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<td>Wada, Fumiko; Murase, Kunihiko; Yoshio, Yoko; Onita, Ken; Ikeda, Koki; Furusu, Hisashi; Yamao, Takushi; Isomoto, Hajime; Takeshima, Fuminao; Omagari, Katsuhisa; Mizuta, Yohei; Nakagoe, Toru; Murata, Ikuo; Tomonaga, Michio; Kohno, Shigeru</td>
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NAOSITE: Nagasaki University’s Academic Output SITE
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A 29-yr-old Japanese man presented with left lumbar pain. Laboratory tests were suggestive of an inflammatory disease but serological, bacteriological, and markers of auto immunity were all negative. Gastroduodenal endoscopy showed slight mucosal congestion of the gastric antrum but the duodenum showed normal villi. Colonoscopy showed no abnormalities, and small bowel enema and jejunoscopy were normal. Abdominal ultrasonography and CT scans showed wall thickening of the small intestine. At laparoscopically-assisted wedge biopsy of jejunum, focal edema was noted mainly affecting the midjejunum together with enlarged mesenteric nodes. Histopathological examination of the surgical biopsy material showed focal neutrophilic infiltration in the mucosa and submucosa without granuloma. The lymph node showed nonspecific changes with no granulomas. Although the etiology could not be identified, the patient responded well to clarithromycin treatment.

Key words: nonspecific, chronic, jejunitis, laparoscopy

Introduction

Jejunitis or jejunal ulceration is rare in the patients with abdominal pain. Diagnostic procedure in such patient may include small bowel endoscopy and radiological examination, although it is often difficult to establish a correct diagnosis based on these examination, and a firm diagnosis is usually made at exploratory laparostomy. Recent studies have emphasized the use of minimal invasive laparoscopy-assisted colectomy and small bowel resection. We report a case of chronic nonspecific jejunitis, which was diagnosed using this procedure.

CASE REPORT

A 29-year-old male was admitted to branched hospital in July 1998 for investigation of three years history of recurrent lumbar dull pain. Laboratory data demonstrated mild inflammatory change (total leucocyte count; 11840/ml, CRP; 1.20mg/ml). Due to persistent of abdominal pain, patient was referred to our hospital for further evaluation. Medical history included treatment of predonisolone and mesalamine for ulcerative colitis diagnosed three years earlier. There was no history of ingestion of NSAIDs or enteric-coated potassium tablets. On physical examination, the abdomen was flat with tenderness in the left periumbilical region. There was no skin lesions or palpable lymphadenopathy. The laboratory tests showed red blood cell count of 419 X 10^{12}/ml, hemoglobin; 12.5 g/dl, white blood cell count; 14000/ml (with 3 % eosinophils, 68 % polymorphonuclears, 21 % lymphocytes, 8 % monocytes), CRP; 2.22 mg/ml, total protein; 6.5 g/dl. Serum gastrin level was normal, and antinuclear antibody test was negative. Stool examination was negative for enteric pathogens, ova, and parasites were negative. Serological test for Yersinia and amoeba were negative.

Gastroduodenal endoscopy showed slight congestion of the gastric antrum and a normal duodenum. Multiple biopsies from duodenum showed non-specific mucosa with normal villous architecture (Fig 1). Colonoscopy showed no abnormalities, and lack of mucosal edema, congestion, easy bleeding, or mucosal ulceration suggesting active ulcerative colitis. Biopsy specimens from the rectum were normal. Abdominal ultrasonography and CT scans (Fig 2) showed thickened loops of the small intestine and enlarged (1 cm in diameter) mesenteric
Fig. 1. Photomicrograph of duodenal biopsies showing non-specific change without villous atrophy. Magnification x 100

Fig. 2. CT scans showing small intestinal wall thickening.

Fig. 3. Small bowel enema showing normal.

Fig. 4. (A): Histopathological examination of the jejunal specimen showing submucosal edema with inflammatory cell infiltrate. Magnification x 40
(B): Note the marked neutrophilic infiltration in the mucosa and submucosa without granuloma. Magnification x 200
lymph nodes. Small bowel enema was normal (Fig 3). Jejunoscopy was also normal. At laparoscopically-assisted wedge biopsy of the jejunum, focal edema was noted affecting mainly midjejunum, together with enlargement of mesenteric nodes. Histopathological examination of the jejunal surgical biopsy showed focal neutrophilic infiltration in the mucosa and submucosa without granuloma. There were no evidence of infection (bacterial, parasitic), neoplasia, ischemic change, or vasculitis (Fig 4). Examination of the lymph node biopsy showed nonspecific changes of reactive hyperplasia without granulomas.

The patient was treated with a course of clarithromycin (400 mg/day for six months). At last follow-up examination, 25 months after discharge from the hospital, the patient was free of abdominal pain, and CRP was negative.

Discussion

The small bowel study of our case showed a unique pattern did not fit into any textbook-described disease category. The differential diagnosis of jejunitis includes an extensive list such as Crohn's disease, celiac sprue (nongranulomatous ulcerative jejunitis), Whipple's disease, eosinophilic jejunitis, Schonlein-Henoch purpura, lymphoma, Zollinger-Ellison syndrome, ischemia, vasculitis, infectious enteritis (nonspecific jejunitis), parasitic infection, drug induced jejunitis (NSAIDs, and enteric-coated potassium tablets), and idiopathic jejunitis.

At first, inflammatory bowel disease was strongly considered in our case based on the medical history. However, we ruled out ulcerative colitis by colonoscopy and rectal biopsy specimens. Crohn's disease is a systemic inflammatory disease that may involve all regions of the gut, although the involvement of the upper gastrointestinal tract seems to be low.(13-15,25,27) Jejunal involvement occurs in 4-10 % of patients with ileitis, ileocolitis or colitis.(13,14) In adults with Crohn's disease, isolated jejunal involvement represents approximately 1 % of cases.(13) In our case, we could not detect granuloma in the jejunal biopsy specimens, but we could not completely rule out mucosal Crohn's disease. Epithelial granuloma is considered as the histological hallmark of gastric Crohn's disease, but such granuloma is only found in 2-5% of patients with Crohn's disease affecting the stomach.(26,27) Recent studies have described focal active gastritis, which constitutes a form of inflammation that seems to be typical of gastric Crohn's disease.(28,29) We have presented that focal active gastritis, diagnosed by immunostaining for mast cells and macrophages, is the histological hallmark of gastric Crohn's disease, and reported the presence of focal active gastritis in 28% of patients with Crohn's disease.(30) These findings suggest the gastric involvement in Crohn's disease is not rare as previously thought.(26,30)

Celiac sprue is characterized specific clinical features including abdominal pain, fever, steatorrhea, and protein losing enteropathy.(41-43) Tissue specimens of the small intestine show diffuse ulceration, intense mononuclear infiltration, and villous atrophy with increased intraepithelial lymphocytes.(32) This disease was ruled out in our patient due to the lack of classical signs of steatorrhea and hypoproteinemia, as well as absence of specific pathological findings. Furthermore, this disease is very rare in Japan, and we have not experienced this disease in our department.

Based on the clinical symptoms, laboratory data, and histological examination using H & E and PAS stain sections, we also ruled out Whipple's disease, eosinophilic jejunitis, Schonlein-Henoch purpura, lymphoma, Zollinger-Ellison syndrome, ischemia, vasculitis, parasitic infection (giardiasis, cryptosporidiosis), and drug induced jejunitis. Infectious enteritis was not likely in our patients, because clinical course is long for infections, and stool cultures were negative. However, this diagnosis could not be eliminated with certainty. The case was also reviewed by Professor R.H. Riddell (Chief of Pathology, McMaster University, Ontario, Canada), including all biopsy materials, who suggested a possible diagnosis of chronic infectious enteritis.

Abdominal CT is an useful non-invasive diagnostic tool that allows detection of malignant lesions in the small intestine.(33) Previous studies reported the use of contrast-enhanced CT, which allows recognition of wall thickening in inflammatory jejunal diseases.(24,35) In our case, CT showed jejunal wall thickening, but a definite diagnosis could not be established by CT only.

Several groups have stressed that small bowel endoscopy is well tolerated and clinically useful procedure for directly examination of the jejunal mucosa.(36,37) Using endoscopy, Morris et al were able to identify jejunal or ileal ulceration in 47% patients with rheumatoid arthritis receiving NSAIDs who had chronic ocult gastrointestinal bleeding.(36) Furthermore, Perez-Cuadrado et al used oral video push endoscopy and identified four out of eight patients (50%) with lesions of Crohn's disease in the small intestine.(37) We have also used the push type small bowel endoscopy (model XSIF230Y2, Olympus) in patients with unexplained upper gastrointestinal bleeding to examine the mucosa of the region extending from the duodenum to jejunum near the ligament of Treitz, but we could not find the mucosal erosions or ulcers in our series (unpublished data).

Clarithromycin, a broad-spectrum macrolide antibiotic
with good penetration into macrophages, could be effective in eradicating certain microorganisms. Other studies have also reported the effectiveness clarithromycin for Crohn’s disease.\(^{[19,20]}\) We also used clarithromycin in our patient, which produced alleviation of abdominal pain and improvement of CRP, suggesting that this antibiotic may be effective in these diseases.

In conclusion, we have described a rare case of chronic jejunitis. No etiological factor could not be identified but the patient responded macrolide treatment. CT is a useful non-invasive diagnostic tool for chronic jejunitis, and laparoscopy-assisted wedge biopsy should be used for the diagnosis of jejunitis.

Acknowledgement

We gratefully acknowledge the pathological advice of Professor R. H. Riddell (Chief of Pathology, McMaster University, Ontario, Canada).

References