Volume 3 Number 1, June 2026

NASARAWA JOURNAL OF MEDICINE

ISSN: 2992-426X (Print)
2992-443X (Online)

Published by
MEDICAL AND DENTAL CONSULTANTS' ASSOCIATION OF NIGERIA (MDCAN)
FEDERAL UNIVERSITY TEACHING HOSPITAL, LAFIA, NASARAWA STATE

Volume 3 Number 1 June 2026

REVIEW ARTICLE

A Practical Guide to Paediatric ECG Interpretation for General Practitioners: From Age-Related Normality to Recognition of Pathology

Bello Abdullahi Ibrahim[1], Surajudeen Oyeleke Bello[2]

Abstract

The electrocardiogram (ECG) is a vital diagnostic tool in children presenting with syncope, chest pain, palpitations, and suspected heart conditions. However, paediatric ECG interpretation presents unique challenges due to significant age-related physiological changes affecting heart rate, axis, intervals, and wave morphology; hence, what is normal for a neonate may be abnormal for an adolescent. General practitioners (GPs) are often the first point of contact for children with these presentations, yet many report limited confidence in interpreting paediatric ECGs. Recent evidence from a 2025 study demonstrated a mean accuracy of 76.6% among clinicians, with notable misdiagnosis rates in emergency and resource-limited settings.

This article presents a practical, evidence-informed framework for paediatric ECG interpretation, developed through synthesis of peer-reviewed literature, published normative data, and established cardiology guidelines. We provide an overview of developmental physiology, a structured five-step approach to ECG analysis, and discussions of common arrhythmias and red flags requiring urgent cardiology referral. Reference tables offer age-adjusted normal parameters, and a summary algorithm supports clinical decision-making. Technical considerations, including lead placement, motion artefact, and the limitations of automated interpretations, are addressed to reduce misdiagnosis. The goal is to bolster GPs' confidence in distinguishing benign from dangerous conditions, enhance patient safety, and optimise the utilisation of specialist care.

Keywords: Paediatric electrocardiography, ECG interpretation, age-related changes, arrhythmias, medical education, general practice

Introduction

The electrocardiogram (ECG) is one of the most frequently performed non-invasive cardiovascular investigations in paediatric practice. Indications include evaluation of syncope, chest pain, palpitations, suspected arrhythmias, drug ingestion, electrolyte disturbances, and initial assessment of suspected congenital or acquired heart disease. [1,2] General practitioners (GPs) are often the first point of contact for children with these presentations, yet many lack formal training in paediatric ECG interpretation.

Despite the widespread availability of ECG machines, paediatric ECG interpretation remains a source of anxiety for many medical officers and general practitioners. This unease is understandable: the paediatric ECG is not simply a smaller version of the adult tracing. It is a dynamic record that evolves with the physiological changes of the growing heart, and findings that would be pathological in an adult may be entirely normal in a child. [3,4]

The clinical significance of this challenge is underscored by recent evidence. A 2025 study evaluating ECG interpretation skills among paediatric trainees and faculty reported a mean accuracy score of only 76.6%, with major over- or under-diagnosis (errors with potential for patient harm) occurring in 12.3% of interpretations.[5] These findings align with

Date Received- 01 March 2026, Date Accepted- 03 August 2026
Date Published online- 25 August 2026

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Published by: MEDICAL AND DENTAL CONSULTANTS' ASSOCIATION OF NIGERIA (MDCAN)
FEDERAL UNIVERSITY TEACHING HOSPITAL, LAFIA, NASARAWA STATE
Citation: Ibrahim BA, Bello SO. A Practical Guide to Paediatric ECG Interpretation for General Practitioners: From Age-Related Normality to Recognition of Pathology, *Nasarawa Journal of Medicine* 2026: 3 (1) 01-10

NASJM

earlier work demonstrating that misinterpretation of paediatric ECGs in emergency department settings can lead to diagnostic delays and inappropriate clinical decisions.[6]

Despite the availability of numerous educational resources on paediatric ECG interpretation, many are either comprehensive textbooks that are impractical for rapid clinical reference or online materials that lack a systematic approach and age-specific normative data. Consequently, non-specialist frontline clinicians have limited access to concise, clinically oriented guidance that integrates developmental physiology, age-adjusted normal values, a structured interpretation algorithm, and clear referral criteria. This review aims to address this gap by providing an evidence-informed, stepwise approach to paediatric ECG interpretation for general practitioners and other frontline clinicians. It outlines the developmental basis of age-related ECG changes, presents practical reference tables of normal values, discusses common normal variants and clinically important abnormalities, highlights red flags requiring specialist referral, and reviews technical factors that influence ECG quality and interpretation.

Methodology

Evidence synthesis

This article is an evidence-informed narrative review developed to provide a practical clinical guide for frontline clinicians. Although not conducted as a formal systematic review, the literature search and evidence synthesis followed a structured and transparent approach consistent with recommendations for high-quality narrative reviews.

Literature search

Electronic searches were performed in PubMed, Google Scholar (for supplementary citation tracking), and the Cochrane Database of Systematic Reviews for publications from January 2000 to December 2025. Search terms included paediatric ECG, paediatric electrocardiogram, ECG interpretation, normal ECG values in children, age-related ECG changes, paediatric arrhythmias, long QT syndrome in children, and ECG referral criteria. Reference lists of key publications and relevant guidelines were also manually screened to identify additional eligible sources.

Eligibility criteria

Eligible publications included peer-reviewed original research, systematic reviews, clinical practice guidelines, consensus statements, and authoritative textbook chapters addressing paediatric ECG interpretation, normal age-specific values, cardiac conduction abnormalities, or arrhythmia recognition in children (0–18 years). Non-English publications, conference abstracts, editorials, and adult-only studies were excluded. Case reports were included only when illustrating clinically important diagnostic findings.

Evidence synthesis

Evidence was synthesized narratively, with emphasis on studies providing age-specific normative ECG data, diagnostic accuracy, and clinically relevant interpretation strategies. Greater weight was given to large population-based studies, internationally recognised reference datasets (including those of Davignon *et al*. and Rijnbeek *et al*.),[12][13] and current professional society guidelines. Where discrepancies existed between sources, recommendations were based on the best available evidence, prioritising contemporary guidelines and consistently validated reference values. Recommendations for referral criteria were cross-referenced with published cardiology society guidelines.

Limitations

As a narrative review, this article did not include a formal risk-of-bias assessment or quantitative meta-analysis. Consequently, recommendations should be interpreted alongside clinical judgement, institutional protocols, and specialist consultation where appropriate.

The Developmental Physiology of the Paediatric ECG

Understanding why the paediatric ECG changes with age requires an appreciation of the dramatic circulatory transition at birth and subsequent cardiac maturation.

The Foetal and Neonatal Circulation. In utero, the placenta serves as the organ of gas exchange. The pulmonary vasculature is constricted, resulting in high pulmonary vascular resistance, while the systemic circulation offers relatively low resistance. The right ventricle pumps against a high-pressure system and becomes the dominant chamber, hypertrophying to exceed the left ventricle at birth.[4,7] This right ventricular dominance explains why neonatal ECGs differ markedly from adult tracings.

Postnatal Transition. With the first breaths after delivery, the lungs expand, pulmonary vascular resistance falls dramatically, and systemic vascular resistance increases as the umbilical cord is clamped. The foramen ovale and ductus arteriosus close, establishing adult-type circulation. Over subsequent weeks and months, the left ventricle gradually assumes its role as the systemic pump, while right ventricular mass regresses relatively as the left ventricle hypertrophies in response to increasing systemic demands. [8] This ventricular remodelling is reflected in the gradual leftward shift of the QRS axis and the progression of precordial R-waves throughout childhood. [9] Adult-type R-wave progression typically emerges by 3–4 years of age, though individual variation is considerable.

Electrophysiological Correlates. The smaller physical dimensions of the infant heart mean electrical impulses travel shorter distances, resulting in shorter durations of all ECG intervals. The PR interval, QRS duration, and QT interval all increase progressively with age as the heart enlarges and conduction pathways elongate. [10] Similarly, the resting heart rate is highest in the neonatal period, when cardiac output is heart rate-dependent due to the relatively noncompliant immature ventricle, and gradually declines to adult values by adolescence. [4]

Special Considerations for Preterm Infants. Infants born prematurely may exhibit ECG patterns more typical of their corrected gestational age than chronological age. Normative data in this population are limited, and cardiology input is advisable when significant abnormalities are suspected in ex-preterm infants. [2]

These developmental changes mean that a single set of "normal" values cannot apply to all children. Age-specific normative data are essential, and the clinician's first step in interpretation must always be to note the child's age.

Limitations of ECG as a Diagnostic Tool

Clinicians must be aware that the ECG has important

limitations in paediatric practice:

1. A normal ECG does not exclude structural heart disease. A child with a significant ventricular septal defect, aortic stenosis, or even hypertrophic cardiomyopathy may have a normal or near-normal ECG. Clinical decision-making must always integrate history and physical examination.
2. Poor sensitivity for certain conditions. The ECG has low sensitivity for detecting atrial septal defects, mild ventricular hypertrophy, and early cardiomyopathy.[14] A normal ECG should not provide false reassurance when clinical suspicion is moderate or high.
3. Age-related overlap. Normal and abnormal values overlap considerably at age extremes (neonates and adolescents), making borderline findings challenging to interpret without serial tracings or echocardiography.
4. Technical limitations. Paediatric ECGs are more susceptible to artefact from crying, movement, and lead misplacement than adult tracings. Poor-quality recordings may simulate pathology.
5. Machine interpretation is unreliable. Automated ECG reports frequently misclassify normal paediatric variants as abnormal and may miss significant abnormalities, particularly prolonged QTc intervals and subtle pre-excitation patterns.

Therefore, the ECG should be viewed as one component of clinical assessment, not a standalone diagnostic test. Over-reliance on ECG findings without appropriate clinical correlation can lead to both under-diagnosis and over-investigation.

Technical Considerations Before Interpretation

Before interpreting any paediatric ECG, the clinician must ensure the tracing is technically adequate. Errors in lead placement, motion artefact, and crying can all produce abnormalities that mimic pathology. [11]

Lead Placement. Precordial lead misplacement is common and can dramatically alter R-wave progression. Key points:

· V1 and V2 should be placed in the 4th intercostal space, right and left of the sternum.
· V3 is midway between V2 and V4.
· V4 should be placed in the 5th intercostal space at the midclavicular line (not simply "under the nipple," as nipple position varies).
· Misplacement of V1 and V2 superiorly can produce an rSr' pattern mimicking right ventricular conduction delay. [11]

Motion Artefact. Young children rarely lie still during ECG recording. Crying, limb movement, and respiratory variation can produce baseline wander, muscle tremor artefact, and apparent arrhythmias. If the tracing is significantly degraded, repeating the ECG with adequate distraction, swaddling, or after feeding an infant is preferable to interpreting a poor-quality tracing.

Effects of Crying and Tachypnoea. Crying increases heart rate and can produce transient ST-segment changes due to tachycardia and altered ventricular repolarisation. Sinus tachycardia from crying should not be mistaken for supraventricular tachycardia; the gradual onset and offset, along with rate variability during crying, typically distinguish it. [11]

Half-Standardisation Recognition. Some paediatric ECGs are recorded at half-standardisation (5 mm/mV instead of 10 mm/mV) when the voltages are very high. This should be noted on the tracing (e.g., "5 mm/mV" or "half gain"). When half standardisation is used, voltage measurements must be doubled for interpretation. Conversely, double standardisation (20 mm/mV) may be used for low-voltage tracings, thereby halving the required voltage measurements. Always check the calibration marker at the beginning of the tracing before measuring voltages.

When to Repeat. If the tracing shows significant artefact, if lead placement is uncertain, or if findings are discordant with the clinical picture, repeating the ECG is always appropriate. A suboptimal tracing interpreted with false confidence is more dangerous than delaying interpretation for a technically adequate recording.

A Stepwise Approach to Paediatric ECG Interpretation

A systematic, reproducible approach minimises the risk of overlooking abnormalities. The following five-step framework is adapted from established paediatric cardiology guidelines and is suitable for routine clinical use. [2,4,12]

Always begin by confirming that the ECG belongs to the correct patient and checking the recording speed (standard 25 mm/s) and voltage calibration (standard 10 mm/mV). A one-page summary algorithm is provided in Figure 1.

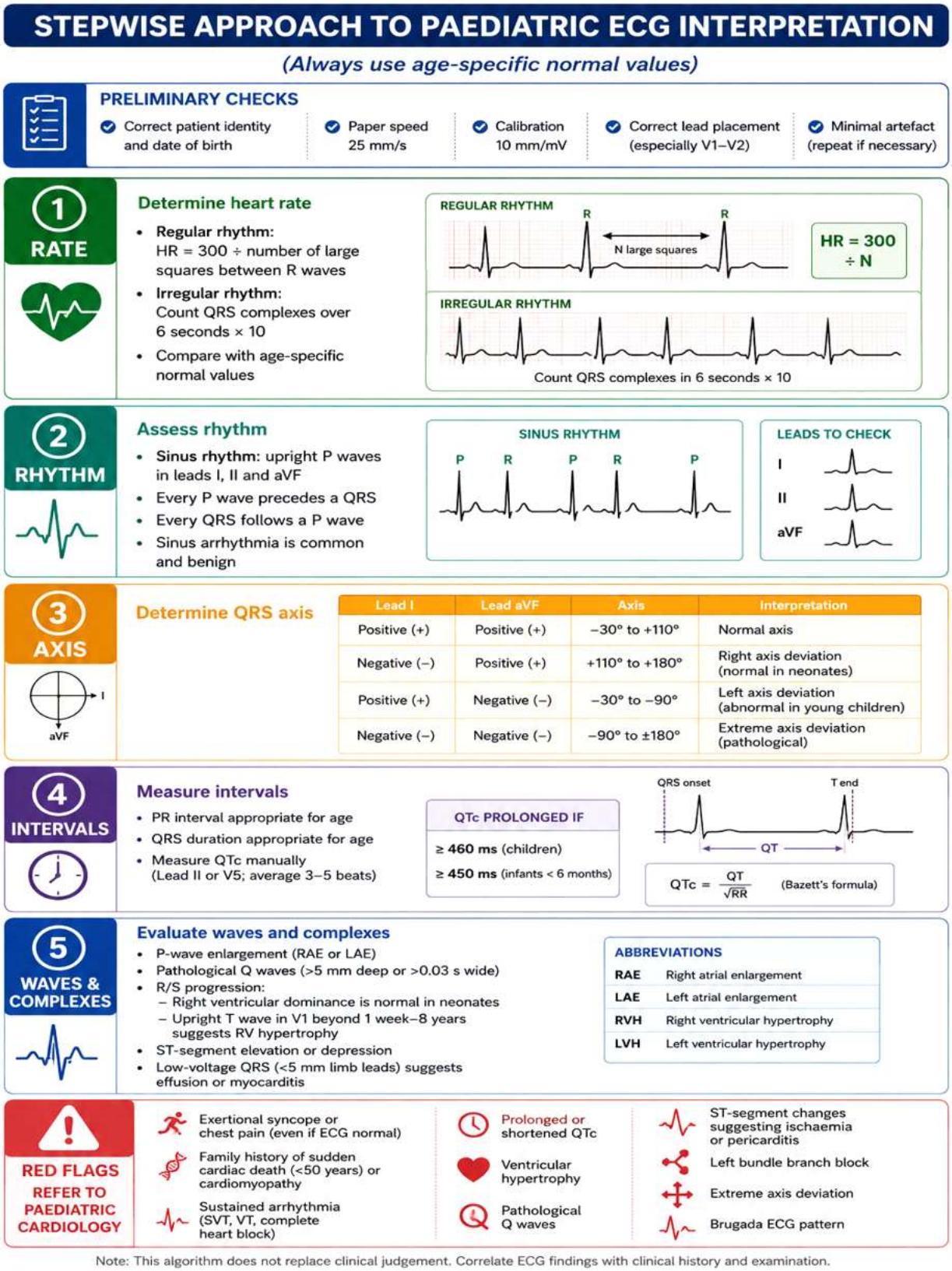
Step 1: Rate – Is It Appropriate for Age?

Heart rate varies inversely with age. Tachycardia or bradycardia must be assessed against age-specific reference ranges (Table 1). An approximate rate can be calculated by dividing 300 by the number of large squares (0.2 s) between consecutive R waves (for regular rhythms). For irregular rhythms, count the number of complexes over a 6-second interval (30 large squares at 25 mm/s) and multiply by 10 to obtain the rate in bpm.

Table 1: Age-Specific Normal Heart Rates and Upper Limits of Intervals

Age	Heart Rate (bpm)	QRS Axis (°)	PR Interval (ms) (upper limit)	QRS Duration (ms) (upper limit)	QTc Upper Limit (ms)†
Preterm infant*	120–180	60–180	120	70	460
Neonate (0–30 days)	90–180	30–180	120	70	450–460‡
Infant (1–12 months)	110–160	5–125	140	75	450
1–5 years	100–150	5–110	170	75	440
6–12 years	80–120	20–120	190	85	440
12+ years	60–100	–30–105	210	100	440 (males) / 460 (females)

Compiled from references [2,4,12,13]; normative data for preterm infants are limited—values shown are approximations. †All QTc values require **manual measurement**; automated readings are unreliable; ‡Some sources use 460 ms for all children under 6 months; clinical correlation advised.



Note: Manual interpretation is recommended for all paediatric ECGs as machine-generated reports may be unreliable in children. Always verify the heart rate, QTc interval, and the presence of delta waves/pre-excitation [Developed by the authors based on published evidence]

Figure 1: Paediatric ECG Interpretation Algorithm

Step 2: Rhythm – Is It Sinus?

Sinus rhythm is present when: (1) P waves are upright in leads I, II, and aVF (indicating a normal P-wave axis of 0° to +90°); (2) a QRS complex follows every P wave; and (3) every QRS complex is preceded by a P wave. [2] The rhythm should be roughly regular, although **sinus arrhythmia**, cyclic variation in the R-R interval with respiration is an extremely common and entirely benign finding throughout childhood (see Figure 2). [15]

Step 3: Axis – Is the QRS Axis Normal for Age?
The net vector of ventricular depolarisation determines the mean QRS axis in the frontal plane. The normal axis shifts leftward with age (Table 1). In neonates, right axis deviation (up to +180°) is normal; by adolescence, the axis should fall within the adult range (–30° to +105°).

Simplified Bedside Axis Determination: For clinicians less comfortable with vector calculations, a rapid assessment can be made:

- **Normal axis:** QRS predominantly positive in both lead I and lead aVF.
- **Right axis deviation (RAD):** QRS predominantly negative in lead I but positive in aVF.
- **Left axis deviation (LAD):** QRS predominantly positive in lead I but negative in aVF.
- **Extreme ("north-west") axis:** QRS negative in both lead I and aVF (range –90° to ±180°). This is always pathologic.

Left axis deviation (less than –30°) is abnormal in young children and may suggest conditions such as atrioventricular septal defect, tricuspid atresia, or left ventricular hypertrophy. [2,15] Extreme axis deviation warrants urgent echocardiography.

Step 4: Intervals – PR, QRS, and QTc
PR Interval. Measured from the onset of the P wave to the onset of the QRS complex (best in lead II), the PR interval reflects conduction time through the atria and AV node. It increases with age and decreases with faster heart rates. A **short PR interval** (below the **lower** limit for age) may be a normal variant but should prompt a search for delta waves suggestive of Wolff-Parkinson-White (WPW) syndrome. [12] A **prolonged PR interval** (above the upper limit for age) indicates first-degree AV block, which can be idiopathic, seen in myocarditis, or associated with congenital heart disease such as Ebstein anomaly or atrial septal defect. [2]

QRS Duration. Measured from the onset of the Q wave to the termination of the S wave (best in lead II or V5/6), QRS duration reflects ventricular depolarisation time and increases with age as ventricular mass increases (Table 1). Prolongation beyond age-specific norms suggests bundle branch block or ventricular conduction delay. **Right bundle branch block** (RSR' pattern in V1 with wide terminal S in V6) is common after cardiac surgery but can occur as an isolated finding. **Left bundle branch block** is rare in children and, if present, almost always indicates underlying structural heart disease. [2,16]

QT Interval and QTc. The QT interval varies inversely with heart rate, necessitating correction (QTc). The Bazett formula (QTc = QT / √RR) remains the most widely used method despite its limitations at extremes of heart rate, i.e., it tends to overcorrect at fast rates (>150 bpm) and undercorrect at slow rates. In infants with tachycardia, alternative formulae (Fridericia, Framingham) may be more accurate, but cardiology review is advisable if QTc is borderline. [17]

Critical Point: Automated QTc readings are unreliable and must not be used in isolation. Measurement should be performed manually in lead II or V5, averaging 3–5 beats to account for respiratory variation. [2] Prolonged QTc (≥460 ms in children, ≥450 ms in infants under 6 months) is a marker for potentially life-threatening ventricular arrhythmias and requires cardiology referral. [18] Causes include congenital long QT syndromes, electrolyte disturbances (hypokalaemia, hypocalcaemia, hypomagnesaemia), and medications. A QTc < 340 ms (short QT syndrome) is also associated with arrhythmia risk and should prompt specialist review. [15]

Step 5: Waves and Complexes – P, QRS, ST, T
P Waves. Normal P waves are ≤3 mm in amplitude and <0.10 s duration in children (<0.08 s in infants). [2] Tall, peaked P waves (P pulmonale) suggest right atrial enlargement; broad, notched P waves (P mitrale) suggest left atrial enlargement.
QRS Morphology and Voltage. The normal evolution of precordial R-wave progression reflects the changing balance of ventricular forces. In neonates and infants, dominant R waves in the right precordial leads (V1–V3) with deep S waves in the left precordial leads are normal, mirroring right ventricular dominance.[4] Adult-type R-wave progression (R > S in V5–V6, S > R in V1–V2) typically emerges by 3–4 years of age. An RSR' pattern in V1 without QRS prolongation (incomplete right bundle branch block pattern) is a common normal variant, present in up to 10% of healthy children. [16]

Simplified Screening for Ventricular Hypertrophy:

- **Suspect RVH if:** R wave in V1 > 98th percentile for age; upright T wave in V1 between 1 week and 8 years of age; pure R wave or qR pattern in V1. [12,16]
- **Suspect LVH if:** S in V1 + R in V6 (or V5) > age-specific 98th percentile; R wave in V6 > 25 mm in infants or >30 mm in older children; inverted T waves in V5–V6 (strain pattern). [12,16]

While detailed age-based 98th percentile criteria have been published (Davignon et al., 1979) [13], we recommend simplified voltage thresholds (e.g., RV6 > 2.5 mV or SV1 > 2.0 mV for LVH) during initial screening. These fixed cut-offs are derived from the upper limits of normal and offer a practical, bedside-friendly alternative without compromising diagnostic utility for the purposes of this review. Regardless of which ECG criteria you choose (percentile-based or simplified), it is essential to remember that echocardiography remains the gold standard for confirming ventricular hypertrophy, given the inherent low sensitivity of ECG

Q Waves. Small Q waves (≤5 mm depth, ≤0.03 s duration) are normal in the inferior (II, III, aVF) and left precordial (V5–V6) leads after the first few days of life. Pathological Q waves, i.e., deep (>5 mm) and/or wide (>0.03 s) suggest myocardial infarction or fibrosis and may be seen in anomalous left coronary artery from the pulmonary artery (ALCAPA) or hypertrophic cardiomyopathy. [2,15]

ST Segment and T Waves. The ST segment should be isoelectric. Minor ST elevation (≤1 mm in limb leads, ≤2 mm in precordial leads) can be normal, particularly in adolescents with early repolarisation.[15] Significant ST depression or elevation suggests ischaemia, pericarditis, or ventricular strain. Low-voltage QRS (<5 mm in limb leads) may indicate pericardial effusion or myocardial dysfunction and warrants echocardiography.

T-wave interpretation is highly age-dependent. At birth, T waves may be upright in all precordial leads. By **day 3–7 of**

life, the T waves in V1–V3 normally invert, reflecting changing haemodynamics and ventricular regression. This **"juvenile T-wave pattern"** of inverted T waves in the right precordial leads can persist into adolescence (typically resolving by 12–14 years, though variable) and is a normal finding. [4,8] However, T waves should remain upright in the inferior and lateral leads (I, II, aVF, V5–V6) after the first week. **Persistently upright T waves in V1 between 1 week and approximately 8 years of age suggest right ventricular hypertrophy until proven otherwise.** [2,16] Some normal infants may retain upright T waves in right precordial leads beyond day 7 without pathology, but correlation with other RVH signs is essential.

Common Normal Variants in Children

Several findings that might trigger concern in an adult ECG are entirely benign in children. Recognising these variants prevents unnecessary investigation and anxiety.

- **Sinus Arrhythmia:** Phasic variation in heart rate with respiration is nearly universal in children and represents normal autonomic modulation. [15]
- **Juvenile T-Wave Pattern:** Inverted T waves in leads V1–V3 (and occasionally V4) are normal throughout childhood and may persist into early adulthood; they typically resolve by adolescence. [4,8]
- **Incomplete Right Bundle Branch Block:** An rSR' pattern in V1 with a normal QRS duration is a frequent finding (up to 10% of healthy children), attributed to delayed activation of the right ventricular outflow tract. Longitudinal studies confirm its benign nature in isolation. [16,19]
- **Sinus Bradycardia of the Athlete:** Well-conditioned adolescents may exhibit resting heart rates in the 40s–50s bpm, similar to adult athletes. [15]
- **Early Repolarisation:** Concave ST elevation with notching at the J point, most prominent in the lateral precordial leads, is a common normal variant in adolescents, particularly males. [15]
- **Neonatal Right Ventricular Dominance:** Dominant R waves in V1–V3 with deep S waves in V5–V6 are normal in the first months of life, gradually transitioning to the adult pattern by 3–4 years. [4]

Recognising Common Pathologies (With Example ECG Tracings)

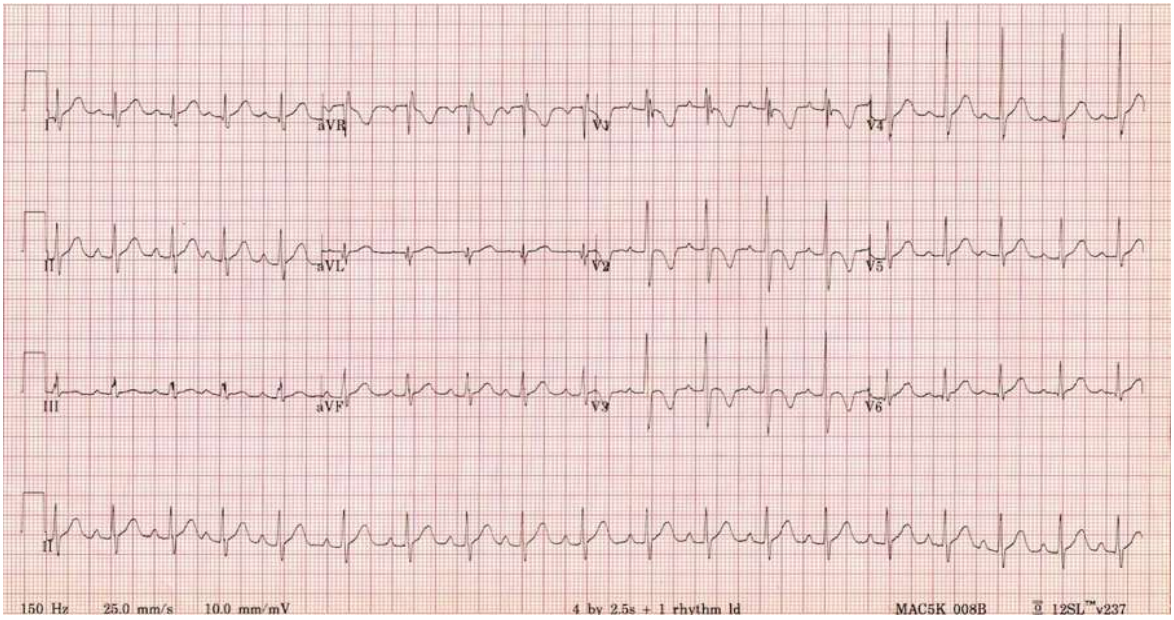


Figure 2: 12-lead ECG of an 8-year-old female illustrates normal paediatric ECGs(It also shows: Heart rate of 110 bpm (normal for age), Juvenile T-wave pattern (T wave inversion in V1-3), Dominant R waves in V1-3, RSR' pattern (partial RBBB morphology) in V1.[8] **Written informed consent was obtained from the patient's guardian for publication of this image.**

ECG recorded using a MAC 5K device with Marquette™ 12SL ECG Analysis Program v237, GE HealthCare.

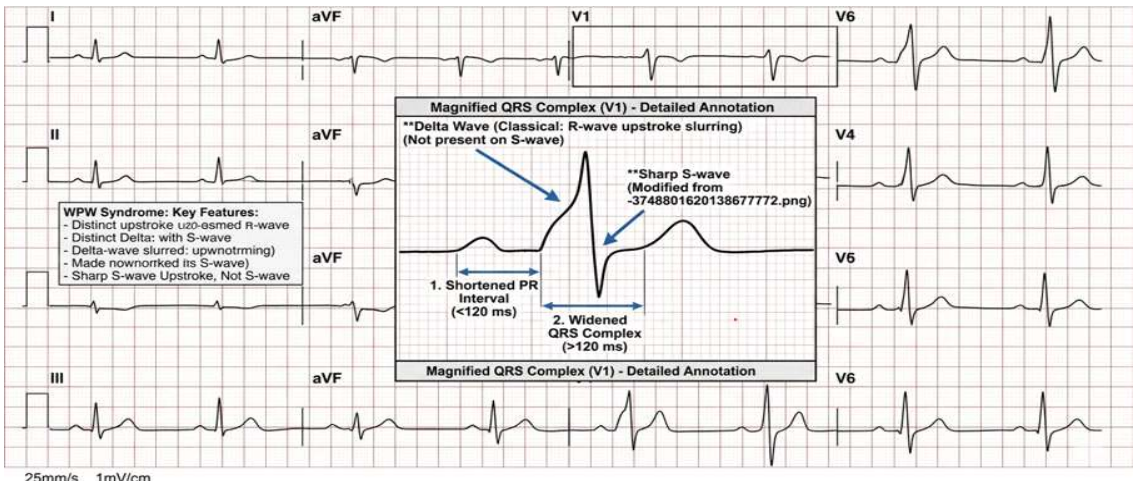


Figure 3a: shows the characteristic delta wave of Wolff-Parkinson-White (WPW) syndrome [Focus on slurred upstroke of the R]. The image is generated with CorelDRAW 13 as a teaching aid based on the information in Park MK [8]

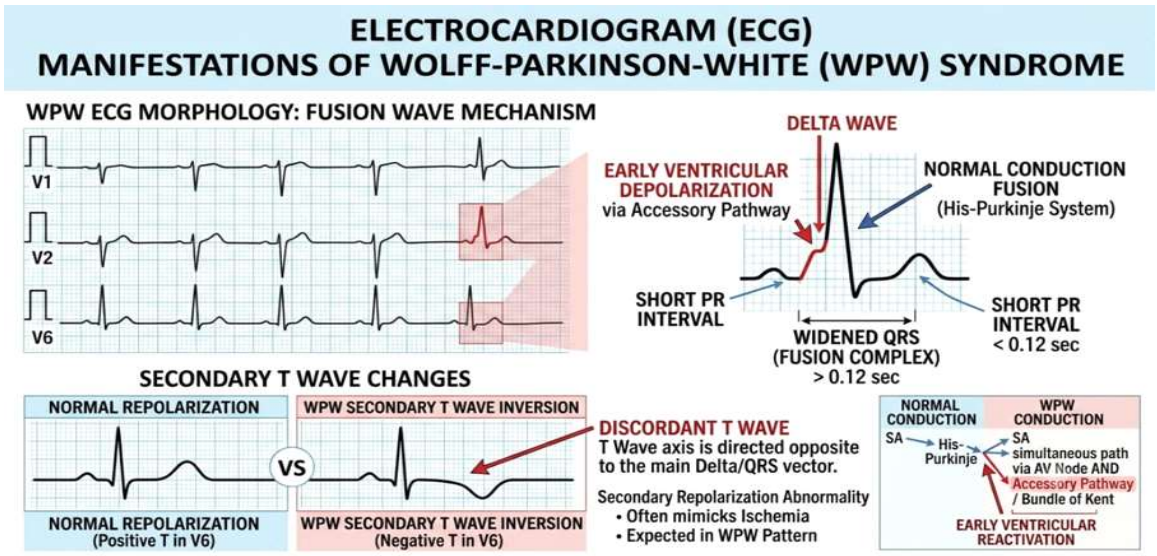


Figure 3b: T wave changes in Wolff-Parkinson-White syndrome. Schematic showing early ventricular activation with fusion of normal and pre-excited conduction leading to early ventricular depolarisation. Figure redrawn by the authors using CorelDRAW 13 based on Park MK.[8]

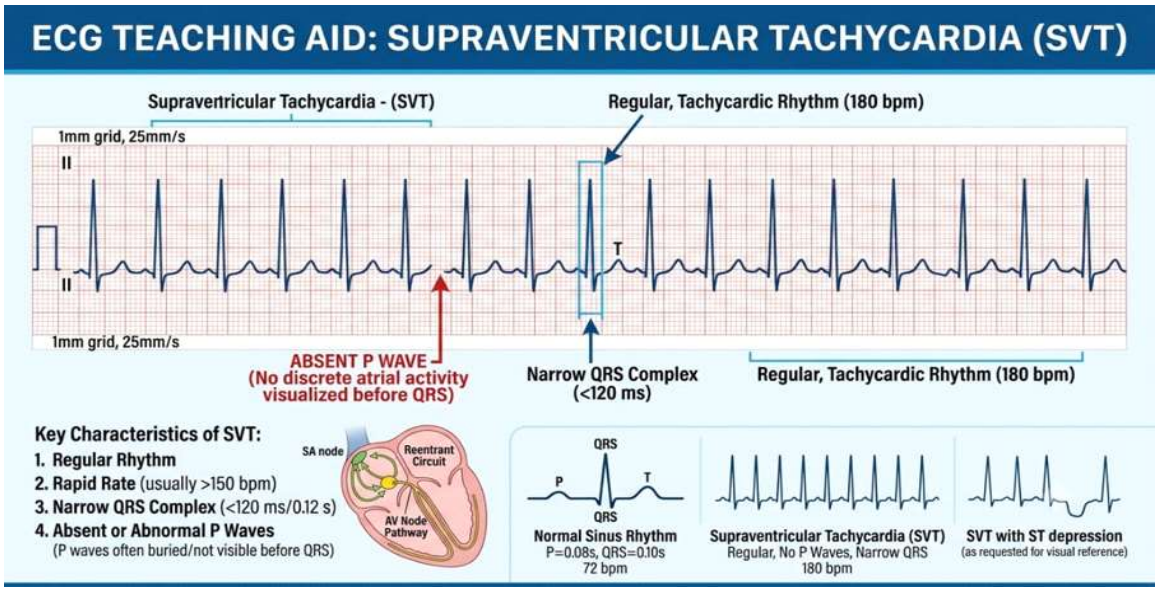


Figure 4 shows narrow QRS complexes and absent P waves seen in supraventricular tachycardia (SVT). Figure redrawn by the authors using CorelDRAW 13 based on Park MK.[8]

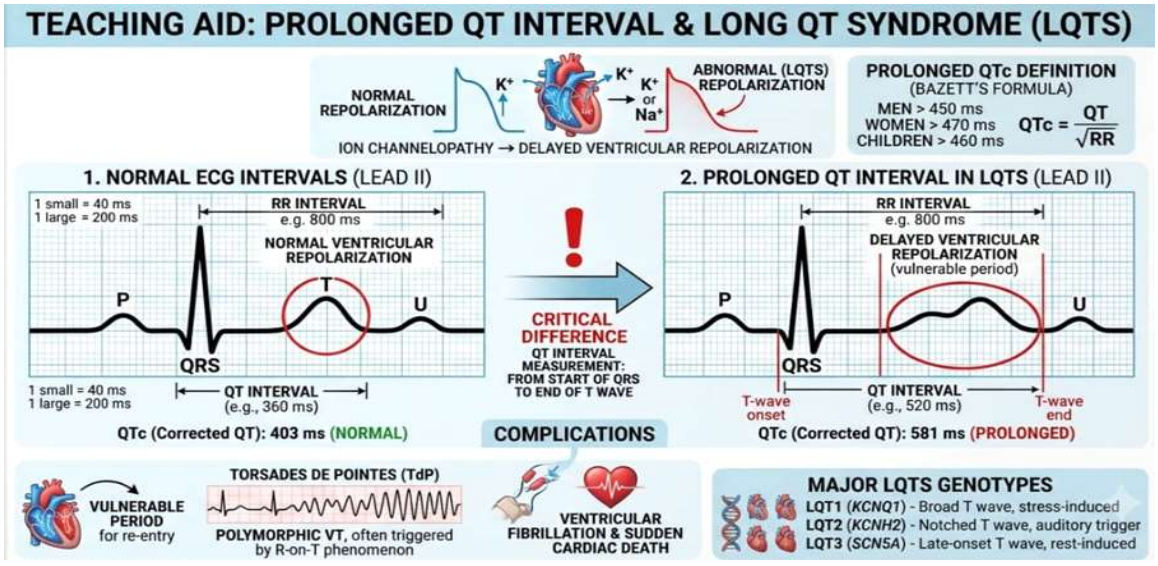


Figure 5 shows a prolonged QT interval in long QT syndrome. The image is generated with CorelDRAW 13 as a teaching aid based on the information in Park MK.[8]

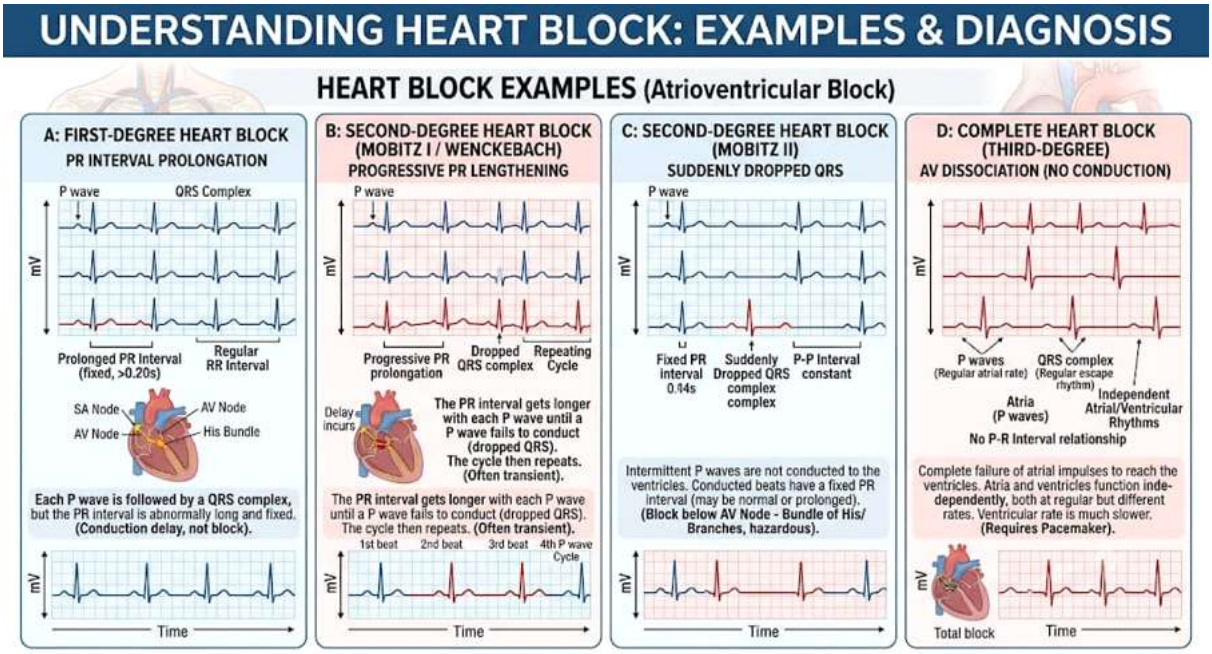


Figure 6 illustrates examples of heart block (first-degree [A], second-degree Mobitz I [B] and II [C], and complete heart block [D]). Figure redrawn by the authors using CorelDRAW 13 based on Park MK.[8]

Supraventricular Tachycardia (SVT)

SVT is the most common symptomatic tachyarrhythmia in children, with an incidence

of approximately 1 in 250–1,000. [20] Most cases are due to accessory pathways (WPW syndrome) or AV nodal re-entry (Figure 4).

ECG Features: Narrow-complex tachycardia (QRS duration normal for age) with a regular, rapid rate. P waves are often absent or difficult to discern. Rates typically exceed **220 bpm in infants** and **180 bpm in older children.** [4,12] Rates **>300 bpm in infants** should prompt consideration of atrial flutter, which may have visible flutter waves. In WPW syndrome, the baseline (sinus rhythm) ECG shows a short PR interval and a delta wave (slurred upstroke of the QRS complex), indicating ventricular pre-excitation.

Clinical Tip: SVT has an abrupt onset and termination, in contrast to sinus tachycardia, which accelerates and decelerates gradually. Haemodynamic status depends on rate, duration, and age; infants may present with poor feeding, irritability, or signs of heart failure, while older children report palpitations or chest tightness.[20]

Ventricular Hypertrophy

Right Ventricular Hypertrophy (RVH): ECG findings include (1) R wave in V1 exceeding the 98th percentile for age; (2) upright T wave in V1 between 1 week and 8 years of age; (3) pure R wave or qR pattern in V1; (4) right axis deviation for age. [12,16] Causes include pulmonary stenosis, tetralogy of Fallot, and pulmonary hypertension.

Left Ventricular Hypertrophy (LVH): Findings include (1) S wave in V1 + R wave in V6 (or V5) > age-specific 98th percentile; (2) R wave in V6 exceeding the 98th percentile; (3) inverted T waves in V5–V6 (strain pattern); (4) left axis deviation. [12,16] A **simplified screening** is R wave in V6 >25 mm in infants or >30 mm in older children, and this should warrant formal voltage calculation. Causes include aortic stenosis, coarctation of the aorta, hypertrophic cardiomyopathy (HCM), and large left-to-right shunts such as ventricular septal defect.

Long QT Syndrome

Congenital long QT syndrome is an important cause of syncope, seizures, and sudden cardiac death in children and young adults. The hallmark ECG finding is a prolonged QTc interval (≥460 ms in children; ≥480 ms in symptomatic patients or those with a strong family history). [18] Additional features may include T-wave alternans or notched T waves. **Manual measurement in lead II or V5 is essential as automated readings are unreliable and may miss up to 40% of cases with prolonged QTc.** [18] Beta-blocker therapy and lifestyle modification are mainstays of treatment, underscoring the importance of accurate identification (Figure 5).

Myocarditis and Pericarditis

These conditions may present with chest pain, fever, and elevated cardiac enzymes. ECG findings in pericarditis include diffuse ST-segment elevation (concave up) and PR-segment depression, which later evolve into T-wave inversion. Myocarditis may produce low-voltage QRS complexes (<5 mm in limb leads), ST-T wave changes, and conduction abnormalities. [2] Low voltage with clinical suspicion of myocarditis or effusion warrants urgent echocardiography.

Arrhythmias Requiring Immediate Attention

- **Ventricular Tachycardia:** Wide QRS tachycardia (QRS duration > upper limit for age) at a rate >120 bpm or 20% faster than sinus. Requires immediate assessment and cardiology involvement.
- **Complete Heart Block:** Atrial rate normal for age but with complete AV dissociation; ventricular escape rate often slow (40–60 bpm in children, slower in infants). May be congenital (associated with maternal lupus) or acquired (post-surgical) (Figure 6).
- **Brugada Pattern:** Coved-type ST elevation in V1–V2 (type 1 Brugada pattern) is rare but important to recognise in children with syncope or a family history of sudden death. [14]

Table 2: Quick Reference for Common Clinical Scenarios

Clinical Scenario	Key ECG Feature to Assess	ECG Findings to Document	Common Diagnoses	Action
Palpitations/ tachycardia	Rate, P waves, QRS width	Rate, regularity, P wave visibility, QRS duration	SVT (narrow complex, no P waves, rate >220 infant/>180 child); Atrial flutter (>300 bpm in infants)	Vagal manoeuvres, adenosine; refer
Syncope (exertional)	QTc, ventricular hypertrophy, pre-excitation	QTc (manual), voltages, delta waves	Long QT, HCM (LVH, deep Q waves), WPW (delta waves)	Urgent cardiology referral
Syncope (non-exertional)	Rhythm, pauses, QTc, Brugada pattern	Baseline rhythm, pauses, QTc, ST in V1-V2	Vasovagal (normal ECG), long QT, Brugada (rare)	Refer if QTc is prolonged or Brugada pattern
Chest pain	ST segments, T waves, hypertrophy	ST elevation/depression, T-wave inversion, voltages	Pericarditis (diffuse ST elevation), normal variant (early repolarisation), HCM (LVH)	Treat cause; refer if abnormal
Murmur	Ventricular voltages, axis	R and S voltages, axis	RVH (RV dominance beyond infancy), LVH (VSD, AS, HCM)	Echocardiogram if suspected
Incidental bradycardia	Rhythm, PR interval, heart rate	Rate, PR interval, AV relationship	Sinus bradycardia (athlete), heart block (AV dissociation)	Check symptoms; refer if AV block
Suspected myocarditis	Voltages, ST-T changes	QRS amplitude, ST segments	Low voltage, ST changes, conduction delay	Urgent echocardiogram; refer

Red Flags and Indications for Referral

While many paediatric ECGs are normal, certain findings mandate discussion with or referral to a paediatric cardiologist:

1. Any ECG from a child with exertional syncope, chest pain, palpitations, or a family history of sudden cardiac death (first-degree relative <50 years) or cardiomyopathy in a family member**,** irrespective of whether the ECG appears normal. [1]
2. Sustained arrhythmias, including SVT, ventricular tachycardia, or complete heart block.
3. **QTc prolongation** (≥460 ms in children, ≥450 ms in infants under 6 months) or QTc shortening (<340 ms).
4. **Ventricular hypertrophy** criteria, particularly if associated with symptoms or abnormal examination.
5. **Pathological Q waves** suggesting myocardial ischaemia or cardiomyopathy.
6. **ST-segment changes** suggestive of ischaemia or pericarditis.
7. Unexplained conduction abnormalities such as left bundle branch block, Mobitz II second-degree block, or advanced first-degree block (PR >200 ms in older children) with symptoms.
8. **Extreme axis deviation** (north-west axis, –90° to ±180°) or left axis deviation in a young infant (< –30°).
9. **Low-voltage QRS** (<5 mm in limb leads) with suspected effusion or myocarditis.
10. **Brugada pattern** (type 1) in any child.

Key Points for Clinicians

- Paediatric ECG interpretation **must be age-adjusted** as normal values for rate, axis, and intervals change continuously from birth through adolescence.
- A **systematic approach** (rate, rhythm, axis, intervals, waves/complexes) reduces the risk of missing clinically significant abnormalities.
- Manual interpretation is recommended over sole reliance on machine-generated reports. At minimum, clinicians should manually verify the heart rate in young children, check for delta waves, and carefully measure the QTc interval. Automated readings are unreliable for QTc measurement; therefore, manual confirmation is essential, as QTc prolongation (defined as ≥460 ms in children and ≥450 ms in infants under 6 months) carries significant clinical implications for the risk of sudden cardiac death.
- **Recognised normal paediatric variants to avoid overdiagnosis**: Right ventricular dominance, juvenile T-wave patterns, and sinus arrhythmia are **normal findings** in children and should not be mistaken for pathology.
- Supraventricular tachycardia (SVT) is the most common paediatric tachyarrhythmia; rates exceeding 220 bpm in infants and 180 bpm in children are highly suggestive; rates >300 bpm in infants should prompt consideration of atrial flutter.
- Any ECG with abnormalities suggestive of structural heart disease, or obtained from a child with exertional symptoms or a concerning family history (sudden death in a first-degree relative <50 years, cardiomyopathy in a family member), warrants **senior cardiology review and management** regardless of the tracing's appearance.
- **Technical quality and clinical context determine**

ECG accuracy: Correct lead placement, minimisation of artefact, and calibration (half or double standardisation) are crucial to interpretation. A normal ECG does not exclude structural heart disease and should never replace comprehensive clinical assessment.

Conclusion

Paediatric ECG interpretation is a core skill for general practitioners and frontline clinicians caring for children. The key to accurate interpretation lies in understanding the profound age-related changes that characterise the normal paediatric ECG and applying a systematic, stepwise approach to analysis. Age-specific normative data are indispensable references and should be readily accessible in all clinical areas where children are assessed. Attention to technical quality, i.e., correct lead placement, recognition of artefact, verification of calibration, and repetition of poor-quality tracings, is an essential prerequisite to accurate interpretation. By recognising both common normal variants and red-flag abnormalities that signal underlying pathology, frontline clinicians can confidently distinguish between benign and life-threatening conditions, ensuring timely specialist referral when indicated. As with all clinical investigations, the ECG must be interpreted within the full context of the history and examination; a normal tracing does not exclude cardiac disease in a symptomatic child, and an abnormal tracing in an asymptomatic child requires careful correlation before decisive action is taken.

Conflict of Interest Statement

The authors declare no conflicts of interest.

Funding: No specific funding was received for this work.

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ORIGINAL ARTICLE

Bacterial and Fungal Isolates from Surgical Site Infections in a Point Prevalence Survey for Healthcare-Associated Infections in Nasarawa State, Nigeria

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Abstract

Background: Surgical site infections (SSIs) are a group of healthcare-associated infections (HAIs) that pose a major public health challenge due to their contribution to increased morbidity, mortality, and increased cost of healthcare. Although bacteria are the usual agents associated with SSIs, fungi are now increasingly associated as well. Over 70% of bacteria associated with healthcare-associated infections are reported to be antimicrobial-resistant. Identifying these pathogens and their antimicrobial susceptibility is important in guiding therapy. This study aimed to determine the prevalence of surgical site infections, identify the associated bacterial and fungal pathogens, and determine their antimicrobial susceptibility patterns.

Methodology: The study was a point prevalence survey conducted among hospitalised patients in public health facilities across the State. The ECDC point prevalence survey protocol for HAIs was adopted for data collection. Data were collected from 208 eligible patients between February and March 2025. Wound swab samples were collected from six patients who met the case definition for SSIs. Samples were processed to isolate and identify bacterial and fungal pathogens and to determine their antimicrobial susceptibility patterns.

Results: The study found an SSI prevalence of 2.9%, constituting 33.3% of all HAIs among hospitalised patients in this study. Both bacteria and fungi were isolated from surgical sites, with *Escherichia coli* and *Aspergillus niger* being the most common bacteria and fungi, respectively. All the bacterial isolates were found to be resistant to several antimicrobial agents, while the fungal isolates had low zones of inhibition to the antifungal agents tested.

Conclusion: The co-occurrence of bacterial and fungal infections in SSI and their resistance to several antibiotics demonstrates the need to have a high index of suspicion for polymicrobial infections in surgical sites to effect appropriate treatment early and reduce the risks associated with HAIs in hospitalised patients

Keywords: Surgical Site Infections, Isolates, Antimicrobial Susceptibility.

INTRODUCTION:

Surgical site infections (SSIs) are a group of healthcare-associated infections (HAIs) that pose a major public health challenge due to their contribution to increased morbidity, mortality, and increased cost of healthcare.[1] Surgical site infections refer to infections occurring in patients within 30 days of receiving healthcare or within a year of an implant being inserted.[2] Most reports show that SSIs are the most common HAIs globally, with the burden higher in developing countries. [3]Surgical site infections are estimated to constitute about 20% of all HAIs in the United States (US) and Europe and cost the US about $900 million and 400,000 extra hospital days annually. [4]Between 2 to 36% of patients who undergo surgery are reported to develop SSIs, depending on the type of procedure, with the highest rates in patients who have undergone orthopaedic procedures. [1] The impact of SSIs mirrors that of HAIs, as the effects are both on the patient and the health system. Surgical site infections,

Date Received - 21 February 2026, Date Accepted - 12 April 2026
Date Published online - 13 August 2026

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NASJM

Published by: MEDICAL AND DENTAL CONSULTANTS' ASSOCIATION OF NIGERIA (MDCAN)
FEDERAL UNIVERSITY TEACHING HOSPITAL, LAFIA, NASARAWA STATE
Citation: ES Audu, A Chuku, GA Obande, SO Bello, AA Audu, Z Amadou et al. Bacterial and Fungal Isolates from Surgical Site Infections in a Point Prevalence Survey for Healthcare-Associated Infections in Nasarawa State, Nigeria *Nasarawa Journal of Medicine* 2026: 3 (1) 11-16

like other HAIs, increase morbidity, healthcare costs, the risk of hospital readmission, disability, and mortality.[2] They also increase healthcare expenditure, workforce demands, staff workload and burnout, antimicrobial use, and the risk of antimicrobial resistance.[1,5,6]

Although bacteria are the usual agents associated with SSIs, fungi are now increasingly associated as well.[7,8] A variety of pathogens have been associated with SSIs, the majority of them being of bacterial and fungal origin. The common bacteria associated with SSIs include Gram-positive organisms, especially members of the genus Staphylococcus: both methicillin-resistant (MRSA) and methicillin-sensitive (MSSA), as well as coagulase-negative Staphylococci. A review report indicates that Staphylococci are the second most common organisms associated with SSIs in Africa. [9] However, most reports suggest that Gram-negative bacilli are the most common cause, with *Escherichia coli* reported as the most common Gram-negative organism associated with SSIs. [10,11] Other common Gram-negative organisms associated with SSIs are *Pseudomonas aeruginosa*, *Proteus mirabilis*, and *Acinetobacter spp*. [11]

Over 70% of all bacteria associated with healthcare-associated infections are reported to be antimicrobial-resistant.[11] Antimicrobial resistance increases the chance of HAIs and other co-morbidities getting worse, and the worsening of the patient's condition leads to prolonged treatment and the need for more antimicrobial agents, predisposing to additional risk of development of resistance. [6]

The commonest fungal pathogens associated with SSIs are Candida spp.[12] Candida infection may occur alone or, in most cases, as a mixed infection with bacteria. [13] Candida is reported to have the ability to form biofilms, hence an important cause of device-associated SSI in patients with implants. [14]

Identifying these pathogens and their antimicrobial susceptibility patterns will guide antibiotic therapy and the implementation of infection prevention and control measures. This study, therefore, aimed to determine the prevalence of healthcare-associated surgical site infections, the associated bacterial and fungal pathogens, and their antimicrobial susceptibility pattern in Nasarawa State, Nigeria.

MATERIALS AND METHODS:

Study location and population: The study was conducted in Nasarawa State, North-central Nigeria, among hospitalised patients in public secondary and tertiary health facilities across the State's three senatorial zones.

Study design: The study was a cross-sectional study, using the point prevalence survey (PPS) design. The European Centre for Disease Control (ECDC) protocol for HAIs and Antibiotic Use was adopted for data collection.[15]

Sample size: The sample size was determined using the formula below for cross-sectional studies, where the variables are qualitative.[16]

$n = z^2 p (1 - p)/d^2$

Where:

n = sample size

z = standard normal variation at 5% ($p < 0.05$), which is 1.96

p = expected prevalence of HAIs in the population based on previous studies is 14.3%.[17,18]

d = precision level determined to be 0.05.

Therefore,

$n = 1.96^2 \times 0.143(1 - 0.143) / 0.05^2$

$n = 3.84 \times 0.12255 / 0.0025$

n = 188.4. If an attrition rate of 10%, (18.84), is added, this was 207.24, giving a minimum sample size of 207. A total of 208 patients from one tertiary and four secondary facilities were included, drawn from one of the two tertiary health facilities and four secondary health facilities across the three senatorial zones of the state.

Sampling method: Stratified random sampling method was used. Of the two tertiary facilities in the state, one was randomly selected. The secondary facilities were stratified into the three senatorial zones in the state. One facility was selected from each zone. When the sample size was not met, an additional facility was randomly selected from the remaining secondary facilities to make up the sample size.

The sampled secondary facilities were General Hospitals Doma, Nassarawa Eggon, Keffi and Obi. While the sampled tertiary facility was Dalhatu Araf Specialist Hospital (DASH), now Federal University Teaching Hospital (FUTH), Lafia.

Ethical consideration: Ethical approval was obtained from the State Health Research Ethics Committee (NHREC Protocol No: 18/06/2017) and the Health Research Ethics Committee of the tertiary health facility (FUTHREC/534). All data was handled confidentially. Consent was waived for data collection from patient's medical records. However, individual consent was sought and obtained from all patients who met the case definition for SSIs before wound swabs were collected.

Data were collected from Patients' medical records into a proforma. All data from the secondary facilities and each inpatient ward of the tertiary facility were collected on the same day.

Survey data collection:

Data was collected over two weeks between February and March 2025. All data from each secondary facility were collected on the same day, and in the tertiary facility, data from each ward were collected on the same day. In all selected health facilities, all hospitalised patients were included, except those admitted after 8 am on the survey date, those in Emergency wards, and those receiving same-day care. Data collection was carried out on working days in all the facilities. Patient data were extracted from patients' medical records (electronic and physical). The case definition for the different types of HAIs was the presence of signs and/or symptoms of the HAI type on the survey date, or a documented diagnosis by a physician, and/or microbiological confirmation. The case definition for surgical site infections (SSI) was based on diagnosis by a clinician, and/or pain, redness, swelling or warmth to touch, and/or purulent discharge or abscess, or spreading cellulitis at the surgical site, or Microbiological (MCB) confirmation.[19,20]

Sample collection and processing:

After obtaining consent from patients who met the case definition for SSIs, wound swab samples were collected from them. The wounds were cleaned with sterile saline, and a sterile cotton swab was gently rotated on the wound. Two swabs were collected from each patient and labelled

appropriately. The samples were transported to the Medical Microbiology Laboratory of the DASH (now FUTH), Lafia. One of the wound swab pairs was inoculated into Blood Agar and MacConkey Agar and incubated at 35-37°C for 24 hours. The second swab pairs were inoculated into Sabouraud Dextrose Agar and incubated at room temperature for up to 2 weeks. The bacterial isolates were identified using colonial morphology, Gram stain, and appropriate biochemical tests in line with standard protocols. [21] The fungal isolates were identified by their growth characteristics, colonial morphology, Germ tube test, and microscopic features in line with standard protocols.[21]

Antimicrobial susceptibility testing:
The Kirby-Bauer method of antimicrobial susceptibility testing was used for both the bacterial and fungal isolates. Mueller-Hinton Agar and modified Mueller-Hinton Agar were used for bacterial and fungal isolates, respectively, in line with standard methods.[22] The antibiotic discs used were Ceftazidime, Cefuroxime, Erythromycin, and Ceftriaxone at 30 µg, Levofloxacin, Amoxicillin, Augmentin, and Rifampicin at 20 µg, and Streptomycin, Gentamicin, Pefloxacin, and Azithromycin at 10 µg. The zones of inhibition were measured and interpreted in accordance with the Clinical and Laboratory Standards Institute (CLSI) interpretative charts.[23] Staphylococcus aureus CB008 and Escherichia coli NCTC CB002 were used as controls. The Antifungal discs used for fungal isolate susceptibility testing were Fluconazole (25 µg), Itraconazole (10 µg), Griseofulvin, and Nystatin. While the susceptibility tests against bacterial pathogens were interpreted in line with the CLSI guidelines, the antifungal susceptibility tests are only descriptive.

Statistical analysis
Data were entered and analysed using SPSS software. A descriptive statistical analysis was conducted for all variables. Categorical variables, i.e., age, sex, facility types, bacterial and fungal isolates, were presented as frequencies and percentages, while continuous variables were summarised using measures of central tendency and dispersion. The age of study participants was reported as mean and standard deviation (SD). Age was further categorised into age groups for analysis of SSI distribution. The overall prevalence of surgical site infection (SSI) was calculated as the number of patients with SSI divided by the total number of hospitalised patients surveyed. Antimicrobial susceptibility patterns for both bacterial and fungal isolates were reported as the number and percentage of isolates that were resistant, intermediate, or susceptible to each tested agent, based on CLSI/EUCAST breakpoints.

RESULTS: The study participants were aged from less than one to over 70 years with a mean age of 32 ± 19.8 years. The majority of them were from the tertiary health facility (73.1%) and female (59.6%) as shown in Table 1.

Five of the six patients who had SSI were males. Two patients with SSI each were aged between 21 and 30 years and 51 years and older, respectively, while the remaining two were less than 20 years old.

The overall prevalence of surgical site infection (SSI) among the 208 hospitalised patients in this point prevalence survey for healthcare-associated infections was 2.9%. Surgical site infections were the most common type of HAI, constituting 33.3% of all the HAIs. (Table 2)

Bacterial growth was detected in five (83.3%) of the six SSI specimens. *Escherichia coli* was the predominant isolate (3/6, 50.0%), followed by *Klebsiella pneumoniae* and *Staphylococcus aureus* (1/6, 16.7% each), Table 3. In addition, fungal growth was detected in four (66.7%) specimens. *Aspergillus niger* was the most frequently isolated fungus (2/6, 33.3%), while *Aspergillus fumigatus* and *Candida albicans* were each isolated from one specimen (16.7%). No fungal growth was observed in two specimens (33.3%).

Antimicrobial susceptibility testing results
Results of susceptibility testing of both the bacterial and fungal isolates to conventional antibacterial and antifungal agents showed a high level of resistance by all the isolates.

Antibacterial susceptibility testing
As shown in Table 4, all bacterial isolates exhibited multidrug resistance. All *Escherichia coli* isolates were resistant to Azithromycin, Erythromycin, Amoxicillin, Cefotaxime, Rifampicin, Ciprofloxacin, Cefuroxime, Gentamicin, Ceftazidime, Ceftriaxone, and Augmentin, while two were also resistant to Pefloxacin. *Klebsiella pneumoniae* demonstrated resistance to a similar range of antibiotics. The *Staphylococcus aureus* isolate was resistant to penicillins, aminoglycosides, and macrolides. However, all bacterial isolates remained susceptible to streptomycin. In addition, all isolates from the tertiary facility were susceptible to levofloxacin, whereas both isolates from the secondary facility were resistant.

Antifungal susceptibility testing
All fungal isolates exhibited inhibition zones ranging from 0 to 18 mm against the antifungal agents tested (Table 5). *Candida albicans* was resistant to all antifungal agents tested. *Aspergillus niger* showed inhibition zones of 10–18 mm with ketoconazole, Fluconazole, and Griseofulvin, whereas *Aspergillus fumigatus* was resistant to all agents except nystatin.

Table 1. Demographic and facility characteristics of study participants

Characteristic		Frequency	Percentage (%)
Age (years)			
Mean age 32.3 ± 19.8 years			
	0 – 10	33	15.9
	11 – 20	25	12.0
	21 – 30	48	23.1
	31 – 40	38	18.3
	41 – 50	26	12.5
	> 51	38	18.3
Gender			
	Male	84	40.4
	Female	124	59.6
Facility type			
	Tertiary	152	73.1
	Secondary	56	26.9

Table 2: Types of Healthcare -associated infections among study participants

Characteristics	Frequency n (N=208)	Percentage (%)
Bloodstream Infections	1	0.5
Gastrointestinal infections (Diarrhoea)	1	0.5
Neonatal sepsis	5	2.4
Surgical site infection	**6**	**2.9**
Skin and soft tissue infections	4	1.9
Urinary Tract Infection	1	0.5

Table 3: Bacterial and fungal isolates from Surgical Site Infections among the participants

Isolates	n	Percentage
Bacterial		
Escherichia coli	3	50.0
Klebsiella pneumoniae	1	16.7
Staphylococcus aureus	1	16.7
No bacterial growth	1	16.7
Fungi		
Aspergillus niger	2	33.3
Aspergillus fumigatus	1	16.7
Candida albicans	1	16.7
No fungal growth	2	33.3

Table 4: Antibacterial susceptibility patterns of bacterial isolates from SSI in Nasarawa state.

Patient ID	Levofloxacin	Azithromycin	Erythromycin	Amoxicillin	Ciprofloxacin	Ceftazidime	Cefuroxime	Rifampicin	Streptomycin	Gentamicin	Pefloxacin	Ceftriaxone	Cefoxitin	Augmentin
	Zone of inhibition in millimeters													
F1	**26**	0	0	0	**28**	0	0	0	18	0	0	0	9	0
F2	**25**	0	0	0	**25**	0	0	0	20	0	**18**	0	0	0
F3	**24**	0	0	0	0	0	0	0	22	0	0	0	8	0
K1	0	0	0	0	0	0	0	0	12	0	7	0	0	0
K2	0	12	0	0	0	0	0	0	23	0	19	0	0	0
CLSI (E)	≥17	*	≥23	≥19	≥21	≥21	≥16	NA	≥15	≥16	≥15	≥13	*	≥19
CLSI (S)	≥21	*	≥15	≥19	≥21	≥17	*	≥20	NA	≥16	*	*	≥21	≥25

*No official breakpoint F: Tertiary facility; K- Secondary facility

Keys: F1- *Escherichia coli*; F2- *Escherichia coli*; F3- *Klebsiella spp*; K1- *Escherichia coli*; K2- *Staphylococcus aureus*; S- Susceptible; I- Intermediate; R- Resistant; NA-Not applicable. CLSI (E)- standard zone for Enterobacteriaceae; CLSI (S)- standard zone for Staphylococci

Table 5: Antifungal susceptibility of fungal isolates from SSI in Nasarawa state.

Patient ID	Isolate	Itraconazole	Nystatin	Ketoconazole	Fluconazole 25 µg
F1	*Aspergillus niger*	12	18	0	0
F2	*Aspergillus niger*	15	0	0	0
F3	*Aspergillus fumigatus*	0	10	0	0
F4	*Candida albicans*	0	0	0	0
CLSI standard		**			*≥19

*CLSI breakpoints for Candida albicans ** No CLSI breakpoints for disk diffusion for *Candida spp* and *Aspergillus spp*.[22]

DISCUSSION

This study found the prevalence of surgical site infections to be 2.9% among hospitalised patients, representing 33.3% of all healthcare-associated infections. This estimate is comparable to the 3.7% previously reported by Ikrama *et al*.[24] and is consistent with the finding from another study that used similar methodology.[18] However, it is substantially lower than estimates reported in many other studies, including the 14.5% prevalence reported by Olowo-Okere *et al*. in a 2019 systematic review and meta-analysis.[25]

The study found the SSIs were polymicrobial, with both bacteria and fungi isolated from the surgical sites. *Escherichia coli* was the most prevalent bacterium, followed by *Staphylococcus aureus* and *Klebsiella pneumoniae.* This finding is consistent with a similar hospital-based study conducted in North-Central Nigeria.[24] However, the finding contrasts with other reports, which found the predominant SSI-associated bacteria to be *Staphylococcus aureus.*[26,27] Although Aspergillus species were the most common fungi isolated in this study, this differs from other reports in which Candida species were the predominant SSI-associated fungi. However, the small sample size limits the generalizability of this finding and may not accurately represent the true fungal profile of SSIs.[14,27]

All the bacterial and fungal isolates in this study displayed resistance to multiple antimicrobial agents. The bacterial isolates, including *Escherichia coli, Staphylococcus aureus,* and *Klebsiella pneumoniae,* all showed resistance to more

than three classes of antibiotics. This finding aligns with other reports in Nigeria and globally, where healthcare-associated pathogens are increasingly becoming multidrug-resistant.[28,29] However, our findings are limited by the small sample size, and the interpretation of the findings may not be a true representation of the antimicrobial susceptibility pattern of all pathogens associated with surgical site infections.

Although the fungal isolates in this study had low sensitivity to the antifungal agents tested, including the Imidazole, Triazoles, Polyenes, and Heterocyclic Benzofurans, this result does not necessarily indicate resistance because the method used for the antifungal susceptibility did not have validated CLSI disc diffusion breakpoints for the organisms and the antifungal agents tested.

Other reports, such as those by Ejembi *et al*[5] and Abban *et al*,[7] who previously reported increasing resistance by healthcare-associated fungal isolates. However, the antifungal susceptibility findings in this study should be interpreted with caution because of the small sample size and the limited range of antifungal agents tested. Although the high level of multidrug resistance observed among the bacterial isolates is concerning because of its potential impact on patient outcomes and healthcare costs, these findings require confirmation in larger studies to better characterize antimicrobial resistance patterns and inform strategies for improving the management of surgical site infections.

Conclusion: This study found an SSI prevalence of 2.9% in Nasarawa state, Nigeria from a point prevalence study. The co-occurrence of both bacteria and fungi in surgical sites is a finding that should influence decisions regarding the care and treatment of SSIs. While *Escherichia coli* and *Aspergillus species* were the most common bacterial and fungal pathogens in this study, all the bacterial isolates demonstrated multidrug resistance. There is a need for regular microbiological testing and guidance, and the need to strengthen antimicrobial stewardship programs in all public health facilities. Furthermore, the strengthening of infection prevention and control programs in public health facilities will reduce the burden of surgical site infections with their associated consequences.

Limitations: This study used a point prevalence design and may not fully represent pathogens associated with SSIs, as it excluded post-discharge infections and patients with short hospital stays. The small sample size for pathogen identification also limits the generalizability of the findings to Nasarawa State.**Conflict of interest**: The authors declare no conflict of interest.

Source of funding: No external funding was received for this study.

Author's contributions:
ESA conceived the study, developed the theoretical framework, implemented the research, and drafted the manuscript. AC, AAA, GAO, and ZA contributed to the study design and implementation. SOB, ZA, LYW, AAA, and MH contributed to the design, implementation, and data analysis. All authors discussed the findings and contributed to the final manuscript.

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ORIGINAL ARTICLE

Pattern and Outcome of Congenital Heart Diseases at the Emergency Paediatric Unit of a Tertiary Hospital in Azare, North-Eastern Nigeria

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Abstract

Background: Congenital heart diseases (CHD) significantly impact childhood health in low-resource settings. This study aimed to assess the hospital-based proportion, clinical patterns, complications, and outcomes of CHD among children seen at the Emergency Paediatric Unit of the Federal University of Health Sciences Teaching Hospital, Azare, North-Eastern Nigeria, over one year.

Materials and Methods: A retrospective analysis was conducted on children aged 1 month to 15 years with echocardiographically confirmed CHD from January 1 to December 31, 2024. Data were collected from medical records and analysed using IBM SPSS version 25. Binary logistic regression identified mortality predictors, with statistical significance set at $p<0.05$.

Results: Of 2,973 admissions, 42 (1.41%) had confirmed CHD. Median presentation age was 9 months (IQR: 3-26), with male predominance (1.2:1). Commonest lesions included ventricular septal defect (33.3%), tetralogy of Fallot (21.4%), and patent ductus arteriosus (14.3%). Complications featured heart failure (50.0%), severe pneumonia (31.0%), and hypoxic spells (14.3%). Overall mortality was 35.7% (15/42), with 80% of deaths in infants. Only 7.1% accessed a surgical referral. Independent mortality predictors were age <6 months (AOR: 3.41, 95%CI: 1.38-8.42, p=0.008), delayed presentation (AOR: 2.89, 95%CI: 1.18-7.06, p=0.021), heart failure at presentation (AOR: 3.92, 95%CI: 1.54-9.97, p=0.004), complex CHD (AOR: 4.56, 95%CI: 1.72-12.08, p=0.002), and lack of surgical referral (AOR: 7.23, 95%CI: 1.51-34.62, p=0.013).

Conclusions: CHD significantly contributes to paediatric morbidity and mortality in this region. Many infants present late and face substantial barriers to surgical care. Enhancing early detection and improving access to cardiac services are essential to reducing mortality.

Keywords: Congenital heart disease, morbidity, mortality, emergency unit, Nigeria, Sub-Saharan Africa

Introduction

Congenital heart diseases (CHD) represent the most common group of congenital anomalies globally, affecting 8-12 per 1,000 live births.[1] These structural malformations range from simple lesions that may close spontaneously to complex anomalies requiring timely surgical intervention.[2] CHD accounts for nearly one-third of all major congenital anomalies and remains a leading cause of infant mortality worldwide.[3]

An estimated 1.35 million newborns are born with CHD annually, with the highest burden concentrated in low- and middle-income countries (LMICs).[4] Sub-Saharan Africa bears a disproportionate share, where limited diagnostic facilities, shortage of paediatric cardiologists, and absence of cardiac surgical services result in delayed diagnosis and

Date Received- 1 April 2026, Date Accepted- 28 June 2026
Date Published online- 25 August 2026

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Published by: MEDICAL AND DENTAL CONSULTANTS' ASSOCIATION OF NIGERIA (MDCAN)
FEDERAL UNIVERSITY TEACHING HOSPITAL, LAFIA, NASARAWA STATE
Citation: Ibrahim BA, Yusuf MO, Bello SO, Pius S, Audu LI, Buba F. Pattern and Outcome of Congenital Heart Diseases at the Emergency Paediatric Unit of a Tertiary Hospital in Azare, North-Eastern Nigeria *Nasarawa Journal of Medicine* 2026:3(1) 17-23

poor outcomes.[5] The region's health systems, already strained by infectious diseases, are not well equipped to address the growing recognition of CHD as a significant contributor to childhood mortality.[6]

Nigeria, with Africa's largest population, lacks comprehensive national data on CHD. Hospital-based reports from Southern and Western Nigeria suggest CHD constitutes 10-20% of paediatric cardiology consultations, with ventricular septal defect (VSD) being most frequently reported.[7-9] However, data from North-Eastern Nigeria are scarce, a region characterised by significant healthcare challenges including health worker shortages, infrastructure deficits, and recurrent humanitarian crises.[10]

Children with undiagnosed or late-diagnosed CHD frequently present to emergency paediatric units with life-threatening complications: heart failure, severe pneumonia, hypoxic spells, and infective endocarditis.[11] The emergency unit serves as the frontline for managing these acute presentations, where timely recognition and appropriate intervention can be lifesaving. Yet, in resource-constrained settings, the absence of echocardiography, limited availability of paediatric cardiologists, and lack of surgical referral pathways contribute to missed diagnoses and preventable deaths.[12]

A recent study from our centre documented overall paediatric morbidity and mortality patterns in 2024, identifying severe malaria, malnutrition, and pneumonia as leading causes of admission and death.[13] However, that study did not specifically examine the burden of CHD. Understanding the current pattern and outcome of CHD in our emergency unit is essential to highlight the hidden burden of cardiovascular disease, inform resource allocation, guide health worker training, and provide baseline data for advocacy and policy development.

Sustainable Development Goal (SDG) 3.2 aims to end preventable deaths of newborns and children under five by 2030.[14] Addressing congenital anomalies, including CHD, is integral to achieving this target. Furthermore, the World Health Assembly resolution on birth defects (2010) urged member states to develop surveillance systems and strengthen capacity for prevention and care of congenital anomalies.[15]

This study aimed to describe the hospital-based proportion, clinical patterns, echocardiographic findings, complications, and outcomes of congenital heart diseases among children presenting to the emergency paediatric unit at the Federal University of Health Sciences Teaching Hospital, Azare, North-Eastern Nigeria, during the 2024 calendar year.

Materials and Methods

Study Design and Setting

This retrospective cross-sectional study was conducted at the Emergency Paediatric Unit (EPU) of Federal University of Health Sciences Teaching Hospital, Azare (FUHSTHA), located in Katagum Local Government Area, Bauchi State, North-Eastern Nigeria. The hospital is a tertiary facility receiving referrals from primary and secondary health centres across Bauchi, Jigawa, and Yobe States, serving an estimated population of 3-4 million people.

The EPU is a 30-bed ward managing children aged 1 month to 15 years with emergent medical conditions. Staffing includes nurses, medical officers, paediatric residents, and consultant paediatricians on a 24-hour rotational basis. Children are triaged, stabilised, and either discharged, transferred to general paediatric wards, or referred elsewhere for specialised care. The unit recorded 2,973 admissions in 2024.[13]

Paediatric cardiology services at FUHSTHA remain limited. Echocardiography became available in 2023 through a single non-portable machine operated by a paediatrician with echocardiography training, with an adult cardiologist available for older patients (>15 years). No cardiothoracic surgeon is on staff, and children requiring surgical intervention are referred to centres in Southern Nigeria or abroad, which is financially prohibitive for most families.

Study Population

All children aged 1 month to 15 years who presented to the EPU from January 1 to December 31, 2024, and had echocardiographically confirmed CHD were included.

Inclusion and exclusion criteria:

The study included children aged 1 month to 15 years with echocardiographically confirmed congenital heart disease (CHD) whose medical records were available for review. Patients who were brought in dead, had suspected but unconfirmed CHD, or had acquired heart diseases (including rheumatic heart disease, myocarditis, and pericarditis) were excluded. Records that were absent or grossly incomplete, defined as having more than 5% missing data on key study variables, were also excluded.

Data Collection

A structured proforma was developed to extract information from patients' medical records, admission registers, and echocardiography records. Two senior paediatric residents trained as research assistants participated in data extraction. The lead researcher reviewed all entries for completeness and consistency. Medical records with incomplete data were excluded.

Data collected included:

1. **Demographic characteristics:** Age, gender, residence, caregiver's education, referral status
2. **Clinical presentation:** Presenting complaints, age at symptom onset, age at diagnosis, delayed presentation (>6 months from symptom onset)[24]
3. **Examination findings:** Vital signs, oxygen saturation, cyanosis, clubbing, heart murmurs, hepatomegaly, heart failure (modified Ross criteria)
4. **Echocardiographic findings:** CHD type (acyanotic/cyanotic, simple/complex), associated anomalies, pulmonary hypertension, ventricular dysfunction
5. **Complications at presentation:** Heart failure, severe pneumonia, hypoxic spells, infective endocarditis, arrhythmias, cerebrovascular accident
6. **Management and outcome:** Medical therapy, surgical referral, hospital stay duration, outcome (discharged, died, DAMA, absconded, lost to follow-up)

Variables and Definitions

- **Age groups:** Infants (<1 year), toddlers/under five (1-4.9 years), school age (5-12 years), adolescents (13-15 years)
- **CHD classification** based on echocardiography:
  - *Acyanotic CHD:* Left-to-right shunts (VSD, ASD, PDA, AVSD) and obstructive lesions (pulmonary stenosis, aortic stenosis, coarctation)
  - *Cyanotic CHD:* Tetralogy of Fallot, transposition of

great arteries, tricuspid atresia, pulmonary atresia, total anomalous pulmonary venous connection, truncus arteriosus, Ebstein's anomaly, double outlet right ventricle

- *Complex CHD:* Lesions requiring surgical intervention in infancy (within the first year of life) or those with multiple associated intracardiac defects regardless of age, including TOF, TGA, DORV, truncus arteriosus, pulmonary atresia, tricuspid atresia, AVSD, and severe VSD with pulmonary hypertension.[2]

- **Delayed presentation:** Presentation >6 months after symptom onset.[24]
- **Heart failure:** Modified Ross criteria requiring tachypnoea, tachycardia, tender hepatomegaly, and cardiomegaly (clinically or on chest radiograph)
- **Hypoxic spell:** Acute episode of cyanosis and hyperpnoea in cyanotic CHD
- **Lost to follow-up:** Failure to return within 1 month for a scheduled appointment after discharge

Ethical Considerations

Ethical approval was obtained from the Health Research Ethics Committee of FUHSTHA (Approval number: NHREC/FUHSTHA/11/11/24). Individual informed consent was waived for this retrospective record review. Patient information was anonymised and handled confidentially in accordance with the Declaration of Helsinki.

Data Analysis

Data were entered into Microsoft Excel® 2016 and analysed using IBM SPSS version 25. Continuous variables were tested for normality using the Shapiro-Wilk test. Normally distributed variables were expressed as mean ± standard deviation (SD), skewed variables as median with interquartile range (IQR). Categorical variables were presented as frequencies and percentages.

Categorical comparisons were performed using Pearson's chi-square test or Fisher's exact test (where any expected cell count was <5). Factors associated with mortality were analysed using binary logistic regression to estimate odds ratios (ORs) and 95% confidence intervals (CIs). Variables with $p<0.2$ in univariate analysis were entered into a multivariate logistic regression using forward stepwise selection (likelihood ratio) to identify independent predictors of mortality. Model fit was assessed using the Hosmer-Lemeshow goodness-of-fit test. Statistical significance was set at $p<0.05$.

Given the small sample size (n=42, 15 mortality events), the multivariate model was limited to 5 predictors to avoid overfitting, in line with the recommended maximum of 1 predictor per 3-5 events.

Results

Demographic Characteristics

During the one-year study period (January 1 to December 31, 2024), 2,973 children were admitted to the EPU. Of these, 42 (1.41%) had echocardiographically confirmed CHD.

Median age at presentation was 9 months (IQR: 3-26). Infants (<1 year) constituted 29 (69.0%) of cases; toddlers/under five (1-4.9 years) 9 (21.4%); school-age (5-12 years) 3 (7.1%); and adolescents (13-15 years) 1 (2.4%). Males numbered 23 (54.8%) and females 19 (45.2%), giving a male: female ratio of 1.2:1.

Most children (38, 90.5%) resided in Bauchi State, with the remainder from Yobe (3, 7.1%) and Jigawa (1, 2.4%). Rural dwellers accounted for 32 (76.2%), while 10 (23.8%) lived in urban/semi-urban areas. Direct home referrals numbered 25 (59.5%), while 17 (40.5%) were referred from other facilities. Caregivers with no formal education comprised 23 (54.8%), while 10 (23.8%) had primary, 6 (14.3%) secondary, and 3 (7.1%) tertiary education. Table 1 summarises demographic characteristics.

Clinical Presentation

Median age at symptom onset was 3 months (IQR: 1-14), while median age at CHD diagnosis was 9 months (IQR: 3-26), indicating a median diagnostic delay of 6 months (IQR: 2-12). Delayed presentation (>6 months after symptom onset) occurred in 26 (61.9%) children.

The commonest presenting complaints were difficulty breathing (33, 78.6%), recurrent respiratory infections (23, 54.8%), and poor weight gain/failure to thrive (20, 47.6%). Table 2 presents complete clinical features.

Table 1: Demographic Characteristics of Children with CHD (N=42)

Characteristic	Frequency n (%)
Age group	
<1 year	29 (69.0)
1-4.9 years	9 (21.4)
5-12 years	3 (7.1)
13-15 years	1 (2.4)
Gender	
Male	23 (54.8)
Female	19 (45.2)
Residence state	
Bauchi	38 (90.5)
Yobe	3 (7.1)
Jigawa	1 (2.4)
Settlement	
Rural	32 (76.2)
Urban/Semi-urban	10 (23.8)
Referral status	
Brought directly from home	25 (59.5)
Referred from another facility	17 (40.5)
Caregiver education	
No formal education	23 (54.8)
Primary	10 (23.8)
Secondary	6 (14.3)
Tertiary	3 (7.1)

Table 2: Clinical Presentation (N=42)

Clinical Feature	Frequency n (%)
Difficulty breathing	33 (78.6)
Recurrent respiratory infections	23 (54.8)
Poor weight gain/failure to thrive	20 (47.6)
Cyanosis	16 (38.1)
Poor feeding	15 (35.7)
Easy fatiguability	12 (28.6)
Febrile illness	9 (21.4)
Cough	8 (19.0)
Diarrhoea/vomiting	6 (14.3)
Seizures	3 (7.1)
Body swelling	2 (4.8)
Palpitations	1 (2.4)

Median oxygen saturation at presentation was 87% (IQR: 74-94%). Cyanosis was clinically evident in 16 (38.1%), and clubbing in 9 (21.4%). Heart murmurs were audible in 38 (90.5%): pansystolic (18, 42.9%), ejection systolic (12, 28.6%), and continuous (5, 11.9%). Hepatomegaly (≥2 cm) occurred in 22 (52.4%), and heart failure (modified Ross criteria) in 21 (50.0%).

Echocardiographic Findings

Ventricular septal defect was the commonest lesion (14, 33.3%), followed by tetralogy of Fallot (9, 21.4%) and patent ductus arteriosus (6, 14.3%). Table 3 presents the complete CHD spectrum.

Acyanotic CHD comprised 27 (64.3%) cases; cyanotic CHD, 15 (35.7%). Among acyanotic lesions, left-to-right shunts predominated (25, 92.6%), while obstructive lesions accounted for 2 (7.4%). Among cyanotic lesions, TOF was most frequent (9, 60.0%).

Complex CHD occurred in 18 (42.9%) children. Associated extracardiac anomalies were identified in 5 (11.9%): Down syndrome (2, 4.8%), other chromosomal anomalies (1, 2.4%), and musculoskeletal anomalies (2, 4.8%). Pulmonary hypertension was present in 15 (35.7%), with 10 (23.8%) having moderate-severe elevation. Ventricular dysfunction (ejection fraction <50%) occurred in 6 (14.3%).

Complications at Presentation

Complications were present in 35 (83.3%) children at presentation (Table 4). Heart failure was most frequent (21, 50.0%) and was more common in acyanotic lesions (17, 81.0% of heart failure cases). Severe pneumonia occurred in 13 (31.0%), predominantly in infants <6 months (9, 69.2%). Hypoxic spells affected 6 (14.3%), all with cyanotic CHD (TOF, 5; DORV, 1).

Table 3: Pattern of Congenital Heart Disease (N=42)

Type of CHD	Frequency n (%)
Acyanotic CHD	**27 (64.3)**
Ventricular septal defect (VSD)	14 (33.3)
Patent ductus arteriosus (PDA)	6 (14.3)
Atrial septal defect (ASD)	4 (9.5)
Pulmonary stenosis (PS)	2 (4.8)
Atrioventricular septal defect (AVSD)	1 (2.4)
Cyanotic CHD	**15 (35.7)**
Tetralogy of Fallot (TOF)	9 (21.4)
Transposition of great arteries (TGA)	2 (4.8)
Double outlet right ventricle (DORV)	1 (2.4)
Truncus arteriosus	1 (2.4)
Pulmonary atresia	1 (2.4)
Tricuspid atresia	1 (2.4)

Table 4: Complications at Presentation by CHD Type

Complication	Acyanotic CHD (n=27)	Cyanotic CHD (n=15)	Total (N=42)	p-value*
Heart failure	17 (63.0%)	4 (26.7%)	21 (50.0%)	0.028
Severe pneumonia	10 (37.0%)	3 (20.0%)	13 (31.0%)	0.043
Hypoxic spells	0 (0%)	6 (40.0%)	6 (14.3%)	<0.001
Pulmonary hypertension	12 (44.4%)	3 (20.0%)	15 (35.7%)	0.032
Failure to thrive	9 (33.3%)	11 (73.3%)	20 (47.6%)	0.016
Infective endocarditis	0 (0%)	1 (6.7%)	1 (2.4%)	0.357
Arrhythmias	1 (3.7%)	1 (6.7%)	2 (4.8%)	0.584
Cerebrovascular accident	0 (0%)	1 (6.7%)	1 (2.4%)	0.357

**Fisher's exact test was used for all comparisons*

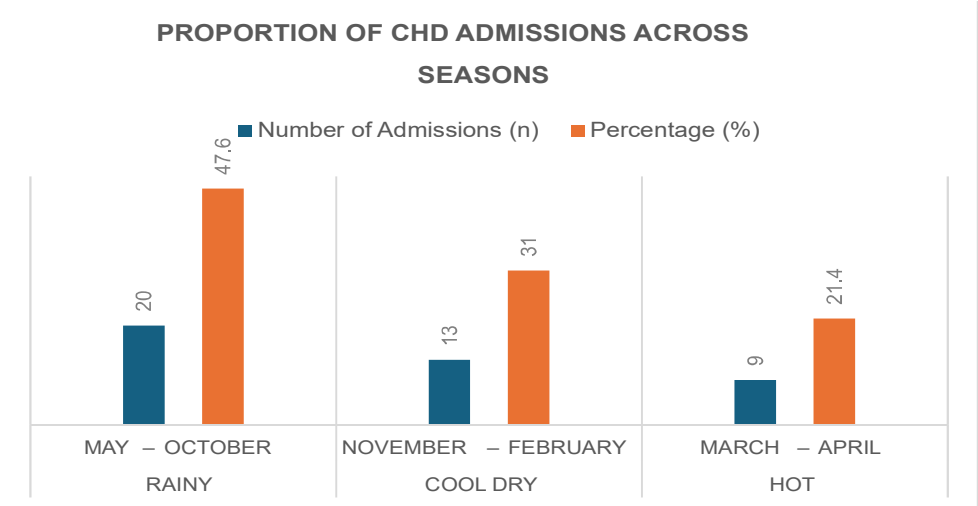


Figure 1: Seasonal Variation of Admissions

Seasonal Variation of Admissions

CHD admissions showed seasonal variation (Figure 1). Of the 42 cases, 20 (47.6%) occurred during the rainy season (May-October), 13 (31.0%) during the dry season (November-February), and 9 (21.4%) during the hot season (March-April). This pattern aligns with overall EPU admission trends reported previously.[13]

Management and Outcomes

Medical management: All 42 children received medical therapy: antibiotics (35, 83.3%), oxygen (26, 61.9%), diuretics (21, 50.0%), digoxin (10, 23.8%), and iron/folate (18, 42.9%). Hypoxic spells were managed with knee-chest position, morphine, oxygen, and propranolol.

Surgical referral: Only 3 (7.1%) children accessed surgical referral. One underwent surgery (VSD closure) in Southern Nigeria; 2 were awaiting surgery at the time of data collection. The remaining 39 (92.9%) did not access surgical care due to financial constraints (32, 82.1%), lack of referral centres within the country (4, 10.3%), or caregiver refusal (3, 7.7%).

Hospital stay: Median duration was 8 days (IQR: 4-15). Sixteen (38.1%) stayed <7 days, 19 (45.2%) 7-14 days, and 7 (16.7%) >14 days. Prolonged stay was associated with complications (p=0.004) and the need for intensive care (p=0.002).

Outcomes: Of 42 children, 24 (57.1%) were discharged home on medical therapy, 15 (35.7%) died during admission, 2 (4.8%) were discharged against medical advice (DAMA), and 1 (2.4%) absconded. Among 15 deaths, 12 (80.0%) occurred in infants, and 10 (66.7%) in children with complex cyanotic CHD.

Documented causes of death included refractory heart failure (7, 46.7%), severe pneumonia with respiratory failure (4, 26.7%), hypoxic spells (2, 13.3%), and sepsis (2, 13.3%).

Follow-up: Of 24 discharged children, only 9 (37.5%) returned for at least one follow-up visit; 15 (62.5%) were lost to follow-up. Median follow-up duration among returnees was 2 months (IQR: 1-5).

Factors Associated with Mortality

Univariate analysis identified factors significantly associated with mortality (Table 5). Multivariate logistic regression (Table 6) revealed independent predictors: age <6 months (AOR: 3.41, 95%CI: 1.38-8.42, p=0.008), delayed presentation (AOR: 2.89, 95%CI: 1.18-7.06, p=0.021), heart failure at presentation (AOR: 3.92, 95%CI: 1.54-9.97, p=0.004), complex CHD (AOR: 4.56, 95%CI: 1.72-12.08, p=0.002), and lack of surgical referral (AOR: 7.23, 95%CI: 1.51-34.62, p=0.013). The final multivariate model demonstrated good fit (Hosmer-Lemeshow test: χ^2=6.23, df=8, p=0.621; Nagelkerke R^2=0.487).

Table 5: Univariate Analysis of Factors Associated with Mortality

Variable	Died (n=15)	Survived (n=27)	OR (95% CI)	p-value
Age at presentation				
<6 months	10 (66.7%)	8 (29.6%)	4.22 (1.89-9.41)	0.001
≥6 months	5 (33.3%)	19 (70.4%)	Reference	
Gender				
Male	8 (53.3%)	15 (55.6%)	0.92 (0.41-2.06)	0.842
Female	7 (46.7%)	12 (44.4%)	Reference	
Delayed presentation				
Yes (>6 months)	12 (80.0%)	14 (51.9%)	3.71 (1.56-8.83)	0.003
No	3 (20.0%)	13 (48.1%)	Reference	
Heart failure at presentation				
Present	11 (73.3%)	10 (37.0%)	4.68 (2.01-10.89)	<0.001
Absent	4 (26.7%)	17 (63.0%)	Reference	
CHD type				
Cyanotic	10 (66.7%)	5 (18.5%)	3.82 (1.67-8.74)	0.001
Acyanotic	5 (33.3%)	22 (81.5%)	Reference	
Complex CHD				
Yes	12 (80.0%)	6 (22.2%)	6.12 (2.58-14.53)	<0.001
No	3 (20.0%)	21 (77.8%)	Reference	
Pulmonary hypertension				
Present	8 (53.3%)	7 (25.9%)	3.02 (1.31-6.96)	0.009
Absent	7 (46.7%)	20 (74.1%)	Reference	
Surgical referral				
No	14 (93.3%)	25 (92.6%)	9.33 (1.89-46.12)	0.006
Yes	1 (6.7%)	2 (7.4%)	Reference	

Table 6: Multivariate Logistic Regression - Independent Predictors of Mortality

Table 6: Multivariate Logistic Regression - Independent Predictors of Mortality

Variable	Adjusted OR (95% CI)	p-value
Age <6 months	3.41 (1.38-8.42)	0.008
Delayed presentation	2.89 (1.18-7.06)	0.021
Heart failure at presentation	3.92 (1.54-9.97)	0.004
Complex CHD	4.56 (1.72-12.08)	0.002
Lack of surgical referral	7.23 (1.51-34.62)	0.013

Hosmer-Lemeshow goodness-of-fit: χ^2=6.23, df=8, p=0.621; Nagelkerke R^2=0.487

Discussion

This one-year study provides contemporary data on the burden of congenital heart disease among children presenting to an emergency paediatric unit in North-Eastern Nigeria. The hospital-based proportion of 1.41% among 2,973 admissions aligns with reports from Lagos (1.4%)[16] and Benin City (1.6%).[17] The slightly higher proportion in this cohort may reflect improved case detection following the introduction of regular paediatric echocardiography services in September 2023. Notably, while CHD constituted only 1.41% of admissions, it accounted for 4.8% of all deaths (15 of 310 total deaths) in the EPU during 2024, confirming that CHD carries a disproportionately high death risk (3.4 times the overall mortality rate) compared to other childhood illnesses in this setting.

The median presentation age of 9 months, with 69.0% being infants, mirrors findings from Ibadan[18] and Kano[19] where most CHD presents in the first year of life. However, this represents a significant diagnostic delay (median 6 months from symptom onset) compared to high-income countries, where prenatal diagnosis and neonatal screening enable detection within weeks of birth.[20] Delayed presentation occurred in 61.9% of our cohort, higher than the 32% reported in a South African study[21] but consistent with other Nigerian reports.[22-24]

The predominance of children from rural areas (76.2%) and caregivers without formal education (54.8%) highlights socio-demographic factors that may contribute to delayed diagnosis and poor outcomes, though formal association testing with mortality was limited by sample size. Late CHD presentation has been documented in Cameroon[25] and Ethiopia.[26] Community-based health education targeting rural populations could improve early recognition of CHD symptoms such as difficulty feeding, tachypnoea, and cyanosis.

Ventricular septal defect (33.3%) was the commonest lesion, consistent with global[27] and Nigerian epidemiological data.[7,9,28] The high proportion of symptomatic VSDs likely reflects the fact that moderate-to-large defects present early with heart failure and recurrent infections, making them more likely to be diagnosed in hospital settings.[29]

Tetralogy of Fallot (21.4%) was the predominant cyanotic lesion, similar to reports from Ibadan (19.8%) [30] and Lagos (22.3%) [31]. The relatively high TOF proportion may reflect survival beyond infancy without intervention, with progressive cyanosis eventually bringing children to medical attention. In contrast, duct-dependent lesions like TGA and pulmonary atresia often result in early death without detection, potentially leading to under-representation in hospital-based studies.[32]

The proportion of complex CHD (42.9%) exceeds figures from some other Nigerian centres[18] but is comparable to reports from other Sub-Saharan African countries.[33] This high proportion may reflect referral bias; children with complex lesions are more likely to be symptomatic and reach tertiary care, while those with simple lesions may remain undiagnosed in the community. The 11.9% prevalence of associated extracardiac anomalies, particularly Down syndrome (4.8%), underscores the importance of CHD screening in children with dysmorphic features.[34]

The high frequency of respiratory difficulty (78.6%), recurrent infections (54.8%), and failure to thrive (47.6%) reflects the haemodynamic consequences of CHD. Left-to-right shunts produce pulmonary overcirculation, leading to tachypnoea and recurrent chest infections, while cyanotic lesions cause chronic hypoxaemia, impairing growth.[35]

Heart failure at presentation in 50.0% of children is concerning but consistent with Nigerian studies: Ekure et al.[31] reported 52% in Lagos, and Ogunbiyi et al.[18] found 41-48% in Ibadan. Heart failure in infants with CHD carries high mortality without timely intervention[36] and its high prevalence in our study reflects delayed diagnosis and limited treatment access.

The 14.3% incidence of hypoxic spells among cyanotic CHD patients is comparable to 12-18% reported elsewhere.[37] Hypoxic spells are emergencies requiring immediate intervention and are associated with increased morbidity if poorly managed.[38] The cerebrovascular accident in one TOF patient highlights thromboembolic risks in cyanotic CHD, particularly with iron deficiency and dehydration.[39]

The finding that 47.6% of CHD admissions occurred during the rainy season (May-October) aligns with the overall EPU admission pattern reported by Yusuf et al. [13], where 43.5% of total admissions occurred during this period. This seasonal clustering may reflect increased respiratory infections during the rains, which precipitate heart failure in children with underlying CHD.

The 35.7% mortality rate is distressingly high, but mirrors reports from other Nigerian centres without cardiac surgery facilities such as Ibadan (31%)[18] and Kano (36%).[19] These figures contrast sharply with <5% mortality in high-income countries, where early diagnosis and prompt surgical intervention are standard.[40]

The independent mortality predictors identified, i.e., age <6 months, delayed presentation, heart failure, complex CHD, and lack of surgical referral provide actionable targets for intervention. Infants under 6 months had 3.4-fold higher mortality odds, reflecting vulnerability during the transitional circulation period.[41]

The strongest predictor of mortality was lack of surgical referral (AOR: 7.23). Only 7.1% of children accessed surgical care, a stark illustration of the treatment gap in Nigeria, where fewer than 10 centres offer paediatric cardiac surgery for over 200 million people.[42] Financial constraints (82.1% of non-referred cases) were the predominant barrier, as most families cannot afford the out-of-pocket costs of surgery abroad. Similar challenges are reported across Sub-Saharan Africa.[43,44]

Given the delayed presentation, limited diagnostic capacity, and poor access to definitive treatment observed in this study, training primary healthcare workers to recognise early signs of congenital heart disease and implementing routine newborn pulse oximetry screening[45] could reduce diagnostic delays. Strengthening echocardiography services, expanding basic echocardiography training for paediatricians, and establishing telemedicine links[46] with paediatric cardiology centres would improve diagnostic capacity and facilitate timely referral. To address the severe treatment gap, regional cardiac surgical centres with coordinated referral pathways[47] should be developed, and paediatric cardiac surgery should be included in the National Health Insurance benefit package to

lessen the financial burden[49] that prevented 82% of families from accessing surgery. International partnerships and charitable collaborations[50] remain important to expand surgical volumes while local capacity is being built.

Some limitations must be acknowledged. This was a single-centre, retrospective study of only 42 patients with 15 deaths, limiting generalisability and the stability of multivariate estimates; the identified predictors should be regarded as exploratory. Neonates were excluded and follow-up was short, so the full spectrum and long-term outcomes of CHD are not captured. Selection bias toward severe, hospitalised cases probably overestimates complication and mortality rates. The small sample precluded adjustment for important confounders such as nutritional status. Multi-centre prospective studies with larger cohorts are needed to validate these findings.

Conclusion

Congenital heart diseases significantly contribute to paediatric morbidity and mortality in Azare, North-Eastern Nigeria, accounting for 1.41% of admissions and 4.8% of deaths in 2024, with a case fatality rate of 35.7%. Infants are primarily affected, often presenting late with complications including heart failure and pneumonia, while access to surgical care is extremely limited (7.1%). Independent mortality predictors include age under six months, delayed presentation, heart failure at presentation, complex CHD, and lack of surgical referral. To meet SDG 3.2 targets, Nigeria must enhance early detection, diagnostic capacity, and financial protections for affected families. This study's data aims to inform policy and resource allocation for paediatric cardiac services in underserved regions.

Conflict of Interest: None

Author's contribution: BAI, MOY, SOB, and FB conceptualised and designed the study. BAI, MOY, SP, and LIA were responsible for data acquisition and clinical management. BAI, SOB, and FB performed the data analysis and interpretation. BAI drafted the manuscript. All authors critically revised the manuscript for important intellectual content, approved the final version, and agreed to be accountable for all aspects of the work.

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ORIGINAL ARTICLE

Assessment of Combined Left Ventricular Function by Myocardial Performance Index in Children with Sickle Cell Anaemia in Steady State at a Nigerian Tertiary Hospital: A Comparative Study

Falaye Adejoke Monsurat[1], Liman Biliyaminu Ibrahim[2]

Abstract

Background: Children with sickle cell anaemia (SCA) are at risk of early cardiac dysfunction due to chronic anaemia and volume overload. Conventional echocardiographic indices may underestimate myocardial impairment, as they are load-dependent. The myocardial performance index (MPI) provides a relatively load-independent assessment of combined ventricular function.

Objective: To assess combined (systolic and diastolic) left ventricular (LV) function using MPI in children with SCA in steady state and compare findings with age- and sex-matched controls.

Methods: This comparative cross-sectional study involved 340 children with SCA in steady state and 340 HbAA controls aged 2–15 years at Usmanu Danfodiyo University Teaching Hospital, Sokoto. All participants underwent clinical evaluation and transthoracic echocardiography. LV MPI was calculated as (IVCT + IVRT)/ET. Conventional systolic and diastolic indices were also assessed. Data were analysed using SPSS version 23. Statistical significance was set at $p < 0.05$.

Results: The mean LV MPI was significantly higher in subjects than controls (0.52 ± 0.20 vs 0.48 ± 0.10; $p = 0.002$). A higher proportion of subjects had combined LV dysfunction using MPI (65.6%) compared with EF (15.0%) and FS (23.8%). MPI showed a moderate positive correlation with IVRT ($r = 0.652$; $p < 0.001$) and weak negative correlations with EF, DT, and E/A ratio.

Conclusion: Children with SCA in steady state exhibit significant combined LV dysfunction detectable by MPI despite relatively preserved conventional systolic indices. MPI is a sensitive tool for early identification of myocardial dysfunction and should be incorporated into routine cardiac evaluation of children with SCA.

Keywords: Sickle cell anaemia; Myocardial performance index; Left ventricular function.

Introduction

Sickle cell anaemia (SCA) is a chronic inherited haemoglobin disorder resulting from a point mutation in the β-globin gene, leading to the production of haemoglobin S. It is characterised by chronic haemolytic anaemia, episodic vaso-occlusion, and progressive multi-organ damage.[1] Globally, over 300,000 children are born annually with SCA, with sub-Saharan Africa accounting for more than 75% of the burden[2]. Nigeria alone contributes the highest number of affected births worldwide, making SCA a major public health concern in the country.[3]

Improvements in childhood survival due to better infection control, vaccination, and supportive care have led to a growing population of children and adolescents living with SCA. Consequently, chronic complications such as cardiovascular involvement are increasingly recognised contributors to morbidity and reduced quality of life. [4] The heart is particularly vulnerable in SCA because of sustained haemodynamic stress from chronic anaemia, recurrent

Date Received- 24 February 2026, Date Accepted- 14 July 2026
Date Published online- 25 August 2026

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Published by: MEDICAL AND DENTAL CONSULTANTS' ASSOCIATION OF NIGERIA (MDCAN)
FEDERAL UNIVERSITY TEACHING HOSPITAL, LAFIA, NASARAWA STATE

Falaye AM, Liman BI: Assessment of Combined Left Ventricular Function by Myocardial Performance Index in Children with Sickle Cell Anaemia in Steady State at a Nigerian Tertiary Hospital: A Comparative Study. *Nasarawa Journal of Medicine* 2026: 3 (1) 15-20

NASJM

hypoxic injury, endothelial dysfunction, and microvascular occlusion.[5]

Chronic anaemia in SCA results in a persistently high-output circulatory state. To compensate, there is an increase in stroke volume and cardiac output, leading to left ventricular (LV) dilatation and eccentric hypertrophy. Over time, these adaptive changes may progress to maladaptive remodelling, impaired ventricular relaxation, and eventual systolic dysfunction.[6] Autopsy and imaging studies have demonstrated myocardial fibrosis and microinfarction even in children and young adults with SCA, suggesting that myocardial involvement begins early in life. [7]

Echocardiography is the most widely used non-invasive tool for cardiac assessment in children with SCA. Conventional indices such as ejection fraction (EF) and fractional shortening (FS) are commonly employed to evaluate LV systolic function.[8] However, these parameters are highly dependent on preload and afterload, which are significantly altered in chronic anaemia. As a result, EF and FS may remain within normal limits despite underlying myocardial dysfunction, thereby underestimating the true burden of cardiac involvement in SCA. [9]

Diastolic dysfunction has been increasingly recognised as an early manifestation of sickle cell cardiomyopathy.[10] Abnormalities in ventricular relaxation and compliance may precede overt systolic dysfunction and are associated with adverse outcomes, including exercise intolerance and pulmonary hypertension. [11] Conventional Doppler indices of diastolic function, such as E/A ratio and deceleration time, are also influenced by loading conditions and heart rate, limiting their sensitivity in isolation.[12]

The myocardial performance index (MPI), also known as the Tei index, is a Doppler-derived parameter that combines systolic and diastolic time intervals to provide a single measure of combined ventricular performance.[13] It is calculated as the sum of isovolumetric contraction time and isovolumetric relaxation time divided by ejection time. The MPI is relatively independent of ventricular geometry, heart rate, and loading conditions, making it particularly useful in conditions characterised by altered haemodynamics, such as SCA. [13]

Recent studies from different regions have demonstrated the utility of MPI in detecting subclinical myocardial dysfunction in children with SCA.[14,15] However, data from Nigeria and much of sub-Saharan Africa remain limited, despite the high disease burden. Establishing baseline data on MPI in Nigerian children with SCA is crucial for early identification of cardiac dysfunction and for guiding long-term surveillance strategies. Therefore, this study assessed combined left ventricular (LV) function using MPI in children with SCA in steady state and compared the findings with those of age- and sex-matched healthy controls to determine the proportion of children with evidence of combined LV dysfunction.

Materials and Methods

Study Design and Setting

This was a comparative cross-sectional study conducted at the Sickle Cell Clinic and Paediatric Cardiology Unit of Usmanu Danfodiyo University Teaching Hospital (UDUTH), Sokoto, a major tertiary referral centre in Northwestern Nigeria.

Study Population

The study population consisted of children aged 2–15 years with confirmed sickle cell anaemia (HbSS) who attended the Sickle Cell Clinic at UDUTH. The sickle cell clinic runs on Wednesday every week with an average of 50 to 60 children seen per day, both new and old patients. (An estimate of about 2,400 children yearly). The lower age limit of 2 years was selected because the earliest cardiac manifestations of sickle cell anaemia are typically reported from this age [16] while the upper limit of 15 years reflected the hospital's paediatric age cutoff. Age- and sex-matched children with normal haemoglobin genotype (HbAA), attending the general paediatric outpatient clinic for minor ailments, served as controls.

Eligibility Criteria

Children with SCA were included if they were in steady state. This was defined as the absence of acute painful crisis, febrile illness, blood transfusion, or hospitalization in the preceding three months.[17] Study controls were children with HbAA, on follow-up at the paediatrics outpatient department clinic for minor illnesses such as uncomplicated malaria, upper respiratory tract infection, and other non-chronic illnesses not known to affect cardiac function.

Exclusion criteria included the presence of congenital or acquired heart disease. Children with a tricuspid regurgitation velocity (TRV) of ≥2.5 m/s were excluded because this threshold was considered suggestive of elevated pulmonary artery pressure and increased cardiopulmonary risk in sickle cell disease. The TRV was preferred because it is a practical, non-invasive echocardiographic screening parameter that could be measured during the study examination. Children with Hb SC, Hb CC, chronic kidney disease, chronic liver disease, malnutrition or those using cardioactive medications such as Digoxin, Dobutamine, Epinephrine, etc., were also excluded. Controls with anaemia (packed cell volume <30%) were also excluded.

Ethical considerations

The study protocol was reviewed and approved by the Ethics and Research Committee of the Usmanu Danfodiyo University Teaching Hospital, Sokoto (UDUTH/HREC/2019/754). Informed consent was obtained from parents in their local languages before participation in the study. Confidentiality of patient data was also maintained throughout the study.

Sample Size and Sampling Technique

A minimum sample size was calculated using standard formulae for comparative studies, based on previous reports of MPI differences in children with SCA.[9] A total of 340 children with sickle cell anaemia and 340 age- and sex-matched controls were enrolled. Participants with sickle cell anaemia were selected by simple random sampling from clinic attendance registers. If a selected participant had already been enrolled or did not meet the eligibility criteria, another participant was selected using the same sampling procedure. Controls were recruited from the follow-up and general outpatient clinics and matched individually to cases by age (±6 months) and sex. In nine instances where a sex-matched control was unavailable, a control of the opposite sex was enrolled to maintain the required sample size. Data collection was conducted over 24 months, from August 2020

to July 2022.

Data Collection

A structured self-administered questionnaire adapted from related studies on LV function assessment using MPI[7,11] was used to collect socio-demographic data, clinical history, and physical examination findings. The instrument was reviewed by experts for content validity and pretested among approximately 10% of the sample in a comparable health facility. Reliability testing produced a Cronbach alpha coefficient of 0.80. Anthropometric measurements were obtained using standard techniques.

Packed cell volume was measured for all participants. About 4 mL of venous blood was collected into EDTA bottles from each control for haemoglobin electrophoresis.

Echocardiographic Assessment

Transthoracic echocardiography was performed using a Sonoscape SSI-5000 machine with a 3.5–7.0 MHz transducer. Studies were conducted with participants in the left lateral decubitus position. Standard parasternal long-axis, short-axis, and apical four-chamber views were obtained according to American Society of Echocardiography guidelines.

LV systolic function was assessed using ejection fraction (EF) and fractional shortening (FS) using the Teichholz method. Diastolic function was evaluated using transmitral Doppler indices including E and A wave velocities, E/A ratio, isovolumetric relaxation time (IVRT), and deceleration time (DT). Myocardial performance index was calculated as:

MPI = (IVCT + IVRT) / ET

Cut off Value for normal MPI = 0.34 ± 0.03[17,18]

All measurements were averaged over three cardiac cycles. To minimise inter-observer variability, all echocardiographic examinations were performed by the same experienced paediatric cardiologist, who conducted approximately 8–10 studies per week.

Statistical Analysis

Data were checked for completeness and consistency at the point of collection and before entry into SPSS. The clinical and echocardiographic assessments were completed during the study visit, and the original proforma and stored echocardiographic records were consulted where clarification was required. Complete data for the primary and principal echocardiographic variables were available for all 680 participants included in the analysis. Therefore, no statistical imputation of missing data was undertaken. Data were analysed using SPSS version 23. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. An independent t-test was used to compare means between groups. A chi-square test was used for categorical variables. Pearson correlation analysis was performed to assess relationships between MPI and other echocardiographic indices. Statistical significance was set at $p < 0.05$.

Results

A total of 680 children were recruited, comprising 340 subjects and 340 controls aged 2 to 15 years, with no significant difference in mean age or age categories between the two groups. Among the subjects, males and females were equally represented, while the controls had slightly more females than males, as shown in Table1 below

Baseline characteristics of the study population

The mean weight, mean height and mean body surface area (BSA) in controls were higher than in the subjects, but the differences were not statistically significant. In addition, the mean pulse rate among the subjects was 96.48 ± 14.9 beats per minute, compared with 94.70 ± 15.1 beats per minute among the controls. Although the mean pulse rate was slightly higher among the subjects, the difference was not statistically significant (t = 1.54, p = 0.124), as shown in Table II below.

Mean values for MPI, systolic and diastolic function parameters of the left ventricles

Table III compares the myocardial performance index (MPI) and conventional echocardiographic indices of left ventricular function between subjects and controls. The mean LV MPI (0.52 ± 0.2) for the subjects was significantly higher than that of the controls (0.48± 0.01), p= 0.002.

Table I: Distribution of the subjects and controls by age and gender

Variables	Subjects; **n** (%)	Controls; **n** (%)	χ^2	p-values
Age categories (years)				
≤ 5	62(18.3)	83(24.4)		
>5 -10	165(48.5)	169(49.7)	7.035	0.060
>10 -15	113(33.2)	88(25.9)		
Mean	8.6±3.4	8.4 ± 3.7		
Gender				
Male	170(50.0)	161(47.4)	0.540	0.462
Female	170(50.0)	179(52.6)		

Table II : Baseline characteristics of subjects and controls

Variables	Subject (HbSS) Mean ±SD	Control (HbAA) Mean ±SD	t-Test	p-value
Weight(kg)	22.11±8.2	24.50±12.6	8.141	0.070
Height(cm)	119.08±21.3	121.88±18.2	7.761	0.085
BSA	0.86±0.2	0.88±0.3	25.053	0.250
Mean pulse rates (bpm)	96.48±14.9	94.70±15.1	1.54	0.124

Furthermore, Subjects had a significantly lower ejection fraction (61.44 ± 9.5% vs. 63.34 ± 7.3%; $p = 0.006$), shorter deceleration time (111.81 ± 28.5 vs. 129.40 ± 25.6 ms; $p < 0.001$), and higher E/A ratio (1.77 ± 0.3 vs. 1.61 ± 0.2; $p < 0.001$) and E-wave velocity (105.78 ± 15.3 vs. 98.43 ± 14.1 cm/s; $p < 0.001$) than controls. Fractional shortening, isovolumic relaxation time, and A-wave velocity were comparable between the groups ($p > 0.05$).

Myocardial Performance Index

Table IV shows that the prevalence of abnormal left ventricular function was significantly higher among subjects than controls based on the myocardial performance index (65.6% vs. 43.2%; $p = 0.014$), ejection fraction (15.0% vs. 9.4%; $p = 0.004$), and fractional shortening (23.8% vs. 11.5%; $p < 0.001$). Significant between-group differences were also observed for deceleration time ($p < 0.001$) and E/A ratio ($p = 0.017$), whereas the prevalence of abnormal isovolumic relaxation time (IVRT) was comparable between the groups ($p = 0.155$).

Correlation Analysis

IVRT was the only variable exhibiting a significant positive correlation with LV MPI in both groups (Subjects: r=0.652; Controls: r=0.472; both p<0.001), indicating it is the most consistent determinant of MPI irrespective of disease status (Table V).

In Controls, additional modest but significant correlations emerged, i.e., DT (negative), E velocity (positive), and A velocity (positive). In Subjects, however, the diastolic filling profile, i.e., E/A ratio and E velocity, showed a significant negative correlation while A velocity lost significance. Notably, EF and FS showed no correlation with LV MPI in either cohort.

MPI showed a moderate positive correlation with IVRT (r = 0.652; p < 0.001) and weak negative correlations with EF, FS, DT, E/A ratio, and E velocity. These findings suggest that worsening combined ventricular performance is predominantly related to impaired diastolic function, as shown in Table V below

Discussion

This study demonstrates that children with sickle cell anaemia in steady state have a significantly higher myocardial performance index than age- and sex-matched healthy controls, indicating early combined left ventricular dysfunction. Importantly, this dysfunction was detected despite relatively preserved conventional systolic indices, highlighting the limitations of relying solely on ejection fraction and fractional shortening in this population.

The pathophysiology of cardiac involvement in SCA is

Table III: MPI and other conventional echocardiographic indices of LV function among the study population

Variables	Subjects (Mean ±SD)	Controls (Mean ±SD)	t-Test	P-value
MPI	0.52 ± 0.2	0.48± 0.01	3.128	0.002
EF(%)	61.44±9.5	63.34± 7.3	2.747	0.006
FS(%)	33.30± 7.3	33.98± 5.7	1.279	0.201
IVRT(m/s)	67.19± 11.9	66.49±13.4	0.678	0.498
DT(m/s)	111.81± 28.5	129.40±25.6	7.953	<0.001
E/A ratio	1.77± 0.3	1.61± 0.2	7.296	<0.001
E vel.(cm/sec)	105.78± 15.3	98.43± 14.1	6.110	<0.001
A vel.(cm/sec)	61.90± 27.3	61.98± 13.0	0.047	0.962

Table IV: Combined ventricular dysfunction using MPI and conventional echocardiographic indices in subjects and controls

Echocardiographic index	**Subjects, n** (%) abnormal	**Controls, n** (%) abnormal	χ^2	**p-value**
MPI	223 (65.6)	147 (43.2)	6.41	0.014
Ejection fraction	51 (15.0)	32 (9.4)	9.04	0.004
Fractional shortening	81 (23.8)	39 (11.5)	25.44	<0.001
IVRT	102 (30.0)	119 (35.0)	2.29	0.155
Deceleration time	71 (20.9)	128 (37.6)	27.80	<0.001
E/A ratio	73 (21.5)	98 (28.8)	6.10	0.017

Table V: Correlation between LV MPI and conventional echocardiographic indices of LV functions in subjects and controls.

Variables	**Subjects**		**Controls**	
	r	p-value	r	p-value
EF	-0.069	0.234	-0.013	0.818
FS	-0.081	0.160	-0.005	0.925
IVRT	0.652	**<0.001**	0.472	**<0.001**
DT	-0.040	0.489	-0.128	0.027
E/A ratio	-0.121	0.037	0.023	0.692
E velocity	-0.117	0.042	0.114	0.048
A velocity	0.069	0.236	0.182	0.002

multifactorial. Chronic anaemia results in sustained volume overload and increased cardiac output, leading to LV dilatation and eccentric hypertrophy. While these changes are initially adaptive, prolonged haemodynamic stress eventually leads to myocardial remodelling, fibrosis, and impaired relaxation. [11,12,13] In addition, recurrent vaso-occlusive episodes and microvascular obstruction may cause repeated subclinical myocardial ischaemia, further contributing to myocardial dysfunction.[11,12]

In the present study, EF was significantly lower among subjects compared with controls; however, most values remained within normal limits. This finding is consistent with several recent studies from Nigeria and other African countries, which have reported preserved or mildly reduced systolic function in children with SCA. [9,18,19] The apparent preservation of systolic function may reflect compensatory mechanisms related to chronic anaemia rather than true myocardial health.

Diastolic abnormalities were more prominent in this study, as evidenced by prolonged IVRT and altered transmitral flow indices. Diastolic dysfunction has been increasingly recognised as an early manifestation of sickle cell cardiomyopathy and has been associated with exercise intolerance and adverse clinical outcomes. [9,11,13,14] The strong correlation observed between MPI and IVRT in this study supports the notion that impaired relaxation plays a central role in early myocardial dysfunction in SCA.[9,14]

The myocardial performance index integrates both systolic and diastolic components of ventricular function and is relatively independent of loading conditions. [9] This makes it particularly valuable in SCA, where preload and afterload are chronically altered. In this study, MPI identified combined LV dysfunction in nearly two-thirds of subjects, far exceeding the proportion detected using EF or FS alone. Similar findings have been reported in recent international studies, underscoring the sensitivity of MPI in detecting subclinical myocardial impairment. [11,14,15]

Studies from other regions have also demonstrated the prognostic value of MPI in chronic anaemic states.[5,6,7,13] Elevated MPI has been associated with increased risk of heart failure and mortality in various paediatric and adult populations. [9,13,14] Although long-term outcome data were beyond the scope of this study, the high prevalence of abnormal MPI among children with SCA raises concerns about future cardiovascular risk and underscores the need for early intervention and monitoring.

From a clinical perspective, the incorporation of MPI into routine echocardiographic evaluation of children with SCA could facilitate earlier detection of myocardial dysfunction, allowing for closer follow-up and timely management. This is particularly relevant in resource-limited settings, where advanced imaging modalities may not be readily available.

This study has some limitations. Its cross-sectional design precludes assessment of temporal changes in myocardial function or causal relationships. In addition, the study was conducted at a single tertiary centre, which may limit generalizability. Nevertheless, the large sample size and inclusion of well-matched controls strengthen the validity of the findings. Although using a single experienced paediatric cardiologist improved measurement consistency, it may have introduced systematic observer bias. In addition, intra observer reproducibility was not assessed, particularly for operator dependent time interval measurements such as IVRT and IVCT.

Conclusion

Children with sickle cell anaemia in steady state exhibit significant combined left ventricular dysfunction as assessed by myocardial performance index, even in the absence of overt systolic impairment on conventional echocardiography. MPI is a sensitive and practical tool for early detection of myocardial dysfunction in this population.

Recommendations

Routine echocardiographic surveillance of children with SCA should include assessment of MPI to facilitate early identification of cardiac involvement. Longitudinal studies are recommended to evaluate the prognostic significance of elevated MPI and to determine the impact of early interventions on long-term cardiovascular outcomes.

Author's contribution: FAM conceived and designed the study, drafted the manuscript, and, together with LBI, revised the manuscript and approved the final version. LBI contributed to data analysis and interpretation.

Acknowledgements: The authors acknowledge the staff of the Paediatric Cardiology Unit and Sickle Cell Clinic of Usmanu Danfodiyo University Teaching Hospital, Sokoto, as well as the children and caregivers who participated in this study.

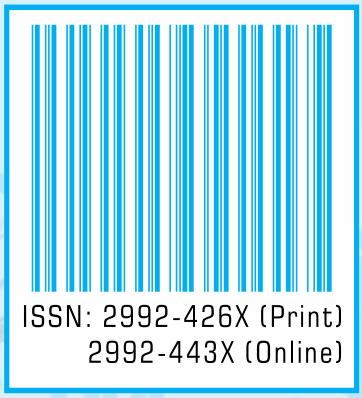
Conflict of Interest: The authors declare no conflict of interest.

Funding: This research was self-funded by the investigators.

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ISSN: 2992-426X (Print)
2992-443X (Online)