have had throughout their history by the different health services.[6] Equally, we must go as far as possible to make the public aware that they should avoid self-medication, even with medicines that are popularly considered to be ''safe drugs'', and that we should not let our guard down in the event of adverse effects because, as we have seen in this case, they can be very serious.

Conflicts of interest

There is no financial support for this project nor conflicts of interests by any of those that took part in it.

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Anisakis and colonic polyp, a rare association☆

Anisakis y pólipo en colon, una asociación infrecuente

Anisakiasis is a parasitic infection caused by ingesting sea fish infected by nematode larvae. It was first diagnosed in Holland in 1960.[1] Japan is currently the country with the highest incidence. It most commonly affects the stomach and small intestine, and rarely the colon. Clinical diagnosis is very difficult due to its non-specific symptoms.

In the literature, there are only 4 cases of anisakiasis linked to bowel cancer.[2–5]

A 73-year-old male undergoing a colonoscopy for polyp monitoring, where a flat polyp of around 4 cm was identified in the caecum with a lateral extension LST-G 0-IIa + 0-IIb according to the Paris Classification, affecting two consecutive folds not susceptible to endoscopic resection, from which biopsies were taken (Fig. 1). The pathology study reported fragments of tubulovillous adenoma from the large intestine with high grade epithelial dysplasia/adenocarcinoma *in situ*, where a Haggitt level could not be established as there were several fragments. In preoperative tests, the patient's blood work showed mild eosinophilia and no significant tumour disease was observed in the imaging tests.

The patient underwent a laparoscopic right hemicolectomy and no macroscopic changes were observed during the procedure and there were no postoperative incidents.

In the pathology study of the colon, the raised, oedematous mucosa in the area where the adenoma biopsies were carried out were macroscopically observed in the caecum. Microscopically, a predominantly transmural eosinophilic inflammatory infiltrate area was observed with microabscess formation, in connection with *Anisakis* larva, located in the muscle layer of the caecum, with low grade dysplasia in the underlying mucosa (Fig. 2), with no significant lesions in 33 isolated lymph nodes.

Anisakis involvement is less common in distal locations of the GI tract and its presence in the colon is rare.[6,7] Some 75 cases of colorectal anisakiasis have been described, the majority of which were in the ascending colon.[2,8]

The acute forms of anisakiasis can present as acute abdominal pain or anaphylactic reaction after consuming products with *Anisakis* in them. Its diagnosis is based on medical history relating the recent consumption of fish and it can be confirmed with the presence of specific antibodies for *Anisakis*, as well as direct observation *via* endoscopy.

On the other hand, the chronic form is more difficult to diagnose given that the antibody titres can decrease and it could be more difficult to see the larva on the endoscopy because it could enter into the mucosa. The differential diagnosis includes appendicitis, Crohn's disease, cancer, intestinal tuberculosis..., in some cases surgical treatment is needed for its diagnosis. In early stages, it can be

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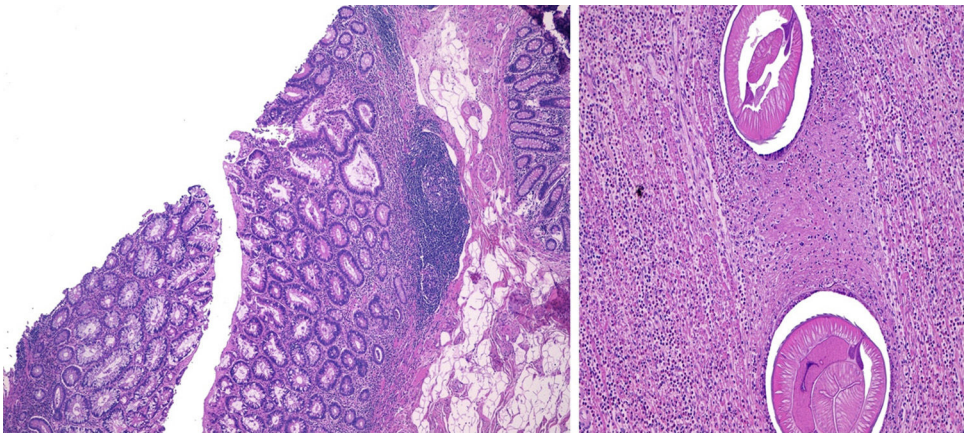

Figure 1 Pathology with *Anisakis* larvae presence.

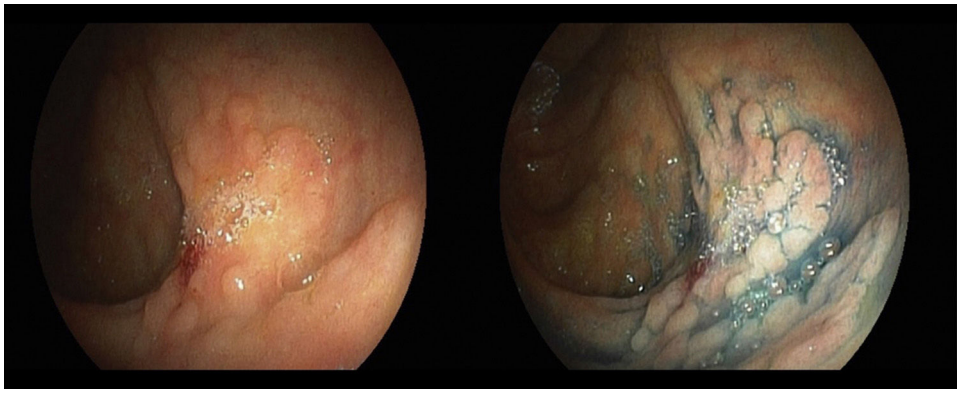

Figure 2 Caecum polyp, with and without indigo carmine staining.

characterised by the presence of eosinophilic granuloma and later, it can turn into abscess tissue.

Of the 4 described cases,[2–5] the larva is found in the ileum in one of them, in the ascending colon in 2 of them (although in one, the tumour is in the sigmoid colon) and another in the sigmoid colon. It is difficult to diagnose pre-operatively, and almost all are described by chance afterwards.

The coexistence of cancer and parasite colonisation has been suggested to be favoured by the changes in the intestinal immunological layers.[4] Furthermore, Petithory et al.[9] question whether *Anisakis* infection could be a factor in the development of carcinoma.

The case we present deals with a patient with a history of bowel polyps, which means it is difficult to determine the cause/effect of the association. One hypothesis could be that the parasite has taken advantage of the changes in the intestinal mucosa to pass through it, which makes the theory that parasite colonisation causes the abnormalities less plausible.

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Acute gastroenteritis and *Haemophilus parainfluenzae*: An unreported but predictable association☆

Gastroenteritis aguda y *Haemophilus parainfluenzae*: una asociación previsible pero no reportada

The term acute gastroenteritis is applied to symptoms of diarrhoea or vomiting ascribable to an infection of the proximal segment of the small intestine or the colon. It is amongst some of the most common infectious diseases and it involves a heightened morbimortality, particularly among the elderly, malnourished and those with underlying illnesses.[1]

The *Haemophilus* spp. coccobacilli make up the normal bacterial flora of the upper respiratory tract, where *Haemophilus influenzae* is the predominant type. In turn, *Haemophilus parainfluenzae (H. parainfluenzae)* has been related to different respiratory tract infections and conjunctivitis. Less commonly, it can cause infective endocarditis and more rarely white tissue abscesses, septic arthritis, genital tract infections, osteomyelitis, wound infections and in very rare cases, meningitis and brain abscesses.[2,3] In recent years, cases of intrabdominal infections of the bile duct, hepatic or pancreatic abscesses, peritonitis and appendicitis have been published.[3–7]

In this article we propose to describe a case of bacterial gastroenteritis by *H. parainfluenzae*.

A 43-year-old male born in Spain, with no previous pathologies of interest nor recent trips, that was seen at our centre presenting a 4-day history consisting of colic-like abdominal pain, vomiting and diarrhoea with no pathological products, accompanied by a high fever and chills.

Upon physical examination, he displayed a heart rate of 103 bpm, blood pressure of 98/63 mmHg and a temperature of 38 °C, with no signs of dehydration. Upon abdominal examination, diffuse pain was noted, more intense in the epigastrium but with no signs of peritonitis and no organomegalies were felt. He also presented cold-sores. The rest of the physical examination was normal.

The blood test showed: white blood cells 12,100 μl with a neutrophil percentage of 82.5%, platelets 95,000 μl, haemoglobin 12.1 mg/dl, C-reactive protein 163.5 mg/l with normal kidney liver function, water-electrolyte balance, amylase and coagulation. HIV infection was ruled out. Cultures were started he was admitted to hospital, receiving Ciprofloxacin as treatment. Whilst he was admitted, he underwent an abdominal CAT scan that reported the presence of findings suggestive of infectious or inflammatory non-specific colitis, affecting segments of the ascending colon. The rest of his abdominal structures were normal. On his fourth day in hospital, *H. parainfluenzae* was isolated in the 2 blood cultures, sensitive to most antibiotics, including quinolones. The stool culture was negative, showing only the presence of conventional mixed flora.

His clinical progress was favourable. In the subsequent outpatient follow-ups 2 months later, the patient remained asymptomatic.

We believe that the patient showed symptoms of acute gastroenteritis caused by *H. parainfluenzae*. The initial clinical symptoms, radiological findings and absence of data suggestive of an alternative diagnosis paired with the isolation of the pathogen in the blood enabled this suspected diagnosis to be confirmed. We cannot rule out that the patient may suffer gastroenteritis from another microorganism, which would allow *H. parainfluenzae* to pass through to the bloodstream. However, taking into account the symptoms of bacteraemia and the absence of other intestinal pathogens, this hypothesised diagnosis does not seem to be substantiated. We must point out that the current culture mediums are not designed to isolate *H. parainfluenzae* in the faeces samples obtained for stool cultures.

In recent years, studies of the bacterial flora in the GI tract have been gaining great importance for their potential relationship with different diseases such as: pseudomembranous colitis, inflammatory bowel disease, irritable bowel syndrome and even chronic constipation and obesity. In this regard, potentially pathogenic microorganisms that were formerly considered to be completely unrelated to this field have gained greater relevance, like with *H. parainfluenzae*, assiduously isolated between the microbacterial flora of the GI tract.[8]

Thus, Palmer GG isolated *H. parainfluenzae* in the intestinal mucosa and suggested that it could possibly act as a pathogen if the gastrointestinal acidity decreased or if the mucosa was altered.[9] Later, Mégraud et al., postulated that the GI tract might work as a reservoir for *H. parainfluenzae*, and they also suggested a possible relationship between the cases of bacteraemia caused by this microorganism, with apparently unknown origins and intercurrent gastrointestinal processes.[10]

Ultimately, we consider out patient to represent a first documented case of acute gastroenteritis by

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