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"Saved by the Pacing Catheter": Gigantic Right Atrial Thromboembolus as a Pulmonary Embolism

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ABSTRACT

Pulmonary embolism (PE) related to the presence of right heart thromboemboli entails a higher mortality rate than PE alone. Right heart thromboemboli are often associated with deep venous thrombosis. The most effective therapy for haemodynamically unstable patients remains unknown, although recent data suggest that thrombolytic therapy is associated with a better outcome.¹ We report the case of an 83-year-old woman, hospitalized with PE consequent to right heart thrombus-in-transit, in whom investigation revealed deep venous thrombosis. She required thrombolysis, given the high mortality risk that is traditionally associated with this condition.

LEARNING POINTS

- Pulmonary embolism secondary to right-sided cardiac thromboembolus entails a higher mortality rate than PE alone.
- Echocardiography is useful for the rapid detection of cardiac thrombi in patients with suspected PE.
- There is no consensus on the best therapeutic approach in this context, although there is some evidence that supports thrombolysis.

KEYWORDS

Pulmonary embolism, right heart thromboembolus, deep venous thrombosis, thrombolysis

INTRODUCTION

Right intracardiac thrombi are rare events. Nevertheless, they do have important diagnostic and therapeutic implications. Right heart thromboembolism (PE)^[1]. Predisposing factors include indwelling wires/catheters, dilated cavities, atrial fibrillation (AF), mechanical heart valves and other potential embolic sources. Ante-mortem diagnosis was unusual before the advent of echocardiography, as described by Covarrubias et al.^[2] in 1977. Right-sided cardiac intracavitary thromboembolus, unlike local thrombosis, is a condition frequently associated with deep venous thrombosis. Material that is temporarily lodged in the right atrium or ventricle, a condition frequently associated with deep venous thrombosis, is often depicted as a highly mobile mass, with a serpentine shape, that wanders inside the right atrium or ventricle, sometimes prolapsing through the tricuspid and/or pulmonary valves during the cardiac cycle. The embolus is normally seen as a narrow pedicle. The echocardiographic characteristics are important to distinguish it from a mural thrombus: less mobility, a wider implantation base and the occasional presence of small focal calcifications^[2,3].

The true incidence is not known, although it is estimated to be between 3% and 23% in patients with PE. Right atrial thrombus-related PE is associated with a higher mortality rate (30%) than PE alone (8%)^[2,3,4].

Despite this evidence, there is no consensus on the best therapeutic approach. The largest retrospective study comparing anticoagulation, surgery and thrombolysis involved 177 cases of cardiac right thromboemboli, 98% of which were associated with deep venous thrombosis. The global mortality rate was 27.1%; however, patients submitted to thrombolysis had a higher survival rate ($p < 0.05$) with anticoagulation or surgical therapies. The major complication reported was bleeding^[5]. Furthermore, a prospective study by Pierre-Justin and Pierard^[4] concluded that, although the incidence of right cardiac thrombi is low in patients admitted to hospital, therapy with recombinant tissue plasminogen activator (rt-PA) seems to be rapidly efficient in improving pulmonary perfusion in a subgroup of patients with PE secondary to right heart thromboembolus.

Hence, there is data supporting thrombolytic therapy as the first-line treatment in this context.

CASE REPORT

The patient was an 83-year-old Caucasian woman, with a medical history of hypertension, ischemic cardiomyopathy and a VVI pacemaker for 10 years. She presented in the emergency room (ER) complaining of a twinge-like left hemithoracic

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Laboratory findings

Elevation of inflammatory markers: leucocytosis with neutrophilia and increased C-reactive protein (CRP)

High D-dimer ($>5.0 \mu\text{g/ml}$)

Arterial blood gas analysis (room air): hypoxaemia and hypocapnia (SatO_2 90%, pO_2 69 mmHg, pCO_2 30 mmHg)

Table 1: Laboratory findings

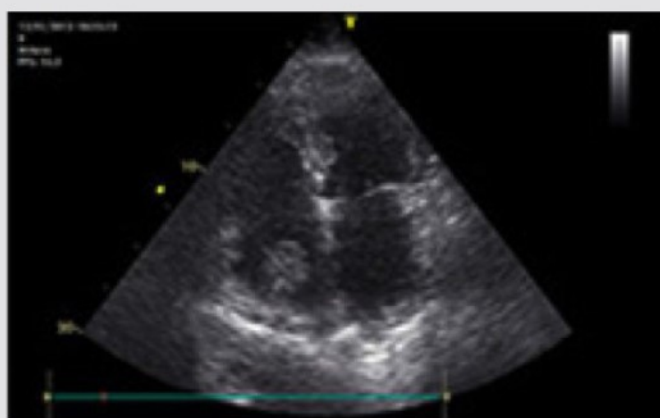


Figure 1: Thromboembolus in the right atrium, adhering to the pacing catheter.



Figure 2: Mobile thromboembolus, serpentineous, prolapsing through the tricuspid valve.

characteristics, emphasizing having suffered fatigue for months, which had worsened during the previous week. She or other complaints. On admission: afebrile, slight dyspnoea, blood pressure (BP) 160/90 mmHg, irregular pulse on auscultation revealed an aortic grade II/VI intensity mesosystolic murmur. Concerning laboratory findings are listed in Table 1. Transthoracic echocardiogram showed mild aortic valvular stenosis (fibrodegenerative), bilateral atrial dilatation, normal left ventricular systolic function and a normal right ventricle. A bulky image was visible on the right atrium, suggestive of a thrombus adherent to the pacing catheter (greatest diameter of 23 mm). Pulmonary Artery Systolic Pressure (PASP) was estimated at 45 mmHg (Figs. 1 and 2).

Results of the contrasted thoracic CT scan were as follows: "Pulmonary artery repletion defects, concerning the left main branch thoroughly involving the superior lobe branch and its segmental branches. On the right, there is a small repletion defect involving the middle branch and a distal thrombus in the right pulmonary artery that does not cause repletion defect of the inferior or middle branches. The patient was admitted to an Intermediate Care Unit and heparinized. A multidisciplinary consultation (including a cardiac surgeon) concluded surgery would have no benefit in this context, given her advanced age and the high surgical risk. By Day 2, she showed no improvement and facing the imminent risk of massive embolization, it was decided to initiate thrombolysis with Alteplase (rtPA). Eight hours later, the patient presented with hypotension, tachycardia and decreased breath sounds on the left. Her hemoglobin level drop stood out. Thoracic CT revealed: "Atelectasis of the left lung associated with major left pleural effusion. A chest tube was placed with immediate drainage of 1000 cc of haematic content; volaemic resuscitation with fluids and blood transfusion was successful in restoring haemodynamic stability. Anticoagulation was reinstated with unfractionated heparin, with periodic monitoring of Partial Thromboplastin Time (aPPT) evaluation.

By Day 3 post-thrombolysis, the echocardiography showed complete resolution of the intracardiac thromboembolus. PASP was 35 mmHg; the patient was stable and the chest tube was removed. On Day 4, a venous echodoppler of the lower limbs showed no thrombosis on the left femoral and popliteal veins. On Day 5, bridging with warfarin was initiated. During the next 10 days the patient progressively began to sit up, remaining stable and asymptomatic. She was discharged on Day 18 and referred to her primary care appointment, with complete resolution of her illness.

CONCLUSION

There is no consensus on the therapeutic approach for patients with PE secondary to intracardiac thromboembolism. Literature on how to tackle the ideal therapy in this context is scarce: it considers anticoagulation, thrombolysis or surgery. In a haemodynamically unstable patient, thrombolytic therapy is considered the best choice, however there is no agreement on the dose and timing. In an otherwise stable patient, anticoagulation is preferred. In the reported case, the patient had predisposing factors for cardiac thrombi such as Atrial Fibrillation and an indwelling wire. However, the presence of deep venous thrombosis seemed to be the major player, and, in our opinion,

presence of the pacing catheter clutching to those emboli, misfortune would surely have ensued. The thrombus' dimensions and the risk for massive PE were fundamental determinants in the decision to undertake thrombolytic therapy.

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