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EDITORIAL COMMENT

A challenging dilemma: Can we predict which premature ventricular contractions may cause cardiomyopathy?

Um verdadeiro desafio: será possível predizer que tipo de sístoles prematuras ventriculares poderão causar cardiomiopatia?

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Premature ventricular contractions (PVCs) are the most frequent ventricular arrhythmia and are commonly associated with heart failure, ventricular arrhythmias and sudden cardiac death.[1]

The development of a cardiomyopathy has also been documented with frequent ventricular ectopic beats. The prevalence of left ventricular (LV) dysfunction is about 10–29% in different series of patients with frequent PVCs.[2]

A common clinical problem is determining whether the PVCs are the primary cause of the patient's cardiomyopathy, or whether the PVCs are only a sign of an underlying cardiomyopathy, misdiagnosed due to relative mild forms of the disease, without clinical evidence.[3]

The mechanism of compromised left ventricular function is not fully understood. Some authors have reported dyssynchrony as the main cause, while others have suggested irregular rhythm, AV-uncoupling and mechanical bradycardia. The lack of pulse pressure and systolic volume effectivity during PVCs has been shown to lead to secondary dilatation of LV and an increase in diastolic and systolic volumes. Dyssynchrony was the most reported cause, and the mechanism is identical to that described in left branch bundle block.[4–6]

Some authors have reported the existence of underlying disease, however, studies with cardiac magnetic resonance imaging with gadolinium have not shown an increase in myocardial enhancement or more fibrosis and scarring. Frequent PVCs can occur in a normal heart.[7]

The PVC burden to induce cardiomyopathy has been cited as more than 5–10% of all the daily heart beats during an unknown interval of time.[1,2]

However, not all patients with frequent PVCs will develop cardiomyopathy. Indeed, some patients with a high PVC burden never seem to develop any LV dysfunction, so attempts have been made to a search for risk factors and hypothesis to predict the occurrence of this type of cardiomyopathy.

Factors identified included patient age, gender (male), comorbidities, PVC characteristics and site of origin. PVCs with longer QRS duration, epicardial location and interpolation (bigeminy) were related to further LV dysfunction. PVCs with longer QRS are usually from free wall and outflow

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tracts; the right outflow tract is the most frequent site of origin of PVCs.[1,4–6,8]

PVC-induced cardiomyopathy is a potentially reversible condition in which left ventricular dysfunction can improve and even normalize with pharmacological suppression or ablation techniques.[9,10]

Echocardiography is important in the study of these patients to analyse dilatation of LV, quantification of global function through measurement of ejection fraction (EF) and global longitudinal strain (GLS), the presence of regional wall motion abnormalities as dyssynchrony, and occurrence of mitral regurgitation.

However, LV ejection fraction (LVEF) and GLS may be difficult to assess in patients with frequent PVCs or bigeminy. Repeat evaluations should be performed to assess LVEF.

In this paper, Doğan et al.[11] aimed to evaluate the effects of successful ablation on impaired left ventricular global longitudinal strain (LV-GLS) in patients with frequent PVCs, as well as the potential risk factors of impaired LV-GLS. Based on the cut-off value of >−16% of GLS, the authors defined two groups, and found differences concerning coupling interval, non-ejecting PVCs and post-extrasystolic potentiation.

The authors measured a selection of echocardiographic parameters related to normal E wave (sinus wave), PVC E wave and post-extrasystolic E wave. E wave was measured at the time of PVC (PVC E wave) and the peak velocity was multiplied by the duration. The same was done for post-extrasystolic E wave. Systolic volume (LVOT VTI) was also measured during the same events. The group with poor longitudinal strain had the following characteristics: low PVC E wave flow and low PVC systolic volume and post-extrasystolic potentiation. These echocardiographic characteristics are a hallmark in the identification of patients at risk of developing LV dysfunction. In Group 1, the authors also found a longer coupling interval time between PVCs and normal sinus complex, with relative bradycardia caused by non-ejecting PVCs.

This article highlights the utility of echo parameters in the analysis of PVCs and their association with the development of cardiomyopathy. With the measurements of systolic flow before, during and after a PVC, the authors noted that some PVCs do not have an effective systolic volume or pressure for aortic valve opening, leading to an inefficient mechanical systole with ''relative'' bradycardia, and consequent decrease in cardiac output and increase in LV diastolic pressures, volume overload, LV dilatation, and LV systolic dysfunction.

Left ventricular strain has become popular in a lot of situations as a precocious parameter of LV dysfunction. Even in cases of preserved LVEF, recent studies have shown significant decreased LV-GLS in patients with frequent PVCs. However, strain analysis requires good imaging and stability of cardiac rhythm to be calculated and, in patients with frequent PVCs, can be misleading.

Beyond the interest of this topic, further and greater studies are needed to support these findings.

Conflicts of interest

The author has no conflicts of interest to declare.

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