


Rapidly Fatal Bacteremia Caused by Shigella sonnei without Preceding Gastrointestinal Symptoms in an Adult Patient with Lung Cancer

To the Editor—Shigella sonnei is a rare cause of fatal bacteremia in adult patients [1–5]. We describe an unusual case of a patient with S. sonnei bacteremia who presented with adult respiratory distress syndrome and multiorgan failure that led to rapidly fatal outcome.

A 62-year-old man with underlying diabetes mellitus, hypertension, and hyperlipidemia visited our hospital after experiencing shortness of breath and palpitation for 3 weeks. He was a merchant and lived in Indonesia. He visited a local hospital in Indonesia and received a diagnosis of hepatomegaly and multiple hepatic nodules. Neither cardiovascular abnormalities nor evidence of acute coronary syndrome were noted. Seven days prior to admission to our hospital, he visited a regional hospital in Taiwan. Computed tomography disclosed a hilar mass in the right mediastinum and several nodules in the right lower lung field and the liver. Transectional biopsy of the hepatic tumor revealed small cell lung cancer. He was transferred to our hospital to receive chemotherapy for lung cancer with liver metastasis.

On initial examination, he was aware and alert but appeared to be chronically ill. He was afebrile (body temperature, 36.3°C), had a pulse rate of 101 beats per min, and had a respiratory rate of 20 breaths per min. Icteric sclerae and hepatomegaly were noted. His white blood cell count was 4920 cells/μL, his hemoglobin level was 11.5 g/dL, and his platelet count was 129,000 cells/μL. Biochemical examinations revealed a bilirubin level (total/direct) of 17.8/11.2 mg/dL, an alanine aminotransferase level of 363 mg/dL, an aspartate aminotransferase level of 359 mg/dL, and a serum creatinine level of 1.8 mg/dL. Chest radiography disclosed a right hilar mass and a small amount of pleural effusion. Chemotherapy with cisplatin (70 mg once per day) and etoposide (100 mg once per day) was initiated. Chest tightness developed on the second day after treatment initiation. Electrocardiography revealed inverted T waves over the precordial leads (V4 to V6), elevated creatine kinase levels (579 U/L; reference range, 38–160 U/L), and an elevated creatine kinase–muscle-brain fraction (186.5 U/L; reference range, <16 U/L). A diagnosis of non–ST-segment elevation myocardial infarction was made, and the patient was transferred to the intensive care unit for further treatment. Ten hours later, a high spiking fever (temperature, 40°C) and loss of consciousness occurred. Profound shock with systolic blood pressure of 80 mm Hg also developed (baseline systolic blood pressure was ~160 mm Hg), and the isotropic agents norepinephrine and dopamine were administered. Piperacillin-tazobactam (3000 mg of piperacillin plus 375 mg of tazobactam every 6 h) was administered empirically. Persistent high spiking fever, leukopenia (white blood cell count, 880 cells/μL), anuria, and adult respiratory distress syndrome developed rapidly. Rapid deterioration of liver function (alanine aminotransferase level, 5727 mg/dL; aspartate aminotransferase level, 25,615 mg/dL) and renal function (blood urea nitrogen level, 90.9 mg/mL; serum creatinine level, 3.9 mg/dL) were also noted. Shock and adult respiratory distress syndrome progressed, and the patient died 26 h after the first episode of spiking fever. The patient’s fibrinogen level was 322.3 mg/dL (reference range, 163.5–362.7 mg/dL), his fibrinogen degradation production level was 10 μg/mL (reference level, <4.1 μg/mL), and his D-dimer level was 2.4 μg/mL (reference level, <2.09 μg/mL), indicating the possible presence of disseminated intravascular coagulopathy. No gastrointestinal symptoms, such as diarrhea, nausea, vomiting, or abdominal pain, were noted before or after the febrile episode.

Two sets of blood cultures (Bactec 9240; Becton Dickinson) both yielded S. sonnei growth on the day after the patient’s death. The isolate was identified to the species level with use of conventional biochemical methods, serogrouping, and 3 commercial identification systems: the Vitek Gram Negative Identification Card (identity probability, 99%), the API 20E (identity, 99%; T value, 0.92; bioMérieux), and the Phoenix System (confidence value, 99%; NMIC/ID-4; Becton Dickinson). The isolate was susceptible to ampicillin (minimum inhibitory concentration [MIC], ≤4 μg/mL), ciprofloxacin (MIC, ≤0.5 μg/mL), piperacillin-tazobactam (MICs, ≤4 and ≤4 μg/mL), and cefotaxime (MIC, ≤1 μg/mL) but resistant to trimethoprim-sulfamethoxazole, as determined by the Phoenix System. Stool cultures were not performed because of a lack of diarrhea during hospitalization.

Shigella species often cause diarrhea or gastrointestinal inflammation in humans and are rarely associated with bacteremia [1, 2]. Children are more susceptible than adults, and the most common causative organism is Shigella flexneri [1, 2]. Among the 9 reported cases of S. sonnei bacteremia...
The patient's poor underlying condition (lung cancer and myocardial infarction) probably contributed to the rapidly fatal outcome.

In summary, we report a rapidly fatal case of *S. sonnei* bacteremia without any associated gastrointestinal symptoms. In immunocompromised hosts, *S. sonnei* should be included in the list of etiologies associated with severe sepsis and disastrous complications, even in the absence of preceding gastrointestinal diseases.

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**References**


Efficacy of Early Pegylated Interferon α-2b Monotherapy for Acute Hepatitis C in HIV-Infected Patients

To the Editor—There is no consensus regarding the best treatment for patients with acute hepatitis C and human immunodeficiency virus (HIV) infection. We present our experience with 12-week monotherapy with pegylated interferon α-2b (1.5 µg/kg/week) in 7 patients (4 men and 3 women). Diagnosis of acute hepatitis C was made as described by Jaekel et al. [1], with active surveillance for an increase in the alanine aminotransferase level to detect hepatitis C virus (HCV) infection. Only 1 patient was symptomatic. Five patients had documented seroconversion for anti-HCV antibodies, and 2 patients demonstrated evidence of a new risk factor for acute hepatitis C. The median age of the patients was 33 years (range, 23–61 years), and the median interval between diagnosis and initiation of therapy was 31 days (range, 14–60 days). The median alanine aminotransferase level at the time of diagnosis was 777 IU/L (range, 196–1200 IU/L), and the median HCV RNA level at the initiation of therapy was 3.56 × 10⁶ IU/mL (range, 615–7.59 × 10⁶ IU/mL). Acute hepatitis C was caused by HCV genotype 1 in 3 patients, genotype 4 in 2 patients, and genotype 2 in 2 patients. Three patients were receiving highly active antiretroviral therapy when they received the diagnosis and continued to receive treatment. The median CD4 cell count was 539 cells/µL (range, 340–712 cells/µL). All patients received 100% of the scheduled treatment for acute hepatitis C, and sustained virological response (SVR) was achieved in 4 (66.6%) of 6 patients (1 patient was excluded because of early reinfection with a different genotype). Among the 2 patients who did not achieve SVR, 1 did not respond to treatment, and 1 experienced an early relapse. All patients infected with HCV genotype 1 achieved SVR, compared with 1 of 2 patients infected with genotype 4. The median baseline HCV RNA level was significantly lower among patients who achieved SVR than among those who did not achieve SVR (8199 vs. 2,878,000 IU/mL; *P* = .034). A rapid virological response was observed in only 3 patients who achieved SVR.

The median duration of follow-up after the end of treatment was 229 weeks (range, 50–303 weeks). Among the 4 patients who achieved SVR, 3 (75%) still have undetectable HCV RNA levels and 1 patient apparently experienced relapse with the same genotype at 48 weeks after the end of treatment. The seventh patient, who experienced an early reinfection with a different genotype, spontaneously cleared the low-grade viremia that was caused by reinfection after 96 weeks.

Treatment of acute hepatitis C in HIV-infected patients is not yet standardized; discussion of this treatment often involves timing, treatment duration, and the necessity of ribavirin. Most studies have used a 24-week treatment regimen [2]. For example, among 101 patients treated with pegylated interferon, with or without ribavirin, for a median duration of 25 weeks, a 64% rate of SVR was reported, with no effect of variation in ribavirin or duration [3]; this finding was