Ventricular Aneurysms in Chagas Heart Disease

In a recent scientific letter, Bravo et al. (1) presented a unique and interesting case of Chagas disease ventricular aneurysm. Taking into account that chronic Chagas heart disease with ventricular aneurysms is the origin of malignant arrhythmias, thromboembolism (mainly pulmonary) and stroke, it is necessary to detail some important milestones in the history of such a peculiar pathology.

In 1981, Oliveira et al. (2) published a seminal work in which they described the pathological anatomy of ventricular aneurysms of chagasic origin. In 2007, Acquatella (3) stablished that echocardiographic and Doppler techniques provided structural and functional information useful in the detection of early myocardial injury, disease progression and the management of patients with Chagas disease. Several years before, in 1991 (4), the first serial histopathological studies in resected ventricular aneurysms of chagasic patients had been carried out with the aim of demonstrating possible circuits generating malignant arrhythmias. The studies describe that the ventricular aneurysm has a thinned ventricular wall formed by fibrous tissue, thickened endocardium and myocardial patches, with thrombi attached to the endocardium (4). The myocardium shows a chronic inflammatory reaction, myocytolysis and fibrosis, with patches of normal, with incipient damage or necrotic myocardial tissue, surrounded by fibrous tissue. The ultrastructural study reveals myocyte hypertrophy and complete or partial loss of myofibrils; mitochondria swelling, with crysta disruption; accumulation of lipofuscin granules and intracellular edema. The most striking alteration is myocyte, vascular endothelium and smooth muscle cell basal membrane thickening. The superposition between the border of healthy fibers and of those with incipient damage observed in serial sections produces an ideal configuration for reentry circuits (Figure 1). The final proof that arrhythmias originate in these endocardial regions was their elimination by aneurysm resection.

Therefore, although it could be considered that knowing the histopathological characteristics of the diseased tissue that becomes a substrate for ventricular arrhythmias would provide the possibility of using radiofrequency ablation techniques, surgical resection continues to be mandatory.

However, following authors such as Acquatella (3), and in a still theoretical arena, it would be possible to investigate by echocardiographic monitoring the presence of predictive elements of Chagas heart disease, in order to implement pharmacological or minimally invasive measures aimed at avoiding the deleterious evolution of the disease. The integrated evaluation from an electrophysiological approach together with echocardiographic methods would allow the detection of dyssynchrony, which, by means of resynchronization therapy, could prevent the development of serious structural abnormalities in the cardiac chambers.

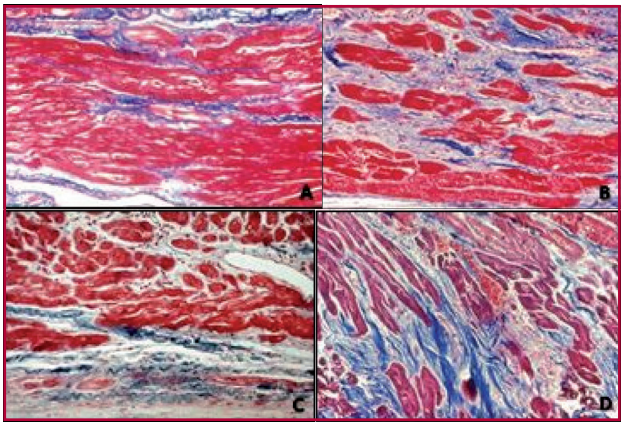

Fig. 1. Different regions of serial sections of Chagas ventricular aneurysm. **A.** Thick tracts, separated by a small amount of connective tissue (blue), with interspersed fibers with varying degrees of cell damage. **B.** Alternating atrophic fibers, separated by loose connective tissue (blue) and fibers aligned in thin bundles, suggestive of belonging to reentry circuits. The thickness of the fibers was 4.5 to 56.6 µm. **C.** "Islands" in subendocardial areas, with relatively preserved myocardium, separated by connective tissue (above) and Purkinje cells belonging to the left bundle of His branch, interrupted by collagen tissue (below). **D.** Longitudinal section of atrophic fibers. Mallory's trichrome stain. A and B: 400X, C: 200X and D: 100X magnification

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Authors' response

With respect to what was stated by the authors, we fully agree on the histopathological and echocardio-

graphic knowledge of the disease evolution, especially in its more advanced stages.

The endemic condition of Chagas disease in Latin America has been known for more than 100 years, as indicated in our publication. However, even today we continue to treat its complications at their maximum expression (ventricular aneurysm, ventricular thrombi and heart failure) by aneurysmectomy and, perhaps, heart transplantation. Even knowing the evolution of the disease (the chronic form takes at least 20 years to manifest and passes through 4 stages), we have not created guidelines to prevent ventricular dilation and, consequently, malignant ventricular arrhythmias, despite follow-up by echocardiography and electrophysiological studies. (1) That would be the first step to achieve the regression of the disease; while we hope that one day the last step will be achieved: its complete suppression by eradicating its vector.

We would like to make an analysis from our position, since surgery is indicated as the, so far, mandatory intervening actor. In our center we have performed several successful procedures for ventricular aneurysms, one of which one was published, and we can say that we completely agree that fibrosis is the main thrombogenic and arrhythmogenic factor of the left ventricle. For this reason, in most cases, these problems are solved once the aneurysm is removed, but it should be realized that such a procedure is highly effective and safe for the patient as long as he presents without serious ventricular function impairment or significant coronary disease, conditions that considerably increase postoperative morbidity and mortality in these patients.

In the Latin American guidelines for the diagnosis and treatment of Chagas heart disease, the pharmacological and invasive treatment for each of its complications has been very explicit. As the authors express, it may be time to generate criteria for identifying reversible changes in stages A and B1 of the disease, especially as representatives of the epidemiological group 1. (1, 2).

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