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Drug Resistance Genes and Source of Methicillin Resistant *Staphylococcus aureus* Strains amongst HIV Patients in Fako Division of Cameroon: A Case-Control Study

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Abstract

Background: Methicillin Resistant *Staphylococcus aureus* and severe antimicrobial resistance is worsened in HIV positive patients, driven by the presence of drug resistance traits. This study aimed to determine the prevalence of MRSA, antibiotic susceptibility, source of infection and the distribution of drug resistance genes amongst HIV positive and HIV negative participants in Fako-Cameroon. **Methods**: Urine and nasal samples were cultured on CLED and MSA agar respectively following standard procedures. The clinical isolates were characterized biochemically and comfirmed by *nuc* gene detection of *S. aureus*. Antimicrobial susceptibility with selected locally prescribed antibiotics was done using the Kirby Bauer disc diffusion technique. Drug resistance genes (*vanA*, *mecA*, *ermA*, *sul3* and *ermC*) were determined using PCR amplification. Data was analyzed using Microsoft Excel 2016 and SPSS version 25; Chi square was set at P ≤ 0.05. **Results**: The overall prevalence of MRSA was 28.8%. MRSA was higher (P = 0.0001) in HIV positive patients (19.4%) than in HIV negative individuals (9.4%). Amongst HIV positive and HIV negative participants, high sensitivity of urogenital *S. aureus* was observed amongst Aminoglycosides [Amikacin (26.3% vs 52.9%)] and Nitrofurans [Nitrofurantoin (25.2% vs 53.1%)]. Resistance was seen against Ciprofloxacin [39.5% vs 41.2%] in HIV positive and HIV negative participants respectively. MAR index of urogenital *S. aureus* was higher amongst HIV positive patients (0.9) compared to HIV negative individuals (0.7). Majority (38.0%) of *S. aureus* clinical isolates cultured from the urine of HIV positive patients were not identical to isolates from the nostrils of the same patients. The overall detection

rate of drug resistance genes were distributed as follows; *vanA* (30.8%), *ermA* (29.6%), *mecA* (28.6%), *sul3* (21.6%) and *ermC* (18.0%). **Conclusion**: Methicillin resistant *Staphylococcus aureus* is more common amongst HIV positive patients, with the clinical isolates being, more resistant to commonly prescribed antimicrobial agents. The clinical isolates causing urinary tract infections in HIV positive patients are usually not from the normal flora (the nostrils).

Keywords

MRSA, Drug Resistance Genes, Cameroon

1. Introduction

Microorganisms such as *Staphylococcus aureus* are disease causative agents, with the ability to cause infections in different human body sites; remarkably the anterior nares, skin, armpit, vagina, upper respiratory tracts, sometimes serving as a reservoir for most opportunistic infections [1]. *S. aureus* have been reported as a urogenital pathogen; which presents doubt as to whether the bacteria that infects the urinary tract actually originate from the nasal tract as a normal flora. Increase in MRSA have prevailed globally due to frequent exposure of *S. aureus* bacteria isolates to commonly used antibiotics. MRSA is one of the abundant pathogen of human existence that colonizes patients both within the hospitals and around the environments [2]. However, *S. aureus* isolated from different body parts often possess different antimicrobial resistance profiles [3]. This is worsened in HIV positive patients with a compromised immune system, enhancing bacterial susceptibility and heightened antimicrobial resistance; which may further complicate treatment. There is a growing concern of *S. aureus* antimicrobial resistance, especially with the activities of Methicillin resistant *Staphylococcus aureus* (MRSA) [4]. *S. aureus* antimicrobial susceptibility has been extensively studied globally; a study reported in Iran observed varying resistance to different antibiotics; Erythromycin (70%), Clindamycin (45%) and 100% sensitivity to Vancomycin, with *ermA* gene (5%) and *ermC* gene (10%) being detected [5]. Other studies have accessed *S. aureus* genotypic variants in Africa. A study conducted in Ethiopia reported that *mecA* gene was detected in 27.5% of *S. aureus* bacteria isolates [6]. In another study carried out in Southwestern Nigeria, *S. aureus* resistance was observed respectively between HIV positive and HIV negative participants as follows; Ampicillin (93.3% vs 84.0%), Ceftriaxone (73.3% vs 32.0%), Augmentin (60.0% vs 16.0%) and Bactrim (66.7% vs 80.0%) [7]. In a study reported by Morgan *et al.*, in 2023, nasal carriage *S. aureus* isolates were resistant to Ampicillin (100%), Cefixime (81.82%), while urogenital *S. aureus* was resistant to Ampicillin (100%) and Cefixime (95%) [3], in Buea-Cameroon. Most MRSA bacteria isolates have been reported to be resistant to Penicillins and Cephalosporins [8]. However, Vancomycin stands as one of the most sensitive antibiotics for MRSA treatment

amidst new antibiotics [9]. Though Vancomycin resistance has been reported globally; Africa (16%), Asia (5%) and South America (4%), MRSA should be considered a top priority pathogen because of its public health importance. Resistance to methicillin is due to beta lactamase production by *S. aureus* isolates, which help the organism to modify penicillin-binding proteins (PBPs), reducing the antibiotic's ability to bind and kill the bacterium; enhancing resistance to Penicillins, Beta lactams and Cephalosporins. *S. aureus* isolates that are resistant to Macrolides (Erythromycin, Clarithromycin) and Lincosamides (Clindamycin) actually make use of the Methylase (*erm*) gene which help to methylate 50S ribosomal binding sites, leading to antimicrobial resistance [10].

However, *S. aureus* isolates from the urinary tract may share resistance traits with isolates from the nostrils, although that relationship remains unclear. This stands a great knowledge gap with direct implications on treatment outcomes and preventive measures from *S. aureus* infected patients. Few studies in HIV endemic areas including Fako-Cameroon have reported the antimicrobial resistance profiles and drug resistance gene profiles in *S. aureus* isolates; isolated from the urinary tract and nasal tracts of HIV positive and HIV negative individuals. Providing this data will therefore strengthen clinical decision (empirical therapeutic guidance), Antimicrobial surveillance, and enhance decolonization strategies in HIV patients. We hypothesize that urogenital *S. aureus* isolates will exhibit higher resistance to antimicrobials due to selective pressure in HIV positive patients. This study aimed to determine the prevalence of MRSA, antibiotic susceptibility, source of infection and the distribution of drug resistance genes amongst HIV positive and HIV negative participants in Fako-Cameroon

2. Materials and Methods

2.1. Study Area and Study Population

Five subdivisions (Buea, Tiko, Limbe, Muyuka and Idenau) make up the Fako division, where the study was conducted. Different levels of health facilities are found in Fako division; which are both private owned and public owned hospitals. Sub-divisional health centres provide health services in rural areas, standing as the smallest unit of health facilities in Cameroon. This is followed by the district hospitals, which are located in the urban areas; towns and provide health care to the population. Referral hospitals named Regional hospitals are located in different regions and receive referral cases from the district hospitals and health centres. Fako division is cosmopolitan, made up of civil servants, traders, farmers and students; comprised of the dry season (from November to February) and the rainy season (from March to October).

2.2. Study Design and Sampling of Study Participants

This study was a hospital—based case-control study and with non-matched variables. It was conducted from March 2024 to April 2025. The cases group comprised of people aged 18 years and above, living with HIV and who were already

on treatment for at least three months. For the control group, they were individuals who were not living with HIV and were 18 years and above. All persons who had not taken antibiotics for more than three weeks who willingly gave their consent were included in the study. Participants who were bed-ridden, and or pregnant women were excluded from the study. Study participants were recruited from health facilities in Fako division by convenience and purposeful sampling methods. HIV positive patients were purposively selected based on predefined inclusion criteria such as confirmed HIV status. This method ensured that only individuals with the characteristics relevant to the study's objective were included. All the HIV positive patients were recruited following the same inclusion criteria. Consecutively, HIV negative controls who met with the inclusion criteria and presented at the same health care facilities during the study period were recruited. This method ensured that the sample represented the typical HIV negative population accessing care during the study period.

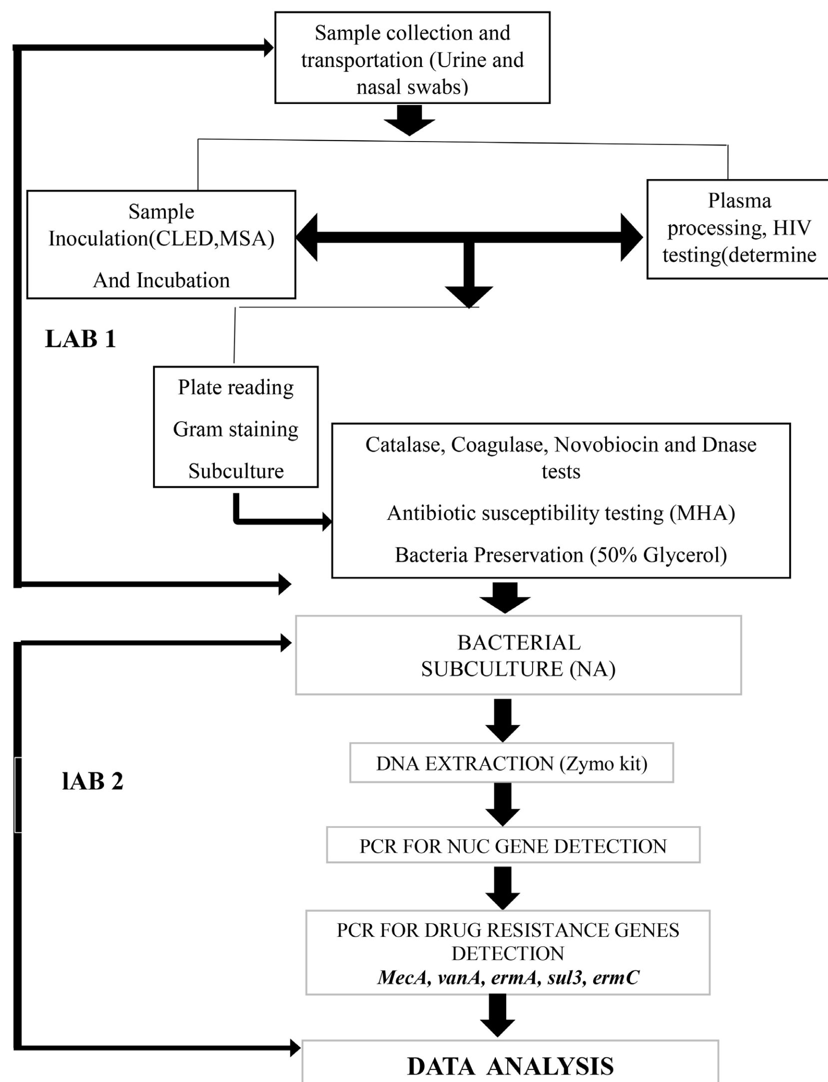

Figure 1. Summary of the research protocol.

The Buea regional hospital is the only referral health facility with an accredited laboratory in the region. Other facilities were selected because they have been empowered to provide health services to HIV patients in the sud-divisions of Fako. The study participants were included in the study by consecutive sampling, where urine and nasal samples were collected. Both the symptomatic and asymptomatic population gave their urine and nasal samples; following the inclusion and exclusion criteria of the study. Demographic data was obtained using well-structured questionnaires; urine, nasal and blood samples were collected from 250 HIV positive and 250 HIV negative individuals. Urine and nasal swabs were used for culture and isolation of *S. aureus* and determination drug resistance genes while blood samples were used to determine the HIV status of study participants (**Figure 1**).

2.3. Bias

In order to avoid measurement bias, laboratory protocols, questionnaires and all procedures were carried out on both HIV positive and HIV negative participants. As for selection bias, we used the same inclusion exclusion criteria for both HIV positive and HIV negative individuals. Participants were recruited from the same health institutions and on the same day.

2.4. Sample Size Calculation

Sample size was calculated using the formula proposed by Kasiulevicius [11];

$$(N) = r + 1(P^*)\ (1 - P^*)(Z\beta + Z\alpha/2)2/1\ (D)2.$$

Where, r = Ration of controls to cases to be recruited in the study, $Z\beta$ = Standard normal variate for power 80%; 0.84, $Z\alpha/2$ = Standard normal variate for a level of significance at $P < 0.05$, $P^* = D = (p1 - p2)$ = Difference in proportions expected based on previous studies, P1 = Pre-study estimate of the proportion of bacteria in HIV patients = 48% [7]. P2 = Pre-study estimate of the proportion of bacteria in Non-HIV patients = 52% [7],

$$D = \text{minimum expected difference} = |48\% - 52\%| = -4\%$$

$$P^* = (0.70 + 0.51)/2 = 0.6$$

Numerical computations

$$N = 1 + 1(0.6)(1 - 0.6)(0.8 + 0.05)2/1\ (-0.04)2,\ N = 237$$

A minimum of 237 participants were to be recruited for each group but we recruited 250.

2.5. Ethical Consideration

Study participants willingly gave their informed consent to participate in the study; they were properly educated using the best languages well understood by the participants; on the benefits of the research as participants. Ethical clearance was obtained from the institutional review board of the faculty of Health Sciences, University of Buea (2024/2057-03/UB/SG/IRB/FHS) and administrative authorization was obtained from the Regional delegation of public health for the South

West region of Cameroon (RII/MINSANTE/SWR/RDPH/PS/129/161).

2.6. Data and Sample Collection

Socio-demographic data; gender, age, occupation, Educational status and marital status) were collected with the help of well-structured questionnaires. Mid-stream urine samples were collected in sterile wide-mouth leaked proof cups. Nasal samples were collected using sterile cotton tipped swabs dipped in sterile physiological saline. Two milliliters of whole blood were collected into EDTA tubes; for plasma, extraction and confirmation of HIV status of participants using determine (Abbott) rapid test strip. Samples were collected in different days and transported to the Buea regional hospital laboratory where urine and nasal samples were inoculated and incubated within two hours of sample collection.

3. Laboratory Analysis

3.1. Bacteriological Culture of Samples and Antibiotic Susceptibility Testing of Clinical Isolates

Two culture media were used for the primary isolation of bacteria isolates. Urine samples were seeded using a sterile calibrated wire loop (0.01 ml) on Cysteine lysine electrolyte deficiency agar (Titan Biotech lmt India). Nasal samples were inoculated on Manitol salt agar (Liofilchem Srl Italy) and the plates were incubated at 37˚C for 24 hours aerobically. Bacteria growth on CLED agar with golden yellow, circular shaped and 1 – 3 mm in diameter were *S. aureus* presumptive isolates, which where subcultured on MSA agar for 24 hours. All bacteria isolates on MSA agar with raised colonies and yellow coloration of the MSA media were further incubated anaerobically for 24 hours at 37˚C. Colonies, which were large, round, smooth and golden-yellow, were gram stained and observed microscopically for purple-clustered cocci organisms. *S. aureus* presumptive isolates were biochemically characterized using the catalase, coagulase, Novobiocin and DNASE tests; as reported in other studies [12]. Bacteria isolates were considered *S. aureus* presumptive isolates if the catalase test, coagulate test, Novobiocin and or DNase tests were positive. Antibiotic susceptibility testing of *S. aureus* clinical isolates was done using the disk-diffusion technique on Mueller Hinton agar plates; following standards operating procedures as recommended by the Clinical Laboratory Standard Institute [13]. The antibiotics used in this study included Amoxicillin (10 µg), Augmentin (30 µg), Ceftriaxone (30 µg), Amikacin (30 µg), Ciprofloxacine (5 µg), Erythromycin (15 µg), Clindamycin (10 µg), Chloramphenicol (30 µg), Bactrim (30 µg), Vancomycin (10 µg) and Nitrofurantoin (30 µg). Although Nitrofurantoin is not a first-line treatment for systemic *S. aureus* infections due to poor tissue penetration and limited efficacy, it is occasionally used for uncomplicated urinary tract infections (UTIs) caused by *S. aureus* in our clinical settings. We included it to assess whether local resistance patterns (particularly in urinary isolates) might justify its restricted use, reinforcing current guidelines. Its inclusion also serves as a negative control to highlight antibiotics that should not be

empirically prescribed for *S. aureus* infections. Amikacin, an aminoglycoside, was selected because it retains activity against some methicillin-resistant *S. aureus* (MRSA) strains and is used in combination therapies for severe infections. In our region, aminoglycoside resistance patterns are underreported, and testing Amikacin helps identify potential salvage therapy options for multidrug-resistant (MDR) isolates. Both antibiotics had breakpoints of ≥17 mm considered susceptible, 15 - 16 mm Intermediate and ≤14 mm considered resistant. All presumptive isolates were subsequently inoculated in 1 ml of nutrient broth in a sterile Eppendorf tubes, vortexed and incubated overnight at 37˚C. the Eppendorf tubes were centrifuged at 8000 g for few minutes and 0.7 ml of the supernatant was discarded and 0.7 ml of 50% glycerol was added to the sediment, vortexed for 10 seconds and stored at −20˚C; awaiting molecular analysis. Sterility Testing: A plate from each media batch was incubated without inoculation to confirm absence of contamination. Growth Consistency: Each sample was inoculated onto two plates of the same media to verify reproducibility of microbial growth.

3.2. Molecular Screening of Presumptive Clinical *S. aureus* Isolates and Determination of Drug Resistant Traits

Presumptive clinical isolates of *S. aureus* were thawed and sub-cultured in nutrient agar at 37˚C for 18 - 24 hours. The extraction of bacteria DNA was done with the help of a compliance bacteria based and compatible DNA extraction kit, according to the manufacturer's instructions and stored at −20˚C, for PCR analysis [14]. The entire DNA extracted was confirmed using 1.5% agarose gel electrophoresis as previously described [15]. The DNA extracted from clinical isolates was used to determine the presence of *S. aureus nuc* gene (F: GCGATTGATGGTGATACGGTT R: AGCCAAGCCTTGACGAACTAA AGC) obtained from Inquaba Biotech [16]. The presence of *nuc* genes indicated that the presumptive bacteria isolates were actually *S. aureus* isolates. The *nuc* gene cycling conditions were as follows; 35 cycles of denaturation at 94˚C for 2 minutes, annealing step at 55˚C for 30 seconds, extension at 72˚C for 30 seconds and final extension at 72˚C for 10 minutes [16]. *S. aureus* DNA confirmed having *nuc* gene, were further investigated for the detection of *S. aureus* drug resistance variants (*ermA*, *vanA*, *mecA*, *sul3*, and *ermC* gene). The *vanA* gene (F: ATG AAT AGA ATA AAA GTT GC and R: TCA CCC CTT TAA CGC TAA TA) with 45 PCR cycles was summarized as follows; Initial denaturation at 98˚C for 2 minute, denaturation at 98˚C for 10 minutes, annealing at 50˚C for 60 seconds, extension at 72˚C for 1.5 minute and final extension at 72˚C for 10 minutes [17]. The *mecA* gene (F: TGGCTATCGTGTCACAATCG and R: CTGGAACTTGTTGAGCAGAG) with 30 PCR cycles as was summarized as follows; Initial denaturation at 94˚C for 5 minutes, denaturation at 94˚C for 30 seconds, annealing at 55˚C for 30 seconds, extension at 72˚C for 5 minute and final extension at 72˚C for 10 minutes [18]. The *sul3* gene (F: TCAAAGCAAAATGATATGAGC and R: TTTCAAGGCATCTGATAAAGA) with 30 PCR cycles was summarized as follows; Initial denaturation at 94˚C for 4 minute, denaturation at 94˚C for 30 seconds, annealing at 55˚C for 30 seconds, extension at

72˚C for 1 minute and final extension at 72˚C for 7 minutes [19]. The *ermA* gene with primer sequences (F: AAGCGGTAAACCCCTCTGA and R: TTCGCAAATCCCTTCTCAAC) and *ermC* gene (F: AATCGTCAATTCCTGCATGT and R: TAATCGTGGAATACGGGTTTG); both had 30 PCR cycles summarized as follows; Initial denaturation at 94˚C for 3 minutes, denaturation 94˚C for 30 seconds, annealing at 55˚C for 30 seconds, extension at 72˚C for 30 seconds and final extension at 72˚C for 4 minutes [20]. All DNA amplicons were separated on 1.5% agarose gel electrophoresis in 1x Tris borate acetate EDTA (TAE) buffer (BioConcept Ltd., Basel, Switzerland) along with a 1 kb DNA ladder (Solis Biodyne) and visualized by ethidium bromide staining under UV transilluminator imaging system (BIO-RAD, USA). Band sizes were as follows; *vanA* gene (1032 bp), *ermA* (190 bp), *mecA* (309 bp), *sul3* (787 bp) and *ermC* (299 bp). Sterile nuclease-free water was included as a no-template control (NTC) in every run to rule out contamination. PCR results were considered valid only if the NTC showed no amplification.

3.3. Data Analysis

Data that was collected using well-structured questionnaires was cleaned and analyzed using Microsoft excel 2016 and the statistical software SPSS version 25. Demographic features of HIV positive and HIV negative participants was analyzed using descriptive statistics. The chi square statistical test was used to compare antimicrobial susceptibility profiles between HIV positive patients and HIV negative individuals. Binary logistic regression was used to determine the association of drug resistance variants and antibiotic susceptibility. The *S. aureus* bacteria isolates from the urine and nostrils of the same participants were compared using their antibiotic susceptibility profiles and the presence of identical drug resistance genes. Clinical isolates from the nostrils were considered putatively identical to those from urine; if they both had the same antimicrobial susceptibility profiles and identical drug resistance genes profiles. The chi square test was used to determine the whether the putatively identical *S. aureus* isolates were significantly associated with HIV positive patients and HIV negative participants. These comparisons may suggest relatedness but require additional genomic or high-resolution typing methods (whole-genome sequeng) to confirm clonality.

4. Results

4.1. Study Participants

At the beginning of the research, we met with 980 individuals, whereby 415 were HIV positive patients and 370 were HIV negative individuals (**Figure 2**). Of these, 370 HIV positive patients and 455 HIV negative individuals were examined potential participants; 155 potential eligible participants refused to be examined for eligibility. Amongst HIV positive and HIV negative participants examined, 257 and 282 people respectively were eligible for the study and 286 participants were excluded based on our inclusion and exclusion criteria. For the eligible participants,

252 HIV positive patients and 259 HIV negative individuals gave their informed consent to participate in the study, while 28 eligible participants refused to give their informed consent. The study included 250 HIV positive patients and 250 HIV negative individuals at the end because 09 participants were unable to give the required samples, they were excluded.

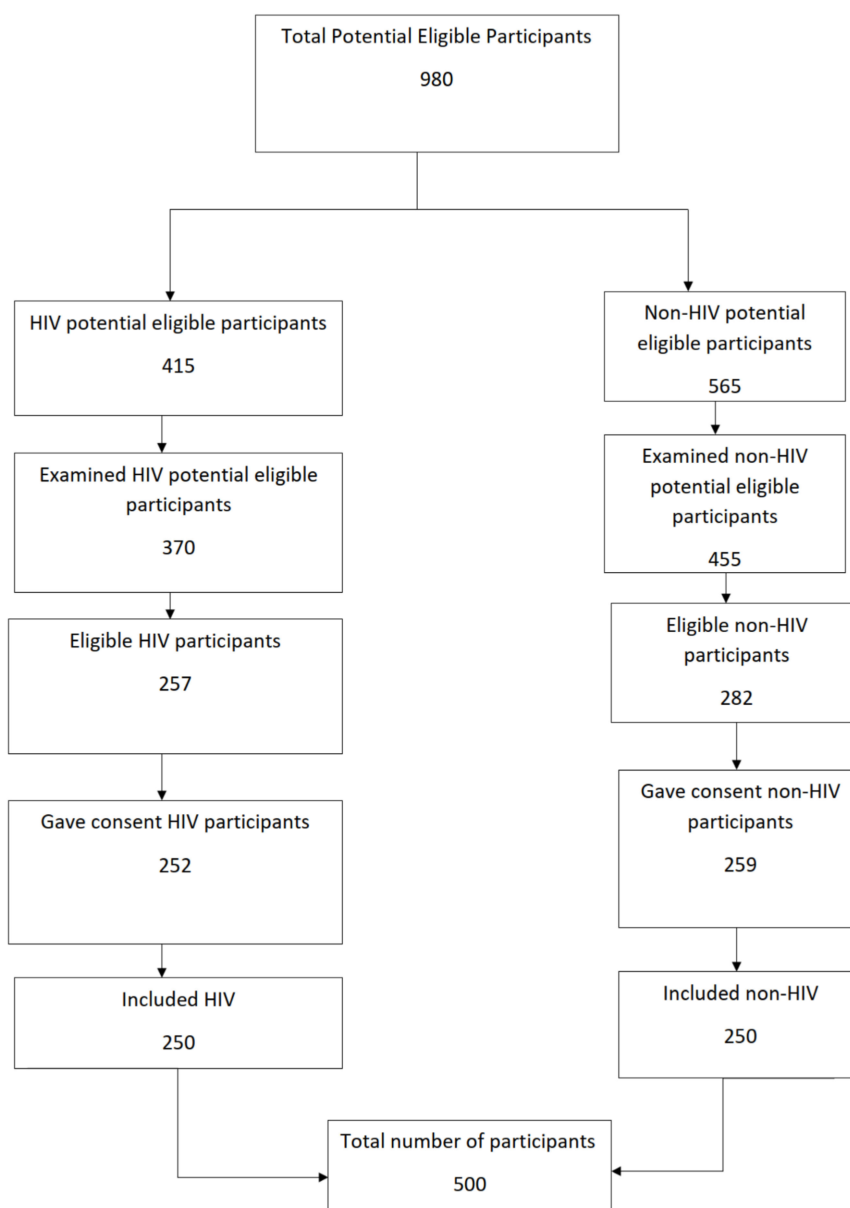

Figure 2. Flow chart showing the summary of study participants.

4.2. Sociodemographic Characteristics of the Study Participants

A total of 500 participants were recruited in this study, 250 were HIV positive patients and 250 HIV negative individuals as shown in Table 1. Majority 165/250 (66.0%) of HIV positive patients were females, as opposed to 158/250 (63.2%) females observed amongst HIV negative persons. Majority of the study participants

were HIV patients within the age range of 41 - 50 years 110/250 (44.0%), who attained primary level of education 141/250 (56.4%) and were married 102/250 (40.8%). For the control group, most of participants were in the age range ≥ 51 years [78/250 (31.2%)], who attained secondary level of education [90/250 (36.0%)], were married persons [122/250 (48.8%)] and involved in business [124/250 (49.6%)].

Table 1. Socio-demographic properties of the study participants.

Variables	HIV Positive		HIV Negative	
	Frequency	Percentage (%)	Frequency	Percentage (%)
Gender				
Female	165	66.0	158	63.2
Male	85	34.0	92	36.8
Total	250	100.0	250	100.0
Age Range				
≤30	7	2.8	57	22.8
31 - 40	44	17.6	53	21.2
41 - 50	110	44.0	62	24.8
≥51	89	35.6	78	31.2
Total	250	100.0	250	100.0
Educational status				
Informal	10	4.0	6	2.4
Primary	141	56.4	88	35.2
Secondary	89	35.6	90	36.0
Tertiary	10	4.0	66	26.4
Total	250	100.0	250	100.0
Occupation				
Employed	101	40.4	85	34.0
Unemployed	8	3.2	11	4.4
Business	134	53.6	124	49.6
Student	2	0.8	25	10.0
Retired	5	2.0	5	2.0
Total	250	100.0	250	100.0
Marital status				
Single	65	26.0	84	33.6
Married	102	40.8	122	48.8
Widow/Widower	57	22.8	32	12.8
Divorced	26	10.4	12	4.8
Total	250	100.0	250	100.0

4.3. General Prevalence of MRSA and MSSA by *mecA* Gene Detection in the Study Population

As indicated in Figure 3, majority of *S. aureus* bacteria isolates detected in the study population where Methicillin susceptible [356/500 (71.2%)] and fewer bacteria detected were Methicillin Resistant *Staphylococcus aureus* [144/500 (28.8%)] isolates.

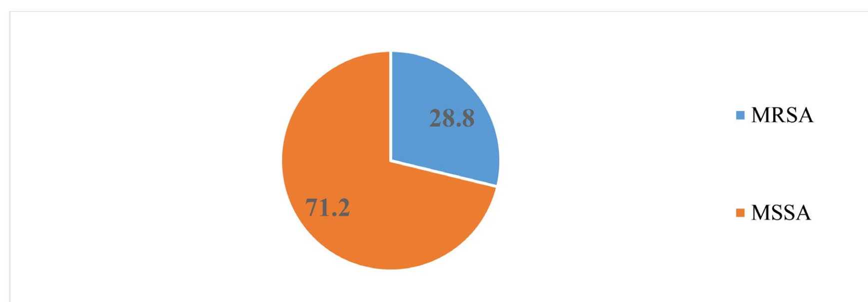

Figure 3. Prevalence of MRSA *and* MSSA isolates in HIV and Non-HIV participants.

4.4. Prevalence of MRSA and MSSA by *mecA* Gene Detection in HIV Positive and HIV Negative Individuals

Methicillin resistant *Staphylococcus aureus* was twice higher 97/500 (19.4%) in HIV positive patients as compared to the 47/500 (9.4%) detected in HIV negative participants. More [203/500 (40.6%)] HIV negative persons had Methicillin susceptible *Staphylococcus aureus* as opposed to 153/500 (30.6%) observed in HIV positive patients as indicated in Table 2.

Table 2. Distribution of MRSA and MSSA isolates in the study population.

Variables	HIV	Non-HIV	Total	P. Value (P ≤ 0.05)
	Frequency (%)	Frequency (%)		
MRSA	97 (19.4)	47 (9.4)	144 (28.8)	
MSSA	153 (30.6)	203 (40.6)	356 (71.2)	0.0001
Total	250 (50.0)	250 (50.0)	500 (100.0)	

4.5. Antimicrobial Susceptibility Testing of Urogenital *S. aureus* Clinical Isolates from Clinical Samples

Table 3(a) and Table 3(b) shows that *S. aureus* isolates in HIV positive patients [106/403 (26.3)], were less susceptible (P = 0.0001) to Amikacin compared to HIV negative participants [213/403 (52.9%). Nitrofurantoin showed great bacteria clearance in both HIV positive patients [102/403 (25.2%)] and HIV negative individuals 214/403 (53.1%), where all bacteria isolates were susceptible (P = 0.0001). Moderate (P = 0.0001) sensitivity to Vancomycin was seen in HIV positive patients [78/403 (19.4%)] compared to 212/214 (52.6%) seen amongst HIV negative participants. Ciprofloxacin recorded the highest (P = 0.0001) degree of

antimicrobial resistance in HIV positive patients [159/403 (39.5%)] compared to 166/403 (41.2%) seen in HIV negative participants. Resistance to Augmentin was observed to be higher (P = 0.0001) in isolates from HIV patients [148/403 (36.7%0] than HIV negative participants [165/403 (40.9%)]. Cephalosporins; Ceftriaxone had huge resistance in HIV positive patients [133/403 (33.0%)], which was lower (P = 0.0001) than HIV negative individuals 214/403 (53.1%), where all clinical isolates resisted all antibiotics which were tested. Resistance to Bactrim, that is routinely used as prophylaxis in HIV patients was lower (P = 0.0001) in HIV positive patients (135/403 (33.5%) than 165/403 (40.9%) observed in HIV negative individuals.

Table 3. (a) Antibiotic susceptibility profiles of urogenital *S. aureus* in HIV positive and HIV negative patients; (b) Antibiotic susceptibility profiles of urogenital *S. aureus* in HIV positive and HIV negative patients.

(a)

Antibiotics	Susceptibility	Group n (%)			P. Value (P ≤ 0.05)
		HIV	Non-HIV	Total	
Augmentin	S	25 (6.2)	49 (12.2)	74 (18.4)	0.0001
	I	16 (4.0)	0 (0.0)	16 (4.0)	
	R	148 (36.7)	165 (40.9)	313 (77.7)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	
Ceftriaxone	S	39 (9.7)	0 (0.0)	39 (9.7)	0.0001
	I	17 (4.2)	0 (0.0)	17 (4.2)	
	R	133 (33.0)	214 (53.1)	347 (86.1)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	
Amikacin	S	106 (26.3)	213 (52.9)	319 (79.2)	0.0001
	I	24 (6.0)	0 (0.0)	24 (6.0)	
	R	59 (14.6)	1 (0.2)	60 (14.9)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	
Ciprofloxacin	S	17 (4.2)	0 (0.0)	17 (4.2)	0.0001
	I	13 (3.2)	48 (11.9)	61 (15.1)	
	R	159 (39.5)	166 ((41.2)	325 (80.67)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	
Erythromycin	S	38 (9.4)	0 (0.0)	38 (9.4)	0.0001
	I	11 (2.7)	48 (11.9)	59 (14.7)	
	R	140 (34.7)	166 (41.2	306 (75.9)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	

Continued

(b)					
Clindamycin	S	64 (15.9)	163 (40.4)	227 (56.3)	0.0001
	I	20 (5.0)	11 (2.7)	31 (7.7)	
	R	105 (26.1)	40 (9.9)	145 (36.0)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	
Chloramphenicol	S	41 (10.2)	0 (0.0)	41 (10.2)	0.0001
	I	20 (5.0)	11 (2.7)	31 (7.7)	
	R	128 (31.8)	203 (50.4)	331 (82.1)	
	Total	189 (46.9)	214 (53.1)	403 (20.0)	
Bactrim	S	30 (7.4)	37 (9.2)	67 (16.7)	0.045
	I	24 (6.0)	12 (3.0)	36 (8.9)	
	R	135 (33.5)	165 (40.9)	300 (74.4)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	
Vancomycin	S	78 (19.4)	212 (52.6)	290 (72.0)	0.0001
	I	13 (3.2)	0 (0.0)	13 (3.2)	
	R	98 (24.3)	2 (0.5)	100 (24.8)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	
Nitrofurantoin	S	102 (25.2)	214 (53.1)	316 (78.4)	0.0001
	I	18 (4.5)	0 (0.0)	18 (4.5)	
	R	69 (17.1)	0 (0.0)	69 (17.1)	
	Total	189 (46.9)	214 (53.1)	403 (100.0)	

S = Sensitive I = Intermediate R = Resistant.

4.6. Antimicrobial Susceptibility Testing of Nasal Carriage *S. aureus* Clinical Isolates from Clinical Samples

Nasal carriage *S. aureus* clinical isolates were more sensitive to commonly used antibiotics. *S. aureus* clinical isolates from HIV positive patients were ironically more sensitive to tested antibiotics, compared to those from the control group (Table 4). The sensitivity patterns of 167 *S. aureus* isolates were observed between HIV positive and HIV negative participants as follows; Clindamycin [77 (46.1)] vs 30 (18.0%) P = 0.0001], Chloramphenicol [70 (41.9%) vs 36 (21.6%) P = 0.004] and Amikacin [65 (38.9%) vs 60 (35.9%) P = 0.0001. Resistance to some of the tested antibiotics between HIV positive and HIV negative was seen as follows; Amoxicillin [84 (50.3%) vs 65 (38.9%) P = 0.024] and Ciprofloxacin [70 (41.9%) vs 58 (34.7%) P = 0.013].

4.7. Multiple Antibiotic Resistance Indices

Multiple antibiotic resistance indices of antibiotics used in the treatment of urogenital *S. aureus* observed in the study population were high; in both HIV positive

and HIV negative participants. *S. aureus* MAR index in HIV positive patients was 0.9 and in HIV negative participants, it was 0.7. The MAR index of antibiotics used in the treatment of nasal carriage *S. aureus* in HIV positive and HIV negative persons was the same (0.4) as seen in Table 5.

Table 4. Antibiotic susceptibility profiles of nasal carriage *S. aureus* in HIV and Non-HIV.

Antibiotics	Susceptibility	Group n (%)			P. Value (P ≤ 0.05)
		HIV Positive	HIV Negative	Total	
Amoxicillin	S	11 (6.6)	2 (1.2)	13 97.8)	0.024
	I	5 (3.0)	0 (0.0)	5 (3.0)	
	R	84 (50.3)	65 (38.9)	149 (89.2)	
	Total	100 (59.9)	67 (40.1)	167 (100.0)	
Amikacin	S	65 (38.9)	60 (35.9)	125 (74.5)	0.0001
	I	29 (17.4)	0 (0.0)	29 (17.4)	
	R	6 (3.6)	7 (4.2)	13 (7.8)	
	Total	100 (59.9)	67 (40.1)	167 (100.0)	
Ciprofloxacin	S	30 (18.0)	9 (5.4)	39 (23.4)	0.013
	I	0 (0.0)	0 (0.0)	0 (0.0)	
	R	70 (41.9)	58 (34.7)	128 (76.6)	
	Total	100 (59.9)	67 (40.1)	167 (100.0)	
Erythromycin	S	30 (18.0)	14 (8.4)	44 (26.3)	0.0001
	I	1 (0.6)	12 (7.2)	13 (7.8)	
	R	69 (41.3)	41 (24.6)	110 (65.9)	
	Total	100 (59.9)	67 (40.1)	167 (100.0)	
Clindamycin	S	77 (46.1)	30 (18.0)	107 (64.1)	0.0001
	I	1 (0.6)	16 (9.6)	17 (10.2)	
	R	22 (13.2)	21 (12.6)	43 (25.7)	
	Total	100 (59.9)	67 (40.1)	167 (100.0)	
Chloramphenicol	S	70 (41.9)	36 (21.6)	106 (63.5)	0.004
	I	0 (0.0)	6 (3.6)	6 (3.6)	
	R	30 (18.0)	25 (15.0)	55 (32.9)	
	Total	100 (59.9)	67 (40.1)	167 (100.0)	

Table 5. Multiple antibiotic resistance index of *S. aureus* and *E. coli* isolates.

Isolates	HIV		Non-HIV	MAR Index
	Resistant Antibiotics (a)	MAR Index	Resistant Antibiotics (a)	
Urogenital *S. aureus*	17	0.9	14	0.7
Nasal *S. aureus*	8	0.4	8	0.4

MAR Index = a/b, Where b (Total number of antibiotics tested) = 18.

4.8. General Prevalence of Drug Resistance Genes of *S. aureus* Isolates in HIV Positive and HIV Negative Persons

In this study, five virulence genes (*vanA, ermA, mecA, sul3* and *ermC*) of *S. aureus* were investigated. The overall prevalence of *vanA* gene 154/500 (30.8%) predominated all other genes. The *ermA*, *mecA, sul3* and *ermC* genes occurred as follows; 148/500 (29.6%), 143/500 (28.6%), 108/500 (21.6%) and 90/500 (18.0%) respectively in the study population (Figures 4-7).

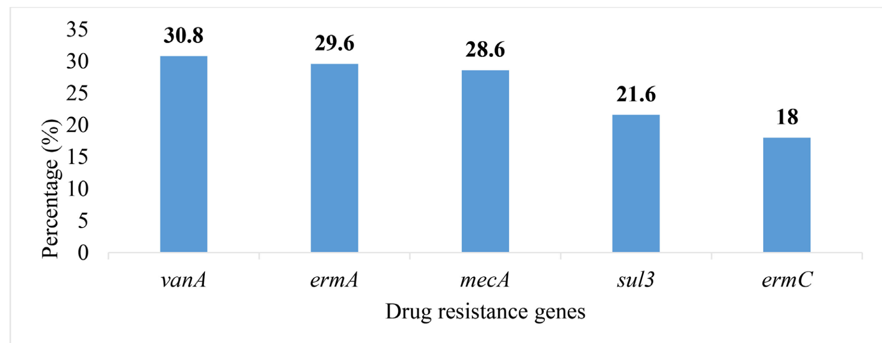

Figure 4. Overall prevalence of drug resistance genes in HIV positive and HIV negative persons.

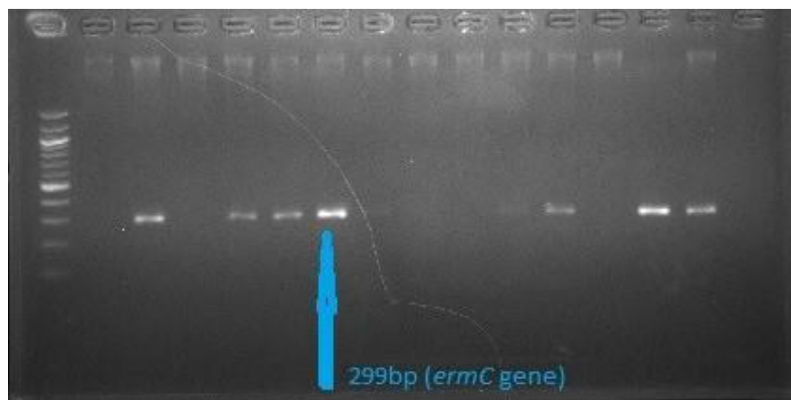

Figure 5. Electrophoresis gel showing *ermC* gene.

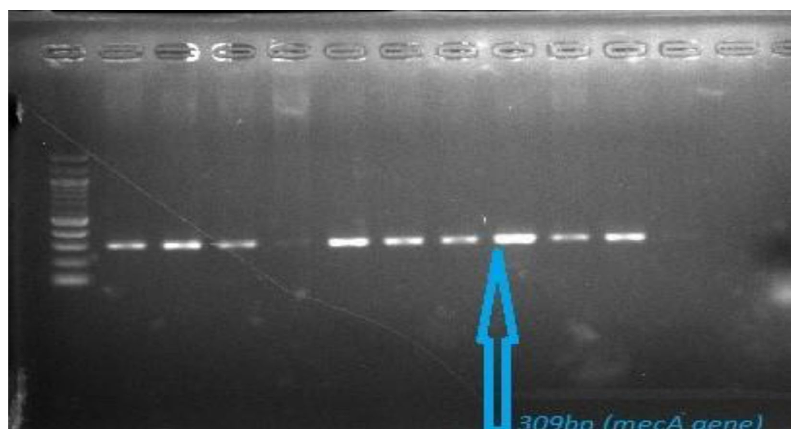

Figure 6. Electrophoresis gel showing *mecA* gene.

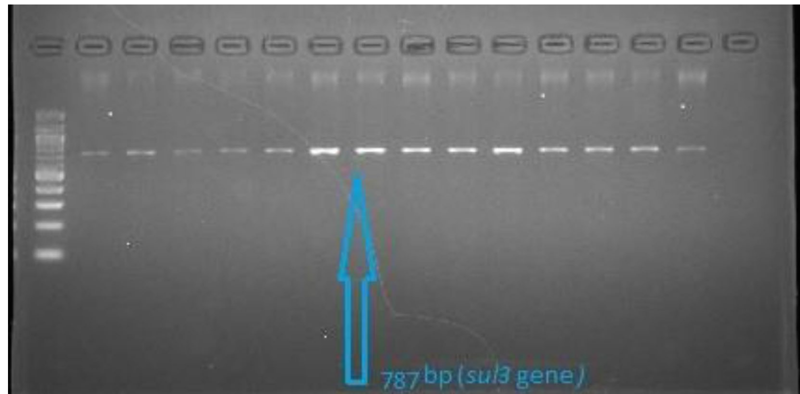

Figure 7. Electrophoresis gel showing *sul3* gene.

4.9. Association of Drug Resistance Genes and Antibiotic Susceptibility

Drug resistance genes were observed to be significantly associated to *S. aureus* resistance to commonly prescribed antibiotics (Table 6). The Macrolide gene (*ermA*) was strongly associated (P = 0.032) to resistance to Chloramphenicol [102/171 (59.6%)]. The Sulfonamide gene (*sul3*) was associated to the following antibiotics; Levofloxacin [66/171 (38.6%), P = 0.029], Ciprofloxacin [72/171 (42.1%), P = 0.029], Bactrim [60/171 (35.1%), P = 0.008] and Augmentin [68/171 (39.8%), P = 0.0001].

Table 6. Association between drug resistance genes and antibiotic resistance.

Antibiotics	Characteristics	Drug resistance gene n (%) n = 171			P. Value (P ≤ 0.05)
		Present	Absent	Total	
		***ermA* gene**			
Chloramphenicol	Sensitive	25 (14.6)	1 (0.6)	26 (15.1)	0.032
	Intermediate	11 (6.4)	6 (3.5)	17 (9.9)	
	Resistant	102 (59.6)	26 (15.2)	128 (74.9	
		***Sul3* gene**			
Levofloxacin	Sensitive	12 (7.0)	11 (6.4)	23 (13.5)	0.029
	Intermediate	20 (11.7)	27 (15.8)	47 (27.5)	
	Resistant	66 (38.6)	35 (20.5)	101 (59.1)	
Ciprofloxacin	Sensitive	7 (4.1)	6 (3.5)	13 (7.6)	0.029
	Intermediate	6 (3.5)	27 (15.8)	46 (26.9)	
	Resistant	72 (42.1)	40 (23.4)	112 (65.5)	
Bactrim	Sensitive	24 (14.0)	34 (19.9)	58 (33.9)	0.008
	Intermediate	14 (8.2)	5 (2.9)	19 (11.1)	
	Resistant	60 (35.1)	34 (19.9)	94 (55.0)	

4.10. Genotypic Similarities of Bacteria Isolates

Our findings indicated that 15/79 (19.0%) of HIV positive patients had *S. aureus* clinical isolates in their urine and nasal samples, which were identical to each other and 30/79 (38.0%) of the clinical isolates were not identical. These findings were significantly not different (P = 0.135) from what was observed amongst HIV negative participants; where the *S. aureus* clinical isolates were equally distributed as identical isolates [17/79 (21.5%)] and non-identical isolates [17/79 (21.5%)], Table 7.

Table 7. Molecular similarity of *S. aureus* clinical isolates between HIV positive and HIV negative patients.

Identity status	HIV Positive n (%)	HIV Negative n (%)	Total n (%)	P. Value ($P \leq 0.05$)
Identical	15 (19.0)	17 (21.5)	32 (40.5)	
Non-identical	30 (38.0)	17 (21.5)	47 (59.5)	0.135
Total	45 (47.0)	34 (43.0)	79 (100.0)	

5. Discussion

The prevalence of MRSA based on *mecA* detection by PCR was 28.8%; whereby 19.4% was detected amongst HIV positive patients and 9.4% detected amongst HIV negative individuals. Sub-group profiling of urogenital and nasal carriage bacteria showed that *S. aureus* clinical isolates, which were detected both in the urine and in nostrils of HIV positive population, were not identical to each other. The difference in the similarity of isolates amongst HIV positive patients (19.0%) and HIV negative individuals (21.5%) was not significant (P = 0.135). Drug resistance genes investigated in this study were distributed as follows; *vanA* (30.8%), *ermA* (29.6%), *mecA* (28.6%), *sul3* (21.6%) and *ermC* (18.0%). Urogenital *S. aureus* clinical isolates were resistant to locally prescribed antimicrobial agents. Resistance was observed to be higher in HIV negative individuals and HIV positive patients. Resistance against Cephalosporins was severe, while Aminoglycosides showed great clearance of bacteria isolates.

In this study, questionnaires were filled by participants which could lead to information bias; history of antibiotic consumption, thereby reducing the number of potential research participants.

MRSA has been associated to most bacterial infections in both hospital based and community acquired human infections [21]. Colonization of *S. aureus* has been reported in HIV positive patients, with nasal carriage being the reservoir source of the infection [22]. In this study, 500 HIV positive patients and HIV negative participants were recruited, MRSA was detected by *mecA* gene presence in 28.8% of the study population; a greater portion of the clinical isolates were MSSA isolates (71.2%). Amongst HIV positive patients (19.4%), MRSA was more prevalent (P = 0.0001) than HIV negative persons (9.4%). On the other hand, MSSA was slightly less prevalent in HIV positive patients (30.6%), as opposed to 40.6%

detected in HIV negative individuals. Higher colonization rate is an avenue for more serious and complicated diseases in Fako. Several studies have been carried out globally and locally on the prevalence of MRSA. In India, a study reported a 68% prevalence for MRSA [23]. Regional prevalence in Africa has been reported as follows; South Africa 24.4%, Sub-Saharan Africa and Central Africa (40.4%), Middle East (47.5%) [24]. South African report concurs with our findings but others did not. A study conducted by Worku in Ethiopia had similar findings with our results [6]. Other studies reported a relatively lower detection rates; a study in Ghana reported that MRSA was 8.2% in HIV positive patients and 91.8% of the isolates were MSSA [25]. In a similar study conducted by Neupane, MRSA prevalence was 13.8% amongst HIV positive patients and 6.7% amongst HIV negative individuals [26], Kyle *et al.*, MRSA was 20% and 11% in HIV positive and HIV negative controls respectively [27]. In Cameroon, detection rate of MRSA varied with studies; 8.0% in Yaounde [28], 2.0% in Yaounde [29], 12.8% in Buea [3]. The prevalence of MRSA seen in HIV positive patients could be because of the difference in attitude towards antibiotic use. Immune status could play a vital role in MRSA colonization in HIV positive patients. Frequent hospitalization, which is likely to occur amongst HIV patients, could open them to hospital-acquired infections. This study provides valuable knowledge of MRSA infections amongst HIV positive patients but did not explore in detail how different antiretroviral therapy (ART) regimens may influence MRSA colonization, infection rates, or resistance patterns. Given the potential interactions between ART and bacterial pathogens; including effects on immune reconstitution, microbial flora, and antibiotic pharmacokinetics; future research may investigate whether specific ART drugs or combinations alter MRSA risk or antimicrobial resistance profiles. The economic burden of methicillin-resistant *Staphylococcus aureus* (MRSA) infections on local healthcare systems is substantial, comprising direct medical costs, indirect societal costs, and long-term healthcare inefficiencies. MRSA infections require longer hospital stays (additional 6 - 10 days) compared to susceptible strains. Higher costs arise from isolation protocols, advanced diagnostics (PCR testing), and delayed effective treatment. Second-line antibiotics (Vancomycin, Linezolid and Daptomycin) are costlier and may require therapeutic drug monitoring. Treatment failures lead to repeated hospital visits and escalated care. Prolonged illness and disability reduce workforce participation. Caregivers also face income loss due to absenteeism. AMR infections increase demand for specialized isolation units, infection control teams, and antimicrobial stewardship programs. Hospitals face penalties for hospital-acquired infections (HAIs), affecting funding; insurance claims rise, increasing premiums and public health expenditures.

Antimicrobial agents have been widely abused in Africa and around the world, leading to serious antimicrobial resistance by *S. aureus*. This has led to treatment failure and life threatening bacterial infections. In this study, most of the *S. aureus* clinical isolates were resistant to frequently used antibiotics. The highest degree of drug resistance was observed in the Penicillins family (Augmentin) and least in

the Aminoglycoside family (Amikacin). The results of this study revealed that urogenital clinical isolates in both HIV positive and HIV negative participants were sensitive to Amikacin (26.35% vs 52.9%), Nitrofurantoin (25.2% vs 53.1%), Vancomycin (19.4 vs 52.6). Most of the clinical isolates were resistant to commonly prescribed antibiotics. Antimicrobial resistance was observed lesser in HIV positive participants than HIV negative individuals were as follows; Ciprofloxacin (39.5% vs 41.2%), Augmentin (46.9% vs 53.1%), and Bactrim (33.5% vs 40.9%). These findings ironically did not agree with most studies where most clinical isolates were observed to be resistant amongst HIV positive patients while few studies observed severe resistance amongst the healthy population. In a study conducted in Central Nepal, 100% sensitivity to Vancomycin was observed in both HIV positive and HIV negative participants, Gentamicin (27.3% vs 66.7%) in HIV positive and HIV negative participants respectively. Resistance to antibiotics was observed between HIV positive and HIV negative individuals as follows; Erythromycin (63.9% vs 66.7%), Bactrim (45.5% vs 54.5%) and Ciprofloxacin (27.3% vs 33.3%) [26]. In Nigeria, a study reported a high resistance in HIV positive patients compared to HIV negative persons as follows; Augmentin (62.5% vs 33.3%), Ceftriaxone (40% vs 22.2%), Erythromycin (62.5% vs 44.4%), Chloramphenicol (70% vs 44.4%), Ampicillin (65% vs 33.3%) and Ciprofloxacin (22.5% vs 22.2%) [3].

A local research carried out in Bamenda Cameroon observed high sensitivity of uropathogenic *S. aureus* clinical isolates to commonly prescribed antibiotics; Ceftriaxone (69.2%), Augmentin (64.1%), Ciprofloxacin (64.7%), Gentamicin (84.6%), Vancomycin (79.5%) and resistance was observed against Bactrim (66.7%) [30]. These results suggest a great regional difference in antimicrobial susceptibility in Cameroon. This difference could to attached to the community's poor knowledge on antibiotic use, automedication, incomplete regimens and improper disposition of hospital used vials. Vancomycin sensitivity could be attributed to its local expensive, unavailability, and parenteral nature, which makes it difficult for abuse by the local population. Urogenital *S. aureus* clinical isolates amongst HIV positive patients in Tigray showed varied susceptibility with isolates being 100% sensitive to Bactrim, far higher than 25.2% observed in our study, Erythromycin and Clindamycin both showed 100% sensitivity, contrary to the findings of this study where very few clinical isolates were sensitive to local antimicrobial agents [30]. This could be as a result of the country's policies regulating the use of antibiotics. Nasal carriage *S. aureus* clinical isolates showed great sensitivity to antibiotics in the HIV positive population. Sensitivity was as follows; Amikacin (38.9% vs 35.9%), Cefotaxim (39.5% vs 28.7%), Clindamycin (46.1% vs 18.0%), chloramphenicol (41.9% vs 21.6%). These findings contrast reports from most studies. A report by Goyitom *et al.*, in Ethiopia indicated 100% resistance to Amikacin and 33.7% resistance to Clindamycin [31]. A Buea based study by Morgan *et al.*, reported a 67.65% resistance to Amikacin and 50% resistance to Clindamycin [3]. In this study, resistance to Erythromycin (41.3%) was far higher than 10%

seen in Ghana [32] and contrast with the findings of Amit in Yaounde [23]. Erythromycin is a common antibiotic that has been gradually abused due to indiscriminate use of the antibiotic. Other studies indicated a sensitivity of 75% to Erythromycin [29]. This high level of antibiotic sensitivity amongst HIV positive patients could be attributed to consistent medical surveillance involving HIV patients; including prophylactic treatment with Cotrimoxazole administration. The study population may have had a limited exposure to previous antibiotics, thereby reducing selective pressure. However, nasal carriage bacteria represent the normal flora, which are eventually less virulent and less invasive. This study highlights the need for continuous antibiotic monitor and restricted use of antibiotics both in HIV positive and HIV negative individuals. *S. aureus* clinical isolates from urine samples had a MAR index of 0.9 amongst HIV positive patients and 0.7 amongst HIV negative individuals. These indices are generally high; insinuating that the clinical isolates were resistant to majority of tested antibiotics. This contrasted with the 0.2 MAR index reported by Igwe [33] in Nigeria. Environmental availability of antibiotics can enhance community acquired drug resistance. The clinical impact of antibiotic resistance on routine treatment failures is profound, affecting patient outcomes, healthcare systems, and public health. Antibiotic resistance often renders first-line antibiotics ineffective, leading to delayed appropriate treatment. This also forces reliance on broader-spectrum agents with "last resort" antibiotics, with increasing toxicity risks. Moreover, infections caused by resistant pathogens exhibit significantly worse outcomes due to limited salvage therapies. In most cases, resistance leads to persistent infections; requiring multiple antibiotic courses. However, Treatment failures often increases risks of sepsis, abscess formation, or metastatic infections from untreated resistant *Staphylococcus aureus*. Resistant infections require longer ICU stays, advanced diagnostics (PCR for resistance genes), and expensive antibiotics. Immunocompromised patients face life-threatening infections when first-line therapies fail.

Drug resistance genes have influenced antimicrobial resistance, illisudating the importance of evaluating antibiotic resistance traits such as *mecA* gene, *vanA* gene, *ermA* gene, *ermC* and *sul3* genes; for *S. aureus* clinical isolates using PCR. In this study, the *S. aureus* presumptive clinical isolates were screened using PCR and 28.6% of the isolates carried the *mecA* gene, 30.8% carried the *vanA* gene, 29.6% carried the *ermA* gene, 216% carried the *sul3* gene and 18.0% carried the *ermC* gene. The *vanA* gene predominated other genes in our study. The results of this study corroborated with findings of Ahmed *et al.*, with a *vanA* detection rate of 29.45 [34]. This *vanA* detection rate was closer to 21.43% observed in Nigeria [35]. Though the study was far lower than 81.2% reported in Egypt in 2022 [36].

50% detected amongst hospitalized patients in Nigeria in 2023 [37] and 48.5% reported in Tanzania, in 2024 [38].

This indicates serious health risk towards *S. aureus* antimicrobial resistance, especially the Methicillin resistant *S. aureus*. The *mecA* gene is used to detect MRSA in clinical isolates. Our findings were in agreement with those of Martin *et*

al., in 2024 [35] but did not agree with the higher (95%) detection rate observed by Rosa *et al.*, in Mexico city [39] and 96.8% observed by Muhammad in Pakistan [40]. In Nepal, 100% detection rate was reported amongst people living HIV and health individuals [41]. Our results were also similar to the findings reported in Yaounde Cameroon by Eyoh *et al.* [42]. This indicates a high MRSA prevalence hence, multiple antibiotic resistance, requiring alternative treatment options, which may be relatively expensive and unavailable in rural area in Fako. This is a major concern and need for urgent surveillance and stewardship programs to prevent the spread of drug resistance traits. Macrolides including Erythromycin and Clarithromycin are used to treat *S. aureus* infection in humans. Most of the resistance to Macrolides has been attributed to erm gene traits, which encodes methylase and inducing drug resistance. Our findings indicated that *ermC* detection rate of 18.0% greatly aligns with the 18.7% reported in Nepal [43], 17.86% reported by Martin in 2024 [35] and 20.5% Iran [44]. However, these findings differed with the findings of other studies which were higher; 4 4.4%, 36.7% and 33.0% all reported in different regions in Iran [45]-[47] but higher than the study reported by Fatma in 2015, with a 10% detection rate [48]. The detection rate of *ermA* gene was higher than most studies; 3% reported by Gholam *et al.*, in 2016 [47], 11% reported in Iran [44], 11.1% in Iran [45], 15.6% reported in Nepal [43]. These results were lower than the 46.7% and 81.9% reported by Saeed *et al.*, and Fatma *et al.*, respectively [48] [49]. This presents serious threats of infection using Clindamycin where it is often used as alternative to Penicillin-allergic patients. The *ermA* gene is also capable of inducible resistance; detectable by the D test. Treatment may fail should the D test be missed or not conducted in a normal antimicrobial susceptibility test. A call for control strategies to curb the spread of drug resistant bacteria.

The study sub-population whose *S.* aureus clinical isolates were profiled showed that majority of the urogenital and nasal carriage *S. aureus*, isolated from HIV positive patients were putatively not identical to each other. Amongst these HIV positive patients, isolates were observed as identical (19.0%) and non-identical (38.0%). On the other hand, there was equal distribution of identical clinical isolates (21.5%) and non-identical clinical isolates (21.5%) amongst HIV negative individuals, although the difference in the distribution was not significant. These results suggest that *S. aureus* clinical isolates from HIV positive patients were ironically exogenous while those isolated from HIV negative individuals could be endogenous. The need for further research with a wider population to ascertain this similarity. Our findings however did not align with the postulations of Keneh *et al.*, in 2023 [49].

Sequencing the genome of *S. aureus* is ideal for comparing clinical isolates using their drug resistance genes. This will ascertain that the isolates are 100% similar or not and to be considered as bacteria of the same strain. The genes were not sequenced in this study and this limitation does not affect our conclusions because the isolates were not identical, based on the presence or absence of drug resistance genes. These findings strongly suggest that opportunistic infections amongst HIV

patients are most at times, not from the normal flora.

6. Conclusion

This study revealed a high prevalence of Methicillin resistance *S. aureus* and severe antibiotic resistance amongst *S. aureus* clinical isolates with a high detection rate of drug resistance genes. *S. aureus* clinical isolates from the urine of HIV positive patients were not from the normal flora (nostrils). These findings indicate a future antibiotic resistance hazard with Methicillin Resistance *Staphylococcus aureus*; a clarant call for antibiotic stewardship and surveillance, effective combination therapies, rational antibiotic policies and continuous research on the mechanisms of drug resistance traits transfer amongst people living with HIV.

Data Availability

The data analyzed and used in this study are available to those who request it.

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Contributions of the Authors

Anumbondem Augustine, Pokam Benjamin Thumamo and Nyasa Babila Raymond conceived this research. Anumbondem Augustine and Ndille Noelar Elua did sample collection and culture of research samples. Anumbondem Augustine, Nyasa Babila Raymond, Pokam Benjamin Thumamo, Ayang Blessing Enjong and Ghogomu Mbigha Stephen carried out PCR procedure. Data analysis was done by Anumbondem Augustine, Nkai Gideon Nyenty and Nyasa Babila Raymond. Anumbondem Augustine wrote the first draft of the manuscript. Raymond Babila Nyasa and Pokam Benjamin Thumamo reviewed the manuscript. All the authors read and approved the final version of this manuscript

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Conflicts of Interest

The authors declare no competing interest.

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