The definitive pathology result was infiltrating low-grade adenocarcinoma pT4aN2b, so adjuvant chemotherapy was indicated. Currently, the patient is being followed-up in the outpatient clinic and continues with exclusively oral nutrition.

Coloplasty is a surgical alternative after oesophagogastrectomy, although it is associated with considerable morbidity and mortality. Reported complications include: ischemia of the coloplasty, dehiscence of the anastomosis, dysmotility and stenosis.[1,2]

In the literature, there are few cases that describe the development of adenocarcinoma in a coloplasty. It has been suggested that the physiopathology of its appearance is the same as for a colon neoplasm, although some authors propose that the action of the bile reflux on the mucosa of the bowel can favor dysplastic changes. Our group always conducts upper gastrointestinal reconstructions with a Roux-en-Y loop to avoid bile reflux that can be incapacitating. The time until the development of a neoplasm in a coloplasty is very variable and can appear from 3 to 55 years after surgery.[3–6]

As for treatment, surgery should respect oncologic criteria with wide resection margins and lymphadenectomy for correct staging. Adjuvant chemotherapy, in this case for colon cancer, should be administered according to the stage and following the TNM classification, while radiotherapy is not recommendable due to the possibility of radiation enteritis.[1,7,8]

Although to date screening is not necessary for all patients with a coloplasty, we believe it is important to consider this rare diagnosis at the onset of dysphagia.

Authorship

All the authors have contributed to the creation of this article and have read and approved of the manuscript. The requirements for authorship have been met.

Conflict of Interests

The authors declare having no conflict of interests.

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Transient Intestinal Ischemia Caused by Aortic Spasm☆

Isquemia intestinal transitoria por espasmo aórtico

Aortic spasm is an unknown clinical entity about which there is little mention in the international literature. In contrast, however, arterial vasospasms are a relatively frequent condition caused by numerous factors,[1,2] such as autoimmune diseases, drugs and recessive autosomal diseases, but there are no data about it affecting the aorta. It is a very rare clinical condition and difficult to diagnose, which could explain the lack of reports in the literature, together with the fact that

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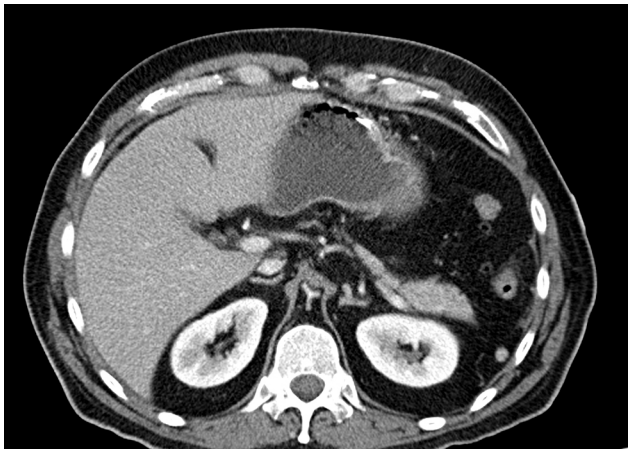

Fig. 1 – Abdominal-pelvic CT scan identifying the aortic spasm.

Fig. 2 – 3D reconstruction of the aorta.

intensive medical treatment can lead to its disappearance, with no imaging studies to confirm the patient's clinical profile.

The patient is a 56-year-old male with no prior medical history of interest who came to the emergency department with sudden-onset abdominal pain together with general decline in his physical status. Blood work showed: leukocytes 19 000 mm^3 (range: 4000–10 000), neutrophils 87% (range: 40–60), lactate dehydrogenase (LDH) 534 IU/L (range: 105–333), C-reactive protein 20 mg/L (normal level: up to 6), pH 7.30 (range: 7.35–7.45); activated partial thromboplastin time (APTT) 75 s (range: 20–40).

Given the suspicion of an acute abdominal process, probably intestinal ischemia, computed tomography (CT) scans with intravenous contrast were ordered of the abdomen and pelvis. Far from identifying signs suggestive of such a process within the arteries that are most frequently affected, complete aortic spasm was observed (Figs. 1 and 2) in a fleur-de-lis pattern that completely occluded the lumen and impeded the passage of contrast material to the superior and inferior mesenteric arteries. No intestinal pneumatosis was found, although there was important dilatation of the small bowel loops. The presumed diagnosis established was mesenteric ischemia due to aortic spasm.

Radiologically, no signs of ischemia were found, although *livedo reticularis* was observed in the lower limbs, while the lab work-up showed elevated serum creatinine (2.5 g/dL), although this could have been due to the toxicity caused by intestinal ischemia.

Given the origin of the entity, we decided to maintain conservative medical treatment with intense serum therapy and vasodilators (nifedipine and captopril). The patient was once again interviewed in order to identify predisposing factors, such as the consumption of associated drugs (ergotamine) or autoimmune diseases, which the patient denied.

After 3 h of intense medical treatment, the symptoms slowly began to recede. The analytical parameters 6 h after the arrival of the patient showed: leucocytes 13 000, neutrophils 855, LDH 460, pH 7.35 and APTT 90%. Other parameters were unaltered. Another abdominal-pelvic CT scan with contrast was ordered, which confirmed the disappearance of the images from the previous scan (Fig. 3). The patient remained in observation for 48 h, during which time the symptoms did not recur, and the patient was discharged. The patient was referred to the Internal Medicine Unit for complete studies and follow-up, although the aetiology was not determined.

Intestinal ischemia is relatively frequent in older patients with predisposing factors. Likewise, there are no reports of intestinal symptoms caused by aortic spasm. Thanks to conservative management, the episode was resolved without the need for surgery, which would have entailed an important risk for morbidity and mortality given the involvement of organs and arteries.

When treating symptoms of intestinal ischemia, one must consider the possibility of autoimmune and/or systemic

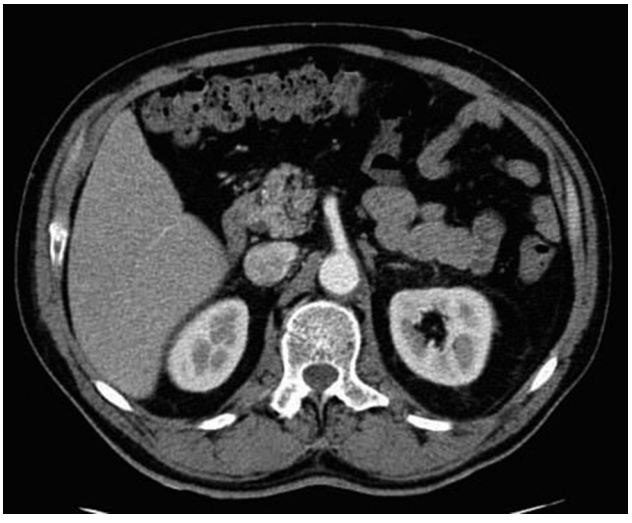

Fig. 3 – Abdominal-pelvic CT scan after the remission of symptoms and normalisation of analytical parameters.

diseases, or rather the use of drugs with associated activity, such as ergotamine or noradrenaline, as triggering factors of the disease. A detailed patient medical history is therefore necessary to be able to rule out any of these processes.

To the best of our knowledge, there have been no reports in the international literature to date about spontaneous aortic spasms with no predisposing factors. This could therefore be considered the first case described in the literature.

Conflict of Interests

The authors have no conflict of interests to declare.

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Tuberculous Periportal Lymphadenitis: A Diagnostic Challenge☆

Linfadenitis periportal tuberculosa: un reto diagnóstico

The existence of a conglomerate of lymph nodes in the periportal region can present in patients with haematological and abdominal neoplasms, or due to infectious causes.[1,2] They usually do not cause symptoms, but they can lead to portal hypertension or obstructive jaundice.[2–5] Diagnosis is complex and can require laparotomy.[2] We present the case of a patient with HIV infection and Burkitt lymphoma who presented with periportal lymphadenopathies and fever caused by tuberculosis (TB).

The patient is a 37-year-old male who underwent surgery for morbid obesity in 2012 at another hospital (gastric band). In April 2014, he was diagnosed with HIV infection and stage IV-B Burkitt lymphoma, which was treated with chemotherapy (R-CODOX-M/R-IVAC) from 24 April to 8 July 2014, which achieved complete remission. In October 2014, he presented with symptoms of fever (up to 39.5 °C) and shivering that had been progressing over the course of 10 days, with no infectious focus. The patient had been treated with cefuroxime and later levofloxacin, which did not resolve the symptoms, so he was hospitalised. Physical examination was normal and no other symptoms were detected. Blood work showed: leukocytes 5700/mm,[3] high percentage of polymorphonuclear cells, prothrombin activity 63% and platelets 84 300 μl. The biochemistry study was normal and the hepatic profile demonstrated levels of GGT 231 U/L and alkaline phosphatase 171 U/L. Abdominal computed tomography (CT) scan revealed a poorly defined lymph node conglomerate in the hepatic hilum, which surrounded the hepatic artery and portal vein. The central area was hypodense and compatible with an area of necrosis. There were also multiple lymphadenopathies in the hepatic hilum, the interaortocaval region and left lateral aortic area (Fig. 1). The blood culture and urine culture series were sterile. The transthoracic echocardiography and ophthalmoscopy were normal. As it was impossible to perform radiology-guided percutaneous needle biopsy due to the location of the lesions, a laparoscopic approach was initially decided upon, but, given the intraoperative findings (increased periportal circulation), a right subcostal laparotomy was done, which revealed multiple large periportal lymph nodes (>2 cm). The largest (3 cm) was compressing the posterior of the left portal vein in the region of the hepatic artery and celiac trunk. There was no ascites, and the liver parenchyma was normal.

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