

Comparison of Nasopharyngeal Versus Anterior Nares Sampling for *Staphylococcus aureus* Detection in Children with Sickle Cell Anaemia: A Cross-Sectional Study in Nigeria

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Abstract:

➤ **Background:**

Staphylococcus aureus carriage is a major risk factor for invasive infections in children with sickle cell anaemia (SCA). The optimal sampling site for comprehensive carriage detection in this high-risk population remains undefined. We compared nasopharyngeal versus anterior nares sampling for *S. aureus* and methicillin-resistant *S. aureus* (MRSA) detection.

➤ **Methods:**

This comparative cross-sectional study enrolled 100 children with SCA (aged 1-14 years) attending two tertiary facilities in Kano, Nigeria. Paired nasopharyngeal and anterior nares swabs were collected from each participant and processed using standard microbiological methods. Using combined results from both sites as the reference standard, detection rates, concordance (Cohen's kappa), and sensitivity for each sampling method were calculated.

➤ **Results:**

Overall *S. aureus* carriage was 48.0% (48/100). Nasal sampling detected 41.0% (41/100) versus 38.0% (38/100) for nasopharyngeal sampling ($p=0.66$). Concordance between sites was moderate ($\text{kappa} = 0.58$, 95% CI 0.43-0.73). Nasal sampling demonstrated a sensitivity of 85.4% (95% CI 72.2-93.9%) and specificity of 100%, while nasopharyngeal sampling showed 79.2% (95% CI 65.0-89.5%) sensitivity and 98.1% specificity. Combined sampling increased detection by 17.1% over nasal alone. MRSA prevalence was 7.0% (7/100), with similar detection rates between sites. Nasal sampling was better tolerated (12% vs 28% discomfort, $p=0.006$).

➤ **Conclusion:**

Neither sampling site alone detects all *S. aureus* carriers among children with SCA. For research and comprehensive clinical assessment, combined nasopharyngeal and nasal sampling is recommended. For routine screening in resource-limited settings, nasal sampling alone provides acceptable sensitivity (85%) and superior tolerability.

Keywords: *Staphylococcus aureus*; MRSA; Sampling Method; Nasal Carriage; Nasopharyngeal Carriage; Sickle Cell Anaemia; Paediatric; Nigeria.

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I. INTRODUCTION

Staphylococcus aureus is a leading cause of morbidity and mortality in children with sickle cell anaemia (SCA) worldwide [1,2]. In Nigerian paediatric SCA populations, *S. aureus* accounts for 23-33% of culture-positive infections, including bacteraemia, osteomyelitis, and pneumonia [3-5]. The emergence of methicillin-resistant *S. aureus* (MRSA) has further complicated clinical management, with these strains exhibiting resistance to all beta-lactam antibiotics and frequently demonstrating multidrug resistance [6].

Nasal carriage of *S. aureus* is a well-established risk factor for subsequent invasive infection, and colonised individuals serve as important reservoirs for transmission [7,8]. The anterior nares are considered the primary ecological niche for *S. aureus*, and most carriage surveillance studies have focused on this site [9]. However, accumulating evidence suggests that *S. aureus* can colonise multiple upper respiratory sites, including the nasopharynx and oropharynx, with variable site-specific carriage rates across populations [10].

The nasopharynx deserves particular attention as the portal of entry for respiratory pathogens and the site from which organisms may ascend to cause sinusitis and otitis media or descend to cause pneumonia [11]. Additionally, the nasopharynx is a dynamic microbial ecosystem where bacterial interference among species, including *Streptococcus pneumoniae*, influences colonisation dynamics [12]. The introduction of Pneumococcal conjugate vaccines (PCV) has altered nasopharyngeal microbial composition, with some studies reporting increased *S. aureus* carriage following PCV implementation [13,14].

For children with SCA, who experience functional asplenia and increased susceptibility to bacterial infections, the optimal sampling strategy for *S. aureus* carriage assessment has not been systematically evaluated. This knowledge gap has important implications: epidemiological surveillance studies across Africa have employed inconsistent sampling methods [15-18], rendering direct comparisons difficult; decolonisation protocols targeting only the anterior nares may fail if a substantial proportion of children harbour *S. aureus* exclusively in the nasopharynx [19]; and infection control efforts in outbreak settings require maximising carrier detection to prevent transmission [20].

Previous comparative studies have yielded inconsistent results. In general adult populations, nasal sampling is often considered sufficient for *S. aureus* detection [21], but paediatric studies suggest nasopharyngeal sampling may identify additional carriers [22,23]. In HIV-infected children, nasopharyngeal sampling detected 15% more carriers than nasal sampling alone [24]. No study to date has directly compared sampling sites specifically in children with SCA.

This study therefore aimed to: (1) compare *S. aureus* and MRSA detection rates between paired nasal and nasopharyngeal swabs in children with SCA; (2) determine the sensitivity and specificity of each sampling site using combined results as reference; and (3) evaluate the incremental yield and clinical implications of combined-site sampling.

II. MATERIALS AND METHODS

➤ Study Design and Setting

A comparative cross-sectional study was conducted from September to December 2024 at two healthcare facilities in Kano, Nigeria: Aminu Kano Teaching Hospital (AKTH) and Khalifa Sheikh Isiyaka Rabi Paediatric Hospital (KSIRPH). The study followed the Standards for Reporting Diagnostic Accuracy (STARD) guidelines [25].

Kano State is the most populous state in Nigeria, with approximately 14 million residents, of whom 47% are children under 15 years [26]. AKTH is a 700-bed tertiary hospital serving as the main referral centre for Kano and five neighbouring states, with a paediatric sickle cell clinic of approximately 1,500 registered children. KSIRPH is a state-owned secondary paediatric facility with a sickle cell clinic serving approximately 160 registered children.

➤ Study Population

We included children aged 1-14 years with confirmed haemoglobin SS phenotype attending the sickle cell clinics at AKTH and KSIRPH. All participants were required to be in steady state at recruitment, defined as the absence of acute illness, pain, fever, acute chest syndrome, or other SCA-related complications, and no blood transfusion in the preceding four weeks [27].

- Inclusion criteria: age 1-14 years; confirmed haemoglobin SS phenotype; steady state; parental/guardian written informed consent; written assent from children aged ≥ 7 years.
- Exclusion criteria: systemic antimicrobial use (other than penicillin V prophylaxis) within three months; known immunosuppressive conditions (HIV, malignancies, chronic corticosteroids); hospitalisation within two weeks; acute illness at recruitment; refusal of either sampling procedure; anatomical nasal abnormalities precluding safe swab insertion.

➤ Sample Size Determination

Sample size was calculated to detect a 15% difference in detection rates between sampling sites, based on previous studies showing 10-20% incremental yield from multi-site sampling [24,40]. Assuming a baseline carriage rate of 45% [17] with 80% power and $\alpha=0.05$ (two-tailed), a minimum of

89 participants was required. To account for potential dropouts, we enrolled 100 children.

➤ *Sampling Strategy*

Consecutive sampling was employed. All eligible children presenting during the study period were invited to participate until the target sample size was reached. Proportional allocation was used based on registered children at each site: 90 from AKTH and 10 from KSIRPH.

➤ *Sample Collection Procedures*

Each participant underwent two sampling procedures in random order (computer-generated random sequence). All samples were collected by two trained paediatric research assistants.

- Nasal swab: A sterile cotton swab moistened with sterile normal saline was inserted approximately 1-2 cm into the anterior naris and rotated gently against the nasal mucosa for 5 seconds, completing five full rotations. The same swab was sampled from the other nostril using an identical technique [28].
- Nasopharyngeal swab: With the child's head slightly tilted backwards, a sterile flexible minitip swab (Kingphar Medical Group) moistened with sterile normal saline was inserted through the nostril along the nasal floor until resistance was felt (posterior nasopharyngeal wall). The swab was rotated 180 degrees twice and left in place for 10-15 seconds before withdrawal. The same swab sampled the other nostril [29,30].

Swabs were placed immediately in separate labelled containers with Stuart's transport medium (Micropoint Bioscience, Inc) and transported to the Microbiology Laboratory of AKTH within three hours.

➤ *Laboratory Procedures*

- Isolation and identification: Swabs were inoculated onto Mannitol Salt Agar (Oxoid Ltd, Basingstoke, UK) and incubated aerobically at 35°C for 24-48 hours. Yellow colonies (mannitol fermenters) were subcultured onto blood agar. Gram-positive cocci in clusters showing catalase and coagulase production (tube coagulase test with rabbit plasma) were identified as *S. aureus* [31].
- MRSA identification: Antimicrobial susceptibility testing was performed by Kirby-Bauer disk diffusion on Mueller-Hinton agar following Clinical and Laboratory Standards Institute (CLSI) guidelines [32]. Cefoxitin (30 µg) disks were used; isolates with inhibition zone ≤ 21 mm were phenotypically classified as MRSA [33]. Additional susceptibility testing was performed for erythromycin, clindamycin (including D-zone test), trimethoprim-sulfamethoxazole, tetracycline, and vancomycin (by E-test).
- Quality control: *S. aureus* ATCC 25923 (MSSA) and ATCC 43300 (MRSA) were used as quality control strains. Laboratory personnel were blinded to participant clinical data and to the results of the paired sample from the other anatomical site.

➤ *Data Collection*

Demographic and clinical data were collected using a structured questionnaire: age, sex, ethnicity, socioeconomic status (modified Oyedeji classification [34]), PCV vaccination status (verified from vaccination cards), penicillin V prophylaxis, hospitalisation in the past year, and antibiotic use in the past year (excluding prophylaxis). Tolerability of each sampling procedure was assessed immediately after collection using a 3-point scale (no discomfort, mild discomfort, significant discomfort/pain).

➤ *Statistical Analysis*

Data were analysed using IBM SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were summarised as frequencies and percentages.

- Comparison of detection rates: McNemar's test for paired proportions compared detection rates between sites.
- Concordance: Agreement between sites was assessed using Cohen's kappa coefficient with 95% confidence intervals, interpreted per Landis and Koch [35]: <0.20 poor, 0.21-0.40 fair, 0.41-0.60 moderate, 0.61-0.80 substantial, 0.81-1.00 almost perfect.
- Diagnostic accuracy: Using combined results from both sites as the reference standard for true carriage (a child positive at either site considered a true carrier), we calculated sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for each sampling site with 95% confidence intervals (Wilson score method). We acknowledge that this reference standard is imperfect, as it may miss carriers at other body sites and cannot distinguish true colonisation from contamination, but it represents the best available approach for comparing sampling methods [51].
- Incremental yield: Additional detection from combined sampling was calculated as: (combined positives - site-specific positives) / combined positives $\times 100\%$. The Number needed to screen (NNS) with the additional site to detect one extra carrier was calculated as $1 / (\text{incremental yield})$.
- Subgroup analyses: Detection rates were compared by age group (<5, 5-9, 10-14 years) and sex using Chi-square test. These analyses were exploratory; therefore, p-values were not adjusted for multiple testing, and findings should be interpreted cautiously.

Statistical significance was set at $p < 0.05$ (two-tailed). Complete data were available for all participants; no imputation was required.

➤ *Ethical Considerations*

Ethical approval was obtained from the Health Research Ethics Committee of Aminu Kano Teaching Hospital (Ref: AKTH/HREC/2023/089) and the Kano State Ministry of Health (Ref: MOH/HREC/2023/112). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from parents/guardians, and written assent from children aged ≥ 7 years. Participants with culture-proven *S. aureus* carriage

were prescribed intranasal 2% mupirocin twice daily for five days for decolonisation.

and were included in the final analysis. The mean age was 7.3 ± 3.5 years (range 1-14 years). Females comprised 55.0% (55/100). The majority were of Hausa ethnicity (95.0%) and from a lower socioeconomic class (93.0%). Table 1 presents participant characteristics.

III. RESULTS

➤ Participant Characteristics

A total of 100 children with SCA were enrolled in the study. All participants completed both sampling procedures

Table 1 Baseline Characteristics of Children with Sickle Cell Anaemia (N=100)

Characteristic	Category	n	%
Age group	<5 years	27	27.0
	5-9 years	42	42.0
	10-14 years	31	31.0
Sex	Male	45	45.0
	Female	55	55.0
Ethnicity	Hausa	95	95.0
	Other	5	5.0
Socioeconomic class	Lower	93	93.0
	Middle/Upper	7	7.0
PCV vaccination (≥3 doses)	Yes	83	83.0
Penicillin V prophylaxis	Yes	84	84.0
Hospitalisation (past year)	Yes	51	51.0
Antibiotic use (past year)*	Yes	69	69.0

*Excluding Penicillin V Prophylaxis

➤ Detection Rates by Sampling Site

Using combined results from both sites, *S. aureus* was detected in 48.0% (48/100) of children (95% CI 38.0-58.2%).

Nasal sampling detected *S. aureus* in 41.0% (41/100, 95% CI 31.5-51.2%). Nasopharyngeal sampling detected *S. aureus* in 38.0% (38/100, 95% CI 28.7-48.3%). The difference was not statistically significant (McNemar's test, p=0.66). Table 2 presents the cross-tabulation of results.

Table 2 Cross-Tabulation of *S. aureus* Detection by Sampling Site

	Nasopharyngeal Positive	Nasopharyngeal Negative	Total
Nasal Positive	31	10	41
Nasal Negative	7	52	59
Total	38	62	100

➤ Concordance Between Sampling Sites

Overall agreement was 83.0% (83/100): 31 both positive, 52 both negative. Discordant results occurred in 17.0% (17/100): 10 positives only by nasal swab, 7 positives only by nasopharyngeal swab.

The kappa coefficient was 0.58 (95% CI 0.43-0.73), indicating moderate agreement [35].

➤ Diagnostic Accuracy

Using combined results of both sampling sites as reference (48 true carriers, 52 true non-carriers), Table 3 presents test characteristics for each sampling site.

Table 3 Diagnostic Accuracy of Nasal and Nasopharyngeal Sampling for *S. aureus* Detection

Parameter	Nasal Sampling	Nasopharyngeal Sampling
True positives	41	38
False negatives	7	10
True negatives	52	51
False positives	0	1*
Sensitivity (% , 95% CI)	85.4 (72.2-93.9)	79.2 (65.0-89.5)
Specificity (% , 95% CI)	100 (93.2-100)	98.1 (89.7-99.9)
PPV (% , 95% CI)	100 (91.4-100)	97.4 (85.8-99.9)
NPV (% , 95% CI)	88.1 (78.6-93.8)	83.6 (74.6-89.9)

*Using combined-site results as reference, the child positive only by nasopharyngeal swab is classified as a true positive for nasopharyngeal sampling. The false positive

count shown reflects an alternative classification and is not applicable to our primary analysis.

➤ *MRSA Detection*

MRSA was detected in 7.0% (7/100) of children (95% CI 3.0-14.0%). Among the 7 MRSA carriers, 5 (71.4%) were male, with mean age 5.7 ± 2.8 years. All MRSA isolates were susceptible to vancomycin and trimethoprim-sulfamethoxazole; 4 (57.1%) exhibited inducible clindamycin resistance.

• *By Sampling Site:*

- ✓ Nasal sampling: 6.0% (6/100)
- ✓ Nasopharyngeal sampling: 5.0% (5/100)
- ✓ Four children (4.0%) were positive at both sites
- ✓ Two positives only by nasal swab
- ✓ One positive only by nasopharyngeal swab

Sensitivity for MRSA detection (using combined sites as reference) was 85.7% (6/7, 95% CI 42.1-99.6%) for nasal sampling and 71.4% (5/7, 95% CI 29.0-96.3%) for

nasopharyngeal sampling. These estimates are imprecise due to small numbers.

➤ *Incremental Yield of Combined Sampling*

Compared to nasal sampling alone (41 carriers), adding nasopharyngeal sampling detected an additional 7 carriers, increasing detection by 17.1%. Number needed to screen (NNS) with nasopharyngeal swab in addition to nasal swab to detect one extra carrier: 14.3.

Compared to nasopharyngeal sampling alone (38 carriers), adding nasal sampling detected an additional 10 carriers, increasing detection by 26.3%. NNS with nasal swab in addition to nasopharyngeal swab: 10.0.

➤ *Subgroup Analyses*

Table 4 Detection Rates by Age Group and Sampling Site

Age Group	n	Nasal Positive n (%)	Nasopharyngeal Positive n (%)	Combined Positive n (%)
<5 years	27	9 (33.3)	8 (29.6)	11 (40.7)
5-9 years	42	19 (45.2)	17 (40.5)	22 (52.4)
10-14 years	31	13 (41.9)	13 (41.9)	15 (48.4)

No significant differences in detection rates were observed between age groups or by sex (data not shown). These exploratory analyses were underpowered for definitive conclusions.

➤ *Tolerability and Practical Considerations*

All 100 children completed both procedures. Discomfort (mild or significant) was reported by 12.0% (12/100) for nasal sampling versus 28.0% (28/100) for nasopharyngeal sampling ($p=0.006$). No significant adverse events occurred. Mean sampling time was 30 seconds (range 20-45) for nasal versus 75 seconds (range 45-120) for nasopharyngeal swabs.

Processing samples from both sites required approximately double the laboratory resources compared to a single site. The estimated incremental cost per additional carrier detected by adding nasopharyngeal to nasal sampling was approximately ₦4,200 (~\$5.60 USD) based on local consumables and personnel costs (excluding overheads and equipment depreciation).

IV. DISCUSSION

This direct comparison of nasal and nasopharyngeal sampling for *S. aureus* detection in children with SCA yielded five key findings: (1) overall carriage prevalence of 48.0%; (2) nasal sampling detects slightly more carriers than nasopharyngeal sampling (41.0% vs 38.0%), though the difference is not significant; (3) concordance between sites is moderate (kappa 0.58), with 17% discordance; (4) nasal sampling demonstrates acceptable sensitivity (85.4%) and excellent specificity (100%); and (5) combined sampling increases detection by 17% over nasal alone.

The 48.0% carriage prevalence in our cohort is within the range reported in other sub-Saharan African SCA studies (33.3-57.9%) [15,17]. Variation likely reflects differences in population characteristics, antibiotic exposure, and laboratory methods. Our 7.0% MRSA prevalence aligns with the 5-10% range from recent Nigerian studies [36,37].

The finding that nasal sampling detects slightly more carriers aligns with the anterior nares being the primary *S. aureus* niche [9].

Our observed nasal sampling sensitivity (85.4%) is consistent with a Dutch study in healthy adults, where nasal sampling detected 84% of carriers identified by multi-site sampling [40]. In HIV-infected Ghanaian children, Donkor et al. found nasopharyngeal sampling detected 44.9% carriers versus 41.0% by nasal sampling [24], similar to our pattern.

The moderate concordance (kappa = 0.58) is comparable to other paediatric populations [41] and indicates that the two sites are not interchangeable. Discordant results may arise from true biological variation (site-specific colonisation due to local host factors [42,43]), sampling technique variation [44], density-dependent detection [45], or temporal variation [46]. The finding that 20.8% of carriers were positive only by nasal swab and 14.6% only by nasopharyngeal swab highlights the limitation of single-site sampling.

Routine clinical screening: In resource-limited settings, nasal sampling alone provides acceptable sensitivity (85%) and is simpler, faster, and better tolerated. The 15% false-negative rate means approximately 1 in 7 carriers will be missed—acceptable for population surveillance but potentially problematic for individual patient decisions.

When clinical suspicion is high despite a negative nasal swab, clinicians should consider nasopharyngeal sampling.

Decolonisation protocols: Standard intranasal mupirocin targets the anterior nares. Our finding that 14.6% of carriers harbour *S. aureus* exclusively in the nasopharynx raises the hypothesis that standard decolonisation may fail in these individuals, warranting investigation in prospective decolonisation studies [47]. If confirmed, enhanced protocols incorporating nasopharyngeal decontamination (e.g., chlorhexidine mouthwash) may be necessary [48].

Research studies: Sampling site significantly affects carriage prevalence estimates. Studies using only nasal sampling would report 41% prevalence, those using only

nasopharyngeal sampling 38%, while the true prevalence (by combined sampling) is 48%. This 7-10 percentage point difference must be considered when comparing studies using different methods. Meta-analyses should incorporate the sampling method as a potential source of heterogeneity.

Infection control and MRSA surveillance: In outbreak investigations where maximising carrier detection is critical, combined multi-site sampling should be standard practice [49]. The finding that 3 of 7 MRSA carriers would have been missed by single-site sampling underscores the importance of comprehensive sampling for accurate MRSA surveillance, given the clinical significance of MRSA in SCA.

➤ *Recommended Sampling Strategy*

Table 5 Recommended Sampling Strategy for *S. aureus* Carriage Assessment in Children with SCA

Context	Recommended Sampling	Rationale
Research prevalence studies	Combined nasal + nasopharyngeal	Most accurate estimate
Routine clinical screening	Nasal alone	85% sensitivity, better tolerated
Pre-decolonisation assessment	Combined	Maximise detection of persistent carriage
Outbreak investigation	Combined	Comprehensive carrier detection
MRSA surveillance	Combined	Enhanced detection given clinical significance

This study is, to our knowledge, the first to directly compare nasal and nasopharyngeal sampling specifically in children with SCA in Kano, Nigeria. The paired sampling design eliminates inter-individual variation as a confounder. The sample size (100) was adequately powered for primary comparisons. We used standardised, reproducible microbiological methods with rigorous quality control and assessed multiple outcomes, including diagnostic accuracy, concordance, incremental yield, and tolerability.

V. LIMITATIONS

This study has several limitations. First, sampling was performed at a single time point, and therefore the temporal stability of *Staphylococcus aureus* carriage could not be assessed. Some discordant results between nasal and nasopharyngeal samples may therefore reflect intermittent carriage rather than true site-specific colonisation. Second, quantitative cultures and molecular typing were not performed, limiting the ability to evaluate bacterial density and determine whether isolates recovered from different anatomical sites represented identical strains. Third, the reference standard used in this study—combined nasal and nasopharyngeal results—is imperfect, as *S. aureus* may colonise other unsampled sites such as the oropharynx [53] or skin, potentially leading to misclassification of some carriers. Fourth, the study was conducted during a single season (September–December), and seasonal variation in *S. aureus* carriage has been reported in some settings. [52]. Fifth, participants were recruited from hospital clinics, and the predominance of Hausa ethnicity and lower socioeconomic status among participants may limit the generalisability of the findings to more diverse populations. Finally, oropharyngeal sampling was not included and the number of MRSA carriers was relatively small, limiting the precision of MRSA-specific analyses and the ability to perform detailed subgroup analyses.

Despite these limitations, this study provides the most comprehensive evidence to date on optimal sampling strategies for *S. aureus* carriage assessment in children with SCA.

VI. CONCLUSIONS

This study demonstrates that neither nasal nor nasopharyngeal sampling alone detects all *S. aureus* carriers among children with SCA in Nigeria. Nasal sampling detects slightly more carriers (41% vs 38%) with acceptable sensitivity (85.4%) and is better tolerated. Combined sampling increases detection by 17% and should be preferred for research, decolonisation assessment, and outbreak investigations. For routine screening in resource-limited settings, nasal sampling alone provides a pragmatic balance of sensitivity, tolerability, and cost-effectiveness. These findings have important implications for prevalence studies, decolonisation protocols, infection control practices, and clinical care in this high-risk paediatric population.

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➤ *Author Contributions*

YYO, FHH, BAI and SUA: Conceptualisation. YYO, FHH, BAI, SUA, MOY and IAI: Methodology. YYO, BAI, MOY, IAI: supervision of data collection and laboratory procedures. YYO, BAI, IKM and LIA: Statistical analysis. YYO and BAI drafted the initial manuscript. YYO, FHH,

BAI and SUA: Resources. FHH, SUA and BAI: Supervision. YYO: Project administration. YYO, MOY, IAI: Data curation. YYO, FHH, BAI, SUA, MOY, IAI, IKM and LIM: Writing- review and editing. All authors have read and approved the final version of the manuscript.

➤ *Conflict of Interest Statement*

The authors declare no conflicts of interest.

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➤ *Data Availability Statement*

The data supporting this study's findings are available from the corresponding author upon reasonable request.

➤ *Ethics Approval*

This study was approved by the Health Research Ethics Committee of Aminu Kano Teaching Hospital (Ref: AKTH/HREC/2023/089) and the Kano State Ministry of Health (Ref: MOH/HREC/2023/112).

➤ *Patient Consent*

Written informed consent was obtained from parents/guardians of all participants, and written assent was obtained from children aged 7 years and above.

REFERENCES

[1]. Booth C, Inusa B, Obaro SK. Infection in sickle cell disease: A review. *Int J Infect Dis.* 2010;14(1): e2-e12.

[2]. Ochocinski D, Dalal M, Black LV, et al. Life-threatening infectious complications in sickle cell disease: A concise narrative review. *Front Pediatr.* 2020; 8:38.

[3]. Brown B, Dada-Adegbola H, Trippe C, Olopade O. Prevalence and etiology of bacteremia in febrile children with sickle cell disease at a Nigeria tertiary hospital. *Mediterr J Hematol Infect Dis.* 2017;9(1): e2017039.

[4]. Musa A, Ogunrinde OG, Mamman AI, Saad YM, Ibrahim A, Yakubu AM. Prevalence and pattern of bacterial isolate in febrile children with sickle cell anemia in a tertiary hospital in Northern Nigeria. *Sub-Saharan Afr J Med.* 2018;5(3):80-85.

[5]. Ibrahim HA, Yakubu YM, Farouk AG, Ambe P, Gadzama GB. Profile of bacterial pathogens causing infections in children with sickle cell anaemia in Maiduguri. *Niger Postgrad Med J.* 2021;28(3):218-224.

[6]. Lakhundi S, Zhang K. Methicillin-resistant *Staphylococcus aureus*: Molecular characterization, evolution, and epidemiology. *Clin Microbiol Rev.* 2018;31(4): e00020-18.

[7]. Raineri EJM, Altulea D, van Dijnl JM. Staphylococcal trafficking and infection—from 'nose to gut' and back. *FEMS Microbiol Rev.* 2022;46(1): fuab041.

[8]. Shankar N, Soe PM, Tam CC. Prevalence and risk of acquisition of methicillin-resistant *Staphylococcus*

aureus among households: A systematic review. *Int J Infect Dis.* 2020; 92:105-113.

[9]. Sollid JUE, Furberg AS, Johannessen M, Hanssen AM. *Staphylococcus aureus*: Determinants of human carriage. *Clin Microbiol Infect.* 2014;20(6):531-541.

[10]. Mertz D, Frei R, Jaussi B, et al. Throat swabs are necessary to reliably detect carriers of *Staphylococcus aureus*. *Clin Infect Dis.* 2007;45(4):475-477.

[11]. González-García S, Hamdan-Partida A, Bustos-Hamdan A, Bustos-Martínez J. Factors of nasopharynx that favour the colonization and persistence of *Staphylococcus aureus*. In: *Pharynx - Diagnosis and Treatment.* London: IntechOpen; 2021:1-22.

[12]. Pettigrew MM, Laufer AS, Gent JF, Kong Y, Fennie KP, Metlay JP. Upper respiratory tract microbial communities, acute otitis media pathogens, and antibiotic use in healthy and sick children. *Appl Environ Microbiol.* 2012;78(17):6262-6270.

[13]. Rocha LC, Carvalho MOS, Nascimento VML, et al. Nasopharyngeal and oropharyngeal colonization by *Staphylococcus aureus* and *Streptococcus pneumoniae* and prognostic markers in children with sickle cell disease from the Northeast of Brazil. *Front Microbiol.* 2017; 8:217.

[14]. Kielbik K, Pietras A, Jablonska J, et al. Impact of pneumococcal vaccination on nasopharyngeal carriage of *Streptococcus pneumoniae* and microbiota profiles in preschool children in South East Poland. *Vaccines (Basel).* 2022;10(5):791.

[15]. Appiah VA, Pesewu GA, Kotey FCN, et al. *Staphylococcus aureus* nasal colonization among children with sickle cell disease at the Children's Hospital, Accra: Prevalence, risk factors, and antibiotic resistance. *Pathogens.* 2020;9(5):329.

[16]. Schaumburg F, Biallas B, Feugap EN, et al. Carriage of encapsulated bacteria in Gabonese children with sickle cell anaemia. *Clin Microbiol Infect.* 2013;19(3):235-241.

[17]. Dayie NTKD, Sekoh DNK, Kotey FCN, et al. Nasopharyngeal carriage of methicillin-resistant *Staphylococcus aureus* (MRSA) among sickle cell disease (SCD) children in the pneumococcal conjugate vaccine era. *Infect Dis Rep.* 2021;13(1):191-204.

[18]. Mutagonda RF, Bwire G, Sangeda RZ, et al. Nasopharyngeal carriage and antibiogram of pneumococcal and other bacterial pathogens from children with sickle cell disease in Tanzania. *Infect Drug Resist.* 2022; 15:4407-4418.

[19]. Liu C, Bayer A, Cosgrove SE, et al. Clinical practice guidelines by the Infectious Diseases Society of America for the treatment of methicillin-resistant *Staphylococcus aureus* infections in adults and children: Executive summary. *Clin Infect Dis.* 2011;52(3):285-292.

[20]. Calfee DP, Salgado CD, Milstone AM, et al. Strategies to prevent methicillin-resistant *Staphylococcus aureus* transmission and infection in acute care hospitals: 2014 update. *Infect Control Hosp Epidemiol.* 2014;35(7):772-796.

- [21]. Kluytmans J, van Belkum A, Verbrugh H. Nasal carriage of *Staphylococcus aureus*: Epidemiology, underlying mechanisms, and associated risks. *Clin Microbiol Rev.* 1997;10(3):505-520.
- [22]. Nilsson P, Ripa T. *Staphylococcus aureus* throat colonization is more frequent than colonization in the anterior nares. *J Clin Microbiol.* 2006;44(9):3334-3339.
- [23]. Lautenbach E, Nachamkin I, Hu B, et al. Surveillance cultures for detection of methicillin-resistant *Staphylococcus aureus*: Diagnostic yield of anatomic sites and comparison of provider- and patient-collected samples. *Infect Control Hosp Epidemiol.* 2009;30(4):380-382.
- [24]. Donkor ES, Kotey FCN, Dayie NTKD, et al. Colonization of HIV-infected children with methicillin-resistant *Staphylococcus aureus*. *Pathogens.* 2019;8(1):35.
- [25]. Cohen JF, Korevaar DA, Altman DG, et al. STARD 2015 guidelines for reporting diagnostic accuracy studies: Explanation and elaboration. *BMJ Open.* 2016;6(11):e012799.
- [26]. National Bureau of Statistics. Demographic Statistics Bulletin 2020. Abuja: NBS; 2020.
- [27]. Ismail A, Yusuf AA, Kuliya-Gwarzo A, Ahmed SG, Tabari AM, Abubakar SA. Correlating transcranial arterial doppler velocities with haematologic parameters and haemolytic indices of Nigerian children with sickle cell anaemia. *Ultrasound.* 2019;27(2):101-110.
- [28]. Centers for Disease Control and Prevention. Nasal swab collection for *Staphylococcus aureus*. Atlanta: CDC; 2020.
- [29]. World Health Organization. Manual for the laboratory identification and antimicrobial susceptibility testing of bacterial pathogens of public health importance in the developing world. Geneva: WHO; 2003.
- [30]. Gurung RR, Maharjan P, Chhetri GG. Antibiotic resistance pattern of *Staphylococcus aureus* with reference to MRSA isolates from pediatric patients. *Future Sci OA.* 2020;6(4):FSO464.
- [31]. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 32nd ed. CLSI supplement M100. Wayne, PA: CLSI; 2022.
- [32]. Boutiba-Ben Boubaker I, Ben Abbes R, Ben Abdallah H, et al. Evaluation of a cefoxitin disk diffusion test for the routine detection of methicillin-resistant *Staphylococcus aureus*. *Clin Microbiol Infect.* 2004;10(8):762-765.
- [33]. Oyedeji GA. Socio-economic and cultural background of hospitalised children in Ilesha. *Niger J Paediatr.* 1985;12(4):111-117.
- [34]. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics.* 1977;33(1):159-174.
- [35]. Okwu MU, Olley M, Akpoka AO, Izevbuwa OE. Methicillin-resistant *Staphylococcus aureus* (MRSA) among patients with sickle cell disease in a tertiary hospital in Benin City, Nigeria. *J Infect Dev Ctries.* 2020;14(5):493-499.
- [36]. Ogunrinde OG, Kuti KM, Anoba S, Oseni SB. Nasal carriage of *Staphylococcus aureus* and methicillin-resistant *S. aureus* among children with sickle cell disease in Zaria, Nigeria. *Niger J Paediatr.* 2021;48(2):87-92.
- [37]. Krismmer B, Weidenmaier C, Zipperer A, Peschel A. The commensal lifestyle of *Staphylococcus aureus* and its interactions with the nasal microbiota. *Nat Rev Microbiol.* 2017;15(11):675-687.
- [38]. Bogaert D, van Belkum A, Sluifster M, et al. Colonisation by *Streptococcus pneumoniae* and *Staphylococcus aureus* in healthy children. *Lancet.* 2004;363(9424):1871-1872.
- [39]. Bode LG, Kluytmans JA, Wertheim HF, et al. Preventing surgical-site infections in nasal carriers of *Staphylococcus aureus*. *N Engl J Med.* 2010;362(1):9-17.
- [40]. Lebon A, Labour JA, Verbrugh HA, et al. Dynamics and determinants of *Staphylococcus aureus* carriage in infancy: The Generation R Study. *J Clin Microbiol.* 2008;46(10):3517-3521.
- [41]. Kaspar U, Kriegeskorte A, Schubert T, et al. The culturome of the human nose habitats reveals individual bacterial fingerprint patterns. *Environ Microbiol.* 2016;18(7):2130-2142.
- [42]. Lemon KP, Klepac-Ceraj V, Schiffer HK, Brodie EL, Lynch SV, Kolter R. Comparative analyses of the bacterial microbiota of the human nostril and oropharynx. *mBio.* 2010;1(3):e00129-10.
- [43]. Warnke P, Warning L, Podbielski A. Some are more equal—A comparative study on swab types and detection agents for *Staphylococcus aureus* and *Pseudomonas aeruginosa* biofilms on medical implant materials. *Eur J Clin Microbiol Infect Dis.* 2015;34(9):1829-1836.
- [44]. VandenBergh MF, Yzerman EP, van Belkum A, Boelens HA, Sijmons M, Verbrugh HA. Follow-up of *Staphylococcus aureus* nasal carriage after 8 years: Redefining the persistent carrier state. *J Clin Microbiol.* 1999;37(10):3133-3140.
- [45]. van Belkum A, Verkaik NJ, de Vogel CP, et al. Reclassification of *Staphylococcus aureus* nasal carriage types. *J Infect Dis.* 2009;199(12):1820-1826.
- [46]. Septimus EJ, Schweizer ML. Decolonization in prevention of health care-associated infections. *Clin Microbiol Rev.* 2016;29(2):201-222.
- [47]. Simor AE, Phillips E, McGeer A, et al. Randomized controlled trial of chlorhexidine gluconate for washing, intranasal mupirocin, and rifampin and doxycycline versus no treatment for the eradication of methicillin-resistant *Staphylococcus aureus* colonization. *Clin Infect Dis.* 2007;44(2):178-185.
- [48]. Siegel JD, Rhinehart E, Jackson M, Chiarello L; Healthcare Infection Control Practices Advisory Committee. Management of multidrug-resistant organisms in health care settings, 2006. *Am J Infect Control.* 2007;35(10 Suppl 2):S165-S193.
- [49]. Goss CH, Muhlebach MS. Review: *Staphylococcus aureus* and MRSA in cystic fibrosis. *J Cyst Fibros.* 2011;10(5):298-306.

- [50]. Rutjes AW, Reitsma JB, Coomarasamy A, Khan KS, Bossuyt PM. Evaluation of diagnostic tests when there is no gold standard: A review of methods. *Health Technol Assess.* 2007;11(50):iii, ix-51.
- [51]. Bojang A, Kendall L, Usuf E, et al. Prevalence and risk factors for *Staphylococcus aureus* nasopharyngeal carriage during a PCV trial. *BMC Infect Dis.* 2017;17(1):588.
- [52]. Marshall C, Spelman D. Re: Is throat screening necessary to detect methicillin-resistant *Staphylococcus aureus* colonization in patients upon admission to an intensive care unit? *J Clin Microbiol.* 2007;45(11):3855.