# Prevalence of mecA, icaA, hlα, sea genes in multi-drug resistance *Staphylococcus aureus* isolated from vaginosis in Iraqi women

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## ABSTRACT

**Background:** *Staphylococcus aureus* has become resistant to many antibiotics worldwide due to their widespread use. The development of *Staph. aureus* resistance refers to many mechanisms involving the generation of enzymes that suppress antimicrobial factors**. Objective**: The current study used genetic markers to investigate *Staph. aureus*'s prevalence, resistance, and virulence. **Methodology**: 150 vaginal swab samples were collected from pregnant and nonpregnant women admitted to Ibn Al-Baladi and Al-Imamain Al-Kadhimain Hospital / Iraq. The study was performed from July to October 2023. **Results:** The results showed highly resistant isolates of *Staph. aureus* toward Cefoxitin, Benzylpenicillin, Amoxicillin, Oxacillin (100%), Amikacin, and Erythromycin (70%). Moderate resistance against Tetracycline, Fusidic Acid (55%), Gentamicin, and Tobramycin (50%).MDR (multidrug resistance) was estimated at 60%. In comparison, XDR ( extensive drug resistance) was estimated at 40% .Further, the results showed that 13 isolates of *S.aureus* have *mecA* genes at (86.7%), while 11 isolates have *icaA* gene (73.3%), and 8 isolates have *sea* gene (53.3%), whereas *hla* gene found in all 15 isolates (100%). Sequences of the *mecA* gene had a similarity of 100% with the reference strain KR936061.1 in GenBank.The *icaA* gene had a similarity of 99.7%with CP123742.1 . The *sea* gene had a similarity of 98% with LC020109.1. The results sequence of *hla* gene had a similarity of 99.4% with reference strain EF543163.1 in gene bank. **Conclusions:** There was a prevalence of methicillin resistance *in Staph. aureus due to the presence of the* resistance gene (*mecA* gene 86.7%) in most isolates. In isolates, the existence of virulence genes icaA, sea, and hla was at (73.3%,53.3%, and 100%, respectively).

**Keywords:** *Staphylococcus aureus*, *mecA,icaA,hlα, sea*.
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## INTRODUCTION

In the last decades, *Staphylococcus aureus* has been resistant to many drugs worldwide due to the standard antibiotics used. The development of *Staph. aureus* resistance is referred to by many mechanisms, involving the generation of enzymes that suppress antimicrobial factors, the activation of antimicrobial efflux pumps, the reduction of permeability in the cell wall of bacteria to antibiotics, and the modulation of objective position to antibiotics (1).

*Staph. aureus* causes critical infections in humans, including urinary infections, mastitis, pneumonia, meningitis, endocarditis, and osteomyelitis. It represents the leading cause of nosocomial infection in surgical wounds (2,3). *Staphylococcus* spreads in different habitats and can colonize the skin, dermal glands, and mucosal membranes of humans and animals (4,5). Bacterial infection associated with preterm birth causes morbidity in fetal and neonatal mortality. Although medical progress and targeted interventions, 15 million cases of preterm birth happen annually, one in ten births, highlighting a need for prophylactic measures to prevent disease(6). *Staph. aureus* is a common element of the skin microbiota, and nosocomial and community-acquired strains have been given attention as established causes of chorioamnionitis and preterm, especially in individuals with extensive healthcare exposure(7). A previous study detected *S. aureus* in healthcare workers' nasal cavities. Where the prevalence of MRSA was 94%

among *Staph. aureus* (8); it was also isolated from the nasal cavities of nurses working in two different hospitals in Baghdad, with infection rates of 38.4% and 37.5% (9). The appearance of virulent and potent drugs of *Staph. aureus*, especially MRSA (methicillin-resistant *Staphylococcus aureus*), causes a critical challenge for the therapy of *Staphylococcus* infections. The methicillin-resistant strains are more resistant to many antibiotics (beta-lactams, macrolides, aminoglycosides) and, therefore, difficult to treat infections. The penicillin resistance mechanism is beta-lactamase production, which suppresses penicillin by destroying the beta-lactam ring. Another mechanism related to the presence of penicillin-binding protein 2a, encoded via the *mecA* gene, is found in SCCmec (*Staphylococcus* cassette chromosome mec) (10).*Staph. aureus* pathogenicity is associated with different virulence genes, such as *S. aureus* enterotoxins (SEs) and hemolysins (hlα and hlβ), which contribute to adhesion, colonization, and tissue invasion , thereby inducing pathogenicity(11). SEA (Staphylococcal enterotoxin A) is among the most commonly involved enterotoxins in food poisoning outbreaks (12). The *icaA* (intercellular adhesion gene A) is responsible for PIA (polysaccharide intercellular adhesin) poly-N-succinyl β-1-6 glucosamine formation, which has a distinct role in synthesizing biofilm and cell adhesion. *Staph. aureus* produces several biofilm layers, each formed within a slime or glycocalyx layer, which exhibit distinct protein expression patterns (13). Molecular diagnostic applications for pathogenic bacteria have improved outcomes in septic cases, particularly in synchronism with antibiotic management programs (14). MRSA contagion is still an essential reason for illness and death in the world and forms a challenge to efficient treatment (15). The detection of the *mecA* gene is considered the primary methicillin resistance determinant in *Staphylococcus* species, enabling rapid molecular screening to distinguish MRSA (methicillin-resistant *Staph. aureus*) from MSSA (methicillin-sensitive *Staph. aureus*) via PCR. Direct detection of MRSA in clinical samples remains challenging, as the *mecA* gene may be present in methicillin-resistant coagulase-negative *Staphylococcus* strains isolated from different clinical samples (16). This study aimed to investigate the prevalence of *Staph. aureus* resistance and the role of these genes in drug resistance.

## METHODOLOGY

### 1. STUDY DESIGN AND SAMPLE COLLECTION

A total of 150 samples were collected from pregnant and non-pregnant women by vagina swabs. The study was conducted from July to October 2023 at Ibn Al-Baladi and Al-Imamain Al-Kadhimain hospitals in Iraq. Inclusion criteria were age (15-72 years), symptoms of infection, history of contraceptive use, and receipt. There were excluded women who suffered from bleeding vaginas. The swab was inserted into half of the space between the introitus and the cervix to prevent contamination by uterine mucus. After that, the swab is pressed gently on the vagina walls, swirled to collect the sample, and carefully removed to avoid touching another part of the body (17).

### 2. ETHICAL APPROVAL

All specimens were obtained in compliance with institutional regulations, safety requirements, and ethical approval was granted (Approval No.108607, dated 17/1/2023). Written informed consent was obtained from all participants prior to their enrolment in the study.

### 3. IDENTIFICATION OF BACTERIA AND ANTIBIOTIC SENSITIVITY

The swabs were cultured on Blood agar plates, and mannitol agar plates, and incubated for 24 hours at 37 °C to promote bacterial growth. The bacterial isolates were identified using the Vitek2 compact system (ID GP card:21342, bioMérieux/France. These isolates were recultured and stored at 4 °C for use in subsequent analyses. According to manufacturer instructions, the antibiotic sensitivity test was performed using a Vitek 2 compact system with an AST card.

### 4. DETECTION OF *mecA, icaA, hla,* and *sea* GENES BY PCR

Genomic DNA from bacteria was extracted using ABIOpure Total DNA/USA. A Quantus Fluorometer(Promega /USA) was used to measure DNA concentration. It detected the presence of genes in *Staph. aureus* using specific primers for these genes (Macrogen company /Korea), as in Table (1). The PCR (Polymerase chain reaction) technique was applied to 15 *Staph. aureus* isolates that were highly resistant to antibiotics. The PCR reaction contents (20 µL ) included 10µL GoTaq Green master mix PCR (2X) (Promega, USA), 1 µL for each forward and

reverse primer (10 picomoles/µL) (Macrogen/ Korea), 3µL of DNA template, and 5µL of free nuclease water. The PCR program for genes is shown in Table (2). Electrophoresis was also applied using agarose gel (2%) to reveal the PCR amplicon using staining with ethidium bromide.

**Table (1): Primers sequences of *mecA,icaA, hla, and sea* genes**

| Gene | 5`-3` sequence | Annealing temp($^0$) | Product size (bp) | References |
|---|---|---|---|---|
| ***mecA*-F** | TCACCTTGTCCGTAACCTGA | 58 | 530 | |
| **mecA-R** | GGTACTGCTATCCACCCTCA | | | |
| ***sea*-F** | TTG GAA ACG GTT AAA ACG AA | 55 | 120 | (19) |
| ***sea*-R** | GAA CCT TCC CAT CAA AAA CA | | | |
| ***icaA*-F** | AACCAACGCGAGCTAAAAGC | 59 | 776 | |
| ***icaA*-R** | GCGTTAACAAACCGGTCACT | | | |
| ***hla*-F** | GTGCAATTGGTAGTCATCACG | 59 | 219 | |
| ***hla*-R** | TGGAACCCGGTATATGGCAA | | | |

**Table (2): PCR amplification for *mecA,icaA, hla, and sea* genes**

| Genes | Initial Denaturation | Denaturation | Annealing | Extension | Final Extension | Hold |
|---|---|---|---|---|---|---|
| ***mecA*** | 95°C(5min) 1cycle | 95°C30(sec) 30cycle | 58 °C(30sec) 30cycle | 72°C(30sec) 30 cycle | 72°C(7min) 1 cycle | $10^0$C(10min) 1cycle |
| ***sea*** | | | 55°C(30sec) 30cycle | | | |
| ***icaA*** | | | 59°C(30sec) 30cycle | | | |
| ***hla*** | | | 59°C(30sec) 30cycle | | | |

## 5. SEQUENCING *MECA,ICAA, HLα,* and *SEA* GENES

It was conducted using sequence-based PCR to amplify the genes of Interest from *S. aureus* isolates by Macrogen DNA sequencing company (Korea). PCR products were purified and sequenced using the BigDye Terminator v3.1 Cycle Sequencing kit with a forward primer on an ABI 3130 Genetic Analyzer (Applied Biosystems, Foster City, CA). Nucleotide sequences were aligned with global isolates by NCBI's Basic Local Alignment Search Tool (BLAST) and Bio Edit program to identify NCBI (http://www.ncbi.nlm.nih.gov ).

## 6. Phylogenetic Analysis

Phylogenetic analysis was performed using nucleotide sequences of the icaA gene. Sequences were aligned using ClustalW, and a phylogenetic tree was constructed in MeGA X using the Neighbor-Joining method. Evolutionary distances were calculated using the Kimura 2-parameter model. The robustness of the tree topology was evaluated based on branch length analysis.

## 7. STATISTICAL DATA ANALYSIS

Chi-square tests were performed using MedCalc Software (2023) online; $p < 0.05$ was considered statistically significant, whereas $p \geq 0.05$ indicated no significant difference.

## RESULTS

### 3.1. IDENTIFICATION OF BACTERIA AND ANTIBIOTIC SENSITIVITY

The Vitek2 compact system identified 60 isolates with a 96% probability of being *S. aureus* isolates. Furthermore, the antibiotic resistance outcomes indicated varying levels of resistance among the twenty isolates, as indicated by the AST Vitek2 Compact results (Table 3 and Figure 1). The results showed significant differences (P<0.05) among highly resistant *S. aureus* isolates for Cefoxitin, Benzylpenicillin, Amoxicillin, Oxacillin (100%), Amikacin, and Erythromycin (70%). Moderate resistance among isolates was against Tetracycline, Fusidic Acid (55%), Gentamicin, and Tobramycin (50%). In contrast, weak resistance among isolates was observed against Clindamycin (45%), Levofloxacin, Moxifloxacin (30%), Linezolid, Vancomycin, Tigecycline, Rifampicin, and Trimethoprim/Sulfamethoxazole (5% each). In contrast, they did not appear resistant to Teicoplanin or Nitrofurantoin (0%), with no significant differences (P > 0.05).

**Table ( 3 ): Antibiotic resistance of *S.aureus* isolates**

| Antibiotic | | | Resistant | Sensitive | P value |
|---|---|---|---|---|---|
| **Cefoxitin Screen** | | | 20(100%) | 0(0%) | P < 0.001 |
| **Benzylpenicillin** | | | 20(100%) | 0(0%) | P < 0.001 |
| **Amoxicillin** | | | 20(100%) | 0(0%) | P < 0.001 |
| **Oxacillin** | | | 20(100%) | 0(0%) | P < 0.001 |
| **Amikacin** | | | 14(70%) | 6(30%) | P < 0.001 |
| **Gentamicin** | | | 10(50%) | 10(50%) | P> 0.05 |
| **Tobramycin** | | | 10(50%) | 10(50%) | P> 0.05 |
| **Levofloxacin** | | | 6(30%) | 14(70%) | P < 0.001 |
| **Moxifloxacin** | | | 6(30%) | 14(70%) | P < 0.001 |
| **Erythromycin** | | | 14(70%) | 6(30%) | P < 0.001 |
| **Clindamycin** | | | 9(45%) | 11(55 %) | P < 0.001 |
| **Linezolid** | | | 1(5%) | 19(95%) | P < 0.001 |
| **Teicoplanin** | | | 0(0%) | 20(100%) | P < 0.001 |
| **Vancomycin** | | | 1(5%) | 19(95%) | P < 0.001 |
| **Tetracycline** | | | 11(55 %) | 9(45%) | P < 0.001 |
| **Tigecycline** | | | 1(5%) | 19(95%) | P < 0.001 |
| **Nitrofurantoin** | | | 20(0%) | 0(0%) | P < 0.001 |
| **Fusidic Acid** | | | 11(55 %) | 9(45%) | P < 0.001 |
| **Rifampicin** | | | 1(5%) | 19(95%) | P < 0.001 |
| **Trimethoprim/ Sulfamethoxazole** | 1(5%) | 19(95%) | P < 0.001 | | |

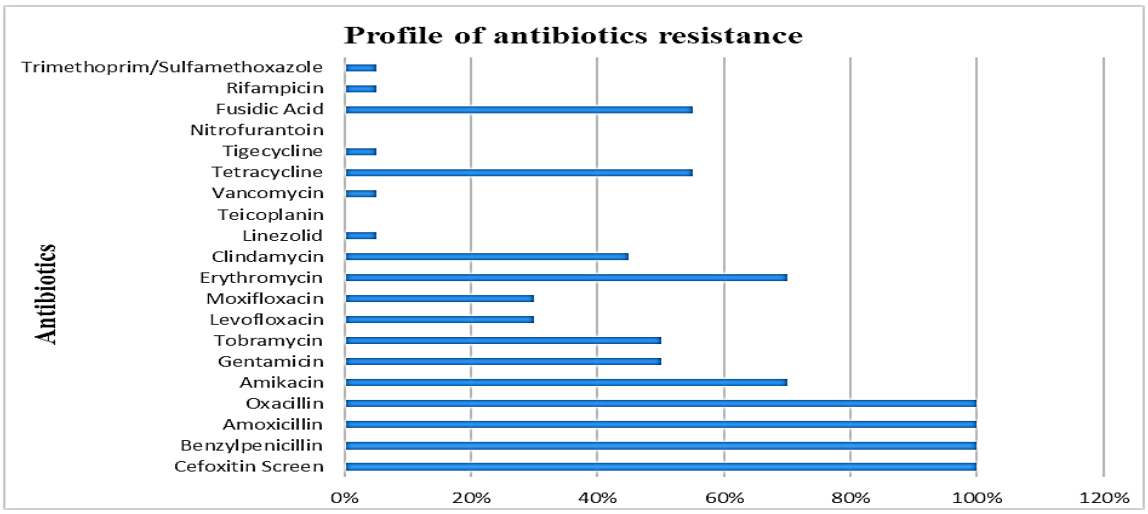
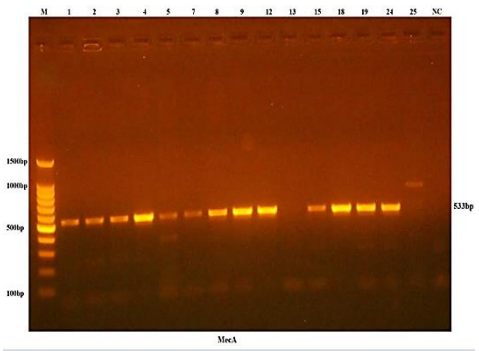



**Figure (1): Profile of antibiotic resistance for *S. aureus* isolates**

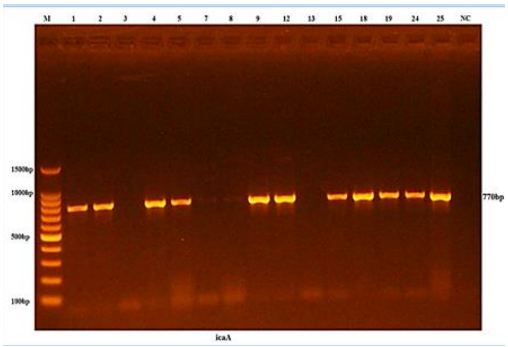
The findings for *S. aureus* isolates' antibiotic resistance indicated resistance to one or more antibiotic categories, with multidrug resistance (MDR) estimated at 60%. While XDR (extensive drug resistance) was estimated at 40%.

### 3.2. *MECA, ICAA, HLα,* and *SEA* GENES DETECTION

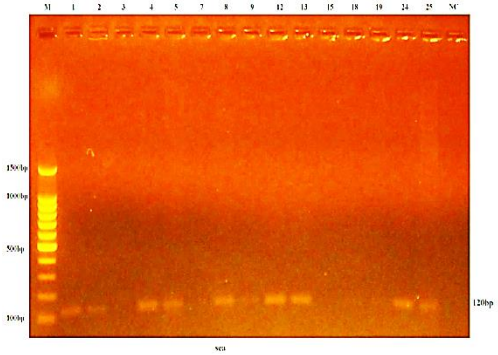
The results showed that 13 isolates of *S. aureus* have the *mecA* gene ( 86.7%),11 isolates have the *icaA* gene (73.3%), and eight isolates have the *sea* gene (53.3%), while the *hla* gene was found in all 15 isolates (100%)as shown in Figures (2,3,4,5) .



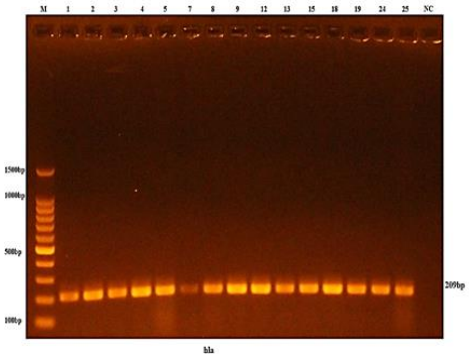

**Figure(2):The amplification PCR product of *mecA* gene (530bp) for *S.aureus* isolates was electrophoresed on 2% agarose gel. M: 100bp ladder marker.**




**Figure(3):The amplification PCR product of *icaA* gene (776bp) for *S. aureus* isolates was electrophoresed on 2% agarose gel. M: 100bp ladder marker.**



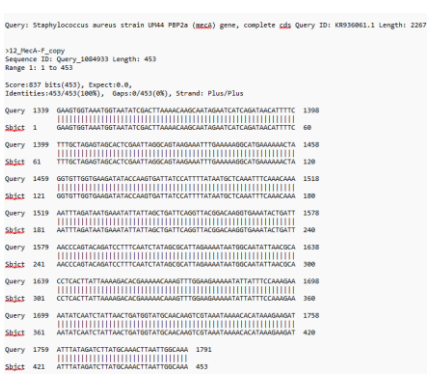

**Figure(4):The amplification PCR product of sea gene (120bp) for *S. aureus* isolates was electrophoresed on 2% agarose gel. M: 100bp ladder marker.**




**Figure(5):The amplification PCR product of hla gene (219bp) for *S.aureus* isolates was electrophoresed on 2% agarose gel. M: 100bp ladder marker.**

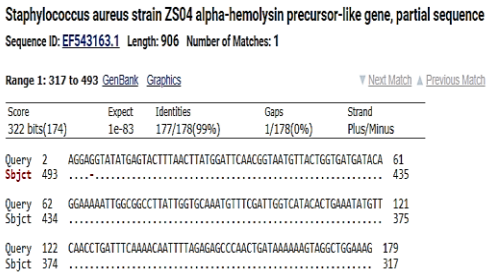
### 3.3 .SEQUENCING OF *MECA, ICAA, HLα,* and *SEA* GENES

The results sequence of the *mecA* gene (Figure 6)in the current study had a similarity of 100% with the reference strain in GenBank( KR936061.1). The sequence of *the icaA gene* (Figure 7) in the current study had a similarity of 99.7% with the reference strain in GenBank( CP123742 1). The sequence of the hla gene (Figure 8) had a similarity of 99.4% with the reference strain in GenBank (EF543163.1). The *sea* gene (Figure 9) had a similarity of 98% with the reference strain in GenBank( LC020109.1).

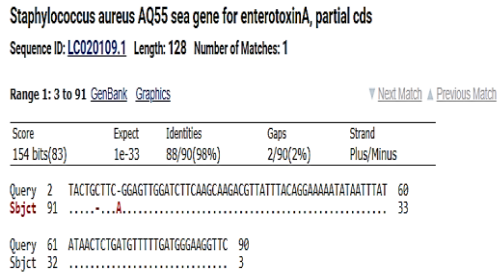



**Figure(6): Results Alignment of sequence for *mecA* gene for *Staph. aureus* isolate 12.**


**Figure(7): Results Alignment of sequence for *icaA* gene for *Staph. aureus* isolate 12.**




**Figure(8): Results of Alignment of sequence for *hla* gene for *Staph. aureus* isolate 12.**




**Figure(9):Results of Alignment of sequence for *sea* gene for *Staph. aureus* isolate 12.**

## Phylogenetic Analysis of *icaA* gene

Results from Figure (10) indicate that isolate 12_icaA-F clustered closely with strains retrieved from the NCBI RefSeq database. The short branch lengths between the isolate and related strains indicate a high degree of sequence similarity, suggesting conservation of the icaA gene within this clade.

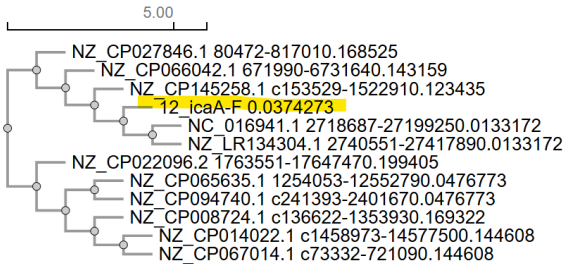



**Figure (10) : Phylogenetic Analysis for *icaA* gene for *Staph. aureus* isolate 12.**

## DISCUSSION

The current study is consistent with prior studies. These results reveal significant differences in antibiotic sensitivity in *Staph. aureus* strains. The outcomes are consistent with another study conducted in Iran that showed most *Staph. aureus* isolates were resistant to Benzylpenicillin at 71.92% and oxacillin at 58.97%. Whereas susceptible against ciprofloxacin at 5.12%, chloramphenicol at 2.56%, sulfamethoxazole-trimethoprim at 2.56%, vancomycin at 0%, and gentamicin at 0% (20). The microorganisms that cause illness may vary with geographic distribution, and appropriate therapies within a region can be identified by identifying resistant strains (21). Virulence factors such as enzymes, adhesins, cell-surface proteins, and toxins that are produced by *Staph. aureus* has a significant role in causing different diseases (22).

Additionally, one Iraqi study reported that *S. aureus* was isolated from burn wounds at a high percentage of 25% compared with other bacteria (23).Other Iraqi studies diagnosed *Staph.aureus* infection in the urinary system of pregnant women(24). The current study also agreed with another study that showed the resistance of *Staph. aureus* (100%) to oxacillin and penicillin, whereas the low resistance appeared with ciprofloxacin, gentamicin, and amikacin (30.8% and 26.90% each of them)(25). Another study indicated that *Staph. aureus* strains in urine samples have recorded a decline in resistance to amikacin and gentamicin (26). Qodrati *et al*. (27) observed that Methicillin resistance was an indicator of resistance to erythromycin and clindamycin (90.9% and 85.4%, respectively ). At the same time, gentamicin, moxifloxacin, ciprofloxacin, levofloxacin, and trimethoprim-sulfamethoxazole were 58.3%. The resistance percentage was zero for linezolid, daptomycin, vancomycin, and tigecycline. The spread of multi-drug resistance (MDR) isolates was 48.5%.

One Iraqi study that diagnosed *Staph. aureus* in hospitals using PCR found that 97.05% were diagnosed as *Staph. aureus*, whereas 80.88% were diagnosed as MRSA (28). Methicillin-resistant *Staph. Staph. aureus* strains are a common cause of nosocomial infections and are a significant public health concern. *Staph. aureus* is considered a bloodstream-causing infection that is common in most developed countries (29).

Moreover, in one study, the *mecA* gene was detected in 60% of *Staph. aureus* isolates from Iraqi patients with furunculosis (30). Vestergaard *et al*.(31) showed in their study that *Staph. aureus* in clinical samples is resistant to all antibiotic classes, and its resistance may evolve due to mutations of genes in chromosomes or by the acquired horizontally transmitted resistance factors

Some studies concur with our study in identifying the *icaA gene* and its association with resistance to antibiotics. Abdrabaa and Aburesha (30) showed in their study that *Staph. aureus* (MRSA) isolates from different clinical samples that produced biofilm had high *icaA* gene expression. Additionally, another study reported that 100% resistance to Ampicillin and Penicillin, and resistance rates of 91% to 86.66% and 86.66% resistance to Methicillin, Oxacillin, and Amoxicillin, respectively (32).

Abdulmanea *et al*. (10) showed that there is a relationship between *Staph. aureus*, MRSA, and the presence of *icaA, hla* genes. Furthermore, the appearance of high resistance in isolates carrying these genes. Baz *et al*.(33) recorded in their study that 32.6% were positive for the *sea* gene in *Staph. aureus* isolates that were resistant to ampicillin and amoxicillin. This variation in resistance could be attributed to the degree of contact between *Staph. aureus* strain and antibiotics. Many hypotheses demonstrated the increase in antibiotic impedance, not just the excessive utilization of antibiotics in medical care, but also the deficiency of organization of the marketing and use of antibiotics. This is increased due to self-therapy with high doses. The smuggled traffic of drugs is considered a factor in the appearance of resistant antibiotics (25).

## Conclusion

Our study demonstrated the presence of the virulence genes mecA, *icaA, hla*, and *sea* in *Staph. aureus* isolates. This indicates that the resistance isolates are resistant to antibiotics. Also, there was a prevalence of methicillin-resistant *Staph. aureus* due to the presence of the resistance gene (mecA gene 92%) in most isolates.

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# مدى انتشار الجينات *mecA* و*icaA* و*hlα* و *sea* في مقاومة الأدوية المتعددة للمكورات العنقودية الذهبية المعزولة من التهاب المهبل لدى النساء العراقيات

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**الخلاصة**

**خلفية البحث:** تسبب المكورات العنقودية الذهبية*S.aureus* حالات عدوى خطيرة لدى البشر مثل التهابات المسالك البولية، والتهاب الضرع، والالتهاب الرئوي، والتهاب السحايا، والتهاب الشغاف، والتهاب العظم والنقي. وهو يمثل السبب الرئيسي لعدوى المستشفيات في جروح العمليات الجراحية . **الهدف** :هدفت هذه الدراسة إلى معرفة مدى انتشار مقاومة بكتريا المكورة العنقودية الذهبية وضراوتها باستخدام الجينات المسؤولة عن الضراوة . **طرق العمل**: تم جمع 150 عينة من المسحات المهبلية من نساء حوامل وغير حوامل في مستشفى ابن البلدي والإمام الكاظم/العراق. أجريت الدراسة في الفترة من تموز إلى تشرين الاول 2023. **النتائج**: أظهرت النتائج مقاومة عالية لعزلات بكتريا المكورة العنقودية الذهبية تجاه سيفوكسيتين، بنزيل بنسلين، أموكسيسيلين، أوكساسيلين (100%)، أميكاسين، وإريثرومايسين (70%). وكانت مقاومة متوسطة للعزلات ضد التتراسيكلين وحمض الفوسيديك (55%) والجنتاميسين والتوبراميسين (50%). وقُدرت المقاومة للأدوية المتعددة MDR بـ 35%. بينما قدرت مقاومة XDR (المقاومة الشاملة للأدوية) بـ 42.9%. كما أظهرت النتائج أن 13 عزلة من بكتريا *S.aureus* تمتلك جين *mecA* بنسبة (86.7%)، و11 عزلة تمتلك جين icaA (73.3%)، و8 عزلات تمتلك جين sea. (53.3%)، في حين وجد جين *hla* في جميع العزلات الـ 15 (100%) كان لتسلسل جين *mecA* تشابه بنسبة 100% مع السلالة المرجعية KR936061.1. وكان لجين *icaA* تشابه بنسبة 99.79% مع CP133660.1. 1. وكان للجين *sea* تشابه بنسبة 98% مع LC020109.1. كان لتسلسل نتائج جين *hla* تشابه بنسبة 99.4% مع السلالة المرجعيةEF543163.1 في بنك الجينات. **الاستنتاجات**: كان هناك انتشار لمقاومة الميثيسيلين لبكتريا *Staph. aureus* بسبب وجود جين المقاومة (*mecA* gene 86.7%) في معظم العزلات. وكان وجود جينات الضراوة *icaA* وsea و*hla* بنسبة (73.3%، 53.3%، و100% على التوالي) في العزلات.

**الكلمات المفتاحية:** بكتريا *Staphylococcus aureus* , sea, hlα, icaA, mecA.