Terminal Ileitis Caused by Yersinia enterocolitica and Presents with Intussusception Mimicking Crohn’s Disease

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The pathological importance of the infection by Yersinia enterocolitica has been increasing. Even though Yersinia has been implicated in Crohn’s disease (CD), its role in CD is controversial. We present a case of a child with terminal ileitis which is due to Yersinia enterocolitica infection and mimics CD. This case demonstrates that clinical manifestations, sonographic, radiologic, endoscopic, and histologic findings of Yersinia enterocolitica enteritis presenting with intussusception. We compared the endoscopic findings and fecal calprotectin between the acute infection phase and the recovery phase after conservative treatment. We also compared previous cases of children with Yersinia enterocolitica infection mimicking CD. Yersinia enterocolitica infection should be included in the differential diagnosis of patients suspicious of CD.

Keywords: Child; Crohn disease; Intussusception; Yersinia enterocolitica

INTRODUCTION

Yersinia enterocolitica may present with gastrointestinal manifestations such as diarrhea, abdominal pain. Gastrointestinal symptoms may mimic inflammatory bowel disease (IBD), especially Crohn’s disease (CD). The pathogenesis of CD is multifactorial [1] and the association between IBD and gastrointestinal chronic inflammatory process have been suggested, but not confirmed [2-4]. Hereby, we described a case of a child with terminal ileitis due to Yersinia enterocolitica infection, presenting with intussusception mimicking CD.

CASE REPORT

A 6-year-old boy with chronic abdominal pain visited the emergency department with acute right-sided abdominal pain that suddenly worsened, and was referred to the pediatric department. He had no significant family or medical history. The abdominal pain was developed suddenly, localized at the right upper quadrant with a numeric rating scale of 8, and recurred at frequent intervals. He had diarrhea with Bristol stool scale of 5 to 6.

His body temperature was 36.9°C, heart rate was 100 beats/min, blood pressure was 120/80 mm Hg, and respiratory rate was 23 breaths/min. There was diffuse abdominal tenderness on the right upper quadrant but no palpable mass was palpated. Laboratory tests showed elevated white blood cell counts of 16,280 cells/μL, neutrophil counts of 13,860 cells/μL, erythrocyte sedimentation rate of 38 mm/hr, and C-reactive protein level of 2.26 mg/dL. He had hemoglobin level of 10.4 g/dL, hematocrit of 31.6%, ferritin of 14 ng/mL, iron of 17 μg/dL, total iron binding capacity of 426 μg/dL, which suggest iron deficiency anemia. Albumin, blood urea nitrogen, and creatinine levels were normal and interferon-γ release assay were negative. Stool calprotectin level was elevated to 3,000 mg/kg.

Ultrasound revealed target like lesion at the right lower quadrant. We diagnosed the patient with ileocolic intussusception. Air-reduction was performed on the day of admission, relieving colicky abdominal pain after the procedure. Diffuse abdominal pain, diarrhea, and low-grade fever were remained. Since intussusception is not common in 6-year-old child, we decided to perform abdomen computed tomography (CT). Abdomen CT showed thickening through terminal ileum, cecum, proximal ascending colon, and the polypoid lesion was suspected in the ter-

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minal ileum (Fig. 1).

In the suspicion of IBD or polyp of the terminal ileum, we decided to perform endoscopy. The examination revealed the presence of a few small-sized ulcers in descending colon and cecum, and multiple aphthous ulcers in the terminal ileum in colonoscopy (Fig. 2). Pathology finding was chronic inflammation with cryptal distortion, which was consistent with a diagnosis of CD. These findings were consistent with the diagnosis of CD, A1-L3-B1 based on the Montreal classification. Exclusive enteral nutrition (EEN) and peroral mesalazine were commenced. On the 9th day after admission, stool bacteria multiplex real-time polymerase chain reaction (multiplex RT-PCR) revealed *Yersinia enterocolitica* infection. EEN and oral mesalazine were discontinued and supportive management was provided. His symptoms and signs improved gradually over 7 days. The patient was discharged on the 11th day after admission. Since CD was not yet completely ruled out, follow-up colonoscopy was scheduled. At the 2 months of follow-up, colonoscopy did not reveal any significant findings such as inflammation or ulcers. His calprotectin was normalized to 33 mg/kg. The patient did not suffer any signs and symptoms such as abdominal pain or diarrhea during 2-year follow-up. This case report was approved the institutional review board of Soonchunhyang University Bucheon Hostpiatl (IRB approval no., 2019-08-005). The patient provided written informed consent for the publication of clinical details and images.

**DISCUSSION**

This case report demonstrates a child of *Yersinia enterocolitica* ileitis accompanied by intussusception, which was similar to CD. Delayed diagnosis of *Yersinia* enterocolitis was due to slow output of stool examination and his presentations. The patient had chronic abdominal pain before the acutely aggravated severe abdominal pain which was consistent with CD, but turned out to be functional abdominal chronic pain without red flag signs. Furthermore, colonoscopy and biopsy also supported with CD with chronic inflammation and crypt distortion. There are few reports compare the endoscopic findings and fecal calprotectin between the acute phase of infection and the recovery phase after conservative treatment.

The etiopathogenesis CD is multifactorial with various environmental, genetic, immune-mediated, and gut microbial factors. Although the association between IBD and gastrointestinal infection such as *Yersinia* have been suggested, the mechanisms involved in the pathogenesis of CD are still not clear. Baumgart and Carding [1] suggested that IBD is more common after gastrointestinal infections, and people with disease generally have higher concentrations of mucosal bacteria than healthy people do. Also, Lamps et al. [2] reported pathogenic *Yersinia* DNA in a series of CD patient

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**Fig. 1.** Computed tomography of the abdomen of a 6-year-old boy with acute abdomen pain. Bowel wall thickening through terminal ileum, cecum, and proximal ascending colon. Suspected polypoid lesion in terminal ileum (arrow).

**Fig. 2.** Colonoscopic findings showed multiple aphthous ulcers at terminal ileum, ascending colon and descending colon.
Yersinia enterocolitis is a gram-negative bacillus with a worldwide distribution, causing a variety of clinical manifestations ranging from self-limited enteritis to life-threatening systemic infection. Yersinia enterocolitica enteritis can mimic appendicitis, intussusception, toxic megacolon, gastrointestinal perforation, and peritonitis that would require pediatric surgical treatment. In our case of terminal ileitis mimicking CD subsequently diagnosed with Yersinia enterocolitis, Le Baut et al. [4] revealed Yersinia could be found in inflammatory and non-inflammatory parts of the ileum in CD patients. This suggests that Yersinia enterocolitica infection should be included in the differential diagnosis of patient suspicious of CD. Although Yersinia enterocolitica infections may mimic CD, other infectious causes of terminal ileitis should be ruled out after careful examination. Differential diagnosis of terminal ileitis is sometimes extremely challenging, and not every inflammation of terminal ileum is CD. There are few case reports on patients, who are not CD, being misdiagnosed as CD [6-9]. Terminal ileitis may be caused by a wide variety of diseases. We compared previous cases report of terminal ileitis caused by Yersinia species (Table 1).

Our case suggests that Yersinia enterocolitica infection should be included in the differential diagnosis of patient suspicious of CD. Although Yersinia enterocolitica infections may mimic CD, other infectious causes of terminal ileitis should be ruled out after careful examination. Differential diagnosis of terminal ileitis is sometimes extremely challenging, and not every inflammation of terminal ileum is CD. There are few case reports on patients, who are not CD, being misdiagnosed as CD [6-9]. Terminal ileitis may be caused by a wide variety of diseases. We compared previous cases report of terminal ileitis caused by Yersinia species (Table 1).

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Year</th>
<th>Authors</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Yersinia species</th>
<th>Intussusception</th>
<th>Symptoms</th>
<th>Abdomen CT</th>
<th>Colonoscopy</th>
<th>CD classification</th>
<th>Histologic finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>1999</td>
<td>Tuohy et al. [7]</td>
<td>3</td>
<td>F</td>
<td>Y. enterocolitica</td>
<td>Not accompanied</td>
<td>Fever, pain, vomiting, diarrhea</td>
<td>Mesenteric lymphadenopathy, thickened bowel wall</td>
<td>Oval aphthae from rectum to cecum</td>
<td>A1-L3-B1 (Montreal)</td>
<td>Severe active colitis with cryptitis, crypt abscesses</td>
</tr>
<tr>
<td>3</td>
<td>2012</td>
<td>Ijichi et al. [8]</td>
<td>5</td>
<td>F</td>
<td>Y. pseudotuberculosis</td>
<td>Not accompanied</td>
<td>Fever, pain, diarrhea</td>
<td>Ascites, mesenteric lymphadenitis</td>
<td>Multiple mucosal aphthae from the rectum to ileocecal valve, without ulceration.</td>
<td>A1-L3-B1 (Montreal)</td>
<td>Focal infiltration of active neutrophils without cryptitis or crypt abscesses</td>
</tr>
<tr>
<td>5</td>
<td>2017</td>
<td>Current case</td>
<td>6</td>
<td>M</td>
<td>Y. enterocolitica</td>
<td>Accompanied</td>
<td>Pain, diarrhea</td>
<td>Bowel wall thickening at terminal ileum, cecum, proximal ascending colon. Polypoid lesion was suspected in terminal ileum</td>
<td>Multiple aphthous ulcers at terminal ileum, ascending colon and descending colon</td>
<td>A1-L3-B1 (Montreal)</td>
<td>Cryptal distortion</td>
</tr>
</tbody>
</table>

CT, computed tomography; CD, Crohn’s disease; M, male; F, female.