Reply to Mateu de Antonio et al.

To the Editor—We thank Mateu de Antonio and colleagues for their comments on our article [1]. We agree with their opinion that baseline differences between groups could affect the results. However, unlike among the general population, age is not an independent factor associated with the development of thrombocytopenia in critically ill patients, such as the patients in our study [2, 3]. One large series study found that 74% of patients with idiopathic thrombocytopenic purpura were younger (age, <40 years old) [4]. In our study, thrombocytopenia was acquired and did not fit the definition of idiopathic thrombocytopenic purpura [5]. In our study, a higher percentage of older patients (>60 years old) did not have thrombocytopenia before receiving linezolid therapy, compared with younger patients (9 [17.0%] of 53 patients vs. 7 [18.4%] of 38 patients; P = 1.000). In this model, the effect of end-stage renal disease on thrombocytopenia is adjusted by modified sequential organ failure assessment (m-SOFA) score.

The methodology used in safety evaluations must be defined in detail. In our practice, linezolid therapy was discontinued if thrombocytopenia was detected in patients who were initially nonthrombocytopenic or if the platelet count decreased by at least 25% after initiation of linezolid therapy in patients who were initially thrombocytopenic. The use of different definitions of initial thrombocytopenia makes it difficult to compare our findings with those of Gerson et al. [6]. Analysis including initial thrombocytopenia status revealed that patients with initial low platelet counts were more likely to develop thrombocytopenia than not (14 [28.5%] of 49 patients vs. 2 [3.8%] of 42 patients; P = .002). The independent risk factors for thrombocytopenia in the multivariate logistic regression analysis were m-SOFA score (OR, 1.30 [95% CI, 1.09–1.56]; P = .004), initial thrombocytopenia (OR, 13.3 [95% CI, 1.74–100.0]; P = .013), central catheter–related infection (OR, 8.12 [95% CI, 1.57–42.07]; P = .013), and end-stage renal disease (OR, 5.62 [95% CI, 1.30–24.39]; P = .021). After adjustment for initial thrombocytopenia, end-stage renal disease remained a significant independent risk factor. Patients with initial anemia did not have a significantly higher incidence of post-treatment anemia. (18 [45.0%] of 45 patients vs. 25 [54.3%] of 46 patients; P = .210).

We did not use any of the well-known potential thrombocytopenia–inducing agents in the study groups, except for heparin, which is widely used during hemodialysis. Despite repetitive heparin exposure, the prevalence of heparin-induced thrombocytopenia among dialysis patients is no greater than that anticipated for other patient populations [7]. Because all patients with end-stage renal disease received dialysis before starting linezolid treatment, the possibility that dialysis or heparin induced thrombocytopenia or anemia after the start of linezolid therapy is less likely. However, the thrombocytopenia group had a higher rate of platelet transfusion (73.5% vs 42.9%, P = .003), which was not an independent risk factor for thrombocytopenia (P = .091). The anemia and nonanemia groups had similar rates of packed RBC transfusion during linezolid therapy (60.5% vs. 50.0%, P = .400).

Finally, this was a 2-year retrospective study that included all patients who received linezolid therapy. We agree that there is significant heterogeneity and a wide dispersion of data in our study population. However, the point of this manuscript is to alert the physician about the range of possible hazards of using linezolid in the broad population of patients with end-stage renal disease. Additional prospective studies are required to examine hypotheses about the hazards of linezolid use in specific subpopulations of patients with end-stage renal disease.

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Potential conflicts of interest. All authors: no conflicts.

References


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Diagnostic Insufficiency in Africa

To the Editor—I read with interest and some solace the viewpoint article by Petti et al. [1] and the accompanying editorial commentary [2] published in the 1 February 2006 issue of Clinical Infectious Diseases. Diagnostic insufficiency in Africa, highlighted by both articles, is severely