A study of *Staphylococcus aureus* nasal carriage, antibacterial resistance and virulence factor encoding genes in a tertiary care hospital, Kayseri, Turkey

M Oguzkaya-Artan, C Artan[1], Z Baykan[2], C Sakalar[3], A Turan[3], H Aksu[3]

Deparment of Medical Services and Tecniques, Halil Bayraktar Health Services Vocational College, Erciyes University, [1]Department of Medical Microbiology, Kayseri Training and Research Hospital, Chest Diseases Clinic, [2]Departments of Medical Education and [3]Medical Biology, Erciyes University, Medical School, Kayseri, Turkey

Abstract

Aim: This study was to determine the virulence encoding genes, and the antibiotic resistance patterns of the *Staphylococcus aureus* isolates, which were isolated from the nasal samples of chest clinic patients.

Materials and Methods: The nasal samples of the in-patients (431) and out-patients (1857) in Kayseri Training and Research Hospital's Chest Clinic, Kayseri, Turkey, were cultured on CHROMagar (Biolife, Italiana) *S. aureus*, and subcultured on sheep blood agar for the isolation of *S. aureus*. Disc diffusion method was used for antimicrobial susceptibility testing. The occurrence of the staphylococcal virulence encoding genes (enterotoksins [*sea, seb, sec, see, seg, seh, sei, sej*], fibronectin-binding proteins A, B [*fnbA, fnbB*], toxic shock syndrome toxin-1 [*tst*]) were detected by polymerase chain reaction.

Results: Forty-five of the 55 (81.8%) *S. aureus* isolates from inpatients, and 319 (90.6%) isolates from tested 352 out-patient's isolates were suspected to all the antibiotics tested. methicillin-resistant *S. aureus* (MRSA) was detected in 1.2% of *S. aureus* isolates. Rifampin, trimethoprim-sulfamethoxazole, clindamycin, erythromycin, gentamicin resistance rates were 1.2%, 1.7%, 2.0%, 8.8%, and 1.2%, respectively. The isolates were susceptible to teicoplanin and vancomycin. The genes most frequently found were *tst* (92.7%), *seg* (85.8%), *sea* (83.6%), *fnbA* (70.9%). There was no statistical significance detected between MRSA and mecA-negative *S. aureus* isolates in encoding genes distribution ($P > 0.05$).

Conclusion: Our results show that virulence factor encoding genes were prevalent in patients with *S. aureus* carriage, whereas antibiotic resistance was low. These virulence determinants may increase the risk for subsequent invasive infections in carriers.

Key words: Carriage, *Staphylococcus aureus*, virulence factors

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Introduction

Staphylococcus aureus is a dangerous pathogen that causes a variety of severe diseases. Nasal colonization is the cause of recurrent staphylococcal infections. The host tissue colonization by *S. aureus* is an important factor in disease pathogenesis. *S. aureus* expresses fibronectin-binding protein A (*fnb*A), which mediates the adhesion to

Address for correspondence:
Dr. Muge Oguzkaya-Artan,
Halil Bayraktar Health Services Vocational College, Erciyes University, Kayseri, Turkey.
E-mail: martan38@gmail.com

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fibrinogen, elastin and fibronectin. In addition, *S. aureus* isolates associated with invasive diseases, including endocarditis, primary septic arthritis and/or osteomyelitis were more likely to have both genes *fnbA* and *fnbB*. The pathogenity of *S. aureus* results from its ability to produce specific toxins and hydrolytic enzymes. The virulence of *S. aureus* is defined by a large repertoire of virulence factors, among which secreted toxins play a preeminent role.[1,2] Staphylococcal enterotoxins (SEs), toxic shock syndrome toxin-1 (*tsst-1*), and exfoliative toxins produced by some *S. aureus* isolates causes staphylococcal food poisoning, staphylococcal toxic shock syndrome (tss), staphylococcal scalded skin syndrome.[3-5] The arginine catabolic element (ACME) prevalent, especially in methicillin-resistant *S. aureus* (MRSA) isolates of sequence type 8 and staphylococcal cassette *mec* type IVa (USA300) and evidence suggest that ACME enhances the ability of ST8-MRSA-IVa to grow and survive on its host. ACME has been identified in a small number of isolates belonging to other MRSA clones but is widespread among coagulase-negative staphylococci.[6] A mobile genetic element–encoded gene, *sasX* has been attributed to the acquisition of virulence determinants by horizontal gene transfer, and the researchers demonstrate that *sasX* has a key role in MRSA colonization and pathogenesis, substantially enhancing nasal colonization, lung disease and abscess formation and promoting mechanisms of immune evasion.[7]

Antimicrobial resistance of bacteria is a growing global problem. In low-resource countries, where extensive empirical use of antibiotics is common, the extent and the impact of this phenomenon tend to be even larger than in industrialized countries. The increase of MRSA in hospital settings and in the community is worrisome. Increasing resistance to antibiotics and the growing prevalence of MRSA can be connected to antibiotic overuse in primary care and requires to be addressed promptly.[5,6,8]

In this study, we detected: (i) The occurrence of the virulence genes: *tsst-1*, SEs (*sea, seb, sec, see, seh, sei, sej, seg*) and *fnbA* and *fnbB*. (ii) The antibiotic resistance patterns of the inpatients and outpatients in a chest clinic who colonized with *S. aureus*.

Materials and Methods

This study is a part of a Erciyes University Scientific Research Project called "Determination of *S. aureus* nasal carriage, investigation of risk factors, and virulence traits among out-patients and in-patients in Kayseri Training and Research Hospital's Chest Clinic"(no: 4674). In this study, data about the virulence factors and antibiotic resistance patterns of *S. aureus* isolates obtained from in-patients and out-patients are given. This study was conducted in January from 1 to 31 2014. The nasal swabs were taken from 1857 out-patients and 431 in-patients. The aim of this study was explained to all of the volunteers, and they signed a written consent form afterwards.

Nasal swabs were obtained from the anterior nares of the volunteers by rotating a sterile swab of Stuart agar gel transport medium. The microbiological study was realized in Erciyes University Halil Bayraktar Health Services Vocational College's Microbiology Laboratory (Kayseri, Turkey). The swabs were immediately streaked on CHROMagar *S. aureus* (Biolife, Italiana). After incubation for 24 h at 37°C suspected violent colonies were isolated and subcultured on to 5% sheep blood agar plates. After incubation for 24 h at 37°C suspected colonies were confirmed to be *S. aureus* by standard biochemical techniques and conventional methods (colony morphology, Gram stain, catalase activity, tube coagulase test). All *S. aureus* isolates were tested for antibiotic resistance. Test for methicillin-resistance was performed by the Kirby–Bauer disc diffusion method, using cefoxitin disc on Muller Hinton agar with 24 h incubation at 35.8°C. We also used CHROMagar MRSA for detecting of MRSA. The tested antimicrobial agents were: Cefoxitin (30 μg), gentamicin (10 μg), trimethoprim-sulfamethoxazole (TMP-SMX, 1.25/23.75 μg), erythromycin (15 μg), clindamycin (2 μg), teicoplanin (30 μg), vancomycin (30 μg), rifampin (30 μg) (all Bioanalyze, Turkey). Erythromycin-induced clindamycin resistance was detected by disk approximation test (D-test). Results were interpreted according to the criteria of Clinical and Laboratory Standards Institute 2007 guidelines.[9] The reference strain *S. aureus* ATCC 29213 was used as internal quality control.

All *S. aureus* isolates from inpatients (55 isolates) and 51 isolates from out-patients, which were taken by systematic sampling were studied for virulence factors determination. The *S. aureus* isolates from out-patients were ranked by their number and the samples were selected by systematic sampling for virulence factors determination (352/50 = 7, taken in 7th). The determination of species specific parts of the genes was performed with oligonucleotide primers, listed in Table 1. DNA of the isolates was prepared with the PureLink™ Genomic DNA Kits (Invitrogen, Carlsbad, USA) according to the instructions of the manufacturer with the use of lysozyme (20 mg/ml, Sigma) in the cell lysis step. The polymerase chain reaction (PCR) reaction mixture consists of PCR master mix (Thermo Scientific, ×2, Califronia, USA), forward primer (0.4 um), reverse primer (0.4 um), template DNA (~30 ng) and nuclease-free water (thermo scientific). The DNA amplification was carried out with the following cycling conditions: Initial denaturation (95°C, 3 min), 35 cycles of denaturation (95°C, 30 sec), annealing (49–51°C, 30 sec), extension (72°C, 1 min) and final extension (72°C, 5 min). Amplicons obtained by PCR amplification were

run in 2% agarose gel at 70 V for 90–120 minutes and visualized in ChemiDoc MP Imaging System (BIORAD, USA). 100 bp DNA marker was used (abm, Canada). The presence of the following genes were studied: *Tst*, SEs (*sea, seb, sec, see, seh, sei, sej, seg*) and *fnb*A and *fnbB* listed in Table 1.

This study was planned and performed in accordance with the Helsinki declaration and was approved by the Ethics Committee of the Erciyes University Medical Faculty. Work permit from the Kayseri Training and Research Hospital's Scientific Committee was also taken.

The data are shown in numbers and frequencies. The evaluation of the difference in categorical variants made by Chi-squared Fisher's exact test. A $P < 0.05$ indicated a significant difference statistically.

Results

In this study, 2283 patients were tested for nasal *S. aureus* carriage. The 352 outpatients ($n = 1852$, 19.0%), and the 55 in-patients ($n = 431$, 12.8%) were positive for carriage. The demographic characteristics of the patients were shown in Table 2. All the *S. aureus* isolates were tested for antibiotic resistance ($n = 407$). In Table 3, the antibiotic resistance patterns of these isolates were shown.

Inpatients

Only erythromycin resistance in three isolates; cefoxitin, clindamycin and erythromycin resistant one isolate; rifampin, cefoxitin, clindamycin, erythromycin and gentamycin resistant one isolate; rifampin, cefoxitin, clindamycin, erythromycin, gentamycin two isolates; rifampin, clindamycin, erythromycin and gentamycin one isolate; clindamycin, erythromycin resistance found in one isolate and two isolates were positive for D-test. The resistance rates for the following antibiotics were: Rifampin, cefoxitin, trimethoprim-sulfamethoxazole, clindamycin, erythromycin, gentamicin, vancomycin, teicoplanin, 9.1%, 9.1%, 0, 9.1%, 18.2%, 9.1%, 3.6%, 0, 0, respectively.

In outpatients

Only trimethoprim-sulfamethoxazole resistance in 7 isolates; erythromycin resistant in 24 isolates; clindamycin, erythromycin resistant one isolate; trimethoprim-sulfamethoxazole, erythromycin and D-test positivity in one isolate and there 22 D-test positivity. The resistance rates for the following antibiotics were: Rifampin, cefoxitin, trimethoprim-sulfamethoxazole, clindamycin, erythromycin, gentamicin, vancomycin, teicoplanin, 0, 0, 2%, 0.9%, 7.4%, 0, 0, 0 respectively.

Of all the isolates ($n = 407$) the antibiotic resistance rates were rifampin, cefoxitin, trimethoprim-sulfamethoxazole, clindamycin, erythromycin, gentamicin, vancomycin, teicoplanin; 1.2%, 1.2%, 1.7%, 2%, 8.8%, 1.2%, 0, 0 respectively [Figure 1].

Table 1: Oligonucleotide primers used for the amplification of the toxin gens and *fnb A*, *fnb B*

Target gen	Oligonucleotide sequence 5'-3'	Size of amplified product (bp)	Reference
Sea	GGTTATCAATGTGCGGGTGG	102	Mehrotra *et al.*[10]
	CGGCACTTTTTTCTCTTCGG		
seb	GTATGGTGGTGTAACTGAGC	164	Mehrotra *et al.*[10]
	CCAATAGTGACGAGTTAGG		
sec	AGATGAAGTAGTTGATGTGTATGG	451	Mehrotra *et al.*[10]
	CACACTTTTAGAATCAACCG		
see	AGGTTTTTTCACAGGTCATCC	278	Mehrotra *et al.*[10]
	CTTTTTTTTCTTCGGTCAATC		
seg	CGTCTCCACCTGTTGAAGG	327	Monday and Bohach[11]
	CCAAGTGATTGTCTATTGTCG		
seh	CAACTGCGATTTAGCTCAG	360	Monday and Bohach[11]
	GTCGAATGAGTAATCTCTAGG		
sei	CAACTCGAATTTTCAACAGGTAC	465	Monday and Bohach[11]
	CAGGCAGTCCATCTCCTG		
sej	CATCAGAACTGTTGTTCCGCTAG	142	Casagrande Proietti *et al.*[12]
	CTGAATTTTACCATCAAAGGTAC		
fnbA	GCGGAGATCAAAGACAA	1279	Yapar and Oğuz[13]
	CCATCTATAGCTGTGTGG		
fnbB	GGAGAAGGAATTAAGGCG	812	Yapar and Oğuz[13]
	GCCGTCGCCTTGAGCGT		
tst	ACCCCTGTTCCCTTATCAATC	326	Mehrotra *et al.*[10]
	TTTTCAGTATTTGTAACGGCC		

fnbA=Fibronectin-binding proteins A; *fnbB*=Fibronectin-binding proteins B; *tst*=Toxic shock syndrome toxin-1

Table 2: The sociodemographic characteristics of the patients

Features (n=2283)	n (%)	
	Outpatients (n=1852)	Inpatients (n=431)
Age groups		
<65	1545 (83.4)	235 (54.5)
65 and over	307 (16.6)	196 (45.5)
Sex		
Male	809 (43.7)	177 (41.1)
Female	1043 (56.3)	254 (58.9)
Having a chronic disease		
Yes	1070 (57.8)	311 (72.2)
No	782 (42.2)	120 (27.8)
Recent antibiotic usage		
No	1670 (90.2)	291 (67.5)
Yes	182 (9.8)	140 (32.5)

Table 3: Antibiotic resistance patterns observed in *Staphylococcus aureus* isolates

	n (%)			P
	Staphylococcus aureus isolates from outpatients (n=352)	*Staphylococcus aureus* isolates from inpatients (n=55)	Total (n=407)*	
Rifampin				
Sensitive	352 (100.0)	50 (90.9)	402 (98.8)	<0.001
Resistant	-	5 (9.1)	5 (1.2)	
Cefoxitin				
Sensitive	352 (100.0)	50 (90.9)	402 (98.8)	<0.001
Resistant	-	5 (9.1)	5 (1.2)	
Trimethoprim-sulfamethoxazole				
Sensitive	345 (98.0)	55 (100.0)	400 (98.3)	0.601
Resistant	7 (2.0)	-	7 (1.7)	
Clindamycin				
Sensitive	349 (99.1)	50 (90.9)	399 (98.0)	0.002
Resistant	3 (0.9)	5 (9.1)	6 (2.0)	
Erythromycin				
Sensitive	326 (92.6)	45 (81.8)	371 (91.2)	0.018
Resistant	26 (7.4)	10 (18.2)	36 (8.8)	
Gentamicin				
Sensitive	352 (100.0)	50 (90.9)	402 (98.8)	<0.001
Resistant	-	5 (9.1)	5 (1.2)	
D-test pozitivity				
Negative	329 (93.5)	53 (96.4)	382 (93.9)	0.554
Positive	23 (6.5)	2 (3.6)	25 (6.1)	

*All the isolates were sensitive to vancomycin, teicoplanin

The studied isolates virulence factor encoding genes [Figures 2 and 3] and comparison of inpatients and outpatient's isolates distribution were shown in Table 4.

Table 4: Comparison of virulence factors among inpatients and outpatients *S. aureus* isolates

	n (%)			P
	S. aureus isolates from outpatients (n=51)	S. aureus isolates from inpatients (n=55)	n=106	
seb				
Negative	8 (15.7)	35 (63.6)	43 (40.6)	<0.001
Positive	43 (84.3)	20 (36.4)	63 (59.4)	
sec				
Negative	12 (23.5)	23 (41.8)	35 (33.0)	0.063
Positive	39 (76.5)	32 (58.2)	71 (67.0)	
seg				
Negative	1 (2.0)	8 (14.5)	9 (8.5)	0.032
Positive	50 (98.0)	47 (85.5)	97 (91.5)	
sei				
Negative	24 (47.1)	34 (61.8)	58 (54.7)	0.172
Positive	27 (52.9)	21 (38.2)	48 (45.3)	
sea				
Negative	1 (2.0)	9 (16.4)	10 (9.4)	0.017
Positive	50 (98.0)	46 (83.6)	96 (90.6)	
sej				
Negative	46 (90.2)	26 (47.3)	72 (67.9)	<0.001
Positive	5 (9.8)	29 (52.7)	34 (32.1)	
seh				
Negative	47 (92.2)	51 (92.7)	98 (92.5)	1.000
Positive	4 (7.8)	4 (7.3)	8 (7.5)	
see				
Negative	43 (84.3)	40 (72.7)	83 (78.3)	0.165
Positive	8 (15.7)	15 (27.3)	23 (21.7)	
fnbA				
Negative	11 (21.6)	16 (29.1)	27 (25.5)	0.504
Positive	40 (78.4)	39 (70.9)	79 (74.5)	
fnbB				
Negative	37 (72.5)	51 (92.7)	88 (73.0)	0.009
Positive	14 (27.5)	4 (7.3)	18 (17.0)	
tst				
Negative	4 (7.8)	4 (7.3)	8 (7.5)	1.000
Positive	47 (92.2)	51 (92.7)	98 (92.5)	

S. aureus=*Staphylococcus aureus*; *fnbA*=Fibronectin-binding proteins A; *fnbB*=Fibronectin-binding proteins B; *tst*=Toxic shock syndrome toxin-1

There was no statistically significant difference detected between MRSA and mecA-negative *S. aureus* (MSSA) isolates in encoding genes distribution ($P > 0.05$).

All the 106 isolates had multiple virulence genes (100%; min: 2, max: 8). Two virulence genes in one isolate (0.9%), three virulence genes in four isolates (3.8%) and follows as: Four in seven isolates (6.6%), five in 19 (17.9%), six in 37 (34.9%), seven in 28 (26.4%), eight in 10 isolates (9.4%).

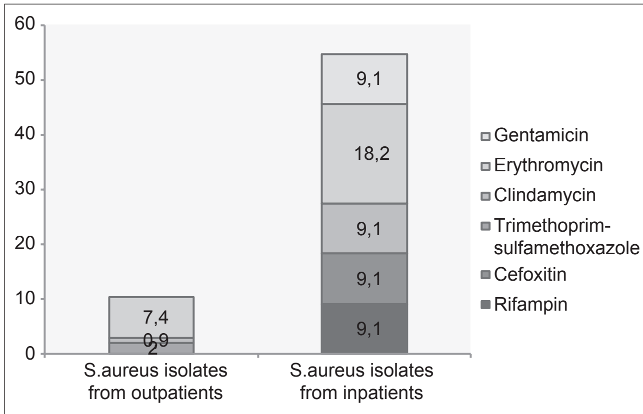

Figure 1: Percentage of isolates to various antibiotics

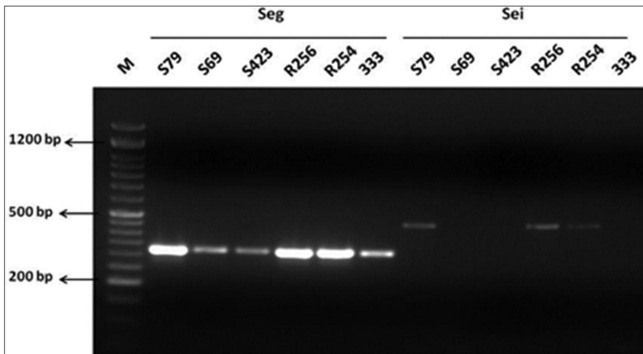

Figure 2: Agarose gel electrophoresis of polymerase chain reaction (PCR) products of *Staphylococcus aureus* isolates. Genomic regions containing the target genes (*Seg, Sei*) were amplified by PCR and PCR products of 327 bp and 465 bp were obtained. Amplified DNA fragments were run in 2% agarose gel and visualized. Presence of PCR products was confirmed. Each number represents a *S. aureus* isolate. Arrows represent the band sizes of the 100 bp DNA marker. M = DNA Marker, bp = base pair

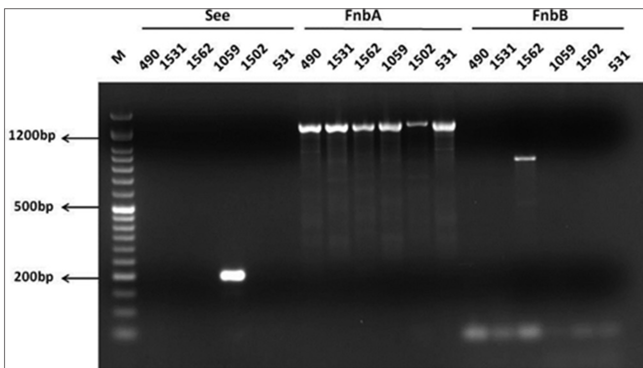

Figure 3: Agarose gel electrophoresis of polymerase chain reaction (PCR) products of *S. aureus* isolates. Genomic regions containing the target genes (*See, fnbA, fnbB*) were amplified by PCR and PCR products of 209 bp, 1279 bp and 812 bp were obtained. Amplified DNA fragments were run in 2% agarose gel and visualized. Presence of PCR products was confirmed. Each number represents a *S. aureus* isolate. Arrows represent the band sizes of the 100 bp DNA marker. M = DNA Marker, bp = base pair

Discussion

In adults, *S. aureus* demonstrates a niche preference for the anterior nares.[14] The nasal carriage of *S. aureus* represents a risk factor for subsequent invasive infection of patients and also interpatient transmission. For both clinicians and antibiotic policy makers, it is important to identify populations at risk for infections with resistant microorganisms and to better understand the epidemiology of antibiotic resistance.[15] In our study, the MRSA carriage rate was 1.2% in *S. aureus* isolates, all of them were from inpatients. There were a statistically significant differences between outpatients and inpatients (<0.001). There were also statistically difference between resistance patterns of rifampin, clindamycin, erythromycin and gentamicin in outpatients and inpatients (<0.001, 0.002, 0.018, <0.001 respectively). A large number of studies have been done all over the world. Scribel *et al*.[16] from Brasil found no MRSA from the patients at a primary healthcare unit. Sfeir *et al*.[17] found 1.5% cefoxitin, gentamycin; 21.4% erythromycin; 16.8% clindamycin resistance and all strains found susceptible to vancomycin and rifampin in 1526 outpatients. Choi *et al*.[18] found 2.5% gentamicin, erythromycin, clindamycin; 1.2% rifampicin; 1.2% oxacillin resistance in health adults. Netsyetayeva *et al*.[5] found erythromycin-induced clindamycin resistance 33.3% in MRSA isolates, and 5.6% in MSSA isolates. They found erythromycin-induced clindamycin resistance rate significantly higher among MRSA isolates than MSSA isolates ($P < 0.05$). From Turkey Cıtak *et al*.[19] found 25.7% MRSA carriage in in patients and 27.6% from out patients. Their group were smaller than ours. In patients 191, outpatients 123 patients. They found no statistically significant difference in MRSA carriage in these groups ($P > 0.05$). In our study it is pleasing to see the antibiotic resistance was lower than the studies stated. Even Versporten *et al*.[20] validated 2011 total national whole scale antibiotic-use data of six southern and eastern European countries and regions. According to European Surveillance of Antimicrobial Consumption project Turkey had the highest antibiotic use in Europe, and they also mentioned that Turkish government already published a Rational Drug Use National Action Plan 2013–2017. The study showed that in our country the most used antibiotics were β-lactam antibacterial, penicillins, and the second group was cephalosporins. Especially, the combinations of penicillins, including β-lactamase inhibitors is the most used drugs. In this study, we were used the antibiotic discs which were used in tertiary care referral center's antibiogram schema for *S. aureus* isolates.

Our patient group was specific, patients who have upper or lower respiratory tract diseases welcomed in this clinic. MRSA was found only in patients, outpatients had no MRSA, especially erythromycin, and clindamycin resistance was important. D-test positivity was 6.1%. Nestyetayeva

et al.[5] found 33.3% D-test positivity. Whenever clindamycin is intended to be used for *S. aureus* infections, D-test should be performed.

According to the results of the present study all the 106 isolates had multiple virulence genes (100%; min: 2, max: 8). The genes most frequently found were *tst* (92.7%), *seg* (85.8%), *sea* (83.6%), *fnbA* (70.9%). Demir *et al*.[3] showed that 79 isolates (65.8%) were positive for one or more virulence genes. Tekeli *et al*.[21] showed virulence genes in 86% isolates of *S. aureus* from blood cultures of patients in Ankara, Turkey. These different results may be due to geographical differences or different ecological origins of the isolates. Demir *et al*.[3] found 14.2% harbored isolates in *S. aureus* isolates from Mustafa Kemal University Hospital, Turkey. Nashev *et al*.[4] found only two positivity for *tst* in 44 *S. aureus* isolates. Lozano *et al*.[6] found *tst* in 28.3% carriers of *S. aureus* from Spain. Nashev *et al*.[4] from nasal carriers found that *seg* and *sei* is the most prevalent whereas Schaumburg *et al*.[22] found *sea* (34.4%), *seg* (33.1%), *sei* (33.1%) the most prevalent genes in carriers. Yapar *et al*.[13] studied 50 *S. aureus* isolates from child carriers and found *fnbA* (28%), and *fnbB* (10%). Nashev *et al*.[4] were reported as 100% for *fnbA*, 40% for *fnbB* in 16 healthy carriers. Nestyetayeva *et al*.[5] detected *fnbA* in 59% of *S. aureus* isolates of healthy Ukrainian adults. Zmantar *et al*.[23] focusing on isolates from soft tissue infections the rate of *fnbA* was reported as 76.1%.

There was no statistically significant difference between MRSA and MSSA isolates among gene distribution in our study. Clindamycin resistance was 14% in *seb* negative isolates, in *seb* positives the resistance rate was 1.6% (Fisher's Chi-square, $P = 0.017$). Clindamycin resistance was 12.1% in *sei* negative isolates, in *sei* positives the resistance was not observed (Fisher's Chi-square, $P = 0.015$). Erytrhromycin resistance was 22.4% in *sei* negative isolates while in *sei* positives the rate was 4.2% (Fisher's Chi-square, $P = 0.010$). Yılmaz *et al*.[24] showed that the isolates harbored with sea had multiple antibiotic resistances, especially MRSA (85.7%).

In our study we found that most of the *S. aureus* isolates were susceptible to all antimicrobial agents. However, high prevalence of virulence factor encoding genes were detected. To gain knowledge of *S. aureus* isolates circulating among carriers as well as the capacity of these isolates to produce virulence factor encoding genes, and antibiotic susceptibility patterns in order to understand the complex evolution of *S. aureus* and MRSA infections.

Conclusion

It is important for primary care physicians to know the antibiotic susceptibility patterns of *S. aureus* isolates, which are circulating among carriers in their region as it shows differences among regions and also virulence factors which the isolates posed for the empirical therapy.

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