Exacerbation of ulcerative colitis due to *Edwardsiella tarda* infection

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Received - 09 August 2017 Initial Review - 21 September 2017 Published Online - 19 October 2017

ABSTRACT

Infectious gastroenteritis can lead to exacerbations of inflammatory bowel disease (IBD) such as ulcerative colitis, which warrants appropriate and timely control with the initiation of specific antibacterial therapy. Herein, we discuss a patient who had presented with an exacerbation of ulcerative colitis due to an infectious gastroenteritis caused by *Edwardsiella tarda* and had responded to the appropriate antibacterial therapy. This study outlines the importance of a culture of stool samples in all cases of IBD with acute exacerbations.

Key words: *Edwardsiella tarda, Gastroenteritis, Inflammatory bowel disease*

Ulcerative colitis is an inflammatory bowel disease characterized by acute and chronic inflammatory changes, commonly affecting the colon and proximal rectum. Exacerbations are common in patients with ulcerative colitis and enteric pathogens are known to induce such exacerbations (1-4). Members of the genus Edwardsiella are usually associated with fresh water life forms and human infections with them are relatively uncommon. Herein, we discuss a patient who had presented with an exacerbation of ulcerative colitis due to an Edwardsiella tarda

CASE REPORT

A 30-year-old female presented with lower abdominal pain and bloody diarrhea for more than 3 months. She was admitted under the medical gastroenterology department and was evaluated further. On clinical examination, the patient had mild dehydration. Systemic examination was clinically normal except for mild lower abdominal tenderness. Her hematological parameters were found to be normal except for mild anemia (9.7 GMs%). Her blood glucose and renal parameters were within the normal limits. Tests for human immunodeficiency virus 1 and 2, hepatitis B virus, and hepatitis C virus markers were found to be negative. No abnormalities were detected by the ultrasound of the abdomen and pelvis. A stool sample was sent for the microbiology department. Microscopy of the stool sample showed plenty of pus cells and red blood cells (RBCs). No parasitic forms were observed. The culture of the stool sample yielded no growth. A colonoscopy was performed, and the histopathology of colonic biopsy revealed active colitis, with crypt micro abscesses suggestive of ulcerative colitis. The patient was treated with mesalamine enema, oral sulfasalazine, and oral hematinics. She was discharged after symptomatic improvement. After 1 month, she had a similar episode of lower abdominal pain and bloody diarrhea for more than 1-week duration. She was readmitted, and her stool sample was sent for culture.

Microscopy of the stool sample showed plenty of pus cells and RBCs. Stool culture showed non-lactose fermenting colonies and red colonies with black centers on MacConkey agar and xylose lysine deoxycholate agar, respectively. Gram stain from culture showed short, uniformly stained Gram-negative bacilli with rounded ends. The bacilli were found to be motile by hanging drop and mannitol motility medium. The organism was found to be catalase positive, oxidase negative, and nitrate-reducing fermentative bacteria. Methyl red was positive, and Voges–Proskauer was negative. Indole was formed. Citrate and urease were negative. Kligler iron agar showed alkaline slant and acidic butt with gas and hydrogen sulfide. Glucose was fermented with gas production while sucrose, lactose, and mannitol were not fermented. There was no deamination of phenyl pyruvic acid, and beta-galactosidase was not produced. Lysine and ornithine were decarboxylated. Arginine was not dihydrolyzed. This isolate was, hence, identified as *Edwardsiella tarda* [1].

The isolate was susceptible to all antibiotics tested, namely, amikacin (30 mcg), gentamicin (10 mcg), ceftazidime (30 mcg), ceftriaxone (30 mcg), ciprofloxacin (5 mcg), meropenem (10 mcg), and cotrimoxazole (1.25/23.75 mg), as per the Clinical Laboratories Standards Institute (CLSI) using the Kirby-Bauer disc diffusion method [2]. The patient was started on oral ciprofloxacin 500 mg, 12 hourly for 5 days, and was discharged after symptomatic improvement.

DISCUSSION

Ulcerative colitis is an inflammatory bowel disease (IBD) affecting primarily the colon and proximal part of the rectum, characterized by ulcerations, hemorrhage, and edematous changes limited to the mucosal surface of the entire length of the colon. Histopathology of ulcerative colitis is characterized by the presence of acute and chronic inflammatory changes with both polymorph nuclear leukocytes and mononuclear cells, crypt microabscesses, distortion of the mucosal glands, and loss of goblet cells [3]. Reports suggest that many enteric pathogens can induce exacerbations of ulcerative colitis [4-8]. Members of the genus *Edwardsiella* inhabit freshwater life forms and cold-blooded animals such as fishes, amphibians, and reptiles. Human infections with *E. tarda* is uncommon and has been rarely isolated from wound infections, blood, and liver abscess [9-11]. Although reported as a cause of gastroenteritis, its recognition as an established agent of gastrointestinal infections has been slow [5,12]. The literature search reveals few previous reports of *E. tarda* enteritis being involved in exacerbations of Crohn's disease [5]. Enteritis due to *Edwardsiella* results in manifestations ranging from secretory diarrhea to even bloody diarrhea. Dietary habits, notably eating of raw or undercooked fish, have been incriminated in most cases as reported from the tropical and subtropical regions of the world [13]. In the index case, the patient had bloody diarrhea, and the stool culture showed a predominant growth of *E. tarda*. We differentiated between various species of *Edwardsiella* such as *E. tarda*, *Edwardsiella hoshinae*, and *Edwardsiella ictaluri* based on the conventional biochemical tests as mentioned [14]. *Edwardsiella piscicida* is a fish pathogen, and its differentiation from other *Edwardsiella* species is possible only by DNA-DNA hybridization method [14], which was out of the scope of the present study. Simultaneous testing of the stool sample for *Campylobacter, Yersinia enterocolitica*, diarrheagenic *Escherichia coli,* and parasites were found to be negative. None of the viruses such as *Cytomegalovirus* known to be associated with relapse of IBD was looked for. Hence, in the absence of evidence of any other infectious etiology, the isolation of this agent as a single predominant pathogen from the stool sample and the response of the patient to specific antibacterial therapy, *E. tarda,* was considered a pathogen here.

It is now well accepted that microorganisms may cause an initial mucosal insult leading to a cascade of immunological events culminating in the onset of IBD and are also responsible for reactivating the disease during its dormancy. Failure to recognize such infections in such settings can have disastrous outcome considering that immunosuppressive agents such as steroids are used for maintenance therapy for IBDs such as ulcerative colitis. We were fortunate to recognize this agent due to the predominance of this pathogen in culture over other expected normal flora.

CONCLUSION

This case report supports the role of *E. tarda* enteritis in causing exacerbations of IBD, thus highlighting the importance of stool culture in all such cases.

REFERENCES

1. Farmer JJ, Farmer MK, Holmes B. The *Enterobacteriaceae*: General characteristics. Topley Wilsons Microbiol Microb Infect; 2010 Available from: http://www.onlinelibrary.wiley.com/doi/10.1002/9780470688618.taw0048/full. [Last cited on 2017 Jul 23].
2. Clinical and Laboratory Standards Institute. Performance Standards for Antimicrobial Susceptibility Testing. 26th ed. Wayne, PA: CLSI; 2016.
3. Hendrickson BA, Gokhale R, Cho JH. Clinical aspects and pathophysiology of inflammatory bowel disease. Clin Microbiol Rev. 2002;15(1):79-94.
4. Mylonaki M, Langmead L, Pantes A, Johnson F, Rampton DS. Enteric infection in relapse of inflammatory bowel disease: Importance of microbiological examination of stool. Eur J Gastroenterol Hepatol. 2004;16(8):775-8.
5. Arya AV, Rostom A, Dong WF, Flynn AN. Crohn's disease exacerbation induced by *Edwardsiella tarda* gastroenteritis. Case Rep Gastroenterol. 2011;5(3):623-7.
6. Engel JJ, Martin TL. *Edwardsiella tarda* as a cause of post dysenteric ulcerative colitis. Int J Colorectal Dis. 2006;21(2):184-5.
7. García Rodríguez LA, Ruigómez A, Panés J. Acute gastroenteritis is followed by an increased risk of inflammatory bowel disease. Gastroenterology. 2006;130(6):1588-94.
8. Meyer AM, Ramzan NN, Loftus EV Jr, Heigh RI, Leighton JA. The diagnostic yield of stool pathogen studies during relapses of inflammatory bowel disease. J Clin Gastroenterol. 2004;38(9):772-5.
9. Frumberg DB, Epstein SE, Jauregui JJ, Pivec R, Maheshwari AV. Prosthetic hip infection with *Edwardsiella tarda* in sickle cell beta thalassemia disease: A case report. J Clin Orthop Trauma. 2016;7 Suppl 1:130-3.
10. Ohara Y, Kikuchi O, Goto T, Yoshida T, Mori H, Matsueda K, et al. Successful treatment of a patient with sepsis and liver abscess caused by *Edwardsiella tarda*. Intern Med. 2012;51(19):2813-7.
11. Nishida K, Kato T, Yuzaki I, Suganuma T. *Edwardsiella tarda* bacteremia with metastatic gastric cancer. IDCases. 2016;5:76-7.
12. Spencer JD, Hastings MC, Rye AK, English BK, Ault BH. Gastroenteritis caused by *Edwardsiella tarda* in a pediatric renal transplant recipient. Pediatr Transplant. 2008;12(2):238-41.
13. Janda JM, Abbott SL. Infections associated with the genus *Edwardsiella*: The role of *Edwardsiella tarda* in human disease. Clin Infect Dis. 1993;17(4):742-8.
14. Reger PJ, Mockler DF, Miller MA. Comparison of antimicrobial susceptibility, beta-lactamase production, plasmid analysis and serum bactericidal activity in *Edwardsiella tarda*, *E. ictaluri* and *E. hoshinae*. J Med Microbiol. 1993;39(4):273-81.

Funding: None; Conflict of Interest: None Stated.

How to cite this article: Shankar U, Chitra G, Mandal J, Mohan P, Hamide A. Exacerbation of ulcerative colitis due to *Edwardsiella tarda* infection. East J Med Sci. 2017;2(3):47-48.