

pancreaticoduodenectomy and review of the literature. *J Gastrointest Surg.* 2008;12:1465–8.

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Superior mesenteric artery pseudoaneurysm due to chronic pancreatitis[☆]



Pseudoaneurisma de arteria mesentérica superior por pancreatitis crónica

The formation of visceral artery pseudoaneurysms (VAPAs) as a vascular complication of pancreatitis is a very rare phenomenon. Even more exceptional is the formation of localised pseudoaneurysms in the superior mesenteric artery (SMA).

We report the case of a 58-year-old female patient with a history of chronic alcoholism, hypertension, diabetes mellitus, alcoholic liver cirrhosis, and chronic alcoholic pancreatitis with a 32 × 43 mm pancreatic pseudocyst, according to the last CT scan performed in May 2011. No surgical history.

The patient came to the emergency department due to 6 h of intense epigastric pain, which irradiated to both hypochondria and was accompanied by nausea and vomiting. No fever or accompanying symptoms were reported. An analysis was carried out showing hyperamylasaemia of 2215 U/l, AST/ALT 32/18 U/l, total bilirubin 2.7 mg/dl, leukocytes 21,910 mm³ (91%N, 4%L), Hb 15.1 g/dl, Hct 42.9% and INR 1.32. Initially, it was considered an exacerbation of chronic pancreatitis, and it was decided to admit her to the gastrointestinal department to start treatment and monitoring.

At 24 h after arrival, the patient remained haemodynamically stable, although the intense abdominal pain persisted. In the follow-up analysis, acute anaemisation was observed with Hb 10.7 g/dl and Hct 29.9%. A CTA was performed which showed signs of chronic pancreatitis, chronic liver disease, diffuse ascites (perihepatic, perisplenic, between bowel loops and in the pelvis) and a heterogeneous collection of blood in various stages of evolution with signs of recent bleeding encompassing SMA, which suggests a contained rupture of a pseudoaneurysm of the superior mesenteric artery (PSMA) (Fig. 1).

The case was consulted with the vascular surgery department, and it was decided to perform an arteriogram with

intention to treat with endovascular treatment (EVT) to exclude the PSMA.

Via a left humeral access, the SMA was catheterised using a multipurpose catheter (COOK[®]), and a 6F introducer was placed in the origin of the SMA. A selective arteriogram was performed, showing a large wide-neck pseudoaneurysm with an irregular true lumen of approximately 25 mm. Using a telescoping technique, the aneurysm sac was catheterised with a Progreat[®] (Terumo) microcatheter, which was inserted into the pseudoaneurysm. To prevent the coils from migrating to the SMA, a scaffolding technique was used on the sac, initially embolising it with two controlled-release 25 × 25 mm and 20 × 50 mm type DCS (COOK[®]) J microcoils creating a “cage” and then filling the ball-shaped sac with 15 × 8 mm and 10 × 8 mm DCS spiral microcoils. A control angiograph was performed, showing a contrast image remaining at the entrance to the aneurysm sac. It was assessed whether to continue with this technique, although due to the high risk of arterial lumen invasion by a coil, it was decided to leave the Progreat[®] microcatheter inside the pseudoaneurysm sac and place a coated 5 × 28 mm stent (BeGraft[®]) in the SMA. Then it was embolised with a new 10 × 8 mm DCS spiral coil to finish excluding the pseudoaneurysm.

In verifying the arteriogram, complete exclusion of the aneurysmal sac with SMA permeability was observed and with preserved collaterality except in the area where the stent was placed where there is no contrast of various jejunal branches (Fig. 2).

During her hospital stay, the patient had no complications related to vascular disease, and did not develop anaemia again. She was discharged 20 days after the EVT.

She was later monitored in outpatient consultations via CTA at one and three months after the intervention. No growth or effusions were observed in the aneurysm cavity, the stent remained permeable and there was no stenosis. The patient remained clinically asymptomatic.

In light of the previous case, we can highlight that the incidence of pseudoaneurysms as a complication after pancreatitis is low and not well established. Some reported case series establish a range of incidence of 1.2–14%,¹ with an incidence of 1–6% in acute pancreatitis and a higher incidence of 7–10% in chronic pancreatitis due to its frequent association with pancreatic pseudocysts.²

Different mechanisms have been suggested to explain the formation of pseudoaneurysms. The two most accepted theories are related to: the presence of a pseudocyst that erodes and weakens the wall of an artery adjacent to the pancreas, leading to the formation of a pseudoaneurysm,

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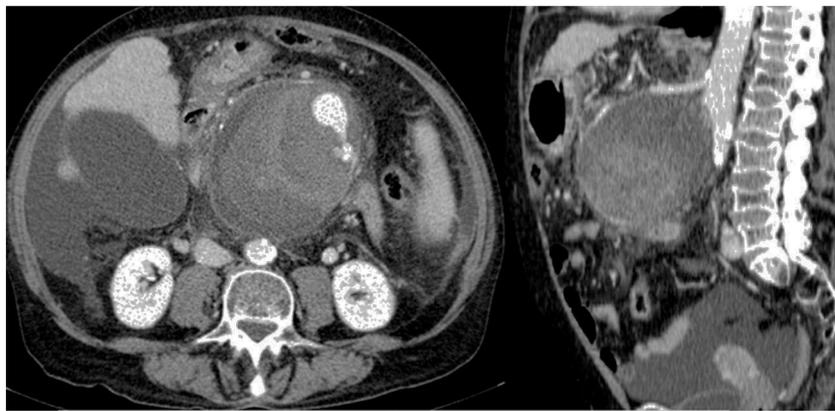


Figure 1 Abdominal CTA: showing the PSMA (102 × 98 × 103 mm [AP × LL × CC]).

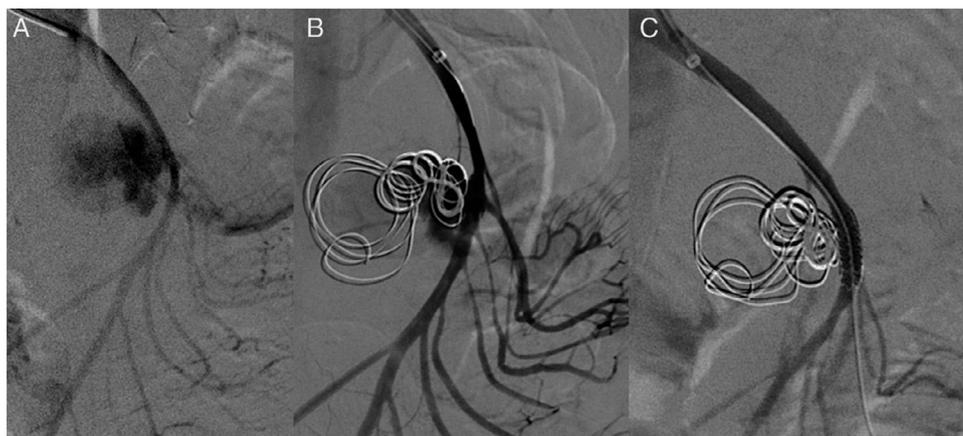


Figure 2 Selective arteriogram: (A) permeability of the PSMA; (B) embolisation of the sac with coils, and (C) placement of the stent.

and the activation of proteolytic enzymes within pancreatic tissue, which causes auto-digestion of pancreatic tissue and of the adjacent structures.^{3,4}

The arteries most affected by proximity to the pancreas are the splenic artery (50%), followed by the gastroduodenal artery (20–50%) and the pancreaticoduodenal arteries (20–30%). The rest of the visceral arteries tend to be affected in 10% of cases.^{1,5}

Some of the factors that increase the risk of vascular complications include necrotising pancreatitis, multiple organ failure, sepsis, abscesses, pseudocysts or pancreatic necrosis and anticoagulant therapy.^{2,6}

Unlike true aneurysms, the majority of VAPAs are symptomatic. Abdominal pain is common and, in the case of rupture, different types of bleeding include: *hemorrhage into the retroperitoneum and abdominal cavity*, among others.⁶ Regarding the diagnosis and study of VAPAs, CTA is the first-choice test, although the gold standard in these cases would be an arteriogram, which allows for endovascular treatment at the same time.^{7,8}

VAPAs always require treatment, independent of their size and the symptoms. Conservative treatment is not recommended due to the high rate of rupture and a mortality of up to 90% in untreated cases. The main objective of

treatment is exclusion of the aneurysm sac.⁹ Classic treatment includes reconstructive surgery or ligation, but due to its high morbidity and mortality (50–100%) it has been replaced in most cases by endovascular treatment, since it is related to a low risk of complications and lower mortality (13–50%).³

The objective of EVT is exclusion of the aneurysm sac via embolisation and/or placement of a stent in the affected artery.⁹ The global technical success is high: 79–100%. However, in 6–55% of cases, more than one procedure is necessary to achieve full exclusion of the pseudoaneurysm.³

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Partial duodenectomy as a therapeutic option in multiple duodenal gastrointestinal stromal tumour associated with neurofibromatosis type 1[☆]



Duodenectomía parcial como opción terapéutica de un tumor del estroma gastrointestinal duodenal múltiple, asociado a neurofibromatosis tipo 1

The incidence of gastrointestinal stromal tumours (GIST) is 10–20/10⁶ inhabitants/year.¹ It is the most common tumour of mesenchymal origin in the digestive tract and constitutes 1–2% of gastrointestinal neoplasms.² The duodenal location is rare, corresponding to 5–7%, behind GISTs in a gastric (50–70%) and small intestinal (20–30%) location, and the association with neurofibromatosis type 1 is infrequent.² The most accepted treatment is surgical resection with free margins, along with imatinib therapy in metastatic cases or recurrent disease and as neoadjuvant therapy for large lesions or complex locations.³ The duodenal location of GISTs is the most complex with relation to surgical treatment, although there are different options based on the duodenal area where the lesion is located and the relationship to adjacent organs.⁴ There are few articles published on this topic, generally isolated cases or short series.^{3–6}

We present the case of a 40-year-old man with neurofibromatosis type 1 undergoing treatment for anaemia of 2 months evolution, and in the last full blood count he presented haemoglobin of 6 mg/dl. The patient was asymptomatic, with the exception of dark faeces. The upper gastrointestinal tract endoscopy (UGIE) of the third part

of the duodenum showed a 3–4 cm ulcerated tumour with a stable adhered clot. No biopsies were taken due to the risk of bleeding. A second UGIE was performed 4 days later, with a pathological anatomy outcome of duodenal ulcer with regenerative changes on the borders. After the abdominal CT scan, which identified a lesion in the third part of the duodenum, without being able to reach a conclusion about the nature thereof, a PET/CT scan was performed (Fig. 1A), diagnosing him with a 4 cm poorly-delimited hypermetabolic duodenal lesion, suggestive of a neoplastic lesion, with no other lesions (Fig. 1B). A surgical intervention was carried out, finding, via visual inspection and palpation, two lesions smaller than 1 cm of neoplastic appearance in the proximal jejunum together with two others (1 × 1 and 4 × 3 cm) on the non-pancreatic side of the third part of the duodenum, with the latter probably causing the symptoms of gastrointestinal bleeding. No other lesions were found in the intestine or stomach. Given the lack of a histological perioperative diagnosis of the lesion and with this being important for the surgical approach to follow, an intraoperative biopsy was performed, reporting a GIST. Since the lesion was located in the third part of the duodenum, it had to be ensured that the opening of the bile duct—located in the second part of the duodenum, next to the resection area—was not injured, therefore a cholecystectomy was performed along with identification of the ampulla of Vater by means of bile duct canalisation with a Fogarty probe via the cystic duct. After identification, a duodenectomy of the third and fourth parts of the duodenum was performed, including the lesions in the contiguous proximal jejunum (Fig. 2A). The intestinal tract was reconstructed via duodenal-jejunal termino-terminal anastomosis and placement of a TachoSil[®] patch (Fig. 2B). The post-operative period proceeded without complications, with discharge after 8 days. The pathological anatomy report showed the presence of a multiple stromal tumour (4 lesions) of low degree of malignancy (<4 cm and <5 mitotic figures per 50 HPF) and positive *c-kit*, receiving no adjuvant oncological treatment. Two months after the surgical intervention, a capsule endoscopy was performed, ruling out the existence of other lesions throughout the digestive tract that may not have been detected intra-operatively. The patient was asymptomatic and had no relapses at 15 months.

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