Angiographic Features of Rheumatic and Nonrheumatic Mitral Regurgitation

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SUMMARY

Left ventricular cineangiography was performed in 37 patients with mitral regurgitation. Twenty-one patients had rheumatic mitral valve disease determined by careful surgical and pathologic examination, 10 had papillary muscle dysfunction, and six had chordae tendineae rupture. Fourteen of the 16 patients considered to have nonrheumatic mitral regurgitation were confirmed surgically; the other two patients developed mitral regurgitation acutely following myocardial infarction. Cineangiograms were evaluated for valve calcification, appearance and mobility of mitral valve leaflets, character of the regurgitant jet, and ventricular contractility. By these criteria, rheumatic mitral regurgitation was distinguished from papillary muscle dysfunction and chordae tendineae rupture in every case. A correct diagnosis of papillary muscle dysfunction was made when focal areas of myocardial dysfunction, determined by comparing tracings of end-systolic and end-diastolic cine frames, occurred at the base of the papillary muscle. However, half the cases of papillary muscle dysfunction had apparently normal contractility. The involved leaflet was correctly identified in the six cases of chordae tendineae rupture.

Additional Indexing Words:
Mitral regurgitation Papillary muscle dysfunction Chordae tendineae
Cineangiography Rheumatic valve disease

The clinical presentation of patients with mitral regurgitation is diverse, ranging from an asymptomatic phase in early rheumatic involvement to fulminant pulmonary edema secondary to acute myocardial infarction and papillary muscle dysfunction or rupture.1-3 Conventional clinical and laboratory methods may prove inadequate to differentiate severe mitral regurgitation due to rheumatic disease from that due to nonrheumatic disorders such as papillary muscle dysfunction or chordae tendineae rupture. Likewise, it is often difficult to differentiate between papillary muscle dysfunction and chordae tendineae rupture in patients with the acute onset of mitral regurgitation.4 Since the etiology of the incompetent mitral valve has prognostic significance and may influence the course of therapy,1 diagnostic left ventricular angiography has been utilized in the evaluation of patients with mitral regurgitation. Initially, our primary consideration was quantitation of the regurgitant fraction, but more recently we have developed diagnostic criteria that allow separation of patients into etiologic categories based upon the appearance of the left ventricle and mitral valve during left ventriculography.

It is the purpose of this report to extend the radiographic criteria generally used for assessment of nonrheumatic mitral regurgitation, and to demonstrate the application of these
criteria in the evaluation of a series of patients with acute and chronic mitral regurgitation.

Methods

From July 1968 to December 1970, 37 patients were selected in whom the clinical findings, cardiac catheterization results, and angiocardiograms were adequate and in whom convincing proof of the etiology of the mitral regurgitation was obtained. Of these 37, 21 were secondary to rheumatic heart disease. Diagnosis of rheumatic heart disease was established by surgical excision and pathologic examination of the mitral valve in all of these patients. Ten patients had papillary muscle dysfunction, and six had chordae tendineae rupture. Of the 16 nonrheumatic patients, 14 had surgical confirmation. Two patients in whom mitral regurgitation developed as an acute event following myocardial infarction never underwent surgery. There were no cases of rheumatic heart disease with superimposed chordal rupture in this series. Left ventricular angiography was performed in all patients. Catheters were preshaped into simple loops (pigtail design),* fabricated from radiopaque polyethylene,* with multiple side holes and an open end hole. Catheters were introduced by percutaneous retrograde femoral technique, and the loop was passed as far to the apex of the left ventricle as possible without inducing ventricular extrasystoles. Fifty cubic centimeters of meglumine diatrizoate (Renografin-76) were injected at 12–15 cc/sec. Low-pressure injections over 4–6 sec were selected as they produce a minimum of premature ventricular contractions, which interfere with quantitation of the regurgitant fraction and with motion of the mitral valve leaflets. Cineangio-
graphy was performed at 50–60 frames/sec with a 9-inch Phillips image intensifier and a 35-mm Arriflex camera. Filming was in the right anterior oblique projection, with the plane of the mitral valve perpendicular to the plane of the screen.

Criteria

Nine angiographic features were evaluated. Three of these observations were considered to be important criteria in distinguishing rheumatic from nonrheumatic insufficiency: (1) presence or absence of valvular calcification; (2) mobility and appearance of valve leaflets; and (3) character of the regurgitant jet. An evaluation of left ventricular contractility was useful in identifying cases of papillary muscle dysfunction. The remaining five features were judged by us to be of no diagnostic value in distinguishing rheumatic from nonrheumatic mitral insufficiency, although the magnitude of the regurgitant jet was an indicator of the severity of the lesion.

Calcification

Ten of the 21 rheumatic valves had radiographically visible calcifications by cine examination. None of the 16 patients with nonrheumatic mitral insufficiency had calcification of the valve leaflets.

Conclusion

The presence of calcium in the mitral valve leaflets is diagnostic of rheumatic mitral valve involvement. The absence of calcification does not exclude a rheumatic etiology. Calcification of the mitral valve annulus is probably unrelated to the rheumatic process, more likely being the end result of a degenerative process.6

Valve Mobility and Appearance

Eighteen of the 21 rheumatic valves could be evaluated for these criteria. Each of these was thickened or irregular. Seventeen of these valves either exhibited diminished motion or domed during diastole into the left ventricle. This appearance was seen only in association with rheumatic heart disease and was independent of the diastolic gradient. Two patients with rheumatic valves had excessive motion or eversion of the posterior leaflet during ventricular systole; however, in both cases there were calcifications present in the leaflet. In the nonrheumatic patients the leaflets appeared thin or filmy (13 patients) or could not be identified with certainty (three patients). In five of 10 patients with papillary muscle dysfunction, valve mobility was judged to be excessive, projecting behind the annulus during ventricular systole. In two patients with papillary muscle dysfunction and five of six patients with chordae tendineae rupture a valve leaf would appear to evert almost completely into the left atrium during ventricular

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*We have used 0.066 RPX material (B-D). Catheters of this design are available commercially from Cook, Inc., and Cordis Corp. The latter fabricates the catheter from radiopaque braided polyurethane.
systole. In every instance, except one, the visualization of an everting or excessively mobile leaflet correctly identified the abnormal leaflet.

**Conclusion**

In rheumatic mitral regurgitation the valve leaflets are almost always thickened and open during diastole with decreased motion (dome). Although not seen in these cases, thickening of the prolapsed leaflet may result from long-standing nonrheumatic insufficiency, possibly as a response to the turbulent regurgitant stream. Mitral leaflet mobility is usually abnormally great in nonrheumatic regurgitation with prolapse of the leaflet behind the annulus visible during ventricular systole. When excessive motion or prolapse of a leaflet is identified, a nonrheumatic etiology affecting that leaflet should be suspected.

**Character of Regurgitant Jet**

A regurgitant jet was visible in all cases studied. In 13 of the 16 cases of nonrheumatic insufficiency the jet was eccentric in direction. Twenty of 21 patients with rheumatic heart disease had a diffuse or primarily central jet. One rheumatic valve had a calcified everting posterior leaflet and an upward directed jet, whereas two other rheumatic valves exhibited jets that began centrally but shifted into an upward or downward direction in late systole. These findings are similar to the observation of marginal jets in mitral regurgitation reported by Raphael and associates.

With chordae tendineae rupture a portion of the mitral valve leaflet is unsupported during ventricular systole and prolapses into the atrium. This prolapsing leaflet may deflect the regurgitant stream resulting in eccentric regurgitation. When the anterior leaf is involved, the stream is generally directed inferoposteriorly. Posterior-leaf involvement causes the jet to be directed in a superior direction against the atrial septum (fig. 1). Inferoposterior jets due to anterior-leaf involvement are easily seen in the right anterior oblique cineangiogram. However, the superior jet is best visualized as a counterclockwise swirling of contrast material in the left atrium, as the jet itself is often hidden by the overlying aorta and left ventricular outflow tract. The regurgitant jet was eccentric in all of our patients with chordae tendineae rupture. By means of the jet as the

![Figure 1](http://circ.ahajournals.org/content/44/6/1082/F1)

**Figure 1**

Left ventriculogram showing asymmetric regurgitant jet directed anterosuperiorly in a patient with ruptured chordae tendineae. The jet is partially hidden by the aortic root. Contrast material has swirled in a counterclockwise direction to fill the enlarged left ventricle. A crescentic lucency represents the edge of the posterior leaflet which is everting behind the mitral annulus. Abbreviations for all figures: Ao = aorta; LA = left atrium; LV = left ventricle. The arrow indicates the direction of the regurgitant jet.
sole criterion, the affected leaflet was predicted correctly in the six cases of ruptured chordae.

Papillary muscle dysfunction probably produces eccentric regurgitation by a similar mechanism—that is, deflection of the regurgitant stream by the prolapsed portion of the valve leaflet (fig. 2). However, the direction of the jet correctly predicted the affected papillary muscle in only six of 10 patients with papillary muscle dysfunction in our series. This lack of specificity is due to the fact that each papillary muscle gives chordae to both mitral valve leaflets.

Twenty of 21 patients with rheumatic heart disease had central jets (fig. 3). Since the rheumatic process results in fusion of the edges of the thickened valve leaflets, insufficiency of the valve is due to the inability of the central portion of the valve leaflets to coapt. The magnitude of the resultant jet is dependent, in part, on the area of the valve that is fixed in an open position.

**Conclusion**

Central jets are indicative of rheumatic mitral regurgitation due to inability of the central portion of the valve leaflets to coapt.
An eccentrically directed jet is characteristic of nonrheumatic disease. An upward-directed jet indicates an abnormality affecting the function of the posterior leaflet; a downward jet is due to malfunction of the anterior leaflet.

**Left Ventricular Contractility**

Tracings of the left ventricle from cine frames representing end-systole and end-diastole were compared. Localized abnormalities of ventricular contraction were seen in four of 10 patients with papillary muscle dysfunction. Combined anterior and posterior infarction accounted for the diffusely abnormal contraction pattern seen in a fifth patient. In these patients the area of diminished ventricular contraction corresponded to electrocardiographic localization of myocardial infarction. This relationship has been noted by others. The involved papillary muscle was located in the area of abnormal contraction. In five patients with papillary muscle dysfunction, ventricular contractility was judged normal. An explanation for this phenomenon is the unique arterial supply of the papillary muscles. Extensive infarction of the underlying myocardium may not involve the papillary muscle, and, conversely, infarction of the papillary muscle may occur in the absence of mural infarction.

Generalized decrease in contractility with dilatation of the left ventricle may also cause mitral regurgitation. The etiology of the dilatation may be idiopathic, secondary to rheumatic involvement, or secondary to left ventricular failure of some other cause. In some cases, a generalized decrease in contractility is secondary to coronary artery disease. In these cases, the resultant mitral regurgitation is caused by inefficient contraction of abnormally directed papillary muscles.

**Conclusion**

In patients with mitral regurgitation, focal abnormalities in ventricular contraction are useful in indicating probable papillary muscle dysfunction. Normal cardiac contractility or diffusely abnormal contractility does not exclude this diagnosis.

**Other Features**

The volume of the regurgitant jet was considered to be large in the majority of the patients studied. The cause of the mitral regurgitation was not correlated with the magnitude of the jet. Ventricular enlargement, atrial enlargement, atrial systolic expansion, and pulmonary venous reflux were of no diagnostic value in differentiating nonrheumatic from rheumatic valvular disease. These features were more directly related to the time interval from onset of mitral regurgitation and the compliance of the left atrium.

**Discussion**

**Diagnostic Accuracy**

With use of the three primary criteria in combination, rheumatic was separated from nonrheumatic mitral regurgitation in every case. Five of the 10 patients with papillary muscle dysfunction exhibited local or generalized abnormalities of contraction. The remaining five patients had normal contractility and were thought to have ruptured chordae. Differentiation between chordal rupture and papillary muscle dysfunction may be extremely difficult in the acute stage following myocardial infarction when we have observed normal ventricular contractility. Two such patients with proven infarction or rupture of papillary muscles were studied initially while in cardiogenic shock. Repeat ventriculography after surgery showed abnormalities of contraction that were not apparent initially. An area of dyskinesis in proximity to a papillary muscle is the basis for differentiation of chordal rupture from papillary muscle dysfunction. However, infarction or ischemia of a papillary muscle may exist when the adjacent myocardium is normal. A diagnosis of chordae tendineae rupture is, therefore, made by exclusion when ventricular contractility appears normal.

To summarize our approach: (1) Look for restricted mobility, doming, or calcification of the mitral valve. If positive, the diagnosis is rheumatic mitral regurgitation. (2) If the leaflets are thin, mobile, and noncalcified, prolapse or excessive mobility of a leaflet.
indicates a nonrheumatic etiology and identifies the abnormal leaflet. (3) An eccentric jet suggests a nonrheumatic etiology. In most cases the direction of the jet implicates the involved leaflet. (4) The ventricular wall is inspected for abnormal contractility. If an area of dyskinesis is in proximity to a papillary muscle, papillary muscle dysfunction is the most likely diagnosis. If contractility is normal or the abnormal area is unrelated to a papillary muscle, the diagnosis of papillary muscle dysfunction cannot be made angiographically. Some of these patients will have papillary muscle dysfunction undetectable by present investigative techniques.

Clinical Application

Although clinical differentiation of rheumatic mitral insufficiency from mitral insufficiency due to papillary muscle dysfunction or chordae tendineae rupture is sometimes possible, laboratory documentation is essential before surgical treatment. The acute development of a regurgitant mitral murmur in association with myocardial infarction or in a patient previously known to have no heart murmur suggests nonrheumatic insufficiency, whereas a long history of a mitral murmur or a history of rheumatic heart disease suggests a rheumatic etiology.1,11 The association of a mitral diastolic murmur favors a diagnosis of rheumatic heart disease although mitral diastolic flow murmurs are frequently noted in association with papillary muscle dysfunction or chordae tendineae rupture. Since the differentiation of these conditions is not always possible by clinical means, left ventriculography is indicated. Clinical symptoms, hemodynamic abnormalities, and observations during volume angiography determine the need for surgical intervention in patients with mitral insufficiency. However, the prognosis of the patient and the need for immediate surgery may be influenced by the etiology of the mitral valve lesion. For example, in papillary muscle dysfunction and chordae tendineae rupture the severity of left ventricular dysfunction will be the primary factor limiting long-term survival, and the magnitude of the regurgitant jet will determine the need for immediate or early treatment. On the other hand, in rheumatic mitral insufficiency left ventricular function, left atrial pathology, and the duration and severity of pulmonary vascular disease all contribute to prognosis. Surgery is often delayed as long as possible to avoid the morbidity associated with prosthetic valves or the as yet unknown fate of homografts and heterografts. For these reasons, radiologic differentiation of the various etiologies of mitral insufficiency is important.

In summary, high-quality left ventricular cineangiography permits an etiologic evaluation of patients with mitral insufficiency when the clinical and routine laboratory studies are not definitive. Catheterization and angiography appear indicated in all patients with mitral insufficiency when surgical intervention is being considered. With use of the techniques of analysis outlined it should be possible to determine the precise etiology in most patients.

References


Circulation, Volume XLIV, December 1971


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Circulation. 1971;44:1080-1086
doi: 10.1161/01.CIR.44.6.1080
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1971 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/44/6/1080

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