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Determinants of MRSA Colonization and Presence of the Panton-Valentine Leukocidin Gene (LUK-PVL) Among MRSA Isolates from People Living with HIV/AIDS at Irrua Specialist Teaching Hospital, Edo State, Nigeria

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Abstract **Original Research Article**

Background: Understanding which host characteristics predict Methicillin-resistant *Staphyloccus aureus* (MRSA) colonisation status and multi-site burden is essential for designing targeted infection-control strategies among people living with HIV/AIDS (PLWH). Concurrently, the Panton-Valentine Leukocidin gene (LUK-PVL), the molecular marker of hypervirulent communitY-associated MRSA (CA-MRSA), has not been characterized in any PLWH cohort from Edo State, Nigeria. This study investigates the participant-level and multi-site determinants of MRSA colonisation, and characterizes LUK-PVL prevalence and the clinical profile of PVL-positive isolates, in 176 PLWH at Irrua Specialist Teaching Hospital (ISTH).

Methods: 176 PLWH were screened for MRSA using three-site swabbing (nasal, axilla, groin; 528 specimens); 131 (74.43%) were MRSA-positive, yielding 230 isolates. Determinants of MRSA carriage status (N=176 participants) and multi-site colonization (≥2 positive sites, n=148 recoverable profiles) were assessed by chi-square, Mann-Whitney U, and binary logistic regression. LUK-PVL was detected by multiplex PCR (McClure protocol; amplicon 433 bp) in all 230 isolates.

Results: MRSA carriage was significantly associated with age group (χ^2=9.900, p=0.042), occupation (χ^2=7.173, p=0.047), WHO clinical stage (χ^2=3.663, p=0.040), and CD4 count category (χ^2=3.824, p=0.047). A clear immunosuppression gradient was observed: MRSA positivity was 86.5% at CD4 <200 cells/µL, declining to 69.6% at CD4 >500. Multi-site colonisation (41.2% of profiles) was independently predicted by hospitalisation within 6 months (aOR=4.30, p=0.011), within 12 months (aOR=4.59, p=0.010), and younger age (aOR=0.97/year, p=0.046); CD4 count was non-predictive (p=0.794). LUK-PVL was detected in 1 of 230 isolates (0.43%), from a recently hospitalised male with preserved CD4 (680 cells/µL), active skin infection, and a mecA+/SCCmecV+/SCCmecII− profile consistent with CA-MRSA.

Conclusion: MRSA colonisation in PLWH is determined by immune status at the level of initial carriage acquisition, and by healthcare contact at the level of multi-site dissemination. The near-absence of PVL confirms predominantly HA-MRSA lineage circulation. These findings support CD4-stratified MRSA screening and hospitalisation-linked decolonisation as the most targeted preventive strategies in this population.

Keywords: LUK-PVL, Panton-Valentine leukocidin, MRSA determinants, multi-site colonization, CD4 count, WHO clinical stage, hospitalization, PLWH, HIV, Nigeria, Edo State, logistic regression.

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INTRODUCTION

Methicillin-resistant *Staphyloccus aureus* (MRSA) colonisation in PLWH operates at levels that are not governed by the same factors. Whether a person acquires MRSA at all appears to be driven largely by immune status: progressive CD4 depletion, impairs epidermal defence, reduces IL-17-mediated antimicrobial peptide production, and diminishes *S. aureus* clearance from colonised skin, creating conditions that favour persistent MRSA carriage.[1,2] Whether that colonisation then spreads to multiple body sites is a different question, shaped more by exposure to nosocomial MRSA reservoirs and healthcare procedures that disrupt skin integrity than by baseline immune function.[3,4]

Evidence specifically linking CD4 count to MRSA carriage risk in African PLWH cohorts is limited. Crum-Cianflone et al.[5] observed an inverse CD4-MRSA relationship in US military HIV outpatients but not inpatients, suggesting the association may depend on the healthcare setting and exposure intensity. Several sub-Saharan African studies have reported overall MRSA prevalence in PLWH without CD4 stratification, making immune-dose comparisons impossible. Whether CD4 count consistently and dose-dependently predicts MRSA carriage in Nigerian Antiretroviral (ART) clinic settings has therefore remained unanswered. Similarly, whether hospitalisation specifically drives multi-site, rather than single-site, colonisation burden in this population has not been examined.

The Panton-Valentine Leukocidin toxin, encoded by lukF-PV and lukS-PV (collectively LUK-PVL), is a pore-forming cytotoxin that targets neutrophils and macrophages.[6,7] Strains carrying LUK-PVL are associated with necrotising skin and soft tissue infections, recurrent furunculosis, and haemorrhagic pneumonia, and are the virulence hallmark of major CA-MRSA lineages including USA300, USA400, and several African ST8 and ST88 strains.[8–12] In PLWH, whose neutrophil function is already compromised by HIV and antiretroviral effects, PVL-carrying MRSA may cause more severe infection than in immunocompetent hosts.[13] PVL prevalence in community MRSA from African studies ranged mostly from 30% to over 80%, reflecting the wide circulation of CA-MRSA lineages across the continent.[14,15]

This study characterises the sociodemographic, clinical, and immunological determinants of MRSA carriage at the participant level among 176 PLWH; the determinants of multi-site MRSA colonisation among recoverable participant profiles; and the prevalence and clinical profile of LUK-PVL gene carriage among 230 MRSA isolates.

METHODS

Study design, site, and participants

This study was conducted at the Department of Medical Microbiology and Parasitology, Irrua Specialist Teaching Hospital (ISTH), Irrua, Edo State, Nigeria. ISTH operates an ISO 9001-certified Medical Microbiology Laboratory and one of the South-South zone's largest ART clinics, with 7,903 attendances recorded in 2019. A cross-sectional hospital-based study enrolled 176 PLWH on ART for ≥6 months by systematic random sampling. Three rayon swabs per participant were collected from the nasal vestibule, axillary fold, and groin (528 specimens). MRSA was confirmed by cefoxitin disc diffusion per CLSI 2021 guidelines; 131 participants (74.43%) were MRSA-positive, yielding 230 confirmed MRSA isolates from three sites. Ethical approval was obtained from the ISTH Ethics and Research Committee; written informed consent was obtained from all participants.

Sample size and sampling

Sample size was calculated using the standard formula for proportions in large populations (N > 10,000): $N = Z^2pq/d^2$. Using Z = 1.96 (95% confidence interval), p = 0.161 (prevalence of MRSA in PLWH reported from Port Harcourt, Nigeria[19], used as the best available Nigerian estimate), q = 0.839, and d = 0.05 (precision),

the calculation yielded N = 207.6. An additional 10% was added to account for attrition (n = 20.76), resulting in a minimum sample size of 228.36, rounded up to 230.

Participants were recruited by systematic random sampling with a k-value of 5.4, calculated from the average number of ART clinic patients per three-month study period (approximately 1,248) divided by the target sample size (230). In practice, every fifth eligible patient was enrolled. The starting point was selected by ballot (number 3), and subsequent patients were selected at five-patient intervals across four clinic days per week.

Eligibility criteria

Inclusion criteria: PLWH aged 2 years and above, attending the ART clinic, and on antiretroviral therapy for six months or longer, regardless of sex.

Exclusion criteria: Patients on Highly Active Antiretroviral Therapy (HAART) for less than six months at the time of enrolment. This cut-off was applied to ensure a minimum period of immune reconstitution and to exclude early-treatment immune dysregulation that might confound MRSA acquisition risk.

Ethical considerations

Ethical approval was obtained from the Ethics and Research Committee of Irrua Specialist Teaching Hospital before commencement. Written informed consent was obtained from all adult participants before enrolment. For paediatric participants (age <18 years), written parental/guardian consent and child assent were obtained. All participants were interviewed in a private, screened examination room to ensure confidentiality and minimise stigmatisation. Participant identities were replaced with numeric codes throughout data collection and analysis. No personal identifiers were retained in the research database. All COVID-19 infection control measures were maintained throughout the study period. Research costs were fully borne by the investigators.

Bacterial culture and DNA extraction

MRSA isolates were stored at −70°C in 16% glycerol broth immediately after primary identification. For PCR analysis, each isolate was sub-cultured from glycerol stock onto mannitol salt agar (MSA) and incubated aerobically at 35°C for 18–24 hours to obtain viable single colonies. A single well-isolated colony morphologically consistent with S. aureus (golden-yellow, mannitol-fermenting) was selected and inoculated into 1 mL of tryptic soy broth, then incubated overnight at 35°C on an orbital shaker at 150 rpm to obtain a bacterial suspension of adequate density for DNA extraction.

DNA was extracted from 1 mL of overnight broth culture using the Norgen Biotek Bacteria and Fungi Genomic DNA Purification Kit (Norgen Biotek Corp., Thorold, Canada) following the manufacturer's standard protocol for Gram-positive organisms. Briefly, this involved enzymatic cell lysis with lysozyme (50 mg/mL) at 37°C for 30 minutes, followed by proteinase K digestion, binding to a silica spin column, washing with proprietary wash buffers to remove inhibitors, and elution with 50 µL of RNase-free elution buffer. Extracted DNA quantity and purity were assessed spectrophotometrically using a NanoDrop instrument; samples with A260/A280 ratios between 1.7 and 2.0 were accepted for PCR. Extracted DNA was stored at −20°C until use.

mecA PCR protocol

Polymerase Chain Reaction (PCR) amplification of the mecA gene was performed in a final volume of 25 µL comprising: 5 µL of 5X FIREPol Blend Master Mix (Solis Biodyne, Tartu, Estonia; containing 1.5 mM $MgCl_2$, dNTPs at 200 µM each, and 2 units of Hot FIREPol DNA polymerase), 25 pmol of each primer (mecA forward: 5'-GTGAAGATATACCAAGTGATT-3'; mecA reverse: 5'-ATGCGCTATAGATTGAAAGGAT-3'; expected amplicon 147 bp), 5 µL of extracted template DNA, and nuclease-free water to volume.

Amplification was performed in an Eppendorf Vapo Protect Nexus Series thermocycler with the following programme: initial denaturation at 95°C for 5 minutes; 30 amplification cycles of denaturation at 95°C for 30 seconds, annealing at 52°C for 60 seconds, and extension at 72°C for 90 seconds; followed by a final extension at 72°C for 10 minutes and a hold at 4°C. Each PCR batch included a positive control (S. aureus ATCC 700699, mecA-positive) and a negative control (S. aureus ATCC 29213, mecA-negative). Batches in which either control failed were invalidated and repeated.

Gel electrophoresis

PCR products were resolved on 1.5% agarose gels prepared in 1× TAE buffer (40 mM Tris, 20 mM acetic acid, 1 mM EDTA, pH 8.0), cast in a horizontal electrophoresis tank at room temperature. A 100-bp DNA ladder (Solis Biodyne, Tartu, Estonia) was loaded in the first lane as molecular weight reference. Electrophoresis was conducted at 80 V for 90 minutes. Gels were stained with ethidium bromide (0.5 µg/mL) for 20 minutes and de-stained in distilled water for 15 minutes before visualisation under UV transillumination. Band sizes were confirmed against the 100-bp ladder; a band at 147 bp was recorded as mecA-positive.

LUK-PVL PCR

The Panton-Valentine Leukocidin gene (LUK-PVL) was detected by PCR on all 230 MRSA isolates using primers: forward 5'-ATCAATAGGTAAAATGTCTGGACATGATCCA-3', reverse 5'-GCATCAAATGTATTGGATAGAAAAGC-3' (expected amplicon: 433 bp).[4] Reactions used Solis Biodyne 5X FIREPol Blend Master Mix in 25-µL volumes. Thermal cycling (Eppendorf Vapo Protect): 95°C 5 min; 30 cycles of [95°C 30 s, 55°C 60 s, 72°C 90 s]; 72°C 10 min. Each run included a PVL-carrying *S. aureus* positive control and a mecA-negative negative control. Products were resolved on 1.5% agarose gels (80V, 90 min) with ethidium bromide staining and UV visualisation.

Statistical analysis

Participant-level MRSA determinants (N=176) were assessed by Pearson chi-square and Fisher's exact test for categorical variables and Mann-Whitney U for continuous variables (CD4+ count, age; Shapiro-Wilk $p<0.001$ for both, confirming non-normality).

Multi-site colonisation determinants used identical bivariate methods, followed by binary logistic regression (outcome: multi-site [≥2 sites] vs single-site). Variables with bivariate $p<0.20$ were entered simultaneously into the multivariable model. Model fit was assessed by AIC, McFadden pseudo-R^2, and Nagelkerke R^2. Given that only 1 of 230 isolates was LUK-PVL positive, no formal statistical association analysis was performed for LUK-PVL; the isolate is described as a case profile. All analyses used SPSS v27.0 (IBM Corp.) at $\alpha=0.05$ (two-tailed).

RESULTS

Participant-level determinants of MRSA carriage

Table 1 presents bivariate analyses. Age group (χ^2 = 9.900, p = 0.042) and occupation (χ^2 = 7.173, p = 0.047) were significant sociodemographic predictors. MRSA positivity was highest among participants aged 21–30 years (85.7%) and lowest among those older than 60 years (50.0%). Among occupational categories, skilled workers had the highest MRSA positivity (90.3%), followed by professionals (84.2%) and unskilled workers (69.6%). Sex, marital status, educational level, and residential area were not significantly associated with MRSA carriage status.

Among immunological and clinical variables, both WHO clinical stage (χ^2=3.663, p=0.040) and CD4 count category (χ^2=3.824, p=0.047) were significantly associated with MRSA carriage (Figure 1). MRSA positivity formed a clear immunosuppression gradient: 86.5% at CD4 <200 cells/µL, 73.3% at CD4 201–500 cells/µL, and 69.6% at CD4 >500 cells/µL.WHO staging showed a parallel trend: 72.6% positivity at stage I, rising to 88.2% at

stage II and 100% at stage III. Viral load category was not independently significant (p=0.661). All clinical and behavioural risk factors, including hospitalisation history, nose-picking, keeping of long nails, invasive device use, cotrimoxazole use, prior antibiotic use, skin infection at visit, and any current medication, were non-significant determinants of MRSA carriage status at the participant level (all p>0.10).

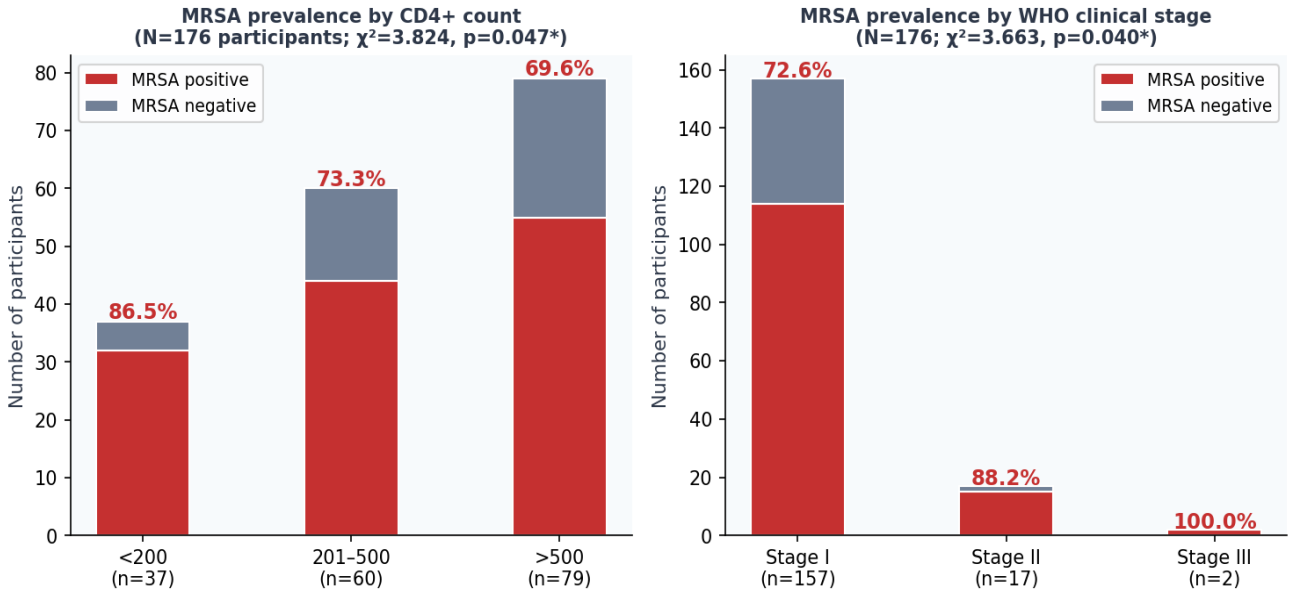

Figure 1: MRSA positivity by CD4+ count category (left; N=176, χ^2=3.824, p=0.047) and WHO clinical stage (right; χ^2=3.663, p=0.040*). An immunosuppression gradient is present for both metrics: positivity is highest at CD4 <200 cells/µL (86.5%) and at WHO stage III (100%).*

Table 1: Significant Determinants of MRSA Carriage Status at Participant Level (N=176)

Variable	MRSA positive n (%)	MRSA negative n (%)	χ^2	p-value
Age group (years)				
≤20	10 (55.6)	8 (44.4)	9.900	0.042*
21–30	12 (85.7)	2 (14.3)		
31–40	36 (80.0)	9 (20.0)		
41–50	38 (80.9)	9 (19.1)		

Variable	MRSA positive n (%)	MRSA negative n (%)	χ^2	p-value
51–60	29 (72.6)	11 (27.5)		
>60	6 (50.0)	6 (50.0)		
Occupation				
None/not applicable	7 (63.6)	4 (36.4)	7.173	0.047*
Unskilled	80 (69.6)	35 (30.4)		
Skilled	28 (90.3)	3 (9.7)		
Professional	16 (84.2)	3 (15.8)		
WHO clinical stage				
Stage I	114 (72.6)	43 (27.4)	3.663	0.040*
Stage II	15 (88.2)	2 (11.8)		
Stage III	2 (100.0)	0 (0.0)		
CD4+ count category				
<200 cells/µL	32 (86.5)	5 (13.5)	3.824	0.047*
201–500 cells/µL	44 (73.3)	16 (26.7)		
>500 cells/µL	55 (69.6)	24 (30.4)		

**p<0.05. Sex, marital status, education, residential area, and all behavioural/clinical risk factors were non-significant (all p>0.10). n=131 MRSA positive; n=45 MRSA negative.*

Multi-site colonisation pattern and determinants

Of 148 participant profiles, 61 (41.2%) were multi-site carriers (≥2 positive sites) and 87 (58.8%) were single-site carriers. Figure 2 presents the colonisation pattern and its relationship with hospitalisation history. In bivariate analysis, hospitalisation history was the only significant predictor of multi-site carriage (χ^2=10.741, p=0.030): participants hospitalised within the previous six months had markedly higher multi-site colonisation rates than those with no hospitalisation history. Age showed a borderline trend (median single-site 44 years vs multi-site 42 years; p=0.086). CD4 count was not associated with multi-site carriage (median 466 vs 424 cells/µL; p=0.794).

In multivariable logistic regression (N=148, AIC=203.3, McFadden R^2=0.096, Nagelkerke R^2=0.131), three variables were independently significant (Table 3, Figure 3): hospitalisation within the last 6 months (aOR=4.30, 95%CI 1.39–13.32, p=0.011), hospitalisation within the last 12 months (aOR=4.59, 95%CI 1.44–14.68, p=0.010), and younger age (aOR=0.97 per year, 95%CI 0.94–1.00, p=0.046). CD4 count, underlying disease, invasive device use, and residential area were all non-significant in the multivariable model.

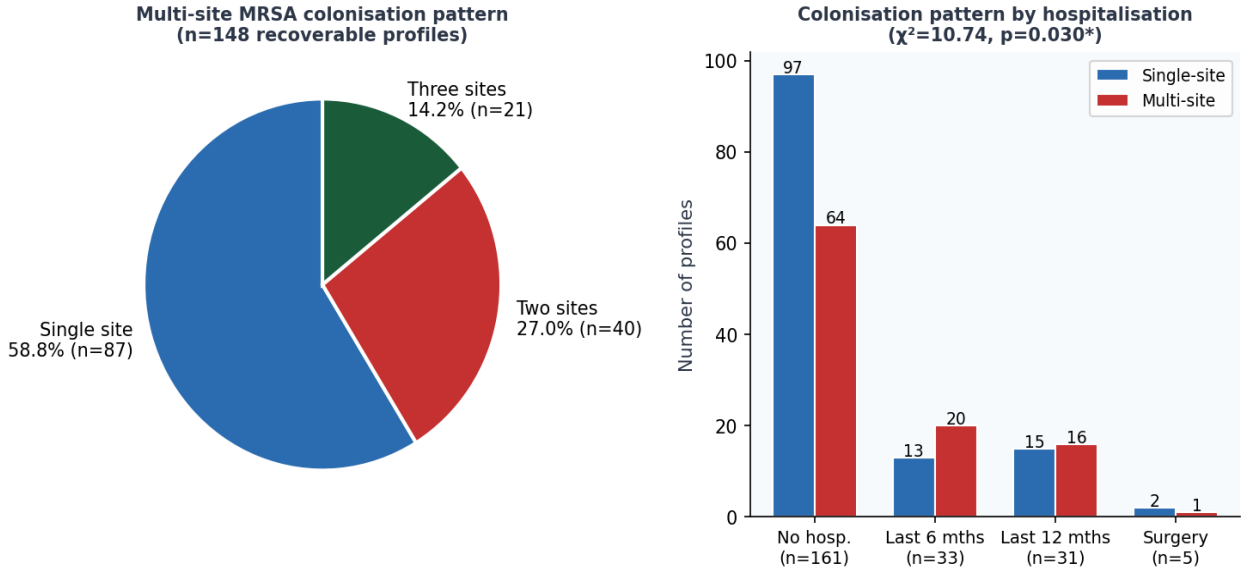

Figure 2: Multi-site MRSA colonisation. Left: colonisation pattern among 148 profiles — 41.2% multi-site, 58.8% single-site. Right: colonisation pattern by hospitalisation history; recently hospitalised participants showed substantially higher multi-site rates (χ^2=10.74, p=0.030*).

Table 2: Bivariate Analysis: Determinants of Multi-Site MRSA Colonisation (n=148 Profiles)

Variable	Single-site n (%)	Multi-site n (%)	Test statistic	p-value
Hospitalisation history	—	—	**χ^2=10.741**	**0.030***
Not hospitalised	97	64	—	—
Last 6 months	13	20	—	—
Last 12 months	15	16	—	—
Age (median, years)	44	42	U=2448	0.086 (trend)

Variable	Single-site n (%)	Multi-site n (%)	Test statistic	p-value
CD4+ count (median, cells/µL)	466	424	U=2229	0.794
Underlying disease — yes	33 (37.9%)	25 (41.0%)	χ^2=0.07	0.797
Invasive device use — yes	30 (34.5%)	31 (50.8%)	χ^2=1.15	0.283
Cotrimoxazole use — yes	73 (83.9%)	53 (86.9%)	χ^2=0.69	0.405
Sex: female	64 (73.6%)	46 (75.4%)	χ^2=0.06	0.814
**p<0.05. n=87 single-site; n=61 multi-site. All non-hospitalisation clinical variables non-significant.*				

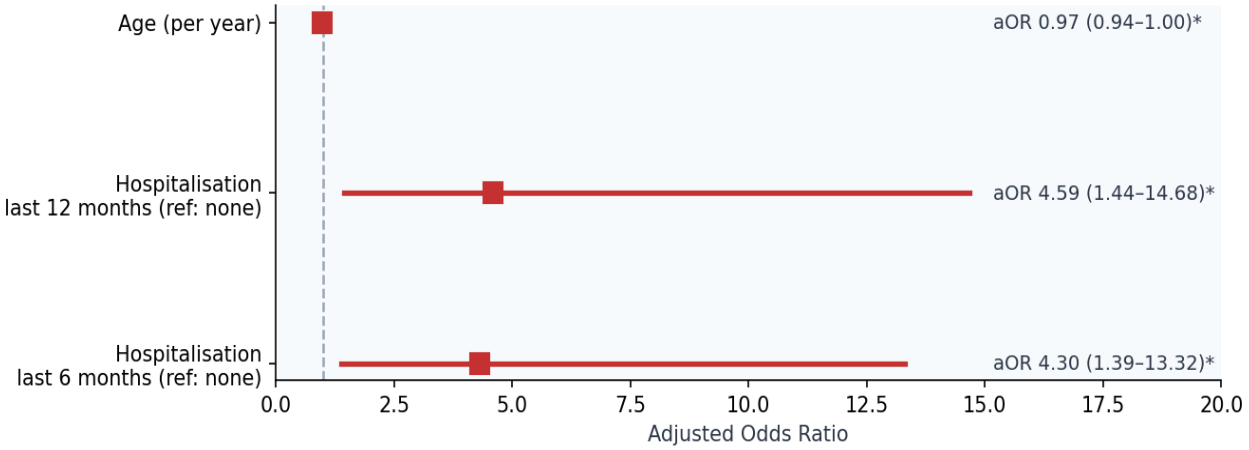

Figure 3: Forest plot: independent determinants of multi-site MRSA colonisation (N=148 profiles, McFadden R^2=0.096). Hospitalisation within 6 and 12 months and younger age were independently significant (* p<0.05).

Table 3: Logistic Regression: Determinants of Multi-Site MRSA Colonisation (N=148)

Variable	cOR (95%CI)	aOR (95%CI)	p (adjusted)	Sig.
N=148, AIC=203.3, McFadden R^2=0.096, Nagelkerke R^2=0.131. Outcome: multi-site (≥2 sites) vs single-site.				
Hospitalisation — last 6 months	4.50 (1.64–12.3)	**4.30 (1.39–13.32)**	**0.011***	✓
Hospitalisation — last 12 months	3.60 (1.42–9.10)	**4.59 (1.44–14.68)**	**0.010***	✓

Variable	cOR (95%CI)	aOR (95%CI)	p (adjusted)	Sig.
Age (per year increase)	0.98 (0.96–1.01)	**0.97 (0.944–0.999)**	**0.046***	✓
Living conditions: urban (ref: rural)	0.43 (0.16–1.20)	0.39 (0.12–1.28)	0.119	
Underlying disease	1.19 (0.60–2.37)	1.15 (0.50–2.66)	0.738	
Invasive device use	1.54 (0.74–3.21)	1.37 (0.57–3.28)	0.483	
CD4+ count (continuous)	Ref.	1.000 (0.999–1.001)	0.794	

**p<0.05. cOR=crude OR; aOR=adjusted OR. Enter method; all listed variables entered simultaneously.*

LUK-PVL gene prevalence and case profile

LUK-PVL was detected in 1 of 230 MRSA isolates (0.43%). Given that only a single positive case was identified, no formal statistical association analysis was performed; the isolate is described as a case profile. Table 4 presents the full molecular and clinical profile of the PVL-positive isolate. It was recovered from the nasal swab of a 37-year-old married male with secondary education, unskilled occupation, and suburban residence. The participant had a preserved CD4 count of 680 cells/µL, had been hospitalised within the preceding six months, had an active skin infection at clinic attendance, reported use of an invasive device, and carried an underlying comorbid disease. He was on cotrimoxazole prophylaxis and had never been observed picking his nose.

The isolate's molecular profile was mecA-positive, SCCmec type V-positive, and SCCmec type II-negative.

Table 4: Profile of the Single LUK-PVL-Positive MRSA Isolate

Characteristic	Value
Molecular profile	
mecA gene	Positive
SCCmec type II	Negative
SCCmec type V	Positive
LUK-PVL	**Positive (433 bp confirmed)**

Characteristic	Value
Swab site	Nasal cavity
Lineage inference	CA-MRSA (SCCmecV + PVL, without SCCmecII)
Patient demographics	
Sex / Age	Male, 37 years
Marital status / Education	Married / Secondary
Occupation / Residence	Unskilled / Suburban
Clinical and immunological profile	
CD4+ count	680 cells/µL (preserved immunity)
Hospitalisation	Within last 6 months
Underlying comorbid disease	Yes
Active skin infection at visit	Yes
Cotrimoxazole / Invasive device	Yes / Yes
Only PVL-positive isolate (0.43%, 1/230). No statistical PVL associations computed due to n=1. The SCCmecV+/LUK-PVL+/SCCmecII− profile is consistent with CA-MRSA; recent hospitalisation suggests healthcare acquisition of a community-origin strain.	

DISCUSSION

Methicillin-resistant *Staphyloccus aureus* colonisation in PLWH at ISTH is mostly governed by two distinct factors. Immune status, measured by CD4 count and WHO clinical stage, determines whether a participant carries MRSA at all. Hospitalisation history determines whether colonisation spreads to multiple body sites. These are not conflicting findings; they reflect different stages of the colonisation process, with immunosuppression facilitating initial acquisition and nosocomial exposure driving anatomical dissemination.

The CD4 gradient observed here, from 86.5% MRSA positivity at CD4 below 200 cells/µL to 69.6% above 500, is consistent with established immune pathophysiology. CD4 depletion reduces Th17-mediated IL-17 and IL-22 output at epithelial surfaces, impairing antimicrobial peptide production against S. aureus.[16,17] Crum-Cianflone et al.[5] found comparable inverse CD4-MRSA associations in US HIV outpatient settings, with low CD4 count independently predicting CA-MRSA infection. The WHO stage gradient, rising from 72.6% positivity at stage I to 100% at stage III, corroborates this and may serve as a practical MRSA risk surrogate in settings where CD4 testing is not routinely available. These findings argue for including CD4 thresholds, particularly below 200 cells/µL, in MRSA screening criteria at ART clinics.

The hospitalisation effect on multi-site colonisation (aOR 4.30 to 4.59) is unsurprising given the concentration of MRSA reservoirs in ward environments, on healthcare workers, and through invasive procedures that disrupt skin at multiple sites. The effect held after adjusting for invasive device use and underlying disease, indicating that hospitalisation carries independent risk beyond these proximate mechanisms. Huang et al.[18] showed that universal decolonisation substantially reduces MRSA infection rates in hospital settings; the magnitude of the hospitalisation aOR here supports applying similar protocols to PLWH following recent admissions. The association between younger age and multi-site carriage (aOR=0.97 per year) likely reflects higher healthcare utilisation, more acute clinical presentations, and less consistent ART adherence in younger patients.

The 0.43% LUK-PVL prevalence contrasts sharply with rates exceeding 80% in community MRSA from West African cities documented by Breurec et al.[19] This near-absence of PVL marks the dominant circulating MRSA as HA-MRSA in character, resistant but not hypervirulent. The clinical implication is practical: decolonisation and resistance-informed treatment should take priority over empirical coverage for necrotising PVL-mediated disease. The single PVL-positive isolate, in a recently hospitalised patient with an active skin infection and a CA-MRSA molecular profile, most plausibly reflects a one-off hospital acquisition of a community strain rather than sustained community CA-MRSA transmission within the ART clinic.

The non-significance of all behavioural variables, including nose-picking, long nails, invasive device use, cotrimoxazole, and prior antibiotic use, points to a risk profile shaped more by structural factors than individual behaviour. Immunosuppression and healthcare contact, both consequences of disease stage and health system access rather than personal choices, account for the meaningful associations. MRSA prevention efforts in this population are therefore better directed at CD4-stratified screening and post-discharge decolonisation than at behavioural modification campaigns.

Limitations include the cross-sectional design, which precludes causal inference, and the use of clinic records for CD4 and viral load rather than concurrent measurement, introducing potential temporal discordance. The n=148 multi-site cohort is smaller than the full N=176, limiting power for weaker associations. The single PVL-positive case cannot support inferential analysis, and the absence of whole-genome sequencing precluded definitive lineage assignment.

CONCLUSION

MRSA colonisation in PLWH at ISTH is shaped by immune status at the point of initial acquisition with CD4 <200 cells/µL associated with 86.5% positivity and WHO stage III with 100% and by healthcare exposure at the point of multi-site dissemination, with recent hospitalisation carrying fourfold increased odds of multi-site carriage regardless of immune status. LUK-PVL was present in only 1 of 230 isolates (0.43%), confirming predominantly HA-MRSA lineage circulation with low hypervirulence potential.

Together, these findings support CD4-stratified MRSA screening at Nigerian ART clinics, with particular attention to participants with CD4 below 200 cells/µL and those returning from recent hospital admissions, as the two highest-risk groups for MRSA carriage and multi-site burden, respectively.

AUTHORS' CONTRIBUTIONS

Ogbue Itohan Joan conceived the idea and conceptualized the study. She was actively involved in all aspects of the study including, literature search, development of research proposal, samples collection and analysis, data collection and analysis, manuscript drafting and publishing. Adewuyi GM and Samuel OS were supervising Consultants for the project and actively participated at all stages of the study. In addition, Adewuyi GM is the Head of the Department of HIV/AIDS, ISTH.

DECLARATIONS

Conflict of interest: None declared.

Funding: Research costs were self-funded by the investigators.

Ethics: Ethics and Research Committee, Irrua Specialist Teaching Hospital.

Data availability: Available from the corresponding author on reasonable request.

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