Ablation of frequent premature ventricular complex in an athlete

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Premature ventricular complexes (PVC) are a common finding in the exam of many athletes. There is no extensive scientific evidence in the management of this situation particularly when associated with borderline contractile function of the left ventricle. In this case report, we present a 35-year-old asymptomatic healthy athlete with high incidence (over 10 000 beats in 24 h) of premature ventricular complex and left ventricular dilatation with dysfunction, which persisted after a resting period of 6 months without training. We performed radiofrequency ablation of the premature ventricular complex focus. After 1-year follow-up, he was asymptomatic without arrhythmia and the left ventricle normalized its size and function as shown by echocardiogram and cardiac magnetic resonance.

Premature ventricular complexes (PVC) are a common finding in the exam of many people who are asymptomatic and are especially prevalent in athletes (Biffi et al., 2011). The management of this arrhythmia is conditioned by the number of PVC in 24 h, the presence of complex ventricular arrhythmia, the association with structural heart disease (Penela et al., 2013), and the symptomatology (Maron & Zipes, 2005). Additionally, the presence of frequent PVC in patients without apparently structural heart disease has also been associated with the development of the left ventricular systolic dysfunction, which could in turn be potentially reversed with the elimination of PVC. Recently, percutaneous radiofrequency ablation of frequent PVCs has been shown to decrease left ventricular size and brain natriuretic peptide levels even among patients with normal left ventricular ejection fraction (Sarrazin et al., 2009). The management of athletes with frequent PVC and whether a period of detraining is useful remains controversial (Giada et al., 2010). It has been related to training intensity and indeed, reducing the intensity of training has been related to a reduction in the frequency of PVC in athletes (Biffi et al., 2011). However, most of the athletes present with normal left ventricular ejection fraction or at most, borderline ejection fraction (inferior limit of normality). We present the case of an athlete who presented with asymptomatic frequent PVC and left ventricular systolic dysfunction that was managed with radiofrequency ablation.

Clinical case

A 35-year-old sportsman, who came to the cardiology outpatient clinic for routine cardiovascular check up, was evaluated as he was performing high-intensity training. In the anamnesis, the patient reported no family medical history or any past personal medical records, he reported a normal electrocardiogram (ECG) 5 years before, without any PVC, and he referred no symptoms and no impairment in his regular performance level. He had been a regular cyclist since he was 20 year olds (8 h per week) and he had also started running during the last year. At the time of diagnosis, the athlete was performing six sessions of training (cycling and running) per week and had performed an average of 11 h/week in the last year. He did not take any medication and denied any supplementary treatment. Physical examination and blood tests were normal.

The ECG showed sinus rhythm with frequent ventricular complex. PVC had inferior axis and right bundle branch block morphology with reverse transition V6 (Fig. 1). All PVCs had the same morphology.

A treadmill exercise test according to the Bruce protocol was performed. During the test, the patient had no symptoms and no ST segment changes suggestive of ischemia; frequent monomorphic PVC even with bigeminy were seen at baseline, which decreased with increasing heart rates during exercise. The exercise test was stopped after reaching 91% of the theoretical maximum heart rate (i.e., 168 bpm) and 14 METs.
The transthoracic echocardiogram showed a severely dilated left ventricle (LV) (Gjerdalen et al., 2014), LV end-diastolic diameter: 70 mm (39 mm/m²), the LV ejection fraction was reduced (LVEF: 44%) with a diffuse hypokinesis (Fig. 2(a,b)). The right ventricle was also moderately hypokinetic. There was minimal secondary mitral regurgitation with other valves normally functioning. The study was completed with a cardiac magnetic resonance imaging (MRI) that confirmed the presence of a dilated LV (LV end-diastolic volume: 266 mL, 150 mL/m²), without wall hypertrophy, and a slight decrease in overall motion (LVEF 39%) without segmental wall motion abnormalities.

The end-diastolic volume of the right ventricle was also slightly increased (175 mL, 99 mL/m²), with preserved global motion. The origin of the coronary arteries was normal and there were no late myocardial enhancement focus or any sign of right ventricular dysplasia.

The 24-h ECG registry showed sinus rhythm throughout the record, with a minimum heart rate of 47 bpm, average of 70 bpm, and maximum of 126 bpm. Very frequent monomorphic PVC were recorded constituting up to 21% of the beats analyzed during the whole day, half of them were recorded as bigeminy; there were no sustained or complex ventricular arrhythmias.
The patient stopped doing regular physical activity for a period of 6 months and started the treatment with beta-blockers and inhibitors of angiotensin-converting enzyme, but the incidence of extrasystoles and LV dysfunction remained without significant changes to baseline.

Percutaneous radiofrequency ablation was proposed and performed through both venous and arterial access. A quadripolar catheter was positioned for diagnosis in the right ventricular apex and an irrigated ablation catheter (Navi-Star™, Biosense Webster, Diamond Bar, California, USA) was inserted into the LV via a retroaortic approach. Firstly, an electro-anatomical reconstruction of the ascending aorta was merged with a previously acquired computed tomography angiography and activation mapping during ventricular ectopic activity was performed. Earlier ventricular activity was identified at the basal segment of the LV lateral wall close to the mitral annulus. Electrical precocity at that level was 32 ms ahead of the QRS complex. Radiofrequency was applied four times at that site (50 W, 45°C, 26 mL/min) until PVC disappeared without any complications. The patient was discharged of the hospital with the prescription of aspirin 100 mg daily for a month.

One month later, the ECG showed sinus rhythm without arrhythmias. The echocardiogram showed a clear improvement with decreased LV end-diastolic diameter (LV end-diastolic diameter: 64 mm) and improved LV ejection fraction (LVEF: 54%) (Fig. 2(c,d)). Aspirin was suspended and patient was allowed to return to progressive moderately intense aerobic physical activity.

At 6-month follow-up, the patient persisted asymptomatic, performing routinely sports at a high weekly volume. The control 24-h ECG registry showed sinus rhythm throughout the record and infrequent ventricular extrasystoles, 235 PVC in 24 h (<1% of analyzed beats). A cardiac MRI performed at 12 months follow-up confirmed the finding on echo at 1 month and even more reversion of LV dilatation (LV end-diastolic volume: 60 mm) and dysfunction (LV ejection fraction: 51%) (Fig. 3(c,d)).

Discussion

Frequent monomorphic ventricular extrasystole can sometimes be a manifestation of structural heart disease but in many cases occur in healthy hearts; they are usually associated with a benign course and a good prognosis. However, the presence of highly frequent (high density) PVC together with the presence of LV dilatation and systolic dysfunction poses a completely different scenario, particularly in the setting of a young, otherwise healthy, subject who wants to keep on practicing sport.

In the absence of structural heart disease, the treatment of PVC is particularly indicated if the patient is symptomatic for palpitations or dyspnea. Medical therapy is considered when incidence of arrhythmia is above 10 000 beats in 24 h and if this is not tolerated because of side effects or it is not effective in controlling the arrhythmia, radiofrequency ablation can be considered (Aliot et al., 2009). However, when the proportion of PVC in relation to the beats originating in the sinus node is more than 20% for 24 h, it has been
recently shown that there is a clear association with increased LV dilation, increased mitral regurgitation, and reduced LV ejection fraction (Niroomand et al., 2002). The pathophysiologic mechanism by which PVC of the outflow tract of the right ventricle generate ventricular dysfunction is not fully elucidated, yet it could have a relationship with ventricular asynchrony producing an effect similar to the left bundle branch block, which causes LV diastolic dysfunction and mitral regurgitation (Littmann & Symanski, 2000). While it has not been clarified that the pathophysiology of PVC originates in the left ventricle, it could produce similar abnormalities in ventricular contraction and the associated deformation.

In the present case, the high density of the ventricular arrhythmia was associated with the existence of LV dysfunction; and according to current recommendations, we decided to perform an ablation which was indeed successful in terms of eliminating the arrhythmia but also in reversing LV dilatation and dysfunction. This was particularly interesting considering that the patient was a physically active sportsman who was not willing to leave sport practice.

**Perspective**

We believe that radiofrequency ablation might be an excellent therapeutic option in selected athletes with frequent PVC, particularly if accompanied by LV dilatation and mild dysfunction. Longer-term follow-up of athletes, such as the presented case, is warranted in order to better understand the potential relationship between training and PVC with ventricular dysfunction.

**Key words:** Premature ventricular complexes, radio-frequency catheter ablation, athlete’s heart.

**References**


