CASE REPORT

Normal Variant T-Wave Changes in an Athlete with Structurally Normal Cardiac Anatomy and Function

Adam M. Noyes, M.D.,* and Peter Schulman, M.D.†

From the *University of Connecticut, Internal Medicine, Farmington, CT; and †University of Connecticut, Cardiology, Farmington, CT

Athletes who perform regular and intensive physical activity may undergo structural and electrical remodeling of the heart that results in electrocardiographic changes that can cause concern. Marked T-wave inversion may represent one such physiologic change. On the other hand, T-wave inversion could be a sign of inherited heart muscle disease or may be a normal variant. Therefore, it is imperative to determine whether abnormalities on an athlete's electrocardiography (ECG) reflect underlying cardiac disease that could place the athlete at risk for sudden cardiac death. For athletes who present with markedly abnormal ECGs, the echocardiography and cardiac magnetic resonance imaging should be considered to evaluate the potential for cardiac disease. We report the case of a high-intensity athlete with concerning ECG changes who required additional studies to exclude cardiac disease.

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Regular and intensive physical activity in athletes may result in structural and electrical remodeling of the heart resulting in changes seen on the electrocardiogram (ECG). Up to 60% of athletes demonstrate ECG changes,¹ posing dilemmas for sports cardiologists tasked with providing medical clearance for intense training and competition. Abnormal ECG findings may be an expression of underlying cardiac disease, placing the athlete at risk of sudden cardiac death (SCD). Therefore, it is imperative to distinguish physiologic from pathologic ECG abnormalities. The ECG findings need to be considered on an individualized basis, as they may vary according to age, sex, ethnicity, and length and level of training.^{2,3} Certain repolarization variances, specifically T-wave inversions (TWIs), are rare in athletes but are common manifestations in individuals with hypertrophic cardiomyopathy (HCM),⁴ which would preclude participation in most sports due to its risk of SCD. The prevalence of TWIs in adult athletes is 3–4%,⁵ and varies by race.^{2,3}

We present a case of a minimally symptomatic athlete displaying marked TWI on the ECG with a review of the cardiology and sports medicine

literature regarding the significance of TWI in athletes demonstrating structurally normal hearts.

CASE PRESENTATION

A 26-year-old African American professional (American) football player was referred to the cardiology clinic because of an abnormal ECG on a preparticipation physical exam for a new team. His resting ECG showed normal sinus rhythm at 58 beats per minute, a normal axis, a PR interval of 106 milliseconds, with voltage criteria for left ventricular hypertrophy (LVH). There was marked asymmetric TWIs in lead V₄, V₅, V₆, biphasic T waves in V₃, and asymmetric inverted T waves in lead I, II, III aVF. (Fig. 1) The patient also described occasional episodes of a very fast heartbeat, unrelated to exertion, associated with mild dizziness but never with lost consciousness. He also occasionally experienced mild shortness of breath associated with these episodes, but denied ever having chest pain. The duration of these episodes were highly variable, and there was no clear precipitating or relieving factors. He denied exertional-related syncope, chest pain, or shortness of breath. He

Address for correspondence: Adam MacArthur Noyes, M.D., Department of Internal Medicine University of Connecticut School of Medicine, 263 Farmington Ave, Farmington, 06103-1235, CT, USA. Tel/Fax: +1 (860) 679-2562; Email: adam.noyes@lifespan.org

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Figure 1. 12-lead electrocardiogram.

used no over-the-counter or prescription medications, herbal products, caffeinated energy drinks, or illicit drugs. He never smoked and he drank small amounts of alcohol. His past medical history is only positive for sports related joint injuries to bones and tendons. His father had an enlarged heart in his 20s–30s, but remains alive and well.

On physical examination, he weighed 204 pounds and was 6 feet 4 inches tall. His blood pressure was 120/62, pulse of 72 beats per minute, and a respiratory rate of 12 per minute. On examination, he was a well-built, comfortable appearing African American male. Cardiovascular examination reveals a normal apex impulse, a normal S1, and a physiologically split S2. There were no murmurs or other abnormal sounds. The remainder of his examination was unremarkable.

A transthoracic echocardiogram was obtained (Fig. 2). Left ventricular cavity size and function were normal with an ejection fraction (EF) of 55–60%. There were no valvular abnormalities.

There was a minimal increase in left ventricular (LV) wall thickness with slightly greater thickness in the apical region. Cardiac magnetic resonance (CMR) was performed (Fig. 3). The CMR revealed normal indexed LV volumes and EF, with no evidence of HCM or other abnormalities.

Based on these findings, he was medically cleared to continue participating in high-intensity football. Subsequently, he had no complications or events and continues to have a successful football career 3 years later.

DISCUSSION

This athlete, without a family history of SCD, displayed marked TWI in the setting of normal cardiac imaging. The adaptation of the athletic heart to the constant increase in preload and afterload results in a form of physiological cardiac

Figure 2. 2D transthoracic echocardiogram in a four-chamber view demonstrating mild increased left ventricular wall thickness.

Figure 3. Gadolinium-enhanced T₁-weighted cardiac MRI in the horizontal long axis (four-chamber view).

remodeling, which may have a significant impact on the athletes resting 12-lead ECG. These cardiac changes reflect the intensity and duration of training, with T-wave changes representing the demands of sustained regular physical training.

Abnormal TWI is defined as >1 mm in depth in two or more leads V₂-V₆, II and aVF, or I and aVL (excludes leads III, aVR and V₁). Deep TWIs within the ECG are of major concern

because these are a recognized manifestation of pathological changes on the heart. Moreover, deep inverted T waves are one of the most important alarming indicators of cardiomyopathy in athletes.⁶ Alarming, TWIs found in the lateral or inferolateral leads should raise the possibility for HCM. In a series of asymptomatic athletes ≤35 years old with HCM confirmed by CMR, 62% exhibited TWI.⁷ In a database of 12,550 athletes,⁸ 881 athletes

Table 1. Seattle Criteria for Physiologic and Pathologic ECG Changes¹⁶

Normal ECG findings in athletes	Abnormal ECG findings in athletes
Sinus bradycardia (≥ 30 bpm)	T-wave inversion > 1 mm in depth in two or more leads V_2 – V_6 , II and aVF, or I and aVL (excludes III, aVR and V_1)
Sinus arrhythmia	Left atrial enlargement
Ectopic atrial rhythm	Pathologic Q waves > 3 mm in depth or > 40 ms in duration in two or more leads (except for III and aVR)
Junctional escape rhythm	Ventricular preexcitation
First-degree AV block (PR interval > 200 ms)	Premature ventricular contractions ≥ 2 PVCs per 10 s tracing
Mobitz type I (Wenckebach) second-degree AV block	Intraventricular conduction delay
Incomplete RBBB	Any QRS duration ≥ 140 ms
Isolated QRS voltage criteria for LVH Except: QRS voltage criteria for LVH occurring with any nonvoltage criteria for LVH such as left atrial enlargement, left axis deviation, ST segment depression, T wave inversion or pathological Q waves	Complete LBBB
Early repolarization (ST elevation, J-point elevation, J waves, or terminal QRS slurring)	RVH pattern
Convex (“domed”) ST segment elevation combined with T wave inversion in leads V_1 – V_4 in black/African athletes.	Brugada-like ECG pattern
	ST segment depression ≥ 0.5 mm in depth in two or more leads

were found with diffusely deep inverted T-waves (≥ 2 mm in at least three leads), with 6% ultimately found to have cardiomyopathies upon imaging. This implies that the seemingly healthy athletes who are found with markedly abnormal ECGs may represent the initial expression of underlying cardiomyopathies, of which may not manifest until many years later and that may ultimately be associated with adverse outcomes.

TWIs found on the ECG of the elite athlete have been suggested to be as common as those of amateur level and even those of sedentary controls. Pelliccia et al.⁶ reported a 2.7% prevalence of TWI in 1005 highly trained athletes and 2.3% in a large population of 32,652 young amateur athletes, while Sharma et al.⁹ reported the prevalence of TWIs is similar among elite athletes and sedentary controls (4.4% vs 4.0%, respectively). However, as prevalent as TWI may be, an echocardiographic study of athletes performed by Bialy et al. revealed that LV mass of the dynamic athletes increased compared with the nonathletes.¹⁰

Race may have an impact on the ECG abnormalities in athletes, suggesting that TWIs may be associated with an “ethnic variant of athlete’s heart.” Data from an Italian population of over 40,000 adult Caucasian athletes of ≤ 35 years of

age revealed that $< 5\%$ exhibited TWI (excluding V_1 and aVR),¹ whereas TWI in the anterior leads (V_1 – V_4) is more common in adult black athletes. In a study of 240 black female athletes, prevalence of TWIs was higher when compared with matched nonblack athletes (14% vs 2%).² Additionally, an examination of 904 black athletes and 1819 white athletes aged 14–35 years participating in 22 different sporting disciplines revealed that TWI were present in up to 25% of athletes and half of these individuals exhibit deep (-0.2 mV) TWIs.³ TWI beyond V_2 is a rare abnormality found in only 0.1% of Caucasian adolescent athletes older than 16 years.³ Therefore, TWI in the anterior precordial leads may be part of a normal variant pattern of repolarization in black athletes consisting of convex (“dome” shaped) ST segment elevation followed by TWI in V_1 – V_4 . On the basis of current data, TWIs preceded by ST segment elevation are present in the anterior precordial leads in up to 13% of black athletes and do not require further assessment in the absence of symptoms, positive family history, or abnormal physical examination.^{3,11} Importantly, TWI in the lateral or inferolateral leads (V_5 – V_6 , I and aVL, II and aVF), regardless of ethnicity, is considered abnormal and requires additional testing to rule out HCM.

Approximately 100 young athletes die from sudden cardiac arrests in the United States every year.¹² Current guidelines for screening remain under debate. However, both the American Heart Association (AHA)¹³ and the European Society of Cardiology (ESC)¹⁴ advocate preparticipation cardiac screening using a comprehensive history and physical examination of young athletes, but the European guidelines recommend obtaining routine ECGs largely based on one observational study conducted in Italy.¹⁵ However, the Italian study did not assess the routine use of ECGs compared with more defined screening based on history and physical examination, thus making it difficult to determine if the ECG added to the other components of the examination. Inclusion of a 12-lead ECG in the screening protocol should be performed if the individual is thought to be at risk based on history and physical, and be interpreted by a provider with the ability to distinguish physiologic from pathologic ECG changes (Table 1).¹⁶

The football player identified in this case presented a difficult scenario. He had a history of palpitations and had a family history that was concerning for an inherited cardiomyopathy. His ECG was highly abnormal, and his transthoracic echo showed mild hypertrophy. However, the CMR findings were critical in eliminating a serious heart muscle disorder, and he was able to resume his career at a professional level. Therefore, CMR can be an indispensable tool in the evaluation of these athletes with symptoms possible pathologic ECG findings. After a comprehensive workup, the authors concluded that the ECG abnormality was a normal variant.

REFERENCES

1. Pelliccia A, Culasso F, Di Paolo FM, et al. Prevalence of abnormal electrocardiograms in a large, unselected population undergoing pre-participation cardiovascular screening. *Eur Heart J* 2007;28:2006-2010.
2. 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;121:1078-1085.
3. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J* 2011;32:2304-2313.
4. Ryan MP, Cleland JGF, French JA, et al. The standard electrocardiogram as a screening test for hypertrophic cardiomyopathy. *Am J Cardiol* 1995;76:689-694.
5. Papadakis M, Basavarajaiah S, Rawlins J, et al. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. *Eur Heart J* 2009;30:1728-1735.
6. Pelliccia A, Maron BJ, Culasso F, et al. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation* 2000;102:278-284.
7. Rowin EJ, Maron BJ, Appelbaum E, et al. Significance of false negative electrocardiograms in preparticipation screening of athletes for hypertrophic cardiomyopathy. *Am J Cardiol* 2012;110:1027-1032.
8. Pelliccia A, Di Paolo FM, Quattrini FM, et al. Outcomes in athletes with marked ECG repolarization abnormalities. *N Engl J Med* 2008;358:152-161.
9. Sharma S, Whyte G, Elliott P, et al. Electrocardiographic changes in 1000 highly trained junior elite athletes. *Br J Sports Med* 1999;33:319-324.
10. Bialy D, Derkacz A, Nowosad H, et al. Electrocardiography and echocardiography in athletic heart imaging. *Biol Sport* 2003;20:195-208.
11. Di Paolo FM, Schmied C, Zerguini YA, et al. The athlete's heart in adolescent Africans: An electrocardiographic and echocardiographic study. *J Am Coll Cardiol* 2012;59:1029-1036.
12. Maron BJ, Doerer JJ, Haas TS, et al. Sudden deaths in young competitive athletes: Analysis of 1866 deaths in the United States, 1980-2006. *Circulation* 2009;119:1085-1092.
13. Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to pre-participation screening for cardiovascular abnormalities in competitive athletes: 2007 update. *Circulation* 2007;115:1643-1655.
14. Corrado D, Pelliccia A, Bjornstad HH, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: Proposal for a common European protocol. *Eur Heart J* 2005;26:516-524.
15. Corrado D, Basso C, Pavei A, et al. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *JAMA* 2006;296:1593-1601.
16. Drezner JA, Ackerman MJ, Anderson J, et al. Electrocardiographic interpretation in athletes: The 'Seattle criteria.' *Br J Sports Med* 2013;47:122-124.