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Scientific letters

Groove Pancreatitis in the Differential Diagnosis of Pancreatic Adenocarcinoma

Pancreatitis del surco en el diagnóstico diferencial del adenocarcinoma de páncreas

The pancreaticoduodenal groove is a space outlined by the pancreas, duodenum and common bile duct. Pancreatitis is one of the diseases that can affect this anatomical area. It was described for the first time in 1973 by Becker and Bauchspeinchel[1] and, in 1982, Stolte et al. coined the term *groove pancreatitis*. It is a rare entity characterized by chronic segmental[2] pancreatitis and has an uncertain pathogenesis.[3] Its identification is important due to the diagnostic problems that may arise with other serious diseases that affect the head of the pancreas, such as pancreatic cancer.

We present the case of a 42-year-old woman with a history of thrombophilia and a heavy drinking habit who, in August 2011, complained of continuous epigastric abdominal pain radiating to both hypochondria during the previous 5 months. The pain had worsened in the last week and was accompanied by postprandial heaviness, vomiting, intermittent 38 °C fever and weight loss. On examination, the patient showed only mild fever and epigastric tenderness.

The lab work only showed alterations in hemoglobin (11.3 g/dl) and leukocyte (15.000/μl) levels, with elevated PCR. Tumor markers CEA and CA 19.9 were normal.

Abdominal computed tomography (CT) revealed a mass measuring 50 mm×65 mm in the head of the pancreas/uncinate process that seemed to encompass the duodenum, along with increased density of the perilesional fat and retroperitoneal lymphadenopathies. Upper gastrointestinal endoscopy showed no lesions. Endoscopic ultrasound, however, revealed extrinsic compression in the second part of the duodenum with normal mucous membranes and a heterogeneous echogenic mass with irregular edges in the head of the pancreas/uncinate process. Fine-needle aspiration reported an inflammatory process. Magnetic resonance imaging (MRI) confirmed the CT findings. The possibility of an autoimmune process was ruled out, as antibodies (antinuclear, anti-lactoferrin, anti-neutrophil cytoplasmic PR3 and MP0) and immunoglobulin (IgG, IgG4, IgA and IgM) were within normal ranges.

The patient was symptomatic after hospital discharge and, given the suspicion of an inflammatory process, and was monitored with periodical follow-up analyses and radiological studies, which continued to indicate an inflammatory process.

One year after hospitalization, the patient once again presented similar symptoms, with minimal elevation of serum amylase (203 U/l). CT confirmed the previous findings, and the patient was discharged after improvement in the symptoms.

Three months later, an MRI study showed a solid-cystic heterogenous mass measuring 7.6 mm between the head of the pancreas and the duodenum; it was hypointense in sequence T1 and hyperintense in sequence T2, with no contrast uptake. Residual inflammatory changes were also observed in the pancreaticoduodenal space as well as wall thickening of the second part of the duodenum, consistent with focal groove pancreatitis (Figs. 1 and 2).

With the diagnosis of groove pancreatitis, symptomatic treatment and abstinence from alcohol were continued, and the patient remains asymptomatic to date.

Groove pancreatitis is a relatively unknown presentation of chronic pancreatitis, which consists of the appearance of fibrous-scar tissue in the fatty plane of the pancreaticoduodenal groove. It most frequently affects middle-aged men with a history of alcoholism.[4] Two types have been described (pure and segmental) according to whether only the groove is affected or whether the dorso-cranial part of the head of the pancreas is affected as well.[5]

Symptoms include postprandial abdominal pain, vomiting, weight loss and, less frequently, jaundice.[4]

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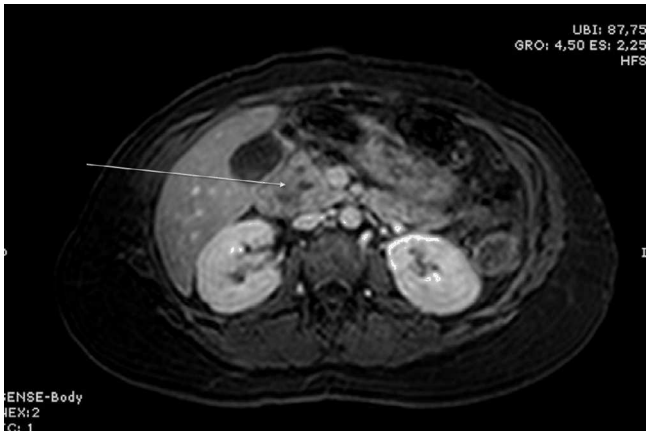

Fig. 1 – MRI image (sequence T1) showing a hypointense cystic lesion in the head of the pancreas.

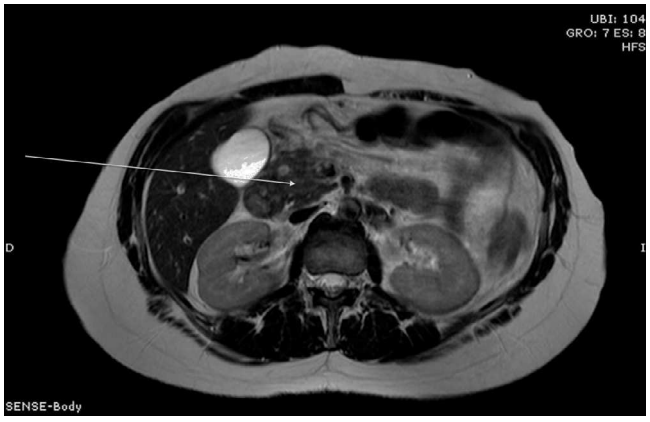

Fig. 2 – MRI image (sequence T2) showing a hyperintense lesion.

The pathogenesis is uncertain, and different causes have been proposed: peptic ulcer, gastric resection, duodenal wall cysts, presence of heterotopic pancreas in the duodenal wall and anatomical variations in the region of the minor papilla, associated with high alcohol consumption, which would increase the density of the pancreatic fluid and its proteins.[3]

Diagnosis is based on clinical suspicion and different diagnostic tests. For many authors, endoscopic ultrasound is the diagnostic test of choice as it has greater sensitivity (86%) and specificity than conventional abdominal ultrasound and biopsies can be taken.[6] Gastroesophageal studies and upper gastrointestinal endoscopy are also useful as they are able to identify duodenal stenosis, and ERCP can display mild stenosis of the main pancreatic duct. CT usually identifies a mass with laminar morphology between the head of the pancreas and the second part of the duodenum that is hypodense with enhancement after administering contrast, although the findings are not completely specific. MRI generally locates a laminar mass in the pancreaticoduodenal groove that is hypointense compared with the pancreatic parenchyma in T1 and isointense or mildly hyperintense in T2, with delayed enhancement after administering gadolinium,[6] as occurred in our case.

According to Gabata et al.,[7] the differential diagnosis between groove pancreatitis and adenocarcinoma cannot be done exclusively with CT and MRI studies. This is especially true if there are no cysts within the mass or in the thickened walls of the duodenum, requiring duodenal biopsy or arteriography. In our case, the diagnosis was based on clinical suspicion after a biopsy taken with endoscopic ultrasound that suggested an inflammatory process. MRI images later confirmed the groove pancreatitis.

In pure forms, the differential diagnosis should be made with cholangiocarcinoma and acute pancreatitis with abscess in the area of the groove. The segmental form requires differential diagnosis with pancreatic adenocarcinoma, which is difficult but of great importance; in some cases, the differential diagnosis is only achieved after pancreaticoduodenectomy.[8]

Conservative therapy based on analgesics, pancreatic rest and abstinence from alcohol are the mainstays of groove pancreatitis treatment. These measures usually succeed initially and must be regularly re-evaluated according to the symptoms, imaging studies and lab determinations.[3,6,8,9] On occasion, the symptoms are resistant to medical treatment and surgical intervention may be required. Pancreaticoduodenectomy is the procedure of choice,[9] and there are case reports of resection of the head of the pancreas with duodenal preservation[10] or even bypass in high-risk patients.[10] Surgery may be necessary if it is not possible to definitively rule out pancreatic cancer.

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Laparoscopic Duodenojejunostomy as a Treatment for Superior Mesenteric Artery Syndrome

Duodenoyeyunostomía laparoscópica como tratamiento del síndrome de la arteria mesentérica superior

Wilkie's syndrome or superior mesenteric artery (SMA) syndrome was first described in 1842 by Rokitanski, although it was Wilkie who published the first series of 75 patients in 1927.[1] It is a rare cause of upper intestinal obstruction caused by compression of the third part of the duodenum between the abdominal aorta and superior mesenteric artery at its origin. We present the case of a patient with this syndrome who had initially been treated conservatively but later required surgical treatment due to persisting symptoms.

The patient is a 29-year-old woman, with no medical or surgical history of interest, who, over the course of the previous 18 months, had presented postprandial fullness and abdominal distension, accompanied by nausea and occasional vomiting.

She came to the Emergency Department due to intolerance to oral intake, epigastric pain and bilious vomiting, which had begun abruptly. She was hemodynamically stable with mild signs of dehydration. Abdominal examination was normal, and the work-up showed normal blood tests. Simple radiography (Fig. 1A) revealed gastric dilatation. Conservative treatment was started with no oral intake, nasogastric suction and fluid therapy. An upper gastrointestinal (GI) series (Fig. 1B) showed that the esophagus, stomach and duodenal bulb had normal characteristics. There was, however, difficult emptying in the third part of the duodenum, which was consistent with functional disorder or SMA. On magnetic resonance imaging (MRI) study, the angle between the SMA and aorta was observed to be 17°.

The patient evolved favorably and a clinical follow-up was scheduled. During the following year, however, the symptoms reappeared with greater intensity. At this stage, surgical treatment was decided on after an upper GI series that confirmed the diagnosis.

The patient was operated on laparoscopically using 4 trocars. The procedure included dissection of the duodenal c-loop with an infra-mesocolic approach and mechanical duodenojejunostomy between the second and third parts of the duodenum and the jejunum, about 25 cm away from the angle of Treitz (Fig. 2A and B) with a 45 mm linear endostapler using 2.5 mm staples (white load). The postoperative period was uneventful, and the patient was discharged on the third day post-op with normal digestive tolerance.

Another upper GI series performed 3 months after the surgical intervention showed that the contrast passed from the normal-sized duodenum to the jejunal loops, with no images of stenosis. Two years after the operation, the patient remains asymptomatic.

The possible causes of SMA syndrome are debilitating disease (AIDS, cancer), eating disorders, postoperative effects (orthopedic surgeries and adhesiolysis due to obstruction of the small intestine), severe trauma and spinal column deformities. Recently, cases have been reported related to rapid weight loss after bariatric surgery.[2]

The incidence of SMA syndrome is 0.2%. It may occur at any age, but is most common in adolescents and young adults, especially women (2:1).[3]

Symptoms are nonspecific and may include postprandial epigastric pain, bloating, early satiety, nausea and vomiting. The pain is alleviated in the knee–chest position and in left lateral decubitus.[4] The causes of death are due to severe electrolyte alterations, gastric perforation, obstructive bezoar or gastric or portal pneumatosis.[3]

Radiologic studies are essential. Historically, upper GI series and arteriography were the standard diagnostic tests, but more recently, CT angiography and MRI have shown greater sensitivity.

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