

# Non-surgical management of an abrupt cavitation and large oval-shaped lung abscess secondary to acute thromboembolic pulmonary infarction: a case report

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Guofeng Ma<sup>1</sup> , Dan Wang<sup>2</sup>, Chao Yan<sup>1</sup>,  
Liang Li<sup>1</sup>, Xiaoling Xu<sup>1</sup>, Xiaohong Wu<sup>1</sup> and  
Kejing Ying<sup>1</sup> 

## Abstract

Infected cavitating pulmonary infarction is a rare complication of pulmonary embolism with a high mortality rate. Surgical excision for this complication has been used in past decades. Abrupt cavitation and a large oval-shaped lung abscess caused by acute thromboembolic pulmonary infarction during anticoagulation are rare. We present a 70-year-old man who suffered from pleuritic pain and breathlessness, accompanied by nausea and vomiting for 1 day. A physical examination showed tachycardia and tachypnea with moist rales in the left upper chest. High D-dimer levels, leukocytosis, respiratory failure and left upper lobe consolidation were found on plain computed tomography (CT). CT pulmonary angiography was performed 2 days after the previous CT scan because pulmonary embolism was suspected. This scan showed emboli in the main, right upper, middle, lower and left upper pulmonary arteries with deteriorated left upper lobe consolidation and cavitation. Thromboembolic pulmonary infarction and an abscess were diagnosed. Enoxaparin 60 mg was administered every 12 hours for 10 days, followed by rivaroxaban, antibiotics and drainage of the hydrothorax. The patient improved after the strategy of non-surgical treatment and was discharged approximately 1 month later. The patient had an uneventful course during rivaroxaban 20 mg once daily for 1 year.

## Corresponding author:

Kejing Ying, Regional Medical Center for the National Institute of Respiratory Diseases, Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, No. 3 East Qingchun Road, Shangcheng District, Hangzhou 310016, China.  
Email: ykjsrrsh@zju.edu.cn

<sup>1</sup>Department of Respiratory Diseases, Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, Hangzhou, China

<sup>2</sup>Department of Radiology, Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, Hangzhou, China



## Keywords

Embolism, infarction, anticoagulation, pulmonary cavitation, lung abscess, infection, rivaroxaban

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## Introduction

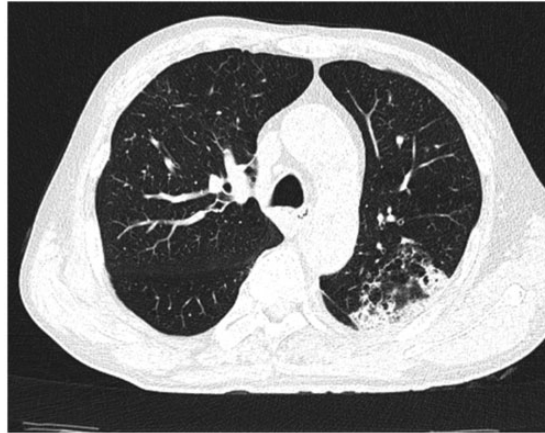
Acute pulmonary embolism (PE) is a life-threatening disease, which leads to pulmonary infarction in almost 31% of cases.<sup>1</sup> Recent studies have indicated that patients with infarction are often young and otherwise healthy before embolism.<sup>1</sup> Because of the dual blood supply and oxygen delivery of the lungs, a semicircular or cushion-like density with focal hyperlucency is frequently observed in computed tomographic (CT) scans of infarction.<sup>2</sup> Pulmonary cavitation complicates 4% to 7% of all cases and has been documented for longer than 6 days.<sup>3–5</sup> Infected pulmonary cavitation can lead to a pulmonary abscess in some patients and these patients mainly present with a fever and purulent bloody sputum.<sup>1,4</sup> We present a rare case of acute PE, which led to large cavitation and a subsequent pulmonary abscess. The patient recovered uneventfully after treatment with anticoagulation, antibiotics and thoracic drainage.

## Case report

A 70-year-old male smoker presented to the Emergency Department complaining of left pleuritic pain and shortness of breath, accompanied by nausea and vomiting for 1 day. He had a history of primary hypertension for 30 years and type 2 diabetes for 10 days. On evaluation, he was weak, with a body temperature of 37.2°C, pulse rate of 108 beats/minute, blood pressure of 149/87 mmHg, and respiratory rate of 22 breaths/minute. Auscultation revealed mild crackles in the left upper chest. No

other abnormal findings were found in the rest of the physical examination.

A laboratory investigation showed that the patient's white blood cell count was 13,100 cells/mL (91.3% neutrophils and 3.6% lymphocytes), and the hemoglobin level and platelet count were normal. Arterial blood gas analysis showed that the oxygenation index was 230 mmHg and the partial pressure of carbon dioxide was 40 mmHg. Serum procalcitonin and high-sensitivity C-reactive protein (hs-CRP) levels were 0.97 ng/mL (normal range, <0.5 ng/mL) and 18.5 mg/L (normal range, <6 mg/L), respectively. The fasting serum glucose level was 226 mg/dL (normal range, 65–109 mg/dL), the hemoglobin A1C value was 8.3% (normal range, 4%–6%) and the D-dimer level was 5930 mg/L (normal range, <500 mg/L). The values for B-type natriuretic peptide, troponin I, serum electrolytes, liver function tests, anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, anticardiolipin antibodies and tumor markers were within the normal range. Protein S, protein C and antithrombin III were negative. Sputum Gram and anti-acid stains, and cultures for bacteria and fungi showed negative results. Transthoracic echocardiography showed a normal left ventricular function, mild tricuspid regurgitation, and no evidence of infective endocarditis or right ventricular strain. Chest CT showed left upper lobe consolidation and mild pleural effusion (Figure 1). The patient was admitted and received oxygen therapy and intravenous piperacillin/tazobactam 4.5 g every 8 hours.



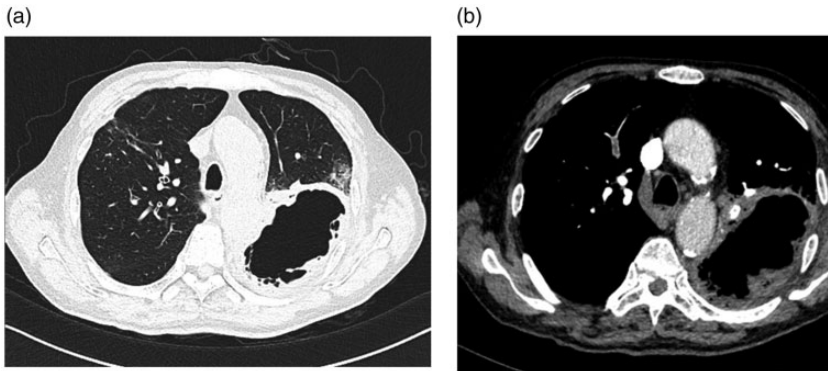
**Figure 1.** Initial computed tomographic scan showing left upper lobe peripheral consolidation, presenting as pulmonary embolism.



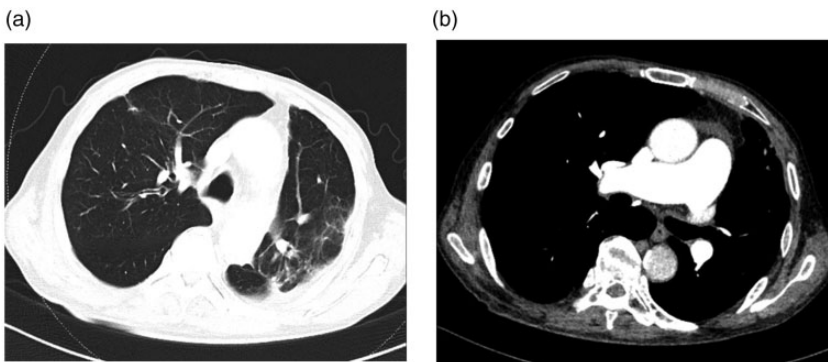
**Figure 2.** (a) Computed tomographic pulmonary angiography showing progress of left upper lobe consolidation after 10 hours. (b) Filling defects in the branches of both upper pulmonary arteries with left upper lobe infarction. (c) Right main pulmonary filling defect and a small amount of left pleural effusion.

CT pulmonary angiography (CTPA) showed multiple filling defects in the main, right upper, middle, lower and left upper pulmonary arteries, with deteriorated left upper lobe consolidation 10 hours after the first CT scan (Figure 2a, 2b and 2c). Repeated transthoracic echocardiography showed mild tricuspid regurgitation with moderate pulmonary hypertension. Compressed ultrasonography was performed and the presence of thrombosis in the lower extremities was excluded. Enoxaparin was initiated at a dose of 60 mg every 12 hours. During the following

3 days, the patient experienced purulent, bloody sputum with a continuous fever, left chest pain and dyspnea. Repeated procalcitonin and hs-CRP levels were 6.54 ng/mL and 133.5 mg/L, respectively. CTPA showed aggravation of left upper consolidation and pleural effusion with large pulmonary cavitation, even though multiple emboli in the pulmonary arteries had partially resolved (Figure 3a and 3b). The patient improved after replacement with intravenous linezolid and meropenem, and simultaneously, ultrasound-guided percutaneous thoracic drainage was also



**Figure 3.** (a) Computed tomographic pulmonary angiography showing a large oval-shaped cavity ( $10 \times 5 \text{ cm}^2$ ) with an irregular inner margin in the left upper lobe. (b) Improved filling defects of both upper pulmonary arteries.



**Figure 4** (a) Computed tomographic pulmonary angiography showing complete resolution of the left upper lobe abscess with ipsilateral peri-pleural fibrous focus at 1 year of follow up. (b) No filling defect in both pulmonary arteries.

implemented. The hydrothorax was mild, bloody, complicated, parapneumonic effusion without a positive culture.

After 10 days of treatment with enoxaparin, the patient was switched to rivaroxaban 20 mg once daily and was discharged 19 days later. His symptoms totally disappeared 3 months later. Follow-up CTPA showed complete resolution of the left upper lobe abscess and pulmonary embolism with ipsilateral peri-pleural fibrous focus (Figure 4a, 4b). Therefore, anticoagulation was stopped 1 year later without any complications.

## Discussion

Our patient presented with severe left chest pain and dyspnea without a fever at the onset of illness. The characteristic radiological features suggested acute PE as the cause of the infected cavitation and abscess. The main differentiation was septic PE with bilateral, peripheral, pulmonary nodules that often manifest cavitory changes on a typical CT scan.<sup>6</sup> Our patient had only one extremely large cavity with no history of underlying infection or indwelling catheters, which is inconsistent with septic PE.

Risk factors for pulmonary infarction with cavitation include being young and healthy, a taller body height, a low body mass index, and active smoking, in addition to a lack of congestive heart failure and chronic lung diseases.<sup>1,3</sup> Pulmonary infarction is frequently associated with complete obstruction of peripheral pulmonary arteries of 3 mm or less in diameter compared with larger arteries.<sup>7,8</sup> The mean time to cavitation for infected pulmonary infarction is 18 days (range: 6–40 days),<sup>3</sup> which is much later than that found in our patient. Possible explanations for the shorter time to cavitation in our patient are an older age and primary hypertension and diabetes, which cause susceptibility to infection and insufficiency of blood supply to the bronchial arteries.

The etiology of pulmonary cavitation is broad.<sup>3,9</sup> A fever, hemoptysis accompanied by purulent sputum, breathlessness and pleuritic pain are convincing signs of infected pulmonary cavitation, as in our patient. Typical findings of an afebrile status, a dry cough, cavitory size stability, cavitory margin irregularity and an absence of air-fluid level may favor aseptic cavitation, such as cancer, collagen vascular diseases and noninfectious pulmonary embolism. However, radiographic features of a single, right-sided and thin-walled lung abscess with scalloped inner margins, following filling defects in pulmonary arteries, suggest infected cavitating pulmonary infarction.<sup>3</sup> These findings are different from our patient's CT findings of an abrupt, large, left-sided, oval-shaped cavitation. The most common infections are from gram-negative bacteria (*Escherichia coli*, *Pseudomonas aeruginosa*, and *Proteus*), which helps clinicians to choose appropriate antibiotics.<sup>5</sup>

Previous studies have suggested prompt surgery owing to a 73% mortality rate in medical treatment of infected pulmonary cavitation, which is theorized to be due to

an insufficient blood supply and a risk of persistent infection.<sup>4</sup> Successful experiences of some separate cases of infected cavitating pulmonary infarction have been reported,<sup>3,4,10</sup> but no recent series have mentioned the prognosis of infected pulmonary cavitation followed by acute PE. Studies published in the past decades have consistently shown a progressive reduction in the fatality rate in patients with acute PE. A previous study on patients with PE from Germany between 2005 and 2015 showed that in-hospital mortality for these patients decreased from 20.4% to 13.9% because of improved management of PE.<sup>11</sup> The mortality rate is likely to be higher with a lung abscess combined with PE compared with PE alone. Our patient improved with broad-spectrum antibiotics and careful anticoagulation.

In conclusion, we experienced a rare case of infected pulmonary cavitation caused by acute PE, which occurred in 3 days. Diagnosis of this condition should be considered in patients with recent PE, a fever, yellowish sputum and leukocytosis. Left, large, oval-shaped cavitation is a rare CT manifestation of PE. Although the mortality rate is still high for this condition, surgical resection may not be the first choice, unless there is failure of medical and interventional treatment.

The reporting of this study conforms to CARE guidelines.<sup>12</sup>

### Author contributions

GM wrote the case report. DW helped choose the images. XW, LL and XX treated the patient. KY and CY helped revise the manuscript. All authors read and approved the final manuscript.

### Declaration of conflicting interest

The authors declare that there is no conflict of interest.

### Ethics statement

Written informed consent was obtained from the patient for treatment and publication of this case

report with accompanying images. The study did not need to be approved by an ethics committee because all data used were from previous medical records.

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### ORCID iDs

Guofeng Ma  <https://orcid.org/0000-0001-9360-3350>

Kejing Ying  <https://orcid.org/0000-0002-6396-4339>

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