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CASE REPORT

Myocardial Infarction Caused by an Enclosed Thrombus in a Patent Foramen Ovale

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Abstract

Paradoxical embolism in coronary artery is a rarely diagnosed clinical entity. In the majority of reported cases; the diagnostic of this pathology is « presumptive » based on certain criteria. It can be considered "proven" when the embolus is found lodged in the abnormal communication between the venous and arterial circulation; which is very rare. We herein report a case of myocardial infarction caused by a proven paradoxical coronary embolism through a patent foramen ovale. The authors highlight through this paper the contribution of echocardiography and particularly transesophageal echocardiography, especially if performed soon after presentation, for early diagnosis.

Keywords: Myocardial infarction with non-obstructive coronary arteries, Paradoxical embolism, Patent foramen ovale

1. Introduction

Patent foramen ovale (PFO) is caused by defective fusion of the septum primum coverage of the fossa ovalis area after birth. PFO is present in approximately 25% of the general population [1]. In most cases it never leads to any health issues, but PFO has been recognized as a possible source of paradoxical embolism since the late 18th century [2]. Paradoxical embolism (PDE) in association with PFO is well documented commonly giving rise to embolic stroke or extremity vaso-occlusion [3]. Paradoxical coronary embolism is a rare phenomenon with a very few cases reported in literature. Almost exclusively the case reports suggest suspected PDE as the reason for acute myocardial infarction (MI) in patients with PFO and normal coronary arteries [2].

We describe a rare case of myocardial infarction (MI) due to PDE confirmed by the presence of a thrombus crossing through a PFO.

1.1. Case report

A 36-year-old man was admitted to the hospital 72 hours after acute chest pain. He has no prior history of angina, and his only cardiovascular risk factor was occasional smoking.

His hemodynamic state was stable with a blood pressure at 110/60 mmHg, heart rate at 70 beats/min, respiratory rate at 20 c/min, and normal physical examination. Electrocardiogram (EKG) showed sinus rhythm with necrosis sequelae in inferior and basal territory, inverted T waves in V4 toV6, and some monomorphic ventricular extrasystole (VES) (Fig. 1). Chest x-ray was normal.

Transthoracic Echocardiography showed dilation of the left ventricle with akinesia of the inferior and posterior wall (especially the basal and middle segment) with basal hypokinesia of both lateral wall and septum. Ejection fraction was 37% and global longitudinal strain of − 12%. Atrial septal aneurysm (ASD) was showed, without evidence of a left-right shunt to the color doppler (Fig. 2) troponin was raised to 40 times normal value.

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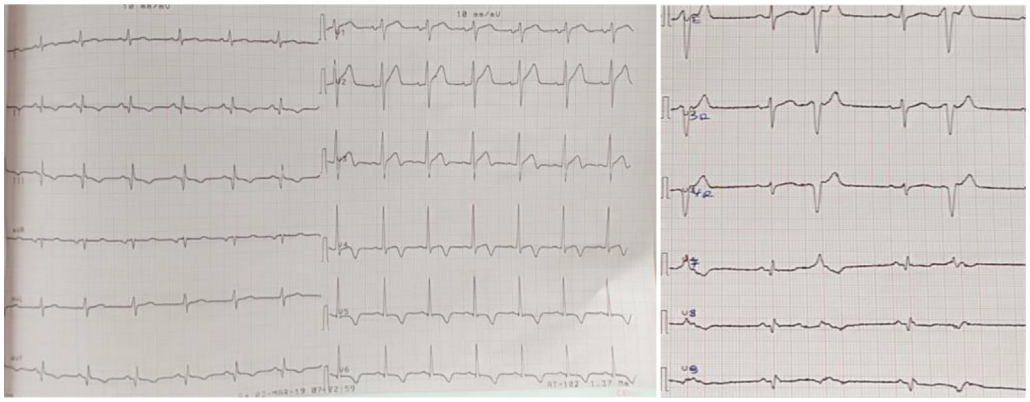

Fig. 1. Electrocardiogram showing extensive posterior repolarization disorders (inferior, basal and lateral territory).

Coronary angiography found normal coronary arteries leading to diagnosis of myocardial infarction with non obstructive coronary (MINOCA).

Evolution was marked by a well tolerated ventricular tachycardia treated by Amiodarone and Bisoprolol.

Cardiac magnetic resonance imaging (MRI) confirmed the diagnosis of non-viable inferior and lateral wall infarction with no reflow, and a small sequela of subendocardial myocardial infarction in the septal and basal wall (Fig. 3).

Following the discovery of the ASD, we realized transesophageal echocardiography (TOE) which demonstrated PFO with an enclosed floating thrombus (Fig. 4). Other biological analyzes were carried out, in particular coagulation studies which revealed increase in anti-hemophilic factor A. Cerebral MRI and venous echodoppler of the lower limbs were normal.

During hospitalization, the patient received, in addition to ischemic heart disease treatment, a continuous anticoagulation with unfractionated heparin, and then he was discharged on oral anticoagulant. Reviewed 2 months later, TOE control showed persistence of thrombus. A surgical treatment was proposed to the patient. The surgery made it possible to extract the thrombus and close the PFO. Postoperative follow-up was uneventful.

2. Discussion

PDE generally refers to a condition in which embolic material from the systemic venous circulation reaches the systemic arterial circulation through communication between the venous and arterial systems [1]. Johnson [4] suggested that PDE should be considered "presumptive" when (1) there is evidence of arterial embolization in the absence of a source in the left heart, (2) there is a source of embolism in the venous system and (3) there is an abnormal communication between the venous and arterial circulation. PDE can be considered "proven" when the embolus is found lodged in the abnormal communication between the venous and arterial circulation [1].

The embolisms have preference to be transmitted primarily into the cerebral arteries and there to the

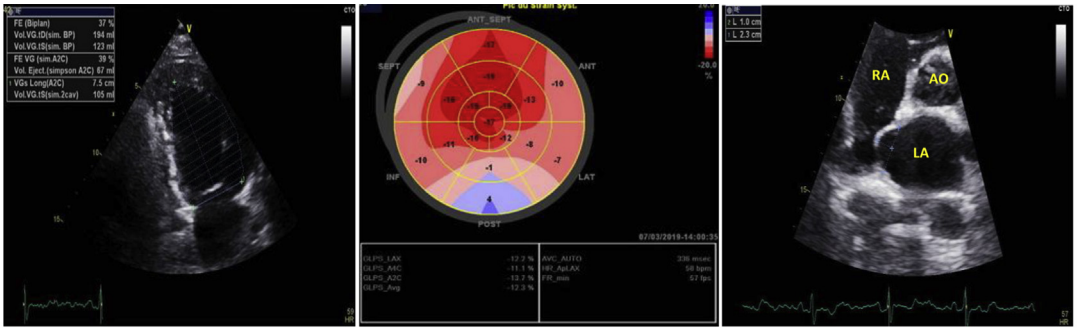

Fig. 2. A: severe left ventricular dysfunction with 37% of ejection fraction. B: Alteration of the left ventricular global longitudinal strain mainly affecting the posterior, inferior and lateral walls. Global longitudinal strain is calculated at −12%. C: Aneurysm of interauricular septum measuring 10 mm × 23 mm. LA = left atrium, RA = right atrium, AO = aorta.

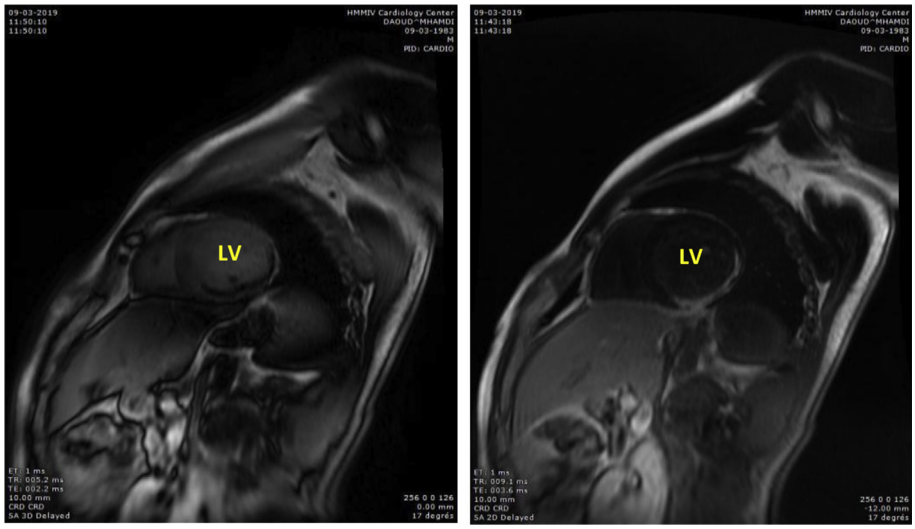

Fig. 3. MRI of non-viable inferior and latéral wall infarction. LV = left ventricle.

posterior vessels [2]. But we also found a subset of patients with MI caused by this pathophysiology, and associations with thrombophilia, pulmonary embolism (PE) and stroke have been reported [5–7]. Most cases described in literature are presumed with no evidence of actual transit of thrombus across a left-right intracardiac shunt. The prevalence of coronary thromboembolism including paradoxical embolism, as a potential cause of MINOCA is thought to be low [8]. In a recent study; among 6502 patients with MI, Kleber Fx et al. found 33 (0.51%) with presumed PDE as the most likely pathophysiology [2]. The diagnosis of PDE requires the demonstration of a PFO. Transthoracic contrast echocardiography (TTEc) is the most effective examination to identify a spontaneous intracardiac right-left shunt with visualization of the left atrium bubble before the third cardiac cycle following opacification of the right atrium. The sensitivity of the TTEc increases with a Valsalva maneuver. PFO associated or not with interauricular septal aneurysm is later identified by TOE [9]. The absence of venous source detected absolutely does not rule out the diagnosis of paradoxical embolism. It is well known that, in a significant percentage of cases, the quest for the venous source remains unsuccessful. The source may not be identified because of size of the thrombus or its unusual localization. In the pelvic veins for example, doppler echo is not enough to exclude thrombi in, so eventually either MRI angiography or flebography should be done to exclude thrombus. Also, thrombus could originate in the PFO tunnel itself due to local stasis in a patient with coagulation abnormalities. Some studies have sought, in a series of patients having presented PDE and in whom a FOP had been detected, a venous source of embolism. On small numbers of patients, these studies are contradictory and it is difficult to draw conclusions [10]. High levels of factor VIIIc (anti-hemophilic factor A) is associated with an increased risk of venous thrombosis (relative risk ranging from 2.2 to 10.3 [95% CI]) [11]. But the definition of normality threshold is difficult for this factor. This is a likely constitutional anomaly whose prevalence in the general population ranges from 6 to 36%. Having regard to its exceptional nature, to date, there are no recommendations concerning the management of MI by paradoxical coronary embolism. According to European and American guidelines; based on studies that have been concerned with ischemic stroke; the closure of a PFO after a first transient ischemic attack or stroke is not recommended or recommended with a low

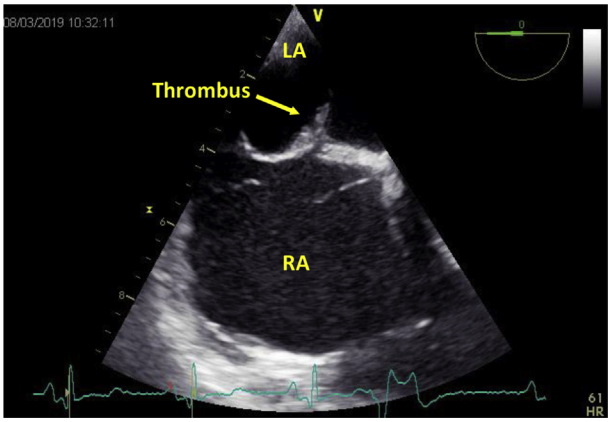

Fig. 4. TOE revealing a mobile thrombus inserted in the PFO. LA = left atrium, RA = right atrium.

level of evidence [9]. However, the results of the CLOSE and REDUCE studies [12, 13] will soon modify the therapeutic algorithm of cryptogenic stroke in young subjects with a recognized place for interventional treatment. Kleber Fx and al offered interventional closure of the interatrial communication to all patients with MI if a PFO, PFO-like atrial septal defect (ASD), or an ASD with or without ASA was confirmed by TEE and no other cause for systemic thromboembolism and no local arterial disease were found [2]. Dao and Tobis reported 1.9% myocardial infarction among their indications for PFO occlusion [14]. A percentage somewhat lower than found by Kleber Fx and al (5.4% in the 1st and 17.7% in the 2nd series) [2].

3. Conclusion

The entity of PDE through PFO remained a diagnostic challenge. The no-guidelines land about patients with non-cerebral paradoxical embolization, requires an individualized approach and justifies PFO closure after careful consideration.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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