Incidence of Ruptured Chordae Tendineae in the Mitral Valvular Prolapse Syndrome*  
An Echocardiographic Study  
P. A. N. Chandraratna, M.D.; and Wilbert Aronow, M.D., F.C.C.P.

Echocardiographic studies were performed on 190 consecutive patients with mitral valvular prolapse. All patients had either mid-systolic posterior motion of the mitral valve or holosystolic hammock-like movement of the valve in systole. Thirteen patients (7 percent) were noted to have ruptured chordae tendineae. In four patients, a combination of abnormalities was observed. Five patients had clinical and bacteriologic evidence of infective endocarditis, two of whom had severe intractable pulmonary edema consequent to acute mitral regurgitation which required mitral valvular replacement.

At surgery, one of these patients had ruptured chordae tendineae to both leaflets, and the other had chordal rupture of the posterior leaflet. The other patients probably had spontaneous rupture of the chordae tendineae. A spectrum of clinical findings was noted. Six patients had marked mitral regurgitation, while two had isolated systolic clicks. Thus, chordal rupture does not always result in severe hemodynamic deterioration. Serial echocardiographic studies will be of value in studying the natural history and progression of disease in patients with chordal rupture.

The frequent occurrence of mitral valvular prolapse and its benign clinical course in most patients are well established;1-3 however, some patients with this condition suffer serious sequelae such as infective endocarditis, malignant ventricular arrhythmias, and severe mitral regurgitation.4-6 Echocardiographic studies have proven to be useful in the diagnosis of mitral valvular prolapse and in the detection of ruptured chordae tendineae.4-6 Although the surgical literature has stressed the importance of chordal rupture in the genesis of severe mitral regurgitation in the floppy mitral valve syndrome, its incidence in an unselected group of patients with mitral valvular prolapse has not been determined.8,10 The purposes of this investigation are (1) to define the incidence of echocardiographically detectable ruptured chordae tendineae in patients with mitral valvular prolapse and (2) to study the influences of chordal rupture on the clinical course and auscultatory features associated with this entity.

Materials and Methods

Echocardiographic studies were performed on 190 consecutive patients with the mitral valvular prolapse syndrome.

*From the Division of Cardiology, University of Oklahoma Health Sciences Center, Oklahoma City; the Veterans Administration Hospital, Long Beach, Calif; and the University of California, Irvine. Manuscript received June 5; revision accepted August 11.

The patients were supine during the echocardiographic examination. A 2.25-MHz, 0.5-inch transducer with a 10-cm focus, an ultrasonic scope (Ekoline 20), and a multichannel recorder (Honeywell 1856 or Irex) were used. An M-mode sweep from the aortic root to the left ventricle was performed on each patient. Care was taken to avoid inferior angulation of the transducer during the mitral valvular recording.11 All patients had either mid-systolic posterior motion of the mitral valve or holosystolic hammock-like posterior motion of the valve in systole.

The echocardiographic criteria used for the diagnosis of chordal rupture were as follows: (1) chaotic diastolic flutter of the anterior mitral leaflet; (2) systolic fluttering of the mitral valve; (3) irregular diastolic fluttering of the posterior mitral leaflet; (4) anterior motion of the posterior mitral leaflet in early diastole; and (5) abnormal echoes in the left atrium during systole.7,8,12-14 Increased amplitude and rate of opening of the anterior mitral leaflet were not included in these criteria, because these abnormalities have been noted in patients with stretched chordae or aneurysmal mitral leaflets, without chordal rupture.12

Results

Thirteen (7 percent) of the 190 patients studied were noted to have echocardiographic evidence of ruptured chordae tendineae. The relevant clinical and echocardiographic data of these 13 patients are summarized in Table 1. None of the patients gave a history of trauma to the chest or a history of rheumatic fever. Five patients had clinical and bacteriologic evidence of infective endocarditis. Two of them developed severe mitral regurgitation and pulmonary edema, which necessitated mitral valvular replacement.
replacement (patients 4 and 9). Patient 4 had rupture of chordae tendineae to both mitral leaflets and vegetations on the valve. Patient 9 had rupture of multiple chordae supplying the posterior leaflet and flail posterior leaflet. The anterior leaflet was large and redundant, but its chordae were intact.

The symptoms of our patients varied from severe pulmonary edema to complete absence of symptoms. Two patients had pulmonary edema, four patients were asymptomatic, five had dyspnea on exertion, and three had atypical pain in the chest (one of whom also complained of dyspnea). The hearts of all of the patients were in sinus rhythm, and none had arrhythmias on the electrocardiogram at rest.

Six of the 13 patients had marked mitral regurgitation, as determined by the presence of a harsh holosystolic murmur, third heart sound, left ventricular enlargement, and left atrial enlargement (five patients). Three others had holosystolic murmurs without a third sound. Two patients had isolated midystolic clicks, one had an isolated late systolic murmur, and one subject had a short crescendo-decrescendo systolic murmur.

**Echocardiographic Findings**

The following abnormalities consistent with ruptured chordae tendineae were noted: (1) chaotic diastolic fluttering of the anterior mitral leaflet, nine patients; (2) systolic fluttering of the mitral valve, four patients; (3) irregular diastolic fluttering of the posterior leaflet, six patients, one of whom (patient 4) had surgical confirmation of ruptured chordae to the posterior leaflet; and (4) paradoxical anterior motion of the posterior leaflet in early diastole, two patients (patients 4 and 9), who had mitral valvular replacement. Rupture of chordae to the posterior leaflet was evident at operation in both patients. Abnormal systolic echoes in the left atrium were present in two. In four patients a combination of these abnormalities was observed.

The left atrium was enlarged (left atrium to aortic root ratio of more than 1.2) in five patients, and the left ventricle was enlarged in six patients. All of the patients with left ventricular dilatation demonstrated hyperdynamic motion of the interventricular septum and posterior wall of the left ventricle, consistent with left ventricular volume overload.

The echocardiogram of the mitral valve of patient 3 is illustrated in Figure 1. Mitral valvular prolapse and systolic flutter of the mitral valve are seen. Figure 2 is an echocardiogram of the mitral valve from the same patient, which was obtained with a slightly different angulation of the transducer. Flutter of the posterior leaflet, which could not be appreciated in Figure 1, is evident.

An M-mode sweep from patient 4 is depicted in Figure 3. Diastolic fluttering of the posterior leaflet and an abnormal echo that demonstrates fluttering in systole is seen in the left atrium. The echo in the left atrium could represent a flail portion of the posterior leaflet. Left ventricular dilatation and vigorous motion of the interventricular septum and left ventricular posterior wall are evident.

| Patient, Infective Endocarditis | Symptoms* | Physical Signs** | Diastolic Flutter† | Mitral Valve Systolic Flutter | Left Atrial Dimension, cm | Ratio of LA/AO§ Dimension, cm|| | Left Ventricular Dimension, cm|| |
|---|---|---|---|---|---|---|---|---|
| 1, M, 59 | + DOE | PSM; S₃ | - - | + - | - | 5.2 | 1.3 | 6.2 | 2.2 |
| 2, M, 46 | - | - | - | + | - | - | 2.7 | 1.0 | 4.9 | + | + |
| 3, F, 40 | + DOE; AP | PSM; S₃ | - - | + - | + | 3.1 | 1.1 | 4.0 | + | + |
| 4, M, 43 | + PE | PSM; S₃ | - - | + - | + | 3.1 | 1.1 | 4.0 | + | + |
| 5, M, 25 | - DOE | PSM; S₃ | - - | + - | + | 3.1 | 1.1 | 4.0 | + | + |
| 6, M, 25 | - | - | - | + | - | + | 2.4 | 0.9 | 4.2 | + | + |
| 7, M, 45 | - DOE | PSM; S₃ | - - | + - | + | 3.1 | 1.1 | 4.0 | + | + |
| 8, F, 43 | - | - | - | + | - | - | 2.2 | 1.1 | 4.9 | + | + |
| 9, M, 28 | + PE | PSM; S₃ | - - | + - | + | 3.1 | 1.1 | 4.0 | + | + |
| 10, F, 55 | - | - | - | + | - | - | 2.2 | 1.1 | 4.9 | + | + |
| 11, F, 19 | - | - | - | + | - | - | 2.2 | 1.1 | 4.9 | + | + |
| 12, F, 17 | - | - | - | + | - | - | 2.2 | 1.1 | 4.9 | + | + |
| 13, M, 51 | + DOE | PSM | - - | + - | + | 3.1 | 1.1 | 4.0 | + | + |

*DOE, Dyspnea on exertion; AP, atypical pain in chest; and PE, pulmonary edema.

**PSM, pansystolic murmur; S₃, third heart sound; MSC, midystolic click; LSM, late systolic murmur; and ESM, ejection (crescendo-decrescendo) systolic murmur.

†AMV, Anterior mitral leaflet; and PMV, posterior mitral leaflet.

‡DAM, Paradoxical early diastolic anterior motion of posterior leaflet.

§Ratio of left atrial dimension to aortic root dimension (upper limit of normal, 1.2).

||Upper limit of normal, 5.4 cm.
had surgical evidence of posterior chordal rupture, five others had flutter of the posterior leaflet); and (5) abnormal echoes in the left atrium demonstrating systolic flutter are occasionally seen in patients with chordal rupture. It should be noted that all patients with systolic fluttering of the mitral valve or paradoxical diastolic anterior motion of the valve or abnormal echoes in the left atrium had clinical evidence of marked mitral regurgitation. These echocardiographic signs are probably indicative of severe disorganization of the valve. Left ventricular enlargement with increased motion of the wall and excessive systolic posterior motion of the left atrial posterior wall, with or without left atrial enlargement, may be observed, depending on the severity and duration of mitral regurgitation.

Aortic regurgitation may cause fine, high-frequency diastolic flutter of the mitral leaflets. Chordal rupture produces irregular, low-frequency diastolic fluttering of the mitral valve, which is quite different from that seen in patients with aortic regurgitation.

Chordal rupture could be attributed to infective

**Discussion**

Ruptured chordae tendineae may cause the following abnormal echocardiographic patterns:7,8,15-14

1. Chaotic diastolic flutter of the anterior mitral leaflet may be seen in rupture of the chordae supplying this structure;7 (2) extensive chordal rupture to the posterior mitral leaflet produces paradoxical anterior motion of the posterior leaflet in early diastole;7,18 (3) systolic fluttering of the mitral valve may at times be noted in patients with ruptured chordae tendineae;14 (4) irregular fluttering of the posterior mitral leaflet may be seen with posterior chordal rupture (besides the patient who

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**Figure 1.** Echocardiogram of mitral valve (patient 3). Note midsystolic mitral valvar prolapse (vertical arrow) and systolic fluttering (F) of mitral valve. AMV, anterior mitral leaflet; and PMV, posterior mitral leaflet.

The echocardiogram of the mitral valve of patient 4 is shown in Figure 4. Irregular diastolic fluttering of the anterior mitral leaflet, paradoxical anterior motion of the posterior leaflet in early diastole, and mitral valvar prolapse are seen. Note that during systole, besides the portion of the valve that prolapses, another echo demonstrating erratic fluttering is seen. This abnormal echo probably arises from a flail part of the mitral valve. Thus, this patient's echocardiogram was suggestive of ruptured chordae tendineae to both leaflets and extensive disorganization of the valve, features which were confirmed at surgery.

**Figure 2.** Echocardiogram of mitral valve (patient 3), recorded with slightly different angulation of transducer. Flutter of posterior mitral leaflet (F) is evident. AMV, anterior mitral leaflet; and PMV, posterior mitral leaflet.
endocarditis in five of our patients. Indeed, all five patients with infective endocarditis had echocardiographic evidence of chordal rupture. This observation attests to the importance of antibiotic prophylaxis against endocarditis in patients with mitral valvular prolapse. There was no history of trauma to the chest or other precipitating factors which could have been responsible for ruptured chordae in the rest of our patients. These patients probably had spontaneous rupture of chordae tendineae. Pomerance observed three cases of ruptured chordae tendineae in a series of autopsies on 35 patients with “ballooning deformity of atrioventricular valves.” One of them had a staphylococcal vegetation, and the other two had no evidence of a current or previous bacterial infection of the valve.

We observed an incidence of 7 percent (13/190 patients) for ruptured chordae in our group. The sensitivity of echocardiographic studies in the diagnosis of chordal rupture is not known. Therefore, it is conceivable that our study underestimates the actual incidence of chordal rupture in patients with mitral valvular prolapse. A spectrum of clinical findings was noted in our group of patients. While six patients had physical signs compatible with severe mitral regurgitation (holosystolic murmur and third heart sound), two others had systolic clicks without systolic murmurs, which suggested that they had little or no mitral reflux. One of these patients underwent left ventricular angiographic studies and was noted to have mitral valvular prolapse with no mitral regurgitation (patient 2). The degree of mitral regurgitation that results from chordal rupture probably depends on the position of the ruptured chord, the thickness of the chord, and the number of chordae damaged.

Roberts and Perloff described the division of...
primary chordae arising from papillary muscular heads into secondary and tertiary chordae. Each primary chorda gave rise to about five tertiary chordae. Haller and Morrow observed that severe mitral regurgitation results when one or two primary chordae in dogs were ruptured by inserting fish-hooks into the left ventricle. In contrast, Perloff postulated that "rupture of a tertiary chord may pass unnoticed." It is conceivable that the patients in our study who had no evidence of mitral regurgitation had rupture of one or more tertiary chordae.

Lam and his associates proposed a different classification, whereby the chordae were named according to their mode of insertion as follows: (1) commissural chordae; (2) rough-zone chordae; (3) cleft chordae; and (4) basal chordae. Two of the rough-zone chordae to the anterior leaflet, which were thicker and larger than the others, were termed "strut chordae." Lam et al postulated that discontinuity of a strut chord might cause more marked mitral regurgitation than rupture of other types of chordae. The degree of hemodynamic deterioration that occurs depends on the severity of mitral regurgitation and the rapidity of its onset. For a given degree of mitral regurgitation, the more acute the onset, the greater the rise in left atrial pressure (because the left atrium is smaller and less compliant than in cases with a long history of mitral regurgitation); and, hence, the hemodynamic deterioration is more severe.

Chordal rupture may alter the auscultatory features in such a manner as to mask the characteristic physical signs of mitral valvular prolapse; for example, patient 12 had an ejection (crescendo-decrescendo) systolic murmur which was not holosystolic. It was not possible to make a clinical diagnosis of mitral valvular prolapse in this patient. Also, in those patients with holosystolic murmurs, the diagnosis of prolapse could not be made on clinical grounds.

Once myxomatous degeneration of the chordae and consequent chordal rupture occur, even if the initial hemodynamic deterioration is slight, it is conceivable that progressive chordal rupture may occur. Therefore, serial echocardiographic studies to detect further damage to the valve and changes in the sizes of chambers may be of value.

Unless a great deal of attention is paid to echocardiographic technique, some cases of chordal rupture may be missed. It is important to scan the entire mitral valve; for example, patient 4 had diastolic flutter of the anterior leaflet and posterior leaflet systolic flutter of the mitral valve, paradoxical anterior motion of the valve in diastole, and abnormal echoes in the left atrium. Each of these abnormalities was best demonstrated by a different angulation of the transducer. Figure 1 illustrates systolic flutter of the mitral valve in patient 3; with a slightly different angulation, flutter of the posterior leaflet (Fig 2), which was not demonstrated in Figure 1, was seen. Systolic flutter of the mitral valve may be overlooked if the echocardiogram is not recorded at 50 or 100 mm/sec.

Cooley and his associates reviewed the findings in 50 patients with the "floppy valve syndrome" who had undergone mitral valvular replacement. Nineteen of their patients (38 percent) had ruptured chordae tendineae, and four others had ruptured papillary muscles. Based on the clinical progress, their patients were divisible into three groups. The first group had myxomatous changes confined to the mitral leaflets, and they had a long history of cardiac murmurs and insidious onset of symptoms. The second group had a slowly progressive course until rupture of chordae (or papillary muscles) occurred, when exacerbation of their symptoms occurred. The third group was relatively asymptomatic until chordal rupture and consequent acute mitral regurgitation occurred. Thus, occurrence of chordal rupture is an important landmark in the natural history of patients with mitral valvular prolapse.

Mills and co-workers studied the natural history of mitral valvular prolapse in 53 patients and noted that five developed progressive mitral regurgitation. Two of their patients underwent mitral valvular replacement, and one of them was found to have ruptured chordae tendineae. It is possible that one or more of the other three patients had chordal rupture, but there was no evidence to document this. Although it has been thought that chordae of the posterior leaflet rupture more frequently, most series show that the two leaflets are affected more or less equally.

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