Anterolateral Papillary Muscle Rupture Caused by Myocardial Infarction

Faisal Dalak, Abulaziz Albardie, Fawaz Almutairi, Fahad AlGofaili

KSHC, King Fahad Medical City, Riyadh, Saudi Arabia.

Abbreviations: **MI:** Myocardial Infraction; **CK:** Creatinine kinase; **LVEF:** Left Ventricular Ejection Fraction; **MR:** Mitral Regurgitation; ERO: Effective Regurgitatant Volume; PISA: Proximal Isovelocity Surface Area: OM1: Obtuse Marginal Artery 1; **OM2:** Obtuse Marginal Artery 2; **NYHA:** New York Heart Association. **PCI:** percutaneous coronary intervention.

Key words: Papillary Muscle Rupture: Myocardial Infarction

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Rupture of a papillary muscle is an uncommon but often fatal complication of acute myocardial infarction (MI) which is responsible for approximately 5% of death after MI (1,2). The characteristics of the underlying coronary disease will define the clinical presentation and prognosis; the mortality could be as high as 80% during the first week of post MI. The rupture of the posteromedial papillary muscle is most common, seen in about 75% of cases. The posteromedial muscle has a single blood supply from the posterior descending branch of a dominant right coronary artery, and is associated with inferior wall infarctions. The rupture of the anterolateral muscle is less common, occurring in 25% of cases, as it has dual blood supplies: from the first obtuse marginal, originating from the left circumflex; and from the first diagonal branch, originating from the left anterior descending. The rupture of the latter is seen with anterior or postero-lateral MI[3,4].

A 62-year-old Sudanese male presented to the emergency room with chest pain for 6 hours. He had mild concomitant shortness of breath. There was no history of orthopnea or paroxysmal nocturnal dyspnoea. Past medical history was remarkable for hypertension, non-compliant with medications and he was an active smoker. There was no family history of coronary artery disease. He had similar chest pain 5 days prior to presentation but he did not seek medical advice due to eligibility issues. On physical examination the patient was conscious and oriented, with a blood pressure of 150/80 mmHg, heart rate 100 bpm and regular, and respiratory rate 20/min. Neck veins were not distended and he had no ankle edema. On examination of the cardiovascular system he had a regular S1 and S2. No murmur was heard. He had normal vesicular breathing in both lungs. Initial EKG showed ST elevation and the inferior leads (II, III and aVF) with ST depression at V2, V3 and V4 (fig 1). The chest radiography showed normal size of the heart with normal lung parenchyma.

Patient was taken to the cath lab within 40 minutes at acute STEMI. Coronary angiogram revealed: Dominant right coronary artery which was totally occluded at the mid segment. Left circumflex artery with 60 % disease at the proximal segment, with small caliber first obtuse marginal (OM1) and second obtuse marginal (OM2), both sub-totally occluded at the proximal end. Underwent primary PCI (percutaneous intervention) to the right coronary artery (fig 2).

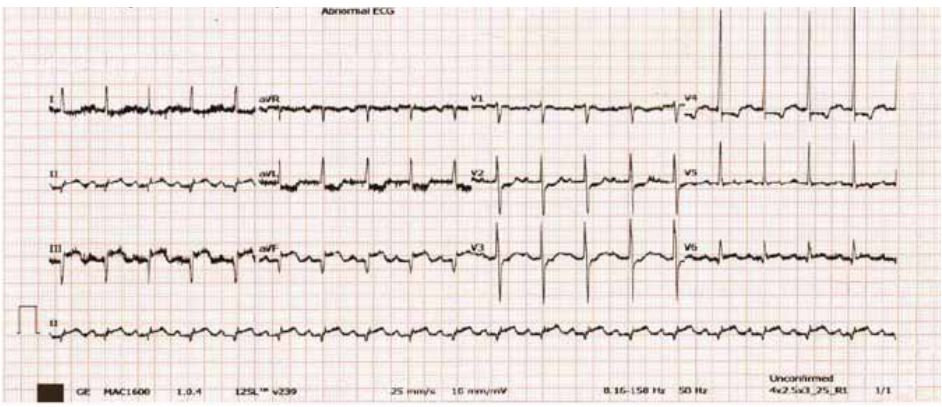

Figure 1. EKG showing normal sinus rhythm, HR 105/min, left axis deviation and ST-elevation at the inferior leads, ST depression in the v2/v3 with upright T wave (Infero-posterior myocardial infarction)

The patient was admitted to the coronary care unit with the diagnosis of ST elevation myocardial infarction post successful primary PCI to the RCA. Subsequently lab showed (CK 213), TroponinT (1265 ng/L).

Suddenly, one hour after coronary intervention, the patient had significant shortness of breath (Killip class IV), with no significant chest pain. Examination showed a significantly distressed patient with diffuse course crepitations in both lungs. Cardiovascular examination showed blood pressure of 77/40 mmHg and grade II systolic murmur at apex of the heart.

Transthoracic echocardiogram

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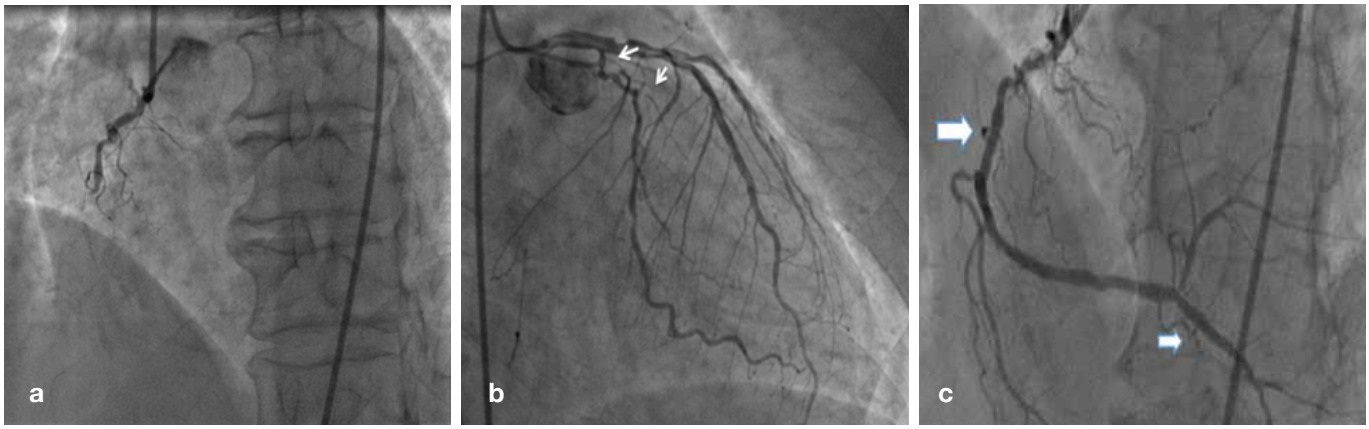

Figure 2 a: Totally occluded RCA at the mid semgent. **b:** Small calibre OM1 and OM2 with significant disease. **c:** Final result after 2 stents in the RCA: one at the mid segment and another one at the PDA, with good final result

was performed, revealing a left ventricular ejection fraction (LVEF)= 40%, with akinesis at the inferior and Inferolateral walls of the left ventricle. There is severe mitral regurgitation (MR) with possible ruptured anterolateral papillary muscle (fig 3a). A transesophageal echocardiogram was performed after that, which showed flail A2 and A3 scallops of the anterior mitral valve leaflet seen in 3 D TEE (fig 3d). Color flow Doppler revealed severe MR. Effective Regurgitant Orifice (ERO) was 5 mm2, regurgitant volume by PISA was 60 ml, regurgitation fraction was 60%, and the Vena Contracta width was 8 mm. There was a systolic flow reversal of the pulmonary vein. Complete ruptured anterolateral papillary muscle was seen in the trans gastric view (fig 3b).

The patient underwent a mitral valve replacement with a Bio-prosthetic Mitral valve and concomitant coronary artery bypass grafting of the Right coronary artery, left circumflex artery. He had an uneventful recovery. We presented the case of a patient admitted status post-acute myocardial infarction secondary to the occlusion of the right coronary artery with first obtuse

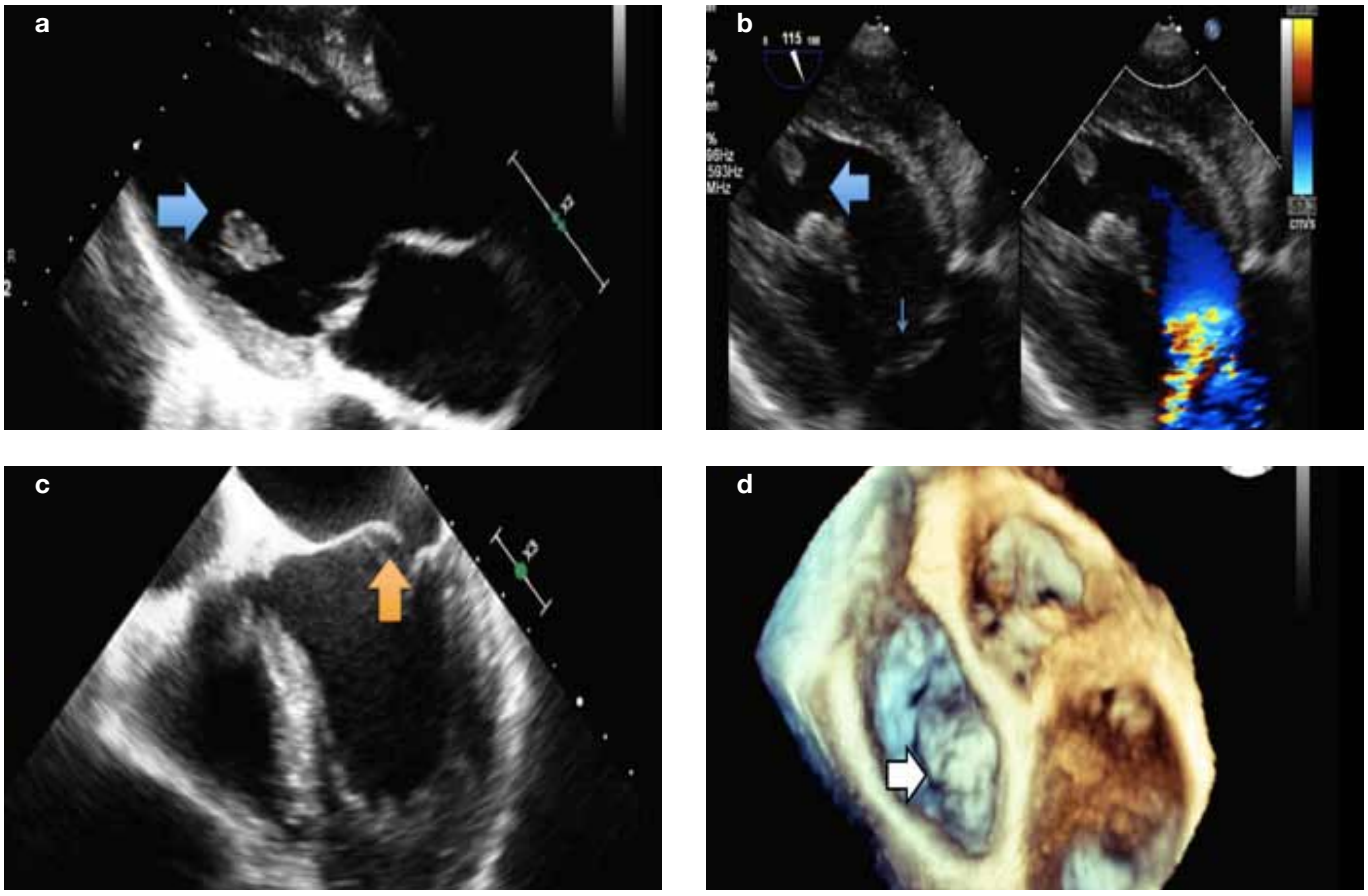

Figure 3 a: Mass in the LV likely ruptured papillary muscle. **b:** Trans-gastric view of TEE, shows complete rupture of anterolateral papillary muscle with significant eccentric mitral regurgitation. **c:** TEE shows flail anterior mitral valve leaflet. **d:** 3D TEE, looking to the mitral valve from the left atrium, with flail A2 and A3 scallops of the anterior mitral valve leaflet.

* Corresponding author. E-mail: fdalak@kfmc.med.sa

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Figure 4: Specimen of the resected anterolateral papillary muscle.

marginals OM1 and OM2, with consequent Mitral regurgitation as a mechanical complication of the myocardial infarction and congestive heart failure. When the transesophageal echocardiogram was performed the MR was found to be secondary to the complete rupture of the anterolateral papillary muscle, which could not be accurately appreciated in transthoracic echocardiogram.

The clinical presentation and severity of a papillary muscle rupture depends on the involved coronary artery and left ventricular performance[5]. This is usually clinically apparent 3-5 days post-acute MI, compatible with the presentation of our patient, as he had chest pain 5 days prior to presentation and did not seek the medical advice since he was not eligible for treatment. As stated previously, the anterolateral papillary muscle is less often involved in a rupture than the posterior papillary muscle, because of its dual blood supply.

Different types of lesions to the papillary muscle may occur as a complication of ischemia; prolapse, elongation or rupture in different degrees, complete rupture being the most common type of rupture. The precise diagnosis of papillary muscle rupture can be difficult to establish by transthoracic echocardiography, as the ruptured head may not prolapse into the left atrium, making transesophageal echocardiography a more sensitive and useful tool for diagnosis[6].

Due to the high mortality rates with the medical management of papillary muscle rupture urgent surgical intervention may be required, the timing of intervention being dictated by the patient's hemodynamic stability[7,8]. The survival rates seem to be related to the extent of papillary muscle rupture, with the best results occurring when a small portion of the tip is ruptured, related to small infarction and limited coronary disease[9-13].

This case confirms the importance of an immediate echocardiographic evaluation in establishing the diagnosis, whenever an acute mechanical complication from an acute MI is suspected. The definitive therapy is surgical valve repair or most often, replacement, which should be undertaken as soon as possible because clinical deterioration in these patients can be sudden[14].

Statement of ethical publishing

The authors state that they abide by the statement of ethical publishing of the International Cardiovascular Forum Journal[15].

Authors' contributions

Faisal Dalak made the diagnosis using transthoracic and transesophageal echocardiography and was responsible for writing the paper and looking up the back ground references. Abdulaziz Albaradie, was the surgeon taking care of the patient. Fawaz Almutairi was the interventionist made the coronary angiography and intervention. Fahad AlGofaili, was a senior consultant cardiac surgeon shared in the management of the patient.

Conflicts of Interest

None declared.

Address for correspondence:

Faisal Ali Dalak
E-mail: fdalak@kfmc.med.sa

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