Cellulitis: getting it right

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Tellulitis seems at first glance to be a straightforward infection that should respond to readily available antibiotics, guaranteeing excellent outcomes. Certainly most emergency physicians treat it as such, giving it little extra thought in their busy clinical workload. Yet, in this issue,

Murray and colleagues have prospectively identified an overall failure rate of 18.7% when treating cellulitis in the outpatient setting.1 There is no way to know if this is a high or low rate of failure, for we appear to lack historical data describing treatment failure rates for inpatients. It is entirely possible that the identified failure rate may be the best we can achieve within practical

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the clinician — at times I wonder if I know anything at all.

We have once again learned that what was thought to be simple bly attaining higher tissue levels in their study; there again levels were not measured.2 It would be interesting to see if the IV treatment failures in Murray and colleagues' study clustered more in the cefazolin/probenicid arm than the ceftriaxone arm, for we are reasonably certain about 24-hour tissue levels for the latter but not the

bining both oral and intra-

venous (IV) therapy, proba-

practice patterns; then again it might not. As the authors state, the emergency medicine literature has been to date woefully inadequate in studying this change in clinical practice. We can only hope that this lacune will soon be corrected.

There is no magic in admitting a patient: irrespective of venue, if (infected) tissue levels of antibiotics surpass the established minimal inhibitory concentration for greater than 60% of the time, treatment success should be assured. If emergency physicians treated patients with the same antibiotic regimen as presently performed on the ward, they former when providing a once-a-day regimen.

This study did not identify pathogens or their resistance patterns. This is not a criticism, for it is a difficult process at best for cellulitis, and rarely done in the emergency department with immunocompetent patients. It raises the question, however, whether some of the treatment failures were due to bacterial resistances, or whether bacteria other than those covered were responsible. When considering any standardized algorithm, these questions will have to be addressed. This has become crucial with the rapid emergence of community-based MRSA (methi-

should expect to have the same results. Unfortunately, an

observational study such as the one by Murray and col-

leagues could not establish that patients receiving therapy

achieved the criterion of adequate tissue levels. Brown and

associates reported a much lower failure rate when com-

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Received: May 2005; accepted: June 7, 2005

Can J Emerg Med 2005;7(4):235-6

cillin-resistant Staphylococcus aureus) skin infections.3

We require even more inclusive studies than this one if we are to define severity. Murray and colleagues state that there was poor inter-rater reliability in judging severity. They have made this observation after having excluded from their cohort almost all patients we would normally consider at risk: diabetics, immunocompromised patients, or those they considered a priori to have a severe infection. It might be difficult to further break down grades of severity when the above groups are excluded. Given the unreliability of "severity assessment," future studies should not exclude those traditional high-risk patients, or at least should limit exclusion criteria to enroll the most externally valid patient sample.

We have once again learned that what was thought to be simple is not. Evidence is required to better guide the clinician — at times I wonder if I know anything at all.

Competing interests: None declared.

Key words: cellulitis; therapy

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