distance from the entry point, as the larva must travel from the mouth to the colon, and its survival is affected by the pH changes that occur along the gastrointestinal tract.[3,4] Colonic intussusception, i.e. the telescoping of a segment of colon into the lumen of the adjacent segment, is very rare, and is usually caused by a malignancy. For all these reasons, anisakiasis is an exceptional cause of colonic intussusception. Only 6 cases of colonic intussusception due to anisakiasis have been described in the literature, in addition to this case.[1,4–8]

Accurate diagnosis is difficult, especially in the absence of a history of fish consumption. In cases of diagnostic suspicion, colonoscopy can reduce the intussusception and confirm the diagnosis of anisakiasis.[3] Ultrasound can be useful in the diagnosis of intussusception, but the CT scan is the method of choice because it can pinpoint the site of intussusception and assess the extent of the obstruction and intestinal viability.[5] Three CT patterns considered pathognomonic of intussusception have been described: the target-like pattern (round mass with intraluminal soft-tissue and eccentric fat density due to the invaginated mesentery), the reniform pattern (a bilobed mass with high peripheral attenuation due to thinning of the bowel wall) or lesion with alternating areas of attenuation related to the bowel wall, mesentery, fluid, contrast material, or gas.[2]

When the diagnosis is suspected, treatment can be conservative, with nil by mouth and fluid therapy, anthelmintics and corticosteroids to reduce parietal oedema, in addition to endoscopic reduction of the intussusception. However, in the absence of a definitive diagnosis, the optimal treatment is not clearly established. Surgery tends to be necessary in most cases due to obstruction, perforation or suspicion of underlying malignant lesions, since anisakis infection produces submucosal tumours that are the cause of the intussusception and require a histological differential diagnosis.[1,4,5]

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Single colonic polyp as a presentation of mantle cell lymphoma☆

Pólipo único colónico como forma de presentación de linfoma del manto

Mantle cell lymphoma, which was first described in 1991 and recognised by the World Health Organisation (WHO) as a disease in 1994, is a type of B-cell non-Hodgkin lymphoma originating from B lymphocytes located in the mantle of a lymph node. It is characterised by expression of B-cell lineage markers (CD19, CD20 and CD5), where CD3, CD10 and CD23 are negative and a chromosomal translocation t(11; 14) causes overexpression of cyclin D1.[1] Mantle cell lymphoma is a rare type of B-cell non-Hodgkin lymphoma, and accounts for just 3–10% of all non-Hodgkin lymphomas.[2] Its incidence in Spain is very low (0.5 per 100,000 inhabitants/year),[3] mainly affecting men (2:1) and individuals over 60 years of age. This aggressive lymphoma has a mean survival of 3–5 years after diagnosis,[1] but, thanks to therapeutic advances and intensive strategies, survival has doubled in the last decade (60% survival at 5 years).[4]

Gastrointestinal involvement, with an incidence of 10–25%, is rare, with the most common manifestation being multiple lymphomatous polyposis, in which multiple lymphoid polyps are identified in the large and small intestine. The polyps usually occur in the ileocaecal region, although they can develop at any site from the stomach to the rectum.[5] They affect the colon and rectum in 90% of cases, the small intestine in 69%, the stomach in 57%, and the duodenum in 52%.[6] Endoscopic diagnosis is rare. Cases with microscopic invasion and normal mucosa on examination have been reported.[4]

We present the case of a 41-year-old man with a history of hypercholesterolaemia. He was seen by the gastroenterologist due to an increase in the number of bowel movements and rectorrhagia lasting several days, with no other accompanying symptoms and no weight loss,

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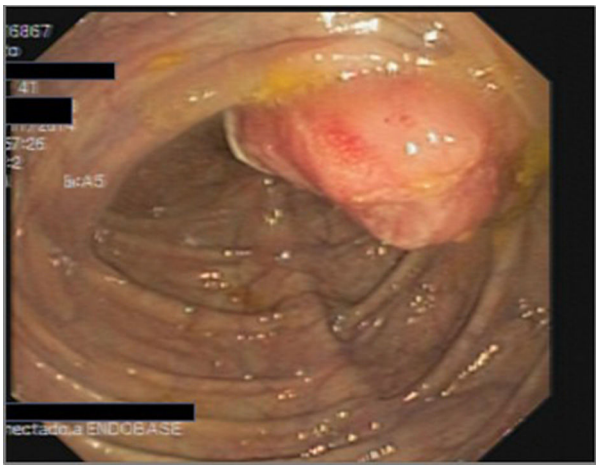

Figure 1 Caecal polyp.

abdominal pain or fever. The physical examination revealed splenomegaly. Laboratory tests found significantly elevated LDH levels; other biochemical parameters and blood count were normal. A colonoscopy was requested, which revealed a friable, ulcerated sessile polypoid lesion (Fig. 1) measuring about 30–40 mm located in the caecum, from which multiple biopsies were taken. It was impossible to pass the scope through the ileocaecal valve, despite several attempts. Based on our findings, we requested a contrast-enhanced thoracoabdominal CT scan (Fig. 2), which showed multiple mediastinal, mesenteric, retroperitoneal and pelvic adenopathies, as well as a large mass extending over the superior retroperitoneal vessels.

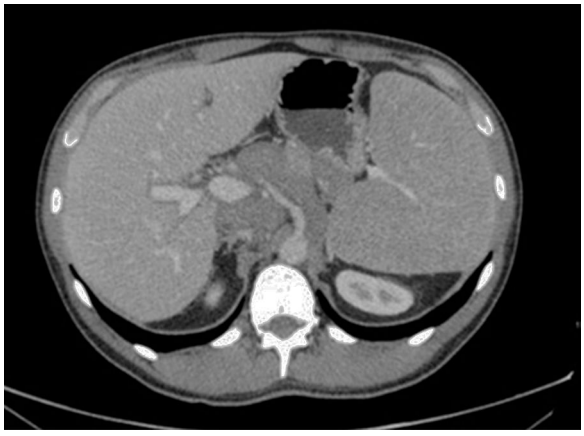

Figure 2 Mass covering the retroperitoneal vessels.

The histological outcome (Fig. 3) of the endoscopic biopsies showed an atypical lymphoid proliferation with strong expression of CD20, CD5 and cyclin D1 in over 75% of B lymphocytes, compatible with infiltration by B-cell non-Hodgkin mantle cell lymphoma. The study was completed with a PET/CT scan that showed supra- and infra-diaphragmatic lymphatic involvement, lymphomatous splenomegaly, diffuse bone marrow and colon involvement, compatible with metabolic stage IV of the disease.

The patient was started on an alternating regimen of 6 cycles of rituximab-cyclophosphamide, vincristine, prednisone (R-macro CHOP)/rituximab-dexamethasone, cisplatin, cytarabine (R-DHAP), with complete response. The 6-month follow-up thoracoabdominal PET/CT scan showed

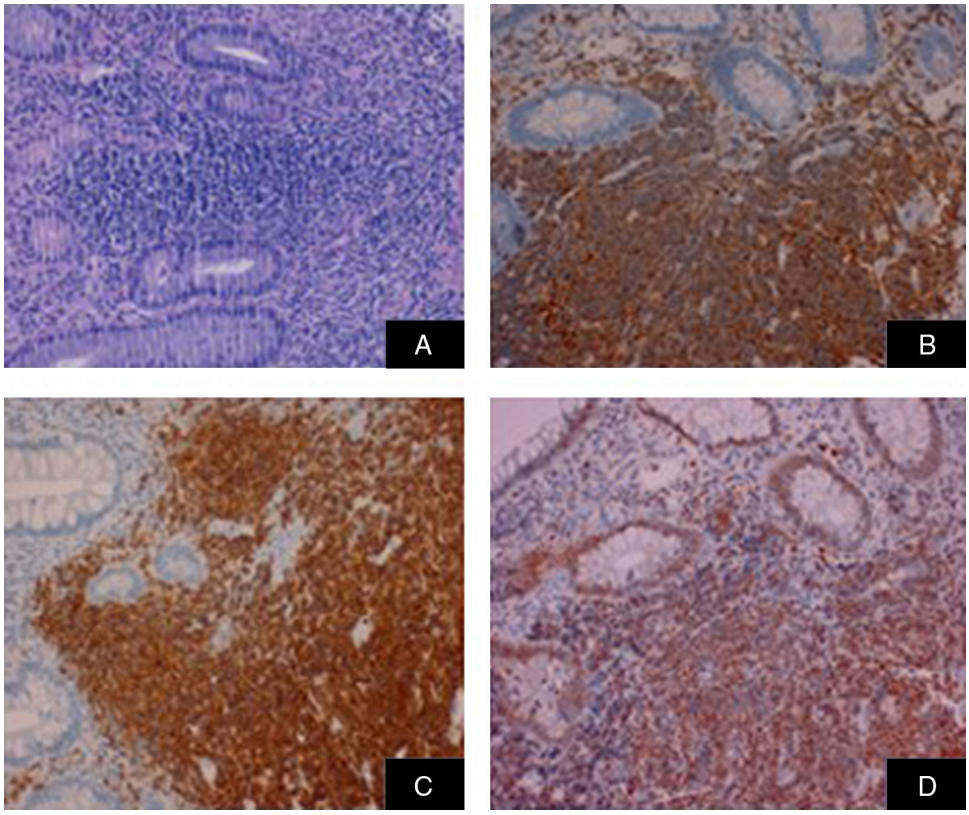

Figure 3 Polyp biopsy: (A) lymphoid population. (B) Immune response positive for CD5. (C) Positive for CD20. (D) Positive for cyclin D1.

no evidence of pathological radiotracer uptake. Follow-up gastroscopy and colonoscopy showed no lesions. Given the patient's good response to intensive therapy, treatment was consolidated with autologous peripheral blood stem cell transplantation (PBSCT). One year after the PBSCT, the patient remains in full remission on maintenance treatment with rituximab.

Our patient is under 60 years of age, with the distinctive characteristic of presenting with a single large polyp instead of multiple lesions. In 90% of patients, symptoms are non-specific, and include: weight loss, asthenia, lethargy, fatigue, anaemia, palpable abdominal or rectal mass and palpable lymphadenopathy. Bone marrow involvement is seen in advanced stages.[7] In the case of gastrointestinal involvement, digestive symptoms have been reported in 15–30% of patients. Endoscopic studies are recommended mainly if there is abdominal pain, changes in bowel movement or rectal bleeding, which was observed in our patient.[6]

Mantle cell lymphoma is considered an aggressive, rapidly progressive type of lymphoma, and 80% of patients are diagnosed at advanced stages.[7] A prognosis must be performed to improve treatment. Prognostic indices permit clinicians to develop treatment strategies based on the patient's particular risk factors. The Mantle Cell Lymphoma International Prognostic Index (MIPI) is used for prognosis in mantle cell lymphoma.[8] In patients under 60 years of age and/or with a high MIPI index score, intensive therapeutic strategies with R-CHOP, R-bendamustine and/or R-DHAP followed by PBSCT are recommended.[9] Our patient had a score of 8 points on the MIPI, which indicates a high risk (estimated average survival of 29 months), and for this reason received intensive therapy with R-macro CHOP/R-DHAP followed by consolidation with PBSCT. He remains in full remission 30 months after the initial chemotherapy treatment.

Prognosis is poorer in older patients with advanced disease at diagnosis, low albumin, splenomegaly, anaemia and elevated LDH levels.[9]

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Catastrophic antiphospholipid antibody syndrome presenting as severe ischaemic colitis☆

Colitis isquémica grave como presentación del síndrome por anticuerpos antifosfolípidos catastrófico

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Antiphospholipid syndrome (APS), or Hughes syndrome, is an acquired autoimmune thrombophilia characterised by venous or arterial thrombosis and/or recurrent miscarriages due to the presence of autoantibodies to phospholipid-binding plasma proteins, such as the lupus anticoagulant, anticardiolipin antibody (aCL), and the anti-beta-2-glycoprotein antibody (aβ2GPI).[1] There are some primary forms that are not associated with another autoimmune disease. Catastrophic antiphospholipid syndrome (CAPS) is a severe and rapidly progressive form that affects multiple organs simultaneously and causes multiple organ failure. Microangiopathy is more commonly found