The treatment of choice is percutaneous drainage, guided by ultrasound or CT, along with antibiotic therapy (ampicillin-sulbactam, third-generation cephalosporins or quinolones).[4,8]

In conclusion, the clinical suspicion is essential to direct the study and not delay intravenous antibiotic therapy and percutaneous drainage. Keeping its virulence in mind, when *K. pneumoniae* is confirmed in blood cultures and/or abscess cultures, the capsular serotype should be identified (string test) and radiological signs with the greatest plausibility of spreading haematogenically should be identified, such as thrombophlebitis or gas production.

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Gastric solitary fibrous tumour☆

Tumor fibroso solitario gástrico

A solitary fibrous tumour (SFT) is a rare mesenchymal neoplasm of fibroblastic origin, representing less than 2% of all soft tissue tumours.[1,2] Originally it was described in the pleura, and was initially considered a serous tumour[1]; however, in recent years cases have been documented arising in virtually any anatomical location and organ[3]; currently it is estimated that extrapleural origin is more common.[1,4] We present a case of abdominal SFT, dependent on the minor gastric curvature, without any associated symptoms, which was found casually during an abdominal computed tomography (CT) for another reason.

The case is a 69-year-old male with a history of type II diabetes mellitus, hypertension, ischaemic cardiomyopathy, chronic obstructive pulmonary disease and sleep apnoea syndrome, undergoing treatment with metformin, glimepiride, sitagliptin, acetylsalicylic acid, torasemide, eplerenone, bisoprolol, enalapril and valsartan. He reported symptoms which had lasted several months of diarrhoea, asthenia and weight loss. The physical examination showed no significant abnormalities. A gastroscopy and colonoscopy were performed, with no relevant findings. An abdominal CT scan was carried out, in which a solid, homogeneous mass was observed, 4.3 cm in its largest diameter, located on the gastrohepatic ligament and related to the minor gastric curvature (Fig. 1). With a suspicion of resectable gastrointestinal stromal tumour (GIST), surgery was scheduled. During the surgery a tumour was observed in the minor gastric curvature, protruding towards the anterior fold of the lesser omentum. A laparoscopic resection was conducted, including spindle-shaped excision of the full thickness of the gastric wall without notable incidents. The histological study showed a $6 \times 5 \times 4.5$ cm tumour, well delimited, made up of a homogeneous proliferation of fusiform cells with a

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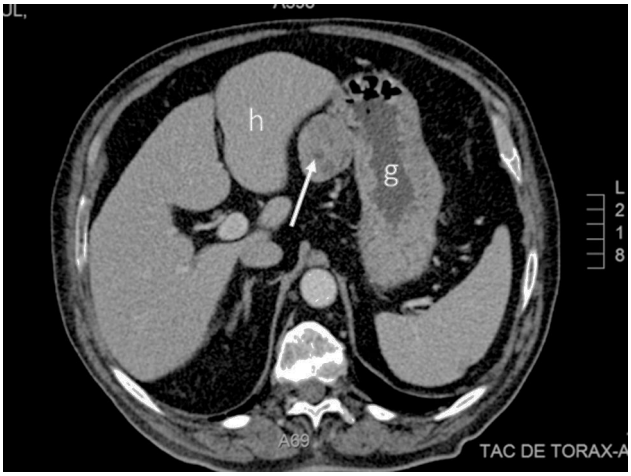

Figure 1 Abdominal CT. Axial section at the level of the upper portion of the abdomen in which a 4 cm solid lesion (arrow) located between the liver (h) and stomach (g) can be observed.

whorled pattern, discrete cellular atypia, occasional mitotic figures, as well as a fibrous stroma and abundant vascular structures with perivascular hyalinisation, consistent with SFT. The resection margins were free of tumour infiltration. The mitotic index was less than 2 mitotic figures/10 high power fields and the proliferative index was low (Ki-67 of 1%). The immunohistochemical study was positive for CD34, vimentin and Bcl-2, and negative for CD99, calretinin, c-Kit, DOG-1, D2-40, S-100, desmin and actin. The post-operative period elapsed without complications.

In the current classification of soft tissue tumours, the term 'SFT' encompasses haemangiopericytomas, as well as SFTs themselves, previously considered independent conditions.[5,6] It is a rare neoplasm, of fibroblastic origin, that affects adults aged 20–70. Histologically, it is characterised by hypo- and hypercellular areas, with fusiform cells surrounded by areas of fibrosis and collagenous stroma and a vascular pattern with branched vessels. The immunohistochemical study tends to be positive for CD34 and, less commonly, for CD99 and Bcl-2.[3,5] It has recently been established that SFT is characterised by the fusion of NAB2 and STAT6 genes of chromosome 12, leading to the overexpression of STAT6, its activation and migration into the cell's nucleus. This change probably constitutes the initial pathogenic mechanism that explains the tumour growth. Also, high diagnostic sensitivity and specificity has been observed in the nuclear positivity of STAT6 in the immunohistochemical study.[3,6]

Although SFT was initially considered a tumour of pleural origin, later reports established that it could take hold in any organ, including the peritoneum, retroperitoneum, liver, lungs, stomach, bladder and prostate, and it can affect head and neck structures such as the parotid, thyroid, orbit and mouth, subcutaneous cellular tissue and, more rarely, skin.[3] Currently it is considered that 60–70% of cases have an extrapleural origin.[4,7] In the most comprehensive series, abdominal location is notable for its frequency, in 30% of cases[4,7]; however, a gastric or gastrohepatic ligament origin is exceptional.[8,9]

SFT is generally a localised and benign tumour, although in 15–20% of cases it exhibits aggressive behaviour leading to local invasion or metastatic disease.[1] The factors that have been associated with this evolution are age, tumour size and the presence of a high mitotic index, cellular atypia, necrosis, haemorrhages or an infiltrative tumour border.[1,4,5] Recently a risk stratification model has been developed based on age, tumour size and mitotic index.[4]

The treatment of choice for SFT is surgical removal. The efficacy of radiotherapy and conventional chemotherapy is very limited. The abundant vascular component of these tumours suggests that anti-angiogenic agents could be of use.[1] Also, understanding the basis of tumourigenesis (NAB2-STAT6 fusion) opens the door to the possibility of new treatments directed at these molecular targets in cases of non-resectable or advanced disease.[3]

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Non-responsive coeliac disease: Coeliac crisis vs. refractory coeliac disease with response to corticosteroids☆

Enfermedad celíaca no respondedora: crisis celíaca vs. enfermedad celíaca refractaria con respuesta a corticoides

Coeliac disease (CD) is an immune-mediated enteropathy, triggered by the ingestion of gluten in genetically predisposed individuals, with various gastrointestinal and systemic manifestations.[1] Most patients respond to a gluten-free diet (GFD), but there is a percentage of patients with non-responsive CD, with the main cause being poor adherence to a GFD.[2] With lesser frequency, the lack of response is due to refractory coeliac disease (RCD).[3] Coeliac crisis (CC) is a rare and potentially fatal complication of CD.[4]

A 63-year-old woman with a history of hypertension and hyperthyroidism, undergoing treatment with carbimazole and torasemide. She was admitted to hospital for chronic diarrhoea with symptoms of severe malnutrition and an organic psychotic mania episode secondary to CD (diagnosis confirmed with intestinal biopsy consistent with Marsh 3a and positive tissue transglutaminase-IgA antibodies [tTG-IgA]: 45 U/ml [normal value: <20 U/ml]), which responded clinically and analytically to a GFD (tTG: 2 U/ml at 2 months after the episode). At 3 months from diagnosis, she was readmitted for disassociated psychosis and severe diarrhoea of rapid progression, with protein-calorie malnutrition, electrolyte imbalances and vitamin deficiencies, despite good adherence to the GFD. Intestinal infections were ruled out (negative faecal cultures and negative viral, bacterial and mycobacterial cultures in intestinal tissue samples). The tTG were determined at admission. They were positive at low titers (25 U/ml) and became negative on the fifth day of admission after GFD (9 U/ml). The genetic test was positive (HLA-DQ2) and the intestinal biopsies showed mucosal atrophy (Marsh 3b in the jejunum and Marsh 3c in the duodenum). During her stay, the patient required blood concentrate transfusions, electrolyte replenishment and parenteral nutrition. After one month without responding to the GFD, the patient developed pneumonia with severe respiratory failure, which was treated with levofloxacin and linezolid, as well as intravenous corticosteroids (methylprednisolone 1 mg/kg), after which she had an excellent response both in the respiratory and digestive symptoms. At discharge, the corticosteroid treatment regimen was tapered off with good response. After one year of follow-up and good adherence to the GFD, the patient was asymptomatic with negative tTG (2 U/ml).

Non-responsive CD is defined as CD that does not respond after 6–12 months on a GFD.[5] Up to 10–20% of patients with CD develop non-responsive CD,[2] with non-adherence to the GFD being the main cause of the lack of response.[3]

tTG values tend to turn negative after a variable amount of time on a GFD. However, they can remain positive in up to 20-30% of patients with RCD despite good adherence to the GFD.[3,6] In our case, the patient had positive tTG values at low titers at admission which could be a false positive or contamination with trace amounts of gluten. The rapid negativisation of said values is notable, given that the antibody levels usually decrease gradually after removing gluten from the diet (half-life of 6–8 weeks).[7] These disparate levels in a short period of time caused confusion in the diagnosis.

Given the suspicion of contamination with trace amounts of gluten, the differential diagnosis of CC could be suggested. CC is a fulminant presentation of CD, with very few cases having been reported in adult patients. It is defined as an acute or rapidly progressing onset of the gastrointestinal symptoms attributable to CD, requiring hospitalisation and/or parenteral nutrition along with at least two objective signs of malnutrition, dehydration or electrolyte imbalance.[4] In this disease, tTG normally have high titers, since the majority of patients who have a CC did not have a prior diagnosis of CD and, therefore, did not follow a GFD, although others may develop it after diagnosis when they do not adequately adhere to a GFD.[4,8] The treatment for CC is a GFD, but some patients require steroids.[4]

On the other hand, in light of the possibility that the initially positive tTG values could have been a false positive, given that they were negative a few days later, a differential diagnosis with RCD was suggested, after ruling out other conditions such as collagenous sprue, tropical sprue and bacterial overgrowth, among others.[9,10]

RCD is defined as the persistence of villous atrophy and clinical malabsorption that do not respond to a GFD. The refractory nature can be primary, if the patient never responded to the GFD, or secondary, if they had an initial response.[5,10] It is a rare condition (1–1.5% of patients with CD).[2] RCD can be classified as type I or type II. Type II RCD is characterised by abnormal T-cells in the intestines and is associated with a higher mortality than type I (56% vs. 7% at 5 years), mainly due to the risk of developing intestinal T-cell lymphoma.[6] With respect to treatment, steroids briefly

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