CONCISE COMMUNICATION

Efficacy of Infection Control Strategies to Reduce Transmission of Vancomycin-Resistant Enterococci in a Tertiary Care Hospital in Korea: A 4-Year Follow-Up Study

Sung Won YoonChang, CIC; Kyong Ran Peck, MD; Og Son Kim, RN; Jang Ho Lee, MT; Nam Yong Lee, MD; Won Sup Oh, MD; Jae-Hoon Song, MD

To determine the effectiveness of infection control strategies to reduce transmission of vancomycin-resistant enterococci (VRE), a cohort study was performed in a university hospital. Contact precautions alone were not effective in reducing transmission of VRE. Strict isolation of affected patients in private rooms, in addition to use of contact precautions, showed a significantly improved reduction in the transmission of VRE.

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Since vancomycin-resistant *Enterococcus* (VRE) was first reported in Korea in 1992,[1] the VRE isolation rate has rapidly increased in hospitals nationwide. In intensive care units (ICUs) in 10 Korean tertiary care hospitals, VRE rectal colonization rates were reported to range from 9.4% to 51.6% in 1999.[2] One Korean hospital reported that the number of cases in which VRE was isolated from clinical specimens had increased 10-fold, from 7 cases in 1996 to 77 cases in 1997.[3] We had a similar experience with a sudden increase in the frequency of VRE isolation at our institution between 1998 and 1999.[4] Our infection control team investigated this increasing trend and set up VRE infection control strategies in 2000. Here, we evaluate the effectiveness of these strategies over 4 years.

METHODS

This was a prospective, cohort study conducted from March 1, 2000, to December 31, 2003, at a 1,250-bed university hospital in Korea. Only *Enterococcus faecium* and *Enterococcus faecalis* were investigated. Nosocomial acquisition was defined according to the guidelines of the Centers for Disease Control and Prevention (CDC); cases that did not meet this definition were categorized as community acquired.

Period A, the contact precaution period (from March 2000 through May 2000), was the set-up period for VRE infection control strategies. The strategies included the following: prompt reporting to physicians within 8 hours after isolation of VRE, restriction of antibiotic prescription by infectious disease physicians, and education of healthcare workers (HCWs) concerning hand hygiene and contact precautions. Rectal samples for surveillance cultures were obtained weekly from patients from whom VRE had been isolated and from the patients sharing their rooms. Environmental surveillance cultures of samples from patients' rooms and the equipment used to treat them were performed after, as well as before, terminal cleaning and disinfection using a quaternary ammonium compound. Precautions were discontinued if weekly follow-up cultures did not grow VRE for 3 consecutive weeks. Period B (from June 2000 through December 2000) was the strict isolation period, during which strict isolation in private rooms was used for patients with culture results positive for VRE, in addition to the infection control strategies employed during period A. During period C (from January 2001 through December 2003), infection control strategies were modified as follows: patients with cultures positive for VRE were strictly isolated in private rooms, samples for rectal culture were obtained only from those patients, and samples for surveillance cultures of the environment and equipment were obtained only after terminal disinfection. Period C was longer than periods A or B, which allowed us to evaluate the long-term effect of our strategies. Data were analyzed with SPSS 10.0 for Windows (SPSS).

RESULTS

A total of 123 VRE isolates were recovered from clinical specimens obtained from 121 patients (Table). Patients with rectal carriage detected with surveillance cultures were excluded from this analysis. There were no significant differences in the distributions of patients by age, sex, or admission department between the 3 study periods. The median duration of hospitalization at the time of VRE isolation was 23 days. Forty-seven percent of patients from whom VRE was isolated had a history of prior hospitalization. Seventy-four percent had been admitted to the ICU previously, and 63% had had surgery during the current hospitalization. Sixty-three percent of the VRE infections were nosocomial, and 37% were community acquired (including those in patients transferred from other hospitals). *E. faecium* accounted for 89% of VRE isolates, and *E. faecalis* accounted for 9%; both organisms were isolated from 2 patients (Table). *E. faecalis* was isolated significantly more often during periods B and C, compared with period A ($P = .02$). As for the phenotypes of glycopeptide resistance, VanA was the major phenotype found during periods A and B. However, VanB was the major type found during period C ($P < .001$). VanC was excluded from this study.

The rectal colonization rate for VRE among patients from whom VRE had been isolated was 43.0% (34 of 79 patients), and among other patients sharing their rooms, the rate was 17.1% (12 of 70 patients). The VRE contamination rate for HCWs' hands was 2.3% (4 of 172 HCWs). The contamination

TABLE. General Characteristics of 121 Patients From Whom 123 Vancomycin-Resistant Enterococci Isolates Were Obtained

Variable	*n*	Period A (contact precautions)	Period B (strict isolation)	Period C (follow-up)	*P*[a]
No. of patients	121	19	7	95	
Sex, no. of patients					.357
Male	66	8	3	55	
Female	55	11	4	40	
Class of acquisition, no. of patients					.834
Hospital-acquired					
Infection	59	7	4	48	
Colonization	17	9	0	8	
Community-acquired					
Infection	32	1	2	29	
Colonization	13	2	1	10	
Enterococcus species isolated, no. of samples					.02
E. faecium	108	18	3	87	
E. faecalis	11	1	3	7	
E. faecium and *E. faecalis*	2	0	1	1	
Specimens obtained, no. of patients					.378
Urine	34	7	3	24	
Wound discharge	27	7	2	18	
Blood	24	3	0	21	
Sputum	1	0	0	1	
Stool	15	0	0	15	
Bile	3	0	0	3	
Other	17	2	2	13	
Phenotypes found, no. of samples					<.001
VanA	61	18	7	36	
VanB	62	1	1	60	

[a] Comparison of period A with periods B and C.

rates for the environment were 8.76% (39 of 445 samples) before terminal disinfection and 0.76% (2 of 263 samples) after terminal disinfection. Pulsed-field gel electrophoresis (PFGE) demonstrated some of the same PFGE patterns among isolates from different patients during period A, although many varieties of PFGE patterns were observed. By contrast, no shared PFGE patterns were observed among isolates from different patients during periods B and C (data not shown).

The incidence rate for VRE was 1.45 cases per 10,000 patient-days during the contact precaution period, which was similar to the rate before the study (Figure). The incidence rate decreased significantly to 0.75 cases per 10,000 patient-days during period B ($P = .003$). The efficacy of infection control strategies for VRE continued during period C, with an incidence rate of 0.88 cases per 10,000 patient-days, which was significantly lower than that of period A ($P = .009$) and was similar to that of period B.

DISCUSSION

There are published guidelines for preventing the emergence and transmission of VRE.[5,6] We adopted and modified these guidelines and set up our own hospital infection control policies. This study demonstrated that our policies had been very effective in controlling VRE. Several studies have shown that enhanced infection control strategies reduce VRE transmission, but they have failed to assess how individual measures resulted in successful control of VRE.[7,8] Our study demonstrates that strict isolation in private rooms significantly reduced VRE transmission, whereas contact precautions alone were not effective.

Although the proportion of patients with community-acquired VRE colonization or infection was 37% in this study, all of these patients were transferred from other hospitals; there were no patients with true community acquisition that we could identify. (The possibility exists that the transferred patients did have true community-acquired VRE colonization or infection.) Therefore, screening for VRE at the time of transfer might be needed to reduce the influx VRE into hospitals. About half of the patients from whom VRE was isolated and one-sixth of the patients sharing their rooms already had rectal colonization at the time VRE was isolated from the clinical specimens obtained in this study. This demonstrates that rectal colonization with VRE, before isolation from clin-

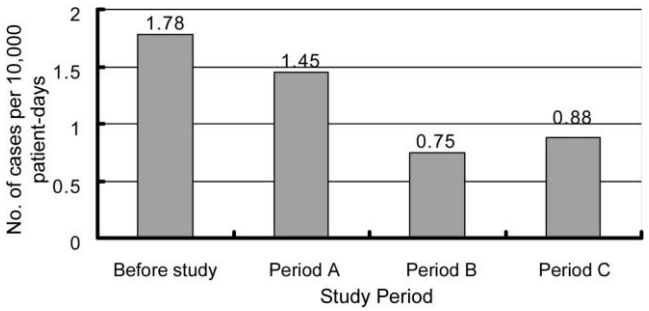

FIGURE. Incidence density rates of patients from whom vancomycin-resistant enterococci (VRE) were isolated, before and after implementations of VRE infection control strategies. The rate did not decrease significantly during period A, compared with the rate before the study. It decreased significantly during period B and during period C ($P < .05$), when there was strict isolation of patients with VRE in private rooms, in addition to use of contact precautions. The VRE incidence density rates between the periods were compared by general estimating equations.

ical specimens, can lead to transmission of VRE to other patients.[7] Contamination of the hands of HCWs indicates that hand washing and use of barrier precautions, including use of gloves and gowns, must be enforced to prevent the spread of VRE. Even though terminal cleaning and disinfection effectively reduced surface contamination, VRE were not completely eradicated. Therefore, we continued to do random surveillance cultures of environmental surfaces after terminal cleaning to improve compliance with cleaning guidelines and to assure the cleanliness of patient rooms.

On the basis of the PFGE patterns of the VRE isolates, we conclude that there might have been a single clonal spread from a single source patient during period A. However, this possible epidemic clone seemed not to contribute to the VRE isolates recovered during periods B and C. This suggests that our infection control policies, which included strict isolation of patients, effectively prevented the clonal dissemination of VRE. As for the phenotypes of glycopeptide resistance, VanB became the major type during period C. This finding is similar to the results of another Korean study.[9] Incongruence between the phenotype and genotype of VRE, in which a VRE isolate has a VanB phenotype and a *vanA* genotype, has been reported.[10] The proportions of incongruent VRE were not evaluated in our study, because genotypic analysis was not performed.

Our study has several limitations. First, the contact precaution period may have favorably influenced adherence to strict isolation procedures, because this study was conducted with a cohort of patients. Second, the use of differing intervals for each period may have allowed seasonal effects to influence our results, although no seasonal variation in VRE infection has been reported. Finally, our study cannot confirm the importance of each infection control measure in reducing VRE transmission, except for the isolation of patients in private rooms, because many infection control measures were implemented simultaneously. Nonetheless, we believe that this study demonstrates that strict isolation in a private room, in addition to use of contact precautions, resulted in a significantly improved reduction in the transmission of VRE. The infection control strategies for VRE in our hospital have now been working effectively for several years.

From the Infection Control Office (S.W.Y., O.S.K.), the Division of Infectious Diseases (K.R.P., W.S.O., J.H.S.), and the Department of Laboratory Medicine (J.H.L., N.Y.L.), Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea.

Address reprint requests to Kyong Ran Peck, MD, Division of Infectious Diseases, Samsung Medical Center, 50 IL-won dong, Kangnam-gu, Seoul, 135-710, Korea (krpeck@smc.samsung.co.kr).

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