Case report

A rare ischemic complication: tricuspid pillar rupture after anterior myocardial infarction

ABSTRACT:

The rupture of the papillary muscle (PRM) after a myocardial infarction (MI) became rare since the introduction of primary angioplasty. In most cases in literature, it's reported in the mitral valve. Followed by a PMR of the tricuspid valve (TV), which is extremely rare. We described a case of a 78-year-old patient, who was admitted to the emergency room for a delayed anterior ST elevation myocardial infarction. It was later complicated by papillary muscle rupture after a percutaneous coronary intervention (PCI) of the right coronary artery involving a stent. The patient underwent surgical repair of the TV with fatal evolution.

Keywords*: acute myocardial infarction, ischemic complication, papillary muscle rupture, Tricuspid valve*

INTRODUCTION

Myocardial infarction (MI) remains the leading cause of death worldwide [1]. Its associated Mechanical complications have significant in-hospital morbidity and mortality. They can lead to rapid clinical deterioration and fatal outcome, with patient survival dependent on prompt recognition and intervention. Since the introduction of primary angioplasty, and with recent improvements in both the medical and percutaneous management of patients, ruptured papillary muscles (RPM) have become rare [2]. This entity is often neglected in the right side, as the rupture of an infarcted right papillary muscle is exceptional. We describe a case of a rare mechanical complication: a partial rupture of the tricuspid pillar after a MI.

CLINICAL CASE PRESENTATION

We report the case of a 78-year-old man, with a history of hypertension for 2 years, well balanced on Angiotensin II receptor blockers (ARBs) without any history of smoking or dyslipidemia, who presented to the emergency room for retrosternal chest pain evolving for 3 days before admission, without improvement under symptomatic treatment, associated with NYHA stage III dyspnea without orthopnea. On clinical examination, the patient was hemodynamically stable without any signs of right or left heart failure. The ECG showed a third-degree AV block, a complete Right Bundle Branch Block (RBBB) associated with an Anterior ST-Segment Elevation (Figure 1). Transthoracic echocardiography showed hypokinesia of the entire anterior wall, the septal walls, and the RV free wall as well as akinesis of the left ventricular apex with severe Left Ventricular dysfunction, LVEF at 28% calculated in SBP. The RV was dilated at 46 mm, systolic dysfunction S' at 9 cm/s and Tricuspid annular plane systolic excursion (TAPSE) at 16 mm. The right atrium was dilated without dilation of the pulmonary artery. There was no insufficiency finding in either the TV or the mitral valve and no

signs of elevated LV filling pressures or pulmonary hypertension. Ultrasensitive troponins were at 4000ng/ml for a normal value below 38 ng/ml. The result of the other biological parameters was normal.

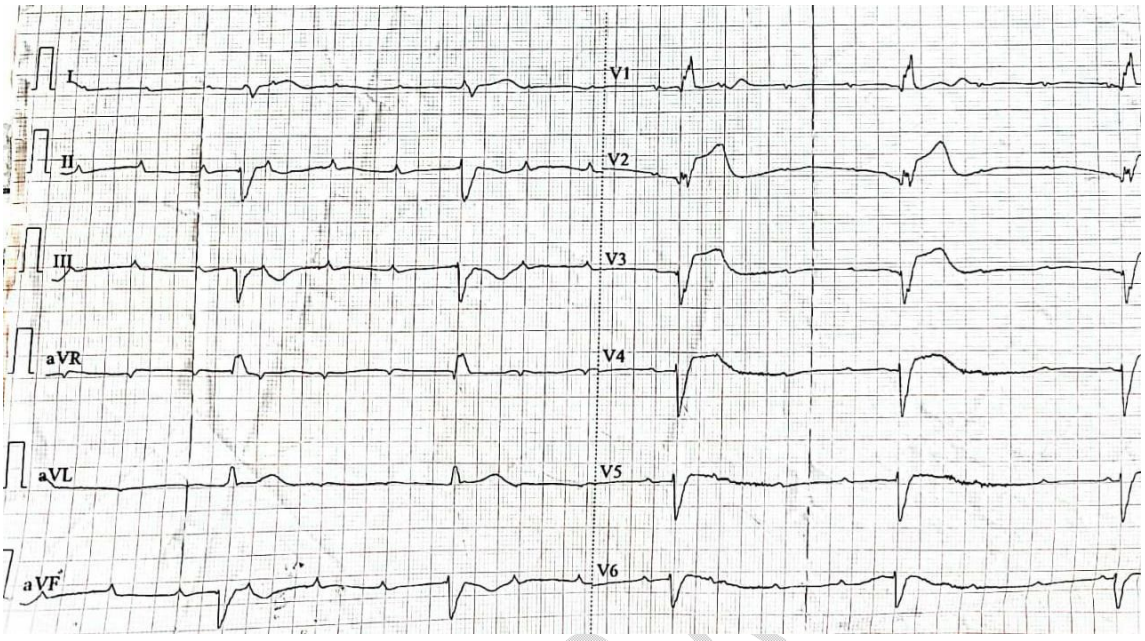

Figure 1: ECG on admission shows third-degree AV block, complete Right Bundle Branch Block (RBBB) associated with an Anterior ST-Segment Elevation

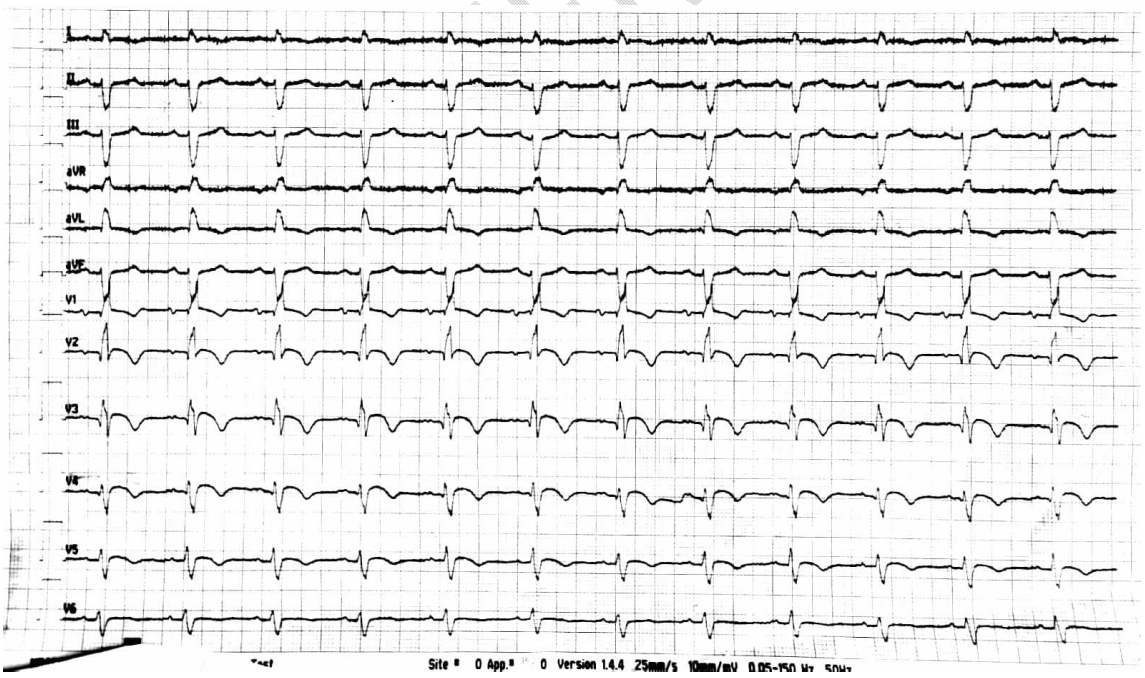

Figure 2: ECG post angioplasty shows sins rhythm and regression of ST-elevation with negative waves in anterior

Coronary angiography was performed, visualizing an acute thrombotic occlusion of the LAD with TIMI 0 flow, dilated urgently by an active stent. Calcified sub-occluded stenosis of the mid-right coronary artery with TIMI 1 flow stented during the same procedure given the context of the right ventricular dysfunction and ischemic AV block (Figure3). An intermediate circumflex artery stenosis and a

significant stenosis of the 3rd marginal artery were found and were medically treated. The results of angioplasty were as follows: primary success of angioplasty of medium IVA after an aspiration thrombectomy with the placement of an active stent under Tirofiban + primary success of DC angioplasty with a placement of an active stent under Tirofiban. The patient is then put on an anti-ischemic treatment + Dual Antiplatelet Therapy (DAPT).

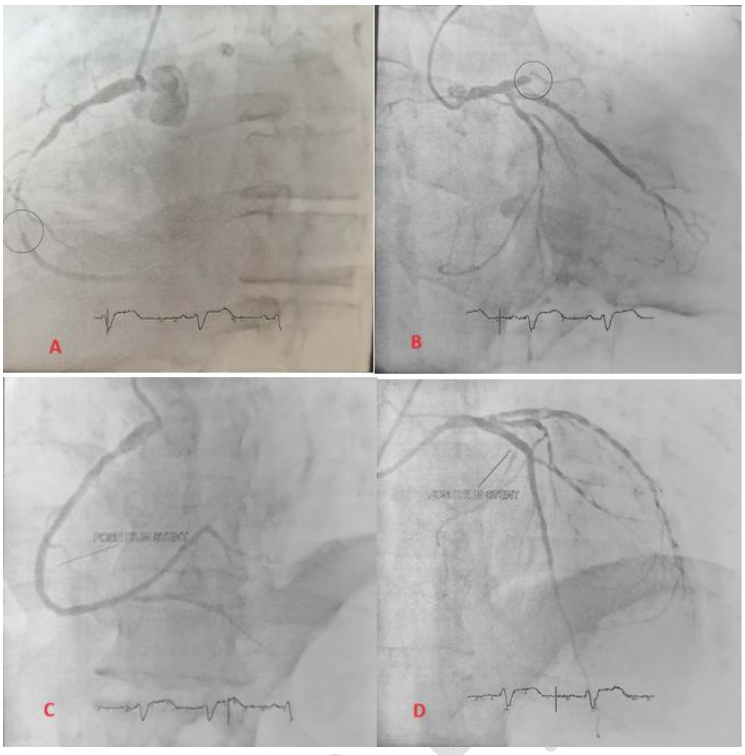

Figure 3: (A) Coronary angiography showing a suboccluded stenosis of the middle right coronary artery, (B) thrombotic occlusion of medium IVA with TIMI 0 flow and results after implantation of stent in (C) RCA and (D) IVA.

Five days later, the patient presented a dyspnea on exertion and fatigue. A Transthoracic echocardiography revealed a hypermobile homogeneous Hyperechogenic mass on the ventricular side of the anterior leaflet with prolapse movement inside the RA causing a tricuspid insufficiency which is difficult to quantify(Figure 4). The diagnosis of infective endocarditis was ruled out by the DUKE criteria: the negativity of the biological assessments and the clinical evolution. Thus, an anticoagulant was started to exclude probability of thrombus, but no reduction in the size of the mass was noted. So, we suspected an ischemic tricuspid papillary muscle rupture or a valvular tumor. Based on these findings, the decision of a surgical repair of the TV was taken after a medical and surgical staff meeting to decide, and the results were in favor of a surgical treatment, given the high risk of complications. The patient underwent emergency surgery. Surgical exploration revealed a rupture at the base of the anterior pillar of the tricuspid valve, along with annular dilatation (Figure 4).

The surgical repair involved reinserting the pillar at the level of the ansiform band using 4/0 prolene suture. Then, a rigid contour 3D ring No. 34 was inserted using 2/0 cardioxyl suture. Water testing revealed moderate leakage. It was decided to perform an EDGE-TO-EDGE repair according to the Alfieri technique with good sealing.The patient presented a hemodynamic shock postoperatively, and

was put on mechanical ventilation with positive inotropic supports without improvement, leading to death after two days post-op.

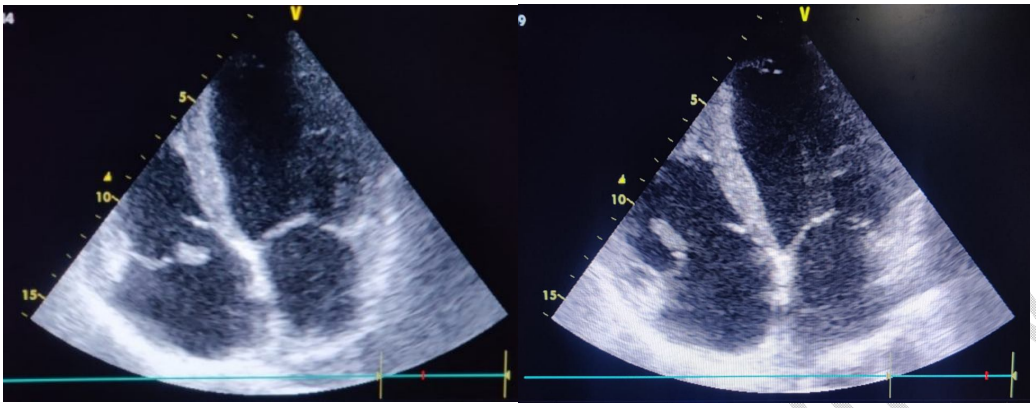

Figure 5: Transthoracic echocardiography revealing a hyperechoic hypermobile mass on the ventricular side of the anterior

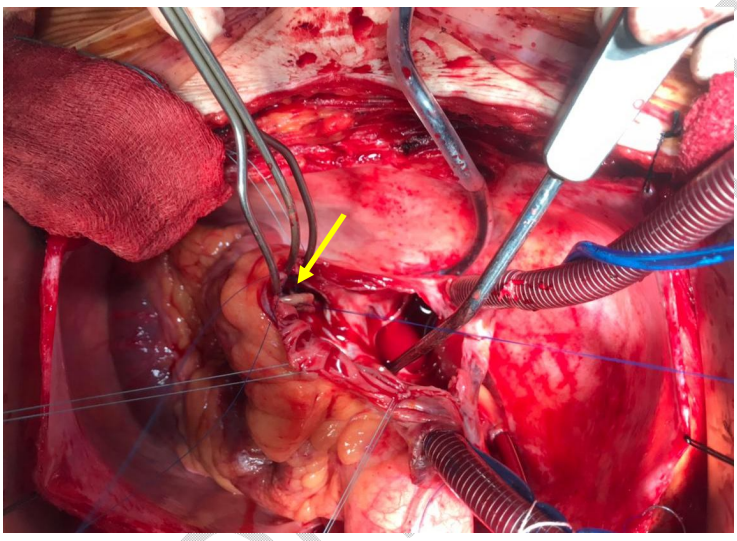

Figure 6: Intraoperative image showing papillary muscle rupture of the tricuspid anterior leaflet.

DISCUSSION

The rupture of the papillary muscle is a rare complication after a myocardial infarction, it is estimated to occur in 1% to 5% of patients with acute myocardial infarction. This is thought to be due to fast diagnosis and early revascularization techniques via percutaneous coronary interventions to limit ischemia. When rupture does occur, there is a high mortality rate without surgical intervention, estimated to be as high as 50% within 24 hours with complete rupture. It used to go up until 90% within the first 24 hours, prior to the initiation of cardiac surgical procedures to repair injuries [3] [4]. Almost all reported cases arise in the papillary muscle of the mitral valve. There are very few studies on a tricuspid papillary muscle rupture; the tricuspid valve was once referred to as the forgotten valve. Development of tricuspid regurgitation after right ventricular myocardial infarction due to a rupture of the papillary muscle is extremely rare.

First, the tricuspid valve is the largest and most apically positioned of the four cardiac valves [5]. Papillary muscles are muscular extensions that are intricately involved in the mechanical activity of the valve. The tricuspid muscle set is constituted of anterior, posterior, and septal papillary muscles attached to the anterolateral ventricular wall, the inferior portion of the septum and the infundibular septal wall, respectively [6]. With the anterior usually being the most mobile and the longest, and the septal being the least mobile and the shortest. These muscles attach to the tricuspid valve leaflets via the chordae tendineae and functionally prevent regurgitation of ventricular blood via tensile strength by preventing prolapse or inversion of the valves during systole. The main blood supply to these muscles comes from the right coronary artery (RCA) that branches into the posterior descending artery and the septal branches, feeding into the interventricular septum [6] [7]. In the setting of acute of the right coronary artery leading to a myocardial infarction and RV involvement, TV papillary muscle rupture may occur, as described in the current case. Aside from ischemia, a rare causal factor, more common causes of tricuspid papillary muscle rupture include endocarditis, myxomatous degeneration, external cardiac massage, and both penetrating and nonpenetrating trauma [8] [9]. Because of the anterior position of the right ventricle in relation to the chest wall, nonpenetrating blunt trauma incidents, such as high-velocity motor vehicle accidents, are prevalent sources of both tricuspid ruptured papillary muscles and torn chordae tendineae. [10].

On the other side, we can summarize the cause of the rarity of an infarcted tricuspid papillary muscle in several anatomic, epidemiologic, and physiologic factors; The low incidence of an isolated right ventricular infarction being one of them. The tricuspid valve (TV) and the papillary muscles (PM) are resistant to ischemia because the systolic pressure of the right ventricle (RV) is lower than that of the LV. An additional protective factor lies in the anatomic arrangement of the tricuspid papillary muscles. The large anterior and small conus papillary muscles of the tricuspid valve are relatively constant in position, but the posterior muscle, which arises from the diaphragmatic wall of the right ventricle, is represented by a group of muscles that are inconstant in number and position. The anterior PM are attached to the moderator band and supplied by the RV branches of the LAD [11]. Furthermore, each of the papillary muscles attaches to two adjacent leaflets of the tricuspid valve, and, even should rupture occur, the resultant tricuspid insufficiency might not be clinically significant unless dilatation of the annulus supervened. The clinical signs of a ruptured mitral papillary muscle, with the pulmonary capillary bed behind the insufficient valve, are likely to be much more dramatic [12].

PMR occurs most frequently within 2–7 days after MI. Our patient experienced the MI for 7 days prior to PMR [3]. The diagnosis clinically can be difficult at times because, often, there is not an audible regurgitant murmur. This is due to either the equalization of pressures between the atria and the ventricle or an eccentric regurgitation. Patients who do have murmurs can have mid, late, or holosystolic murmurs. Transthoracic echocardiography may demonstrate a flail mitral valve leaflet with a prolapse into the atrium during systole, a visualization of a ruptured papillary muscle head with erratic movements in the ventricle, or a mobile mass attached to the chordae tendineae. The sensitivity of TTE to visualize the structural abnormalities has been reported to be between 65-85%. It is recommended to utilize a transesophageal echocardiography with unequivocal TTE results, as TEE sensitivity has been reported to be as high as 92-100%. Many of these patients are too hemodynamically unstable to undergo invasive procedures, and thus TTE is typically the initial diagnostic method used. [13] [14].

After good research of the literature, we found 5 clinical cases of Tricuspid PMR after a myocardial infarction in the world. The first case of a rupture of the tricuspid pillar secondary to myocardial infarction was reported in 1964. All previous published cases were non-ischemic and were reported

on four occasions in young people between the ages of 20 to 30 years, and were either the result of a bacterial endocarditis (3 cases) or trauma (1 case) [5] [6]. This first case was about A 72-year-old woman, who was admitted to the hospital for a congestive heart failure with pulmonary oedema and a history of angina pectoris; The ECG revealed QS in inferior leads and elevated ST segments in anteroseptal leads complicated further by a third-degree heart block. The patient deteriorated hemodynamically and neurologically and she died on Day 20 of her admission. The diagnosis of a ruptured tricuspid septal papillary muscle following acute myocardial infarction was retained on the post-mortem pathologic examination [2]. Then, in 2009, Jonathan R et all reported the case of a 85 old year patient with intractable hypoxia in the setting of an acute inferior STEMI with posterior and RV involvement complicated by the rupture of the posterior tricuspid valve (TV) papillary muscle with right-to-left shunting across a patent foramen ovale (PFO). The culprit lesion was the occlusion of the proximal right coronary artery, which was treated with a successful angioplasty. Moreover, a percutaneous closure of the PFO has been done because of the ongoing hypoxemia [15].

The occurrence of a rupture of the pillar can happen at a distance from the infarction and even after revascularization. This is the case of a 70-year-old patient, published in 2018, who presented with a massive tricuspid regurgitation caused by a papillary muscle rupture, three months after a percutaneous coronary intervention to the right coronary artery involving a stent, knowing that the TTE control after 15 days of revascularization was without abnormalities [16].

Despite progress in the management of infarction, the prognosis remains poor even in the event of a percutaneous or a surgical treatment, especially if associated with other mechanical complications such as septal rupture. Given that VSP increases RV systolic pressure, the TVPM is susceptible to ischemia. This was the case for 2 reported patients, who benefited from the treatment of the culprit ischemic lesions with a surgical repair of a ruptured tricuspid papillary muscle as well as a ruptured ventricular septum. The patients died of multiorgan failure [17] [18]. And like with our patient, the prognosis remains fatal, especially in old patients with heart failure and probably chronic myocardial ischemia. Thus, prevention of cardiovascular disease is necessary and there should be a good clinical evaluation with early and well-thought-out management.

CONCLUSION

To the best of our knowledge, this is the first case of a rupture of a tricuspid papillary muscle due to anterior myocardial infarction to be reported in the Moroccan and the African literature, and apparently the second in the worldwide literature. The tricuspid valve was once deemed the forgotten valve. However, clinicians should remain aware and be able to diagnose this potential complication in not only the mitral valve but also the right side.

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