

Myocardial Delayed Enhancement by Cardiac Magnetic Resonance Imaging in Pulmonary Arterial Hypertension: A Marker of Disease Severity

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The study of Bessa et al.¹, published in this issue of *Arquivos Brasileiros de Cardiologia*, studied 30 patients with pulmonary hypertension (PH) using cardiac magnetic resonance imaging. They evaluated the presence and extent of delayed enhancement in these patients and correlated the percentage of delayed enhancement mass with severity markers in pulmonary hypertension. Delayed enhancement was found in 93% of patients with HP in the anterior and inferior septa, in the septal-RV free wall attachment zones, commonly called delayed enhancement of ventricular junction pattern. The delayed enhancement mass was corrected to the left ventricular mass. The percentage of delayed enhancement was then used for analysis. This study showed a higher percentage of myocardial fibrosis in patients with signs of Right Ventricular Failure (RVF), Functional Class (FC) IV, 6-Minute Walk Test (6MWT) < 300 m, Cardiac Index (CI) < 2.0 and right atrial pressure > 15. The presence of RHF, the impairment of FC and the 6MWT walking distance and low CI are classic markers of prognosis in HP. The percentage of fibrosis was able to identify patients with RVF (clinical evaluation), FC IV, 6MWT < 300 m and CI < 2.0 L/min.m² with good accuracy.

Despite some progress in understanding the physiopathology of the disease and the discovery of new treatments in recent decades, pulmonary hypertension is still a disease with poor prognosis². Non-invasive markers to better assess the severity of the disease and that may help determine which patients require more aggressive treatments are needed. Delayed enhancement is a tool that was initially used to evaluate areas of myocardial fibrosis in patients who have had myocardial infarction. The contrast injected is quickly rinsed in normal areas, but when there is increased extracellular tissue, such as in fibrosis, the contrast is retained and is slowly eliminated

from these areas. When images are acquired late (5-10 min after contrast injection), the areas in which the myocardium is intact do not retain the contrast, but the areas with fibrosis retain the contrast, hence the term delayed enhancement.

In patients with HP, three studies demonstrated the presence of delayed enhancement in most patients and delayed enhancement was found mainly in the RV septal attachment zone and in the septal wall³⁻⁵. Fibrosis in these areas can also be found in hypertrophic cardiomyopathy^{6,7}, unlike other cardiomyopathies such as the Chagas disease⁸, with predominance of fibrosis in the basal and apical left ventricular (LV) inferolateral wall, or viral myocarditis with diffuse pattern⁹, among other patterns suggestive of specific etiologies of cardiomyopathies. In most of these diseases, the presence of delayed enhancement appears to be associated with increased risk of arrhythmias and worse prognosis. Delayed myocardial enhancement (fibrosis) of ventricular junction pattern appears to be associated with Right Ventricular (RV) overload. An explanation for this preferential location of delayed enhancement is the overload sustained by the septum with increased RV afterload. As the RV overload increases, it dilates and pushes the septum toward the LV, overloading the septal RV attachment zones and the septum itself. Shehata et al. demonstrated the inverse relationship of delayed enhancement mass with Eccentricity Index (EI), that is, the higher the septal bulging toward the left ventricle, and consequently the lower the EI, the greater the delayed enhancement mass¹⁰. In experimental studies, these are the areas subjected to maximum stress in normal ventricular contraction, and these areas are also the first to produce natriuretic peptide type A in HP models, reflecting greater mechanical stress. The study of Bessa et al. demonstrated that most patients with HP had delayed enhancement and ventricular junction pattern, confirming the literature data¹. An echocardiographic study in patients with HP of a specific etiology associated with schistosomiasis also demonstrated a relationship of increased pulmonary pressure with disease severity, suggesting that in various etiologies of HP, a delayed enhancement of similar pattern may occur (Armstrong)¹¹.

The strength of this study was that all patients underwent right cardiac catheterization within 72 hours after cardiac magnetic resonance imaging. Previous studies have shown the relationship of delayed enhancement mass with RV dysfunction and hemodynamic variables, but this study was the first to demonstrate the relationship of myocardial fibrosis with clinical, hemodynamic and functional markers.

The evaluation of right ventricular function is emerging as an independent prognostic marker in HP and the study

Keywords

Magnetic Resonance Secfroscopy/methods; Hypertension, Pulmonary; Myocardium; Endomyocardial Fibrosis.

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of Sheata et al. also demonstrated an inverse correlation of delayed enhancement mass with RV ejection fraction¹⁰. Unfortunately, this study did not evaluate the fibrosis mass percentage in relation to RV dysfunction.

Although this study has evaluated a small number of patients with HP and although it is a cross-sectional study in which it is not possible to show the prognostic role of delayed enhancement, the fact that the percentage of fibrosis is increased in patients who have markers of worse prognosis suggests that delayed enhancement may prove to be an important noninvasive prognostic marker in patients with HP. It would be interesting if the authors conducted long-term follow-up of these patients, so that the prognostic role of delayed enhancement is confirmed and fibrosis may show its prognostic role in HP, thus helping clinical decisions. The study of Bessa et al. also opens up the possibility of

comparing other forms of HP, such as those belonging to the other groups of classification (secondary to left ventricular dysfunction, diseases of the pulmonary parenchyma, chronic pulmonary thromboembolism, for example) in order to analyze the existence or not of different patterns of fibrosis.

Despite these limitations, the manuscript of Bessa et al. is another original scientific contribution indicating that myocardial fibrosis detected by cardiac resonance imaging correlates directly with the severity of disease and possibly with prognosis. Therefore, another marker of severity of cardiomyopathy associated with pulmonary hypertension is reaffirmed and can be identified by magnetic resonance imaging. The evaluation of interstitial myocardial fibrosis through myocardial T1 mapping by resonance imaging may bring in the future more information on the myocardial state and prognosis in this important and challenging clinical scenario.

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