caused by a small pulmonary embolism and that a more extensive embolism may have happened on the day of the visit. After 1 day of aggressive intravenous anticoagulant therapy, the patient had not improved, and there was no evidence of worsening hemorrhagic complications. Therefore, thrombolytic agents were administered. It is crucial to differentiate complicated diseases adequately so the appropriate therapeutic decisions can be made.

FOLLOW-UP

At a 3-week follow-up appointment, the patient had no symptoms on exertion, and CECT showed a trend of resolution of the pulmonary emboli. Therefore, his rivaroxaban dose was reduced to 15 mg/day, as approved by the Pharmaceuticals and Medical Devices Agency of Japan.[4] We plan to continue rivaroxaban until the pulmonary thrombus and lower

extremity venous thrombus disappear on computed tomography.

CONCLUSIONS

A patient with LPE secondary to massive pulmonary embolism underwent antithrombotic therapy. Before initiating antithrombotic therapy, other differential diagnoses must be excluded on the basis of the patient's medical history.

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KEY WORDS pulmonary edema, pulmonary embolism

APPENDIX For a supplemental Discussion section, table, figures, and videos, please see the online version of this paper.