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CASE REPORT

Recurrent repolarisation abnormalities in an athlete

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Introduction

Electrocardiographic changes due to regular physical exercise are well recognised and result from a combination of structural cardiac changes and increased vagal tone [1–6]. However, abnormalities on the athlete's resting electrocardiogram (ECG) may be the first indication of an underlying electrical or structural cardiac disorder, and may precede symptoms and macroscopic structural abnormalities by several years [7]. The first manifestation of such disorders may be a malignant arrhythmia or sudden cardiac death (SCD) [3]. Considering that physical exertion is a trigger for fatal arrhythmias in cardiomyopathies or primary electrical cardiac disorders, differentiating cardiac pathology from physiological adaptation in athletes is crucial [3, 8, 9].

Athletes of African/Afro-Caribbean (black) ethnicity comprise a rapidly growing population of athletes in Western Europe and such individuals frequently develop a greater magnitude of left ventricular hypertrophy (LVH) compared with their Caucasian counterparts (white athletes) and display a high frequency of repolarisation anomalies that overlap with the hypertrophic cardiomyopathy phenotype [1, 2, 4, 6, 7]. Differentiating between the 'athlete's heart' and potentially lethal cardiac abnormalities is occasionally challenging, particularly in black athletes.

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Case

A 20-year-old black athlete engaged in middle-long distance running and soccer underwent a cardiovascular assessment, including a health questionnaire and ECG. He was asymptomatic and had no family history for SCD or inherited cardiac diseases. He was not taking regular medication or performance-enhancing drugs. A 12-lead ECG revealed voltage criteria for LVH and widespread repolarisation abnormalities with convex ST-segment elevation in leads V2-V4, biphasic T waves in lead II, and T-wave inversion in leads III, aVF, V1-6 (Fig. 1). Subsequent echocardiography demonstrated normal biventricular systolic function. The left ventricular dimension at end diastole measured 55 mm with a maximum left ventricular wall thickness of 12 mm. Indices of diastolic function were normal and there were no other features to suggest an underlying cardiomyopathy. Considering the distinct repolarisation abnormalities on his ECG, a cardiopulmonary exercise test (CPEx) and cardiac magnetic resonance imaging (CMRI) were performed. The CMRI revealed mild biventricular enlargement with normal ventricular function and no evidence of late gadolinium enhancement to indicate myocardial fibrosis. The athlete demonstrated a maximal oxygen consumption of 42.57 ml/min/kg (95 % predicted) with normal blood pressure response and no ECG changes or arrhythmias. A 24-h Holter monitor did not reveal any arrhythmias.

We were unable to confirm or refute the diagnosis of an underlying cardiomyopathy with these results, but when reviewed in our outpatient clinic to discuss the test results it appeared that the athlete had been forced to stop exercising for 4 weeks because of a lower limb injury. The ECG at this point demonstrated complete resolution of the T-wave inversions in all leads except lead III and V1 (Fig. 2). Following recovery he resumed training and an ECG performed 6 weeks afterwards revealed recurrence of marked repolarisation changes (Fig. 3).

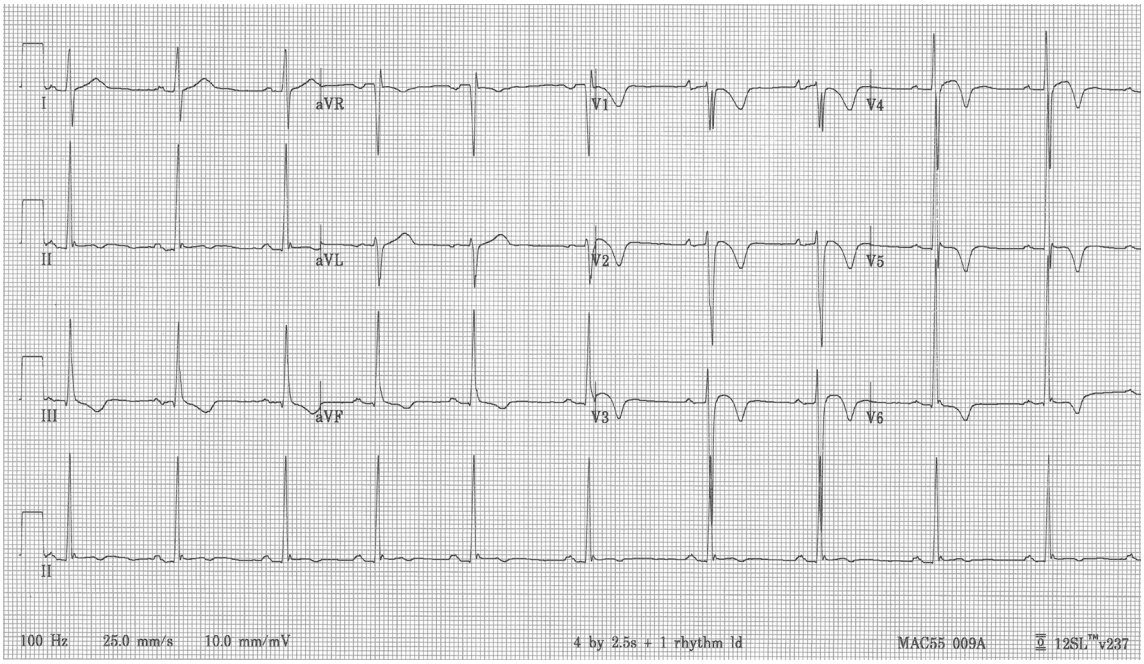

Fig. 1 The athlete's first ECG with voltage criteria for LVH, convex ST-segment elevation in leads V2-V4, biphasic T waves in lead II and T-wave inversion in leads III, aVF, V1-6

Discussion

The extent of cardiovascular changes induced by regular physical exercise is influenced by several factors including gender, sports classification (static, dynamic or combined), age and ethnicity. In highly trained black athletes, repolarisation abnormalities are a common finding, with up to 22.8 % of individuals demonstrating T-wave inversion in two or more contiguous leads versus 3.7 % of white athletes [4]. These ECG changes overlap with those observed in several conditions associated with SCD in athletes, including arrhythmogenic right ventricular cardiomyopathy (ARVC) and hypertrophic cardiomyopathy (HCM) [1, 2, 4–6, 10]. SCD due to HCM is reported to be commoner in black athletes

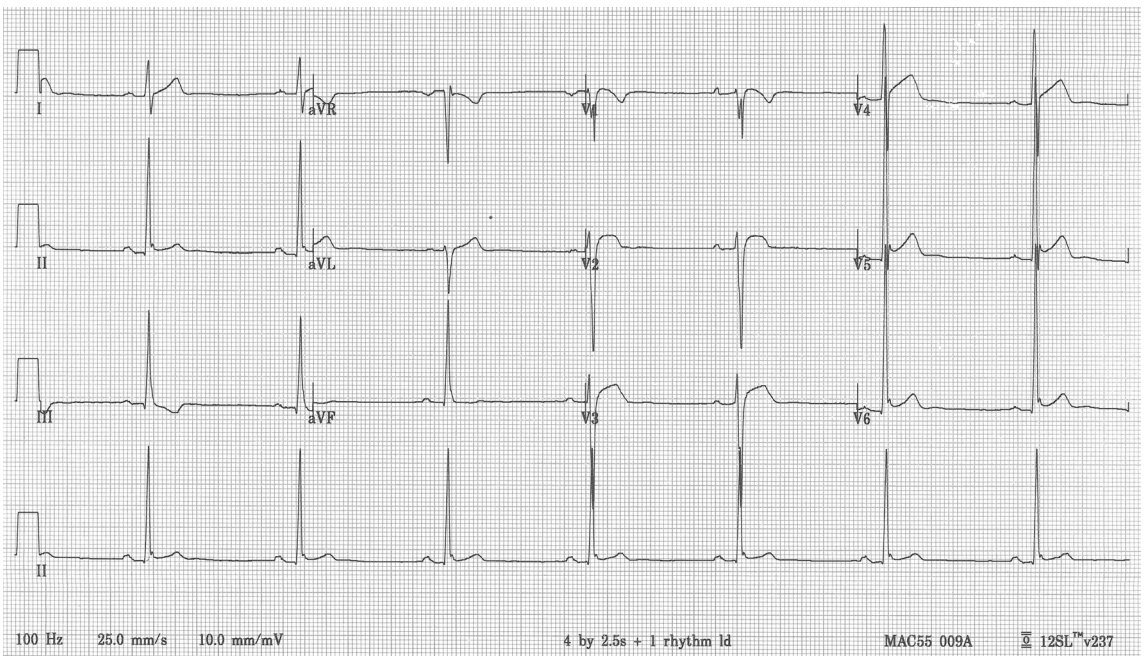

Fig. 2 The ECG after detraining, with resolution of the T-wave inversion in leads II, aVF, V2-V6

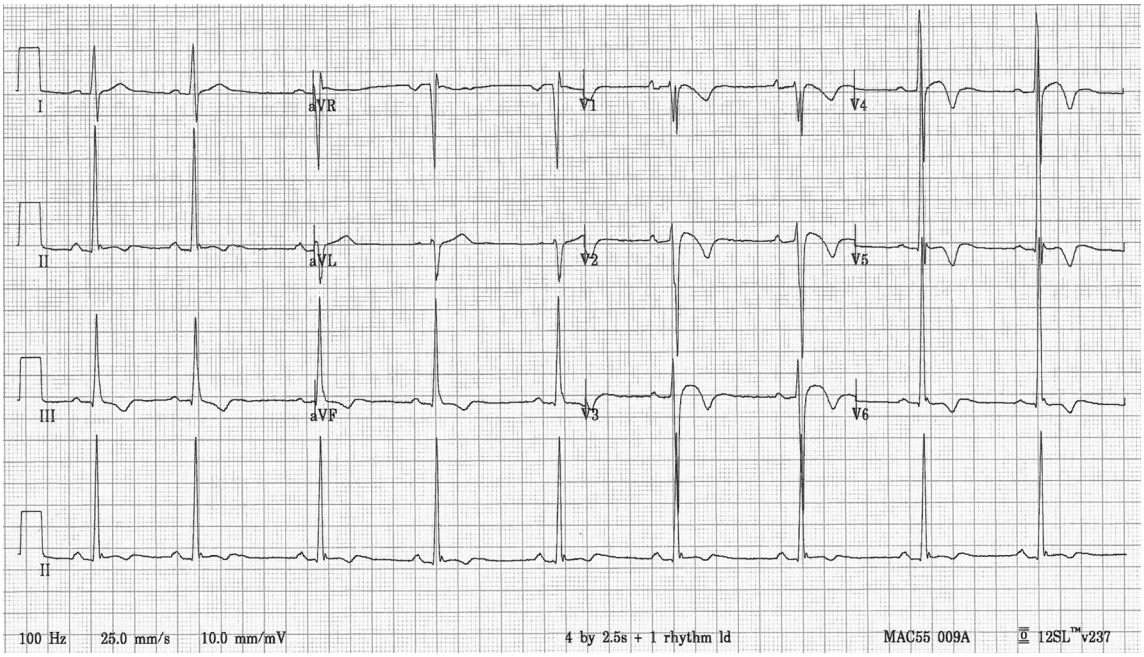

Fig. 3 The athlete's ECG 6 weeks after resuming his training, with reappearance of the biphasic T waves in lead II and inverted T waves in leads aVF and V2-6

than in white athletes and therefore the combination of repolarisation abnormalities and LVH pose a major conundrum in black athletes [9].

From observations and long-term follow-up in large cohorts of black athletes, T-wave inversion in leads V1-V4 preceded by a convex ST-segment elevation in an otherwise asymptomatic black athlete with no family history of premature SCD or cardiomyopathy and a normal cardiovascular examination may be considered an ethnic variant in response to training and does not require further testing [1, 4, 10]. In contrast, we have noted aborted sudden cardiac death and development of cardiomyopathy in a small minority of black athletes with inferolateral T-wave inversion. Given the high prevalence of T-wave inversion in the inferolateral leads in patients with HCM (>80 %), we recommend that this particular pattern of repolarisation changes should be viewed with caution and trigger comprehensive evaluation [4, 11].

The differentiation between physiological cardiac adaptation and a cardiomyopathy requires a combination of several imaging modalities, exercise stress test with simultaneous gas exchange measurements and heart rhythm monitoring as well as evaluation of first-degree relatives [2]. If the echocardiogram is inconclusive a cardiac MRI is recommended, considering its superior spatial resolution and the possibility of demonstrating late gadolinium enhancement, which is associated with exercise-related ventricular arrhythmias [1, 12]. A period of physical inactivity (detraining) to demonstrate a regression of training-related changes in order to exclude an underlying cardiomyopathy is often regarded as the last resort [13, 14]. In our athlete an accidental detraining period of just 4 weeks resulted in the complete resolution of the repolarisation abnormalities and the evaluation failed to show any of the broader phenotypic features of hypertrophic cardiomyopathy. Our case illustrates that T-wave inversion in the inferolateral leads, although common in black patients with HCM, does not necessarily implicate underlying pathology in a black athlete. A relatively short period of deconditioning could be used to resolve this clinical dilemma.

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