

Prevalence, molecular characteristics and antimicrobial susceptibility patterns of MRSA in hospitalized and nonhospitalized patients in Barbados

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Abstract

The epidemiology and clonality of methicillin-resistant *Staphylococcus aureus* (MRSA) has not been investigated, as not much research or surveillance has been undertaken to identify and characterize the circulating MRSA strains in Barbados. Prevalence rates, molecular characteristics and antimicrobial susceptibility pattern of MRSA infections in hospitalized and nonhospitalized patients were investigated. A total of 293 isolates were included in the study, with 100 from the hospital and 193 from the public health laboratory. Isolates were collected over a period of 1 (2015–2016) and 3 years (2013–2016) respectively. MRSA was identified using standard microbiologic techniques and was further analysed by multiplex PCR for the presence of the *spa*, *mec* gene complex typing and *PVL* genes (*lukS-PV* and *lukF-PV*). A prevalence rate of 19.7% was calculated for those hospitalized. All hospital isolates were sensitive to vancomycin, rifampin, linezolid and cotrimoxazole (trimethoprim/sulfamethoxazole), whilst 82% were sensitive to clindamycin. The *PVL* gene was detected in 76% of hospital isolates. In the community isolates, resistance was observed in erythromycin (100%), ciprofloxacin (97.4%), clindamycin (13%) and cotrimoxazole (5.7%). There was no resistance to vancomycin. The *PVL* gene was detected in 97.9% of the isolates, the *mecA* gene in only 2.1% and the *mecC* gene in 0%. Most MRSA isolates were community acquired in both settings, and the antimicrobial susceptibility profile was similar, suggesting transmission of community-associated MRSA into the hospital environment. Further harmonization of antimicrobial policy for the treatment of MRSA (and by extension other pathogens) should be implemented to quell ongoing transmission. We found that 93.4% of MRSA in Barbados treated in the primary healthcare system were sensitive to

cotrimoxazole. By typing MRSA isolates and drawing inferences on transmission on the basis of genetic relatedness, transmission pathways may be tracked. Further studies must be performed for this high level of comprehensiveness so that with the surveillance of MRSA, effective strategies may be developed to prevent or limit transmission.

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Introduction

Methicillin, a semisynthetic penicillin that is poorly hydrolyzed by penicillinase, was first used clinically in 1960. Only 1 year later, *Staphylococcus aureus* strains that showed resistance to methicillin were reported [1]. Resistance to methicillin confers resistance to all penicillinase-resistant penicillins and cephalosporins. Since then, methicillin-resistant *Staphylococcus aureus* (MRSA) strains have appeared in countries worldwide, and these strains continue to be one of the most common hospital pathogens [2]. It has been shown that methicillin-susceptible *S. aureus* (MSSA) strains become MRSA strains through the acquisition of a staphylococcal cassette chromosome *mec* (SCC*mec*) element carrying a *mecA* gene, which is responsible for methicillin resistance [3,4].

Barbados, a populous island country of 287 212 persons in the Eastern Caribbean, is 34 km (21 miles) long and 23 km (14 miles) wide, covering an area of 432 km² (167 square miles) [5]. Historically, the economy of Barbados has been dependent on sugarcane cultivation and related activities but has diversified into the manufacturing and tourism since the late 1970s and early 1980s. The tourism industry generates more than 50% of the country's foreign exchange, where approximately 14 000

people are directly employed, and contributes 12% of GDP [6]. With globalization and the increasing rates of travel, dissemination of infectious agents is an important determinant in the healthcare—and the health of the public. In many parts of the world, epidemiologically and genotypically defined community-associated (CA) MRSA strains have emerged to become frequent causes of hospital infection. Until the emergence of CA-MRSA in the late 1990s [7,8], infection was predominantly due to healthcare-associated (HA) strains associated with advanced age, comorbidities, surgical procedures or indwelling medical devices [9–11]. CA-MRSA later emerged as a cause of infection in the community in previously healthy individuals of all ages, with no history of hospital contact and none of the risk profiles that are typical of healthcare exposure [12,13]. Recently, however, CA-MRSA strains have emerged as a cause of HA infection in some parts of the world [13], challenging definitions of CA-MRSA based on clinical epidemiology and where disease manifests [14–16] in favour of genotype-based definitions [17–19]. Nonetheless, CA-MRSA strains retain a number of important characteristics, notably the association with infection in previously healthy individuals in the community [7,8,12,16,20]. Outbreaks in hospitals and nursing homes are today caused by both HA-MRSA and CA-MRSA clones [21–23], and it has been suggested that it is no longer useful to regard HA-MRSA and CA-MRSA as separate entities [24].

Healthcare settings are regarded as the epicentre for MRSA transmission in many countries worldwide. Hospital admission of unknown MRSA carriers, lack of MRSA admittance screening and spread of MRSA among nursing home residents could all be part of the explanation of this mélange of HA-MRSA and CA-MRSA in hospitals [25]. This would increase our knowledge of the prevalence and management of such infections in light of increasing antibiotic resistance and the overuse of more potent antibiotics in the community clinics that could compromise future treatment regimens by creating antibiotic resistance isolates.

The aims of this study were to determine the antimicrobial susceptibility patterns of MRSA in the in Barbados healthcare system (polyclinics, hospital and satellite clinics); and to characterize by molecular techniques the strains of MRSA in hospitalized and nonhospitalized patients.

Patients and methods

Hospitalized patients

Isolates of MRSA were collected at the Department of Microbiology, The Queen Elizabeth Hospital Barbados, a 600-bed facility, from December 2014 to December 2015. One hundred isolates were collected from the following specimens: blood, bone, ear, fluids, surgical drains, tissue, urine and

wounds. Samples were submitted to the microbiology department from wards in the hospital, including the intensive care units. They were categorized as HA or CA. HA was classified as a patient whose MRSA isolate was cultured more than 48 hours after admission, while CA was classified as a patient whose MRSA isolate was cultured within 48 hours after admission. This information was provided by the infection control department.

Community-based patients (nonhospitalized)

Samples were collected as convenience samples from routine testing that were suspected to be *Staphylococcus aureus* species from the period July 2013 to January 2016. These samples were received at the laboratory from the healthcare institutions on the island. These include the eight polyclinics, their satellite clinics and three other healthcare institutions (termed 'other' in this study).

Processing

Samples were received in the microbiology department and cultured according to standard operating procedures for each sample type. Cultures were incubated at 35°C for 18 to 24 hours. Culture plates were examined the next day, and isolates of *Staphylococcus* species were identified and subcultured on 5% sheep's blood agar and MacConkey agar, and incubated as described above. Gram staining as well as catalase and coagulase tests were performed for confirmation of the organism.

All hospital isolates were confirmed to be *Staphylococcus aureus*. These were speciated using the MicroScan Dried Gram Positive MIC/Combo panel with Cefoxitin Screen well (CfxS) using the MicroScan WalkAway system, which is a fully automated identification and susceptibility system. Amoxicillin/clavulanate acid, ampicillin, ceftriaxone, ciprofloxacin, clindamycin, erythromycin, gentamicin, levofloxacin, linezolid, moxifloxacin, nitrofurantoin, oxacillin, penicillin, rifampin, tetracycline, cotrimoxazole (trimethoprim/sulfamethoxazole) and vancomycin are included in the panel.

Antimicrobial susceptibilities of community isolates were determined by the Kirby-Bauer disc diffusion method on Müller-Hinton agar. The following antibiotic discs were applied to the plate: erythromycin (E), penicillin (P), oxacillin (OX), cephalothin (CF), cefuroxime (CXM), ceftazidime (CAZ), ciprofloxacin (CIP), cotrimoxazole (SXT), clindamycin (CC), cefoxitin (FOX), amoxicillin/clavulanic acid (AMC) and vancomycin (VAN). Susceptibilities determined by Clinical and Laboratory Standards Institute (CLSI) guidelines. Data were analysed by WHONET 5.6 (<http://www.whonet.org/software.html>).

All MRSA isolates were stored in brain–heart infusion broth with 20% glycerol at –20°C before extraction of DNA.

DNA extraction

Cultures that were stored in brain–heart infusion broth were inoculated on 5% sheep's blood agar and incubated overnight. Two to three colonies were picked and suspended in 100 µL of lysis buffer (Instagene Matrix; Bio-Rad, Hercules, CA, USA) in a 1.5 mL Eppendorf tube. The suspension was vortexed and incubated at 56°C for 1 hour. The suspension was further incubated at 95°C for 1 hour. After the second incubation, the suspension was vortexed and centrifuged at 13 200 rpm for 5 minutes. DNA extractions were stored at –20°C for PCR [26].

Polymerase chain reaction

A multiplex PCR method developed by the European Union Reference Laboratory–Antimicrobial Resistance was used for confirmation of methicillin resistance by amplification of both *mecA* and *mecC*, identification of *S. aureus* by amplification of the *spa* gene (also used for typing) and detection of the Panton-Valentine leukocidin (*PVL* or *LukF-PV*)-encoding gene [26]. A PCR run consisted of 15 patient samples and three controls; *Staphylococcus aureus* subsp. *aureus* Rosenbach ATCC 25923D-5TM, *Staphylococcus aureus* subsp. *aureus* Rosenbach ATCC 6538D-5TM and ATCC 1556D-5. A negative control was also included in each run, which consisted of the master mix and deionized water. After the preparation of the master mix, 2 µL of sample DNA was added to 23 µL of the master mix to give a PCR volume of 25 µL; this was amplified according to the above-mentioned protocol. Briefly, each PCR contained 0.4 µM of the primers as follows: *mecA* primers (*mecA* P4, 5'-TCCA-GATTACAACCTCACCAGG-3'; *mecA* P7, 5'-CCACTTCA-TATCTTGTAACG-3'), *spa* primers (*spa*-1113F, 5'-TAAAGACGATCCTTCGGTGAGC-3'; *spa*-1514R, 5'-CAG-CAGTAGTGCCGTTTGCTT-3'), 1 µM *PVL* primers (*PVL*-F, 5'-GCTGGACAAAATTCTTGGGAATAT-3'; *PVL*-R, 5'-GATAG-GACACCAATAAATTCTGGATTG-3') and *mecA*_{LGA251} primers (*mecA*_{LGA251} Multi FP, 5'-GAAAAAAGGCTTA-GAACGCCTC-3'; *mecA*_{LGA251} Multi RP, 5'-GAAGATC TTTCCGTTTTTCAGC-3') [26,27]. Amplification was performed in a PX 0.2 thermal cycler manufactured by Thermo Electric (West Chester, PA, USA) with 5 minutes at 94°C hot start, followed by 30 cycles of 30 seconds at 94°C, 1 minute at 59°C and 1 minute at 72°C, with a final 10 minutes at 72°C. Amplicons were electrophoresed in a 2% agarose gel and visualized under ultraviolet light [26,27].

This study was approved by the institutional review board of the University of the West Indies (Cave Hill) and by the Ministry of Health and the ethics committee of Queen Elizabeth Hospital. No patient information was disclosed.

Results

HA isolates

Over the 1-year period from December 2014 to December 2015, a total of 4696 hospital isolates were identified, of which 688 (19.7%) were MRSA positive, occurring in 44% in male and 56% female subjects ranging in age from 1 to 87 years. Patients were admitted from the accident and emergency department to the following units: surgical 35%, paediatric 15%, internal medicine 10%, orthopaedics 10% and urology 8%. Other patients were admitted to other wards on the basis of the availability of space. When analysed by site of infection, 25% were isolated from blood, 2% urine, 4% surgical drains, 30% tissue, 24% wounds, 9% bone, 4% fluid and 2% ear.

One hundred isolates of MRSA were identified over a period of 1 year. Ninety of these isolates were CA-MRSA, and the other ten were HA-MRSA. The 90 CA-MRSA isolates were made up of tissue ($n = 30$), wound swabs ($n = 24$) and blood ($n = 17$). Of the ten HA-MRSA, eight were blood and two surgical drains (Table 1).

Overall, 100% of isolates were resistant to oxacillin (MIC >2 µg/mL), with a positive cefoxitin screen well of MIC >4 µg/mL. Antimicrobial susceptibility testing revealed that all isolates were resistant to ceftriaxone and ciprofloxacin, and 90% of isolates were resistant to erythromycin. All isolates were sensitive to vancomycin, rifampin, gentamicin, linezolid and cotrimoxazole; 82% were sensitive to clindamycin, with 2% inducible clindamycin resistance.

Multiplex PCR analysis confirmed the presence of the *spa*, *mecA* gene in 77 of 100 MRSA isolates. The *PVL* gene was negative in one of 77 isolates. A further 15 isolates showed only *spa* and *PVL* genes, with the remaining eight isolates showing no *spa*, *mecA/C* and *PVL* gene but that were identified as MRSA by the MIC values (Table 2).

CA isolates

A total of 193 samples were collected from 12 clinical sites over the 3-year period 2013 to 2016 (Fig. 1). There were 117 female (60.6%) and 76 male (39.4%) subjects included in the study. Most of the isolates were from wound swabs (71%), followed by nasal swabs (8.8%), with penile (0.5%) and vaginal (0.5%) swabs being the least common specimen types submitted for testing (Table 3).

Of the 193 samples tested, nine, or 4.7%, gave D-zones for clindamycin induction. A total of 94.3% susceptibility was recorded to cotrimoxazole, in 92.3% female and 96.1% male subjects. A susceptibility to vancomycin of 97.4% was observed;

TABLE 1. HA-MRSA and CA-MRSA of hospital isolates

Site of specimen	Total no. of isolates	CA-MRSA isolates, n (%)	HA-MRSA isolates, n (%)
Blood	25	17 (18.9)	8 (80)
Bone	9	9 (10)	0
Ear	2	2 (2.2)	0
Fluid	4	4 (4.4)	0
Surgical drain	4	2 (2.2)	2 (20)
Tissue	30	30 (33.3)	0
Urine	2	2 (2.2)	0
Wound swab	24	24 (26.7)	0
Total	100	90	10

CA, community acquired; HA, hospital acquired; MRSA, methicillin-resistant *Staphylococcus aureus*.

TABLE 2. Demographic data and presence of PVL gene of hospital-acquired MRSA

No.	PVL gene present	Ward, sex	Site	Age (years)	Sex
1	No	Medical, M	Surgical drain	67	M
2	Yes	Medical, M	Blood	57	M
3	Yes	Medical, F	Surgical drain	29	F
4	Yes	Medical, M	Blood	58	M
5	Yes	Medical, F	Blood	60	F
6	Yes	Surgical, mixed	Blood	59	F
7	Yes	Medical, M	Blood	57	M
8	Yes	Medical, M	Blood	71	M
9	Yes	Surgical, mixed	Blood	60	F
10	Yes	Surgical, mixed	Blood	69	F

clindamycin susceptibility averaged 89.6%. All isolates were resistant to the β -lactam antibiotics and macrolides. When analysed by age, of those aged ≤ 18 years (16.1%), 94.5% of the isolates were susceptible to cotrimoxazole, vancomycin (100%), clindamycin (93.5%) and ciprofloxacin (0). Of the study

population aged 19 to 54 years (35.2%), 97.1% of the isolates were susceptible to cotrimoxazole, 100% to vancomycin, 86.8% to clindamycin and 1.5% to ciprofloxacin. Of the study population aged ≥ 55 years (39.3%), 92.1% of isolates were susceptible to cotrimoxazole, 100% to vancomycin, 80.3% to clindamycin and 1.3% to ciprofloxacin (Table 4).

The *PVL* gene was amplified in 185 (95.9%) of the samples tested, along with the *spa* and *mecA* genes. The other eight isolates had amplification of the *spa* and *mecA* genes only and were obtained from clinic 1 ($n = 1$), clinic 2 ($n = 1$), clinic 3 ($n = 2$), clinic 4 ($n = 2$) and clinic 5 ($n = 2$) (Fig. 2).

For the eight (4.2%) of 193 isolates where only the *spa* and *mecA* genes were amplified, four of these isolates were resistant to vancomycin, whilst one isolate was resistant to cotrimoxazole, ciprofloxacin and vancomycin. Six isolates were resistant to clindamycin. There was a missing age demographic for the single patient with an isolate with multiple resistances (Table 5).

Discussion

To our knowledge, our study is the first in Barbados to determine the prevalence and molecular characteristics of MRSA isolates from both hospitalized and nonhospitalized patients. To generate definitive ideas regarding the types and presence of MRSA circulating within the country, consecutive isolates submitted to the both the public health laboratory from 2012 to 2016 and the hospital laboratory from 2015 to 2016 (prospective) were analysed, with a total of 293 isolates analysed.

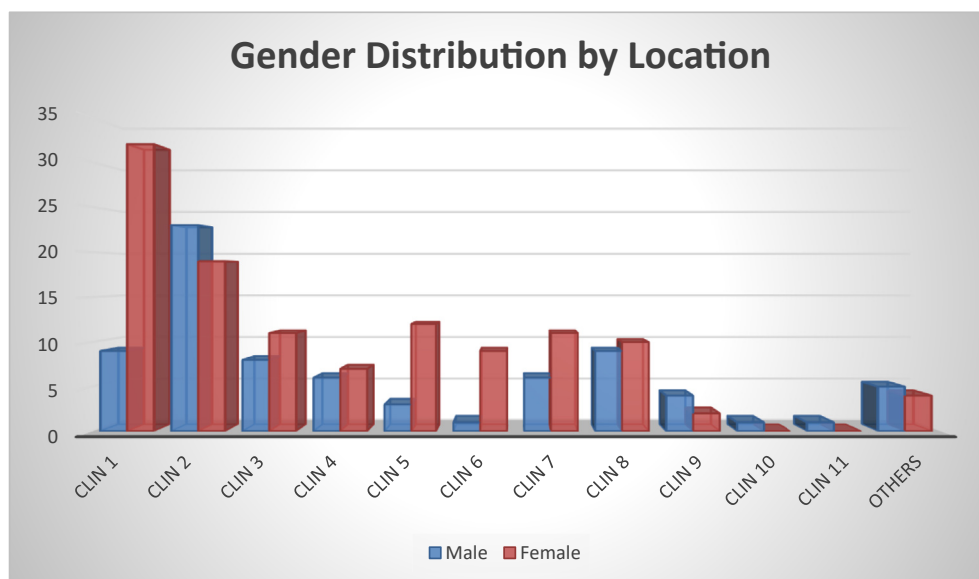
**FIG. 1.** Sex distribution of nonhospitalized patients.

TABLE 3. Specimen types collected in community

Site of specimen	Swabs, n (%)	Cotrimoxazole	Vancomycin	Clindamycin	Ciprofloxacin
Abscess	16 (8.3)	16	16	15	0
Aspirate	1 (0.5)	1	1	0	0
Axilla	4 (2.1)	4	4	4	0
Groin	2 (1.0)	2	2	2	0
Eye	2 (1.0)	2	2	0	0
Ear	6 (3.1)	6	6	6	0
Nasal	16 (8.3)	12	13	16	0
Penis	1 (0.5)	1	1	1	0
Vaginal	1 (0.5)	1	1	1	0
Pus	5 (2.5)	5	5	2	3
Ulcer	6 (3.1)	5	6	4	0
Wound	132 (68.4)	127	130	114	3
Skin	1 (0.5)	0	1	0	0
Total	193	182	188	169	6

TABLE 4. Age distribution and antibiotic susceptibility in community isolates

Age (years) (%)	Percentage susceptible to:			
	Cotrimoxazole	Vancomycin	Clindamycin	Ciprofloxacin
<18 (16.1%)	94.5	100	93.5	0
19–54 (35.2%)	97.1	99	86.8	1.5
≥55 (39.3%)	92.1	97	80.3	1.3
Average	94.6	98.7	86.9	0.9

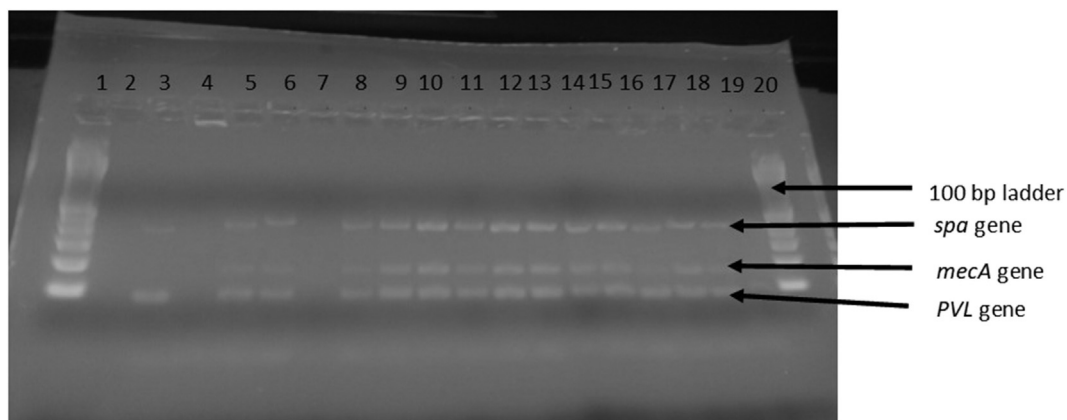


FIG. 2. Agarose gel electrophoresis of PCR-amplified products showing portions of *spa* (variable), *mecA* (162 bp) and *PVL* gene (83 bp). Lanes 1 and 20, 100 bp ladder; lanes 2–4, controls; lanes 5–19, isolates.

TABLE 5. Patient demographics, susceptibility and molecular characteristics of nonhospitalized patients with variant genotypic characteristics

Patient No.	Clinic	Specimen	Age (years)	Sex	D test results	Antibiotic				Amplified gene			
						CC	SXT	CIP	VAN	<i>spa</i>	<i>mecA</i>	<i>mecC</i>	<i>PVL</i>
9	5	Ear	40	F	—	S	S	R	S	+	+	—	—
13	2	Wound	—	M	—	S	R	R	S	+	+	—	—
14	5	Wound	58	F	—	R	S	R	S	+	+	—	—
16	1	Wound	38	F	—	R	S	R	S	+	+	—	—
47	3	Nasal	80	F	—	R	S	R	S	+	+	—	—
48	3	Nasal	67	F	—	R	S	R	S	+	+	—	—
49	4	Wound	41	M	—	R	S	R	S	+	+	—	—
70	4	Wound	48	F	—	R	S	R	S	+	+	—	—

CC, clindamycin; CIP, ciprofloxacin; R, resistant; S, susceptible; SXT, cotrimoxazole (trimethoprim/sulfamethoxazole); VAN, vancomycin.

There is limited information available on the antimicrobial susceptibility and prevalence of MRSA in the Caribbean region, particularly their genotypic characterization. Several studies in the Caribbean showed that MRSA isolates were predominantly SCCmec types IV and V, but no antimicrobial resistance patterns were documented [28]. Another study demonstrated the presence of the circulating sequence type (ST) 239 MRSA III clone in Trinidadian patients from 294 clinical isolates by microarray hybridization. The *mecA* gene was found in 15.3% of isolates compared to 285 (97.3%) of 293 of MRSA isolates from our study. The *PVL* gene was detected in 261 (89%) of 293 of clinical isolates, comprising of 76% from hospitalized patients and nonhospitalized patients (261/293, 63.1%). This gene was not analysed in that Trinidadian study, so no comparisons can be made [29]. A high proportion of patients in St Kitts and Nevis were found to have MRSA, with a prevalence of 45% compared to 17.9% of Barbadian patients, which seems to be similar to other Caribbean islands [29–33]. No *mecC* genes were detected, indicating that none of the MRSA isolates from Barbados were associated livestock-associated MRSA lineages. Of the hospital isolates, no *spa*, *mecA/mecC* or *PVL* genes were detected in eight patients, and in 15, *mecA* was undetected; however, all isolates were ceftioxin-resistant strains (MIC >4 µg/mL). The latter phenomenon could be associated with improper characterization of the strain or challenges in the basic microbiologic investigation. However, *spa*-deficient *S. aureus* has been isolated from Danish patients, which was either truly missing the *spa* gene or was associated with deletions in the immunoglobulin G-binding domain C, where the upstream primer used for *spa* typing is located [34]. Hence, nontypeable *spa* genes might not have been detectable with the set of primers used.

A predominant feature of CA-MRSA is the presence of the *PVL* genes that encode a *S. aureus* exotoxin that induces lysis of monocytes and neutrophil granulocytes and harbours SCCmec types IV and V [35–37]; a relationship between CA-MRSA, SCCmec type IV and V, and *PVL* has been confirmed in some studies [38–40]. The resistance of *S. aureus* to methicillin is caused by the *mecA* gene, located on a mobile genetic element, the staphylococcal cassette chromosome *mec* (SCCmec). This lack of a *mecA* gene and β-lactam resistance has been attributed to mutations in various genes, including those encoding penicillin-binding proteins [39,41]. Eight nonhospitalized patients had a similar pattern; these patients had isolates that were susceptible to vancomycin, analogous to the hospitalized patients. In addition, SCCmec type IV and V are known to be small and highly mobile elements. Their dissemination in a community population is mostly by transfer of strains from carriers to other individuals; from MRSA strains to MSSA strains; or even from coagulase-negative staphylococci strain to an MSSA strain

[42]. This was detected in the nares of two such patients, indicating the possibility of easy transfer within the community. These strains need further investigation to confirm the mechanisms of resistance due to the plausibility of detecting vancomycin-resistant *Staphylococcus aureus*, as seen with the strains exhibiting resistance.

In the present study, resistance to antibiotics both the community and hospital settings was high, particularly to fluoroquinolones (290/293, 98.9%), macrolides (community, 100%; hospitalized, 284/293, 97%) and β-lactams (100%) inclusive of methicillin. These findings are important because of the increasing antimicrobial resistance within the community and hospital settings. All hospital isolates were sensitive to vancomycin, linezolid, gentamicin and cotrimoxazole. Of the isolates from nonhospitalized subjects, 24 were resistant to clindamycin compared to 18 in hospitalized subjects, with positive inducible clindamycin results in 24 (8.2%) and two (0.7%) isolates respectively, indicating that clindamycin cannot be used in 4.5% of the population. Most isolates were susceptible to cotrimoxazole (186/193, 96.4%, of nonhospitalized and 100% of hospitalized patients). Some similarities in antibacterial susceptibility patterns to vancomycin, clindamycin, ciprofloxacin, erythromycin and cotrimoxazole have been observed in Trinidad, Jamaica, Antigua and the French territories [31,43–45].

Incongruous antibiotic usage, hygienic practices, ineffectual infection control and extensively used antimicrobials in agricultural practice has led to an increase in antimicrobial resistance in the community setting, which can be transferred to the hospital setting. Our findings show that the molecular characteristics and antimicrobial susceptibilities of isolates in hospitalized and nonhospitalized patients are similar. One isolate from a hospitalized patient was negative for the *PVL* gene and was designated a true HA-MRSA. This patient was first hospitalized in 2013 for diabetic complications, then returned in January 2015, where MRSA screening of nasal, axilla and groin was negative. Subsequently MRSA was isolated from surgical drains in February 2015, followed by positive blood culture, which remained positive until March 2015, upon this patient's death. The patient was administered vancomycin; however, because of an allergy, he was instead administered linezolid, but he never recovered. His isolates were sensitive to vancomycin, cotrimoxazole, gentamicin and linezolid and resistant to ceftriaxone, ciprofloxacin, clindamycin and erythromycin. This isolate could be classified as a true HA-MRSA due to the absence of the *PVL* gene. However, the remaining HA-MRSA isolates showed antimicrobial profiles similar to those characterized as CA-MRSA, so it can be surmised that the isolate of MRSA circulating is primarily from the community and that the incidence of true HA-MRSA is low and can be attributed to the stringent infection control measures set up by the Department

of Infection Control. Proper surveillance, antibiogram development and molecular characterization should be implemented in the community and hospital environment to monitor and guide treatment of these infections. In addition, all types of MRSA were found to be cotrimoxazole sensitive, suggesting this drug's likely use within both populations.

Conflict of interest

None declared.

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