

Cystic duct obstruction due to gallstones, or exceptionally a tumour, increases the intramural pressure causing mural necrosis and, in the course of time, perforation.^{3,5,9,10} On extremely rare occasions, the gallbladder attaches to the wall and ultimately perforates the subcutaneous tissue. It eventually connects to the skin, giving rise to a CCF.^{8,9}

CCF can present in 2 forms: as a cutaneous orifice that discharges bile, pus or calculi, with no changes in the patient's general condition or few symptoms; or as a subcutaneous tumour in the right hypochondrium, with inflammatory signs, that can be accompanied by impairment of the general condition.⁶

A CCF accompanied by an internal fistula has been reported on only 3 occasions, 2 to the duodenum and 1 to the small intestine.^{6,9} We found no previous cases of cholecystocholedochal fistula (Mirizzi II) and concomitant CCF in our literature search.

The abscess and sometimes the fistula track can be seen on abdominal CT or magnetic resonance imaging.^{1,4,5,8} A fistulography may occasionally be necessary.^{1,2,8} Differential diagnosis should consider infected epidermal cyst, tuberculosis, gangrenous pyoderma and costal osteomyelitis.^{2,10}

Treatment should be adapted to the form of onset and the patient's medical conditions. If the symptoms start as a wall abscess, surgical drainage should be performed and antibiotics administered.^{3,8} Although the CCF closes in around 20% of patients, recurrence is practically universal.^{1,9} Closure is only advisable in very elderly patients and those with severe comorbidities. Deferred treatment consists of cholecystectomy, drainage of the subcutaneous collection and excision of the fistula track.^{4-6,8} Percutaneous cholecystostomy has been used as a bridging treatment between the initial phase and the definitive surgery.⁶

The presence of an orifice or abscess in the right hypochondrium, or more rarely in another location, that drains bile or gallstones should raise the possibility of a CCF. If only pus is observed, the diagnosis is more difficult and fistulography may be useful. Immediate or deferred laparotomic cholecystectomy is usually the most effective treatment.

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A case of sclerosing angiomatoid nodular transformation of the spleen: Imaging and histopathological findings



Un caso de transformación nodular angiomatoide esclerosante del bazo: hallazgos radiológicos e histopatológicos

A 62-year-old woman presented with left upper quadrant pain and anorexia for 1 month. She had a past medical history of nephrolithiasis, extramembranous glomerulonephritis and a relevant atopic background.

Physical examination as well as laboratory tests were unremarkable. Abdominal computed tomography, performed without endovenous contrast due to patient's

atopic history, highlighted a nodular density between the pancreatic tale and splenic hilum. On unenhanced magnetic resonance imaging this corresponded to a vascular structure, next to the spleen, with a "serpentine" shape and apparently in continuity with this organ. Additionally, within the spleen there were three, well circumscribed, macronodular lesions, with lobular borders, the biggest measuring 2.5 cm in greater diameter. These lesions were isointense on T1-weighted sequences and hypointense with mildly hyperintense septa on T2 and FAT-SAT Fiesta sequences (Fig. 1). No other relevant lesions were identified.

Considering the limitations of unenhanced radiological examinations, concerns about malignancy and the potential for splenic vein thrombosis, splenectomy was indicated. Intraoperatively, the splenic vein presented with a tortuous and nodular configuration, which corresponded to the struc-

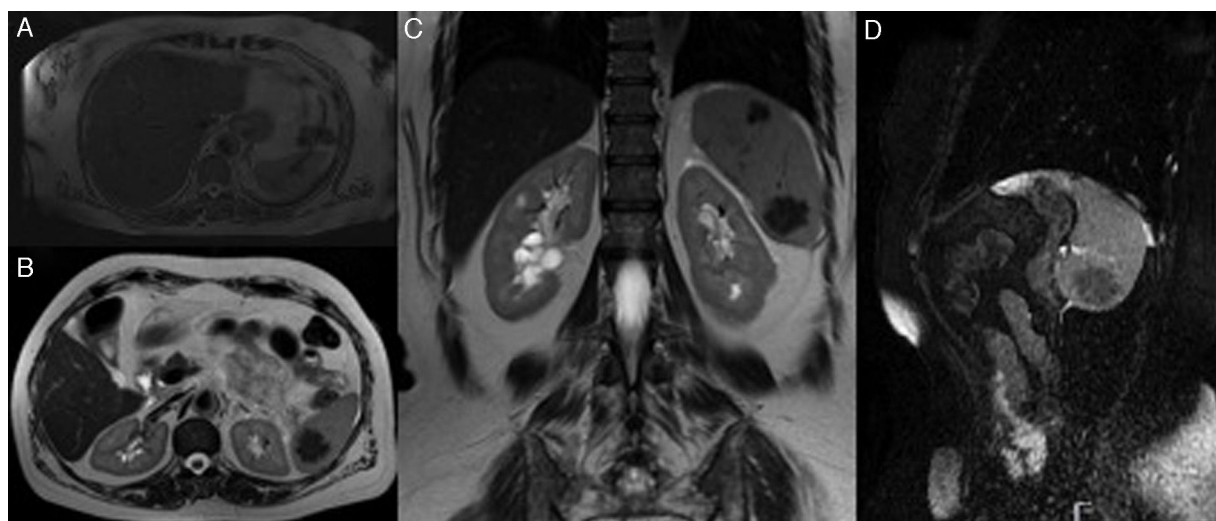


Figure 1 Magnetic resonance imaging. (A) T2-weighted sequences (axial plane) showing a vascular structure in the splenic hilum with a nodular configuration. A macronodular lesion is also seen in this image, within the spleen, exhibiting low signal. (B and C) T2-weighted sequences (axial and coronal plane, respectively) showing nodular lesions, in the upper spleen and the lower spleen, with lobular borders, hypointense, sketching internal micronodular formations. (D) Fiesta FATSAT (sagittal plane) sequence showing a hypointense lesion in the lower spleen, with discrete hyperintense septa.

ture previously described in the splenic hilum. Splenectomy was performed without complications.

The resected spleen weighed 154g and had a round-shaped prominent lesion. On sectioning, the lesion was solid, white in color, with a yellowish center, well-circumscribed,

lobulated and measured 2.5cm in diameter. There were two other similar lesions within the splenic parenchyma (1.2 and 1.8 cm in diameter). Microscopically, each lesion was composed of multiple angiomatoid nodules surrounded by sclerotic tissue. These angiomatoid nodules consisted

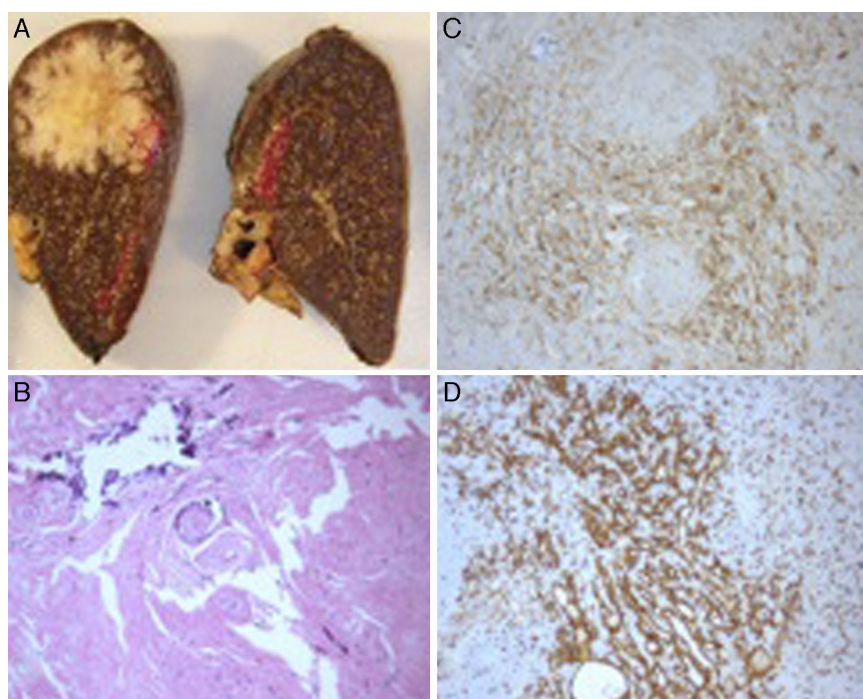


Figure 2 (A) Sectioning images of the resected spleen showing the bigger lesion within splenic parenchyma. On macroscopy, it is a well-circumscribed, round-shaped, lobulated lesion. (B) Microscopy image showing angiomatoid nodules surrounded by a stroma with intense fibrosis, hyalinization and calcifications (hematoxylin and eosin staining, 100 $\times$). (C) Immunohistochemical staining for CD31 showing the abundant vascular structures (capillaries, sinusoids and veins) (100 $\times$). (D) The sinusoids showing strong immunoreactivity for CD8 (100 $\times$).

of capillaries, sinusoids and small veins as evidenced by immunohistochemical staining (CD8, CD34 and CD31 positivity) (Fig. 2). Nuclear atypia, mitotic figures and necrosis were absent. The diagnosis of sclerosing angiomatoid nodular transformation of the spleen (SANT) was established. The patient remained asymptomatic with no recurrence after 10 months of follow-up.

SANT of the spleen is a rare benign vascular lesion, first described by Martel et al.,¹ in 2004, with few more than a 100 cases published in the literature.² It is most commonly encountered in middle-aged adults as an incidental finding on imaging.^{1,3,4} When symptomatic, abdominal pain predominates.^{1,3,4}

According to former studies, there is a slight female preponderance.^{1,3} The pathogenesis of this recently described entity is still unclear.^{1,3} Some authors hypothesize that SANT may represent a peculiar transformation of the red pulp of the spleen in response to an exaggerated stromal proliferation.^{1,3}

The differential diagnosis of SANT includes both benign and malignant vascular lesions such as hemangiomas, hamartomas, lymphangiomas, hemangioendotheliomas, littoral cell angiosarcomas, inflammatory myofibroblastic lesions, angiosarcomas (the commonest nonlymphoid malignant primary tumor of the spleen)⁵ or nodular transformation of the splenic red pulp in response to metastatic carcinoma.⁶

Typical pattern on computed-tomography (CT) and magnetic resonance imaging (MRI) is a well-circumscribed splenic mass with smooth or lobular borders.^{2,5,7} Although more frequently solitary, multiple nodules (as in our case) have been described.^{8,9} It has iso to mild hypodensity compared to surrounding parenchyma on non-enhanced CT.^{2,5,9} For this reason, in our case, it went unnoticed on the first imaging study. On MRI, SANTs most commonly show low to intermediate signal intensity on T1-weighted sequences. On T2-weighted sequences, lesions have typically low signal, in contrast to most differential diagnosis.^{2,5,7} Several authors describe a "spoke-wheel" appearance after contrast administration on CT, MRI and on contrast-enhanced ultrasound that may suggest the diagnosis. This pattern refers to peripheral and septal enhancement with a hypo-enhancing central stellate scar, which correlates to the pathological findings.^{2,5,7,10} However, there are no completely reliable radiological features for the diagnosis of SANT and concerns for malignancy and the potential for splenic rupture often lead to splenectomy.³

In our case, patient's relevant history of atopy made it prudent to perform the investigation with non-enhanced radiological examinations. The findings on MRI namely on T2-weighted and Fiesta FATSAT sequences suggested the diagnosis though not completely reliable. In fact, histopathological characterization appears to remain the diagnostic "gold standard".³ Furthermore, the patient presented with abdominal pain and had a concurrent vascular anomaly in the splenic hilum, which made us to consider the potential for splenic vein thrombosis. At the end, this anomaly corresponded to the splenic vein with a peculiar configuration, not previously described in association with SANT.

Microscopic findings include multiple angiomatoid nodules with a distinctive immunohistochemical profile, in a fibrosclerotic background. These angiomatoid nodules are composed of three types of vessels which

resemble the normal vascular structure of splenic red pulp: the cord capillary-type (CD31+/CD34+/CD8-), the sinusoid-type (CD31+/CD34-/CD8+) and the small vein-type (CD31+/CD34-/CD8-).^{1,3} Other splenic vascular lesions such as hemangioma, hamartoma and littoral cell angiosarcoma lack the nodular pattern of SANT and these mixture of vessels that gives rise to its characteristic immunophenotype.^{1,6} Our case is in accordance with the pathological findings previously described.

Splenectomy is useful and effective without described recurrence after surgery.⁴

The authors report a new case of symptomatic SANT. As more cases are reported a complete characterization of this disease becomes possible. We highlight the imaging and pathological features that may suggest this uncommon lesion and facilitate differential diagnosis and patient's management.

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Conflicts of interest

None declared.

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Acute pancreatitis associated with the intragastric balloon[☆]



Pancreatitis aguda asociada a balón intragástrico

We present the case of a young woman who developed a rare complication secondary to an intragastric balloon, requiring its removal. This has only been previously described once in the literature.¹

The prevalence of overweight is increasing worldwide. At the same time, the intragastric balloon is becoming established as an effective, safe and well-tolerated treatment in patients with morbid obesity, especially in cases in which dietary, pharmacological and behavioural modification therapies have failed, or as a step prior to bariatric surgery.^{2,3} This, together with the fact that weight loss is maintained in almost half of patients 1 year after removing the balloon,^{2,3} has led to an increase in the use of this technique.

A 20-year-old woman with a history of an eating disorder (bulimia nervosa) and implantation of an intragastric balloon 5 months previously came to the emergency department for severe epigastric pain radiating to the left hypochondrium that had commenced the previous day, associated with nausea and vomiting. She denied dietary transgression and alcohol or drug ingestion. On examination, her abdomen was soft and non-tender, with no signs of peritonism. Blood tests revealed: amylase 875 U/L (normal: 28–100), lipase 187 U/L (normal: 16–36), C-reactive protein 28.78 mg/dL (normal: 0–0.5), and leukocytes 18,170/mm³ (normal: 4800–10800) with neutrophilia. The rest of the biochemistry tests (including triglycerides and calcium), full blood count and coagulation were normal.

Abdominal ultrasound found an acalculous gallbladder and head and body of the pancreas with no abnormalities, although the tail could not be visualised due to the presence of the intragastric balloon, which measured around 10 cm. Computed tomography (CT) scan of the abdomen and pelvis without contrast showed an area with decreased uptake in the tail of the pancreas measuring around 23 × 24 mm, with rarefaction of the adjacent fat, consistent with focal pancreatitis of the tail (Balthazar stage C) with less than 30% pancreatic necrosis; the intragastric balloon and mini-

mal free fluid in the pouch of Douglas were also visualised (Figs. 1 and 2). Once other causes of acute pancreatitis, such as congenital malformations, trauma or tumour, had been ruled out by the CT images, the diagnosis of acute pancreatitis secondary to compression of the tail of the pancreas by intragastric balloon was established by exclusion. The patient progressed well with symptomatic treatment, with the abdominal pain receding and laboratory parameters returning to normal levels. On discharge, it was recommended that the balloon be removed in the centre where it had been implanted.

Acute pancreatitis is a common inflammatory process, with an incidence in the United States of around 40 cases per 100,000 persons.⁴ Global mortality in hospitalised patients with acute pancreatitis is approximately 10% (range: 2–22%), reaching 30% in the subset with necrotising acute pancreatitis.⁵

Acute pancreatitis is caused by gallstones and excessive alcohol consumption in 75–85% of cases. Hypertriglyceridaemia is the most common metabolic cause, accounting for 1–4% of cases,^{5,6} followed by hypercalcaemia (in hyper-



Figure 1 Computed tomography image of the abdomen and pelvis, in which an area of low uptake can be seen in the tail of the pancreas with rarefaction of the adjacent fat, consistent with focal pancreatitis (white arrow).

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