Enterococcal Bloodstream Infection After Hematopoietic Stem Cell Transplant: Experience of a Center With a Low Prevalence of Vancomycin-Resistant Enterococci

To the Editor—We read with interest the article by Vydra and colleagues about enterococcal bloodstream infections (EBSIs) in hematopoietic stem cell transplant (HSCT) recipients [1]. In their experience, the incidence of EBSI was 12%, and in 61 adult patients 66% of enterococci were resistant to vancomycin (VRE) [1]. The mortality 30 days after EBSI was 38% for both VRE and vancomycin-susceptible enterococci (VSE), and EBSI, particularly due to VRE, was associated with lower 1-year survival [1]. Using the same statistical approach, we reviewed our experience, based on new data and a reanalysis of a previous study [2].

From 2004 to 2011, 67 cases of EBSI were diagnosed among 582 adult allogeneic HSCT recipients, with a cumulative incidence of 11.5% (95% confidence interval [CI], 9.1%–14.2%). However, only 13% of infections (9/67) were caused by VRE. Pharyngeal colonization with enterococci (irrespective of their susceptibility to vancomycin), severe mucositis, treatment with cephalosporins, and poorer general conditions (expressed by lower Karnofsky score) were significant risk factors for early (within 30 days from HSCT) EBSI, whereas previous empirical therapy with vancomycin decreased the risk [2]. The mortality 30 days after BSI was 26% for VSE and 11% for VRE. The 1-year posttransplant overall survival was 23% (95% CI, 12%–36%) for all EBSIs, 24% (95% CI, 12%–37%) for VSE, 24% (95% CI, 4%–54%) for VRE, and 65% (95% CI, 61%–69%) for patients without EBSI, compared with 48% for VSE, 23% for VRE, and 63% for no EBSI reported by Vydra and colleagues. The 1-year nonrelapse mortality was 63% (95% CI, 47%–80%) for all EBSIs, 64% (95% CI, 47%–81%) for VSE, 44% (95% CI, 13%–90%) for VRE, and 23% (95% CI, 19%–27%) for patients without EBSI, compared with 33% for VSE, 53% for VRE, and 22% for no EBSI reported by Vydra and colleagues. However, when evaluating the hazard ratio for all-cause mortality in patients with EBSI, we found that the impact of EBSI was most prominent during the first 3 months after HSCT, although it persisted at 12 months as well (Table 1).

In summary, 2 main differences emerged between the 2 studies. First, the overall mortality associated with EBSIs, irrespective of vancomycin susceptibility, was as high as the one reported for VRE by Vydra and colleagues, even though most of the infections were due to VSE and vancomycin was included in initial empirical therapy. Thus, in our experience, and contrary to what has been reported by others [2, 3], we did not find any difference in mortality depending on vancomycin susceptibility. Second, EBSI, due to both VSE and VRE, had a significant impact not only on the long-term but especially on the short-term risk of all-cause mortality as compared to patients without EBSI, data that were not shown by Vydra and colleagues.

We confirm that EBSI is a marker of poor short- and long-term outcome. More clinical and microbiological data are necessary to explain why in our center VSE were associated with lower survival compared to Vydra and colleagues, despite no difference between the 2 studies in overall survival and nonrelapse mortality among patients without EBSI and lower mortality 30 days after EBSI.

Note

Potential conflicts of interest. All authors: No reported conflicts.

References


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Clinical Infectious Diseases 2012;55(12):1744

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DOI: 10.1093/cid/cis785