Rupture of the Anterolateral Papillary Muscle Caused by a Single Diagonal Branch Obstruction

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This report presents the first case of anterolateral papillary muscle rupture caused by a diagonal branch occlusion only. Although the patient was in shock on admission, he was successfully treated by emergency surgery after hemodynamic stabilization by a percutaneous cardiopulmonary support system. This case implies that the anterolateral papillary muscle has a single blood supply and that it can rupture by an occlusion of only one diagonal branch even after balloon angioplasty. (*Circ J* 2002; 66: 872–873)

Key Words: Myocardial infarction; Papillary muscle rupture; Percutaneous cardiopulmonary support system; Shock

A 70-year-old Japanese man was referred to the coronary care unit 1 h after the onset of a first episode of severe chest oppression. On admission, he presented in cardiogenic shock with profound sweating. Physical examination revealed blood pressure 80/50 mmHg, heart rate 130 beats/min, gallop heart sounds, crackles in both lungs, and alert consciousness. Neither of 2 experienced cardiologists (T.Y., H.W.) could detect a systolic murmur. ECG showed sinus tachycardia and ST-segment elevation in leads I and aVL, and ST depression in leads II, III and aVF. Atrial blood gas analysis showed PaO₂ 53 torr and PaCO₂ 48 torr. Echocardiography revealed akinesis in the lateral wall of the left ventricle and moderate mitral regurgitation caused by prolapse of the anterior mitral leaflet, although the echo image quality was not satisfactory because of tachypnea, tachycardia and the patient's restlessness. The size of infarcted area was limited to the lateral wall only, and the ejection fraction was 65%. Emergency coronary angiography was performed 30 min after admission. There was an occlusion at the proximal site of the first large diagonal branch (Fig 1). The left circumflex coronary artery was hypoplastic, and fixed stenosis was not detected in the other coronary arteries. The hemodynamic parameters became more critical just after coronary arteriography: the systolic blood pressure fell to 60 mmHg. To ascertain the cause of the hemodynamic deterioration, we immediately re-examined the echocardiography images. On color Doppler, a ruptured anterolateral papillary muscle could be seen moving freely with massive mitral regurgitant flow (Fig 2). To improve the hemodynamic state, a percutaneous cardiopulmonary support system and intra-aortic balloon pumping were promptly started

instead of revascularization. Emergency mitral valve replacement was performed on the same day. Revascularization of the occluded diagonal branch by coronary artery bypass grafting was not performed because its territory was relatively small and did not affect global left ventricular function. The anterolateral papillary muscle was completely ruptured at the proximal site and on the histological examination, had neutrophil invasion without fibrosis, which was compatible with an extremely early inflammatory phase of acute myocardial infarction (AMI) (Fig 3). The patient recovered after the surgery and was discharged on foot on the 20th day.

Fig 1. (A) ECG on admission showed sinus tachycardia and ST-segment elevation in leads I and aVL, and ST depression in leads II, III and aVF. (B) Emergency coronary arteriography revealed an occlusion at the proximal site of the first large diagonal branch (indicated by an arrow) without any other significant stenosis in the left coronary artery (LAO view). (C) The left circumflex artery was hypoplastic (RAO view).

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Fig 2. (A) Echocardiography from the apex revealed the ruptured anterolateral papillary muscle, moving freely (indicated by an arrow). (B) Massive mitral regurgitant flow was detected by color Doppler echocardiography. (C) The anterolateral papillary muscle was absent on the left ventricular wall in the short axis view (indicated by an arrow).

Fig 3. (A) The ruptured anterolateral papillary muscle resected during the surgery. (B) A specimen of the base of the papillary muscle (H&E, $\times 10$). (C) Neutrophil invasion (arrow) and spot bleeding sites (triangle) without fibrosis are compatible with an extremely early inflammatory phase of myocardial infarction ($\times 200$).

Discussion

Papillary muscle rupture is a rare but critical mechanical complication of AMI. In particular, rupture of the anterolateral papillary muscle occurs in 10% of cases compared with rupture of the posteromedial papillary muscle, because the latter has a single blood supply from the right coronary artery or the left circumflex coronary artery (LCX). The anterolateral papillary muscle has a dual blood supply from the left anterior descending artery (LAD) and the LCX.^{1,2} To our knowledge, the present report is the first to document that a single obstruction of the diagonal branch led to an anterolateral papillary muscle rupture. Previous reports have found that rupture of the anterolateral papillary muscle followed an obstruction in the LCX or a trunk of the LAD;^{3–6} but not in only one diagonal branch. The present patient's anatomy of a hypoplastic LCX increased the possibility of papillary muscle rupture by an occlusion of only one diagonal branch and is a warning to cardiologists that there is a possibility of a single blood supply for the anterolateral papillary muscle and there is a risk of its dysfunction or rupture by an occlusion even after balloon angioplasty.

The interval between the onset of myocardial infarction and papillary muscle rupture varies from 6 h to 7 days,¹ but in the present case it occurred 1 h after the onset of AMI. At first we did not assume a papillary muscle rupture because the time elapsed was too short for myocardial rupture. This case taught us not to exclude papillary muscle rupture from the differential diagnosis of the shock state in AMI, even within 1 h after the onset.

The mechanism of a myocardial rupture in such an extremely early phase of AMI remains unknown, but is

probably related to the excessive power of the hyperkinetic motion of the neighboring wall and the relatively small transmural infarcted area. Once a ruptured papillary muscle is diagnosed, mitral valve replacement needs to be carried out as soon as possible and as a bridge to the surgery, a combination of percutaneous cardiopulmonary support system with intra-aortic balloon pumping is a useful strategy in such a hemodynamically deteriorated patient.⁷

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References

1. Nishimura RA, Shaff HV. Papillary muscle rupture complicating acute myocardial infarction: Analysis of 17 patients. *Am J Cardiol* 1983; **51**: 373–377.
2. Voci P, Biolotta F, Caretta Q. Papillary muscle perfusion pattern: A hypothesis for ischemic papillary muscle dysfunction. *Circulation* 1995; **91**: 1714–1718.
3. Salvi L, Barbier P. Circulatory support with hemopump in cardiogenic shock secondary to papillary muscle rupture. *Cardiologia* 1995; **40**: 865–868.
4. Kranidis A, Kouloouris S. Mitral regurgitation from papillary muscle rupture. Role of transesophageal echocardiography. *J Heart Valve Dis* 1993; **2**: 529–532.
5. Sharma SK, Secjler J. Clinical angiographic and anatomic findings in acute severe ischemic mitral regurgitation. *Am J Cardiol* 1992; **70**: 277–280.
6. Barbour DJ, Roberts WC. Rupture of a left ventricular papillary muscle during acute myocardial infarction: Analysis of 22 necropsy patients. *J Am Coll Cardiol* 1986; **8**: 558–565.
7. Yasu T, Murata S, Katsuki T, Fujii M, Kubo N, Ohmura N, et al. Acute myocarditis successfully treated by percutaneous cardiopulmonary support applied by a newly developed heparin-binding oxygenator and circuits. *Jpn Circ J* 1997; **61**: 1037–1042.