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CASE REPORT

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Massive mitral regurgitation secondary to acute ischemic papillary muscle rupture: The role of echocardiography

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Abstract

Papillary muscle rupture is an infrequent but often fatal mechanical complication of acute myocardial infarction (AMI). We report the case of an AMI complicated by the development of an abrupt cardiogenic shock due to the rupture of the head of the postero-medial papillary muscle with echocardiographic demonstration of severe mitral regurgitation due to flail posterior mitral valve leaflet. After initial stabilization with medical therapy and diagnostic coronary angiography, the patient was referred for urgent cardiac surgery and successfully underwent mitral valve replacement with implantation of a bioprosthesis. This case confirms the importance of transthoracic echocardiography in diagnosing mechanical acute complications during an AMI and in the decision making of patients with sudden onset of hemodynamic compromise. Transthoracic echogardiography should be immediately carried out in all patients in whom a mechanical complication during an AMI is suspected. (Cardiol J 2010; 17, 4: 397–400)

Key words: echocardiography, papillary muscle, rupture

Introduction

Papillary muscle rupture is an infrequent but often fatal mechanical complication of acute myocardial infarction (AMI). Despite its rarity, it is an important cause of severe mitral regurgitation (MR) usually proceeding to heart failure and, if not corrected, to cardiogenic shock and eventually death [1]. Echocardiography is one of the non-invasive imaging assessment techniques that can identify mechanical complications such as acute MR in the setting of AMI [2], and it allows for precise location of the papillary muscle rupture and leaflet involvement, modality and entity of MR, and hemodynamic complications. We report the case of an AMI complicated by the rupture of the head of the postero-medial papillary muscle with echocardiographic demonstration of severe MR due to flail posterior mitral valve leaflet.

Case report

A 68-year old hypertensive woman, not known to have other coronary risk factors, was initially evaluated in another hospital after sudden onset of dyspnoea without chest pain. Physical examination noted a systolic murmur compatible with MR, pulmonary rales and crackles in the bilateral lung basis. The patient presented with blood pressure of 150/ /100 mm Hg and a pulse rate of 90 beats per minute.

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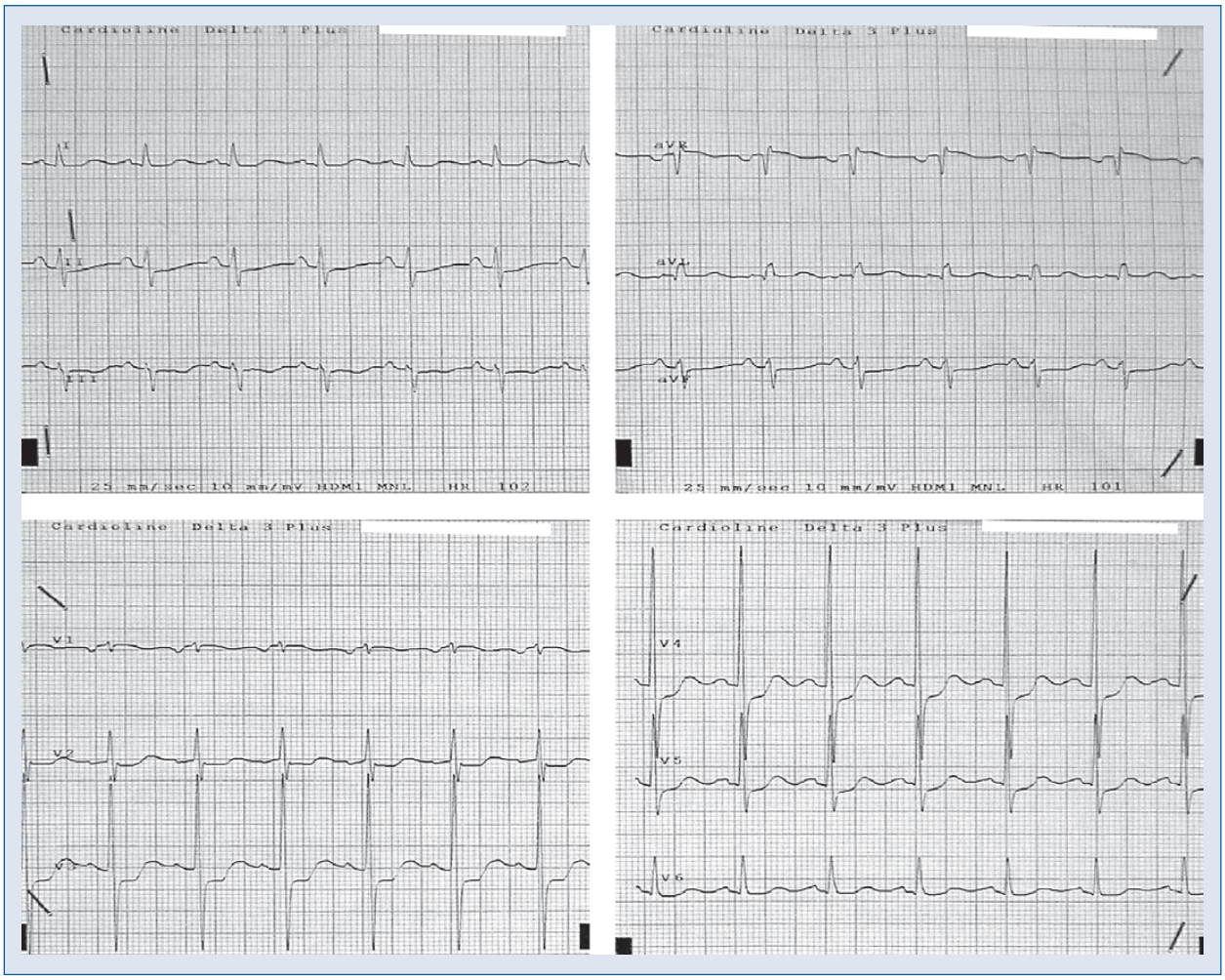

Figure 1. Electrocardiograph showing normal sinus tachycardia, heart rate 110/min, normal axis and ST segment depression in the II/III/aVF and V5–V6 (inferolateral ischemia).

Mild room air oxygen desaturation was present, requiring non-invasive ventilation with continuous positive airway pressure. Electrocardiography revealed sinus tachycardia with ST segment depression in precordial leads (Fig. 1).

The patient suddenly became hemodynamically unstable: pulse rate raised to 110 beats per minute, blood pressure dropped to 90/60 mm Hg, with a room air oxygen saturation of 60% and a respiratory rate of 40 acts per min, associated with evidence of peripheral hypoperfusion. Intravenous inotropes and diuretics were given and endotracheal intubation and ventilation with positive endexpiratory pressure were also immediately instituted.

Electrolytes, liver and kidney function tests were within normal limits. The patient's creatine kinase (CK) levels peaked at 298 UI/L, the serum troponin I was 5.05 ng/mL, consistent with myocardial infarction.

Chest X-ray demonstrated acute pulmonary oedema. Diagnosis of non-ST elevation acute coronary syndrome was established. After the initial stabilization, the patient was transferred to our tertiary center to undergo urgent coronary angiography. A pre-procedural transthoracic echocardiogram (TTE) showed a posterior mitral valve flail with a massive MR (Fig. 2) due to the complete rupture of the head of the posteromedial papillary muscle (Fig. 3). Other findings included hypercontractile left ventricle (LV) with a mid anterolateral hypokinesis and ejection fraction of 70%, along with normal left and right ventricular size and dimensions of left atrium in parasternal long axis and apical four chamber projections.

Coronary angiography revealed a multi-vessel atherosclerotic disease with a sub-occlusive 99% stenosis of a first marginal branch (most likely the culprit lesion) and 75% stenosis of distal left ante-

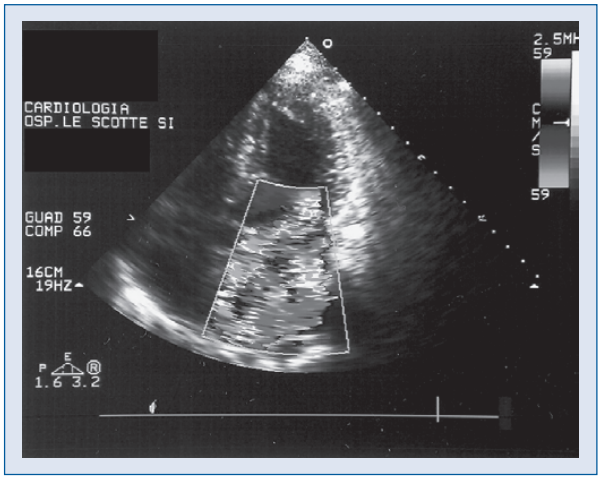

Figure 2. The transthoracic echocardiography showed a posterior mitral valve flail with a severe mitral regurgitation into the left atrium because of the rupture of the papillary muscle; dimensions of left atrium in apical four-chamber projections were normal.

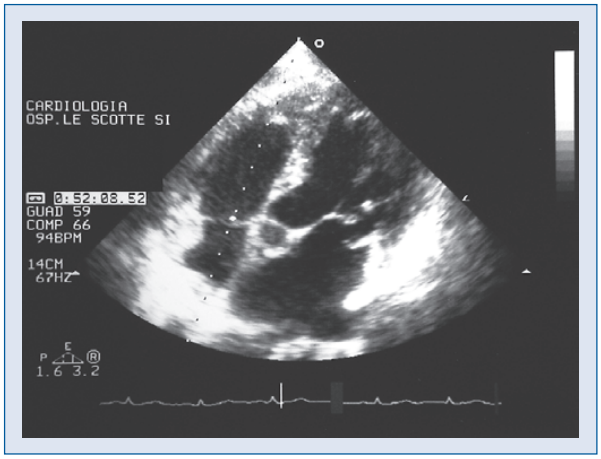

Figure 3. A transthoracic echocardiographic apical four--chambers view illustrating (in upper panel) a flail posterior mitral valve leaflet with ruptured papillary muscle. In lower panel the transthoracic echocardiography showing the complete rupture of the head of the posteromedial papillary muscle.

rior descending and of first diagonal. Overall, the patient was not deemed suitable for revascularization (Fig. 4), with minor lesions also involving the right coronary artery (Fig. 4). Left ventriculography confirmed severe mitral regurgitation and uncovered significant LV kinetic alterations. The patient was thus referred for urgent cardiac surgery. She successfully underwent open heart surgery with mitral valve replacement with implantation of

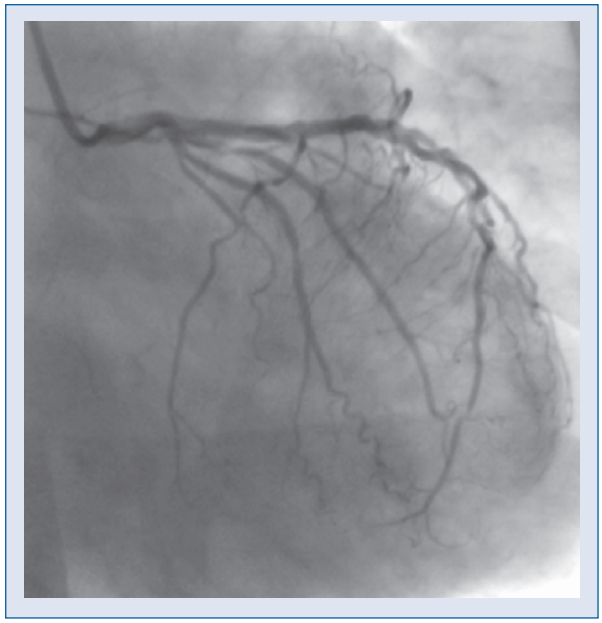

Figure 4. Coronary angiogram showing first obtuse marginal (OM1) occluded by plaque ateromatosa with an important stenosis (stenosis 99%).

a bioprosthesis. The post-operative course was uneventful and she was discharged home on day 8 after surgery.

Discussion

The abrupt rupture of papillary muscle determines massive MR, virtually always complicated by acute heart failure and pulmonary oedema. It has been reported in 1–5% of all patients suffering an AMI. The rupture is more frequent during the first week after AMI, and accounts for approximately 5% of deaths in post-AMI patients [3]. Without surgical treatment nearly 90% of patients with ruptured papillary muscle die within a week [4].

The involvement of the postero-medial papillary muscle is 6–12 times more common than that of the antero-lateral. Indeed, postero-medial papillary muscle vascularization is provided only by the interventricular posterior coronary artery originating even from the right coronary or from the circumflex coronary artery, and it may aggravate infarction heralded by occlusion in such vessels [5].

In patients developing a rupture of a papillary muscle, the surgical approach is conditioned by the threat of a quick and unexpected deterioration with respect to an early hemodynamic instability, and urgent surgery has to be considered irrespective of the clinical presentation [6].

Our case confirms the importance of TTE in diagnosing mechanical acute complications during an AMI and in the decision making of patients with sudden onset of hemodynamic compromise.

Echocardiography is the imaging technique of choice for detecting complications of acute infarction including myocardial free wall rupture, acute ventricular septal defect, and mitral regurgitation secondary to papillary muscle rupture or ischemia [2].

TTE is able to identify a papillary muscle rupture with a diagnostic sensitivity of 65–85% [7]. Transesophageal echocardiography is more sensitive than TTE, but in this case the TTE image was consistently clear and diagnostic with no need for further characterization. TTE is particularly useful, not only for studying LV size and function, but also for directly showing complications of ruptured papillary muscle such as the 'flail' of mitral flaps and the papillary stump interested by the prolapsing rupture in left atrium.

Acknowledgements

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