BMJ Case Reports

Myth exploded

T-wave inversions and the role of de-training in the differentiation of athlete's heart from pathology: is 6 months too long?

Gregory Whyte,[1] John Somauroo,[2] Mathew Wilson,[3] Sanjay Sharma[4]

[1]Research Institute for Sport and Exercise Science, Liverpool John Moores University, Liverpool, UK;
[2]Cardiology Department, Countess of Chester Hospital, Chester, Cheshire, UK;
[3]Department of Sports Medicine, National Orthopaedic Hospital, Doha, Qatar;
[4]Department of Cardiology, King's College Hospital, London, UK

Correspondence to Professor Gregory Whyte, gregwhyte27@yahoo.co.uk

Summary

Electrocardiographic changes are common in athletes. Differentiation of a physiological from a pathological substrate is important as ECG changes may indicate underlying cardiac disease placing the athlete at increased risk of sudden cardiac death. Deep T-wave inversions are uncommon in Caucasian athletes however; appear more prevalent in black athletes. Irrespective of the ethnic origin of the athlete, deep T-wave inversions require thorough follow-up. At present, 6 months de-training is recommended to assist in the differentiation of physiologic and pathologic changes where a definitive diagnosis is elusive through standard diagnostic techniques. This case study examines findings from a black and a Caucasian athlete presenting with deep T-wave inversions following a brief (ca.3 week) period of de-training resulting in normalisation of T-wave. These cases suggest that a shorter period of time may be sufficient in differentiating physiological from pathological mechanisms for deep T-wave inversions.

BACKGROUND

Sudden cardiac death in young athletes is highly publicised events that have profound effects upon the athlete's family, the sport and the community as a whole. While they are uncommon events, the sudden cardiac death of an athlete may be avoidable. Differentiation physiologically induced changes in the athlete's heart from pathological causes is important in preventing death. [1-3] Unfortunately, this is not always simple.[4 5] The present case report adds new and important information in this differential diagnostic dilemma.

CASE PRESENTATION

Case 1 A 27-year-old black South African Premiership and International football player presented following an episode of presyncope and an episode of transient loss of consciousness (reported as a 'few seconds'). Both episodes occurred on hot days following training concomitant to symptoms of nausea and overheating without chest pain, palpitations or breathlessness.

Case 2 A 19-year-old Caucasian male Premiership rugby player presented following an episode of prolonged palpitations during a rugby game, which lasted for 15 min before half time and persisted throughout half time and for a further 15 min into the second half (45 min total with heart rate was documented at 170 beats/min with symptoms including dyspnoea, lethargy and light headedness).

INVESTIGATIONS

Case 1 He was otherwise asymptomatic with no family history of note. Physical examination was normal. Resting ECG revealed Sokolow-Lyon voltage criteria for left ventricular hypertrophy (LVH) and marked repolarisation changes in leads V1–V3 comprising of deep T-wave inversion (>−0.2 mV) in lead V1 and convex ST segment elevation in V2 and V3 (figure 1a). Echocardiography revealed mild LVH (13 mm) and mild left ventricular cavity enlargement (60 mm) with normal function. The origins of both coronary arteries were identified and were normal. Integrated cardiopulmonary exercise stress testing (CPEX) and cardiac MRI were entirely normal.

Case 2 He was subsequently transferred to hospital and underwent an ECG which showed normal sinus rhythm. The ECG demonstrated deep T wave inversion (>−0.2 mV) in V2–V4 and widespread T-wave inversion in most of the other leads (figure 2a). His subsequent echocardiogram, 48 h tape and CPEX were entirely normal. Cardiac MRI was entirely normal including the coronary origins and initial course, and absence of perfusion or gadolinium abnormalities. There was no family history of note and the athlete denied any illicit drug use.

DIFFERENTIAL DIAGNOSIS

- Athlete's heart (physiological adaptation)
- Hypertrophic cardiomyopathy (HCM)
- Arrhythmogenic right ventricular cardiomyopathy (ARVC).

TREATMENT

In light of the grossly abnormal ECG both athletes underwent detraining.

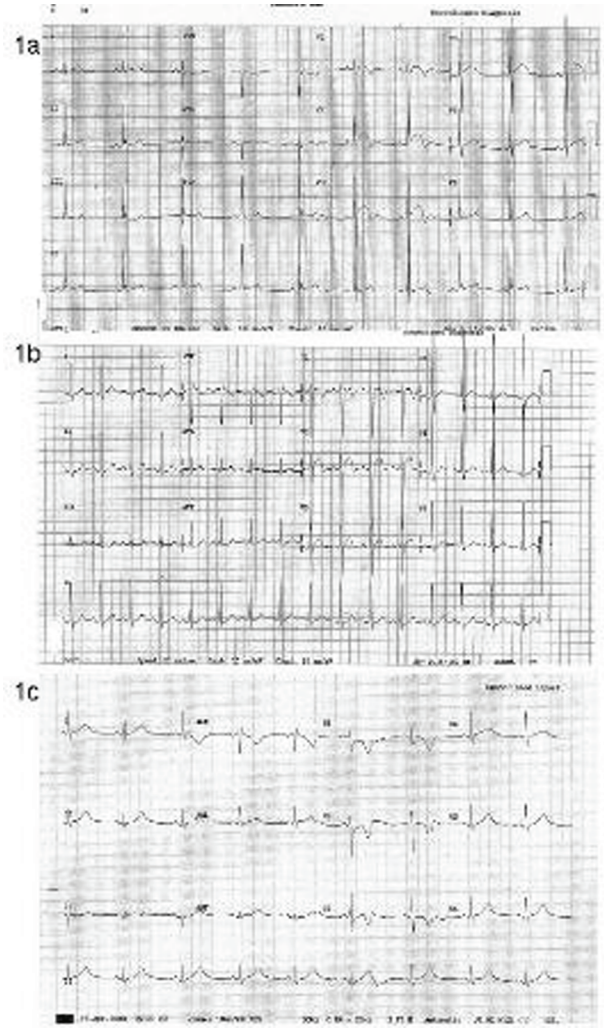

Figure 1 Case 1: Black South African football player ECG demonstrating: (a) Sokolow-Lyon voltage criteria for LVH and marked repolarisation changes in leads V1–V3 comprising of deep T-wave inversion in lead V1 and convex ST segment elevation in V2 and V3; (b) Normalisation of T-wave inversions following 3 weeks de-training; (c) Follow-up ECG at 3 months demonstrating the re-appearance of T-wave inversions in V1–V3 following return to play.

Figure 2 Case 2: Caucasian rugby player ECG demonstrating: (a) deep T-wave inversion in V2–V4 and widespread T-wave inversion in most of the other leads; (b) Normalisation of T-wave inversions following 4 weeks de-training; (c) Follow-up ECG at 6 months demonstrating the re-appearance of T-wave inversion in V1 with convex ST segment elevation and T-wave inversion in V2 and V3.

OUTCOME AND FOLLOW-UP

Case 1 A repeat ECG at 3 weeks demonstrated normalisation of the ST segment (figure 1b). As a result, the athletes' symptoms were felt to represent vaso-vagal episodes and the ECG changes were deemed physiological rather than pathological and the athlete was cleared for training and competition. At 3 month follow-up, the T-wave inversions had returned (figure 1c).

Case 2 A repeat ECG at 4 weeks demonstrated normalisation of the ST segment (figure 2b). Accordingly, the ECG changes were deemed physiological and the athlete was cleared for training and competition. At 6 month follow-up, the T-wave inversions had returned (figure 2c).

DISCUSSION

Deep T-wave inversions in the precordial leads are a major concern as these ECG alterations are a recognised manifestation of HCM and ARVC.[6] Inverted T-waves may represent the only sign of an inherited heart muscle disease even in the absence of any other features or before structural changes in the heart can be detected. T-wave inversions are uncommon in Caucasian athletes however; recent studies have demonstrated that African/Afro–Caribbean black athletes present with more striking repolarisation changes on the ECG and exhibit a greater prevalence of inverted T-waves and magnitude of LVH than Caucasian athletes of similar age and size participating in identical sports.[7 8] Indeed, up to 25% of black athletes exhibit either repolarisation changes or LVH that overlap with morphologically mild HCM.[8] Whether the repolarisation changes observed in the black football player in the present case study reflect an adaptation due to regular, intensive exercise or his ethic origin remains unknown, as almost all studies of athlete's heart have been performed on European Caucasian individuals.[9] Irrespective of the ethnic origin, the presence of T-wave inversions in the precordial leads (except V1) requires

BMJ Case Reports 2012; doi:10.1136/bcr.06.2011.4403

thorough investigations given the potentially deleterious outcome.

Following exhaustive clinical follow-up without conclusive diagnosis, the European Society of Cardiology recommend a period of up to 6 months de-training to assist in the differentiation of physiology and pathology.[10] A period of 6 months abstinence from training and competition is often unpalatable for an athlete and based upon the two cases presented here may be unnecessarily long. Serial investigations at short time periods (ca.3 weeks) throughout the period of de-training are recommended to ensure the athlete does not spend unnecessary time out of training and competition.

The two players identified in this case study presented a diagnostic conundrum. Despite a comprehensive clinical investigation and the reversal of the T-wave anomalies following de-training, the authors were unable to explain the clinical significance of the deep T-wave inversions. Recently, Pelliccia *et al*.[11] reported on 81 athletes (from a database of 12 550) with diffusely distributed and deeply inverted T-waves, with no apparent cardiac disease, whom had undergone serial clinical, ECG, and echocardiographic studies for 9±7 years (range, 1 to 27 years). From the 81 identified, five (6%) ultimately proved to have cardiomyopathies, including one who died suddenly from undetected ARVC. The authors suggest that these abnormal ECG's may represent the initial expression of genetic cardiac disease that may not be evident for many years. A recent case study by Zeller and colleagues[12] recognised the difficulty in providing medical clearance for athletes with marked T-wave inversions after the cardiac death of a young black African footballer following 60 min of strenuous exercise. Remarkably, 15 days prior to death, the player underwent full cardiovascular screening. Denying personal symptoms or family history of sudden cardiac death, the ECG demonstrated diffuse T-wave inversions in V3–V6. Cardiopulmonary exercise testing and echocardiography were normal. Autopsy, however; revealed mild fibrosis of the septum but without myofiber disarray, and with no signs of ARVC or anomalous coronary arteries. In line with these findings, we have recommended an annual review of the two players presented in this case study.

Learning points

- T-wave inversions require thorough follow-up investigations.
- The clinical significance of T-wave inversions remains elusive.
- Requesting a period of de-training to identify the aetiology of these changes should be accompanied by serial follow-up at short time intervals (ca.3 weeks) to avoid unnecessary delay to return to training and competition.
- The continued clinical surveillance and clinical follow-up for these athletes is important; even in the absence cardiac symptoms and/or clinically demonstrable heart disease.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Oakley CM.** The electrocardiogram in the highly trained athlete. *Cardiol Clin* 1992;**10**:295–302.
2. **Sharma S,** Whyte G, Elliott P, *et al.* Electrocardiographic changes in 1000 highly trained junior elite athletes. *Br J Sports Med* 1999;**33**:319–24.
3. **Barbier J,** Ville N, Kervio G, *et al.* Sports-specific features of athlete's heart and their relation to echocardiographic parameters. *Herz* 2006;**31**:531–43.
4. **Corrado D,** Basso C, Thiene G. Essay: sudden death in young athletes. Lancet 2005;**366**:S47–8.
5. **Maron BJ,** Epstein SE, Roberts WC. Causes of sudden death in competitive athletes. *J Am Coll Cardiol* 1986;**7**:204–14.
6. **Corrado D,** Pelliccia A, Heidbuchel H, *et al.* Recommendations for interpretation of 12-lead electrocardiogram in the athlete. *Eur Heart J* 2010;**31**:243–59.
7. **Magalski A,** Maron BJ, Main ML, *et al.* Relation of race to electrocardiographic patterns in elite American football players. *J Am Coll Cardiol* 2008;**51**:2250–5.
8. **Basavarajaiah S,** Boraita A, Whyte G, *et al*. Ethnic differences in left ventricular remodeling in highly-trained athletes relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2008;**51**:2256–62.
9. **Rawlins J,** Carre F, Kervio G, *et al.* Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. *Circulation* 2010;**121**:1078–85.
10. **Corrado D,** Pelliccia A, Bjørnstad HH, *et al.* Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus statement of the study group of sport cardiology of the working group of cardiac rehabilitation and exercise physiology and the working group of myocardial and pericardial diseases of the european society of cardiology. *Eur Heart J* 2005;**26**:516–24.
11. **Pelliccia A,** Di Paolo FM, Quattrini FM, *et al.* Outcomes in athletes with marked ECG repolarization abnormalities. *N Engl J Med* 2008;**358**:152–61.
12. **Zeller L,** Giladi H, Golan R, *et al.* Sudden death in a young soccer player with marked electrocardiographic repolarization abnormalities. *Clin J Sport Med* 2010;**20**:66–8.