SciMedCentral

Journal of Translational Medicine & Epidemiology

Case Report

The Diagnostic Dilemma between Pulmonary Embolism with Positive Chest Imaging and Pneumonia: A Case Report and Literature Review

Kan Xu[1], Xinjun Tang[2], Yuanlin Song[2] and Zhihong Chen[2]*

[1]Geriatrics Division of Zhongshan Hospital, Fudan University, Shanghai 200032, China
[2]Respiratory Division of Zhongshan Hospital, Fudan University. Shanghai Institute of Respiratory Disease, Shanghai 200032, China

Corresponding author
Zhihong Chen, Shanghai Insititute of Respiratory Diseases. Respiratory Division of Zhongshan Hospital, Fudan University. 180 Fenglin Road, Shanghai, China, Tel: 86-021-64041990-2445; Fax: 86-021-64187165; E-mail: czh60@hotmail.com

Submitted: 15 September 2014

Accepted: 06 February 2015

Published: 04 May 2015

ISSN: 2333-7125

Keywords
• Pulmonary embolism
• Pneumonia

Abstract

Background: Although the last decades have witnessed more pulmonary embolism (PE) in China, PE in young adults is rare. Here we reported a young man of saddle PE with several lung infiltrations in imaging which was misdiagnosed as pneumonia for three times.

Case review: A 25-year-old man, presenting no obvious risk factors for PE, was finally diagnosed as saddle PE. However, during the whole process of diagnosis and treatment, the patient was misdiagnosed as pneumonia three times. He presented with left chest pain with difficulty breathing, mild cough, and sputum. Considering the imaging, and clinical manifestations, the patient was diagnosed as pneumonia. He suffered from sudden right chest pain, dyspnea, and tachycardia later. The D-dimer increased significantly, accompanied by mild hypoxemia and hyperventilation. CT pulmonary angiography (CTPA) revealed a saddle PE (medium risk).

Conclusions: The clinical features of pneumonia and PE differ. PE has a more sudden onset, and the dyspnea is more prominent than cough and sputum. PE usually has a dyspnea unmatching with the changes in imaging, and has no response to antibiotics. Pneumonia has a progressive onset, with more prominent cough, sputum, and fever. The dyspnea of pneumonia basically matches with the pulmonary infiltrates in imaging. Pneumonia is sensitive to antibiotics. Therefore, attention should be paid to young PE patients without obvious incentives. The differentiation between pneumonia and PE should be carefully conducted, and the diagnostic procedure of PE should be correctly applied, in order to timely diagnose and treat this type of disease.

INTRODUCTION

The diagnose rate of pulmonary embolism (PE) has been increasing in China in recent years. Most of PE does not have lung infiltration in chest imaging, because lung tissue receives oxygen supply from multiple sources including pulmonary artery, bronchial artery and alveolar [1,2]. When PE occurs, the blood pressure of the distal pulmonary artery decreases, and the oxygen-rich blood in pulmonary vein hence can nourish the lung tissue retrogradely, resulting in fewer infarction among the PE [3,4]. When PE is accompanied by pulmonary infiltrates in imaging, it should be differentiated from diseases such as pneumonia, tuberculosis, pulmonary fungal disease, pulmonary vasculitis, etc. Here we reported a case of young adult with saddle PE, which was misdiagnosed as pneumonia 3 times before the right diagnosis was made. Literatures in regard to the clinical characteristics of both PE and pneumonia were also reviewed.

CASE PRESENTATION

A 25-year-old man, visited the outpatient of Zhongshan hospital on April 11, 2013, because of left chest pain and dyspnea. He had an earlier history of catching cold and then suffered from left chest pain, cough, and activity-associated dyspnea. The pain located in the intercostal region between 5-6 ribs in his front, left chest. It was a persistent dull pain and aggravated when coughing and deep breathing. There was no sputum, hemoptysis, fever, etc. Chest X-ray showed a slight inflammation

in the left lower lung and slightly-thickened left pleura. The blood routine was as follows: WBC, 9.90×10^9/L; N, 63.5%; and L, 25.4%. ECG (electrocardiography) indicated sinus rhythm and T-wave changes (flattened leads II, aVF, and V5-6). The patient was diagnosed with left lower lobe pneumonia and treated with levofloxacin (0.2 po bid), however with no good effect. The patient visited outpatient clinic again on April 12. Chest CT showed slight inflammation of the left lung with a small amount of pleural effusion, as well as thickened left pleura (Figure 1). B-ultrasound indicated left pleural effusion of 15 mm depth. The blood routine was as follows: WBC, 9.94×10^9/L; N, 73.7%; L, 15.8%; and D-dimer, 2.71 mg/L. The patient was diagnosed with left lower lobe pneumonia again and treated with cefuroxime (3.0 ivgtt bid), with, however, no significant improvement in symptoms. From April 13, the patient started to run high fever (body temperature, 39.5 °C). On April 14, he was reviewed at outpatient by chest CT, which showed progressed bilateral pulmonary inflammation (mainly left lung), compared with the previous imaging; (Figure 1). B-ultrasound showed no significant bilateral pleural effusion. The blood routine was as follows: WBC, 10.0×10^9/L; N, 75%; and L, 14.0%. The patient was diagnosed again with bilateral pneumonia and treated with compound aminopyrine (2 mL im st) for fever and moxifloxacin (0.4 ivgtt qd) for infection. Two days later, the patient's temperature gradually dropped to 37.4 °C, with improved cough symptoms and alleviated chest pain, but no significant relief of the activity-associated dyspnea. The patient presented with dyspnea even when taking a ground walk or a bath. He had no symptoms such as bloody sputum, fainting, etc. After 3 days of intravenous anti-infection, the patient was given moxifloxacin (0.4 po qd) orally. On April 22, the patient had sudden severe pain in the upper right chest, located close to the right sternum in the intercostal region between 2-6 ribs, with a nature similar to that of the left chest pain, i.e., persistent dull pain, exacerbated with cough and deep breathing. The patient visited the emergency department then. Review chest CT indicated pulmonary inflammation; compared to the previous CT on April 14, there was some kind of absorption of the left lower lobe lesion, but new lesion emerged in the middle lobe of the right lung (Figure 1). ECG showed sinus tachycardia (heart rate, 124 beats/min) and T-wave changes (flattened and slightly inverted in leads II, aVF, and V3-6). The blood routine was as follows: WBC, 10.13×10^9/L; N, 75.0%; L, 14.5%; D-dimer, 23.37 mg/L (much higher than normal); the arterial blood gas (oxygen, 2 L/min) was as the following: pH, 7.43; PCO_2, 37 mmHg; PO_2, 94 mmHg; HCO_3^-, 24.6 mmol/L; and SO_2, 98.0%. Echocardiography indicated right atrioventricular enlargement and mild pulmonary hypertension (PASP, 43 mmHg). CTPA showed thrombus in the main pulmonary artery, more severe in left pulmonary arteries, and wide embolization of the remaining arterial branches (central floating type) (Figure 2). Lung window indicated consolidation of the lower left lung, with slightly decreased volume, as well as a wedge-shaped shadow in the middle lobe of the right lung, close to the chest wall (Figure 2). Until then the patient was finally diagnosed with acute PE (medium risk).

Treatment and follow up

The patient was admitted into respiratory intensive care unit (RICU) on April 22, 2013, and immediately received thrombolytic therapy (actilyse 50 mg iv st). The patient subsequently received anticoagulant therapy with low molecular weight heparin (fraxiparine, 0.8 mL iH q12h), then maintained with oral warfarin. The results of other examinations during the hospitalization were as follows: venous ultrasound of the legs, negative; full set of blood tumor markers, negative; full set of rheumatism antibodies

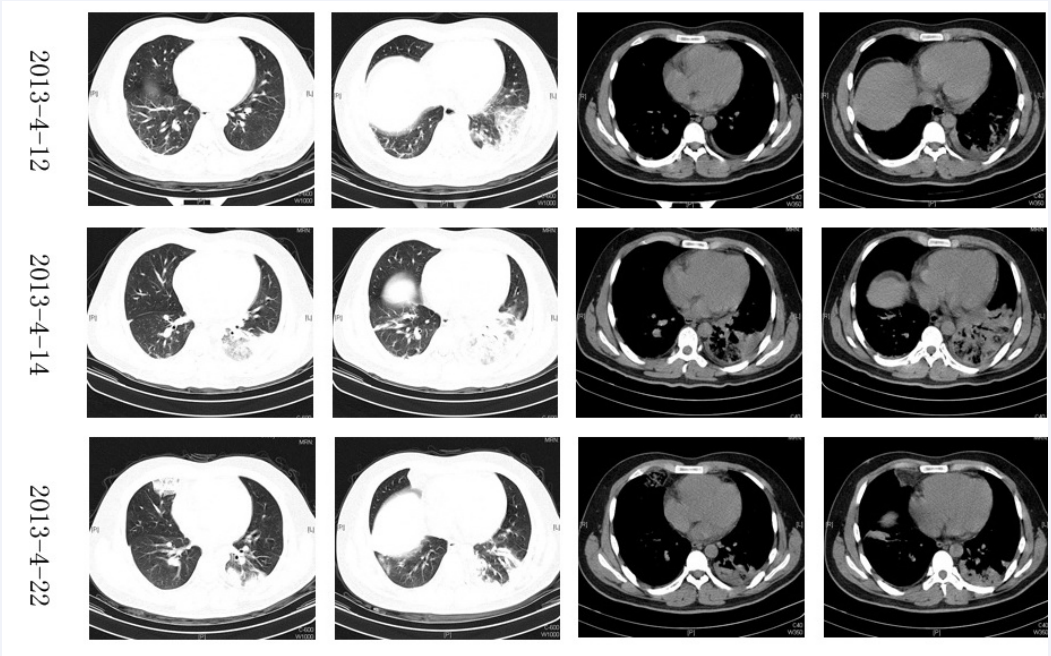

Figure 1 Evoluation of CT imaging from onset of disease to definite diagnosis. On Apr 12th, triangle-shaped opacity was in left lower lobe with a small amount of pleural effusion. It evolved into large consolidation on Apr 14th. On Apr 22th, part of the left lower lobe consolidation was absorbed, while a new wedge-shaped shadow appeared in right middle lobe.

and anti-cardiolipin antibodies, negative; antithrombin III and protein C, normal; and protein S was 18.7% (Normal range: 75%-130%): decreasing significantly.

On April 29, after receiving 7 days of therapy, the patient's status became stable, and the chest pain and breathing difficulties were evidently mitigated. The D-dimer decreased to 4.20 mg/L. CTPA indicated embolism of bilateral pulmonary arteries and their branches, were slightly decreased compared with the previous imaging on April 22 (Figure 2). Three months after the onset of PE, (August 14, 2013), CTPA showed no significant filling defects with bilateral pulmonary arteries, and only a few fiber cords left at the lower left lung (Figure 2). The patient was followed up for a year. He took warfarin regularly and was in a stable condition, with no recurrent PE.

DISCUSSION

The patient was misdiagnosed with pneumonia three times, because he was young, with atypical PE symptoms and positive chest imaging similar to pneumonia, without common PE risk factors. **A**ge is an important risk factor for PE. The Revised Geneva Scoring System adds 1 point to patient older than 65 years in evaluating PE risk factors [5]. Sakuma et al. [6,7] statistically analyzed 11,367 cases of autopsy-confirmed PE in Japan from 1987 to 1998 (from 396,982 copies of autopsy reports) and showed the number of PE increased dramatically in the population older than 40, while the prevalence of PE in 20-39 years population remained low, accounting for 7.7% of the total fatal PE. However, from the perspective of a single cause of death for young population, PE is one of the major causes of the death of healthy young people. A total of 255 cases of PE patients were admitted and treated in our hospital (Zhongshan Hospital of Fudan University, China) from 1999 to 2009. Among them, 13 cases (13/255, 5.1%) were younger than 30 years, and 22 (22/255, 8.6%) were between 31-40 years, indicating that there are quite some PE cases among the young population in China (data not shown)

When considered individually, symptoms, signs, or common laboratory tests have limited diagnostic power of PE. So clinical probability assessment is an important step to diagnose it. The Canadian model introduced by Wells at al.[8,9] is the most frequently used prediction rule for suspected PE. It includes seven variables of which three refer to well-recognized risk factors for PE. The model heavily depends on the subjective judgement, so the Wells' model seems better suited to rule out rather than to rule in the diagnosis of PE. The present patient was first evaluated by Wells criteria on April 12, and his clinical risk score of PE was 0. On April 14, with deterioration of breathless and no response to antibiotics, the likelihood of PE increased with Wells score of

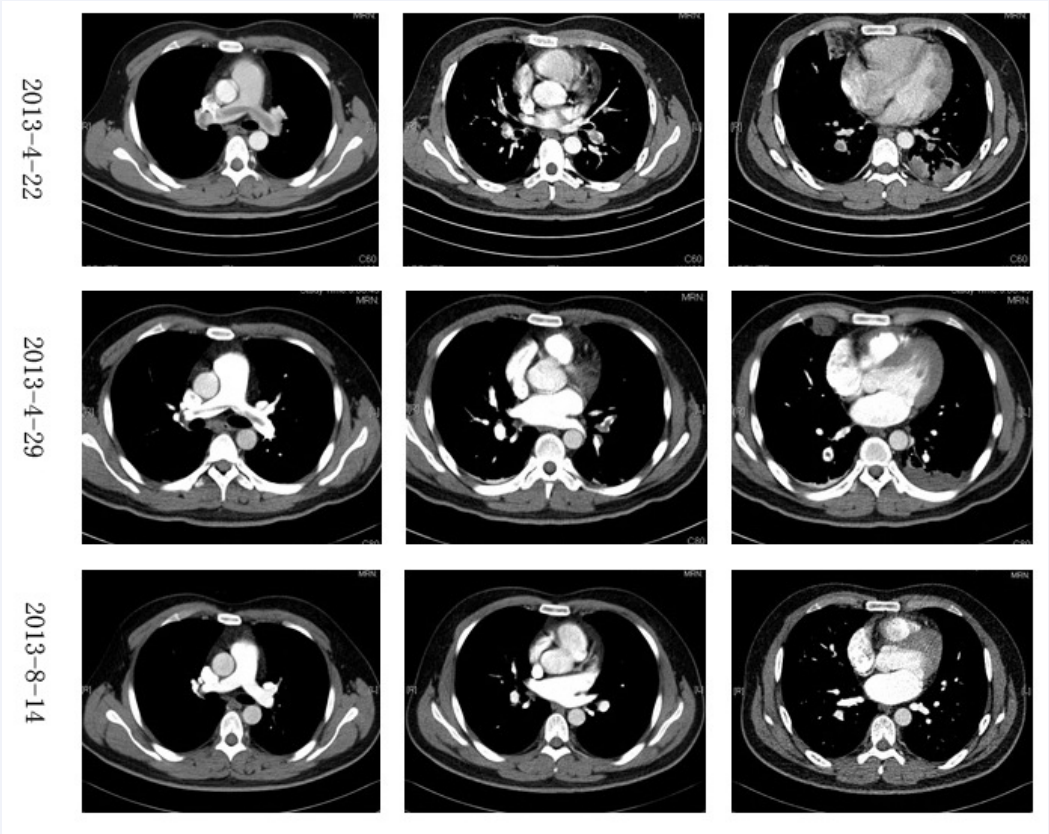

Figure 2 CTPA (Computor Tomography pulmonary angiography) evolution from the diagnosis day to 4 month later. Saddle thromboembolism showed in the main pulmonary artery. Filling-defects were in both lower pulmonary arteries and their branches. Subpleural crescent consolidation was in the left lower lobe and wedged shaped shadow was in the right middle lobe. One week later, the review of CTPA showed most of the filling defects absorbed. 4 months after the initial thrombolytic therapy, the imaging went back to normal.

3. On April 22, the assessed risk score increased to 4.5 (based on Wells criteria, 0-1 means low risk; 2-6 indicates medium risk; and >6 represents high risk).

Imaging tests are important in confirming diagnosis in suspected PE. PE with positive chest imaging needs to be differentiated from many other diseases such as pneumonia, tuberculosis, pulmonary fungal disease, pulmonary vasculitis, lung abscess, pulmonary vascular sarcoma, leiomyosarcoma, etc. In clinic, there are subtle differences between them. For example, the order of the clinical manifestations of the 2 diseases is different, i.e., pneumonia usually has a gradual onset, starts with fever, cough, sputum, probably accompanied by subsequent chest pain and dyspnea. However, the onset of PE is usually more sudden; it starts with chest pain and post-activity dyspnea, possibly mixed with fever and cough in the late stage. Relative to the imaging changes, more severe clinical manifestations are observed with PE. A peripheral wedge-shaped opacity on CT is associated with PE, but the finding is not specific. They may be sometimes attributed to pneumonia. When MDCT has the limitation to differentiate PE with pneumonia, V/P SCAN should be performed. The basic principle for the diagnosis of PE based upon V/P SCAN is to recognize lung segments without perfusion but preserved ventilation, i.e. mismatch. If absence of ventilation and perfusion exists at the same time, but is matched. It predicts the probability of pneumonia [10]. In a previously reported case, a 25 years old of young woman was firstly diagnosed as pneumonia with CT scan, but further examination with V/P SCAN confirmed PE in presence of pneumonia [11]. In the present case, the proper diagnosis is PE combined with pneumonia.

Risk factor may be crucial clues to diagnose PE, especially in case without history of venous thromboembolism. The present patient is a computer fan, with partially introverted personality. He is quiet with little activities, which may be one incentive of his disease. In addition, The present patient showed obviously decreasing protein S level. Therefore, reduced anticoagulant factors may partly explain the onset of PE in the present case [12,13].

In short, saddle PE complicated with lung infiltration, occurred, especially, in young patients in the absence of obvious risk factors, is relatively rare. But if the physicians can be highly alert of PE, actively screen for relevant clues, and correctly follow the diagnostic procedure of PE [14], this type of diseases can be diagnosed and promptly treated.

AUTHORSHIP AND CONTRIBUTIONS

All the authors have substantially contributed to the manuscript. Dr Xu and Dr. Chen took care of the patient. Dr. Tang searched related English literatures. Dr Xu, and Dr.Tang prepared the draft of the manuscript. Dr Song provided great ideas about the conception of the work and critically made the revision of the manuscript.

REFERENCES

1. Coldhaber SZ. Pulmonary thromboembolism. Harrison's principles of internal medicine.16th edition, McGraw-Hill publishing Division. 2005; 1563-1564.
2. Tapson VF. Acute pulmonary embolism. N Engl J Med. 2008; 358: 1037-1052.
3. Bai C. Pulmonary embolism. In Practical Internal Medicine. Ed. 11. Chen Y. Beijing: People's Health Publishing House. 2001; 1637-1640.
4. Wang C, Wu Y. The pathology and pathophysiology of pulmonary thromboembolism. Chin J Tuberc Respir Dis. 2001; 24:707-708.
5. Le Gal G, Righini M, Roy PM, Sanchez O, Aujesky D, Bounameaux H. Prediction of pulmonary embolism in the emergency department: the revised Geneva score. Ann Intern Med. 2006; 144: 165-171.
6. Sakuma M, Nakamura M, Takahashi T, Kitamukai O, Yazu T, Yamada N. Pulmonary embolism is an important cause of death in young adults. Circ J. 2007; 71: 1765-1770.
7. Christiansen LR, Collins KA. Natural death in the forensic setting: a study and approach to the autopsy. Am J Forensic Med Pathol. 2007; 28: 20-23.
8. Wells PS, Anderson DR, Rodger M, Ginsberg JS, Kearon C, Gent M. Derivation of a simple clinical model to categorize patients probability of pulmonary embolism: increasing the models utility with the SimpliRED D-dimer. Thromb Haemost. 2000; 83: 416-420.
9. Genna Rollins. The Challenge of Diagnosing Pulmonary Embolism, Clinical Laboratory News. 2011: 37; 110-115.
10. Bajc M, Neilly JB, Miniati M, Schuemichen C, Meignan M, Jonson B; EANM Committee. EANM guidelines for ventilation/perfusion scintigraphy: Part 1. Pulmonary imaging with ventilation/perfusion single photon emission tomography. Eur J Nucl Med Mol Imaging. 2009; 36: 1356-1370.
11. Bajc M. Value of ventilation/perfusion SPECT detecting extensive pulmonary embolism in a patient with pneumonia. Thromb Haemost. 2005; 93: 993-994.
12. Caspers M, Pavlova A, Driesen J, Harbrecht U, Klamroth R, Kadar J. Deficiencies of antithrombin, protein C and protein S - practical experience in genetic analysis of a large patient cohort. Thromb Haemost. 2012; 108: 247-257.
13. Lipe B, Ornstein DL. Deficiencies of natural anticoagulants, protein C, protein S, and antithrombin. Circulation. 2011; 124: e365-368.
14. Bajc M, Neilly JB, Miniati M, Schuemichen C, Meignan M, Jonson B. EANM guidelines for ventilation/perfusion scintigraphy : Part 2. Algorithms and clinical considerations for diagnosis of pulmonary emboli with V/P(SPECT) and MDCT. Eur J Nucl Med Mol Imaging. 2009; 36: 1528-1538.

Cite this article

Xu K, Tang X, Song Y, Chen Z (2015) The Diagnostic Dilemma between Pulmonary Embolism with Positive Chest Imaging and Pneumonia: A Case Report and Literature Review. J Transl Med Epidemiol 3(1): 1039.