Value of Exploratory “Scanning” in the Echocardiographic Diagnosis of Ruptured Chordae Tendineae

By T. D. Giles, M.D., G. E. Burch, M.D., and E. C. Martinez, M.D.

SUMMARY
Echocardiographic diagnosis of rupture of the chordae tendineae to the anterior and posterior leaflets of the mitral valve may be difficult to make. Two patients, one with rupture of chordae tendineae to the posterior leaflet and the other with rupture of chordae tendineae to the anterior leaflet of the mitral valve, are reported to illustrate the use of an exploratory “continuous scanning” technique to facilitate and verify the echocardiographic diagnosis of these disorders. The elimination of confusing echocardiograms is emphasized.

Additional Indexing Words:
Mitral regurgitation  Mitral valve leaflet prolapse  Mitral valve echoes  Left atrial echoes

The echocardiogram is useful in the diagnosis of ruptured chordae tendineae of both posterior1, 2 and anterior1, 2, 3 leaflets of the mitral valve. However, echocardiograms recorded from patients with ruptured chordae tendineae are subject to variations in individual interpretation. Thus, the level of confidence in echocardiographic diagnosis of this disorder likewise varies.

We have recently studied echocardiographically two patients with surgically proven ruptured chordae tendineae. In one patient chordae tendineae to the anterior leaflet of the mitral valve were ruptured, whereas in the other patient, the chordae tendineae to the posterior leaflet were ruptured. This report is concerned both with the echocardiographic technique for the study of chordae tendineae rupture and with the resultant recordings showing characteristic diagnostic manifestations.

Echocardiographic Technique
The echocardiograph used was a Smith-Kline ultrasonoscope (Ekoline-20A) utilizing a 2.25 MHz transducer, 1.6 cm in diameter with a repetition rate of 1,000 impulses/sec. Permanent records were obtained using a Honeywell Visicorder. Paper speed was 50 mm/sec.

The patients were examined in the recumbent position with the torso elevated approximately 45° above the horizontal. An aquasonic gel was used to insure airless contact of the transducer with the chest wall. Initially, the transducer was placed at the left sternal border in the fourth intercostal space and the ultrasound beam directed posteriorly, inferiorly and slightly to the left. Damping and gain adjustments were made to obtain satisfactory echoes from the interventricular septum, mitral valve leaflets and posterior left ventricular wall. Then the transducer was slowly rotated superomedially toward the patient’s right shoulder until echoes from the aortic root and left atrium were obtained. By recording continuously while slowly rotating the ultrasound beam, a continuous exploratory “sector scan” was obtained which reflected anatomic and temporal continuity between the mitral valve region of the left ventricle and the aortic root and left atrium.

Report of Patient 1
J.E., a 54-year-old man, was admitted to the Tulane Cardiology Service at Charity Hospital of Louisiana at New Orleans on October 16, 1972 for evaluation and treatment of severe mitral regurgitation. Twelve years prior to admission, he sustained an accidental compressing injury of the chest which was followed by a brief period of dyspnea. Two years later, he had the insidious onset of easy fatigability and dyspnea on exertion. He also reported occasional episodes of substernal chest pain which were associated with effort at times. However, he had several instances of sudden, fairly severe left anterior chest pain associated with dyspnea.

Eight years prior to admission, mitral regurgitation was recorded for the first time on physical examination and mild left ventricular congestive heart failure and angina pectoris were present. A diagnosis of rheumatic
valvular heart disease and concomitant coronary arteriosclerosis was made. Despite treatment, his congestive heart failure progressively worsened until the time of admission. There was no past history of rheumatic fever.

Physical examination on admission revealed a chronically ill man in mild respiratory distress. Blood pressure was 112/92 mm Hg, pulse rate 92 beats/min, and respiratory rate 22/min. The neck veins were not distended. The apex impulse of the heart was large and displaced laterally into the anterior axillary line. A left parasternal heave and a systolic thrill were palpated at the apex. The first heart sound was of normal intensity, and the second heart sound was widely split with an accentuated pulmonic component. A protodiastolic gallop rhythm was present. A loud, high-pitched pansystolic crescendo-decrescendo murmur was heard at the apex, which radiated best to the left sternal border and neck and poorly to the left axilla and back. The remainder of the physical examination was unremarkable.

The electrocardiogram showed a regular sinus rhythm with a slightly prolonged PR interval. Changes of left ventricular hypertrophy were present as well as ST segment and T wave changes indicative of papillary muscle ischemia. Teleradiograms of the chest revealed generalized cardiomegaly and pulmonary vascular congestion. An echocardiogram was recorded on October 17, 1972. The pertinent findings are illustrated by the exploratory "continuous sector scan" shown in figure 1. The left atrium was large and its posterior wall bulged posteriorly during ventricular systole. Prominent echoes were recorded from the left atrial cavity posteriorly. As the scan was followed to the mitral valve region of the left ventricle, the echoes recorded from within the left atrium were seen to merge with those from the posterior leaflet of the mitral valve. The anterior leaflet of the mitral valve moved normally during diastole.

The anterior and posterior leaflets of the mitral valve did not coapt during ventricular systole and remained at least 0.5 cm apart with maximal separation during mid-systole. This separation was primarily due to a posterior displacement of the posterior leaflet of the mitral valve during ventricular systole. An early diastolic anterior "overshoot" of the posterior leaflet of the mitral valve was present.

From the echocardiogram, the diagnosis of ruptured chordae tendineae to the posterior leaflet of the mitral valve was made. The ECHO tracing was interpreted as showing a flail posterior mitral valve leaflet with its attached chordae tendineae prolapsing into the left atrium during systole. Because the echoes from the prolapsed leaflet were recorded far into the left atrium, and because of the scattered nature of the echoes (fig. 1), it was deduced that the chordae tendineae attached to the prolapsing posterior mitral valve leaflet were long. Some abnormality of the anterior leaflet of the mitral valve was suspected because of the horizontal systolic CD segment of the echogram.

On December 19, 1972, the patient underwent surgery for plication of the posterior leaflet of the mitral valve and annuloplasty for ruptured chordae tendineae. At operation, the mitral valve ring was noted to be markedly dilated. The anterior leaflet of the mitral valve appeared essentially normal. The posterior mitral valve leaflet was found to be unsupported from approximately its midpoint to the posteromedial commissure. At least four chordae tendineae, each one ruptured approximately one centimeter from its attachment to the posterior leaflet, were present near the posteromedial commissure. A plication procedure was carried out on the posterior leaflet. The patient died intraoperatively.

**Report of Patient 2**

D.M., a 19-year-old woman, was referred to the Tulane Cardiology Service at Charity Hospital of Louisiana at New Orleans for evaluation for fever and a heart murmur. Two months prior to admission, she developed a "flu-like" illness. She received antibiotics. Two weeks later she continued to have fever in association with nausea, vomiting and backache. She was treated for a urinary tract infection with antibiotics without improvement. A cardiac murmur was first noted at that time.

Seven days prior to admission, she was hospitalized elsewhere with a diagnosis of acute pyelonephritis. No improvement in the clinical state occurred, and she subsequently developed pulmonary edema. A loud apical holosystolic murmur was noted. The patient was treated with digitalis, diuretics, oxygen, bed rest and a low salt diet with little benefit. After five days of this therapy she was referred to Charity Hospital of New Orleans for further evaluation and treatment.

There was no past history of rheumatic fever.

Upon physical examination on admission the patient was found to be lethargic and dyspneic. Blood pressure was 108/70 mm Hg and the pulse 103/min. The oral temperature was 101°F. There were no signs of systemic embolization. The apex beat of the heart was in the fifth intercostal space in the mid-clavicular line and was hyperkinetic. A systolic thrill was palpated at the apex. A loud, pansystolic murmur was heard at the apex, left axilla and back. A mid-diastolic rumble and a protodiastolic gallop rhythm were present. A faint diastolic blowing murmur was heard at the left sternal border. The second heart sound exhibited normal splitting and had a loud pulmonic component. Crepitant rales were heard at the lung bases bilaterally.
Teleroctgenograms of the chest revealed generalized cardiomegaly with pulmonary vascular congestion and left atrial enlargement. Several electrocardiograms recorded during hospitalization revealed a sinus tachycardia.

Laboratory examinations revealed mild anemia and leukocytosis. An increase in numbers of red blood cells and white blood cells and some fine and coarse granular casts were found in the urine. Numerous blood cultures were negative. The patient was treated empirically for bacterial endocarditis and congestive heart failure. After one week of therapy, she became afebrile. However, she remained in congestive heart failure. Eight days following admission electrocardiograms were recorded.

A representative echocardiographic tracing obtained by an exploratory “continuous sector scan” is shown in Figure 2. There was marked excursion of the interventricular septum and left ventricular posterior wall. The anterior leaflet of the mitral valve exhibited marked early diastolic anterior motion, almost touching the interventricular septum. Coarse “fluttering” of the anterior leaflet of the mitral valve was recorded. During systole, multiple parallel echoes were recorded from the anterior leaflet of the mitral valve, partially obscuring the posterior leaflet. In exploring to the region of the aorta and left atrium, the anterior leaflet of the mitral valve was found to prolapse into the anterior portion of the left atrial cavity during ventricular systole. This echocardiogram was therefore considered to indicate a flail anterior leaflet of the mitral valve secondary to ruptured chordae tendineae at the margin of the valve leaflet.

On the 38th day of hospitalization, replacement of the mitral valve with a prosthesis was undertaken. At surgery, several chordae tendineae were found to be ruptured near their attachment to the anterior leaflet of the mitral valve. The flail leaflet prolapsed freely into the left atrium. Vegetations were present on the anterior mitral valve leaflet. The posterior leaflet appeared normal. The patient died immediately postoperatively.

Discussion

Numerous echocardiographic criteria for the diagnosis of ruptured chordae tendineae to the posterior leaflet of the mitral valve have been reported. Echoes recorded from within the left atrial cavity which move toward the posterior left atrial wall during ventricular systole may originate from a flail posterior leaflet. When such echoes are associated with echocardiographic evidence of recent onset of mitral insufficiency, i.e., posterior systolic motion of the left atrium, almost normal left atrial size, and marked systolic motion of the interventricular septum, then rupture of chordae tendineae to the posterior mitral valve leaflet is particularly likely. Lack of systolic coaptation of the mitral valve leaflets, early systolic posterior motion of the posterior mitral valve leaflet and an early diastolic “overshoot” of the valve leaflet with further posterior displacement associated with atrial contraction have also been observed.

The above criteria were all fulfilled by the echocardiogram recorded from patient 1. However, when each criterion is viewed individually, its interpretation is difficult. For example, we and others have noted the presence of multiple linear echoes within the region of the left atrium of patients with left atrial thrombi. Such echoes may be artifactual in nature. Moreover, the echoes recorded from the flail posterior leaflet of the mitral valve of patient 1 exhibited random motion as well as posterior motion toward the left atrial posterior wall during ventricular systole. There is no doubt that some of the random echoes were from chordal material. Likewise, the systolic posterior motion of the posterior mitral valve leaflet recorded from our patient has been noted in other disease states of the mitral valve, e.g., floppy mitral valve or mid-systolic click syndrome. However, the very early systolic posterior movement of the posterior leaflet of the mitral valve which was also noted in our patient is a more reliable sign of ruptured chordae tendineae. The findings associated with mitral regurgitation of recent onset are, of course, nonspecific.

The diagnosis of ruptured chordae tendineae to the posterior leaflet of the mitral valve becomes reliable when the exploratory “continuous sector scan” is studied. Echoes recorded from within the left atrial cavity are found to blend into those from the posterior leaflet of the mitral valve, thus establishing not only prolapse of the valve leaflet, but a flail, posterior mitral valve leaflet as well. Because abnormal echoes were recorded so far into the left atrial chamber and exhibited marked random motion in our patient 1, it was assumed,
and later proved at surgery, that the ruptured chordae tendineae attached to the flail leaflet were long.

Rupture of the chordae tendineae to the anterior mitral valve leaflet is somewhat more difficult to diagnose echocardiographically than rupture of those to the posterior leaflet. Echocardiographic criteria for the diagnosis of the former include marked, erratic diastolic fluttering and multiple linear systolic parallel echoes recorded from the anterior leaflet of the mitral valve. Increased amplitude during diastole of echoes recorded from the mitral valve leaflets and movement of the anterior leaflet of the mitral valve into the left atrium during ventricular systole have been reported. Echocardiograms recorded from patient 2 showed all of these findings. Once again, however, these findings would be difficult to interpret without the use of the exploratory "continuous sector scanning" technique.

Diastolic fluttering of the anterior leaflet of the mitral valve is seen in patients with aortic insufficiency, atrial flutter or fibrillation, as well as in technically inadequate recordings. Multiple parallel systolic echoes may also originate from a normal anterior leaflet of the mitral valve. Finally, movement of the anterior leaflet of the mitral valve into the left atrium may occasionally be missed if only standard (isolated segmental) echocardiographic recording techniques are used.

Utilizing the exploratory "continuous scanning" technique, a dynamic and panoramic view of a flail anterior leaflet of the mitral valve is obtained. Most importantly, movement of the anterior leaflet of the mitral valve into the left atrium during ventricular systole may easily be seen. Furthermore, detection of echoes from the anterior mitral valve leaflet region of the anterior portion of the left atrial wall denotes a flail leaflet.

Thus, exploratory "continuous sector scanning" is a valuable technique in rendering a confident echocardiographic diagnosis of ruptured chordae tendineae either to the posterior leaflet of the mitral valve or to the anterior leaflet. We feel that exploratory "continuous scanning" between several anatomic regions of the heart should be done routinely in patients undergoing echocardiographic examination. Such echocardiographic exploration clearly requires the direction of a physician acquainted with normal and pathologic anatomy as well as the clinical intricacies of the disease process.

References
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