

Original Article

Molecular identification of *erm A* and *erm B*, *erm C* genes in methicillin-resistant *Staphylococcus aureus* isolates from burns patients and their association with multidrug resistance

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Abstract



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Methicillin-resistant *Staphylococcus aureus* (MRSA) is a significant hospital-acquired pathogen, particularly concerning in burn patients due to its multidrug resistance. This study aimed to assess the antibiotic sensitivity profile and identify the presence of *erm* genes (*ermA*, *ermB*, and *ermC*) associated with erythromycin resistance in MRSA isolates from burn patients. A total of 80 *S. aureus* isolates were collected from burn cases, with initial diagnoses performed using conventional culture and microscopic methods. MRSA isolates were confirmed using chromogenic agar media, and antibiotic susceptibility was determined via the disc diffusion method. Polymerase chain reaction (PCR) was employed to detect the *erm* genes responsible for macrolide resistance. Among 80 samples, 40 were identified as *S. aureus*, of which 18 were confirmed as MRSA. PCR analysis revealed the prevalence of *ermA*, *ermB*, and *ermC* genes at rates of 12%, 33%, and 11%, respectively. All MRSA isolates exhibited multidrug resistance to antibiotics, highlighting the challenge of treating infections in burn patients. This study underscores the critical need for molecular characterization of MRSA strains to inform effective therapeutic strategies and control their spread in burn wards.

Keywords: *Staphylococcus aureus*, MRSA, Hichrom agar, Burns, *erm*.

1. Introduction

Staphylococcus aureus is a pathogenic bacterial species, a spherical, gram-positive bacterium. These organisms are naturally found in various parts of the human body without causing any symptoms. However, in some cases, they may become opportunistic, causing serious diseases [1-4]. Methicillin-resistant *Staphylococcus aureus* which frequently exhibits multidrug resistance in clinical infections, is one of the most serious and sometimes fatal pathogens. Patients with severe burns are at high risk for MRSA infection due to the loss of their protective skin barrier and suppression of the immune system. Therefore, The presence of methicillin-resistant *Staphylococcus aureus* (MRSA) in burn patients is a major health concern [5-8]. According to the 2013 update of the Global Burden of Disease (GBD), Burns are ranked as the sixth leading cause of death worldwide. A study supported by autopsy results showed that more than 60% of burn victims died from infectious complications [9, 10]. The burn wound (BW) is rapidly colonized by bacteria, causing dynamic exchange with the external environment, forming biofilms. MRSA

strains isolated from burn patients have been shown to show resistance to several antibiotics [11-13]. MRSA has emerged as a major cause of acute hospital-acquired infections with high mortality rates, often exhibiting resistance to multiple drugs, particularly beta-lactam antibiotics, due to the production of a modified penicillin-binding protein (PBP2a) encoded by the *mecA* gene located within the SCCmec cassette chromosome [14-16]. MRSA is remarkably adaptable and highly diverse. It is constantly evolving new mechanisms to benefit its survival. Worldwide, MRSA is the most common cause of hospital-acquired infection. It remains the most tolerant and can survive harsh conditions. In recent years, there has been a significant rise in MRSA isolates, posing a serious challenge as resistance to this pathogen implies resistance to all beta-lactam antibiotics [17-19]. Macrolide antibiotics, such as erythromycin, are among the most commonly used treatments for Gram-positive cocci bacteria. However, resistance to erythromycin in Gram-positive bacteria arises through various mechanisms, including modification of the ribosomal target site mediated by methyltransferases encoded by

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erm genes (erythromycin resistance methylase) and efflux pumps encoded by macrolide-specific resistance genes (*msrA* and *msrB*). Among these mechanisms, methylation mediated by erm genes is the primary factor contributing to macrolide resistance. In *Staphylococcus aureus*, three major erm genes—*ermA*, *ermB*, and *ermC*—have been identified as key contributors to erythromycin resistance [19, 20]. The increasing antibiotic resistance of bacterial isolates has made it essential to gather epidemiological data on the susceptibility of MRSA strains isolated from burn injuries. Such data can guide the selection of appropriate drugs for controlling hospital-acquired infections and improving patient treatment outcomes. Accordingly, this study aimed to evaluate the antibiotic sensitivity profile and determine the prevalence of erm genes (*ermA*, *ermB*, and *ermC*) in methicillin-resistant *Staphylococcus aureus* isolates from burn patients admitted to hospitals in Baquba city, Iraq.

2. Materials and methods

2.1. Samples collection

Dry swab samples were taken from eighty human samples from patients with burns from Baquba General Hospital - Specialized Burns Center after consent was obtained from patients and their companions. The samples were taken under the supervision of the specialized medical staff using sterile swabs (saturated in saline solution 0.9). They were transferred directly to the laboratory to conduct the necessary tests

2.2. Phenotypic diagnosis of *S. aureus*

All samples were directly cultured on mannitol salt agar (Hi-Media -India), which is a suitable medium for isolating *Staphylococcus aureus* due to its high salt content that inhibits the growth of other microbes. Then, the colonies were prepared on a slide to be stained with Gram stain and observed under the microscope as Gram-positive clusters. Then, a som of biochemical tests were conducted, such as catalase, oxidase, Coagulase, and urease.

2.3. Detection MRSA isolates

2.3.1. HiCrome MeReSa agar base

HiCrome MeReSa agar medium (Hi-Media, India) was used for the detection of MRSA isolates. This chromogenic medium is specifically designed for MRSA detection. The medium was prepared according to the manufacturer's instructions by dissolving 41 g of the media in 500 mL of distilled water. After cooling, a selective supplement (FD 229) was added along with 5 mL of distilled water to each methicillin vial containing 2 mg of methicillin, mixed thoroughly, and poured into sterile Petri dishes. Isolates exhibiting positive growth, indicated by blue colonies, were

identified as MRSA, while other strains were considered MSSA. All isolates were initially cultured on mannitol salt agar and incubated at 37°C for 24–48 hours.

2.3.2. Cefoxitin disk method

A broth culture of all *S. aureus* strains was grown on Mueller-Hinton agar (Hi-Media, India). A 30 µg cefoxitin disk (Bioanalysis, Turkey), along with other antibiotic disks, was then placed on MHA plates and incubated at 37°C for 18 h. The zone of inhibition was measured according to CLSI guidelines. A zone of inhibition >22 mm around the cefoxitin disk was interpreted as MSSA, while a zone of inhibition <21 mm around the cefoxitin disk was interpreted as MRSA.

2.3.3. Molecular detection of MRSA

Molecular identification of MRSA isolates was used to extract DNA using PCR primers manufactured by (Macrogen Laboratories- Korea) according to NCBI sequences (Table 1)

2.4. DNA extraction

DNA extraction for bacterial strains in this study was performed using a Genomic DNA Extraction kit (Zymo Research, USA) according to the manufacturer's instructions. The resulting PCR products were sequenced using Sanger sequencing performed by Shanghai Majorbio Biopharm Technology Co., LTD.

2.5. Polymerase chain reaction program

The final solution was placed in a centrifuge and centrifuged at 15,000 rpm for 15 seconds. The tubes were then carefully transferred to a thermal cycler to complete the amplification process for each primer, following the program outlined in Table 2. Subsequently, the reaction products were loaded onto an agarose gel, and electrophoresis was performed for one hour at 100 volts. The gel was stained with ethidium bromide, and the bands were visualized and photographed according to the instructions provided by Macrogen (South Korea). Finally, the recovered sequence chromatograms were manually analyzed using SnapGene Viewer to accurately interpret the nucleic acid peaks.

2.6. Antibiotic susceptibility test

Antibiotic susceptibility testing for all isolates was performed using the Kirby-Bauer method [21], as described by Bauer and Kirby. Briefly, 5 ml of nutrient broth was inoculated with pure colonies aged 18-24 hours and incubated at 37°C. The bacterial suspension was then compared with McFarland solution to achieve an approximate concentration of 1.5×10^8 cells/ml. 1 ml of the bacterial suspen-

Table 1. Primers used for PCR amplification of *ermA*, *ermB*, and *ermC* genes in MRSA isolates.

Genes	Pr.	Sequence 5'—3'	Annealing Temp. (C°)	Size (bp)	Ref.
<i>ermA</i>	F:	AAGCGGTAAACCCCTCTGA	55C°	190	(21)
	R:	TTCGCAAATCCCTTCTCAAC			
<i>ermB</i>	F:	CATTTAACGACGAAACTGGC	60C°	425	
	R:	GGAACATCTGTGGTATGGCG			
<i>ermC</i>	F:	AATCGTCAATTCCTGCATGT	55C°	299	
	R:	TAATCGTGGAATACGGGTTTG			

Table 2. PCR amplification program for detection of *ermA*, *ermB*, and *ermC* genes in MRSA isolates

Steps	Temp. °C	Time m: s	No. Cycles
In. Denaturation	95	5 m	1- Cycles
Denaturation	95	30 s	
Annealing			
<i>ermA</i>	55	30 s	30- Cycles
<i>ermB</i>	60		
<i>ermC</i>	55		
Extension,	72	30 s	
Final extension	72	7 m	
Hold	10	10 m	1- Cycles

Table 3. Antibiotic discs used for susceptibility testing of MRSA isolates: concentrations and interpretive criteria.

Antibiotic	Conc. µg/ml	Sensitive	Intermediate	Resistant
Amikacin	30	≥17	15-16	≤14
Azithromycin	15	≥18	14-17	≤13
Ciprofloxacin	5	≥21	16-20	≤15
Clindamycin	10	≥17	15-16	≤14
Erythromycin	15	≥23	14-22	≤13
Gentamicin	10	≥15	13-14	≤12
Imipenem	10	≥16	15-16	≤13
Levofloxacin	5	≥ 19	16-18	15≤
Penicillin	10U	≥29	-	≤28
Tetracycline	30	≥19	15-18	≤14

sion was transferred to Mueller-Hinton agar and allowed to dry. Antibiotic discs (Bioanalysis, Turkey), as detailed in Table 3, were carefully placed on the agar surface, and the plates were incubated for 24 hours at 37°C. Following incubation, the diameters of the zones of inhibition were measured and interpreted according to the specifications in Khan et al., 2024 [22].

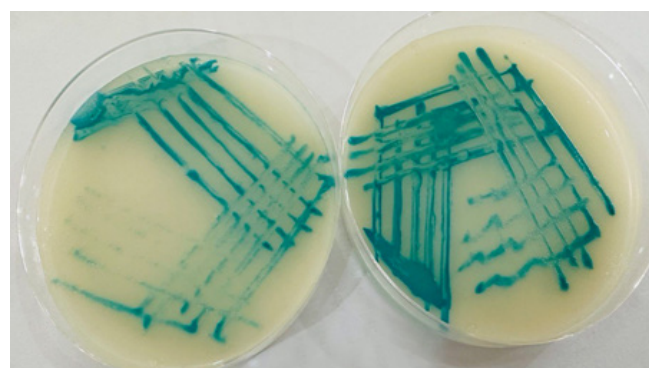
3. Results

3.1. Identification of MRSA isolation

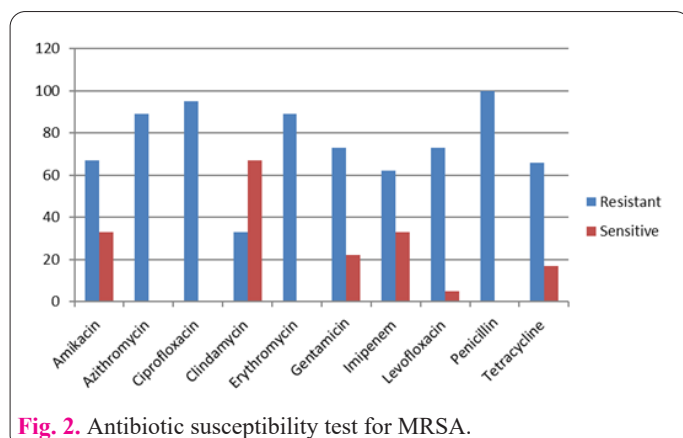
Approximately eighty human medical samples were collected from hospitalized patients with first- and second-degree burns using swabs saturated with saline solution, under the supervision of specialized medical staff. All swabs were cultured directly in the laboratory on Mannitol salt agar, then incubated at 37°C for 24 h. Forty (50%) samples showed positive growth on Mannitol salt agar, 23 (29%) samples did not show growth, and 17 (21%) showed unknown growth. Subsequently, biochemical tests and Gram staining were performed for accurate identification. The isolates were then cultured on HiCrome MeReSa selective medium to accurately identify MRSA based on the color indicator. Eighteen samples (36%) showed blue color on the media, indicating MRSA, as shown in Fig. 1.

3.2. Antibiotic susceptibility test

Performed susceptibility test for 18 isolates of MRSA to 10 antibiotics was determined by using the Kirby-Bauer Method (Antibiotic disk diffusion method) according to Clinical Laboratory Standards Institute [22] as shown in Table 4 and Fig 2. MRSA isolates were 100% resistant to penicillin, followed by Ciprofloxacin at 95%, while the antibiotics Azithromycin and Erythromycin had a resistance rate of 89%, while the antibiotic Clindamycin showed the

**Fig. 1.** Growth of MRSA insolent in HiCrome MeReSa media.**Table 4.** Antibiotic susceptibility profiles of MRSA isolates from burn patients.

Antibiotic	Resistant		Intermediate		Sensitive	
	%	No.	%	No.	%	No.
Amikacin	67	12	-	-	33	6
Azithromycin	89	16	11	2	-	-
Ciprofloxacin	95	17	5	1	-	-
Clindamycin	33	6	-	-	67	12
Erythromycin	89	16	11	2	-	-
Gentamicin	73	13	5	1	22	4
Imipenem	62	11	5	1	33	6
Levofloxacin	73	13	22	4	5	1
Penicillin	100	18	-	-	-	-
Tetracycline	66	12	17	3	17	3



lowest resistance rate of 33%. In a related context, all the isolates of MRSA showed multiple antibiotic resistance, they were resistant to most of the antibiotics under study.

3.3. Multidrug antibiotics Resistant (MDR)

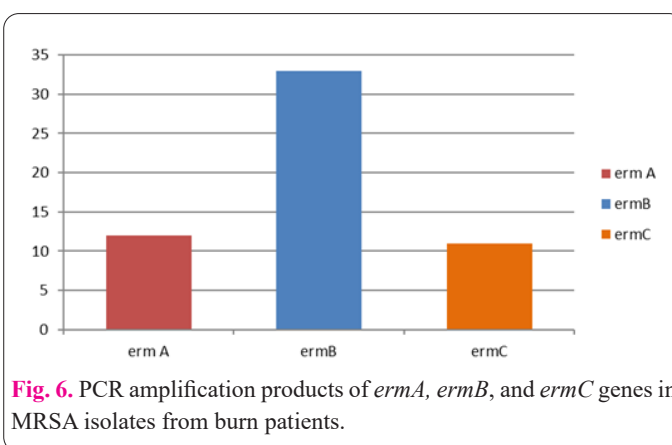
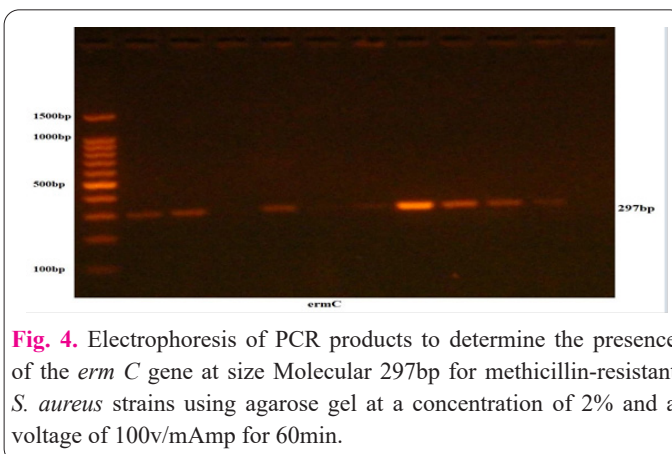
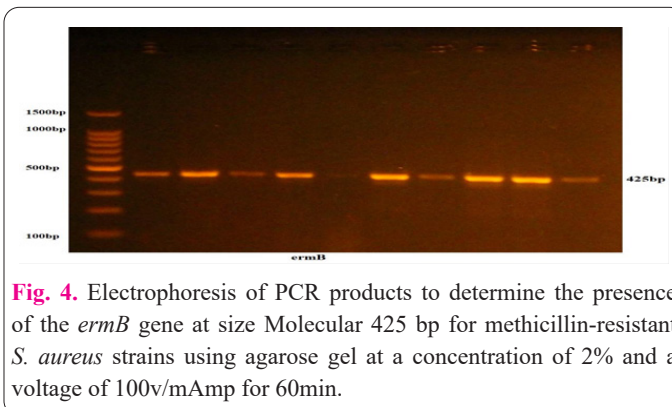
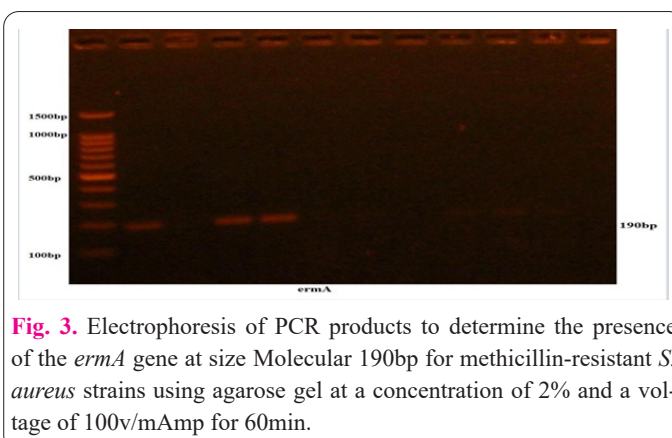
Results showed that all isolates diagnosed as MRSA showed multiple drug resistance (MDR) to the antibiotics used in the study. All MRSA isolates were resistant to eight antibiotics or more.

3.4. Detection *erm* (A, B, and C) genes of MRSA isolate

MRSA isolates were assayed for the presence of resistance genes, including *ermA*, *ermB*, and *ermC*, using PCR (Figures 3, 4, and 5). The results indicated that the *ermA* gene, encoding erythromycin resistance (Figure 3) with a molecular weight of 190 bp, was identified in 3 isolates (12%); the *ermB* gene (Figure 4), with a molecular weight of 425 bp, was identified in 6 isolates (33%); and the *ermC* gene (Figure 5), with a molecular weight of 297 bp, was identified in 2 isolates (11%), as shown in Figure 6.

4. Discussion

Patients lying in the burn wards are more vulnerable to MRSA infection than those lying down in another ward. Wound in burn patients is a difficult and complex problem due to the demolition of the skin, which reduces the effectiveness of cellular immunity [23, 24]. Many studies have demonstrated the involvement of MRSA in causing infections in burn cases, making it a significant cause of mortality. Therefore, detecting the spread of MRSA and understanding its resistance mechanisms in burn patients is crucial. This study revealed the prevalence of MRSA in burn cases and characterized the antibiotic sensitivity profiles of the isolates. Furthermore, we analyzed the molecular characteristics of MRSA isolates. In our study, approximately 80 *S. aureus* samples were collected from burn cases at Baquba Teaching Hospital from November 2023 to August 2024, with an MRSA isolation rate of 36%. This isolation rate is lower than previous reports, possibly due to strengthened hospital infection control management or the inappropriate long-term use of broad-spectrum antibiotics. It is worth noting that MRSA drug resistance is primarily due to the *mecA* gene, which encodes and regulates the expression of penicillin-binding protein (PBP2a) [25]. The prevalence of MRSA in burn patients was 36%, which aligns with other studies conducted in Baghdad, where the incidence of MRSA among burn patients reached 30% [26]. The current study provides data on the prevalence of macrolide resistance genes in MRSA strains isolated from



burn patients. Polymerase chain reaction revealed that the most prevalent genes in the MRSA strains were *ermB* and *ermA*, with frequencies of 33% and 12%, respectively. The *ermC* gene was detected at a lower frequency of 11%. This distribution may be explained by the transfer of resistance

genes via mobile genetic elements such as plasmids. Other studies have reported varying prevalence rates of *erm* genes; for example, the *ermA* gene has been reported to be the most prevalent in clinical *S. aureus* isolates, ranging from 21% to 67%. A study conducted in Denmark found that the *ermA* gene was responsible for 80% of erythromycin resistance. In our study, all MRSA isolates were resistant to erythromycin and exhibited multidrug resistance to other antibiotics. Similarly, a recent study in Nigeria involving 100 MRSA samples indicated that 85% showed multidrug resistance and carried erythromycin resistance genes such as *ermA* and *ermC* [20]. Several studies have reported that the *ermA* gene is frequently found in *S. aureus* isolates from clinical sources, with prevalence rates ranging from 21% to 67% [27]. Another study conducted in Denmark found that the *ermA* gene was responsible for 80% of erythromycin resistance [20]. Our study revealed that all MRSA isolates were resistant to erythromycin and exhibited multidrug resistance to other antibiotics. A recent Nigerian study involving approximately 100 MRSA samples indicated that 85% showed multidrug resistance and carried erythromycin resistance genes such as *ermA* and *ermC* [28]. These findings reinforce the need to monitor antibiotic resistance and develop strategies to reduce the spread of infection. In our study, the multidrug resistance index revealed that most isolates were resistant to eight or more antibiotics, indicating a lack of response to a wide range of antibiotic treatments. This high level of resistance can be attributed to the presence of multidrug resistance genes within the bacterial genome, enabling the bacteria to withstand a broad spectrum of antibiotics. The antibiotic susceptibility of isolates is influenced by several factors, including the number and source of isolates, as well as their ability to evade the effects of antibiotics. In this regard, numerous studies have indicated that the source of the isolate is a critical factor in determining multidrug resistance, particularly in cases of *S. aureus* causing hospital-acquired infections [29]. Multidrug resistance has also been associated with the overexpression of efflux pumps, proteins that allow bacteria to expel a wide range of chemical compounds, including antibiotics. These pumps contribute to the development of bacterial strains with great capabilities to survive even in toxic environments [30]. It should be noted that our study has some limitations: (1) the samples were collected from a single hospital, which limits the generalizability of the findings to other healthcare settings; and (2) the number of MRSA strains analyzed for molecular characteristics was relatively small. To strengthen the research, future studies should involve collaboration with multiple hospitals to broaden the sample size and include a larger number of cases.

In conclusion, our study highlights the significant prevalence of multidrug-resistant MRSA in burn patients at Baquba Teaching Hospital, with a notable presence of *erm* genes, particularly *ermB*, contributing to macrolide resistance. These findings underscore the importance of continuous surveillance of antibiotic resistance patterns and the implementation of robust infection control measures to limit the spread of MRSA in burn units. Further research, involving larger sample sizes and multiple hospitals, is warranted to gain a more comprehensive understanding of the molecular epidemiology of MRSA and to guide the development of effective treatment strategies for

burn patients.

Conflict of interests

The author has no conflicts with any step of the article preparation.

Consent for publications

The author read and approved the final manuscript for publication.

Ethics approval and consent to participate

No human or animals were used in the present research.

Informed consent

The authors declare that no patients were used in this study.

Availability of data and material

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Authors' contributions

Sarmad Qassim Mohammad & Sinda Zarrouk & Hussam Sami: contributed to study design, literature review, and analysis, contributed to experimental analysis, sample collection, and obtaining ethical approval. Sinda Zarrouk: contributed supervision and follow-up to data analysis, experimental analysis and auditing. Idriss Saleh Jalil: Contribute to providing resources and collecting samples. All authors contributed to the writing, reviewing, and final proofing of the article.

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