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CASE REPORT

Right Atrial Thrombus as a Cause of Acute Pulmonary Embolism: A Case Report

Trombo en la aurícula derecha como causa de embolia pulmonar aguda: reporte de un caso clínico

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ABSTRACT

Thrombi mainly form in the left heart chambers, particularly in dilated atria and in the context of atrial fibrillation. The presence of thrombi in the right atrium is uncommon; they are more often the result of peripheral venous circulation emboli, and less frequently, they originate in situ. In Latin America, the incidence of deep vein thrombosis is approximately 1 case per 1000 people per year, and that of pulmonary embolism is 0,5 cases per 1000 people per year. This clinical case presents a 64-year-old male patient diagnosed with a serpentine thrombus in the right atrium and dilated right heart chambers, successfully treated with oral anticoagulation.

Keywords: Right Atrial Thrombus; Acute Pulmonary Embolism; Echocardiography; Computed Tomography; Oral Anticoagulation.

RESUMEN

Los trombos se forman principalmente en las cavidades izquierdas del corazón, especialmente en aurículas dilatadas y en presencia de fibrilación auricular. La presencia de trombos en la aurícula derecha es poco común; con mayor frecuencia se trata de émbolos procedentes de la circulación venosa periférica y, con menor frecuencia, se originan in situ. En América Latina, la incidencia de trombosis venosa profunda es de aproximadamente 1 caso por cada 1000 personas por año, y la de embolia pulmonar es de 0,5 casos por cada 1000 personas por año. Este caso clínico presenta a un paciente masculino de 64 años diagnosticado con un trombo serpenteante en la aurícula derecha y dilatación de las cavidades cardíacas derechas, tratado exitosamente con anticoagulación oral.

Palabras clave: Cesárea; Prevalencia; Desigual en Atención Medica; Historias Clínicas; Estudios Retrospectivos.

INTRODUCTION

Intracardiac masses often pose a significant diagnostic and therapeutic challenge. In many cases, the mass is discovered incidentally and subsequently confirmed upon further evaluation.[1] Among the possible diagnoses in this broad category are thrombi, which are more frequently located in the left heart chambers

than in the right, and within the right chambers, more often in the atrium than in the right ventricle.[2]

Thrombi in transit in patients with pulmonary embolism are reported in the literature in 4–18 % of cases. The mere presence of thrombi in the right heart chambers indicates a poor prognosis, and their management remains controversial.[3] These thrombi most commonly originate from deep veins in the lower limbs, although a minority are formed in situ, particularly in cases involving venous stasis (e.g., right atrial dilation, atrial fibrillation) or in association with foreign bodies (such as venous catheters or pacemaker leads). These are often immobile, heterogeneous masses adhered to the wall with distinct intracavitary margins.[4]

The pathogenesis of venous thromboembolism is classically described by Virchow's triad: blood stasis, endothelial injury, and hypercoagulability. Known risk factors include immobility, certain surgical procedures, cancer, sedentary lifestyle, obesity, long-distance travel, heart failure, antiphospholipid syndrome, use of oral contraceptives, hereditary thrombophilia, hyperhomocysteinemia, among others.[5]

Echocardiographic evaluation—both transthoracic (TTE) and especially transesophageal (TEE)—can provide valuable diagnostic and therapeutic information in selected patients with venous thromboembolism.[6] The detection of intracavitary thrombotic material and/or structural septal abnormalities can offer crucial data on disease severity and the underlying anatomical cause, particularly in cases of pulmonary or paradoxical embolism. The risk of systemic embolization in patients with a patent foramen ovale is associated with high mortality, exceeding 45 %.[7]

There are no controlled trials in the literature that define optimal management for these situations. Only isolated case reports exist, describing the use of anticoagulation, systemic thrombolysis, or surgical thrombectomy—with varying outcomes.[5]

We present a case of pulmonary embolism in a patient in whom TEE revealed a serpentine thrombus in the right atrium extending across the foramen ovale. We discuss the diagnostic and therapeutic strategies considered.

CASE REPORT

Clinical Case Presentation

A 64-year-old Caucasian male patient with a history of smoking for approximately 40 years, chronic alcoholism for over 20 years, essential arterial hypertension, and stable coronary artery disease.

Approximately three weeks prior to admission, the patient began experiencing exertional dyspnea with minimal effort, which worsened significantly in the last seven days. He also reported a sudden episode of loss of consciousness with spontaneous recovery.

On physical examination, notable findings included mucocutaneous pallor, lower limb edema with poor Godet sign, not extending beyond the proximal third of both legs, decreased vesicular breath sounds in both lung fields without rales or wheezes, rhythmic heart sounds with low tone, a grade III/VI systolic murmur at the lower left sternal border, blood pressure of 105/60 mmHg, and heart rate of 90 bpm.

Basic laboratory tests revealed elevated liver enzymes, while cardiac enzymes remained within normal limits. D-dimer testing could not be performed due to lack of availability.

Electrocardiogram (ECG) showed sinus rhythm, irregular, with a markedly right-deviated QRS axis, exhibiting an S I, Q III, T III pattern, along with an S I, II, III pattern, qR in V1, R/S ratio >1 in V1 and <1 in V6.

Chest X-ray revealed a low-density radiopaque image with poorly defined borders affecting the right apex and infraclavicular region. This was associated with bilateral hilar thickening, more prominent on the right side, with well-defined contours suggestive of a vascular origin. The cardiothoracic index was at the upper limit of normal. The right diaphragmatic dome was visible.

Transthoracic echocardiography (TTE) revealed dilatation of the right heart chambers, right ventricular systolic dysfunction, and severe pulmonary hypertension. A mass was visualized in the right atrium, suggestive of thrombus. There was also systolic paradoxical motion of the interventricular septum, indicative of elevated right-sided pressures.

Given these findings, a transesophageal echocardiogram (TEE) was performed, which confirmed the presence of a long, serpentine mass. One end of the mass moved in and out through the tricuspid valve, while the other end was attached to the interatrial septum and extended through the foramen ovale, with a portion visualized in the left atrium (figure 1A).

A contrast-enhanced chest CT scan (figure 1B) revealed a hypodense, elongated image measuring 62 × 15 mm at the level of the pulmonary artery trunk, extending into both right and left pulmonary branches, producing a contrast filling defect consistent with thrombosis at this level.

Additionally, a hyperdense lesion with poorly defined borders was noted in the right pulmonary apex, both infraclavicular regions, and the left basal hilum. The latter showed cavitation, all of which are findings suggestive of massive branch pulmonary embolism (figure 2).

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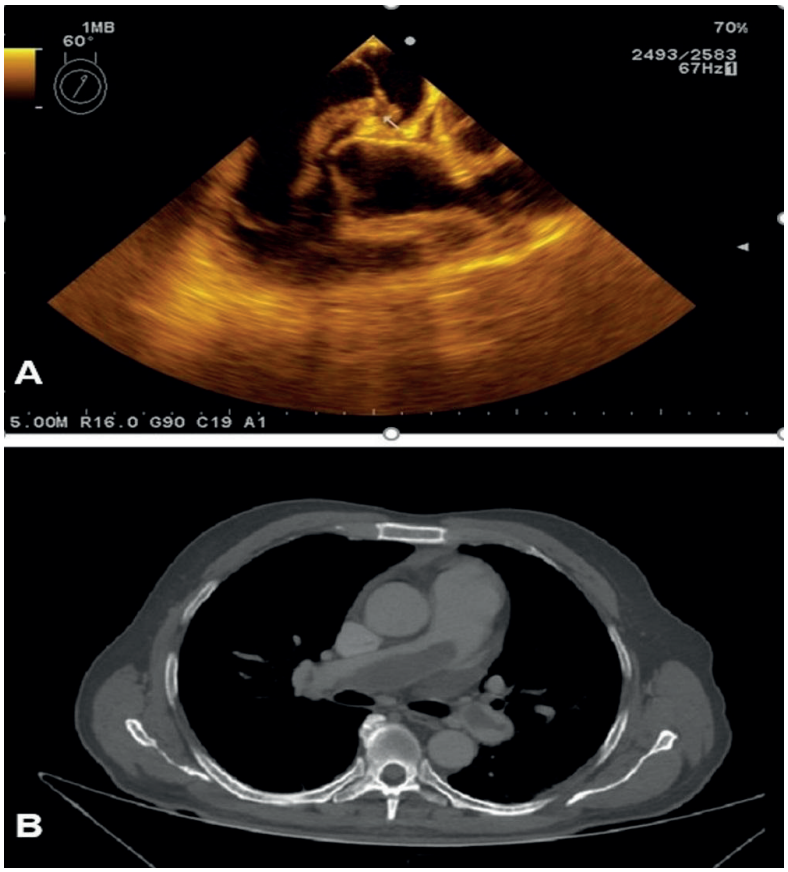

Figure 1. A. Transesophageal echocardiogram at 60º showing a serpentine thrombus in the right atrium (RA) traversing the interatrial septum (IAS) through the foramen ovale (FO) and entering the left atrium (LA). **B**. Contrast-enhanced chest CT scan showing thrombus in both the right and left pulmonary arteries, occupying nearly the entire lumen of the vessels

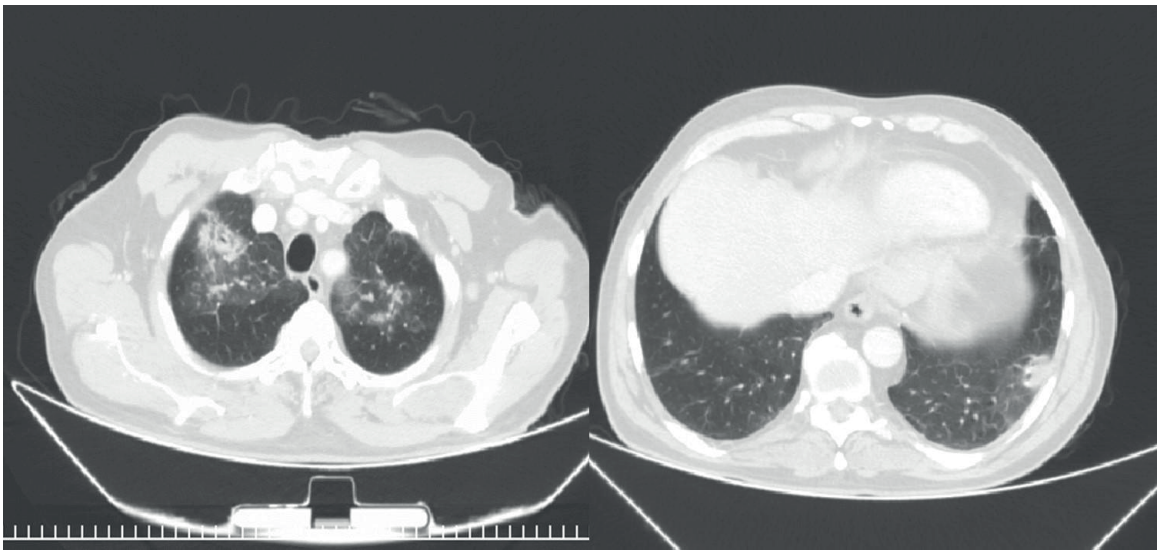

Figure 2. A hyperdense image is observed in the right pulmonary apex, both infraclavicular regions, and the left basal hilum, the latter showing cavitation

The patient was admitted with a diagnosis of a serpentine thrombus in the right atrium extending through a patent foramen ovale (PFO) into the left atrium, as the underlying cause of a massive branch acute pulmonary embolism with associated risk of paradoxical embolism.

After a multidisciplinary team discussion, the decision was made to initiate treatment with oral anticoagulation and low molecular weight heparin (LMWH), which was continued until the international normalized ratio (INR) reached the therapeutic range.

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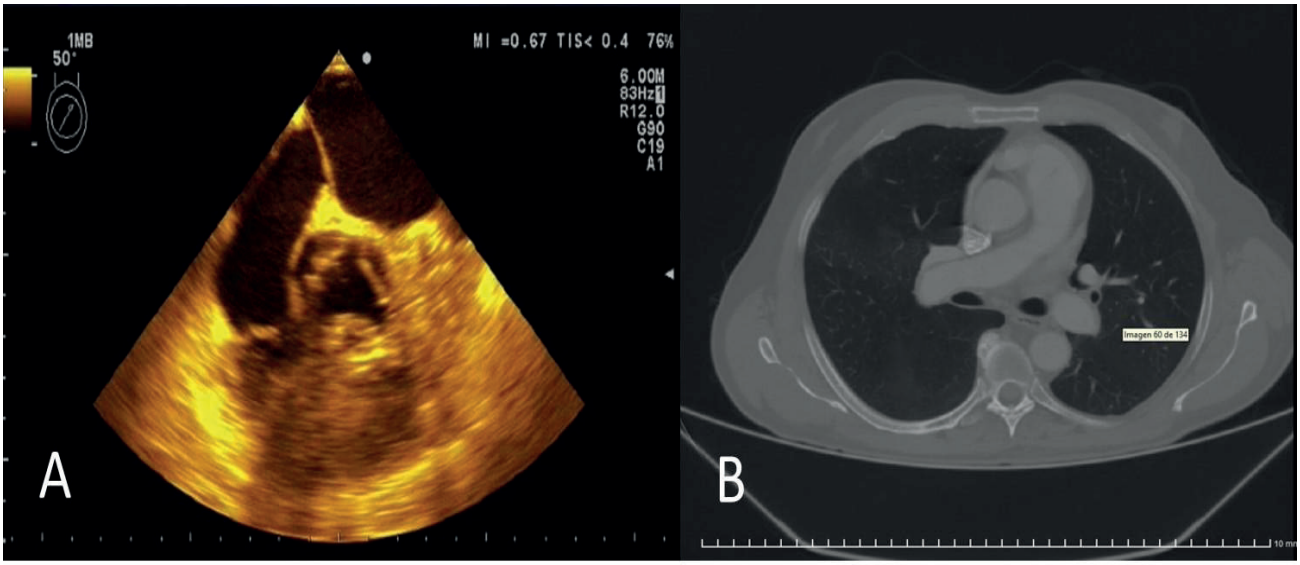

Figure 3. A. Transesophageal echocardiogram at 50º showing absence of thrombus in the right atrium and an intact interatrial septum. **B.** Contrast-enhanced chest CT scan showing no evidence of thrombus in the lumen of the right or left pulmonary arteries

After 21 days of hospitalization and clinical improvement, the patient was discharged and scheduled for monthly follow-up. At the three-month follow-up visit, a transesophageal echocardiogram (TEE) and contrast-enhanced chest CT scan were performed to assess clinical and radiological evolution (figure 3).

As observed three months after treatment, imaging studies confirmed the absence of thrombi in both the right atrium and pulmonary arteries. Clinically, the patient showed significant improvement, remained on oral anticoagulation with a therapeutic INR, and continued follow-up under the care of the internal medicine team.

DISCUSSION

Few cases like ours have been reported in the literature, and it represents a diagnostic and early therapeutic challenge.[8] Previous studies agree that mortality in the first 24 hours exceeds 45 %.[9]

Right heart thromboembolism is defined as deep vein thrombosis that embolizes to the right heart chambers, mainly the right atrium, or forms de novo in that location.[5]

Thrombi are laminar or spherical mobile masses, non-infiltrative to the wall, with echogenic density greater than that of the adjacent myocardium. They are usually located in the left atrial appendage or at the junction between the inferior vena cava and the right atrium. Left ventricular thrombi affect 5–15 % of patients with acute myocardial infarction. Right-sided cardiac thrombi are rare, with an incidence of 10,8 %.[8]

Three types of right heart thrombi are described in the literature: [11,12]

Type A: Serpentine, extremely mobile thrombi, associated with pulmonary embolism and/or deep vein thrombosis (78–98 %).

Type B: Immobile thrombi, morphologically similar to those found in the left ventricle. Associated with pulmonary embolism in 35–40 % of cases.

Type C: Extremely mobile thrombi, non-serpentine in shape, with an intermediate risk of pulmonary embolism (~65 %).

Most thrombi are diagnosed by echocardiography, though this remains a diagnostic challenge in patients with poor acoustic windows, suboptimal endocardial border definition, or difficulties in apical image acquisition.[6]

The limited visualization of the right heart chambers in some patients using transthoracic echocardiography (TTE) makes transesophageal echocardiography (TEE) a crucial tool for diagnosis in these cases. Visualizing the interatrial septum is especially important, as a defect at this level increases mortality due to the risk of paradoxical embolism.[4]

Currently, anticoagulant therapy remains the treatment of choice according to some authors in the management of pulmonary embolism. Other therapeutic options, as previously mentioned, include thrombolysis and surgical thrombectomy.[5]

In our clinical case, the combination of clinical manifestations, electrocardiographic findings indicative of acute pulmonary embolism, and imaging evidence from echocardiography (a serpentine thrombus in the right atrium crossing through the foramen ovale with risk of paradoxical embolism), later confirmed on chest CT, leads us irrefutably to the diagnosis of acute pulmonary embolism caused by a thrombus originating in the deep veins and lodged in the right atrium.

The changes observed in follow-up imaging studies after three months may be explained by two possible

scenarios:

- Thrombus detachment, which is unlikely, as this would have likely resulted in a fatal outcome.
- Thrombus dissolution due to anticoagulant therapy, which is more consistent with the patient's clinical improvement following treatment.

As mentioned earlier, an alternative therapeutic option would have been thrombolysis. Contreras et al. noted that floating right atrial thrombi are associated with high mortality, and any delay in treatment can be fatal.[13] In their case, thrombolysis was chosen. However, this was not selected in our case due to the high bleeding risk in this patient.

It is essential to consider various factors when making clinical decisions in these scenarios, such as:

- The patient's hemodynamic status,
- The level of the healthcare facility,
- Available therapies,
- The attending physician's experience.

CONCLUSIONS

Both transthoracic and transesophageal echocardiography are essential for diagnosing thrombi in the right heart chambers, and contrast-enhanced chest CT remains the main imaging modality for diagnosing pulmonary embolism.

In cases with a high bleeding risk, anticoagulation remains a safe and effective management strategy.

An increased number of reported cases and further studies on this pathology are necessary to determine the optimal first-line treatment in this type of patient.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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