Spontaneous thrombosis of a gastroduodenal artery pseudoaneurysm manifesting as a subepithelial lesion☆

Trombosis espontánea de un seudoaneurisma de la arteria gastroduodenal manifestado como una lesión subepitelial

A 54-year-old man with a history of heavy alcohol consumption, muscle wasting, and recent hospitalization due to acute alcoholic pancreatitis, presented at the emergency room with epigastric pain and vomiting. Clinical examination and laboratory tests were normal. An upper endoscopy was performed that revealed a 30-mm tubular lesion at the upper edge of the duodenal bulb, covered with normal mucosa, suggestive of a vascular lesion (fig. 1A and B). A computed tomography scan identified a predominantly solid and heterogeneous mass, between the duodenum and pancreatic head, measuring 40 x 15 mm, with indefinite borders, that had no contrast enhancement (fig. 1C and D). Endoscopic ultrasound confirmed the extra-pancreatic location. The color Doppler ultrasound exam was negative for vascular flow, but no other information was obtained (fig. 2A and B). Two weeks later, a magnetic resonance imaging scan described a spontaneous, hyperintense lesion on T1 and T2 in the proximity of the gastroduodenal artery that showed no enhancement after gadolinium administration (figs. 2C and D). The imaging features were suggestive of a thrombosed pseudoaneurysm of the gastroduodenal artery. The patient refused clinical follow-up and continued with heavy alcohol consumption.

Aneurysms of the visceral arteries occur in up to 5% of patients with acute or chronic pancreatitis (gastroduodenal: 10-15%) and are associated with a mortality rate of 25 to 50%.[1,2]

Two types of aneurysms have been described: true aneurysms, which develop when the inflammatory process causes the partial digestion of the arterial wall, and pseudoaneurysms, when pancreatic enzymes from a pseudocyst erode into an adjacent artery leading to bleeding (our case).

The most common clinical presentation is gastrointestinal bleeding secondary to rupture (52%), followed by abdominal pain (46%). The mortality rate in the setting of rupture is about 40% and according to the medical literature, spontaneous thrombosis is one of the most

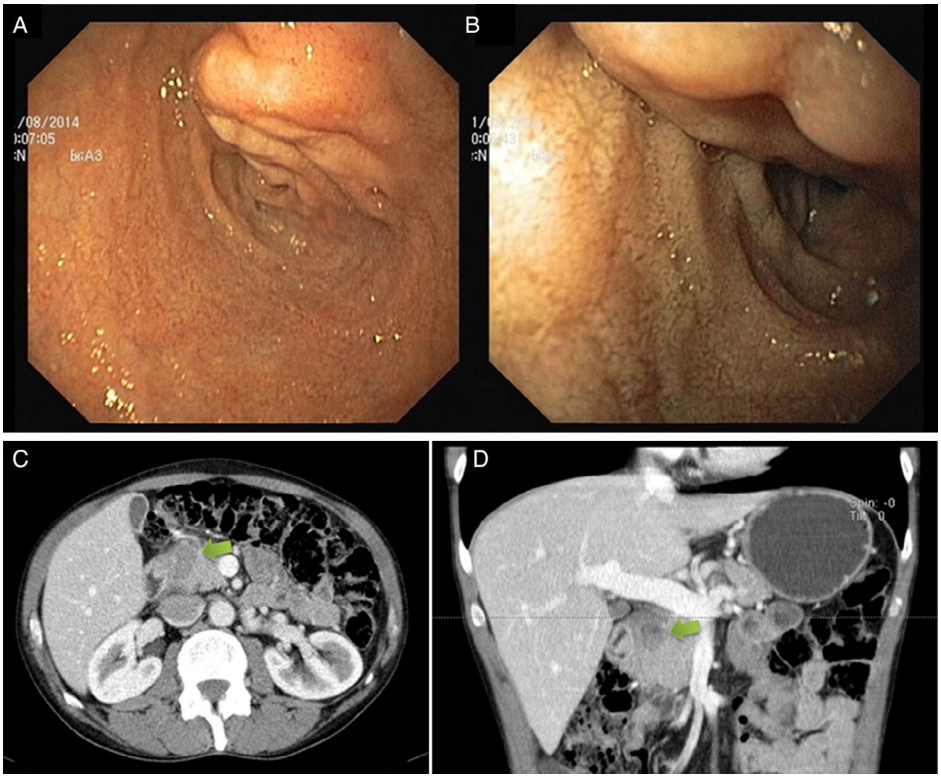

Figure 1 A and B) Upper endoscopy showing a tubular lesion, suggestive of vascular damage, at the upper edge of the duodenal bulb. C and D) CT scan revealing a solid and heterogeneous mass, between the duodenum and pancreatic head, with indefinite borders and no contrast enhancement.

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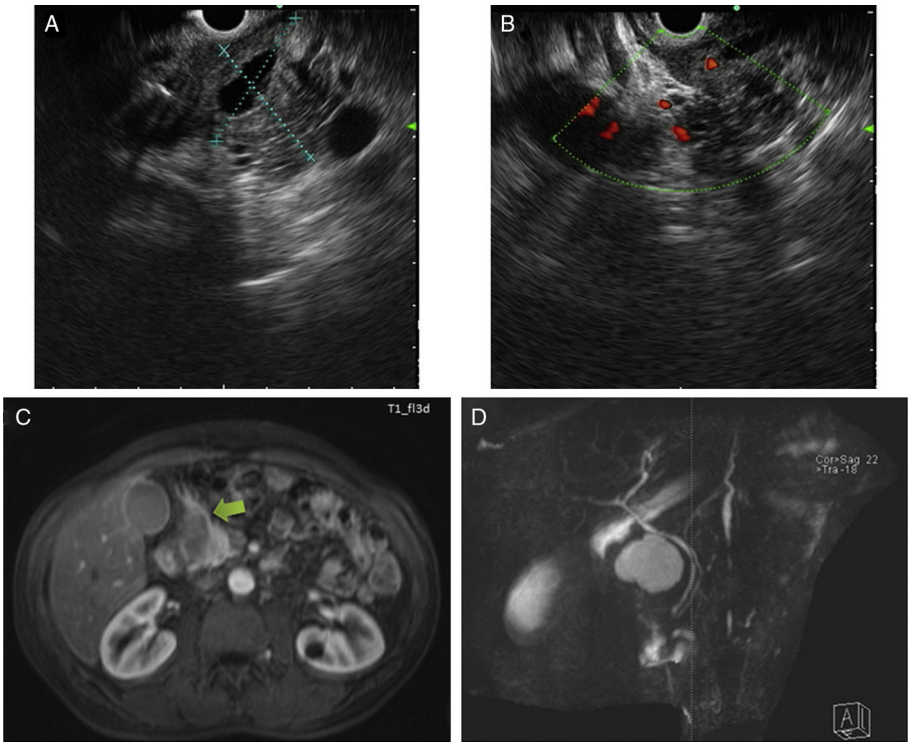

Figure 2 A and B) Endoscopic ultrasound defined a largely hypoechoic lesion, with some anechoic areas and poorly defined limits. Color Doppler ultrasound was negative for vascular flow. C and D) The magnetic resonance imaging scan shows a spontaneous hyperintense lesion on T1 and T2 in the proximity of the gastroduodenal artery, with no enhancement after gadolinium administration. No communication with the common bile duct or pancreatic duct was observed.

infrequent forms of clinical presentation of gastroduodenal artery pseudoaneurysm.[3–5] The gold standard for diagnosis continues to be visceral angiography. The use of alternative methods with good sensitivity, such as computed tomography, magnetic resonance imaging, and endoscopic ultrasound, has already been reported.[3]

Spontaneous thrombosis is an unpredictable event and was previously reported in association with factors that decrease blood flow and increase coagulation, such as hypotension, dehydration, vasospasm, local damage to the arterial wall, and occult malignancies.[4–7] However, given the high mortality rate, physicians should not wait for this to occur. Endovascular repair with coil embolization or stent placement is currently the first line of treatment. Surgery is reserved for patients with failed endovascular repair or that present with hemodynamic instability.[3,8–10]

Ethical responsibilities

Protection of persons and animals. The authors declare that no experiments were performed on humans or animals for this study.

Data confidentiality. The authors declare that they have followed the protocols of their work center in relation to the publication of patient data.

Right to privacy and informed consent. The authors have obtained the informed consent of the patients and/or subjects referred to in the article. This document is in the possession of the corresponding author.

Conflict of interest

The authors declare that there is no conflict of interest.

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Acute appendicitis in a patient with intestinal malrotation☆

Apendicitis aguda en un paciente con malrotación intestinal

Acute appendicitis is the most frequent acute surgical illness in the adult. Typical symptoms include: periumbilical pain that migrates to the right iliac fossa, anorexia, fever, and signs of peritoneal irritation.[1] Due to the variety of appendicular positions, one third of the patients with acute appendicitis have pain that is located outside of the right lower quadrant. Left side pain is rarely observed.[2] In both children and adults, the left cecal appendix can present in situs inversus totalis or in intestinal malrotation.

A 49-year-old man had a history of morbid obesity, laparoscopic gastric band placement, and pulmonary thromboembolism 7 years earlier. He came to the emergency service complaining of 24-h progression of sudden, colicky, generalized abdominal pain predominantly in the left lower quadrant, abdominal distension, nausea, and pasty stools. Physical examination showed him to be afebrile, dehydrated, with important abdominal distension, reduced peristalsis, involuntary muscle resistance, and pain upon palpation and decompression of the left iliac fossa. His biochemical parameters were: leukocytes of 9.7 10^3/ml, neutrophils of 70%, hemoglobin of 17.2 g/dl, hematocrit of 52.7%, and platelets of 449 10^3/ml. Blood chemistry, serum electrolytes, and urinalysis were normal. Initial diagnostic suspicion was diverticular disease. An abdominal computed tomography scan revealed a probable sigmoid volvulus vs colonic malrotation. A barium enema was then carried out that confirmed colonic malrotation (fig. 1). In a new analysis of the tomography scan, acute appendicitis was identified (fig. 2), with a delay in the diagnosis and treatment of approximately 8 hours. Laparoscopic appendectomy was performed, washing the abdominal cavity and placing drains. The findings were perforated appendicitis in the left iliac fossa and generalized purulent peritonitis. The patient's progression was adequate and he was released from the hospital on the 4th postoperative day. The histopathologic study confirmed perforated acute appendicitis.

Whereas appendicitis is the abdominal pathology that most commonly requires surgical intervention, intestinal malrotation in the adult is rare. Gastrointestinal tract rotation and fixation abnormalities are frequently associated with abdominal wall anomalies and diaphragmatic hernia. Filston and Kirks[3] reported an association with lesions, such as atresias and upper gastrointestinal tract stricture, intussusception, and Hirschsprung disease, of up to 62%.[3,4]

Intestinal malrotation is an anatomic variant defined as the lack of rotation or incomplete rotation of the intestine. This is caused by a defective rotation of the middle intestine around the axis of the superior mesenteric artery, between weeks 4-12 of fetal life, and the subsequent abnormal fixation to the parietal peritoneum.[5,6] It is a broad term that encompasses a wide variety of abnormalities of intestinal rotation and fixation. This alteration can be asymptomatic throughout the patient's lifetime or can produce a fatal acute abdomen, if not appropriately diagnosed and treated. The clinical presentation of malrotation is: intermittent colicky pain, vomiting, chronic diarrhea, and malabsorption. In cases of exacerbation of another pathology, the typical symptoms appear, as occurred with our patient.

To understand malrotation it is necessary to be aware of the normal embryonal development of the intestine. During fetal development, the digestive tract is a short and straight tube. This tube then becomes elongated and takes up its orderly and stable arrangement in the peritoneal cavity. This process is known as intestinal rotation and fixation, according to the classic description by Snyder and Chaffin.[7,8]

To better understand the abnormalities of rotation and fixation, they are grouped according to the stage of development in which they were produced, as follows:

- Non-rotation. The small bowel is found in the right part of the abdomen and the colon and cecum in the left part. The distal ileum crosses the midline from right to left to reach the cecum. Our patient had this type of rotation.

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