Research Article

Determination of Virulence Factors and Resistance Profile of Methicillin-Resistant Staphylococcus aureus Strains among Different Types of spa, agr, and SCCmec

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In order to restrict the spread of methicillin-resistant S. aureus (MRSA) in hospitals, it is necessary to characterize isolates rapidly and precisely. The objective of this study was to determine virulence factors and resistance profiles of MRSA strains among spa, agr, and SCCmec types. In total, 55 MRSA isolates were collected from clinical specimens. The MRSA isolates were characterized by antimicrobial susceptibility testing, virulence genes, agr typing, spa typing, and SCCmec typing. According to our findings, all MRSA strains were resistant to cefoxitin; 88% and 86.7% of which were resistant to erythromycin and clindamycin, respectively. Type II agr was predominant with 54.54% frequency. Among 27 different spa types, type t030 was most frequently (25.45%). Most MRSA isolates (63.3%) were SCCmec type III. The pvl and tst genes were found in 25.3% and 32.7% of MRSA isolates, respectively. Among the MRSA strains, ermA, ermB, and ermA were present in 50%, 33.3%, and 57.3% of cases, respectively. In addition, 43 of the 55 MRSA strains (78%) harbored aminoglycoside resistance genes. The results of our study revealed that the MRSA rate in our region is dramatically high. Better infection control guidelines in hospitals, as well as ongoing epidemiological surveillance studies, could be strongly suggested for effective prevention of the spread of MRSA to inpatients.

1. Introduction

Among human pathogens, Staphylococcus aureus (S. aureus) is one of the most common ones [1]. Methicillin-resistant S. aureus (MRSA) strains have emerged as a major problem in hospitals, owing to the increased mortality rate associated with some of these infections. MRSA strain outbreaks have a significant impact on morbidity, mortality, and healthcare costs [2–4]. MRSA strains express virulence factors that play a key role in infection progression. The accessory gene regulator (agr) system regulates the expression of numerous virulence factors in S. aureus, and four major agr types have been identified to date. Different agr types have different properties and distributions in various geographic regions; thus, identification of the predominant types in each location would be of benefit [5, 6].

It is clear that the spread of MRSA around the world is constantly evolving, with new strains emerging in a variety of geographical regions. The mecA gene, which confers beta-lactam resistance, is found on the staphylococcal cassette chromosome mec (SCCmec) of MRSA strains. MRSA is divided into distinct epidemiological types based on the presence of the SCCmec element. The SCCmec typing method can help distinguish community-associated MRSA (CA-MRSA) from hospital-acquired MRSA (HA-MRSA) infections [7].

Continuous MRSA surveillance in each location necessitates monitoring the epidemiology, host characteristics, and
transmission routes of emerging strains [8]. Therefore, clinicians must have a thorough understanding of MRSA’s molecular epidemiology in order to assess the efficiency of preventative strategies and provide effective prophylaxis [9].

Prevention of MRSA transmission by screening patients, personnel, and the environment is a critical goal of infection control [9]. However, investigating the origins and routes of transmission of MRSA is possible only through the use of typing approaches, which are necessary for genetic characterization. There is a variety of molecular epidemiological methods used for MRSA surveillance, including multilocus sequencing typing (MLST), pulsed-field gel electrophoresis (PFGE), staphylococcal protein A (spa) gene sequencing, agr, and SCCmec typing [10]. Although each of these methods has a pretty good discriminating power, it has been demonstrated that combining genotyping methods is beneficial and advantageous for distinguishing distinct MRSA clones.

*spa* typing is a valuable typing instrument owing to its ease of use, cost-effectiveness, and uniform nomenclature, which is based solely on assessment of the repeat pattern in the *spa* gene [11]. The *spa* region polymorphism, which encodes a part of the *spa* protein, is characterized by variations in tandem repeats as well as variations in base sequences within repetitions. In other words, in any strain of *S. aureus*, each motif consists of 24 base pairs, which are referred to as unique sequence repeat motifs. The order of the repeats determines the *spa* type for a strain [12]. The *spa* types are important for identifying *S. aureus* outbreak isolates and infection control policies around the world. Over the last decade, studies have been conducted on the distribution of *spa*, *agr*, and SCCmec types in various geographic areas [13, 14]. Therefore, the current research is aimed at determining virulence and antimicrobial resistance profiles of MRSA isolates using *spa*, *agr*, and SCCmec typing.

2. Materials and Methods

2.1. Ethical Considerations. Ethics approval to perform this study was obtained from the institutional review board of Al-Zahra Hospital in Isfahan (Iran), then wards (emergency, men, women, children, and intensive care unit) in Al-Zahra Hospital in Isfahan (Iran), then referred to the hospital laboratory. The *S. aureus* was identified using growth on mannitol salt agar, showing beta-hemolysis on 5% sheep blood agar, and being gram positive as well as producing catalase, coagulase, and DNase. The presence of the *mecA* gene was confirmed by PCR in all *S. aureus* isolates (Table 1).

2.3. Antimicrobial Susceptibility Testing and Detection of MRSA. The following antibiotics were tested for antibiotic susceptibility using the disk diffusion technique on Mueller-Hinton agar, and the results were recorded after incubation for 18 hours at 37°C and in accordance with the CLSI guidelines [15]: penicillin (10 μg), erythromycin (15 μg), gentamicin (10 μg), tetracycline (30 μg), clindamycin (2 μg), rifampicin (5 μg), cefoxitin (30 μg), linezolid (5 μg), and trimethoprim-sulfamethoxazole (5 μg) (Mast, Merseyside, UK). The presence of the 310-base pair (bp) PCR product of the *mecA* gene was examined in all *S. aureus* isolates (Table 1), and cefoxitin (30 μg) discs on Muller-Hinton agar plates were used to screen for MRSA isolates.

2.4. Genomic DNA Extraction. A DNA Mini Kit (Qiagen GmbH, Hilden, Germany) was used for genomic DNA extraction. Fresh colonies harvested from agar plates were washed with 500 μl TE 1x and centrifuged for 10 minutes at 5000 rpm according to manufacturer’s protocol. A suspension was then prepared in 200 μl TE 1× with 20 μl lysostaphin (200 μg/ml final concentration) and incubated at 37°C for 20 minutes. Finally, the obtained DNA was dissolved 50 μl RNase-DNase-free water (Sigma). DNA concentration was measured with a spectrophotometer.

2.5. SCCmec Typing. As described previously, multiplex PCR was used to identify various MRSA isolates using genomic DNA as the template [16]. The amplification started with a 3-minute denaturation step at 94°C, then 35 cycles of 30 seconds at 94°C, 1 minute at 55°C, 1 minute at 72°C, and finally 5 minutes at 72°C for final extension.

2.6. Detection of Virulence and Resistance Genes. To detect virulence genes such as hemolysin A (hla), toxic shock syndrome toxin (lst), staphylococcal enterotoxins (sea, seb, and sec), and Panton-Valentine leukocidin, PCR was used (pvl). As previously described, PCR assays were used to investigate the common aminoglycoside resistance genes (aac (6′)-aph (2″), aph3, ant4) and macrolide resistance genes (ermA, ermB, ermC) [17, 18].

2.7. Detection of *agr* Types. Multiplex PCR was performed to detect *agr* types using a set of primers containing a common forward primer (Pan) and reverse primer (agrI, agrII, agrIII, and agrIV) that are unique to each *agr* group [19]. The primer sequences are shown in Table 1.

2.8. Detection of *spa* Types. The identified MRSA strains were subjected to PCR to detect the *spa* gene (Table 1). The amplification reaction consisted of an initial denaturation step at 94°C for 5 min followed by 35 cycles of denaturation at 94°C for 40 second, hybridization at 56°C for 40 second, and extension to 72°C for 50 second, followed by final extension to 72°C for 5 minutes [11]. Sequencing was then performed on the PCR products. Also, after sequencing, the spa database server (http://spaserver.ridom.de/) was used to determine different types.

2.9. Statistical Analysis. For statistical analysis, SPSS Statistics 22.0 for Windows was used. Data were presented using descriptive statistics (frequency, percentage, mean, and standard deviation).
3. Results

3.1. Detection and Isolation of MRSA. In this study, 55 clinical MRSA isolates were recovered from blood samples (n = 12; 21.83%), nasal (n = 14; 24.5%), urine (n = 7; 12.72%), trachea (n = 8; 14.54%), wound (n = 12; 21.83%), and synovial (n = 2; 3.63%). According to our data, the nasal specimen has the highest frequency of MRSA (26%). The patients were divided into 29 (52%) males and 26 (48%) females. Participants in the study ranged in age from 9 to 86. Most of the study participants were in the 21–60-year-old group (66%).

3.2. Antibiotic Susceptibility Tests. Antibiotic susceptibility testing was performed on all MRSA isolates. All were resistant to cefoxitin and penicillin; 88% and 86.7% of them were resistant to erythromycin and clindamycin, respectively. On the other hand, all MRSA isolates were sensitive to linezolid (Table 2).

3.3. agr Typing. According to agr typing, 55 of the MRSA isolates belonged to one of agr types I, II, III, or IV. By using the agr typing method, 29.09% (n = 16), 54.54% (n = 30), 10.9% (n = 6), and 5.45% (n = 3) of isolates belonged to agr types I, II, III, and IV, respectively.

3.4. Prevalence of SCCmec Types, Virulence, and Resistance Genes. Most MRSA isolates (63.3%) were SCCmec type III. Also, the frequency of SCCmec types II and IX was 10.7% for each, 9.3% as SCCmec type V, 4% as SCCmec type I, and 2% as SCCmec type IV. In addition, 43 of the 55 MRSA strains (78%) harbored aminoglycoside resistance genes, with the presence of aac(6’)-aph(2”), aph3, and ant4 genes among MRSA isolates, were 54%, 32.7%, and 31.3%, respectively. Among the 55 MRSA strains, macrolide resistance genes ermC, ermH, and ermB were detected in 35 (63.6%), 11 (20%), and 9 (16.4%) isolates, respectively. In our study, genes encoding staphylococcal enterotoxins sea, seb, and sec were found in 48%, 25.5%, and 12% of MRSA isolates, respectively. In addition, the pvl and tst genes were found in 25.3% and 32.7% of MRSA isolates, respectively.

3.5. spa Typing. Twenty-seven spa types were observed in this study. There were 19 spa types that were found only once in all of the 55 strains analyzed. Accordingly, single types of spa are extremely important in MRSA strains. Table 3 shows that t030 and t037 predominate in clinical samples, especially in blood and nasal samples. As well as, phenotypic and genotypic traits of all our isolates are presented in Table 4 [21].

4. Discussion

MRSA strains are one of the leading causes of infections in hospitals, but infections from community-related MRSA have become a global public health threat over the recent decades [22]. The widespread occurrence of multidrug-resistant (MDR) MRSA augments the cost of antibiotic therapy and limits treatment options. During the last two decades, the widespread use of beta-lactam antibiotics in Iranian hospitals and medical facilities led to increased resistance to these antibiotics [23]. The results of the current study revealed that MRSA strains are resistant to erythromycin (88%), clindamycin (86.6%), tetracycline (68%), rifampicin (57%), and gentamicin (54.6%). Based on these results, linezolid was the most effective drug for MRSA in the study area. In addition, more than 93% of our MRSA isolates were not included. As a protein synthesis inhibitor, erythromycin is widely utilized for the treatment of staphylococcal infections [24]. According to Mahdiyoun et al., the frequency of MRSA resistance tested for erythromycin was 84.4% [25]. A study conducted in Taiwan reported that the resistance rates for erythromycin and clindamycin were 94.9% and 86.5%, respectively [26]. High frequency of resistance to erythromycin and clindamycin antibiotics in the present study was consistent with the findings of previous research in Iran [24] and India [27].

We also found SCCmec type III in a high percentage of MRSA isolates. Similarly, studies in Iran and other Asian
countries have reported the high prevalence of SCCmec type III [28–30]. In MRSA isolates, SCCmec mobile genetic factor leads to an expansion of antibiotic resistance determinants as well as virulence factors, which can act as a large reservoir of resistance genes, enterotoxins, and other virulence factor genes. Our results showed that among MRSA with SCCmec type III, sea, hla, and seb were the most frequently found genes encoding virulence factors. Herein, the majority of the isolates with type III were resistant to erythromycin and clindamycin (55% and 54%) while all the isolates were

| Type of specimen | ERY | CD | SXT | GM | TE | RA |
|------------------|-----|----|-----|----|----|----|
| Blood            | 1.3 | 20.0 | 0.7 | 20.7 | 9.3 | 12.0 |
| Nasal            | 3.3 | 22.7 | 3.7 | 22.7 | 12.7 | 13.3 |
| Urine            | 4.7 | 8.7 | 2.7 | 10.7 | 6.7 | 6.7 |
| Trachea          | 0.7 | 14.0 | 2.0 | 12.7 | 8.0 | 6.7 |
| Wound            | 1.3 | 20.0 | 4.7 | 16.7 | 10.7 | 13.3 |
| Synovial         | 0.7 | 2.7 | 0.0 | 3.3 | 2.7 | 0.7 |
| Total            | 12.0 | 88.0 | 13.3 | 86.7 | 50.0 | 50.0 |

| Spa type | Blood | Nasal | Urine | Trachea | Wound | Synovial | Total |
|----------|-------|-------|-------|----------|-------|----------|-------|
| t275     | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t4679    | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t7685    | 1 (50) | —     | 1 (50) | —        | —     | —        | 2     |
| t3236    | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t790     | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t030     | 4 (28.6) | 5 (35.7) | 1 (7.1) | 2 (14.3) | 2 (14.3) | —       | 14    |
| t037     | 1 (12.5) | 2 (25) | 2 (25) | 1 (12.5) | 1 (12.5) | 1 (12.5) | 8     |
| t3769    | —     | —     | —     | —        | —     | —        | 1 (100) | 1     |
| t3204    | —     | 1 (50) | —     | —        | —     | —        | 2     |
| t314     | —     | —     | —     | —        | —     | —        | 1     |
| t5163    | —     | —     | —     | —        | —     | —        | 1     |
| t0325    | 1 (25) | —     | —     | 1 (25) | 2 (50) | —        | 4     |
| t1587    | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t223     | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t5593    | —     | —     | 1 (100) | —        | —     | —        | 1     |
| t131     | —     | —     | —     | —        | 1 (100) | —        | 1     |
| t15871   | —     | —     | —     | —        | 1 (100) | —        | 1     |
| t159     | —     | —     | —     | —        | 1 (100) | —        | 1     |
| t1360    | —     | —     | —     | —        | 2 (100) | —        | 2     |
| t692     | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t2976    | —     | —     | 1 (100) | —        | —     | —        | 1     |
| t2104    | —     | —     | —     | —        | 1 (100) | —        | 1     |
| t1258    | —     | —     | —     | 2 (100) | —     | —        | 2     |
| t1403    | —     | 1 (100) | —     | —        | —     | —        | 1     |
| t2457    | 1 (100) | —     | —     | —        | —     | —        | 1     |
| t3182    | 1 (100) | —     | —     | —        | —     | —        | 1     |
| t459     | 1 (100) | —     | —     | —        | —     | —        | 1     |
| Total    | 10 (18.2) | 16 (29) | 6 (11) | 8 (14.55) | 12 (21.8) | 3 (5.45) | 55    |
Table 4: Phenotypic and genotypic traits of all MRSA isolates in this study. Some data were collected with the study of Latifpour et al. [21].

| Isolate number | spa type | agr type | Sample type | SCCmec | Virulence genes | Resistance profile | Resistance genes |
|----------------|----------|----------|-------------|--------|----------------|--------------------|------------------|
| 1.             | t030     | II       | Blood       | III    | hla, sea, seb  | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), aph3, ermC |
| 2.             | t030     | II       | Blood       | III    | tst            | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), aph3, ermB |
| 3.             | t030     | IV       | Wound       | III    | pvl, hla, sea, seb, sec | FOX, ERY, CD, SXT, GM, TE | aac (6′)-aph (2″), ant4, ermC |
| 4.             | t030     | II       | Trachea     | III    | hly, sea      | FOX, ERY, CD, SXT, GM, RA | aac (6′)-aph (2″), aph3 |
| 5.             | t030     | I        | Nasal       | III    | hly, sea      | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), aph3 |
| 6.             | t030     | II       | Nasal       | III    | pvl, tst, hly, sea | FOX, ERY, CD, TE | ermC |
| 7.             | t030     | II       | Blood       | III    | tst            | FOX, ERY, CD, SXT, GM, RA | aac (6′)-aph (2″), aph3 |
| 8.             | t030     | I        | Nasal       | III    | hla, sea      | FOX, SXT, GM, TE, RA | aph3, ant4, aac (6′)-aph (2″) |
| 9.             | t030     | II       | Nasal       | IX     | hla           | FOX, ERY, CD, SXT, GM, TE, RA | ermC |
| 10.            | t030    | I        | Nasal       | V      | —             | FOX, GM | ant4, aph3, ermA |
| 11.            | t030    | I        | Blood       | V      | pvl, hla, sea | FOX, ERY, TE | ermC, ermA |
| 12.            | t030    | IV       | Wound       | II     | hla, sea, seb | FOX, ERY, CD, SXT, TE, RA | ermC, ermB |
| 13.            | t030    | I        | Trachea     | II     | hla, tst     | FOX, ERY, TE | ermC, ermB |
| 14.            | t030    | I        | Urine       | IX     | hla, tst     | FOX, ERY, CD | ermC, ermA |
| 15.            | t037    | III      | Urine       | II     | hla, sec     | FOX, TE | — |
| 16.            | t037    | I        | Urine       | III    | pvl, tst, hla, sea, seb | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), ant4, aph3 |
| 17.            | t037    | I        | Nasal       | III    | hla           | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), aph3 |
| 18.            | t037    | III      | Synovial    | III    | hla           | FOX | — |
| 19.            | t037    | II       | Wound       | III    | hla, sea     | FOX, TE | — |
| 20.            | t037    | II       | Nasal       | IX     | pvl, tst, hla, sea, seb | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), aph3 |
| 21.            | t037    | I        | Trachea     | IX     | hla, sea     | FOX | — |
| 22.            | t037    | I, II    | Blood       | IX     | hla, sea     | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), ermC |
| 23.            | t1258   | I        | Trachea     | III    | tst, hla, seb | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), aph3, ermA |
| 24.            | t1258   | I        | Trachea     | I      | pvl, hla, sea | FOX, ERY, CD, TE, RA | aac (6′)-aph (2″), aph3, ermA |
| 25.            | t1360   | I, II    | Wound       | III    | hla, sea     | FOX, ERY, CD, SXT, RA | aac (6′)-aph (2″), ermC |
| 26.            | t1360   | III      | Wound       | III    | hla, sea, sec | FOX, ERY, CD, SXT, GM, RA | aac (6′)-aph (2″), aph3, ermC |
| 27.            | t131    | II       | Wound       | III    | pvl, hla, sea | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), ermC |
| 28.            | t5163   | II       | Trachea     | III    | pvl, hla, seb | FOX, ERY, CD, GM, TE | aac (6′)-aph (2″), ermC |
| 29.            | t5163   | I        | Wound       | III    | hla          | FOX, ERY, CD, TE | ant4, ermC |
| 30.            | t7685   | II       | Urine       | III    | tst, hla     | FOX, ERY, CD, SXT, GM, TE | aac (6′)-aph (2″), aph3, ermC |
| 31.            | t7685   | III      | Blood       | III    | hla, tst, sea | FOX, ERY, CD, SXT, GM | aac (6′)-aph (2″), ant4, ermC |
| 32.            | t1587   | II       | Nasal       | III    | hla, sea     | FOX, ERY, CD, GM, TE | aac (6′)-aph (2″), aph3, ermB |
| 33.            | t1403   | II       | Nasal       | V      | tst, hla, sea, seb | FOX, ERY, SXT, GM | aac (6′)-aph (2″), ant4 |
| 34.            | t15871  | II       | Trachea     | III    | pvl, hla     | FOX, ERY, CD, GM, TE, RA | ant4, ermC |
| 35.            | t159    | I        | Trachea     | III    | hla, sea, seb, sec | FOX, ERY, CD, GM, TE, RA | ant4, ermC |
| 36.            | t2104   | II       | Wound       | III    | hla, sea     | FOX, ERY, CD, TE, RA | aac (6′)-aph (2″), ermC |
| 37.            | t275    | II       | Nasal       | III    | pvl, hla     | FOX | — |
| 38.            | t223    | II       | Nasal       | V      | tst, seb     | FOX, ERY, CD, TE | ant4, ermA |
| 39.            | t2457   | II       | Blood       | III    | pvl, hly, seb | FOX, ERY, CD, GM, TE, RA | aac (6′)-aph (2″), aph3 |
| 40.            | t314    | Wound    | III         | hla    | FOX, ERY, CD, GM, TE | aac (6′)-aph (2″), ermC |
| 41.            | t2976   | II       | Urine       | III    | pvl, tst, hla, sea | FOX, ERY, CD, SXT, GM, TE, RA | aac (6′)-aph (2″), aph3, ermA |
| 42.            | t3182   | I        | Blood       | III    | pvl, tst, hla, sea | FOX, ERY, CD, GM, TE | ant4, ermC |
| 43.            | t3236   | III      | Nasal       | III    | pvl, hla     | FOX, ERY, CD, GM, TE | aac (6′)-aph (2″), ermC |
resistant to gentamicin. The SCCmec typing has also been done in other parts of Iran in accordance with our study. A study by Ebrahim-Saraie et al., Moshtagheian et al., and Parhizgari et al. reported that SCCmec type IV dominated MRSA isolates; however, in line with our findings, most studies conducted in Iran described SCCmec type III as the predominant SCCmec type [31–33]. In the present study, among MRSA strains, ermC (63.6%) was the most commonly detected macrolide resistance gene, followed by ermA (20%) and ermB (16.4%). Also, the most frequently identified aminoglycoside resistance gene was aac (6′)-aph (2″) (54%). These findings are in contrast with the study by Hau et al. [34], which showed clinical MRSA in the United States in which an incidence of 91.5% for ermA and 12.7% for ermB was reported. A similar finding was also reported by Yilmaz and Aslantaş [35] that the ermC and aac (6′)-aph (2″) genes were detected in 91.5% and 50% of S. aureus isolates, respectively.

In view of the widespread of MRSA isolates, it is imperative that the treating physician encourages the preservation of glycopeptides and linezolid only in the case of MRSA. In Iran, a major MRSA-associated problem is the result of increased incidence and hospitalization rates. Therefore, for screening, epidemiology, surveillance, and infection control, rapid and accurate typing of MRSA isolates is crucial [23]. Several genotyping techniques are available for identifying S. aureus strains in epidemiological studies. Sequence-based typing methods, such as MLST and spa typing, have several obvious advantages; for example, they are easily used, portable, reproducible, and able to provide comparable results compared to tape-based methods, such as small macrorestrictrion analysis [14]. One of the key regulators of S. aureus, which is involved in the regulation of bacterial virulence factors, is the agr system. There are currently four agr types identified in S. aureus strains (I, II, III, and IV). According to our study, the predominant agr type among the 55 MRSA isolates was type II, with a frequency of 54.54%.

### 5. Conclusion

According to recent studies, presence of these common SCCmec (III), spa (t030), and agr (II) types indicates that these MRSA strains are actively circulating in the healthcare setting of Isfahan Province. Despite the high diversity of our spa types (27/55) in this study, most of them are classified as t030 and t037 and are probably phylogenetically related. There is a possibility that these strains may spread to other parts of Iran or the world in relation to the tourism industry in Isfahan Province. Therefore, the current study emphasizes the importance of molecular typing in tracking global trends in the emergence, spread, and persistence of epidemic MRSA strains. Better infection control guidelines in hospitals, as well as ongoing epidemiological surveillance studies,
could be strongly suggested for effective prevention of bacteria spread to inpatients and control nosocomial infections.

Data Availability

The authors confirm that the data supporting the findings of this study are available within the article.

Consent

Consent is not necessary.

Conflicts of Interest

The authors declare that there is no conflict of interests regarding the publication of this paper.

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