



# Cutaneous bacterial infections: Changing trends in bacterial resistance

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Normal skin is colonized with resident bacterial flora, usually *Staphylococcus epidermidis*, other coagulase-negative *Staphylococci*, *Corynebacteria* and *Propionibacterium acnes*. These bacteria form a protective layer and prevent the adhesion and multiplication of potential pathogens.

Cutaneous infections arise whenever there is a break in the continuity of the skin or as a part of systemic infection. The spectrum of bacteria associated with primary cutaneous infections has remained the same over the years though the frequency of their association has changed. *Staphylococcus aureus*, *Streptococci*, *Corynebacterium spp*, *Erysipelothrix*, *Enterobacteriaceae*, *Pseudomonas* and anaerobes have been implicated in a variety of cutaneous infections with polymicrobial infections also being reported. The aerobic Gram-positive cocci accounted for more than 60% of cutaneous infections in the 70's. Though *Staphylococcus aureus* continues to be the predominant pathogen, *Enterobacteriaceae*, *Pseudomonas* and *Enterococci* are being increasingly reported.<sup>1,2</sup> This spectrum is seen even in HIV-infected patients and intravenous drug users.<sup>3-5</sup>

Infections caused by antibiotic resistant strains have become a global problem. The increasing prevalence of multi-drug resistant organisms with few or no treatment options such as methicillin resistant *Staphylococcus aureus* (MRSA), vancomycin resistant

enterococci (VRE) and the extended spectrum beta-lactamase (ESBL) producing Gram-negative bacilli both in hospitalized patients and, to a lesser extent, in the community are a serious cause for concern. This trend in antibiotic resistant strains is true for infection at any site of the body.

In order of frequency, *Staphylococcus aureus* accounts for 30-50% of skin and soft tissue infections, followed by the *Enterobacteriaceae*, non-fermenters, *Streptococci* (beta-hemolytic Group A and others) and anaerobes.<sup>6,7</sup> On an average more than 90% of *Staphylococcus aureus* elaborate penicillinases or beta-lactamases (a trend seen even in community acquired strains) and 20-30% of *Staphylococcus aureus* are methicillin resistant.<sup>8</sup> The prevalence of MRSA in India is also on the rise and there are reports of detecting MRSA in community-acquired infections though the prevalence is much lesser.<sup>9,10</sup> MRSA strains also demonstrate a high degree of resistance to other antibiotics especially beta-lactams, erythromycin and aminoglycosides. This changing trend calls for changes in empirical therapy based on current susceptibility patterns. Beta-lactams or erythromycin cannot be considered as the standard treatment options today, especially in hospitalized patients. A combination of beta-lactam/beta-lactamase inhibitor (amoxicillin/clavulanic acid, ampicillin/sulbactam) will elicit a better response. Vancomycin is considered as the drug of choice for the treatment of infection due

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to MRSA but not for carriage or colonization.<sup>8</sup> Favorable outcomes have also been reported with the use of teichoplanin and linezolid (an oxazololidinone). Local eradication is usually achieved with mupirocin, bacitracin or chlorhexidine.

Though there are no reports of penicillin resistant *Streptococcus pyogenes*, these organisms are demonstrating increasing tolerance to this antibiotic. ESBLs are beta-lactamases that hydrolyze extended-spectrum cephalosporins with an oxyimino side chain. These cephalosporins include cefotaxime, ceftriaxone, and ceftazidime, as well as the oxyimino-monobactam aztreonam.<sup>11</sup> Between 10% and 60% of enterobacteriaceae associated with skin and soft tissue infections have been reported as ESBL producers.<sup>1,2,6,7</sup> Beta-lactamase inhibitors such as clavulanate, sulbactam or tazobactam in vitro inhibit most ESBLs, but the clinical effectiveness of beta-lactam/beta-lactamase inhibitor combinations cannot be relied on consistently for therapy. In fact, the therapy of choice for infections caused by ESBL-producing members of the *Enterobacteriaceae* is a carbapenem.<sup>11</sup>

The presence of open skin lesions in hospitalized dermatology patients predisposes them to colonization with resistant organisms, which, if not adequately cared for, may lead to infection. The time is therefore apt not only to treat the infection and eradicate the colonization or carriage but also to educate the patient at the time of discharge about the appropriate use of antibiotics and proper wound care. This will reduce the chances of dissemination of resistant pathogens in the community.

It is widely acknowledged that the rapidity of development of resistance to an agent increases with the magnitude of its use. Therefore newer agents should be used judiciously only on isolation of a resistant pathogen from definite infection and not for treatment of colonization or contamination.

Thus the antimicrobial options for treatment of cutaneous infections have undergone a sea change. In choosing the appropriate antimicrobial therapy, one must take into account the resistance profile of the

infective agent, the antibacterial profile of the antimicrobial agent and its pharmacokinetic properties. A combination of beta-lactam/beta-lactamase inhibitor may be a better option in community acquired infections, whereas in hospitalized patients culture sensitivity reports will be essential to decide on therapy.

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