# Incidence and Antibiotics Resistance of *Staphylococcus aureus* isolated from Skin Lesion of Psoriasis Patients

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## Abstract

Isolation of *Staphylococcus aureus* was ought to be the reduced local defense factors in the lesion due to local and systemic immunosuppressant drugs like cytotoxic drugs and corticosteroid used for the treatment of psoriasis. The study aim to investigate the incidence and antibiotic resistance ability of these bacteria associated with psoriasis. Skin swab was taken from lesional skin of psoriasis patients for isolating of *S. aureus*. Isolates identified by conventional tests and confirmed with 16SrRNA specific primers. Among all patients, 37(52.9%) isolates were identified as *S. aureus*. All isolates tested for methicillin susceptibility using an initial screening test. The results showed 34(91.9%) isolates that resisted to pencillin, 29(78.4%) isolates resisted to methicillin and 22(59.5%) isolates that resisted to oxacillin. All isolates showed sensetivity to vancomycin and linezolid and then confirmed by used genotypic detection for presence of *mecA* gene. From this isolates, *mecA gene* was detected in 14 isolates. There were differences in resistance to antibiotics observed between *mecA*-positive and *mecA*-negative isolates.

**Key Words: *Staphylococcus aureus* , Antibiotic resistance, Psoriasis.**

## Introduction

Skin infections with *Staphylococcus aureus* are an important cause of morbidity and even mortality. Infections by these bacteria not only cause disease directly, but also thought to be important triggers in worsening inflammatory skin disease ranging from atopic dermatitis to psoriasis [1]. *S. aureus* is a commensal organism that colonizes up to 50% of humans[2]. The organism most often colonizes the anterior nares and, from there, may colonize other body surfaces, including other mucous membranes and damaged skin. It can produces enzymes and toxins, which include coagulase, an enzyme that clots plasma and coats staphylococcal cells, which prevents the cells from being phagocytosed and destroyed by macrophages[3]. Bacterial and viral infections may be linked to psoriasis. *S. aureus*, *Malassezia furfur*, and *Candida albicans* may play a role in exacerbation of chronic psoriasis [4,5,6].

Antibiotic resistant staphylococci are major public health concern since the bacteria can be easily circulated in the environment. Antibiotic resistance and multi-antibiotic resistance have been developed in *S. aureus*, which made the treatment of *S. aureus* infections more difficult [7,8]. Methicillin resistance is due to the presence of *mecA* gene coding for penicillin-binding protein (PBP2A) with a low affinity for β-lactam antibiotics. This gene is carried on Staphylococcal Cassette Chromosome (SCC)*mec*, a unique mobile genetic element, integrated into the staphylococcal chromosome[9].

In Najaf province, however, studies pertaining to molecular characterization of methicillin resistant *S. aureus* (MRSA)-conferring *mecA*-genes and the epidemiology of antibiotics resistance, especially among patients with psoriasis, are rare. This study concluded that *S. aureus* isolated from lesional skin of psoriasis patients have a relationship with psoriasis and there was a significant association between the resistant of these bacteria that harbored mecA gene with psoriasis.

## Materials and Methods

**Patients and Samples**: Seventy patients with psoriasis were included in this study. All patients attended Al-Sadder Medical City, outpatient clinic of Dermatology in Najaf city during the period from May 2011 to October 2011. The study included patients who have not received any treatment for their skin condition whether topical or systemic for at least 3 weeks before taking the swab, have not any other skin diseases which may interfere with the results (such as superficial bacterial infections as, impetigo, abscess and folliculitis) and recent exacerbation of their disease. For isolation and identification of *S. aureus*, all samples were collected using sterile swab taken from skin lesion of patients then directly inoculated onto a plate of mannitol salt agar and incubated at 37ºC aerobically for 24 hr. The suspicious colonies identified by Gram stain then further identified to species level using routine biochemical test such as coagulase test, Voges-Proskauer test and catalase production.

**Antibiotics susceptibility Test:** Antibiotics susceptibility was carried out against 19 antibiotics using the disk diffusion method on Muller-Hinton agar medium. Bacterial growth inhibition zones around the disks measured and interpreted as recommended by the National Committee for Clinical Laboratories Standard guidelines[10]. All isolates were tested for methicillin susceptibility using an initial screening test according to the guidelines of the CLSI (2010)[10.] The isolates were initially screened for methicillin susceptibility by disk diffusion method using methicillin, oxacillin and cefoxitin antibiotic disks. Multiple antibiotic resistance (MAR) phenotypes were determined for *S. aureus.* In this study, MDR isolates were defined as isolates demonstrating non-susceptible to ≥ 1 agent in ≥ 3 antibiotics of the fifteen categories of antimicrobial drugs While, the definition of XDR: An isolate that is non-susceptible to ≥ 1 agent in all but ≤ 2 categories. PDR when isolates non-susceptible to all fifteen antimicrobial categories tested[11].

**DNA Extraction:** For molecular detection, DNA extraction was done according to Sambrook *et al*,1989 [12]. The extract was stored in freezer at -20°C until used. Primers used in this study for mecA gene were F: TCAGGTTACGGACAAGGTGA and R: AAACCACCCAATTTGTCTGC. The PCR tubes were placed on the PCR machine and the right PCR cycling program were applied. The PCR conditions were as follows: Initial denaturation at 95°C for 2 minutes followed by 30 cycles of amplification with 95°C for 30 seconds, annealing at 56°C for 30 seconds, extension at 72°C for 40 seconds and final extension at 72°C for 5 minutes.

## Results

Among 70 staphylococci recovered from 70 psoriatic patients, 40 (57.1%) isolates were considered as *S. aureus* using traditional biochemical tests. The forty isolates were additionally investigated by PCR amplification of species-specific parts of the gene encoding the 16SrRNA with the oligonucleotide primers. The result revealed that 37 (92.5%) isolate showed positive amplification of 756 bp confirming that these isolates were *S. aureus* Three isolates (7.5%) gave negative results when detected by using 16SrRNA amplification were excluded in this study. However, although no significant difference was seen between *S. aureus* carriers and non-carriers psoriasis patients there was a relationship found between the present *S. aureus* and psoriasis patients (Fig.1). The psoriasis patients who carried *S. aureus* were recorded in 19 males (51.4%**)** and 18 female (48.6%). Males were recorded slightly higher percentage than females, there was no significant difference between them ($P > 0.05$) (Tab. 1).

The overall resistance of the isolated *S. aureus* to antibiotics is shown in Tab (2). All isolates had antibiotic resistance, the results showed that high resistance rate against penicillin G (91.9%) followed by methicillin (78.4%) and oxacillin (59.5%). All the isolates were susceptible to linezolid and vancomycin. Multiple antibiotic resistance (MAR) phenotypes were also determined. The occurrence of multidrug resistance (MDR), extensive drug resistance (XDR) was investigated among (24= 64.9%) and (1=2.7%) respectively, and no isolates were identified as pandrug resistant (PDR).

Resistance for penicillinase-stable penicillins and cefoxitin by disk diffusion was originating in 20 (54.1%) isolates for methicillin, oxacillin and cefoxitin. The remaining 9/29 (31%) *S. aureus* isolates which establish as resistant against methicillin, were determined as susceptible to cefoxitin. The 20 cefoxitin, methicillin and oxacillin resistant isolates were initially interpreted as MRSA. The gene of *mecA* was detected in 14 of these isolates. The relationship between antibiotic resistances of individual *S. aureus* and the presence of *mecA* gene is shown in Tab (3). All *mecA* positive isolates were resistant to penicillin G, oxacillin, methicillin, cefoxitin, and piperacillin-tazobactam. Also it is determined that vancomycin, linezolid, teicoplanin chloramphenicol and gentamicin were the most effective antibiotics against these isolates with resistant rates 0.0%, 0.0%, 7.1%, 7.1% and 14.1%, respectively.

**Table (1): The relationship between sex and distribution of psoriasis patients with or without *S. aureus* isolates**

| Gender | Psoriasis | | | | Total |
|---|---|---|---|---|---|
| | *S. aureus* carrier | % | *S. aureus* non-carrier | % | |
| Males | 19 | 51.4 | 15 | 45.5 | 34 |
| Females | 18 | 48.6 | 18 | 54.5 | 36 |
| Total | 37 | | 33 | | 70 |
| P value | 0.622 | | | | |

**Table (2): Antibiogram susceptibility proportions of isolated *S. aureus* to tested antimicrobial agents with disk diffusion methods (n= 37)**

| **Antibiotic** | **P** | **OX** | **ME** | **AMC** | **PRL** | **CEP** | **CTX** | **FOX** | **MEM** | **CIP** |
|---|---|---|---|---|---|---|---|---|---|---|
| **Resistant isolate (%)** | 34 (91.9) | 22 (59.5) | 29 (78.4) | 18 (48.7) | 21 (56.8) | 21 (56.8) | 14 (37.8) | 20 (54.1) | 4 (10.8) | 9 (24.3) |
| **Antibiotic** | **L** | **VA** | **AZM** | **TMP** | **CD** | **TP** | **C** | **TE** | **GM** | |
| **Resistant isolate (%)** | - | - | 16 (43.2) | 13 (35.1) | 10 (27) | 1 (2.7) | 1 (2.7) | 11 (29.7) | 2 (5.4) | |

P:Pencillin,OX:Oxacillin, ME: Methicillin, AMC: Amoxicillin-Clavulanic acid, PRL: Pipracillin - tazobactam, CEP: Cephalothin, CTX: Cephotaxime, FOX: Cefoxitin, MEM: Meropenem, CIP: Ciprofloxacin, TMP: Trimethoprim, CD: Clindamycin, AZM: Azithromycin, L:Linezolid,VA:Vancomycin, TP: Teicoplanin, C: Chloramphenicol, TE: Tetracycline and GM:Gentamycine

**Table (3): Relationship between antibiotics resistant *S. aureus* isolates and the presence of *mecA* gene**

| Type of Antibiotic | No.(%) of antibiotic resistant isolates | | Type of Antibiotic | No.(%) of antibiotic resistant isolates | |
|---|---|---|---|---|---|
| | *mecA*-positive isolates (n=14) (%) | *mecA* - negative isolates (n=23) (%) | | *mecA*-positive isolates (n=14) (%) | *mecA*-negative isolates (n=23) (%) |
| Penicillin G | 14 (100.0) | 20 (87.0) | Trimethoprim | 10 (71.4) | 3 (13.0) |
| Oxacillin | 14 (100.0) | 8 (34.8) | Clindamycin | 6 (42.9) | 4 (17.4) |
| Methicillin | 14 (100.0) | 15 (65.2) | Azithromycin | 11 (78.6) | 5 (21.7) |
| Amoxicillin-clavulanic acid | 13 (92.9) | 5 (21.7) | Linezolid | - | 0 (0.0) |
| Piperacillin-tazobactam | 14 (100.0) | 7 (30.4) | Vancomycin | - | 0 (0.0) |
| Cephalothin | 12 (85.7) | 9 (39.1) | Teicoplanin | 1 (7.1) | 0 (0.0) |
| Cefotaxime | 12 (85.7) | 2 (8.7) | Chloramphenicol | 1 (7.1) | 0 (0.0) |
| Cefoxitin | 14 (100.0) | 6 (26.1) | Tetracycline | 9 (64.3) | 2 (8.7) |
| Meropenem | 4 (28.6) | 0 (0.0) | Gentamicin | 2 (14.3) | 0 (0.0) |
| Ciprofloxacin | 7 (50.0) | 2 (8.7) | | | |

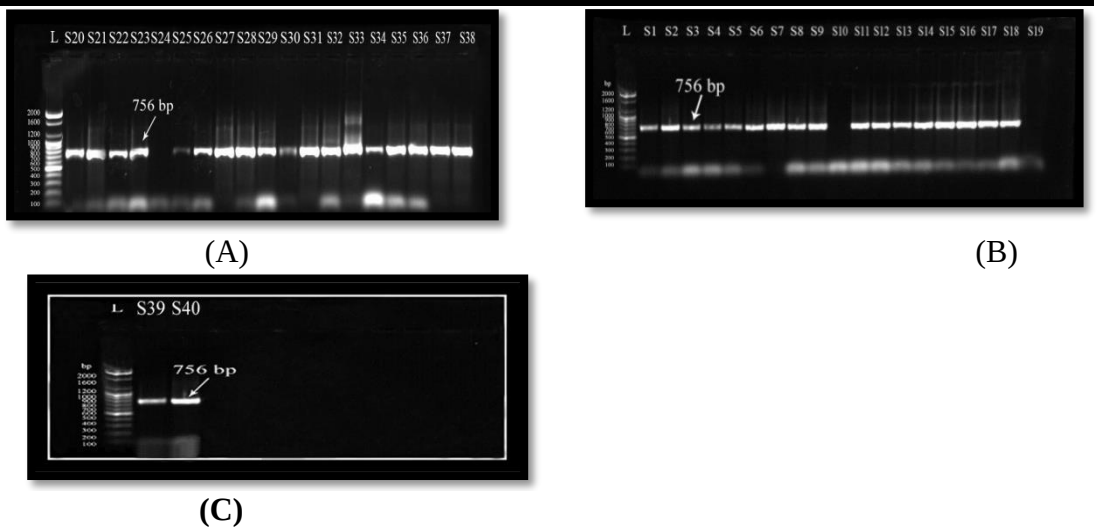


**Figure (1 A, B and C): Ethidium bromide-stained agarose gel of monoplexPCR amplified products from extracted DNA of *S. aureus* isolates and amplified with *16SrRNA* gene primers. The electrophoresis was performed at 70 volt for 1.5 hr. Lane (L), DNA molecular size marker (2000-bp ladder), Lanes 1, 2, 3, 4, 5, 6 ,7,8, 9, 11, 12, 13, 14, 15, 16, 17, 18, 20, 21, 22, 23, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39 and 40 shows positive results with *16SrRNA* gene. Lanes S10, 19 and 24 shows negative results with *16SrRNA* gene.**

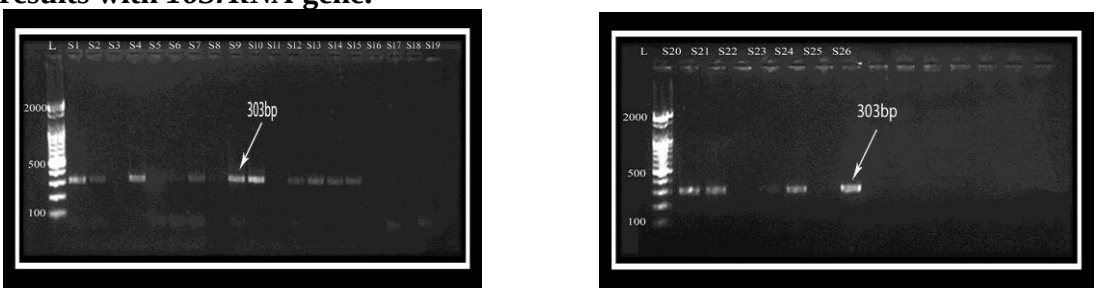


**Figure (2 A and B): Ethidium bromide-stained agarose gel of PCR amplified products from extracted DNA of *S. aureus* isolates and amplified with primer *mecA*. The electrophoresis was performed at 70 volt for 1.5hr. Lane (L), DNA molecular size marker (2000 bp ladder), Lanes (S1, 2, 4, 7, 9, 10, 12, 13, 14, 15, 20, 21, 24 and 26) show positive results with *mecA* gene (303 bp), Lanes (S3, 5, 6, 8, 11, 16, 17, 18, 19, 22, 23, 25, 27, 28, 29, 30,31, 32, 33, 34, 35, 36, 37, 38, 39 and 40) show negative results with *mecA* gene.**

## Discussion

Psoriasis is a chronic immunologically mediated inflammatory disease of the skin, which has been assessed to affect 1-2% of the general population. Several researchers reported that psoriasis occurs in any gender or race with equal amount of male and female [13, 14]. The present study showed a slightly female predominance. Psoriasis can manifest at any age, from infancy to old age [15]. In this study, psoriasis was found to be higher in patients of age groups 21-30 and 31-40 years old; the numbers of patients in these age groups were 40/70 (57.1%). This result corresponds to an Egyptian study mention where psoriasis was higher in individuals of age group 18-40 years old (49, 62.8%) and in both sexes (28 males and 21 females) [16]. This finding was also in accordance with what was detected by other study [17, 18]. In the

studied psoriatic patients, our research isolated *S. aureus* in 40 (57.1%) of all the skin lesions examined from which 37 (92.5%) were positive by PCR for gene encoding the 16SrRNA and confirmed as *S. aureus*. The present findings were unexpected, as the idea that psoriatic plaques may be heavily colonized by staphylococci had seemed a reasonable one. The disease, with its obvious disturbance of normal skin structure, might well have lacked some of the factors, which normally help to rid the skin of bacteria[19]. The results concur with previous studies [20, 21, 22] who found that the cultivation in psoriatic lesional skin was significantly higher than that in normal skin. The rate of isolation of *S. aureus* from psoriatic skin lesion in this study was also correlated to other previous reports [21, 22, 23, 24] that showed about 50% of all the psoriatic cases were colonized with *S. aureus* isolates. The presence of *S. aureus* colonization in lesional skin of psoriasis patients may due to contamination from carriage sites[25]. However, the colonization rate of *S. aureus* in psoriasis has been reported to be between 27.0%-33.0% in other studies[26, 27] which is lower than present study. This variation in isolation of *S. aureus* may be explained by the effect of several factors like environmental factors, personal immunological background, genetic susceptibility, personal hygiene on colonization of psoriatic patients and sampling method.

In this study asserts that an investigation of the antibiotic-resistance profiles of these isolates may serve as a tool in assessing the health risks that humans may encounter when infected by antibiotic-resistant isolates. However, *S. aureus* can adapt rapidly to the selective pressure of antibiotics. Antibiotic resistance and multi-antibiotic resistance have been developed in *S. aureus*, which made the treatment of *S. aureus* infections more difficult [7, 8]. Penicillin G was the drug to which a large proportion of the isolates were resistant. The study showed 91.9% of the isolates were resistant to this drug. Previous studies in Najaf have already discussed the resistance of *S. aureus* to penicillin, [28, 29] found that 90.8% and 83.3% of *S. aureus* isolated from hospitals and holy shrine respectively were resistant to this antibiotic. In response to β-lactam chemotherapy, *S. aureus* has sequentially acquired resistance genes, *blaZ*, which codes for a β-lactamase and confers resistance to penicillins only[30]. However, 64.9% of isolates were confirmed as MDR. The occurrence of MDR *S. aureus* isolates in Najaf hospitals setting is a serious issue that requires urgent attention.

According to cefoxitin resistance results, 54.1% of the isolates were phenotypically considered as MRSA. One study reported that cefoxitin disk diffusion test was 100% sensitive for MRSA detection. Alternatively, 78.4% and 59.5% isolates were resistant to methicillin and oxacillin, respectively[31]. This means that disk diffusion testing using cefoxitin disk is far superior to most of the currently recommended phenotypic methods and is now an accepted method for the detection of MRSA by many reference groups including CLSI (2010)[10].

*mecA*-based PCR methods has accepted as "gold standard" for detection of MRSA [9,32,33]. In this work, among the 37 isolates, 14 (37.8%) were *mecA* positive, indicating that *mecA* is responsible for methicillin resistance in those isolates. In this study, MRSA lacking *mecA* gene are classified as false resistant by the oxacillin and cefoxitin disk diffusion method and it was considered that it may due to another resistance mechanism such as hyperproduction of β-lactamase. The study was possible to conclude that *mecA* gene negative cefoxitin and oxacillin resistant isolates were potentially MRSA[34]. Additionally, previous investigation reported that found among the 13 *S. aureus* strains as methicillin resistant phenotypically, only 4 (30.7%) were positive for *mecA* gene[35]. The expression of *mecA* gene makes MRSA isolates resistant to all types of β-lactam, in addition to many other antibiotics[10]; these may

lead to a difficult treatment of skin lesion and may increase severity of the disease. In one study reported that the present of MRSA isolates and its relation with psoriasis disease might be attributed to role of *S. aureus* in triggering and exacerbation of psoriasis[36]. Moreover, other study found that *mecA* was recognized in 22.6% *S. aureus* isolated from patients with psoriasis[22]; this finding support a potential association of the *mecA* gene with psoriasis, and reported that methicillin resistance gene (*mecA*) may be associated with psoriasis.

The study concluded that *S. aureus* isolated from lesional skin of psoriasis patients have a relationship with psoriasis and there was association between the resistant of these bacteria that harbored mecA gene compared with mecA-gene negative isolates in patients with psoriasis.

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# مدى مقاومة المضادات الحيوية للمكورات العنقودية الذهبية المعزولة من منطقة الجلد المصاب للمرضى المصابين بداء الصدفية

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**جامعة الكوفة \ كلية الطب**

## الخلاصة

ان عزل بكتريا المكورات العنقودية الذهبية من المرضى المصابين بداء الصدفية يعود الى اختزال عوامل الدفاع الموقعية في منطقة الاصابة نتيجة العقاقير المثبطة مناعيا مثل العقاقير السامة للخلايا والستيرويدات التي تستخدم لعلاج الصدفية . تهدف هذه الدراسة لفحص مدى قابلية المقاومة للمضادات الحيوية لبكتريا المكورات العنقودية الذهبية المقترنة بداء الصدفية. لقد تم اخذ مسحات جلدية من منطقة الاصابة لمرضى الصدفية لعزل البكتريا العنقودية الذهبية. حددت العزلات بواسطة الاختبارات التقليدية وعززت بالطرق الجزيئية باستخدام البادىء 16SrRNA المتخصص .بينت النتائج ان من بين كل المرضى هناك 37 (9و52%) عزلة حددت بانها مكورات عنقودية ذهبية. اختبرت جميع العزلات لحساسيتها للمثسلين باستعمال اختبار الحص البدائي وقد تبين ان هناك 34 (91.9%) عزلة مقاومة للبنسلين و 29(78.4%) عزلة مقاومة للمثسلين و 22(59.5%) عزلة مقاومة للاوكساسلين.اظهر جميع العزلات حساسيتها لكل من الفنكومايسين والليزوليد. ثم عززت هذه النتائج بتحديد الجينوتايب لوجود جين mecA وقد اظهرت الدراسة 14 عزلة حاملة لهذا الجين وقد لوحظ ان هناك فرق لمقاومة المضادات الحيوية بين العزلات الحاملة لهذا الجين والعزلات الفاقدة له. توصلت هذه الدراسة الى الاستنتاج بان بكتريا المكورات العنقودية المعزولة من منطقة الجد المصابة لمرضى داء الصدفية لها علاقة بالمرض وهناك ارتباط معنوي بين مقاومة هذه البكتريا التي تمتلك mecA جين وبين داء الصدفية . حيث تبين ان لهذه البكتريا دور في زيادة حدة هذا المرض عند امتلاكها للmecA جين.