Am. J. Trop. Med. Hyg., 99(5), 2018, pp. 1239–1245
doi:10.4269/ajtmh.17-0554
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Antibiotic Resistance Patterns and Molecular Characterization of Methicillin-Resistant *Staphylococcus aureus* in Clinical Settings in Rwanda

Florence Masaisa,[1]* Etienne Kayigi,[2] Jeremiah Seni,[3] Freddie Bwanga,[4] and Claude Mambo Muvunyi[1]

[1]*Department of Clinical Biology, School of Medicine and Pharmacy, College of Medicine and Health Sciences, University of Rwanda, Kigali, Rwanda;* [2]*National Reference Laboratory Division, Biomedical Services, Rwanda Biomedical Center, Kigali, Rwanda;* [3]*Department of Microbiology and Immunology, Catholic University of Health and Allied Health Sciences, Mwanza, Tanzania;* [4]*Department of Medical Microbiology, School of Biomedical Sciences, Makerere University College of Health Sciences, Kampala, Uganda*

Abstract. The escalating burden of infections attributable to methicillin-resistant *Staphylococcus aureus* (MRSA) in East African countries is calling for interventional strategies to control the spread of this strain. The present study aimed at determining the prevalence, antimicrobial profiles, and staphylococcal cassette chromosome *mec* (SCC*mec*) typing of MRSA strains. This was a cross-sectional laboratory-based study involving 226 non-duplicated *S. aureus* isolates from different clinical samples of patients attending a referral hospital in Kigali. Kirby–Bauer disk diffusion method was used for drug susceptibility testing. Methicillin-resistant *S. aureus* were confirmed using polymerase chain reaction (PCR) assay for the *mec*A gene and SCC*mec* type PCR assay was used for genotyping. Of 138 *S. aureus*, 39 (31.2%) were found to be MRSA strains. The mean age of the patients was 21.9 years. The incidence of MRSA increases with age and was 27.1% in patient age group younger than 18 years, 33.3% in the age group between 19 and 65 years, and 66.7% in patient age group older than 65 years. There was a significant association between geographic regions and incidence of MRSA (P = 0.02) with the high MRSA isolates from Northern (61.5%) and Western (50%) provinces. Methicillin-resistant *S. aureus* strains were found to be mostly susceptible to linezolid (93.5%). Among the MRSA strains, SCC*mec* type I and SCC*mec* type IV were the most prevalent at 56.4% and 17.9%, respectively. A high prevalence of MRSA was found in Rwanda. Staphylococcal cassette chromosome *mec* type I (52.2%) was the most predominant. A continuous surveillance of MRSA strains, particularly in the hospital settings, should be an enduring exercise in Rwanda.

INTRODUCTION

Staphylococcus aureus remains a major public health problem worldwide as it causes local infections such as impetigo, folliculitis, cellulitis, wound sepsis, and invasive diseases such as bacteremia, necrotizing pneumonia, osteomyelitis, meningitis, endocarditis, toxic shock syndrome, and sepsis.[1]

The evolution of resistance in *S. aureus* has a profound historical background since penicillin was introduced in the market in the early 1940s as the treatment of choice for *S. aureus*.[2] Twenty years later, *S. aureus* isolates account for 80% of penicillin resistance, thus development of methicillin (β-lactamase–resistant penicillins) as an alternative solution to this problem.[2,3] In 1961, 2 years following introduction of methicillin, the first strain of methicillin-resistant *S. aureus* (MRSA) was reported in the United Kingdom and shortly after, the strain spread all over the world and became pandemic in various health facilities.[4,5] Resistance to methicillin is due to the presence of chromosomal *mec*A gene, which encodes for low-affinity penicillin-binding protein 2a responsible for the resistance to methicillin and all other β-lactam antibiotics.[4,6] The *mec*A gene is found on a mobile genetic element known as staphylococcal cassette chromosome *mec* (SCC*mec*).[7] Depending on molecular size, five main types of SCC*mec* (type I–V) have been identified. Types I, II, and III are associated with hospital-acquired MRSA (HA-MRSA), whereas types IV and V are associated with community-acquired MRSA (CA-MRSA).[4] In addition, SCC*mec* harbor genes responsible for β-lactam and non-β-lactam antimicrobial agents.[4] Multiplex PCR which can detect the SCC*mec* types has been shown to be relatively simple and less costly to be used in developing countries compared with other molecular typing methods such as multilocus sequence typing and pulsed-field gel electrophoresis (PFGE).[8–10]

Methicillin-resistant *S. aureus* is a global health threat both in developed and developing countries, and its burden is projected to escalate and confer negative health impact across regions.[1,4,11] A study conducted in eight African countries in 2003 showed an overall prevalence of 15%.[12] Moreover, a significant genotypic diversity has been recently shown by a systematic review across African countries, necessitating the need to have country-specific surveillance for effective MRSA-associated infections prevention and control.[1] In Eastern African Community Region (EAC), the proportion of MRSA among *S. aureus* isolates has been reported at a rate of 15%, 38%, and 84% in Tanzania, Uganda, and Kenya, respectively.[10,13–15] Despite the prevailing information on the magnitude of MRSA and genotypic diversity in EAC and other African countries,[1] limited information exists regarding MRSA in Rwanda. Therefore, the present study aimed at determining the prevalence, antimicrobial resistance patterns, and SCC*mec* genotypic of MRSA strains from clinical specimens among patients attending a University teaching hospital in Kigali, Rwanda, in order to have a baseline information for the management of MRSA-associated infections as well as infection prevention and control.

MATERIAL AND METHODS

Study design, sites, and sampling methods. This was a cross-sectional laboratory-based study conducted from April to May 2014 using 226 archived *S. aureus* isolates. The study was conducted at Center Hospitalier Universitaire de Kigali (CHUK), Rwandan Biomedical Center National Reference Laboratory Division (RBC/BIOS-NRL), and Molecular Biology

* Address correspondence to Florence Masaisa, Department of Clinical Biology, School of Medicine and Pharmacy, College of Medicine and Health Sciences, University of Rwanda, Kigali, Rwanda. E-mail: masaisa2009@gmail.com

Laboratory Makerere University College of Health Sciences (MakCHS).

Center Hospitalier Universtaire de Kigali is a 441-bed referral hospital located in Kigali city; it receives approximately three-fourth of all referral cases in Rwanda, particularly from Kigali city, Northern, Eastern, and some districts of Western province. Collected *S. aureus* were from various clinical specimens obtained from inpatients and outpatients who attended this hospital from June 2013 to April 2014 and were kept at −80°C freezer.

The RBC/BIOS-NRL Division is one of the divisions of Rwanda Biomedical Center and is located at Boulevard de la Revolution in Kigali, Rwanda. The laboratory is responsible of developing policies regulating laboratories in Rwanda, training laboratory personnel, supervising laboratories, and providing external quality control of health facilities in Rwanda.

The Molecular Biology Laboratory is one of the teaching and research laboratories of the Department of Medical Microbiology in MakCHS, located at Mulago Hill in Kampala, Uganda. This laboratory helped in performing the *mec*A gene and SCC*mec* typing molecular assays.

Data collection and laboratory procedures. Achieved non-duplicated *S. aureus* isolates were recorded into the study log book by using codes and were subcultured on blood agar supplemented with 5% sheep blood. Phenotypic reidentification of *S. aureus* was based on catalase, slide, and tube coagulase tests.[16] Following identification, antimicrobial susceptibility testing (AST) was performed by Kirby–Bauer disk diffusion method on Muller–Hinton agar plate according to Clinical and Laboratory Standards Institute recommendations.[17]

Antimicrobial agents used to determine *S. aureus* drug susceptibility patterns included penicillin (10 units), cefoxitin (30 µg), erythromycin (15 µg), clindamycin (2 µg), trimethoprim–sulfamethaxazole (1.35/23.75 µg), tetracycline (30 µg), linezolid (30 µg), rifampin (5 µg), chloramphenicol (30 µg), ciprofloxacin (5 µg), gentamycin (10 µg), and oxacillin (1 µg).[18] All *S. aureus* strains with zone inhibition ≤ 21 mm of diameter to cefoxitin, were considered as MRSA and all strains with zone inhibition ≥ 22 mm were considered as MSSA.[19] Oxacillin disk was also used in phenotypic detection of MRSA as previously described.[16]

Pure colonies of *S. aureus* isolates were inoculated into labeled cryovials of brain heart infusion supplemented with 20% glycerol and were triple-packaged during transportation to the Molecular Biology Laboratory in MakCHS into a cool box containing ice plastic bottles to maintain low temperature required for survival of isolates. A Material Transfer Agreement was signed between the RBC/BIOS-NRL Division and the Molecular Biology Laboratory, MakCHS, before shipment of isolates to Uganda for *mecA* gene and SCC*mec* typing assays.

Molecular assays. DNA was extracted from *S. aureus* isolates using the crude DNA extraction method as previously described.[18]

Forward primer P4: 5′-TCCAATTACAACTTCACCAGG-3′ and reverse primer P7: 5′-CCACTTCATATCTTGTAACG-3′ were used to amplify segment of *mec*A gene by PCR method as described before.[10] The *mec*A gene detection is universally considered as a golden standard in the diagnosis of MRSA as it detects the gene which encodes for methicillin resistance and other β-lactam antibiotics.

Staphylococcal cassette chromosome *mec* gene was amplified by using four forward and four reverse primers shown in the following table to detect the five major types of SCC*mec* (type I–V) using a multiplex PCR method as previously described.[8] Two multiplex PCR were performed; the first multiplex amplified CCrC (CCrC F and R) and mecA-*IS*431 (5R*mecA* and 5R431), whereas the second multiplex amplified ccrA2-B (beta and alpha-3) and *IS*1272 (1272F1 and 1272R1).[10] In case no band was observed, the respective isolate was regarded as "non-typeable."

Name of primer	Sequence of primer (5′ 3′)	Band size in bp	Target gene
β and α3	ATTGCCTTGATAATAGC CYTCTTAAAGGCATC AATGCACAAACACT	937	ccrA2-B
ccrCF and ccrCR	CGTCTATTACAAGATGT TAAGGATAAT CCTTTATAGACTGGATT ATTCAAAATAT	518	ccrC
1272F1 and 1272R1	GCCACTCATAACATA TGGAA CATCCGAGTGAAACC CAAA	415	IS1272
5R*mecA* and 5R431	TATACCAAACCCGACA ACTAC CGGCTACAGTGATAA CATCC	359	*mecA*-IS431

The amplicons were analyzed by electrophoresis using a 2% TAE agarose gel in 1× TAE (Tris-acetate-EDTA) buffer run at constant voltage of 120 for 1 hour. The images were visualized and captured using the Bio-imager (UVP, LLC, Upland, CA).

Quality control. *Staphylococcus aureus* ATCC 25923 and *Staphylococcus epidermidis* ATCC 12944 were used as positive control and negative control for identification tests. For AST, *S. aureus* ATCC 25923 was used to quality control antibiotic disks.[17] *Staphylococcus aureus* ATCC 25923 and *S. aureus* ATCC 43300 were used as negative control and positive control for *mec*A gene detection, respectively.[17]

Data analysis. Sociodemographic and clinical information of included isolates were retrieved from laboratory registers and hospital information management system and transferred to the Microsoft Excel for consistency check. The Statistical Package for the Social Sciences software word version 20.0 was used to analyze the data.

Study permission and ethical considerations. Ethical permission was obtained from Rwanda National Ethics Committee and Rwanda Ministry of Education, Science, Technology and Research.

RESULTS

Description of study population and incidence of MRSA in subgroups. The study involved 138 confirmed *S. aureus* isolates from a total of presumed 226 non-duplicated *S. aureus* isolates; other isolates were excluded for various reasons (Figure 1). Demographic characteristics of the study participants were available in 125 individuals (90.6%) and are described in the Table 1. Of total isolates, 39 isolates (31.2%) were found positive for MRSA (Table 1). The study population comprised 70 (56%) males, with 31.4% MRSA prevalence, and 55 (46%) females, with 30.8% MRSA prevalence. The mean age of the patients was 21.9 years (standard deviation, 18.9 years), with a range of 8 months to 73 years. The incidence of MRSA increases with age and was 27.1% in patient

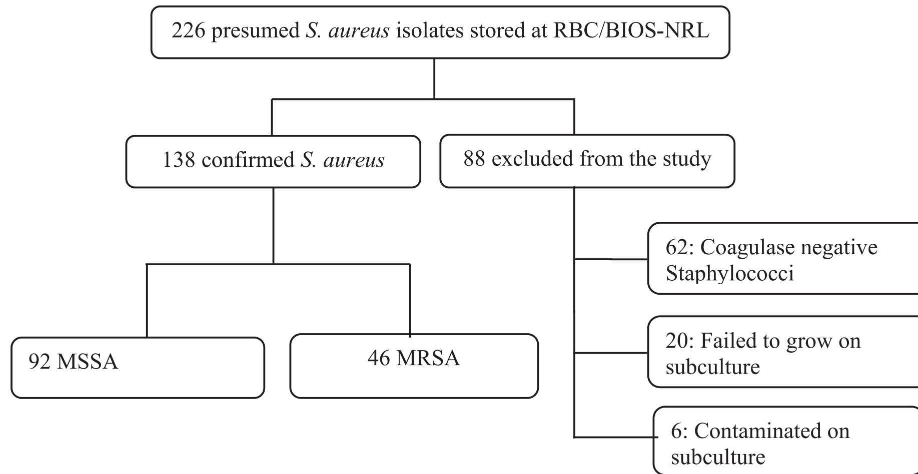

FIGURE 1. Enrollment of isolates. RBC/BIOS-NRL = Rwanda Biomedical Center National Reference Laboratory; MRSA = methicillin-resistant *S. aureus*; MSSA = methicillin-sensitive *Staphylococcus aureus*.

age group less than 18 years, 33.3% in the age group between 19 and 65 years, and 66.7% in patient age group older than 65 years. However, no significant difference was seen between MRSA and age groups (P = 0.309). The majority of isolates (80.8%) were retrieved from pus swabs followed by blood sample (11, 2%). There was a significant association between province and incidence of MRSA (P = 0.02) with the high MRSA isolates from Northern (61.5%) and Western (50%) provinces.

TABLE 1
Distribution of MRSA in different groups

	Total (%) (*N* = 125)	*N* (%) MRSA (*N* = 39)	*P* value
Sex			
Male	70 (56.0)	22 (31.4)	0.950
Female	55 (44.0)	17 (30.9)	–
Age, years, mean = 21.9 ± (18.9)			
≤ 18	59 (47.2)	16 (27.1)	0.308
19–64	63 (50.4)	21 (33.3)	–
≥ 65	3 (22.4)	2 (66.7)	–
Specimen			
Pus	101 (80.8)	28 (27.7)	0.208
Blood culture	14 (11.2)	7 (50)	–
Nasal swab	2 (1.6)	0 (0)	–
Skin swab	2 (1.6)	1 (50)	–
*Others	6 (4.8)	3 (50)	–
Ward			
Surgical	47 (37.6)	14 (29.8)	0.723
Pediatrics	28 (22.4)	7 (25)	–
Internal medicine	15 (12.0)	6 (40)	–
Gynecology Obstetrics	9 (7.2)	4 (44)	–
Outpatient department	4 (3.2)	2 (50)	–
Theater	5 (4.0)	2 (40)	–
Others†	17 (13.6)	4 (23.5)	–
Province			
Kigali city	53 (42.4)	11 (20.8)	0.020
Eastern	26 (20.8)	9 (34.6)	–
Northern	13 (10.4)	8 (61.5)	–
Western	14 (11.2)	7 (50)	–
Southern	19 (42.4)	4 (21.1)	–

MSSA = methicillin-sensitive *Staphylococcus aureus*.
* Other specimens included alveolar punction, ascite punction, eye pus, skin pus, synovial punction, and urine.
† Other wards included emergency ear, nose, and throat; neonatology; dermatology; and ophthalmology.

Susceptibility of *S. aureus* to different antibiotics. The majority of the *S. aureus* isolates were resistant to penicillin (96.4%), trimethoprim–sulfamethaxazole (34.1%), and tetracycline (34.1%). Most of the *S. aureus* isolates were susceptible to chloramphenicol (83.3%), ciprofloxacin (85.5%), gentamicin (87%), and linezolid (94.9%) (Table 2).

Prevalence of MRSA among *S. aureus* isolates. Based on the presence of *mecA* gene which is a gold standard, the proportion of MRSA among *S. aureus* isolates was 31.2% (39/138), whereas the proportion of MRSA by both phenotypic tests (cefoxitin and oxacillin disks) was 27.2% (34/138). Thus, the gold standard test detected three more MRSA isolates compared with phenotypic methods (Figures 2 and 3).

Resistance of MRSA to other antibiotics. Methicillin-resistant *S. aureus* strains showed a high resistance to trimethoprim–sulfamethaxazole 47.8%, erythromycin 41.3%, and tetracycline 39.1%, but low resistance to linezolid (6.5%) (Table 3).

Genetic diversity of SCC*mec* in MRSA isolates. A multiplex PCR targeting four different MRSA genes was performed to differentiate the strains into HA-MRSA and CA-MRSA using SCC*mec* typing (Figure 4). Staphylococcal cassette chromosome *mec* type I was found to be the most prevalent with 52.2% (24/46, followed by SCC*mec* type IV 15.4% (7/46) and SCC*mec* type II was not found among MRSA strains in

TABLE 2
Staphylococcus aureus antimicrobial susceptibility pattern (*N* = 138)

Antimicrobials	Susceptible *N* (%)	Intermediate *N* (%)	Resistant *N* (%)
Penicillin	5 (3.6)	NA	133 (96.4)
Oxacillin	89 (64.5)	13 (9.4)	36 (26.1)
Erythromycin	98 (71.0)	15 (10.9)	25 (18.1)
Clindamycin	107 (77.5)	13 (9.4)	18 (13.0)
Trimethoprim–sulfamethaxazole	86 (62.3)	5 (3.6)	47 (34.1)
Tetracycline	87 (63.0)	4 (2.9)	47 (34.1)
Chloramphenicol	115 (83.3)	6 (4.3)	17 (12.3)
Ciprofloxacin	118 (85.5)	8 (5.8)	12 (8.7)
Gentamicin	120 (87.0)	NA	18 (13.0)
Linezolid	131 (94.9)	NA	7 (5.1)

MRSA = methicillin-resistant *Staphylococcus aureus*; NA = not applicable.

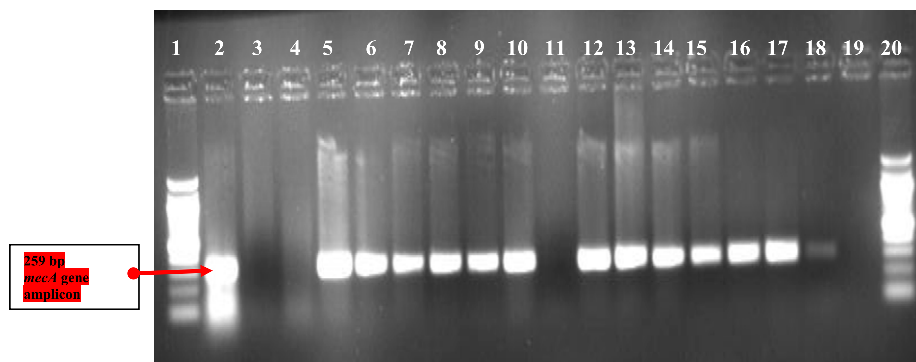

FIGURE 2. *mecA* PCR agarose gel electrophoresis. Lanes 1 and 20 are 100-bp DNA ladder. Lanes 2 and 19 show the positive and negative controls. Lanes 3, 4, and 11 show *mecA* negative, whereas the rest show *mecA*-positive *Staphylococcus aureus* isolates. This figure appears in color at www.ajtmh.org.

Rwanda. Non-typeable MRSA strains also have a high proportion of 26.6% (12/46).

Distribution of SCC*mec* types among MRSA and in different groups is shown in Table 4. The majority of the MRSA strains belonged to SCC*mec* type I (22 stains, 56.4%). Staphylococcal cassette chromosome *mec* type IV a SCC*mec* type III were found in seven strains (17.9%) and in one strain (2.6%), respectively. Staphylococcal cassette chromosome *mec* type V was not confirmed in any of the MRSA strains, whereas nine strains (23.1%) could not carry any of the previously described SCC*mec* types. Social demographic data showed a disproportional distribution of SCC*mec* type in the different groups. The occurrence of SCC*mec* type I and type IV was similar in the age groups of less than 18 years (10 strains, 58.8% and three strains, 17.6%, respectively) and 19–64 years (11 strains, 52.4% and four strains, 19%, respectively). Females had high proportion of SCC*mec* type I 63.6% (14/22) compared with males 47.1 (8/17). Staphylococcal cassette chromosome *mec* type I was more predominantly carried out in MRSA strains from surgical department (81.3% [13/16]) and pus specimens (60.7 [7/28]), and Eastern Province (70% [7/10]).

DISCUSSION

Staphylococcus aureus causes local and invasive infections of public health importance both in the community and hospital settings.[19–21] For the past six decades, this species has progressively developed resistance to β-lactams and other antibiotics posing treatment challenges, which in turn necessitates a need for continuous surveillance strategies to curb its spread both in the community and hospital settings.[1,2,4] To our knowledge, this is the first study from Rwanda that provides the local epidemiological data and genetic diversity of MRSA. Although, not statistically significant, MRSA prevalence seems to increases with age, which is consistent with previous report showing an association between age and both the rate of MRSA.[12] Kigali Province contributed to almost half of isolates (42.4%), most likely as it is the most populated province and the majority of patient admitted at CHUK are from the province. Northern Province had the highest prevalence of MRSA (61.5%). There are no clear reasons for this geographic variation of MRSA in this study; however, this might be explained by geographic variation in the prevalence of organisms in the hospitals across the country.

Although penicillin is among the cheapest antibiotics and therefore the most affordable for developing countries with limited resources, this study proved it to be ineffective in Rwanda health facilities as almost 100% of *S. aureus* isolates were resistant to the drug; these findings are similar to another study conducted in Uganda.[10] The finding about high rates of penicillin resistance is not surprising and is also in line with several studies carried out in Africa that also reported a greater than 90% resistance to penicillin.[1] Interestingly, *S aureus* isolates in the present study were susceptible to other non-β lactam antibiotics such as gentamicin, ciprofloxacin, and chloramphenicol which can be potential treatment options in Rwanda. The susceptibility to gentamicin (87.5%) is similar to another study in Uganda,[22] but in contrary to other studies which showed low susceptibility to gentamicin and ciprofloxacin in approximately 15.9% and 25.6% respectively.[13,23] The present study found a high prevalence of MRSA (33.3%) which may present the management challenges of infections associated with this strain because the readily available and cheap β-lactam drugs cannot be used, whereas the cost of the second-line drugs such as vancomycin is prohibitive. The MRSA prevalence in Rwanda is similar to the proportions ranging from 25% to 37.5% found in three studies conducted in Uganda[10,22,24] but higher than 10% reported from Tunisia,

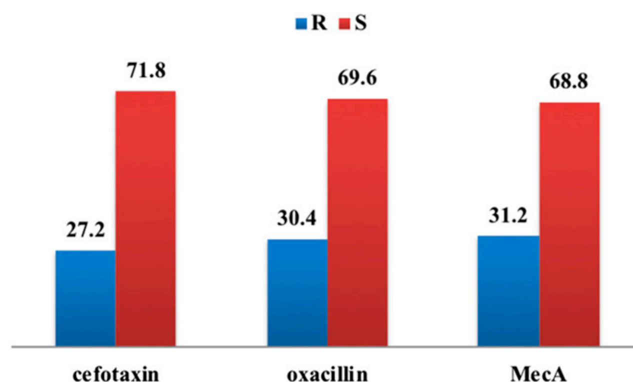

FIGURE 3. Prevalence of methicillin-resistant *Staphylococcus aureus* based on different methods. This figure appears in color at www.ajtmh.org.

Table 3
Resistance of methicillin-resistant *Staphylococcus aureus* (N = 46) to antibiotics

Antimicrobials	Susceptible N (%)	Intermediate N (%)	Resistant N (%)
Trimethoprim–sulfamethaxazole	24 (52.2)	0 (0.0)	22 (47.8)
Erythromycin	20 (43.5)	7 (15.2)	19 (41.3)
Tetracycline	25 (54.3)	3 (6.5)	18 (39.1)
Gentamicin	29 (63.0)	0 (0.0)	17 (37.0)
Clindamycin	24 (52.2)	9 (19.6)	13 (28.3)
Chloramphenicol	31 (67.4)	2 (4.3)	13 (28.3)
Ciprofloxacin	31 (67.4)	4 (8.7)	11 (23.9)
Rifampin	36 (78.3)	2 (4.3)	8 (17.4)
Linezolid	43 (93.5)	0 (0.0)	3 (6.5)

Malta, and Algeria.[15] Moreover, the prevalence of MRSA in this study is low compared with 46%, 59.8%, and 84% found in northern India, China, and Kenya, respectively.[13,23,25] There is also variation in the rate of MRSA isolates resistant to vancomycin, refered as vancomycin-intermediate *S. aureus* (VISA), which is the last drug of choice. Although, this study could provide data VISA, the moderate rate of MRSA may suggest similar observation with reports of other studies in Tanzania, Kenya, and Uganda where no resistance to vancomycin was reported.[10,13,15] The difference on the resistance rates may be due to different antimicrobial use policies, infection control practices, and different studied populations in these countries, but the finding about VRSA in Rwanda calls for further analysis with reliable antibiotic susceptibility method to ascertain its occurrence, source, and promptly control and prevent its spread.

Previous evidences suggest that SCC*mec* types I, II, and III tend to be more related to HA-MRSA, whereas SCC*mec* types IV and V are associated with CA-MRSA. This study found that SCC*mec* type I (56.4%) was the most predominant among MRSA strains, whereas SCC*mec* type II and V were not found among *mecA* gene-positive isolates in Rwanda. The majority of these isolates were collected from surgical wards, which accounted for 81.3% of the SCC*mec* type I followed by type IV (18.6%). Similar to other studies in Uganda, the predominance of SCC*mec* type I in surgical ward may indicate the possibility of transmission of these strains in the hospital setting[10,26] and thus a need to emphasize on strengthening of infection control practices in Rwanda to reduce MRSA nosocomial infections. As opposed to SCC*mec* type I, the predominance of SCC*mec* type III (57.6%) and SCC*mec* type II (22.0%) was found in another study conducted in China.[25] The low proportion of CA-MRSA may pin point proper usage of antibiotics in community settings in Rwanda.

However, 26% of MRSA isolates could not be assigned to any SCC*mec* types, and this may probably reflect the low discriminatory power of the method and thus calling for the need to use other cost-effective methods with high discriminatory power such as *spa* sequence typing.[10]

Limitations. This study did not use the gold standard for molecular typing of strains which is PFGE; despite this, a baseline SCC*mec* typing to delineate nosocomial versus community-associated MRSA strains circulating in Rwanda has been conducted.

CONCLUSION

For the first time, high prevalence of MRSA (31.2%) in Rwanda has been documented and is associated with certain age groups and geographic regions, which has an important implication for developing prevention programs.

Staphylococcus aureus isolates from Rwanda are more susceptible to gentamicin, ciprofloxacin, chloramphenicol, and linezolid, whereas MRSA are mostly susceptible to linezolid. Staphylococcal cassette chromosome *mec* type I (52.2%) was found to be the most predominant MRSA genotype and mostly isolated from surgical pus specimens suggesting nosocomial MRSA transmission. A continuous surveillance of MRSA strains, particularly in the hospital settings, should be an enduring exercise in Rwanda so as to curb the transmission of MRSA strains in this setting.

Received July 12, 2017. Accepted for publication April 30, 2018.

Published online September 10, 2018.

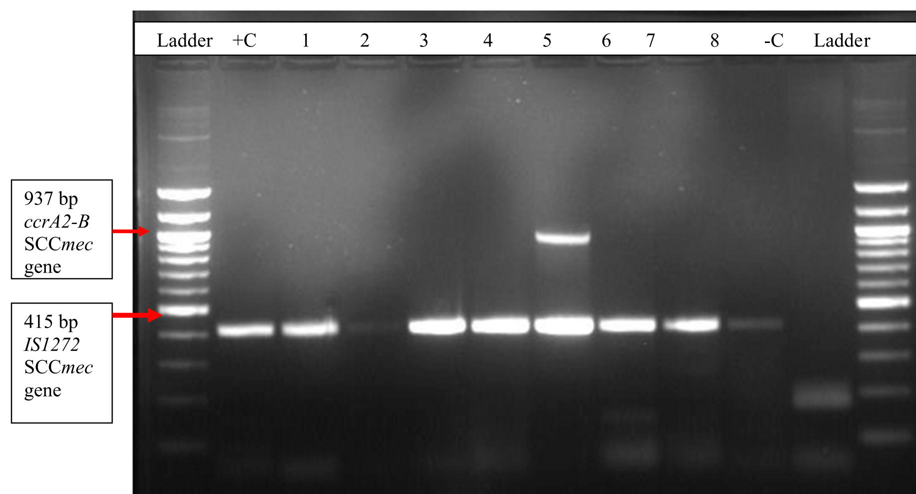

Figure 4. *IS1272* and *ccrA2-B* genes on PCR agarose gel electrophoresis; 100-bp DNA ladder. Lanes +C and –C show the positive and negative controls. Lanes 1, 2, 3, 4, 6, 7, and 8 show *IS1272* positive staphylococcal cassette chromosome *mec* (SCC*mec* type I), whereas lane 5 shows *IS127 and ccrA2-B*–positive *MRSA* (SCC*mec* type IV). This figure appears in color at www.ajtmh.org.

Table 4
Prevalence of SCC*mec* type in different groups ($N = 39$)

Subgroup (*n*)		SCC*mec* types			
		Non-typeable 9 (23.1)	Type I 22 (56.4)	Type III 1 (2.6)	Type IV 7 (17.9)
Age group	≤ 18 (17)	3 (17.6)	10 (58.8)	1 (6)	3 (17.6)
	19–64 (21)	6 (28.6)	11 (52.4)	0 (0.0)	4 (19)
	≥ 65 (1)	0 (0.0)	1 (100)	0 (0.0)	0 (0.0)
Gender	Female (22)	5 (27.7)	14 (63.6)	1 (4.6)	2 (9.1)
	Male (17)	4 (23.5)	8 (47.1)	0 (0.0)	5 (29.4)
Specimen	Pus (28)	5 (17.9)	17 (60.7)	0 (0.0)	6 (21.4)
	Blood culture (7)	2 (28.6)	3 (42.8)	1 (14.3)	1 (14.3)
	Others* (4)	2 (50)	2 (50)	0 (0.0)	0 (0.0)
Ward	Surgical (16)	1 (6.3)	13 (81.3)	0 (0.0)	3 (18.6)
	Pediatrics (7)	2 (28.6)	2 (28.6)	1 (14.2)	2 (28.6)
	Internal medicine (5)	1 (20)	3 (60)	0 (0.0)	1 (20)
	Gynecology obstetrics (4)	2 (50)	1 (25)	0 (0.0)	1 (25)
	Outpatient department (2)	1 (50)	1 (50)	0 (0.0)	0 (0.0)
	Others† (4)	2 (50)	2 (50)	0 (0.0)	0 (0.0)
Province	Kigali city (10)	4 (40)	5 (50)	0 (0.0)	1 (10)
	Eastern (10)	1 (10)	7 (70)	1 (10)	1 (10)
	Northern (9)	2 (22.2)	4 (44.4)	0 (0.0)	3 (33.4)
	Western (6)	1 (16.7)	4 (66.6)	0 (0.0)	1 (16.7)
	Southern (4)	1 (25)	2 (50)	0 (0.0)	1 (25)

SCC*mec* = staphylococcal cassette chromosome *mec*.
* Other specimens included alveolar punction, ascite punction, eye pus, skin pus, synovial punction, and urine.
† Other wards included emergency ear, nose, and throat; neonatology; dermatology; and ophthalmology.

Acknowledgments: We thank Thadee Bwanakweri (RBC/BIOS-NRL Bacteriology Section); Edgar Kigozi and Fred Ashaba Gatabazi (Molecular Biology/Department of Medical Microbiology, Makerere University); and other staff members in Rwanda and Uganda for their excellent technical assistance and support.

Financial support: This study was supported by the Swedish International Developments Cooperation Agency (*Sida*) funds in cooperation with the University of Rwanda, Coordination Office of Research Activities

Authors' addresses: Florence Masaisa, Department of Clinical Biology, College of Medicine and Health Sciences, University of Rwanda, CHUK-Kigali, Kigali, Rwanda, E-mail: masaisa2009@gmail.com. Etienne Kayigi, Biomedical Services, Rwanda Biomedical Center, Kigali, Rwanda, E-mail: kayigi2003@yahoo.fr. Jeremiah Seni, Department of Medical Microbiology, Makerere University College of Health Sciences, Kampala, Uganda, E-mail: jeremiahseni@gmail.com. Freddie Bwanga, Department of Microbiology and Immunology, Catholic University of Health and Allied Health Sciences, Mwanza, Tanzania, E-mail: freddie.bwanga@case.edu. Claude Mambo Muvunyi, Department of Clinical Biology, School of Medicine and Pharmacy, College of Medicine and health Sciences, University of Rwanda, Kigali, Rwanda, E-mail: clmuvunyi@gmail.com.

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