electrocardiographic findings and ASD but not yet apical left ventricular hypertrophy. Besides, we supposed that in our patient, the prolonged therapy with ACE-inhibitor could have delayed the symptoms and cardiac concentric remodelling.

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Mobile right heart thrombus and massive pulmonary embolism

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Abstract The current report describes a patient with pulmonary embolism, treated unsuccessfully with heparin. Transthoracic echocardiography revealed free-floating right heart thrombus. Migrating deep vein thrombus to the right heart was suspected. Transesophageal echocardiography confirmed origin of the thrombus in the inferior cava vein. Mortality rate of mobile right heart thrombus is over 40%, therefore urgent surgical embolectomy was performed with relief of symptoms.

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Case report

A 47-year-old man with a history of deep vein thrombosis was treated for pulmonary embolism with intravenous heparin for 4 days. Despite therapy, he complained of progressive dyspnea and chest pain. On examination, blood pressure was 110/65 mmHg and saturation 97%. The ECG showed sinus rhythm, left heart-axis and negative T-waves in leads V2—V5 (Fig. 1).

Transthoracic echocardiography (Fig. 2) was performed, demonstrating a worm-like, mobile mass in the right heart. The right ventricle was enlarged, hypertrophied and paradoxical septal motion was present, indicating right ventricular pressure overload. Systolic tricuspid valvular gradient was 56 mmHg. Transesophageal echocardiography (Fig. 3) showed a thrombus in the right atrium, originating from the inferior vena cava with thrombus in the pulmonary artery. The European Guidelines on diagnosis and management of acute pulmonary embolism do not routinely advise the use of echocardiography in haemodynamically stable patients with pulmonary embolism.[1,2] However, echocardiography was performed in this patient because of worsening symptoms despite therapy. Subsequent spiral computed tomography (Fig. 4) confirmed subtotal occlusion of the pulmonary artery with thrombus. No extracardiac tumor was present.

The diagnosis was migrating thrombus to the right heart because of worm-like morphology of the thrombus and its mobility, as well as origin from the inferior caval vein. There were no clinical signs of infection and no leucocytosis, therefore endocarditis was not likely.

Laboratory testing demonstrated a platelet count of 41×10^9/L, a drop from 341×10^9/L at start of heparin treatment. Development of thrombocytopenia can occur as early as 4 days after start of heparin treatment as a result of heparin-induced thrombocytopenia (HIT).[3] In HIT, antibodies

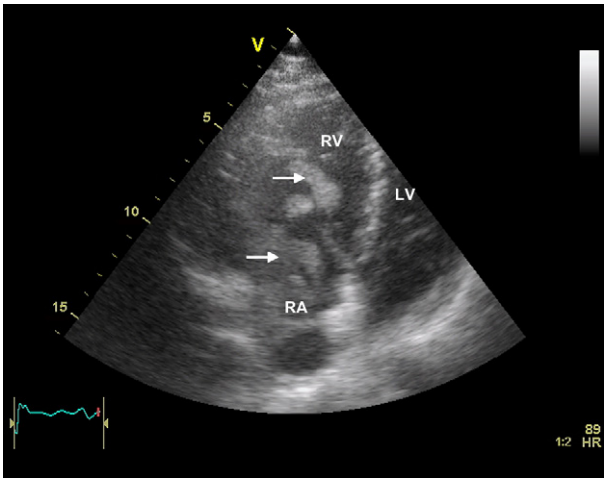

Figure 2 Transthoracic four-chamber view demonstrating a worm-like free-floating mass (arrow) in the right atrium with protrusion in the enlarged and hypertrophied right ventricle during diastole (four-chamber view RVED 5.1 cm, parasternal long-axis RVED 3.3 cm, RV free wall 0.6 cm). RA = right atrium, RV = right ventricle, LV = left ventricle.

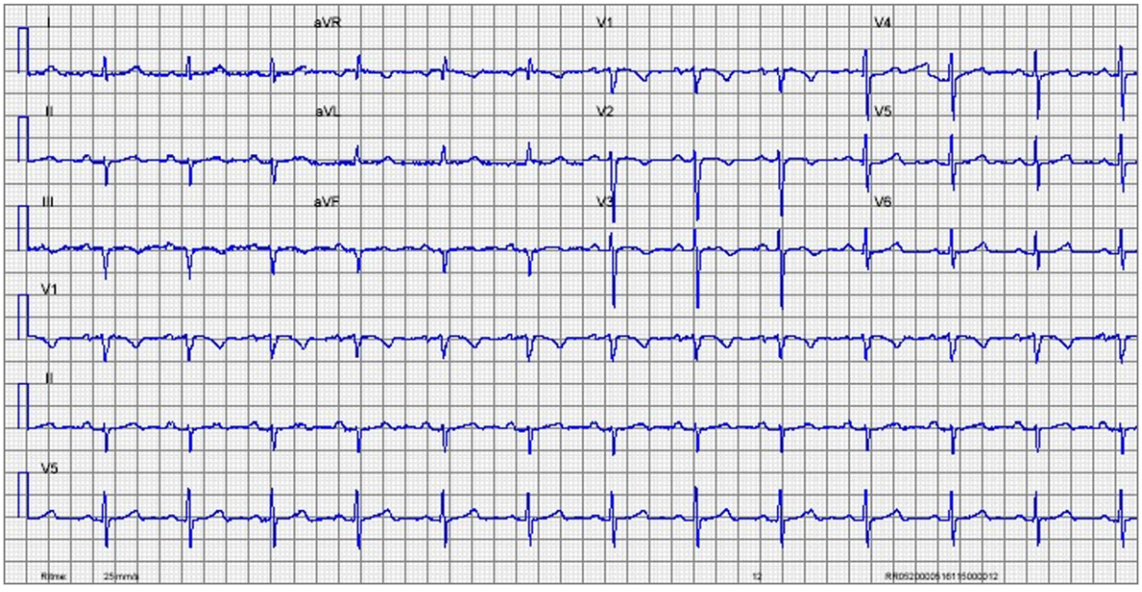

Figure 1 Electrocardiogram (25 mm/s and 10 mv/mm) showing sinus rhythm (90 bpm), normal conduction and left heart-axis, with negative T-waves in leads V2—V5 indicating right ventricular overload.

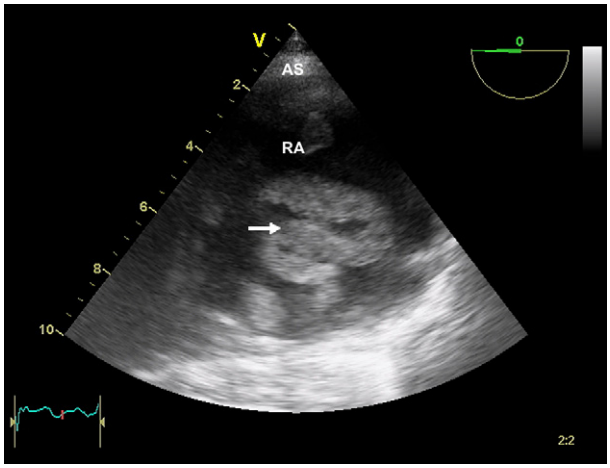

Figure 3 Transesophageal echocardiography confirming the free-floating, worm-like, homogeneous thrombus (3×7 cm, arrow) in the right atrium, originating from the inferior vena cava. RA = right atrium, AS = atrial septum.

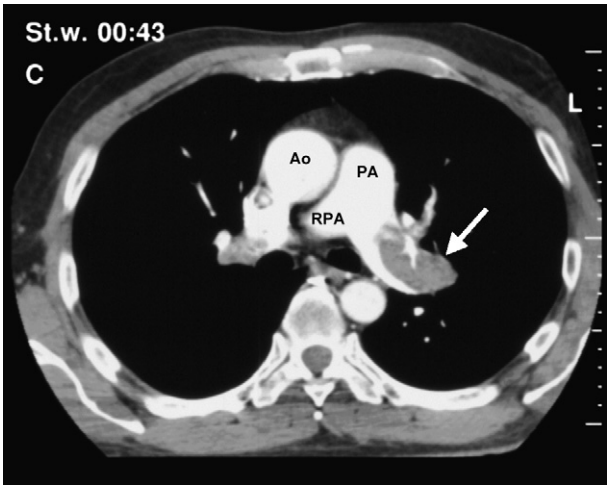

Figure 4 Computed tomography demonstrating thrombotic material in both pulmonic arteries; occlusion in of the left pulmonary artery is demonstrated (arrow). RPA = right pulmonary artery, Ao = ascending aorta.

against heparin activate the coagulation system and induce platelet aggregation. Intracardiac thrombus may have been worsened or induced by HIT in this case, though HIT was not confirmed by immunoassay. Heparin therapy was switched to intravenous dalteparin.

In patients with a mobile right heart thrombus, the incidence of pulmonary embolism is 97% and reported mortality is over 44%.[4,5] Investigators have recommended either urgent surgical treatment or thrombolysis of mobile right heart thrombus, although prospective data of optimal treatment are lacking.[4] The patient was not treated with thrombolytic therapy because of the risk of embolisation of large thrombus fragments. In addition, thrombocytopenia was considered a relative contraindication to thrombolysis. Therefore, the patient was scheduled for urgent surgical removal of the mass.

At surgery, a large thrombus was resected from the right atrium and pulmonary artery. Histopathology confirmed thrombus and cultures were negative. Platelet count normalized within a few days. The patient was discharged in stable condition with acenocoumarol thromboprophylaxis.

In conclusion, this case report demonstrated the value of echocardiography to visualize and evaluate a life-threatening right heart thrombus in a patient with pulmonary embolism. In these patients, the choice of thrombolysis or surgery will depend on additional factors.

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