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VALVULAR HEART DISEASE

Ischaemic mitral regurgitation: mechanisms and diagnosis

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Ischaemic mitral regurgitation (MR) is defined as MR caused by changes of left ventricular structure and function related ultimately to ischaemia. However, the acute manifestation of MR following infarction (which usually presents as a haemodynamic crisis) is related to rupture or stretching of the papillary muscle, and is normally categorised with complications of infarction. The term ischaemic MR is usually understood to relate to chronic MR, occurring >2 weeks after infarction and in the absence of structural mitral valve disease. In terms of pathogenesis, this should be considered a disease of abnormal left ventricular (LV) shape and function with a valvular manifestation.

The frequency in ischaemic MR varies according to the technique used for its detection (being more common in echocardiographic than angiographic studies), the management of the patients (more common in non-revascularised patients), the timing post-myocardial infarction (MI) (more common early, before medical treatment is optimised), and infarct size. These selection influences were avoided in the study of a geographically defined MI incidence cohort in which MR was identified at 30 days in 50% of 773 patients, 12% of which were moderate or severe, and in whom the detection by physical examination was unreliable.¹

Ischaemic MR is not only a common but also a serious finding. The community based study of ischaemic MR among 30 day survivors of MI showed moderate or severe MR to be associated with a threefold increase in the risk of heart failure and a 1.6-fold increased risk of death at 5 year follow-up independent of age, gender, ejection fraction (EF), and Killip class.¹ Mortality was increased even with mild MR.²

MECHANISMS OF ISCHAEMIC MR

The mechanism of ischaemic MR requires a combination of both leaflet tethering and reduction of closing forces (table 1). The difficulty posed by separating the role of each component has been addressed by in vitro work suggesting that apical and posterior displacement of the papillary muscles are the dominant features. These effects are all amplified in the setting of a dilated annulus.³

Valvular and subvalvular changes

Disturbances of posterior leaflet displacement (asymmetric pattern)

The classic pattern of ischaemic MR involves a posterior wall motion abnormality with regional

remodelling, leading to posterolateral and apical displacement of the (posterior) papillary muscle, the apical component of which appears to be the most important. This regional remodelling appears to be related to the regional scar burden.² As the papillary muscle contributes chordae to both leaflets, the consequences are: (1) displacement of the posterior leaflet posteriorly; (2) development of a hockey stick deformity of the anterior leaflet due to tethered secondary chordae; and (3) displacement of the mitral coaptation point posteriorly (causing an asymmetric shape). The consequence is anterior leaflet override with a posterior MR jet (fig 1). Usually this arises from the centre of the valve, but the process may disproportionately involve the medial commissure (fig 2), perhaps reflecting papillary muscle elongation.

Left ventricular dilatation (symmetric pattern)

Global enlargement of the left ventricle (LV) usually occurs in association with increased sphericity and annular dilatation, and all contribute to MR. The coaptation point of the MV is moved apically, with a large tenting area,⁴ and both leaflets are involved to a similar degree, causing a central jet (fig 3). This circumstance is typically associated with large infarct size—typically an anterior infarction or multiple infarctions.⁵ Asymmetric and symmetric mechanisms may become mixed, as remodelling of the LV with MR due to papillary muscle displacement may add global to regional remodelling.

Annular dilatation

Annular dilatation and flattening is a frequent accompaniment to global LV remodelling. Loss of the typical saddle shape of the annulus reduces leaflet curvature and thereby increases leaflet stress.⁶ However, patients with lone atrial fibrillation, who develop mitral annular dilation in the absence of ventricular abnormalities, show less MR than those with dilated cardiomyopathy, even after correction for annulus size.⁷ The normal mitral leaflet area is more than double the area of the annulus, indicating a significant reserve before annular enlargement leads to non-coaptation. Annular enlargement is generally uniform, and experimental work has indicated annular enlargement >1.75-fold would be required to engender MR.⁸ Thus, the independent contribution of annular enlargement to ischaemic MR has perhaps been overstated. Its role appears to be adjunctive,

Table 1 Typical features of asymmetric and symmetric patterns of ischaemic mitral regurgitation

	Asymmetric	Symmetric
▶ Leaflet tethering	PML tethered towards posterior wall	Both tethered towards apex
▶ MR jet direction	Eccentric, posterior	Usually central
▶ Tenting area	Increased	Greatest
▶ Annulus	Minor change	Dilated and flattened
▶ LV remodelling	Regional	Global
▶ MI site	Inferior	Anterior/multiple
▶ CAD extent	RCA/Cx	Multivessel

CAD, coronary artery disease; Cx, circumflex coronary artery; LV, left ventricular; MI, myocardial infarction; MR, mitral regurgitation; PML, posterior papillary muscle; RCA, right coronary artery.

with evidence that this increases MR at different levels of papillary muscle displacement.³ The exception to this statement relates to basal inferoposterior infarcts, which may cause asymmetric annular dilatation.⁹

Papillary muscle discoordination

The papillary muscles are the main contributors to acute ischaemic MR. Although traditionally incriminated in chronic ischaemic MR, the importance of associated regional LV dysfunction has been recognised for nearly 40 years.¹⁰ In rare instances, papillary muscle scarring may lead to elongation and mitral prolapse.

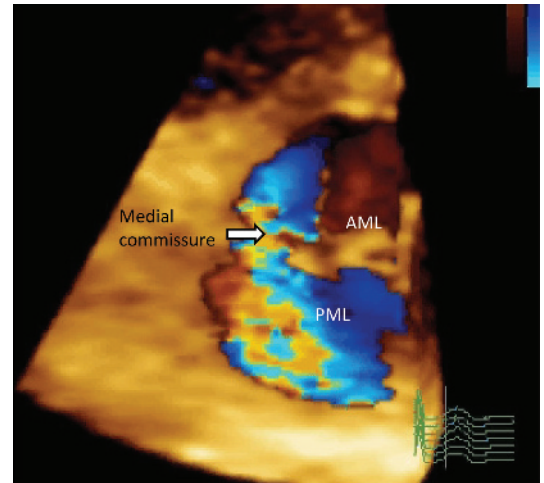
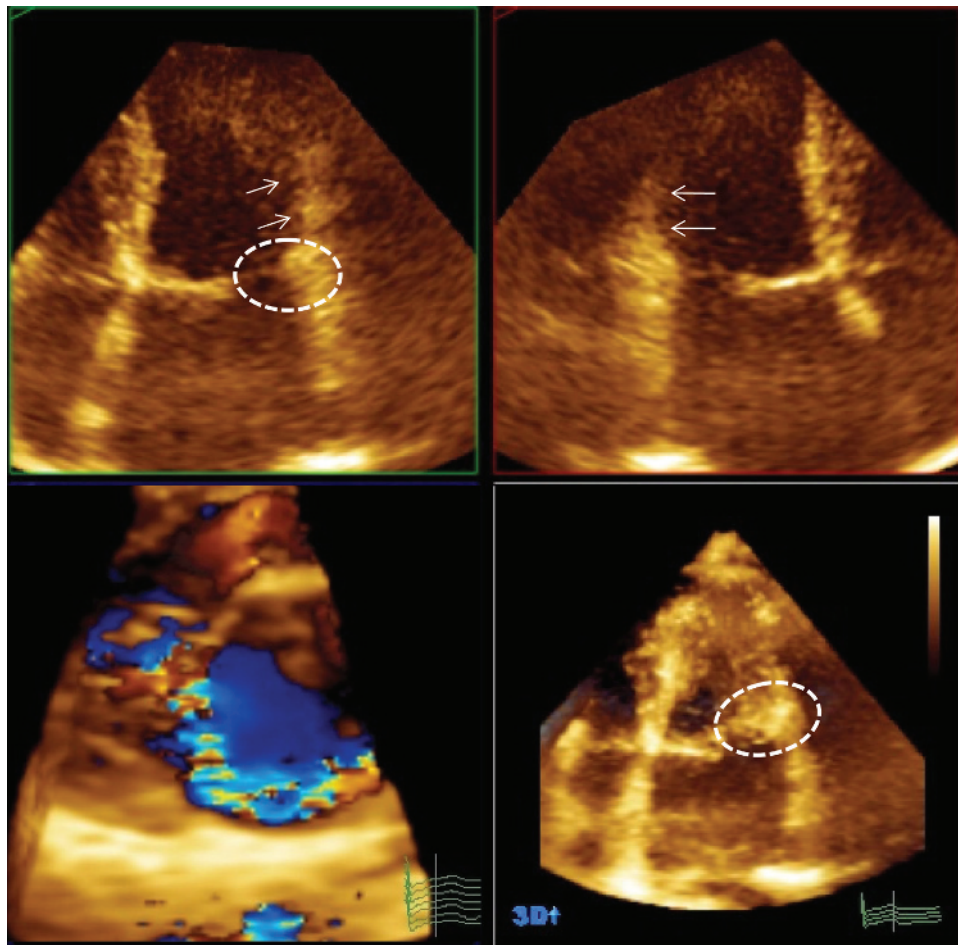


Figure 2 Ischaemic mitral regurgitation with disproportionate involvement of the medial commissure. AML, anterior mitral leaflet; PML, posterior mitral leaflet.

The contribution of papillary muscle dyssynchrony has been more recently recognised as an important component of LV dyssynchrony. In left bundle branch block (LBBB), mechanical activation occurs first in the segment adjacent to the posterior papillary muscle and is delayed in the segment with the anterolateral papillary muscle insertion.

Figure 1 Ischaemic mitral regurgitation (MR) following inferoposterior myocardial infarction. End systolic freeze frames in apical views from a three dimensional echocardiogram (lower right) show how posteriorly directed ischaemic mitral regurgitation arises from remodelling of the posterolateral wall, with displacement of the posterior papillary muscle (arrows). The resulting apical tethering of the posterior leaflet (dashed circle) causes posteriorly directed MR (lower left).



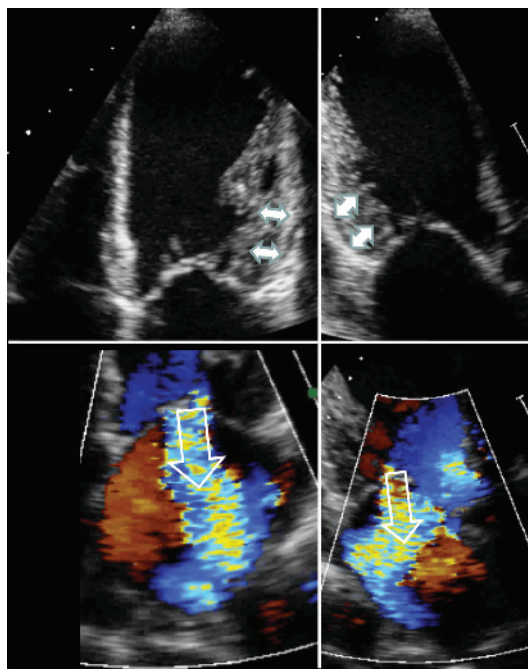


Figure 3 Ischaemic mitral regurgitation (MR) following left anterior descending (LAD) and right coronary artery (RCA) territory infarctions—end-systolic freeze frames show the septum is thinned but there is preserved thickening in the posterior wall (double headed arrows). Left ventricular remodelling has caused displacement of both papillary muscles with failure of coaptation of the mitral leaflets and central MR (open arrow).

With cardiac resynchronisation therapy (CRT), the interpapillary muscle activation time delay is shortened and its magnitude of change, which varies substantially from patient to patient, is significantly correlated with the decrease in mitral regurgitant fraction.¹¹ Reductions in this interpapillary activation delay (fig 4) correlate well with reduction of MR following CRT.

Closing forces

Impairment of LV contractile function is associated with all of the morphological changes listed above. However, it is unusual for LV dysfunction to cause MR in the absence of LV remodelling.³ The severity of ischaemic MR is a dynamic balance between tethering and closing forces (table 2).¹² These interactions result in potentially significant variation of ischaemic MR over time (fig 5). The presence of only mild global dysfunction with preserved closing forces may attenuate the severity of MR that might otherwise arise from the regional changes that cause non-coaptation. Treatments (myocardial revascularisation, CRT, medical therapy, and exercise) which influence closing pressure may significantly modulate ischaemic MR.

Myocardial revascularisation

Revascularisation does not necessarily reverse ischaemic MR. In a study of coronary bypass surgery in 136 patients with moderate ischaemic MR, half showed an improvement in MR, but

resolution of MR occurred in only 9%, and 40% had residual moderate or severe MR.¹³ The effects of revascularisation on closing pressure are likely to depend on the presence and extent of viable myocardium. There is a link between the amount of scarring (defined by contrast enhanced magnetic resonance imaging (MRI)) and the severity of MR,¹⁴ and one study showed that improved posterior wall function after thrombolysis was associated with a lower frequency of MR.¹⁵ The role of viable myocardium in ischaemic MR is probably important but unproven.

CRT

The effects of CRT may not relate purely to papillary muscle resynchronisation, and also relates to improvement in dP/dt with CRT therapy.¹⁶ This contribution to improved closing forces is evidenced by the acute haemodynamic effect of CRT, before the occurrence of reverse remodelling. The acute decrease in mitral effective regurgitant orifice is directly correlated to the increase in LV systolic performance (dP/dt), which counteracts the increased tethering forces that impair mitral valve competence.^{16 17} In addition, diastolic MR is relieved by shortening atrioventricular (AV) delay.¹⁸ After successful CRT implantation, MR improves in most patients, remains unchanged in about 25%, and worsens in a relatively small number of patients.

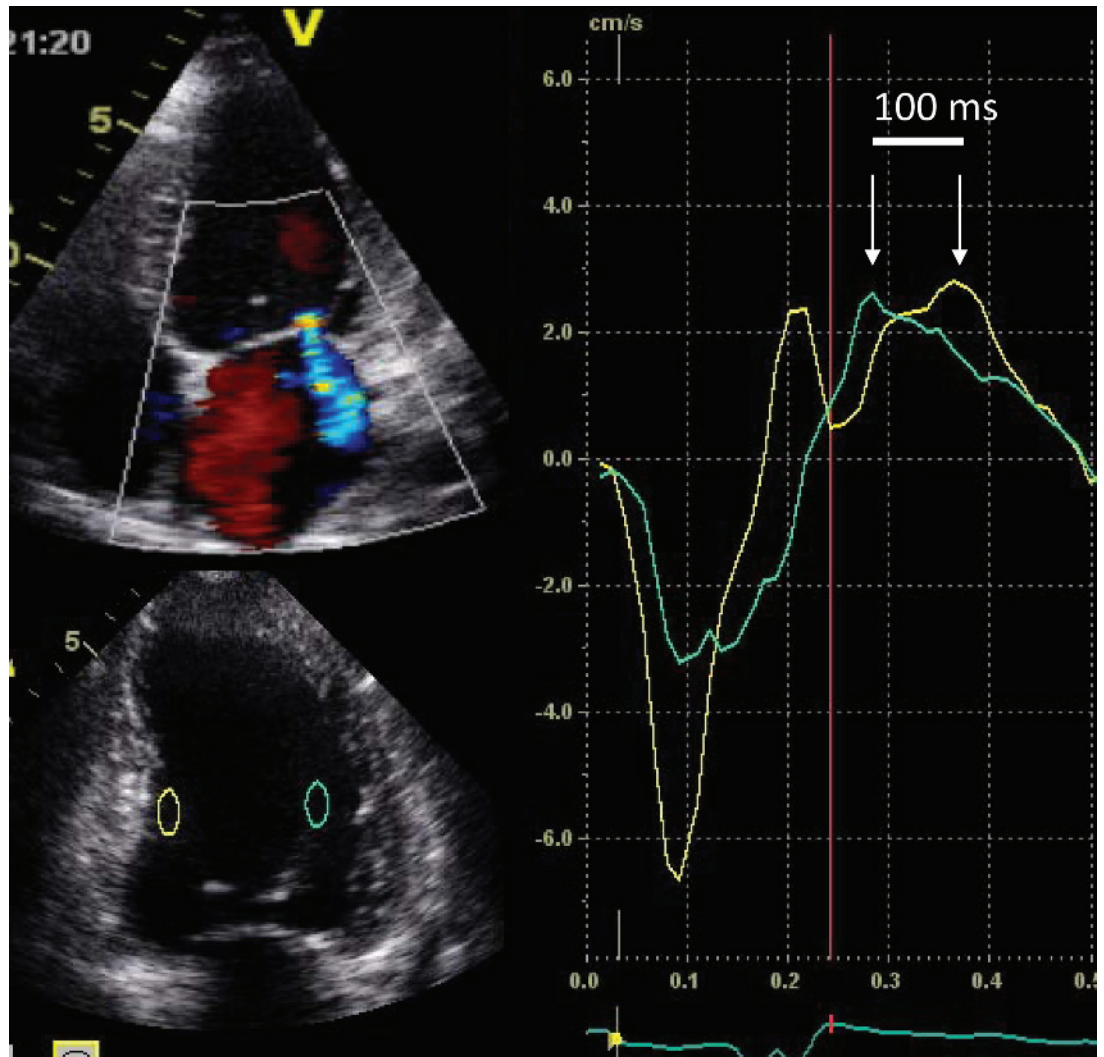
Medical treatment

Treatment is an important contributor to the variation of ischaemic MR over time (fig 5). Dobutamine, which is widely used in the acute setting to support LV dysfunction, rarely provokes MR and more commonly causes no change or reduces the severity of MR, both in groups with impaired and preserved LV function.¹⁹ Other medical treatments have not been well studied. Most patients can be expected to show a reduction of MR in response to vasodilation, but these results were heterogenous and no more associated with dynamic than fixed regurgitant valves.²⁰

Exercise

Exercise is an important cause for variability of ischaemic MR, irrespective of the baseline severity of MR.²¹ The normal response to exercise involves vasodilation (reducing tethering forces), global contractile reserve (increasing closing forces), and regional contractile reserve (normalising function or shape in the posterior wall). However, ischaemia, a hypertensive response to exercise or loss of contractile reserve may all lead to worsening MR with exercise.²¹ An increase of effective regurgitant orifice (ERO) ≥ 13 mm² carries adverse prognostic implications,²² possibly through recurrence of this process causing remodelling, or exercise induced heart failure. The determinants of exercise induced changes of MR include systolic annular area, tenting area, wall motion score, and the degree of apical displacement of the mitral valve.

Figure 4 Inter-papillary activation delay, shown with colour tissue Doppler.



DIAGNOSIS OF ISCHAEMIC MR

The clinical signs of MR and its severity are unreliable in ischaemic MR. Irrespective of imaging technique, the report needs to address findings related to LV morphology and ejection fraction, mitral valve morphology, and the severity of MR, as these features govern the indications for surgery in the current guidelines.²³

LV morphology and function

As ischaemic MR is fundamentally a ventricular disease, the global report should include observations about LV volume and shape (including sphericity). The regional evaluation should include comments about wall thinning, wall motion abnormalities (and if possible, their contractile

reserve), and perhaps a marker of scar thickness (using strain or contrast).

The role (if any) of measuring LV synchrony is controversial. Current guidelines for CRT are based on symptom status, LV function and QRS duration, and the reliability of current measures of mechanical synchrony do not seem to justify their incorporation into decision making.²⁴ On the other hand, observational studies demonstrate an improvement in moderate ischaemic MR and mitral tethering with CRT,²⁵ proportionate to the degree of inter-papillary delay, measured in the short axis view.

Mitral valve morphology

The mitral findings should include consideration of the morphology of the valve as well as MR jet direction. A posterior directed jet is usually associated with eccentric valve involvement, while a central jet marks symmetrical involvement (table 1).

Despite the normal structure of mitral leaflets in ischaemic MR, the displacement of the leaflets into the LV leads to a number of characteristic changes of valve morphology. Annular dimension should be <35 mm (in the anteroposterior axis). A number

Table 2 Tethering and closing forces in ischaemic mitral regurgitation

Tethering forces	Closing forces
▶ Papillary muscle displacement	▶ Reduced LV contractility
▶ LV dilatation and sphericity	▶ LV dyssynchrony
▶ Annular dilatation	▶ Papillary muscle dyssynchrony
	▶ Reduced annular contraction

LV, left ventricle.

Figure 5 Temporal variation of ischaemic mitral regurgitation (MR) severity. This patient presented following a posterior wall myocardial infarction, and at the time of presentation had severe MR with a prominent tenting angle. Following transfer to our facility, in the course of which the patient was treated with inotropes and left ventricular volumes decreased, the MR almost resolved with reduction of tenting angle. After a percutaneous intervention she still had moderate mitral regurgitation, emphasising the role of inotropes in acutely reducing ischaemic MR.

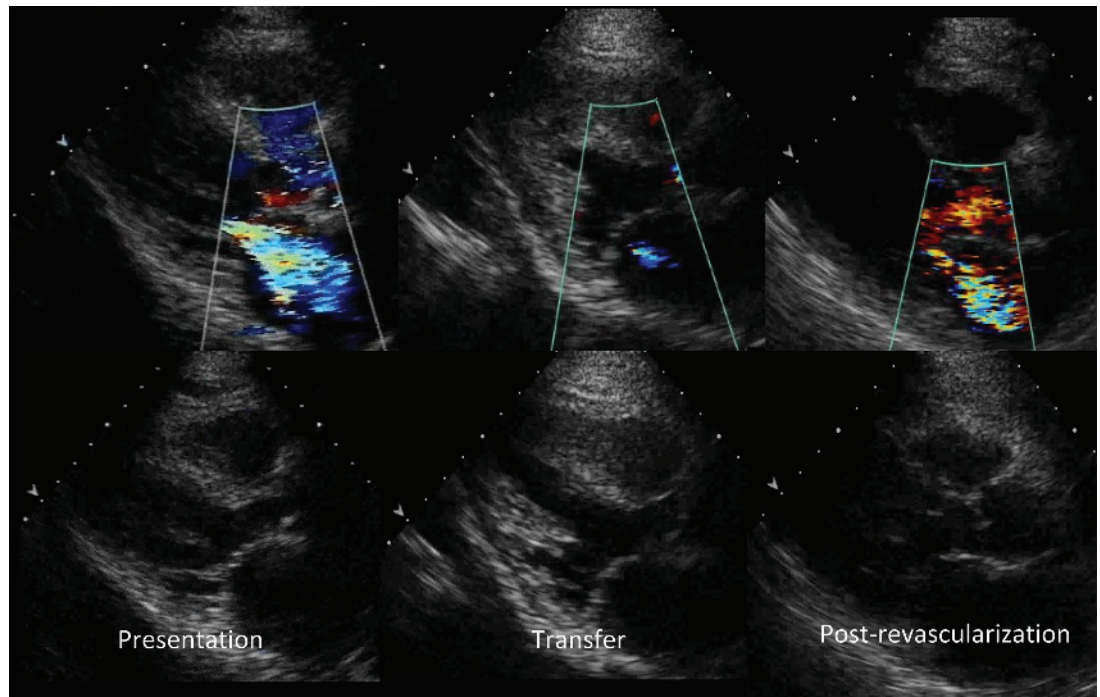
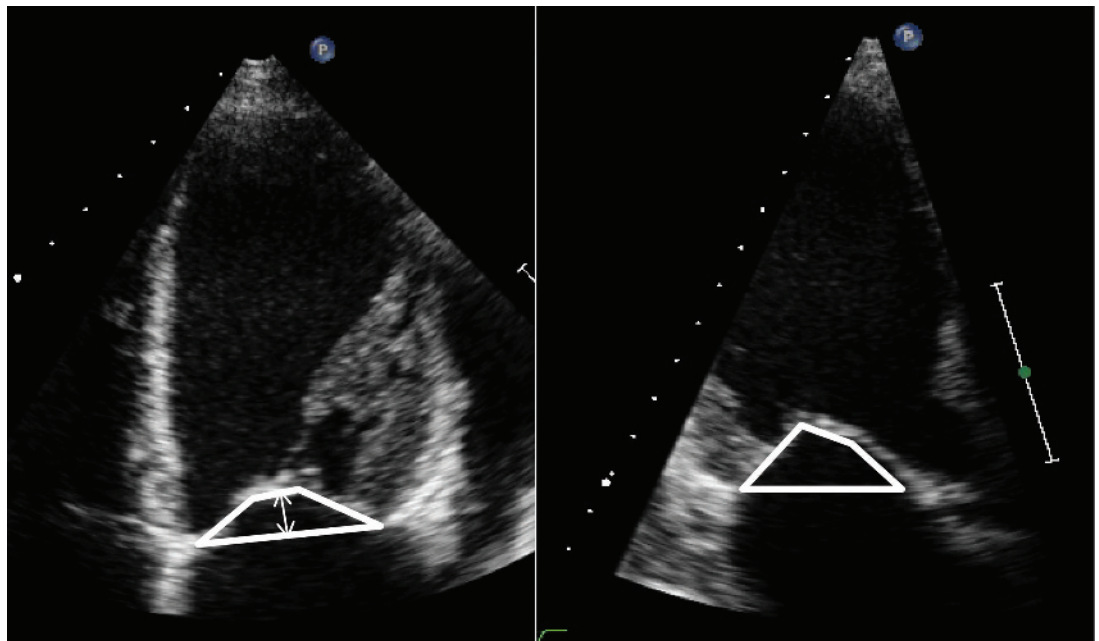


Figure 6 Measurements of abnormal mitral morphology in ischaemic mitral regurgitation. Tenting area 2.6 cm^2 , coaptation distance 1 cm , angle 35° .



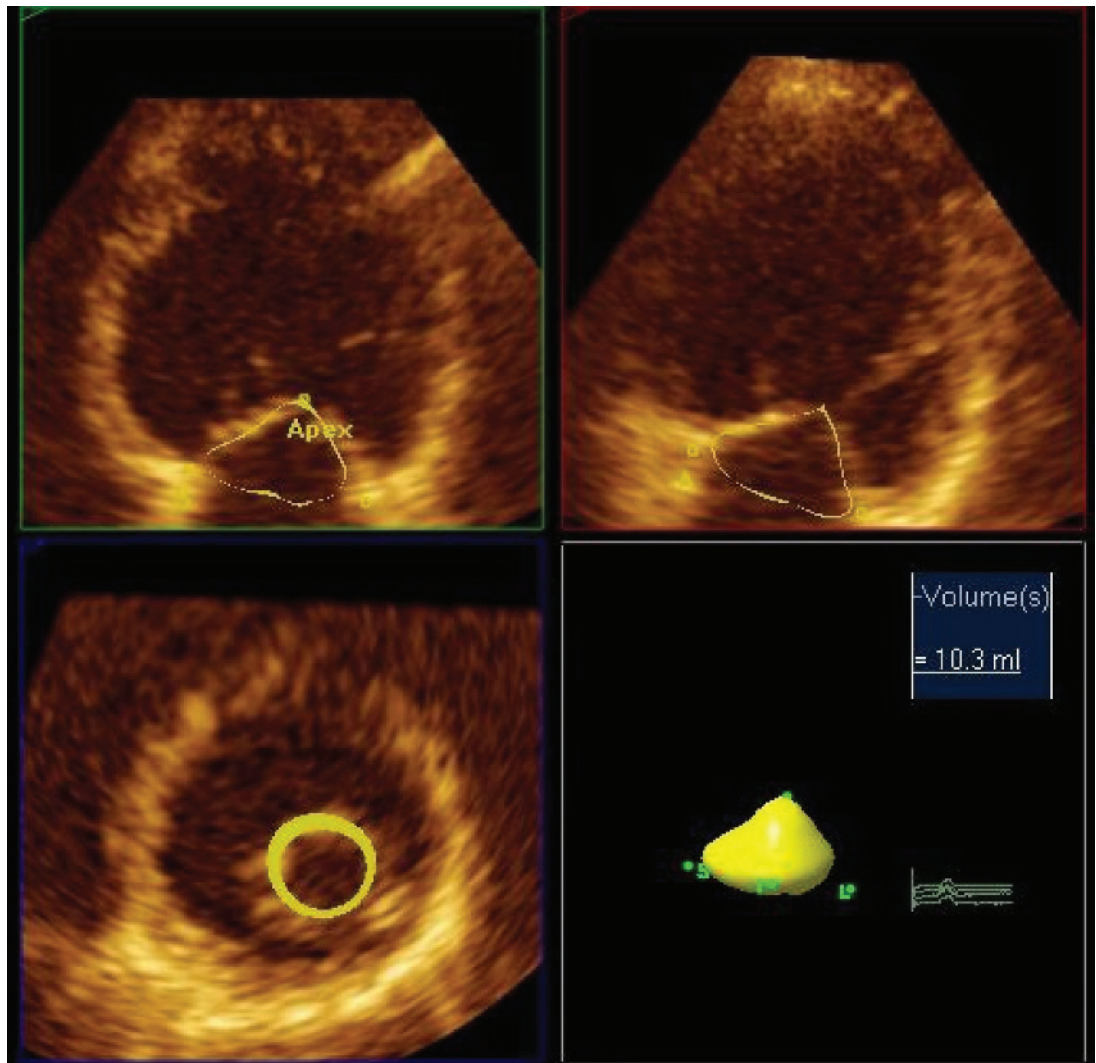
Important contributors to ischaemic MR

- ▶ Structural
 - Disturbances of posterior leaflet displacement (asymmetric pattern)
 - Left ventricular dilatation (symmetric pattern)
 - Annular dilatation
- ▶ Functional
 - Papillary muscle discoordination
 - Closing pressure: viability, ischaemia, medical treatment, exercise

of anatomic measurements can be made that reflect the pathophysiology of ischaemic MR, including tenting area, leaflet angles, coaptation depth, bending distance and leaflet length (fig 6). Greater degrees of morphologic disturbance are predictive of greater likelihood of persistence of MR following mitral annuloplasty, with the optimal cut-offs for distinguishing patients with persistent MR being a coaptation distance of $>0.6 \text{ cm}$, a tenting area of $>2.5 \text{ cm}^2$, or a posterior leaflet angle $>45^\circ$.²⁶ Measures of tenting volume

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Figure 7 Application of three dimensional echocardiography to the measurement of tenting volume.



are now readily available using three dimensional echocardiography (fig 7).

These features of mitral valve configuration may vary from time to time according to loading conditions, and it is not clear as to whether they provide incremental information to standard Doppler measures for the assessment of MR severity.

The imaging report in ischaemic MR

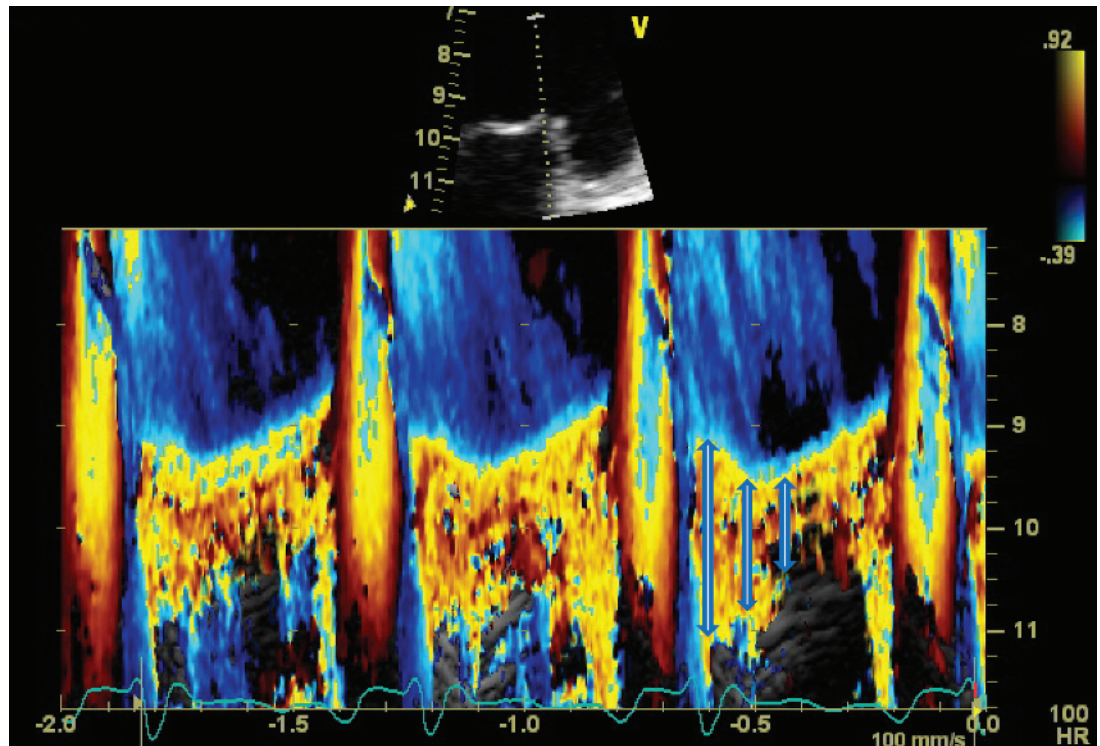
- ▶ Left ventricular (LV) morphology and function
 - LV volume and shape (including sphericity)
 - Wall thinning, wall motion abnormalities
 - Contractile reserve
 - Scar thickness
 - LV synchrony (controversial)
- ▶ Mitral valve morphology
 - MR jet direction (eccentric vs symmetrical LV dysfunction)
 - Annular dimension
 - Tenting area, leaflet angles, coaptation depth
- ▶ Severity
 - Beware inhomogeneity of MR and variability and complexity of jet morphology

Severity

MR severity may be difficult to assess in ischaemic regurgitation. In part, this relates to variations in MR severity during systole. Typically, the severity of MR is greatest at the beginning and end of systole and least in mid-systole, when the LV pressure is maximal, LV volume is reduced, and the mitral leaflets are pushed back into the annular plane (fig 8).²⁷ This phenomenon has important implications with respect to the use of regurgitant orifice area as an index of MR, implying that this should be averaged through systole. The use of volumetric methods for calculating regurgitant volume may also avoid errors due to inhomogeneity of MR. Measurement of vena contracta width may be a preferable means of assessing MR severity, although this is equally influenced by changes in LV pressure and dimensions. There is evidence that three dimensional echocardiography is superior to two dimensional techniques for the measurement of vena contracta, especially with eccentric jets.²⁸

The second contributor to the difficulty of assessing MR severity in ischaemic MR relates to the variability and complexity of jet morphology. Irregularly shaped and multiple jets, caused by a

Figure 8 Non-uniform progression of ischaemic mitral regurgitation through systole (arrows demonstrate narrowing of the vena contracta). Accurate quantitation requires averaging at multiple time points.



variety of mechanisms,²⁹ pose problems for the proximal isovelocity surface area approach.⁴ It is important to remember that relatively moderate ischaemic MR has an adverse prognosis, and therefore particular caution should be paid to recognition of moderate lesions.

CONCLUSIONS

Ischaemic MR is relatively common and carries an adverse prognosis. Although therapeutic strategies are currently limited, recognition of the predominant mechanisms may in the future allow rational therapies, targeted towards specific mechanisms.

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