Anterolateral papillary muscle rupture caused by myocardial infarction: A case report

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Abstract

The rupture of the anterolateral papillary muscle is less common than the posteromedial papillary muscle, as the anterolateral muscle has a dual blood supply, whereas the posteromedial papillary muscle has a single blood supply. We present a 49-year-old male patient who has presented with chest pain and dyspnea. A transesophageal echocardiogram was performed which showed that the mitral valve was normal in thickness with a flail anterior leaflet not coapting with the posterior leaflet due to the coronary artery disease. The patient underwent a mitral valve replacement and left anterior coronary artery and obtuse marginal bypass grafting.

Introduction

Rupture of a papillary muscle is an uncommon but often fatal complication of acute myocardial infarction (MI) which is responsible for approximately 5% of early deaths (1). Papillary muscle rupture is usually seen in relatively small area infarctions, often with the modest coronary disease extend, revealed by angiogram [1]. The condition may result in acute mitral regurgitation, acute pulmonary edema, and/or cardiogenic shock [2]. The rupture of the posteromedial papillary muscle is most common and comprises about 75% of cases. Posterior medial 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 supply 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 anterolateral MI [3,4]. Here, we present a case of anterolateral papillary muscle rupture.

Case Report

A 49-year-old male patient was admitted to emergency room with chest pain and dyspnea. An electrocardiogram is recorded at the time of admission with no sign of ST segment abnormality (Figure 1). A series of troponin and CK measurements were performed with the following results; troponin, 0, 15, 2, 14 and 3, 40; CK; 353, 360, 392 at 1, 2, 3 h respectfully. The patient was then accepted to coronary intensive care unit with the diagnosis of non-ST MI. He had a history of insulin dependent diabetes for 10 years and was smoking a pack of cigarettes for at least 20 years. Upon physical examination, crepitant rhales were heard at the basal segments of the lungs bilaterally. Cardiac auscultation revealed normal S1 and S2, but with a holosystolic murmur at the aortic area. Arterial blood pressure was 140/90 mmHg. A heart rate was 96/min with sinus rhytm.

An emergent coronary angiography was performed with the following findings; left anterior descending artery stenoses 70% before the origin of first the diagonal branch and %40 after the second diagonal, circumflex artery diffuse stenosis after the origin of OM2 branch and subtotal occlusion of OM3 branch, right coronary artery had 30% stenosis at the mid portion (Figure 2). A transthoracic echocardiography was performed and revealed rupture of anterolateral papillary muscle with a regurgitant orifice area of 4.7 mm² (Figures 3 and 4). Inferior segments were found hypokinetic and left ventricular ejection fraction was 50%.

The patient was then taken to the operating theater to perform a mitral valve replacement and coronary artery bypass procedure. Left anterior descending artery and circumflex artery stenoses were bypassed with saphenous vein grafts. Mitral valve replacement was performed with size 25 mechanical mitral valve prosthesis (Figure 5). An echocardiography...
performed 2 weeks postoperatively revealed normal left ventricular function with no sign of regurgitation.

**Discussion**

The clinical presentation and severity of a papillary muscle rupture depends on the involved coronary artery and left ventricular performance. This is usually clinically apparent 6 days after the acute MI phase [1], and compatible with the presentation of our patient. 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 of the papillary muscle may occur as a complication of ischemia; prolapse, elongation or different degrees of rupture, partial rupture being the most common type of rupture [5].

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 [6,7]. Transthoracic echocardiography is useful in the diagnosis of papillary muscle rupture, with a sensitivity of 65-85% [8]. A flail segment of the mitral valve and a severed papillary muscle or chorda can frequently be seen moving freely within the left ventricular cavity. However, in some cases, transthoracic echocardiography is not informative, and transesophageal echocardiography is required to establish the diagnosis. This is most likely to occur when the ruptured head does not prolapse into the left atrium, as was observed in our patient, a feature reported in up to 35% of cases [6].

High mortality rates observed with the medical management of papillary muscle rupture impose urgent surgical intervention. The timing of the intervention is dictated by the patient’s hemodynamic stability [9,10]. 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 [11]. The preferred treatment for PMR is to replace the mitral valve with a prosthetic valve (MVR). The alternative to MVR is mitral valve repair, which consists of reattachment of the ruptured papillary muscle head to the base of the papillary muscle or to the left ventricle with or without ring annuloplasty [12,13]. Although mitral valve repair can be performed with a good outcome in selected cases [12,13], this technique is technically more challenging, especially in friable infarcted tissue. In addition, a failed repair would inevitably cause poor outcomes. In contrast, since MVR is technically straightforward and reproducible, and definitely reduces left atrial volume overload, we consider MVR to be the treatment of choice in these cases.

Conclusion

Emergent surgical intervention remains the treatment of choice for papillary muscle rupture. Surgery involves significant operative mortality (18%), but the recent trends are associated with lower operative risk, particularly with associated CABG (8,7%). Long-term outcome after surgery is the same as that of similar MI without papillary muscle rupture. These encouraging observations emphasize the importance of prompt diagnosis and an aggressive therapeutic approach for patients suffering papillary muscle rupture after MI. This case is unusual in presenting two uncommon features of ischemic papillary muscle: rupture of the anterolateral muscle after MI involving the inferoposterior walls, and the fact that the ruptured muscle head did not prolapse because it had become trapped in the left ventricle by a cord attachment.

References