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## Case report

## A case of pulmonary infarction induced by undiagnosed HIV

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## ABSTRACT

A 25-year-old Chinese man visited our institution due to fever and left chest pain. A chest CT showed infiltrative shadows with pleural effusion. Despite antibiotics treatment, his symptoms gradually worsened. The contrast CT showed deterioration of infiltrative shadows with thromboembolism in pulmonary arteries, suggesting pulmonary infarction. Thereafter, his HIV test turned out to be positive. His symptoms and radiological findings improved after initiation of an anticoagulant therapy. No known risk factors for thromboembolism were identified except HIV infection. The possibility of pulmonary thrombosis should be noted when the HIV patient with acute chest pain and pneumonia-like infiltrative shadow is seen.

## 1. Introduction

Pulmonary infarction (PI) is seen in one-third of patients with acute pulmonary embolism (PE) [1]. Diagnosis of PI is sometimes challenging because the initial symptoms of PI are nonspecific, such as chest pain, fever, and dyspnea. Moreover, the radiological findings of PI resemble bacterial pneumonia or other respiratory diseases. Delayed diagnosis of PI can be seen, especially in healthy young subjects with no known risk factors. The risk factors for thrombosis, also known as Virchow's triad, divided into three categories, namely hypercoagulability, stasis of blood flow, and endothelial injury. Well recognized risk factors are immobilization, surgery, dehydration, malignant tumor, and hereditary diseases such as deficiency of protein C or protein S [2]. In addition, it has been shown that HIV infection is known to induce thrombosis [3,4].

We herein report the case of PI, which had been at first diagnosed with pneumonia and pleuritis. In this case, undiagnosed HIV infection may be strongly associated with the development of PI.

## 2. Case report

A 25-year-old Chinese man with fever and acute left chest pain visited our emergency department. He was diagnosed with syphilis and started oral amoxicillin/clavulanate three days ago. He occasionally smoked one or two cigarettes a day. No travel history within six months or illegal drug use were identified. On admission, his body temperature

was 39.4 °C, blood pressure was 128/59 mmHg, and oxygen saturation on room air showed 96%. An auscultation of the left chest showed coarse crackles. His chest pain worsened during inspiration. The electrocardiogram revealed heart rate of 93 beats per minute and no ST-T change was observed. The chest X-ray showed an infiltrative shadow in the left lower lung field and chest CT revealed the consolidation along with ground-glass opacity in the both lung fields and pleural effusion (Fig. 1a and b). The laboratory findings showed the neutrophil-dominant increase of white blood cell counts (9550/μL) and increase of C-reactive protein (7.96 mg/dL). The cultures for blood and sputum showed negative results. Other laboratory data were mostly within normal limit (Table 1). Combined with the clinical symptoms and radiological findings, his initial diagnosis was bacterial pneumonia with pleuritis.

Piperacillin/Tazobactam was initiated, but his symptoms gradually worsened. The contrast CT one week after admission showed the deterioration of infiltrative shadows and pleural effusions with thromboembolism in pulmonary arteries, suggesting pulmonary infarction (Fig. 2). D-dimer was increased to 26.1 μg/dL (Table 2). The laboratory findings did not indicate any congenital or acquired thrombotic disorders, but HIV test turned out to be positive. Although he had denied the history of any casual sex contacts on admission, he confessed that he was men who have sex with men (MSM) after careful interview. Evaluation of deep thrombosis by the lower limb echo showed no evidence of venous thromboembolism. The heart ultrasound indicated normal left ventricular contraction with no right heart load.

*Abbreviations:* PI, pulmonary infarction; PE, pulmonary embolism; MSM, men who have sex with men.

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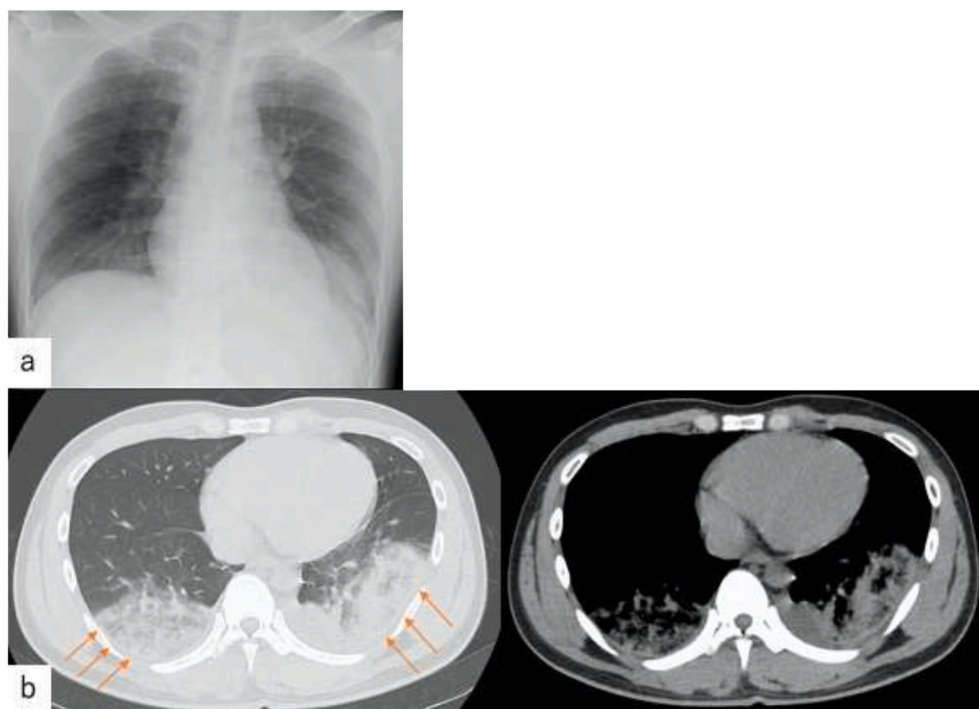
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**Fig. 1.** (a)The chest X-ray on admission showed infiltrative shadow in the left lower lung field. (b)The chest CT showed mixture of infiltrative shadows and ground glass opacity in both lower lobes (arrow) with small amount of left pleural effusion.

**Table 1**  
Blood test on admission.

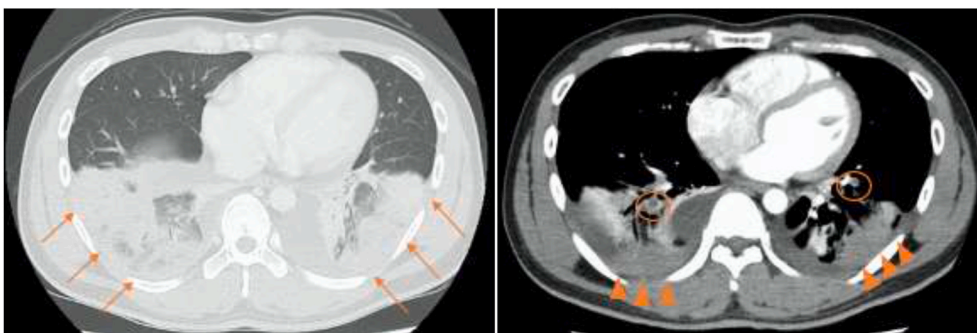
Test items	Results	Criterion values
WBC (/μL)	9550	4000–8600
Neut (%)	86.3	38–70
Lym (%)	8.3	27–45
Mono (%)	4.5	0–7
Eosin (%)	0.8	0–2
RBC (/μL)	$4.65 \times 10^6$	$3.80 \times 10^6$ – $4.80 \times 10^6$
Hb (g/dL)	12.6	12.0–16.0
Hct (%)	39.4	35.0–43.0
Plt (/μL)	$17.2 \times 10^4$	$15.0 \times 10^4$ – $35.0 \times 10^4$
TP (g/dL)	7.3	6.5–8.2
Alb (g/dL)	3.1	3.8–5.1
T-Bil (mg/dL)	1.2	0.2–1.2
AST (U/L)	17	13–33
ALT (U/L)	12	6–31
LDH (U/L)	177	119–229
ALP (U/L)	141	115–359
γ-GTP (U/L)	34	6–46
Cr (mg/dL)	0.92	0.48–0.79
BUN (mg/dL)	8.5	8.0–20.0
CRP (mg/dL)	7.96	0–0.30
B-D glucan (pg/mL)	9.6	<25.0
T-SPOT	negative	negative
Syphilis STS	18.7	<1.0
TPHA	145.2	<0.5

After initiation of an anticoagulant therapy (edoxaban 60 mg/day), his symptoms and radiological findings gradually improved (Fig. 3). Although his CD4<sup>+</sup> lymphocyte counts were decreased to 40/μL, he showed no evidence of opportunistic infection. He started antiretroviral therapy after discharge.

### 3. Discussion

Here, we showed the rare case of PI, which can be induced by undiagnosed HIV infection. Due to his physical symptoms and radiological findings on admission, he was first diagnosed with the bacterial pneumonia and pleuritis. This was because the patient was considered a healthy young subject and no known risk factors for thrombosis were identified on admission. PI usually develops among a patient with PE. PI was once considered a relatively rare lung complication because the lung receives oxygen supply from three sources, namely the pulmonary circulation, the bronchial circulation, and the airways [1]. However, the actual prevalence of PI is higher than previously believed. It has been reported that one third of PE patients developed PI and younger individuals without cardiopulmonary disease were more likely to develop [5].

There are two important lessons in this case. Firstly, the existence of HIV infection alone can cause PI. In this case, we evaluated other risk factors for thrombosis, but no known risk factors were identified except for HIV. Aside from several opportunistic infections through the reduction of CD4<sup>+</sup> T cells, HIV patients can develop non-infectious complications such as malignant tumor, neurological disorders, and thrombosis [6]. HIV infection is known to be a prothrombotic condition due to several factors including the virus itself, host response, and anti-retroviral therapy [3,4]. It has been reported that lower CD4<sup>+</sup> T cell counts, elevated HIV RNA levels, elevated lipid levels, and deficiency of coagulation functions are the risk factors for the development of thrombosis in HIV patients [7]. Among them, lower CD4<sup>+</sup> counts and elevated HIV RNA levels may be associated with thrombosis in this case. HIV/AIDS cases are relatively rare in Japan, however, approximately 1300–1600 cases are still newly diagnosed annually for the past 10 years [8]. When we look back, the history of syphilis infection and MSM status strongly indicated HIV infection [9]. It is important to



**Fig. 2.** The contrast CT one week after admission revealed deterioration of pleural-based infiltrative shadows and ground glass opacity (arrow) with poorly enhanced lung parenchyma (arrowhead). Multiple thromboses were detected in pulmonary arteries (circle). The increase of pleural effusion was seen in both lungs.

**Table 2**  
Blood test 7 days after admission.

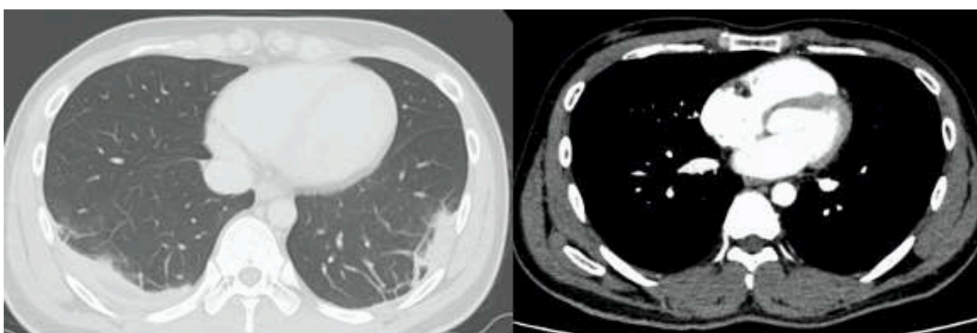
Test items	Results	Criterion values
D-dimer (µg/mL)	26.1	<1.0
Protein C activity (%)	89	64–146
Protein S activity (%)	99	67–164
Protein C antigen (%)	103	70–150
Protein S antigen (%)	117	65–135
Free protein S antigen (%)	118	60–150
Homocysteine (µmol/L)	8.5	3.7–13.5
Antinuclear antibody	<40	<40
Anti ds-DNA-IgG (U/mL)	3.9	<12.0
Anti CL-β2GPI antibody (U/mL)	<0.7	<3.5
Anti CL-IgG antibody (U/mL)	<8	<10
Lupus anticoagulant	negative	negative
HBs antibody	negative	negative
HCV antibody	negative	negative
HTLV-1	negative	negative
HIV antigen/antibody	positive	negative
HIV DNA (copy/mL)	1.1 × 10 <sup>5</sup>	0
CD4 <sup>+</sup> lymphocytes (µL)	40	700–1300
Candida antigen	negative	negative
MAC antibody (U/mL)	negative	negative
Aspergillus antigen	negative	negative
Cryptococcus antigen	negative	negative
Toxoplasma antigen	negative	negative
CMV antigenemia	negative	negative

consider the possibility of HIV infection when a patient has sex contact with unspecified majority and shows atypical clinical course or presentation of disease.

Second, diagnosis of PI by radiological findings is challenging. PI is usually seen in the lower lung lobe and the typical radiological findings include consolidation with internal air lucencies (reversed halo sign), focal decrease in parenchymal enhancement, broad pleural base, and pleural effusion [10–12]. Nevertheless, misdiagnosis such as pneumonia, pulmonary edema, or organizing pneumonia sometimes occurs because these findings shown above are not specific to PI. In addition, the radiological findings of PI changes depending on the location of infarction, disease onset, and severity of ischemic injury. Both first and second chest CT well described the characteristics of PI such as reversed halo sign, broad pleural base, and pleural effusion in this case, but it seemed rather difficult to make a definitive diagnosis by CT findings alone. The combination of clinical course of a patient and laboratory data including radiological findings and coagulation fibrinolytic system are necessary for the diagnosis of PI. The possibility of pulmonary thrombosis should be noted when the antibiotics-resistant, acute chest pain and pleuritis-like infiltrative shadow is seen. In conclusion, the prevalence of HIV infection is relatively rare in Japan; however, we should not forget that HIV infection alone can cause PI.

**Declaration of competing interest**

None declared.



**Fig. 3.** The chest CT one month after treatment revealed improvement of infiltrative shadow and pleural effusion in both lungs. Thromboses in pulmonary arteries disappeared.

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