MRI findings of functional ischemic mitral regurgitation: When, Why and How?

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Learning objectives

- To illustrate with sketches and MR images some geometrical left ventricular (LV) and mitral apparatus mechanisms that have been implicated in the pathogenesis of functional ischemic mitral regurgitation.
- To describe a systematic approach to evaluate functional ischemic mitral regurgitation using MR imaging.
Background

Ischemic mitral valve regurgitation is a condition with a complex and controversial diagnostic-therapeutic pattern and, for this reason, is often associated with uncertain evolution and prognosis. The clinical definition of ischemic mitral regurgitation implies a situation where mitral valve insufficiency occurs after myocardial infarction in the presence of structurally normal mitral valve leaflets and subvalvular apparatus.

The left ventricular papillary muscles are particularly vulnerable to ischemia due to their perfusion by the terminal portion of the coronary arteries. Any vascular disturbance results in papillary dysfunction, particularly in the posterior papillary muscle which is supplied by the posterior descending branch of the right coronary artery. The anterolateral papillary muscle is less frequently infarcted than does the posterior papillary muscle. This difference is probably due to the fact that the anterolateral papillary muscle receives a double supply by diagonal branches of the left anterior descending coronary artery and by marginal branches from the left circumflex artery.

The anatomical and pathophysiological mechanisms responsible for functional ischemic mitral regurgitation are complex and not fully clarified. A number of mechanisms have been invoked in its pathogenesis, including alterations of papillary muscle position, annular dynamics, and intraventricular synchrony, that progressively worsen mitral regurgitation.

The role of imaging methods in decision making concerning this functional regurgitation is fundamental. Comprehensive assessment of ischemic mitral regurgitation requires to understand its pathophysiology. An accurate evaluation of its severity and of the mechanism of the dysfunction is needed to determine the timing for surgical intervention and the type of surgical intervention required. Furthermore, LV volumes and function are necessary to determine the timing and risks of surgery and LV viability.

Ideally, a comprehensive imaging modality should address all information needed in a single imaging session. Such comprehensive assessment is feasible in a single MRI examination but needs a defined protocol, as described with sketches and MR images in this paper.
PATHOPHYSIOLOGY
The mitral apparatus is a complex mechanism that requires functional integrity of all the anatomical elements that form it. Any structural distortion that alters the coordination between them can cause regurgitation.

The main pathophysiological mechanisms proposed in functional ischemic mitral regurgitation include the LV remodeling, geometrical alterations of papillary muscles and mitral valve leaflets and left ventricular dilatation.

Left ventricular remodeling, papillary muscles and valve leaflets
Myocardial infarction of the segments underlying the papillary muscles (typically a lateral or inferior infarct) results in remodeling of that region of the ventricle (Figure 1). This increase of LV sphericity results in distortion of ventricular geometry and led to a retraction of the attachments of the mitral leaflets toward the apex at systole restricting their ability to close effectively at the annular level which results in incomplete closure and finally in mitral regurgitation.
**Fig. 1:** Sketches of left ventricular (LV) remodeling. The majority of patients with ischemic mitral regurgitation have an etiologic basis of prior myocardial infarction (left), not an acute myocardial infarction or papillary ischemic event. Resulting wall motion abnormalities and LV remodeling leading to lateral and apical displacement of papillary muscles (right) are the key pathophysiologic events.

**References:** - A Coruña/ES

**Geometrical alterations of papillary muscles and mitral valve leaflets**

- *Tethering height* is defined as the shortest distance during systole from the coaptation point of the anterior and posterior mitral leaflets to the mitral annular plane (Figure 2).

- *Tenting area* is the smallest area during systole bounded by the leaflets and the mitral annular plane (Figure 2).

**Fig. 2:** Sketches depict closing and tethering forces in the normal ventricle (left) and after inferior myocardial infarct (middle). In the normal ventricle, the mitral leaflets reach the annular plane during systole. Papillary muscle displacement after infarction increases tethering forces, which pull the mitral leaflets away from the annular plane resulting in incomplete mitral leaflet closure in areas supported by the posterior papillary muscle (right).

**References:** - A Coruña/ES

Apical, posterior and lateral shift of papillary muscles secondary to LV remodeling causes:

- An increase of tethering forces on both leaflets.
- An increase of tenting area.
- Decrease of coaptation length and area

Both, the increased tenting area with decreased coaptation surface lead to mitral regurgitation in areas supported by the posterior papillary muscle (Figure 2).
Papillary muscle contractile dysfunction can paradoxically decrease mitral regurgitation from inferobasal ischemia by reducing leaflet tethering to improve coaptation (Figure 3).

**Fig. 3**: Sketches showing (left) inferobasal infarction with papillary muscle still functioning, causing mitral regurgitation by outward distortion of the inferior base. Extension of ischemic zone to papillary muscle can paradoxically diminish mitral regurgitation by reducing tethering so that leaflets can seat better at annular level (right).

**References**: - A Coruña/ES

**Left ventricular dilatation**
LV volume overload secondary to mitral regurgitation and ventricular dysfunction leads to increase LV dilation.

The LV dilatation causes:
- Mitral annular dilation and progressive loss of coaptation over the entire surface of the valve leaflets.

This pathophysiological process then becomes self-perpetuating with progressive deterioration of ventricular function and a degree increasing of ischemic mitral regurgitation.

**STUDY PROTOCOL**
Steady-state free precession (SSFP) cine MRI
Obtained at short-axis multislice-multiphase (Figure 4), two and four-chambers (Figure 5).

**Fig. 4**: Short-axis SSFP cine MR image. To assess and to quantify global and regional left ventricular function.

*References*: A Coruña/ES
Fig. 5: Two (left) and four-cambers (right) SSFP cine MR images at mitral atroventricular plane. Qualitative analysis of mitral valve motility and mitral regurgitation degree and measurements of geometrical parameters.

References: - A Coruña/ES

Utility
- Analysis and quantification of global and regional cardiac function.
- Qualitative analysis of mitral regurgitation degree and mitral valve motility.
- Measurements of geometrical parameters in the mitral atroventricular plane.

Velocity encoded cine MRI
At short-axis view (magnitude and phase images) through mitral atroventricular and ascending aorta planes (Figure 6).
Fig. 6: Magnitude (left) and phase (right) velocity-encoded MR images in short-axis view at mitral atrioventricular plane (upper) and in axial plane through ascending aorta (down) to quantitative analysis of mitral regurgitation degree.

References: A Coruña/ES

Utility
- Quantitative analysis of mitral regurgitation degree (regurgitant volume and regurgitant fraction).

Either with a direct method through the mitral atrioventricular plane or with an indirect methods (that in some times could be more consistent), based on the measure of the regurgitant mitral volume subtracting the forward flow through the ascending aorta from the left ventricular stroke volume obtained with volumetric calculation.

First-pass perfusion MRI
Obtained after intravenous administration of 0.05 mmol/kg of a gadolinium-based contrast agent, at a rate of 4-5 ml/s in short-axis view (Figure 7).

**Fig. 7**: Short-axis first-pass perfusion MR image showing subendocardial perfusion defect of apical inferior and lateral segments.

**References**: - A Coruña/ES

**Utility**
- Analysis of ischemic perfusion defects at rest.

Delayed myocardial enhancement
At 10 minutes after intravenous administration of 0.2 mmol/kg of a gadolinium-based contrast agent in short-axis, two and four-chambers (Figure 8).

Fig. 8: Short-axis (left), two (middle) and four-chambers (right) post-gadolinium delayed MR images showing delayed myocardial enhancement and myocardial viability.

References: - A Coruña/ES
Utility
- Analysis of myocardial viability
- Detection of myocardial necrosis

QUANTIFICATION OF MORPHOLOGICAL PARAMETERS

Left ventricular remodeling
Left ventricular remodeling is obtained from longitudinal and transverse diameters of left ventricle.

Longitudinal diameter (L) in systole and diastole (Figure 9).
- Distance between the tip of the left ventricle and the middle third of the mitral annulus (two-chamber).

Mid-ventricular transverse diameter (T) in systole and diastole (Figure 9).
- Distance between the anterior and septal wall (two-chamber) and septal and lateral wall (four-chamber).
**Fig. 9:** Two-chamber diastolic (upper left) and systolic (upper right) and four-chamber diastolic (down left) and systolic (down right) SSFP cine MR images showing as to obtain the longitudinal (L) and mid-ventricular transverse diameters (T).

**References:** - A Coruña/ES

*Left ventricular sphericity index (%) = (LV longitudinal diameter / LV transverse diameter) x 100.*

The site of LV remodeling, more than its extent, is the more important determinant of whether ischemic mitral regurgitation will develop.

LV dilatation, even when marked, may not cause mitral regurgitation unless accompanied by geometric distortion in the region of the papillary muscles. This explains the high
prevalence of ischemic mitral regurgitation in patients with localized infarction of inferior wall.

**Papillary muscle displacement**
Measurements are obtained during systole at two and four-chambers:

*Anterior papillary muscle displacement (Figure 10)*
- Distance from posterior papillary muscle head to anterior mitral annular commissure (A).

![Fig. 10](image)

**Fig. 10:** Two (left) and four-chambers (right) SSFP cine MR images obtained at systole show the measurement of anterior papillary muscle displacement (A) as the distance from posterior papillary muscle head to anterior mitral annular commissure.  

**References:** - A Coruña/ES

*Posterior papillary muscle displacement (Figure 11)*
Distance from posterior papillary muscle head to posterior mitral annular commissure (B).
Fig. 11: Two (left) and four-chambers (right) SSFP cine MR images obtained at systole show the measurement of posterior papillary muscle displacement (B) as the distance from posterior papillary muscle head to posterior mitral annular commissure.

References: - A Coruña/ES
Ischemic mitral regurgitation is proportional to the degree of deformity of the mitral valve complex, especially the outward displacement of the papillary muscles, rather than to global LV dilatation.

Mitral annular diameter
MRI measurements can be derived in two and four-chamber views and should be performed during systole and diastole to document the sphincter function of the mitral valve annulus.

The slice obtained should represent a true diameter of the mitral annulus and should not be taken off center to prevent underestimation of the real value.

The distance between the anterior and posterior mitral annulus (MA) during systole and diastole are the mitral annular diameters (Figure 12).
**Fig. 12**: Two-chamber diastolic (upper left) and systolic (upper right) and four-chamber diastolic (down left) and systolic (down right) SSFP cine MR images showing as to measure the mitral annular diameters (MA).

**References**: A Coruña/ES

The mitral annulus has been the prime focus for surgical repair techniques.

**QUANTIFICATION OF FUNCTIONAL PARAMETERS**

**Global cardiac function**

Left ventricular ejection fraction (LVEF) is a valuable parameter in patients with ischemic mitral regurgitation. LVEF may be easily derived by MRI values of LV end-systolic and LV end-diastolic volumes.
One of the major advantages of MRI over other conventional imaging techniques in the study of ischemic mitral regurgitation, is its ability to quantify global LV function without assuming an elliptical shape of the ventricle.

To avoid the assumption of the LV elliptical shape, measurements of LV end-systolic and LV end-diastolic volumes should be derived from a series of contiguous short axis slices (Figure 13) of cine-MRI using the Simpson rule (volume = sum of the areas drawn multiplied by the thickness and the cutting interval). Stroke volume, ejection fraction and cardiac output are obtained from LV volumes.

**Fig. 13**: Short-axis SSFP cine-MR images from base to apex of left ventricle to measure systolic, diastolic and stroke volumes, ejection fraction and cardiac output using the Simpson rule.

**References**: - A Coruña/ES

There is a direct relationship between increased LV end-systolic volume and the geometric parameters that influence the development of ischemic mitral insufficiency and an inverse relationship with ejection fraction.

**Regional cardiac function**

**Qualitative analysis**

According to current nomenclature, the left ventricle is divided in 17 segments, as follows (Fig. 14):

- Short-axis. Basal ventricular six segments (anterior, anteroseptal, inferoseptal, inferior, inferolateral, and anterolateral), middle ventricular six segments (anterior, anteroseptal, inferoseptal, inferior, inferolateral, and anterolateral) and apical four segments (anterior, septal, inferior and lateral).

References: - A Coruña/ES

Values are assigned to every segment on the basis of the segmental kinesis: score 1 for normokinesis, 2 for hypokinesis, 3 akinesis, 4 diastolic dyskinesis, 5 systolic dyskinesis (Figure 15).
Fig. 15: Apical short-axis SSFP cine-MR images obtained at diastole (left) and systole (right) show systolic dyskinesis (arrows) of anterior and septal segments.

References: - A Coruña/ES
Scores are added up to obtain a global score that is divided by the number of investigated segments to generate a wall motion score index. A normally contracting ventricle with 17 well functioning segments should have a wall motion score indexing of 1.

Quantitative analysis
Quantitative analysis is obtained by calculating the rate of myocardial thickening using the following formula:

\[
Rate \ of \ thickening \ (\%) = \frac{\text{systolic thickness} - \text{end-diastolic thickness}}{\text{end-diastolic thickness}} \times 100
\] (Figure 16).

Fig. 16: Short-axis SSFP cine-MR images from base to apex of left ventricle show diastolic and systolic thickness to calculate the rate of myocardial thickening (end-systolic thickness - end-diastolic thickness) / end-diastolic thickness x 100).

References: - A Coruña/ES
Mitral valve function

Qualitative analysis

Four-chambers SSSFP cine-MRI at the mitral atrioventricular plane is performed to qualitative assessment of mitral valve regurgitation.

- Mitral valve regurgitation is identified as turbulent blood flow in the left atrium (arrow) during systole (Figure 17).
Fig. 17: Four-cambers SSFP cine MR image at mitral atrioventricular plane showing left atrial turbulent blood flow (arrow) during systole by mitral valve regurgitation. Tricuspid valve regurgitation and pleural effusions also can be seen.

References:
- A Coruña/ES

Quantitative analysis
Short-axis velocity encoded MRI at the mitral atrioventricular plane and through the ascending aorta are obtained to calculate the regurgitant volume and regurgitant fraction.

- Regurgitant volume # 30 ml is a factor indicative of poor prognosis.

Coaptation depth
The coaptation depth is a measure of the degree of leaflet tethering.

**Coaptation depth**

- Maximal distance between the mitral valve annulus plane and the coaptation point (C) at mid systole in the two and four-chambers views (Figure 18).

![Fig. 18: Two (left) and four-chambers (right) SSFP cine MR images obtained at mid systole showing the coaptation depth obtained from the maximal distance between the mitral valve annulus plane and the coaptation point (C).](image)

**References:** - A Coruña/ES
This distance correlates with the presence and severity of ischemic mitral regurgitation.

An objective of therapy for ischemic mitral regurgitation is to reduce the coaptation depth allowing coaptation closer to the annular plane.

**Tenting area**
Tenting area is the maximal area between the anterior mitral valve leaflet, posterior mitral valve leaflet and mitral valve annulus at mid systole in two and four-chambers views (Figure 19).
Fig. 19: Two (left) and four-chambers (right) SSFP cine MR images obtained at mid systole show the tenting area measurement (maximal area between the anterior mitral valve leaflet, posterior mitral valve leaflet and mitral valve annulus).

**References:** - A Coruña/ES
Tenting area is considered a strong determinant of ischemic mitral regurgitation severity.

**Viability and myocardial necrosis**
To identify the myocardial necrosis and viability is important for planning ventricular restoration surgery.

**Quantitative analysis**
Areas of myocardial necrosis and viability are identified in the 17 segments of left ventricle (Figures 14 and 20).
**Fig. 14:** Sketches show on a circumferential polar plot (left), of the 17 myocardial segments and the recommended nomenclature for tomographic imaging of the heart. The short-axis and longitudinal (right) views overlap and complement each other. 

**References:** - A Coruña/ES

**Fig. 20:** Two-chamber (left) and short-axis view (right) post-gadolinium delayed MR images showing transmural enhancement (arrows) at basal inferior and mid inferior segments.

**References:** - A Coruña/ES
Drawing the areas of delayed enhancement (myocardial necrosis) and nulled myocardial signal intensity (viable myocardium), the percentage of myocardial necrosis can be calculated (Figure 21):

**Fig. 21:** Short-axis view post-gadolinium delayed MR image shows transmural enhancement of middle inferior segment (red) and as to calculate the percentage of myocardial necrosis drawing the areas of delayed enhancement (myocardial necrosis) and nulled myocardial signal intensity (viable myocardium).

**References:** - A Coruña/ES

Percentage of necrosis (%) = (# areas segmental necrosis / # segmental areas of viable myocardium) x 100.

There is a direct relationship between myocardial necrosis percentage and mitral regurgitation degree.

**Semiquantitative analysis of posterior papillary muscle necrosis**
The relationship between areas of delayed posterior papillary muscle enhancement and viable papillary muscle can be semi-quantitatively evaluated in each segment using a 4-grade system (Figure 22):

Fig. 22: Short-axis view post-gadolinium delayed MR image shows transmural enhancement (arrow) of posterior papillary muscle (grade III of posterior papillary muscle necrosis) and transmural enhancement of middle inferior segment.

References: A Coruña/ES
Grade 0 = 0%. No delayed enhancement
Grade I = Delayed enhancement less than 50%
Grade II = Delayed enhancement between 50 and 75%
Grade III = Delayed enhancement more than 75%
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Conclusion

To detect and to quantify functional ischemic mitral regurgitation is important because it is one of survival prognostic factors of patients with myocardial infarction.

MRI can detect and quantify objectively many morphological and functional geometric parameters that can contribute to the development of ischemic mitral regurgitation.

Detection and quantification of morphological and functional parameters is also essential to decide the therapy and to choice of appropriate surgical technique.
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