Echocardiographic Diagnosis of Mitral Regurgitation due to Ruptured Chordae Tendineae

By Thomas Sweatman, M.D., Arthur Selzer, M.D., Masanobu Kamagaki, M.D., and Keith Cohn, M.D.

With the technical assistance of Mrs. Judith Basford

SUMMARY

The echocardiographic findings from six patients with ruptured chordae tendineae from the posterial mitral valve leaflet and three with ruptured chordae from the anterior leaflet are reported; in all cases the diagnosis was later proven by open-heart surgery. The constellation of echocardiographic findings suggesting ruptured chordae tendineae to the posterior leaflet are: (1) The appearance of echoes within the left atrial chamber, representing prolapse of the posterior leaflet and flail chordae tendineae. This finding, although not consistently observed, is the most pathognomonic echocardiographic sign. (2) Increased amplitude of systolic excursion of the left atrial wall, in most cases associated with normal or only mildly increased left atrial diameter. (3) Recognition of thin, low-intensity (noncalcified) mitral valve leaflets. (4) The anterior leaflet may also manifest increased amplitude of motion, and may not coapt normally with the posterior leaflet in systole. (5) Increased amplitude of motion of the interventricular septum with an increased estimated stroke volume and normal-to-increased ejection fraction.

The findings suggestive of ruptured chordae tendineae to the anterior leaflet are: (1) A markedly increased amplitude of motion of the anterior mitral valve leaflet is evident. This leaflet appears to approach or touch the left ventricular septum in systole and the left atrial wall in diastole. (2) All of the findings of ruptured chordae to the posterior leaflet are also seen here, except for the observation of the posterior leaflet within the left atrial chamber.

This constellation of findings has not been seen in normal individuals, nor is it present in patients with mitral insufficiency proven to be due to other factors, such as papillary muscle dysfunction or rheumatic heart disease. Hence, this noninvasive technique enables one to diagnose accurately the presence of mitral regurgitation due to ruptured chordae tendineae.

Additional Indexing Words:
Rheumatic heart disease Cardiac surgery Ultrasound Angiography Ejection fraction Left ventricular stroke volume Valve leaflets

In recent years, cardiologists have come to appreciate that ruptured chordae tendineae represent one of the more common forms of mitral regurgitation.1, 2 Although several clinical characteristics, such as unusual radiation of the murmur, fourth heart sound, and a small left atrium, suggest the possibility

From the Division of Cardiology, Presbyterian Hospital, Pacific Medical Center, and the Heart Research Institute, the Institute of Medical Sciences, San Francisco, California.

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Address for reprints: Dr. Keith Cohn, Cardiology Unit, Pacific Medical Center, Clay and Webster Streets, San Francisco, California 94115.

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580 Circulation, Volume XLVI, September 1972
RUPTURED CHORDAE TENDINEAE

of this diagnosis, definitive recognition of ruptured chordae often awaits direct surgical or pathologic inspection of the valve apparatus.

The purpose of this study is to describe the echocardiographic findings from nine patients with mitral insufficiency due to ruptured chordae tendineae. It is evident from the findings reported here that this noninvasive technic enables one to identify this entity with a high degree of accuracy, and to distinguish it from other forms of mitral regurgitation.

Methods

All nine of the patients included in this report were later proven during open-heart surgery to have ruptured chordae tendineae. In six, the ruptured chordae had been attached to the posterior leaflet of the mitral valve, and it was that leaflet that was prolapsing. In three patients, the chordae from the anterior leaflet had ruptured. All patients had right and left heart catheterization, eight of the nine had left ventricular cineangiography performed in the right anterior oblique view, and all had been proven to have severe mitral regurgitation, necessitating surgical correction.

The ultrasound records were obtained from a Sperry reflectoscope utilizing a 2-MHz transducer, 13 mm in diameter. The recordings were made both by taking Polaroid photographs from a cathode-ray oscilloscope and from an Electronics for Medicine DR-8 strip-chart recorder.

The patients were studied in the supine position with the transducer placed in the third or fourth intercostal space just at, or 1 to 2 cm from, the left sternal border. A gel was placed between the skin and transducer to assure proper contact and to avoid artifacts due to air-skin interface. From this location, the transducer (and therefore echo beam) can be aimed in a variety of directions. When directed superiomedially, as in lines 1 and 2 of figure 1, the beam reflects off the aorta, passes through the left atrial chamber, and then reflects from the posterior left atrial wall. When directed more posteriorly and only slightly superiorly, as in line 3, echoes from the anterior leaflet of the mitral valve and either the mitral annulus or the left atrium are obtained. When directed inferiorly and laterally, as in line 4, the beam will reflect off the ventricular septum, pass through the left ventricular cavity, and reflect off the posterior wall of the left ventricle.

The left atrial diameter was measured at the end of ventricular systole as described by Hirata. The movement of the posterior left atrial wall was measured during ventricular systole and ventricular diastole (before atrial contraction), and this difference was taken as the amplitude of motion of the left atrial wall. Left ventricular dimensions and estimated stroke volume were measured just below the mitral valve as described by Feigebaum et al. The width and relative density of the anterior mitral leaflet were estimated by varying the sensitivity setting and comparing the intensity of the echo from the anterior leaflet with the intensity recorded from other structures. All of the measured values obtained from the patients with ruptured chordae tendineae were compared with findings from 30 normal subjects. We have studied 85 patients with other forms of mitral insufficiency with ultrasound; although the details of the echocardiographic findings in this group of patients are too extensive to be included in this paper, comparative and contrasting features will be commented upon when applicable.

Results

Ruptured Chordae Tendineae to the Posterior Cusp

The findings are best presented by referring to the direction of the echo beam in figures 1–5. Figure 2 illustrates the echocardiographic findings when the beam is passed in the direction of line 1, that is, passing through the ascending aorta and into the left atrial chamber. Within the left atrium, echoes, apparently from the flail posterior mitral valve leaflet or torn chordae, are seen. That these are not artifactual, produced by scattering of
The upper left portion of the figure shows a diagrammatic representation of the echo beam passing through the aorta, into the left atrial chamber, reflecting off the flail posterior mitral valve leaflet. The upper right portion of the figure shows the actual echocardiographic recording, with a diagrammatic representation of this at the lower right. The posterior leaflet is seen within the left atrial chamber, moving posteriorly during systole, approaching the left atrial wall.

The ultrasound waves, is evidenced by their persistence when the gain control is lowered, such that the posterior atrial wall nearly disappears. Similar disconnected "dots" of echoes are also seen within the left ventricle in normal individuals, when one aims through the midportion of the left ventricular chamber, reflecting off the margin of the mitral leaflet and off the chordae tendineae. In normal individuals, there is no visible echo within the left atrial chamber.

If the beam is aimed along line 2 (figs. 1 and 3), it again passes through the aorta and bounces off the posterior-medial aspect of the left atrial wall. In normal individuals, the left atrial wall moves posteriorly during systole only a slight degree, usually less than 3 mm. In all of the patients with ruptured chordae tendineae, there was a marked outward

Figure 2

The echo beam passes through the aortic root, and reflects off the posterior-medial aspect of the left atrial wall. The actual echocardiogram in the upper right, and diagram of this in the lower right with a magnified view, show marked posterior systolic motion of the left atrial wall.

Figure 3

The posterior leaflet does not coapt normally with the anterior leaflet but, rather, approaches it only momentarily during systole and then separates from it, moving posteriorly toward the left atrial chamber.

Figure 4
RUPTURED CHORDAE TENDINEAE

Phono-
EKG

Ventricular Septum

Posterior Wall, L.V.

Figure 5

(Left) When the beam is aimed along direction 4, it passes through the ventricular septum (a) and reflects off the posterior wall of the left ventricle (b). Here, the echocardiographic findings in normal individuals are contrasted in diagrammatic form with those in patients with ruptured chordae tendineae (RCT). The posterior wall moves with a normal amplitude in ruptured chordae tendineae, although the septal motion is increased in magnitude. (Right) An actual echocardiographic tracing is shown from a patient with ruptured chordae tendineae, showing increased septal motion.

Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Range</th>
<th>Normal Mean</th>
<th>Ruptured chordae tendineae Range</th>
<th>Ruptured chordae tendineae Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left atrial wall excursion (mm)</td>
<td>2-4</td>
<td>3</td>
<td>7-8</td>
<td>8</td>
</tr>
<tr>
<td>Left atrial diameter (cm/m²)</td>
<td>1.2-2.0</td>
<td>1.5</td>
<td>1.8-3.3</td>
<td>2.6</td>
</tr>
<tr>
<td>Left ventricular stroke volume (ml)</td>
<td>50-87</td>
<td>72</td>
<td>112-415</td>
<td>210</td>
</tr>
<tr>
<td>Ventricular septal motion (mm)</td>
<td>4-8</td>
<td>6</td>
<td>8-25</td>
<td>15</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.34-0.68</td>
<td>0.47</td>
<td>0.52-0.77</td>
<td>0.62</td>
</tr>
</tbody>
</table>

*Circulation, Volume XLVI, September 1972*
posterior wall, enabling estimations of the left ventricular size. By comparing the normal on the left with the ruptured chordae tendineae on the right (fig. 5), one can see that the posterior wall moves with normal amplitude in ruptured chordae tendineae although the septal motion is markedly augmented in this form of mitral regurgitation. Thus, table 1 reveals that the septal wall motion averages 15 mm, this resulting in an increased estimated stroke volume averaging 210 mm/m² and an ejection fraction which is within normal limits, or slightly increased.

By varying the sensitivity control of the apparatus, one can roughly gauge thickness and density of a particular structure. The normal mitral valve is found to be very thin, whereas in rheumatic mitral regurgitation the valve is usually thickened. An increase in the intensity of the echo, suggesting a more dense structure, is also seen when the valve leaflet is calcified. In our cases of ruptured chordae tendineae the valve leaflets appear to be thin and the echo is of low intensity, suggesting a noncalcified structure.

**Ruptured Chordae Tendineae from the Anterior Leaflet of the Mitral Valve**

In this entity, many of the findings were similar to those of ruptured chordae tendineae to the posterior leaflet. An increased systolic expulsive motion of the left atrium, increased left ventricular septal motion, high stroke volume, normal ejection fraction, and thin, low-intensity mitral valve leaflet were noted. No posterior leaflet was seen within the left atrial chamber during systole. There were, in addition, findings considered distinctive for anterior mitral leaflet prolapse. Most prominent among these was a marked increase in the amplitude of motion of the anterior mitral valve leaflet, seen in all three cases. The anterior valve leaflet can be shown to approach, or strike, the septum in diastole, and to approach the left atrial wall in systole (fig. 6). A marked increase in amplitude of motion of the mitral valve leaflet has also been seen in other patients we have studied: those with stretched chordae tendineae and those with aneurysmal leaflets, as seen in Marfan's syndrome. In the patients with ruptured chordae tendineae to the posterior leaflet, the anterior cusp moves with a normal or increased amplitude.

**Discussion**

Recent observations have indicated that nonrheumatic forms of mitral regurgitation occur more frequently than previously believed, the ratio of rheumatic to nonrheumatic cases of mitral regurgitation being estimated at 39 to 61 in a current study. Isolated rupture of chordae tendineae was found to be the second most common form of mitral regurgitation—next to rheumatic—with an incidence of 19%. This well-defined clinical syndrome shows a preferential involvement of chordae attached to the posterior leaflet (91%); the prolapse of that leaflet directs the regurgitant jet toward the septal wall of the atrium, adjacent to the aortic root, producing a systolic murmur in the “aortic and pulmonic” areas of the precordium. Clinical findings often provide sufficient clues to establish the
RUPTURED CHORDAE TENDINEAE

585
diagnosis of this disease entity, but the differentiation from aortic stenosis and other types of mitral regurgitation may be difficult, requiring cardiac catheterization, angiography, and even surgery to clarify the diagnosis.

The availability of echocardiographic examination (a noninvasive technic) as a reliable means to recognize prolapse of the posterior leaflet of the mitral valve due to rupture of chordae tendineae adds a diagnostic tool of paramount importance. The most pathognomonic finding was the posterior leaflet-chordal echoes detected within the left atrium, moving toward the left atrial wall during systole. The other echocardiographic abnormalities in patients with ruptured posterior leaflet chordae aid in identification and in estimation of the severity of mitral regurgitation, and it is the constellation of findings that allows one to make a fairly definitive diagnosis of ruptured chordae tendineae in the majority of cases. The increased amplitude of systolic excursion of the left atrial wall is felt to be due to blood regurgitating into the left atrial chamber during systole. Increased systolic excursion of the left atrial wall has also been noted in patients with hyperkinetic circulations and in patients with left-to-right shunts, either from ventricular septal defect or patent ductus arteriosus (personal observations). The near-normal size of the left atrial chamber is one of the hallmarks of acute or subacute mitral insufficiency, and such findings are seldom seen in chronic mitral regurgitation of great severity. The intensity and width of the echo from the anterior mitral valve leaflet also aid in distinguishing this entity from mitral regurgitation due to rheumatic heart disease, since, in the latter, the mitral valve is thickened and often calcified, whereas in ruptured chordae tendineae unassociated with previous rheumatic valve disease the leaflets remain thin and noncalcified.

Although it is possible to have ruptured chordae tendineae in the presence of rheumatic valvular disease, and thus a thickened valve in the presence of other signs of ruptured chordae, we have not yet seen such a combination. Finally, the increased amplitude of motion of the septum and augmented estimated stroke volume with normal-to-increased ejection fraction are a combination of findings suggesting mitral insufficiency of large magnitude, with normal or near-normal left ventricular contractility. This is an important observation since many patients with primary heart failure and associated mild mitral insufficiency have poorly contracting left ventricles, and these values reside in the abnormally low range.

It should be noted that the E-F slope (rate of posterior motion of the anterior mitral valve leaflet during rapid ventricular filling) has been described as characteristically being increased in rheumatic mitral insufficiency. This value was normal in four of our six patients with ruptured chordae tendineae to the posterior mitral valve leaflet.

The echocardiographic findings from the anterior leaflet prolapse are not as characteristic, but, utilizing the combination of findings described, we have accurately identified this entity in three instances. It should be emphasized that anterior leaflet prolapse from ruptured chordae tendineae is far less common than that of posterior leaflet prolapse and that other characteristics, such as radiation of the murmur to the back and along the spine, enables one to recognize this entity.

It should be pointed out that echocardiographic examination is capable of distinguishing a complete prolapse of a detached cusp from some of its chordae, from a partial prolapse ("billowing") of the mitral cusp, related to stretched chordae or aneurysmal leaflet deformity. This latter entity has an entirely different etiology, clinical significance, and prognosis. In patients with midsystolic clicks and late systolic murmurs one sees an abrupt posterior motion of the anterior or posterior leaflet echo, occurring in midsystole, coincident with the click and onset of murmur.

While the number of cases studied by us utilizing echocardiography is not large enough to make a definitive statement, one can surmise that echocardiographic diagnosis of
the rupture of the chordae to the posterior mitral cusp has a very high specificity. Furthermore, it appears to be a more sensitive method than angiocardiography: prolapse of the mitral valve leaflets was identified by left ventricular angiogram in six of our eight patients. However, in only one of these was the particular leaflet which was prolapsing correctly identified.

References
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