

Colonic Anisakiasis: an exceptional presentation

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ABSTRACT:

- Anisakiasis is a worldwide parasitic disease with 90% of reported cases in Japan. Here we present the case of a Spanish 55 years-old male patient with acute abdominal pain in the right hypochondrium. The final diagnosis was infectious right colitis caused by *Anisakis*.
- **Keywords:** *Anisakis*, Colonic anisakiasis, Parasitic infection, Parasitic disease, Intestinal anisakiasis, Intestinal disease, Ultrasound, Computed tomography.

INTRODUCTION

Anisakiasis is an under-diagnosed zoonotic infection whose incidence is increasing in our environment. It is caused by *Anisakis simplex*, *Anisakis physeteris*, or *Pseudoterranova* species. Marine mammals are natural hosts. Human beings become incidental hosts by eating raw or undercooked, infected fish. It is estimated that 90% of anisakis cases are reported in Japan. Clinical manifestations are due to allergic reactions or the local action of the parasite. The gastric form of anisakiasis is more frequent than intestinal form and the colonic form is rare. The diagnosis is made by direct visualization of the wound by endoscopy. Radiology imaging can be helpful in the diagnostic approach. The treatment is removing the wound by endoscopy and conservative management. The main preventive action is heating to over 70°C (158°F) or freezing to -20°C (-4°F) for 72 hours. This communication aims to review clinical and diagnostic settings of intestinal infestation caused by *Anisakis* to include it in acute abdominal pain differential diagnosis. Spain is a geographical region where its prevalence is low but due to changes in lifestyle and eating habits, its incidence is increasing. Practitioners should be aware of this condition to make a prompt and correct diagnosis.

CASE REPORT

A 55 years-old male patient with only diabetes mellitus as relevant medical history came to the Emergency Department of our hospital having experienced acute abdominal pain for 24 hours in the right hypochondrium. Other symptoms were depicted. Physical examination was normal with leukocytosis and mild elevation of ALT (alanine aminotransferase). Due to clinical suspicion of acute cholecystitis, an abdominal US was performed (Figure 1). Imaging compatible with colo-colonic intussusception in hepatic flexure with intestinal wall thickening was seen. Abdominopelvic computed tomography (CT) was performed with intravenous contrast and the main findings were wall thickening of ascending and transverse colon with stranding of adjacent fat and a minimal amount of ascites (Figure 1). The main suspicion was an infectious disease or inflammatory colitis, the possibilities of ischemic or neoplastic etiologies were less probable.

In a colonoscopy, a worm was observed (Figure 2) and confirmed by the Microbiology Department as *Anisakis*. Besides the colonoscopy, the patient received Albendazole 400 mg daily for 3 days. After treatment, the patient improved clinically and remains asymptomatic nowadays.

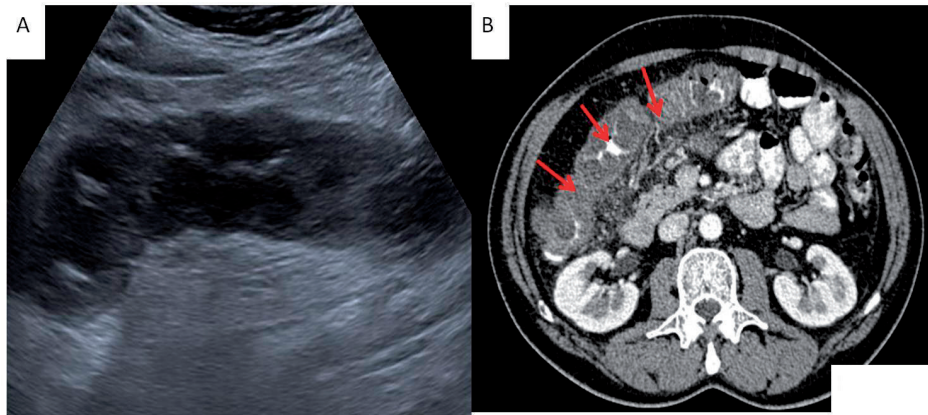


Figure 1. Abdominal US (A) and axial contrast-enhanced abdominopelvic CT (B) shows circumferential mural wall thickening in ascending and transverse colon (arrows in B) with stranding of the adjacent fat and small amount of ascites in the pelvis (not shown).

DISCUSSION

Anisakis is a nematode (roundworm) part of the family Anisakidae (superfamily Ascaroidea)¹, with a worldwide distribution¹ that was first described in 1809 by Rudolphi¹. However, it wasn't until 1955 when human infection was first noted in the Netherlands^{1,2}.

Marine mammals (whales, seals, dolphins) are natural hosts¹⁻⁶, and human beings are incidental hosts^{1,3,5-7}.

Eggs are driven out in marine mammals faeces. They hatch in the water, where they develop throughout the first and second larvaria phase³. These larvae are ingested by small crustaceans like plankton, where the third larvae phase takes place. Fish and cephalopods ingest those infected crustaceans, and so the larvae migrate to muscles and visceral organs. Its life cycle is complete when marine mammals eat contaminated fish, after which, the larvae move to the fourth phase and adult phase so they can produce eggs.

Principal vectors are fish, crustaceans, and cephalopods. The bigger the animal is, the higher its parasite index and, therefore, infestation.

Human beings become accidental hosts by ingesting contaminated raw or undercooked fish (intermediate host)^{3,6}. Larvary stadium 3 is the only one able to infect humans³. After ingestion, the larva penetrates the gastric

and the intestinal mucosa, where it dies and causes an inflammatory reaction and abscess formation with eosinophilic predominance³. Often larvae trespass the intestinal wall forming an abscess inside of peritoneal cavity^{2,3}.

Countries where it is most prevalent are Japan (because of sushi), Scandinavia (cod liver), the Netherlands (herring) and Latin America (ceviche)¹. In Spain, principal causes of infection are anchovies and sardines¹. 20.000 cases are detected worldwide annually, of which, 90% come from Japan^{2,6}. In Spain, center and northeast are the geographical areas with higher prevalence^{1,8}. Madrid's population has shown recently a 12.4 % prevalence of specific anisakis IgE^{1,8}. Sensitization increases with age⁸. Symptoms are due to the parasite's local action or to hypersensitivity reactions⁵.

Orally, the parasite can lead to the following clinical situations¹:

1. Anisakiasis: because of local action of the parasite in the digestive tract with a latency period of 12-48 hours till the onset of the symptom.
 - Gastric anisakiasis is the most frequent form (72%) taking place between 1-8 hours after contaminated fish ingestion¹⁻³. Symptoms include epigastralgia, nausea, and vomiting^{1-4,7}. In analytics leukocytosis^{2,4,5}, increased CRP⁵ is noted and in less than 30% of cases eosinophilia is observed^{1,3,5}.

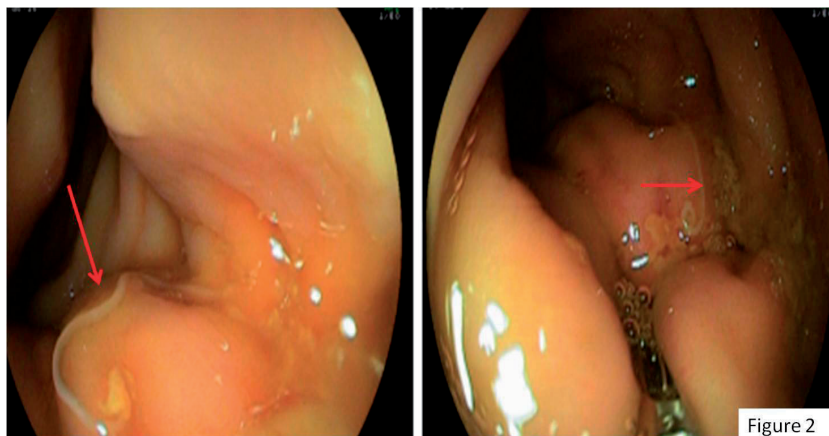


Figure 2. Colonoscopy. A white filiform worm (arrows) is seen in hepatic flexure as well as edematous mucosa due to inflammatory changes.

- The intestinal anisakiasis form develops around 5–7 days post-ingestion, which is difficult to diagnose¹⁻³. Its symptoms include acute abdominal pain and peritonism¹. The main complications are stenosis, obstructive ileus, perforation and pneumoperitoneum¹⁻³. It can occur in any localization, although is more common in terminal ileum². The colonic involvement is rare, the caecum being the more common site⁶.
- 2. Immediate hypersensitivity reaction, an immunoglobulin E (IgE) mediated clinical picture a few hours after the ingestion that depicts banal allergic symptoms as urticaria to angioedema or anaphylaxis^{3,7}.

Causative allergens of anisakis hypersensitivity are the AniS, being 1 and 4 subtypes the most important in this group¹.

In 2001, the Spanish Society of Allergy established a fish sensitization prevalence of 13.1% in the healthy population and 38.1% in subjects with chronic urticaria¹.

- 3. Gastroallergic anisakiasis: mixed between the previous two. Up to 10% of the anisakiasis cases are accompanied by urticarial manifestations¹.
- 4. Infection outside the digestive tract is due to the larva migration¹, which crosses the gastric or intestinal wall and gets into the peritoneal cavity or other visceral organs where it dies and triggers an inflammatory reaction, and consequently, an abscess / eosinophilic granuloma that may be confused with a neoplasm^{2,3}.

When the parasite infects the skin or mucosae, it can result in contact dermatitis, rhinoconjunctivitis or asthma¹. The diagnosis can be challenging. It can be made with compatible symptoms and a complete anamnesis. Without raw or undercooked fish, ingestion is difficult to carry out the diagnosis^{1,5}. Allergic tests demonstrate a previous contact¹. The cutaneous allergic test is the most sensible and less specific¹ because of crossed reactivity^{1,8} with antigens from other parasites (*Ascaris* and *Toxocara*). *Anisakis* induces the immune system to produce a series of specific antibodies that may be detected by different techniques such as indirect immunofluorescence (IFI), radioimmunoassay test (RAST), enzyme-linked immunosorbent assay (ELISA) and Western blot^{2,3}.

Specific anisakis IgE determines the grade of sensitization. It is the most specific with low sensibility. Its value depends on the moment when is taken after exposition¹. It must be taken into account that in a hyper acute onset serology is negative until 10 days after infection². Serial determination of serum IgE concentration can confirm the diagnosis when it decreases more than 50% in 6 months after the symptoms¹.

Radiological imaging is useful for the diagnostic approach, especially in the intestinal form, although imaging findings are not specific³. They are also important for discarding other abdominal pathologies with similar acute abdominal pain^{5,7}.

Main radiological findings are circumferential and symmetric intestinal mural thickness with narrowing of the lumen^{3,4}, folds thickening, dilatation of proximal small intestine loops⁵⁻⁷ and mucosa enhancement (“target sign”) due to submucosa edema^{4,5,7}. It is also frequent to

observe stranding of adjacent mesenteric fat, blood vessel engorgement, collections, ascites and abdominal lymphadenopathy^{5,7}. The differential diagnosis of the intestinal obstruction due to anisakis must include conditions that lead to wall intestinal thickenings like neoplasm, inflammatory bowel disease, ischemic enteritis, eosinophilic enteritis, invagination, appendicitis, diverticulitis, ileitis and other infection caused by parasites (*Strongyloides*, *Ascaris*, *Toxocara*, *Ancylostoma*) or bacterias (*Yersinia*, tuberculosis) mainly^{3,5-7}. To get a certain diagnosis, direct visualization of the parasite in the endoscopy is imperative¹⁻⁴. If treatment is needed in the acute onset, larvae extraction through endoscopy or surgery is the treatment of choice^{1,3,4,7}. In extra digestive affectation, surgery is mandatory³. Symptomatic treatment such as proton-pump inhibitors to decrease local inflammation and relieving abdominal pain¹ and systemic glucocorticoids, antihistaminic or epinephrine if necessary for an allergic reaction, is also used¹. It is still not clear if antihelminthics are used, like Albendazole, whether it is effective for treatment². Preventive measures consist of heating over 70°C for at least 2 minutes; freezing to -20°C for 72 hours^{1-3,6} and avoiding risk food (larvae can survive until 25 days in vinegar and 21 in salting)¹.

Our case was challenging to achieve the diagnosis for both the radiologist and physicians. Intestinal anisakiasis is not usually included in the differential diagnosis of acute abdominal pain for several reasons. It is usually under-diagnosed because of its low frequency, due to the difficulty of knowing about previous ingestion of raw fish 1 week before of symptoms onset and because it is usually misdiagnosed with other acute abdominal pathologies like appendicitis or diverticulitis, more frequently diagnosed.

Besides the rarity of this entity, it should be highlighted that colonic affection due to anisakis is rare because the parasite causes symptoms before reaching the colon in the majority of cases. It is difficult to find a case of anisakiasis with this clinical presentation.

The patient was managed with conventional treatment. Once the *Anisakis* was detected and extracted with the colonoscopy and after 3 days of Albendazole, the patient remains asymptomatic nowadays. Because no complications like abscess or perforation were detected, no surgery was needed.

CONCLUSIONS

Anisakiasis is a growing problem in the Western countries due to changes in nutritional habits. In most cases, worms infest the stomach and less frequently in the lower digestive tract. The solitary involvement of the colon is exceptional. Symptoms and physical examination are widespread and non-specific. Imaging findings may demonstrate edematous wall thickening of the gastric or intestinal mucosa, abscess formation, ileus, perforation... Certain diagnoses can only be made by endoscopic inspection of larvae and it could be therapeutic. Therefore, we must keep anisakiasis in mind, when diagnosing abdominal pain.

AUTHOR CONTRIBUTIONS:

P.C.I. performed the literature search and wrote the manuscript together with J.C.C. Editing of the manuscript draft was done by all authors. The decision of the final version to be published was agreed upon by all authors.

CONFLICT OF INTERESTS:

The authors declare no conflict of interest.

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