Esophageal Anisakiasis Accompanied by Reflux Esophagitis

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A case with esophageal anisakiasis accompanied by reflux esophagitis is described. A 38-year-old man visited our hospital with complaints of heartburn and disturbance of food passage about seven hours after eating raw cuttlefish. The first esophagogastroscopy revealed an anisakis larva invading the squamocolumnar junction. Near the anisakis larva, a whitish exudate was demonstrated in the distal esophagus just proximal to the squamocolumnar junction. An anisakis larva was easily extracted from the esophagus by forceps. Reflux esophagitis with whitish exudative mucosal lesions and an area of linear erythema more than 5mm long were noted endoscopically 8 weeks after treatment with lansoprazole and cisapride. After six months the third endoscopic examination clarified that there was neither exudate nor erythema in the distal esophagus. Judging from the clinical course that he complained of newly experienced heartburn about seven hours after eating raw cuttlefish, and that whitish exudative mucosal lesions and an area of linear erythema did not disappear at three months after extraction of the anisakis larva. It was concluded that an anisakis larva enters the stomach first and then returns to the esophagus by gastroesophageal reflux.

(Key words: heartburn, gastroesophageal reflux, anisakis larva, magnifying electronic endoscope)

Introduction

Anisakis larvae cause acute gastric mucosal lesions with acute symptoms such as epigastralgia, nausea and vomiting. Most patients diagnosed as gastrointestinal anisakiasis were reported to be gastric anisakiasis, and esophageal anisakiasis was a rare disease in comparison (1). We treated a patient with esophageal anisakiasis and reflux esophagitis. It was suggested that the reflux esophagitis resulted from anisakis larvae, or anisakiasis accidentally occurred in a patient with a history of reflux esophagitis. Here, we report a patient with reflux esophagitis, which was triggered by esophageal anisakiasis, and we consider the correlation between reflux esophagitis and esophageal anisakiasis.

Case Report

On November 22, 1993, a 38-year-old man visited our hospital with complaints of heartburn and disturbance of food passage about seven hours after eating raw cuttlefish. He did not complain of epigastralgia, and his past medical history was unremarkable. He was a man weighing 70 kg and 166 cm tall with moderate nutrition. On physical examination, his temperature was 36.9°C, his blood pressure was 128/80 mmHg, and mild tenderness without defense was found in the upper abdomen. Laboratory studies disclosed the following values; white blood cell 18,000/mm³ (stab leukocyte 18%, segmental leukocyte 54%, eosinophil 5%, lymphocyte 22%, monocyte 1%), red blood cell 560x10⁶/mm³, hemoglobin 17.1 g/dl, platelets 32.7x10⁹/mm³, total protein 8.7 g/dl, aspartate aminotransferase 28 IU//, alanine aminotransferase 48 IU//, γ-glutamyl transpeptidase 180 IU//, lactate dehydrogenase 557 IU//, Cr 1.2 mg/dl, blood urea nitrogen 18 mg/dl, alkaline phosphatase 125 IU//, T-cho 184 mg/dl, UA 6.3 mg/dl, amylase 36 IU//, glucose 110 mg/dl, serum pepsinogen (PG) I 68.0 ng/ml, PG II 9.9 ng/ml, PG I/PG II ratio 6.8, and anti-Helicobacter pylori immunoglobulin G antibody 16.6 AU/ml (BML kit, enzyme-linked immunosorbent assay (ELISA)).

The first esophagogastroscopy was performed on the same day using a transparent hood-fitted zooming electronic magnifying endoscope. A conventional endoscopy revealed an anisakis larva invading just into the squamocolumnar junction. Near the anisakis larvae, a whitish exudate was demonstrated in the distal esophagus just proximal to the squamocolumnar junction (Fig. 1A). Neither acute mucosal lesion nor anisakis larva was found in the stomach. Magnifying observation revealed the fine
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Figure 1. The first endoscopic examination on November 22, 1993. A) Conventional observation showing an anisakis larva invading into the squamocolumnar junction. Near the anisakis larva, whitish exudate was demonstrated in the distal esophagus just proximal to the squamocolumnar junction. B) Magnifying observation clearly showing the fine mucosal structure of the distal esophagus and the cardia with whitish exudate just surrounding the anisakis larva.

mucosal pattern of the distal esophagus and the cardia with the whitish exudate just surrounding the anisakis larva (Fig. 1B). After iodine potassium iodine solution was sprayed through the biopsy channel, the anisakis larva stopped moving (Fig. 2). The anisakis larva, could then be extracted from the esophagus using a forceps in an easy and rapid procedure. After that he was treated with 30 mg lansoprazole plus 20 mg cisapride for eight weeks.

The second follow-up endoscopy was done on February 10, 1994. In the conventional observation, reflux esophagitis with whitish exudative mucosal lesions and an area of linear erythema of more than 5 mm long were noted (Fig. 3A). A magnifying endoscopic picture demonstrated the regenerative epithelium of the distal esophagus surrounding whitish exudates (Fig. 3B). Because the whitish exudate did not disappear, the same therapy was continued yet once more for 8 weeks. After that the medical treatment was continued with 40 mg famotidine without a break.

The third endoscopy was performed on August 18, 1994. A conventional endoscopy revealed the esophageal mucosa which was discoloried and thickened. The squamocolumnar junction was transformed into peninsular-like forms of squamous tissue, and was necessary to determine whether the whitish exudate disappeared or not (Fig. 4A). A magnifying observation clarified that there was neither exudate nor erythema in the distal esophagus (Fig. 4B).

Figure 2. Endoscopic pictures with iodine potassium iodine solution spraying method on November 22, 1993. As soon as it was sprayed through the biopsy channel, the anisakis larva stopped moving.
Figure 3. The second follow-up endoscopy on February 10, 1994. A) A conventional observation showing reflux esophagitis with whitish exudative mucosal lesions and an area of linear erythema of more than 5 mm long. B) A magnifying endoscopic picture demonstrating the regenerative epithelium of the distal esophagus surrounding whitish exudates.

Figure 4. The third endoscopy on August 18, 1994. A) A conventional endoscopic picture showing the squamocolumnar junction transformed into peninsula-like forms of squamous tissue, and whether the whitish exudate had disappeared or not, could not be determined. B) A magnifying observation clarifying that there was neither exudate nor erythema in the distal esophagus.
Discussion

One hundred to two-hundred cases of anisakiasis have been reported every year in Japan (2). Human anisakiasis was first reported by Van Thiel et al (3) in 1960. Generally, anisakis larvae are endoscopically found to invade the esophagogastric wall. Anisakis larvae cause acute gastric mucosal lesions with complaints of epigastralgia. Because anisakis larvae tend to penetrate the stomach wall and move to other sites of the stomach, it is not always found by endoscopy (4).

In contrast to gastric anisakiasis, esophageal anisakiasis is a more rare disease. Adachi et al (5) reported that from 1978 to 1987 two cases of esophageal anisakiasis were published in the literature. In another literature search, we found a paper about that three cases of esophageal anisakiasis had been reported from 1988 to 1994. Iino et al (6) described in 1991 that the ratio of esophageal to gastric anisakiasis is 28 to 1,989 based on the results of a questionnaire about anisakiasis collected from medical institutions throughout the Kyushu districts of Japan from March 1962 to June 1990.

Although it is well-known that an anisakis larvae causes an acute gastric mucosal lesion in the stomach, it is unknown whether it causes the same lesion in the esophagus. In previous reports, there was no case with esophageal anisakiasis accompanied by reflux esophagitis. It was not clear why anisakis larvae invaded the esophagogastric junction and not the gastric mucosa. Judging from the clinical course that he complained of heartburn about seven hours after eating raw cuttlefish, we considered that the anisakis larva may have entered the stomach in the first place and then returned to the esophagus by gastroesophageal reflex. In addition, on the grounds that whitish exudative mucosal lesions and the area of linear erythema were still demonstrated at the 12 o'clock position three months after extraction of the anisakis larva and because Fig. 3 shows that the mucosal breaks at 2 and 4 o'clock positions were not caused by the anisakis larva, we suggested that he had reflux esophagitis before penetration of the larvae into the gastroesophageal junction. On the other hand, when gastroesophageal reflux is associated with symptoms but without evidence of tissue injury on endoscopy, the patient is not described as having reflux esophagitis but rather symptomatic reflux (7). Thus endoscopic views and symptoms do not correspond in some patients. Furthermore, it is not always true that patients with reflux esophagitis on endoscopy complain of heartburn. On the assumption that the anisakis larva swallowed with the raw cuttlefish invaded the esophageal wall before entering the stomach, the symptoms should be marked as soon as eating raw cuttlefish. From these viewpoints, although he had no experience of heartburn, it seemed reasonable to suppose that the anisakis larva entered the stomach first and then return to the esophagus by gastroesophageal reflux as in the case Adachi et al (5) reported.

References