the risk of tuberculous infection is high [8, 9]. Our patient, and others described in previous reports [2–4], were in environments where they were heavily exposed to M. tuberculosis. In addition to varying degrees of immunosuppression, high levels of exposure to M. tuberculosis likely contributed to their risk of reinfection.

The case described herein illustrates that diabetes may be a risk factor for exogenous reinfection in patients with prior tuberculous infection. To what extent, if any, exogenous reinfection occurs in immunocompetent hosts is unknown. Stead [10] suggested that healthy tuberculin reactors are at very low risk of reinfection when they are again exposed to M. tuberculosis. Shafer et al. [5] presented a case of exogenous reinfection in an immunocompetent woman who had previously used alcohol heavily. However, it is not clear how previous heavy alcohol use affects the immune system.

The number of cases of tuberculosis resulting from exogenous reinfection is undoubtedly underestimated since the cases described to date have involved only reinfection with isolates resistant to multiple drugs. Perhaps, when molecular epidemiological tools are more broadly used to analyze M. tuberculosis isolates, we will have a truer sense of the role reinfection plays in tuberculosis.

Recurrent Vancomycin-Resistant Enterococcus faecium Bacteremia in a Leukemia Patient Who Was Persistently Colonized with Vancomycin-Resistant Enterococci for Two Years

The enterococcus has become a major nosocomial pathogen, particularly in immunosuppressed patients, and has also acquired resistance to all major therapeutic antibiotics including vancomycin. Mortality was high in reported outbreaks of vancomycin-resistant enterococcal bacteremias, particularly in cancer patients [1, 2]. We report the case of a patient with leukemia who had three separate episodes of vancomycin-resistant enterococcal bacteremia over 2 years.

A 40-year-old man with acute promyelocytic leukemia received induction chemotherapy in October 1993 at the University of Maryland Cancer Center. A rectal surveillance culture performed 18 days after admission to the hospital yielded Enterococcus faecium that was resistant to vancomycin (MIC, >256 μg/mL), teicoplanin (MIC, 64 μg/mL), ampicillin (MIC, >256 μg/mL), and streptomycin (MIC, >1,024 μg/mL); in addition, this isolate had high-level resistance to gentamicin (MIC, 1,024 μg/mL). Blood cultures performed during a febrile episode 6 days later yielded an isolate of E. faecium that was identical to the original isolate on the basis of antimicrobial susceptibility tests and pulse-field gel electrophoretic patterns. The isolate was determined to be a vanB1 genotype by PCR.

The patient did not have sepsis on the basis of subsequent vital signs but had severe neutropenia and mucositis. He had received oral vancomycin therapy as bowel prophylaxis and iv vancomycin therapy for a clinically documented catheter exit site infection. His Hickman catheter was removed. The results of subsequent blood and catheter tip cultures were negative. His neutrophil count recovered 6 days later and was associated with remission of his leukemia; he recovered uneventfully without specific therapy.

Over the next 3 months, rectal surveillance cultures continued to yield an E. faecium isolate that was identical to the first one obtained in cultures performed during his first admission; the isolates were determined to be identical on the basis of antimicrobial susceptibility tests and pulse-field gel electrophoretic patterns. In March 1995 his leukemia relapsed for a second time, and he received chemotherapy. A rectal surveillance culture yielded an identical E. faecium isolate. He again became bacteremic with the same E. faecium isolate.

References

His vancomycin-resistant enterococcal bacteremia had been preceded by treatment with iv vancomycin, imipenem, and ciprofloxacin for prior infections. He again had severe neutropenia and mucositis. He never developed signs or symptoms of sepsis. He received no specific therapy, and subsequent blood cultures were negative for enterococci. His neutropenia resolved after 37 days, and his leukemia went into remission.

In September 1995 the patient’s leukemia relapsed, and he again received chemotherapy. A rectal surveillance culture once again yielded the same E. faecium isolate. After two infections (Candida albicans fungemia 10 days before isolation of vancomycin-resistant enterococci [VRE] in blood cultures and Staphylococcus epidermidis bacteremia 20 days before isolation of VRE in blood cultures), blood cultures again yielded the identical E. faecium isolate. Even though his vascular catheter insertion sites were changed, E. faecium bacteremia persisted and he had severe neutropenia and mucositis. On the fifth day of bacteremia, he developed septic shock with hypotension and respiratory and renal failure. He remained bacteremic throughout the course of therapy with doxycycline and novobiocin (the organism was susceptible to these two antibiotics). He was still severely neutropenic at the time of his death 3 days later.

Although vancomycin-resistant enterococci has become a major nosocomial pathogen, little is known about its epidemiology. Leukemia patients offer a unique opportunity to study the epidemiology of VRE since they are admitted to the hospital numerous times and (at our institution) since rectal surveillance cultures are performed weekly while the patients are hospitalized. In our patient’s case, he was colonized with the same vancomycin-resistant enterococcus clone over a 2-year period despite extended periods (>3 months) outside of the hospital and remission of his leukemia. Of 22 follow-up rectal surveillance cultures, all but one yielded VRE.

At our institution, we maximize the chance of isolating VRE by using selective media (colistin nalidixic acid agar supplemented with defibrinated 5% sheep blood, vancomycin [10 μg/mL], and amphotericin B [1 μg/mL]) for surveillance cultures, which may explain the high proportion of positive cultures. However, the potential for long-term carriage suggests that all cancer patients with a history of colonization with VRE should be isolated during every hospitalization.

Our patient had multiple risk factors for infection due to VRE. He consistently became bacteremic with VRE after induction chemotherapy that caused neutropenia and mucositis, which in turn necessitated the use of therapeutic antimicrobial agents. He had received multiple antibiotics (including oral vancomycin) that select for VRE in stool [3]. The VRE organisms isolated from blood cultures were identical to those colonizing his gastrointestinal tract, a finding that strongly implicates the gastrointestinal tract as the source of bacteremia. Oral vancomycin is no longer used for bowel prophylaxis at our institution.

A case-control study comparing cancer patients infected with VRE to those colonized with VRE found that the total number of days of antibiotics was the only independent risk factor for VRE infection in a multivariate analysis [4]. Another case-control study that compared cancer patients infected with VRE to ward controls found that VRE colonization and antibiotics that cover anaerobes were risk factors for infection due to VRE [2]. In a similar study, increasing APACHE scores, mucositis, and receipt of antibiotics for >80% of hospital days [1] were independent risk factors for infection due to VRE in cancer patients.

Mortality in reported outbreaks of VRE infection in cancer patients has been high (57%-73%) [2, 4]. In our experience, the prognosis of bacteremia due to VRE is strongly associated with the prognosis of the underlying disease; infection due to VRE resolved with neutrophil recovery and disease remission. During our patient’s fourth course of induction chemotherapy, when his chance of remission was low, VRE caused persistent bacteremia and, finally, septic shock. Our patient’s case demonstrates that bacteremia due to VRE may not be lethal if the underlying disease is controlled; however, the risk of infection remains if the underlying disease recurs.

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References

Optic Neuritis: A Rare Complication of Primary Varicella Infection

Optic neuritis is an extremely rare complication of chickenpox that has been the subject of isolated case reports [1–4]. Selbst et al. reviewed the cases of eight patients aged 3–14 years, and Miller et al. described the first three adult cases [2, 3]. All 11 patients had visual symptoms that occurred during or after the onset of varicella rash and that ranged from 2 to 38 days [2–4]. Visual loss is nearly always bilateral in patients with optic neuritis and can be severe. We describe an unusual case of monocular optic neuritis due to varicella in an adult whose visual symptoms preceded the varicella rash.

A healthy 25-year-old female presented with a 3-week history of progressive blurring of vision of her left eye. She did not have associated eye pain, diplopia, or headache. Five days after the onset of visual blurring, she developed vesicles over her upper lip that spread to the rest of her body. She apparently had not been