

LETTER TO THE EDITOR

VRE fecal colonization/infection in cancer patients

Bone Marrow Transplantation (2007) 39, 567–568.
doi:10.1038/sj.bmt.1705639; published online 12 March 2007

We read with interest the article by Dubberke *et al.*¹ regarding their experience with vancomycin-resistant enterococcal bloodstream infections in a hematopoietic stem cell transplant (HSCT) unit. Experience at our institution, an National Cancer Institute designated Comprehensive Cancer center, is similar to theirs in some aspects, and different in other aspects. We screened 2115 patients for fecal colonization with vancomycin resistant enterococci (VRE), including 955 with hematologic malignancies (leukemia, myeloma, myelodysplastic syndromes), 653 HSCT recipients, and 507 patients with lymphoma.² VRE were recovered from fecal smears of 56 patients with hematologic malignancies (5.9%), 32 HSCT recipients (4.7%) and 11 lymphoma patients (2.2%) for an overall frequency of colonization of 4.7%. Thirty-one episodes of VRE bacteremia occurred in these patients, with all but two episodes occurring in patients with VRE fecal colonization. Thus fecal colonization with VRE had a positive predictive value of 29.3% for the subsequent development of VRE bacteremias. Additionally, the negative predictive value of fecal smears was 99.9% in these high-risk patients. The species distribution of our VRE isolates was as follows: *Enterococcus faecium* – 77%; *E. faecalis* – 13%; and other species (*E. avium*, *E. casseliflavus*, *E. durans*, *E. gallinarum*, *E. raffinosus*) – 10%. Five of the 31 episodes of bacteremia (16%) were polymicrobial with enteric gram-negative bacilli being the predominant co-isolates. Several patients developed infection at other sites without developing bacteremia (Table 1). The urinary tract was the most common site followed by skin and skin structure infection, biliary tract infection, and one episode of meningitis. VRE were also isolated from respiratory samples (sputum; bronchoalveolar lavage (BAL)) of seven patients who did not meet the criteria for pneumonia and were considered to be colonized. The mortality directly attributable to VRE bacteremia was <7%. Eight patients of the 31 with VRE bacteremia (26%) developed and survived a subsequent episode of bacteremia over a 1-year follow-up period, providing further evidence of the low pathogenicity/virulence of these organisms.

We also looked for VRE infection rates in our solid tumor patients over the same study period (Table 1). Fecal smears to detect VRE colonization are not routinely performed in these patients, consequently VRE colonization rates could not be determined. Nevertheless 10 episodes of VRE bacteremia occurred in 8538 patients (0.12%), and 42 episodes of infection (0.49%) occurred at other sites giving an overall infection rate of 0.61%, significantly lower than in patients with hematologic

malignancies/HSCT recipients ($P = <0.001$). Patients with hematologic malignancies were 17.3 times more likely to get VRE bacteremia (95% confidence interval (95% CI): 8.0–37.3) and 3.7 times more likely to get VRE infection at other sites (95% CI: 2.1–6.5) than patients with solid tumors. Similarly HSCT recipients were 11.9 times more likely to get VRE bacteremia (95% CI: 4.8–29.4), and 3.5 times more likely to get VRE infection at other sites (95% CI: 1.8–6.8) than solid tumor patients.

Active surveillance among our high-risk patients has been a successful strategy in the control and prevention of VRE outbreaks and nosocomial spread of infection.³ Such surveillance and strong adherence to infection control policies have reduced the frequency of VRE isolates at our institution from a peak of 38% in 2001 to 17% in 2005 (institutional infection control data). Although most patients respond to therapy and the attributable mortality remains low, the emergence of resistance to linezolid is of considerable concern.⁴ The organisms remain susceptible to new agents such as daptomycin.⁵ Our data indicate that hospitalized patients with hematological malignancies and HSCT recipients are at greater risk of developing VRE infections than patients with solid tumors. These infections occur almost exclusively in patients with VRE fecal colonization. Surveillance programs to detect fecal colonization in high-risk patients reduce the frequency and spread of these infections. They also identify a subset of patients at risk for development of VRE bacteremia and VRE infections at other sites, information that can impact empiric therapy of subsequent febrile episodes. We also ‘red flag’ patients with VRE fecal colonization for subsequent hospital admissions, as our attempts at eliminating fecal colonization in such patients have been singularly unsuccessful.

Table 1 Distribution of infections caused by VRE in various subgroups of patients with cancer

Patient subgroup	No.	No. (%) with VRE bacteremia	No. (%) with other VRE infection sites ^a	Total percent VRE infection rate
Hematologic malignancy	955	19 (1.99)	17 (1.78)	3.77
HSCT	653	9 (1.38)	11 (1.68)	3.06
Lymphoma	507	3 (0.59)	4 (0.79)	1.38
Solid tumor	8538	10 (0.12)	42 (0.49)	0.61
Total	10653	41 (0.38)	74 (0.69)	1.07

^aOther sites of infection included 52 episodes of urinary tract infection, 13 episodes of skin/skin structure infection, eight episodes of biliary tract infection, and one episode of meningitis. Seven patients with VRE isolated from respiratory samples did not meet the criteria for pneumonia, and were considered to be colonized.

KVI Rolston¹, Y Jiang¹ and M Matar²

¹*Department of Infectious Diseases, Infection Control and Employee Health, The University of Texas MD Anderson Cancer Center, Houston, TX, USA and*

²*Department of Infectious Diseases, Infection Control Notre Dame de Secours Hospital, Jbeil, Lebanon
E-mail: krolston@mdanderson.org*

References

- 1 Dubberke ER, Hollands JM, Georgantopoulos P, Augustin K, DiPersio JF, Mundy LM *et al.* Vancomycin-resistant enterococcal bloodstream infections on a hematopoietic stem cell transplant unit: are the sick getting sicker? *Bone Marrow Transplant* 2006; **38**: 813–819.
- 2 Matar MJ, Tarrand J, Raad I, Rolston KVI. Colonization and infections with vancomycin-resistant enterococcus among patients with cancer. *Am J Infect Control* 2006; **34**: 534–536.
- 3 Hachem R, Graviss L, Hanna H, Arbuckle R, Dvorak T, Hackett B *et al.* Impact of surveillance for vancomycin-resistant enterococci on controlling a bloodstream outbreak among patients with hematologic malignancy. *Infect Control Hosp Epidemiol* 2004; **25**: 391–394.
- 4 Raad II, Hanna HA, Hachem RY, Dvorak T, Arbuckle RB, Chaiban G *et al.* Clinical-use-associated decrease in susceptibility of vancomycin-resistant *Enterococcus faecium* to linezolid: a comparison with quinupristin-dalfopristin. *Antimicrob Agents Chemother* 2004; **48**: 3583–3585.
- 5 Prince R, Coyle E, Rolston K, Kapadia M, Kaur A, McCauley S. *In vitro* activity of daptomycin and linezolid against vancomycin-resistant gram-positive pathogens from cancer patients. (Abstract). *17th European Congress of Clinical Microbiology and Infectious Diseases (Munich)*, 2007.