Skin Infections From Community MRSA Rising

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SAN FRANCISCO — Community–associated methicillin-resistant *Staphylococcus aureus*—almost unheard of 10 years ago—has become the single biggest cause of skin infections in the United States, Dr. Greg Moran said at the 12th International Conference on Emergency Medicine.

"We really don't know what's begun this sudden explosion of resistant staph in the community all over the United States, as well as in Canada and Europe," said Dr. Moran, an emergency physician at the Olive View–UCLA Medical Center, Sylmar, Calif. "One thing we do know is that this is not a phenomenon of the hospital strains moving into the community. These are genetically distinct strains."

The hospital strains are usually USA100 and 200, while the overwhelming majority of the community strains are USA300. In his 2006 study, virtually all skin infections cultured from hospitals in 11 cities across the country were caused by community-associated strains; 78% of those were a single clone of USA300. "There is something about this strain that has given it a very, very strong survival advantage in the community," Dr. Moran said. "Almost all of [the skin infections] (98%) carried the Panton-Valentine leukocidin toxin gene and the SCCmec type IV gene."

The SCCmec gene confers methicillin resistance, while the Panton-Valentine leukocidin toxin gene is associated with spontaneous skin and soft-tissue infections, as well as necrotizing pneumonia. The mutations make the community-associated MRSA strains much more likely to cause infections than those MRSA strains found in hospitals, he said.

In addition to authoring a seminal paper on the topic (N. Engl. J. Med. 2006;355:666-74), Dr. Moran has kept track of the MRSA skin infections occurring in his own hospital since 1997. There were 25 cases documented that year. "That number rose to almost 450 per year in 2006 and 2007," he said. "In 2001, 29% of our skin infections were MRSA. That more than doubled by 2003-2004, to 64%. In a very short time, we went from something we virtually never saw in the community, to it being the single largest cause of skin infections."

A few clinical features are associated with an increased risk of the community-associated MRSA infections, Dr. Moran said, including recent antibiotic use, abscess, a history of "spider bite" (insect bite of unknown origin), prior MRSA infection, and close contact with a MRSA-infected individual. But none of those was a strong predictor.

"The reality is you can't use any of these risk factors to decide who you're going to treat for MRSA," he said. Despite their prevalence, most of these infections are not serious and don't grow the "killer flesh-eating super bugs" touted in grocery store tabloids, Dr. Moran said. "We still have a number of antibiotic options. More than 90% of the isolates in our study were susceptible to at least one agent."

Dr. Moran made these comments about the available antibiotic choices:

► **Vancomycin.** "Even though this is the gold standard, we are now recognizing its limitations. We are seeing more resistance to this than we used to."

► **Clindamycin.** "Ninety-five percent of the isolates were susceptible to this in our study, although that appears to be decreasing. In our hospital, susceptibility is now down to about 85%."

► **Linezolid.** "It's very effective, but also very, very expensive. Post hoc data suggest that it may be clinically superior for hospital-acquired MRSA pneumonia, but there are no prospective data on this. It's a good drug, but I think it's prohibitively expensive."

► **Daptomycin.** "Very good for skin infections, but we don't use it for MRSA pneumonia—it binds to the pulmonary surfactant and is inactivated."

► **Tigecycline.** "This is a good choice when you want both gram-negative and gram-positive activity."

► **Trimethoprim sulfa.** "This is close to 100% effective in vitro, but there isn't much clinical data for its use in skin infections."

► **Tetracycline.** "It's a cheap generic with reasonable effectiveness."

Current studies conclude that there's no real benefit to adding antibiotics to the treatment regimen, he said. However, there are many limitations to those studies: Many had small treatment numbers and were done before the MRSA phenomenon, he added. Therefore, more aggressive treatment may be warranted now. "The truth is, we don't know the answer."

For most uncomplicated skin infections, he performs an incision and drainage, and doesn't give antibiotics. However, "I do give antibiotics if there is a fever, significant associated cellulitis, immune or vascular compromise, if the lesion is in a high-risk area like the hands or face, or if the patient has already failed an incision and drainage," he explained. ■

THE EFFECTIVE PHYSICIAN

Evaluating New ICU Fever

BY WILLIAM E. GOLDEN, M.D., AND ROBERT H. HOPKINS, M.D.

Background

Managing critically ill adults is a complex and resource-intensive undertaking. The American College of Critical Care Medicine and the Infectious Diseases Society of America recently updated guidelines for the evaluation of fever in the ICU to assist in cost-effective, targeted patient care.

Conclusions

Measurement by pulmonary artery thermistor is the gold standard for core temperature. Comparably accurate temperature measurements can be obtained from bladder catheter thermistors, esophageal probes, or rectal probes. Infrared ear thermometry and oral probes are acceptably accurate. Temporal artery thermometers, axillary thermometers, and chemical strips correlate poorly with pulmonary artery measurement. Axillary temperatures should not be used. Rectal thermometers are uncomfortable for the patient and could promote transmission of infectious agents in the ICU.

Antibiotic removal devices in blood cultures remain a controversial technology. They can help identify staphylococcus and yeast, but are not helpful for gram-negative infections.

Most cases of inconsistent blood culture findings reflect blood drawn from an indwelling line turning positive with negative cultures from venipuncture.

Fever is common in the first 48 postoperative hours and usually does not represent infection. Infection is more likely when fever is present at 96 hours and later.

Urine cultures from catheter systems with more than 10^3 colony-forming U/mL are reliable indicators of bacteriuria or funguria, but might not represent the etiology of the patient's fever. Gram stains of centrifuged urine are reliable for identifying predominant infectious agents.

Cytomegalovirus infections can cause hectic fevers after blood transfusion in otherwise asymptomatic, immunocompetent patients. Immunocompromised patients can develop severe sequelae from this condition.

Common causes of noninfectious fever include drug fever, cholecystitis, myocardial infarction, stroke, gout, pancreatitis, pulmonary emboli, transplant rejection, and adrenal insufficiency.

Drug fever can take several days to resolve after discontinuation of the causative agent.

Implementation

In general, patients with temperature higher than 38.3° C (101° F) should have clinical assessment to determine if infection is present. Immunocompromised patients might require a lower temperature threshold. Similarly, patients with new temperatures below 36.0° C (96.8° F) should also be assessed for presence of infection. Patients with stable temperature but with unexplained hypotension, tachycardia, confusion, oliguria, or abnormal white blood cell counts should also have infection in their differential.

Clinical assessment of new fever should replace automatic, standing-order laboratory and radiologic tests in the ICU.

Accuracy of blood cultures depends on the sampling technique and sufficient blood volume, ideally 20-30 mL per site. Yield is optimized by taking three to four blood cultures of adequate volume from different sites in the first 24 hours of bacteremia or fungemia. Blood drawn from multiple ports on the same catheter should not count as separate culture sites. Blood cultures should not be obtained from a peripheral intravenous site at the time of insertion to avoid contamination. All cultures should be carefully labeled with time obtained and collection site.

To assess possible catheter-related sepsis, the catheter should be removed and cultured. Short catheters should have their tips cultured, while longer catheters should have the tip and intracutaneous segments sent to the lab. Pulmonary artery catheters should have the tip and introducer sent for culture. It is rarely necessary to culture infusate specimens.

Chest x-rays should be obtained for suspected pulmonary infections. A specimen from the lower respiratory tract should be obtained and sent to the lab within 2 hours of collection. If appropriate, pleural fluid should be tapped under ultrasound guidance and sent for culture and Gram stain.

Enteric stool cultures are rarely useful for evaluating diarrhea not present on admission.

A single stool specimen for *Clostridium difficile* common antigen, enzyme immunoassay (EIA) for toxins A and B, or tissue culture is usually sufficient to assess for pseudomembranous colitis. A second evaluation is appropriate for persistent symptoms after a negative EIA toxin assay, but is not useful after a negative common antigen test. Empiric treatment is discouraged to avoid the development of antibiotic resistence.

Urine cultures should be sampled from a port in the tubing and not from the collection bag. Specimens should be sent to the lab for plating within 1 hour of collection or refrigerated if testing will be delayed.

Dipstick testing of urine from catheter systems is not useful for decision making.

For patients with suspected sinusitis as a cause of ICU fever, CT scans should be obtained. Patients whose sinusitis is not responsive to antibiotics could benefit from puncture and aspiration of the infected cavity.

Reference

O'Grady NP, et al. Guidelines for evaluation of new fever in critically ill adult patients: 2008 update from the American College of Critical Care Medicine and the Infectious Diseases Society of America. Crit. Care Med. 2008;36:1330-49.

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