# Antibiogram in Community Acquired Skin and Subcutaneous Infections due to *Staphylococcus Aureus*

I HUSSAIN F U REHMAN Z RANI T S HAROON
*Department of Dermatology, King Edward Medical College/Mayo Hospital, Lahore.*
*Correspondence to Dr. Ijaz Hussain Assistant Professor of Dermatology*

**We isolated 25 strains of *Staphylococcus aureus* causing skin and subcutaneous infections in community and determined their β-lactamase activity and *in vitro* sensitivity to 10 antibiotics. The majority (72%) were β-lactamase positive. The antibiogram revealed sensitivity to pencillin in 28%, erythromycin in 60%, amoxycillin-clavulinic acid in 64%, gentamicin in 72%, cefoperazone in 72%, ceftazidime in 80%, ceftriaxone in 84%, oxacillin in 88%, clindamycin in 96% and vancomycin in 96% of patients. The study suggests that majority of isolates of *S. aureus* were sensitive to commonly used antibiotics barring penicillin.**

**Key words: Antibiogram, skin and superficial infections, staphylococcus aureus**

The skin and subcutaneous infections (SSIs) constitute a common group of dermatological infections. Aerobes and facultative anaerobes especially staphylococci and strepococci are the usual causative pathogens. According to the previous work, *Staphylococcus aureus* is the main pathogen of SSIs.[1-4] Generally, systemic antibiotics are used for treatment purposes. According to an estimate over 250 antibiotics of different classes are available in the market world over.[5] Overuse of antibiotics by medical practitioners, quacks, and due to self-medication has given rise to resistant strains of bacteria. The present study focused on the isolation of different strains of *S. aureus* causing community-acquired SSIs and their sensitivity to 10 commonly used antibiotics namely, penicillin, erythromycin, amoxycillin-clavulinic acid, gentamicin, oxacillin, ceftriaxone, cefatazidime, cefoperazone, clindamycin and vancomycin.

## Patients and methods

In this pilot study, 25 pus specimens from 20 patients (10 males and 10 females; mean age 27.7±11.7 years), suffering from community-acquired skin and subcutaneous infection, were taken. The patients who were admitted in hospitals or who had used systemic/local antibiotics within last 72 hours were not included. Specimens were taken by swabs after removing crusts; or needle aspiration where overlying epidermis was intact. These were cultured for aerobic and anaerobic pathogens. Isolated colonies of *S. aureus* were further subcultured on blood agar plates incubated at 37°C aerobically and then examined at 24 and 48 h. β-lactamase activity was determined using the chromogenic cephalosporin analogue 87/312 methodology.[6] Antibiotic sensitivities were determined by diffusion method as described by Bauer and Kirby.[7] Different antibiotics used for this purpose were penicillin, erythromycin, amoxycillin-clavulinic acid, gentamicin, oxacillin, ceftriaxone, cefatazidime, cefoperazone, clindamycin and vancomycin.

## Results

Twenty-five isolates of *S. aureus* were identified on culture. These were grown from lesions of furuncle (n=9), folliculitis (n=4), paronychia (n=3), abscesses (n=3), impetigo (n=2), ulcers (n=2), superficial burns (n=1), and cellulitis (n=1). The different sites of infection were lower limbs (n=8), trunk (n=7), upper limbs (n=4), nailfolds (n=3), and face (n=3).

Of these 18 (72%) strains were β-lactamase positive and 7 (28%) did not show β-lactamase activity. The antibiotic sensitivity/resistance of different strains is shown in Table 1. The antibiogram revealed that three-fourth of isolates were resistant to penicillin and about one-third were resistant to erythromycin and amoxycillin/clavulinic acid. Resistance to gentamicin, cefoperazone, ceftazidime, ceftriaxone and oxacillin was occasional. Although resistance to clindamycin was rare, more than 50% showed intermediate sensitivity. Almost all isolates were sensitive to vancomycin.

Table 1 Antibiotic sensitivity of *S. aureus* isolates (n=25)

| Antibiotic | Sensitive n (%) | Inter-mediate sensitive n (%) | Resistant n (%) |
|---|---|---|---|
| Penicillin | 4 (16) | 3 (12) | 18(72) |
| Erythromycin | 9 (36) | 6 (24) | 10 (40) |
| Amoxy/clavulin* | 10 (40) | 6 (24) | 9 (36) |
| Gentamicin | 11 (44) | 7 (28) | 7 (28) |
| Cefoperazone | 9 (36) | 9 (36) | 7 (28) |
| Ceftazidime | 10 (40) | 10 (40) | 5 (20) |
| Ceftriaxone | 10 (40) | 11 (44) | 4 (16) |
| Oxacillin | 19 (76) | 3 (12) | 3(12) |
| Clindamycin | 9 (36) | 15 (60) | 1(4) |
| Vancomycin | 20 (80) | 4 (16) | 1(4) |

* Amoxycillin/clavulinic acid

## Discussion

In addition to the primary and secondary SSIs, skin and subcutaneous tissues might be a target to various exotoxins

produced by *S. aureus* e.g. toxic shock syndrome toxins I and II, exfoliatin etc. Recent data suggests that this organism may enhance the inflammatory process by superantigen-mediated T-cell activation and may have aggravating role in atopic dermatis[8], psoriasis and cutaneous T-cell lymphoma[9]. This necessitates systemic antibiotic for infections caused by *S. aureus*. The different antibiotics recommended for the treatment of *S. aureus* include penicillins, cephalosporins, macrolides, clindamycin, quinolones and vancomycin. We selected 10 commonly prescribed antibiotics for SSIs to determine the sensitivity of *S. aureus* to these agents.

The present study highlights that majority of the isolated strains of *S. aureus* were β-lactamase positive; hence the majority were resistant to penicillin, a β-lactamase-sensitive antibiotic. The integrity of β-lactam ring is essential for the antibiotic activity. Such resistance is carried by plasmids and is acquired by transduction[10]. Erythromycin and amoxycillin-clavulinic acid are considered effective against β-lactamase-producing pathogens. However, about one-third of isolates showed resistance to them. These two agents are commonly prescribed to out-patients. This figure is close to that by Misko et al, who reported 26% of *S. aureus* strains resistant to erythromycin[11].

Other antibiotics like gentamicin, ceftazidime, cefoperazone, ceftriaxone, clindamycin, oxacillin and vancomycin exhibited high *in vitro* antistaphylococcal activity. Resistance to vancomycin was rare. These results are in agreement with some studies but differ from others. Nishijima et al, determined *in vitro* susceptibility of 130 isolates of *S. aureus* to 19 antibiotics and reported 19.2% of isolated strains resistant to methicillin.[1] In another study by the same researchers, resistance was the highest to gentamicin and lowest to ofloxacin.[12] The antibiotic sensitivity of different strains may vary in different populations and even in the same population at different times.[10] The sensitivity/resistance pattern in hospital-acquired infections is expected to be quite different.

According to our results, the majority of *S. aureus* strains in our community are β-lactamase positive and resistant to benzyl penicillin. However, other groups of antibiotics like cephalosporins, gentamicin and amoxycillin-clavulinc acid are still effective in majority of cases. Needless and excessive therapy should be avoided and newer antibiotics should be reserved for non-responders or hospital-acquired infections to circumvent the problem of emerging resistant strains of *S. aureus*.

**References**


1. Nishijima S, Nakagawa M, Sugiyama T, et al. Sensitivity of *Staphylococcus aureus*, isolated from skin infections in 1994, to 19 antimicrobial agents. J Int Med Res 1995; 23: 328-34.
2. Brook I. Microbiology of secondary bacterial infections in scabies lesions. J Clin Microbiol 1995; 33: 2139-40.
3. Summanen PH, Talan DA, Strong C, et al. Bacteriology of skin and soft-tissue infections: comparison of infections in intravenous drug users and individuals with no history of intravenous drug use. Clin Infect Dis 1995; 20 (Suppl 20): S279-82.
4. Powers RD. Soft tissue infections in the emergency department: the case for the use of simple antibiotics. South Med J 1991; 84: 1313-5.
5. Harris BD, Adal KA, Tomecki KJ. New and emerging therapies in infectious diseases. Dermatol Clin 2000; 18: 31-35.
6. O'Callaghan DH, Morris A, Kirby SM, Shingler AH. Novel method for detection of beta-lactamase by using a chromogenic cephalosporin substrate. Antimicrob Agents Chemother 1972; 1: 283-8.
7. Baker FJ, Breach MR, eds. Medical Microbiological Techniques. London: Butterworths & Co., 1980: 343-50.
8. McFadden JP, Noble WC, Camp RD. Superantigenic exotoxin-secreting potential of staphylococci isolated from atopic eczematous skin. Br J Dermatol 1993; 128: 631-2.
9. Chakravatri D, LaMorte VJ, Nelson MC, et al. Role of CBP/P300 in nuclear receptor signalling. Nature 1996; 383: 99-102.
10. Sherris JC, ed. Medical Microbiology: An introduction to infectious diseases. Staphylococci. New York: Elsevier Science Publishing Co., 1984: 150-61.
11. Misko ML, Terracina JR, Diven DG. The frequency of erythromycin-resistant *Staphylococcus aureus* in impetiginized dermatoses. Pediatr Dermatol 1995; 12: 12-5.
12. Nishijima S, Namura S, Kawai S, et al. Sensitivity of *Staphylococcus aureus* and *Streptococcus pyogenes* isolated from skin infections in 1992 to antimicrobial agents. J Dermatol 1994; 21: 233-8.