We report the results of a study carried out in 825 young football club members, all of whom underwent screening for hypertrophic cardiomyopathy using a 12-lead ECG. Echocardiographic assessment was performed in only those with positive ECG results, as defined by the European Society of Cardiology. Echocardiography proved necessary in 61 (7%) individuals with positive ECG findings, of whom 7 had echocardiographic findings indicative of left ventricular hypertrophy: five were in the “gray zone,” 1 was judged to have athlete’s heart, and one had been diagnosed with hypertrophic cardiomyopathy. In all these cases, ECG showed repolarization abnormalities, and 4 satisfied criteria for left ventricular hypertrophy.

Key words: Sudden death in athletes. Hypertrophic cardiomyopathy. Sports screening.

INTRODUCTION

Although incidence is low, the sudden, unexpected death of athletes has substantial medical and social repercussions. Preparticipation screening in competitive sports has aroused debate and controversy. As Boraita et al suggest, a test with sufficient sensitivity and specificity to cut false positives and negatives to the absolute minimum is required. However, above all, healthcare systems must recognize the need to dedicate resources to developing systematic, preventative measures that enable us to identify individuals at risk.

Hypertrophic cardiomyopathy (HCM), present in 1/500 births, is the principal cause of sudden death in athletes in the US and a frequent cause in Spain. Suárez-Mier et al found that, in Spain, arrhythmogenic dysplasia was the principal, identifiable cause of sudden death in athletes aged <30 years, followed by severe left ventricular hypertrophy. Cause of death could not be determined in >30% of cases.

Hypertrophic cardiomyopathy can be diagnosed by 12-lead electrocardiogram (ECG) in 75%-95% of patients. However, in Italy, where a rigorous program of preparticipation screening for competitive sports exists, arrhythmogenic right ventricular dysplasia is the most frequent cause of sudden death in athletes and the lower
incidence of deaths from HCM is attributed to the screening program. This has moved cardiology societies (European Society of Cardiology [ESC], Spanish Society of Cardiology [SEC]) to recommend ECGs be included in basic preparticipation screening.4-6 In Spain, however, no legal obligation to screen athletes exists and sports federations do not require athletes to undergo screening prior to accepting their affiliation.

The objective of our study is to determine the usefulness of SEC and ESC recommendations on preparticipation cardiovascular testing prior to affiliating footballers.

METHODS

From September thru December 2006, we studied 825 consecutive teenage and adult footballers, affiliated to the Football Federation of the autonomous region of Asturias in northwestern Spain. All attended appointments at the Escuela de Medicina Deportiva de Asturias where they underwent basic cardiologic screening including medical history with personal and family antecedents and physical examination. All underwent resting 12-lead ECG. Positivity criteria are in Table 1 and are those accepted by the ESC (2005). Athletes with electrocardiographic criteria of positivity were referred for echocardiography with Sonos 5500 equipment. We defined left ventricular hypertrophy as >13 mm interventricular septal thickness and, in line with earlier publications, considered up to 55 mm left ventricular diastolic diameter to be normal. In all cases, we analyzed systolic and diastolic function. Where exercise testing was indicated, this was conducted on an exercise bicycle following a protocol with 50 W increases every 2 min. In athletes with left ventricular hypertrophy, after the echocardiogram, we took blood samples for genetic analysis (analyzing MYBPC3 gene), which was conducted in the molecular genetics laboratory of our center. Results are expressed as mean (SD).

RESULTS

We studied 825 affiliated footballers (95.5% men; mean age, 21.7 [5.4] years); 61 fulfilled ESC electrocardiographic positivity criteria (Table 1) and were referred for echocardiography. Mean hours of training per day were 2 (0.1) on 3.2 (1.4) days per week over 11 (4) years.

Medical History

Two teenage athletes referred for echocardiographic examination reported a history of non-effort related syncope and had no relevant family history. Another 2 reported undocumented family history of sudden death and, finally, in 2 clinical records we found a history of palpitations with no associated clinical condition. In infancy, 1 individual had been operated for interventricular communication and periodically attended follow-up.

Electrocardiographic Findings

In 35 athletes, we identified repolarization abnormalities defined as ST-segment depression or negative T-wave in 2 or more leads according to ESC criteria; right bundle branch block (RBBB) in 8; voltage criteria in 4; Wolff-Parkinson-White (WPW) syndrome in 4 (2 right posteroseptal pathways and 2 left lateral pathways);
borderline QT in 1; right axis deviation to the in 5; and ectopic ventricular beats in 4.

**Echocardiographic Findings**

Mean septal thickness was 11 (2) (8-23) mm and mean posterior wall thickness, 10 (1.3) (8-14) mm. In 7 footballers we found >13 mm septal thickness, defined as left ventricular hypertrophy. In these 7, the ECG showed repolarization abnormalities (ST-segment depression or negative T-wave) and in 4 of them, left ventricular growth criteria; we included 5 in the “gray area” (13-15 mm septal thickness, non-dilated left ventricle, with normal systolic and diastolic function) and 1 was considered as athlete’s heart with physiologic hypertrophy associated with left ventricular dilatation. Finally, the last individual, who was asymptomatic, presented 23 mm septal thickness. The electrocardiographic finding was ventricular growth with voltage criteria and ST-segment depression; the echocardiographic study revealed no other findings characteristic of the disease such as systolic anterior motion (SAM) or subaortic gradient. This individual was advised against participating in competitive sports. We conducted family screening and genetic analysis in the individual (a carrier of the MYBPC 3-Arg495Trp mutation in exon 18) and his family members (mother and grandfather were both carriers of the same mutation), and they also underwent echocardiography; we encountered HCM criteria in the grandfather. Other echocardiographic findings were residual interventricular communication, 1 case of mild pulmonary stenosis and 1 of mitral and tricuspid valve dysplasia, both redundant and thickened, without hemodynamic repercussions. None of the athletes with preexcitation syndrome reported symptoms and all underwent stress-exercise testing with sudden disappearance of preexcitation in 3 cases, and intermittent pathways in the fourth. In all we recommended regular follow-up. In the case of the individual with asymptomatic, borderline QT, without family history, we also performed exercise testing, which showed normalization of QT interval, and did not advise against practicing sports.

**DISCUSSION**

From time to time, when the sudden death of an athlete occurs, the controversy over compulsory preparticipation medical screening for athletes and the inclusion of the ECG in this, reappears. Our results coincide with Pellicia et al, who evaluated 4450 Italian athletes, and concluded that ECG is an efficient weapon in the identification of HCM. Moreover, they consider the inclusion of the echocardiographic study as a standard test in the screening programs would not be necessary to detect HCM. However, the difficulty arises in the so-called “gray zone,” and the question is: Are we only dealing with athlete’s heart? According to ESC recommendations, in cases of ECG abnormalities with high voltages, T-wave inversion or deep precordial Q wave, a more exhaustive examination should be conducted including a family study, echocardiogram, and diastolic parameter study, and 24 hour Holter. If doubt persists, cardiovascular resonance imaging should be conducted and athletes should be recommended to temporarily stop participating in competitive sports in order to determine whether findings are reversible or not. Similarly, identifying WPW syndrome, long QT, or Brugada syndrome would entail more exhaustive screening.

Our results demonstrate the ECG is a useful, accessible weapon in basic preparticipation sports screening—something already proven in Italy—not only to determine HCM, but also to rule out other entities. We must make special mention of arrhythmogenic dysplasia and coronary abnormalities, as screening for these seems less efficient.

In view of our results, and in line with earlier publications, we should consider screening athletes for HCM and preventing sudden death through the use of 12-lead ECG. Echocardiography should be reserved for individuals with positive ECG. Electrocardiographic analysis would facilitate the detection of other potentially lethal entities.

Healthcare professionals should try to guarantee that practicing sport entails no risks, especially when we encourage the practice of physical exercise as a healthy lifestyle habit. Consequently, we are obliged to promote and apply screening prior to practicing sports in line with scientific societies’ clinical practice guidelines.

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