Community-Associated Methicillin-Resistant *Staphylococcus aureus*: Is It in Your Community and Should It Change Practice?

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In this issue of *Annals*, Frazee et al1 have reported a phenomenon that many emergency physicians have already experienced—community-associated methicillin-resistant *Staphylococcus aureus* (MRSA) now appears to be among the most common etiologies of skin and soft tissue infections. This study is a good example of how emergency department (ED) surveillance can be critical for making sentinel observations of community events on the basis of examination of clinically important syndromes. Past reports have noted increased proportions of *S. aureus* isolates that are community-associated MRSA from specimens submitted to microbiology laboratories, but these studies suffer from biases associated with culture acquisition, poorly defined patient groups, and lack of prospective data collection to confirm absence of MRSA risk factors (eg, nursing home residence) in order to establish prevalence rates in otherwise healthy community members.2 Because this study is from one urban center with a large proportion of higher-risk patients such as intravenous drug users and the homeless, we must be cautious in assuming that its findings can be generalized to other areas. However, considering the increasing reports from various sources and sites, it appears that MRSA may be replacing methicillin-susceptible *S. aureus* (MSSA) as the typical community staphylococcal strain.

Antimicrobials that appear to have in vitro activity against US community-associated MRSA isolates include vancomycin, clindamycin, trimethoprim-sulfamethoxazole, rifampin, and linezolid. Most MRSA isolates are resistant to macrolides and quinolones, and many are resistant to tetracyclines. As mentioned by Frazee et al,1 some macrolide-resistant strains of MRSA have what has been termed “inducible” clindamycin resistance. This is found by the performance of a “D-test” in the microbiology laboratory, a procedure that is not routine in many centers. There have been case reports of treatment failures or recurrences associated with clindamycin therapy for these strains, but it appears that most patients will recover with clindamycin therapy. Concern about inducible clindamycin resistance is probably not a reason to completely avoid clindamycin for skin and soft tissue infections. Although data on treatment of community-associated MRSA–related skin and soft tissue infections are sparse, one study demonstrated that clindamycin can be used successfully for invasive community-associated MRSA infections in children.3

We know that antibiotics such as cephalexin are inactive in vitro against community-associated MRSA, but even in areas with a high prevalence of community-associated MRSA (eg, >50% of culturable infections), it is unclear that we need to change our empiric antimicrobial therapy for skin and soft tissue infections. Most skin abscesses, even when caused by MRSA, can be cured with adequate drainage in the absence of antimicrobials. A study of MRSA abscesses in immunocompetent children found no difference in cure rates between those with organisms susceptible or resistant to prescribed antimicrobials.4 We are not aware of any published comparative investigations that demonstrate a clinical outcome advantage among patients with MRSA skin and soft tissue infections who were treated with an antimicrobial with in vitro MRSA activity compared with another lacking this activity (eg, vancomycin versus cephazolin intravenously or clindamycin versus cephalixin orally). However, there are currently many newer antimicrobials (eg, linezolid, daptomycin, dalbavancin, oritavancin) that possess in vitro MRSA activity that are in or have recently completed Phase III clinical trial evaluation, which may soon shed some light on this issue.

Nonetheless, it is difficult to justify using drugs like cephalexin empirically if it is known that the majority of patients will be infected with resistant isolates. Antimicrobial selection may have more of an impact on outcomes with cellulitis and open wound infections, because drainage will not be the main treatment. However, it is likely that a greater proportion of cellulitis is caused by *Streptococcus pyogenes.*5 Although clindamycin has in vitro activity against *S. pyogenes,* many are resistant to trimethoprim-sulfamethoxazole.6 Because of concern about *S. pyogenes,* including cephalexin in a trimethoprim-sulfamethoxazole regimen or using clindamycin may be preferable for cellulitis without abscess.

Despite the lack of good studies for this scenario, emergency physicians still have to make decisions regarding empiric antimicrobial treatment in patients for whom MRSA is a possibility. Culturing these infections has now become more important; first, to help evaluate and follow the local MRSA prevalence, and second, to ensure that patients who do not improve can be given an antibiotic with in vitro activity. In areas in which MRSA prevalence is low or unknown, it is still
reasonable to give cephalixin to many of these patients, accompanied by instructions to return if there is no improvement. Treatment could be adjusted on the basis of culture results at that point. In areas with a high community-associated MRSA prevalence, a change in empiric therapy to a combination of trimethoprim-sulfamethoxazole and rifampin should be considered. Whether the addition of rifampin provides any benefit in this situation is unclear. Use of this combination is based on extrapolation from studies showing that eradication of nasal carriage of MRSA is greater with combination therapy including rifampin.\(^7\) Aside from the risk of drug interactions, rifampin is an inexpensive and safe drug. Elimination of nasal carriage does appear to reduce the likelihood of recurrent furunculosis\(^8\) and may be an additional benefit of adding rifampin. Oral clindamycin alone is another option, but it is more expensive, must be dosed more frequently, and may be associated with a higher incidence of adverse reactions. For patients with skin and soft tissue infections requiring hospital admission, intravenous clindamycin is a reasonable alternative to vancomycin, although perhaps not as well studied for severe infections. In areas with community-associated MRSA, it is now appropriate to include vancomycin in the empiric regimen for patients with any life-threatening infection that may be due to \(S\) aureus, including severe pneumonia, endocarditis, and sepsis. 

Once an MRSA infection is identified, patients are typically placed into contact isolation in private rooms, with gowns and gloves used by health care workers.\(^9\) In many institutions, this now appears to be somewhat of a farce, and contributes to the broader problem of ED crowding with patients awaiting hospital admission. It is not practical in most institutions to initiate isolation for all patients admitted with skin and soft tissue infections. We find ourselves admitting these patients to nonisolation rooms, only to have them placed in isolation 2 days later when MRSA is confirmed. When these strains are so prevalent in the community, it becomes less clear that contact isolation in the hospital will have an impact. Thus, in communities with a high prevalence of community-associated MRSA, maintaining contact isolation solely on the basis of finding MRSA is now questionable despite current guidelines. Whether the infection is caused by MRSA or MSSA, standard precautions should be used for any patient with a purulent wound to prevent exposing other patients or personnel to infected material.

What is abundantly clear is that we need more research on this new phenomenon of community-associated MRSA. Specifically, broad geographic and prospective epidemiologic studies of ED patients with clinically important skin and soft tissue infections should be conducted to better define the extent of community-associated MRSA and patterns of in vitro antimicrobial susceptibility. As opposed to typical Phase III prelicensing trials that often include many patients with abscesses, meaningful comparative antimicrobial studies should be conducted that exclude patients with infections that do not require antibiotics and focus on those that do (eg, purulent open wound infections).\(^10\) The role of eliminating colonization with nasal mupirocin or antiseptic skin cleansers is unclear in community-associated MRSA-related skin and soft tissue infections.\(^11\) Finally, the impact of (perhaps outdated) infection control practices on nosocomial MRSA transmission and more global outcomes, such as ED flow, need to be evaluated.

**Funding and support:** The authors report this study did not receive any outside funding or support.

**Publication dates:** Available online January 14, 2005.

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