Estructura y función ventricular izquierda en la estenosis aórtica: el enemigo interior

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Concentric hypertrophy is developed in aortic stenosis (AS). This hypertrophy is characterized by a decrease in the relationship between the end-diastolic diameter and the left ventricular wall thickness (LV) (r/h) with a normal sized cavity. If thickness-radius relationship and LV systolic pressure remain constant, the hypertrophy is appropriate. An increase in r/h represents an increase in wall stress and this is associated with an inappropriate hypertrophy. (1, 2)

The increase in myocyte mass and interstitial fibrosis is linked with diastolic and systolic dysfunction that may remain after valve replacement. (3)

Another aspect to consider is the contribution of the systemic vascular resistance to the LV post-load in patients with aortic stenosis; due to this situation, concurrent hypertension with valvular heart disease increases the LV load and it may affect the assessment of AS severity, and also its structure and function. (4)

These assertions create two questions: Are these considerations enough and conclusive for the management of this group of patients? Is there any direct relationship between clinical-paraclinical consequences and the degree of myocardial structure?

From the physiopathological point of view, the evolution to myocardial failure has its foundations on the modifications that the myocardium suffers in its structure and function throughout the adaptation of the left ventricular hypertrophy (LVH). (5)

In a first approach, the increase of the left ventricular mass seems appropriate to maintain the parietal stress; however, data from another 23 studies showed an inverse relationship between the ejection fraction (Ef) and the angiographic LV mass (r = -0.59). (6)

Studies that used endomyocardial biopsy to research on the LV structure and function in AS showed that the increase in interstitial fibrosis is directly related to contractile worsening and, even more, advanced degrees of pump failure in patients with AS are associated with more advanced degrees of alteration in the myocardial structure. (7)

The mass of muscle cells, in general, is greater than the mass of interstitial tissue and the mass of muscle cells is closely related to the decrease of the peak of the LV ejection fraction. (6, 7)

These studies lead us to a first consideration: LV hypertrophy in AS is not "mild" and limiting of the ventricular contractile worsening, but it is limiting of the LV adaptive process that may be tolerated in great or lesser extent in relation to the progression of the valvular heart disease and its time evolution.

These results are important from the point of view of the hypertrophy regression after aortic valve replacement, since residual hypertrophy is directly related to the degree of pre-operative interstitial fibrosis.

Patients with symptomatic severe AS, compensated LVH and preserved pump function present systolic and diastolic alterations related to the changes in the myocardial structure and myocyte hypertrophy is positively correlated with the decrease of +dP/dt max / LVEDP. The aforementioned was shown in Hita et al.'s study (8)

These findings allow us to consider if patients with AS or LVH are adapted "appropriately" or "inappropriately" in response to cardiac work, especially in the asymptomatic subgroup in which the decision criteria are not uniform.

The compensated role of LVH has been studied in hypertension where patients with inappropriate LVH were related to adverse events, alteration of the metabolic profile, concentric geometry, LV dysfunction and adverse prognosis. (9)

More recently, a significant part of patients with AS presented excessive LVH which was correlated with concentric structure and alteration of the systolic function. (10)

Adjusting clinical (age, diabetes) and echocardiographic (peak and mid gradient, extension of the calcification) variables, the presence of inappropriate LVH preserves independent value of death by any cause or admission due to pump failure or non-mortal AMI. (11)

LVH is considered inappropriate or excessive when there is a difference of 10% or greater between observed and expected values. The expected LV mass was determined by the expulsion work and the patient's sex and height.

If this adaptive process is progressive and if it may be identified in early stages of the disease, this process may help us to differentiate risk groups.

In asymptomatic patients with severe AS, the lack of reliable predictors does not allow us to move comfortably towards a more aggressive behaviour. On the other hand, the fact that doctors derive asymptomatic patients to a valve surgery is not an

infrequent behaviour. This behaviour is induced by the perception that the patient prognosis is really more adverse than the estimated one.

Do we have to measure the severity of the obstruction or its physiological consequences in asymptomatic patients with severe AS or do we have to focus on the valve and its consequences "of (bad) adaptation" in a better approximation?

Many initial efforts were performed to focus on the valvular obstruction and to assess the degree of severity. There is no specific cutting point to determine the prognostic value of the peak aortic jet velocity and the valve area at peak velocity, since patients with a range of 4.0-5.5 m/sec and 0.5-1.3 cm² have presented sudden events.

In the last years, physiological consequences (increase of LV mass, alteration of systolic and diastolic function) have gained space in the bibliography to be included in the decision process in asymptomatic patients with severe AS. (7, 10, 11)

Steward et al. (12) have studied the role of Tissue Doppler to estimate systolic and diastolic function in asymptomatic patients with severe AS (AVA = 0.81 cm²), Ef > 50% and peak velocity > 3 m/sec).

During an average monitoring (31 months), 57% of patients developed symptoms attributed to AS. These patients presented a greater peak gradient, a minor AVA, a greater LV mass and a minor velocity of the tissue systolic wave. However, in a multivariate analysis, the aortic peak velocity was the only significant predictor.

Regardless of individual study results, the approximation to the patient with AS should be integrative. The degree of valvular obstruction, the repercussion of LV structure (concentric LVH, mass and RWT), LV function (systolic and diastolic), LA dilation and presence of fibrosis in the NMR will allow us to establish a risk group pattern and to refine our clinical perception.

Hita et al. showed us that the "inner enemy" is present, with a harmful and silent effect in the natural evolution of this group. For this reason, the value of his work reminds us that the patient's approach should be complemented with information of physiological consequences of this process.

Again, old concepts within reach of modern detection techniques allow us to complement without the replacement of our clinical criterion.

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