PAPERS

Echocardiographic visualization of the anatomic causes of mitral regurgitation resulting from myocardial infarction

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Summary
A murmur of mitral regurgitation in patients with congestive heart failure after an acute myocardial infarction suggests a surgically correctable cause of the heart failure. Six patients who presented in this manner and who later underwent surgery have been evaluated by two-dimensional echocardiography (2DE). The underlying anatomical cause of the mitral regurgitation was correctly identified as papillary muscle rupture (2 cases), ruptured chordae tendineae (1 case) and papillary muscle fibrosis (3 cases).

Two-dimensional echocardiography is useful in evaluating patients with congestive heart failure who develop a systolic murmur after acute infarction, as it can detect surgically correctable structural defects.

Introduction
Congestive cardiac failure after a myocardial infarction generally indicates extensive muscle necrosis, usually not curable by surgery. The appearance of a pansystolic murmur in this clinical setting, however, suggests the possibility of a surgically correctable lesion (Fox, Glassman and Isom, 1979).

Cardiac catheterization and angiography are generally required to assess the severity of the mitral regurgitation and to exclude an acquired ventricular septal defect. The development of two-dimensional echocardiography (2DE) gives the clinician an alternative bedside method of studying the sequelae of myocardial infarction. Experience with this technique in the detection of the underlying anatomic causes of mitral regurgitation occurring after myocardial infarction is reported.

Patients and methods
Six patients (5 male, 1 female) aged 42 to 67 (mean age 61) years with congestive cardiac failure and recent onset of mitral regurgitation following myocardial infarction were studied prospectively by 2DE before invasive study. Two-dimensional echocardiography was performed using an HP 77020A phased array sector scanner. Complete echocardiographic studies were obtained from the standard transducer positions including the parasternal long and short axis views (Fig. 1a and b), the apical and subxiphoid views. The mitral valve apparatus and function were analysed from these multiple imaging planes (Tajik et al., 1978). All patients had cardiac catheterization and conventional angiographic studies and later underwent surgery where the anatomic pathology was confirmed.

Results
Papillary muscle rupture
Two patients had acute left heart failure after an inferior infarction and rupture of a part of a papillary muscle. In each case there was a flail mitral valve leaflet (anterior in one case, posterior in the other) visualized by 2DE, particularly in the apical views. During systole, the tip of the affected leaflet was seen overshooting the other leaflet into the left atrium, with loss of systolic coaptation. The length of the flail segment was relatively long and the increased thickness of the tip of the flailing segment was suggestive of a mass lesion (Fig. 2). At surgery this mass was seen to be a detached head of the posteromedial papillary muscle. Both patients survived mitral valve replacement.

Ruptured chordae tendineae
A 67-year-old patient developed severe mitral regurgitation due to a ruptured chordae affecting the posterior mitral valve leaflet following an anterior infarction. In the parasternal long axis view (Fig. 3) and apical views there was rapid systolic motion of the tip of the leaflet into the left atrium with loss of normal leaflet coaptation. The vibrating motion of the ruptured chordae could also be seen.
Fig. 1. (a) Long axis view of the left ventricle. The aorta (A) is to the right and the apex of the left ventricle (LV) is to the left of the image. The right ventricle (R) is anterior and the left atrium (la) is posterior. Normal systolic coaptation of the anterior (a) and posterior (p) leaflets of the mitral valve (Mv) is seen; pw=posterior left ventricular wall. (b) Cross-sectional short axis view of the left ventricle at the level of the papillary muscles. The anterolateral (AL) and posteromedial (PM) papillary muscles are seen in the left ventricular cavity. A=anterior; P=posterior; L=lateral ventricular wall segments; e=endocardium; S=interventricular septum.
in the short axis view of the left ventricle during diastole. At surgery the antero-lateral papillary muscle was fibrotic and the two major medial fixing chordae to the posterior cusp were ruptured. This resulted in prolapse of a large segment of the posterior cusp into the left atrium. The flail segment was excised and the valve repaired. Additional revascularization to the circumflex and diagonal vessels was undertaken. The postoperative course was uneventful.

The posterobasal left ventricular wall was akinetic in the patient with an inferior infarction and posteromedial papillary muscle involvement. Reduced thickness, increased wall density and decreased motion of anterolateral segments (Fig. 4b) were present in the other two patients with anterior infarcts and anterolateral papillary muscle involvement. The abnormal ventricular wall motion involved the corresponding papillary muscle, which also appeared denser than normal (Fig. 5). One of these patients also had an anteropapical thrombus visualized by ultrasound (Fig. 5). At surgery, the papillary muscle and chordae were intact, but the affected papillary muscles were scarred. The presence of a partially organized apical mural thrombus in one of the cases was confirmed. In addition to surgical revascularization procedures, these patients underwent mitral valve replacement. One patient with anterolateral papillary muscle dysfunction died in the perioperative period.

**Angiographic results**

The 6 patients underwent left ventricular angiography and coronary arteriography. In 5 an A–P left ventriculogram was performed and in one both A–P and lateral views were made. In each case severe mitral regurgitation was demonstrated, but in no case was the underlying morphology visualized. The presence of coronary artery disease was shown in each case.

**Discussion**

Mitral valve competence is dependent on normal and co-ordinated function of the mitral valve annulus, leaflets, chordae, papillary muscle and left ventricular myocardium (Silverman and Hurst, 1968). The diagnosis of mitral regurgitation can usually be made at the bedside; the appearance of the murmur following myocardial infarction suggests a surgically correctable lesion. Although cardiac catheterization is required to exclude septal rupture and to assess the severity of the mitral regurgitation, conventional angiographic views provide little information as to the anatomic cause leading to valve insufficiency. Oblique axial left ventricular angiography (Fig. 6) may at times demonstrate a flail mitral leaflet. Using 2DE, it was possible to identify non-invasively the underlying anatomic cause of the mitral regurgitation in the 6 patients with angiographically documented severe mitral regurgitation resulting from coronary artery disease.

Papillary muscle rupture is an unusual cause of mitral regurgitation complicating about 1% of acute myocardial infarctions. The posteromedial papillary muscle is the one most frequently involved (Sanders, Neuberger and Ravin, 1957). In the 2

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**Papillary muscle dysfunction**

Three patients had papillary muscle dysfunction following myocardial infarction. None had flail mitral leaflet. Although systolic coaptation of the leaflet was preserved, it occurred inferiorly and mitral valve closure was incomplete (Fig. 4). Diastolic motion of the affected leaflet was reduced; it appeared to be retracted and held in a rigid position.

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**Fig. 2.** Apical view from one of the patients with severe mitral regurgitation from a ruptured papillary muscle. Systolic frame. The flail posterior (p) mitral leaflet (thin arrow) overshoots the anterior leaflet (a) and lies within the left atrium. The increased thickness of the tip of the flailing segment (large arrow) is suggestive of a mass lesion, in this case the head of the papillary muscle. (Legend as in Fig. 1).
Fig. 3. Long axis view from the patient with severe mitral regurgitation from ruptured chordae tendineae and flail posterior mitral leaflet. The tip of the posterior leaflet (arrows) moves past the anterior mitral leaflet into the left atrium during systole. Maximal abnormal motion occurs at the tip and there is loss of normal leaflet coaptation. (Legend as in Fig. 1).

Fig. 5. Parasternal short axis view from a patient with papillary muscle fibrosis following an anterolateral infarction, showing a protruding apical thrombus (large arrows) and fibrosis of the anterolateral papillary muscle (small arrows) which appears denser than the posteromedial papillary muscle. (Legend as in Fig. 1).
Fig. 4. Papillary muscle dysfunction and anterior left ventricular wall abnormality leading to mitral regurgitation (a) Long axis view, systolic frame. There is stretching and retraction of the leaflets into the body of the left ventricle (arrow); mitral valve closure is incomplete. (b) Short axis view, the abnormal anteroseptal wall is best seen in this view which shows reduced thickness, increased wall density and decreased motion (arrows) of the anterolateral wall involving the anterolateral (AL) papillary muscle. (Legend as in Fig. 1).
patients in which an inferior myocardial infarction caused rupture of the posteromedial papillary muscle leading to a flail leaflet, the anterior mitral leaflet was involved in one case and the posterior leaflet (Fig. 2) in the other. Most patients who survive the acute event have rupture of one or two heads of one papillary muscle, as in the two cases described; rupture of an entire papillary muscle is usually fatal (Kremkau, Gilbertson and Bristow, 1973).

Fig. 6. Left ventricular angiogram, left anterior oblique view with caudocranial angulation. A flail mitral leaflet (arrows) in a patient with non-ischaemic mitral regurgitation is visualized. Ma = mitral annulus; LA = left atrium; LV = left ventricle; A = aorta.

Ruptured chordae tendineae is a common cause of acute mitral regurgitation. Although most patients with this condition have no underlying heart disease (Sanders et al., 1967) it can occur secondary to other conditions including myocardial infarction. Ruptured chordae (Fig. 3) secondary to ischaemia was visualized in one patient who had significant mitral regurgitation documented angiographically following an anterior infarction.

Because both chordal and papillary muscle rupture can produce a flail mitral leaflet detectable by 2DE, it may not always be possible to determine the precise site of rupture by this technique. Increased thickness in the tip of the flailing segment (Fig. 2) is suggestive of papillary muscle rupture. However, as both conditions produce severe mitral regurgitation, the clinical and surgical implications are similar (Fox et al., 1979).

The papillary muscles are sensitive to ischaemia because of their precarious blood supply and relatively increased oxygen demand. Extreme forms of papillary muscle necrosis and acute fibrosis associated with ventricular wall injury resulting from coronary artery disease may lead to shortening and retraction of one of the leaflets into the body of the left ventricle. This results in disruption of the normal line of anterior and posterior leaflet closure (Fig. 4) and leads to mitral regurgitation (Shelburne, Rubinstein and Gorlin, 1969).

Patients with papillary muscle fibrosis and dysfunction are indistinguishable clinically from those with papillary muscle rupture (Glancy et al., 1973). These two lesions can, however, be differentiated by two-dimensional echocardiography and this is important because papillary muscle rupture implies significant mitral regurgitation requiring surgery. Patients with papillary muscle fibrosis and dysfunction with severe, surgically curable mitral regurgitation cannot however be separated by 2DE from those with papillary muscle dysfunction and mild regurgitation; the severity of the regurgitation has to be assessed angiographically.

Because echocardiographic technique permits the non-invasive assessment of diffuse or segmental wall motion abnormalities, mitral insufficiency secondary to ventricular dysfunction can be differentiated from mitral regurgitation due to valvar disease (Mintz et al., 1979).

The sensitivity of 2DE in the detection of these structural defects involving the mitral valve is not known. Findings similar to the cases described here have been recently reported (Mintz et al., 1981). The echocardiographic identification of the potentially correctable causes of severe mitral regurgitation after a myocardial infarction should in some cases obviate the need for left ventricular angiography and expedite surgical intervention.

References


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