

Case Report

Underdiagnosed Pulmonary Artery Thrombosis and Pulmonary Embolism in Rheumatic Heart Disease

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Abstract

Pulmonary embolism is a life-threatening acute cardiovascular disease and not easy to make an immediate diagnosis in clinical arena. A 46-year-old female with underdiagnosed pulmonary artery thrombus and pulmonary embolism in rheumatic heart disease, the patient's presentation, diagnostic procedure and successful treatment are presented.

Keywords: Pulmonary artery thrombus; Pulmonary embolism; Rheumatic heart disease

1. Clinical Case

A 46-year-old female presented with a 24-hour history of sustained left epigastrium ache, accompanied with abdominal distension, cough, short of breath, and severe lower extremity edema. The initial diagnosis and treatment were concentrated in gastro-enteritis at rural health center for 7 days. However, the mentioned symptoms were non-significantly relieved. Subsequently, she was admitted to local tertiary hospital and diagnosed with rheumatic heart disease (RHD). After 10 days of pharmacological treatment (diuresis and inotropic medications), she was been referrals to our department. Physical examination exhibited the patient was in poor general condition, blood pressure of 90/60 mmHg, pulse rate of 106 beats per minute, respiratory rate of 32 breaths per minute with 92% oxygen saturation of blood on oxygen therapy (1 L/ml), with typical malar flush and mild cyanotic lips, pulmonary auscultation showed crackles and lower breath sounds in left lung, cardiac auscultation indicated arrhythmic and mild-diastolic murmur at

the apical region, engorgement of the jugular vein and moderate lower extremity edema. The diuretic and inotropic treatment was continued. Transthoracic echocardiography (TTE) confirmed moderate rheumatic mitral valve stenosis and regurgitation, moderate aortic valve regurgitation, moderate tricuspid regurgitation and significantly decreased left ventricular ejection fraction (40%). The estimated pulmonary artery systolic pressure was 60 mmHg. After that, coronary computed tomography angiography revealed normal coronary artery, filling defect of distal right pulmonary artery and atelectasis (Figure 1). Finally, pulmonary artery thrombus (PTA) and PE were detected by computed tomography pulmonary angiography (CTPA), the PTA was riding on the pulmonary bifurcation (Figure 2A). Pulmonary infarction was synchronously detected (Figure 2B). Furthermore, the D-dimer testing was 65.37 µg per litre. Deep venous thrombosis was excluded with ultrasonography and cancer was ruled out with computed tomography scan. The patient experienced urgent cardiopulmonary bypass assisted cardiac surgery and pulmonary embolectomy. The PAT was removed via an incision of pulmonary artery (Figure 2C). The patient was quickly recovered and discharged with oral anticoagulation on the 15th day postoperative.

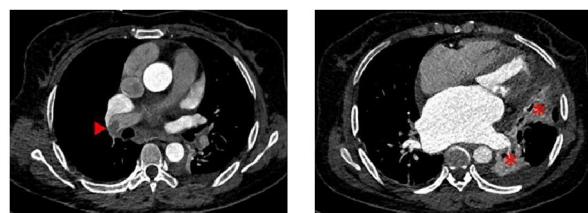


Figure 1: Coronary computed tomography angiography showing pulmonary embolism (►) and atelectasis (*).

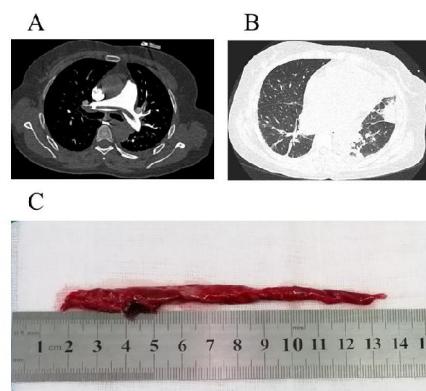


Figure 2: (A). Computed tomography pulmonary angiography demonstrating massive pulmonary artery thrombi and pulmonary embolism; (B). Pulmonary infarction; (C) The removed pulmonary artery thrombus.

2. Discussion

PAT has been reported in Eisenmenger syndrome, pulmonary artery aneurysm, pulmonary artery stump and the other disease conditions [1, 2]. According to current diagnostic algorithms of PE, Wells rule and Geneva score [3], the PE should be ruled out in this case. The D-dimer test has been extensively applied in the diagnosis of PE and the age-

adjusted D-dimer threshold for non-invasive exclusion of PE has been proposed by Marc Righini and colleagues [4]. However, the result of D-dimer testing in this case was 65.37 µg per litre. Acute PE is characterized by substantial clinical manifestations which are result from a complex interplay between different organs. In most patients of dyspnea, chest pain, haemoptysis, syncope, and unilateral extremity swelling, PE should be suspected [3]. In this case, the symptoms were variable and nonspecific, included left epigastrium pain, abdominal distension, cough, short of breath, and lower extremity edema were manifested. Parts of these symptoms are overlap with RHD, this is the primary cause of belated diagnosis. Therefore, a high index of clinical suspicion is a critical factor in the establishing diagnosis of PE. The left epigastrium pain might be related to the disturbances in left lower pulmonary circulation and local pleural involvement. In spite of the pharmacological treatment was continued more than 2 weeks, there was not in complete relief of clinical signs and symptoms in this case. These conditions suggested that clinical must consider the possibility of some other disease may combine in this case. Finally, the CTPA confirmed the diagnosis of PTA and PE. Surgical pulmonary embolectomy has been successfully performed before the introduction of medical treatment [3]. The remove of PAT can ameliorate right ventricular dysfunction, lung ventilation-perfusion match and pulmonary hypertension. The mechanism of PAT and PE in RHD patient has not been fully understood. Here, we infer that PAT and PE may result from mitral stenosis related pulmonary hypertension, left atrial blood turbulence and hypercoagulability.

3. Conclusion

This case highlights not only the frequent problem of PE is easily missed diagnosis, but also remind clinicians should maintain a high index of suspicion. We assume that the diagnosis of RHD in this case was an incidental finding and that she did not suffer from PTA, PE and right heart ventricular failure.

Conflict of Interest

None declared

References

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