Objectives: Staphylococcus aureus is a nosocomial pathogen that provides a major challenge in the healthcare environment, especially in burns units where patients are particularly susceptible to infections. In this study, we sought to determine molecular types of S. aureus isolates collected from burns patients, based on staphylococcal protein A and coagulase gene polymorphisms.

Methods: Antibiotic susceptibility testing of 89 S. aureus strains isolated from burn wounds of patients was assessed using the Kirby-Bauer disk diffusion method. Strains were characterized by spa typing, coa typing, and resistance and toxin gene profiling.

Results: A total of 12 different spa types were identified with the majority being t790 (18%). Panton-Valentine leucocidin encoding genes were identified in spa types t044 (5.6%), t852 (2.2%) and t008 (2.2%). The most commonly detected antibiotic resistance gene was ant (4')-Ia (60.7%). Ten different coa types were detected and the majority of the tested isolates belonged to coa III (47.2%). All the high-level mupirocin-resistant and low-level mupirocin resistant strains belonged to coa type III.

Conclusion: The present study illustrated that despite the high frequency of coa III and spa t790 types, the genetic background of S. aureus strains in Iranian burns patients was diverse. The findings obtained are valuable in creating awareness of S. aureus infections within burns units.

Introduction

Burns patients are at an increased risk of colonization and subsequent infections by nosocomial pathogens due to the disruption of skin protective barrier and reduction of immune responses, which can lead to poor clinical outcomes and increased morbidity and mortality rates [1]. More than 70% of deaths in burns patients results from infection with nosocomial microorganisms. Based on the literature, many pathogens are responsible for infection in burns patients, but it is well established that Staphylococcus aureus, in particular, methicillin-resistant S. aureus (MRSA) strains, are one of the most common nosocomial pathogens of burn wounds [2,3]. They could originate from the patient or transmitted easily by direct (i.e. contaminated hands or droplet) and indirect contact (i.e airborne infection). Many of the published data in Iran indicates that multidrug-resistant (MDR) bacteremia and wound infections are the most important causes of mortality in burns patients [2,4,5]. Our previous study showed that there is a high rate of infection with MRSA in burns patients, which is a serious threat for the individual, and also a hazard to public health. Infection with MDR MRSA in burns patients may increase the economic burden of the healthcare system, and causes limitations to the therapeutic options available for
treatment [1-4].

A major concern with managing MRSA infections in burns patients is the lack of awareness of the molecular and resistance patterns of MRSA. Various molecular typing techniques have been employed for *S. aureus* isolates include pulsed-field gel electrophoresis, staphylococcal cassette chromosome mecA (SCCmec) typing, the accessory gene regulator (agr) typing, the mec-associated hypervariable region (dru), multilocus sequence typing, coagulase (coa) typing, and staphylococcal protein A (spa) typing [6]. Although the pulsed-field gel electrophoresis technique is a standard method, it is acknowledged that polymerase chain reaction based methods such as coa and spa typing due to its cost-benefit, rapidity and high throughput ability could be an effective method for routine typing of MRSA isolates [7,8].

The coagulase protein is genetically and antigenically divergent and is an important virulence factor for *S. aureus*. Sequence analysis of staphylococcal coagulase (SC) showed 6 regions including a signal sequence, N-terminal D1 region and D2 region, central region, 27-amino acid-repeat region and C-terminal sequence. Ten main types of SC have been described as a result of sequence diversity in the SC region [8-10].

The staphylococcal protein A (spa) gene contains 3 distinct regions including Fc, X and C regions; typing is based on the number of tandem repeats and the sequence variation in region X of the protein A gene [7]. The distribution of spa types of MRSA strains isolated from different geographical areas of the world, show different patterns [11].

In addition, coa typing is also simple to perform, easily interpreted, and requires minimal laboratory skills and equipment, and therefore could be a useful addition to spa typing for genotyping of *S. aureus*, including MRSA strains [8,9]. In Iran, there is very little information about the genetic diversity of *S. aureus* isolated from burns patients, therefore, the present study aimed to determine the resistance pattern, carriage of resistance determinants, and molecular characteristics of MRSA isolates collected from burns patients based on coa and spa gene polymorphism analysis.

### Materials and Methods

#### 1. Sampling and data collection

In the current cross-sectional study, 89 *S. aureus* isolates were investigated from the burn wounds of patients during a 12-month study starting from October, 2016 to November, 2017. The Ethics Committee of Shahid Beheshti University of Medical Sciences (Tehran, Iran) also approved this study (IR. SBMU. MSPREC.1396.878). Swab sticks and scalpel blades were used for sampling burn wounds with pus and hemorrhaging tissues. Specimens were submitted to the laboratory within 4 hours of collection and were processed immediately. Standard biochemical tests, such as growth on mannitol salt agar, colony morphology on blood agar, and coagulase, Dnase, and catalase production assays, were used to identify *S. aureus* isolates. A PCR assay targeting the *S. aureus*-specific *nuc* gene was performed to provide definitive identification of *S. aureus* isolates [1]. *S. aureus* isolates were stored at -70°C in Tryptic Soy Broth (TSB, Merck co., Germany) containing 20% glycerol until molecular analysis.

#### 2. Antibacterial susceptibility testing and MRSA screening

*S. aureus* isolates were tested for *in vitro* susceptibility to ampicillin, amikacin, gentamicin, clindamycin, erythromycin, tetracycline, linezolid, teicoplanin, rifampicin, and quinupristin-dalfopristin (Mast Diagnostics Ltd, Merseyside, UK) by the disk diffusion method, based on the direction of the Clinical and Laboratory Standards Institute (CLSI) guide [12].

The minimum inhibitory concentration (MIC) for vancomycin and mupirocin was determined by the broth microdilution method in accordance with the CLSI recommendation. The MIC cutoff points for vancomycin based on the CLSI criteria were as follows: resistant, ≥ 16 µg/mL; intermediate, 4-8 µg/mL; and susceptible, ≤ 2 µg/mL. Strains were considered to demonstrate low-level mupirocin resistance (LLMUPR) if the MIC value was between 8-256 µg/mL, and high-level mupirocin resistance (HLMUPR) if the MIC value was ≥ 512 µg/mL. MDR isolates are defined by resistance to 3 or more unique antibiotic classes [1,13]. ATCC29213 and ATCC25923 (*S. aureus*) were considered as the reference strains for quality control purposes. For MRSA screening, phenotypic growth was investigated around the cefoxitin disc (30 µg) placed on Mueller-Hinton agar plates (Merck, Germany), containing 4% NaCl. PCR was applied for genotypic amplification of mecA genes [12].

#### 3. DNA preparation

Genomic DNA from 24-hour cultures of *S. aureus* isolates were extracted using the InstaGene Matrix kit (BioRad, Hercules co., CA, USA) based on the manufacturer’s protocols. Briefly, for each sample, lysostaphin (Sigma–Aldrich co., USA) was added at a final concentration of 15µg/mL for cell wall lysis. After extraction, the purity of DNA was assessed using a nanometer.

#### 3.1. Detection of resistance and toxin encoding genes

PCR was performed to determine the presence of resistance (*mupA, mecA, erm(A), vanA, msr(A), mupB, erm(B), vanB, msr(B), erm(C), tet(M), aac(6′)-Ie-aph(2′), aph(3′)-IIIa, and ant(4′)-Ia*) and toxin (*eth, eta, pvl, tst*) encoding genes as described by Rashidi et al [13] and Goudarzi et al [1].
4. Typing of the S. aureus Protein A locus

PCR amplification was carried out with specific primers for spa typing as suggested by Harmsen and colleagues [7]. DNA fragments containing the spa gene were purified using the QIAgen PCR purification kit, and were subjected to DNA sequence analysis and nucleotide sequences on both strands were determined using an ABI Prism 377 automated sequencer (Applied Biosystems, Perkin-Elmer Co., Foster City, CA). Sequence-editing was performed using the Chromas software (Version 1.45, Australia). The edited sequences were assigned to particular spa types according to the guidelines described by a Ridom SpaServer database (http://www.spaserver.ridom.de).

5. Coa typing

Four sets of multiplex PCR reactions were used for assigning SC types (I-X) according to the procedure of Hirose et al [5]. Set A contained primers for identifying SC types I, II, III, IVa, IVb, Va, and VI, while set B contained primers for identifying SC types VII, VIII, and X. Set 3 was used for identifying SC types IX and Vb. SC types IVa and IVb were distinguished using set 4 primers [8].

Results

1. Sampling and antibiotic susceptibility

There were 89 S. aureus strains obtained from burns patients. All the strains under study were confirmed as MRSA due to the presence of the mecA gene as well as phenotypically. The results of antimicrobial susceptibility showed that all the isolates were susceptible to linezolid, teicoplanin, and clindamycin (51.7%), rifampicin (33.7%), and quinupristin-dalfopristin (25.8%).

The MICs of vancomycin showed that 31 (34.8%) isolates had MIC of 0.5 µg/mL, 18 (20.3%) had MIC of 1 µg/mL, 23 (25.8%) had MIC of 2 µg/mL and 17 (19.1%) had MIC of 8 µg/mL. Five different resistant phenotypes were identified among our MRSA isolates. The predominant resistance pattern included resistance to 4 antibiotics (52.8%), followed by 6 antibiotics (15.7%), 8 antibiotics (13.5%), 5 antibiotics (11.3%), and 7 antibiotics (6.7%), simultaneously.

2. Antimicrobial resistance and toxin genes

All MRSA strains in the current study harbored at least 2 antibiotic resistance genes. The most commonly detected antibiotic resistance genes were ant (4’)-Ia (60.7%), aac (6’)-le/aph (2’) (55.1%), aph (3’)-IIa (46.7%), tet(M) (43.8%), erm(A) (29.2%), erm(C) (23.6%), erm(B) (20.2%), msr(B) (18%), msr(A) (13.5%), and mupA (13.5%). vanA, vanB and mupB genes were not detected among tested isolates. Analyzing toxin genes revealed that the most prevalent gene was tst (14; 15.7%), followed by pvl (9; 10.1%), eta (2; 2.2%) and etb (2; 2.2%).

3. Spa typing

A total of 12 different spa genotypes were identified. t790 accounting for 18% (16/89 each) was found to be the predominant spa type, followed by t064 (13.5%; 12), t030 (11.3%; 10), t044 (10.1%; 9), t852 (9%; 8), t223 (7.9%; 7), t421 (6.7%; 6), t008 (6.7%; 6), t019 (6.7%; 6), t021 (4.5%; 4), t005 (3.4%; 3), and t10795 (2.2%; 2). PVL (lukS-lukF)-encoding genes were identified in 5 strains (55.6%, 5/9) with spa types t044, 2 strains with t852 (22.2%, 2/9), and 2 strains with t008 (22.2%, 2/9), while tst was identified among spa types t790 (64.3%, 9/14) and t223 (35.7%, 5/14). Of the 13 LLMUPR strains, 11 strains belonged to t790 (84.6%), and 2 strains (15.4%) to t008. spa types t064 (10 isolates; 83.3%) and t008 (2 isolates; 16.7%) were observed among 12 HLMUPR-MRSA strains.

4. Sc typing

In the present study, 10 different types were detected and the most common coa type was type III (42; 47.2%) with spa types t790 (16; 18%), t064 (10; 11.2%), t030 (10; 11.2%) and t008 (6; 6.8%). The coa type II was observed in 13 isolates (14.6%) with spa types t019 (5; 5.6%), t044 (4; 4.5%) and t021 (4; 4.5%). The coa type VIII with spa types t852 (22.2%, 2/9), while tst was identified among spa types t790 (64.3%, 9/14) and t223 (35.7%, 5/14). Of the 13 LLMUPR strains, 11 strains belonged to t790 (84.6%), and 2 strains (15.4%) to t008. spa types t064 (10 isolates; 83.3%) and t008 (2 isolates; 16.7%) were observed among 12 HLMUPR-MRSA strains.
Table 1. Characteristics of the MRSA strains isolated from burns patients.

| SC type | spa type | Toxin genes (No.%) | Antibiotic resistance profile (No.%) | Antibiotic resistance genes (No.%) | Total n (%) |
|---------|----------|-------------------|-------------------------------------|----------------------------------|-------------|
| I       | t223     | tst (2;50)        | AP, CD, E, AK, GM, T (2;50)         | Meca (4;100), erm(C) (4;100), mrsr(B) (2;50), erm(A) (3;75), ant (4')-la (4;100), aac (6')-le/aph (2') (3;75) | 4 (4.5) |
|         |          |                   | AP, CD, E, AK, GM, RPT (2;50)       |                                  |             |
|         | t852     | pvl (1;33.3)      | AP, CD, E, AK, GM, MYC (2;66.7)     | Meca (3;100), ant (4')-la (3;100), aac (6')-le/aph (2') (3;100), aph (3')-Illa (3;100) | 3 (3.4) |
|         | t005     |                   | AP, CD, E, AK, GM, RP, T (1;50)     | Meca (2;100), erm(A) (2;100), mrsr(B) (1;50), tet(M) (2;100), ant (4')-la (2;100) | 2 (2.2) |
| II      | t019     | eta (2;40)        | AP, GM, CD, E (1;20)                | Meca (5;100), tet(M) (4;80), ant (4')-la (3;60), aac (6')-le/aph (2') (4;80), aph (3')-Illa (2;40) | 5 (5.6) |
|         | t044     | pvl (3;75)        | AP, CD, E, AK, GM (3;75)            | Meca (4;100), tet(M) (3;75), ant (4')-la (4;100), aac (6')-le/aph (2') (3;75), aph (3')-Illa (4;100) | 4 (4.5) |
|         | t021     |                   | AP, E, AK, GM, SYN (2;50)           | Meca (4;100), ant (4')-la (4;100), aac (6')-le/aph (2') (3;75), aph (3')-Illa (2;50), erm(A) (2;50), erm(B) (4;100) | 4 (4.5) |
|         | t790     | tst (9;56.3)      | AP, CD, E, AK, GM, RP, T, MUP (2;20) | Meca (16;100), erm(C) (12;75), mrsr(A) (10;62.5), ant (4')-la (12;75), aac (6')-le/aph (2') (5;80), tet(M) (9;62.5), aph (3')-Illa (9;56.3) | 16 (18) |
|         | t064     |                   | AP, CD, E, AK, GM, RP, T, MUP (8;80) | Meca (10;100), erm(C) (5;50), mrsr(A) (8;80), ant (4')-la (9;90), aac (6')-le/aph (2') (5;50), aph (3')-Illa (3;30), tet(M) (9;90), mupA (10;100) | 10 (11.2) |
|         | t008     | pvl (2;33.3)      | AP, E, GM, T, SYN, MUP (2;20)       | Meca (6;100), mrsr(B) (5;83.3), mrsr(A) (2;33.3), erm(A) (5;83.3), ant (4')-la (4;46.7), aac (6')-le/aph (2') (5;83.3), aph (3')-Illa (4;66.7), mupA (2;33.3) | 6 (6.7) |
|         | t030     | etb (2;20)        | AP, GM CD, E (6;60)                 | Meca (10;100), aac (6')-le/aph (2') (8;80), aph (3')-Illa (6;60), erm(A) (6;60), mrsr(B) (8;80), tet(M) (6;60) | 10 (11.2) |
| IVa     | t0795    |                   | AP, CD, E, AK, GM (2;100)           | Meca (2;100), ant (4')-la (2;100), | 2 (2.2) |
| IVb     | t421     |                   | AP, CD, E, AK, GM (2;100)           | Meca (4;100), erm(B) (3;75), ant (4')-la (4;100), | 4 (4.5) |
| Va      | t005     |                   | AP, CD, E, AK, GM (1;25)            | Meca (1;100), tet(M) (1;100), | 1 (1.1) |
| VI      | t223     | tst (3;100)       | AP, CD, E, AK, GM, T (2;66.7)       | Meca (3;100), aph (3')-Illa (3;100) | 3 (3.4) |
|         | t421     |                   | AP, CD, E, AK, GM (1;33.3)          | Meca (1;100), aac (6')-le/aph (2') (1;100), erm(B) (1;100), | 1 (1.1) |
| VII     | t019     |                   | AP, GM, RP, T (1;100)               | Meca (1;100), erm(B) (1;100), | 1 (1.1) |
| VIII    | t852     | pvl (1;20)        | AP, E, AK, GM, SYN (3;60)           | Meca (5;100), aac (6')-le/aph (2') (4;80), aph (3')-Illa (5;100), erm(B) (4;80), | 5 (5.6) |
|         | t044     | pvl (2;40)        | AP, E, AK, GM, SYN (2;40)           | Meca (5;100), erm(B) (5;80), | 5 (5.6) |
| X       | t064     |                   | AP, E, AK, GM, SYN (2;100)          | Meca (2;100), ant (4')-la (2;100), | 2 (2.2) |
|         | t421     |                   | AP, GM, RP, T (1;100)               | Meca (1;100), ant (4')-la (1;100), | 1 (1.1) |

AP = ampicillin; CD = clindamycin; E = erythromycin; AK = amikacin; GM = gentamicin; MRSA = methicillin-resistant *Staphylococcus aureus*; MUP = mupirocin; RP = rifampicin; SYN = quinupristin-dalfopristin; T = tetracycline.
Discussion

Several studies have revealed different findings of resistance rate of *S. aureus* isolated from burns patients which may be linked to various bacterial detection methods [10]. Although the prevalence rate of mupirocin-resistant *S. aureus* strains depends on various geographic areas and/or patient populations, a relatively high prevalence of mupirocin-resistant *S. aureus* strains (28.1%) was found in this study, isolated from burns patients which may be linked to various bacterial detection methods [10].

In the current study, the second most frequent type was *tst* carriage in *t790* isolates in this study were carrying the *tst* gene (56.3%). LLMUPR was detected in 11 strains with *t790* (12.4%). Although, virulence markers and drug resistance patterns in *t790* isolates may vary, resistance to mupirocin and *tst* carriage in *t790* isolates has been reported by several investigators [1,20].

The present study reported the existence of *t790* as the predominant spa type in 18% of isolates which were all *coa* type III. The low frequency of *t790* in the study was in line with Udo’s study from Kuwait, 2016 [21], and a study conducted by Goudarzi et al [1] who analyzed *S. aureus* strains isolated from burns patients in Iran (13.2%). More than half of *spa* *t790* isolates in this study were carrying the *tst* gene (56.3%). LLMUPR was detected in 11 strains with *t790* (12.4%). Although, virulence markers and drug resistance patterns in *t790* isolates may vary, resistance to mupirocin and *tst* carriage in *t790* isolates has been reported by several investigators [1,20].

In the current study, the second most frequent *spa* type was *t064* (13.5%), distributed in *coa* types III (11.2%) and X (2.3%). All these strains were carrying the *mupA* gene and confirmed as LLMUPR strains. In agreement with our study, mupirocin resistance in *t064* strains has been reported previously from Nigeria [29], Kuwait [21] and Ireland [20].
The third most common spa type was t030 detected in 11.3% of isolates, that all belonged to coa type III. This finding contrasts with our previous study in burns patients that reported t030 as one of the most common spa types amongst the tested isolates (24.5%) [1]. Similarly, the authors of a study of MRSA in Kuwait reported a low prevalence of spa type t030 (2.7%) [21].

Another spa type in our study was t044 (10.1%; 9 isolates), that of those with 5 of those isolates found to be positive for the pvl encoding gene (55.6%). Of 9 isolates with the t030 spa type, 4 isolates belonged to coa type II (44.4%) and 5 isolates to coa type VIII (55.6%). A review performed by Asadollahi and colleagues in 2018, to determine distribution of the most prevalent spa types among clinical isolates of methicillin-resistant and -susceptible S. aureus, indicated that t044, distributed in 11 countries, was one of the most common spa types [11]. This spa type was also previously reported [30].

In agreement with studies performed in Kuwait [31], and Saudi Arabia [32], PVL-positive, multi-resistant spa type t852 isolates (9%) were found in our survey, pointing to their spread in the Gulf Persian Cooperative Council countries.

In this study, the spa type t223 was found in 7.9% of isolates for the first time in Iran. The resistance and virulence profiles of the t223 isolates (PVL-negative, tst-positive) was similar to t223 isolates reported in S. aureus strains isolated from children and parents in the Gaza Strip [33], and also t223 recovered from healthy individuals in Jordan [34], which may suggest the origin of spa type t223.

The t421 isolates was detected in small numbers, similar to Wang et al [35], who revealed that out of 99 MRSA isolates classified as ST239, 92 were ST239-spa t037 and 4 were ST239-spa t421.

In the present study, spa type t008 was detected at an incidence of 6.7%. The majority of the 108 MRSA isolates were resistant to mupirocin, as has been reported in t008 isolates obtained in Kuwait, the USA, and many European countries [20,21].

The spa type t019 was observed among 6 isolates (6.7%) with 5 isolates belonging to SC II and 1 isolate to SC VII. Two isolates harbored the eta encoding gene and belonged to SC II. spa type t019 has also been reported in the United States, and certain Asian and European countries like Egypt, Japan, Poland, and Taiwan [20]. The findings of the present study are in line with a multicenter study conducted during a 6-year period in 17 countries, that reported a low frequency of spa type t019 (3.6%) among tested isolates [36].

In line with our findings that showed the existence of spa type t021 in 4.5% of isolates, several researchers in Lebanon, Ireland, Romania, and Portugal reported a low frequency of the t021 among MRSA clinical isolates in comparison to other spa types, but not to the same extent [20].

t0195 with SC type IVa accounted for 2.2% of all strains. This was supported by Boswihi’s report from Kuwait [21] that spa type t0195, was one of the spa types associated with the ST772-V, Bengal Bay clone, and was detected in small numbers. The Bengal Bay clone was previously reported in Italy, UK, Kuwait, Malaysia, Saudi Arabia, and UAE [21,35,37].

### Conclusion

There were 10 different SCs and the 12 spa types in the present study suggesting infection in burns patients is caused by S. aureus strains harboring different variants of the coa and spa genes. The present study showed the prevalence of coa type III and spa type t790 with a high level of MDR in the burns unit, which highlights the special attention for systematic surveillance and antimicrobial stewardship programs for S. aureus infections within burns patients.

### Conflicts of Interest

The authors declare that they have no conflicts of interest.

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