TABLE
COMPARATIVE PATTERN ANALYSIS OF 62 ENVIRONMENTAL AND 43 CLINICAL ISOLATES OF EXTENDED-SPECTRUM BETA-LACTAMASE–PRODUCING ENTEROBACTERIACEAE RECOVERED FROM THE CARDIAC SURGERY INTENSIVE CARE UNIT PRODUCED BY PULSED-FIELD GEL ELECTROPHORESIS AND RANDOMLY AMPLIFIED POLYMORPHIC DNA

Species and Pattern	No. of Isolates: Environmental (Site)	No. of Isolates: Clinical
Klebsiella oxytoca		
P1	2 (F, D)	1*
P2	21 (C, F, D)	9†
P3	14 (C, F, D)	5‡
P4–9	0	8
Enterobacter cloacae		
P10	6 (C, F)	4§
P11	2 (F, D)	0
P12	3 (C, F)	0
P13	1 (D)	0
P14–21	0	9
Citrobacter freundii		
P22	4 (D)	0
P23	3 (F, D)	0
P24	1 (D)	0
P25	1 (C)	0
P26–29	0	6
C. diversus		
P30	2 (F)	0
P31	2 (F)	0
P32	0	1

C = countertops; F = faucets; D = drains.
*One case of respiratory colonization.
†One case of mediastinitis, one case of pneumonia, and seven cases of rectal colonization.
‡Two cases of rectal colonization, two cases of respiratory colonization, and 1 case of pneumonia.
§One case of rectal colonization, two cases of catheter colonization, and one case of urinary tract infection.

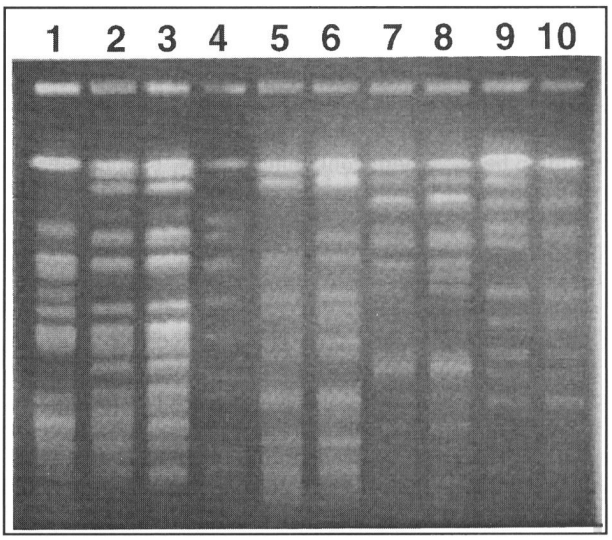

FIGURE. Pulsed-field gel electrophoresis patterns of *Xba*I-restricted DNA from extended-spectrum beta-lactamase–producing Enterobacteriaceae. Lane 1 = *Enterobacter cloacae* clinical strain P14; lane 2 = *Enterobacter cloacae* environmental strain P10; lane 3 = *Enterobacter cloacae* clinical strain P10; lane 4 = molecular size marker RN 450; lane 5 = *Klebsiella oxytoca* environmental strain P1; lane 6 = *K. oxytoca* clinical strain P1; lane 7 = *K. oxytoca* environmental strain P2; lane 8 = *K. oxytoca* clinical strain P2; lane 9 = *K. oxytoca* environmental strain P3; and lane 10 = *K. oxytoca* clinical strain P3.

room from which the P10 strain was isolated, but had been discharged 9 months before the environmental culture. Environmental and clinical strains of *Citrobacter* species had different randomly amplified polymorphic DNA patterns (data not shown).

DISCUSSION

This is the first study to document environmental contamination with ESBL Enterobacteriaceae as high as 26% of all surfaces cultured in a cardiac surgery ICU. This is much higher than the rate found by D'Agata et al.[10] They found that 5% of cultured surfaces were contaminated in two ICUs, including only third-generation, cephalosporin-resistant, gram-negative rods of major importance. The latter study took place during a non-outbreak period, whereas the current investigation was initiated following a major outbreak of these pathogens when infections continued to occur despite control measures. The current study confirms that sinks and countertops, and especially the joints of the countertops, represent commonly unrecognized sources of ESBL Enterobacteriaceae in ICUs. The ability of gram-negative bacteria to survive in moist environments for long periods helps to explain their occurrence in such sites.[11] This underscores the need to keep all inanimate surfaces surrounding patients as dry as possible, particularly in ICUs, where the number of opportunities for cross-transmission of nosocomial pathogens via the hands of healthcare workers is especially high.

In the current study, four species of ESBL Enterobacteriaceae were recovered using cotton-tipped, moistened swabs, which probably did not allow recovery of all bacteria. However, in a recent investigation, the sensitivity of this technique was 74% for the detection of gram-negative bacteria and it therefore should be considered the screening method of choice for these pathogens in the hospital environment. By comparison, the sensitivity of Rodac plates was 43%.[12]

Comparative PFGE analysis revealed that inanimate surfaces in ICUs can be contaminated with several species or strains of ESBL Enterobacteriaceae; one strain can survive for weeks to months at multiple sites; and some clinical strains may be recovered from environmental samples, raising the possibility of an exogenous origin of antibiotic-resistant, gram-negative bacteria. ESBL Enterobacteriaceae are supposed to be cross-transmitted like other nosocomial pathogens of the transient skin flora after direct contact with a colonized patient in the absence of hand hygiene by healthcare workers. However, conta-

mination of the hands of healthcare workers could occur following direct contact with patients or the environment. In moist environments, the contamination of hands could occur paradoxically during handwashing in a contaminated sink. Further studies are needed to determine whether the viable bacteria responsible for hand contamination are sufficient to support transmission to a patient who is not contaminated. Thus, transmission of ESBL Enterobacteriaceae via contaminated inanimate surfaces could not be excluded, particularly when multiple patients without overlapping stays were colonized with the same strain.

The results of this investigation have prompted attention to wet surfaces near sinks and faucets by cleaning staff in the ICU and implementation of quarterly environmental surveys. We believe these findings add evidence to support the hypothesis that moist surfaces may serve as sources of microorganisms in the ICU. Identification of such sources may be helpful in preventing transmission of such nosocomial pathogens.

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