Quantitative Assessment of Left Ventricular Regional Wall Motion in Endomyocardial Fibrosis

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Objective
To analyze left ventricular (LV) regional wall motion in patients with endomyocardial fibrosis (EMF).

Methods
The study comprised 88 patients, 59 of the female sex, with a mean age of 39±13 years (range, 9 to 65) and with echocardiographic and angiographic evidence of left ventricular EMF. The intensity of fibrous tissue buildup on contrast ventriculography was classified as mild, moderate, or severe. The overall left ventricular ejection fraction (LVEF) was determined by using the area-length method on ventriculography. The motion was measured in 100 equidistant chords perpendicular to the centerline drawn in the middle of the final diastolic and systolic contours and normalized to cardiac size. Five left ventricular segments were analyzed: A - apical; AL - anterolateral; AB - anterobasal; IA - inferoapical; IB - inferobasal. Abnormality was expressed in units of standard deviation of the mean motion in a normal population of reference, comprised of 103 patients with normal LV according to clinical and electrocardiographic data, and angiographic standards.

Results
Mean LVEF was 0.47±0.12. Fibrous tissue buildup in the left ventricle was mild in 12 patients, moderate in 40, and severe in 36. The regions with the poorest ventricular wall motion were A (-1.4±1.6 standard deviation/chords) and IA (-1.6±1.8 standard deviation/chords) compared with that in AB (-0.3±1.9 standard deviation/chords), AL (-0.5±1.8 standard deviation/chords) and IB (-0.9±1.3 standard deviation/chords). No relation was observed between the intensity of fibrous tissue buildup and regional ventricular wall motion.

Conclusion
A change in LV regional wall motion exists in EMF, and it is independent of the intensity of fibrous tissue buildup qualitatively assessed. Nonuniform involvement of the LV should be considered when planning surgery for this disease.

Key words
regional contractility, fibrous involvement, endomyocardial fibrosis

Eighty-eight patients with left ventricular endomyocardial fibrosis were studied, 59 of the female sex, with a mean age of 39±13 years (range, 9 to 65), 85 (96%) of whom had associated right ventricular fibrosis. The distribution of functional class according to the New York Heart Association (NYHA) criterion was as follows: I in 8 (9.1%) patients; II in 11 (12.5%); III in 38 (43.2%); and IV in 31 (35.2%). The normal population of reference comprised 103 individuals, 52 of whom were men, with a mean age of 52±10 years (range, 21 to 72) and normal left ventricle according to clinical and electrocardiographic data, and angiographic pattern.

The diagnosis of endomyocardial fibrosis was established based on the following echocardiographic findings present in all cases: obliteration of the ventricular apex; atrial dilation; an increase in endocardial echo reflectivity; restrictive diastolic filling pattern;
Atroventricular regurgitation. The angiographic images of the left ventricle were performed with a pigtail catheter in the anterior right oblique projection at 30º and confirmed the diagnosis. The intensity of the fibrous tissue buildup on contrast cineventriculography was classified as mild, moderate, or severe. The overall left ventricular ejection fraction (LVEF) was determined by the area-length method and the regional left ventricular wall motion quantified by the centerline method, which is a simple model for analyzing ventricular wall motion.

The intensity of fibrous tissue buildup on contrast cineventriculography was divided into mild, moderate, and severe. The regions with the worst contractility were A (-1.4±1.6 standard deviation/chords) and IA (-1.6±1.8 standard deviation/chords), compared with AB (-0.3±1.9 standard deviation/chords), AL (-0.5±1.8 standard deviation/chords), and IB (-0.9±1.3 standard deviation/chords). The left ventricular wall motion did not depend on the intensity of fibrous tissue buildup.

**Results**

The mean LVEF was 0.47±0.12. The left ventricular involvement by fibrous tissue was mild in 12 (14%) patients, moderate in 40 (44%), and severe in 36 (41%). The regions with the worst contractility were A (-1.4±1.6 standard deviation/chords) and IA (-1.6±1.8 standard deviation/chords), compared with AB (-0.3±1.9 standard deviation/chords), AL (-0.5±1.8 standard deviation/chords), and IB (-0.9±1.3 standard deviation/chords). The left ventricular wall motion did not depend on the intensity of fibrous tissue buildup.

**Fig. 1** - Centerline method for analyzing regional left ventricular wall motion. A) final diastolic and systolic endocardial contours on contrast ventriculography; B) 100 chords perpendicular to the centerline delimitated between the final diastolic and systolic contours; C) left ventricle divided into 5 regions, the chords 1 to 10 were excluded because they represented the mitral valve.

**Fig. 2** - Regional wall motion in 5 left ventricular regions showing a reduction in motion in the regions A and IA: AB - anterobasal; A - apical; IA - inferoapical; IB - inferobasal.

**Fig. 3** - The degree of overall fibrosis of the left ventricle was qualitatively analyzed on left ventriculography as mild (12 patients), moderate (40), and severe (36). That fibrosis was related to regional wall motion in each of the 5 left ventricular regions, and it showed no relation to the intensity of the overall buildup of fibrous tissue: A - apical; AL - anterolateral; AB - anterobasal; IA - inferoapical; IB - inferobasal. Overall buildup of fibrous tissue in the LV: 1 - mild; 2 - moderate; 3 - severe.
Discussion

The pathogenic mechanisms of endomyocardial fibrosis are little understood. Some studies have suggested the occurrence of different clinical and histopathological stages in the evolution of the disease, the final stage being characterized by a fibrotic process of the endocardium. Some controversial points exist regarding the disease, which is characterized by a diffuse myocardial inflammatory process, or as having an evolutionary character.

Some detailed descriptions of the disease exist. The lesions reflect the replacement of the endocardium and adjacent ventricular myocardium by fibrous tissue. The lesions are not diffusely located in the endocardium, but usually affect the apex and ventricular inlet. From the left ventricular apex, the fibrotic lesions extend to the posterior wall, involving the posterior leaflet of the aorticvalve, which adheres to the fibrosis. This is a continuous or irregular process, with areas of tissue between the apex and the valvular region which are spared from fibrosis. However, the disease may affect only the apex or the valvular region, and may even be found in regions other than the apex and valves. This morphological information is of great relevance, mainly because new surgical techniques have been reported.

In clinical practice, ventricular wall motion is assessed through echocardiography by use of the wall motion score or through contrast cineventriculography of the left ventricle. The poor reproducibility of that assessment led to the development of the method used in this study, which was described by Sheehan et al in 1982 and introduced at our institution by Toscano et al in 1992. This method promotes a reliable model for analyzing the left ventricular regional function. Our results showed that the A and IA walls had a significant reduction in motion in comparison with that of the other walls. Because both regions are the predominant site of fibrous tissue buildup in endomyocardial fibrosis, we expected that these regional alterations were greater in the ventricles with more intense fibrosis buildup. In our study, however, we did not find such a relation between them. Quantification of the regional left ventricular wall motion had no relation to the qualitative fibrous tissue buildup assessed on cineventriculography, although the latter analysis is useful predominantly for assessing the endocardial involvement, but not the true myocardial involvement.

In our study, we used 103 left ventricular cineventriculograms of healthy individuals to compare with those of patients with endomyocardial fibrosis. Previous studies have shown that the reliable definition of normal left ventricular cineventriculography requires the analysis of 50 healthy individuals. Therefore, in our study, the number of healthy individuals analyzed exceeded the minimum necessary recommended in the literature.

If one admits that endomyocardial fibrosis is a disease characterized by a scar, an interesting question is to know why, in most cases, the apex and diaphragmatic region are the most affected regions of the left ventricle. As already known, in Chagas’ disease, which is characterized by a diffuse myocardial inflammatory process, the apical aneurysm is a very suggestive morphological finding. When the aneurysm is absent, alterations in contractility, which frequently involve the left ventricular posteroinferior wall, may be detected. In nondilated Chagas’ disease, a reduction in the segmentary contractility of the inferoapical wall may also be observed. Both heart diseases lead to segmentary alteration in wall motion, which may be analyzed through that method.

Different types of aggressive agents cause different types of morphologic consequences in the left ventricular cavity. Thus, eosinophilic endomyocarditis leads to endomyocardial fibrosis, a restrictive syndrome with small ventricles, while a chronic diffuse inflammatory process, such as Chagas’ disease, causes dilated cardiomyopathy. Usually, Chagas’ disease causes diffuse active inflammatory cardiomyopathy, while endomyocardial fibrosis is consequent to a previous inflammatory process located mainly in the inner portions of the ventricles. Consequently, in endomyocardial fibrosis, the scar is internal to an almost normal muscular layer, which protects the chamber from dilation. On the other hand, a previous thrombus may become fibrous, reducing the ventricular cavity. However, those 2 different types of cardiomyopathy have one point in common: apical involvement. In the dilated form of Chagas’ disease, the apex seems thinner than the other walls, or with aneurysms of different sizes; in the restrictive form, it is thicker, and usually slightly hypokinetic. Probably, the tension in the wall is different in several regions of the left ventricular chamber. This needs to be better elucidated. In endomyocardial fibrosis, fibrosis may sometimes be apparent neither grossly nor on ventriculography. In addition, the histopathological findings of endomyocardial fibrosis show degenerative subendocardial alterations, which certainly justify the described alterations in motion.

Several studies have shown that that method allows the comparison of the effects of the surgical and clinical treatment for regional left ventricular wall motion. The next step is to compare wall motion in the pre- and postoperative periods in patients with endomyocardial fibrosis.

Our data may be useful for planning surgery and for analyzing the surgical results according to the type of ventricular involvement.

Acknowledgments

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Table I - Descriptive levels referring to the 2-by-2 comparison of ventricular regional wall motion in endomyocardial fibrosis

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RVWM - ventricular regional wall motion; A - apical; AL - anterolateral; AB - anterobasal; IA - inferoapical; IB - inferobasal.
References