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Long QT Syndrome in Athletes: Diagnosis, Risk Stratification, and Management for Safe Sports Participation

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Abstract

Background. Long QT Syndrome (LQTS) is a hereditary cardiac channelopathy that predisposes individuals to malignant arrhythmias and sudden cardiac death. In athletes, physiological cardiac adaptations can mask or mimic LQTS, complicating diagnosis and risk assessment.

Methods. This review synthesizes current evidence on the diagnosis, risk stratification, management, and safe sport participation in athletes with LQTS, drawing from cohort studies, clinical guidelines, and expert consensus.

Results. Diagnosis in athletes requires careful ECG evaluation, exercise testing, and genetic analysis to distinguish pathological QT prolongation from training-related changes. Risk stratification integrates clinical history, genotype, and monitoring tools, including wearable

ECG devices. With appropriate therapy, primarily beta-blockers, and individualized monitoring, many athletes can safely participate in moderate- to high-intensity exercise.

Conclusions. Early recognition, structured assessment, and multidisciplinary management enable safe sports participation for athletes with LQTS while reducing the risk of sudden cardiac death. Genotype-specific approaches and continuous monitoring further optimize safety while supporting athletic performance.

Keywords: long QT Syndrome; athletes; diagnosis; risk stratification; sport participation; arrhythmia

Introduction

Long QT Syndrome (LQTS) is a cardiac channelopathy characterized by a prolonged QT interval on the electrocardiogram (ECG), which reflects abnormal ventricular repolarization and predisposes affected individuals to life-threatening ventricular arrhythmias such as torsades de pointes and sudden cardiac death (SCD) (Corrado *i in.*, 2023). Congenital LQTS is an inherited ion channel disorder most often diagnosed in childhood or early adulthood and is associated with pathogenic mutations in multiple genes encoding cardiac ion channels, primarily LQT1, LQT2, and LQT3 subtypes (Galić *i in.*, 2021).

In athletic populations, the clinical identification of LQTS presents unique diagnostic challenges because intense training can physiologically prolong the QT interval and is often accompanied by marked bradycardia, making interpretation of corrected QT measurements more complex (Schnell *i in.*, 2018). Crucially, vigorous physical activity may trigger arrhythmic events in susceptible individuals, making LQTS a recognized but relatively rare contributor to exercise-related sudden cardiac death among young athletes (Harmon *i in.*, 2015).

Given these risks, current research has increasingly focused on risk stratification, cardiac monitoring protocols, and the development of evidence-based guidelines for safe sports participation among competitive athletes with LQTS. Recent studies further emphasize the importance of individualized approaches to decision making about return to play, balancing the benefits of physical activity with the potential cardiac risks inherent to this condition (Aziz & Saarel, 2017; Tobert *i in.*, 2021).

Epidemiology and Pathophysiology

Congenital long QT syndrome (LQTS) is a hereditary cardiac channelopathy that affects approximately 1 in 2,000 to 1 in 2,500 individuals in the general population, although the true prevalence may be higher due to asymptomatic carriers and variable penetrance of pathogenic variants (Refsgaard *i in.*, 2012; Schwartz *i in.*, 2009). Among elite and competitive athletes, isolated prolongation of the QT interval is relatively uncommon, estimated at approximately 0.4%, yet distinguishing between physiological QT prolongation associated with athletic cardiac remodeling and pathological LQTS remains a critical clinical challenge (Basavarajiah *i in.*, 2007; Christou *i in.*, 2022). Misclassification can lead either to unnecessary restrictions from sports participation or, conversely, to unrecognized risk of potentially fatal arrhythmias during exertion.

LQTS results from mutations in genes encoding cardiac ion channels, predominantly KCNQ1 (LQT1), KCNH2 (LQT2), and SCN5A (LQT3), which respectively affect potassium and sodium currents that are essential for normal cardiac repolarization (Balestra *i in.*, 2024). The electrophysiological consequences of these mutations include delayed ventricular repolarization, increased heterogeneity of action potentials across the myocardium, and heightened susceptibility to early afterdepolarizations, which can trigger for example torsades de pointes or sudden cardiac death (SCD) (El-Sherif, 2001; Schwartz *i in.*, 2017).

Physical exertion acts as a potent trigger for arrhythmic events in athletes with LQTS, especially in those with LQT1, due to increased sympathetic stimulation and heart rate acceleration during intense activity (Darbar, 2010). Additionally, comorbidities, electrolyte imbalances (such as hypokalemia and hypomagnesemia), and certain medications (e.g., antiarrhythmics, psychotropics, antibiotics) can exacerbate QT prolongation, further increasing the arrhythmic risk in athletes with or predisposed to long QT Syndrome (Khan *i in.*, 2024). Understanding these interactions between genetic predisposition, electrophysiology, and physical stress is essential for clinicians managing competitive athletes with LQTS.

Diagnosis in Athletes

The diagnosis of Long QT Syndrome (LQTS) is based on a comprehensive clinical evaluation integrating electrocardiographic findings, clinical history, and genetic testing. In athletes, this process is particularly challenging due to physiological cardiac adaptations associated with intensive training, commonly referred to as the “athlete’s heart” (Finocchiaro *i in.*, 2026). Standard resting electrocardiograms (ECG) are the initial diagnostic tool and are essential for detecting QT interval prolongation, however, athletes often present with sinus bradycardia,

increased vagal tone, and early repolarization patterns, all of which can mimic or mask true QT prolongation (Sharma *et al.*, 2018). Accurate measurement of the corrected QT interval (QTc), using formulas such as Bazett or Fridericia, and comparison with sex- and age-specific norms is essential. According to expert consensus, a QTc ≥ 500 ms on repeated ECG recordings is associated with a significantly increased risk of malignant ventricular arrhythmias and sudden cardiac death (Priori *et al.*, 2013). Further evaluation is indicated in male athletes with QTc ≥ 470 ms and female athletes with QTc ≥ 480 ms. In cases of borderline QT prolongation (QTc 470–499 ms in males and 480–499 ms in females), detailed assessment of personal and family history is crucial, particularly regarding syncope, documented arrhythmias, or sudden cardiac death (Christou *et al.*, 2022). A QTc ≥ 480 ms on repeated recordings is generally considered diagnostic in the appropriate clinical context, whereas values between 450 and 480 ms represent a borderline zone requiring further investigation (Gomez *et al.*, 2016; Johnson & Ackerman, 2009).

Current international recommendations propose a stepwise diagnostic algorithm for athletes with suspected LQTS. If QT prolongation is identified, repeat ECG testing is recommended to confirm persistence. When suspicion remains, additional investigations should be performed, including calculation of the Schwartz score (Table 1), which integrates ECG findings, clinical history, and family history to estimate the probability of LQTS. A Schwartz score ≥ 3.5 indicates a high probability of LQTS (Krahn *et al.*, 2022).

Electrocardiographic findings		Points
A	≥ 480 ms	3
	460–479 ms	2
	450–459 ms (in males)	1
B	QTc 4th minute of recovery from exercise stress test ≥ 480 ms	1
C	Torsades de pointes	2
D	T wave alternans	1
E	Notched T wave in 3 leads	1

F	Low heart rate for age	0.5
Clinical history		Points
A	Syncope	
	With stress	2
	Without stress	1
B	Congenital deafness	0.5
Family history		Points
A	Family members with definite LQTS	1
B	Unexplained sudden cardiac death below age 30 among immediate family members	0.5

Table 1. Schwartz score (Schwartz & Crotti, 2011).

Exercise testing provides further diagnostic value by assessing QT dynamics during heart rate changes and recovery, which may unmask abnormal repolarization patterns not evident at rest. Paradoxical QT prolongation during exercise or a $QTc \geq 480$ ms at the fourth minute of recovery supports the diagnosis of LQTS. In selected cases, pharmacological provocation tests (e.g., with epinephrine) can be employed to reveal concealed LQTS phenotypes (Christou i in., 2022). In cases where clinical and instrumental findings remain inconclusive, genetic testing should be considered, particularly for the major LQTS-associated genes (*KCNQ1*, *KCNH2*, and *SCN5A*), which account for the majority of cases. Genetic testing serves as a confirmatory diagnostic tool for individuals with borderline or ambiguous ECG findings, a suggestive family history, or unexplained syncope (Schwartz i in., 2012). Identification of pathogenic variants in LQTS-associated genes can establish a definitive diagnosis and guide management, including risk stratification and sports participation decisions (Napolitano i in., 2005). Importantly, genotype-phenotype correlations provide prognostic information, as different LQTS subtypes demonstrate distinct triggers and risk profiles—for instance, LQT1 events are often exercise-induced, LQT2 events are frequently associated with emotional or auditory triggers, and LQT3 events typically occur during rest or sleep (Crotti i in., 2008).

The diagnosis of LQTS in athletes requires careful differentiation between physiological and pathological findings, supported by repeated ECG evaluation, clinical assessment, and targeted diagnostic testing. A structured algorithm-based approach is essential to ensure accurate diagnosis while minimizing the risk of both underdiagnosis and overdiagnosis in this unique population.

Risk Stratification and Monitoring

Effective risk stratification and monitoring are essential in managing athletes with Long QT Syndrome (LQTS), aiming to prevent life-threatening arrhythmias while allowing safe sports participation. Individual risk assessment is based on factors such as genotype, QTc duration, history of syncope or cardiac arrest, and family history of sudden cardiac death (Wang i in., 2022). Patients with prolonged QTc, prior symptoms, or high-risk genotypes demonstrate a higher risk of ventricular arrhythmias and require closer surveillance (Priori i in., 2003).

Validated risk prediction tools, including the 1-2-3-LQTS-Risk model, integrate clinical and genetic variables to estimate the likelihood of severe arrhythmic events and support clinical decision-making. These models facilitate individualized management strategies, including pharmacological treatment and, in selected cases, implantable cardioverter-defibrillator (ICD) implantation (Mazzanti i in., 2022).

Monitoring strategies combine traditional and modern approaches. Holter monitoring and exercise testing remain fundamental for assessing QT dynamics and detecting arrhythmias (Krijger Juárez i in., 2025). Additionally, wearable ECG devices enable continuous QTc tracking and have emerged as valuable tools for long-term monitoring, particularly in physically active individuals (Delinière i in., 2024).

Management and Recommendations for Athletes

Management of athletes with Long QT Syndrome (LQTS) requires a multidisciplinary, individualized approach that balances the benefits of physical activity with the risk of life-threatening arrhythmias.

Pharmacologic therapy is the cornerstone of treatment. Beta-blockers remain the first-line therapy for most symptomatic and high-risk patients, effectively reducing adrenergically mediated arrhythmic events (Schwartz, 2011). Non-selective beta-blockers such as nadolol or propranolol are preferred due to their consistent effect on heart rate and sympathetic tone (Hauwanga i in., 2024). Recent studies demonstrate that beta-blocker efficacy can vary depending on LQTS subtype, age, and sex, highlighting the importance of individualized

therapy (Went i in., 2021). In patients with persistent arrhythmic events despite optimal beta-blockade, left cardiac sympathetic denervation (LCSD) or implantable cardioverter-defibrillator (ICD) implantation may be indicated (Goldenberg & Moss, 2008). LCSD has been shown to significantly reduce arrhythmic events, particularly in high-risk LQT1 and LQT2 patients, while ICDs are reserved for those with a history of cardiac arrest or refractory syncope (Schwartz i in., 2004).

Sport participation recommendations should be personalized and based on shared decision-making. Evidence indicates that, under optimal medical therapy and with appropriate monitoring, many athletes with LQTS can safely engage in moderate- to high-intensity exercise (Davydoff i in., 2022). Decisions regarding competitive or recreational sports should also consider the type of activity, frequency, and potential exposure to adrenergic surges.

Lifestyle modifications complement pharmacologic therapy and monitoring. Athletes are advised to avoid QT-prolonging medications, maintain electrolyte balance (particularly potassium and magnesium), and ensure adequate hydration. Structured warm-up and cool-down periods may mitigate adrenergic surges, and training intensity should be adjusted according to individual risk profiles (Cho, 2016; Mazzanti i in., 2025). Pediatric and adolescent athletes benefit from tailored activity plans that preserve quality of life and social engagement while minimizing arrhythmic risk (Chen i in., 2022).

Emerging strategies in LQTS management, including gene therapy and microRNA modulation, are under investigation and may allow future personalization of therapy based on genotype and molecular profile (Yang i in., 2022). While not yet standard of care, these approaches highlight the evolving landscape of precision medicine in inherited arrhythmia syndromes.

In summary, effective management of athletes with LQTS integrates pharmacologic therapy, risk stratification, individualized monitoring, genotype-specific considerations, lifestyle modifications, and shared decision-making. When carefully applied, these strategies enable many athletes to safely participate in sports while minimizing the risk of life-threatening arrhythmias.

Safe Sports Participation Guidelines

Participation in sports for athletes with long QT Syndrome (LQTS) requires careful risk assessment, individualized planning, and ongoing monitoring. Decisions must balance the benefits of physical activity with the potential risk of life-threatening arrhythmias.

Return to sport in athletes with long QT Syndrome (LQTS) should be guided by individualized risk assessment and supported by appropriate medical management. Current evidence indicates

that asymptomatic athletes receiving optimal therapy, particularly beta-blockers, may safely resume training under structured supervision (Aziz *et al.*, 2015). Emphasis is placed on continuous monitoring, training quality control, and shared decision-making to balance cardiovascular safety with the physical, psychological, and performance benefits of regular exercise (Johnson & Ackerman, 2013).

Guidelines for athletes with long QT Syndrome recommend individualized exercise restrictions, such as avoiding water sports in LQT1 and pausing activity in symptomatic individuals until specialist evaluation. Return to competitive sports may be considered after at least three months of being asymptomatic on therapy, including beta-blockers and, when indicated, ICDs, emphasizing ongoing monitoring and shared decision-making (Ritt *et al.*, 2022).

Multidisciplinary collaboration is essential for successful management. The athlete's care team should ideally include a sports cardiologist or electrophysiologist, coach or trainer, and other specialists such as exercise physiologists or quality-of-training experts. This team ensures individualized exercise prescriptions, monitors for warning signs during activity, and provides guidance for safe progression in training and competition (Aziz & Saarel, 2017). Shared decision-making, involving the athlete and family, is recommended to weigh potential risks and benefits while respecting the athlete's preferences.

Conclusions and Future Directions

Early diagnosis, accurate risk stratification, and qualitative monitoring of training are essential to ensure the safety of athletes with long QT Syndrome (LQTS). Advances in genetic testing, ECG-based biomarkers, and wearable monitoring technologies have significantly improved the ability to identify high-risk individuals before the occurrence of life-threatening arrhythmic events.

Despite these advances, knowledge gaps remain, particularly regarding the safe intensity and type of sports participation for athletes with different LQTS genotypes. Current research underscores the need for prospective studies evaluating the impact of competitive and recreational exercise, individualized training modifications, and genotype-guided management strategies.

The future of LQTS management in sport relies on multidisciplinary collaboration, integrating sports cardiologists, electrophysiologists, coaches, exercise physiologists, and quality-of-training specialists. Such collaboration facilitates personalized exercise prescriptions, continuous risk assessment, and the development of standardized safety guidelines for athletes across competitive levels.

In conclusion, early recognition, comprehensive risk evaluation, and rigorous monitoring are pivotal to allowing athletes with LQTS to participate safely in physical activity. Future research should focus on longitudinal studies, genotype-specific risk modeling, and the integration of advanced monitoring technologies. These efforts will refine current guidelines, improve safety, and optimize the balance between athletic performance and cardiovascular protection.

Disclosure

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