Functional Ischemic Mitral Regurgitation: an Open Debate

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Functional ischemic mitral regurgitation is a complex disorder with a poor prognosis. Although the underlying anatomical and pathophysiological mechanisms are not entirely clear, it is known that postinfarction left ventricular remodeling is the most significant factor in the development of this mitral valve lesion. Echocardiography and magnetic resonance imaging have made significant contributions to clarifying the many mechanisms that progressively worsen mitral regurgitation. There is still controversy about the best surgical approach, particularly with regard to whether to replace or repair the valve, the type and size of prosthesis to be used in mitral annuloplasty, the durability of repair, valve-related complications, and medium-term survival. The early recurrence (≤6 months) of mitral regurgitation after surgery has been reported in 17%-29% of patients. Better understanding of the origin and evolution of functional ischemic mitral regurgitation is necessary to enable the adoption of a more effective surgical approach to this enigmatic valvular disease.

Key words: *Coronary artery disease. Mitral insufficiency. Surgery.*

Insuficiencia mitral isquémica funcional: un debate abierto

La insuficiencia mitral isquémica funcional es una enfermedad compleja de mal pronóstico. Los mecanismos anatómicos y fisiopatológicos causales no han sido totalmente aclarados. El remodelado ventricular izquierdo postinfarto es el principal factor inductor de la lesión mitral. La ecocardiografía y la resonancia magnética han contribuido de manera importante al esclarecimiento de los diferentes mecanismos que progresivamente agravan la regurgitación mitral. El tratamiento quirúrgico óptimo es controvertido, sobre todo en lo relativo a la decisión de sustituir o reparar la válvula, el tipo y el tamaño de la anuloplastia mitral, la durabilidad de la reparación, las complicaciones relacionadas con la válvula mitral y la supervivencia a medio plazo. Recientemente se ha descrito la reaparición precoz (≤ 6 meses) de la regurgitación mitral tras la cirugía en el 17-29% de los pacientes. Es preciso aumentar los conocimientos sobre el origen y la evolución de la insuficiencia mitral isquémica funcional con el fin de que sea posible realizar una cirugía más eficiente para esta enigmática enfermedad valvular.

Palabras clave: *Cardiopatía isquémica. Insuficiencia mitral. Cirugía*.

Functional ischemic mitral regurgitation is defined as a complication of coronary artery disease, rather than a chance association with mitral regurgitation of degenerative or some other etiology. It is a complex disease, the natural history of which is indicative of a poor prognosis. Grigioni et al[1] reported a 5-year actuarial survival of 38%±5%, with a significant impact on quality of life. The rate of survival was significantly lower (29%±9%) among patients with an effective regurgitant orifice area of 20 mm^2 or more.

The complex functional anatomical and pathophysiological mechanisms that cause ischemic mitral regurgitation are not entirely clear. Left ventricular remodeling owing to severe myocardial infarction plays an important role in its development, although local involvement of the papillary muscles is also a factor. In contrast to its role in other types of mitral insufficiency, annular dilation is not the direct cause of regurgitation, but the consequence of the different mechanisms leading to a lack of valve coaptation.

It is not always easy to establish the etiology of functional mitral regurgitation in coronary artery disease because the latter presents with degenerative mitral regurgitation in a considerable proportion of patients. Valve lesions due to Barlow disease can be identified by the typical redundant, billowing leaflets with elongated chordae tendineae. In contrast, the coexistence of an isolated degenerative lesion in the posterior leaflet makes the etiological diagnosis of mitral regurgitation more difficult, both on echocardiographic examination and on direct intraoperative inspection. The ischemic origin of the lesion is evident when the posterior papillary muscle appears to be elongated and presents a pearly aspect or partial rupture.

The therapeutic approach to this disease is the subject of controversy since the results of mitral valve

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surgery continue to be unsatisfactory. Functional ischemic mitral regurgitation is currently one of the most widely investigated medical conditions at the international level. In recent years, a number of new aspects of its anatomy and pathophysiology have been discovered, and novel surgical techniques are helping to improve the outcome of the correction of this disease.

The purpose of this editorial is to compile the most relevant and valuable scientific information encompassed in the knowledge of the different underlying mechanisms of functional ischemic mitral regurgitation, as well as the new surgical techniques.

FUNCTIONAL ANATOMY

In patients with nonischemic mitral regurgitation, the abnormal valve structure leads to functional deterioration of left ventricle because of dilation and, eventually, to ventricular dysfunction. In contrast, the ventricular dysfunction in coronary artery disease generates a series of changes in left ventricular geometry and in the various components of the mitral valve (papillary muscles, chordae, leaflets, and annulus), resulting in the complex functional anatomy of ischemic mitral regurgitation. Coronary heart disease can lead to left ventricular dysfunction, with changes in the papillary muscles, rupture or detachment of the chordae tendineae, restricted valve motion, annular dilation, and, consequently, mitral regurgitation. The progressive increase in mitral regurgitation results in an increase in ventricular dilation and wall stress, thus establishing a vicious circle, with progressive deterioration of ventricular function and aggravation of mitral regurgitation.[2-4]

Transthoracic and transesophageal echocardiography have contributed substantially to the understanding of the mechanisms of ischemic mitral regurgitation.[5,6] As it depends on left ventricular function, mitral regurgitation acts as a functional failure, the severity of which varies from one moment to another. Thus, it is important to carefully analyze the valve anatomy, which remains constant, rather than its function, which changes over time. Craig Miller's research team at Stanford University has made a major contribution to the understanding of this complex valve disease through a number of experimental studies in sheep, in which they induced myocardial ischemia for subsequent testing of different annuloplasty devices.[7-9] However, these studies in animals with acute myocardial ischemia do not faithfully reproduce the anatomical and pathophysiological features of the clinical picture in humans and, thus, their value is limited.

Basically, mitral valve anatomy exhibits restricted leaflet motion and annular dilation, resulting in inadequate coaptation. For this reason, it has been included in the Carpentier classification of mitral insufficiency as type IIIb.[10] Since 1983, this classical classification,[10] which has not undergone subsequent revision, groups the different types of mitral insufficiency according to the mobility of the valve leaflets. Type I includes mitral insufficiency with normal valve leaflet mobility, in which the valvular regurgitation is mainly produced by the lack of leaflet coaptation (annular dilation) or perforation (endocarditis or trauma). Type II is comprised of insufficiency with increased valve mobility due to chordal elongation or rupture (degenerative disease). Finally, type III includes mitral insufficiency in which valve motion is restricted (type IIIa: rheumatic valve disease; type IIIb: coronary artery disease). However, recent findings associated with coronary artery disease have limited the usefulness of this classification. Segmental ischemia of left ventricle, akinetic or dyskinetic, and of the papillary muscles —in particular, the posterior papillary muscle—provokes a restriction of valve motion (type IIIb). This is due to the increase in the distance between the mitral annular plane and the papillary muscle base. The excessive tension on the chordae tendineae impedes the normal coaptation of the valve leaflets, a finding that is most evident in the region of the posterior commissure and the region posterior to the septal leaflet. Moreover, mitral insufficiency can also present as a simple functional failure secondary to annular dilation (type I) or, less frequently, with valve prolapse (type II), as recently observed by Jouan et al[11] in one third of their patients. Prolapse usually occurs in the presence of necrosis of the papillary muscle or one of its heads into which marginal chordae tendineae are inserted. This papillary muscle ischemia can provoke its partial rupture or its dysfunction, events that contribute to valve prolapse. Myocardial ischemia usually affects the posterior papillary muscle due to its precarious irrigation, although the anterior papillary muscle may also be involved (in 9% of the cases),[11] despite the fact that its vascularization is better ensured by the left anterior descending and diagonal arteries.

The magnetic resonance imaging study performed in China by Yu et al[12] demonstrated that the mechanism that produces mitral insufficiency is very complex and involves the participation of multiple vector forces originated by the increase in the anteroposterior diameter of the annulus, the abnormal posteroinferior tension on the leaflets, and increases in the distance between the papillary muscles and in the distance between the annulus and the base of these muscles. Using a computer program involving three-dimensional echocardiography, Ahmad et al[13] made some highly interesting observations concerning annular geometry and valve motion in ischemic mitral regurgitation. The annular perimeter was significantly greater than that of the control group (10.7 cm vs 8.6 cm) and there was an increase in the intertrigonal distance (2.8 cm vs 2.1 cm), resulting in a significant increase in the mitral

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