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Original Article

Investigation of the Presence of *mecA* and *mecC* Genes and Antibiotic Resistance Profiles in Methicillin-Resistant *Staphylococcus aureus* Strains Isolated from Clinical Samples

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ABSTRACT

Objective: Methicillin-resistant *Staphylococcus aureus* (MRSA) is widely regarded as one of the most frequent bacteria, causing a wide range of infections in humans and animals. MRSA is associated with significant morbidity and mortality in many parts of the world. The aim of this study was to determine the molecular identification, antibiotic resistance profiles, and presence of *mec*A and *mec*C genes of MRSA strains isolated from clinical specimens at Siirt Training and Research Hospital.

Methods: In our study, we isolated 20 coagulase positive *S. aureus* strains from patients admitted to our hospital between July 2020-July 2021. Antibiotic susceptibility tests were carried out on the bacteria that were isolated and identified by culturing various clinical samples. The *mec*A and *mec*C genes were then examined by polymerase chain reaction (PCR) using the universal primers *mec*A-F-*mec*A-R and *mec*C-F-*mec*C-R following MRSA had been identified in the cultures from the clinical samples. To identify the isolated *S. aureus* strains by 16S ribosomal ribonucleic acid (rRNA) gene analysis, polymerase chain reaction (PCR) amplification was conducted using universal primers 68f and 518r.

Results: We isolated and identified 20 coagulase positive *S. aureus* strains from patients admitted to our hospital. According to the 16S rRNA gene sequences, the strains isolated in this this study, exhibited high similarity (99%-100%) to the 16S rRNA genes of *S. aureus* strains listed in GenBank (https://www.ncbi.nlm.nih.gov/genbank/). While the *mec*A gene was found in 18 of 20 strains, none of the strains had the *mec*C gene. A high percentage of MRSA was found to be penicillin resistant (75%). In conclusion, 20 MRSA strains were isolated from various clinical specimens. Eighteen of these strains were also identified molecularly by 16S rRNA sequence analysis.

Conclusion: The results of this study confirmed the significance of the 16S rRNA, *mec*A, and *mec*C genes in MRSA identification and highlighted the increasing frequency of MRSA in Türkiye

Keywords: MRSA, 16S Rrna, mecA gene, mecC gene

INTRODUCTION

S. aureus is a gram-positive bacterium that causes numerous complex infections, including skin and soft tissue infections, pneumonia, meningitis, osteomyelitis, bone and joint infections, and endocarditis.[1,2] Methicillin-resistant *S. aureus* (MRSA) strains were found to be associated with significant morbidity and mortality in many regions of the world.[3] A leading pathogen of hospital-acquired (HA-MRSA) and community-acquired (CA-MRSA) infections are MRSAs. In addition, the antimicrobial resistance, infectivity, and virulence factors of MRSA make it a big threat to human health worldwide.[4] The emergence of CA-MRSA and HA-MRSA strains today has made MRSA one of the most dangerous pathogens in the world.[5]

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The *mec*A gene, a component of the mobile genetic element Staphylococcal cassette chromosome (SCC) mec (SCCmec), causes methicillin resistance.[4,6] Penicillin-binding protein 2a (PBP2a) is encoded by the *mec*A gene. In comparison to other penicillin-binding proteins, PBP2a's active site has a low affinity for β-lactam antibiotics. In this way, *S. aureus* manages to survive treatment with these antibiotics.[7,8] PBP2a catalyzes the transpeptidation reaction necessary for peptidoglycan crosslinking, enabling the synthesis of cell wall even when antibiotics are present. Resistance to all β-lactam antibiotics is provided in addition to methicillin, due to the inability to interact with the β-lactam moieties of PBP2a encoded by *mec*A.[8,9]

In 2011, researchers discovered the mecALGA251, which is a new *mec*A gene homologue found in both human and dairy cattle isolates.[10] The International Working Group on the Classification of SCC elements has proposed renaming the mecALGA251 gene to *mecC*.[11] The *mec*C positive MRSA isolates have been reported to belong to strains commonly found in cattle (CC130, CC1943, and sequence type (ST) 425), indicating the presence of a zoonotic MRSA reservoir.[10] In many European countries, *mec*C MRSA was found in other host species such as livestock, wildlife, and domestic animals.[2] In humans, a total of 12 691 individuals in Germany (a prevalence of 0.1%) have been observed to have 11 *mec*C-positive CC130 isolates.[12] Methicillin-resistant *S. aureus* with the *mec*C gene has also been discovered in sheep, suggesting that this could be another reservoir.[13] *mec*C MRSA has appeared to be rare in humans so far. There are, however, some interesting geographical differences in prevalence. The recent rise in prevalence in Denmark has prompted researchers to focus their attention on *mec*C MRSA.[14,15]

MAIN POINTS

- The most prevalent bacteria that cause numerous infections in both humans and animals are methicillin-resistant *Staphylococcus aureus* (MRSA) strains.
- Twenty MRSA strains were isolated and identified from various clinical samples.
- The isolated strains displayed a high degree of similarity (99%-100%) to the 16S rRNA genes of *S. aureus* strains deposited in GenBank, as determined by the 16S rRNA gene sequences.
- While the *mec*A gene was detected in 18 of 20 strains, none of the strains had the mecC gene.
- A high percentage of MRSA was found to be penicillin resistant (75%).

The frequency of MRSA varies from country to country and even from 1 hospital division to the next. Whereas the infection rate in Scandinavia is only 1%, it can reach up to 80% in countries like Italy and Greece.[16] Methicillin resistance rates were reported as 43%, 40%, and 35%, respectively, in an antibiotic resistance study that examined the rate of methicillin resistance among *S. aureus* isolates in our country between 2003 and 2005.[17] The antibiotic resistance rates of bacteria isolated from catheter and endotracheal aspirate (ETS) samples of patients receiving mechanical ventilation in the intensive care units of Siirt Training and Research Hospital between January 2018 and November 2019 were recently determined by Acer and Özüdoğru.[18] Methicillin resistance was observed in 82.9% (n = 35) of coagulase-negative Staphylococcus isolated from catheter specimens and 45% (n = 20) of *S. aureus* isolated from ETS. Detection and identification of MRSA in clinical microbiology laboratories are important for appropriate treatment selection and obtaining epidemiological data. For this reason, it is necessary to reduce *S. aureus* identification to the gene level in health services and to do so in a shorter time. Identification of MRSA and detection of resistance antibiotics to be administered to the patient are critical for preventing disease spread. Therefore, in this study, we aimed to determine the molecular identification, antibiotic resistance profiles, and presence of *mec*A and *mec*C genes of MRSA strains isolated from clinical specimens at Siirt Training and Research Hospital.

METHODS

Sample Collection

In this study, we isolated 20 MRSA strains from various clinical samples (blood, urine, wound, sputum, abscess, tissue, etc.) in the Siirt Training and Research Hospital Microbiology Laboratory between July 2020 and July 2021.

Isolation and Identification of Methicillin-Resistant *Staphylococcus aureus*

Samples from hospitalized and non-hospitalized patients were inoculated on Eosin Methylene Blue, 5% sheep blood agar (COS; bioMerieux), chocolate agar, and McConckcey (MCK; bioMerieux) media and incubated at 35-37°C for 18-24 hours. Blood cultures were evaluated in the BacT/Alert 3D (bioMérieux, France) automated blood culture system. Gram staining was performed on the colonies in which bacterial growth was detected in the culture samples, which were evaluated after incubation. Coagulase and catalase tests were performed on bacteria evaluated as Gram-positive cocci. The VITEK 2 automated system (bioMérieux, France) was used to identify bacterial strains and determine their antibiotic susceptibility. As a control strain, *S. aureus* ATTC-29213 standard strain was used.

Antibiotic Susceptibility Test

The Clinical Laboratory Standard Institute (CLSI) recommended Kirby–Bauer disk diffusion method and the VITEK 2 automated system (bioMérieux, France) were used to evaluate the antibiotic susceptibility of all isolates to different antibiotics. An inoculum was formed from positive MRSA colonies transferred in normal saline solution, creating a 0.5 McFarland physiological standard turbidity.[19] The following antibiotic discs were used: erythromycin (15 µg), clindamycin (2 µg), ciprofloxacin (5 µg), tetracycline (30 µg), nitrofurantoin (10 µg), vancomycin (30 µg), gentamicin (10 µg), penicillin (10 µg), linezolid (30 µg), teicoplanin (30 µg), rifampicin (5 µg), and trimethoprim–sulfamethoxazol (25 µg). Antimicrobial susceptibility was calculated and evaluated using CLSI guidelines after the diameters of inhibition zones were measured.

Cefoxitin Disc-Diffusion Method for Methicillin-Resistant *Staphylococcus aureus* Detection

In accordance with CLSI guidance, the modified Kirby–Bauer disk diffusion method was performed to assess the susceptibility of *S. aureus* isolates to cefoxitin (30 g). These *S. aureus* strains were classified as MRSA after they proved to be cefoxitin resistant.[19,20]

Molecular Characterization

16S Ribosomal RNA Analyses

Deoksiribonükleik asit (DNA) isolation of the samples was done by using the PureLink® Genomic DNA Kits. Gene regions targeted for the identification of species were amplified using universal primer pairs 68f-TNANAC ATGCAAGTCGRRCG-518r WTTACCGCGGCTGCTGG in the PCR study. PCR mixture (25 µL) contained 1.5 mM MgCl2, 0.2 mM dNTP mix, 1 µL each primer (10 mM), 0.25 µL Taq DNA Polymerase, 2.5 µL Taq DNA Polymerase buffer, 13.75 µL molecular grade water, and 3 µL DNA template. A Kyratec thermocycler polymerase chain reaction (PCR) system was used for PCR amplification, which was programmed as follows: initial denaturation at 95°C for 5 minutes, then 40 cycles of denaturation at 95°C for 45 seconds, annealing at 57°C for 45 seconds, and extension at 72°C for 1 minute, followed by a final extension at 72°C for 5 minutes. The products of PCR amplification were run for 90 minutes at 100 volts on a 1.5% agarose gel with 1× TAE buffer. Ethidium bromide (1 µL of a concentration of 500 µg/mL) was used to visualize it under ultra violet (UV) light. After PCR for samples, a single band on an agarose gel indicated that the PCR process was successful. The MAGBIO "HighPrep TM PCR Cleaning System" (AC-60005) purification kit, which was acquired and purified in accordance with the kit instructions, was used to clean the single-band samples. The final PCR products obtained were sent to Medsantek (İstanbul) company for Sanger Sequencing.

Bioinformatics Analysis

Raw sequence data (Standard Flowgram Format files) were preprocessed with free molecular biology software (GeneStudio). Contig Express was used to assemble the sequences (Vector NTI, Thermo Fisher Scientific, Waltham, MA, USA). Gaps and missing data in the sequences were removed during the processing process. The partial 16S rRNA gene sequences of the isolates were compared to those in the National Center for Biotechnology Information (NCBI) database using Basic Local Alignment Search Tool (BLAST). The phylogenetic tree was drawn according to the neighbor-joining method using Molecular Evolutionary Genetics (MEGA) 11 version.[21,22] The maximum composite likelihood method was used to calculate the evolutionary distances.[23]

Investigation of the Presence of the *mec*A and *mec*C Genes

Isolation of DNA in samples for *mec*A and *mec*C genes analysis of bacteria was conducted with EurX GeneMATRIX Bacterial & Yeast DNA isolation kit (EurX Ltd., Gdansk, Poland). To control the amount and purity of the DNA obtained after DNA isolation, spectrophotometer measurements were taken with the Thermo Scientific Nanodrop 2000 (USA) device. The PCR study used primers *mec*A-F 5′-CCTAGTAAAGCTCCGGAA-3-*mec*A-R 5′-CTAGTC-CATTCGGTCCA-3′ and *mec*C-F: 5′-TGTTGTAGCAATGTTCACAC 3′- *mec*C-R: 5′-CAAGCAC TTAATATCAACGC-3′ to amplify specific gene regions.[24] PCR mix of 35 µL was prepared as follows: 1.5 mM $MgCl_2$, 0.2 mM dNTP mix, 0.3 µM for each primer, 2 U Taq DNA Polymerase, and 3 µL template DNA. PCR amplification and the amplification products were run on an agarose gel as mentioned above, and their images were taken in UV light using ethidium bromide. Solis Biodyne (Estonia) FIREPol® DNA Polymerase Taq polymerase enzyme was used in the PCR reaction. Obtaining a single band on the agarose gel indicated that the PCR process was successful.

Statistical Analysis

To evaluate the data, the Statistical Package for the Social Sciences (SPSS) statistical package program (International Business Machiness (IBM) Corporation version 22.0 (IBM Corp.; Armonk, NY, USA) was used. Data were presented as mean and standard deviation for groups. The statistical significance level was accepted as $p < 0.05$.

Ethics Approval

This study was approved by Siirt University's Non-Invasive Clinical Research Ethics Committee (Date: October 13, 2020, Number: 2020/01.01).

RESULTS

16S Ribosomal RNA Analyses

Twenty bacterial isolates identified as MRSA isolated from various clinical specimens were evaluated (Figure 1). Eighteen strains with *mec*A gene were selected for molecular characterization. About 500 bp nucleotides of 16S rRNA genes were determined for *S. aureus* strains by 16S rRNA sequencing analysis (Figure 2). The sequences were submitted to GenBank (https://www.ncbi.nlm.nih.gov/genbank/) after analysis in the Basic Local Alignment Search Tool (BLAST) program. The sequences were assigned the following accession numbers: OK624658, OK624659, OK624660, OK624661, OK624662, OK624663, OK624664, OK624665, OK624666, OK624667, OK624668, OK624669, OK624670, OK624671, OK624672, OK624673, OK624674, OK624675. The BLAST results of *S. aureus* strains are given in Table 1. The obtained sequences were compared with the existing sequences in the NCBI database. The BLAST results showed that the 16S rRNA sequences of the *S. aureus* strains in our study were 99% to 100% similar to the *S. aureus* 16S rRNA genes available in GenBank. Using the sequencing data, the phylogenetic tree was drawn according to the neighbor-joining method with the Molecular Evolutionary Genetics Analysis (MEGA) 11 version (Figure 3).

Presence of the *mec*A and *mec*C Genes

To investigate *mec*A and *mec*C genes in *S. aureus* strains, after PCR amplification of desired gene regions with appropriate primers, the amplification products were run on 1.5% agarose gel and then photographed under UV light with ethidium bromide. Figure 4 shows the agarose gel image of the *mec*A gene after PCR. As seen in the figure, a single band was obtained in the gel, indicating the successful of PCR process. In contrast to the *mec*A gene, no band was found for the *mec*C gene in agarose gel electrophoresis after PCR. This shows that the strains do not contain the *mec*C gene.

Antibiotic Resistance of Methicillin-Resistant *Staphylococcus aureus*

Resistance and sensitive status of MRSA strains isolated from various clinical samples to various antibiotics is given in Table 2. Eight strains were resistant to 3 or more antibiotics. Significant differences were observed between the antibiotic resistance/sensitivity responses of the strains used in the study (Table 2). While all strains were resistant to cefoxitin, all strains were susceptible to nitrofurantoin, vancomycin, linezolid, and teicoplanin. When the study results were compared, the results for cefoxitin, nitrofurantoin, vancomycin, linezolid, and teicoplanin were statistically significant ($P < 0.01$). When the other antibiotics used in the study were taken into account, different results were observed. The rates of resistant strains for erythromycin were found to be 20%, while these rates were 15% for clindamycin, 25% for ciprofloxacin, 35% for tetracycline, 15% for gentamicin, 75% for penicillin, 25% for rifampin, and 5% for trimethoprim/sulfamethoxazole (Table 2).

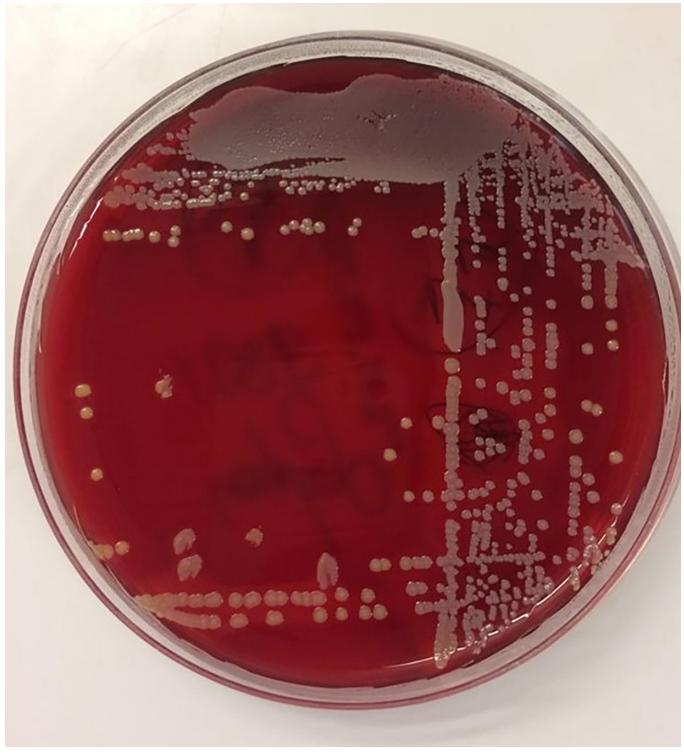


Figure 1. Colony image on blood agar of one of the purely isolated methicillin-resistant *Staphylococcus aureus* strains.

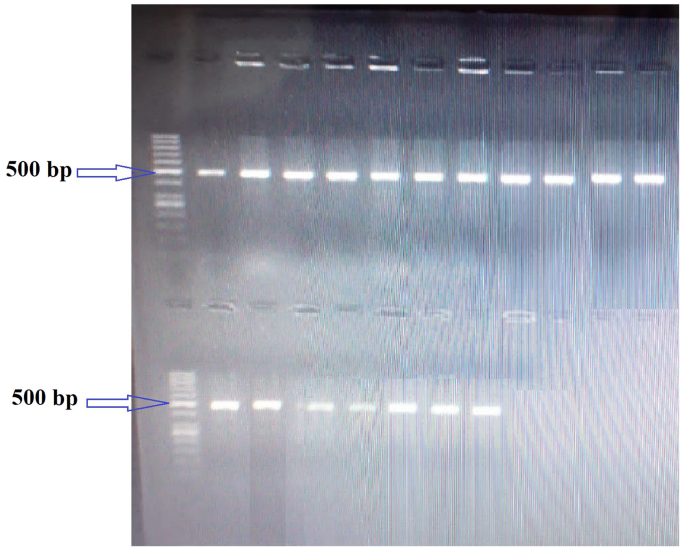


Figure 2. Agarose gel images of polymerase chain reaction-amplified 16S rRNA gene.

Table 1. Phylogenetic Affiliation of Methicillin-resistant *Staphylococcus aureus* Isolates from Various Clinical Samples

Strains	Isolation Source	Closest Sequence GenBank ID	Similarity (%)	GenBank Accession Number
SIU1	Blood culture	*Staphylococcus aureus* strain NBRC 100910	99.78	NR_113956
SIU3	ETA (respiratory secretion) culture	*Staphylococcus aureus* strain ATCC 12600	100	NR_115606
SIU4	Blood culture	*Staphylococcus aureus* subsp. anaerobius strain MVF-7	99.56	NR_036828
SIU5	Wound culture	*Staphylococcus aureus* strain NBRC 100910	99.35	NR_113956
SIU6	Nose culture	*Staphylococcus aureus* strain NBRC 100910	99.56	NR_113956
SIU7	Urine culture	*Staphylococcus aureus* strain S33 R	99.14	NR_037007
SIU8	Blood culture	*Staphylococcus aureus* strain ATCC 12600	99.56	NR_115606
SIU9	Ear culture	*Staphylococcus aureus* strain NBRC 100910	99.56	NR_113956
SIU10	Tissue culture	*Staphylococcus aureus* subsp. anaerobius strain MVF-7	99.56	NR_036828
SIU11	Wound culture	*Staphylococcus aureus* strain S33 R	99.78	NR_037007
SIU12	Blood culture	*Staphylococcus aureus* strain NBRC 100910	99.56	NR_113956
SIU13	Wound culture	*Staphylococcus aureus* subsp. anaerobius strain MVF-7	99.93	NR_036828
SIU15	Urine culture	*Staphylococcus aureus* strain NBRC 100910	99.06	NR_113956
SIU16	Wound culture	*Staphylococcus aureus* strain NBRC 100910	99.57	NR_113956
SIU17	Vaginal culture	*Staphylococcus aureus* strain ATCC 12600	93.88	NR_115606
SIU18	Wound culture	*Staphylococcus aureus* strain NBRC 100910	99.13	NR_113956
SIU19	Urine culture	*Staphylococcus aureus* strain S33 R	99.14	NR_037007
SIU20	Nose culture	*Staphylococcus aureus* subsp. anaerobius strain MVF-7	99.56	NR_036828

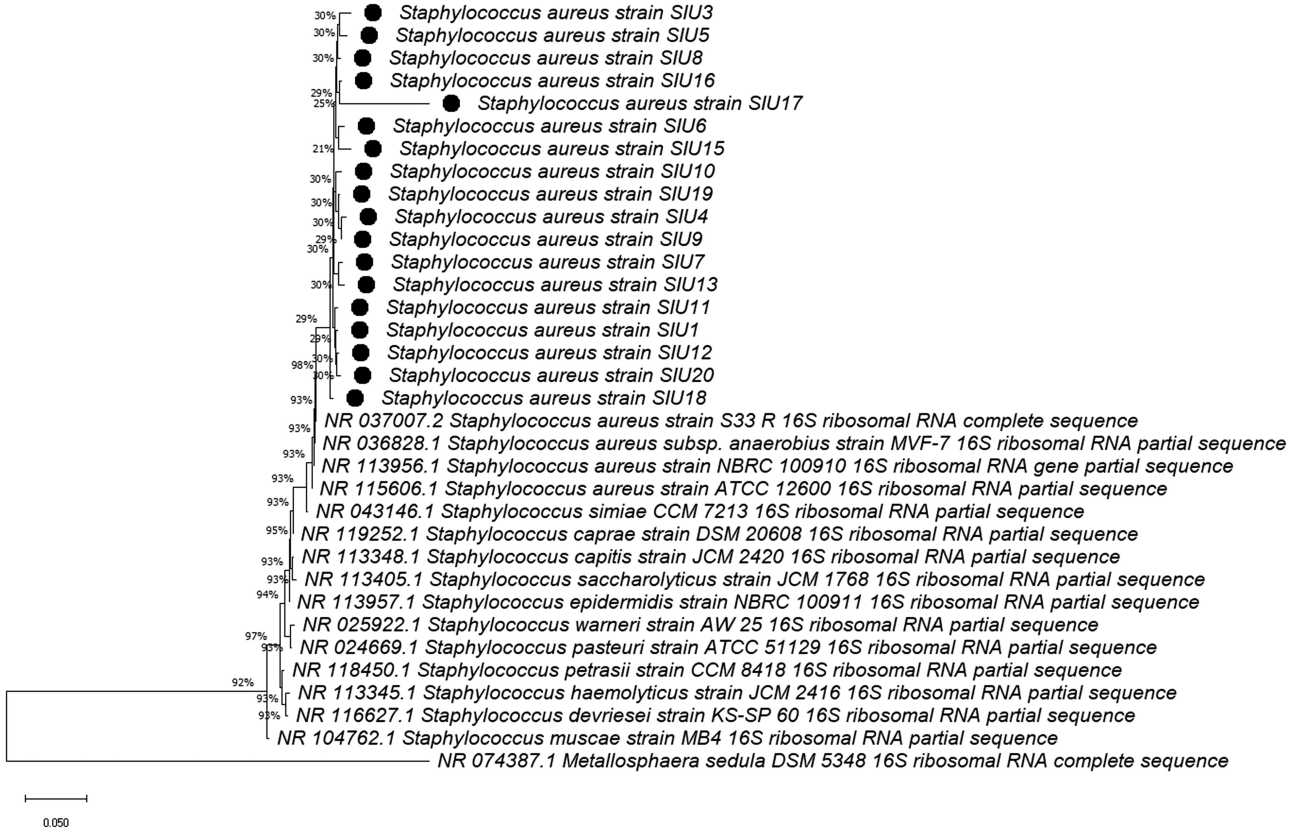


Figure 3. Phylogenetic tree constructed using the neighbor-joining method based on 16S rRNA gene sequences, showing the relationship between isolated *Staphylococcus aureus* strains and closely related species. *Metaallosphaera sedula* DSM 5348 was used as the outgroup.

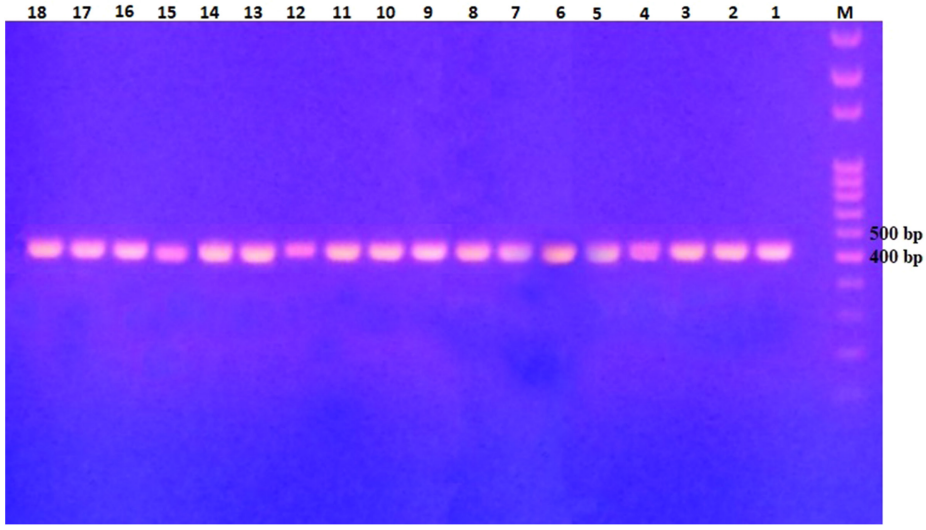


Figure 4. Agarose gel images of polymerase chain reaction-amplified *mec*A gene (M; DNA marker, 1; SIU1, 2; SIU3, 3; SIU4, 4; SIU5, 5; SIU6, 6; SIU7, 7; SIU8, 8; SIU9, 9; SIU10, 10; SIU11, 11; SIU12, 12; SIU13, 13; SIU15, 14; SIU16, 15; SIU17, 16; SIU18, 17; SIU19, 18; SIU20).

DISCUSSION

Methicillin-resistant *Staphylococcus aureus* has spread all over the world in recent years and has become highly endemic in many geographic regions.[25] Multidrug resistance (MDR) in MRSA limits treatment options, making it an important pathogen associated with nosocomial infections.[2] Some researchers suggested that, because of the changing pattern of antibiotic resistance in *S. aureus* and the prevalence of MDR in MRSAs, resistance patterns should be evaluated on a regular basis and antibiotic therapy should be guided by susceptibility testing.[26] In this respect, accurate and rapid identification of MRSA is critical in diagnosing the prognosis of *S. aureus* infections.[27]

Table 2. Resistance and Sensitive Rates of Methicillin-Resistant *Staphylococcus aureus* Strains Isolated from Various Clinical Samples to Various Antibiotics

Antibiyotics	R (%)	S (%)	*P*
Cefoxitin	20 (100)	0 (0)	***P* < 0.001**
Erythromycin	4 (20)	16 (80)	.168
Clindamycin	3 (15)	17 (85)	.134
Ciprofloxacin	5 (25)	15 (75)	.197
Tetracycline	7 (35)	13 (65)	.239
Nitrofurantoin	0 (0)	20 (100)	***P* < 0.001**
Vancomycin	0 (0)	20 (100)	***P* < 0.001**
Gentamicin	3 (15)	17 (85)	.134
Penicillin	15 (75)	5 (25)	.197
Linezolid	0 (0)	20 (100)	***P* < 0.001**
Teicoplanin	0 (0)	20 (100)	***P* < 0.001**
Rifampin	5 (25)	15 (75)	.197
TMP/SXT	1 (5)	19 (95)	**.050**

R, resistant; S, susceptible; TMP/SXT, trimethoprim/sulfamethoxazole.

The cefoxitin disk diffusion method or the oxacillin agar dilution method is used to detect MRSA isolates. Oxacillin keeps its activity longer during storage than methicillin and is more likely to detect strains that are hetero-resistant. Contrarily, tests utilizing cefoxitin are more accurate and reproducible than those using oxacillin as a *mec*A gene stimulator. The cefoxitin disk screen test is also recommended by CLSI. Detection of the *mec*A gene by PCR, on the other hand, is the gold standard method.[2,28,29] In this study, the *mec*A gene was amplified by PCR in 20 *S. aureus* strains that were found to be resistant to cefoxitin by the cefoxitin disc diffusion method. After PCR amplification obtained by using *mec*A primer pairs, the PCR product was run on agarose gel electrophoresis and the *mec*A gene was detected in 18 strains of them. The *mec*A gene was found in 90% (18) of the isolated MRSA strains in current study. The results of this assay were highly correlated with those of conventional disk diffusion susceptibility tests.

In southwest England, *mec*C was first reported to isolate from a bulk tank milk sample.[30] Following the discovery of MRSA with the *mec*C gene, numerous nations have begun investigating the origins and epidemiology of these isolates. *mec*C genes were found in 14 different local and wild animal species from 13 different European countries in a previous study.[31] *mec*C positive *S. aureus* isolates have also been detected in humans.[14] In humans, MRSA strains with the *mec*C gene are less common. However, it may be a public health issue because phenotypic and

genotypic tests lack the ability to detect this gene completely.[32] Only a few studies have examined the *mec*C gene in MRSA strains isolated from people in Türkiye.[2,33] The *mec*C gene hasn't been found in 20 isolated MRSA strains in our study, which was similar to studies in Türkiye. In a recent study in Türkiye conducted by Uçar et al.,[34] similar to our study, it was reported that while the *mec*A gene region was detected in all 68 MRSA strains, no *mec*C gene region was detected in any of the strains.

In our study, it was determined that all MRSA strains were susceptible to vancomycin, nitrofurantoin, linezolid, and teicoplanin while all strains were resistant to cefoxitin. It has also been reported in previous studies that MRSA strains are susceptible to vancomycin, a glycopeptide.[35,36] All MRSA strains in our study were susceptible to the glycopeptides vancomycin and teicoplanin. They may therefore be utilized as the preferable drug for treating MDR-MRSA infections. However, routine testing and frequent monitoring of vancomycin sensitivity are required. Use of vancomycin should be limited to preserve its value. It should be administered only in those cases where there is a clear need. The screening test and MIC determinations are advised for early diagnosis of imminent resistance and monitoring the response to therapy, even though all isolates were determined to be vancomycin -susceptible.[35]

In the current study, a high percentage (75%) of MRSA was found to be resistant to penicillin. In previous studies, Gaire et al.,[20] and Pandey et al.[37] reported similar results. In a recent study, Minami et al.[38] found penicillin susceptibility in 12 of 17 MRSA strains. Their genetic analysis revealed that the IS 1182 mobile genetic element was inserted into the promoter region of the *mec*A gene in class B SCC*mec*. Therefore, they suggested that the *mec*A system was nonfunctional for these strains. Similarly, in another recent study, Harrison et al.[39] revealed that penicillin susceptibility in MRSA strains is due to mutations in the *mec*A promoter region that reduces *mec*A (PBP2a) expression. The reason for the 25% penicillin susceptibility seen in MRSA strains in our study may be due to mutations occurring in the *mec*A promoter region, as stated in these studies.

The 16S rRNA gene sequence has widely been used for identifying bacterial species. The 16S rRNA genes of bacteria typically have 9 "hypervariable regions," which show significant sequence diversity among bacterial species and can be utilized to identify them.[25,40,41] In our study, 18 of 20 strains identified as MRSA by conventional methods were also identified by 16S rRNA sequence analysis.

There were some limitations of our study; first, the number of samples sent to the microbiology-culture laboratory decreased due to the coronavirus disease 2019 pandemic. Secondly, due to the small number of samples, MRSA cases tended to decrease compared to previous years. Finally, there has been a general decrease in the number of patients admitted to the hospital due to the pandemic.

In conclusion, 20 MRSA strains were isolated from various clinical specimens by conventional methods. 16S rRNA sequences revealed that the sequences of the strains isolated in this study were 99% to 100% similar to the *S. aureus* 16S rRNA genes available in GenBank. A high percentage of MRSA was found to be penicillin resistant (75%). The *mec*A gene was detected in 90% (18/20) of the isolated MRSA strains in this study. The results of this assay were highly correlated with those of conventional disk diffusion susceptibility tests. Unlike the *mec*A gene, no *mec*C gene was detected in any of the isolates.

The results of this study confirmed the relevance of employing molecular techniques as an epidemiological tool and highlighted the significance of the mecA, mecC, and 16S rRNA genes in MRSA detection. We also highlighted the rising frequency of MRSA in Türkiye. This is the first study on the molecular characterization of MRSAs in the Siirt region.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Siirt University (Date: October 13, 2020, Number: 2020/01.01).

Informed Consent: Written informed consent was obtained from hospital management where the study was performed.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – O.A., B.I.; Design – O.A., B.I.; Supervision – O.A., B.I.; Resources – B.I., O.A.; Materials – M.E., O.A.; Data Collection and/or Processing – O.A., M.E.; Analysis and/or Interpretation – O.A., B.I.; Literature Search – O.A., B.I., M.E.; Writing Manuscript – O.A.; Critical Review – O.A.; B.I.

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Declaration of Interests: The authors declare that they have no competing interests.

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