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Case Report

Apparently Spontaneous Partial Rupture of Anterolateral Papillary Muscle Requiring Urgent Surgery

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ABSTRACT

Papillary muscle rupture is a rare complication of myocardial infarction. Here we describe a case of an apparently spontaneous rupture of papillary muscle, not associated with coronary obstruction. The patient was a 73-year-old man admitted to our hospital for diabetic ketoacidosis complicated by acute pulmonary edema and cardiogenic shock. Transthoracic and transesophageal echocardiography showed partial rupture of papillary muscle leading to severe mitral regurgitation. Urgent cardiac surgery with mitral valve replacement was performed.

Case Report

We describe the case of a 73-year-old man, with medical history of paroxysmal atrial fibrillation, hypertension, chronic renal failure (stage IV), type 2 diabetes, and peripheral artery disease, admitted to our hospital for acute respiratory failure with concomitant diabetic ketoacidosis and acute renal failure. His blood pressure was 110/60 mmHg. On physical cardio-thoracic examination no cardiac murmurs were present, and the vesicular murmur was preserved. The ECG showed sinus tachycardia and it was otherwise normal. Cardiac troponin was mild elevated (230 pg/ml). Blood exams revealed glucose blood level of 700 mg/dl, lactate of 4,3 mmol/L and severe metabolic acidosis (pH = 6,8). Chest computed tomography (CT) showed multiple and bilateral ground glass opacities more accentuated in lower lobes and bilateral pleural effusion. In suspicion of possible sepsis, the patients were admitted to department of medicine and adequate medical therapy was initiated. Due to a rapid deterioration of general clinical condition with hypotension and pulmonary edema, the patient was transferred to the intensive care unit.

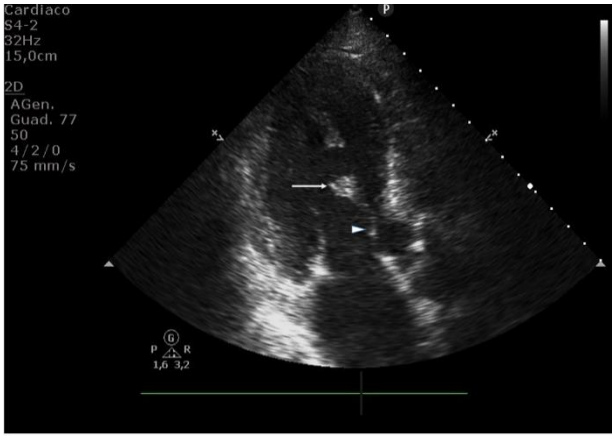

Figure 1: Transthoracic echocardiogram 3 chamber view. Partial rupture of the head of anterolateral papillary muscle (arrow) attached to anterior leaflet (arrowhead).

On physical examination an apical holosystolic murmur appeared. Inotropic support, intravenous diuretic therapy and noninvasive ventilation was started. Transthoracic echocardiogram showed mild left

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ventricular hypertrophy without regional wall motion abnormalities. Ejection fraction was 59 %. Severe mitral regurgitation was present, with eccentric regurgitant jet directed posteriorly. A mobile rounded mass, attached to anterior mitral leaflet, compatible with partial anterolateral papillary muscle (PM) rupture, was seen (arrows, Figures 1 & 2). To confirm the diagnosis transesophageal echocardiogram (TOE) was performed. Two dimensional and three-dimensional TOE showed eversion of anterior mitral valve leaflet secondary to partial rupture of the head of anterolateral PM (arrows, Figures 3, 4 & 5). The head of the PM was still attached to the papillary muscle body through a thin fibromuscular structure.

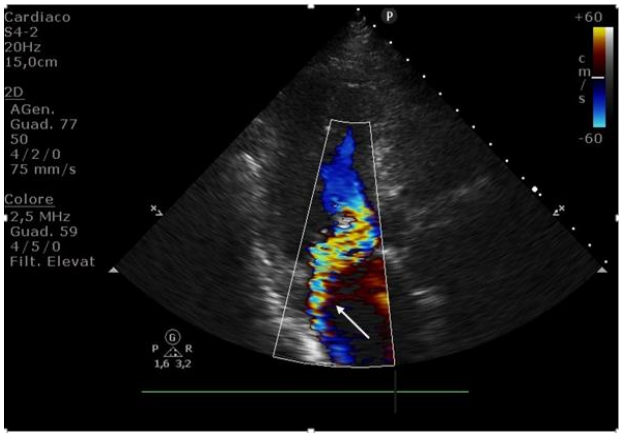

Figure 2: Transthoracic echocardiogram 3 chamber view. The image shows severe mitral regurgitation with eccentric jet (arrow) at color-Doppler.

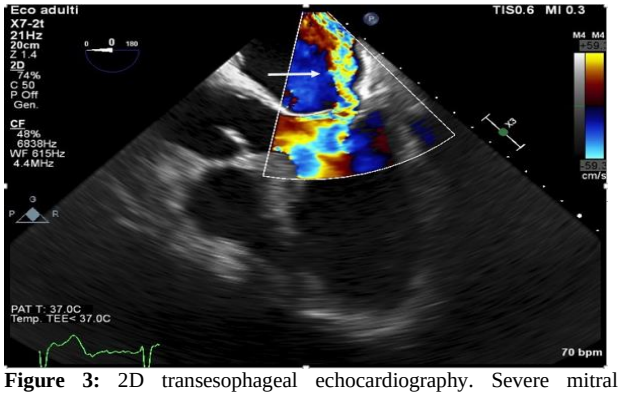

Figure 3: 2D transesophageal echocardiography. Severe mitral regurgitation with eccentric jet (arrow) at color-Doppler.

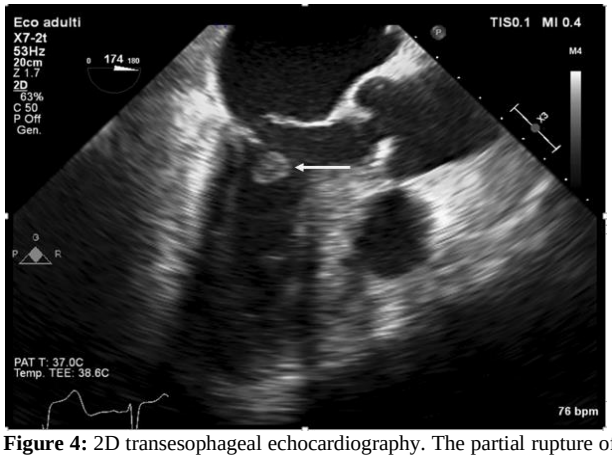

Figure 4: 2D transesophageal echocardiography. The partial rupture of the head of anterolateral papillary muscle (arrow) was confirmed.

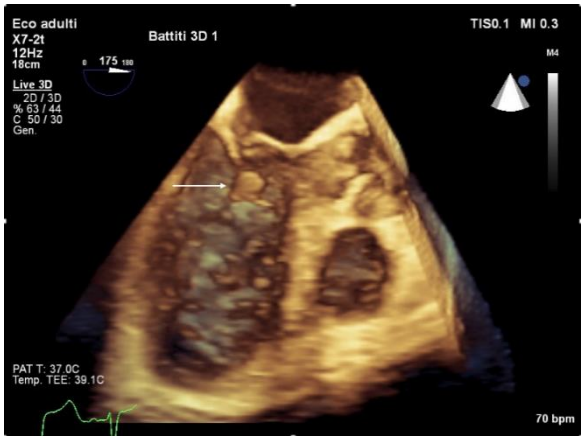

Figure 5: 3D transesophageal echocardiography. The head of anterolateral papillary muscle (arrow) is still attached to the body of the papillary muscle itself.

Urgent coronary angiography showed separate aortic ostium of left anterior descending and left circumflex coronary arteries, in absence of significant coronary artery disease (Figures 6 & 7). Patient underwent urgent cardiac surgery and mitral valve replacement with biological valve prosthesis was performed. Pathology confirmed partial rupture of anterolateral PM (Figure 8) and histological examination revealed myocardial necrosis of the head of the PM. Postoperative transthoracic echocardiogram showed normal function of the mitral valve bioprosthesis, normal ejection fraction and the absence of wall motion abnormalities. Patient was discharged several days after intervention with adequate hemodynamic status and good clinical conditions.

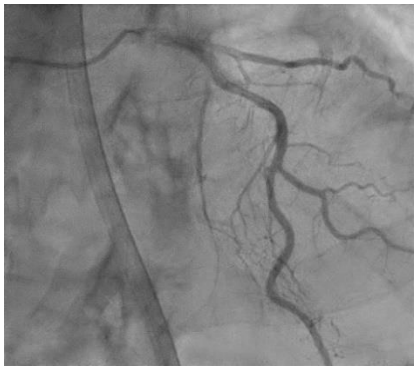

Figure 6: Invasive coronary angiography: non-significant coronary disease of anterior descending artery.

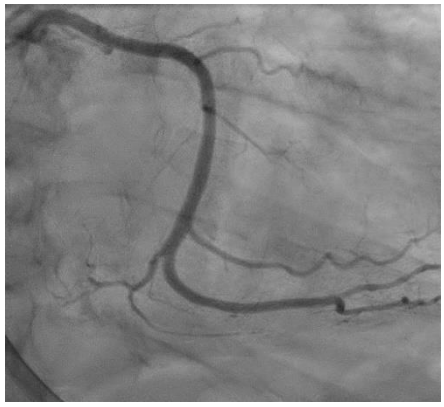

Figure 7: Invasive coronary angiography: normal aspect of circumflex artery.

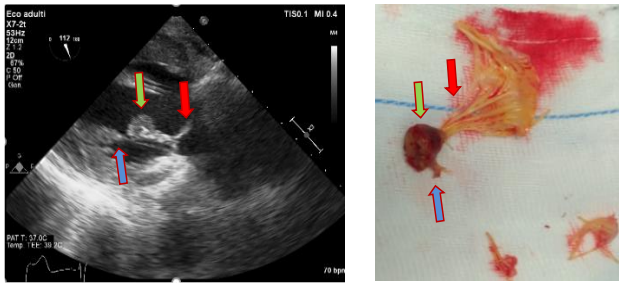

Figure 8: Comparison between echocardiographic and surgical findings. On the left transesophageal echocardiography, on the right the head of anterior papillary muscle removed during surgery. Red arrow: anterior mitral valve leaflet; Green arrow: head of anterior papillary muscle; Blue arrow: residual thin fibromuscular structure connecting the head to the body of papillary muscle.

Conclusion

PM rupture is a rare complication of acute myocardial infarction (AMI) with an incidence of 0.05% [1]. The most common cause of PM rupture is myocardial infarction due to atherothrombotic coronary artery disease (more often ST-segment elevation myocardial infarction, less frequently non-ST segment elevation myocardial infarction), other rare etiologies include: myocardial infarction with non-obstructive coronary arteries (MINOCA), trauma, syphilis, periarteritis nodosa, vegetating valvulitis, myocardial abscess, iatrogenic, and cocaine use [2, 3]. Mortality can reach the 50 % in first 24 hours and the 80 % in the first week if surgically untreated [4]. The typical clinical scenario caused by PM rupture is represented by severe mitral regurgitation, cardiogenic shock, and pulmonary edema, occurring 2-7 days after AMI [5]. Intravenous diuretic, vasodilators, inotropic agents, intra-aortic balloon pump (IABP) and ventilatory support, in preparation for angiography and urgent surgery, are the cornerstone of treatment.

In our case, the absence of obstructive coronary artery disease on invasive coronary angiography in association with transient elevation of cardiac troponin (cTn) levels (>95%) and the histological finding of myocardial necrosis, lead us to hypothesize that this rupture was related to a MINOCA secondary to paroxysmal atrial fibrillation, or acute metabolic distress due to ketoacidosis.

The treatment of PM rupture consists in different interventional approach: mitral valve replacement, surgical mitral valve repair, or transcatheter edge-to-edge repair (MitraClip). Mitral valve replacement is preferred in unstable patients to reduce surgical time, however mitral valve repair could be considered in selected patients; MitraClip implantation procedure should be considered in patients with prohibitively high surgical risk [6, 7]. Surgical team experience and a multidisciplinary evaluation based on morpho-functional findings of valvular apparatus play a key role in decision making process.

In consideration of our experience, of the general condition of the patient and the presence of comorbidities predisposing to alterations of myocardial microcirculation, we opted for surgical mitral valve replacement. Despite medical literature suggest that patients with MINOCA have better clinical and prognostic outcome than patients with obstructive coronary artery disease, an adequate clinical and diagnostic monitoring of AMI complications should be pursued, even in absence of significant acute thrombotic lesions on coronary angiography, in order to prevent fatal outcome.

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