Case Reports in Gastroenterology

Case Rep Gastroenterol 2011;5:623–627
DOI: 10.1159/000334423

Published online: October 29, 2011

ISSN 1662–0631
www.karger.com/crg

Crohn's Disease Exacerbation Induced by *Edwardsiella tarda* Gastroenteritis

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Key Words
Edwardsiella tarda · Bacterial gastroenteritis · Crohn's disease

Abstract
Exacerbations of Crohn's disease are not infrequently associated with bacterial gastroenteritis. The recognition of synchronous infections in such patients is vital for the initiation of appropriate antimicrobial therapy. Furthermore, the detection of active bacterial infections may lead the clinician to delay starting biological therapy. We report here a man presenting with an exacerbation of his Crohn's disease during a trip to Thailand. Stool cultures were positive for the unusual gut pathogen *Edwardsiella tarda*. The patient's symptoms resolved with concurrent antibiotic and steroid therapy. This finding demonstrates the value of performing stool culture in all patients presenting with exacerbations of inflammatory bowel diseases.

Introduction

Crohn's disease (CD) is a chronic remitting and relapsing inflammatory disease of the gastrointestinal tract. The etiology of CD is incompletely understood, but is thought to relate to an inappropriate activation of the mucosal immune system in genetically susceptible individuals. Various inciting triggers for the onset of CD have been postulated, including bacterial gastrointestinal pathogens [1]. The most commonly described pathogen associated with exacerbations of CD is *Clostridium difficile* [2]. However, other, more unusual, bacteria have been detected as synchronous infections in the context of CD flares. *Edwardsiella tarda*, a bacterium isolated from a wide variety of freshwater and marine fish, reptiles, birds, and mammals, can cause a spectrum of

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intestinal and extraintestinal diseases in humans. While *E. tarda* infections occur globally, they are more common in tropical and subtropical regions, which is thought to be due to the consumption of raw or improperly cooked fish [3]. We report the first case of CD exacerbation secondary to *E. tarda* gastroenteritis in a patient returning from Thailand.

Case Report

A 28-year-old Caucasian man presented with a 3-week history of abdominal pain and diarrhea that had begun during a trip to Thailand. His medical profile was significant for a 10-year history of CD which had necessitated maintenance azathioprine therapy (200 mg daily) and a right hemicolectomy the year prior to presentation. Subsequent to his surgery, the patient had a baseline of 5–8 bowel movements per day with ongoing hematochezia. He was therefore due to begin treatment with infliximab, which had been postponed pending his return from Thailand. The patient had been vaccinated against typhoid prior to his trip.

The patient had been in Thailand for 10 days when, within the span of 12 h, he began experiencing abdominal pain and 20–30 bloody and watery bowel movements per day. He did not suffer from nausea or vomiting, but did experience iritis, which had occurred during prior flares of his CD. The iritis manifested as redness, photophobia, and retro-orbital pain, and was treated by the patient with topical steroid drops. The patient's mother and grandfather also complained of symptoms of gastroenteritis during the trip, but did not experience iritis. Of note, while his diet during the trip consisted primarily of vegetables and chicken, the patient reported consuming catfish shortly before the onset of symptoms.

Upon return home, the patient presented to hospital with his gastrointestinal symptoms and a persistent temperature of 38.7°C. He did not have a leukocytosis, but ESR was 47 mm/h and CRP was 230 nmol/l. Stool investigations were negative for *C. difficile* toxin, but cultures were positive for *E. tarda*. Blood cultures were negative. Flexible sigmoidoscopy showed pancolitis to 70 cm with no mucosal sparing (**fig. 1**). Colonic biopsies revealed mucosal ulceration and inflammation indistinguishable from active inflammatory bowel disease (IBD) (**fig. 2**).

The patient was treated with ciprofloxacin, metronidazole, and methylprednisolone on admission with rapid improvement of his symptoms. Repeat stool cultures 3 days following treatment were negative.

Discussion

CD is a form of IBD manifested by chronic transmural inflammation of the gastrointestinal tract. It can affect any location of the gastrointestinal tract from the mouth to the perianal area. The clinical presentation of CD depends on the region(s) of the bowel affected by the disease. Intestinal symptoms can include abdominal pain, diarrhea, melena or hematochezia, malabsorption, strictures, fistulae, or abscesses. Extraintestinal manifestations of CD include arthritis, inflammatory changes of the eyes, rashes, hypercoagulable states, and neurological symptoms.

The etiology of IBD is not well understood; however, it has been postulated that infectious gastroenteritis may be a precipitating or exacerbating factor [1]. In a retrospective study from Denmark, patients with either *Campylobacter* or *Salmonella* gastroenteritis followed for 15 years were 2.9 times more likely to have IBD than unexposed controls [4]. Synchronous enteric infections occur in 10.5–20% of IBD relapses [5, 6]. *C. difficile* is the most common culprit, and it portends a worse clinical outcome [2]. However, more atypical enteric pathogens have also been described in the context of IBD. A 2006 case report described a 48-year-old man with known proctitis who had an exacerbation of symptoms 6 months after infectious gastroenteritis with *E. tarda*.

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Colonic biopsies demonstrated mixed inflammatory infiltrates with crypt abscesses consistent with ulcerative colitis [7].

E. tarda is a Gram-negative bacillus and a member of the *Enterobacteriaceae* family. Members of the genus *Edwardsiella* are predominantly found in freshwater and marine environments [3]. *E. tarda* has been isolated from mammals, birds, reptiles, fish, and amphibians. Asymptomatic carrier rates of *E. tarda* are very low, ranging from 0 to 0.8% in epidemiological studies of children and adults from Asia, Australia, and Central America [3]. Biochemically, it closely resembles the *Salmonella* species and occasionally is misidentified as such. *E. tarda* is susceptible to a wide variety of antibiotic classes, including β-lactams, cephalosporins, aminoglycosides, and fluoroquinolones [3, 8].

E. tarda gastroenteritis can manifest in a number of ways, usually as acute secretory enteritis with nausea, vomiting, abdominal cramps, and low-grade fever (38.0–38.5°C), but occasionally in a more severe form involving bloody diarrhea, colonic ulcerations, and pseudomembranes [3, 8, 9]. *E. tarda* gastroenteritis occurs worldwide, but particularly in tropical and subtropical regions, where the consumption of raw or improperly cooked fish may be common practice. Aquatic pets such as fish and turtles have also been implicated as a source of human infection. In addition, human to human transmission has been documented [3]. Although they are rare, extraintestinal infections with *E. tarda* have been described and have a high mortality rate [8]. These include bacteremia, meningitis, endocarditis, soft tissue infections, and intraabdominal abscesses [10].

As the incidence of IBD increases worldwide, it is becoming important to determine whether patients presenting with flares have a concurrent bacterial gastroenteritis requiring antibiotic therapy. The present report describes a 28-year-old patient with known CD who contracted *E. tarda* gastroenteritis during a trip to Thailand. His presentation was consistent with bacterial infection due to the rapidity of symptom onset as well as the isolation of a single pathogen from stool samples. We favor that this represented an infection-induced IBD flare as the patient experienced an extraintestinal manifestation of his CD, namely iritis. IBD flares have been documented with multiple organisms; however, we believe this is the first documented case of a CD flare with *E. tarda*. This underscores the importance of conducting a rigorous stool culture in all patients presenting with exacerbations of IBD.

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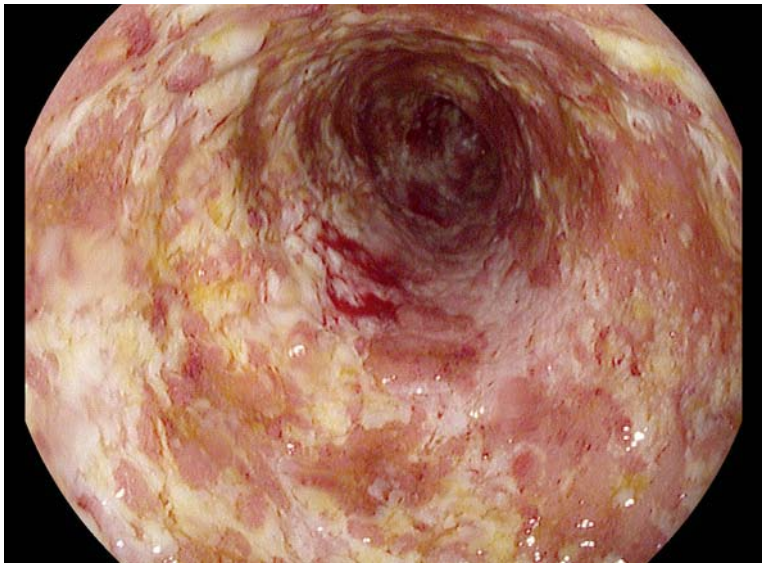

Fig. 1. Endoscopic photograph demonstrating continuous active colitis.

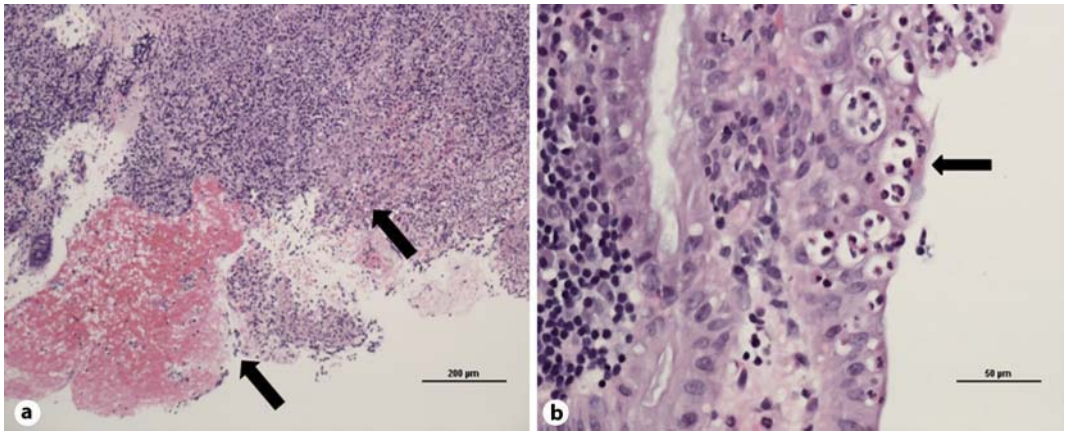

Fig. 2. Biopsy obtained at the time of endoscopy consistent with active IBD. Hematoxylin and eosin-stained colonic biopsies demonstrating mucosal ulceration with fibrin deposits and acute inflammation (arrows) (**a**) and epithelial microabscesses (arrow) (**b**).

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