CASE REPORT

Campylobacter Fetus Meningitis and Bacteremia in a Well-Controlled HIV Patient

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ABSTRACT

We present a case of Campylobacter fetus meningitis and bacteremia in an HIV patient. He was initially admitted due to concern for meningitis. After brief observation, the patient was discharged—once infectious etiologies had been ruled out. Soon after discharge, he was readmitted due to late culture growth demonstrating Campylobacter fetus in his cerebrospinal fluid (CSF) and blood. Our patient declined initially despite being placed on an appropriate, susceptibility-proven antibiotic regimen. He was later treated successfully with carbapenem therapy. This outcome is in alignment with the few previous cases of treatment failure (despite an appropriate antibiotic regimen) and supports the argument that most patients respond best to a carbapenem regimen.

INTRODUCTION

Campylobacter fetus is a rare, opportunistic pathogen that may cause an infection, ranging from mild gastrointestinal illness to severe systemic disease. To our knowledge, only one previous case had concomitant Campylobacter fetus bacteremia and meningitis infection. In this case report, we outline the hospital course, complications, risk factors (to contract this infection), and the treatment for this rare infection. Here, we present a rare case of concomitant Campylobacter fetus bacteremia and meningitis in a patient with well-controlled HIV (human immunodeficiency virus).

CASE PRESENTATION

A 34-year-old male with HIV was seen in the emergency department with vomiting, headache and difficulty sleeping over the last three days. He denied other symptoms—including abdominal pain, diarrhea, melena, and hematochezia. Two weeks prior to presentation, the patient had four teeth extracted due to caries. And he admitted to recently adopting a puppy.

The examination was significant for neck stiffness with decreased range of motion and headache that is exacerbated by neck movements. He otherwise had no rashes. His
abdomen was soft, nondistended, and nontender with normoactive bowel sounds. There were moist mucus membranes with no oral lesions. Initial labs, the complete blood count (CBC) and comprehensive metabolic panel (CMP), were unremarkable. The HIV PCR (polymerase chain reaction) result was 32 copies/mL, and the CD4 count was 514 cells/μL. Both CT (computed tomography) and MRI (magnetic resonance imaging) scans of the brain showed no acute abnormalities.

After a lumbar puncture, he was started empirically on cefepime, vancomycin and acyclovir for presumed meningitis. His cerebrospinal fluid (CSF) studies showed a: white blood cell (WBC) count of 14/ccc, red blood cell (RBC) count of 38/ccc, protein of 77mg/dL, glucose of 60mg/dL, negative meningitis/encephalitis panel, negative cryptococcal antigen PCR, and nonreactive VDRL (venereal disease research laboratory) test. The patient’s symptoms resolved after a day of antibiotics. Blood and CSF cultures were negative at 48 hours, so antimicrobials were discontinued. He was subsequently discharged with a diagnosis of headache of unknown etiology.

Twenty-four hours after discharge, both the CSF and blood cultures grew curved, gram-negative rods. Preliminary MALDI (matrix-assisted laser desorption/ionization) mass spectrometry identified possible Campylobacter fetus, which was later confirmed. The sample was sent out for identification and susceptibility, shown in figure 1. A diagnosis of Campylobacter fetus meningitis and bacteremia was made. The patient was readmitted and placed on ceftriaxone and levofloxacin.

During this admission, he was asymptomatic other than a mild headache. Unlike his previous hospital stay, the patient was more alert and had no nuchal rigidity or neck pain. Seven days after initial presentation, his WBC increased from 7 ×10^3/ccm to 21 ×10^3/ccm, and he had a fever of 102°F. The patient experienced new symptoms of chills, rigors, and vomiting but had no diarrhea, abdominal pain, headache, neck pain or mental status changes. Due to these changes, antibiotic therapy was escalated to meropenem and vancomycin to cover for possible, hospital-acquired infections. He underwent a sepsis workup which was unremarkable. Two days after broadening antibiotics, the patient became afebrile and demonstrated clinical improvement. He was discharged home to complete three total weeks of meropenem and linezolid. The patient was unfortunately lost to follow up after discharge.

DISCUSSION

Campylobacter fetus is a gram-negative, microaerophilic, spiral-shaped bacterium that infects individuals predisposed due to immunosuppression, alcohol use, diabetes, or cancer. While major reservoirs for Campylobacter fetus are sheep and cattle, there are case reports of Campylobacter fetus infection associated with dog exposure.1,2 There are also reports of patients who contracted Campylobacter fetus after tooth extractions or having dental caries. Our patient may have had exposure to the bacteria through these routes or his recent puppy adoption.3,4

Unlike Campylobacter jejuni, treatment of Campylobacter fetus is not well-defined. There are reports of both treatment success and failure with third-generation cephalosporins and ampicillin and of high resistance to erythromycin, fluoroquinolones, and tetracyclines.5-7 Most previous cases have shown treatment success with carbapenem.7 Our patient’s initial clinical decline while on ceftriaxone and levofloxacin resulted in switching to meropenem. Vancomycin was added to cover for possible, hospital-acquired infection. His
delayed fever and leukocytosis while on ceftriaxone and levofloxacin (despite microbiology demonstrating susceptibility) is concerning for inconsistencies between in vitro and in vivo susceptibilities—which has been pointed out in another study.7

**CONCLUSION**

We present a rare case of Campylobacter fetus meningitis and bacteremia in a patient with well-controlled HIV who was successfully treated with carbapenem. We recommend that patients who have confirmed Campylobacter fetus infection receive treatment according to the susceptibility pattern and have an extended observation period to ensure clinical improvement—due to the possibility of resistance despite the susceptibility pattern.

**Notes**

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**REFERENCES**