Editorial

Environmental Contamination with *Staphylococcus aureus* and Outbreaks: The Cause or the Effect?

Neil L. Barg, MD

Determining whether antibiotic-resistant nosocomial bacteria arise from a common source remains a constant problem for the hospital epidemiologist, particularly in view of the recent emergence of bacterial strains resistant to expanded-spectrum antibiotics such as ceftazidime, imipenam, and mupirocin. The article in this issue by Layton et al[1] describes an outbreak of mupirocin-resistant *Staphylococcus aureus* thought to disseminate from an environmental source.

The epidemiology of infections caused by *S aureus* was well described in studies completed during the 1950s and 1960s, when staphylococci were the predominant nosocomial pathogens. Outbreak strains often were identified through the use of phage typing and antibiograms and thus were discriminated from endemic colonizing isolates. A new phage type, or the sudden appearance of a new antibiotic-resistant strain, was sufficiently unusual to permit such discrimination. The source of staphylococcal strains causing nosocomial outbreaks was often the primary concern of the epidemiologists. Extensive analysis using case-control studies and surveillance cultures of patients, personnel, and environmental surfaces led investigators to conclude that environmental surfaces were not an important source of *S aureus.* One editor cautioned, "Care should be taken to avoid the too facile assumptions that an article carrying staphylococci is necessarily implicated in staphylococcal cross infections."[2] Outbreak strains actually were shown to spread from patient to patient by transient carriage of *S aureus* on the hands of hospital personnel.[3]

The sudden appearance of mupirocin-resistant *S aureus* among patient isolates from a dermatology ward prompted a surveillance study by Layton et al.[1] Strains obtained from patients and environmental surfaces were compared using pulsed-field gel electrophoresis (PFGE). The method chosen to compare strains isolated in this epidemic has been shown by other investigators to accurately discriminate among staphylococcal clones.[4-6] In the present study, a predominant clone of S *aureus* resistant to mupirocin was identified by finding a single PFGE pattern. Strains of the same clone also were found on a blood pressure cuff and a shower stall during a single sweep of environmental surfaces. No S *aureus* isolates were recovered from the hands of sampled personnel. These data contrast with the findings of previous studies. For example, Venezia et al, in a study of a similar S *aureus* outbreak, found the epidemic strain on the hands of one nurse but not on environmental surfaces.[7] What do we make of two studies that investigated a similar phenomenon in a similar patient population, yet implicated different sources for antibiotic-resistant *S aureus?* These studies may reflect accurately different circumstances. On the other hand, this apparent contradiction may exist because of insufficient surveillance. In the present study of mupirocin-resistant *S aureus,* the use of PFGE unequivocally established the similarity of the predominant patient strain to the environmental isolates. However, we cannot determine if the environment was the source of isolates subsequently colonizing patients or if person-to-person spread was the major route of dissemination, with the environment as an innocent bystander. A single sampling of the environment and a single sampling of personnel hands were performed.

From St. Thomas Hospital, Nashville, Tennessee.

Address reprint requests to Neil L. Barg, MD, Assistant Professor of Medicine, St. Thomas Hospital, PO. Box 380, Nashville, TN 3 7202.

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Because only one sampling of each was performed, we do not know how long the environmental surfaces were colonized nor at what point during the epidemic these surfaces became colonized. If other environmental surfaces were colonized during this 14-month outbreak, then the cuff and the shower were less likely to be the predominant sources of the epidemic isolate. In addition, the cuff and the shower were less likely to serve as the source of the epidemic strain if the implicated surfaces were not colonized until late in the epidemic. Environmental contamination with S *aureus* would be expected on a ward of desquamating patients colonized with *S aureus*. To establish the source with more certainty, additional conventional epidemiologic techniques could have been used to support and strengthen the molecular findings. For instance, the authors could have determined whether colonized patients were more likely to have used the implicated shower or blood pressure cuff than noncolonized patients. This would have established the relevance of contact with these objects.

The environment also was considered an important reservoir by the authors because the epidemic strain was not recovered from the hands of nursing personnel. However, hand carriage of staphylococci is recognized as transient. Because of the transient nature of carriage, a second surveillance study, or a more inclusive surveillance, might have recovered the epidemic strain. Other modes of transmission were also possible. Doctor-patient contact or patient-patient contact may have been important. Patients housed on dermatology wards often are ambulatory; colonized patients may have been had frequent contact with noncolonized patients, thus facilitating dissemination. Indeed, each patient who became colonized or infected during this outbreak was concomitantly hospitalized with a prior colonized or infected patient. The authors also thought the environment was a source of this epidemic because the eradication of S *aureus* from environmental surfaces with disinfectants was associated with the decline of the colonization and infection of patients with the epidemic S *aureus* strain. However, coincident with disinfection of the environment, barrier techniques were instituted. Proper implementation of barrier precautions alone may have been sufficient to reduce or eliminate dissemination of epidemic strains of S *aureus*. Finally, follow-up cultures are available for eight months, only slightly longer than the longest interval between colonized or infected patients. Perhaps a longer follow-up period may have shown return of the epidemic strain with the environmental surfaces remaining free of the epidemic isolate.

Therefore, it is plausible that the environmental surfaces passively harbored the outbreak strain on a ward of colonized patients with desquamating skin diseases. Through the use of current molecular techniques that precisely identified the outbreak strain, the authors demonstrated the dissemination of a mupirocin-resistant strain of S *aureus* among dermatology patients with apparent involvement of environmental surfaces. Additional surveillance and case-control associations, implicating an environmental source, would have strengthened the authors' conclusions. Whether the environment served as a reservoir for the dissemination of the epidemic strain in this outbreak remains unresolved.

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