Thrombus-in-transit and Paradoxical Embolism in Patient with ST-Segment Elevation Acute Coronary Syndrome

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Introduction

Paradoxical embolism (PE) was defined by Julius Cohnheim, in 1877, as a venous thromboembolism that moves to the systemic circulation by right-left intrathoracic shunt.[1] It usually occurs in combination with pulmonary thromboembolism (PTE), which suddenly increases the right heart pressure, favoring the thrombus displacement.[2] When this association occurs, the incidence of serious complications such as acute myocardial infarction (AMI) is high.

Risk factors for thrombotic events, anatomic venoarterial defects and right-left pressure gradient in the shunt are clinical conditions necessary for an EP to be present.[2] Although generally presumptive, its diagnosis is certain when thrombus-in-transit is detected due to defects between the right and left areas of the heart. The most common one is the patent foramen ovale (PFO).

The most frequent initial presentation of PE consists of an acute ischemic cerebrovascular event, which may be transient or not, due to the tendency of migration to the posterior cerebral arteries.[1] However, about 10 to 15% of the cases are acute coronary syndromes (ACS). Although described, this association is rare and it is estimated that, in the general population, less than 1% of AMI is caused by PE.[3] Among patients aged up to 35, about 25% of all acute coronary events have this etiology.[4]

Since PTE and AMI are differential diagnoses for chest pain and dyspnea, it is important to associate them in cases of PE. This report describes a case of ACS as unusual etiology of PE due to PTE and rare thrombus-in-transit.

Case Report

R.A.R., male, 38 years old, former smoker, with a history of trauma in the left lower limb (LLL) in 2004, evolving with chronic venous insufficiency after vascular surgery. In January 2017, he presented progressive edema, pain and hyperemia in LLL. A Doppler ultrasonography was performed, diagnosing deep vein thrombosis (DVT) of superficial and deep femoral veins, the treatment for which consisted of oral anticoagulation. The patient sought emergency treatment due to temporary loss of peripheral vision, followed by sudden dyspnea, tightness in the chest, palpitations and lipotomy, and was hospitalized for investigation. While in hospital, he presented a new episode of chest pain, with sudden and intense dyspnea, associated with pre-syncope and profuse sweating. On physical examination, he was tachycardic and tachypsistic, with a universally reduced vesicular murmur, and presented palpation pain, edema and hyperemia in the LLL. Due to the acute chest discomfort, electrocardiogram (ECG) was performed (Figure 1), revealing extensive anterior infarction by ST-segment elevation in V1 to V6 and D1 and AVL. Before the ECG, coronary angiography was performed (Figure 2) and, due to total thromboembolic occlusion in the anterior descending artery, the procedure was thrombus aspiration and angioplasty with stent with thrombolysis in myocardial infarction (TIMI) II grade flow. Chest angiotomography diagnosed acute extensive bilateral PTE due to obstruction of the pulmonary artery trunk and transthoracic echocardiogram (TTE), which revealed mobile and friable thrombi in the interatrial septum (thrombus-in-transit), measuring, in the left atrium, 1.3 × 1.8 cm and, in the right, 4.5 × 1.3 cm (Figure 3).

EP was then suggested as the etiology for infarction. Transesophageal echocardiography (TEE) with microbubble test (Video 1) revealed PFO and extensive DVT was diagnosed with Doppler ultrasound of the LLL. The medical management consisted of full anticoagulation, and thrombolysis was not performed due to hemodynamic stability and maintenance of right ventricular function. In the control TTE, no new thrombi were detected. The patient evolved with hospital discharge, with recommendation of antiplatelet therapy and anticoagulation, and a proposed PFO repair and investigation for thrombophilia with a hematologist.

Discussion

Coronary embolism is an important non-atherosclerotic cause of AMI, with prevalence of around 4 to 13%.[5] Atrial fibrillation, cardiomyopathies and valvular diseases are the main sources of coronary emboli. However, a rare cause is paradoxical coronary embolization, usually from lower limb DVT, accounting for only 0.67% of AMI etiologies.[3] This PE is possible due to an atrial septal defect, and is favored if associated with pulmonary embolism.[2]

Although the PTE is frequent in patients with severe heart diseases due to prolonged immobilization, its simultaneous occurrence with AMI is relatively rare and is usually because the PE derives from PFO (atrial septal defect with a prevalence

Keywords

Embolism; Paradoxal; Acute Coronary Syndrome; Foramen Ovale; Patent; Pulmonary Embolism; Echocardiography/methods; Anticoagulants; Ultrassonography; Doppler/methods.

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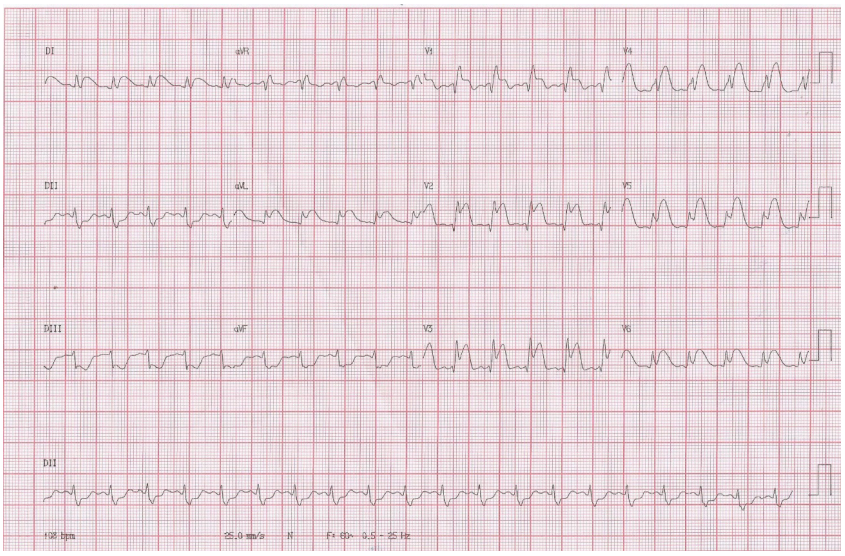


Figure 1 – *Electrocardiogram showing ST-segment elevation in V1 to V6 and D1 and AVL.*

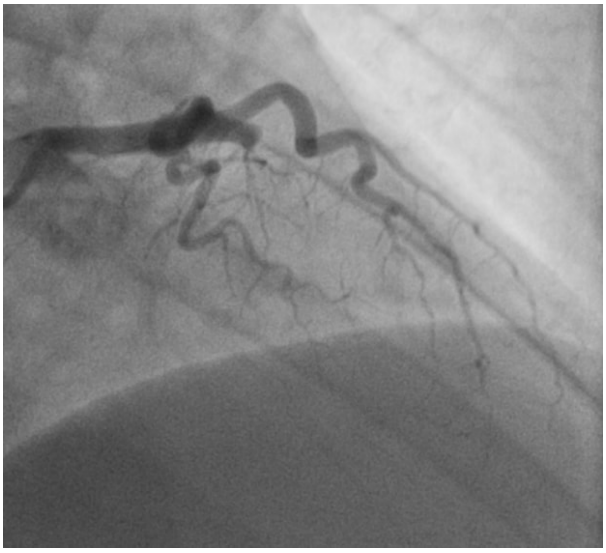


Figure 2 – *Coronary angiography showing total obstruction in the proximal third of the anterior descending artery (without other lesions in the other arteries), suggesting thromboembolic cause.*

of 27% in the general population).[6] Diagnostic errors may occur due to the similarity between findings of acute PTE, ACS and heart failure. Therefore, one must be vigilant of any signs of dyspnea without an apparent cause, elevation of D-dimer, or distension of the right ventricle.

Diagnosis of PE is generally presumptive and consists of: arterial thromboembolism in the absence of atrial fibrillation, left heart disease or severe atherosclerosis; detection of right-left shunt by PFO or atrial septal defect; and presence of venous thromboembolism or pulmonary embolism.

Diagnosis of certainty of PE requires demonstration of thrombus by venoarterial defect by angiotomography or echocardiography, as in the report. However, because it is a transitory phenomenon, detection of thrombus passage is quite rare.[3] The first case diagnosed by echocardiography was described by Nellessen et al.[7] in 1985 and, in a review by Fauveaua et al.,[8] only 88 cases of thrombus crossing the PFO were described in the literature. However, due to the greater availability of imaging techniques, the identification of this entity tends to be more frequent, and the physician

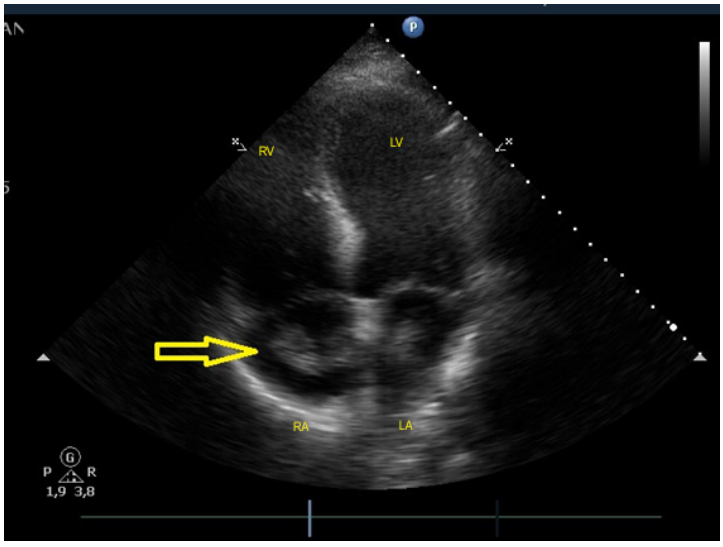
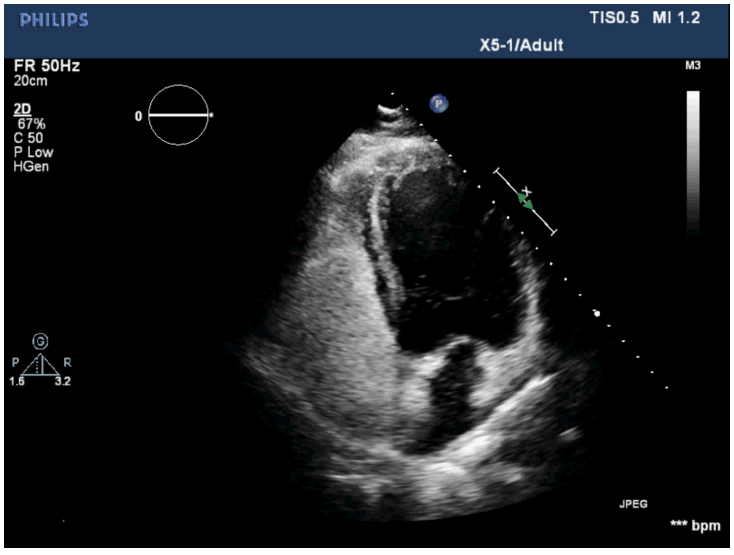


Figure 3 – *Two-dimensional echocardiography with 4-chamber view showing extensive thrombus-in-transit through the interatrial septum measuring, on the left atrial face, 1.3 × 1.8 cm and, on the right atrial face, 4.5 × 1.3 cm. RV: right ventricle; LV: left ventricle; RA: right atrium; LA: left atrium.*



Video 1 – *Two-dimensional echocardiography with 4-chamber view, revealing the passage of microbubbles from the right atrium to the left atrium through the patent foramen ovale. Access the video here: http://departamentos.cardiol.br/dic/publicacoes/revistadic/2018/v31_4/video_v31_4_249_ingles.asp.*

must know its management. In hemodynamically stable patients, thrombectomy associated with PFO closure is a choice in case of low surgical risk. However, anticoagulation is a choice for patients with small PFO or comorbidities that increase surgical risk. In hemodynamically unstable patients, thrombolysis is the method of choice.[9]

The management of rare cases of AMI due to PE is similar to that of cases of atherosclerotic etiology. Manual aspiration of coronary thrombus, with or without stent angioplasty (use of dual antiplatelet therapy), is the treatment of choice, in association with anticoagulation in cases of confirmed embolic nature.[4]

Secondary prevention of another PE event includes shunt closure, anticoagulation, or both. Recent meta-analyses have shown benefits of percutaneous occlusion of PFO in the prevention of recurrence, compared to drug therapy, but this is solely based on studies on cryptogenic stroke.[10] Recurrent pregnancy, low adherence to anticoagulant therapy, age and patient choice should be determining factors in the decision between PFO occlusion and oral anticoagulant therapy throughout life.[11]

It is concluded that, in the context of a young patient admitted because of a condition suggestive of AMI, the

etiology of PE should be considered, especially if coronary angiography shows obstruction without evidence of atherosclerotic lesions and suspected associated PTE/DVT, in which case echocardiography and screening for venous thromboembolism are important.

Authors' contributions

Research creation and design: Aguiar DL, Plens ICM, Bezerra GN, Belém LS, Lino DOC; Data acquisition: Aguiar DL, Bezerra GN, Plens ICM, Belém LS, Lino DOC; Data analysis and interpretation: Aguiar DL, Bezerra GN, Plens ICM; Statistical analysis: Aguiar DL, Belém LS, Lino DOC; Funding: Aguiar DL, Belém LS, Lino DOC; Manuscript writing: Aguiar DL, Bezerra GN, Plens ICM, Belém LS, Lino DOC; Critical revision of the manuscript as for important intellectual content: Aguiar DL, Bezerra GN, Plens ICM, Belém LS, Lino DOC.

Potential Conflicts of Interest

There are no relevant conflicts of interest.

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Academic Association

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