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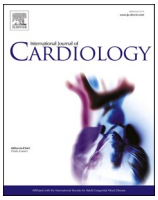

Interpretation and management of T wave inversion in athletes: An expert opinion statement of the Italian Society of Sports Cardiology (SICSPORT)

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ABSTRACT

T wave inversion (TWI) on the electrocardiogram (ECG) is a relatively common finding in athletes. It poses a diagnostic challenge, as it can indicate either a benign physiological pattern or an early sign of serious cardiac pathology. This expert opinion statement provides a comprehensive review of the current understanding of TWI in athletes, emphasizing the importance of its localization, associated clinical features, and demographic factors in guiding its interpretation and management. We explore the potential causes of TWI, including physiological adaptations such as the juvenile pattern and training-induced repolarization variants, as well as pathological conditions like cardiomyopathies, ion channel diseases, and other cardiac abnormalities. Additionally, we discuss the implications of TWI in different ECG leads—anterior, inferior, and lateral—and the diagnostic work-up needed to exclude underlying disease. The importance of follow-up in athletes with TWI is highlighted, particularly for young athletes, to monitor the potential development of cardiomyopathy. Finally, we address

Abbreviations: ACM, arrhythmogenic cardiomyopathy; CAD, coronary artery disease; CMR, cardiac magnetic resonance; CVD, cardiovascular disease; DCM, dilated cardiomyopathy; ECG, electrocardiogram; HCM, hypertrophic cardiomyopathy; LBBB, left bundle branch block; LQTS, long QT syndrome; LV, left ventricle; LVNC, left ventricular non-compaction; MVP, mitral valve prolapse; PM, papillary muscle; RBBB, right bundle branch block; RV, right ventricle; SCD, sudden cardiac death; TWI, T wave inversion; WPW, Wolff-Parkinson-White.

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considerations for sports eligibility in athletes with TWI, stressing the need for a balanced approach that ensures athlete safety without imposing unnecessary restrictions and investigations.

1. Introduction

T wave inversion (TWI) on a 12-lead electrocardiogram (ECG) in athletes, defined as a negative T wave of ≥1 mm in depth across two or more contiguous leads (excluding V1, aVR, and III), presents a significant diagnostic challenge for sports physicians and consulting cardiologists [1,2]. While TWI can indicate serious cardiovascular diseases (CVD) [3,4], it may also be observed in apparently healthy athletes [5]. The clinical significance of TWI varies according to its lead distribution on the ECG, namely anterior (leads V1-V4), lateral (V5-V6, I and aVL) and inferior (II and aVF). Therefore, careful evaluation is required to distinguish between benign and potentially life-threatening conditions [1,6].

This expert opinion statement aims to provide an updated overview of the literature on TWI in athletes, focusing specifically on its interpretation and management, including recommendations for eligibility or disqualification from competitions.

2. The normal T wave

Frank N. Wilson has compared the ventricular activation process to a "wave's crest" preceded by positive electrical charges and followed by negative electrical charges [7]. This concept is fundamental to electrocardiography, where an activation front propagating through the heart can be represented by a single dipole that projects positive potentials ahead of it and negative potentials behind it [7]. The QRS complex represents cardiac depolarization, while the T wave represents the repolarization of the ventricles at end-systole, following the QRS complex. The normal T wave is positive in unipolar chest leads, except V1, while in the bipolar limb leads the T vector is directed leftward and inferiorly, and the T waves are always upright in leads I and II and inverted in lead aVR [7]. They may be upright or inverted in leads III and aVL, depending on whether the T vector is more vertical or more horizontal. In lead aVF, the T wave is usually upright but occasionally flat or slightly inverted.

Despite the traditional attribution of normal T wave genesis to a transmural gradient (longer action potential duration in endocardial cells compared to epicardial cells), modern theory suggests that it results from the heterogeneity of ventricular repolarization across different heart regions, with two main gradients contributing to its formation: the transmural gradient (epicardium vs. endocardium) and the apico-basal gradient (apex vs. base) [8–10]. The transmural gradient predominantly influences the morphology of the T wave in unipolar precordial leads, as these leads are positioned closer to the chest wall and are sensitive to the electrical differences between the epicardial and endocardial surfaces. On the other hand, the apico-basal gradient has a greater impact on bipolar limb leads, as these leads reflect the global electrical activity of the heart and are influenced by the differences in repolarization timing between the apex and base of the ventricles. Both gradients are necessary to accurately replicate the morphology of the T wave in clinical ECGs, as shown by computational models and body surface potential mapping studies [8,9].

3. Possible causes of TWI in the athlete

There are many causes of TWI in athletes (Table 1).

3.1. Non-pathological causes of TWI

3.1.1. The juvenile pattern

Interpreting TWI in the anterior precordial leads can be challenging in children and adolescents. While anterior TWI is typically pathological in most adult athletes [10,11], it may be considered a normal age-related pattern when confined to the anterior precordial leads in these younger age groups. The term "juvenile" ECG pattern denotes TWI or a biphasic T wave beyond lead V1 in children and adolescents who have not reached physical maturity [12]. After puberty, the T wave is usually inverted in lead V1 and upright in leads V2-V6. Indeed, pubertal development has been shown to significantly influence repolarization in young athletes, while training plays a limited role [13–15].

Therefore, in the absence of symptoms and/or a family history of sudden cardiac death (SCD) or cardiomyopathies, anterior TWI up to lead V3 in pre-pubertal children should not prompt further investigations, and a yearly follow-up is suggested until the positivization of anterior TWI [12] (Fig. 1). A longitudinal study on 2227 children (mean age 12.3 ± 2.0 years) screened for pre-participation in sports competitions demonstrated that, during a 4-year follow-up, the vast majority of children who originally had anterior TWI showed T wave positivization (94 %), with only 6 % of children still exhibiting anterior TWI after the period of observation, in the absence of structural heart disease. On the contrary, in the same study, the authors found that infero-lateral TWI is rare, persistent and may be accompanied by structural heart disease [16]. Therefore, infero-lateral TWI should not be interpreted as physiologically related to age, development or training.

After complete pubertal development, TWI becomes significantly less common (0.1 %) and raises the issue of differential diagnosis between a developing heart muscle disease and a benign juvenile pattern of repolarization [15–19]. In these cases, further investigations are recommended to exclude the presence of early cardiomyopathy, particularly arrhythmogenic cardiomyopathy (ACM), which typically starts to become overt after puberty [5,19]. It is difficult to define a clear cut-off age, and the decision to proceed with further investigations should also take into account clinical, demographic, and biological data. Reviewing previous ECG traces, if available, can be useful in cases where the clinical significance of anterior TWI during the peri-pubertal age is uncertain, as a history of normal repolarization patterns does not support the benign persistence of juvenile TWI.

3.1.2. The "Black athlete" repolarization variant

Early criteria for the ECG interpretation in the athlete were associated with high rates of abnormal and false-positive results [20]. One of the most notable caveats of these criteria was that they were primarily derived from Caucasian cohorts without considering large representations of Black athletes. Subsequent work by Papadakis et al.[17] studied

Table 1
Causes of TWI in athletes.

Non-pathological	Juvenile pattern
	Early repolarization variant
	Upright position or chest conformation
Equivocal	Borderline anatomical variants (apical hypertrophy, papillary muscle abnormalities)
	Dynamic or heart-rate dependent
	Training-induced
Pathological (secondary to)	Depolarization abnormalities (bundle branch block, ventricular pre-excitation, pacing…)
	Acute myocardial inflammation (acute ischemia, acute myocarditis, trauma)
	Chronic structural heart disease (cardiomyopathies, valvular heart diseases, congenital heart diseases…)
	Ion channel disease (Brugada syndrome and some long-QT syndrome variants)

TWI in Afro-Caribbean athletes, revealing a significantly higher prevalence of TWI compared to sedentary Black individuals and white athletes. Additionally, compared to Black individuals with hypertrophic cardiomyopathy (HCM), TWIs in Black athletes were more often found in the V1-V4 leads, whereas TWIs in HCM typically involved the inferior and/or lateral leads. Furthermore, none of the athletes with TWIs in the V1-V4 leads were diagnosed with HCM, whereas some of the athletes with TWIs in the inferior and/or lateral leads were subsequently diagnosed with HCM. Finally, TWIs in the anterior leads of Black athletes, which were generally asymmetric or biphasic, were typically preceded by a repolarization variant consisting of J-point elevation and convex ST-segment elevation ("domed-type" early repolarization), whereas HCM patients typically showed ST-segment depression [17].

These findings suggest that "domed-type" early repolarization associated with TWI restricted to V1-V4 could represent a variant of the athlete's heart and physiological adaptations in black athletes, while TWI in the inferior and/or lateral leads or ST-segment depression warrants further evaluation to rule out pathology. For this reason, beginning with the 2013 Seattle Criteria and continuing with the more recent 2017 International Criteria for ECG Interpretation, the presence of an early repolarization pattern typical of black athletes has been progressively recognized and attributed to a benign variant. This recognition and other adjustments have led to a significant reduction in abnormal and false-positive ECGs [21]. Furthermore, the accurate definition of the "domed-type" early repolarization pattern as a J-point elevation >0.1 mV, associated with a ratio of ST-segment elevation at the J-point to ST-segment elevation at J + 80 ms <1, can also be used to distinguish early repolarization with near certainty from the "coved-type" Brugada type 1 pattern [22].

It should be noted that, although a positive T wave typically characterizes the early repolarization pattern of white athletes, they may occasionally show the variant with TWI [23,24], which may represent a specific issue in distinguishing between normal conditions and ACM [25].

In summary, anterior TWI is usually a normal ECG variant when accompanied by J-point elevation and convex ST-segment elevation followed by TWI in V2-V4 [12]. This variant is particularly common in black athletes but may occasionally be observed in athletes with other ethnical backgrounds (Fig. 2). Conversely, when anterior TWI is associated with other ECG anomalies, and/or when the characteristics of anterior TWI do not suggest a benign pattern of repolarization (e.g., TWI preceded by ST-segment depression or deep/symmetrical TWI), or when TWI extend beyond leads V1-V4, further investigations are recommended [2].

3.1.3. *Upright position or chest conformation*

Various factors, including changes in body position or the specific conformation of the chest, can influence TWI in an ECG. When an individual transitions from lying down to standing (upright position), several mechanisms may contribute to TWI:

- Autonomic Nervous System Activation: Standing up activates the adrenergic component of the autonomic nervous system, alters the autonomic (sympathetic vs. vagal) balance, increases the heart rate, and overall electrical activity of the heart;
- Changes in Venous Return: Altering body position impacts venous return to the heart, potentially affecting the heart's electrical conduction system;
- Diaphragmatic Position: The diaphragm moves downward in a standing position, which may shift the heart's position and the direction of the repolarization vectors.

For this reason, in athletes referred for TWI, it is of paramount importance to verify that the ECG was acquired in the supine position and, when in doubt, repeat it before starting an unnecessary diagnostic work-up (Fig. 3).

In addition, TWI can be linked to conditions affecting chest anatomy:

- Chest Conformation: Structural and anatomical differences, such as those seen in conditions like pectus excavatum, where the chest wall is sunken, can alter the spatial orientation of the heart. This change can affect how electrical impulses are detected by ECG leads, potentially causing TWI;
- Obesity: Excessive adipose tissue can shift the position of the heart and alter the electrical vectors detected by the ECG, sometimes resulting in TWI;

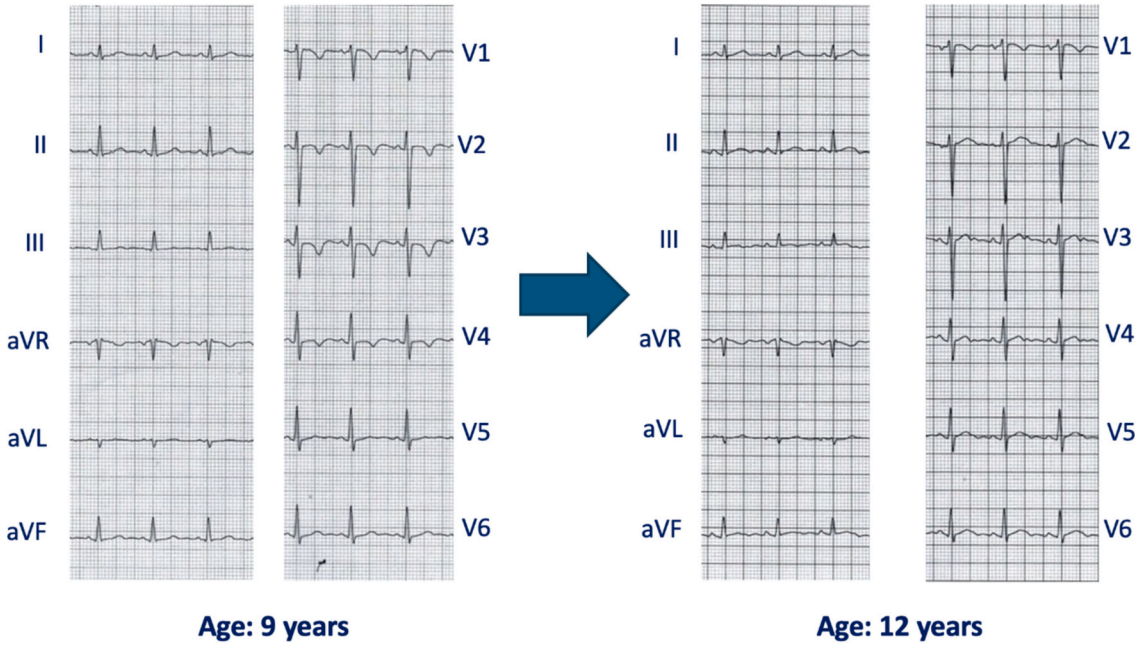

Fig. 1. Dynamic changes of TWI in childhood. The ECG on the left demonstrates a typical juvanile T wave inversion pattern observed in pre-pubertal children (9 years old). T waves are inverted in leads V1-V4, consistent with the juvanil pattern. The ECG on the right depicts a normalized pattern after pubertal development, where T waves in V2-V4 become upright, indicating physiological maturation of the conduction system (12 years old).

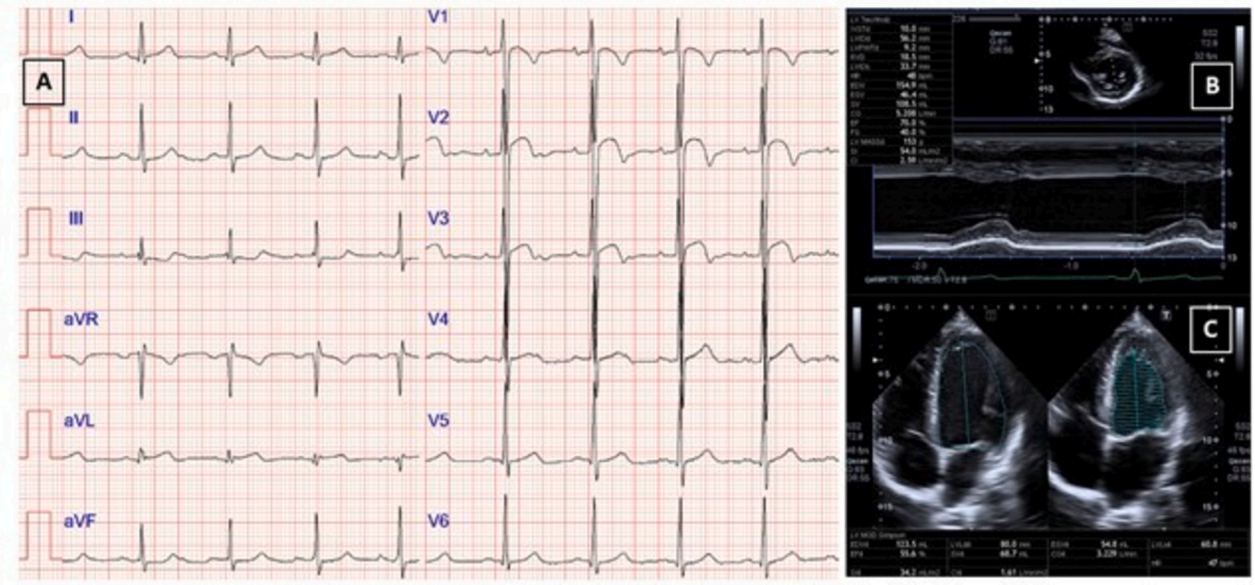

Fig. 2. Professional soccer player (defender), aged 23, of Central African origin. The resting electrocardiogram (ECG, panel A) and echocardiogram (B and C) were done during his preparticipation screening, as mandated by Italian law. The ECG shows the typical repolarization pattern of the black athlete, with a dome-shaped ST-segment elevation followed by negative T waves from V1 to V3. At heart ultrasound, no presence of signs of cardiomyopathies or pathological left ventricular hypertrophy.

- Skeletal Abnormalities: Conditions like scoliosis can change the heart's position or the path of electrical conduction, affecting ECG readings;
- Pulmonary Conditions: Diseases like chronic obstructive pulmonary disease can alter the position and dynamics within the chest, impacting the ECG and inducing TWI.

Historical studies have shed light on the clinical relevance of these variations. In 1935, Leimdorfer first noted changes in the T wave from positive to negative with changes in posture from lying to standing. He initially attributed these ECG changes to latent cardiac diseases, but subsequent studies contradicted this view. In 1938, Sigler discovered that posture-induced ECG changes do not necessarily indicate latent cardiac diseases, emphasizing the importance of knowing the patient's position during ECG recording, especially when comparing different ECGs from the same patient [26]. Later research by White in 1941 [27] affirmed these findings, noting that the position of the diaphragm is significant and that its elevation at full expiration tended to normalize T waves, though not as effectively as lying down. Studies in the 1960s by Hinkle [28] and later Lachhman [29] highlighted that orthostatic T wave changes are common and often benign, occurring in up to 16 % of healthy young subjects without any evident cardiac abnormality. Bellet's study in 1965 [30] further supported these findings, showing that postural changes were more frequent in subjects with angina or positive exercise tests than those with negative exercise tests. This study, along with others, led to a consensus that isolated T wave changes should not be over-interpreted as indicators of coronary artery disease (CAD) or other pathologies. Instead, they are often benign and rarely constitute the sole criterion for a positive exercise test.

3.2. Equivocal causes of TWI

3.2.1. Borderline anatomical variants

In clinical practice, after careful investigation, some athletes with suspicious TWI remain free from a diagnosis of HCM or other cardiomyopathies, suggesting that either TWI precedes the phenotypic development of CVD over a long period or that other non-pathologic abnormalities should be considered [6].

Among the non-pathologic anomalies, a few studies have described the association between TWI and isolated hypertrophy and/or apical displacement of the papillary muscles (PMs) [31]. De Lazzari et al. investigated a cohort of 53 athletes with inferior-lateral TWI, free from cardiomyopathies, after a thorough investigation inclusive of cardiac magnetic resonance (CMR) [32]. Compared with controls, athletes with TWI showed significantly greater diameters, areas, volumes, and mass of both papillary muscles. Namely, PM mass was greater by about 40 % in subjects with TWI compared to controls. Indeed, PMs were displaced apically in most athletes with TWI (47 % versus 17 % of controls; $p < 0.001$). Specifically, both the PMs appeared displaced, although isolated malposition of either the anterolateral or the posterolateral papillary muscle was possible. The increased mass of the PMs was more prominent than left ventricle (LV) mass only in athletes with TWI; i.e., the ratio of PM mass to LV mass was 4 % versus 3 % in controls ($p < 0.001$), suggesting that the increase in PM mass was not the consequence of sport-related LV remodeling. In fact, in a few athletes with TWI who underwent a detraining period, PM mass remained unchanged [33]. Clinically, it was reported that during a short-term (3-year) follow-up period, none of the athletes with TWI and PM hypertrophy/displacement showed the incidence of cardiac disease, namely HCM. Finally, a few of these athletes (9, or 17 %) underwent molecular screening of a large panel of genes, which failed to identify any pathogenic mutations.

It has been speculated that morphological PM abnormalities can be responsible for the development of apical hypertrophy, even in the absence of a sarcomeric gene mutation. The study by Lee et al. [31] demonstrated that an apically displaced anterolateral PM might be associated with hypertrophy of the LV apico-lateral segments (but not the apico-septal), morphologically similar to apical HCM, also in association with giant TWI. This finding suggests that an abnormal implant of the anterolateral papillary muscle may cause repolarization abnormalities by itself.

Regardless of PM displacement or hypertrophy, in a portion of patients with TWI, the LV apex may lack the physiologic myocardium tapering, defining a pattern of only relative apical hypertrophy (Fig. 4). Morphologically, these patients may show absolute LV apical thickness in a range of 11–12 mm (i.e., greater than 5.6 mm/m^2) which is nowadays considered a legitimate criterion for diagnosing apical HCM [34].

Although this remains a debated point, these observations suggest

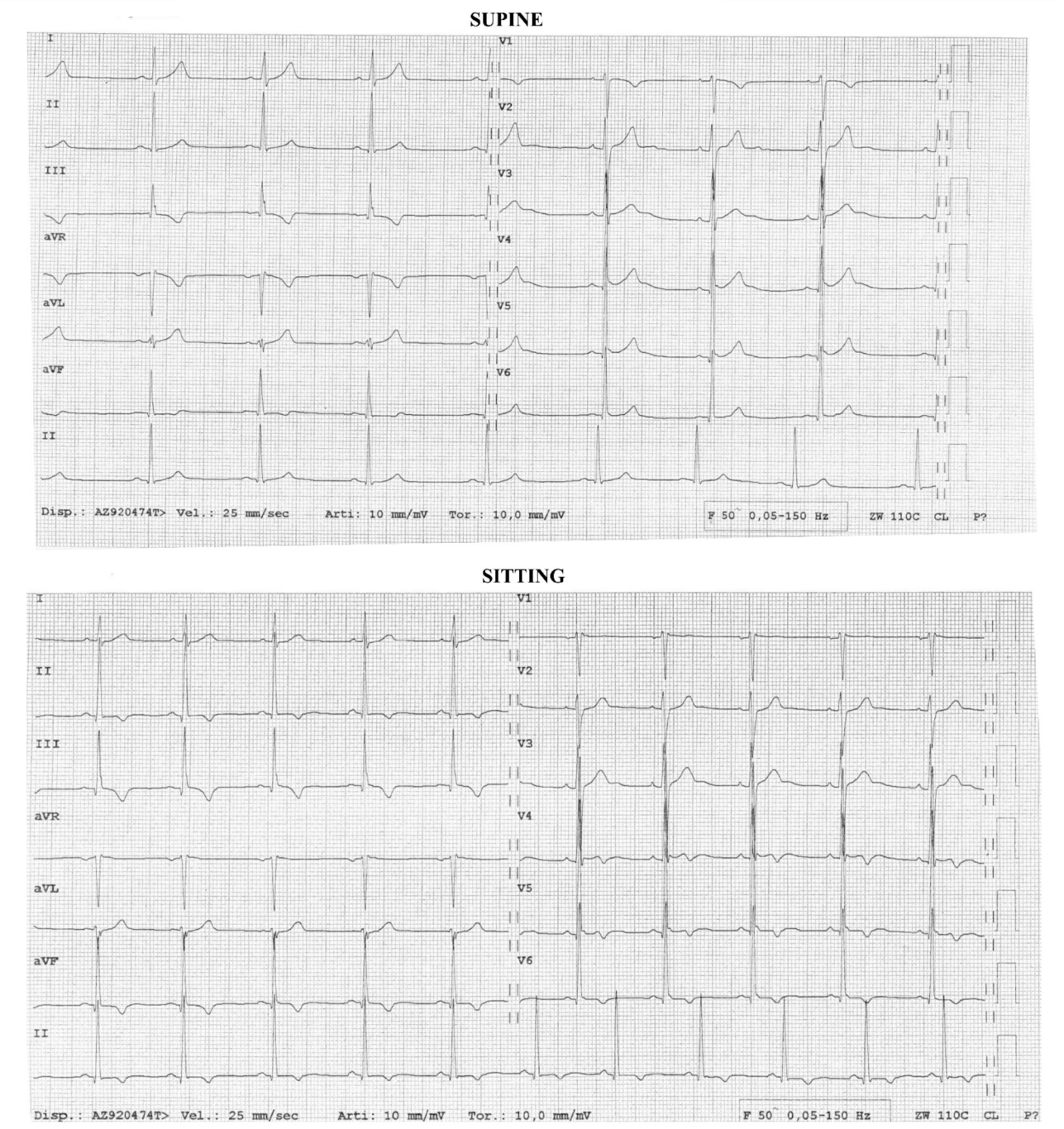

Fig. 3. Two ECGs were recorded from a 25-year-old football player, one in the supine position (top) and one in the sitting position (bottom). In the supine ECG, T waves are negative in leads V1-DIII and isodiphasic in aVF. In the sitting ECG, T waves become negative in all inferior leads and leads V4-V6.

that TWI in association with even mild apical LV thickening, or with PM hypertrophy and/or displacement, should be viewed with caution, potentially representing a phenotypic expression of non-physiologic hypertrophy and a distinct pattern of apical HCM [34]. Indeed, it is worth mentioning that TWI in the number of patients may precede the development of hypertrophy over a long period [35]. Consequently, the identification of TWI in individuals (including athletes) with mild hypertrophied LV apex and/or displaced PMs warrants serial evaluations to detect the development of HCM over time. Finally, it should be noted that certain patients with TWI and the absence of any morphologic LV abnormality may not develop a clear LV hypertrophy even after a long-term period and present a favorable clinical outcome, suggesting that, even if present, an abnormal genetic background might not express morphologically [36].

3.2.2. *Exercise-induced normalization of TWI (pseudonormalization)*

T wave pseudonormalization is defined as the presence of an inverted T wave $\geq$1 mm in any lead at rest, which becomes positive at peak exercise. When associated with symptoms such as chest pain and U-wave inversion in anterior precordial leads, it is indicative of CAD [37]. In

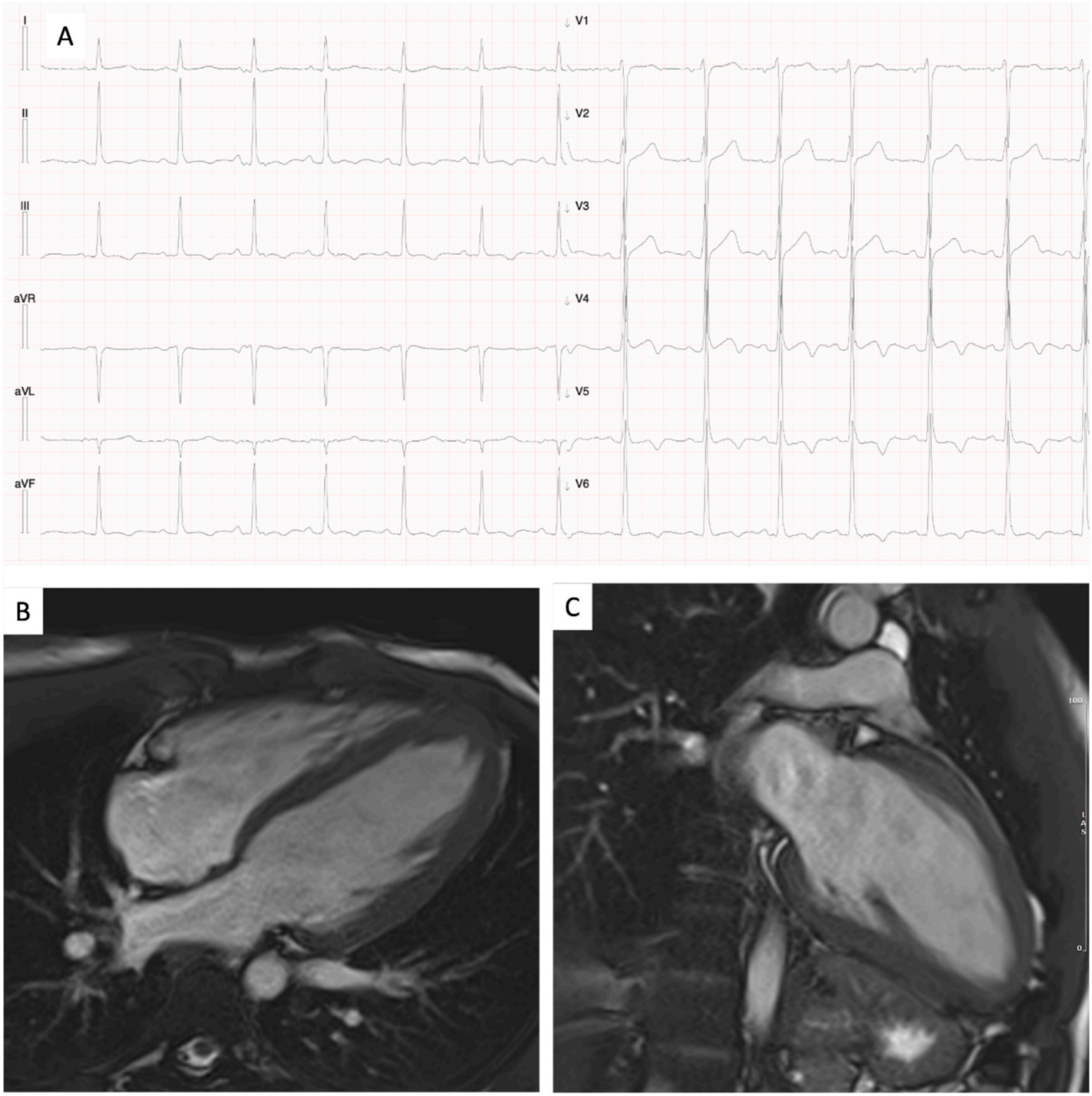

Fig. 4. Case of a 27-year-old basketball player. The baseline ECG (Panel A) displays negative T waves in the inferolateral leads. Cardiac magnetic resonance imaging (Panels B and C) reveals an absence of the normal apico-basal gradient in left ventricular wall thickness, consistent with relative apical hypertrophy. Panel B shows a 4-chamber cine view in diastole, while Panel C shows a 2-chamber cine view in diastole.

patients with post-myocardial infarction, pseudonormalization of T waves in leads related to the previous myocardial infarction is an indicator of residual myocardial viability and predictive of survival at a 6-month follow-up [38]. In healthy subjects with a low risk of CAD, this is a non-diagnostic finding [39].

Initially, in top-level athletes without clinical signs of structural heart disease, the presence of TWI at baseline with pseudonormalization of T waves by isoproterenol infusion or maximal physical effort was considered functional and benign [40]. However, it has been shown that T wave pseudonormalization can also occur in patients with CVD. A study found that partial or complete exercise-induced pseudonormalization of right precordial T wave inversion occurred in some patients affected by ACM and was unrelated to the clinical phenotype and for this reason, changes in negative T waves in right precordial leads (V1-V3) were inaccurate in differentiating between ACM patients and benign repolarization abnormalities [41]. In patients with HCM, stress ECG is considered less accurate for ischemia detection due to the significant repolarization abnormalities at rest ECG and peak exercise, including T wave pseudonormalization [42]. In summary, normalization of TWI present at baseline should not be considered a finding suggesting the benign nature of the repolarization abnormality, and thus warrant further investigations.

3.2.3. Training-induced TWI

Evidence on the phenomenon of training-induced TWI remains limited in scientific literature. Zaidi et al. [43] hypothesized that TWI and biventricular dilation are physiological cardiac adaptations resulting from regular exercise. They compared athletes with normal ECGs, athletes with TWI, and individuals with ACM using ECG, echocardiography, and CMR. Their findings showed no differences in any electrical, structural, or functional cardiac parameters between athletes with and without TWI, in contrast to the distinct differences observed in ACM patients. This led them to conclude that TWI and balanced biventricular dilation in athletes are likely benign manifestations of training. Brosnan et al. [44] found that TWI in leads V1-V3 was more prevalent in endurance athletes than in the general population, attributing this to the lateral displacement of the right ventricle (RV) and its apex toward the axilla, a physiological adaptation to the thoracic spatial constraints resulting from cardiac enlargement. By analyzing the angle of the interventricular septum relative to the thoracic midline using CMR, they discovered a correlation between this septal angle and the presence of TWI in V1-V3. Since the RV is activated after LV and its repolarization typically results in negative T waves, they concluded that this lateral shift could be the cause of the observed TWI in these athletes, suggesting that these are benign adaptations to increased cardiac demands. Finally, there are reports in the literature that both healthy athletes [45] and HCM athletes [46], with TWI, show a regression of this phenomenon with a period of detraining, suggesting how this aspect is not always a marker of a being nature of the TWI.

3.3. Pathological causes of TWI

3.3.1. Depolarization abnormalities

Secondary T wave changes can be due to concomitant depolarization abnormalities and cardiac memory phenomena. From an electrophysiological perspective, a change in the ventricular activation sequence can provoke variations in the duration of the excited state, which alters the ventricular gradient and subsequently leads to T wave abnormalities [47]. In these cases, the diagnostic and prognostic significance of these anomalies is not linked to the T wave itself but rather to the importance of the underlying intraventricular conduction disorder (e.g., left bundle branch block - LBBB) [48].

The phenomenon of electrical T wave memory has been well-known for some time and refers to the persistence of TWI after ablation of an anomalous pathway [49], or in the phenomenon of T wave memory in intermittent ventricular stimulation from pacemakers. Even more important is the role of T wave alterations secondary to intermittent disturbances of intraventricular impulse conduction [50]. The underlying mechanisms involve ion channel modulation and calcium handling, leading to short-term memory (minutes to hours) and long-term memory (lasting weeks to months). Short-term memory is mediated by signaling pathways affecting ion channel trafficking, while long-term memory involves changes in gene transcription, particularly influencing calcium and potassium channels [51].

3.3.2. Acute myocardial inflammation

In athletes, TWI may be a sign of acute or chronic myocardial inflammation due to ischemia, myocarditis or trauma. All these situations share a characteristic TWI pattern with symmetrical branches in the leads exploring the inflamed area. These dynamic abnormalities generally normalize within a few weeks and are related to myocardial edema [52]. Although usually accompanied by symptoms such as chest pain or dyspnea, myocardial inflammation (particularly of non-ischemic origin) may be mildly symptomatic or asymptomatic: hence, when dynamic TWI are detected by preparticipation screening, this possibility should be taken into account.

In the context of CAD, the so-called Wellens' syndrome has been described as characterized by deeply inverted T waves in leads V1-V4 that suggest anterior wall ischemia due to critical stenosis of the proximal left anterior descending artery [53]. Additionally, deep TWI usually occurs during the evolving phase of a Q-wave myocardial infarction and sometimes with a non-Q-wave MI as well. These TWIs result from a delay in regional repolarization caused by the ischemic edema [24].

Myocarditis is another cause of acute myocardial inflammation and is a common concern for sports cardiologists [54,55]. Mild subclinical myocarditis is often clinically asymptomatic, making it a difficult but important diagnosis, as it poses the athlete at risk of SCD. Diagnosing subclinical myocarditis depends primarily on an ECG at the initial stage of the disease: new-onset TWI without ST elevation and sometimes premature ventricular contractions should raise suspicion of myocarditis [56].

Acute myocardial inflammation may also occur due to trauma. Once considered exceedingly rare, the number of reported cases is rising [57]. Maringhini et al. described a case involving a healthy 17-year-old male athlete who suffered an open-handed blow to the anterior part of the chest. He did not show any arrhythmias and did not require resuscitation, but he did exhibit TWI in the inferolateral leads [58].

3.3.3. Chronic structural heart diseases

An ECG demonstrating TWI may represent the first and only sign of a myocardial disorder in which the heart muscle is structurally and functionally abnormal. This condition can be caused by an inherited muscle disease (cardiomyopathy) or be secondary to an overload of volume or pressure, such as hypertension, valvular disease, CAD, or congenital heart disease.

TWI can be found in some of the most relevant cardiomyopathies:

- Dilated cardiomyopathy (DCM). These patients show a wide spectrum of electrical and functional abnormalities that can change over time, but compared to other cardiomyopathies, repolarization abnormalities are not very common, with a prevalence of 15 %–45 % [59]. The leads most frequently involved are the inferior, antero-lateral, and infero-lateral;
- ACM. A 12-lead ECG is an integral part of the evaluation of ACM patients, and repolarization abnormalities are commonly observed, with a reported prevalence of up to 85 % [60]. According to current diagnostic criteria, TWI in the right precordial leads V1, V2, and V3 or beyond is a major criterion for RV ACM in individuals with complete pubertal development (usually age $\geq$ 14 years). Excluded from the diagnostic criteria are TWI in V1 to V3 combined with J-point/ST-segment elevation due to early repolarization and those associated with complete right bundle-branch block (RBBB) secondary to the conduction defect. TWI in the right precordial leads V1 and V2 are classified as a minor criterion for RV-ACM in males $\geq$14 years old, in the absence of RBBB and J-point/ST-segment elevation. TWI extending beyond V3, either in the presence of complete RBBB or individuals <14 years old, is also a minor criterion. The presence of TWI in the left precordial leads (V4–V6), with or without the involvement of inferior leads, in the absence of LBBB is a minor criterion for the LV phenotype [61];
- HCM. The ECG shows TWI in up to 90 % of HCM cases, usually deep TWI ($\geq$2 mm) in infero-lateral leads associated with ST-segment depression. Sometimes, the so-called "giant T waves" (>10 mm), a typical finding in the apical form of HCM, are observed [62]. It should be emphasized that ECG abnormalities in HCM patients may precede echocardiographic findings of the disease (see Fig. 5);

TWI can also be observed in:

- Ventricular overload/enlargement. RV hypertrophy is caused by a pressure load (e.g., pulmonary hypertension or pulmonary valve stenosis), and the ECG may show anterior TWI while RV volume overload can be caused by congenital disease with left-to-right shunts or tricuspid valve regurgitation. On the other hand, LV

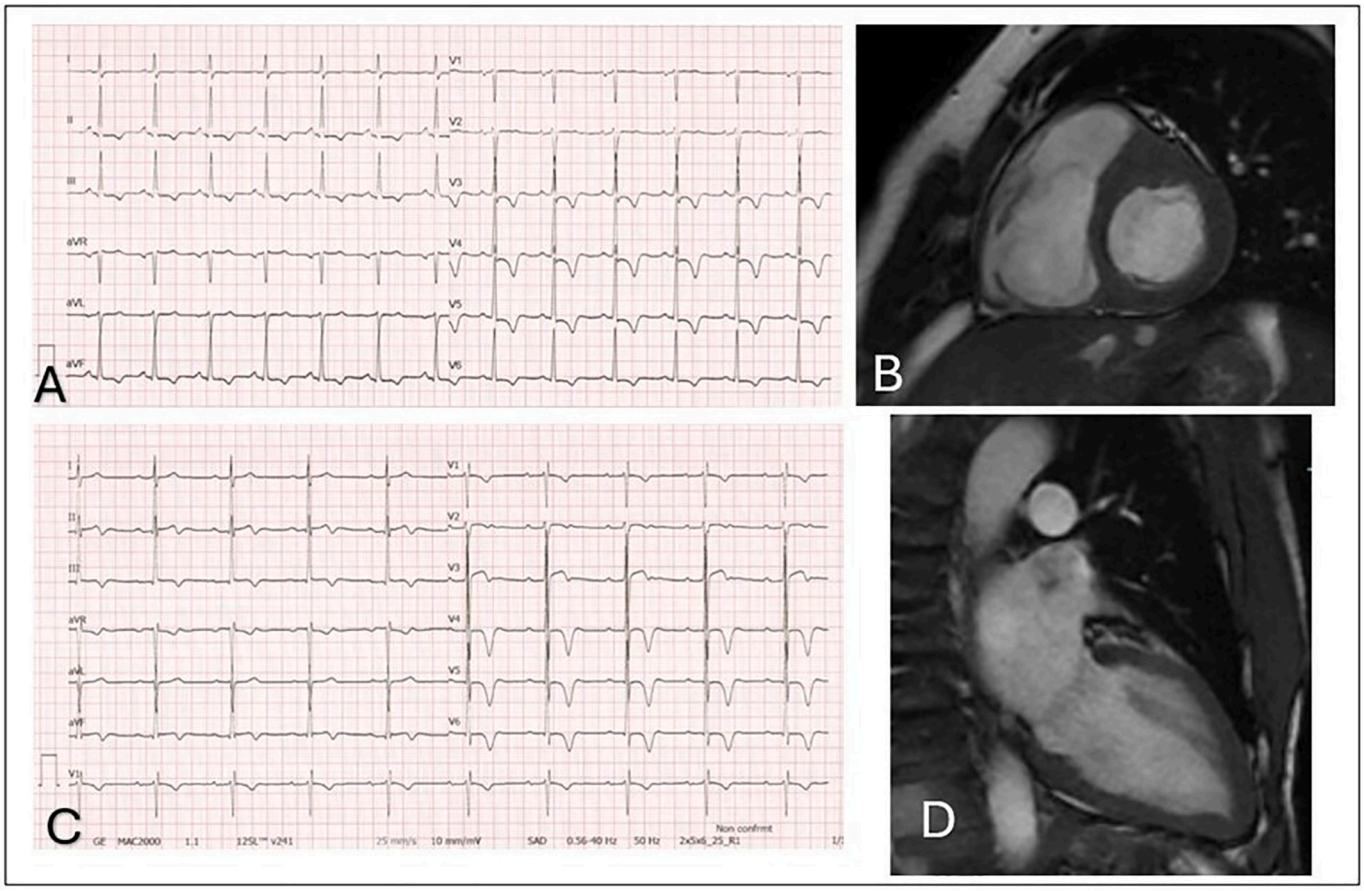

Fig. 5. ECG and cardiac magnetic resonance (CMR) findings in a 26-year-old basketball player with non-obstructive HCM and underlying MYH7 pathogenic variant. Twelve-lead ECG showing ST-segment alterations and TWI in inferior and precordial leads (A). CMR images in the short axis show asymmetric hypertrophy of the LV (B). CMR and ECG findings in a case of apical HCM in a 57-year-old marathon runner. ECG shows signs of left ventricular hypertrophy and TWI in lateral leads (C). CMR reveals loss of the usual apical wall thickness tapering due to apical wall thickness exceeding basal wall thickness (D).

hypertrophy may underline pressure (hypertension, aortic stenosis) or volume (mitral or aortic regurgitation) overload in the LV [5]. The repolarization abnormalities associated with LV hypertrophy typically include ST-segment depression with TWI in the lateral leads that also show tall R waves. Furthermore, in the case of ventricular dilation, the finding of TWI is helpful in the differential diagnosis between a pathological condition and an athlete's heart [19,63].

- Arrhythmic Mitral Valve Prolapse (MVP). The so-called "Barlow disease" is a syndrome characterized by predominantly bi-leaflet myxomatous degeneration of the mitral valve with or without

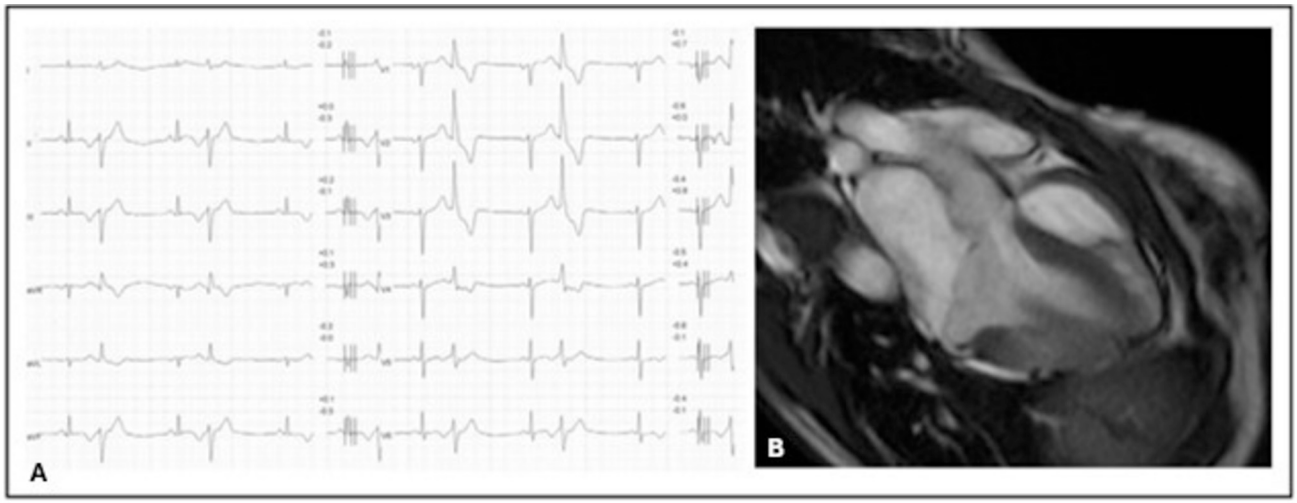

Fig. 6. A 19-year-old female with an arrhythmic mitral valve prolapse. Twelve-lead ECG shows TWI in the inferolateral leads and monomorphic premature ventricular beats with RBBB/superior axis morphology (A). In the Cine CMR, 3-chamber, systolic frame, long-axis view, you can see mitral valve prolapse with myocardial annular dysfunction (B).

significant regurgitation, morpho-functional abnormalities of the mitral annulus, complex ventricular arrhythmias with RBBB morphology, and TWI in the inferolateral leads (Fig. 6) [64]. The extent of TWI is associated with a higher degree of lateral myocardial fibrosis, suggesting diffuse fibrosis as the underlying substrate for ECG repolarization abnormalities and an increased arrhythmic risk in patients with MVP [65]. Therefore, this finding suggests that athletes with clear evidence of MVP and T wave abnormalities in the inferior leads should undergo a clinical evaluation, including at least echocardiography, 24-h 12-lead Holter ECG monitoring, and exercise testing [66]. CMR is advised in the presence of MVP with TWI and clinically relevant ventricular arrhythmias [63,67,68].

3.3.4. Ion channel diseases

Long QT syndrome (LQTS) is a congenital (cLQTS; familial) or acquired (aLQTS) cardiac disorder of myocardial repolarization. The diagnostic criteria for cLQTS have been codified by Schwartz, combining clinical features with family history and abnormalities of repolarization on the ECG (including T wave alternants and notched T wave in 3 leads), to provide a low (≤1 point), intermediate (1.5 to 3 points), or high (≥3.5 points) probability of disease [69]. Multiple subtypes of cLQTS have been described to date. The three most common are: LQT1, caused by mutations in KCNQ1, which accounts for 45 % of genotyped cases; LQT2, caused by mutations in KCNH2 (40 % of genotyped cases); and LQT3, caused by mutations in SCN5A (10 % of genotyped cases). These three common subtypes can be differentiated by characteristic T wave morphology patterns, which include early onset broad T waves in LQT1, bifid (notched) T waves in LQT2, and late-onset T waves in LQT3 [70]. Although TWI is not a typical finding in these three most common inherited LQTs variants, other rare genotypes may be characterized by TWI: the Triadin-related form shows extensive TWI in precordial leads, and giant negative T waves can also be found in other genetic conditions associated with LQTS, such as Timothy syndrome and Ankyrin-B syndrome. Giant negative T waves can also be observed in acquired LQTS, such as hypokalemia, hypothyroidism, cerebral stroke, pheochromocytoma, and Tako-Tsubo syndrome [70].

Brugada syndrome is a hereditary disease linked to a mutation of sodium channels (15–30 % of cases involve SCN5A loss of function) and is characterized by the association of malignant cardiac arrhythmias with a typical ECG pattern [71–73]. Brugada syndrome is categorized into three types based on ECG findings, but the only diagnostic pattern is the type 1, that features ST elevation with at least 2 mm J-point elevation and a gradually descending ST segment followed by a negative T wave (coved pattern). The presence of a negative T wave is crucial for diagnosing Type 1, helping to differentiate the Brugada pattern from early repolarization patterns, which typically feature J-point elevation followed by ascending ST and positive T waves [74].

4. Diagnostic work-up in athletes with TWI

For a proper interpretation of TWI patterns in athletes, it is important to ensure that the ECG is conducted with the patient lying down, not tachycardic (as some individuals exhibit heart rate-dependent repolarization abnormalities) and noting any variations with respiratory acts. Moreover, it is crucial to consider primarily the localization of the TWI, which may help to identify specific cardiac pathologies in conjunction with the family and personal history and the clinical correlates of the TWI (Table 2) [75]. It is important to remember that TWI may represent the initial expression of cardiomyopathies that may not become evident for many years and could ultimately be associated with adverse outcomes; therefore, follow-up is necessary in doubtful cases. Although most evidence discussed in this manuscript was collected in young athletes with an age range 14–35 year-old, the recommendations potentially apply to the entire age spectrum of athletes, although using CMR in very young athletes may be challenging and should be evaluated on a case-by-case basis.

Table 2
Differential diagnosis of different localization of TWI.

TWI localization	Findings suggesting athlete's heart	Findings suggesting potential pathologies
aVR, III and V1 (in isolation)	They should not raise the suspicious of disease	
Anterior leads (V1-V4)	Common in women (especially if in V1-V2 only), Black athletes, or those with incomplete pubertal development. TWI preceded by J-point and/or ST-segment elevation is a repolarization variant often seen in Black athletes and occasionally in other ethnicities	Pathological conditions may be indicated by minimal or absent J-point elevation (<1 mm), a coexistent depressed ST-segment preceding TWI, deep TWI, or other ECG abnormalities such as low limb lead voltages, prolonged S-wave upstroke, premature ventricular beats and epsilon waves. Furthermore, in peripubertal athletes, normal T waves in previous ECG is concerning
Inferior leads (II, III, avF)	If isolated, it may not necessarily indicate pathology and could be related to normal physiological variations, particularly if the QRS axis is horizontal	Suspicion of disease should be heightened if symptoms, additional ECG anomalies, or abnormal morphologic cardiac findings accompany TWI in these leads.
Lateral leads (V4-V5-V6)	After ruling out pathological substrates, the presence of anatomical variants observable on imaging, such as hypertrophic papillary muscles or false tendons, might explain them	Should always raise suspicion of diseases, especially when linked with symptomatic presentation or other diagnostic indicators of cardiac pathology. Consider repeating the evaluation during follow-up.

4.1. Anterior leads (V1-V4)

Anterior TWI is typically a normal variant in pre-pubertal young athletes (juvenile pattern), female athletes (especially in V1 and V2) [76], especially endurance ones [77,78], and individuals of Afro-Caribbean descent, often preceded by J-point elevation and coved ST-segment [79]. Therefore, this sign may be interpreted as non-pathological in these specific populations. However, when anterior TWI extends beyond V4 or is present in combination with other potential ECG signs of pathology [80], further cardiovascular investigations are needed to rule out an underlying cardiomyopathy. First-line investigations should include echocardiography, exercise testing and 24-h ambulatory ECG monitoring targeting at evaluating the presence of ventricular arrhythmias. In case of abnormal findings at such investigations or when the clinical suspicion is high (e.g. positive family history of CVD or suspicious symptoms), further investigations including CMR are indicated.

4.2. Inferior leads (II, III, avF)

The clinical significance of isolated inferior TWI remains uncertain. Inferior TWI has been reported in a certain proportion of normal individuals, including healthy athletes, but it may also be present in subjects affected by cardiomyopathy. In patients with ACM, the prevalence of inferior TWI can reach 31 %, while in healthy athletes, the prevalence is lower, around 3 % [81].

The 2017 International Criteria for Electrocardiographic Interpretation in Athletes [12] highlighted that the significance of TWI exclusively restricted to inferior leads is controversial and cannot be attributed to the physiological remodeling of the athlete's heart. In both cases, a possible association with cardiac muscle diseases, including HCM, DCM, and myocarditis is emphasized, although with a low positive predictive value if not associated with other abnormalities or clinical elements. For this reason, the document suggests always second-level investigations with echocardiography, but CMR only in cases

with additional suspicious elements. One limitation of these criteria is the increase in false positives in studies conducted on non-Caucasian populations [82]. A 2022 review analyzed the impact of different ethnicities on the correct interpretation of the athlete's ECG, highlighting that T waves in the inferior leads are more common in Black athletes (ranging from 1.3 % to 6.1 % depending on the studies) compared to Caucasian ones (where they are found with percentages ranging from 0.5 % to 1.7 %) [83].

On the other hand, there are also isolated cases of an association between TWI in the inferior leads and cardiac masses [84]. Recently, TWI in inferior leads has also been associated with non-ischaemic LV scar in athletes with RBBB morphology ventricular arrhythmias [85], so the morphology of premature ventricular beats must always be evaluated [86,87]. Another aspect concerns the association with MVP. In the study by Basso et al. in 2015 [88], negative T waves in the inferior leads alone were found in 83 % of cases who died from arrhythmic MVP for whom ECGs were available. In the European Hearth Rhythm Association expert consensus statement on arrhythmic MVP [89], negative T waves in the inferior leads are included among the "phenotypic risk features," along with mitral annular disjunction, late gadolinium enhancement, and multiple polymorphic premature ventricular contractions [64]. These recent studies on arrhythmic MVP and non-ischemic LV scars highlight the importance of not dismissing inferior negative T waves as normal finding [90].

In summary, the presence of inferior TWI is relatively common in young athletes, and its clinical significance is uncertain when present in isolation but can also be related to CVD. For this reason, it appears reasonable to perform always second-line investigations and to proceed with further testing in cases of abnormal findings or high clinical suspicion [5,19].

4.3. Lateral leads (V4-V5-V6)

The presence of TWI in leads I and aVL, V5, and/or V6 defines lateral TWI, while TWI appearing in leads II, aVF, V5-V6, I, and aVL is classified as infero-lateral TWI [80]. Lateral TWI occurs in 0.3 % to 1.5 % of athletes, with its prevalence reportedly ten times higher in Black athletes compared to White ones [91]. This pattern is linked to cardiomyopathies in a significant number of cases [92,93], and thus, it should invariably raise concerns about underlying heart disease and necessitate a comprehensive evaluation to exclude a life-threatening cardiac condition [80].

Notably, in a study involving 12,550 athletes identified 81 with diffusely distributed and deeply inverted T waves, who initially showed no apparent CVD but were followed for up to 27 years, cardiomyopathy developed in five of these athletes (6 %), alongside other CV disorders in six athletes (7 %). In all cases who developed a disease during follow-up, TWI involved the lateral leads (± other leads) [35]. Additionally, research by Schnell et al. on 155 asymptomatic athletes found cardiomyopathies in 41 % of those with lateral TWI, with CMR uncovering 24 cases of disease where echocardiograms were normal or inconclusive [93]. Moreover, an investigation by Sheikh et al. revealed that 21 % of asymptomatic athletes with TWI were diagnosed with cardiac disease, with a higher prevalence in Caucasians than Black athletes (30 % vs. 12 %) [91]. Recently, Maestrini et al. confirmed that in a large cohort of 504 athletes evaluated prospectively, CMR was able to detect cardiac conditions, primarily HCM, in 34 % of those presenting with infero-lateral TWI [94].

These findings underscore the importance of comprehensive evaluation for all athletes exhibiting lateral or infero-lateral TWI. Even if initial echocardiography does not indicate structural heart disease, CMR is anyway recommended for its superior capability in evaluating HCM, especially at the LV apex, and for its unique ability to detect myocardial fibrosis [2]. Moreover, if no unifying diagnosis is evident, it is advisable to conduct annual echocardiographic surveillance, particularly for athletes under 35 years old. Repeating CMR approximately every 2–3 years following the initial diagnosis is also suggested, as lateral TWI may precede the full phenotypic expression of cardiomyopathies, particularly HCM [5,95,96].

5. Sports eligibility and follow-up

The presence of TWI is not inherently indicative of CVD. Therefore, when TWI is identified, it is crucial to undertake further evaluations to either confirm or exclude any underlying pathological conditions, as outlined in the proposed algorithm (Fig. 7).

In athletes with TWI, sports eligibility is evaluated according to the underlying CVD [95] and the sports discipline, as suggested by current recommendations [96,97]. If a heart disease, whether structural or functional (such as channelopathies), is diagnosed [85], sports eligibility must be primarily based on the severity of the disease, taking into account the possible unfavorable evolution of some conditions precipitated by physical activity. This is the case, for example, with ACM [98].

It is important to note that, in many instances, a markedly abnormal ECG may not reveal any features of CVD even after a comprehensive work-up. In such cases, the athlete should not be restricted from participating in competitive sports [2]. However, considering the potential for developing cardiomyopathy later in life, regular follow-up is highly recommended (at least annually and particularly during adolescence and young adulthood) [35]. Indeed, while cardiac imaging may be negative during initial clinical evaluations in children/adolescents with suspected TWI, pathological LV hypertrophy can be detected later in life. Genetic testing is not indicated in TWI in the absence of any structural abnormalities or confirmed family history of cardiomyopathy [99]. Athletes should also be educated on the importance of recognizing incident cardiac symptoms as a red flag that requires re-evaluation.

6. Conclusions

In conclusion, TWI in athletes is a complex phenomenon with a broad spectrum of potential causes, either benign or potentially malignant. While some forms of TWI may be indicative of early-stage cardiomyopathies, others may represent normal variants associated with athletic training or anatomical differences. Therefore, a comprehensive diagnostic work-up is essential to accurately differentiate between these possibilities, including regular follow-up, particularly in young athletes, to monitor for the potential development of cardiomyopathies over time. The goal is to balance the need for early detection of life-threatening conditions with the recognition of benign variants, thereby safeguarding athletes' health while allowing them to continue their sports careers without unnecessary restrictions.

CRediT authorship contribution statement

Stefano Palermi: Writing – review & editing, Writing – original draft, Project administration, Conceptualization. **Lucia Tardini:** Writing – review & editing, Writing – original draft, Conceptualization. **Francesca Graziano:** Writing – review & editing, Writing – original draft, Conceptualization. **Massimiliano Bianco:** Writing – review & editing, Writing – original draft. **Alessandro Bina:** Writing – review & editing, Writing – original draft. **Silvia Castelletti:** Writing – review & editing, Writing – original draft. **Elena Cavarretta:** Writing – review & editing, Writing – original draft. **Maurizio Contursi:** Writing – review & editing, Writing – original draft. **Domenico Corrado:** Writing – review & editing, Conceptualization. **Flavio D'Ascenzi:** Writing – review & editing, Writing – original draft. **Giuseppe Inama:** Writing – review & editing, Writing – original draft. **Lucio Mos:** Writing – review & editing, Writing – original draft. **Antonio Pelliccia:** Writing – review & editing, Writing – original draft. **Zefferino Palamà:** Writing – review & editing, Writing – original draft. **Antonio Scarà:** Writing – review & editing, Writing – original draft. **Luigi Sciarra:** Writing – review & editing, Writing – original draft. **Fabrizio Sollazzo:** Writing – review & editing, Writing –

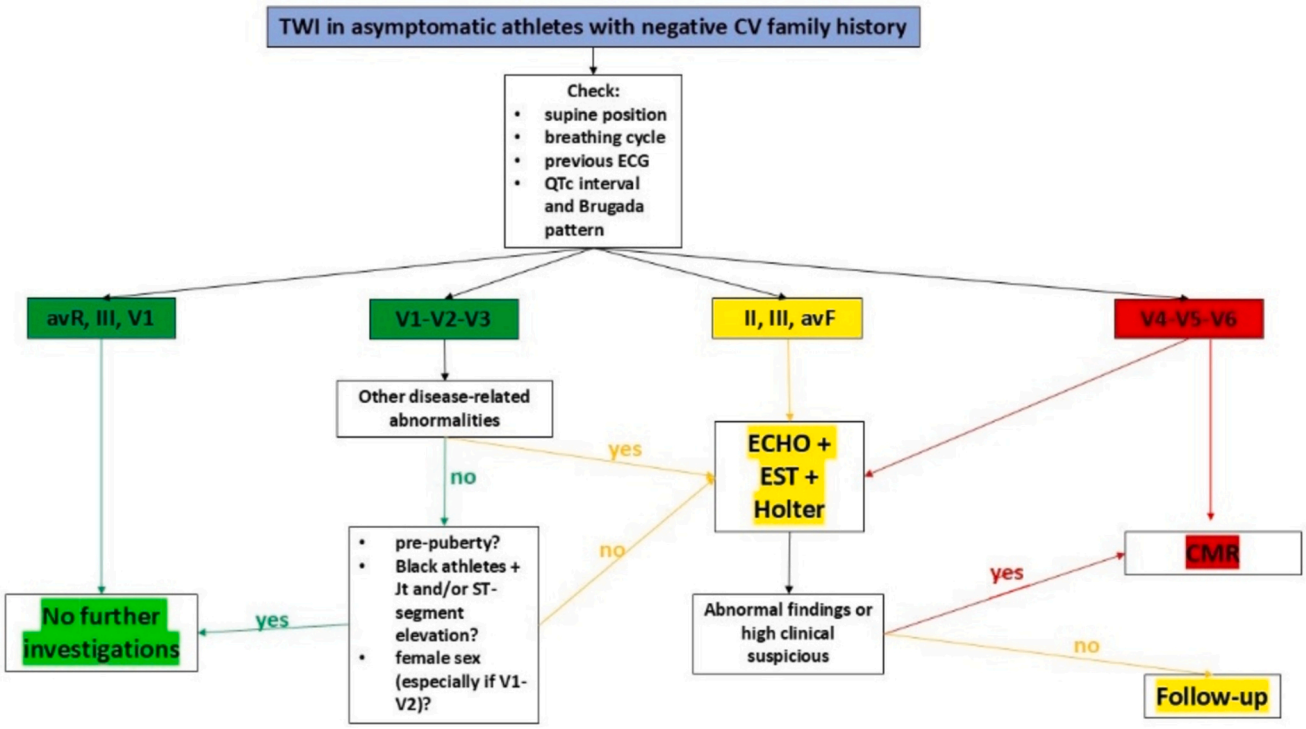

Fig. 7. Proposed algorithm to rule out the presence of an underlying disease in case of a TWI finding in an asymptomatic apparently healthy athlete. The algorithm outlines a stepwise approach, beginning with an assessment of the athlete's supine position, breathing cycle, and previous ECGs. Special attention is given to specific ECG leads with the TWI. Pre-puberty status and factors such as the presence of QTc interval prolongation, Brugada pattern, and demographic considerations (e.g., Black athletes with ST-segment elevation and female athletes) must always be taken into account. Based on pre-test probability and the results of first-line tests (echocardiography - ECHO, exercise stress testing - EST and 24-h ambulatory ECG monitoring including a training session - HOLTER), further investigations particularly with CMR may be warranted.

original draft. **Giampiero Patrizi:** Writing – review & editing, Writing – original draft. **Teresina Vessella:** Writing – review & editing, Writing – original draft. **Alessandro Zorzi:** Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Conceptualization.

Declaration of competing interest

the authors declare no conflict of interest.

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