



Hepatic Anisakiasis Presented as a Suspected Metastatic Cancer

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Abstract

Anisakiasis is a zoonotic disease caused by ingesting nematode larvae found in raw seafood dishes such as sushi, sashimi, ceviche, and marinated herring. While it primarily affects the digestive tract, cases outside have been reported. We present the case of a young Spanish patient who arrived at the emergency department with abdominal pain and fever. A thoraco-abdominopelvic CT scan revealed irregular thickening of the gastric wall, suspected to be a lesion, along with multiple hepatic lesions suggestive of secondary appearances and nodules in the greater omentum, indicative of peritoneal carcinomatosis. This case details a rare instance of hepatic anisakiasis initially mistaken for metastatic cancer, underscoring the importance of considering hepatic anisakiasis as a differential diagnosis in patients with diets rich in raw fish. Patient consent was obtained for the publication of this case.

Subject Areas

Gastroenterology & Hepatology

Keywords

Anisakiasis, Zoonotic Disease, Nematode Larvae, Raw Seafood Abdominal Pain, Hepatic Lesions, Peritoneal Carcinomatosis

1. Introduction

Anisakiasis is a zoonotic disease caused by the ingestion of nematode larvae found in raw seafood dishes such as sushi, sashimi, ceviche, and marinated herring. To date, more than 15,000 cases have been reported, with Japan being the most affected country due to its dietary habits. The pathological changes in the gastrointestinal tract during an Anisakis simplex infection result from both the direct action of the larvae invading the tissues and the complex interaction between the

host's immune system and the parasite. This disease is currently considered an emerging public health concern.

In non-endemic regions, the risk of misdiagnosis is high due to a lack of awareness of the disease. Atypical presentations, such as acute abdominal pain, severe allergic reactions, or nonspecific gastrointestinal symptoms, can be mistaken for other conditions like ulcers or Crohn's disease. Diagnostic challenges include the need for a detailed dietary history and the use of specific techniques such as endoscopy and medical imaging to identify larvae.

Increasing awareness among clinicians in non-endemic areas is essential to improve recognition and management of the disease, thereby reducing the risk of misdiagnosis and enhancing patient outcomes.

2. Clinical Case

Mr. J.L., of Spanish origin, presented to the emergency department for the management of diffuse abdominal pain lasting for more than three weeks, associated with intense asthenia and a fever of 39°C.

The abdominal examination revealed tenderness in the epigastric area and the right iliac fossa, while the rest of the clinical examination was unremarkable.



Figure 1. Hepatic lesion in segment IV with a secondary appearance.



Figure 2. Hepatic lesion in segment VII.

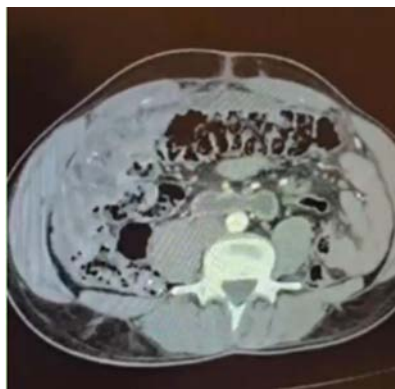


Figure 3. Thickening of the greater omentum with the appearance of omental cake.

A biological assessment was performed in the emergency department, which showed a C-reactive protein level of 190 mg/L, platelet count of 726 G/L, leukocytes at 9.6 G/L, no hepatic cytolysis, GGT at 252 U/L, PAL at 278 U/L, lipase at 22 U/L, and total bilirubin at 5 μ mol/L.

An abdominal CT scan performed in the emergency department revealed irregular thickening of the gastric wall, particularly in the anterior wall, measuring 13 mm in thickness, along with secondary-looking hepatic lesions and nodular thickening of the greater omentum consistent with peritoneal carcinomatosis (**Figures 1-3**).

Gastroscopy revealed a budding appearance of the bulb, non-stenotic and non-ulcerated (multiple biopsies taken). Presence of the worm in the duodenum (larvae removed using biopsy forceps) (**Figure 4**).

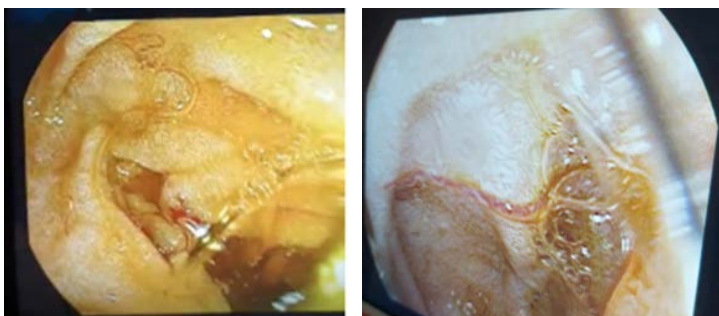


Figure 4. Anisakis larvae embedded in the duodenum.

The histopathological study of the gastric biopsies revealed an inflammatory mucosa with eosinophilic granulomas.

A serology for anisakidosis was performed, showing: specific IgE at 1.53 KUA/L (reference value < 0.10) and specific IgG4: P4 Anisakis at 0.1 mgA/L (reference value < 0.1).

To determine the origin of the hepatic lesions, a biopsy of a hepatic nodule in segment VIII was performed. The histological examination of the hepatic biopsies revealed epithelioid and gigantocellular granulomatous inflammation with necro-

sis, suggesting an infectious etiology.

Given the endoscopic and histological findings, as well as the history of consuming raw seafood, Anisakis infection was diagnosed as the cause of the gastric and hepatic manifestations. The patient was treated with albendazole at a dose of 400 mg twice a day for 14 to 21 days.

During hospitalization, there was a clear clinical improvement: the pain and fever disappeared, and the abdominal examination was strictly normal. The C-reactive protein level decreased to 50 mg/l. The decision to discharge was made, but unfortunately, the patient was lost to follow-up.

3. Discussion

Anisakiasis is a cosmopolitan zoonosis whose incidence has been steadily increasing over the past two decades [1].

The first zoonotic case attributed to the *Anisakis* species was described in the Netherlands by Van Thiel *et al.* in 1960. Anisakiasis is divided into three types: gastric anisakiasis, intestinal anisakiasis, and extra-gastrointestinal anisakiasis [2]. Ishikura *et al.* reported that gastric anisakiasis accounts for 95.7% of all cases, intestinal anisakiasis for 3.8%, and extra-gastrointestinal anisakiasis for only 0.5%. Among extra-gastrointestinal anisakiasis cases, hepatic anisakiasis is extremely rare.

Humans become infected by ingesting raw fish containing *Anisakis simplex* in its larval form. The disease may be secondary either to infestation by a single larva or to a massive infestation [3].

The clinical presentation is most often mild or even asymptomatic (dyspeptic syndrome, transient skin allergy), but it can also manifest in a more severe form with peritoneal complications or severe allergic reactions.

Most cases have been reported in Japan due to dietary habits; however, it is increasingly being recognized in Western countries [4].

Hepatic anisakiasis can occur when *Anisakis* larvae migrate from the digestive tract wall to the peritoneal cavity, invade the surface of the liver, and form a tumor at the hepatic margin. It is also presumed that the larvae from the digestive tract could reach the liver via the portal vein, but there are no reports of hepatic anisakiasis invasion through any pathway other than the surface of the liver [5].

Anisakiasis of the digestive system can be confused with several other conditions due to the similarity of symptoms. Acute appendicitis, characterized by sharp abdominal pain, is often considered in the differential diagnosis. Similarly, gastroenteritis, which presents with pain, nausea, and diarrhea, can be challenging to distinguish from anisakiasis. Gastro-duodenal ulcers, with their gastrointestinal discomfort and pain, as well as Crohn's disease, which causes similar intestinal symptoms, are also possibilities. Intestinal obstruction, manifesting as abdominal pain and vomiting, should also be considered. Accurate diagnosis requires thorough clinical examinations, medical imaging, and sometimes endoscopy to differentiate these conditions and provide appropriate treatment.

For a positive diagnosis, ultrasound or CT scans may show intestinal wall thickening, decreased peristalsis, intraperitoneal effusion, or a pseudo-tumoral appearance [6]. Hepatic anisakiasis tumors present as a small low-density area at the hepatic margin on CT. A biopsy may be an option for a small low-density tumor at the hepatic margin, unless there is a strong suspicion of malignant disease.

The parasitological examination of stool samples is not contributory [7].

Direct visualization, through endoscopy, of the parasite embedded in the gastric or intestinal wall allows for a definitive diagnosis. The examination will simultaneously enable the extraction of the larvae and resolution of the symptoms. The endoscopic lesion may show simple mucosal edema, more or less pronounced depending on the duration of the infestation [8].

Histologically, the study of the surgical specimen in patients who underwent surgery generally reveals significant edema of the mucosa and submucosa, which can sometimes be transmural and present a pseudo-tumoral appearance. It may sometimes show an eosinophilic granuloma, centered around a larva with a digestive tube and lateral branches, but it most often only provides a probabilistic argument.

The use of serological techniques faces several challenges. There are numerous cross-reactions with other parasitic infections (such as *Ascaris*, *Toxocara canis*, *Trichinella*, or *Echinococcus granulosus*). Antibodies typically become positive only several days after infection, making them of limited use for diagnosing acute infection. Finally, the detection of specific antibodies does not indicate the timing of the infestation but only shows that the subject has had contact with the parasite at some point in their life (IgE levels can remain elevated for several months or even years). For example, it is considered that 15% to 27% of the general population has a positive serology [9]. Therefore, serology is not very specific for acute infection and must always be correlated with the patient's history and symptoms [10]. In our case, the patient's dietary history and pathological results, including significant infiltration of inflammatory cells, edema, the formation of granulation tissue, and the visualization of the worm in the duodenum, were compatible with a diagnosis of anisakiasis.

The absence of patient follow-up is a significant limitation of our study. This restricts our understanding of the long-term progression of the disease and the effectiveness of the treatment. Follow-up is crucial for evaluating recurrences or late complications, which would help improve management strategies and ensure better outcomes for patients.

4. Conclusion

Hepatic anisakiasis remains a rare condition in Western countries, highlighting the crucial importance of obtaining a detailed dietary history to guide diagnosis. The diagnosis is confirmed through biopsy results, while serology allows for retrospective evaluation. Since 2004, prevention of the disease has relied on well-codified measures for fishing and fish preservation.

It is essential to increase clinician awareness of this condition to improve recognition and management. Proposing a diagnostic algorithm could help standardize the clinical approach, incorporating key steps such as assessing dietary history, utilizing medical imaging, and confirming with biopsy. These strategies will help reduce the risk of misdiagnosis and optimize patient care.

Conflicts of Interest

The author declares no conflicts of interest.

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