

Distribution of antibiotic resistance genes in *Staphylococcus epidermidis* clinical isolates

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ABSTRACT

Background: *Staphylococcus epidermidis* is an opportunistic pathogen associated with hospital- acquired infections and increasing antibiotic resistance. This study aimed to determine the prevalence of *Staphylococcus epidermidis* in selected clinical samples and to investigate the distribution of antibiotic resistance genes in isolates.

Methods: A total of 164 clinical samples were collected from Al-Ramadi Teaching Hospital and Al-Ramadi Maternity & Children Teaching Hospital in Al- Anbar, Iraq, during the period from August 2021 to December 2021. Samples were obtained from wounds, kidney stones, eyes, and skin, representing common clinical sources of *Staphylococcus* infections. Identification of isolates was performed using standard biochemical tests and the VITEK 2 Compact system with GP- ID cards. Polymerase chain reaction (PCR) was used to detect selected antibiotic resistance genes (*mecA* , *ermA* , *ermC* , *tetK* , and *aacA- D*) associated with resistance to commonly used antibiotics.

Results: Out of 164 clinical samples, 24 (14.63%) were positive for *Staphylococcus epidermidis* , while 140 (85.37%) were negative for *Staphylococcus epidermidis* . PCR analysis revealed that the *mecA* gene was detected in 24/24 isolates (100%), the *ermC* gene in 24/24 isolates (100%), the *ermA* gene in 3/24 isolates (12.5%), the *tetK* gene in 24/24 isolates (100%), and the *aacA-D* gene in 10/24 isolates (41.7%).

Conclusion: The findings indicate a high prevalence of antibiotic resistance genes among *Staphylococcus epidermidis* isolates in the studied hospitals. However, the study is limited by the relatively small number of *S. epidermidis* isolates and its restriction to specific healthcare facilities in Al- Anbar, Iraq. Further studies with larger and more diverse sample populations are recommended to better understand the epidemiology of antibiotic resistance in *S. epidermidis*.

EDITORIAL NOTE

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INTRODUCTION

Staphylococcus epidermidis is a normal component of the human microbiota and is frequently found on the skin and mucous membranes. *S. epidermidis* is able to establish a lifelong commensal relationship with the host by binding to tissue surface molecules via specific adhesins, starting early in human life [1]. The most frequently isolated member of the coagulase-negative staphylococci group is *S. epidermidis*. This group can be distinguished from *Staphylococcus aureus* by its inability to produce coagulase. Due to its prominence as an important cause of nosocomial infections, *S. epidermidis* has received considerable attention. *S. epidermidis* was long thought to be comparatively harmless; however, it is now widely acknowledged to be a pathogen. *S. epidermidis* is considered an opportunistic pathogen because a susceptible host may allow it to change from a usual resident of human skin to an infectious agent [2]. The most prevalent pathogenic isolate from human epithelial cells is *S. epidermidis*, which mostly colonizes the axilla, head, and nose. Immunocompromised patients have been linked to it as a cause of nosocomial infections, which is a problem made worse by the rising antibiotic resistance of hospital-isolated pathogens [3]. *S. epidermidis* strains typically show resistance to several antibiotic classes including tetracyclines, aminoglycosides, cephalosporins, fluoroquinolones, penicillins, and macrolides. Therefore, hospitals face a serious problem with resistant *S. epidermidis* strains [4]. Therefore, the present study aimed to investigate the distribution of antibiotic resistance genes in clinical isolates of *Staphylococcus epidermidis*.

METHODS

Sample collection

From August 2021 to December 2021, 164 samples were collected from two hospitals in Al-Anbar: Al-Ramadi Teaching Hospital and Al-Ramadi Maternity & Children Teaching Hospital. Samples were collected from patients with suspected bacterial infections using sterile swabs or sterile containers depending on the type of specimen. All specimens were collected under aseptic conditions by trained medical personnel and immediately transported to the microbiology laboratory for analysis. These included wound, kidney stone, eye, and skin samples.

Isolation and identification of *S. epidermidis*

Each specimen was cultured on 5% sheep blood agar and incubated at 37 °C under aerobic conditions. After 24 hours, the bacteria that grew on blood agar were transferred to mannitol salt agar to isolate *staphylococci*. Morphological and biochemical identification of *S. epidermidis*, including Gram staining, catalase and coagulase tests, and hemolysis testing, was performed and the isolates were presumptively identified as *S. epidermidis*. Standard positive and negative control strains were used during biochemical testing to ensure the accuracy and reliability of the identification procedures. Automated identification was also performed using the VITEK 2 Compact system with GP-ID cards.

DNA extraction and quantification

Genomic DNA was extracted from bacterial cultures using DNA extraction kits (Geneaid, Korea) according to the manufacturer's instructions. DNA concentration and purity were assessed using a NanoDrop spectrophotometer. DNA was then kept at -20 °C to avoid degradation. Molecular weight markers (100 bp), 1% agarose gel, and 1× TAE buffer were prepared in accordance with the manufacturer's instructions.

PCR reaction mixtures

Amplification of *mecA*, *ermA*, *ermC*, *tetK*, and *aacA-D* was performed using standard PCR with *mecA* primers F-AAAATCGATGGTAAAGGTTGGC and R-AGTTCTGCAGTACCGGATTTGC, *ermA* primers F-AAGCGGTAAACCCCTCTGA and R-TTCGCAATCCCTTCTCAAC, *ermC* primers F-AATCGTCAATTCTGCATGT and R-TAATCGTGAATACGGGTTTG, *tetK* primers F-

GTAGCGACAATAGGTAATAGT and R- GTAGTGACAATAAACCTCCTA, and *aacA-D* primers F-TAATCCAAGAGCAATAAGGGC and R-GCCACACTATCATAACCACTA [4]. A 20 µL reaction mixture was prepared according to the manufacturer's instructions (BIONEER, Korea). The conditions for PCR thermal cycling included an initial denaturation step at 94 °C for 4 min followed by 35 cycles of denaturation at 94 °C for 1 min, annealing at 58 °C for 45 sec, and extension at 72 °C for 1 min, with a final extension step at 72 °C for 7 min. The annealing temperature of 58 °C was selected based on previously published protocols and primer characteristics reported earlier [4].

RESULTS

Isolation of *S. epidermidis*

Among 164 samples, 24 (14.63%) were identified as *S. epidermidis*, and 140 (85.37%) were not identified as *S. epidermidis*. Among the 24 *S. epidermidis* isolates, the distribution was as follows: wounds (n=2), kidney stones (n=3), eyes (n=5), and skin (n=14).

Identification of *S. epidermidis*

The isolates were presumptively identified as *S. epidermidis*, and specific biochemical tests were performed for additional verification. All isolates were Gram-positive and catalase-positive, while coagulase and hemolysis tests were negative. To confirm that the isolates belonged to *S. epidermidis*, the automated VITEK 2 Compact system was used with GP-ID cards containing 64 biochemical tests. The results demonstrated that all 24 (100%) isolates were confirmed as *S. epidermidis*. This technique enabled rapid bacterial identification.

DNA concentration and purity

A NanoDrop spectrophotometer was used to measure the extracted DNA concentration and purity; the results showed that the DNA concentration ranged from 44.11 to 207.26 ng/µL, while the purity of extracted DNA from the studied isolates ranged from 1.717 to 2.014.

Molecular Detection of Antibiotic Resistance Genes

The *mecA* gene was detected in all 24 *S. epidermidis* isolates (24/24, 100%). The results showed that 24/24 isolates (100%) had the *tetK* gene. The *ermC* gene was detected in all *S. epidermidis* isolates (24/24, 100%), while the *ermA* gene was detected in 3/24 (12.5%) isolates. The results revealed that 10/24 (41.7%) isolates had *aacA-D* gene.

DISCUSSION

The prevalence of *mecA* in our study was higher than that reported in another study in Iran, where the gene was present at 92.2% [5]. Another study in Iran reported a prevalence rate of 80% [6]. The *mecA* gene encodes the protein PBP2A ([penicillin-binding protein 2A](#)), a transpeptidase that helps form the [bacterial cell wall](#). PBP2A possesses a lower affinity for beta-lactam antibiotics, allowing cell wall synthesis to proceed in their presence [7]. The presence of the *mecA* gene may contribute to beta-lactam resistance.

Another study in South Africa reported that the *tetK* gene was present in 80% of isolates [1]. The *tetK* gene encodes a tetracycline efflux protein that expels tetracycline from the cell cytoplasm [8]. This high *tetK* gene prevalence in our study may be associated with resistance to tetracycline antibiotics.

In China, the prevalence of the *ermC* and *ermA* genes was close to the results of our study; the prevalences were *ermC* (94.8%) and *ermA* (6.2%) [9]. In Tunisia, a study by Bouchami *et al.*, [10] found that the percentages of the *ermC* and *ermA* genes were 53% and 32%, respectively. The *ermC* and *ermA* genes are associated with ribosomal modification, which is associated with constitutive macrolide- lincosamide- streptogramin B (cMLS_B) resistance and high- level resistance to MLS_B antibiotics [1]. The presence of the *ermC* and *ermA* genes may contribute to antibiotic resistance in *S. epidermidis* isolates.

A similar study in Iraq reported an identical prevalence of 41.7% of *aacA-D* gene [11]. In another study in Japan, the prevalence of the gene was close to the results of our study (42.5%) [12]. A

study in Belgium reported a gene prevalence of 53% in *S. epidermidis* [13]. The *aacA-D* gene encodes aminoglycoside-modifying enzymes. The *aacA-D* gene is associated with resistance to aminoglycoside antibiotics [14]. This may contribute to aminoglycoside resistance in some *S. epidermidis* isolates.

CONFLICT OF INTEREST

There is no conflict of interest to be declared by authors regarding publicizing this research.

AUTHOR CONTRIBUTIONS

Both the authors contributed equally to conceptualization, execution and reporting of this study.

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