Clostridium difficile Infections among Hospitalized Children, United States, 1997–2006

To the Editor: Zilberberg et al. described a notable increase in rates of *Clostridium difficile* infection (CDI)—related hospitalizations of children during 1997–2006 on the basis of analysis of data from 2 national administrative databases (1). As the authors acknowledge, they used administratively coded databases, which have inherent misclassification and testing biases.

Detection of *C. difficile* toxin indicates that bowel flora have been perturbed. However, the clinical role of toxin detection or isolation of *C. difficile* organisms in children is controversial. Although primary CDI is a recognized pathologic entity in children, one needs to consider whether another etiology related to a concomitant infection, antimicrobial drug administration, or alteration in enteral nutrition may be the precipitating event resulting in *C. difficile* toxin production.

It is our clinical observation that availability of testing for *C. difficile* and rapidity of assay results play a role in the submission of stool specimens for analysis. In 2007, we conducted a 5-month retrospective chart review of *C. difficile* testing practices at 2 local tertiary-care pediatric hospitals. Of 796 stool specimens submitted, 42 (5%) were notable for the detection of toxin A or B; these samples represented 35 patients (2). Medical coders likely face the same challenges as clinicians who must interpret toxin assay results and their clinical role with regard to hospitalized children. Although the ~2-fold increase in CDI-associated hospitalization rates reported by Zilberberg et al. in their time series and cross-sectional analyses is notable, these results should be interpreted within the context of clinical and epidemiologic factors contributing to generation of this data.

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References


2. Vindigni SM, Sullivan DH, Shane AL. To treat or not to treat? Optimizing pediatric *Clostridium difficile* management. Poster presented at Fifth Decennial International Conference on Healthcare-Associated Infections. 2010 Mar 18–22; Atlanta, GA, USA.

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In Response: I appreciate the letter by Vindigni and Shane pointing out the need for a cautious approach to treatment for infection with *Clostridium difficile* isolated from the stool of children, given their propensity for colonization by this organism (1). I could not agree more. In our report, we noted an increase over time in the rate of hospitalizations for not only *C. difficile* infections (CDIs) but also for rotavirus infections in children. This finding led us to acknowledge the possibility of a reporting bias for CDI (2). Other studies have detected a similar increase in CDIs among hospitalized children and have reported greater severity of associated disease (3–5). Such epidemiologic data, combined with emergence of the BI/NAP1/027 hypervirulent strain of *C. difficile* in the United States and abroad, support
a real increase in CDIs in a population for which the clinical definition is likely less specific than for adults. Although a more precise clinical definition for CDI in children (primarily those <2 years of age) is needed, studies like ours, which are necessarily limited methodologically, can serve to alert clinicians to be more vigilant to the possibility of disease caused by this evolving pathogen, even in a population thought to be at low risk.

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