



Antibiotic Resistance Patterns of MRSA associated with Hospital-Acquired Infections in Tertiary Healthcare Facilities in Rwanda

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Citation: Usengimana A, Niyikora N, Bisanukuri E, Emmanuel M, Uwumuremyi F, et al. (2026) Antibiotic Resistance Patterns of MRSA associated with Hospital-Acquired Infections in Tertiary Healthcare Facilities in Rwanda. J Infect Dis & Pati Care 3: 33.

Abstract

Background: Methicillin-Resistant *Staphylococcus aureus* (MRSA) is a leading cause of Hospital-Acquired Infections (HAIs), contributing to increased morbidity, mortality and healthcare costs. Despite reports of MRSA in Rwanda, its specific role in HAIs within local hospitals remains poorly characterized.

Objectives: In this study, we aimed to investigate antimicrobial resistance patterns of MRSA, characterize *mecA* gene presence and SCCmec types and identify MRSA associated with nosocomial infections in the Rwandan hospital settings.

Methods: MRSA isolates were obtained from *S. aureus*-positive clinical samples. Antimicrobial susceptibility testing was performed using a panel of antibiotics. MRSA-associated HAI cases were documented. PCR was used to confirm *mecA* gene presence and SCCmec typing was conducted to determine genetic diversity.

Results: Results of the 110 *S. aureus* analyzed, 48% were methicillin-resistant. MRSA cases were distributed across hospital wards: Pediatrics (5), Surgery (3), ICU (3), Neonatology (3), Dialysis (2), PICU (2) and Internal Medicine, HDU, Gynecology and Urology (1 each). MRSA strains exhibited high resistance to penicillin (98%), ampicillin (92%), erythromycin (77%) and ciprofloxacin (73%), while most remained susceptible to linezolid, levofloxacin and vancomycin. Among MRSA-positive cases, 27% were associated with HAIs. Molecular characterization revealed the presence of SCCmec types I, II, IV and V, along with untippable genes, indicating substantial genetic diversity among MRSA strains.

Conclusions: MRSA is prevalent among hospital-acquired infections in Rwanda and is associated with multidrug resistance and diverse genetic profiles. These findings underscore the need to strengthen infection prevention and control strategies to mitigate MRSA-related morbidity and healthcare burden in Rwanda.

Keywords: MRSA; Resistance; Nosocomial infections; Tertiary healthcare

Received date: April 17, 2026; **Accepted date:** May 13, 2026; **Published date:** May 15, 2026



Introduction

Staphylococcus aureus (*S. aureus*) remains a major public health threat and is associated with a wide range of clinical manifestations, including local and invasive infections [1]. Management of *Staphylococcal* infections is challenging due to the emergence of Methicillin-Resistant *S. aureus* (MRSA) strains.

MRSA was first detected in the 1960s, just one year after methicillin was introduced for treating *Staphylococcal* infections and previous studies have shown that methicillin resistance is mediated by the *mecA* gene, which encodes penicillin-binding protein 2a (PBP2a), conferring low affinity for β -lactam antibiotics [2-4]. The World Health Organization (WHO) has classified MRSA as a high-priority pathogen due to its association with high mortality, prolonged hospital stays and elevated treatment costs [5]. Currently, *S. aureus* has been implicated in both hospital- and community-acquired infections. Although MRSA is well-documented in high-income countries, data are limited in developing countries, including Rwanda.

MRSA has been frequently reported in patients with prolonged hospital stays and also immunocompromised individuals [6,7]. The frequency of MRSA varies significantly by region. In Africa, the prevalence of *Staphylococcal* infections ranges from 12%-80% [8]. This variability has been attributed to inadequate infection control policies and poor antibiotic stewardship. Studies conducted in East Africa have reported the prevalence of MRSA infections in hospital settings to range between 31% and 82% [8,9]. Recent data from Gabon indicate that 3%-20% of *S. aureus* isolates from skin and soft tissue infections over an 11-year period were MRSA, highlighting the urgent need for strengthened infection prevention and control measures [10].

MRSA is most commonly spread through direct contact in hospitals, particularly in high-risk wards such as intensive care units, surgical units, dialysis centers and pediatric wards [7]. Active decontamination programs and antibiotic stewardship initiatives, alongside genomic surveillance, have shown potential in reducing healthcare-associated MRSA transmission [6,11]. However, resource constraints in many African healthcare settings hinder the widespread adoption of these strategies.

In Rwanda, earlier studies have reported MRSA prevalence in clinical settings, yet there remains a lack of recent hospital-level data on MRSA antimicrobial resistance pattern [12]. In addition to methicillin, data are scarce on the efficacy of other antibiotics in treating MRSA associated infections. This study aimed to investigate MRSA among hospital-acquired infections and associated antibiotic resistance profiles in Rwanda.

Materials and Methods

The present study was conducted in Rwanda. A consecutive sampling strategy was employed. All routine

clinical specimens with confirmed *Staphylococcus aureus* isolates between March and June 2025 were included. 110 *S. aureus* isolates were collected and considered for further investigations. Ethical clearance for this study was obtained including the use of isolates collected routinely for diagnostic purposes.

Bacterial isolation and identification

Clinical specimens collected from hospital wards and the Outpatient Department (OPD) were received and registered in the microbiology laboratory. Samples were initially examined microscopically. Specimens suspected of containing *Staphylococcus aureus* indicated by gram staining showing Gram-positive cocci in clusters were inoculated onto blood agar plates supplemented with 5% defibrinated sheep blood for culture and further identification. The inoculated plates were incubated at 37°C for overnight. After incubation, colonies were evaluated for characteristic morphology; whitish colonies demonstrating β -hemolysis (clear zones surrounding colonies) were subjected to further testing. Gram staining was repeated to confirm Gram-positive cocci in clusters.

Phenotypic identification of *S. aureus* involved mannitol fermentation on mannitol salt agar, catalase testing and both slide and tube coagulase assays. In addition, the VITEK® 2 Compact system (bioMérieux, France) was employed for confirmatory identification. Pure colonies were suspended in sterile saline to a 0.5 McFarland turbidity standard and inoculated into ID-GP cards specific for Gram-positive bacteria. The platform automatically processed the biochemical reactions and provided organism identification based on its unique metabolic profile using the VITEK® 2 software.

Antimicrobial Susceptibility Testing (AST)

Antimicrobial Susceptibility Testing (AST) of the confirmed *Staphylococcus aureus* isolates was conducted in two different sites. The Kirby-Bauer disk diffusion method was performed manually on Mueller-Hinton agar following the Clinical and Laboratory Standards Institute (CLSI) 2024 guidelines. Antibiotic discs tested included penicillin (10 units), cefoxitin (30 μ g), erythromycin (15 μ g), clindamycin (2 μ g), trimethoprim-sulfamethoxazole (1.25/23.75 μ g), tetracycline (30 μ g), linezolid (30 μ g), chloramphenicol (30 μ g), ciprofloxacin (5 μ g), gentamicin (10 μ g), ampicillin (30 μ g), daptomycin (30 μ g), levofloxacin (5 μ g) and oxacillin (1 μ g). Methicillin resistance was primarily determined by cefoxitin disk diffusion, with isolates exhibiting a zone of inhibition $\leq$ 21 mm classified as MRSA and those $\geq$ 22 mm as Methicillin-Susceptible *S. aureus* (MSSA). Oxacillin disk diffusion was also used as a supplementary phenotypic test for MRSA detection. In addition to for some isolates, antimicrobial resistance was performed using the automated VITEK® 2 Compact system (bioMérieux, France), employing AST-GP67 cards. These cards contain Minimum Inhibitory Concentration (MIC) panels for antibiotics including cefoxitin (for MRSA screening), penicillin, oxacillin, clindamycin, erythromycin,



linezolid, vancomycin, daptomycin, tetracycline/doxycycline, trimethoprim-sulfamethoxazole, gentamicin, moxifloxacin, levofloxacin and rifampicin. Results were interpreted according to CLSI 2024 standards. All confirmed MRSA isolates were preserved in skim milk and stored at -20°C for subsequent molecular analyses.

Detection of MRSA resistance genes (mec A gene)

To confirm methicillin resistance, the presence of the *mecA* gene was detected using conventional Polymerase Chain Reaction (PCR). Stored MRSA isolates were first sub-cultured on Mannitol Salt Agar (MSA) and incubated overnight at 37°C. Bacterial colonies were then harvested and suspended in nuclease-free water. DNA was extracted using a heat-lysis method by heating the bacterial suspension at 95°C for 20mins. The *mecA* gene was amplified using the following primers (Integrated DNA Technologies, IDT, USA): Forward primer (*mecAF*): 5'-GTAGAAATGACTGAACGTCCGATAA-3', Reverse primer (*mecAR*): 5'-CCAATTCCACATTGTTTCGGTCTAA-3'. Each PCR reaction contained 0.5 µM of each primer, 3 µL of DNA template and 2X Master Mix in a final volume of 20 µL. The following program was used; Initial denaturation at 95°C for 15 mins (1 cycle), Denaturation at 95°C for 30 seconds (34 cycles), Annealing at 58°C for 30 second, Extension at 72°C for 1 minute and final extension at 72°C for 5 minutes. PCR products were analyzed by electrophoresis on a 2% agarose gel prepared in 1 × TAE (Tris-acetate-EDTA) buffer. Electrophoresis was run at a constant voltage of 235 V for 20mins. DNA fragments were stained with ethidium bromide and visualized under UV light using a Bio-imager (UVP). A 1000 bp DNA ladder was included as a molecular weight marker. Isolates were considered confirmed MRSA when phenotypic methicillin resistance correlated with the presence of the *mecA* gene by PCR and only these isolates were included in further analysis.

Assessment of Hospital-Acquired Infections (HAIs) and SCCmec typing

To determine the proportion of MRSA cases associated with Hospital-Acquired Infections (HAIs), patient medical records were reviewed. An HAI was defined as any infection occurring more than 48hrs after hospital admission, without clinical signs of prior incubation, in accordance with the World Health Organization (WHO) guidelines. MRSA isolates confirmed in the microbiology laboratory were cross-referenced with patient records to identify those that fulfilled the HAI criteria. To further characterize these MRSA isolates, Staphylococcal Cassette Chromosome *mec* (SCCmec) typing was conducted using conventional PCR targeting the Joining (J) regions of SCCmec elements, which are specific to types I-V. A single pair of universal primers was used for amplification (Table 1):

Forward primer (SCCmec-J-F): 5'-AGTTGTAGTTGTCGCGAGT-3'
Reverse primer (SCCmec-J-R): 5'-TTGAGGATGGAGCGAGTT-3'

PCR amplification yielded type-specific amplicons with the following product sizes: 613 bp (type I), 398 bp (type II), 280 bp (type III), 776 bp (type IV) and 325 bp (type V). PCR products were resolved on a 2% agarose gel stained with GreenStar™ Nucleic Acid Staining Solution and band sizes were estimated using a molecular weight DNA ladder. SCCmec types were assigned based on the observed band sizes.

Table 1: Primers used for detection of MRSA resistance genes.

Name of primer	Sequence of primer (5' 3')	Primers length in bp	Target gene
<i>mecAF</i> and <i>mecAR</i>	GTAGAAATGACTGAACGTCCGATAA CCAATTCCACATTGTTTCGGTCTAA	25	<i>mecA</i>
Jl region primers (I-V) F-R	AGTTGTAGTTGTCGCGAGT TTGAGGATGGAGCGAGTT	19 18	Types I, II, III, IV, V

Quality control

For quality assurance of identification and susceptibility procedures, the following American Type Culture Collection (ATCC) strains were used: *Staphylococcus aureus* ATCC 25923 as a quality control strain for disk diffusion testing and positive control for identification; *Staphylococcus epidermidis* ATCC 12228 was used as a negative control for identification tests. For MIC-based testing with the VITEK® 2 system, *S. aureus* ATCC 29213 was used as the quality control strain.

Graphical depiction and data analysis

GraphPad Prism v10 software (GraphPad Software, La Jolla CA, USA) and Excel were used to depict all statistics.

Results

Description of the study population and MRSA distribution

A total of 110 *Staphylococcus aureus* isolates were analyzed, of which 53 (48.2%) were methicillin-resistant (*S. aureus*, MRSA). MRSA was slightly more common in males (51%) than in females (49%). The mean age of patients with MRSA was 28.4 ± 5 years. Patients of less than 10 years old had the highest proportion of MRSA cases (34%). Individuals above 60 years old accounted for 6.4% of MRSA cases. The majority of MRSA isolates were obtained from blood samples 71.7% (38/53), followed by pus 17% (9/53) and urine 5.7% (5/53). A few isolates came from cerebrospinal fluid 1.8% (1/53), while no MRSA was detected in vaginal swabs. Tracheal aspirates accounted for 3.7% (2/53) of MRSA-positive samples. MRSA was most frequently isolated from patients in the pediatric ward which represented 18.9% (10/53), ICU 17%, (9/53) internal medicine and surgery 15.1%, (8/53) and



neonatology 5.7% (3/53). Other wards such as dialysis, HDU, emergency, OPD orthopedics, PICU, gynecology and urology each accounted for a small proportion of cases (Table 2).

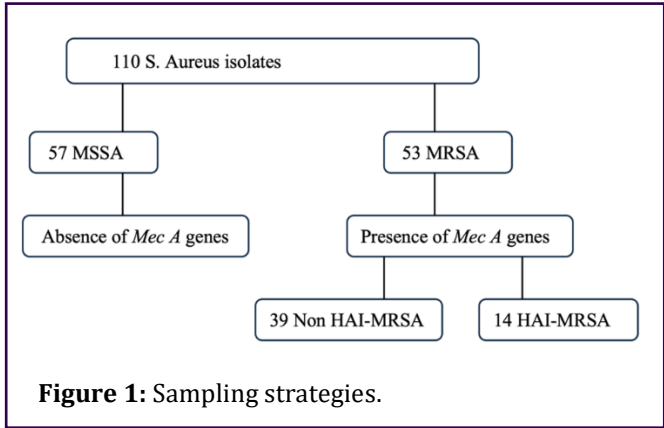


Table 2: Demographics and distribution of *S. aureus* and MRSA isolates.

Demography		Total (n=100)	MRSA (53)	P value
		N (%)	N (%)	
Sex	Male	59 (53.6)	27 (51)	0.36
	Female	51 (46.4)	21 (49)	
Age group (years)	0-10	33 (30)	18 (34)	0.706
	10-20	15 (13.6)	6 (11.3)	
	21-30	16 (14.5)	7 (13.2)	
	31-40	15 (13.6)	6 (11.3)	
	41-50	15 (13.6)	6 (11.3)	
	51-60	6 (5.5)	3 (5.7)	
	>60	10 (9.1)	7 (6.4)	
Specimen	Blood	73 (66.4)	38 (71.7)	0.324
	Pus	24 (21.8)	9 (17)	
	Urine	6 (5.5)	3 (5.7)	
	Vaginal swab	3 (2.7)	0 (0)	
	Trachea asp.	3 (2.7)	2 (3.7)	
	CSF	1 (0.9)	1 (1.8)	
Wards	Pediatric	24 (21.8)	10 (18.9)	0.672
	IM	20 (18.2)	8 (15.1)	
	ICU	15 (13.6)	9 (17)	
	Surgery	18 (16.4)	8 (15.7)	
	Dialysis	4 (3.6)	3 (5.7)	
	HDU	2 (1.8)	1 (1.9)	
	Neonatology	5 (4.5)	3 (5.7)	
	Emergency	3 (2.7)	1 (1.9)	
	OPD	3 (2.7)	2 (3.8)	
	Orthopedic	1 (0.9)	1 (1.9)	
	PICU	3 (2.7)	3 (5.7)	

	Gynecology	9 (8.7)	3 (5.7)
	Urology	3 (3.7)	2 (3.8)

Methicillin-Resistant *Staphylococcus aureus* (MRSA) exhibits resistance to a broad spectrum of antibiotics

Staphylococcus aureus exhibited increased resistance to a wide range of antibiotics (Table 3). Out of the total MRSA isolates analyzed, 48% were identified as methicillin-resistant *Staphylococcus aureus* (MRSA), while 52 % were susceptible to *S. aureus* (MSSA) (Figure 2A). The antibiotic susceptibility testing of MRSA isolates revealed high resistance rates to several commonly used antibiotics. The highest resistance was observed for penicillin (98%), followed closely by ampicillin (94%) and erythromycin (81%), indicating widespread β -lactam resistance. Moderate levels of resistance were observed against, ciprofloxacin (77%), Trimethoprim/sulfamethoxazole (58%) and piperacillin tazobactam (50%). All isolates remained 100% susceptible to Vancomycin and Linezolid suggesting that these antibiotics may still retain efficacy against MRSA. These findings underscore the multidrug-resistant nature of MRSA and highlight the critical need for ongoing antimicrobial surveillance and stewardship programs in Rwanda.

Table 3: Resistance pattern of methicillin resistant *Staphylococcus aureus*.

Antimicro bials	<i>S. aureus</i> isolates (N=110)			MRSA isolates (N=53)	
	Suscep tible	Interme diate	Resis tant	Suscep tible	Resis tant
	N (%)	N (%)	N (%)	N (%)	N (%)
Penicillin	6 (5)	NA	104 (95)	1 (2)	52 (98)
Erythromy cin	46 (42)	NA	64 (58)	10 (19)	43 (81)
Clindamyci n	85 (77)	NA	25 (23)	37 (70)	16 (30)
Trimethop rim- Sulfameth oxazole	63 (57)	NA	47 (43)	22 (42)	31 (58)
Tetracycli ne	78 (71)	NA	32 (29)	34 (64)	19 (36)
Chloramph enicol	91 (83)	NA	19 (17)	42 (79)	11 (21)
Ciprofloxa cin	49 (45)	NA	61 (55)	12 (23)	41 (77)
Gentamyci n	77 (70)	1 (0.9)	32 (29)	39 (74)	14 (26)
Linezolid	105 (96)	NA	5 (4)	51 (96)	2 (4)



Ampicillin	16 (14)	NA	94 (86)	3 (6)	50 (94)
Daptomycin	91 (83)	NA	19 (17)	39 (74)	14 (26)
Vancomycin	93 (84)	1 (0.9)	16 (15)	45 (85)	8 (15)
Piperacillin-Tazobactam	61 (56)	NA	49 (44)	26 (49)	27 (51)
Levofloxacin	99 (90)	NA	11 (10)	45 (85)	8 (15)
Cotrimoxazole	53 (48)	NA	57 (52)	21 (40)	32 (60)
Nitrofurantoin	92 (84)	NA	18 (16)	41 (77)	12 (23)
Doxycycline	64 (58)	NA	46 (42)	28 (53)	25 (47)

type I was the most frequently identified (30%), followed by types II and V (20% each) and type IV (10%). Notably, 20% of the HAI-MRSA isolates were non-typeable using the primers applied, indicating the possible presence of novel or variant SCCmec genes (**Figure 3D**).

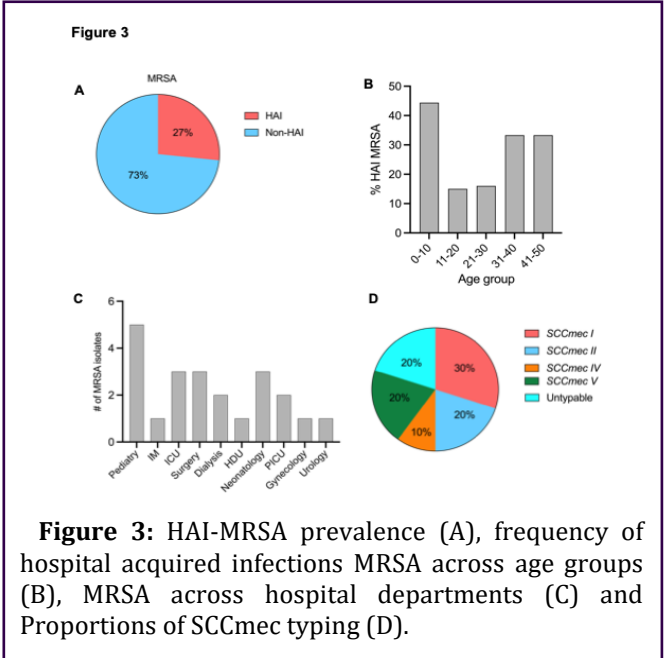


Figure 3: HAI-MRSA prevalence (A), frequency of hospital acquired infections MRSA across age groups (B), MRSA across hospital departments (C) and Proportions of SCCmec typing (D).

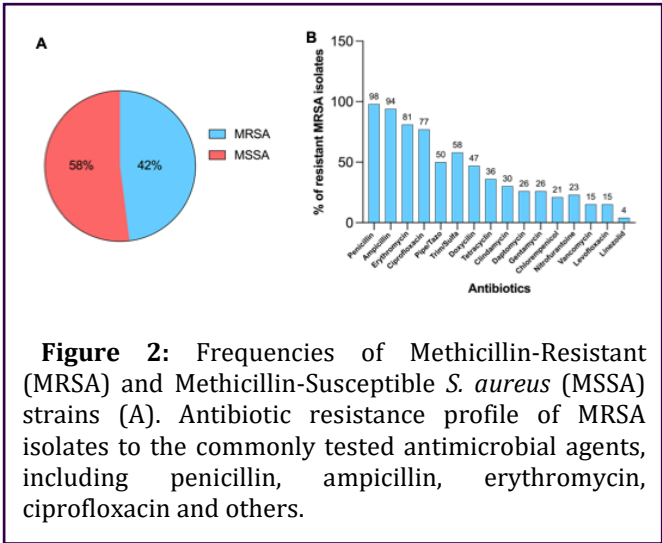


Figure 2: Frequencies of Methicillin-Resistant (MRSA) and Methicillin-Susceptible *S. aureus* (MSSA) strains (A). Antibiotic resistance profile of MRSA isolates to the commonly tested antimicrobial agents, including penicillin, ampicillin, erythromycin, ciprofloxacin and others.

A substantial proportion of MRSA cases are associated with hospital-acquired infections

Out of the 53 confirmed MRSA isolates, 14 (27%) were associated with hospital-acquired infections (HA-MRSA) (**Figure 3A**). These cases were distributed across various hospital wards: five in Pediatrics, three each in Surgery, ICU and Neonatology, two in Dialysis, two in PICU and one each in Internal Medicine (IM), High Dependency Unit (HDU), Gynecology and Urology. Children aged 0-10 years represented the largest proportion of HA-MRSA cases (44.4%, 8/14). Other affected age groups included those aged 31-40 years and 41-50 years, with each group having 33.3% of their MRSA cases classified as healthcare-associated (**Figure 3B**).

Molecular typing of the MRSA isolates was performed using primers specific to SCCmec resistance genes. SCCmec

Discussion

Over the past decades, Methicillin-Resistant *Staphylococcus aureus* (MRSA) has emerged globally as a significant public health concern, characterized by resistance to β -lactam antibiotics, including methicillin and other penicillins [3]. This resistance poses significant therapeutic challenges especially in developing countries including Rwanda, underscoring the need for active surveillance of MRSA. Our findings indicate that the MRSA infection rate was 48 %, with the highest distribution observed among children aged 0-10 years (18.9%). This pattern is consistent with global trends, emphasizing the epidemiological significance of MRSA in younger populations. A study by Said et al. (2025) similarly reported a 32% MRSA prevalence among children and adolescents, highlighting increased burden in these age groups [13]. The elevated prevalence in young children may be attributed to several factors, including immature immune defenses, frequent contact with healthcare environments and recurrent or inappropriate antibiotic use [10]. The predominance of MRSA cases in children under 10 was also documented in previous hospital-based surveillance studies [14].

In comparison with other sample types, blood was identified as the predominant source of MRSA isolates, accounting for 71.7%, which highlights a substantial burden of MRSA-associated bacteremia. In line with this finding, a recent study reported that *Staphylococcus aureus* constituted 25% of Gram-positive bloodstream infections in Rwanda [15]. The study also identified the pediatric (18%), internal medicine (17%) and ICU (17%)



departments as having the highest rates of *S. aureus* infections. This distribution pattern is consistent with reports from other tertiary hospitals, where MRSA is frequently isolated from bloodstream infections across various departments [1,15]. The presence of MRSA across multiple units including pediatrics, ICU, internal medicine and surgical wards poses a significant challenge to infection prevention and control strategies in hospitals.

Antimicrobial susceptibility patterns revealed a multidrug-resistant phenotype among MRSA isolates; this study proved the isolated *S. aureus* were resistant to penicillin. These results reflect the global trend of increasing β -lactam resistance among MRSA strains due to the *mecA* gene encoding penicillin-binding protein 2a (PBP2a), which confers resistance to all β -lactam antibiotics [15]. A study by Masaisa et al. 2018 reported 100% of penicillin resistance, in referral hospitals in Rwanda [12]. Moderate resistance to other first-line antibiotics such as erythromycin and ciprofloxacin, was also limiting therapeutic options. However, relatively low resistance to linezolid, levofloxacin and vancomycin, provides some hope for treatment, although the emergence of resistance to these agents has been increasingly reported and warrants close monitoring [16].

Of note, our study also revealed that Hospital-Acquired MRSA (HA-MRSA) accounted for 27% (n=14) of all MRSA cases. These findings align with previous reports from both regional and global studies. A study in Ethiopia by Asrat et al. (2021) found that over 60% of MRSA isolates in high-dependency as one of critical care unit underscore the vulnerability of patients requiring intensive monitoring or chronic interventions [17]. In dialysis-related units, hemodialysis patients are at an elevated risk for nosocomial infections due to frequent vascular access, repeated contact with healthcare settings and underlying immune compromise. Central venous catheters, in particular, serve as direct portals for pathogen entry, including MRSA. Similar findings were reported by Masaisa et al. (2018) in Rwanda, where critical care and specialized units showed higher rates of HA-MRSA than general wards, likely due to invasive procedures, immunocompromised patients and prolonged hospital stays [12].

The neonatology ward also exhibited a high proportion of HA-MRSA cases (66%), which is consistent with findings from Kenya by Omuse et al. (2020), where neonatal units were identified as hotspots for MRSA transmission, primarily due to underdeveloped immunity in neonates and frequent contact with healthcare providers (9). Moreover, the pediatrics (30%) and PICU (33.3%) wards also demonstrated a substantial burden, aligning with data from Uganda by Kateete et al. (2019), who reported significant HA-MRSA colonization among pediatric patients, particularly in PICUs [18].

In contrast, internal medicine recorded the lowest HA-MRSA rate (12.8%). This divergence might reflect differences in patient risk profiles and less frequent exposure to invasive procedures compared to surgical or

critical care units. However, this finding contradicts some findings, that highlighted higher MRSA incidence in internal medicine due to the presence of elderly patients with comorbidities [14].

Regarding age distribution, the data indicate that children aged 0–10 years represented the largest proportion of HA-MRSA cases (44.4%), despite being only a subset (18/53) of the total MRSA isolates. This is in agreement with a global trend highlighting the growing burden of HA-MRSA among pediatric populations. A multicenter surveillance study by Said et al. (2025) found a 32% MRSA prevalence among children and adolescents, noting that immature immune systems and frequent hospital visits contribute significantly to increased susceptibility [13]. Interestingly, no HA-MRSA cases were identified among patients older than 60 years, despite the presence of MRSA isolates in that group. This observation contrasts with studies from higher-income countries, where older adults are frequently overrepresented in HA-MRSA cases due to factors like frailty, polypharmacy and institutionalization [20]. In the current setting, it is possible that older patients were admitted for conditions unrelated to invasive care or had community-onset MRSA, which requires further molecular differentiation.

The most prevalent SCCmec type identified in this study was SCCmec I (30%), followed by SCCmec II (20%), both of which are typically associated with hospital-acquired MRSA (HA-MRSA). These types carry additional resistance genes and are frequently found in strains isolated from healthcare settings. This correlates well with our earlier finding that 27% of MRSA cases were hospital-acquired, predominantly from High Dependency Unit (HDU) and the dialysis unit, with 100% and 66.6% of MRSA isolates being healthcare-associated, respectively. With Similarity in the neonatology ward (66%), those hospital areas are commonly linked with HA-MRSA due to high antibiotic pressure, invasive procedures and immunocompromised patients. SCCmec I and II are known for their multidrug resistance and are rarely found in community settings. Their presence supports the classification of these isolates as HA-MRSA. A recent study by Gashegu et al. (2024) in Rwanda also reported high frequencies of SCCmec I and II among ICU and surgical ward isolates, reinforcing the idea that these types are hospital-associated in the East African region [15].

SCCmec IV and V, on the other hand are more commonly linked with community-acquired MRSA (CA-MRSA) due to their smaller cassette size and limited resistance genes. However, the detection of SCCmec IV (10%) and V (20%) within our isolates some of which originated from outpatients suggests the presence of community-associated MRSA (CA-MRSA) strains. Their occurrence in both community and hospital settings indicates a potential infiltration of CA-MRSA clones into healthcare environments. This phenomenon has been increasingly documented in Africa and globally. According to Said et al. (2025), the traditionally distinct boundaries between CA- and HA-MRSA are becoming blurred due to increased patient mobility, outpatient care transitions and



overlapping healthcare exposures [13]. From an infection prevention perspective, this trend is concerning as CA-MRSA strains are often more transmissible and may complicate conventional hospital-based MRSA control strategies, thereby underscoring the need for strengthened surveillance and molecular epidemiological monitoring.

The presence of 20% untypable SCCmec genes may indicate: Novel or hybrid SCCmec cassettes, genetic recombination or limitations in the typing method (J I-V primers). This suggests a potential genetic evolution of MRSA strains within local healthcare settings. A similar observation was made in Kenya by Mwachari et al. (2023), where 18% of isolates were untypable and showed novel mec gene arrangements, possibly contributing to diagnostic and treatment challenges [21]. These findings highlight the limitations of conventional SCCmec typing methods and highlight the need for more comprehensive molecular approaches, such as whole-genome sequencing, to fully characterize circulating MRSA clones.

Conclusion

Our findings demonstrate an increased prevalence of multidrug-resistant MRSA, particularly among pediatric patients and in bloodstream infections, highlighting the clinical significance of MRSA. The widespread resistance of MRSA to multiple antibiotics including β -lactams, macrolides and fluoroquinolones limits treatment options and underscores the urgent need for robust antimicrobial stewardship programs. The detection of diverse SCCmec types, including non-typeable strains, reflects ongoing genetic evolution and necessitates enhanced molecular surveillance and infection control efforts. Addressing the spread of MRSA will require an integrated strategy involving continuous monitoring, improved diagnostic capabilities and antibiotic stewardship in clinical settings in Rwanda.

Data Availability Statement

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

Source of Funding

This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

Conflict of Interest

The authors declare no conflicts of interest.

References

1. Chambers HF, DeLeo FR (2009) Waves of resistance: *Staphylococcus aureus* in the antibiotic era. *Nat Rev Microbiol* 7: 629-641. [Crossref] [Google Scholar] [Indexed]
2. Barber M (1961) Methicillin-resistant staphylococci. *J Clin*

Pathol 14: 385-393. [Crossref] [Google Scholar] [Indexed]

3. Katayama Y, Ito T, Hiramatsu K (2000) A new class of genetic element, staphylococcus cassette chromosome mec, encodes methicillin resistance in *Staphylococcus aureus*. *Antimicrob Agents Chemother* 44: 1549-1555. [Crossref] [Google Scholar] [Indexed]
4. Hartman B, Tomasz A (1981) Altered penicillin-binding proteins in methicillin-resistant strains of *Staphylococcus aureus*. *Antimicrob Agents Chemother* 19: 726-735. [Crossref] [Google Scholar] [Indexed]
5. World Health Organization (2024) WHO updates list of drug-resistant bacteria most threatening to human health.
6. David MZ, Daum RS (2010) Community-associated methicillin-resistant *Staphylococcus aureus*: Epidemiology and clinical consequences of an emerging epidemic. *Clin Microbiol Rev* 23: 616-687. [Crossref] [Google Scholar] [Indexed]
7. Otto M (2013) Community-associated MRSA: What makes them special? *Int J Med Microbiol* 303: 324-330. [Crossref] [Google Scholar] [Indexed]
8. Falagas ME, Karageorgopoulos DE, Leptidis J, Korbila IP (2013) MRSA in Africa: Filling the global map of antimicrobial resistance. *PLoS ONE* 8: e68024. [Crossref] [Google Scholar] [Indexed]
9. Maina D, Omuse G, Revathi G, Adam RD (2016) Spectrum of microbial diseases and resistance patterns at a private teaching hospital in Kenya: Implications for clinical practice. *PLOS ONE* 11: e0147659. [Crossref] [Google Scholar] [Indexed]
10. Breurec S, Zriouil SB, Fall C, Boisier P, Brisse S, et al. (2011) Epidemiology of methicillin-resistant *Staphylococcus aureus* lineages in five major African towns: Emergence and spread of atypical clones. *Clin Microbiol Infect* 17: 160-165. [Crossref] [Google Scholar] [Indexed]
11. Köser CU, Holden MTG, Ellington MJ, Cartwright EJP, Brown NM, et al. (2012) Rapid whole-genome sequencing for investigation of a neonatal MRSA outbreak. *N Engl J Med* 366: 2267-2275. [Crossref] [Google Scholar] [Indexed]
12. Masaisa F, Kayigi E, Seni J, Bwanga F, Muvunyi CM (2018) Antibiotic resistance patterns and molecular characterization of methicillin-resistant *Staphylococcus aureus* in clinical settings in Rwanda. *Am J Trop Med Hyg* 99: 1239-1245. [Crossref] [Google Scholar] [Indexed]
13. Said KB, Alshammari K, Ahmed RME, Alshammari F, Jadani AH, et al. (2025) MRSA profiles reveal age- and gender-specificity in a tertiary care hospital: High burden in ICU elderly and emerging community patterns in youth. *Microorganisms* 13: 1078. [Crossref] [Google Scholar] [Indexed]
14. Klein EY, Sun L, Smith DL, Laxminarayan R (2013) The changing epidemiology of methicillin-resistant *Staphylococcus aureus* in the United States: A national observational study. *Am J Epidemiol* 177: 666-674. [Crossref] [Google Scholar] [Indexed]
15. Gashegu M, Ndahindwa V, Rwagasore E, Tuyishime A, Musanabaganwa C, et al. (2024) Diversity, distribution and resistance profiles of bacterial bloodstream infections in three tertiary referral hospitals in Rwanda between 2020 and 2022. *Antibiotics* 13: 1084. [Crossref] [Google Scholar]



[Indexed]

16. Liu C, Bayer A, Cosgrove SE, Daum RS, Fridkin SK, et al. (2011) Clinical practice guidelines by the infectious diseases society of America for the treatment of methicillin-resistant *Staphylococcus aureus* infections in adults and children. Clin Infect Dis 52: e18-e55. [Crossref] [Google Scholar] [Indexed]
17. Kahsay AG, Hagos DG, Abay GK, Mezgebo TA (2018) Prevalence and antimicrobial susceptibility patterns of methicillin-resistant *Staphylococcus aureus* among janitors of Mekelle University, North Ethiopia. BMC Res Notes 11: 294. [Crossref] [Google Scholar] [Indexed]
18. Kateete DP, Namazzi S, Okee M, Okeng A, Baluku H, et al. (2011) High prevalence of methicillin resistant *Staphylococcus aureus* in the surgical units of Mulago hospital in Kampala, Uganda. BMC Res Notes 4: 326. [Crossref] [Google Scholar] [Indexed]
19. Mzee T, Kazimoto T, Madata J, Masalu R, Bischoff M, et al. (2021) Prevalence, antimicrobial susceptibility and genotypic characteristics of *Staphylococcus aureus* in Tanzania: A systematic review. Bull Natl Res Cent 45: 162. [Crossref] [Google Scholar]
20. Kourtis AP, Hatfield K, Baggs J, Mu Y, See I, et al. (2019) Vital signs: Epidemiology and recent trends in methicillin-resistant and in methicillin-susceptible *Staphylococcus aureus* bloodstream infections-United States. MMWR Morb Mortal Wkly Rep 68: 214-219. [Google Scholar] [Indexed]
21. Khasabuli O, Ngugi C, Kiiru J (2020) Diversity of SCCmec elements in Methicillin-Resistant *Staphylococcus aureus* (MRSA) recovered from a healthy student population in Kenya. [Crossref] [Google Scholar]