

Prevalence, Patterns, and Factors Associated with Hearing Impairment Among Children and Adolescents with Sickle Cell Disease in Eastern Uganda: A Cross-Sectional Study

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Background: Sickle cell disease (SCD) imposes a substantial burden in sub-Saharan Africa, and the cochlea is vulnerable to the vaso-occlusion and chronic anaemia that characterise the condition. Hearing impairment in children with SCD remains poorly described in Uganda, including the eastern districts around Mbale that carry one of the highest SCD burdens in the country. This study determined the prevalence, patterns, and factors associated with hearing impairment among children and adolescents with SCD attending Mbale Regional Referral Hospital.

Methods: A hospital-based cross-sectional study enrolled 225 children and adolescents aged 5 to 18 years attending the sickle cell clinic in a steady clinical state. An audiologist performed pure-tone audiometry, and hearing impairment was defined as a worse-ear average air-conduction threshold above 20 dB at 500, 1000, and 2000 Hz, following the World Health Organization definition. Factors associated with hearing impairment were examined using Firth penalised logistic regression, with crude and adjusted odds ratios reported with 95% confidence intervals.

Results: Hearing impairment was present in 52 of 225 participants, a prevalence of 23.1% (95% CI 18.1 to 29.0). Impairment was unilateral in 29 and bilateral in 23, and most losses were mild. A central nervous system event (adjusted odds ratio [aOR] 3.54; 95% CI 1.01 to 12.43; $p = 0.049$) and a history of ear infection (aOR 2.42; 95% CI 1.01 to 5.82; $p = 0.048$) were independently associated with hearing impairment. Three or more transfusions showed a comparable association that just missed significance (aOR 2.16; 95% CI 0.99 to 4.73; $p = 0.054$).

Conclusion: One in four children and adolescents with SCD at this referral hospital had hearing impairment, and the affected children carried markers of severe disease and middle-ear pathology. Audiometry should be integrated into routine sickle cell care to detect impairment before it harms language and learning.

Keywords: sickle cell disease, hearing impairment, sensorineural hearing loss, audiometry, children, adolescents, Uganda, sub-Saharan Africa

Introduction

Sickle cell disease (SCD) is the most common inherited haemoglobin disorder worldwide, and its burden falls disproportionately on sub-Saharan Africa. The Global Burden of Disease Study 2021 estimated that 515,000 children

were born with the condition in 2021 and that 7.74 million people were living with it, with about four in five affected births occurring in sub-Saharan Africa.¹ When deaths from all contributing causes are counted rather than only those assigned to a single underlying cause, the total mortality burden reached 376,000 in 2021, nearly eleven times the cause-specific figure, and the gap was widest in sub-Saharan Africa.¹ Uganda carries one of the heaviest burdens on the continent. National surveillance of more than 300,000 infants confirmed a SCD prevalence near 0.9%, and the trait is far more common, reaching 20 to 28% historically in the eastern districts of Mbale and Sironko.^{2,3} Stroke and other central nervous system events are recognised complications of the disease in Ugandan children and add to its neurological morbidity.⁴

The cochlea is richly perfused and metabolically demanding, which makes it susceptible to the same vascular and haematological stresses that characterise SCD. Hearing loss is a large and rising public health problem worldwide. An estimated 1.57 billion people lived with some degree of hearing loss in 2019, and around 97.83 million of those affected were children and adolescents younger than 20 years.^{1,5} The World Health Organization (WHO) defines hearing loss as a threshold poorer than 20 dB in the better ear, and even mild loss at this level can disturb speech perception, schooling, and social development in a growing child.⁶ In low-income and middle-income settings, where specialist ear and hearing services are scarce, much of this burden goes undetected and untreated.^{6,7}

Repeated vaso-occlusion within the cochlear microvasculature, compounded by chronic anaemia and haemolysis, can injure the organ of Corti and the stria vascularis and produce sensorineural hearing loss.⁸ A global meta-analysis of 17 paediatric studies found that children with SCD carried more than three times the risk of sensorineural hearing loss compared with the general population.⁹ Imaging work supports a vascular mechanism, with inner-ear signal abnormalities detectable on magnetic resonance imaging in about a fifth of children, even when they have no symptoms.⁸ The shared blood supply places the vestibular apparatus at similar risk, and a recent systematic review described labyrinthine haemorrhage and dizziness among affected patients.¹⁰ Children with SCD are also prone to pneumococcal infection and recurrent otitis media, which add a conductive pathway to hearing loss, and rare genetic observations such as SLC44A2 deficiency further connect red-cell biology to the preservation of hearing.¹¹

Evidence from Africa, although still limited, points to a substantial and clinically important problem. Comparative studies in Nigeria reported hearing impairment in 35% of children with SCD and confirmed higher odds of loss in those with the homozygous genotype, while a prospective study in northwest Nigeria found bilateral sensorineural hearing loss in 25.6% of affected children and linked it to raised white-cell and platelet counts, poor clinic attendance, and frequent crises.^{12,13} Hearing impairment in this population is not benign, since it is associated with poorer school performance and weaker cognitive function in attention and calculation.¹⁴ Despite this growing concern, the prevalence and correlates of hearing impairment among Ugandan children with SCD have not been described, and routine audiometry is not part of sickle cell care in the region. This study addressed that gap by determining the prevalence, patterns, and factors associated with hearing impairment among children and adolescents attending the sickle cell clinic at Mbale Regional Referral Hospital (MRRH) in eastern Uganda.

Methods

Study Design and Setting

This was a hospital-based cross-sectional study conducted at the sickle cell clinic of MRRH, a public tertiary teaching hospital for Busitema University that serves Mbale city and the surrounding districts of eastern Uganda, about 220 km east of Kampala. The sickle cell clinic runs every Wednesday and attends approximately 130 patients, comprising about 70 children and 60 adolescents, with 450 adolescents and 300 children registered for follow-up.¹⁵ The hospital is a public tertiary teaching hospital for Busitema University and serves the districts of eastern Uganda. The catchment area carries one of the highest sickle cell trait frequencies in the country, which makes the clinic a high-volume site for paediatric and adolescent sickle cell care. Audiometric assessment is not part of the routine clinic package, so participation offered the first formal hearing evaluation for most participants.

Study Population and Eligibility

The study enrolled children and adolescents aged 5 to 18 years with a diagnosis of SCD confirmed by haemoglobin electrophoresis who were in a steady clinical state, defined as the absence of an acute vaso-occlusive crisis, febrile illness, or blood transfusion in the four weeks before assessment. The lower age limit of five years was set because reliable pure-tone audiometry requires a child able to follow test instructions. Children with a known congenital malformation of the ear, a diagnosed non-sickle cause of hearing loss, or those who were critically ill and could not be transferred for hearing testing were excluded, so that the estimated burden reflected SCD rather than unrelated or transient causes.

Sample Size and Sampling

The sample size was calculated using the Cochran formula for a single proportion, with a 95% confidence level and a 5% margin of error, anchored to a hearing impairment prevalence of 17% reported in an earlier Ugandan study.² This yielded a minimum of 217 participants and total of 225 children and adolescents were enrolled.

Data Collection and Measurements

Trained study staff administered a structured, pre-tested, interviewer-administered questionnaire and abstracted clinical and laboratory information from clinic records. Collected variables covered sociodemographic characteristics, housing and environmental exposures, disease history including vaso-occlusive episodes and transfusions in the preceding year, hydroxyurea and sulfadoxine-pyrimethamine use, central nervous system events, and physical findings such as jaundice, pallor, tonsillar enlargement, and adenoid hypertrophy. Laboratory values for haemoglobin, haematocrit, white-cell count, platelets, bilirubin, and the aminotransferases were recorded from the most recent steady-state results.

Each participant underwent otoscopic ear examination followed by pure-tone audiometry, performed by an audiologist with a calibrated Interacoustics audiometer in the quietest available room. Air-conduction thresholds were measured at 500, 1000, 2000, 4000, and 8000 Hz, and bone-conduction thresholds were obtained where indicated. Tympanometry was performed to assess middle-ear function. The pure-tone average across 500, 1000, and 2000 Hz was used to define hearing status. Thresholds were recorded across 500 to 8000 Hz, and the average of 500, 1000, and 2000 Hz was used for classification because these frequencies define the World Health Organization grades of hearing loss, which keeps the estimate comparable with the international literature.

Outcome Definition

Hearing impairment was defined from pure-tone audiometry as a worse-ear average air-conduction threshold above 20 dB across 500, 1000, and 2000 Hz, in line with the WHO definition of disabling and mild hearing loss.⁶ The pure-tone average of each ear was calculated, and the poorer ear determined the participant's hearing status. Among affected participants, impairment was described by laterality, either unilateral or bilateral, and by severity using the standard grades of mild, moderate, and moderately severe or worse.

Statistical Analysis

Data were cleaned and coded in Microsoft Excel and analysed in R. Continuous variables were summarised as the mean with standard deviation or the median with interquartile range, and categorical variables as frequencies and percentages. The few missing values, which affected fewer than 25% of records for every variable, were completed during data preparation using multiple imputation by chained equations, and all descriptive and regression analyses were performed on the completed data. Prevalence was reported with a 95% Wilson confidence interval overall and by age and sex.

Factors associated with hearing impairment were examined with Firth penalised logistic regression, which was chosen because the number of affected children was modest and some categories were sparse. Age was modelled as a continuous term per year, since a test for departure from linearity using a quadratic term was not significant. Each factor was tested unadjusted, and those with a p -value < 0.2 were entered into a multivariable model alongside age and sex, which were retained as confounders. Crude and adjusted odds ratios were estimated with 95% confidence intervals, and a p -value $<$

0.05 was treated as significant. Multiple candidate factors were examined without adjustment for multiplicity, so associations near the significance threshold should be interpreted with corresponding caution.

Results

Sociodemographic Characteristics

The 225 children and adolescents who enrolled and had an interpretable audiogram formed the analytic sample, with a mean age of 11.9 years. Just under half were aged 10 to 14 years ($n = 103$, 45.8%), the cohort was slightly more female ($n = 117$, 52.0%), and most families lived in permanent housing ($n = 156$, 69.3%). More than half of the children shared a sleeping room with more than two people ($n = 131$, 58.2%), and exposure to loud noise was reported for 36 (16.0%). The sociodemographic characteristics are shown in [Table 1](#).

Clinical and Laboratory Characteristics

About seven in ten participants had experienced at least one vaso-occlusive episode in the preceding year, and 71 (31.6%) reported three or more, while half had received at least one blood transfusion and 52 (23.1%) had received three or more. Hydroxyurea use was almost universal ($n = 213$, 94.7%), jaundice was present in 144 (64.0%), and the mean haemoglobin was 8.3 g/dL. Adenoid hypertrophy was present in 83 (36.9%) and tonsillar enlargement in 111 (49.3%), while a central nervous system event had occurred in 12 (5.3%). The clinical and laboratory characteristics are presented in [Table 2](#).

Table 1 Sociodemographic Characteristics of Study Participants ($n = 225$)

Characteristic	Frequency	Percent
Age in years		
Mean (SD)	11.9 (3.7)	
5 to 9	63	28.0
10 to 14	103	45.8
15 to 18	59	26.2
Sex		
Female	117	52.0
Male	108	48.0
Type of house		
Permanent	156	69.3
Semi-permanent	69	30.7
Persons per sleeping room		
Two or fewer	94	41.8
More than two	131	58.2
House ventilation		
Well ventilated	211	93.8
Not well ventilated	14	6.2
Exposure to loud noise		
Yes	36	16.0
No	189	84.0

Abbreviation: SD, standard deviation.

Table 2 Clinical and Laboratory Characteristics of Study Participants (n = 225)

Characteristic	Frequency	Percent
Vaso-occlusive episodes in past year		
None	67	29.8
1 to 2	87	38.7
3 or more	71	31.6
Blood transfusions in past year		
None	112	49.8
1 to 2	61	27.1
3 or more	52	23.1
Hydroxyurea use		
Yes	213	94.7
No	12	5.3
Sulfadoxine-pyrimethamine use		
Yes	220	97.8
No	5	2.2
Central nervous system event		
Yes	12	5.3
No	213	94.7
Symptoms or signs of anaemia		
Yes	99	44.0
No	126	56.0
Jaundice		
Yes	144	64.0
No	81	36.0
Pallor on examination		
Mild	183	81.3
Moderate	30	13.3
Severe	12	5.3
Tonsillar enlargement		
Normal	114	50.7
Large	111	49.3
Adenoid hypertrophy		
Yes	83	36.9
No	142	63.1
History of ear infection		
Yes	29	12.9
No	196	87.1

(Continued)

Table 2 (Continued).

Characteristic	Frequency	Percent
Laboratory indices		
Haemoglobin, g/dL, mean (SD)	8.3 (2.0)	
Haematocrit, %, mean (SD)	26.0 (6.3)	
White cell count, 10 ³ /μL, median (IQR)	12.6 (9.7–16.3)	
Platelets, 10 ³ /μL, median (IQR)	336.3 (235.8–448.6)	
Total bilirubin, mg/dL, median (IQR)	10.2 (7.1–14.1)	
Aspartate aminotransferase, U/L, median (IQR)	23.6 (19.2–34.8)	
Alanine aminotransferase, U/L, median (IQR)	21.1 (17.1–27.7)	

Abbreviations: SD, standard deviation; IQR, interquartile range.

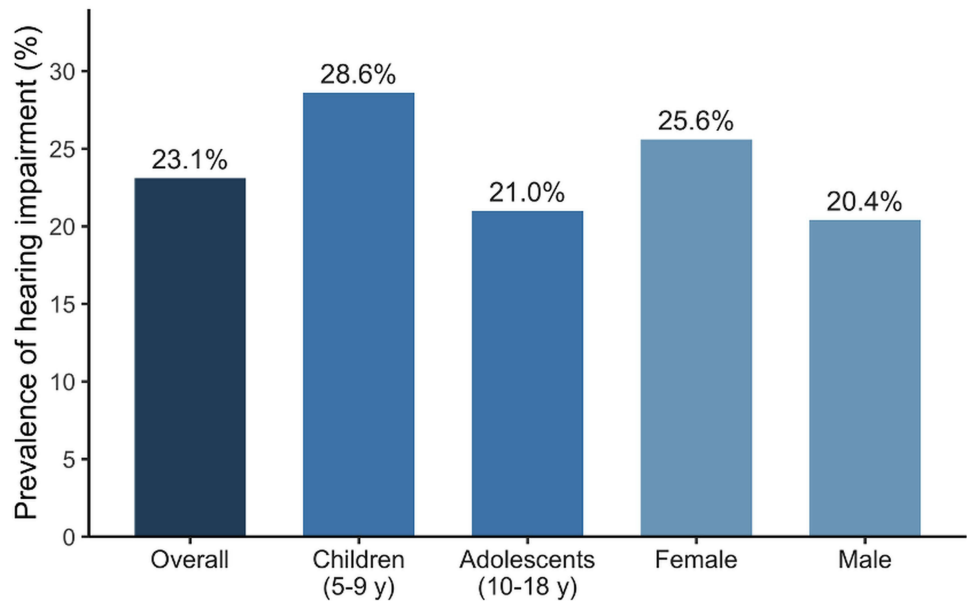


Figure 1 Prevalence of hearing impairment among study participants, overall and by age category and sex.

Prevalence and Patterns of Hearing Impairment

Hearing impairment was present in 52 of the 225 participants, a prevalence of 23.1% (95% CI 18.1 to 29.0). (Figure 1). Among the 52 affected participants, impairment was unilateral in 29 and bilateral in 23, and the mean worse-ear threshold was 35.2 dB. Most losses were mild, with 32 of the 52 affected falling in the 20 to 34 dB range and 8 reaching 50 dB or worse. The detailed patterns are presented in Table 3.

Factors Associated with Hearing Impairment

On bivariate analysis, three factors were associated with hearing impairment. A central nervous system event carried about four times the odds (cOR 3.82; 95% CI 1.19 to 12.34; $p = 0.025$), a history of ear infection about 2.6 times the odds (cOR 2.64; 95% CI 1.16 to 5.99; $p = 0.020$), and receiving three or more blood transfusions in the past year about 2.3 times the odds (cOR 2.32; 95% CI 1.13 to 4.77; $p = 0.022$). Jaundice and moderate or severe pallor showed weaker associations that were not statistically significant, and age, sex, adenoid hypertrophy, crowding, and the remaining factors were not associated.

In the multivariable model, which adjusted for age, sex, blood transfusions, history of ear infection, central nervous system event, jaundice, and moderate or severe pallor, two factors remained independently associated with hearing

Table 3 Patterns of Hearing Impairment Among Affected Participants (n = 52)

Characteristic	Frequency	Percent
Laterality		
Unilateral	29	55.8
Bilateral	23	44.2
Severity (worse-ear pure-tone average)		
Mild (20 to 34 dB)	32	61.5
Moderate (35 to 49 dB)	12	23.1
Moderately severe or worse (≥ 50 dB)	8	15.4

impairment. A central nervous system event carried about 3.5 times the odds (aOR 3.54; 95% CI 1.01 to 12.43; $p = 0.049$), and a history of ear infection about 2.4 times the odds (aOR 2.42; 95% CI 1.01 to 5.82; $p = 0.048$). Three or more blood transfusions carried about twice the odds but was not significant (aOR 2.16; 95% CI 0.99 to 4.73; $p = 0.054$). Age was not associated with the outcome (aOR 0.95 per year; 95% CI 0.87 to 1.04; $p = 0.292$), and sex, jaundice, and pallor were also not significant. The crude and adjusted estimates are presented together in Table 4.

Discussion

This study aimed at determining the prevalence, patterns, and factors associated with hearing impairment among children and adolescents with SCD in eastern Uganda. About one in four children assessed had hearing impairment, the loss was mostly mild and frequently confined to one ear, and the children affected were those who had experienced a central nervous system event or a history of ear infection, with frequent transfusion showing a similar though weaker association.

The prevalence of 23.1% sits within the range reported across paediatric sickle cell populations and close to several recent African estimates. A prospective study in northwest Nigeria found bilateral sensorineural hearing loss in 25.6% of affected children, a Brazilian cohort reported sensorineural loss in 25.8%, and comparative work in Nigeria documented hearing impairment in 35% of children with the disease.^{12–14} Lower estimates have come from cohorts of similar age but with narrower outcome definitions or different genotype mixes. A prospective French series of 89 children aged 5 to 19 years applied the same 20 dB threshold used here and reported hearing impairment in 11.2%, although most of that loss was conductive otitis media with effusion rather than sensorineural, and just over a quarter of the cohort carried the milder HbSC or S β^+ genotype.¹⁶ An earlier Nigerian study of 52 children aged 6 to 19 years reported a 13.4% prevalence when the outcome was restricted to sensorineural hearing loss, against a control rate of 6.2%.¹⁷ The global meta-analysis that pooled these single-site reports placed the risk of sensorineural hearing loss in children with SCD at more than three times that of the general population, which frames the Mbale estimate as consistent with a real and substantial excess rather than an outlier.⁹

The pattern of impairment was predominantly mild and mostly unilateral, which has practical consequences. Mild and one-sided losses are easy to miss in a busy clinic, yet even mild hearing loss can disturb speech processing and classroom learning in a child.⁶ The mean worse-ear threshold of 35.2 dB places a meaningful share of affected children at the boundary where everyday communication begins to suffer. A largely mild and unilateral distribution is consistent with early or slowly progressive cochlear injury, which is the stage at which detection is most useful, because monitoring and support can begin before the loss deepens. Longitudinal audiometric work in this population describes gradual change rather than abrupt deafness, which strengthens the case for periodic rather than one-off assessment.¹⁸

The factors that stood out point toward severe disease and middle-ear pathology rather than toward age or anaemia alone. A central nervous system event was the strongest association, with more than three times the odds of hearing

Table 4 Bivariate and Multivariable Analysis of Factors Associated with Hearing Impairment

Characteristic	No, n (%)	Yes, n (%)	cOR (95% CI)	p	aOR (95% CI)	p
Age, years						
Per one-year increase	12.0 (3.6)	11.7 (4.0)	0.98 (0.90–1.06)	0.573	0.95 (0.87–1.04)	0.292
Sex						
Female	87 (74.4)	30 (25.6)	I		I	
Male	86 (79.6)	22 (20.4)	0.74 (0.40–1.38)	0.341	0.65 (0.33–1.27)	0.208
Type of house						
Permanent	122 (78.2)	34 (21.8)	I			
Semi-permanent	51 (73.9)	18 (26.1)	1.33 (0.69–2.57)	0.394		
Persons per sleeping room						
Two or fewer	77 (81.1)	18 (18.9)	I			
More than two	96 (73.8)	34 (26.2)	1.52 (0.79–2.92)	0.212		
Exposure to loud noise						
No	147 (77.8)	42 (22.2)	I			
Yes	26 (72.2)	10 (27.8)	1.32 (0.58–2.96)	0.507		
Anaemia severity (Hb, g/dL)						
Mild to moderate (≥ 7)	124 (75.6)	40 (24.4)	I			
Severe (< 7)	49 (80.3)	12 (19.7)	0.78 (0.38–1.60)	0.504		
Vaso-occlusive episodes						
None	54 (80.6)	13 (19.4)	I			
1 to 2	68 (78.2)	19 (21.8)	1.15 (0.53–2.51)	0.731		
3 or more	51 (71.8)	20 (28.2)	1.60 (0.73–3.51)	0.242		
Blood transfusions						
None	89 (79.5)	23 (20.5)	I		I	
1 to 2	52 (85.2)	9 (14.8)	0.71 (0.31–1.63)	0.424	0.69 (0.30–1.61)	0.396
3 or more	32 (61.5)	20 (38.5)	2.32 (1.13–4.77)	0.022	2.16 (0.99–4.73)	0.054
Hydroxyurea use						
No	111 (91.7)	1 (8.3)	I			
Yes	162 (76.1)	51 (23.9)	2.38 (0.42–13.44)	0.327		
Central nervous system event						
No	167 (78.4)	46 (21.6)	I		I	
Yes	6 (50.0)	6 (50.0)	3.82 (1.19–12.34)	0.025	3.54 (1.01–12.43)	0.049
Symptoms of anaemia						
No	101 (80.8)	24 (19.2)	I			
Yes	72 (72.0)	28 (28.0)	1.59 (0.84–3.01)	0.156		
Jaundice						
No	67 (83.8)	13 (16.2)	I		I	
Yes	106 (73.1)	39 (26.9)	1.79 (0.87–3.67)	0.112	1.54 (0.71–3.30)	0.272

(Continued)

Table 4 (Continued).

Characteristic	No, n (%)	Yes, n (%)	cOR (95% CI)	p	aOR (95% CI)	p
Pallor, moderate or severe						
No	145 (79.2)	38 (20.8)	I		I	
Yes	28 (66.7)	14 (33.3)	1.84 (0.87–3.86)	0.108	1.85 (0.82–4.18)	0.139
Tonsillar enlargement						
Normal	91 (79.8)	23 (20.2)	I			
Large	82 (73.9)	29 (26.1)	1.42 (0.76–2.66)	0.276		
Adenoid hypertrophy						
No	113 (79.6)	29 (20.4)	I			
Yes	60 (72.3)	23 (27.7)	1.52 (0.78–2.97)	0.220		
History of ear infection						
No	156 (79.6)	40 (20.4)	I		I	
Yes	17 (58.6)	12 (41.4)	2.64 (1.16–5.99)	0.020	2.42 (1.01–5.82)	0.048

Abbreviations: cOR, crude odds ratio; aOR, adjusted odds ratio; CI, confidence interval.

impairment after adjustment, which fits a shared vascular mechanism in which the cerebral and cochlear circulations are injured by the same vaso-occlusive process.^{8,10} The association with three or more transfusions in the preceding year, most plausibly marks disease severity, since frequently transfused children are those with the most crises and complications, and the direction of effect matched the signal from central nervous system events. A history of ear infection roughly doubled the odds of impairment, which is consistent with the conductive pathway that recurrent otitis media and pneumococcal susceptibility add to the sensorineural injury of SCD.¹¹ Reports from Nigeria similarly tied hearing loss to frequent crises, poor clinic attendance, and raised inflammatory indices, and a study in Calabar found type B tympanograms only in children with the disease, which supports a genuine middle-ear contribution.^{13,19} Otitis media is among the leading causes of childhood hearing loss in the region, which makes this conductive pathway both plausible and preventable.⁷

Audiometry is not currently part of routine sickle cell care in this setting, yet nearly a quarter of the children assessed had a detectable hearing deficit, much of it mild and therefore clinically silent. Embedding periodic hearing assessment into sickle cell clinics, with particular attention to children who have had a central nervous system event, who are frequently transfused, or who have a history of ear infection, would allow impairment to be identified while intervention still helps. This matters most in a low-resource setting where undetected hearing loss compounds the educational and social disadvantage that children with a chronic illness already face, and where the association between hearing loss and weaker school performance has already been demonstrated in this population.¹⁴ Pneumococcal vaccination and prompt treatment of otitis media offer a preventable route to reducing the conductive component, and the same audiology pathway could serve the broader population of children with hearing loss in the region, where otitis media remains a leading and treatable cause.^{7,20}

Several limitations shape how these results should be interpreted. The cross-sectional design captures hearing status at a single point and cannot establish whether the associated factors precede or follow the impairment, so the relationships described are associations rather than proven causes. Genotype was not recorded, which prevented comparison between the homozygous and compound heterozygous forms that other studies have found important.¹² Classification of impairment relied on air-conduction thresholds with tympanometry rather than on full bone-conduction audiometry in every child, so severity and laterality were described without separating sensorineural from conductive loss in all cases, and the worse-ear definition, although aligned with the WHO standard, registers unilateral loss that a better-ear definition would not. Recruitment at a single referral hospital may favour children with more severe disease, which could raise the

prevalence relative to a community sample. Missing values were completed during data preparation, which preserved the full sample but carries the usual assumptions of that approach. Audiometry was performed in the quietest available room rather than a sound-treated booth, so ambient noise may have elevated some thresholds and slightly inflated the prevalence, although reported noise exposure was not associated with impairment, which makes noise-induced loss an unlikely explanation. Finally, several adjusted associations clustered close to the conventional significance threshold, so they are best read as a consistent signal toward severe disease and middle-ear pathology rather than as precise effect sizes. The three-frequency pure-tone average follows the World Health Organization definition but is less sensitive to the high-frequency loss that sickle cell disease can cause, so the reported prevalence is conservative, and the high-frequency thresholds already collected will be analysed separately.

Conclusion

Hearing impairment affected about one in four children and adolescents with SCD at this eastern Ugandan referral hospital, and the children most affected were those who had experienced a central nervous system event, who were frequently transfused, or who had a history of ear infection. The losses were mostly mild and often unilateral, which makes them easy to overlook and important to find. Integrating periodic audiometry into routine sickle cell care, alongside attention to preventable middle-ear disease, would allow impairment to be detected and managed before it undermines language, learning, and development. Longitudinal and genotype-resolved studies are now needed to confirm these associations and to clarify the role of hydroxyurea and iron overload in hearing outcomes.

Data Sharing Statement

The de-identified dataset is available from the corresponding author on reasonable request and in accordance with institutional data-sharing policy.

Ethics Approval and Consent to Participate

The study was approved by the Busitema University Faculty of Health Sciences Research and Ethics Committee (approval number BUFHS-2025-641), and administrative clearance to conduct the research was obtained from Mbale Regional Referral Hospital. Written informed consent was obtained from a parent or guardian, and assent was obtained from participants old enough to provide it. The study was conducted in accordance with the Declaration of Helsinki.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agreed to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests.

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