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Invasive Gastric Anisakiasis. The Fisrt Confirmed Foodborne Parasitosis Case Report from Valencian Community (Spain).

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Abstract

Anisakiasis, in an environment like ours with high fish consumption, is an emerging zoonosis caused by the nematode of the Anisakidae family (Anisakis simple) acquired by ingesting raw or undercooked fish contaminated by larvae of the parasite. Being frequent in northern European countries and mainly in Japan, the incidence of reported cases in Spain (mainly the clinical forms of gastric presentation) is still low due to the low suspicion of diagnosis and the difficulty of performing an urgent gastroscopy in some hospitals. The clinical picture generally begins 5-7 hours after the ingestion of the suspicious fish (generally in our environment, raw or vinegar anchovies) with epigastric pain, nausea and vomiting (gastric anisakiasis), frequently accompanied by an allergic picture (angioedema, urticaria, anaphylaxis) in cases of intestinal anisakiasis. The clinical forms of gastric presentation have been little described in our setting (35 cases until 2016). To our knowledge, this is the first case of invasive gastric anisakiasis reported in the Valencian Community. Gastroscopy allows a diagnosis of certainty (possibility of direct visualization of free or partially embedded intragastric nematodes) and proceed to endoscopic extraction. Conclusion: In our setting, the history of recent ingestion of suspicious fish must be asked and investigated in patients with acute-onset digestive symptoms. Early gastroscopy is the technique of choice in gastric anisakiasis and allows the extraction of the larvae if it is visualized.

CASE REPORT

We present a case of a 79-year-old man seen in Emergency with a 24- to 36-hour history of intense abdominal pain – diffuse but predominantly epigastric – with nausea and vomiting. The

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day before symptoms onset, he had eaten fresh anchovies in vinegar (not previously frozen). He had no fever or other symptoms (diarrhoea, urticarial or anaphylaxis reaction). His medical history included high blood pressure; dyslipidaemia and type 2 diabetes; aortobifemoral bypass (2007); single bypass of the left anterior descending coronary artery 6 months previously, and acute non-ST-elevation myocardial infarction treated by primary angioplasty with drug-eluting stent implantation; permanent atrial fibrillation; moderate tricuspid regurgitation with pulmonary hypertension; right retinal vein occlusion (2008); and prostatic adenocarcinoma. The patient was taken the following usual treatment: rivaroxaban, acetylsalicylic acid, torasemide, omeprazole, valsartan/hydrochlorothiazide, atorvastatin, amlodipine, ranolazine, repaglinide and metformin/sitagliptin.

Physical examination showed haemodynamic stability and diffuse, predominantly epigastric abdominal pain with no signs of peritoneal irritation. The only significant blood test finding was neutrophilia without leukocytosis and a C-reactive protein level of 2.6 mg/dL. Abdominal ultrasound showed biliary microlithiasis with no evidence of acute cholecystitis; and CT scan showed severe gastric wall thickening with significant oedema, compatible with infection/inflammation or gastric ischaemia. A gastroscopy confirmed gastric thickening, revealing a nodule below the cardia and a winding structure compatible with *Anisakis*, which was removed with suitable endoscopic forceps (Images 1 and 2). Biopsies showed eosinophilic gastritis and gastric metaplasia compatible with *Anisakis* infection. Immunoglobulin E (IgE) levels were 580 IU/mL (normal: 0-100) and *Anisakis*-specific IgE was 63.30 kU/L (normal: 0-0.35). The patient improved rapidly and was discharged without symptoms 24 h after endoscopy. Patient was reviewed at 30 and 90 days from hospital discharge, remaining asymptomatic.
Final diagnosis: Gastric anisakiasis treated by endoscopic removal of the parasite.

DISCUSSION:

Anisakiasis is a zoonosis caused by the nematodes *Anisakis simplex*, *Anisakis physeter is*, *Anisakis pegreffi* and *Pseudoterranova*. Marine mammals (whales, seals, sea lions and walruses) are the natural hosts of these parasites. *Anisakis* nematodes can cause several health problems in humans who become infected after eating raw or undercooked fish or squid [1].

The types of fish most frequently associated with infection are herring, hake, mackerel, anchovies and cod. Cases of anisakiasis are occurring with increasing frequency in western countries, probably owing to cultural influences on eating habits, and a greater consumption of foods containing raw fish (e.g. sushi) [3]. Freezing the fish or squid at -20°C and/or cooking it at 70°C are the best ways of preventing infection [1].

The life cycle of the parasite begins with the excretion of nonembryonated eggs in the faeces of marine mammals. The larvae are ingested by crustaceans and migrate to the muscle tissue. These larvae are transferred to fish and squid through predation. When marine mammals ingest contaminated crustaceans or fish, the larvae develop into worms that become embedded in the gastric mucosa, then lay eggs that are shed in the faeces. The same process occurs in human hosts [1].

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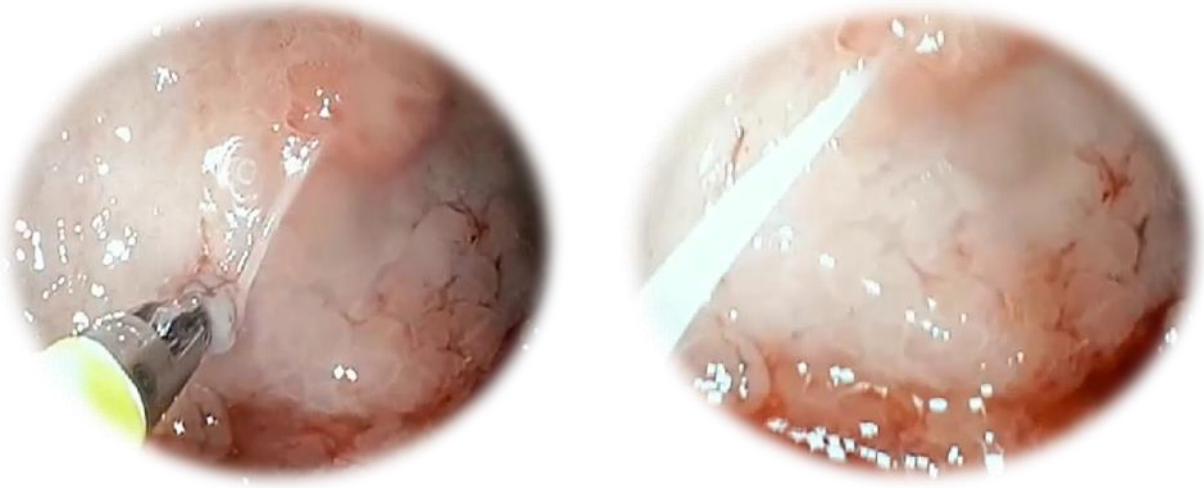
The main clinical symptoms of *Anisakis* infection are gastric, intestinal, extra gastrointestinal and allergic diseases. After ingesting the infected raw or undercooked fish, some people experience pruritus and tingling in the posterior oropharynx. Gastric anisakiasis usually develops 1 to 8 hours after ingestion and manifests as acute epigastric pain, nausea and vomiting. Intestinal anisakiasis normally develops after 5 to 7 days, manifesting as abdominal distension and intense abdominal pain, and often a palpable inflammatory mass obstructing the intestine. Infected people may also have symptoms characteristic of eosinophilic gastroenteritis or enterocolitis. If the ileocecal region is affected, anisakiasis can mimic acute appendicitis, often resulting in surgery [1, 3]. The *Anisakis* larvae occasionally penetrate the peritoneal cavity or other visceral organs (extragastrointestinal anisakiasis), causing eosinophilic granuloma that can be confused with neoplasm [1].

Anisakis can cause allergic reactions ranging from mild urticaria (pruritus and skin lesions) to life-threatening anaphylaxis. Fever and eosinophilia are common, though if the disease is diagnosed early, eosinophil levels normally stay within normal limits (as in our case). A major allergenic protein (Ani s7) has been identified in *Anisakis spp*., although no Ani s7-specific IgE test is commercially available at present [2]. The larvae of the parasites must be viable for this allergic reaction to take place: neither skin tests nor oral challenges with lyophilised parasites have produced a reaction [3].



Figures 1 and 2: taken during the endoscopic removal of the Anisakis *worm from the patient's stomach, in the Endoscopy Room of San Juan de Alicante University Hospital.*

Visual examination of the worm in the patient's vomit or by endoscopy can lead to a diagnosis of anisakiasis. The larvae may be clearly visible for an endoscopist, or the endoscopic examination may reveal an ulcerated lesion in the stomach or duodenum with the worm in the centre (as in the

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images above, which show an ulcerated lesion from which the *Anisakis* was removed). Barium swallow tests may show narrowing of the intestinal lumen in areas with inflamed mucosa. CT scans may show mucosal wall thickening in the stomach or bowel [1]. Total and *Anisakis*-specific IgE levels are usually high, especially in patients who suffer an allergic reaction after infection. Immunological tests such as ELISA have been developed for diagnosing anisakiasis, but they are not widely available [1, 3].

The differential diagnosis of small bowel obstruction due to this parasite includes tumours, Crohn's disease, primary eosinophilic gastroenteritis and other parasites (*Strongyloides*, *Ascaris*, *Toxocara*, *Ancylostoma*), bacterial infections (*Yersinia*, tuberculosis), intussusception and ischaemia [1].

In most cases, symptoms are self-limiting and improve with symptomatic treatment and gastroprotectant drugs [3]. The gold standard for the diagnosis and treatment of anisakiasis is endoscopic removal of the parasite (we used this technique for our patient in view of his persistent symptoms) [1]. In patients with symptoms of bowel obstruction, corticosteroids reduce local inflammation to facilitate bowel movements and thereby eliminate the need for surgery [3]. Antihistamines as well as corticosteroids reduce symptoms of allergic reactions. Anaphylaxis is potentially life-threatening and requires administration of adrenaline [2]. Unlike other human helminths, Anisakis larvae do not respond well to common anthelmintics such as mebendazole, albendazole or thiabendazole [1, 3].

Spain is a country with high fish consumption and, after Japan, is considered the second country with the highest incidence of anisakiasis, although no extensive series are described. Among the two largest series published in our country, only 16 cases correspond to the gastric form [4,5]. In the Valencian Community, two cases has been previously described, corresponding to an intestinal and an allergic form of presentation [6,7]. To the best of our knowledge, our case illustrates the first invasive gastric anisakiasis described from Valencian Community, a typical Spanish region with high touristic affluence and high fish consumption.

In summary, when faced with a clinical picture consisting of acute-onset epigastric pain and vomiting, the patient should be asked about recent consumption of raw or undercooked fish (not always self-reported), and an anisakis infection should be suspected and ruled out among the presumptive diagnoses. Therefore, a good anamnesis is key. The most effective diagnostic and therapeutic technique is endoscopic visualization and removal of the parasite.

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