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Rash as a Prognostic Factor in West Nile Virus Disease

To the Editor—As West Nile virus (WNV) becomes widespread throughout the United States, larger cohort data allow investigators to more precisely identify determinants of clinical outcomes of disease. We commend Bode et al. [1] for uncovering additional factors associated with adverse events in patients hospitalized with WNV infection during an outbreak in 4 Colorado counties in 2003. In this population with high morbidity, multivariate analysis of the presence of rash as a prognostic factor for severe disease and mortality would have been of interest.

Two recent studies of large-scale outbreaks of WNV in the United States have highlighted this finding. In 2002, the Illinois Department of Public Health reported 884 cases of WNV infection and 66 deaths due to infection [2]. Rash was a common finding among all patients for whom information was available (301 [46%] of 654 patients), as well as among patients with neuroinvasive disease (151 [39%] of 390 patients). Among patients with reported rash, age-adjusted risks were significantly decreased for encephalitis (relative risk, 0.67; 95% CI, 0.53–0.84), encephalitis plus death (relative risk, 0.44; 95% CI, 0.21–0.92), and death (relative risk, 0.39; 95% CI, 0.19–0.81). In 2003, the Colorado Department of Public Health and Environment reported 2947 cases of WNV infection and 63 deaths due to infection throughout the entire state [3]. Of a total of 1564 patients (60%) had signs of rash among evaluable cases. Age-adjusted risks for meningitis (OR, 0.7; 95% CI, 0.6–0.9), encephalitis (OR, 0.5; 95% CI, 0.3–0.6), and death (OR, 0.3, 95% CI, 0.1–0.8) were also similarly decreased in patients with reported rash [4]. A characteristic rash typically appears transiently in a diffuse maculopapular pattern at the height of febrile symptoms [4–6]. Few studies have examined the histopathological characteristics of rash lesions in WNV infection. In 1 case series, skin biopsy revealed superficial perivascular lymphocytic infiltrates seen commonly in viral exanthems [6]. Whether the development of rash in WNV infection reflects a functional immunoprotective response to circulating viral antigens requires further investigation and validation. Future surveillance activities should include prospective studies assessing features of rash that might account for its apparent favorable effect against severe disease and mortality in WNV disease.

Acknowledgments


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To the Editor—We congratulate Fournier and Richet [1] on their insightful article on the hot topic of the epidemiology and control of Acinetobacter baumannii infection in health care facilities. We believe that the authors’ statement that “potentially severe A. baumannii infection, such as bacteremia or pneumonia in patients in the intensive care unit [ICU] who are undergoing intubation, do not seem to be associated with a higher attributable mortality or increased length of hospital stay” [1, p. 692] needs some clarification.

The authors referred to 2 matched cohort studies performed in critically ill patients by Blot et al. [2] and Garnacho et al. [3]. In both studies, the observed difference in mortality between patients with and without A. baumannii infection was not statistically significant. However, in the study by Blot et al. [2], an ICU stay in excess of 5 days was found in patients with A. baumannii bacteremia (the median length of ICU stay for patients with and without A. baumannii bacteremia was 25 days and 20 days, respectively). This finding was statistically significant ($P = .04$).

García-Garmendia et al. [4] also performed a matched case-control study that compared the outcomes of patients hospitalized in the ICU with and without A. baumannii acquisition (defined as infection or colonization). They extracted the data from this study regarding the outcomes of the subsets of patients with A. baumannii infection (we excluded patients with A. baumannii colonization). The crude ICU mortality among patients with and without A. baumannii infection was 58% and 15%, respectively—a statistically significant difference ($P < .001$)—resulting in an attributable mortality for A. baumannii infection of 43%. In addition, the median length of ICU stay was 13 days longer for patients with A. baumannii infection. This finding was also statistically significant ($P < .001$).

Furthermore, Weingarten et al. [5] performed a matched case-control study that compared patients, most of whom were hospitalized in ICUs, with and without A. baumannii acquisition. We also extracted the relevant data from this study regarding the subsets of patients with A. baumannii infection. Although data regarding the attributable mortality for A. baumannii infection were not available, a statistically significant longer ICU stay was found among patients with A. baumannii infection.

We recently performed a systematic review of relevant matched cohort and case-control studies to evaluate the attributable mortality of A. baumannii infection among critically ill patients [6]. In the 6 matched cohort and case-control studies included in our review, we found that the attributable in-hospital mortality among patients with A. baumannii infection was 7.8%–23% and that the ICU mortality was 10%–43% [2–4, 7–9]. Although definitive statements regarding the attributable mortality of A. baumannii infection cannot be made from the available studies because of their methodological heterogeneity, we believe that the data from the relevant studies suggest that infection with A. baumannii may be associated with considerable attributable mortality and increased length of ICU stay.

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**Reply to Falagas et al.**

To the Editor—Falagas et al. [1] suggest in their letter to the editor that we underestimated the attributable mortality of Acinetobacter baumannii infections among critically ill patients in our review [2]. To assess the impact of infection in terms of morbidity, functional status, extra costs, or mortality is essential to a better knowledge of those infections. However, such assess-