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Electrocardiographic Abnormalities in Sickle Cell Trait: Prevalence and Clinical Meaning

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Abstract

Sickle cell trait (SCT) has historically been considered a benign carrier state, yet emerging evidence suggests it may confer subtle cardiovascular risks. Electrocardiographic (ECG) abnormalities in SCT carriers—such as ST-T segment deviations, prolonged QT intervals, and nonspecific conduction delays—may reflect subclinical myocardial stress, microvascular ischemia, or predisposition to arrhythmias. The prevalence of these abnormalities varies across populations, and their clinical significance remains incompletely understood. Interpretation is further complicated by the absence of SCT-specific normative ECG data and the transient nature of some changes, which may only manifest under hypoxic, dehydrated, or exertional conditions. Despite these challenges, ECG offers a non-invasive and widely accessible tool for early detection of potential cardiac involvement in SCT, particularly among high-risk individuals such as athletes, military personnel, and those exposed to environmental stressors. This perspective synthesizes current knowledge on the prevalence, patterns, and clinical implications of ECG abnormalities in SCT, highlighting the need for standardized assessment, integration with advanced imaging modalities, and research to clarify prognostic relevance.

Keywords: Sickle cell trait, Electrocardiography, Cardiac conduction, Arrhythmia, Cardiovascular risk

Abbreviations

AV – Atrioventricular
ECG – Electrocardiogram
LV – Left Ventricle
QT – QT Interval
SCT – Sickle Cell Trait
ST – ST Segment
T-wave – T-wave (repolarization wave of the ECG)

Introduction

Sickle cell trait (SCT) affects hundreds of millions of individuals worldwide, with particularly high prevalence in sub-Saharan Africa, the Mediterranean, the Middle East, and among African American populations [1-2]. It results from heterozygosity for the β-globin gene mutation, leading to the coexistence of normal hemoglobin A and sickle hemoglobin S. Historically, SCT has been considered a benign carrier state, with most individuals remaining asymptomatic under normal physiological conditions [3-4].Although SCT is typically asymptomatic, emerging evidence suggests that carriers may experience subtle cardiovascular alterations. Observational studies have linked SCT to renal complications, venous thromboembolism, and sudden exertional deaths in athletes and military personnel. These findings challenge the traditional perception of SCT as entirely benign and suggest that underlying cardiovascular stressors may go unrecognized in this population [5-6].

Electrocardiography (ECG) offers a non-invasive, accessible method to evaluate cardiac conduction, repolarization, and arrhythmogenic risk. In SCT, intermittent red blood cell sickling can impair microvascular perfusion, promote myocardial stress, and trigger transient ischemic changes. Such events may manifest as subtle ECG abnormalities, which, if recognized early, could provide an opportunity for risk stratification and preventive intervention [7-10].Reported ECG changes in SCT carriers include ST-T segment deviations, prolonged QT intervals, T-wave inversions, and nonspecific conduction delays. While these abnormalities are often subtle and may be overlooked as normal variants, they may reflect subclinical myocardial ischemia or predispose to arrhythmias under physiologic or environmental stress. Understanding these patterns is essential for clinicians evaluating Sickle Cell Trait carriers, particularly in high-risk populations [12-13].

Interpreting ECG abnormalities in SCT is complicated by the lack of SCT-specific normative data, variability in clinical expression, and transient nature of changes that may only appear under stress, hypoxia, or dehydration. Conventional ECG interpretation criteria may fail to capture subtle deviations, and existing studies are limited by small sample sizes and heterogeneous methodologies. These factors contribute to under-recognition and uncertainty regarding prognostic significance [14-16].Despite these challenges, ECG abnormalities may have important clinical implications. In high-risk settings—such as athletic training, military service, or exposure to extreme environments—subtle conduction or repolarization disturbances may predispose carriers to arrhythmias, exertional syncope, or sudden cardiac death. Recognizing these changes may guide monitoring, preventive strategies, and individualized counseling for SCT carriers [17-19].This perspective seeks to synthesize current knowledge on ECG abnormalities in SCT, exploring their prevalence, patterns, and potential clinical significance. By highlighting gaps in evidence and proposing avenues for further research, the article aims to improve awareness among clinicians and encourage integration of ECG assessment into cardiovascular risk evaluation for SCT carriers.

Aim

The aim of this perspective is to examine the prevalence, patterns, and clinical significance of electrocardiographic (ECG) abnormalities in individuals with sickle cell trait (SCT). By synthesizing current evidence and highlighting gaps in knowledge, the article seeks to enhance understanding of subclinical cardiac alterations in SCT, inform early risk stratification, and guide monitoring and preventive strategies for at-risk populations.

Methods

This perspective article was developed through a narrative review of the literature, focusing on electrocardiographic (ECG) findings in individuals with sickle cell trait (SCT). A comprehensive search was conducted across PubMed, Scopus, and Web of Science databases, covering publications from inception to August 2025. Search terms included combinations of *"sickle cell trait," "electrocardiography," "ECG abnormalities," "cardiac conduction,"* and *"arrhythmia."* Both clinical and preclinical studies, including original research, case reports, observational studies, and reviews, were considered. Inclusion criteria comprised studies reporting ECG findings in SCT carriers, studies exploring electrophysiologic or microvascular mechanisms relevant to cardiac function in SCT, and literature addressing clinical outcomes associated with ECG abnormalities. Exclusion criteria were studies focused exclusively on sickle cell disease without reference to SCT, non-English publications without available translation, and reports lacking cardiac electrophysiologic data.Given the narrative and perspective-oriented design, formal quality assessment and meta-analysis were not conducted. Instead, emphasis was placed on identifying recurring patterns, pathophysiologic insights, clinical relevance, and gaps in knowledge. Reference lists of key publications were also screened to ensure comprehensiveness. The findings are presented thematically, integrating prevalence data, clinical interpretation, and implications for monitoring and preventive strategies.

Prevalence of ECG Abnormalities in Sickle Cell Trait

Electrocardiographic (ECG) abnormalities have been increasingly reported among individuals with sickle cell trait (SCT), although the true prevalence remains difficult to quantify due to limited large-scale studies and heterogeneous methodologies. Existing evidence, largely derived from small observational cohorts, cross-sectional studies, and case reports, suggests that a significant subset of SCT carriers may exhibit subtle conduction and repolarization changes even in the absence of overt cardiovascular disease [20-22].Commonly reported abnormalities include **ST-T segment deviations**, such as ST depression or T-wave inversions, which may reflect subclinical myocardial ischemia arising from intermittent microvascular obstruction. **Prolonged QT intervals**, another frequently observed finding, could indicate increased susceptibility to ventricular arrhythmias. **Nonspecific conduction delays**, including minor intraventricular conduction abnormalities or first-degree atrioventricular block, have also been documented, potentially reflecting underlying myocardial stress or subtle structural remodeling [23-24].

Prevalence estimates in SCT cohorts vary widely. Some studies report ECG abnormalities in approximately 5–10% of carriers, while others, particularly among high-risk populations such as athletes, military recruits, or individuals exposed to hypoxic or dehydrating conditions, report rates as high as 20–30%. This variability likely reflects differences in study populations, the criteria used to define ECG abnormalities, and the sensitivity of diagnostic methods [25].Importantly, many ECG changes in SCT are subtle and may be interpreted as normal variants in the general population. This contributes to under-recognition and underreporting, highlighting the need for SCT-specific reference ranges and systematic screening protocols. Additionally, the transient nature of some abnormalities—appearing only under physiologic or environmental stress—may further obscure true prevalence [26-27].

Types of Electrocardiographic Abnormalities in Sickle Cell Trait

Electrocardiographic (ECG) abnormalities in individuals with sickle cell trait (SCT) are generally subtle but can provide insight into underlying cardiac electrophysiology and potential arrhythmogenic risk. The most commonly reported abnormalities include conduction disturbances, repolarization changes, arrhythmogenic markers, and variations in autonomic regulation.

Conduction Disturbances: First-degree atrioventricular (AV) block and bundle branch blocks have been observed in SCT carriers. These conduction delays are usually mild and asymptomatic, reflecting minor alterations in electrical signal propagation within the heart. While generally benign, they may warrant monitoring in individuals engaged in strenuous physical activity or those with additional cardiovascular risk factors [26].

Repolarization Changes: Nonspecific ST-T wave abnormalities and early repolarization patterns are frequently noted. These changes indicate subtle alterations in ventricular repolarization and, while often clinically insignificant, can mimic ischemic patterns, particularly under conditions of hypoxia or exercise-induced stress [28].

Arrhythmogenic Markers: Although rare, isolated cases of increased propensity for ventricular or supraventricular arrhythmias have been documented in SCT carriers. Exercise, dehydration, and hypoxic environments may trigger transient arrhythmic episodes, suggesting that SCT may interact with environmental stressors to influence arrhythmia risk [29].

Autonomic Function Variations: Altered heart rate variability, reflecting reduced parasympathetic tone or increased sympathetic activity, has been described. Such autonomic dysregulation may contribute to observed conduction and repolarization changes and may influence exercise tolerance and cardiac response to stress [30].

Pathophysiological Mechanisms of ECG Abnormalities in Sickle Cell Trait

The electrocardiographic (ECG) abnormalities observed in individuals with SCT are likely the result of several interconnected pathophysiological mechanisms. While SCT is typically benign, the presence of hemoglobin S can affect microvascular perfusion and myocardial oxygenation under certain stress conditions, leading to subtle but detectable changes in cardiac electrophysiology (Table 1).

Table 1: Pathophysiological Mechanisms of ECG Abnormalities in Sickle Cell Trait

Mechanism	Description	ECG Manifestation	Clinical Implication
Microvascular Occlusion	Intermittent sickling impairs myocardial microcirculation under hypoxia, dehydration, or acidosis	ST-T wave changes, early repolarization patterns	Usually benign, may mimic ischemia; may influence arrhythmic risk under stress
Hypoxia-Induced Myocardial Stress	Reduced oxygen delivery during exercise, altitude exposure, or acute illness	QT interval variation, T-wave abnormalities	Can increase arrhythmogenic susceptibility, particularly under physiologic stress
Autonomic Dysfunction	Altered sympathetic-parasympathetic balance affecting heart rate variability	Sinus arrhythmia, AV conduction delay	May affect exercise tolerance and predispose to arrhythmias in susceptible individuals
Subclinical Structural Changes	Minor left ventricular remodeling or diastolic dysfunction	Bundle branch block, conduction delays	Generally asymptomatic; highlights the need for monitoring in high-risk settings

Microvascular Occlusion: Intermittent sickling of red blood cells under hypoxic, dehydrated, or acidic conditions can transiently impair myocardial microcirculation. This may cause minor ischemic changes that influence conduction pathways and repolarization, contributing to ECG abnormalities such as ST-T wave changes or early repolarization patterns [31].

Hypoxia-Induced Myocardial Stress: During exercise, altitude exposure, or acute illness, oxygen delivery to myocardial tissue may be transiently compromised. Hypoxia can alter ion channel function, prolong action potentials, and modify ventricular repolarization, producing detectable ECG changes and potentially increasing arrhythmogenic susceptibility [32].

Autonomic Dysfunction: Altered sympathetic and parasympathetic balance has been observed in SCT carriers, leading to variations in heart rate variability and influencing conduction and repolarization patterns. This autonomic modulation can manifest as sinus arrhythmia, subtle AV conduction delays, and repolarization abnormalities [33].

Subclinical Structural Changes: Some studies suggest that SCT carriers may exhibit minor left ventricular remodeling or diastolic functional alterations detectable by echocardiography. These structural changes can impact myocardial conduction properties, further contributing to ECG findings [34].

Clinical Meaning and Interpretation

Electrocardiographic (ECG) abnormalities in sickle cell trait (SCT) carriers, while often subtle, may provide critical insights into subclinical cardiovascular stress and potential arrhythmogenic risk. These changes can reflect underlying pathophysiologic mechanisms, including intermittent microvascular ischemia, myocardial strain, and alterations in cardiac conduction [28].**ST-T segment deviations**, such as ST depression or T-wave inversions, may signal localized ischemia resulting from transient obstruction of the coronary microvasculature by sickled red blood cells under stress conditions like hypoxia, dehydration, or intense physical exertion. Even in the absence of epicardial coronary artery disease, these changes could indicate myocardial oxygen supply-demand imbalance and early cellular injury [29]. **Prolonged QT intervals** and repolarization abnormalities, including flattened or biphasic T waves, may predispose SCT carriers to ventricular arrhythmias. These findings are particularly relevant in high-stress environments such as competitive athletics or military training, where sudden exertional cardiac events have been documented. Although most carriers remain asymptomatic, the presence of these ECG markers could identify individuals at higher risk for arrhythmogenic events [30].

Conduction delays, including minor intraventricular blocks or first-degree atrioventricular delay, may reflect subtle myocardial remodeling or autonomic imbalance induced by repeated microvascular stress. While these abnormalities are generally benign, their detection warrants careful longitudinal monitoring, particularly when combined with other risk factors such as dehydration, anemia, or electrolyte disturbances [31].Interpretation of ECG findings in SCT must consider context. Many abnormalities are transient, appearing only under physiologic stress, and conventional ECG reference ranges may fail to capture SCT-specific deviations. Integrating ECG assessment with clinical history, biomarkers of myocardial injury (e.g., troponins, NT-proBNP), and advanced imaging modalities (e.g., cardiac MRI, PET) can improve understanding of their clinical significance and guide preventive strategies(Table 2) [32-33].

Table 2: Clinical Meaning and Interpretation of ECG Abnormalities in Sickle Cell Trait

ECG Abnormality	Prevalence in SCT	Clinical Interpretation	Potential Implications
First-degree AV block	5–10%	Mild conduction delay; usually asymptomatic	Rarely progresses; monitoring advised in symptomatic individuals or athletes under stress
Bundle branch block	2–5%	Interventricular conduction delay	Generally benign; may indicate subclinical structural changes
Early repolarization pattern	10–15%	Benign variant; ST elevation in precordial leads	Typically no intervention required; important to differentiate from ischemia
Nonspecific ST-T changes	8–12%	Minor repolarization abnormalities	Usually asymptomatic; can mimic ischemia during stress or hypoxia
QT interval variation	3–7%	Mild prolongation or shortening	Monitor for arrhythmogenic risk in extreme exercise or hypoxic conditions
Sinus arrhythmia	15–20%	Physiologic heart rate variability	Often benign; reflects autonomic tone and may influence exercise tolerance
Altered heart rate variability	10–15%	Reduced parasympathetic activity	May predispose to arrhythmia under stress; warrants monitoring

Challenges and Limitations

Understanding electrocardiographic (ECG) abnormalities in sickle cell trait (SCT) presents several intertwined challenges that limit both clinical application and research progress. First, the existing body of evidence is sparse and heterogeneous. Most studies are small, observational, or based on case reports, with varying methodologies and inconsistent definitions of ECG abnormalities. This variability makes it difficult to estimate true prevalence or to generalize findings across diverse populations [34].A key challenge lies in the absence of SCT-specific normative ECG data. Conventional reference ranges are derived from the general population and may fail to detect subtle deviations that are meaningful in SCT carriers. As a result, many minor conduction or repolarization changes are likely under-recognized or dismissed as normal variants, limiting early detection of potential cardiac risk [35].Adding to the complexity, many ECG abnormalities in SCT are transient and stress-dependent. They may manifest only during hypoxia, dehydration, or intense physical exertion, conditions that are not always captured during routine resting ECG assessment. This transient nature reduces the sensitivity of single-timepoint evaluations and underscores the need for dynamic or ambulatory monitoring in research and clinical practice [36].

Another limitation is the uncertain prognostic significance of detected abnormalities. While ST-T deviations, prolonged QT intervals, and minor conduction delays suggest subclinical myocardial stress, there is limited longitudinal data linking these findings to arrhythmias, sudden cardiac events, or long-term cardiac remodeling in SCT carriers. Consequently, clinicians often rely on extrapolation from other populations, which may not fully reflect SCT-specific risk [37].Resource constraints further challenge comprehensive evaluation. Advanced imaging techniques such as cardiac MRI or PET scans, which could complement ECG findings and clarify myocardial involvement, are often inaccessible in regions with high SCT prevalence. Moreover, comorbidities such as hypertension, electrolyte imbalances, or coexisting hemoglobinopathies can confound ECG interpretation, making it difficult to attribute findings solely to SCT [37].

Future Directions

The evolving understanding of electrocardiographic (ECG) abnormalities in sickle cell trait (SCT) underscores the need for focused research and clinical innovation. Future efforts should prioritize establishing SCT-specific normative ECG data to improve the detection of subtle conduction and repolarization abnormalities that may otherwise be dismissed as normal variants. Such benchmarks would enhance clinical interpretation and provide a foundation for standardized screening protocols [38].Longitudinal studies are also essential to determine the prognostic significance of ECG findings in SCT. By following carriers over time, researchers can correlate subtle abnormalities with outcomes such as arrhythmias, myocardial injury, or sudden cardiac events. This knowledge will help identify individuals at heightened risk and guide personalized monitoring and preventive strategies [39].

Integration of ECG with advanced diagnostic modalities, including echocardiography, cardiac magnetic resonance imaging, and PET scans, offers another avenue to improve understanding. These tools can reveal structural or perfusion changes that accompany electrical abnormalities, providing a more complete picture of myocardial health. Stress testing, whether physiologic or controlled, may uncover transient ischemic changes that remain invisible on resting ECGs [40].High-risk populations, including athletes, military personnel, and individuals exposed to hypoxia or dehydration, warrant particular attention. Developing evidence-based screening and monitoring protocols tailored to these groups could reduce the incidence of exertional cardiac events and inform safe participation in physically demanding activities [41].

Pharmacologic and preventive interventions also deserve exploration. Simple measures, such as optimizing hydration or modulating exercise intensity, alongside potential therapies targeting microvascular perfusion or arrhythmia risk, may mitigate the cardiovascular impact of SCT.

Concurrently, genetic and molecular studies could elucidate why some carriers exhibit ECG abnormalities while others remain unaffected, enabling precision medicine approaches [42].Global health considerations must guide future directions. Access to ECG monitoring, advanced diagnostics, and clinician education remains limited in many regions with high SCT prevalence. Strengthening infrastructure, raising awareness, and implementing cost-effective screening strategies are critical to ensuring that at-risk populations benefit from early detection and intervention [43-44].

Conclusion

Electrocardiographic (ECG) abnormalities in sickle cell trait (SCT) carriers, while often subtle and underrecognized, may reflect subclinical myocardial stress, microvascular ischemia, and potential arrhythmogenic risk. Patterns such as ST-T segment deviations, prolonged QT intervals, and nonspecific conduction delays have been documented, particularly in high-risk populations exposed to physiological or environmental stressors. Although most carriers remain asymptomatic, these findings highlight the need for careful interpretation, contextualized clinical evaluation, and, when appropriate, longitudinal monitoring. Current limitations—including small study cohorts, lack of SCT-specific ECG reference ranges, transient nature of some abnormalities, and unclear prognostic significance—underscore the importance of further research. Large-scale epidemiological studies, integration with advanced imaging and biomarkers, and longitudinal outcome assessments are needed to clarify the clinical relevance of ECG changes in SCT.

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