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Mesenteric tumor due to chronic anisakiasis

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ABSTRACT
Intestinal anisakiasis is a rare parasitic disease and difficult to diagnose due to symptoms are not specific, so it is considered an underdiagnosed disease. The clinical suspicion with a correct diagnosis of anisakiasis allows the establishment of a correct treatment; in most cases, the resolution is possible with conservative treatment, avoiding unnecessary surgery to the preoperative differential diagnosis of acute abdomen. We report the case of a patient who required urgent surgery secondary to an exacerbation of chronic anisakiasis.

Key words: Anisakiasis. Mesenteric tumor. Acute abdomen.

INTRODUCTION
Anisakiasis is a zoonosis that is acquired after eating raw or undercooked fish contaminated by Anisakis larvae (1,2).
In gastric anisakiasis, Anisakis larvae is usually recognized during the upper endoscopy, but intestinal anisakiasis is usually diagnosed after the combination of imaging and serological tests, so that, anisakiasis is considered an underdiagnosed disease, with an increasing prevalence due to the currently popularity of Japanese food, consisting of undercooked or raw fish (3).
The clinical symptoms include digestive symptoms resulting from the interaction of the parasite on the intestinal wall, and allergic reactions such as urticaria, angioedema, and even anaphylactic
shock due to a hypersensitivity reaction (1).

We report the case of a patient who required emergency surgery, diagnosed with acute abdomen secondary to an exacerbation of chronic anisakiasis.

**CLINICAL CASE**

A 46-year-old male who presented feeling of gastric fullness and bloating for three months, without nausea, vomiting, or pathological products. He went to the emergency room with abdominal pain over the previous fifteen days. Leukocytosis was noted (17,800 cells/µL).

The patient underwent CT of the abdomen that showed a mesenteric mass (41x46x60 mm) with calcified material, adjacent to a small bowel loop, with wall thickening and locoregional lymph nodes, and intra-abdominal free fluid (Fig. 1).

The patient underwent surgery for exploratory laparotomy and was found to have a mesenteric mass in the distal ileum. An intestinal resection was performed with the tumor (Fig. 2A). The postoperative period progressed satisfactorily, was discharged on the fifth postoperative day.

Pathology reported an ovoid formation of 6.5x4 cm. Microscopy showed an inflammatory process, ulcerated mucosa affecting the wall and perivisceral fat, consisting of lymphocytes, plasma cells, polymorphonuclear, neutrophils and eosinophils; also small granulomas consisting of giant cells surrounded by eosinophils were evident. A conglomerate necrotic material with calcium deposits and peripheral epitheliod histiocytes was identified (Fig. 2B). Nearby lymph nodes were affected showing follicular lymphoid hyperplasia. With PAS and Ziehl no fungi or acid-fast bacilli were observed. The morphological characteristics of the process suggested a parasitic etiology.

Subsequently, and as a consequence of the pathological findings, a stool culture (being negative for *Salmonella, Shigella, Yersinia, Campylobacter* and *Aeromonas*) and a direct parasitological stool examination were performed, both tests were negatives. Allergic sensitization finally confirmed, with RAST for anisakis of 0.97kU/L, so moderate class 2 (0.70 to 3.4 kU/L) was considered positive.

**DISCUSSION**

Digestive anisakiasis is the result of the combination of the direct tissue damage, involving the development of eosinophilic granulomas and/or perforations, and allergic reactions (3). Gastric anisakiasis represents approximately 96% of cases, whereas the intestinal forms account for the remaining 4% (4).

Clinical manifestations of acute gastric anisakiasis may present with epigastric pain and can mimic other entities such as peptic ulcer or gastritis; symptoms of intestinal involvement may be
compatible with appendicitis, intussusception or ileitis. The manifestations of chronic anisakiasis present with abdominal pain, vomiting, diarrhea, constipation, intestinal obstruction and/or fullness (5,6).

Pathological findings combine two different mechanisms: Direct tissue damage and allergic reaction. Tissue damage is due to the direct invasion of larvae that favors the development of an eosinophilic granuloma or mucosal perforation, resulting in a transmural edema, vascular congestion and diffuse infiltrate of neutrophils and eosinophils (7).

Intestinal anisakiasis, sometimes, is underdiagnosed due to manifestations of anisakiasis appears at least one week after ingestion of contaminated food; so, it is important to know the radiological manifestations of the disease (6,7). Radiological findings reflect the histological changes described, such as the concentric thickening of the bowel wall, the sign of the target (as a result of different contrast enhancement by the layers of the intestinal wall), intestinal obstruction and/or perilesional free liquid; all as a result of eosinophilic infiltration and edema caused by the passage of the larva to the bowel wall (3,7,8).

Unlike other parasitic diseases, peripheral eosinophilia is uncommon, and also stool tests are negative, because the parasite does not reach the adult form in man, not eliminating eggs in stool. Currently, the gold standard for the diagnosis of anisakiasis is specific antibodies of IgA, IgG and IgE Anisakis simplex, with a sensitivity of 70-80% (1,4,8).

Although the incidence of anisakiasis is increasing by the increased consumption of raw fish, is considered to be an underdiagnosed disease. It is necessary, therefore, to have the clinical suspicion with nonspecific abdominal manifestations, being essential a correct history that included dietary habits. The suspicion of this entity and the correct diagnosis of anisakiasis allow the establishment of a correct treatment, since in most cases the resolution is possible with conservative treatment, avoiding unnecessary surgery to the preoperative differential diagnosis of acute abdomen.

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**Fig. 1.** CT of the abdomen showing a mesenteric mass with calcified material.

**Fig. 2.** A. Open mesenteric mass. B. H-e showing an inflammatory process consisting of diffuse infiltrate of lymphocytes, neutrophils, plasmatic cells and eosinophils.