Mitral Insufficiency Resulting from Rupture of Normal Chordae Tendineae

Report of a Surgically Corrected Case

By L. E. January, M.D., June M. Fisher, M.D., and J. L. Ehrenhaft, M.D.

MitraL INSUFFICIENCY usually results from the distortion or loss of substance of the mitral leaflets associated with chronic rheumatic heart disease. Mitral insufficiency from rupture of chordae tendineae is different because the leaflets themselves may be normal. Insufficiency is due to partial loss of the valve-restraining mechanism causing regurgitation during ventricular systole. The pathologic findings in this condition have been reviewed in recent reports.1,2

The role of infection in the development of the disorder is not clear.3 It is reported that healed subacute bacterial endocarditis, sometimes associated with antecedent rheumatic changes, may weaken the chordae tendineae and permit rupture.4 Rheumatic endocarditis alone,5 crushing chest injuries with compression of the heart,6 and severe cardiac strain from exertion have been implicated. In some instances no definite cause has been found and spontaneous rupture must be considered.7

Spontaneous disruption of the valvular supporting structure has been considered rare but more and more often it is being found as the only cause for mitral insufficiency. Preoperative diagnosis often is possible, and surgical repair is feasible. Attempts at correction by closed plication methods have not been very successful.8 During open cardiotomy the damaged structures can be carefully inspected and repaired directly by reconstructing the chordae tendineae or the valve.9-11 This may or may not necessitate additional plication of a dilated valve ring, sometimes stretched by long-standing mitral insufficiency with left atrial and left ventricular enlargement.

We have observed a patient in whom spontaneous rupture of the chordae tendineae was diagnosed preoperatively and corrected surgically.

Case Report

Miss A.C., a 58-year-old school teacher, had considered herself healthy. Regular physical examinations for years had disclosed no heart murmurs. While walking home from school in a leisurely way on the afternoon of October 3, 1960, she experienced a vague, poorly defined ache in the anterior chest. She said it was "not a real pain," but it was unusual and she hesitated and considered sitting down on the curb. Instead, she walked on home and more or less forgot about it during her usual routine that evening. She retired at about 10:30 p.m., feeling well, only to be awakened at about 1:30 a.m., October 4, 1960, by severe dyspnea, cough and "rattling" in the chest and a fear that she "was going to die." She sat up in a chair the remainder of the night, calling her doctor early in the morning. He hospitalized her immediately.

The only positive physical findings were dyspnea, crepitant rales over the right lung and a grade-II, blowing systolic murmur loudest at the cardiac apex. The heart was not demonstrably enlarged and the rhythm was regular at 112 beats per minute. The blood pressure was 146/80 mm. Hg. The temperature was 101.0 F. The blood count, urinalysis, and electrocardiogram were normal. The chest x-ray (fig. 1a) was interpreted as showing a pneumonitis in the right lung. The blood serology was negative. The maximum SGOT was 30 units. A blood culture was sterile. The white blood cell count did not increase.

"Virus pneumonitis" was suspected. No antibiotic drugs were prescribed. The temperature returned promptly to normal. A sinus tachycardia up to 120 beats per minute persisted and digitalization with digoxin was accomplished. Six days after admission chest fluoroscopy disclosed no abnormality. She was dismissed on the eighth day, still receiving a maintenance dose of 0.1 mg. of digoxin daily.

It was difficult for her to continue at work because of fatigue and mild exertional dyspnea.
She next reported to her doctor on November 3, 1960, having had orthopnea for 1 week. The heart was larger. The systolic murmur had increased to grade IV and was heard widely over the chest. The blood pressure was 170/80 mm. Hg. The neck veins were distended. Crepitant rales were heard in both lungs, and moderate hepatomegaly had appeared. The weight was up six pounds from a month earlier.

The chest x-ray (fig. 1b) confirmed the increase in heart size, and there were small bilateral pleural effusions and pulmonary congestion. The blood count, urinalysis, sedimentation rate, blood urea nitrogen, and the electrocardiogram were normal. Two LE-cell preparations were negative. Blood cultures were sterile.

The maintenance dose of digitoxin was continued, moderate salt restriction was instituted, and she received a mercurial diuretic. Improvement was rapid and the signs of cardiac failure disappeared. The roentgenographic evidence of cardiac failure disappeared in the chest x-ray (fig. 1c) and the heart itself appeared as on the first film (fig. 1a).

She taught the remainder of the school term, but cardiac failure progressed in spite of salt restriction, digitoxin, and chlorothiazide therapy. Her doctor repeatedly noted neck vein distention and hepatomegaly. Pedal edema eventually appeared. Heart size continued to increase, and the systolic murmur grew even louder. The blood pressure rose to 166/110 mm. Hg.

She was admitted to the State University of Iowa Hospitals on June 14, 1961. Neck vein distention, bilateral pulmonary rales, hepatomegaly to 5 cm. below the costal margin, and pitting edema to the knees were noted. Both ventricles were significantly hyperactive and the cardiac apex was at the anterior axillary line, where there was a prominent systolic thrill. The rhythm was regular at 80 beats per minute. The pulmonic second sound was accentuated. A grade-V medium-pitched pansystolic murmur (fig. 2), was loudest at the apex. It was transmitted throughout the chest and into the neck. The blood pressure was 160/100 mm. Hg.

The chest x-ray (fig. 3a), made 2 days after diuresis had reduced her weight by eight pounds, revealed significant enlargement of the heart. The electrocardiogram was normal except for digitalis effect. The routine blood and urine examinations were normal. The serum cholesterol was 242 mg. per cent. The protein-bound iodine was 5.4 meg. per cent.
MITRAL INSUFFICIENCY FROM RUPTURED CHORDAE TENDINEAE

Table 1
Cardiac Catheterization Data

<table>
<thead>
<tr>
<th></th>
<th>Preoperative pressures</th>
<th>Postoperative pressures</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.A.</td>
<td>38/14</td>
<td></td>
</tr>
<tr>
<td>R.V.</td>
<td>75/3</td>
<td>37/1</td>
</tr>
<tr>
<td>R.A.</td>
<td>12/4</td>
<td>5/2</td>
</tr>
<tr>
<td>L.A.</td>
<td>51/7 A = 17</td>
<td>13/7 (9)*</td>
</tr>
<tr>
<td>L.V.</td>
<td>135/11</td>
<td></td>
</tr>
</tbody>
</table>

*Wedge pressure.

Discussion

The history failed to establish pre-existing rheumatic or congenital heart disease. The course of the illness and the observations in another hospital virtually excluded myocardial infarction and bacterial endocarditis. The sudden onset of the disease, the impressive increase in both the loudness of the systolic murmur and the size of the heart, and the relentless progression of cardiac failure lead us to suspect mitral insufficiency from spontaneous rupture of chordae tendineae. We thought that the initial, severe nocturnal dyspnea several hours after the onset of the anterior chest discomfort was due to pulmonary edema and that the first chest x-ray (fig. 1a) showed unilateral pulmonary edema12, 13 instead of a pneumonitis. Less likely possibilities were rupture of a papillary muscle or rupture of the interventricular septum. Neither seemed probable, particularly because there had been no evidence whatsoever of acute myocardial infarction.

Diuresis of 16 pounds was associated with relief from the signs and symptoms of cardiac failure. Cardiac fluoroscopy disclosed no evidence of a ventricular aneurysm or of a left-to-right shunt. The results of cardiac catheterization via the right saphenous vein are shown in table 1. There were severe right ventricular hypertension and evidence of chronic cardiac failure. Left atrial pressure was greatly elevated as measured by transseptal puncture. The left atrial pressure tracing before operation (fig. 4) depicts the typical systolic peak of severe mitral regurgitation with a maximum pressure of 51 mm Hg.

We were secure in the diagnosis of mitral insufficiency and she was scheduled for operation with use of the heart-lung machine. The right atrium was of normal size. The right ventricle was slightly enlarged, and the left ventricle and left atrium were greatly enlarged. Palpation over the posterior aspect of the left atrium revealed a thrill, transmitted into the root of the aorta, suggesting an associated aortic stenosis.14, 15 The left atrium was opened, and inspection of the mitral leaflets revealed severe mitral insufficiency caused by rupture of the chordae tendineae inserting into the posterior portion of the mural leaflet. There was no evidence of scarring or calcification of the mitral leaflets indicative of antecedent rheumatic disease or healed subacute bacterial endocarditis. Furthermore, the chordae tendineae leading to the septal leaflet and those anteriorly attaching to the mural leaflet appeared normal. The insufficiency was repaired by placing two 00 sutures through the base of papillary muscles and
through the margins of the unsupported portion of the mural leaflet at different points and tied in place (fig. 5). This type of repair appeared adequate to prevent prolapse of the mural leaflet above the level of the mitral ring. There was no associated aortic stenosis.

Postoperative Course

During the first 2 weeks after operation there were numerous recurrences of short paroxysms of atrial fibrillation, which were ultimately prevented by quinidine. Her subsequent course has been one of progressive improvement with return to normal health. She has had no further cardiac failure. The heart has become impressively smaller, as shown in figure 3b. The left ventricle is less hyperactive and the apical systolic thrill is gone. There is a sinus rhythm and the heart sounds are normal. The murmur indicated in the phonocardiogram in figure 2 has reduced its intensity to grade III and is less harsh. The residual mitral insufficiency has no apparent dynamic consequences. The blood pressure is 150/90 mm. Hg. The right hemidiaphragm is elevated and immobile, due to injury of the right phrenic nerve at operation.

She has returned to work. The results of cardiac catheterization via the left saphenous vein, performed 6 months after operation, are shown in table 1.

The right ventricular pressure returned to near normal. Transseptal puncture was technically impossible but the pulmonary wedge pressure was only slightly elevated. Figure 4 shows no systolic pressure peak in the wedge position comparable to the prominent direct left atrial pressure peak before surgery.

Summary

Mitral insufficiency resulting from rupture of normal chordae tendineae occurs and can be recognized. It is subject to surgical repair. A patient with refractory cardiac failure due to mitral insufficiency on this basis has been totally rehabilitated by surgical repair of the valvular restraining mechanism.

Acknowledgment

We are grateful to Benjamin J. Wolverton, M.D., Cedar Rapids, Iowa, for the data on this patient prior to her admission to the State University of Iowa Hospitals.
MITRAL INSUFFICIENCY FROM RUPTURED CHORDAE TENDINEAE

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Principle of Inductive Reasoning

Once more we emphasize that the principle of mathematical induction is quite distinct from empirical induction in the natural sciences. The confirmation of a general law in any finite number of cases, no matter how large, cannot provide a proof for the law in the rigorous mathematical sense of the word, even if no exception is known at the time. Such a law would remain only a very reasonable hypothesis, subject to modification by the results of future experience.—Richard Courant and Herbert Robbins, What is Mathematics? An Elementary Approach to Ideas and Methods. England, Oxford University Press, Tenth Printing, 1960, p. 10.
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