

Acute Mitral Regurgitation Requiring Urgent Surgery because of Chordae Ruptures after Extreme Physical Exercise: Case Report

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ABSTRACT

Chordae rupture is the most common cause of severe acute mitral insufficiency. Many different mechanisms can cause an acute chordal rupture: degenerative mitral valve disease, infective endocarditis, myocardial infarction, or a posttraumatic event. We present 2 cases of acute mitral regurgitation requiring urgent surgery due to a posterior leaflet chorda rupture after extreme physical exercise.

INTRODUCTION

Chordal rupture of any of the mitral valve leaflets is the most common cause of acute mitral regurgitation requiring prompt surgery [Hickey 1985]. Previous reports showed that myxomatous changes severely affect the mechanical properties of the mitral valve chordae, leading to elongation and possible rupture [Hickey 1985; Barber 2001]. We report 2 cases of primary chordae rupture of the mitral valve after heavy lifting. Clinical presentation, diagnosis, and surgical treatment are detailed.

CASE 1

A previously healthy 52-year-old man, a state champion bodybuilder, was admitted to the emergency room after experiencing acute onset of chest pain and shortness of breath while bench-pressing 200 kg during his routine workout.

A physical examination showed signs of congestive heart failure: tachycardia, pulmonary edema, and hypertension. A chest radiograph and a chest examination confirmed severe bilateral pulmonary congestion. There was no evidence of myocardial ischemia according to an electrocardiographic evaluation, nor was there any evidence of elevated cardiac enzymes. The patient was emergently intubated and treated with a combination of afterload reduction and diuretics. A transesophageal echocardiogram confirmed the diagnosis of severe mitral regurgitation due to chordal rupture of the posterior leaflet. The patient had normal ventric-

ular function and normal chamber sizes. Once the patient had been stabilized, we performed surgery on an urgent basis on the following day. We performed a primary mitral valve repair with a P2 triangular resection including the ruptured chordae and implantation of an annuloplasty prosthetic ring. Surgical access was obtained through a 7-cm right minithoracotomy. Despite the acute nature of the patient's clinical condition, we used a limited-access approach for cosmetic reasons because of the patient's professional activity. The postoperative recovery was unremarkable. The histologic report showed myxomatous changes of the posterior leaflet.

A 6-year follow-up demonstrated an excellent outcome for the primary repair, with the patient having returned to his normal activity 6 months after surgery.

CASE 2

A 56-year-old carpenter was brought to the emergency room with severe pulmonary edema after having experienced an acute onset of chest pain while lifting "heavy doors." Signs of severe congestive heart failure were found. Although no evidence of myocardial ischemia was found, a coronary angiogram was performed, and normal coronaries arteries were demonstrated. The ventriculogram showed severe mitral regurgitation, however. A transesophageal echocardiogram revealed a normal ejection fraction and a normal left atrial size and confirmed the diagnosis.

The patient was compensated medically and brought to the operating room the next day on a semielective basis. A P2 flail was found. A conventional technique was used to repair the valve. The postoperative course was uneventful. The pathologic report demonstrated fibromyxoid changes.

COMMENT

Although degenerative mitral valve disease is the most common cause of mitral regurgitation with chordae rupture [Hickey 1985], the acute onset and clinical presentations exemplified in these 2 cases have not previously been reported to our knowledge. In a very interesting study, Barber et al demonstrated that chordae arising from a myxomatous mitral valve are likely to flail at a lower tensile stress or a lower absolute load than normal chordae [Barber 2001].

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Studies in strenuously exercised healthy dogs have indicated that a considerable increase in the volume occurs in the filling of the left ventricle, despite marked shortening of the diastolic filling period [Miyazaki 1990]. In addition to the downward shift of the diastolic pressure-volume relation, an increased slope was observed during exercise; the increase slope was suggested to reflect increased chamber stiffness [Nonogi 1988].

Is obvious that these patients have preexisting myxomatous mitral valve disease, and exposure to intense exercise, such as heavy lifting, precipitated an acute rupture of a primary chorda of the posterior leaflet and triggered massive mitral valve incompetence. In patients with acute mitral regurgitation and a normal left atrium size, the atrial pressure rises abruptly, leading to pulmonary edema, marked elevation of pulmonary resistance, and right-sided heart failure [Braunwald 1992].

The nature of the compromise of the valve and subvalvular apparatus dictates the best mode of primary repair. Kazui et al [2004] reported 12 cases of acute mitral valve insufficiency due to chordae rupture; these investigators performed a primary-repair technique with excellent results and a follow-up to 3.8 years. Even though the etiology of the myxomatous mitral valve disease is still unclear, further studies should be conducted to understand the genetics and

the natural evolution of this disease. We believe that this contribution may prompt the development of better surgical strategies and, most importantly, prevent severe complications. Transesophageal echocardiography was invaluable for prompt diagnosis in these cases.

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