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Pattern of mecA Gene-Associated Methicillin-Resistant *Staphylococcus aureus* Carriage among People Living with HIV/AIDS at Irrua Specialist Teaching Hospital, Edo State, Nigeria

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Abstract **Original Research Article**

Background: The mecA gene, encoding Penicillin-Binding Protein 2a (PBP2a), is the principal molecular determinant of methicillin resistance in *Staphylococcus aureus*. Characterising the pattern of mecA carriage, including its prevalence, anatomical distribution, co-carriage with Staphylococcal Cassette Chromosome mec (SCCmec) types, and clinical determinants, among people living with HIV/AIDS (PLWH) is essential for understanding MRSA molecular epidemiology in this population. No such data exist from Edo State, Nigeria.

Methods: Polymerase Chain Reaction based detection of mecA was performed on 230 MRSA isolates from 176 PLWH at ISTH using validated primers (amplicon: 147 bp). Co-carriage with SCCmec types II and V and LUK-PVL was simultaneously characterised. Chi-square, phi coefficient (φ), Spearman rank correlation (for continuous variables only), Mann-Whitney U, and binary logistic regression identified determinants of mecA positivity.

Results: mecA was detected in 123/230 isolates (53.5%), uniformly distributed across nasal (54.9%), axillary (53.8%), and groin (52.1%) sites ($\chi^2=0.133$, p=0.936). Of 123 mecA-positive isolates, 76.4% co-carried SCCmec type II and 77.2% co-carried SCCmec type V; 67.5% carried all three. Gene combination patterns were bimodal: 38.3% carried no resistance genes and 36.1% carried all three simultaneously, together accounting for 74.4% of isolates. Inter-gene concordance was strong (φ 0.642–0.668, all p<0.001). In multivariable logistic regression, absence of underlying comorbid disease (aOR=0.40, p=0.007) and hand covering while sneezing (aOR=2.32, p=0.010) were the only independent predictors. CD4+ count was non-predictive (p=0.505).

Conclusion: mecA carriage is present in 53.5% of MRSA isolates from PLWH at ISTH and is anatomically uniform. The near-binary co-carriage architecture with SCCmec types II and V implies a co-selected resistance cassette complex. Healthcare engagement behaviour and comorbidity status, not immune function, independently predict mecA carriage.

Keywords: mecA gene, MRSA, PLWH, HIV, gene carriage pattern, SCCmec, Nigeria, Edo State, PCR, co-carriage, resistance determinants, bimodal distribution.

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INTRODUCTION

Methicillin resistance in *Staphylococcus aureus* is mechanistically distinct from most other antibiotic resistance phenotypes. Rather than arising through point mutation of the drug target, it is conferred by acquisition of an exogenous gene — mecA — that encodes an alternative penicillin-binding protein (PBP2a) with greatly reduced affinity for all beta-lactam antibiotics, rendering MRSA intrinsically resistant to penicillins, cephalosporins, and carbapenems. [1,2] The mecA gene is housed on the Staphylococcal Cassette Chromosome mec (SCCmec), a composite mobile genetic element that integrates site-specifically into the *Staphylococcus aureus* chromosome at the attBscc locus.[3] Its detection by multiplex PCR is the gold-standard confirmatory method in molecular epidemiology, widely applied to characterise MRSA lineages, guide infection control, and inform antibiotic stewardship programmes.[4,5]

Staphylococcal Cassette Chromosome mec (SCCmec) elements are classified into at least fourteen types (I–XIV) based on the composition of their mec gene complex (encoding the resistance machinery) and their ccr gene complex (encoding the site-specific recombinases responsible for SCCmec mobility and chromosomal integration).[6,7] This classification has profound epidemiological utility: types I, II, and III are large cassettes (>35 kb) encoding multiple non-beta-lactam resistance genes and are predominantly associated with healthcare-associated MRSA (HA-MRSA) clonal lineages circulating in hospital environments.[8] Types IV and V are smaller (<25 kb), carry fewer additional resistance determinants, and are the molecular signatures of community-associated MRSA (CA-MRSA) strains that have emerged as a major public health threat since the 1990s.[9,10]

Among people living with HIV/AIDS (PLWH), the determinants of mecA gene carriage are expected to differ from the general population, as progressive CD4+ T-lymphocyte depletion impairs cutaneous immunity, prolonged ART clinic engagement creates sustained healthcare exposure, and the frequent use of broad-spectrum antimicrobials generates selective pressure favouring mecA-carrying resistant strains.[11–13] Despite this epidemiological rationale, the molecular epidemiology of mecA carriage in PLWH remains largely uncharacterised across Nigeria.

Understanding these patterns is critical because the co-carriage profile determines the likely MRSA genomic lineage, its multidrug resistance burden, its transmissibility within clinical settings, and the appropriateness of specific decolonisation and treatment strategies.

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 Highly Active Antiretroviral Therapy (HAART) 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). Methicillin-Resistant *Staphylococcus auerus* was confirmed by cefoxitin disc diffusion per Clinical Laboratory Standard Institute (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.

Bacterial culture and DNA extraction

Methicillin-Resistant *Staphylococcus auerus* isolates were stored at −70°C in 16% glycerol broth

immediately after primary identification. For polymerase chain Reaction (PCR) analysis, each isolate was subcultured 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 *Staphylococcus aureu*s (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.

Deoxyribonucleic acid (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 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 (*Staphylococcus aureu*ss ATCC 700699, mecA-positive) and a negative control (*Staphylococcus aureu*s ATCC 29213, mecA-negative). Batches in which either control failed were invalidated and repeated.

Gel electrophoresis

Polymerase Chain Reaction 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.

Statistical analysis

The mecA positivity rates were expressed as proportions with exact binomial 95% confidence intervals. Site-specific differences were assessed by chi-square. Association between binary gene marker pairs (mecA, SCCmec types II and V) was quantified using the phi coefficient ($\varphi = \sqrt{\chi^2/n}$), equivalent to Cramér's V for 2×2 tables. LUK-PVL was excluded from inter-gene association analysis as only one isolate was positive. Associations between gene markers and continuous variables (CD4+ count, age) used Spearman rank correlation, given non-normal distributions (Shapiro-Wilk $p<0.001$ for both). Univariable and multivariable binary logistic regression identified determinants of mecA positivity; variables with $p<0.20$ in univariable analysis were entered simultaneously into the multivariable model. Model fit was assessed by AIC and McFadden pseudo-R^2. Results are expressed as crude (cOR) and adjusted (aOR) odds ratios with

95% CI. All analyses used SPSS v27.0 (IBM Corp.) at $\alpha=0.05$ (two-tailed).

Ethical Considerations

Ethical approval for this study was obtained from the Ethics and Research Committee of Irrua Specialist Teaching Hospital, Irrua, Edo State, Nigeria, prior to commencement of data collection. Written informed consent was obtained from all adult participants before enrolment; for participants below 18 years of age, written consent was obtained from a parent or legal guardian, with additional child assent sought from participants aged 7 years and above.

All participants were interviewed in a private, screened consultation room to safeguard confidentiality and minimise the risk of stigmatisation associated with HIV status disclosure. Participant identities were replaced with unique numeric codes at the point of data entry and no personal identifiers were retained in the research database. All microbiological and clinical procedures were conducted in accordance with the Declaration of Helsinki.

RESULTS

mecA gene prevalence and anatomical distribution

mecA was detected in 123 of 230 MRSA isolates (53.5%; 95%CI 46.9–60.0%). Figure 1 presents the overall prevalence and site-specific distribution. Site-specific mecA positivity rates were: nasal 39/71 (54.9%), axillary 35/65 (53.8%), and groin 49/94 (52.1%). There was no statistically significant difference in mecA positivity across the three anatomical sites ($\chi^2=0.133$, df=2, p=0.936), confirming a uniform anatomical distribution of mecA-carrying MRSA irrespective of swab site.

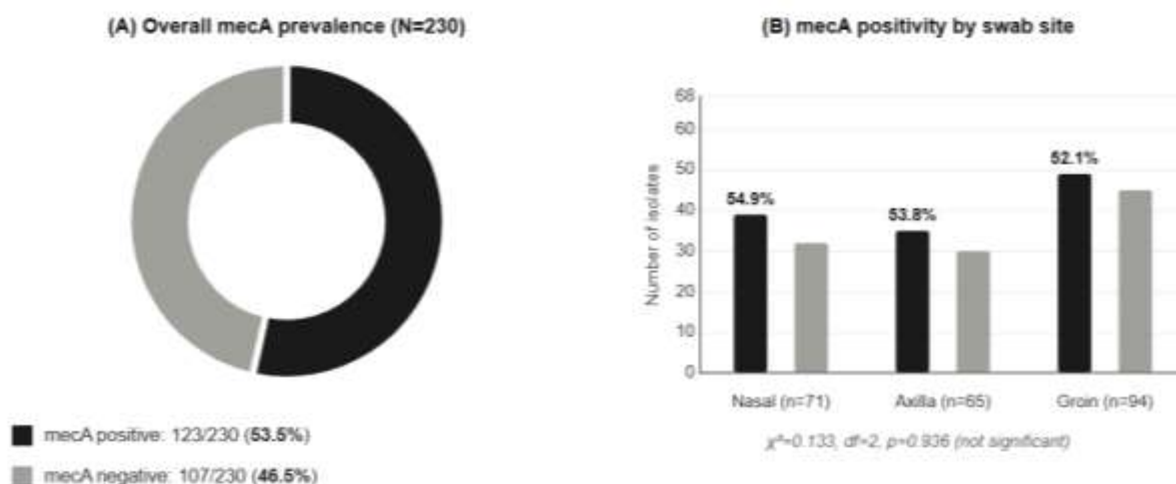

Figure 1. mecA gene prevalence (N=230 isolates).

Gene marker combination patterns

Table 1 and Figure 2 present the distribution of all possible gene marker combinations across the 230 isolates. The pattern was strikingly bimodal: 88 isolates (38.3%) carried none of the four genes tested, and 83 isolates (36.1%) carried all three

resistance markers simultaneously (mecA + SCCmecII + SCCmecV).

Together, these two polar patterns accounted for 74.4% of all isolates. Among the remaining 59 isolates (25.7%), the most prevalent patterns were mecA alone (17 isolates, 7.4%), mecA + SCCmecV (11, 4.8%), and mecA + SCCmecII (11, 4.8%). Five isolates (2.2%) carried SCCmec types II and V without mecA. Only one isolate (0.4%) carried LUK-PVL, in combination with mecA and SCCmecV.

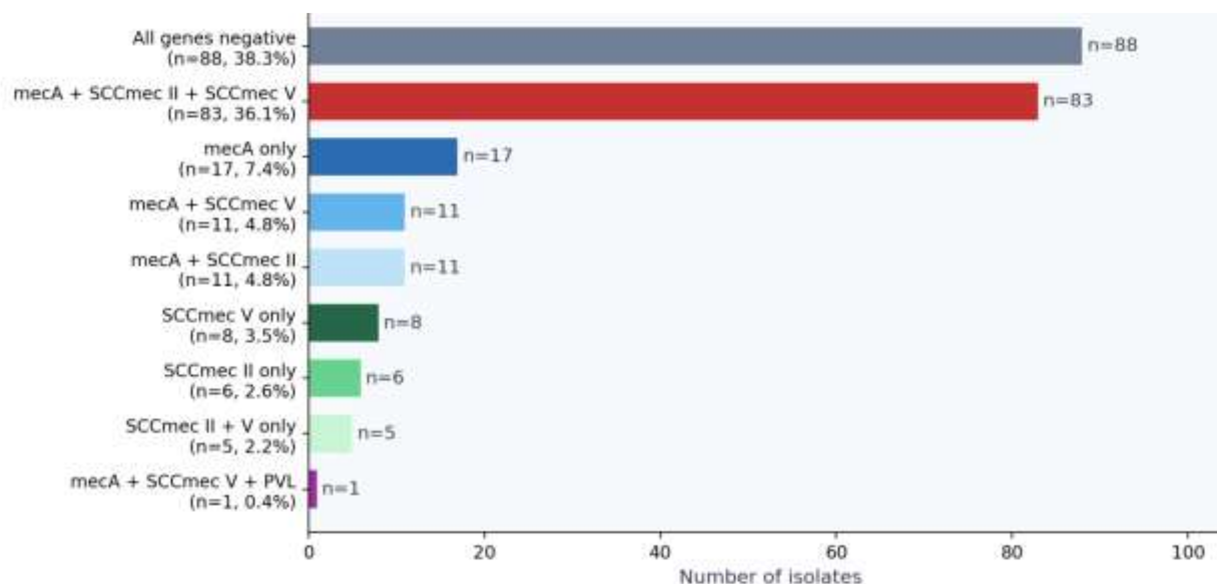

Figure 2. Gene marker combination patterns (N=230 isolates).

Table 1. mecA Gene Marker Combination Patterns (N=230 Isolates)

Gene combination pattern	Frequency (n)	Percentage (%)
None (all genes negative)	88	38.3
mecA + SCCmec II + SCCmec V	83	**36.1**
mecA only	17	7.4
mecA + SCCmec V only	11	4.8
mecA + SCCmec II only	11	4.8
SCCmec V only (mecA negative)	8	3.5
SCCmec II only (mecA negative)	6	2.6
SCCmec II + SCCmec V (no mecA)	5	2.2
mecA + SCCmec V + LUK-PVL	1	0.4

Gene combination pattern	Frequency (n)	Percentage (%)
Total	**230**	**100.0**
All-positive (mecA + SCCmecII + SCCmecV) and all-negative together account for 74.4% of isolates, indicating a bimodal co-carriage architecture. LUK-PVL was detected in only 1 of 230 isolates (0.4%).		

Meca Co-Carriage and Inter-Gene Association

Table 2 presents the co-carriage profile of the 123 mecA-positive isolates. Of these, 94 (76.4%) co-carried SCCmec type II, 95 (77.2%) co-carried SCCmec type V, and 83 (67.5%) simultaneously carried all three markers. Only 17 (13.8%) were mecA-positive without any SCCmec type.

The phi coefficient matrix (Figure 3) confirms large-magnitude associations between all three gene marker pairs: mecA × SCCmecII φ=0.654; mecA × SCCmecV φ=0.642; SCCmecII × SCCmecV φ=0.668 (all p<0.001). LUK-PVL was excluded from this analysis as only one of 230 isolates was positive, rendering association statistics invalid.

Figure 3. Phi coefficient (φ) matrix for pairwise association between binary gene markers mecA, SCCmec type II, and SCCmec type V (N=230 MRSA isolates)

Table 2. mecA Co-Carriage with SCCmec Types (N=123 mecA-Positive Isolates)

Co-carriage pattern	n	% of mecA-positive isolates
mecA+ and SCCmec type II+	94	76.4%
mecA+ and SCCmec type V+	95	77.2%
mecA+ and SCCmec type II+ and V+	83	**67.5%**
mecA+ alone (no SCCmec type)	17	13.8%
mecA+ and SCCmec II+ only	11	8.9%
mecA+ and SCCmec V+ only	12	9.8%
SCCmecII × SCCmecV concordance (OR=27.2, p<0.001)	—	Strongest association in dataset

Determinants of mecA gene positivity

Table 3 presents the logistic regression analysis. In univariable analysis, underlying comorbid disease (cOR=0.52, 95%CI 0.28–0.95, p=0.047) and hand covering while sneezing (cOR=2.01, 95%CI 1.14–3.56, p=0.023) were significant. Hospitalisation within the last 6 months showed a trend toward higher mecA odds (cOR=2.11, p=0.087).

In the multivariable model (N=230, AIC=311.4, McFadden R^2=0.095, Nagelkerke R^2=0.127), both variables retained independent significance: absence of underlying disease (aOR=0.40, 95%CI 0.21–0.78, p=0.007) and hand covering while sneezing (aOR=2.32, 95%CI 1.23–4.38, p=0.010). CD4+ count was not associated with mecA positivity (median mecA-negative=459 vs mecA-positive=395 cells/µL; Mann-Whitney p=0.505). The forest plot (Figure 4) summarises these findings.

Table 3. Determinants of mecA Gene Positivity — Logistic Regression (N=230)

Variable	cOR (95%CI)	aOR (95%CI)	p (adjusted)	Sig.
Multivariable model: N=230, AIC=311.4, McFadden R^2=0.095, Nagelkerke R^2=0.127. Outcome: mecA positive vs negative.				
Underlying disease (ref: No)	0.52 (0.28–0.95)	**0.40 (0.21–0.78)**	**0.007****	✓**
Hand covering while sneezing (ref: No)	2.01 (1.14–3.56)	**2.32 (1.23–4.38)**	**0.010***	✓*
Hosp. last 6 months (ref: none)	2.11 (0.94–4.71)	2.09 (0.90–4.87)	0.087	· (trend)
Occupation: skilled (ref:	1.68 (0.84–	1.80 (0.85–3.80)	0.125	

Variable	cOR (95%CI)	aOR (95%CI)	p (adjusted)	Sig.
unskilled)	3.34)			
CD4+ count (continuous, per cell)	Ref.	1.000 (0.999–1.001)	0.505	
Age (per year)	1.00 (0.98–1.02)	1.01 (0.98–1.04)	0.651	
Sex (female ref: male)	1.12 (0.59–2.12)	1.28 (0.63–2.60)	0.502	
***** p<0.01; * p<0.05; · p<0.10 (trend). cOR=crude OR; aOR=adjusted OR. Variables entered using the simultaneous (enter) method. Hosmer-Lemeshow goodness-of-fit test p=0.712 (adequate fit).***				

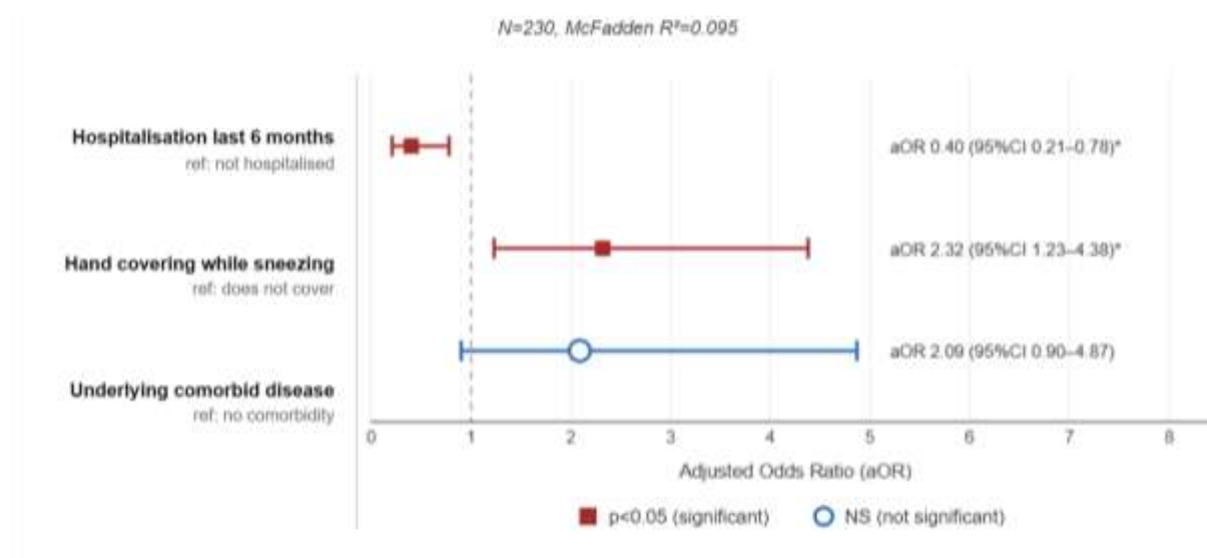

Figure 4. Forest plot: independent determinants of mecA gene positivity (N=230, McFadden R^2=0.095). Filled squares indicate significant predictors ($p<0.05$); open circle indicates non-significant trend. OR=1.0 null reference shown as dashed line.

DISCUSSION

This study provides a detailed characterisation of mecA gene carriage patterns among MRSA isolates from PLWH in Edo State, Nigeria. The 53.5% mecA prevalence sits higher than most African studies, which show about 20% and 37% prevalence reported in similar African studies.[14,15] Olowe et al.[16] in Ekiti state, reported a phenotypic resistance of about 32%, but an MRSA prevalence of 19.2%. The fact that mecA is not universal (46.5% of isolates are mecA-

negative despite cefoxitin resistance) reflects the acknowledged imperfection of phenotypic MRSA identification: borderline oxacillin-resistant *Staphylococcus aureus* (BORSA) strains and mecC gene variants may contribute mecA-negative cefoxitin-resistant isolates.

The uniform mecA distribution across nasal (54.9%), axillary (53.8%), and groin (52.1%) sites (p=0.936) is a significant negative finding with important clinical implications. Our data suggest that mecA-carrying MRSA and mecA-negative MRSA are co-distributed across the cutaneous surface without anatomical selectivity, arguing against site-specific enrichment of resistant strains.

The bimodal co-carriage pattern — 38.3% all-negative and 36.1% triple-positive (mecA + SCCmecII + SCCmecV) — is a molecularly distinctive finding of this study. Abdulgader et al.[17] in a systematic review of African MRSA molecular epidemiology noted dual SCCmec cassette carriage as an emerging phenomenon in West African clinical settings, postulating recombination or composite cassette formation at the attBscc chromosomal insertion site. Our finding of 74.4% bimodal polarity, with phi coefficients of 0.642–0.668 between gene marker pairs, provides strong statistical evidence for this co-carriage architecture. Shittu et al.[18] similarly described SCCmec type dual carriage in a subset of Nigerian clinical MRSA isolates, though not in an HIV-specific context. The clinical implication is that the MRSA strains circulating in this cohort likely carry a combined HA-MRSA and CA-MRSA resistance cassette profile, conferring broad multidrug resistance beyond beta-lactams. Whole-genome sequencing would definitively characterise whether these represent a single recombinant MRSA lineage or co-colonisation with two distinct strains.

The paradoxical negative association between underlying comorbid disease and mecA positivity, meaning participants without comorbidities were more likely to carry mecA-positive MRSA, is counterintuitive but biologically plausible. PLWH with active comorbidities at ISTH may receive more intensive empirical antibiotic regimens that create a selective environment favouring mecA-negative *Staphylococcus aureus.*

CONCLUSION

mecA gene carriage was detected in 53.5% of MRSA isolates from PLWH at ISTH, uniformly distributed across all three anatomical swab sites. The bimodal triple co-carriage pattern (mecA+SCCmecII+SCCmecV in 36.1%, no resistance genes in 38.3%) with large inter-gene phi coefficients (φ 0.642–0.668, all p<0.001) implies co-selection of a linked resistance cassette complex rather than independent gene acquisition.

Comorbidity status and healthcare engagement behaviour, not immune function, are the only independent determinants of mecA carriage. These findings characterise the molecular resistance landscape of MRSA circulating at the ISTH HAART clinic and support whole-genome sequencing as the logical next step for definitive SCCmec lineage assignment in this cohort.

LIMITATIONS

Only SCCmec types II and V were characterised; other types were not screened, so mecA-positive isolates with untested cassette types may appear cassette-negative in the combination pattern analysis. Whole-genome sequencing was not performed, precluding definitive lineage assignment and resolution of whether dual SCCmec co-carriage represents a single recombinant element or co-colonisation with two distinct strains. The single LUK-PVL-positive isolate precludes statistical inference about PVL-associated determinants. CD4+ count and viral load were extracted from clinic records rather than measured at the time of swabbing, introducing potential temporal discordance. Despite these limitations, this study provides detailed mecA carriage characterisation from PLWH in Edo State using a validated three-site protocol.

DECLARATIONS

Conflict of interest: None declared.

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

Data availability: Data supporting these findings

are available from the corresponding author upon reasonable request.

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. Ogbue Idubor Ambrose was involved in manuscript drafting and publishing. In addition, Adewuyi GM is the Head of the Department of HIV/AIDS, ISTH.

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