Fatal Septic Shock with Multiple Organ Failure Due to Campylobacter jejuni

Campylobacter jejuni infections are among the most common causes of gastroenteritis. However, severe extraintestinal infections have been occasionally described, mainly in immunodeficient patients and patients with neoplasia [1, 2]. To our knowledge, we describe the first case of fatal septic shock due to C. jejuni in a thalassemic patient who had undergone splenectomy.

A 41-year-old man was admitted to the hospital with fever (temperature to 38°C), chills, low blood pressure (80/70 mm Hg), tachycardia (pulse rate, 120), respiratory distress, and drowsiness. Two days before admission, he had had diarrhea, abdominal pain, and fever and had been treated with ampicillin. The patient had a complex medical history of thalassemia intermedia for which he had undergone splenectomy 15 years before. Recurrent venous thrombosis necessitated treatment with oral anticoagulants. He underwent frequent blood transfusions because of a recent diagnosis of hemochromatosis with cirrhosis, diabetes mellitus, and right ventricular failure. Owing to the deterioration of his clinical status, he was admitted to the intensive care unit.

Laboratory studies showed hemolytic anemia (hemoglobin level, 9 g/dL; reticulocyte count, 479 x 10^9/L; hemoglobin F level, 39.6%; hemoglobin A1 level, 4.2%; indirect bilirubin level, 51 μmol/L; very low haptoglobin level), severe acidosis (lactate level, 8 mmol/L; pH, 7.26), acute renal failure (creatinine level, 216 μmol/L; activated partial thromboplastin time, 85 seconds [normal, 32 seconds]; normal factor V level), and leukocytosis (WBC count, 19,000/mm³). HIV serology was negative. A chest roentgenogram showed cardiac enlargement and adult respiratory distress syndrome. Abdominal and renal echography disclosed mesenteric lymph nodes and hepatomegaly. Despite aggressive fluid treatment, the patient’s condition further deteriorated, and he had to be mechanically ventilated. A Swan-Ganz catheter was inserted to optimize administration of catecholamines and measurement of right-ventricle filling pressure. Empirical antibiotic treatment with piperacillin/tazobactam and amikacin was started.

Four blood culture specimens obtained during admission yielded a motile gram-negative rod after 2 days of incubation. Subcultures on Campyloselect medium (bioMérieux, Marcy l’Etoile, France) at 42°C under microaerophilic conditions yielded excellent growth of a little gray colony. The microorganism was oxidase-positive, catalase-positive, hippurate-positive, and urease-negative. The isolate was susceptible to nalidixic acid and resistant to cephalothin, amoxicillin, amoxicillin/clavulanate, aminoglycosides, erythromycin, and pefloxacin; it was identified as C. jejuni. Bronchoalveolar lavage fluid, a protected brush specimen, urine, and a rectal swab culture remained negative. The treatment was changed to pefloxacin and amikacin. In spite of all the therapeutic efforts, the patient died 7 days after admission of multiple organ failure, including acute renal failure (which was treated with continuous hemofiltration), adult respiratory distress syndrome with major pulmonary hypertension, septic liver, and myocardial failure.

A MEDLINE search of the English-language medical literature since 1984 (key words: Campylobacter jejuni and septic shock) did not reveal any cases of fatal septic shock due to C. jejuni.

Acute enteritis is the most common presentation of C. jejuni infection and is usually self-limiting. More-severe C. jejuni infections are not frequently encountered. Cases of bacteremia due to C. jejuni have been rarely reported; the frequency of these cases may be underestimated because of difficulties in detecting the growth of C. jejuni in nonautomated systems. In the previously reported cases of campylobacter bacteremia, many patients, including the cohort of 10 patients described by Spelman et al., did not have acute enteritis [3]. Most patients with septicemia recovered after receiving treatment with ampicillin, tetracyclines, or fluoroquinolones [4]. One case of toxic shock syndrome due to Campylobacter intestinalis was reported [5]; this patient recovered with erythromycin therapy. As in our case, severe diarrheic forms of C. jejuni infections have been described. Two fatal cases of C. jejuni infection have been reported by Smith and Blaser [6], but the exact causes of death were not clear.

Campylobacter bacteremia is thought to be serious in patients with humoral immunodeficiency, including those with AIDS [7], agammaglobulinemia, diabetes mellitus, cirrhosis, and complement system disease, as well as in those who are receiving corticosteroid therapy [2]. The severity of septicemia in our patient may have been enhanced by his immunocompromised condition, which was related to his prior splenectomy and to his cirrhosis and diabetes.

This case report stresses the ability of C. jejuni to cause death in spite of early hospitalization and tailored antimicrobial therapy. The mechanism of the pathogenicity of C. jejuni that concerns digestive manifestations is known to be the cholera-like enterotoxin and a cytotoxin [8, 9]. Septic shock could be related to lipopolysaccharides present in these bacteria but also could be related to the enterotoxin that has been proposed as causing toxic shock syndrome in one case [5].

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References
Chorioretinitis Following Liver Transplantation: Detection of Toxoplasma gondii in Aqueous Humor

In immunocompromised hosts, toxoplasmosis is most often a disseminated disease with lethal effects. We report a case of toxoplasmic chorioretinitis following liver transplantation. The diagnosis was made by detection of Toxoplasma gondii in aqueous humor by means of polymerase chain reaction (PCR) analysis.

A 43-year-old woman with fulminant toxic hepatitis underwent liver transplantation in June 1993. The patient's serum was negative for T. gondii before transplantation; in contrast, IgG antibodies were detected in the donor serum. During the first 50 days after the operation, she developed an infectious syndrome; results of all microbiological examinations remained negative. Specific screening tests, including blood cultures, urine cultures, and serologies, were negative for cytomegalovirus (CMV), herpesvirus, Epstein-Barr virus, and fungi. In spite of no apparent ocular disease, ophthalmoscopy of the fundus oculi showed a normal right eye but suggested CMV retinitis in the left eye. She was treated with ganciclovir (500 mg/d) for 15 days without improvement.

At this point, serology showed IgM antibodies to T. gondii. Retinal angiography revealed focal necrotizing retinitis without retinal hemorrhages. Anterior chamber aspiration was carried out, and the presence of T. gondii was confirmed by PCR analysis with B1 gene amplification [1]. Tissue cultures of aqueous humor specimens for detection of T. gondii were not performed for technical reasons, and culture of an aqueous humor specimen on Sabouraud medium was negative. Culture of a liver biopsy specimen was negative for T. gondii. Syphilitic serology was negative. Findings on a cerebral CT scan were unremarkable. Full cicatrization of the fundus oculi was observed 10 days after completion of 6 weeks of treatment with pyrimethamine (150 mg/d for 2 days and then 75 mg/d), sulfadiazine (3 g/d), and folic acid (50 mg/d). One year after transplantation, the patient had not had any relapses of toxoplasmosis.

In our patient's case, transmission of T. gondii from the liver transplant is likely to have occurred. She seroconverted 7 weeks after transplantation, which is the delay usually described for this type of contamination [2]. Furthermore, her medical history did not reveal any risk factor for toxoplasmosis. Toxoplasmosis transmission following liver transplantation is rare; only a few cases have been reported since the work of Ruskin and Remington in 1976 [3]. Speirs et al. [4] reported on 38 liver transplantations; five patients were mismatched (seronegative recipients with seropositive organ donors), and primary T. gondii infection developed in one of these mismatched patients. To our knowledge, only one case of toxoplasmic chorioretinitis following liver transplantation has been reported [5]; in this case, the diagnosis was made after enucleation of the retina for histologic examination.

In immunosuppressed hosts with toxoplasmic chorioretinitis, the retina is fully thickened, white, necrotic, and inflamed, and this condition is often associated with severe overlying vitritis. Anterior granulomatous uveitis is a common finding. Fluorescein angiography may help to distinguish toxoplasmosis from CMV lesions: retinal hemorrhages are uncommon in toxoplasmosis, and in toxoplasmic, the dye tends to accumulate along the edge of the lesion; however, in CMV retinitis, the dye accumulates in the center of the necrotic lesion.

The fact that PCR analysis with gene amplification can detect small amounts of DNA suggests that it may be a sensitive method for the diagnosis of toxoplasmosis [6]. Interest in the use of PCR analysis of aqueous humor specimens for diagnosing toxoplasmic ocular infections in immunocompromised hosts has still not been established. However, in our case the diagnosis was made by means of this procedure.

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