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Acute Hearing Loss and Rickettsial Diseases

To the Editor—Premaratna et al. [1] provided a timely reminder to clinicians in their report describing acute hearing loss due to scrub typhus. These authors, however, do not mention an important fact that is relevant for clinicians who practice in areas where other rickettsial diseases are prevalent. Acute hearing loss with or without other isolated cranial nerve abnormalities may occur in a variety of rickettsial diseases. For example, unilateral or bilateral deafness may occur in patients with Rocky Mountain spotted fever, Mediterranean spotted fever, and murine typhus [2–5]. The mechanism for hearing loss in patients with Rocky Mountain spotted fever has been assumed to be vasculitis-induced cochlear damage [3]. However, it is also possible that hearing impairment or selected other neurological complications of rickettsial infection could be immune mediated, because Guillain-Barré polyneuropathy has been described following infection with *Rickettsia conorii* and *Rickettsia rickettsii* [6, 7].

Acknowledgments

Potential conflicts of interest. D.J.S.: no conflicts.

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References

Relationship of Colonization with Vancomycin-Resistant Enterococci and Risk of Systemic Infection in Patients with Cancer

To the Editor—We read with great interest the article by McNeil et al. [1] regarding vancomycin-resistant enterococci (VRE) colonization and infection in patients undergoing liver transplantation. These organisms are also significant pathogens in patients with an underlying malignancy, accounting for substantial morbidity and mortality among this population [2]. We have conducted a similar study involving patients with hematological malignancies and hematopoietic stem cell transplant (HSCT) recipients at our institution, a National Cancer Institute–designated comprehensive cancer center. For such patients, 3 weekly rectal swab samples are routinely obtained to detect fecal colonization with VRE, while maintaining contact isolation [3]. We collected data on fecal colonization with VRE and subsequent infections over a 1-year period in hospitalized patients with leukemia, lymphoma, and receipt of HSCT. We describe our findings below.

A total of 2115 patients were screened. VRE colonization was documented in 99 (4.7%) of the patients, with VRE colonization in 56 (5.9%) of 955 leukemia patients, 32 (4.9%) of 653 HSCT recipients, and 11 (2.2%) of 507 lymphoma patients. The most common species isolated was *Enterococcus faecalis* (84% of isolates), with 6% of isolates being *Enterococcus faecalis*, and 10% being other species (*Enterococcus avium, Enterococcus durans, Enterococcus casseliflavus, and Enterococcus gallinarum*). Among 99 patients with VRE colonization, 29% developed an episode of bloodstream infection (table 1). Of interest, only 2 patients with leukemia with VRE bacteremia during the study period did not have fecal VRE colonization. Fecal colonization with VRE had a high negative predictive value (99.9%) and a 29.3% positive predictive value for the development of VRE bloodstream infection. Other sites of infection included the urinary tract (28 episodes) and surgical wounds (4 episodes); all episodes occurred in patients with VRE colonization. VRE that were iso-