Case Report

Complete rupture of the anterolateral papillary muscle complicated with acute myocardial infarction due to diagonal branch occlusion

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Summary It is well known that post-infarction papillary muscle rupture of the anterolateral papillary muscle is less frequent than that of the posteromedial papillary muscle. This is thought to be due to a difference in blood supply (single vs dual) of the papillary muscles. Recently, we had two cases in which occlusion of the diagonal branch of the left anterior descending artery was found to be the culprit lesion of acute myocardial infarction leading to complete rupture of the anterolateral papillary muscle. Herein, we report on these two rare successful surgical cases with some review of the literature.

Introduction

Papillary muscle rupture (PMR) complicating acute myocardial infarction (AMI) is rare, but when it occurs, it is often associated with lethal hemodynamic deterioration including cardiogenic shock or acute pulmonary edema [1–9]. Rupture of the anterolateral papillary muscle is known to be less frequent than that of the posteromedial papillary muscle [1–9], and this is thought to be due to the fact that the anterolateral papillary muscle has dual blood supply from both the left anterior descending artery (LAD) and the left circumflex artery (LCX), whereas the posteromedial papillary muscle has only single blood supply from the posterior descending artery of either the LCX or the right coronary artery (RCA). We report on two surgical cases of complete PMR of the anterolateral papillary muscle complicating AMI due to single occlusion of the diagonal branch of the LAD.

Case 1

An 86-year-old man, who had a history of systemic hypertension and cerebral infarction, was transferred to our hospital due to a sudden onset of dyspnea. On initial examination, his blood pressure and heart rate were 70/40 mmHg and 90 beats per minute, respectively. No significant systolic murmur was noted at the apex. Blood laboratory tests revealed a
Papillary muscle rupture due to diagonal branch occlusion

Figure 1 (a) Electrocardiogram showed ST elevation in leads aVR and aVL, and ST depression in leads II, III, aVF and V3—V5. (b) Chest X-ray showed severely emphysematous lungs and mild cardiomegaly with pulmonary congestion.

white blood cell (WBC) count of 9710/mm³, C-reactive protein (CRP) of 6.6 mg/dl, creatine kinase (CK) of 227 IU/L, CK-MB of 3 IU/L, and positive troponin T. An electrocardiogram (ECG) showed ST elevation in leads aVR and aVL, and ST depression in leads II, III, aVF, and V3—V5 (Fig. 1a), and a chest X-ray revealed severely emphysematous lungs and mild cardiomegaly with some pulmonary congestion (Fig. 1b). He was diagnosed with acute coronary syndrome, then transferred to the cardiac catheterization laboratory. On coronary angiography (CAG), there was 90% stenosis of the mid LAD and 99% stenosis of the first diagonal branch (D1). However, there was no significant stenosis in the LCX and the RCA. Successful PCI was performed to revascularize both the LAD and the D1 lesions (Fig. 2a). Despite successful PCI, his systolic blood pressure continued to range from 50 to 70 mmHg requiring inotropic support and his dyspnea worsened. Transthoracic echocardiography (TTE) showed severe mitral regurgitation (MR) due to complete rupture of the anterolateral papillary muscle. An intra-aortic balloon pump (IABP) was inserted immediately, and he was transferred to the operating room for emergency mitral valve replacement (MVR). We found a complete rupture of the anterolateral

Figure 2 (a) Coronary angiography after percutaneous coronary intervention of the left anterior descending artery revealed total occlusion in the first diagonal branch (arrow) in Case 1. (b) Preoperative coronary angiography showed total occlusion in the first diagonal branch (arrow) in Case 2.
papillary muscle. After complete resection of the anterior leaflet and partial resection of the posterior leaflet (Fig. 3a), standard MVR was performed using a 27-mm bioprosthesis. Weaning from cardiopulmonary bypass (CPB) was achieved smoothly with IABP support. The aortic cross-clamp time (ACC) and the CPB time were 54 min and 76 min, respectively. His postoperative course was complicated with acute renal failure and respiratory failure, which required prolonged hospitalization. He was finally discharged 70 days after surgery. At three months post-discharge, he was doing very well. Pathological examination showed coagulation necrosis of the papillary muscle surrounded by inflammatory cell infiltration.

Case 2

A 74-year-old man with no significant prior medical history experienced a sudden onset of dyspnea with chest pain and was referred to our hospital due to cardiogenic shock. In the emergency department, a physical examination revealed a blood pressure of 62/46 mmHg and heart rate of 90 beats per minute. Also, a systolic murmur of grade III/VI was heard at the apex. Blood laboratory tests revealed a WBC count of 12,100/mm³, CRP of 3.7 mg/dl, CK of 352 IU/L, CK-MB of 42 IU/L, and positive troponin T. There were no ST changes in any of the ECG leads. TTE showed severe MR associated with complete rupture of the anterolateral papillary muscle. CAG was performed after an IABP was inserted. There was 90% stenosis of the LAD, with total occlusion of the D1 (Fig. 2b), and normal LCX and RCA. We decided to perform emergency MVR along with coronary artery bypass grafting (CABG) for the LAD stenosis. At surgery, bileaflet prolapse involving lateral side of the mitral valve due to complete rupture of the anterolateral papillary muscle was observed (Fig. 3b). The anterior leaflet was excised and MVR was performed using a 29-mm bioprosthesis with chordal preservation of the posterior leaflet. Then, an aorto-coronary artery bypass of the LAD was made using a piece of a great saphenous vein. Weaning from CPB was achieved smoothly with IABP support. The ACC time and CPB time were 73 min and 154 min, respectively. The postoperative course was uneventful. The patient was discharged on the 17th postoperative day and has been doing well for two and a half years since then. Pathological findings were similar to those of Case 1.

Discussion

PMR is a rare but serious complication after AMI. It usually occurs 2–7 days after AMI with a prevalence of 1–5%, and usually leads to rapid hemodynamic instability [1–8]. If PMR is not treated surgically, the mortality rate is reported to be 70% and 94% within 24 h and 2 months, respectively [1, 6, 7]. Thus, the diagnosis of PMR needs to be made quickly and emergency surgery must be performed.

Rupture of the anterolateral papillary muscle is less frequent than that of the posteromedial papillary muscle, and it occurs in about 10–27% of all PMR cases [3–5, 9]. This is thought to be because the anterolateral papillary muscle usually has a dual blood supply, whereas the posteromedial papillary muscle has a single blood supply. However, Voci et al. analyzed the perfusion pattern of the anterolateral papillary muscle and revealed that about 30% of individuals had only a single blood supply [10]. The two present cases definitely had only a single blood supply from the diagonal branch of LAD. Therefore, it should be carefully noted that even a small area of myocardial infarction due to D1 occlusion can cause catastrophic complications. From an anatomical standpoint, the anterolateral papillary muscle usually has a single head, while the posteromedial papillary muscle commonly has double or multiple heads. Consequently, rupture of the anterolateral papillary muscle, which is most commonly a complete rupture, leads to more serious (often lethal complications) than rupture of the posteromedial papillary muscle [2, 3, 7].

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 [2–5, 9]. Although mitral valve repair can be performed with a good outcome in selected cases [3–5, 9], 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

Figure 3  Surgical specimens of the ruptured anterolateral papillary muscle and the mitral leaflets (a) Case 1; (b) Case 2.
reduces left atrial volume overload, we consider MVR to be the treatment of choice in these cases.

**Conclusion**

We experienced two rare cases of the anterolateral PMR complicated with AMI due to D1 occlusion. We successfully performed emergency MVR with excellent outcomes.

**References**


