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CASE REPORT

OCCLUSION OF PATENT FORAMEN OVALE AFTER MYOCARDIAL INFARCTION DUE TO CORONARY ARTERY EMBOLISM. CASE REPORT

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ABSTRACT

Patent foramen ovale is a congenital heart disease prevalent in about 30% of the adult population and is associated in the genesis of ischemic cerebrovascular events, peripheral arterial occlusions, and less commonly, with acute coronary syndrome. Paradoxical coronary artery embolism is considered rare, being an underdiagnosed cause of acute myocardial infarction in patients with a low risk profile for coronary heart disease. We report a case of acute myocardial infarction with ST-segment elevation of the anterior wall due to presumed paradoxical embolism with subsequent percutaneous occlusion of the patent foramen ovale.

KEYWORDS: FORAMEN OVALE, PATENT; EMBOLISM, PARADOXICAL; MYOCARDIAL INFARCTION; SEPTAL OCCLUSION DEVICE; HEART DEFECTS, CONGENITAL

INTRODUCTION

From the end of the fourth week of embryonic development, the primitive atrium divides into right and left atria by the fusion of two septa, the septum primum and the septum secundum, the latter forming an incomplete division between the atria, receiving the denomination of foramen ovale[1]. It consists of an embryonic formation essential for the maintenance of fetal circulation, since it allows the continuous diversion of oxygenated blood from the right atrium to the left atrium[2]. After birth, due to the increase in pressure in the left atrium, due to the increase in pulmonary venous return, the septum primum is pressed against the septum secundum, adhering to it, succeeding the closure of the foramen and the formation of the fossa ovale. When, for some reason, this fusion does not happen or it occurs inappropriately, we characterize the patent foramen ovale (PFO)[1].

Considered a common abnormality, the prevalence of foramen ovale patency in the adult population is approximately 30%3. The PFO is functionally closed most of the time and in most cases it does not cause systemic repercussions, and may go undetected [4,5]. However, in situations such as deep inspiration, coughing and Valsalva maneuver where the pressure of the right atrium exceeds that of the left atrium, the PFO opens, allowing the passage of emboli from the venous to the systemic circulation, establishing a paradoxical embolism (PE)[4,6]. Deep venous thrombosis (DVT) of the lower limbs is an important emboligenic source, but other mechanisms have been suggested, such as the formation of the thrombus in the foramen itself due to blood stasis[2].

Two diagnostic methods can be used to document PFO: transesophageal echocardiography (TEE), traditionally considered standard, and transcranial Doppler, both sensitized with the bubble study and Vasalva maneuver[7]. In TEE, in patients with PFO, at least three microbubbles should be visualized inside the left atrium, between the third and fifth cardiac cycle after maximum contrast opacification in the right atrium. In transcranial Doppler, the test is confirmed when a hyperintense signal is observed within 10 seconds after infusion of the stirred saline solution8. However, since most patients are asymptomatic, the diagnosis may occur as an occasional finding in tests requested for another purpose[4,5].

In 1877, pathologist Julius Cohnheim hypothesized that the passage of a paradoxical embolism through the PFO could be the cause of a cerebrovascular accident (CVA)[2]. Since then, numerous statistical analyzes have been published demonstrating PFO as an important risk factor for ischemic stroke, especially in patients younger than 55 years of age and with no other apparent cause to trigger the insult9. Although the ischemic cerebrovascular event

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is the predominant clinical manifestation of PE, a fact that can be explained by the predisposition of embolisms to reach the cerebral arteries due to anatomical aspects and the distribution of blood flow in the aortic arch, cases of peripheral arterial occlusions, and less commonly, acute coronary syndrome (ACS) are reported in the literature[5].

The aim of this study is to report a case of acute myocardial infarction (AMI) with ST-segment elevation of the anterior wall due to presumed paradoxical embolism and subsequent PFO occlusion using a percutaneous device.

The Research Ethics Committee of the Hospital de Urgências de Goiânia, linked to Plataforma Brasil, approved the present study (CAAE: 52695421.2.0000.0033).

CASE REPORT

A 48-year-old male, married patient sought care in the cardiology emergency department after waking up at night due to severe tight chest pain accompanied by nausea, vomiting and profuse sweating. In his previous medical history, he reported having systemic arterial hypertension and being treated with olmesartan. He denied other known comorbidities and has no family history of cardiovascular disease.

On physical examination, he was conscious, oriented, cutaneous-mucous pallor, diaphoretic, eupneic, without adventitious sounds on respiratory auscultation and oxygen saturation of 98% on room air. Blood pressure 90/70 mmHg, heart rate 63 beats per minute, regular heart rhythm, two beats, normophonetic sounds, no murmurs. The peripheral pulses of the four limbs were palpable and symmetrical.

The electrocardiogram (ECG) showed ST-segment elevation in leads V2 to V4, establishing the diagnosis of AMI in the anterior wall in the hyperacute phase (Figure 1).

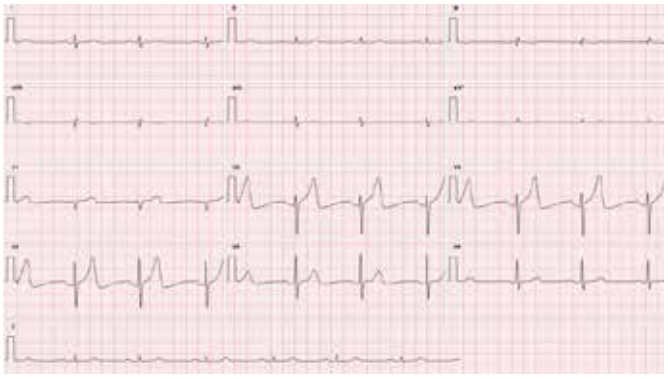

Figure 1. Electrocardiogram image on admission showing ST-segment elevation leads V2 to V4.

In view of the ECG, an urgent coronary angiography was performed, which showed obstruction with high thrombotic load in the proximal third of the anterior descending artery and absence of atherosclerotic lesions (Figure 2 A and B). Intracoronary thrombus aspiration was performed with a catheter associated with an intracoronary glycoprotein IIb/IIIa inhibitor (Tirofiban®) and alteplase (Actilyse®), in addition to heparinization and primary balloon angioplasty. At the end, Thrombolysis in Myocardial Infarction (TIMI) III flow was obtained. Procedure performed without clinical or angiographic complications (Figure 2 C).

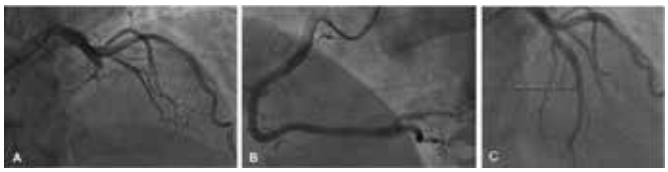

Figure 2. Coronary angiography image. A: total obstruction in the proximal third of the anterior descending artery with no atherosclerotic lesions. B: dominant right coronary artery, with normal angiographic appearance. C: Post-intervention result. Image of residual thrombus is observed, but without flow impairment.

Due to the angiographic appearance with the absence of atherosclerotic plaques, the medical approach consisted of keeping the patient anticoagulated and proceeding with the etiological investigation for possible causes of non-atherothrombotic AMI. The following were performed: lower limb Doppler without evidence of DVT; hematological investigation for thrombophilia without alterations; transesophageal echocardiogram (TEE) with normal ventricular function, contractility and valves, in addition to the absence of thrombi in the left atrial appendage. The infusion test with agitated saline solution was performed, showing the passage of numerous microbubbles from the right atrium to the left atrium during the Valsalva maneuver, compatible with the diagnosis of PFO (Figure 3).

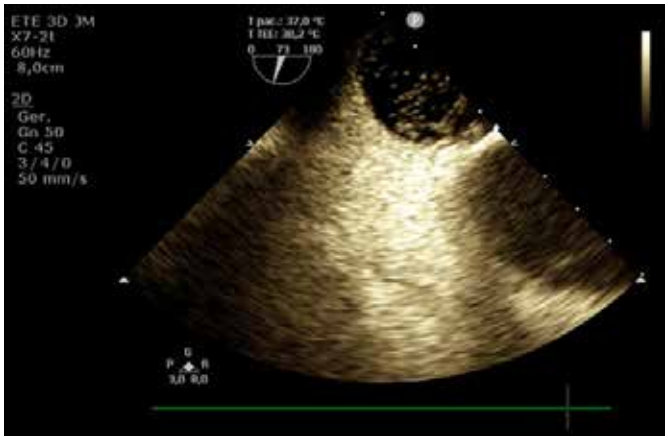

Figure 3. Transesophageal echocardiogram image showing microbubbles crossing from the right atrium to the left atrium through the patent foramen ovale.

There being no other justification for the occurrence of AMI, paradoxical embolism was presumed. Forty-five days

after the infarction and after discussion with the Heart Team, it was decided to perform a new catheterization, which showed a normal angiographic appearance, followed by closing the PFO with an Occlutech® prosthesis, guided by three-dimensional TEE, with an excellent final result (Figure 4 A, B and C).

Figure 4. A and B: Three-dimensional transesophageal echocardiographic image guiding the opening and positioning of the prosthesis. C: Fluoroscopy image demonstrating the prosthesis after its release for PFO closure.

The patient was discharged from the hospital with dual antiplatelet therapy. A control transthoracic echocardiogram was performed one week after the intervention, demonstrating adequate positioning of the prosthesis and absence of residual shunts.

DISCUSSION

In the present case, what stands out is the correlation between AMI in a middle-aged patient with intermediate cardiovascular risk, with PFO and the therapeutic approaches adopted, emphasizing the presumed diagnosis of PE.

It has been recognized since the 1970s that coronary atherosclerosis is the main cause of ACS, however a variety of causes of non-atherosclerotic AMI have been described, including arteritis, Takotsubo cardiomyopathy, aortic stenosis and insufficiency, trauma, spasm, dissection, congenital anomalies and coronary artery embolization10. Paradoxical coronary artery embolism is considered uncommon, representing approximately 10 to 15% of all paradoxical emboli, and in patients younger than 35 years, it corresponds to 25% of acute coronary events[6].

Usually, the clinical and electrocardiographic characteristics are similar to those with classic obstructive atherosclerotic disease10. As a result, PE is, in general, an underdiagnosed cause in individuals with AMI, and should be considered in patients with acute AMI and with a low risk profile for coronary artery disease (CAD)[6]. There are no differences in the approach to AMI by PE in the emergency department. The identification of ST-segment elevation on the ECG associated with the presenting signs and symptoms should trigger a rapid assessment to define the reperfusion strategy10. In this context, the treatment of choice is manual aspiration of the thrombus, followed or not by angioplasty and stent placement associated with antiplatelet agents[6].

The use of a glycoprotein IIb/IIIa inhibitor receives a grade IIb/IIIa recommendation by the American College of Cardiology and American Heart Association (ACC/AHA) guidelines committee for those with ST-segment elevation AMI, especially those with extensive anterior wall and/or large thrombotic burden[11]. On the other hand, the use of thrombolytics via the intracoronary route, in turn, is not routinely recommended due to their potential risk of causing bleeding and the lack of studies demonstrating their effectiveness, and their use is currently restricted to exceptional cases[12]. In the case presented here, in view of the catastrophic situation with high thrombotic load in the proximal third of the left anterior descending artery, we chose to combine drugs with manual thrombectomy, however, we know that the success of the procedure cannot be attributed to such conduct and we recognize that this can be a confounding factor.

The definitive diagnosis of PE is made by autopsy or by direct visualization of the passage of the thrombus through the PFO during echocardiography, being considered a rare diagnosis[13]. However, PE can be considered a presumed cause of infarction when in the presence of a right-to-left shunt and the following conditions are ruled out: coronary artery spasm, atrial fibrillation, vascular and myocardial disease, vasculitis, atherosclerosis and intracavitary thrombus[5]. In our case, it was the most likely cause of AMI, considering the presence of a thrombus in the left anterior descending artery, atherosclerotic plaques not apparent on coronary angiography, tests for hematological disorders without alterations, ultrasound study of normal lower limb veins and exclusion of others. possible causes, in addition to the presence of PFO. We recognize as a limitation of the study the lack of complementary intracoronary imaging to strengthen the diagnostic conclusion of the absence of atherosclerotic plaque, ulcer or endothelial rupture. However, it is known that the panorama of health services in Brazil presents different scenarios and different realities, in addition to regional inequalities with regard to accessibility to more complex services. Thus, a careful analysis of each case individually is necessary, so that there is a balance between the risks and benefits involved in the percutaneous closure of PFO, given its high prevalence in the population.

The indications for secondary prevention in patients with PFO are still the subject of extensive debate. The therapeutic arsenal available includes antiplatelet aggregation, oral anticoagulation and percutaneous or surgical PFO closure. With the advent of the percutaneous approach, the surgical procedure is reserved for selected cases. The results obtained with percutaneous treatment in published series describe results equivalent to those obtained through surgery with the advantage of lower occurrence of complications[14].

There is scientific evidence that percutaneous occlusion in PFO is safe and beneficial in preventing the recurrence of ischemic cerebrovascular events in patients with cryptogenic stroke[3]. In a meta-analysis study published in 2020, involving six randomized trials and 3750 patients, PFO closure demonstrated superiority in reducing the

rate of recurrent stroke over medical therapy alone - risk ratio of PFO closure versus medical therapy of 0.37; 95% confidence interval, 0.17 to 0.78; p=0.01. The occlusion procedure has been implicated in an increased risk of atrial fibrillation[15].

The reference standard for secondary prevention remains controversial and the decision must be individualized and shared, considering age, risk of recurrence of ischemic events, adverse events, long-term clinical consequences and patient preferences[15]. In cases where the outcome was AMI, there are no studies with strong evidence demonstrating that these patients would also benefit from this strategy, however, most authors suggest that the occlusion of the venoarterial communication should follow the same indications[6].

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