Thrombus Migration: An Uncommon Presentation in Obstructive Shock

Migração de Trombo: Uma Apresentação Rara no Choque Obstrutivo

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Resumo

A presença de um trombo em migração no tromboembolismo pulmonar é rara, sendo a sua presença associada a uma maior mortalidade. Esta entidade continua a levantar problemas sobre qual a melhor opção terapêutica a instituir nestes doentes. Os autores apresentam um caso de uma mulher de 76 anos admitida na sala de reanimação em contexto de choque a esclarecer. A realização da ecocardiografia revelou-se fundamental para o diagnóstico de choque obstrutivo em contexto de tromboembolismo pulmonar permitindo atuar de forma precoce e com sucesso.

Palavras-chave: Choque; Ecocardiografia; Embolia Pulmonar; Trombose

Abstract

Thrombus migration in pulmonary embolism is a rare finding, and its presence is associated with an increased mortality. This entity still raises some problems in its approach. We report a case of a 76-year-old woman admitted in the emergency room in shock. Transthoracic echocardiogram was essential in the diagnosis of obstructive shock in the context of pulmonary embolism allowing immediate and successful treatment.

Keywords: Echocardiography; Pulmonary Embolism; Shock; Thrombosis

Introduction

Pulmonary embolism represents one of the most common causes of cardiovascular death,[1,2] just preceded by acute coronary syndromes and cerebrovascular accidents. A thrombus migration is a very rare finding (4%)[2] but should be considered in the presentation of a medical emergency especially due to its potential to evolve to a fatal event such as an acute massive pulmonary embolism, frequently linked to high mortality rates.[3] Nevertheless, it still raises questions about its optimal medical approach in part due to the lack of randomized prospective studies about this entity.

Case Report

Seventy-six-year-old woman, with a previous medical history of arterial hypertension, dyslipidaemia, hypothyroidism and mitral valve plasty (previous rheumatic fever) presents to the emergency department with unspecific complaints of malaise, productive cough and dyspnoea for the past 15 days.

She was hospitalized one month before due to a non-ST elevation myocardial infarction. Coronarography excluded significant coronary lesions, identifying a small, poorly defined fistula between the right coronary artery and the pulmonary artery. Transthoracic echocardiography at discharge was considered normal.

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At admission, the patient was conscient, apyretic, tachycardic (heart rate (HR) 100 bpm), hypotensive (non-invasive arterial pressure (AP) 95/53 mmHg) and with peripheric oxygen saturation of 90% (on room air). No other relevant findings at physical examination. The patient became haemodynamically unstable (AP 67/35 mmHg) and unresponsive. Physical examination revealed poor peripheral perfusion (capillary refill time > 4 seconds), Glasgow coma scale 12 points, peripheral oxygen saturation 80% and HR 136 bpm (sinus rhythm on cardiac monitor).

Electrocardiogram (Fig. 1) with sinus tachycardia (HR 136 bpm), SIQIII pattern and a slight ST elevation in aVR and ST depression in DI, aVL and precordial leads.

Laboratory results and arterial gasometry revealed elevated lactate (8 mmol/L) and a mild elevation of myocardial necrosis markers (troponin I 120 pg/mL – normal range < 14 pg/mL)).

It was assumed shock of unknown etiology. Transthoracic echocardiography (TTE) revealed right cavities dilatation (Fig. 2) and the presence of a serpiginous echogenic mass (Fig. 3) that protruded from the inferior vena cava crossing the right atrium and reaching the right ventricle (thrombus migration).

Angio-computed tomography was not performed due to the patient haemodynamic instability. Thrombolysis (alteplase), fluid challenge and vasopressor (noradrenaline) perfusion were immediately started with a good clinic response.

Hemodynamic stability was achieved 5 hours after the thrombolysis. An unfractionated heparin perfusion was initiated after the thrombolysis and discontinued 24 hours after the patient was admitted. Afterwards the anticoagulant therapy chosen was subcutaneous enoxaparin injections until discharge. The remaining hospitalization was uneventful and the patient was discharged 7 days after on a non-vitamin K antagonist oral anticoagulant - apixaban. Transthoracic echocardiography revaluation before discharge revealed mild dilatation of the right cavities with mild systolic dysfunction of the right ventricle (TAPSE 15.7 mm) and a pulmonary artery systolic pressure of 28 mmHg.

Follow-up at 12 months was also uneventful and the patient displayed no limitation to daily physical efforts.

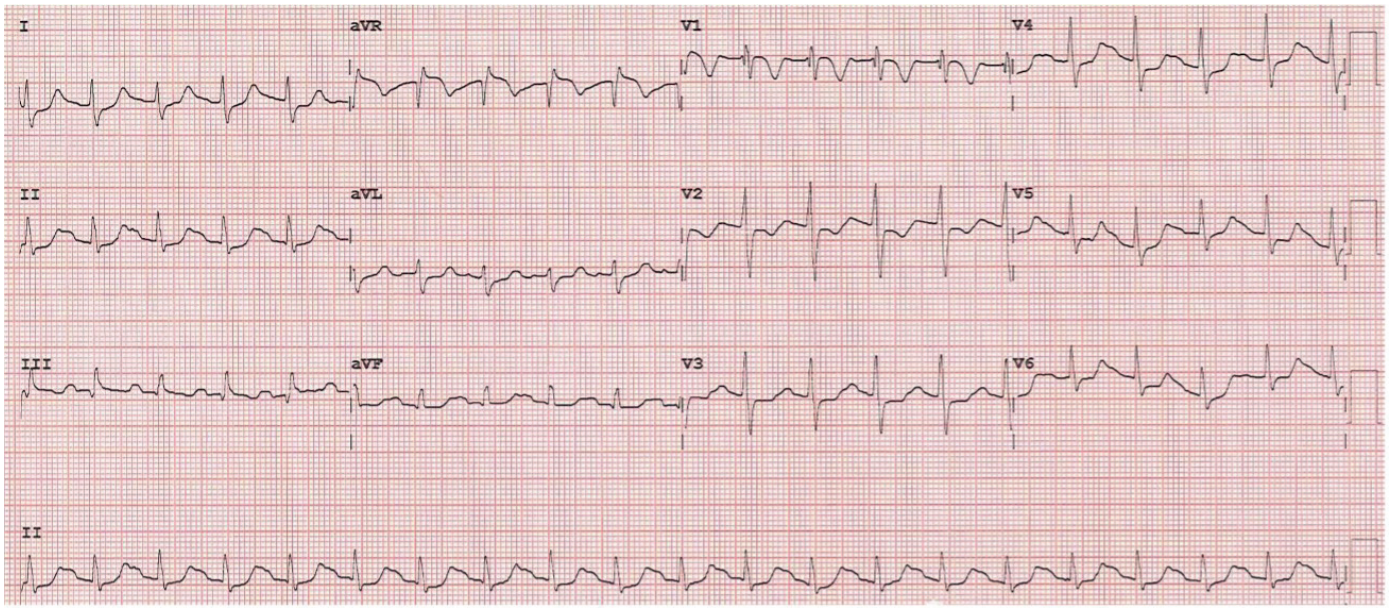

Figure 1. Sinus tachycardia (HR 136 bpm), SIQIII pattern with ST elevation in aVR and ST depression in DI, aVL and precordial leads.

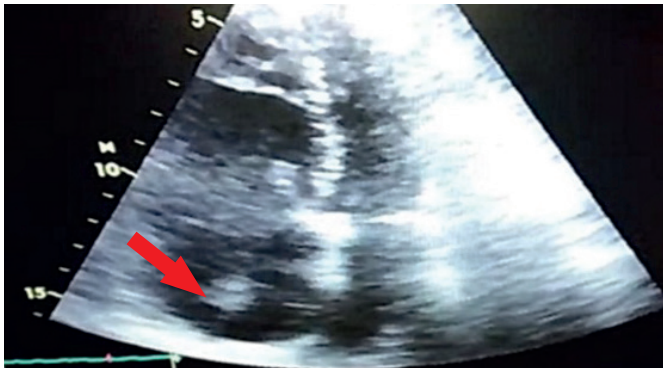

Figure 2. Transthoracic echocardiography (apical 4 chamber view) showing right cavities dilatation and the presence of an echogenic mass in the right atrium (red arrow).

Figure 3. Transthoracic echocardiography (subcostal 4 chamber view) showing the presence of a serpiginous echogenic mass in the right atrium (red arrow).

Discussion

The incidence of thrombus migration is rare.[1,2,4] It is associated with cardiorespiratory arrest and intra-hospital mortality in about 50% of the cases. The presence of a migration thrombus could be seen in deep venous thrombosis or any cardiac pathology that promotes thrombus *in situ*.[5]

A thrombus migration in the presence of pulmonary embolism requires an emergent intervention. Medical literature describes three types of thrombi: 1) Type A, the most common and with high capability of embolization; 2) Type B, small mobile thrombus adherent to the ventricle wall (*in situ*); 3) Type C, the rarest with high mobility and capable of mimicking atrial mixomas.[3,5] This clinical case concerns a thrombus migration, type A. The initial electrocardiogram raised the hypothesis of an acute pulmonary embolism in its classical finding.

Echocardiography is fundamental allowing the evaluation of the hemodynamic status, and helping in the differential diagnosis and risk estratification[6] in a patient in shock. In the case of pulmonary embolism, we have several echocardiographic signs that could be used as prognostic markers. However, the most recent scientific guidelines discourage the use of transthoracic echocardiogram (TTE) as a routine in differential diagnosis of patients with low/intermediate risk[7] of having an acute pulmonary embolism. Nevertheless, TTE is of high value in the case of shock due to its high specificity as a rule-in test, especially when the patient cannot undergo other types of exams.[8]

There is no scientific evidence regarding the best strategy to approach a patient with a migrating thrombus. The actual recommendations are based on case series and few retrospective studies. There is a lot of controversy about the best treatment especially concerning the haemodynamic stable patients.

The risk/benefit of each therapeutic option should be considered individually, and the decision should be made by a multidisciplinary team.

Conclusion

The intervention in acute pulmonary embolism with thrombus migration occurring in a haemodynamic stable patient is not clear. In this case report the patient progression to haemodynamic instability simplified our approach as thrombolysis was the first line treatment.

Responsabilidades Éticas

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