unacceptably high rates of treatment failure [2, 3]. In vitro data indicate that the newer fluoroquinolones are active against Listeria species but have relatively poor penetration into the CNS, and ciprofloxacin used in a murine model of listeriosis was ineffective [4].

Linezolid is active against L. monocytogenes in vitro [5], and CSF concentrations of linezolid that are adequate for treatment have been attained in a rabbit model [6]. Additional data derived from the use of linezolid in neurosurgical settings, as well as the efficacy demonstrated in the treatment of CNS listeriosis in a murine model [7, 8], prompted us to use linezolid in the patient we describe.

Rifampin crosses the blood-brain barrier, penetrates cell membranes, and is also active against L. monocytogenes. In a study of isolates recovered from patients with Listeria meningitis, rifampin and TMP-SMZ were the most potent monotherapeutic drugs tested [9]. Combination treatment involving rifampin and another active antimicrobial may reduce the emergence of resistance to rifampin. Our success with the combination of linezolid and rifampin may offer a valid alternative therapy for brain abscess caused by L. monocytogenes.

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case of hepatitis E were defined as those who had clinical jaundice that appeared after >1 week of hospital stay and who had serum IgM antibodies to HEV that were detectable via ELISA (Abbott Diagnostics). In the absence of a positive serological marker for HEV infection, patients received a presumptive diagnosis if the incubation period was >2 weeks in duration, jaundice occurred during the outbreak period, and the patient had an elevated alanine transaminase level. Other types of viral hepatitis were excluded by serological testing, as was drug-induced hepatitis.

The overall ward attack rate for HEV infection was 15.9% (18 of 113 patients). Ten patients were women, the mean patient age was 27.6 years, and the median incubation period for patients with outbreak cases was 23 days (range, 7–120 days). Compared with control subjects, the group of patients with either proven or presumptive HEV infection received either intravenous dexamethasone (OR, 4.6; 95% CI, 1.4–15.9; P < .008) or intravenous mannitol (P < .001) more frequently, but they did not receive blood transfusions more frequently (OR, 2.71; 95% CI, 0.65–11.79; P < .14). Bacteriological testing of samples of ward and select hospital water sources and outlets found no evidence of fecal contamination. Interviews with nursing staff and other ward employees disclosed the inappropriate practice of administering mannitol and dexamethasone to patients by means of shared intravenous administration sets. The reason given for this practice was a shortage of nursing staff, especially during evening and night hours. Six subjects (33%) died, including a pregnant woman.

Sporadic nosocomial cases of serologically confirmed or clinically presumptive HEV infection have been reported, including a hospital outbreak involving a doctor and 2 nurses who were exposed to a patient’s blood, lochia, and stool during repair of vaginal lacerations [2]. A number of seroepidemiological studies have suggested that HEV may share transmission routes with hepatitis B, hepatitis C, or hepatitis D viruses in populations of patients who receive blood component therapy [3,4]; undergo hemodialysis [5], plasmapheresis [6], or renal transplantation [7]; or use illicit injection drugs [8]. Patients with HEV infection are viremic during the acute phase of illness, and the duration of viremia may occasionally extend beyond the time of biochemical resolution [9].

A major limitation of our study is the unavailability of HEV RNA data to document viremia and support the epidemiological suggestion that HEV was parenterally transmitted during the outbreak. Nevertheless, in this outbreak of hospital-acquired HEV infection in a neurosurgical ward, receipt of dexamethasone or mannitol infusions via shared intravenous administration sets was a risk factor significantly associated with infection, and this strongly implicates a parenteral route of HEV transmission.

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Abacavir Pharmacokinetics in Hepatic Dysfunction

Sir—In our article entitled “Antiretroviral Pharmacokinetics in Hepatitis with Hepatic Dysfunction” [1], we stated that no data existed on the pharmacokinetics of abacavir in persons with hepatic dysfunction. We point out that, although abacavir is not metabolized by the cytochrome P450 system, its metabolism through cytosolic alcohol dehydrogenase and uridine diphosphate–glucuronyltransferases could potentially be altered in patients with hepatic dysfunction. In our article [1], because of the lack of data, we do not recommend any dosing adjustment of abacavir in patients with hepatic dysfunction. Shortly after this article was accepted for publication, the manufacturer of abacavir, GlaxoSmithKline, published an updated version of the package insert for abacavir that included new data on the effect of hepatic dysfunction on abacavir pharmacokinetics, as well as new recommendations for dosing adjustments in this situation [2].

Data provided in the package insert state that a mean increase of 89% in the abacavir area under the curve was found

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