dence-rate ratios from this analysis are somewhat closer to those reported for the high-income cohorts in the original analysis [2]. The sensitivity analysis prompted by the letter from Caluwaerts and Colebunders thus strengthens our conclusions that the reduction in rates of TB during the first year of HAART is similar in low-income and high-income settings.

Acknowledgments

Potential conflict of interest. M.W.G.B. and M.E.: no conflicts

Martin W. G. Brinkhof and Matthias Egger
Department of Social and Preventive Medicine, University of Bern, Bern, Switzerland

References


Reprints or correspondence: Dr. Martin W. G. Brinkhof, Dept. of Social and Preventive Medicine, University of Bern, Finckenhubelweg 11, CH-3012 Bern, Switzerland ( brinkhof @ispm.unibe.ch).

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Table 1. The association of vancomycin treatment group and OR of mortality.

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<td><strong>Treatment group</strong></td>
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<td>VMIC 1.0</td>
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<td>VMIC 1.5</td>
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<td>VMIC 2.0</td>
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<td>VMIC 2.5</td>
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NOTE. Data are summarized from the article by Soriano et al. [1]. NA, receipt of inappropriate empirical therapy; VMIC 1.0, receipt of empirical vancomycin and an isolate with a vancomycin MIC of 1 μg/mL; VMIC 1.5, receipt of empirical vancomycin and an isolate with a vancomycin MIC of 1.5 μg/mL; VMIC 2.0, receipt of empirical vancomycin and an isolate with a vancomycin MIC of 2 μg/mL.

Soriano et al. [1]). The authors placed shock as a covariate in their multivariable model. They give the rationalization that this is a negative confounder of the association between treatment group and mortality.

I would argue, however, that shock should not be in the model, because it is on the causal pathway from treatment group to death. Including this in the model would generate OR estimates for the association of treatment group and mortality, which then are—theoretically— independent of shock.

I am not sure how to interpret their model in this context or the multivariable OR they present. What is the reason to include shock as a covariate?

It would be helpful to see a multivariable model for the association of treatment group and mortality without shock as a covariate in the model. It would also be helpful to see an assessment of the overall statistical significance of the treatment-group effect in the model, in addition to the individual OR by subgroup.

Potential conflict of interest. M.E.L.: no conflicts

Mark E. Lustberg
Division of Infectious Diseases, Department of Internal Medicine, Ohio State University Medical Center, Columbus

Reference


Reprints or correspondence: Dr. Mark E. Lustberg, Div. of Infectious Diseases, Dept. of Internal Medicine, Ohio State University Medical Center, N1147 Doan Hall, 410 W. 10th Ave., Columbus, OH 43210 (mark.lustberg@osumc.edu).

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Vancomycin Minimum Inhibitory Concentration as a Predictor of Mortality in Methicillin-Resistant Staphylococcus aureus Bacteremia: A Second Look

To the Editor—We read with interest the article by Soriano et al. [1] that described vancomycin MIC as a predictor of mortality in patients with methicillin-resistant Staphylococcus aureus bacteremia. The issue of MIC “creep” was documented elsewhere [2], and the conclusion that a higher MIC is associated with an increased risk of mortality is not surprising. In addition, the presence of shock associated with methicillin-resistant Staphylococcus aureus bacteremia was documented elsewhere as a risk factor for mortality [3]. However, the negative association between the development of shock and vancomycin MIC is extremely intriguing. Soriano et al. [1] hypothesize that this relationship could be attributed to a decrease in pathogenicity as resistance increases through a variety of mechanisms. We offer an alternative explanation of the data and address some concerns with the study by Soriano et al. [1].

After examination of the absolute incidence of patient characteristics, it is clear that the development of shock is negatively associated with vancomycin MIC without adjustment for confounding variables (for 1 μg/mL, 28.4%; for 1.5 μg/mL, 20.2%; for 2 μg/mL, 10.9%; P = .007). It is also clear that heart failure occurred in a significantly higher percentage of pa-

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