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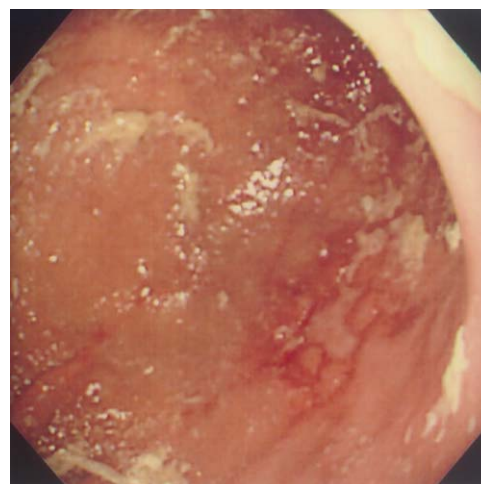


Figure 1. Endoscopic view of irregularly shaped ulcer, approximately 10 mm in diameter, surrounded by shallow, nodular, and marginal elevations on greater curvature of distal gastric body.

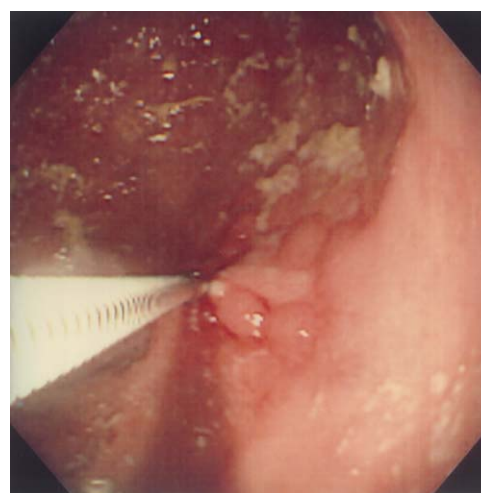


Figure 2. Endoscopic view of biopsy specimen being obtained from ulcer margin.

Anisakis in a biopsy specimen from the edge of a gastric ulcer: report of a case

Gastric anisakiasis is a common disease that is caused by infection by *Anisakis* larvae. Typically, it is from consumption of raw fish. Endoscopic findings are primarily gastric reddening, edema, and small erosions around the

worm penetration site; a transient giant edematous swelling, called a "vanishing tumor" of the stomach, occasionally is noted. As a rare chronic form, gastric anisakiasis also is known to manifest as a submucosal eosinophilic granuloma.¹⁻³ A case of gastric anisakiasis is presented in which the patient had an ulcerative lesion resembling early gastric cancer. However, a fragment of the body of an *Anisakis* larva was detected in a biopsy specimen from the margin of the ulcer, which led to the diagnosis.

Case report. A 51-year-old Japanese woman was hospitalized because of epigastric pain and anorexia of about 10 days' duration. She had been undergoing maintenance hemodialysis since 1991 for chronic renal

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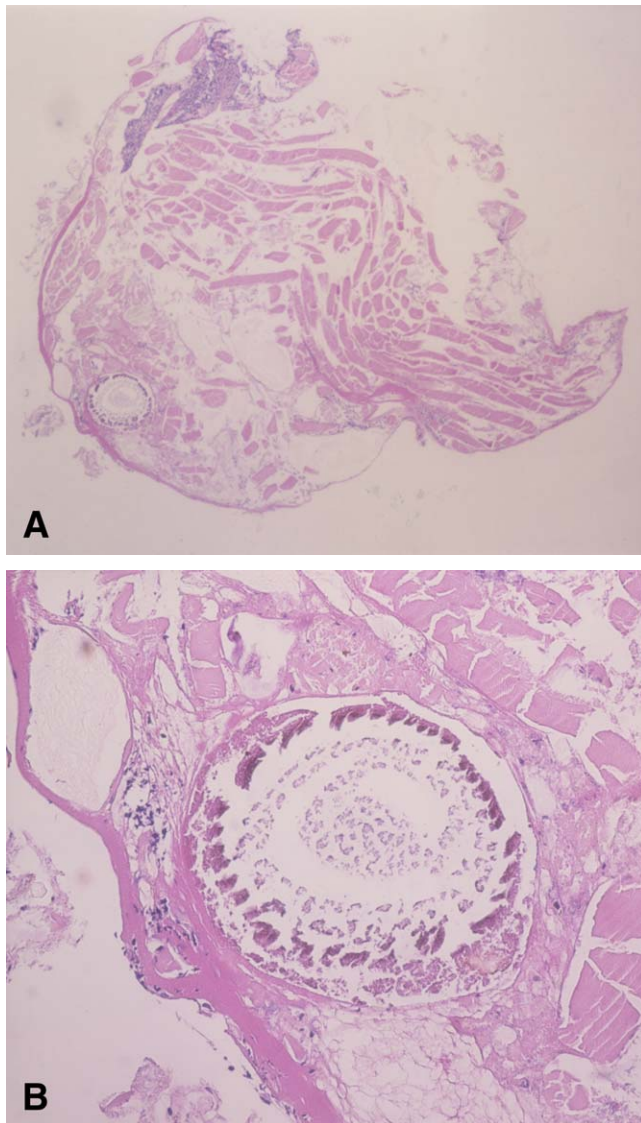


Figure 3. **A**, Photomicrograph of biopsy specimen from edge of ulcer, showing cross section of an *Anisakis* larva in muscle layer (H&E, orig. mag. $\times 36$). **B**, Photomicrograph of biopsy specimen, showing absence of eosinophil infiltration around *Anisakis* body (H&E, orig. mag. $\times 200$).

failure. She did not recall eating raw fish before the onset of symptoms. Eight days after admission, EGD disclosed an irregularly shaped ulcer, about 10 mm in diameter, surrounded by shallow, nodular, and marginal elevations on the greater curvature of the distal gastric body (Fig. 1). Forceps biopsy specimens from 4 sites on the margin (Fig. 2) all revealed hyperplastic changes, but one specimen also contained a cross section of a larva of *Anisakis* in the muscle layer. Eosinophil infiltration was not present around the *Anisakis* body (Fig. 3). The symptoms rapidly improved with treatment (famotidine). At EGD 1 month later, there was an S1 stage ulcer scar, and the marginal elevation was no longer present (Fig. 4).

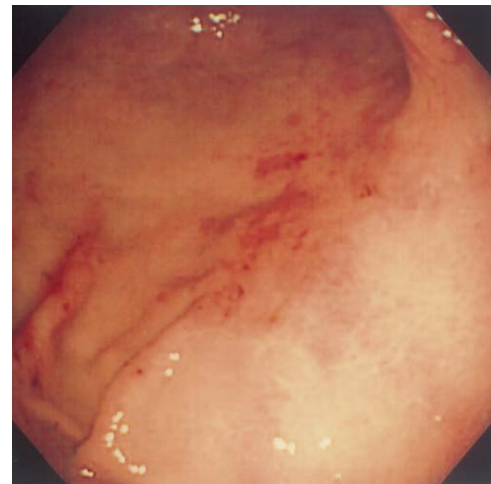


Figure 4. Endoscopic view 4 weeks later, showing ulcer scar on greater curvature of distal gastric body.

Discussion. Because gastric anisakiasis is acquired by eating raw fish, it is relative common in Japan. It now is reported from Western countries,⁴ presumably because of the popularity of sushi. The incidence of gastric anisakiasis in a population is directly related to the consumption of raw fish. Most patients have severe epigastric pain, which can begin within 1 hour of ingestion of the parasite.¹ When the infection is untreated, the pain continues for a few days, until the worms die within the mucosa. At endoscopy, the ringlike larvae may be seen in any area of the gastric mucosal surface. Other endoscopic findings include edema, erosion, and pseudotumor formation. The most effective treatment is endoscopic removal of all visible larvae. If this is successful, the severe pain disappears within a few hours.¹ In the present case, the ingestion, penetration, and death of an *Anisakis* larva probably occurred about 10 days before presentation, when the patient experienced the onset of epigastric pain.

The pathogenesis of gastric anisakiasis is considered to include an allergic reaction to the *Anisakis* antigen. When the larva of *Anisakis* dies after penetration of the mucosa, eosinophil infiltration and proliferation of connective tissue occur around the larval body, followed by formation of an eosinophilic granuloma, which has the appearance of a submucosal tumor. Subsequently, the granuloma usually decreases in size and gradually disappears.^{2,5,6} In the present case, eosinophil infiltration around the organism was absent, possibly because the inflammation associated with penetration of the larva had subsided with time and disappeared because of the collapse of the granuloma.

There are occasional reports of gastric anisakiasis with a morphology that resembles a Borrmann type II advanced gastric cancer or a type IIa + III early gastric cancer.⁷ There also are a few reports of the endoscopic discovery of a worm in the base of a gastric ulcer.^{2,8-10} *Anisakis* also has been discovered in granulomas removed by EMR.¹¹ However, the detection of the body of an *Anisakis* larva after ulceration is rare. No similar report in which the body of the organism was found in a forceps biopsy specimen

from the margin of an ulcer was found in a search of the MEDLINE database (1998-2002). In the present case, the diagnosis of gastric anisakiasis was considered definitive, because the incidental existence of a larvae in the margin of an ulcer would seem to be highly unlikely. When the morphology of an ulcerative gastric lesion suggests gastric cancer without the histopathologic findings of malignancy, anisakiasis should be considered.

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Metastatic Merkel cell carcinoma masquerading as colon cancer

Merkel cell carcinoma (MCC) is an unusual dermal neoplasm of neuroendocrine origin, which occurs predominantly in elderly men.¹ Since the initial description by Toker² approximately 2000 cases have been reported.³ Although rare, MCC has been described as the most malignant primary skin tumor.⁴⁻⁶ In the majority of cases, the presentation is that of a flesh-colored, red or blue, firm, non-tender intracutaneous mass that grows rapidly over a few weeks to months and that may ulcerate.^{3,5} Most patients (70%-80%) with MCC present with localized disease; the remainder have regional lymph-node involvement at initial presentation.³ Distant metastasis is present in 1% to 4% of patients,³ the common metastatic sites being skin (28%), lymph nodes (27%), liver (13%), lung (10%), bone (10%), and brain (6%).^{3,6} A rare case in which MCC presented as colon cancer, with further metastasis to the stomach and the duodenum, is described herein. The importance of early and wide surgical treatment and close follow-up are exemplified by this case.

Case report. A 62-year-old man presented with an enlarged right axillary lymph node that had been present for 3 weeks. Review of systems revealed an unintentional loss of 10 pounds of body weight and an enlarged lymph node in the left groin that had been present for 5 months. The medical history included a myocardial infarction, hypertension, diabetes mellitus, and hyperlipidemia. In addition, a skin lesion on the right side of the neck excised 6 years earlier at another facility had been diagnosed as basal cell carcinoma. He occasionally smoked cigars and consumed alcohol in moderation. On further questioning, the patient acknowledged that he had been evaluated at another facility 5 weeks earlier because of left groin lymphadenopathy. CT of the abdomen and the pelvis had demonstrated a 5 × 7-cm, left groin mass separate from the femoral vessels, along with gastrohepatic and portocaval lymphadenopathy. The CT also demonstrated a 1.5 × 2.0-cm soft tissue lesion in the descending colon.

Examination revealed an 8 × 8-cm firm, non-mobile, enlarged lymph node in the right axilla, and a 10 × 12-cm firm, non-mobile enlarged lymph node in the left groin. There was no scar apparent on the right neck to correlate with the reported excised lesion. Digital rectal examination yielded stool that tested positive for occult blood.

Laboratory tests included a complete blood-cell count, which revealed macrocytic anemia. Biochemical tests of liver function, and serum amylase and lipase were within normal ranges. Colonoscopy disclosed an ulcerating 0.7 × 0.5 × 0.4-cm mass at approximately 45 cm from the anus.

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