Genotype Distribution of Panton-Valentine Leukocidin (PVL)-Positive \textit{Staphylococcus aureus} Strains Isolated from Wound-Related Infections: a Three-Year Multi-Center Study in Tehran, Iran

Mehdi Goudarzi\textsuperscript{1,*}, Maryam Fazeli\textsuperscript{2}, Ramin Pouriran\textsuperscript{3}, and Gita Eslami\textsuperscript{1}

\textsuperscript{1}Department of Microbiology, School of Medicine and \textsuperscript{3}School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran; and \textsuperscript{2}Department of Virology, Pasteur Institute of Iran, Tehran, Iran

SUMMARY: The spread of Panton-Valentine leucocidin (PVL)-carrying \textit{S. aureus} strains in patients with wound infections in both the community and hospitals is increasing in some areas of Iran. In the present study, we determined the molecular characteristics and distribution of PVL-producing \textit{S. aureus} strains isolated from wound infections. Genes encoding resistance, toxins, and staphylococcal enterotoxins were analyzed by polymerase chain reaction assays. Genotyping was performed using multi-locus sequence typing. Aminoglycoside resistance genes including \textit{ant (4')-Ia (57.4\%)} and \textit{aac (6')-Ie/aph (2')} (45.7\%) were the most prevalent genes in isolates. Staphylococcal enterotoxin type A, as the most frequent type, was present in 20.2\% of isolates. Strains belonged to seven clonal complexes. The most frequent clonal complex was CC30 (ST30) (29.8\%), followed by CC22 (ST22) (21.3\%), CC8 (ST8 and ST931) (17\%), CC88 (ST88) (10.6\%), CC59 (ST59 and ST338) (8.5\%), CC1 (ST772 and ST1) (7.5\%), and CC15 (ST15) (5.3\%). Our findings indicated an increasing trend of CC30, carrying a wide range of resistance and toxin genes, which could present an obstacle in the treatment of patients with wound infections. Further studies are required to investigate the carriage of resistance, the antibiotic susceptibility pattern, and toxins encoding genes in different molecular types.

INTRODUCTION

\textit{Staphylococcus aureus} is an opportunistic pathogen that principally colonizes the skin of about two-thirds of the population. This opportunistic pathogen has the capability of causing a wide range of diseases, including skin and soft-tissue infections (SSTIs) (1). Recently, a dramatic increase in the number of patients with wound infections associated with \textit{S. aureus} strains has been reported, representing a significantly high rate of mortality and morbidity in the healthcare setting (1–3).

The expression of virulence factors and the presence of antibiotic resistance genes make \textit{S. aureus} a highly pathogenic microorganism (4–7). Panton-Valentine leukocidin (PVL), a pore-forming toxin that disrupts host cell membranes, reduces immune resistance by leukocyte lysis or apoptosis and targets complement receptors. This leukotoxin is composed of S-related and F-related proteins, which are carried by several lysogenic bacteriophages (8).

In the past few decades, it has been well-documented that SSTIs are frequently associated with \textit{S. aureus} strains harboring PVL (9,10). Recent reports worldwide have shown that hospital-acquired \textit{S. aureus} clones with PVL production and methicillin resistance are rapidly emerging and appear to be driven by the dramatic increase in community-acquired methicillin-resistant \textit{S. aureus} (CA-MRSA) skin infections (11,12). It is well-established that PVL-encoding genes could be found in highly diverse clones of \textit{S. aureus} isolates. Hence, most researchers are currently focused on better understanding the epidemiology of PVL-positive MRSA isolates in order to develop public health interventions for MRSA infection control (13,14).

Despite the high incidence of PVL-positive \textit{S. aureus} in wound infections, the data regarding the prevalence and characteristics of PVL-producing \textit{S. aureus} isolated from patients with wound infections in Iran are limited. Hence, this study was planned to investigate antibiotic-resistant phenotypes and molecular characteristics of PVL-producing \textit{S. aureus} strains isolated from wound infections in Tehran, Iran.

MATERIALS AND METHODS

Study population, bacterial isolation, and PVL genes detection: The present cross-sectional study was performed during a period of three years from April 2015 to 2018. A total of 94 non-duplicate PVL-positive \textit{S. aureus} strains were obtained from 365 \textit{S. aureus} strains isolated from patients with wound infections who were referred to eight hospitals located in different parts of Tehran.

The wound samples were collected using a sterile cotton swab. Standard microbiological methods were applied for preliminary identification (15).
confirmations of isolates were based on distinction of nucA genes by polymerase chain reaction (PCR) (16). The entire strains were also analyzed for the presence of lukF-PV and lukS-PV gene encoding components of PVL. The PCR technique utilized was described by Lina et al. (17). The wound infections in this study included skin abscesses (49/94, 52.1%), surgical wounds (17/94, 18.1%), burn wounds (12/94, 12.8%), traumatic wounds (9/94, 9.6%), and decubitus wounds (7/94, 7.4%). The PVL-positive S. aureus strains were kept in Tryptic Soy Broth (TSB; Merck KGaA, Darmstadt, Germany) containing 20% glycerol at −70°C prior to subsequent analysis (18).

Ethics approval: The study protocol was reviewed and approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences (IR.SBMU.MSP.REC.1396.607).

Antimicrobial susceptibility testing: The antimicrobial susceptibility profiles for PVL-positive isolates were determined by the disk diffusion test for kanamycin, ciprofloxacin, rifampicin, clindamycin, quinupristin-dalfopristin, tetracycline, erythromycin, linezolid, teicoplanin, amikacin, tobramycin, gentamicin, and trimethoprim-sulfamethoxazole (Mast Group Ltd.,Bootle, UK) based on Clinical and Laboratory Standards Institute (CLSI) guidelines (19). S. aureus ATCC29213, S. aureus ATCC25923, and S. aureus ATCC BAA-976 (D-zone test negative), and S. aureus ATCC BAA-977 (D-zone test positive) were employed as quality-control strains. For vancomycin, mupirocin, and fusidic acid (Sigma-Aldrich, St. Louis, MO, USA), the minimal inhibitory concentration (MIC) was determined by the broth microdilution test. The findings for susceptibility were analyzed using the CLSI criteria (19), except for fusidic acid, which was interpreted according to the European Committee for Antimicrobial Susceptibility Testing instructions (20).

Screening for MRSA strains: In order to determine the resistance to methicillin, a cefoxitin disk (30 µg) was used on a Mueller Hinton agar plate, supplemented with 4% NaCl based on the instructions approved by the CLSI (19). Isolates with phenotypic resistance to oxacillin were genetically established by PCR for the presence of the mecA gene, as previously described (21,22).

DNA extraction: Genomic DNA was extracted from overnight pure cultures of PVL-carrying S. aureus strains on 5% sheep blood agar (BA; Merck) by using phenol-chloroform extraction. If the purity was appropriate, it was used as the template for PCR.

Amplification of selected resistance and toxin-encoding genes: The entire isolates were screened for the presence of resistance (mecA, mupA, msp(A), vanA, erm(A), mepB, msp(B), vanB, erm(B), erm(C), aph(3’)-IIIa, tet(M), aac(6’)-Ie-aph(2’)) and ant(4’)-Ia) and staphylococcal toxins and enterotoxin (tst, eta, sea, seh, seb, sec, sed, see, seg, seh, sei, and sej) encoding genes by conventional PCR (7,8,22).

Multi-locus sequence typing (MLST): All the 94 PVL-positive S. aureus isolates underwent MLST as stated by the method of Enright et al. (23) by sequencing an internal fragment of seven unlinked housekeeping genes to identify the following allelic profiles: pta, arcC, tpi, arOE, gmk, yqiL, and glp. Eventually, sequence types (STs) were identified by submission of the DNA sequences to the online MLST database (https://pubmlst.org/).

RESULTS

A total of 94 S. aureus isolates were analyzed in this study. The results for MRSA screening showed that all strains were resistant to methicillin according to the phenotypic method and harbored the mecA gene, which confirmed MRSA. The maximum level of resistance of MRSA isolates with PVL-encoding gene strains was observed against erythromycin (77.7%). Resistance was observed against gentamicin (75.5%), tetracycline (71.3%), ciprofloxacin (58.5%), clindamycin (51.1%), amikacin (47.9%), kanamycin (43.6%), tobramycin (38.3%), rifampicin (40.4%), trimethoprim-sulfamethoxazole (29.8%), mupirocin (18.1%), and quinupristin-dalfopristin (15.9%). The entire strains were susceptible to teicoplanin, linezolid, vancomycin, and fusidic acid. All the strains were inhibited by fusidic acid at a similar MICs and MICso of 0.5 µg/mL. In fact, none of the isolates tested positive for vanA and B. Out of 17 PVL-positive MRSA clinical isolates resistant to mupirocin (18.1%), 12 (12.8%) and five (5.3%) isolates exhibited the HLMUPR and the LLMUPR phenotypes, respectively. All the 12 HLMUPR strains harbored the mupA gene. The mupB gene was not detected in any of the mupirocin-resistant isolates. Overall prevalence of the macrolide and streptogramin type B (MS); inducible macrolide, lincosamide, and streptogramin type B (iMLSb); and constitutive macrolide, lincosamide, and streptogramin type B (cMLSb) phenotype was found to be 11.7%, 14.9%, and 62.8%, respectively. Remarkably, 12 different antibiotic resistance profiles were identified among PVL-positive MRSA strains.

Regarding the prevalence of toxin-encoding genes, the most prevalent gene was sea (n = 19; 20.2%); this was followed by sei (n = 14; 14.9%), seg (n = 13; 13.8%), sej (n = 9; 9.6%), sec (n = 7; 7.4%), seb (n = 6; 6.4%), sed (n = 5; 5.3%) she (n = 2; 2.1%), and eta (n = 2; 2.1%). The see, etb, and tst genes could not be detected.

The analysis of the resistance genes in the PVL-positive MRSA strains revealed that the most prevalent gene was ant(4’)-Ia (54; 57.4%), followed by aac(6’)-Ie-aph(2’)) (43; 45.7%), tet(M) (40; 42.5%), aph(3’)-IIIa (37; 39.4%), erm(C) (37; 39.4%), erm(B) (15; 15.9%), mupA (12; 12.8%), mupB (11; 11.7%), mupB (7; 7.4%), and msp(A) (6; 6.4%) genes. Notably, there was no isolate that carried mupB, vanK, or vanB genes.

94 PVL-positive isolates belonged to seven Clonal Complexes (CCs). The most frequent CC was CC30 (ST30) (29.8%), followed by CC22 (ST22) (21.3%), CC8 (ST8 and ST931) (17%), CC88 (ST88) (10.6%), CC59 (ST59 and ST338) (8.5%), CC1 (ST772 and ST1) (7.5%), and CC15 (ST15) (5.3%). Our results revealed that CC22 decreased from approximately half of tested
isolates in 2016 to 2.9% in 2018. An increasing trend was observed for CC30, which drastically increased to 52.9% in 2018 after reaching a maximum of 9.7% in 2016. The annual overview on PVL-carrying MRSA clinical isolates is presented in Table 1.

In the present study, 12 isolates (12.8%) expressed HLMPUR, all of which belonged to CC8 (ST8). The LLMPUR phenotype was detected in seven isolates (5.3%) that belonged to CC15 (ST15). An inducible resistance to clindamycin was observed in ST8 (8.5% [8/94]), ST22 (4.3% [4/94]), and ST88 (2.1% [2/94]), while the constitutive phenotype was predominant in CC30 (25.5% [24/94]), CC22 (12.8% [12/94]), and CC88, CC59, and CC1 (4.3% [4/94] each). Constitutive phenotypes were not detected in CC8 and CC15. MS

| Strain | April 2015-March 2016 n (%) | April 2016-March 2017 n (%) | April 2017-March 2018 n (%) | Total n (%) |
|--------|-----------------------------|-----------------------------|-----------------------------|-------------|
| CC30   | 3 (9.7)                     | 7 (24.1)                    | 18 (52.9)                   | 28 (29.8)   |
| CC22   | 14 (45.2)                   | 5 (17.2)                    | 1 (2.9)                     | 20 (21.3)   |
| CC8    | 5 (16.1)                    | 6 (20.7)                    | 5 (14.7)                    | 16 (17)     |
| CC88   | 2 (6.5)                     | 5 (17.2)                    | 3 (8.8)                     | 10 (10.6)   |
| CC59   | 3 (9.7)                     | 1 (3.4)                     | 4 (11.8)                    | 8 (8.5)     |
| CC1    | 4 (12.9)                    | 3 (10.3)                    | 0 (0)                       | 7 (7.5)     |
| CC15   | 0                           | 2 (6.9)                     | 3 (8.8)                     | 5 (5.3)     |
| Total  | 31 (33)                     | 29 (30.9)                   | 34 (36.1)                   | 94 (100)    |

### Table 2. Molecular characterization of PVL-positive MRSA isolates

| Clonal Complex (CC) | PVL positive MRSA sequence types | Virulence genes (n ; %) | Antibiotic resistance profile (n ; %) | Antibiotic resistance genes (n ; %) | Hospitals (n ; %) | Total n (%) |
|---------------------|----------------------------------|------------------------|--------------------------------------|-------------------------------------|------------------|-------------|
| CC30                | ST30                             | sea (5;17.9), sei (10;35.7), seg (12;42.9) | E, GM, T, CIP, CD, AK, K, RI (5;17.9) | tet(M) (14;50), erm(C) (11;39.3), erm(B) (2;7.1), ant (4’)-Ia (25;89.3), aac (6’)-Ie/aph (2’) (20;71.4), aph (3’)-IIa (18;64.3), msr(B) (2;7.1) | H1 (2;100), H2 (4;14.4), H3 (2;7.1), H4 (3;10.7), H5 (2;7.1), H6 (3;10.7), H7 (7;25), H8 (2;7.1) | 28 (29.8) |
| CC22                | ST22                             | sec (3;15), sei (3;15), seg (1;5), sea (1;5) | E, GM, T, CIP, CD, AK, K, RI (5;25) | erm(C) (8;40), msr(B) (5;25), erm(B) (10;50), ant (4’)-Ia (12;60), aac (6’)-Ie/aph (2’) (11;55), erm(A) (2;10) | H1 (4;20), H2 (5;25), H4 (2;10), H5 (4;20), H7 (3;15), H8 (2;10) | 20 (21.3) |
| CC8                 | ST8                              | sea (5;41.7), sed (2;16.7) | E, GM, T, CIP, AK, RI, MS (4;20) | erm(C) (10;83.3), ant (4’)-Ia (3;25) | H2 (3;25), H3 (2;16.7), H4 (4;33.3), H7 (3;25) | 12 (12.8) |
| CC88                | ST931                            | seb (3;75), sec (2;50) | E, GM, T, AK, K, RI, TN (4;100) | ant (4’)-Ia (3;75), aac (6’)-Ie/aph (2’) (3;75), aph (3’)-IIa (1;25), tet(M) (3;75) | H1 (1;25), H5 (1;25), H6 (2;50) | 4 (4.2) |
| CC8                 | ST88                             | sea (6;60), sed (3;30), sej (3;30), eta (2;20) | E, GM, T, CIP, AK, K, RI, TN (4;40) | tet(M) (7;70), erm(C) (5;50), erm(A) (7;70), aac (6’)-Ie/aph (2’) (2;20), msr(B) (6;60) | H2 (3;30), H3 (2;20), H4 (3;30), H7 (1;10), H8 (1;10) | 10 (10.6) |
| CC59                | ST59                             | seb (3;42.9), sec (4;57.1) | E, GM, T, CIP, CD, AK, K, RI (2;28.6) | tet(M) (4;57.1), erm(C) (3;42.9), erm(B) (3;42.9), erm(A) (1;14.3), ant (4’)-Ia (2;28.6), aac (6’)-Ie/aph (2’) (2;28.6) | H1 (2;28.6), H7 (3;42.8), H8 (2;28.6) | 7 (7.4) |
| CC1                 | ST772                            | sea (1;20), sei (1;20), sej (2;40) | E, GM, T, CIP, CD (3;60) | ant (4’)-Ia (4;80), aac (6’)-Ie/aph (2’) (3;60), aph (3’)-IIa (4;80), tet(M) (5;100) | H3 (2;40), H4 (1;20), H6 (1;20), H5 (1;20) | 5 (5.3) |
| CC1                 | ST1                              | seb (2;100), sec (1;50) | E, GM, T, CIP, CD (1;50) | tet(M) (1;50), erm(A) (1;50) | H8 (2;100) | 2 (2.2) |
| CC15                | ST15                             | -                       | E, GM, T, CIP, AK, K, MUP (3;60) | aac (6’)-Ie/aph (2’) (5;100), tet(M) (100) | H3 (2;40), H4 (4;20), H7 (1;20) | 5 (5.3) |

1) CD, clindamycin; E, erythromycin; GM, gentamicin; RI, rifampicin; T, tetracycline; CIP, ciprofloxacin; K, kanamycin; SYN, quinupristin- dalfopristin; AK, amikacin; TN, tobramycin; TS, trimethoprim- sulfamethoxazole; MUP, mupirocin; SYN, quinupristin-dalfopristin.
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phenotypes were confirmed in 11 isolates, which were distributed in CC22 (4 isolates), CC88 (3 isolates), CC1 (3 isolates), and CC59 (1 isolate). Resistance profiles, virulence genes, and their distribution among different molecular types of PVL-carrying MRSA strains are presented in Table 2.

**DISCUSSION**

This cross-sectional study provided indispensable findings regarding the molecular typing for PVL-positive MRSA strains isolated from patients with wound infections. Primarily, all the PVL-positive S. aureus strains obtained from wound infections were found to carry the mecA gene. Secondly, PVL-positive MRSA strains were broadly resistant to erythromycin, gentamicin, tetracycline, and ciprofloxacin, but they had greater sensitivity to trimethoprim-sulfamethoxazole, mupirocin, and quinupristin-dalfopristin. Thirdly, PVL-positive MRSA strains displayed a high diversity with a predominance of CC30 (29.8%) followed by CC22 (21.3%), CC8 (17%), CC88 (10.6%), CC59 (8.5%), CC1 (7.5%), and CC15 (5.3%). Eventually, our analysis demonstrated a replacement of the ST22-MRSA predominant type with the ST30-MRSA type over a three-year period.

In the present work, all PVL-positive strains were resistant to mupirocin. The high prevalence of PVL-positive MRSA strains was also reported by several investigators in different geographic regions (24–27). It was attained in this study that out of 94 PVL-carrying MRSA clinical isolates, 18.1% were resistant to mupirocin. These findings are higher than the rates reported by earlier studies performed in France (2.2%) (28). Nonetheless, the result is lower than those obtained from previous studies regarding MRSA strains that were isolated from burn patients (28.3%) (21) and ICU patients (30.5%) (7) in Iran. In our study, 12.8% and 5.3% of mupirocin-resistant MRSA isolates exhibited HLMUPR and LLMUPR phenotypes, respectively. In a study performed by Barakat et al. for two years from 2013–2015 in Egypt, the overall prevalence of HLMUPR and LLMUPR was reported as 3.5% and 4.3%, respectively (29). Our study showed that the overall prevalence of MS, iMLSA, and cMLSA phenotypes was 11.7%, 14.9%, and 62.8%, respectively. Similarly, in a study performed in Iran, Rashidi et al. (7) reported 52.6%, 12.6%, and 5.3% prevalence of cMLSA, iMLSA, and MS phenotypes among the MRSA strains isolated from the intensive care unit. In a Canadian study, Lavallée et al. (30) found that 64.7% and 35.3% of isolates indicated iMLSA and cMLSA phenotypes, respectively.

Several investigators indicated that aminoglycoside resistance genes are dominant in S. aureus clinical isolates (7,8,21). In our study, the ant (4')-Ia gene (57.4%) was the most frequent gene followed by aac (6')-le/aph (2') (45.5%) and aph (3')-IIa (39.4%). The findings of a study carried out in Turkey on 50 staphylococci from in-patients also showed that the majority of the isolates carried aac (6')-le/aph (2') (60.5%) followed by ant (4')-Ia (24%) and aph (3')-IIa (8%) (31). The higher rates of aminoglycoside resistance genes could be due to the over usage of aminoglycoside in the treatment of serious staphylococcal infections and horizontal gene transfer among the strains.

In the present study, 12.8% of isolates carried the mupA gene. They were confirmed as HLMUPR MRSA. This finding is lower than the rate reported by Abbasi-Montazeri et al. in Iran (32) (34%) and González-Domínguez et al. in Spain (33) (27.2%) and higher than the rate reported in Korea (1.8%) (34). Generally, variations in resistance genes are expected, since the mentioned genes are subjected to high but variable selective pressure.

It was observed that 10 STs were grouped into seven CCs of known PVL-positive MRSA lineages, which had noted strain diversity in our hospitals. One significant finding in the present work was that, ST30, which is known as the Southwest Pacific or the USA1100 clone, was the most widespread molecular type detected among PVL-carrying MRSA clinical isolates (29.8%). However, our finding is closer to that obtained in a study done by Goudarzi et al., which stated that ST30 is the second most common type among PVL-positive MRSA clinical isolates (21.5%) (8). Similarly, the emergence of the PVL-positive ST30 MRSA lineage was reported in Italy (36) and Brazil (37), which indicated a world-wide distribution of this type. We also observed an increasing trend for ST30, which drastically increased from 9.7% in 2016 to 52.9% in 2018; hence, the rate could increase more in the near future. These data contrast with the findings of a study conducted in Kuwait (9), which reported a significant decrease in the prevalence of ST30-IV-MRSA from 22% in 2006 to 2.9% in 2010. Our previous study indicated that ST30, accounting for 21.5%, was at a high level (8). These findings suggest that ST30 is a dominant type in these infections.

Our analysis indicated that ST22, a global hospital-associated MRSA pandemic clone, which is known as UK-EMRSA-15, was indeed the second most frequent ST. This is in conformity with the results of numerous studies that stated that PVL-positive ST22 MRSA strains have been specifically associated with resistance to gentamicin, trimethoprim, and ciprofloxacin (4,8). In this collection, half of the isolates were resistant to ciprofloxacin (10/20), and 95% (19/20) and 30% (6/20) were resistant to gentamicin and trimethoprim, respectively.

In the present study, CC8 was the third most common CC detected (17% [16/94]), which was assigned to ST8 (75% [12/16]) and ST931 (25% [4/16]). USA300 is a major global epidemic clone that has been noticed for its rapid dissemination within the community and hospitals (10). The present data demonstrate that USA300 accounted for 12.8% of the tested PVL-positive MRSA isolates. The mentioned PVL-positive MRSA lineage with high prevalence of resistance genes has been previously reported in different parts of the world (9,10). The entire HLMUPR strain belongs to ST8 (12.8%). In accordance with the present work, mupirocin resistance in ST8-MRSA isolates was reported previously by several investigators (9,10). ST931 is a single locus variant of ST8, which is genetically related to MRSA USA300. In line with the results obtained from the present study, ST has also been reported in Spain (38) and Portugal (5).

Particularly, ST88 was the fourth molecular type

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detected among PVL-positive MRSA strains. In fact, ST88 is a predominant pvl-positive MRSA clone in Africa (39). We found ST88 in 10.6% of isolates. Similar to the findings in this research, Karbuz et al. from Turkey (12) and Liu et al. from China (24) reported low prevalence rates of ST88 in 1.1% and 4.8% of their MRSA isolates, respectively.

According to the literature, CC59 clones are mostly reported in the Asia-Pacific region (10). In the present survey CC59 isolates (8.5%) exhibited ST59 (67.5% [7/8]) and ST338 (12.5% [1/8]). In a study conducted by Liu et al. (24) on CA-MRSA strains isolated from outpatients with SSTIs, ST59 was reported as the most common lineage accounting for 76.2% (16/21). The ST59 types disseminated in our hospitals carried seb (3 isolates) and sed (4 isolates). The result obtained by Yu et al. in China (13) described that the isolates were distributed in six main lineages, with the majority of CC5 (52.3%), followed by CC7 (11.7%), CC59 (8.6%), CC88 (6.3%), CC398 (4.7%), and CC121 (3.1%). They also revealed higher carriage of seb and sec among CC59 isolates, which could be due to the marker of this lineage. In our study, only one MRSA isolate was identified to be the ST338 type. The low prevalence of ST338 has been reported previously in Taiwan and the southern region of China (14).

In fact, the results of a previous published study in Iran (21) are contrary to those of the present study, in which a low frequency of the ST15 type among our isolates was demonstrated (5.3%). Several studies described various prevalence of ST15. A study conducted in 25 European countries reported ST15 as the second most frequent clone among their isolates (40); while another study conducted in 11 European countries indicated that this clone was less frequent (41). All 5 LLMUPR strains belonged to CC15, similar to what was indicated in our previous study (21).

CC1 was another CC that was detected among our PVL-positive MRSA strains (7.5%), which exhibited ST772 (71.4% [5/7]) and ST1 (28.6% [2/7]). ST772-MRSA-V, also known as the Bengal Bay clone, emerged for the first time in Bangladesh and was reported in many countries, especially Saudi Arabia, UAE, and the Middle East (9,10). In the present study, 5.3% of PVL-positive MRSA strains belonged to this ST type. In regards to the drug susceptibility profile, our results are in accordance with a survey performed by Shore et al. (11), which stated that all the CC1/ST772-MRSA-V isolates exhibited an MDR pattern and carried multiple resistance genes, including: aac (6')-Ie-aph (2'), ant (4')-Ia, and tet(M). Similar to our previous research (8), we demonstrated the presence of ST772 among PVL-carrying MRSA strains. The aforementioned type has a great tendency to disseminate widely; as a result, it might spread through the community rapidly and cause problems in hospitals and health care settings.

In our study, two out of the 94 isolates were ST1. However, multiple ST1-MRSA-IV clones associated with SSTIs have been reported by several investigators (13,14). In conclusion, seven different CCs and 10 ST types found in the present study suggested that wound infection in patients is caused by S. aureus strains harboring different molecular types. It was ultimately indicated that there is a considerable increasing trend for CC30 versus a decreasing trend for CC22. Further measures such as a global surveillance of PVL-positive MRSA strains and nationwide antimicrobial resistance surveillance to detect changes in their clonal composition and dissemination are needed.

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Conflict of interest None to declare.

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