Complete rupture of the anterolateral papillary muscle caused by coronary spasm

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Received 21 April 2015; received in revised form 21 July 2015; accepted 27 July 2015

Abstract

Papillary muscle rupture usually occurs as a catastrophic complication of acute myocardial infarction in patients with coronary artery stenosis; it is therefore less common in patients without coronary artery stenosis. We report the case of a 67-year old woman without coronary artery stenosis who suffered an acute anterolateral papillary muscle rupture and was successfully treated with mitral valve replacement. Evidence of coronary spasm was found on a coronary vasomotion test, suggesting that a high sensitivity to coronary spasm may explain a mechanism of isolated papillary muscle infarction.

Keywords: Acute mitral regurgitation • Anterolateral papillary muscle rupture • Coronary spasm • Mitral valve replacement

INTRODUCTION

Papillary muscle rupture, a rare mechanical complication of acute myocardial infarction in patients with coronary artery stenosis, is often associated with lethal haemodynamic deterioration, including cardiogenic shock and acute pulmonary oedema [1]. Anterolateral papillary muscle (APM) rupture is less frequent than posteromedial papillary muscle (PPM) rupture and is extremely rare in the absence of coronary artery stenosis. We present a patient with acute severe mitral regurgitation (MR) due to complete rupture of an isolated APM infarction, without coronary artery stenosis and with evidence of coronary spasm, successfully treated with mitral valve replacement (MVR).

CASE REPORT

A 67-year old woman presented to the emergency room with chest pain, severe dyspnoea and general weakness that had begun 2 h prior to admission. She had had mild fatigue for 7 days associated with the onset of mild neck and back pain. Her past medical history was unremarkable, with no hypertension and no medication, but active smoker, with a negative family history of coronary artery disease. Because of rapid respiratory and haemodynamic deterioration, with tachypnoea, anuria, increasing oxygen demand and a drop in blood pressure requiring inotropic support, the patient was immediately transferred to the intensive care unit. A chest X-ray showed severe bilateral pulmonary oedema; the patient was intubated and immediately begun on mechanical ventilation. Laboratory tests revealed a CK-myocardial band of 20 U/l and a positive troponin-T (0.798 ng/ml). Electrocardiogram (ECG) revealed sinus rhythm and no ST-segment changes in any lead. Transthoracic echocardiography (TTE) revealed severe MR due to APM rupture. After insertion of an intra-aortic balloon pump, emergency coronary angiography was performed and revealed no stenotic lesions. We decided to perform emergency mitral valve surgery.

Intraoperative transoesophageal echocardiography (TEE) demonstrated massive MR with flail in Segments A1, A2, anterolateral commissure and P1 secondary to complete rupture of the APM but no wall-motion abnormalities (Fig. 1A and Videos 1 and 2). There was complete rupture of the APM (Fig. 1B). After complete resection of the anterior leaflet and partial resection of the posterior leaflet, standard MVR was performed. Pathological examination showed coagulation necrosis of the papillary muscle surrounded by inflammatory cell infiltration.

On postoperative single-photon emission computed tomography (SPECT)/computed tomography (CT) scan coronary angiography fusion images using thallium-201 (201TI) and iodine-123 beta-methyl-p-iodophenyl-pentadecanoic acid (123I-BMIPP), there was less uptake of 123I-BMIPP than of 201TI in the first obtuse marginal (OM-1) segment, corresponding to the OM-1 artery (Fig. 1C). This indicated the presence of myocardial metabolic dysfunction; i.e. myocardial ischaemia, in this area. To identify the cause of an APM rupture without coronary artery stenosis, we performed a coronary vasomotion test with intracoronary acetylcholine (ACH) provocation. Initial coronary angiography demonstrated no stenotic lesions in either coronary artery (Fig. 2A). After injecting ACH 100 µg into the left coronary artery, there was
severe vasoconstriction at the first diagonal, OM-1 and second obtuse marginal (OM-2) and posterolateral branches, with impaired coronary blood flow distal to these stenoses (90–99%) (Fig. 2B). Although the patient was asymptomatic, ECG showed signs of ST-segment depression in leads V5 and V6. Therefore, nitroglycerine 0.4 mg was injected into the left coronary artery, and after 2 min the stenotic lesions disappeared (Fig. 2C). When ACH 50 μg was infused into the right coronary artery, similar severe vasoconstriction (90–99%) occurred at the site of the AV node artery.

The patient was extubated on postoperative day 3 and discharged from the intensive care unit in good clinical condition, without neurological deficit, on Day 5. Postoperative TTE showed no MR and no wall-motion abnormalities.

**DISCUSSION**

Papillary muscle rupture has been reported to occur—rarely—in the absence of significant coronary artery stenosis—because of endocarditis, blunt chest trauma, Takotsubo cardiomyopathy and acute pancreatitis [2–4]. In most of our patients, the PPM is perfused entirely by either the right coronary artery or its posterolateral branch. The APM is more often perfused by two arteries, the OM-1 and the first diagonal. When one of these is occluded, collateral flow from the patent vessel may prevent dysfunction.

However, after analysis of the perfusion pattern of the APM, Voci et al. [5] reported that about 30% of individuals had only a single blood supply. Similarly, our patient’s APM was likely supplied only by the OM-1 branch. The myocardial metabolic dysfunction of the OM-1 segment, observed on postoperative SPECT/CT angiography, suggested that isolated infarction or ischaemia of the small area of coronary spasm, which resulted from a single blood supply from the OM-1, had led to APM rupture. Although coronary embolism, especially in atrial fibrillation, can cause isolated papillary muscle infarction, no left atrial thrombus was seen on TEE in our patient.

The APM usually has a single head, while the PPM often has two or more heads. Rupture—most commonly complete—of the APM can be more serious than rupture of the PPM. The papillary muscles are subendocardial structures, and small endocardial infarctions can cause papillary muscle ruptures; thus, even a small area of myocardial infarction due to coronary spasm can cause

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**Figure 1:** (A) Transoesophageal echocardiography revealing complete detachment of the head of the anterolateral papillary muscle (white arrow). (B) Bileaflet prolapse on the anterolateral side of the mitral valve resulting from complete rupture of the APM. (C) Dual-isotope single-photon emission computed tomography SPECT using $^{201}$Tl and $^{123}$I-BMI/P/CT coronary angiography fusion image. There was less uptake of $^{123}$I-BMI/P than of $^{201}$Tl in the first obtuse marginal segment, corresponding to the first obtuse marginal branch. APM: anterolateral papillary muscle; AAo: ascending aorta; AC: anterior commissure; LA: left atrium; LV: left ventricle; $^{123}$I-BMI/P: iodine-123 beta-methyl-p-iodophenyl-pentadecanoic acid; $^{201}$Tl: thallium-201.

**Video 1:** Intraoperative movie of transoesophageal echocardiography. Erratic motion of the ruptured anterior head in the left atrium and ventricle.

**Video 2:** Three-dimensional transoesophageal echocardiography demonstrated massive mitral regurgitation with flail in Segments A1, A2, AC and P1 secondary to complete rupture of the anterolateral papillary muscle. AC: anterolateral commissure.
catastrophic complications. The preferred treatment for papillary muscle rupture is to replace the mitral valve with a prosthetic valve. The alternative to MVR is mitral valve repair, which consists of reattaching the ruptured papillary muscle head with or without ring annuloplasty. In cases of ruptured chordae tendineae, repair is usually feasible and is preferred over MVR. Although mitral valve repair can lead to a good outcome, it is more technically challenging than replacement, especially where there is friable infarcted tissue after complete papillary muscle rupture. In contrast, since MVR is technically straightforward and reproducible, we consider MVR to be the treatment of choice for APM rupture, but surgical strategy will be based on a patient’s overall condition.

CONCLUSIONS

We have described an unusual presentation of acute MR due to isolated complete APM rupture in the absence of elevated isoenzymes, wall-motion abnormalities or coronary artery stenosis. In our patient, SPECT/CT coronary angiography fusion imaging suggested myocardial ischaemia in the OM-1 segment; coronary vasomotion testing showed high sensitivity of the OM-1 to coronary spasm. Our findings suggest that a high sensitivity to coronary spasm may be a mechanism of isolated APM infarction.

Conflict of interest: none declared.

REFERENCES