The Clinical Diagnosis and Surgical Management of Ruptured Mitral Chordae Tendineae

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Rupture of the mitral chordae tendineae is one of the less common causes of mitral insufficiency. In our experience, however, it is one form of this disease that is more readily corrected by surgery. The purpose of this paper is to describe six patients with mitral insufficiency secondary to ruptured chordae tendineae whom we have encountered over the preceding 18 months.

Case Reports

Case 1

A white shoe salesman first entered University Hospitals of Cleveland at age 61 stating that he had been rejected from the armed services 23 years previously and that 21 years ago life insurance had been refused because of a heart murmur. He denied having rheumatic fever or other febrile illnesses. His only memory of trauma was being thrown over the handlebars of his bicycle at age 12. From age 40 through age 55, he did well with an equivocal decrease in his exercise tolerance. Four years ago at another hospital cardiac catheterization revealed mitral insufficiency. Shortly thereafter he noted a rapidly progressive increase in dyspnea on exertion, orthopnea, pedal edema and ascites. Two years ago digi- toxin was begun with marked improvement in all of his symptoms. At the time of his admission to University Hospitals he again had increasing symptoms of cardiac decompensation.

His blood pressure was 140/80 and his pulse was 68 and irregular. A systolic thrill was present at the apex, along the left sternal border, and at the right second interspace. The heart was enlarged with a left ventricular heave. The rhythm was atrial fibrillation. There was a grade-III (basis of I to IV) medium-pitched pansystolic murmur at the apex which was transmitted to the axilla. In addition, a grade-II “diamond-shaped” harsh systolic murmur was heard at the right second interspace and was transmitted to the neck. There was a grade-I apical mid-diastolic rumble. Breath sounds were diminished at both bases. The liver edge was palpable 3 to 4 cm. below the right costal margin.

Cardiac fluoroscopy revealed a cardiothoracic ratio of 24.2/31. The enlargement was thought to be both left and right ventricular with marked left atrial enlargement. The electrocardiogram showed atrial fibrillation, right axis deviation, left ventricular hypertrophy, digitalis effect, and frequent premature ventricular beats.

On the rigorous program the patient lost 12 Kg., and was symptomatically improved. He was discharged but returned 2 months later for surgery. His clinical status on return remained unchanged, although his heart size was increased by x-ray (fig. 1).

The chest was entered through a midline sternal incision, and extracorporeal circulation was instituted (as it was in all cases in this report). A huge left atrium with enlargement of both the left and right ventricles was noted. No thrill was felt over the aorta itself, but there was a transmitted systolic thrill from the huge left atrium adjacent to the base of the aorta. A catheter inserted into the left internal mammary artery disclosed a rapid systolic upsweep with a good dicrotic notch. Left ventricular pressure tracings were recorded simultaneously and disclosed no gradient across the aortic valve (fig. 2). Left atrial pressures were elevated with a prominent “V” wave. The mitral ring was dilated and both leaflets appeared to have redundant valvular tissue. There was marked upward ballooning of the posterior leaflet. In the midportion of this leaflet there were two ruptured chordae tendineae. The leading edge of the valve in this region was sutured back down to the papillary muscles in the left ventricle and the valve ring was foreshortened by inserting horizontal mattress sutures in the region of the anterior and posterior commissures. This resulted in complete competency of the valve. The left atrial pressure was measured at the end of the procedure, and, although the mean pressure was somewhat higher than before surgery, there was no longer a peak “V” wave.
Postoperatively the patient did extremely well. At time of discharge 12 days later his apical murmur was grade I and localized to the apex. No thrill or systolic murmur could be heard over the right second interspace, and the second sound in this area was normal. Three months later the patient was free from symptoms of congestive heart failure and there was a marked reduction in heart size by x-ray. A grade-I apical systolic murmur was barely audible.

Case 2
A 28-year-old man had rheumatic fever at age 6. He did well until age 22 when he was hospitalized for 75 days with febrile illness that was treated with penicillin and sulfonamides. He then had moderate exertional dyspnea, but no other symptoms of congestive heart failure. At age 23 he was operated upon elsewhere for mitral valve disease. However, he became hypotensive at the time of the chest incision and the

Figure 1
X-rays, case 1 (upper) and case 2 (lower), before and 3 months after surgery. Note grossly enlarged left atrium.
operation was terminated. Shortly thereafter, he first developed paroxysmal nocturnal dyspnea and ankle edema. He responded to digitalis and diuretic therapy and did well until age 28, when his congestive failure recurred.

The patient was a thin, pale white man. The neck veins were distended at 90°. There was a diffuse left ventricular impulse with a localized apical systolic thrill. The left heart border extended to the midaxillary line. The rhythm was atrial fibrillation. A grade-III pansystolic murmur was present at the apex and extended over the entire precordium and to the axilla. In addition, a grade-II "diamond-shaped" systolic murmur was heard over the aortic area and was transmitted to the neck. There was a grade-I diastolic apical rumble and a questionable grade-I decrescendo high-pitched diastolic murmur along the left sternal border (fig. 3).

The electrocardiogram revealed atrial fibrillation, a mean QRS axis of +100°, left ventricular hypertrophy, and digitalis effect. Cardiac fluoroscopy disclosed a massively enlarged left heart border that depressed the left

![Figure 2](image-url)

*Figure 2*

Left atrial pressure tracings of case 1 (upper) and case 3 (lower) before (left) and after (right) surgery. Left atrial pressure curve is lowermost on graph. The ordinate scale does not apply to the middle tracings, which are simultaneous aortic curves.
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Figure 3

Phonocardiogram (case 2) demonstrating “diamond-shaped” systolic murmur at the right second inter-space with plateau-type murmur at apex.

leaf of the diaphragm. The left atrium was markedly enlarged. Cardiac catheterization pressures included wedge 17 mm., pulmonary artery 30/15, right ventricle 30/5, left ventricle 100/2-14 and aorta 100/80 (fig. 4). The cardiac output was 3.8 L./min. Angiocardiography revealed no aortic regurgitation on injection into the aortic root.

The chest was opened through the right fourth intercostal space. The left atrium was huge; over it a distinct vibration was felt. A needle inserted into the atrium revealed a “V” wave pressure of 25 mm. with a “Z” point pressure of 5 mm. When the left atrium was opened, both leaflets of the mitral valve ballooned upward; although thickened, they were free from calcium. The mitral ring was grossly dilated. There were two centrally placed chordae tendineae of the anterior mitral leaflet that had ruptured. Because of the size of the left ventricle it was impossible to reattach the chordae to the papillary muscle. Figure-eight stitches were placed in the ring in the region of both the anterior and posterior commissures. There was marked improvement in the regurgitant stream and the valve admitted two fingers.

Postoperatively a grade-I pansystolic apical murmur and a grade-I aortic systolic murmur, which did not radiate into the neck, were present. The patient was treated for congestive failure and discharged with a marked reduction in symptoms. Three months later he continued to show improvement.

Case 3

A 50-year-old white woman was asymptomatic until age 49, when she gradually developed cough, paroxysmal nocturnal dyspnea, and dyspnea on exertion. At this time treatment was started for congestive heart failure with improvement. She denied any history of rheumatic fever, severe illnesses, or recent trauma. She did, however, recall being in an automobile accident at the age of 23, when she received a severe blow to her chest.

The blood pressure was 160/90 and the pulse was 100. The heart was clinically enlarged, with a left ventricular heave. A systolic thrill was present at the apex and at the right second interspace. There was a grade-III pansystolic apical murmur that radiated to the axilla and along the lower left sternal border. In addition a grade-I diastolic rumble was present at the apex. There was a grade-II harsh “diamond-shaped” systolic murmur at the right second intercostal space, which radiated into the neck.

Venous pressure, circulation time, and vital capacity were all within normal limits. The electrocardiogram disclosed a mean electrical axis of +70° and left ventricular hypertrophy. Cardiac fluoroscopy revealed enlargement of both right and left ventricles, and marked left atrial enlargement. The cardiothoracic ratio was 15.5/26.

The chest was entered through a midline sternum-splitting incision. A catheter inserted into the left internal mammary artery revealed a normal aortic pressure curve with a rapid ascent and a distinct incisura. The left atrium was markedly enlarged; a systolic thrill was palpable over it in the region of the base of the aorta. Left ventricular pressures were recorded simultaneously with aortic pressures and revealed no gradient across the aortic valve. The left
atrial pressure disclosed a huge "V" wave with peak pressures reaching 40 mm Hg. The valve ring was markedly dilated with stretching of the posterior mitral leaflet. This leaflet ballooned upward but was free from calcium and there was no gross evidence of rheumatic valvulitis. There was, however, rupture of several centrally placed chordae tendineae of the posterior leaflet. A suture was placed in the leading edge of this leaflet in the area of the rupture and inserted into a papillary muscle of the left ventricle. In addition, one suture was inserted in the region of the posterior commissure approximating the ring in this region. Following correction of the deformity, left atrial pressures were again obtained. The "V" wave had disappeared and the mean pressure was 8 mm. No thrill could be palpated over the apex.

Postoperatively the patient did well. A grade-II mid-frequency systolic murmur persisted at the apex. There was no thrill, however, and no murmur was audible at the right second interspace.

Shortly following discharge the patient again developed symptoms and signs of congestive heart failure, and was noted to have a grade-III apical systolic murmur audible over the entire precordium. Reoperation disclosed that the previously placed suture in the papillary muscle had stretched and the valve was again insufficient. In addition, the one horizontal mattress stitch used to approximate the ring had pulled loose from the posterior portion of the ring. The valve was again repaired. Postoperatively, the patient showed signs of cerebral embolization. Despite therapy she died on the fifth postoperative day. No autopsy was obtained.

**Case 4**

A 40-year-old white man had acute rheumatic fever at age 11 and was told at that time of a heart murmur. He had subsequent attacks of migratory arthritis of a similar nature at ages 13 and 15. He was asymptomatic until age 23 when he developed bacterial endocarditis and was treated successfully at another hospital. At age 29 he had a repeated bout of bacterial endocarditis complicated by a ruptured cerebral mycotic aneurysm and focal glomerulonephritis. Treatment with antibiotics was successful, and he left the hospital without sequelae. No changes were noted in his cardiovascular examination at this time. Three months later he first developed dyspnea on exertion, ascites, and pedal edema. He responded well to treatment for congestive heart failure.

The heart was enlarged with a localized apical systolic thrill. There was a grade-III pansystolic medium-pitched apical systolic murmur that radiated to the axilla. A harsher, earlier grade-II aortic systolic murmur was also noted; it radiated to both clavicles. A grade-I diastolic apical rumble and a questionable left sternal border diastolic blow were audible.

The electrocardiogram disclosed atrial fibrillation, a mean QRS axis of +70° and left ventricular hypertrophy. Cardiac fluoroscopy revealed left ventricular and possible right ventricular enlargement, left atrial enlargement, and a prominent pulmonary outflow tract.

![Figure 4](http://circ.ahajournals.org/)

Left ventricular to aortic pullback pressure tracing (case 2) demonstrating no aortic gradient.

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At surgery a large left atrium was noted, and there was marked mitral regurgitation. The valve was minimally thickened but without calcium or commissural fusion. Three chordae tendineae of the anterior mitral leaflet were ruptured. Mattress sutures were placed in the free chordae, and they were brought into direct contact with the papillary muscles. The leak was almost completely corrected by this procedure.

Postoperatively, the patient experienced renal failure, but following peritoneal dialysis he made a good recovery. At the time of discharge, 1 month later, his cardiac symptoms were improved. There was a grade-I apical systolic murmur along the left sternal border but not in the aortic area.

**Case 5**

A 35-year-old Negro woman was hospitalized at age 9 with migratory polyarthritis diagnosed as acute rheumatic fever. She was rehospitalized with “fever and a heart murmur” for 5 months at the age of 15. At age 34 she had increasing dyspnea of 3 months’ duration, and periodic fever, weight loss, pain in her fingertips, and left upper quadrant pain of about 2 months’ duration.

The blood pressure was 140/80. There was a 2 by 2 mm. hemorrhage of the right retina. The heart was enlarged to the anterior axillary line with a left ventricular heave and a systolic thrill at the apex. A grade-III pansystolic apical murmur that radiated to the axilla was audible. A grade-I + diastolic rumble and a loud third sound were present at the apex. A tender spleen tip was palpable.

Repeated blood cultures revealed *Beta hemolytic streptococcus.* The electrocardiogram disclosed a regular sinus rhythm, a mean QRS axis of +40°, left ventricular hypertrophy, left atrial hypertrophy, and digitalis effect. Left ventricular and left atrial enlargement were also noted on cardiac fluoroscopy.

The patient was treated with penicillin, streptomycin, and digitalis. On the twelfth hospital day she developed a left hemiplegia considered secondary to a septic embolus. She improved rapidly, however, and was discharged on digitalis but with no clinical evidence of congestive heart failure. She returned 6 months later for surgical repair of her mitral valve. Her cardiac examination at that time remained unchanged.

The chest was entered through the right fourth intercostal space. The left atrium was large and tense, and pulsed with each ventricular systole. The “V” wave measured 40 mm. Hg with a “Z point” pressure of 10 mm. Two central chordae tendineae of the anterior leaflet of the mitral valve were found to be ruptured. There was some fusion of the commissures. The valve leaflets were minimally thickened. The commissures were split, and the insufficiency was repaired with placement of one stitch in the region of the anterior commissure, thereby narrowing the ring in this area. Two similar sutures were placed in the ring near the posterior commissure. Following closure of the atrium the “V” wave was reduced to 15 mm. but the “Z point” pressure remained elevated.

Postoperatively the patient did well. Only a grade-I apical systolic murmur could be heard at the time of discharge. Follow-up x-rays 2 months later revealed reduction in heart size with no signs of congestive failure.

**Case 6**

A 39-year-old Negro woman had a prolonged febrile illness at age 6. At age 33 she first noted increasing fatigue and minimal pedal edema and was told at this time of a heart murmur. She responded well to digitalis until age 35, when she again noted a progressive decrease in her exercise tolerance. At time of admission she described one-half block dyspnea, two-to-three-pillow orthopnea, and increasing peripheral edema.

Examination revealed atrial fibrillation with an average ventricular rate of 48. There was a left ventricular impulse at the anterior axillary line. A localized apical systolic thrill was felt. The first sound was replaced by a systolic murmur. There was a grade-II + plateau pansystolic apical murmur that radiated to the axilla. A grade-I apical diastolic rumble was present.

Cardiac fluoroscopy disclosed enlargement of both right and left ventricles with a cardiothoracic ratio of 15.5/24.5. There was considerable left atrial enlargement. The electrocardiogram revealed a mean QRS axis of +40° atrial fibrillation, left ventricular hypertrophy, and digitalis effect.

The chest was opened through a submammary right frontal intercostal space incision. Left atrial pressures revealed a “V” wave of 20 mm. Palpation of the mitral valve revealed some fusion of both commissures and a forceful regurgitant stream. Direct inspection of the valve revealed that two chordae of the anterior leaflet had ruptured. Sutures were used to reattach the chordae to the underlying papillary muscle of the left ventricle. The commissures were then opened for a distance of a few millimeters. There was definite thickening of both leaflets with rolling down of the posterior leaflet. One horizontal mattress stitch supported by a patch of Teflon was placed in the region of the posterior commissure and corrected the insufficiency.

Postoperatively the patient’s course was un-
eventful. A grade-II apical systolic murmur persisted at the apex. At the time of discharge there were no clinical signs of congestive heart failure. Three and 6 months later the patient remained free from cardiac symptoms and her heart size had diminished by x-ray.

**Discussion**

Mitrail insufficiency resulting from rupture of one or more chordae tendineae is a relatively uncommon entity. Osmundson et al. found only 20 cases over a 24-year experience at the Mayo Clinic. The occurrence of six cases in an 18-month period in our series appears to be unusually high. This apparent increase in incidence may in part result from the advances in cardiovascular surgery and the recognition of this entity at the operating table.

Certain clinical guides have previously been described that aid in the diagnosis of ruptured mitral chordae. Although there is some variation in the six patients reported herein, there are certain historical features and physical signs that seem characteristic.

The etiology in rupture of the mitral chordae tendineae may vary. Bacterial endocarditis has been implicated as the most common cause, although rheumatic endocarditis and trauma have also been considered significant factors. In certain previously reported cases no definitive etiology could be determined. Clinically, both rheumatic fever and bacterial endocarditis were conclusively noted in cases 2, 4, and 5 of the preceding report. Anatomically, aside from the ruptured chordae, there was no gross evidence, such as leaflet vegetation or ulceration to indicate preceding bacterial endocarditis in any of these. It has previously been pointed out, however, that following bacterial endocarditis residual changes in the valve leaflet may be of a minor nature and rupture of the mitral chordae may represent the dominant lesion. In addition, it is of interest that two of these three patients demonstrated little anatomic evidence of residual rheumatic valvulitis. They disclosed some thickening of the valve tissue and the affected leaflet ballooned upward, but there was no commissural fusion, shortening and thickening of the chordae, or other features of an old rheumatic process. A single patient (case 6) disclosed historical as well as anatomic evidence of a rheumatic infection without satisfactorily documented bacterial endocarditis.

In two of the preceding six instances no etiologic explanation was apparent. Both patients gave a history of chest trauma but in each it preceded the onset of clinical heart disease by so long a period of time that it would seem an unlikely cause. The absence of any clinical evidence and gross pathologic changes suggest that these two cases might be included among those previously described in which there was no satisfactory etiologic explanation. Microscopic confirmation was impossible, however, so that residual abnormalities from an inflammatory process may still have been present.

Previous observations suggest that worsening of symptoms or the sudden onset of congestive heart failure may occur with, or shortly following rupture of the chordae tendineae. Cardiac symptoms increased in our three patients who had documented endocarditis. In one instance cardiac decompensation was first noted during the acute illness. In the second, symptoms were evident within 3 months. There was a distinct reduction in exercise tolerance in the third patient immediately following the endocarditis, although congestive failure was not diagnosed for over 1 year.

The abrupt onset of a systolic murmur also is considered helpful in establishing the diagnosis of rupture of the mitral chordae tendineae. The patients with bacterial endocarditis in our series all had systolic murmurs when they were first seen by us, and there were no changes in the auscultatory findings during the course of their acute illness. It seems reasonable that the sudden onset of a murmur would be consistent with chordae rupture and it may be that in our patients this occurred preceding hospital admission.

Several authors think that there is an anatomic basis for the characteristics of the murmur in patients with ruptured chordae tendineae. Osmundson et al. were able to identify a definite left atrial "jet lesion" in all
of their cases involving chordae of the posterior leaflet. In six of these a basal systolic murmur simulating that of aortic stenosis had been recorded during life. These authors suggest that the involved posterior leaflet arched upward, so that its free edge did not oppose that of its anterior counterpart. As a result the regurgitant stream deflected off the outer portion of the insufficient leaflet striking the anterior septal wall of the left atrium. Thus, the transmission of the murmur to the base and the location of the jet lesion in this area are explained. In two of their cases as well as in other reports, however, basal systolic murmurs were noted in rupture of the anterior mitral leaflet.9,10 No reason for this was readily apparent. Two of our four preceding cases demonstrated a definite aortic systolic thrill, while in four a “diamond-shaped” systolic murmur was audible at the right second interspace. In all of these, a distinct vibration was palpable over the upper aspect of the left atrium at the time of surgery, suggesting that the aortic murmur and thrill resulted from the regurgitant stream striking the atrial wall. The lack of any aortic gradient coupled with the loss of the thrill and disappearance or marked reduction in the aortic murmur postoperatively indicates that the mitral insufficiency was, in fact, the sole cause of the auscultatory findings. Only two of our four patients proved to have rupture of the posterior leaflet, whereas in two the anterior leaflet was involved. No distinct jet lesion was obvious at the time of surgery, although careful inspection of the atrial wall was not performed. The aortic systolic thrill was clinically palpable in the two patients in whom the chordae of the posterior leaflet were involved, whereas none was evident with rupture of the anterior chordae. The thrill was most likely present in the former group because the regurgitant jet struck the anterior septal part of the atrium in the area of aortic outflow. In the latter group the jet flow was probably directed more posteriorly, and, although in close enough proximity to the aortic root to produce a murmur, it was not sufficiently adjacent to produce a palpable vibration. There was no thrill over the upper portion of the atrium at surgery in the two patients who failed to demonstrate an aortic murmur. This suggests that the regurgitant jet in this instance was directed far enough posteriorly so as not to transmit noise to the base of the heart.

Although patients with mitral insufficiency from any cause can have left atrial dilatation, we have been impressed with the extreme atrial enlargement in the six preceding patients with mitral insufficiency secondary to ruptured mitral chordae. The exact pathogenesis of the giant left atrium is not clear. Rogers and Wittels11 suggested that marked atrial dilatation might result from a high-speed regurgitant jet causing “post-stenotic dilatation in reverse.” This interpretation could apply to the patient with ruptured chordae in which there might be a more direct, narrow regurgitant stream. This jet stream would strike the atrial wall with great impact, thus producing dilatation as well as the “diamond-shaped” aortic murmur which seems so characteristic of this entity.

Our experience suggests that patients with mitral insufficiency secondary to ruptured chordae tendineae are good candidates for surgical correction. When possible the flail leaflet should be reattached to the papillary muscle of the involved chordae tendineae. In addition, one or more well-supported sutures should be inserted to reduce the size of the valve ring in order to assure competence and to reduce the amount of stress applied to the reattached leaflet. Case 3 illustrates this need rather clearly in that failure to maintain reduction in the size of the valve ring resulted in recurrence of the insufficiency and breakdown of the repair of the detached chordae.

If desired, the enlarged leaflets can be excised and an artificial valve inserted. With a markedly dilated ring and increased ventricular volume routinely present in these cases, insertion of a valve prosthesis should be technically easy to accomplish. At this time, however, we prefer to repair this type of lesion with the patient’s own excess, pliable valve tissue. The results in this series of patients would confirm the efficacy and desirability of this approach, since there was only
one technical failure. In addition, we feel less concern about embolization and recurrent blood stream infection than we would with artificial valve replacement.

Previous experiences as well as our own indicate that a preceding history of bacterial endocarditis following which symptoms of heart failure may have become apparent; a "diamond-shaped" systolic murmur at the right second interspace in the presence of a loud pansystolic apical murmur; and a markedly enlarged left atrium should make one consider the diagnosis of ruptured mitral chordae tendineae, since this disease appears to be particularly amenable to surgical correction.

Summary

Six patients with mitral insufficiency secondary to ruptured chordae tendineae are presented. Each patient underwent surgical correction of the deformed valve. In two instances the posterior leaflet was involved while in four the anterior leaflet was affected. Three patients had definite bacterial endocarditis that appeared to be related to the onset of cardiac symptoms. In the two instances of posterior valve involvement there were an aortic thrill and a harsh systolic murmur, whereas in two of the four with rupture of the anterior leaflet an aortic systolic murmur was heard but no thrill was felt. The other two with anterior leaflet disease disclosed no murmurs in the aortic area. These findings are considered to be a result of the direction of the regurgitant jet and the area in which they strike the left atrium. None of the patients had hemodynamic evidence of aortic valve disease and in each instance the aortic systolic murmur decreased or disappeared after surgery. Each patient had gross left atrial enlargement. Although relatively uncommon, mitral insufficiency due to rupture of the mitral chordae tendineae is readily corrected surgically.

References

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