Editorial

Staphylococcal Infections in the Hemodialysis Unit: Prevention Using Infection Control Principles

Joseph W. Chow, MD; Michael Sorkin, MD; Angella Goetz, RN, MNEd; Victor L. Yu, MD

Infections are a leading cause of death in patients on chronic hemodialysis, second only to cardiovascular disease.[1-3] Staphylococcus is the major pathogen and the leading cause of access-site infection, bacteremia, and endocarditis in hemodialysis patients and is responsible for more deaths than any other organism.[3-5] In this issue of *Infection Control and Hospital Epidemiology* (pp 534-541), Kaplowitz et al report that *S aureus* caused four of five bacteremias and seven of nine access-site infections over one year in their group of 71 patients on chronic outpatient hemodialysis. In a companion report of the same study in the *Journal of Clinical Microbiology*[6] the authors assess the relationship between microbial colonization and subsequent access-site infection. Practitioners in infection control should find their extensive study especially heartening in that the philosophy and methods applied derive from classical infection control principles.

The authors emphasize that their study is the first prospective study that provides overall infection rates in hemodialysis patients. Their approach was rigorous. The criteria for infections were clearly defined, and the clinical and microbiologic data were collected prospectively. Finally, an impressive amount of data was compiled (Tables 1, 4. 5, 6). Although well done, what is the value of all the data? We suspect they offer somewhat less than they promise.

From the VA Medical Center and the University of Pittsburgh, Pittsburgh, Pennsylvania.

Address reprint requests to Victor L. Yu, MD, Infectious Disease Section, VA Medical Center, University Drive C, Pittsburgh, PA 15240.

The fatal flaw for most surveillance studies is that the rates are not readily extrapolated to other institutions because of inherent differences in patient populations, definitions of infection, methodology of reporting, and overall medical practice. One might argue that incidence rates **could** be useful within an individual institution where definitions could be standardized and patient populations and medical practice would be stable. We remain skeptical. Outbreaks are rarely identified by noting the current infection rate has exceeded the baseline infection rate; they are usually identified by intuitive and empiric observations of the physicians and nurses caring for these patients.

We are well aware that most readers of this journal would disagree with us on this point since the approach of "surveillance without a hypothesis" is ingrained into infection control. But it is notable that the authors themselves did little with the main data other than to compare their rates to those of other investigators. The rates were either comparable or they were not and little else could be said.

On the other hand, surveillance was only one aspect of a larger study on infections in hemodialysis patients. The other aspects are more important, in our opinion, and we will direct our comments to the implications concerning *S aureus* infections.

The reservoir for *S aureus* is the human being. Staphylococci can be found on the umbilicus and perineum in the neonate and colonizes man throughout adulthood. Person-to-person transfer readily occurs, usually via direct contact, although the organism also can spread into the

https://doi.org/10.1086/645771 Published online by Cambridge University Press

environment by aerosolization. The source of the staphylococci is thought to be the nares. Moss showed in 1948 that reduction of nasal staphylococci with application of topical penicillin was followed by a significant decrease of staphylococci on the skin, whereas failure to reduce staphylococcal nasal carriage corresponded to failure to decrease skin carriage.[7] The ease with which staphylococci can spread to other body parts can be strikingly demonstrated by application of fluorescent dye in the nasal vestibule; within a few hours the dye can be found on multiple areas of the subject's face, hands, and clothing.[8]

Patients on hemodialysis have a significantly higher staphylococcal carriage rate than the general population.[9,10] The reason for the increased carriage rate is uncertain, although theories on immunologic, dermatologic. and mechanical factors have been advanced.[10-12]

It has been presumed that in vascular infections in hemodialysis patients, the *Staphylococcus* in the nares is transmitted to the skin, where it subsequently gains access to the systemic circulation via needle punctures for dialysis. *S aureus* isolated from infected dialysis fistulas are usually identical to the staphylococci on the nose, throat, and skin as determined by phage-typing.[13,14] Hemodialysis patients who are cutaneous carriers are significantly more likely to have staphylococcal infections than noncarriers.[9,14]

We have initiated routine cultures of the nares of hemodialysis patients to monitor their staphylococcal carriage state in an attempt to target a population at high risk for access-site infections. In a limited study, we found the nares appeared to be a more sensitive indicator of staphylococcal carriage than the skin; 52% of'nasal cultures were positive for *S aureus* whereas only 6% of simultaneously obtained skin cultures were positive.[14]

However, by culturing more frequently and for a longer duration of' up to a year, Kaplowitz et al were able to culture *Staphylococcus* from the skin in 42% of' patients while the nasal cultures were positive in 49%.[6] On occasion. staphylococci were present on the skin but not in the nares. Oddly enough, the total number of positive staphylococcal cultures from the nares (146) was higher than from the skin (49) (Table 3 in the, / *Clin Microbiol* article). Nevertheless, their data suggest that surveillance cultures of' both the nares and the skin should be considered.

The higher yield of skin versus nares in the Kaplowitz study may arise from methodological differences including number of cultures taken from each site, the total area of the skin cultured, whether a dry or wet swab was used, and when the culture was plated. Exact methodologic details on moistening of the swab, use of one or two nostrils, details of insertion, and timing of culture plating after swabbing should be included in future studies of nasal colonization. Our study was lacking in this regard."

Berman et al did not find any difference in staphylococcal carriage between patients on hemodialysis and medical student controls, and one wonders if their results, which are somewhat contrary to the literature, might also be due to methodologic differences."'

We use a Culturette collection and transport system (Marion Scientific, Kansas City, MO) consisting of two swabs and an ampule of transport medium within a tube. One dry swab is inserted into each anterior nare and rotated gently. The two swabs are then returned to the tube and the ampule is crushed to moisten the swabs. Plating onto Vogel-Johnson (or Mannitol-salt) and blood agar medium is completed within four hours after collection.

Consistent with their findings on skin carriage, Kaplowitz et al found a higher correlation between staphylococcal access site infection and prior skin colonization ($P=0.02$) than with prior nasal colonization ($P=0.08$), suggesting that skin colonization may be more of a risk factor than nasal colonization." However, the number of infected patients was small (7) and the one additional patient with infection who may have had nasal but not skin colonization would have changed the P values substantially (Table 5 in the *J Clin Microbiol* article).

They further showed that the *S aureus* which were colonizers had similar antibiotic susceptibility profiles to the *S aureus* taken from the infected access site. Although phage-typing would have been more specific, their findings are again consistent with the thesis that the colonizing staphylococci become the pathogens.

Kaplowitz et al also found a significant relationship between hygiene and the development of vascular access infection. *S aureus* was significantly more likely to remain, and in a higher concentration, on the skin of patients with poor hygiene than those with good hygiene after application of an antiseptic. (This may have been because staphylococci are colonized in a higher concentration in patients with poor hygiene or that patients with poor hygiene do not cleanse their access sites as thoroughly as patients with good hygiene before dialysis.)

Evaluation of personal hygiene was based on a scale of "good hygiene" (clean skin and clothing), "intermediate hygiene" (skin moderately encrusted with dirt and clothing moderately dirty) or "poor hygiene" (skin heavily encrusted with dirt and clothing soiled). These criteria are somewhat vague and need to be defined more precisely; for example, how is the extent of dirt encrustation determined? Very few of the patients in our dialysis unit would have been classified as "moderately or heavily encrusted with dirt," although until objective criteria are formulated, we really cannot be certain. They are correct in pointing out Improved hygiene through educational measures would certainly be preferable to prophylactic antibiotics.

The authors found no difference in infection rates between sterile and clean skin preparation techniques for the access site prior to needle insertion for hemodialysis. However, closer examination of the two techniques studied shows that the skin preparation was identical for both groups. The difference was the use of sterile gloves and drapes in the "sterile" group. Thus, their results are not surprising and are reassuring to the vast majority of hemodialysis units which routinely use the "clean" technique.

In summary, Kaplowitz and colleagues have performed a comprehensive study using a multidisciplinary approach that should be a foundation for future research

https://doi.org/10.1086/645771 Published online by Cambridge University Press

efforts. Continued investigation in this area by infectious disease specialists and infection control practitioners with their nephrology colleagues should improve our understanding of the ecology and pathogenesis of staphylococcal infections. Cost-effective preventive measures are the long-term goal.

REFERENCES

1. Burton BT, Krueger KK, Bryan FA Jr: National registry of long-term dialysis patients. *JAMA* 1971; 218:718-722.
2. Degoulet P, Legrain M, Reach I, et al: Mortality risk factors in patients treated by chronic hemodialysis: Report of the Diaphane collaborative study. *Nephron* 1982; 31:103-110.
3. Bradley JR, Evans DB, Calne RY: Long-term survival in haemodialysis patients. *Lancet* 1987; 1:295-296.
4. Cross AS, Steigbigel RT: Infective endocarditis and access site infections in patients on hemodialysis. *Medicine* 1976; 55:453-466.
5. Keane WF, Shapiro FL, Raij L: Incidence and type of infections occurring in 445 chronic hemodialysis patients. *Trans Am Artif Intern Organs* 1977; 23:41-46.
6. Kaplowitz LG, Comstock JA, Landwehr DM, et al: Prospective study of microbial colonization of the nose and skin and infection of the vascular access site in hemodialysis patients. *J Clin Microbiol* 1988; 26:1257-1262.
7. Moss B, Squire JR, Topley E, et al: Nose and skin carriage of *Staphylococcus aureus* in patients receiving penicillin. *Lancet* 1948; 1:320-325.
8. Smith IM: *Staphylococcus aureus*, in Mandel GL, Douglas RG Jr, Bennett JE (eds): *Principles and Practice of Infectious Diseases*. New York, John Wiley and Sons, 1979, pp 1530-1552.
9. Goldblum SE, Reed WP, Ulrich JA, et al: Staphylococcal carriage and infections in hemodialysis patients. *Dial Transplant* 1978; 7:1140-1148.
10. Kirmani N, Tuazon CU, Murray HW, et al: *Staphylococcus aureus* carriage rate of patients receiving long-term hemodialysis. *Arch Intern Med* 1978; 138:1657-1659.
11. Goldblum SE, Reed WP: Host defenses and immunologic alterations associated with chronic hemodialysis. *Ann Intern Med* 1980; 93:597-613.
12. Aly R, Shinefield HI, Strauss WG, et al: Bacterial adherence to nasal mucosal cells. *Infect Immunol* 1977; 17:546-549.
13. Martin AM, Clunie GJA, Tonkin RW, et al: The aetiology and management of shunt infections in patients on intermittent haemodialysis. *Proc Eur Dial Transplant Assoc* 1967; 4:67-72.
14. Yu VL, Goetz A, Wagener M, et al: *Staphylococcus aureus* nasal carriage and infection in patients on hemodialysis: Efficacy of antibiotic prophylaxis. *N Engl J Med* 1986; 315:91-96.
15. Berman DS, Schaefler S, Simberkoff MS, et al: *Staphylococcus aureus* colonization in intravenous drug abusers, dialysis patients, and diabetics. *J Infect Dis* 1987; 155:829-831.

https://doi.org/10.1086/645771 Published online by Cambridge University Press