CT Findings of Surgically Verified Acute Invasive Small Bowel Anisakiasis Resulting in Small Bowel Obstruction

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Acute invasive small bowel anisakiasis is an extremely rare cause of small bowel obstruction. The authors report a case of surgically verified small bowel anisakiasis resulting in small bowel obstruction. A 54-year-old man presented with suddenly developed diffuse abdominal pain after ingestion of raw fish. The peripheral blood examination showed leukocytosis without eosinophilia. CT showed a long segment of thickened small bowel accompanied by a focal narrowed portion and combined with ascites. When these findings are noted in patients with a history of recent ingestion of raw or undercooked fish, the diagnosis of small bowel anisakiasis should be considered in order to avoid application of unnecessary surgical treatment, in spite of the severity of the abdominal pain and bowel obstruction.

Key Words: Parasites, anisakiasis, intestine, infection, computed tomography

INTRODUCTION

Anisakiasis is a parasitic disease caused by ingestion of Anisakis larvae present in raw or undercooked fish.1,3 Gastric anisakiasis can be easily confirmed by endoscopy and many reports on it have been published.1,3-9 However, anisakiasis that occurs in the small bowel is difficult to diagnose preoperatively because of its nonspecific symptomatic presentation and inappropriate diagnostic tool. Small bowel anisakiasis is a rare cause of bowel obstruction.10-34 We have managed a patient with surgically verified acute invasive anisakiasis in the small bowel resulting in small bowel obstruction. To our knowledge, this is the first report on the CT findings of this condition.

CASE REPORT

A 54-year-old man was admitted to our hospital for the evaluation of a suddenly developed diffuse abdominal pain. There was no significant medical history that could be associated with the pain. Two days prior to the admission, he ate sea eel as sashimi. On the physical examination, he had direct and rebound tenderness in the right lower quadrant of the abdomen. The peripheral blood examination showed leukocytosis without eosinophilia (WBC count 12,500/mm³ with 2% eosinophils). The plain abdomen film showed the air-fluid level, which suggested intestinal obstruction. The abdominal ultrasonography showed a small bowel dilatation and ascites. For a better evaluation of the cause of ascites and the bowel dilatation, abdominal CT was performed on the same day. On the postcontrast CT, a highly enhanced and thickened small bowel wall was seen as a long segment of more than 7 cm. Contrast enhancement was noted along the whole thickness of the involved segment of the small bowel wall without adjacent mesenteric fat infiltration. The proximal part of the enhanced
bowl showed a focal narrowed portion. The small bowel, proximal to this enhanced segment, was dilated (Fig. 1A). Ascites was seen in the right lower quadrant (Fig. 1B).

In spite of the conservative treatment, the abdominal pain was not relieved after one day therapy. And the air-fluid level was still noted on the plain abdomen film without interval change. In view of consideration of an inflammatory or malignant stricture of the small bowel, the exploratory laparotomy was performed. Focal thickening of the distal jejunum wall was seen without signs of a definite mass during the surgery. A segmental resection of the thickened bowel was done. On the gross examination, the small bowel wall was edematous and a bowel wall invasion by a 2.5 cm long whitish worm was identified (Fig. 2A, B). The histologic examination revealed a dense eosinophilic infiltration along the entire thickness of the bowel wall, especially at the submucosal layer. The worm was identified as Anisakis. The final diagnosis was acute invasive anisakiasis of the small bowel. The postoperative course was uneventful.

DISCUSSION

Occasionally, the larva of the nematode parasites of fish-eating birds or marine mammals

![Fig. 1](image1.png)

Fig. 1. (A) Postcontrast abdominal CT showing the highly enhanced small bowel wall with focal narrowed portion (arrow). Contrast enhancement was noted along the whole thickness of the involved segment of the small bowel wall without definite infiltration to the adjacent mesenteric fat. The small bowel proximal to the enhanced segment was dilated. (B) Postcontrast abdominal CT showing ascites in the right lower quadrant of the abdomen.

![Fig. 2](image2.png)

Fig. 2. (A) Gross specimen of the diseased bowel revealing the penetration of a whitish anisakiasis larva into the bowel wall (arrow). (B) Cut section of the diseased bowel segment demonstrating edematous change, especially in the submucosal layer (arrow).
can inhabit or invade the human gastrointestinal tract, producing a condition known as anisakiasis. The two species most commonly involved are Anisakis simplex and Pseudoterranova decipiens. The adult Anisakis inhabits the intestinal tracts of marine mammals as well as certain birds. The cephalic end of the adult nematodes lies buried within the gastrointestinal mucosa of the host. In the aggregate, they may be so numerous that they may form tumor-like masses. The first case of anisakiasis was described by Van Thiel in 1960. Since then, many reports have been published on anisakiasis, especially in countries such as Japan, Spain and Holland. However, it can occur in nearly all the maritime areas, where raw or undercooked ocean fish or squids are consumed.

Gastrointestinal anisakiasis is classified as a luminal and an invasive form, according to the presence of the bowel wall invasion by Anisakis larva. The luminal form does not cause clinical symptoms. Only the invasive form can cause clinical problems. The invasive form is subdivided into the gastric and intestinal type, according to the penetration site of the Anisakis larvae. Clinical presentations and radiographic findings are different according to the type. Gastric anisakiasis is easily diagnosed by endoscopy and many clinical and radiographic reports on gastric anisakiasis have been published. However, the confirmation of the diagnosis of the small bowel anisakiasis is difficult due to the absence of proper diagnostic tools that could be resorted to for this purpose preoperatively.

In gastric anisakiasis, acute abdominal pain, nausea, and vomiting develop within a few hours after ingestion of the infected fish. Peripheral eosinophilia is commonly found on laboratory examinations. On the upper GI series, mucosal edema is prominent and thread-like filling defect, suggesting the presence of the worm itself, can sometimes be seen. If there is a history of ingestion of raw or undercooked fish several hours ago, we can definitely diagnose anisakiasis with these radiologic findings. The worm can be seen directly with the endoscope; thus, the diagnosis can be confirmed. The frequency of direct visualization of the worm was reported to be higher in examination by endoscopy than the upper GI series. Moreover, treatment of anisakiasis can be provided during the endoscopy by removal of the worm. So, endoscopy is the primary method for the diagnosis and treatment of gastric anisakiasis.

Intestinal anisakiasis causes a diffuse severe abdominal pain and rarely intestinal obstruction can be noted, as seen in our case. Leukocytosis without eosinophilia is a common laboratory finding in this condition, which often leads to the incorrect diagnosis of acute appendicitis or regional enteritis. However, there is no reliable diagnostic method for this condition. So a preoperative diagnosis is extremely difficult and only indirect radiological, clinical and laboratory findings can be used to diagnose intestinal anisakiasis. Seropositivity to the Anisakis antibody can be helpful in making the diagnosis. But it takes some time to develop the seropositivity after the parasitic infestation takes place, and it fails to be helpful in acute conditions but can be useful only in chronic conditions. So, the radiological examination is important in the diagnosis of the acute form of intestinal anisakiasis. The small bowel barium study shows the presence of thickened folds and irregular luminal narrowing. These findings reflect pathologic changes such as marked edema, eosinophilic infiltration, and granuloma formation. In rare cases, the whole worm can be identified as a filling defect in the barium study. Shirahama et al. report that the ultrasonographic findings of a transient and segmental thickening of the small bowel wall and the presence of ascites suggest the possibility of intestinal anisakiasis in patients who complain of abdominal pain. The CT findings of our case were similar to the ultrasonographic findings. However, CT provides more information such as the enhancing pattern and more subjective findings than ultrasonography. Based on the CT findings, the differential diagnosis of the disease included adenocarcinoma of the small bowel and a benign disease such as the inflammatory bowel disease. Adenocarcinoma of the small bowel usually involves only a short segment and on CT presents as an eccentric focal mass or as a circumferential asymmetric and irregular thickening of the bowel wall. There is an abrupt transition and luminal narrowing. The contrast-enhanced soft tissue mass may have a homogeneous density. In
the benign intestinal disease, the hallmark of the CT appearance is the circumferential and symmetric thickening of the bowel wall. The process is usually segmental or diffuse. The contrast-enhanced segment involved shows different ring densities, referred to as a "double halo" or "target" sign. However, the CT findings of the present case showed a relatively long segmental symmetric wall thickening with luminal narrowing and diffuse contrast enhancement on the involved segment. These findings have several features of the benign and the malignant disease. So, when recent history of ingestion of raw fish is present, CT can be helpful in the diagnosis of small bowel anisakisiasis.

The preoperative diagnosis of intestinal anisakiasis is regarded as being crucially important, especially in cases with bowel obstruction, because most often these patients recover only in the result of application of the conservative management for 1-2 weeks after the onset of the symptoms. Bowel obstruction is not an alarming presentation of intestinal anisakiasis. It develops through the luminal narrowing caused by the bowel wall edema. After several days, the thickened bowel wall returns to its normal condition, and the symptoms of the bowel obstruction disappear. However, if a proper diagnosis of anisakiasis is not made, an operation may have to be carried out unnecessarily.

In conclusion, several CT findings such as a long segment of the thickened small bowel wall, a focal luminal narrowing with diffuse contrast enhancement on the involved segment leading to bowel obstruction and ascites in patients with history of a recent ingestion of raw or undercooked fish, could suggest the possibility of small bowel anisakiasis. Therefore early surgical intervention should be avoided in these patients.

REFERENCES