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Reply to Blot et al.

TO THE EDITOR—We appreciate the comments by Blot et al. [1] in this issue of Clinical Infectious Diseases. During 2000–2005, rectal surveillance for extended-spectrum β-lactamase–producing Enterobacteriaceae (ESBL-E) in our hospital demonstrated a 2.3% colonization rate among 17,872 high-risk patients [2]. Bloodstream infection due to extended-spectrum β-lactamase–producing Enterobacteriaceae (ESBL-BI) among screened patients was recorded during the study period. Screen-negative patients developed ESBL-BI in 8.5% of cases, the positive predictive value of our surveillance system. Of 17,459 screen-negative patients, only 11 (0.1%) developed an ESBL-BI; the negative predictive value of our system was 99.9%.

As noted by Blot et al. [1], several European studies have demonstrated a correlation between clinical isolates and pathogens of bloodstream infection in high-risk patients [3–5]. Because our study [2] was not designed to evaluate all bloodstream infections among high-risk patients, we cannot comment on this correlation in our patient population.

Blot et al. [3] found that pathogen prediction with tracheal surveillance cultures in the intensive care unit resulted in higher rates of appropriate antibiotic therapy within 24 h and higher rates of survival in patients with pneumonia-related bloodstream infection [6]. In contrast, our recent subset analysis of ESBL-BI among ESBL-E screen-positive, screen-negative, and unscreened patients did not suggest a difference in rates of appropriate antibiotic treatment or patient mortality [7].

In our study [2], 11 cases of ESBL-BI occurred among screen-negative patients, yielding a sensitivity of 76.1%. Of interest, 5 of these 11 patients had an infection with an ESBL-E clinical isolate (urine [3 patients], wound [1 patient], and respiratory tract [1 patient]) before the ESBL-BI. Although the use of a more comprehensive surveillance system, as described by Blot et al. [3] (urine and sputum cultures in addition to culture of rectal samples), may have yielded a higher sensitivity in prediction of ESBL-BI in our population, the cost-effectiveness of this strategy is unknown [3]. In addition, lower ESBL-E prevalence rates in the United States [8] may limit our ability to produce similar results.

We agree that a cost-benefit analysis of ESBL-E screening may determine the need for an expanded surveillance program in the future. In the meantime, intensive physician education efforts will be needed where active ESBL-E surveillance is currently performed, in an effort to optimize the use of culture results in clinical decision making.

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References


Epidemiology of Invasive Group A Streptococcal Infection in the United States

TO THE EDITOR—We read with interest the study of invasive group A streptococcal infection (IGASI) by O’Loughlin et al. [1]. The authors are to be commended for an informative report on the epidemiology of IGASI in the United States that used data from the Active Bacterial Core Surveillance program.

According to O’Loughlin et al. [1], the
Active Bacterial Core surveillance program has expanded its definition of the risk factor skin condition to include blunt trauma. This is a timely event, given the recent interest in the biological plausibility of the hypothesis that nonpenetrating injury is a risk factor for group A streptococcal necrotizing fasciitis and myonecrosis [2]. We have linked a history of recent blunt trauma to the presence of group A streptococcal necrotizing fasciitis in a group of patients hospitalized for IGASI throughout Florida [3]. The results of our exploratory study could be confirmed by a case-control study using data from the Active Bacterial Core surveillance system.

With regard to the multivariable analysis of risk factors for mortality [1], the investigators have not accounted for confounding by certain laboratory parameters and antibiotic regimens. For example, it has been shown in a regression spline analysis that the serum albumin level at the time of hospital admission appears to be a predictor of mortality in patients hospitalized for IGASI [4]. In addition, patients with a particular manifestation of IGASI, such as necrotizing fasciitis, may be more likely to receive clindamycin therapy. Clindamycin therapy has been shown to reduce the odds of hospital mortality by 89% (adjusted OR, 0.11) among patients admitted to the hospital for IGASI who have necrotizing fasciitis [5]. Perhaps the Active Bacterial Core surveillance program could collect data on these potential confounders and/or effect modifiers for a limited number of sites that participate in this useful surveillance system.

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References


Therapeutic Effectiveness of Ionic Zinc for Common Colds

To the Editor—Caruso et al. [1] reviewed 11 reported quality criteria to assess the therapeutic effectiveness of zinc products for common colds. However, the authors did not consider the most fundamental criterion—the dose response of the active ingredient ionic zinc. It has use in treating colds through antirhinoviral means [2, 3] by reducing the level of intercellular adhesion molecule-1 [4], increasing IFN-γ levels by 10-fold [5], and protecting cell plasma membranes [6].

In 2004, I reanalyzed all of the double-blind, placebo-controlled studies of zinc lozenges for the common cold, including the British Medical Research Council Common Cold Unit’s positive report of zinc gluconate in treating laboratory human rhinovirus type 2 common colds [7, 8]. I researched the data on dose response in these studies and reported a conclusion that was exactly opposite to the conclusion of Caruso et al. [1]. My reanalysis [8] showed statistically significant correlations between total daily dose of ionic zinc and reduction in median duration (P = .005) and mean duration (P < .02) of colds but not between total zinc received and mean duration (P = .12) of colds. This report showed that nearly all single-ligand zinc gluconate and zinc acetate lozenges succeeded in reducing the duration of colds and nearly all multiligand zinc lozenges failed in the reducing the duration of colds.

My report [8] was the only review to consider dose response, and it is unknown why Caruso et al. [1] failed to mention dose response or my report. It was not possible for Caruso et al. [1] to review the upcoming report by Prasad [4], who found a substantial reduction in the duration of colds (3.5 vs. 7.4 days; P < .001) with the use of zinc acetate (13.3 mg zinc) lozenges, which are known to release ionic zinc.

Future studies should focus on plain zinc acetate lozenges that are chemically identical to the lozenges reported to be effective by Petrus et al. [9] in 1998, by Prasad et al. [10] in 2000, and in the upcoming report by Prasad [4]. Such lozenges have been previously described [11] and have been highly successful in treating colds both in clinical trials and in the general population. Although billions of dollars have been spent on zinc lozenges for treatment of colds, money spent on zinc lozenges containing amino, citric, or ascorbic acids may have been wasted.

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