Exercise-induced Acute Mitral Valve Chordae Rupture

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Exercise is a rare cause of acute chordae rupture, which is one of the important and emergent differential diagnoses in approaching a patient presenting with dyspnea. Several common diseases such as rheumatic heart disease and infectious diseases have been known to cause chordae rupture. Severe mitral valve prolapse had been reported to be at increased risk for chordal rupture. Because mitral valve prolapse may present in up to 10% of healthy individuals, primary care practitioners should recognize that certain patients with mitral valve prolapse who engage in athletics may be at increased risk for such a rare but emergent complication.

Introduction

This case report describes a patient who suffered transient chest pain during strenuous exercise, and progressive dyspnea and hemoptysis later. Acute chordae rupture leading to heart failure was diagnosed after serial examinations.

Case report

A 45-year-old man presented to the emergency department because of progressive dyspnea after strenuous weight lifting and push-ups exercise 1 week ago. He was in excellent health and continued performing weight lifting and push-ups regularly. The exercise was discontinued 1 month ago when he began to suffer from gastroesophageal reflux disease. He restarted vigorous exercise 1 week ago, and during exercise he was attacked by transient precordial chest pain. The chest pain subsided spontaneously, but progressive dyspnea and hemoptysis developed during the week. He also complained of orthopnea. Tachycardia (130 beats/min) and desaturation (SaO2 = 92%) were noted when he arrived at the emergency room. His blood pressure was 136/70 mmHg. Physical examinations disclosed engorged jugular veins and grade IV/VI pansystolic murmur over apex. Rales were noted at bilateral lung fields. Neither fever nor peripheral edema was found. The patient’s electrocardiogram showed sinus tachycardia (Fig. 1). Chest radiograph showed cardiomegaly and
bilateral consolidation with pleural effusion (Fig. 2). Thoracentesis disclosed a transudate, and cardiac origin was impressed. The echocardiographic study revealed a ruptured posterior mitral leaflet (Fig. 3A). The color Doppler map showed the moderate–severe mitral regurgitation that was eccentric toward the anterior medial aspect of the left atrium. The anterior-medial eccentric flow implies valvular damage on the posterior-lateral site (Fig. 3B). Under the impression of posterior mitral leaflet chordae rupture with acute heart failure, the emergent mitral valve plasty was performed. P2 and P3 chordae rupture with leaflet prolapse were found at the mitral valve during the operation. Dilated left atrium and annulus were also noted. Follow-up chest radiograph showed bilateral clear lung fields (Fig. 4), and the patient was discharged in a stable condition 9 days after the operation. Echocardiography 2 months later showed only trace mitral regurgitation and good contractility of the left ventricle. During follow-up 6 months later, the patient was noted to have maintained the condition of his health.

Discussion

The impairment of any component (annulus, leaflets, chordae tendineae, and papillary muscle) of the mitral valve may result in mitral valve incompetence. Acute mitral regurgitation is usually caused by acute disruption of the restraining force structures. The strain forces exceeding the stretching threshold of the chordae result in rupture of the mitral chordae tendineae. In contrast with mild chordae rupture involving a single chorda, significant rupture simultaneously involves multiple chords and may cause severe acute mitral regurgitation. Severe rupture may be either immediate onset or a progressive process in which
minimal rupture precedes to a more extensive or even complete rupture.

Myxomatous degeneration, mitral valve prolapse, bacterial endocarditis, and rheumatic heart disease are the leading underlying causes of chordae tendineae rupture [1–3]. Acute mitral regurgitation resulting from rupture of chordae tendineae has also been reported in patients with blunt chest injury, pregnant women, and those who engage in vigorous exercises [4–7]. Spontaneous chordae rupture occurs occasionally, predominantly among men older than 50 years, and is rare in young adults and children [8]. The most common impaired site in patients with rheumatic mitral valve disease is the anterior leaflet. By contrast, the most common site of spontaneous chordae rupture is the posterior leaflet [4]. However, the mechanism of such a difference remains unclear.

Mild chordae rupture rarely causes hemodynamic change and requires neither intervention nor treatment; however, significant rupture is usually a life-threatening emergency requiring immediate surgical intervention. Patients with acute and symptomatic severe mitral regurgitation, patients with New York Heart Association functional class II, III, and IV symptoms with normal left ventricular function, and patients with symptomatic or asymptomatic severe mitral regurgitation with mild or moderate left ventricular dysfunction are considered to have indications for mitral valve surgery [9]. Compared with mitral valve replacement, chordae tendineae reconstruction with or without annuloplasty provides better outcome and life quality, and lower mortality and morbidity.

Severe mitral valve prolapse has been reported to be at increased risk for chordal rupture, whether spontaneous or induced by strenuous physical activity [5]. There were only a few case reports about exercise-related chordae rupture; therefore, there was no available literature report concerning the possible incidence and exercise mode in these patients. These reports showed that the exercises associated with chordae rupture included sit-ups, running, weight lifting, push-ups, and labor [4,5]. It seems that it is not the mode of exercise, but rather the extent of exercise, that is related to the possibility of producing chordae rupture. Guidelines from the Bethesda Conference on Cardiovascular Abnormalities in the Athlete have mentioned the theoretical risk that strenuous exercise may predispose to chordal rupture [10,11]. It would be reasonable to be alert to patients with mitral valve prolapse accompanied with the following presentation: history of syncope; disabling chest pain; complex ventricular arrhythmias, particularly if induced or worsened by exercise; significant mitral regurgitation; prolonged QT interval; Marfan’s syndrome; and family history of sudden death [12]. Because mitral valve prolapse may present in up to 10% of healthy individuals, primary care practitioners should recognize that certain patients with mitral valve prolapse who engage in athletics may be at increased risk for such a rare but emergent complication.
References


