

Chronic Papillary Muscle Rupture: 14-Year Survival without Surgical Treatment

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Papillary muscle rupture is a life-threatening complication of myocardial infarction which is usually refractory to medical treatment. We present a very rare case of a 65-year-old woman who had a myocardial infarction and posteromedial papillary muscle rupture which was only treated with medical therapy, including her corresponding 14-year follow-up. However, surgical intervention is still strongly recommended because the prognosis of acute papillary muscle rupture associated with myocardial infarction remains poor.

Key Words: Complication • Myocardial infarction • Papillary muscle rupture • Survival

INTRODUCTION

Papillary muscle rupture (PMR) is a rare but life-threatening complication of myocardial infarction.¹ It is usually refractory to medical treatment and the mortality rate is high even with cardiac surgery. However, we presented a case with posteromedial PMR who survived for 14 years without surgical treatment.

CASE REPORT

A 68-year-old woman who complained of chest pain was diagnosed with acute inferior-posterior myocardial infarction in 1997. She had a history of hypertension and hyperlipidemia. She had rales at the bases of the lungs (Killip Class II) during admission and a 3/6 systolic murmur best heard at the apex. Transthoracic echo-

cardiography (TTE) visualized a gross cardiac mass with a moderate mitral regurgitation. Left ventricular ejection fraction (LVEF) was calculated 45% and left atrial (LA) anterior-posterior (AP) diameter was 48 mm (Table 1). Urgent coronary angiogram showed a multi-vessel disease, and percutaneous transluminal angioplasty (without stent implantation) for the right coronary artery was performed. Transesophageal echocardiography recordings in 1997 showed a 1.91 × 2.4 cm echo-dense multilobular mass (arrow) attached to the mitral valve (Figure 1A). Coronary artery bypass grafting combined with mitral valve surgery was planned for the next step. However, the patient was unwilling to have an operation although she was clearly informed about the benefits of surgical therapy and the characteristically poor outcomes with medical therapy alone. She was discharged with a daily dose of 10 mg of ramipril, 25 mg of metoprolol, 25 mg of spironolactone, 20 mg of *pravastatin* and 100 mg of acetylsalicylic acid. She presented with severe dyspnea in March 2006. Vital signs revealed a heart rate of 65/min, blood pressure of 120/65 mmHg, respiratory rate of 29/min, and temperature of 36.5 °C. Her physical examination revealed rhythmic heart sounds with a 2/6 pansystolic murmur best heard at the apex. Bibasillary rales were detected. Bilateral mild pretibial edema was palpated. Jugular venous congestion was also visible. Electrocardiogram revealed sinus rhythm

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with 65 beats per minute with a left bundle branch block. Except for high cholesterol levels her biochemistry results were within normal ranges. Transthoracic echocardiography performed in 2006 showed an echodense mass prolapsing into the LA in each systole and moving back into the left ventricle (LV). Moderate systolic dysfunction (LVEF 36%) with a moderate to severe mitral regurgitation and an aneurysm at apex were detected. Left atrial AP diameter was 56 mm (Table 1). For further evaluation of the mass, she was investigated by trueFISP sequence cardiac magnetic resonance imaging which clearly showed rupture of the two heads of posteromedial papillary muscle (PM) (Figure 1B, arrow) and an aneurysm formation (asterisk) at the apex. She again rejected surgical treatment. She was hospitalized four times for decompensated heart failure in 2009 and 2010. Transthoracic echocardiography demonstrated freely swinging ruptured papillary muscles and a moderate to severe eccentric mitral regurgitation in 2009 (LVEF 30%, LA diameter 65 mm) and 2010 (LVEF 25%, LA diameter 68 mm) (Table 1, Figure 1C). She died due to heart failure in 2011.

DISCUSSION

The papillary muscles are contiguous structures of the adjacent LV wall which are vital for mitral valve competence.^{2,3} Both papillary muscles give chordae tendineae to both mitral leaflets which contribute to the mitral valve integrity and simultaneous motion of both mitral leaflets.⁴ Usually one anterior but often two or three posterior papillary muscles are observed. The arterial blood supply of posteromedial papillary muscle is mostly single, either from the right or left circumflex coronary artery, making it more vulnerable to ischemia compared to the anterolateral one which has a dual blood supply.⁵ In our case, posteromedial PMR was due to myocardial ischemia and the underlying hypertension might have also contributed to the ischemia by increasing myocardial wall tension. Rupture of the PM occurs in 1-3% of patients with acute myocardial infarction and accounts for 5% of infarct-related deaths.⁶ Before surgical intervention the prognosis of acute PMR associated with myocardial infarction was poor, with 33% of patients dying immediately, 50% dying within 24 hours,

Table 1. Course of left atrial diameter, severity of mitral regurgitation and left ventricular ejection fraction and LV diameters of the patient during survival

Years	1997	2006	2009	2010
LA diameter	48 mm	56 mm	65 mm	68 mm
Severity of MR	moderate	moderate to severe	moderate to severe	moderate to severe
LVEDD	49 mm	58 mm	66 mm	70 mm
LVESD	40 mm	47 mm	54 mm	60 mm
LVEF	45%	36%	30%	25%

LA, left atrial; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; MR, mitral regurgitation.

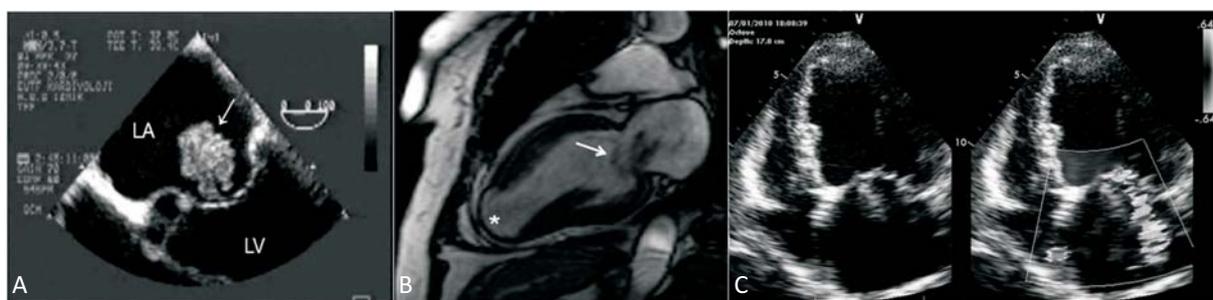


Figure 1. (A) Transesophageal echocardiography imaging in 1997 showing multilobular mass attached to the mitral valve. (B) Cardiac magnetic resonance image in 2006 showing ruptured heads of posteromedial papillary muscle. (C) Transthoracic echocardiography image in 2010 showing ruptured posteromedial papillary muscle and moderate to severe mitral regurgitation.

and only 6% surviving longer than two months.^{7,8} Open surgical treatment or replacement is the standard therapy for patients with PMR and mitral regurgitation although percutaneous mitral valve repair using Mitraclip seems to be the promising alternative therapeutic approach.⁹ Our patient was followed-up with only medical treatment because she strictly rejected surgical treatment or any invasive procedure. To the best of our knowledge, a case with PMR with such a long survival without surgical treatment has not been previously reported. Long-term survival of the patient could possibly be explained by the presence of an enlarged LA due to hypertension before the posteromedial PM ruptured and occurrence of a moderate mitral regurgitation in the patient. The presence of an enlarged LA with high compliance might contribute to adaptation to the abrupt increase in LA pressure when acute mitral regurgitation developed. Involvement of only posteromedial PM during myocardial infarction with relatively preserved LV systolic functions and long-term use of angiotensin-converting enzyme inhibitors and spironolactone therapy might also have contributed to the long-term survival in this patient. By presenting this case, we intended to add novel information about posteromedial PMR to the literature. Our patient had a relatively longer survival than expected, however she died below the average age in our country which could be explained as a premature death due to myocardial infarction complication. To avoid premature death, standard surgical treatment should clearly be advised to each patient with PMR.

CONCLUSIONS

PMR is a life-threatening complication of myocardial infarction. Although we presented a rare case of a patient PMR with 14-year survival without any invasive

therapy, standard surgical treatment is still advised for the long-term reduction of cardiovascular events.

COMPETING INTERESTS

The authors declare no conflict of interest.

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