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Clinical and Aetiological Patterns of Paediatric Heart Failure in Nigeria: Insights Into Soluble ST2 as an Emerging Biomarker

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Research Article

Keywords: Paediatric heart failure, soluble ST2, biomarker, severe malarial anaemia, congenital heart disease, pneumonia, Nigeria

Posted Date: November 18th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11249554/v1

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Additional Declarations: No competing interests reported.

Abstract

Background

Paediatric heart failure remains a major cause of morbidity and mortality in low- and middle-income countries. Soluble suppression of tumorigenicity-2 (sST2) is an emerging biomarker of myocardial stress and remodelling, but evidence in African children is limited. This study assessed the clinical and aetiological patterns of paediatric heart failure and the relationship between heart failure and plasma sST2 levels among Nigerian children.

Methods

This analytical cross-sectional study was conducted at the Emergency Paediatric Unit, University of Benin Teaching Hospital, Nigeria, from March to October 2024. A total of 408 children were enrolled, comprising 204 children with clinically diagnosed heart failure and 204 age- and sex-matched healthy controls. Sociodemographic, clinical, anthropometric and laboratory data were collected. Heart failure severity was assessed using the Modified Ross Classification, while plasma sST2 levels were measured and compared between groups and across aetiological categories.

Results

Children younger than one year accounted for 87 (42.6%) cases, while 139 (68.1%) were from lower socioeconomic groups. Most had mild (107, 52.5%) or moderate (91, 44.6%) heart failure. Pneumonia (76, 37.2%), severe malarial anaemia (65, 31.9%) and congenital heart disease (36, 17.6%) were the leading causes. Cases had significantly higher pulse and respiratory rates and lower BMI-for-age z-scores than controls ($p < 0.001$). Median plasma sST2 was significantly higher in cases than controls (6.26 [3.79–15.98] vs 4.05 [2.25–6.35] ng/mL; $p < 0.001$) and varied significantly by aetiology ($p = 0.037$), with highest levels in malignancy-related and pericardial heart failure. No significant sex-related differences were observed.

Conclusion

Paediatric heart failure was predominantly associated with pneumonia, severe malarial anaemia and congenital heart disease. Plasma sST2 was significantly elevated and varied by aetiology, suggesting potential utility as a biomarker of myocardial stress. Longitudinal studies are warranted to establish its prognostic value.

BACKGROUND

Heart failure (HF) is a major cause of morbidity and mortality in children and represents the final common pathway of several cardiac and non-cardiac disorders.[1,2] Unlike adults, where heart failure is largely attributable to ischemic heart disease and hypertension, paediatric heart failure arises from a diverse range of conditions including congenital heart disease, cardiomyopathies, rheumatic heart disease, severe anaemia, respiratory tract infections, and sepsis.[3,4] The condition is associated with prolonged hospitalizations, increased healthcare costs, and significant mortality, particularly in resource-limited settings.[5]

Globally, an estimated 6 million children and adolescents were living with heart failure in 2021, highlighting its substantial public health burden.[6] In sub-Saharan Africa, paediatric heart failure remains a common cause of hospital admission and death, driven by the dual burden of congenital cardiac disorders and preventable infectious diseases, differing considerably from that seen in high-income countries, with non-cardiac conditions contributing significantly to disease occurrence.[7,8]

In Nigeria, paediatric heart failure is a frequent clinical syndrome encountered in tertiary healthcare facilities. A recent systematic review and meta-analysis reported an overall prevalence of approximately 8.3% among hospitalized children, increasing to over 13% among non-neonatal admissions.[9] Severe anaemia, respiratory tract infections, congenital heart disease, and sepsis were identified as the leading causes, while the pooled case fatality rate was about 16%.[9] Despite advances in diagnosis and management, outcomes remain poor, emphasizing the need for improved approaches to risk assessment and monitoring.

Diagnosis is primarily based on clinical evaluation and echocardiography; however, clinical manifestations may be non-specific, particularly in younger children.[4,10] Consequently, there is growing interest in biomarkers that can aid diagnosis, assess disease severity, and predict outcomes.

Soluble suppression of tumorigenicity-2 (sST2) is an emerging cardiovascular biomarker released in response to myocardial stress, inflammation, and cardiac remodelling, with elevated levels being associated with worsening heart failure, adverse ventricular remodelling, and increased mortality in adults.[11,12] Although sST2 has demonstrated prognostic value in adult heart failure, evidence regarding its utility in paediatric populations remains limited, particularly in Africa.

Given the substantial burden of paediatric heart failure in Nigeria and the paucity of data on novel biomarkers, this study aimed to characterize the clinical and aetiological patterns of paediatric heart failure and explore the potential role of soluble ST2 as an emerging biomarker in affected children. This may contribute to improved diagnosis, risk stratification, and management of paediatric heart failure.

METHODOLOGY

Study Design and Setting

This analytical cross-sectional study was conducted at the Emergency Paediatric Unit (EPU) of the University of Benin Teaching Hospital (UBTH), Benin City, Edo State, Nigeria, between March and October 2024. UBTH is a 900-bed tertiary referral hospital serving Edo State and neighbouring states in southern Nigeria. The EPU manages approximately 1,600 paediatric admissions annually, including children with heart failure.

Study Population

The study included children aged 2 months to 17 years. Subjects were children with clinically diagnosed heart failure, while controls were apparently healthy age- and sex-matched children recruited from outpatient clinics.

Children were diagnosed with heart failure based on the presence of soft tender hepatomegaly and at least two of the following: tachycardia, tachypnoea, dyspnoea, or cardiomegaly. Subjects with congenital heart disease, acquired heart disease, pneumonia, severe malarial anaemia, or other systemic illnesses causing heart failure were eligible for inclusion. Children with conditions known to significantly affect inflammatory responses, including diabetes mellitus, systemic lupus erythematosus, chronic liver disease, collagen vascular diseases, sepsis, chronic pulmonary disease, and neonatal conditions, were excluded.

Controls were recruited from paediatric outpatient clinics and were excluded if they had any medical condition capable of influencing inflammatory or biochemical markers.

Sample Size and Sampling Technique

The minimum sample size was calculated using the formula for comparison of two proportions, based on a prevalence of 16.7%, a confidence level of 95%, and a study power of 90%. After finite population correction and adjustment for possible attrition, a sample size of 204 participants was obtained for each group.

Participants were recruited consecutively. Eligible subjects were enrolled within 24 hours of admission to the EPU, while controls were recruited from outpatient clinics and matched for age and sex.

Data Collection

Data were collected using a semi-structured interviewer-administered questionnaire. Information obtained included sociodemographic characteristics and clinical details. Socioeconomic class was determined using the revised Ibadin classification.[13] Heart failure severity was assessed using the Modified Ross Scoring System[14] and categorized as mild (3–6), moderate (7–9), or severe (10–12).

Anthropometric measurements, including weight, height/length, and body mass index (BMI), were obtained using standard procedures. Weight-for-age (WAZ), height-for-age (HAZ), and BMI-for-age (BAZ) z-scores were generated using World Health Organization growth standards.[15] Vital signs assessed included pulse rate, respiratory rate, blood pressure, and peripheral oxygen saturation (SpO_2).

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 27.0. Continuous variables were summarized as medians and interquartile ranges, while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using the Mann–Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables. Comparisons involving more than two groups were conducted using the Kruskal–Wallis test with post-hoc analyses where appropriate. Statistical significance was set at $p < 0.05$.

Ethical Considerations

Ethical approval

was obtained from the Health Research and Ethics Committee of the University of Benin Teaching Hospital, Benin City (ADM/E22/A/VOL.VII/148301217). The study conformed with the Helsinki Declaration of 1964, as revised in 2013, concerning human and animal rights. Participants consented to the study and all questionnaires were coded to ensure confidentiality. Written informed consent was obtained from parents or caregivers, while assent was obtained from older children where applicable. Confidentiality of participant information was maintained throughout the study.

RESULTS

Sociodemographic Characteristics of Participants

A total of 408 children participated in the study, comprising 204 children with clinically diagnosed heart failure (subjects) and 204 age- and sex-matched healthy controls. Females constituted over half of both groups, accounting for 116 (56.9%), while males were 88 (43.1%). The median age of subjects was 1.25 years (IQR: 0.67–4.00) compared with 2.00 years (IQR: 0.80–4.00) among controls. This difference was not statistically significant ($p = 0.154$). Similarly, median age did not differ significantly between male and female subjects ($p = 0.143$).

The age distribution was comparable between subjects and controls, with children younger than one year accounting for 87 (42.6%) in each group. Those aged 1–5 years constituted 84 (41.2%) subjects and 73 (35.8%) controls, while children older than five years accounted for 33 (16.2%) subjects and 44 (21.6%) controls. Socioeconomic status differed significantly between the groups ($p < 0.001$). Over two-thirds of subjects, 139 (68.1%), belonged to the lower socioeconomic class compared with 87 (42.6%) controls. Conversely, the middle socioeconomic class was more common among controls, accounting

for 111 (54.5%) compared with 46 (22.5%) subjects. The upper socioeconomic class comprised 19 (9.4%) subjects and 6 (2.9%) controls. As illustrated in Table 1

Severity of Heart Failure Among Subjects

Among the 204 children with heart failure, over half, 107 (52.5%), had mild heart failure, while 91 (44.6%) had moderate disease. Severe heart failure was uncommon and occurred in only 6 (2.9%) children. As shown in Fig. 1

Anthropometric and Vital Sign Characteristics

The median BMI-for-age z-score was significantly lower among subjects, − 0.42 (IQR: −1.07–0.37), compared with 0.51 (IQR: −1.37–1.86) among controls ($p < 0.001$). However, no significant differences were observed in weight-for-age z-score, with median values of − 1.48 (IQR: −3.00–0.17) among subjects and − 1.23 (IQR: −2.65–0.30) among controls ($p = 0.825$), or in height-for-age z-score, which was 0.94 (IQR: −0.47–1.42) among subjects and 0.88 (IQR: −0.49–1.22) among controls ($p = 0.591$).

Children with heart failure had significantly higher pulse and respiratory rates than controls. The median pulse rate was 140.00 beats per minute (IQR: 134.00–160.00) among subjects compared with 124.00 beats per minute (IQR: 110.00–130.00) among controls ($p < 0.001$). Similarly, the median respiratory rate was 47.00 breaths per minute (IQR: 40.00–60.00) among subjects and 30.00 breaths per minute (IQR: 24.50–32.00) among controls ($p < 0.001$). Oxygen saturation was significantly lower among subjects, with a median value of 95.00% (IQR: 91.00–96.00) compared with 96.00% (IQR: 96.00–98.00) among controls ($p < 0.001$). Diastolic blood pressure was also significantly lower among subjects, with a median of 60.00 mmHg (IQR: 56.00–64.00) versus 62.00 mmHg (IQR: 60.00–66.00) among controls ($p < 0.001$). There was no significant difference in systolic blood pressure between the groups ($p = 0.426$). As shown in Table 1

Aetiology of Heart Failure

Anaemia was the most common cause of heart failure, accounting for 83 (40.8%) subjects. Severe malarial anaemia constituted the largest proportion, occurring in 65 (31.9%) children, while sickle cell anaemia and malignancy-related anaemia accounted for 14 (6.9%) and 4 (2.0%) cases respectively. Pneumonia was the second most common cause, occurring in 76 (37.2%) subjects. Congenital heart disease was identified in 36 (17.6%) children, while myocardial and pericardial diseases accounted for the remaining cases.

Among the 36 children with congenital heart disease, ventricular septal defect was the predominant lesion, accounting for 18 (50.0%) cases. Atrioventricular septal defect occurred in 12 (33.3%) children, while atrial septal defect and patent ductus arteriosus each accounted for 3 (8.3%) cases. This is illustrated in Fig. 2

Plasma sST2 Levels Across Aetiological Categories

Plasma sST2 levels varied significantly across the different causes of heart failure (p = 0.037). The highest median level was observed among children with malignancies, 15.56 ng/ml (IQR: 14.01–17.11), followed by pericardial diseases, 13.03 ng/ml (IQR: 4.54–21.52), and congenital heart disease, 8.54 ng/ml (IQR: 4.34–17.48). Severe malarial anaemia was associated with a median sST2 level of 7.94 ng/ml (IQR: 3.67–16.92), while lower levels were observed among children with pneumonia, 4.82 ng/ml (IQR: 3.78–11.89), myocardial diseases, 4.42 ng/ml (IQR: 2.52–4.70), and sickle cell anaemia, 4.36 ng/ml (IQR: 3.34–7.53). This summarised in Table 3, Fig. 3.

Plasma sST2 Levels by Age Group

Plasma sST2 levels were significantly higher among subjects than controls across all age categories. Among children younger than one year, the median sST2 level was 5.69 ng/ml (IQR: 4.16–15.19) among subjects compared with 4.05 ng/ml (IQR: 2.68–6.13) among controls (p < 0.001). For those aged 1–5 years, the median sST2 level was 6.84 ng/ml (IQR: 3.39–15.36) among subjects and 3.64 ng/ml (IQR: 1.94–8.01) among controls (p < 0.001). Among children older than five years, the median sST2 level was 7.35 ng/ml (IQR: 3.75–17.34) among subjects compared with 4.25 ng/ml (IQR: 2.77–5.19) among controls (p = 0.007).

Overall, subjects had a significantly higher median plasma sST2 level of 6.26 ng/ml (IQR: 3.79–15.98) compared with 4.05 ng/ml (IQR: 2.25–6.35) among controls (p < 0.001). Although median sST2 levels showed a gradual increase across age groups among subjects, the difference was not statistically significant (p = 0.702). As illustrated in Table 4, Fig. 4

Plasma sST2 Levels by Sex

Male subjects had a median plasma sST2 level of 6.44 ng/ml (IQR: 4.09–15.19), while females had a median level of 6.07 ng/ml (IQR: 3.47–15.98). This difference was not statistically significant (p = 0.480). Similarly, among controls, median sST2 levels were comparable between males, 3.91 ng/ml (IQR: 2.43–6.21), and females, 4.05 ng/ml (IQR: 2.16–6.85), with no significant difference observed (p = 0.829). As shown in Fig. 5

Table 1
Distribution of sociodemographic characteristics of study participants

Characteristics	Subjects (N = 204) n (%)	Controls (N = 204) n (%)	χ^2	df	P-value
Gender					
Male	88 (43.1%)	88 (43.1%)	< 0.001	1	> 0.999
Female	116 (56.9%)	116 (56.9%)			
Age group (years)					
<1	87 (42.6%)	87 (42.6%)	2.334	3	0.312
1–5	84 (41.2%)	73 (35.8%)			
>5	33 (16.2%)	44 (21.6%)			
Socio-economic Class					
Lower	139 (68.1%)	87 (42.6%)	45.635	2	< 0.001*
Middle	46 (22.5%)	111 (54.5%)			
Upper	19 (9.4%)	6 (2.9%)			

Note. χ^2 = chi-square test statistic; df = degrees of freedom. *Statistically significant ($p < 0.05$).

Table 2
Comparison of median anthropometric and vital sign values between subjects and controls

Parameter	Subjects Median [IQR]	Controls Median [IQR]	Z-statistic	P-value
ANTHROPOMETRY				
WAZ	−1.48 [− 3.00–0.17]	−1.23 [− 2.65–0.30]	0.222	0.825
HAZ	0.94 [− 0.47–1.42]	0.88 [− 0.49–1.22]	0.538	0.591
BAZ	−0.42 [− 1.07–0.37]	0.51 [− 1.37–1.86]	6.616	< 0.001*
VITAL SIGNS				
Pulse rate (bpm)	140.00 [134.00–160.00]	124.00 [110.00–130.00]	14.297	< 0.001*
SpO_2 (%)	95.00 [91.00–96.00]	96.00 [96.00–98.00]	7.270	< 0.001*
Respiratory rate (breaths/min)	47.00 [40.00–60.00]	30.00 [24.50–32.00]	15.999	< 0.001*
Systolic BP (mmHg)	90.00 [82.50–100.00]	90.00 [84.00–100.00]	0.796	0.426
Diastolic BP (mmHg)	60.00 [56.00–64.00]	62.00 [60.00–66.00]	4.596	< 0.001*

Note. WAZ = weight-for-age z-score; HAZ = height-for-age z-score; BAZ = BMI-for-age z-score; IQR = interquartile range; SpO_2 = oxygen saturation; BP = blood pressure. *p < 0.05. Mann–Whitney U test.

Table 3
Plasma sST2 levels across aetiological categories of paediatric heart failure

Cause of Heart Failure	Median [IQR] (ng/ml)	Test Statistic (H)	P-value
Congenital Heart Disease	8.54 [4.34–17.48]	13.442	0.037*
Myocardial diseases	4.42 [2.52–4.70]		
Pericardial diseases	13.03 [4.54–21.52]		
Severe malarial anaemia	7.94 [3.67–16.92]		
Malignancies	15.56 [14.01–17.11]		
Pneumonia	4.82 [3.78–11.89]		
Sickle cell anaemia	4.36 [3.34–7.53]		

Note. IQR = interquartile range; H = Kruskal–Wallis test statistic. *Overall p = 0.037. Pairwise: severe malarial anaemia had significantly lower sST2 than pneumonia (p < 0.001), CHD (p < 0.001), myocardial diseases (p = 0.005), and malignancies (p < 0.001).

Table 4
Comparison of median plasma sST2 levels between subjects and controls by age group

Age Group	Subjects Median [IQR] (ng/ml)	Controls Median [IQR] (ng/ml)	Z-statistic	P-value
< 1 year	5.69 [4.16–15.19]	4.05 [2.68–6.13]	4.162	< 0.001*
1–5 years	6.84 [3.39–15.36]	3.64 [1.94–8.01]	4.041	< 0.001*
> 5 years	7.35 [3.75–17.34]	4.25 [2.77–5.19]	2.668	0.007*
Total (All Ages)	**6.26 [3.79–15.98]**	**4.05 [2.25–6.35]**	**6.260**	**< 0.001***

Note. IQR = interquartile range. *p < 0.05. Mann–Whitney U test for between-group comparisons. Within-subject age comparison: Kruskal–Wallis H = 0.707, df = 2, p = 0.702 (NS).

DISCUSSION

This study characterized the clinical and aetiological patterns of paediatric heart failure and explored the potential utility of soluble ST2 (sST2) as an emerging biomarker among Nigerian children. The findings demonstrated that heart failure occurred predominantly in children younger than five years, was more common among those from lower socioeconomic backgrounds, and was largely attributable to preventable conditions such as severe malarial anaemia and pneumonia. Furthermore, plasma sST2 levels were significantly elevated among children with heart failure compared with healthy controls and varied according to the underlying aetiology.

The predominance of infants and young children among heart failure cases is consistent with reports from Okpokowuruk et al.[16] in Akwa Ibom, Nigeria and AbdulKadir et al.[9] in a nationwide meta-analysis, which have shown that heart failure occurs most frequently during early childhood. This pattern may reflect the high burden of congenital heart disease, severe infections, and nutritional vulnerabilities in younger age groups. Younger children are particularly susceptible to rapid cardiovascular decompensation because of limited physiological reserve and increased metabolic demands.[17] The finding highlights the need for early recognition and prompt management of conditions predisposing to heart failure during infancy and early childhood.

A significantly greater proportion of children with heart failure belonged to the lower socioeconomic class. Similar observations have been reported in studies from Nigeria and other low- and middle-income settings.[18–20] Children from disadvantaged households are more likely to experience delayed healthcare seeking, poor nutritional status, recurrent infections, and inadequate access to preventive health services.[21] These factors may increase vulnerability to diseases such as malaria, pneumonia, and anaemia, which remain important causes of paediatric heart failure in resource-constrained settings. This finding highlights the influence of social determinants of health on paediatric cardiovascular outcomes.

More than half of the children presented with mild heart failure, while only a small proportion had severe disease. This finding suggests that many affected children were identified before progression to advanced cardiac dysfunction. Early presentation may be attributable to increasing awareness among caregivers and improved referral practices within the study setting. Nevertheless, the substantial proportion of children with moderate heart failure indicates that delayed presentation remains a challenge and supports continued efforts toward earlier diagnosis and intervention.

Children with heart failure had significantly lower BMI-for-age scores and significantly higher pulse and respiratory rates than healthy controls. These findings are consistent with the well-established pathophysiological consequences of heart failure, including increased metabolic demands, poor feeding, reduced nutritional intake, and chronic catabolic stress.[22,23] The observed lower oxygen saturation and diastolic blood pressure among affected children further reflect impaired cardiovascular performance and reduced tissue oxygen delivery. Collectively, these clinical characteristics reinforce the importance of careful nutritional and physiological assessment in children presenting with heart failure.

A major finding of this study was that severe malarial anaemia and pneumonia together accounted for over two-thirds of all heart failure cases. This finding is consistent with studies by Abdulkadir et al.[9] in a nationwide study, Duru et al.[24] in Bayelsa state, and Abdi et al.[25] in Ethiopia, identified severe anaemia and respiratory tract infections as leading causes of paediatric heart failure. The persistence of these preventable conditions as dominant aetiologies highlights ongoing public health challenges related to malaria control, childhood infections, and access to timely healthcare. Although congenital heart disease remained an important contributor, the predominance of non-cardiac causes distinguishes paediatric

heart failure patterns in Nigeria from those observed in high-income countries, where structural and genetic cardiac disorders predominate.

The study also demonstrated significantly elevated plasma sST2 levels among children with heart failure compared with healthy controls across all age groups. This finding supports the growing evidence that sST2 is a marker of myocardial stress and cardiac remodelling. The consistently higher levels among affected children suggest that sST2 may have potential utility as a biomarker for identifying paediatric heart failure and monitoring disease burden.

Plasma sST2 concentrations differed significantly according to the underlying cause of heart failure. The highest levels were observed among children with malignancy-related and pericardial causes of heart failure, while lower levels were noted in those with pneumonia and sickle cell anaemia. These differences may reflect varying degrees of systemic inflammation, myocardial strain, and tissue remodelling associated with different disease processes. The finding suggests that sST2 may provide additional information regarding the underlying pathophysiological mechanisms contributing to heart failure beyond conventional clinical assessment.

Although plasma sST2 levels tended to increase with age among subjects, the difference was not statistically significant. Similarly, no significant sex-related differences were observed. These findings suggest that sST2 is relatively stable across demographic categories and may therefore be less influenced by age and sex than several other cardiovascular biomarkers. Such characteristics may enhance its clinical applicability in paediatric populations.

LIMITATIONS

This study was conducted in a single tertiary healthcare facility, which may limit the generalizability of the findings to other settings. The cross-sectional design precluded assessment of temporal relationships and prognostic outcomes associated with sST2 levels. Most participants had mild or moderate heart failure, resulting in relatively few cases of severe disease for subgroup analyses. Additionally, serial measurements of sST2 were not performed, preventing evaluation of changes in biomarker levels over time or in response to treatment.

CONCLUSION

Paediatric heart failure in this Nigerian tertiary hospital was predominantly caused by severe malarial anaemia, pneumonia, and congenital heart disease, with most affected children presenting in early childhood and belonging to lower socioeconomic groups. Children with heart failure exhibited significantly higher plasma sST2 levels than healthy controls, and sST2 concentrations varied according to the underlying aetiology. These findings suggest that sST2 may serve as a useful biomarker of myocardial stress in paediatric heart failure and may complement conventional clinical assessment. Strengthening prevention and early treatment of malaria, anaemia, and respiratory infections remains essential for reducing the burden of paediatric heart failure in Nigeria.

RECOMMENDATIONS

1. Routine efforts to prevent and promptly treat malaria, severe anaemia, and pneumonia should be strengthened to reduce the burden of paediatric heart failure.
2. Clinicians should maintain a high index of suspicion for heart failure among young children presenting with severe anaemia, respiratory infections, or congenital heart disease.
3. Further research should establish age-specific reference ranges and clinically useful cut-off values for sST2 in African paediatric populations.
4. Integration of novel biomarkers such as sST2 into paediatric heart failure evaluation protocols should be explored alongside conventional clinical and echocardiographic assessments.

Declarations

FUNDING DECLARATION

This research received no special grant from any funding agency in the public,commercial or not-for-profit sector.

CONSENT FOR PUBLICATION

Not Applicable

CLINICAL TRIAL NUMBER

Clinical trial number is not applicable in this manuscript

Author Contribution

B.N. conceived and designed the study, contributed to data acquisition, analysis and interpretation, and drafted the manuscript. W.E.S. contributed to the study conception and design, interpretation of findings, and critical revision of the manuscript for important intellectual content. M.T.A. contributed to data acquisition and analysis, interpretation of results, and critical revision of the manuscript. B.N., W.E.S., and M.T.A. reviewed and approved the final manuscript and agreed to be accountable for all aspects of the work

Acknowledgement

The authors thank the Children Emergency Room, Paediatric cardiology clinic teams, and Dr Enosakhare Okungbowa (biostatistician/Lecturer)

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Figures

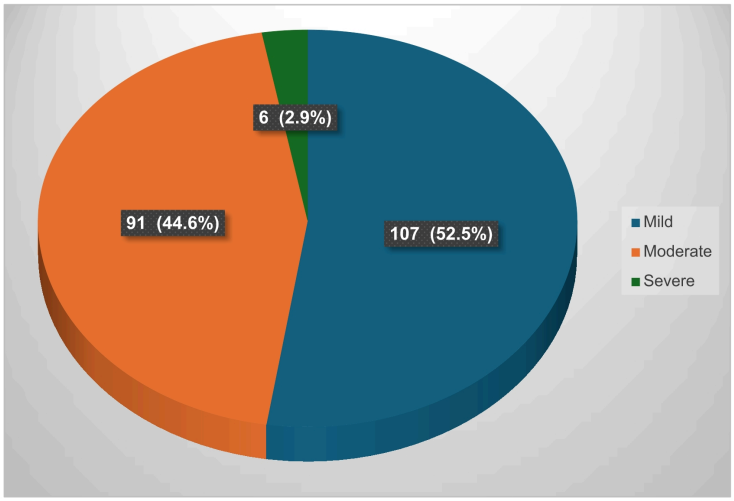


Figure 1

Distribution of heart failure severity at presentation by Modified Ross Classification (N = 204).

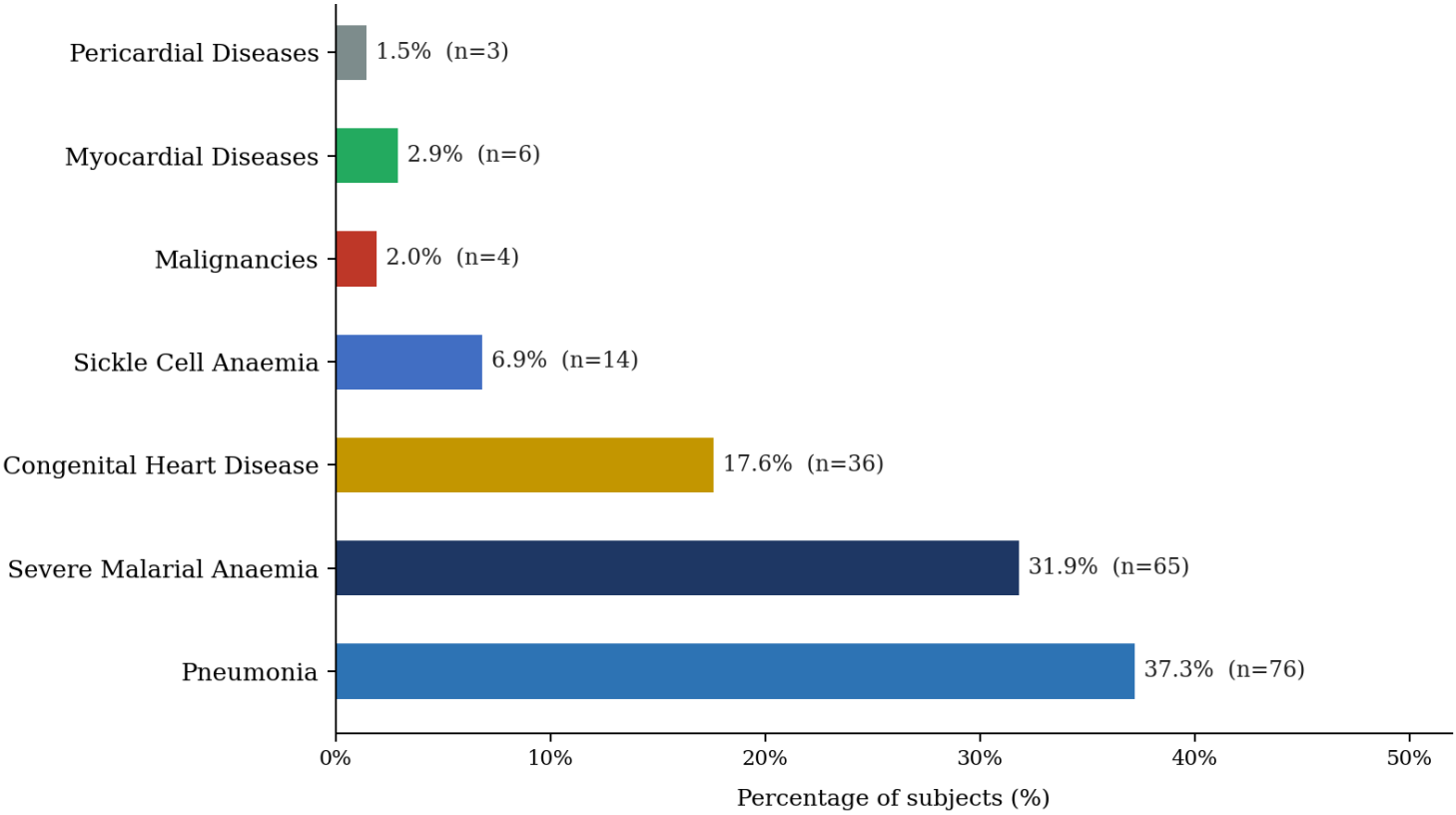


Figure 2

Distribution of subjects by cause of heart failure (N = 204). Severe malarial anaemia was the single most common cause (31.9%).

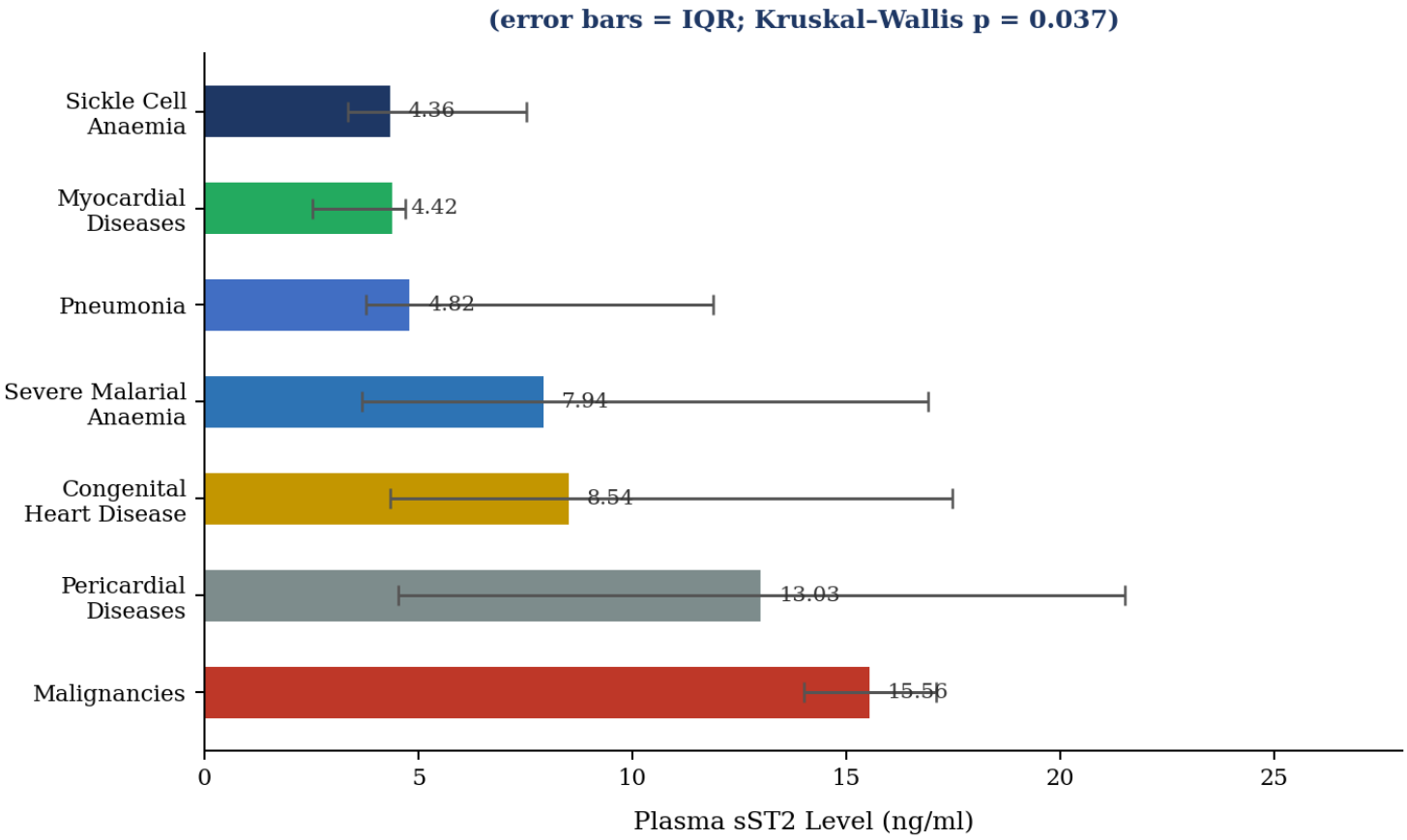


Figure 3

Median plasma sST2 levels by aetiological category

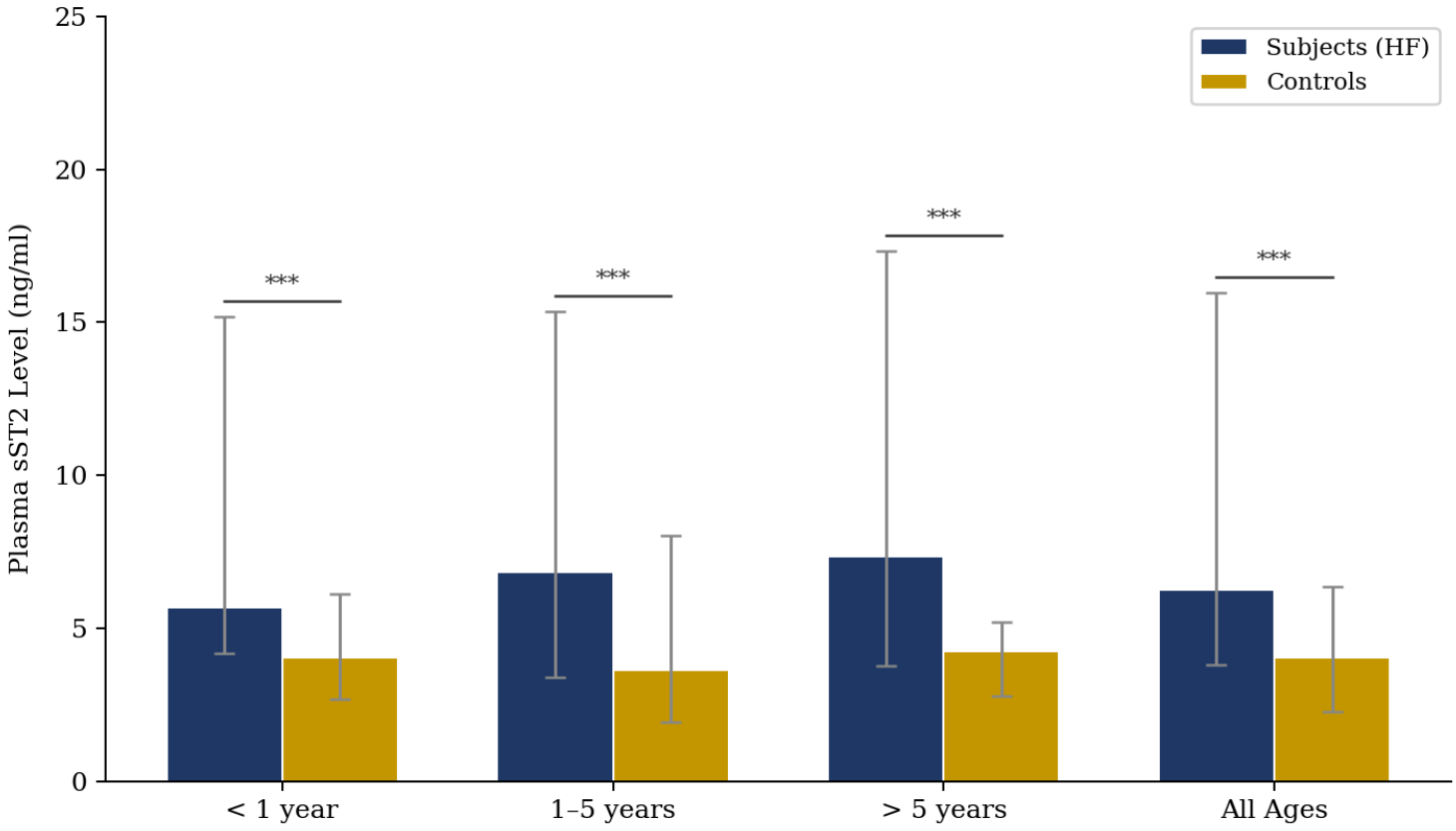


Figure 4

Median plasma sST2 levels by age group — subjects vs. controls (error bars = IQR). *** p < 0.001 for all between-group comparisons (Mann–Whitney U test).

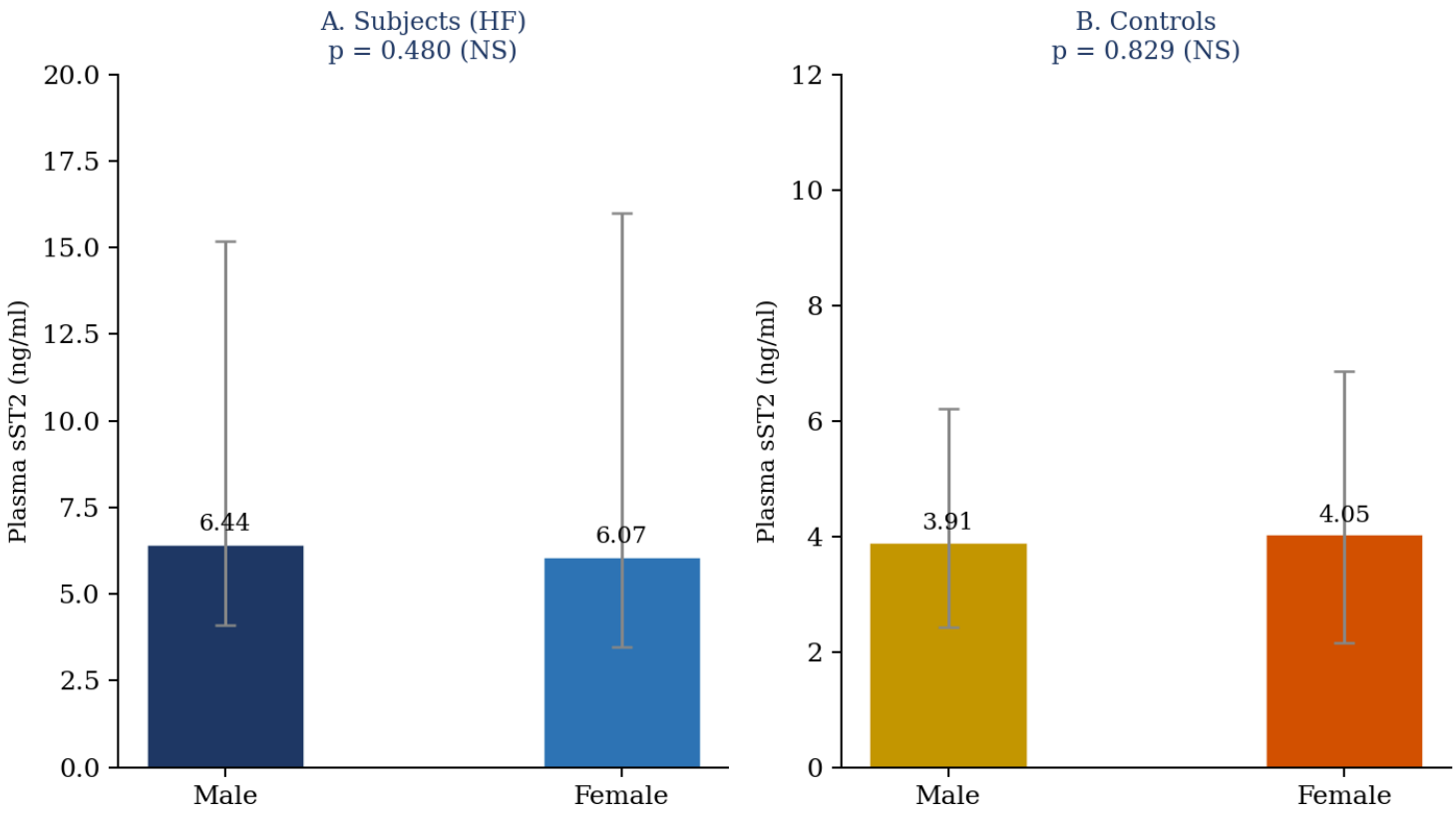


Figure 5

Median plasma sST2 levels by sex in subjects (Panel A) and controls (Panel B) (error bars = IQR). NS = not significant.

Supplementary Files

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