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CASE REPORT

A Case Report on Myocarditis in Asymptomatic Athletes. What Shall We Do?

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Abstract

Myocarditis is a cardiac disease of heterogeneous aetiology characterised by the infiltration of inflammatory cells into the myocardium and a potential risk of impairment of cardiac function. Viral myocarditis appears to be a major cause of unexpected death in persons under 40 years of age, particularly in athletes and persons engaged in moderate physical activity, and may progress to chronic dilated cardiomyopathy. With this article, the author describes two recent cases of young athletes with subacute myopericarditis who underwent cardiovascular imaging investigations as a consequence of a polymorphic ventricular arrhythmia recorded during the examination and electrocardiogram with exercise testing required for competitive sports preparticipation screening.

Keywords: Myopericarditis, Athletes, Cardiac Magnetic Resonance, Echocardiography, Arrhythmias.

1. Introduction

Myocarditis is a cardiac disease of heterogeneous aetiology characterised by the infiltration of inflammatory cells into the myocardium and a potential risk of impairment of cardiac function.[1] Myocarditis is a typical inflammatory disease that leads to myocardial damage with necrosis and/or degeneration of adjacent myocytes, different from the ischaemic damage associated with coronary artery disease.[2] This disease can develop either acutely or chronically and is characterised by inflammatory cell infiltrates, myocyte necrosis or myocyte degeneration, with or without fibrosis. It can be caused by various aetiologies, but in particular by infectious, connective tissue, granulomatous, toxic or idiopathic processes. Associated systemic manifestations of the disease are often present, and sometimes the endocardium or pericardium are involved. Affected athletes may be asymptomatic, have non-specific prodromal symptoms or present with overt congestive heart failure, compromising arrhythmias or sudden death.[3] It is indeed a subtle and under-diagnosed disease, but estimates of its incidence can be made by examining autopsy results.[4]

Viral myocarditis appears to be a major cause of unexpected death in persons under 40 years of age, particularly in athletes and persons engaged in moderate physical activity, and may progress to chronic dilated cardiomyopathy (DCM). Adolescents and adults with suspicious symptoms commonly have a recent history of viral disease, 10-14 days prior to presentation, and may have chronic dilated cardiomyopathy.[5] In this manuscript, the author describes two recent cases of young athletes who underwent cardiovascular imaging investigations as a consequence of a polymorphic ventricular arrhythmia recorded during the examination and electrocardiogram with exercise testing required for competitive sports participation. The case report draws particular attention to the use of cardiac magnetic resonance imaging in athletes

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with stress-inducible ventricular arrhythmias in the setting of sports medical screening, even in the absence of symptoms and/or signs of left ventricular dysfunction, in order to rule out an inflammatory pathology of the myopericardium at high risk of threatening arrhythmias.

2. Case Report

The author presents two anedoctal cases of young athletes who underwent an examination for competitive sports fitness in the setting of forensic sports medicine as required by Italian law. The first case concerns a 21-year-old amateur cyclist who, apparently healthy and in excellent fitness, showed at the resting ECG ventricular repolarization abnormalities (i.c. Negative T waves in the right precordial leads from V1 to V3) suggestive of arrhythmogenic right ventricular disease. The exercise stress test showed the appearance of an inducible ectopic ventricular arrhythmia at low external workload. In particular, the ECG tracing showed multiple, polyfocal and polymorphous BPVs, predominantly from the right ventricle, in the absence of symptoms, throughout the duration of the stress test. An echocardiogram was performed for these findings, which showed mild dilatation of the right heart chambers with preserved global contractility and biventricular systolic function. A subsequent 24 h Holter ECG confirmed the presence of multiple polymorphic BPVs, even in couples, with a frequency exceeding 5000 BPV over 24 h. Consequently the athlete underwent cardiac magnetic resonance imaging, repeated twice only 4 months apart, which revealed functional morphological aspects that could not be interpreted unequivocally due to the presence of a slight biventricular dilatation associated with a slight reduction in global contractility, but above all because of the presence of SIV dyskinesia in the absence of a branch block and fibrosis/scar of the inferior wall of the VDx (Fig. 1). In view of the diagnosis of myopericarditis (i.e. pericardial enhancement and some areas of LGE also affecting the VSn, and greater involvement of the VDx), partly resolved, and the volumetric reduction of the VDx, the most probable hypothesis was that these were myocardial inflammatory damage findings with predominantly VDx involvement, since in favour of a suspected MRI of ARVDc there would only be the presence of the aforementioned late gadolinium enhancement (LGE) but not the presence of adipose infiltration, without significant dilatation and/or dyskinesias of the right ventricle. In this regard, follow -up monitoring (at least 6 months) was suggested. The second case concerned a 32-year-old athlete, an amateur soccer player, who was completely asymptomatic and presented for renewal of his competitive fitness. The clinical course was superimposable on the previous case, as the exercise ECG stress test showed polyfocal, polymorphous BPVs, even in couples, in the absence of symptoms. The 24-hour Holter ECG confirmed the presence of multiple polymorphic and polyfocal ventricular ectopic beats, while an echocardiogram was within normal limits for normal cavity diameters and parietal thicknesses, with preserved biventricular systolic function, and only minimal pericardial effusion. However, a cardiac MRI showed extensive signs of subepicardial late enhancement (LGE) nearly circumferential to the mid segments, at the level of the inferior wall and inferoseptal to the basal segments, and at the level of the inferior wall and inferolateral to the apical segments (Fig. 2), with minimal evidence of pericardial effusion. These findings suggested the diagnosis of extensive subacute myopericarditis. Consequently, this athlete was also suspended from competitive sporting activity and training for six months, as recommended by all sports cardiology experts.

3. Discussion

The presentation of myocarditis in athletes is multifaceted and the precise definition of the diagnosis is difficult, and there is no uniform clinical and work-up protocol. The combined clinical aspects of symptoms, electrocardiography, laboratory tests, echocardiography, cardiac MRI and, in some cases, endomyocardial biopsy help to establish the diagnosis with certainty.6 As reported in the literature, most patients with myocarditis heal spontaneously; however, athletes may be at increased risk of adverse cardiac events. Based on the limited evidence and especially autopsy studies, current recommendations generally recommend suspension from competitive sports for a minimum of 3 to 6 months.

The diagnosis of subacute myopericarditis in asymptomatic athletes is indeed a challenge for physicians and sports cardiology, as its presentation is atypical and subtle. However, the key element for clinical suspicion is always the presence of equivocal features at resting ECG and the presence of complex ventricular ectopy at pre-sports screening. This must always alert the physician certifying competitive sports fitness. It follows that early diagnosis and specific treatment strategies are necessary to reduce mortality and the need for heart transplantation in these patients.7-8

4. Conclusion

In summary, many questions remain unanswered regarding the actual pathogenesis of myocarditis and the role of possible viral infection, immune system, host genetic background and environment in disease progression and prognosis. This manuscript re-proposes the dilemma of imposing even prolonged and perhaps unnecessary disqualification on athletes to avoid adverse cardiac events, which can cause significant disruptions to training programmes and tournament preparation and lead to decreased performance and ability to compete. Therefore, better risk stratification tools are needed.

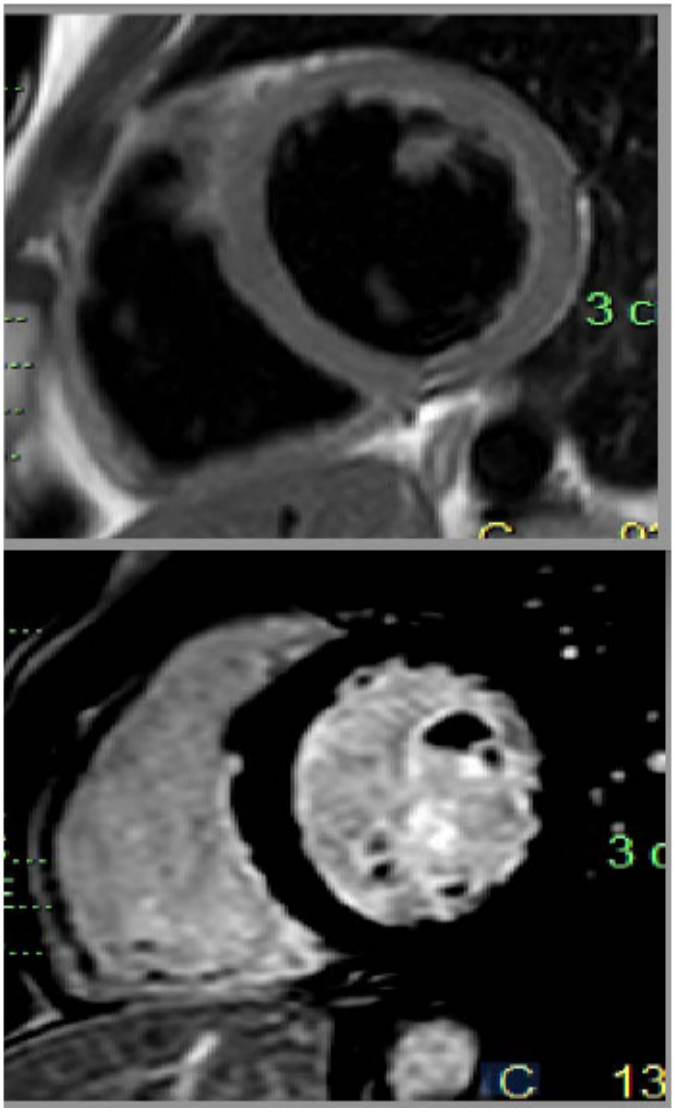

Figure 1. *Showed a slight biventricular dilatation associated with a slight reduction in global contractility, SIV dyskinesia with pericardial enhancement and some areas of LGE also affecting the VSn, and greater involvement of the Vdx, without adipose infiltration*

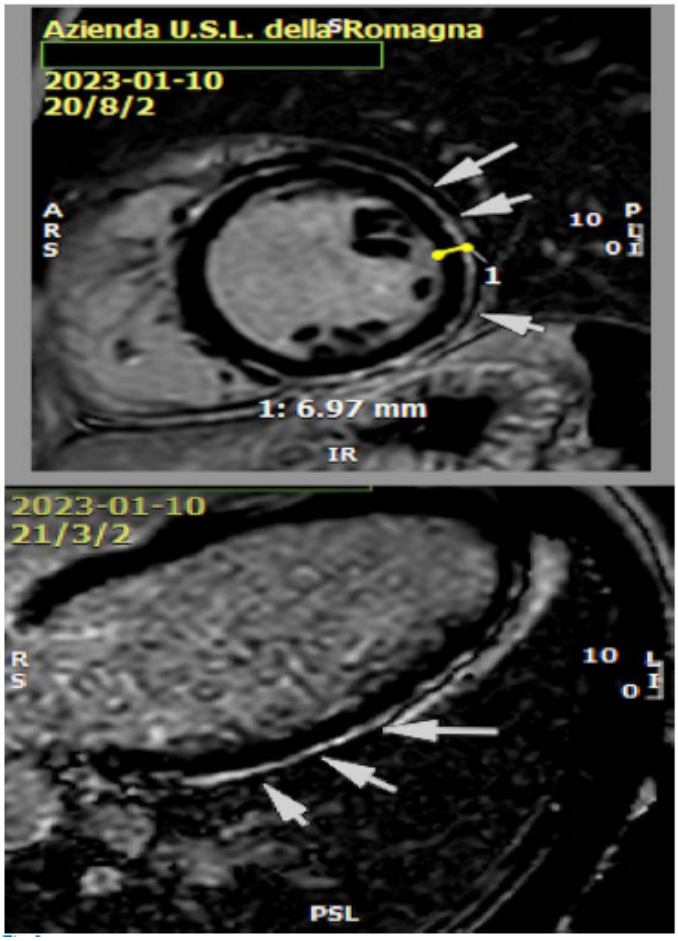

Figure 2. *Showed extensive signs of subepicardial late enhancement (LGE) nearly circumferential to the mid segments, at the level of the inferior wall and inferoseptal to the basal segments, and at the level of the inferior wall and inferolateral to the apical segments*

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