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Editorial

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Ventricular arrhythmias and sudden cardiac death in patients with chagas disease

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Editorial

Chagas disease is a parasitic zoonosis caused by Trypanosoma cruzi which is transmitted by insects belonging to different species of Triatoma. Nevertheless, several other routes of transmission have also been described, such as transmission via blood transfusion, infected organs transplant, and oral transmission.[1–5] Following the acute phase of the infection, untreated Chagas' disease enters a chronic phase that is initially asymptomatic or unrecognized. It is estimated that nearly one third of the patients infected with T. cruzi will develop symptomatic heart disease during the course of their lives.[6–10] The pathogenesis of the Chagas heart disease (CHD) is not clearly understood yet. Current knowledge of the physiopathology of this disease, as well as, the scientific understanding of the medical based evidence points towards a complex etiology. There is evidence of direct involvement of parasites in producing myocardial damage.[11–14] The possibility of a mechanism associated to autoimmune phenomenon was also described.[15–18] Other described pathogenic mechanisms include microvascular alterations and autonomic denervation.[19–21]

Carlos Chagas described the disease that bears his name in 1909. The next year he emphasized the importance of the abnormalities in the cardiac rhythm found in some patients with chronic disease. Hence, he then already suggested the existence of heart involvement in patients with chronic Chagas disease.[22–25] He noticed the presence of premature ventricular contractions (PVC) and stressed the presence of AV block in cases of extreme bradycardia during physical examination as cause of abnormalities in the cardiac rhythm. Carlos Chagas presented cases of patients with heart failure and frequent PVC who were found to have nests of parasites accompanied by interstitial mononuclear cell infiltration in the myocardium at autopsy.[26–28] Therefore, he suggested that PVC on physical examination could herald sudden cardiac death (SCD) in the cardiac form of the chronic disease.[29–31]

Low LV ejection fraction and VO2 max remained independent predictors of all cause mortality in patients with chronic heart failure secondary to chronic CHD. In addition, ventricular tachycardia induced by exercise stress testing was found to be a predictor of sudden cardiac death in patients with chronic CHD.[32–36] Rochitte et al.,[37] quantified myocardial fibrosis by magnetic resonance imaging in patients with chronic CHD. They observed that myocardial fibrosis was present in 85% of patients with this condition, and that LV ejection fraction was inversely correlated to myocardial fibrosis. The authors demonstrated that myocardial fibrosis was present in all patients with ventricular tachycardia, a fact that strongly suggest a role for the fibrosis in the pathogenesis of malignant arrhythmias in patients with chronic CHD.[37]

Chagas heart disease is a fibrotic disease that is generally located in the posteroinferior and apical region of the left ventricle, the sinus node, and the conduction system below the bundle branch. CHD often develop as a form of dilated cardiomyopathy with a tendency towards the formation of apical aneurysms. It has a powerful arrhythmogenic potential leading to ventricular arrhythmias and sudden cardiac death. Also CHD is often associated with bradyarrhythmias due to atrioventricular block or sinus node disease. In the natural course of the disease, the cardiac abnormalities appear progressively around 20 to 30 years following infection.[21,22] On the other hand, during the acute phase of the infection nearly 10% of the patients develop myocarditis that progresses rapidly towards a severe form of CHD.[38,39] Other patients with mild cardiac involvement who are in the chronic phase of the disease may develop sudden exacerbation and acute heart failure when exposed to conditions of immunosuppression.[39]

There is no characteristic pattern of CHD in the conventional electrocardiogram. However, an isolated right bundle-branch block or its association with left anterior hemiblock in patients with positive serology can be considered indicators of chronic CHD.[40–42] Patients with symptomatic CHD should be considered to be at increased risk of SCD. It is important to remember that SCD can often be the first manifestation of CHD.[43,44] It was observed that during the chronic phase of the clinical evolution of the patients suffering from CHD, some of them died suddenly and unexpectedly. Unexpected SCD is one of the main mechanisms of mortality in patients suffering from Chagas disease. SCD occurs in a significant number of subjects in whom no previous symptoms or relevant symptoms were detected during clinical follow-up.[43,44] The presence of repolarization disorders have been described preceding sudden and global asynchrony. The dispersion of the QT interval is present in 20-30% of infected subjects not suffering from any other symptoms. Therefore, it is necessary to evaluate the dispersion of the QT interval and variability of the heart rate in Chagas disease since its association with ventricular arrhythmia and sudden unexpected death in patients infected with T. cruzi has been proven.[7–9]

In this setting even nonspecific symptoms as weakness or dizziness have much more significance as they may be predictive of an episode of syncope. Patients with CHD often develop premature

ventricular contractions and tachycardias, most commonly associated with myocardial damage that generates reentry phenomena. Most sustained ventricular tachyarrhythmias in patients with CHD do not arise from the apical aneurysm of the left ventricle but rather from the inferolateral region.[45,46] Since these malignant arrhythmias develop within a progressive abnormality with multiple arrhythmogenic foci, radiofrequency ablation is not so effective and should not be considered as first choice therapeutic technique. Despite the absence of specific studies in chronic CHD, the implantation of an implantable cardioverter defibrillator (ICD) is recommended to reduce the risk of sudden death in cases of clinical sustained ventricular tachycardia, or in monomorphic sustained ventricular tachycardia developing during the electrophysiological study. The administration of amiodarone is a possibility in patients with nonsustained ventricular tachycardia and normal noninducible electrophysiological study. Amiodarone is also administered empirically in an effort to reduce the frequency of appropriate or inappropriate ICD discharges and lower the likelihood of electrical storm. If the patient receives too many discharges despite this pharmacological treatment, radiofrequency ablation should be considered.[47,48]

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Conflicts of interest

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