The Low Prevalence of Shiga-Toxin Production among Sorbitol Non-Fermenting Escherichia coli Urinary Tract Isolates Does Not Warrant Routine Screening

SIR—Enterohemorrhagic strains of Escherichia coli (EHEC), which cause disease, at least in part, by producing shiga-toxins, have become a serious public health concern [1]. Although hemolytic uremic syndrome most commonly follows hemorrhagic colitis, it has been reported to occur rarely after urinary tract infections with EHEC [2, 3]. It has, therefore, been suggested that direct detection of shiga-toxin should be performed on E. coli isolates from urine specimens [2].

We collected sorbitol non-fermenting E. coli isolates from urine cultures for 1 year (December 1998 through December 1999) and examined the prevalence of shiga-toxin production in these isolates. Eighty-one isolates were collected from a varied patient population. The urine specimens were submitted from the Emergency Department (10), outpatient clinics (44), hospitalized patients (18), or our reference laboratory (9). Seventy-seven of the isolates were from women and 4 were from men. The isolates collected were present in the urine specimens in quantities of at least >10,000 cfu/mL. The organisms were identified and the sorbitol fermentation status determined by use of the Vitek Gram Negative Identification card (bio-Merieux, Hazelwood, MO). The isolates were tested for the presence of shiga-toxin 1 (stx1) and/or shiga-toxin (stx2) by using the Premier EHEC kit (Meridian Diagnostics, Cincinnati, OH) according to the manufacturer’s guidelines. None of the 81 E. coli isolates tested demonstrated the presence of stx1/stx2. A clinical stool isolate of E. coli O157:H7, from a patient with hemorrhagic colitis, was used as the positive control and consistently demonstrated the presence of shiga-toxin.

Rather than screening all E. coli urinary isolates for the presence of stx1/stx2, we screened only sorbitol non-fermenting E. coli isolates, because this EHEC phenotype (O157:H7) is relatively common in North America. We found a complete absence of shiga-toxin producing E. coli isolates in this selected population and, therefore, suggest that the routine screening of E. coli isolates from urine specimens for the presence of stx1/stx2 is not warranted. The screening of E. coli urinary tract pathogens for the presence of stx1/stx2 may be more fruitful if limited to isolates from patients with evidence of hemolytic uremic syndrome or, possibly, hemorrhagic cystitis.

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Appropriate Antibiotic Treatment for Pneumonia

SIR—We read with great interest the article by Gonzalez et al. [1] that described a high mortality rate among patients treated with vancomycin for pneumonia caused either by methicillin-resistant Staphylococcus aureus (MRSA; 50%) or by methicillin-sensitive S. aureus (MSSA; 47%). In contrast, the authors have found that in the subgroup of patients receiving cefoxitin treatment for pneumonia caused by MSSA, the mortality rate was zero [1]. Among intubated patients receiving cefoxitin for pneumonia caused by MSSA, we found a mortality rate of 2.6%; in episodes caused by MRSA and treated with intermittent administration of vancomycin (with serum level monitoring), we found a mortality rate of 54.5% [2]. Moreover, 2 of our patients developed an MRSA episode even though they were receiving treatment with vancomycin. Postmortem cultures performed for 3 of these patients showed that MRSA...
strains persisted even though these patients had received vancomycin. The discrepancies between reports of microbiologic resistance and clinical outcomes may be due to the fact that sustained tissue levels above MIC are far more determinant of clinical outcome.

In our view, the findings reported by both papers [1, 2] indicate that the classical definition of "appropriate antibiotic therapy" for serious infections—that it should be based on obtaining serum antimicrobial levels above the MIC for likely infecting pathogens—is not correct. This definition is appropriate for intravascular infections, such as endocarditis, but clinical results in pneumonia may depend more on tissue penetration than on serum levels. This may explain the unexpected lack of efficacy of vancomycin treatment for cases of pneumonia caused by gram-positive cocci [1, 2], the lack of clinically observed failures in lung infections treated with antibiotics that have poor activity against penicillin-resistant Staphylococcus pneumoniae [3], and the success of ultrashort iv antibiotic regimens [4].

These clinical observations are important for many reasons. First, clinical trials of new antibiotics should no longer accept vancomycin treatment as standard therapy for pneumonia caused by gram-positive cocci. Second, in spite of the reports of its sensitivity, vancomycin should not be considered as first line therapy for gram-positive lung infections. Finally, until more data are available, patients with pneumonia caused by MRSA should receive combination therapy with vancomycin plus another antistaphylococcal agent (such as rifampin). Because vancomycin exhibits concentration-dependent killing, our opinion is that vancomycin should be prescribed in continuous infusion with the goal of maintaining MICs that are >20 mg/mL.

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References