Bloodstream Infections Due to *Micrococcus* spp and Intravenous Epoprostenol

To the Editor—We read with interest the article by Kallen et al1 published in the April 2008 issue of the journal. Kallen et al presented a retrospective cohort study of bloodstream infection (BSI) in patients treated with intravenous prostanoids. The authors concluded that BSI due to gram-negative pathogens was more common in patients who received treatment with intravenous treprostinil than among patients who received treatment with intravenous epoprostenol. The authors reported the organisms that were isolated in blood samples from both groups. We believe that it is important to further examine these results.

Although the novel finding in this report was the higher rate of BSI due to gram-negative pathogens among patients treated with intravenous prostanoids, the authors did not comment on the high rate of BSI due to *Micrococcus* spp in patients treated with epoprostenol. In the latter group, micrococci were the second most common type of bacterium isolated (11 cases); in contrast, micrococci were isolated in none of the patients who received intravenous treprostinil. *Micrococcus* spp have been reported consistently as the second most common etiologic agent of BSI in patients receiving epoprostenol after *Staphylococcus* spp3,4.

In January 2008, we submitted a paper reporting the common occurrence of BSI due to *Micrococcus* spp among patients treated with intravenous epoprostenol at our institution.5 During the period from January 2001 to December 2006, 45 cases of BSI occurred in patients who received intravenous epoprostenol through a Groshong catheter. There were 13 cases of BSI due to *Staphylococcus aureus*, 8 cases of BSI due to *Staphylococcus epidermidis*, and 5 cases of BSI due to *Micrococcus* spp. Because no patients at our institution were being treated with intravenous treprostinil at that time, we reviewed the blood culture results from 657 patients who were using a Groshong catheter during the same period for reasons other than pulmonary hypertension. Strikingly, we did not find any micrococal BSIs in this group of patients.

Why are cases of BSI due to *Micrococcus* spp more frequent in patients treated with intravenous epoprostenol, whereas they are almost nonexistent in other groups with long-term central venous catheters, including patients who are treated with intravenous treprostinil? This question may have interesting answers. Maybe epoprostenol creates the right environment for the growth of micrococci during the preparation, storage, or delivery of the drug. But the most intriguing avenue for investigation, as we also suggested, is to explore the role of the prostanoids in modulation of the immune response in vivo as has already been demonstrated in vitro.6 The answer to our question may have implications for the development of new types of therapy for pulmonary hypertension.

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Martin A. Valdivia-Arenas, MD

From the Department of Internal Medicine, Blanchard Valley Health System, Findlay, Ohio.

Address reprint requests to Martin A. Valdivia-Arenas, MD, Department of Internal Medicine, Blanchard Valley Health System, 1900 South Main Street, Findlay, OH 45840 (martin1990br@yahoo.com).

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REFERENCES


A Tertiary Care Cancer Center Experience of the 2007 Outbreak of *Serratia marcescens* Bloodstream Infection Due to Prefilled Syringes

To the Editor—We read with great interest the article by Su et al1 that describes the 2007 outbreak of *Serratia marcescens* bloodstream infection in Texas due to contaminated prefilled heparin syringes. We had a similar outbreak at our institution2 during the same period, but interestingly the product incriminated in the investigation by Su et al1 was not being used at our institution. Even after the recall of all prefilled heparin syringes on December 20, 2007,3 there were still new cases of *S. marcescens* bloodstream infection occurring among our patients with cancer, with some of these cases acquired nosocomially. The infection control team suspected a second...