Letter to the Editor
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Elite athletes with recurrent ERS

We read with interest the recent paper by Colivicchi et al., which reported on the clinical presentation, diagnostic work-up and long-term outcome of a consecutive series of elite athletes (n = 33) with recurrent exercise-related syncope (ERS).1

The athletes were referred to the authors' syncope clinic after 12-lead ECG and transthoracic echocardiography. The only structural heart disease detected was mitral valve prolapse with mild mitral regurgitation in two individuals. Subsequently, Holter monitoring, exercise testing, electro-encephalography, electrophysiological testing and head-up tilt testing were performed, although the numbers of each investigation performed was not stated. Most of the athletes (66%) had a positive response to tilt testing, while exercise-related hypotension was noted in four athletes, three of whom had a positive response to tilt testing. Follow-up suggested a benign process and outcome such that a completely negative work-up was felt to justify continued participation by the athlete in competitive elite sport, although the role for tilt testing was not stated. The discussion then focussed on the exclusion of structural heart disease as being of prime importance.

We are concerned, however, that the group of athletes investigated is highly selected and that the message being presented may be misleading. The population studied is open to bias as only athletes with two or more episodes of ERS were included. This may lead to exclusion of athletes with potentially lethal underlying heart disease who may experience only one syncopal episode before ceasing competitive sport, or who may not have survived a first episode. There may therefore be less severe or easily diagnosed cardiac disease present in the studied individuals.

The lack of detected significant structural heart disease was very surprising, as was the lack of ion channelopathy disease, such as long QT syndrome. LQT1, for example, is known to be associated with exertional or post-exertional syncope.2 Neither was there any mention of provocative investigation for the ion channel-opathies in general.

In addition, it is well documented that some cases of hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) are associated with either very mild or even normal phenotypes where both ECG and echocardiography are either normal or borderline.3,4 These individuals remain at increased risk of sudden cardiac death (SCD) and can suffer ERS. Ion channelopathies can be notoriously difficult to diagnose or exclude and can still be a cause of ERS and SCD.5 By adopting the authors' recommendations, athletes with an unremarkable work-up in the context of ERS could be wrongly reassured and allowed to continue in competitive sports.

We believe that ERS in a competitive athlete should be considered as an aborted cardiac arrest until exhaustive investigations have ruled out the main causes of sudden death in this group. The investigations should not only look for structural heart disease but also focus on the ion channelopathies, which are important causes of SCD in young athletes in the absence of structural cardiac abnormality. Until this has been done, athletes should avoid competition and strenuous activity.

References


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