presence of metastasis and patients' immunodeficiency, the prognosis of these patients seems to be excellent.

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2444-3824/

Tuberculosis, one more consideration in the differential diagnosis of a pancreatic mass☆

Tuberculosis, un diagnóstico más a tener en cuenta ante una masa pancreática

Pancreatic tuberculosis (TB) is rare, especially in immunocompetent patients. Its manifestation as a pancreatic mass represents a diagnostic challenge as it may be falsely identified as a malignancy of the pancreas.[1]

We present the case of a 70-year-old man who attended our hospital following seven days of left abdominal pain, fever, anorexia and asthenia, with deteriorating general condition. His personal history of note included dilated cardiomyopathy and atrial fibrillation, receiving anticoagulant therapy with coumarin derivatives. The patient reported no substance abuse.

During the physical examination, the patient was sweaty, with BP 95/65 mmHg and a temperature of 37.5 °C. The patient experienced pain in the epigastrium and left upper quadrant upon abdominal palpation, with no signs of peritoneal irritation. The chest and abdominal X-rays found no relevant abnormalities. The blood tests revealed altered liver biochemistry levels (AST 114 IU/l, ALT 94 IU/l, ALP 371 IU/l and GGT 877 IU/l). The blood and urine cultures were negative. A computed tomography (CT) scan revealed a cystic mass in the head of the pancreas measuring 5 × 5 cm in diameter, containing septa, as well as retroperitoneal lymphadenopathies and lymphadenopathies at the hepatic hilum and coeliac artery (Fig. 1). Serum amylase, lipase and CA 19-9 levels were normal. An ultrasound-guided percutaneous fine-needle aspiration biopsy (FNAB) was performed. Material that appeared purulent was extracted, which was found to be negative for malignant cells and consistent with an inflammatory process with polymorphonuclear

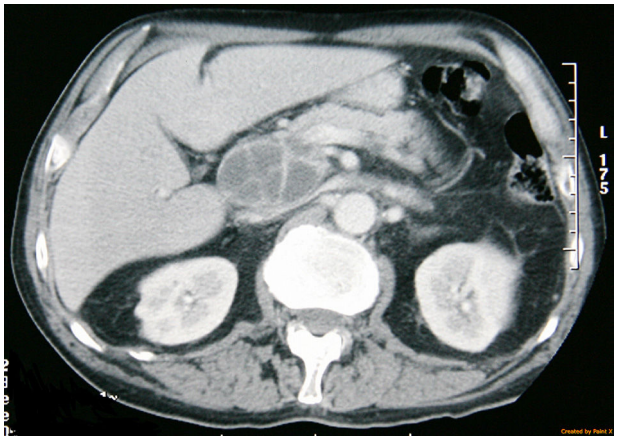

Figure 1 Abdominal CT scan: cystic lesion containing septa and measuring 5 × 5 cm in the head of the pancreas.

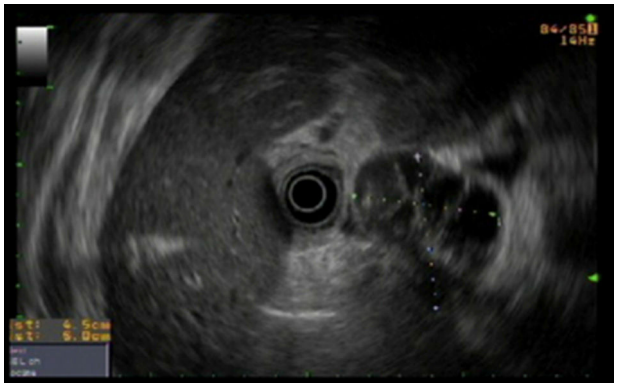

Figure 2 Endoscopic ultrasound: hypoechoic and heterogeneous pancreatic cystic lesion measuring 5 × 4 cm.

☆ Please cite this article as: Mora Cuadrado N, Berroa de la Rosa E, Velayos Jiménez B, Herreros Rodríguez J, Abril Vega CM, de la Serna Higuera C, et al. Tuberculosis, un diagnóstico más a tener en cuenta ante una masa pancreática. Gastroenterol Hepatol. 2017;40:619–621.

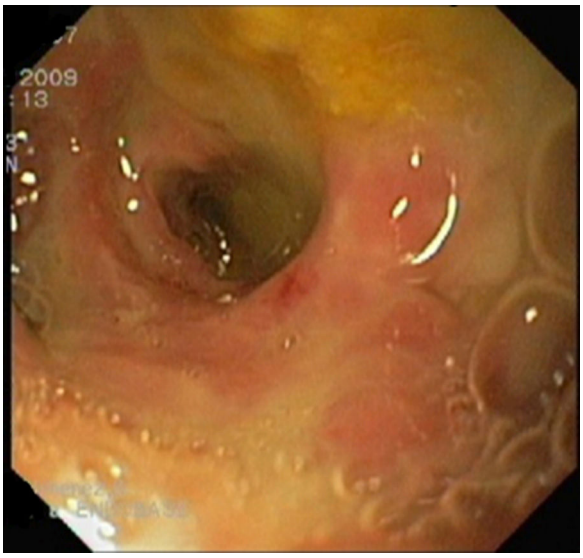

Figure 3 Gastroscopy: the pancreas.

leukocytes and cellular detritus. No bacterial or fungal growth was found.

Despite treatment with imipenem and antipyretics, the clinical course was poor with persistent fever. As a result, it was decided to conduct endoscopic ultrasound-guided drainage. A hypoechoic and heterogeneous mass measuring 5×4 mm was identified adjacent to the wall of the duodenal bulb (Fig. 2) and a round lymphadenopathy measuring 8 mm was found at the hepatic hilum. The mass was punctured from the duodenum and a metallic prosthesis and a nasopancreatic drain were placed.

Given the patient's persistent fever despite modifying antibiotic therapy (tigecycline, ceftazidime, fluconazole) and poor drainage flow rate, the cystic duodenal fistula was reviewed by gastroscopy. The pancreas was accessed, which was found to contain detritus in its walls (Fig. 3), and biopsies were taken. During admission, a round, painful and palpable lymphadenopathy of the left laterocervical region, with a major axis measuring 3 cm, was identified and subsequently resected.

The histological examination of the lymphadenopathy confirmed that it was necrotising granulomatous lymphadenitis. The histological examination of the pancreas biopsies confirmed the presence of necrotic inflammatory tissue. Both examinations found acid/alcohol-fast bacilli (AAFB). Given these findings, antituberculosis treatment with isoniazid, rifampicin and pyrazinamide was prescribed for two months, with rifampicin and isoniazid administered for a further nine months. The patient was asymptomatic by the end of treatment. No abnormalities of the pancreas were found on a CT scan conducted two years later for other reasons.

Pancreatic TB is rare and its isolated manifestation tends to be associated with miliary tuberculosis.[2,3] Abdominal TB accounts for 12% of extrapulmonary TB cases.[4] In order, from most common to least common, it may involve the ileocaecal junction, the peritoneum, the spleen, the liver or the pancreas.[5] It is believed that infection of the pancreas may be caused by lymphatic or haematogenous dissemination, or by reactivation of a previous latent infection.[6] It may manifest as acute or chronic pancreatitis, pancreatic abscess, gastrointestinal bleeding or obstructive jaundice. It may also present as an isolated pancreatic mass mimicking pancreatic carcinoma. Some cases of pancreatic cancer with TB have been reported. This range of different manifestations makes pancreatic TB difficult to diagnose.[3,6,7]

Findings from imaging tests may be consistent with pancreatic TB, but no finding is unique to that disease. As a result, experience and a high level of suspicion are required to establish a definitive diagnosis. Histopathological, cytological or microbiological confirmation is required in all cases. Pancreatic biopsy techniques include ultrasound- or CT-guided percutaneous biopsy, endoscopic ultrasound-guided FNAB or surgical biopsy.[5,7,8] Diagnosis is facilitated by granulomas, epithelioid histiocytes with plasma cells and lymphocytes, and, more rarely, acid/alcohol-fast bacilli. Although the culture is highly specific, it is slow and less sensitive.[8]

Once the diagnosis has been established, antituberculosis treatment should be administered for 6–12 months. The initial phase consists of two months of intensive TB treatment with multiple drugs (rifampicin, isoniazid, pyrazinamide and ethambutol), followed by a continuation phase of rifampicin and isoniazid for at least four months.[9]

In conclusion, pancreatic TB is a rare condition that tends to arise in the context of miliary or disseminated tuberculosis. Like chronic pancreatitis or autoimmune pancreatitis, it may mimic a pancreatic malignancy, making it difficult to diagnose. As such, in patients with a space-occupying lesion of the pancreas and unexplained fever, a diagnosis of pancreatic TB should be considered.

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2444-3824/

Primary follicular lymphoma of the gastrointestinal tract: Endoscopic findings and role of capsule endoscopy☆

Linfoma folicular primario gastrointestinal: hallazgos endoscópicos y papel de la enteroscopia con cápsula

Primary lymphoma of the gastrointestinal tract is rare and is most commonly caused by secondary gastrointestinal invasion. Diffuse large cell lymphoma, MALT lymphoma and mantle cell lymphoma are the most common histological subtypes. Since 2008, the World Health Organisation has recognised a new lymphoma subtype, primary gastrointestinal follicular lymphoma (GI-FL). We present the cases of two patients diagnosed with primary GI-FL at our centre. The first case is a 56-year-old female carrier of an exon 12 mutation in the hMSH-2 gene that was discovered after surgery (hysterectomy with double adnexectomy) for ovarian adenocarcinoma 12 years previously (T1N0M0; TNM classification, 7th edition). Three years ago, she was diagnosed by chance with primary GI-FL of the duodenum after detecting a whitish nodular and elevated lesion close to the major duodenal papilla during a routine gastroscopy (Fig. 1A). The biopsies revealed B-cell lymphoid proliferation in the lamina propria comprising small and irregular lymphocytes and some isolated centroblasts. The immunohistochemical study was positive for the markers CD20, CD10, BCL-2 and BCL-6 and negative for CD3, CD5 and cyclin-D1 (Fig. 1B). The extension study (CT scan of the chest and abdomen, bone marrow biopsy and PET scan) ruled out distant cancer, although three further lesions of similar characteristics were found in the middle and distal jejunum and the terminal ileum during the capsule endoscopy (CE) (Fig. 1C). The patient achieved complete remission after four cycles of treatment with rituximab. A subsequent check-up two years later revealed endoscopic and histological recurrence, but without radiological progression. Given the patient's refusal to receive new treatment, conservative treatment was administered. Twelve months later, the patient remained stable and with no evidence of tumour progression. The second case concerns a 48-year-old woman who was admitted for a six-month history of asthenia, vomiting and abdominal pain caused by menstruation. A conventional endoscopic examination was performed (gastroscopy and ileocolonoscopy), which revealed some polypoid mucosal lesions in the terminal ileum consistent with lymphoid follicular hyperplasia (Fig. 2A). Given the persistent symptoms, an abdominal CT scan was performed, which found thickening of a short segment of the terminal ileum wall as well as regional and retroperitoneal lymphadenopathies, with no other relevant findings (Fig. 2B). The CE confirmed the radiological findings and also revealed a *de novo* mucosal stenosis that was impassable by the capsule (Fig. 2C). After two weeks of treatment with budesonide (9 mg/24 h), the device was expelled naturally without incident. The biopsies obtained previously were restudied, this time observing cell clusters with an immunophenotype consistent with primary GI-FL of the ileum. CHOP chemotherapy + rituximab was initiated, with radiological improvement observed on the control CT scan after two treatment cycles.

Primary GI-FL is a variant of nodal follicular lymphoma.[1] It is a rare lymphoma, accounting for between 1% and 3.6% of all primary lymphomas of the gastrointestinal tract.[2] Prevalence tends to be higher among middle-aged women. Although it is often asymptomatic (43%), the most common symptoms to manifest are abdominal pain (28%), nausea-vomiting (8%) and gastrointestinal bleeding (6%).[3] They tend to be unifocal tumours and most commonly arise in the second part of the duodenum close to the ampulla of Vater. Nevertheless, as was the case with our first patient, multifocal cases of primary GI-FL have been reported. In such cases, capsule endoscopy, which is a harmless and well-tolerated procedure, may be used extremely effectively to detect and locate all synchronous lesions. It can also serve as a guide for biopsy when the conventional endoscopy is negative.[4] Primary GI-FL often presents endoscopically as small polypoid lesions coated in normal mucosa. However, on rare occasions it manifests as ulcers, which can lead to stenosis of the intestinal lumen.[5,6] From a histological standpoint, they present a pattern of nodular growth comprising germinal centres made of centrocytes and centroblasts with an immunophenotype that is typically positive for the markers CD19, CD20, CD22 and CD79a and negative for CD43, CD5 and cyclin-D1.[7] The extension study should include an endoscopic examination, a CT scan of the chest and abdomen and/or PET scan, as well as a bone marrow biopsy.

☆ Please cite this article as: Juanmartiñena Fernández JF, Fernández-Urién I, Iglesias Picazo R, Aznárez Barrio MR, Montes Díaz M, Cebrian García A, et al. Linfoma folicular primario gastrointestinal: hallazgos endoscópicos y papel de la enteroscopia con cápsula. Gastroenterol Hepatol. 2017;40:621–623.