A Semiquantitative Analysis of the Fecal Flora of Patients with Vancomycin-Resistant Enterococci: Colonized Patients Pose an Infection Control Risk

Vancomycin-resistant enterococci (VRE) have rapidly emerged as major nosocomial pathogens in hospitals throughout the United States. VRE fecal carriage can be widespread among hospitalized patients [1]. To determine whether VRE fecal colony counts differ in VRE-colonized and VRE-infected patients, we performed a semiquantitative study of the aerobic fecal flora of hospitalized patients with VRE.

Stool samples were obtained from patients with VRE. Stool suspensions equivalent to $10^{-1} \text{ g/mL}$ were made and serially diluted up to $10^{-12} \text{ g/mL}$ with use of sterile saline. One mL of each dilution was inoculated into trypticase soy broth, Enterococcosel broth (Becton-Dickinson Microbiology Systems, Cockeysville, MD), and Enterococcosel broth plus vancomycin (6 $\mu$g/mL) and incubated at $35^\circ C$ for 48 hours.

Enterococcosel broths (with or without vancomycin) exhibiting growth were plated onto sheep blood agar; trypticase soy broths were plated onto Columbia and MacConkey agars. Three to five colonies of each colony type were isolated from each plate. Enterococci were identified and tested for vancomycin resistance with use of standard methods [2]. Aerobic gram-negative bacilli (GNB) were identified by colony morphology.

VRE-colonized and VRE-infected patients were compared with use of Wilcoxon’s rank sum test or Fisher’s exact test. A log$_{10}$ transformation of VRE cfu per g of stool was performed. Comparisons between mean log$_{10}$ VRE and dichotomous variables were analyzed with use of the unpaired $t$-test. The Friedman test was used to assess differences among VRE, vancomycin-susceptible enterococci (VSE), and GNB cfu/g; the Student-Newman-Keuls test was used to determine which counts differed. $P$ values were determined by two-tailed tests.

VRE were recovered from the stool of 25 patients. Nine patients had a VRE infection, five had VRE colonization of a clinical site in addition to stool, and 11 had VRE recovered only from stool. There were six bloodstream infections, one pancreatic abscess, one case of peritonitis, and one urinary tract infection, with an onset of a median of 10 days (range, 0–23 days) before stool sampling. The 16 VRE-colonized and the nine VRE-infected patients had similar demographics, clinical characteristics, APACHE II (Acute Physiologic and Chronic Health Evaluation) scores, and days of antimicrobial therapy, except that a higher percentage of VRE-infected patients were male (88% vs. 37.5%, $P = 0.02$).

Figure 1. The number of vancomycin-resistant enterococci (VRE), vancomycin-susceptible enterococci (VSE), and aerobic gram-negative bacilli (GNB) recovered from the stool of 25 patients. Each symbol represents the highest number of organisms recovered. The line in each group of symbols indicates the median number of organisms for the group. Patients for whom VSE or GNB were not detected are indicated by log$_{10}$ = none.

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VRE was the aerobic organism recovered in greatest number in stool (median of $10^5$ cfu/g; range, $10^3$–$10^9$ cfu/g), outnumbering GNB (median of $10^7$ cfu/g; range, $0$–$10^9$ cfu/g), and significantly outnumbering VSE (median of $10^5$ cfu/g; range, $0$–$10^9$ cfu/g) (Friedman test $\chi^2 = 5.76; P = .02$) (figure 1). The VRE isolates were Enterococcus faecium for 22 patients and Enterococcus faecalis for three patients. The number of VRE was the same for 20 stool samples for which duplicate cultures were performed.

VRE-colonized and VRE-infected patients had similar VRE fecal colony counts (mean $\pm$ SD log_{10} of $8.3 \pm 1.1$ cfu/g vs. $7.3 \pm 2.1$ cfu/g, $P = .24$). When patients who had VRE recovered from stool only were compared with patients who had VRE isolated from a clinical site, no statistically significant difference in VRE cfu/g of stool was found (mean $\pm$ SD log_{10} of $8.2 \pm 1.3$ cfu/g vs. $7.7 \pm 1.8$ cfu/g, $P = .47$).

In our VRE-colonized and VRE-infected patients, VRE was the predominant aerobic organism present in stool, outnumbering both GNB and VSE. VRE-colonized and VRE-infected patients had similar numbers of VRE cfu/g of stool, suggesting that VRE-colonized patients are as likely as VRE-infected patients to contaminate the immediate environment and/or transmit VRE. The reservoir of colonized patients can be important in the transmission of other nosocomial pathogens, including methillin-resistant Staphylococcus aureus [3] and Clostridium difficile [4]. Our data suggest that the same is true for VRE.

Potential limitations of our study include a relatively small sample size and the selection of patients from a population of known VRE fecal carriers, which could bias results toward patients with higher numbers of VRE. Although Enterococcosel broth can detect as few as one colony of enterococci [5], the use of broths may have underestimated the number of enterococci by up to one log because the number of cfu/g could range from the highest dilution exhibiting growth and continue up to, but not include, the next highest dilution where no growth was detected.

The current U.S. Public Health Service recommendations for reducing transmission of VRE apply to both VRE-colonized and VRE-infected patients [2]. Our study supports infection control policies that emphasize the importance of identifying both VRE-colonized and VRE-infected patients. The failure to prevent VRE transmission may, in part, be explained by the failure to identify and isolate VRE-colonized patients.


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References


Cervical Cytomegalovirus Infection in a Woman with AIDS

Although cytomegalovirus (CMV) infection is a significant source of morbidity and mortality in patients with AIDS, its incidence and clinical significance in the female genital tract in women with AIDS remain unknown. In the three cases reported in the literature, the patients were either asymptomatic [1] or they had menorrhagia [2] or a pelvic inflammatory disease (PID)-like syndrome [3]. These reports predate the widespread use of ganciclovir for treatment of CMV infections. We report the successful treatment of a case of cervical CMV infection that was diagnosed by Papanicolaou smear and that manifested as a PID-like syndrome in a woman with AIDS.

A 37-year-old woman with known AIDS presented for routine gynecologic evaluation. The patient, who had been infected with HIV for 5 years, had a CD4 cell count of 0/mm³. In the 6 months before evaluation, the patient had had an outbreak of rectal herpes simplex virus infection and an episode of CMV esophagitis that was treated with ganciclovir. The patient had not been sexually active for 4 years. She reported intermittent fevers and denied nausea and vomiting. During the patient’s visit, she complained that she had had weakness, fatigue, and pelvic pain for 4 weeks.

Physical examination revealed a thin young woman with a benign abdomen. Pelvic examination demonstrated no external lesions. Specifically, no lesions consistent with herpes simplex virus infection were noted. A small polypoid lesion was noted at the cervical os, and findings of bimanual examination were unremarkable. The results of cultures for gonorrhea and chlamydia were negative. Cytological examination of cervical specimens was remarkable for inflammatory cell changes. Intranuclear inclusions that were morphologically consistent with CMV infection were noted (figure 1). Culture of a cervical specimen yielded CMV.

The patient had persistent pelvic pain, and subsequent examination revealed moderate right-lower-quadrant tenderness. A diagnosis of PID secondary to CMV infection was made, and the patient was treated with iv ganciclovir (twice daily for 14 days). The patient did not receive concurrent antibacterial treatment for PID.