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The Source of Epidural Infection following Epidural Analgesia Identified by Pulsed-field Gel Electrophoresis

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WE report an epidural infection that developed after a thoracic epidural block. The patient had a long-standing atopic dermatitis covering most of the body surface, which had been treated with corticosteroid ointment. The epidural infection responded promptly to antibiotics. The pattern of chromosomal DNA fragments by pulsed-field gel electrophoresis (PFGE) revealed that strains of *Staphylococcus aureus* isolated from the epidural space and the catheter insertion sites and two of three strains isolated from two distant areas of the skin surface with atopic dermatitis were identical.

Case Report

A 22-yr-old man was referred to our pain clinic for treatment of severe pain associated with herpes zoster. Clusters of clear and cloudy vesicles were present over the left T5 dermatome. The patient had a 9-yr history of atopic dermatitis, which had spread over the entire body surface. The patient's back was prepared carefully with 0.5% chlorhexidine in 80% ethanol and then draped. An epidural catheter was inserted at the T6-T7 interspace. Five milliliters bupivacaine, 0.5%, was injected through a micropore filter into the catheter. The bolus injection was followed by an epidural infusion of the same concentration of bupivacaine at the rate of 1.0 ml/h. The pain resolved completely.

Two days later, the patient's body temperature increased to 39.2°C. On the next day, the patient complained of back pain at rest and during epidural bolus injection. Temperature was 39.6°C and the leukocyte count was 10.4×10^9/l. A small amount of purulent discharge was noted at the site of catheter insertion during compression of the surrounding skin. The catheter was removed and the patient received ceftazidime, 4 g, and vancomycin, 2 g daily. Cultures of the purulent discharge at the insertion site and prurient materials present inside the catheter before removal grew oxacillin-susceptible *S aureus*. On the seventh day, the patient's temperature was 35.5°C and leukocyte count was 6.5×10^9/l. The infection and the zoster pain resolved completely. There were no discernible neurologic deficits. The patient was discharged on the fifteenth day.

Microbiologic Evaluation

To identify the source of the epidural infection, bacteriologic samples were obtained from the epidural space (purulent materials inside the epidural catheter), five different areas of the skin surface of the back (two areas at approximately 10 cm and 20 cm away from the catheter insertion site with atopic dermatitis and three other areas located at the right buttock and at both shoulders without atopic dermatitis), and the nasal cavities of the patient and of the anesthesiologist who performed the epidural block. Six strains of *S. aureus* were isolated. Three strains were from two areas of the skin surface with atopic dermatitis (strains A, B, and C). Two strains were from the site of catheter insertion and the epidural space (strains D and E, respectively). A strain of *S. aureus* was isolated from the nasal cavity of the anesthesiologist (strain F). No strain of *S. aureus* was isolated from either of the three areas of the skin surface without atopic dermatitis. The isolate from the patient's nasal cavity was *Staphylococcus haemolyticus*.

Bactericidal activity of the skin disinfectant, 0.5% chlorhexidine in 80% ethanol, was assessed on each of the six isolated strains of *S. aureus* using the method of Sato *et al.*[1] Each strain was inoculated in brain–heart infusion broth and incubated overnight at 37°C. The suspension containing 1.5×10^9 colony-forming units/ml was used as a test inoculum. Ten microliters of each test inoculum was added to 5 ml chlorhexidine, 0.5%, in 80% ethanol and vortexed for 5 s. Control suspension was prepared

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by adding each test inoculum to 5 ml Tween-80, 5%. After the exposure of each inoculum to the skin disinfectant in air for 15, 30, 60, 120, and 240 s at room temperature, the antimicrobial activity of the disinfectant in the suspensions was inactivated by 1:1000 dilution of the suspensions with 5% Tween-80. One hundred microliters of the resulting suspensions were incubated on nutrient agar plates at 37°C for 72 h in duplicate to obtain colony count. No organisms grew after a 15-s exposure of the test inocula to 0.5% chlorhexidine in 80% ethanol.

Chromosomal DNA of *S. aureus* was prepared by the method of Hu *et al.*[2] The DNA from a block embedded in agarose was digested *in situ* with each restriction endonuclease of *Sma* I, *Cpo* I, and *Mlu* I (Takarashuzo Company, Kyoto, Japan). The fragments were separated by PFGE in a CHEF-DRIII apparatus (Nippon Bio-Rad Laboratory, Tokyo, Japan) for 22 h with initial and final pulses of 5 and 70 s, respectively. The gel was stained with ethidium bromide and photographed under ultraviolet transillumination. *Lambda* DNA *ladder* (Takarashuzo Company) was used as a DNA size marker. The pattern of chromosomal DNA fragments by PFGE on a restriction endonucleases *Cpo* I or *Sma* I showed that strain E (*S. aureus* from the epidural space) was identical to strains A and C (*S. aureus* from the skin surface with atopic dermatitis), as well as the strain D (*S. aureus* from the site of catheter insertion) (fig. 1). The pattern of DNA fragments by PFGE in strain F (*S. aureus* from the nasal cavity of the anesthesiologist) was different from that in strain E. The pattern of DNA fragments by PFGE on *Mlu* I showed that strain E was identical to strains A, C, and D (data not shown).

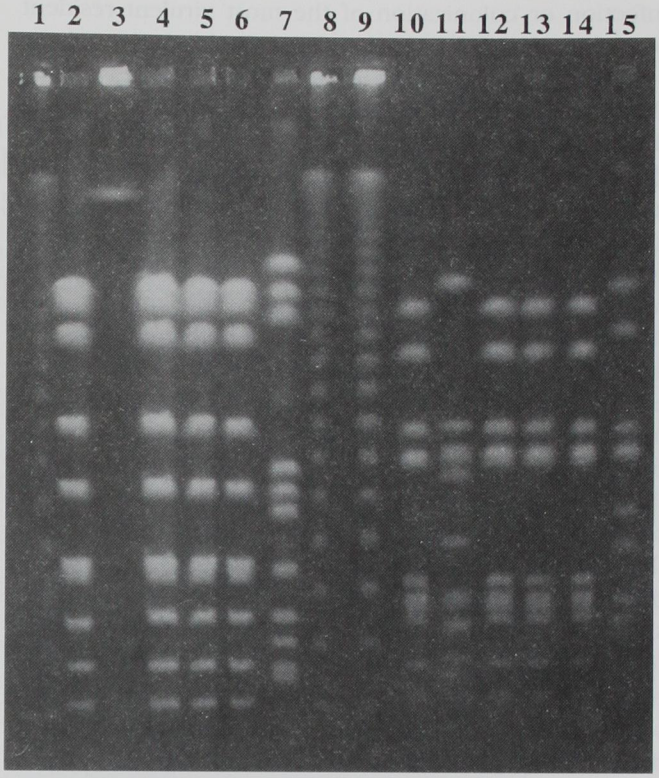

Fig. 1. The pattern of chromosomal DNA fragments digested with *Cpo* I (lanes 2 to 7) or *Sma* I (lanes 10 to 15) enzyme on six strains of *Staphylococcus aureus* isolated from the patient and the anesthesiologist. (*A*) Lanes 2 and 10, (*B*) lanes 3 and 11, and (*C*) lanes 4 and 12 isolated from the skin surface with atopic dermatitis. (*D*) An isolate from purulent discharge at the epidural puncture site (lanes 5 and 13). (*E*) An isolate from the epidural space (purulent material inside the catheter) (lanes 6 and 14). (*F*) An isolate from the nasal cavity of the anesthesiologist (lanes 7 and 15). Lanes 1, 8, and 9 show the size marker.

Discussion

The 15-s exposure to 0.5% chlorhexidine in 80% ethanol killed all six strains of *S. aureus* isolated from the patient. Because of the presence of atopic dermatitis at the site of epidural puncture, we prepared a wide area of the skin surface using a careful aseptic technique. We used disposable syringes and local anesthetics from newly opened vials for each epidural bolus injection. Cultures of local anesthetics from the same lot number used for treatment grew no organisms. Although a strain of *S. aureus* was isolated from the nasal cavity of the anesthesiologist, its pattern of chromosomal DNA fragments on each of three restriction endonucleases was different from those in the strains of *S. aureus* isolated from the epidural space, the catheter insertion site, and the two areas of the skin surface with atopic dermatitis.

Approximate rates for the *S. aureus* carriage sites in normal subjects are as follows: anterior nares: 35%; perineum: 20%; axillae: 5–10%; and toe webs: 5–10%.[3] However, the prevalence of *S. aureus* is low in the ranges of 1 to 2% or less on the skin surface of other parts of the body.[4,5] Lesions of atopic dermatitis are known to carry *S. aureus* even though the lesions are usually judged not to be clinically infected. Leyden *et al.*[6] reported the isolation of a large quantity of *S. aureus* from 90% of chronic plaques and 100% of acute exudative lesions of atopic dermatitis. Aly[7] showed that *S. aureus* predominated the total flora and also isolated the organism from unafflicted skin surfaces in 76% of patients with atopic dermatitis. It is not known whether the *S. aureus* dominance of the flora in the lesions represents secondary

infection or colonization of the most virulent resident organisms in the deeper skin tissue. In this patient, three strains of *S. aureus* were isolated from two areas of the skin surface with atopic dermatitis, although we did not detect *S. aureus* in any of the three areas without atopic dermatitis. The patterns of chromosomal DNA fragments in two of the three strains of *S. aureus* from the afflicted areas were identical to those isolated from the epidural space and the catheter insertion site, suggesting a causal relationship between the infection and one of two strains of *S. aureus* isolated over the afflicted skin surface. Although 0.5% chlorhexidine in 80% ethanol was effective in quickly killing all strains of *S. aureus* isolated from our patient *in vitro*, skin disinfectants, as shown previously,[1] may not penetrate into the deep layers of the skin tissue where colonies of organisms exist. It may be possible that a strain of *S. aureus* was inserted into the epidural space through the needle track at the time of epidural block or that the organisms from the afflicted skin surface spread to the catheter insertion site and further along the indwelling catheter despite precautions. The short time period that elapsed before the onset of fever suggests that the former mechanism may have been responsible for the infection.

Although the skin was prepared carefully and a strict aseptic technique was used during the management of the catheter, an epidural infection developed in the patient, and a strain of *S. aureus* that was genetically identical to the pathogen was isolated from the afflicted skin surface. Although the mechanism of infection was not established clearly, this case suggests that the skin surface with atopic dermatitis may be a potential source of epidural infection, even if the skin surface is prepared strictly and managed carefully.

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