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SHORT REPORT

Aneurysm of the Pancreaticoduodenal Arteries Associated with a Cœliac Artery Lesion

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Introduction

A ruptured aneurysm of the pancreaticoduodenal arteries without acute or chronic pancreatitis but associated with a median arcuate ligament division is an exceptional event described in only 11 cases. The case of a ruptured pancreaticoduodenal artery aneurysm, associated with a cœliac artery lesion which we describe, illustrates the difficulty in diagnosing these rare events promptly and in instituting urgent treatment to arrest the bleeding followed by an elective procedure to prevent recurrence.

Case Report

A 54-year-old man with no history of vascular disease was admitted to a district hospital for investigation of vague abdominal pain mainly affecting the right abdomen, hypotension corrected by infusion of crystalloid and no fever. Laboratory blood chemical findings including a normal hemoglobin, raised leukocyte count and high C-reactive protein concentration. This presentation raised the suspicion of a gall bladder infection and the patient was kept under close observation overnight. The next day, hypotension developed and the patient complained of pain in the right iliac quadrant. An abdominal ultrasound scan showed a large iliac fluid collection, but no lesions involving the gall bladder or liver. Appendicitis was diagnosed and the patient underwent a McBurney operation. During surgery blood was found in the abdomen. An exploratory laparotomy revealed a large retroperitoneal hematoma. The patient was transferred to our vascular surgery unit. A CT scan after contrast injection revealed an intact retroperitoneal hematoma ($16 \times 9 \times 15$ cm), with no bleeding from the aorta or the visceral arteries, and a median arcuate ligament division that compressed the origin of the cœliac trunk. Because these findings suggested a ruptured pancreaticoduodenal artery aneurysm arteriography was planned to confirm the diagnosis and treat the aneurysm by embolization. The patient, who was by now haemodynamically stable, was kept under observation in the ITU and transferred to the vascular surgical unit. On day 1, a CT scan showed that the hematoma had enlarged. The patient was kept under surveillance in the vascular unit and arteriography was planned for the following day. During the night, the patient collapsed but responded to more IV crystalloid and was immediately transferred to the radiological unit. While the patient was being prepared for arteriography, a new CT scan showed the hematoma had now increased in size and had spread to the intraperitoneal space, filling the peri-hepatic and peri-splenic areas as well as the pelvis.

The patient underwent selective arteriography to visualize the stenosis caused by compression of the cœliac axis, to localize the bleeding pancreaticoduodenal artery aneurysm and to proceed to treatment by embolization. Under local anesthesia, a 5-F introducer

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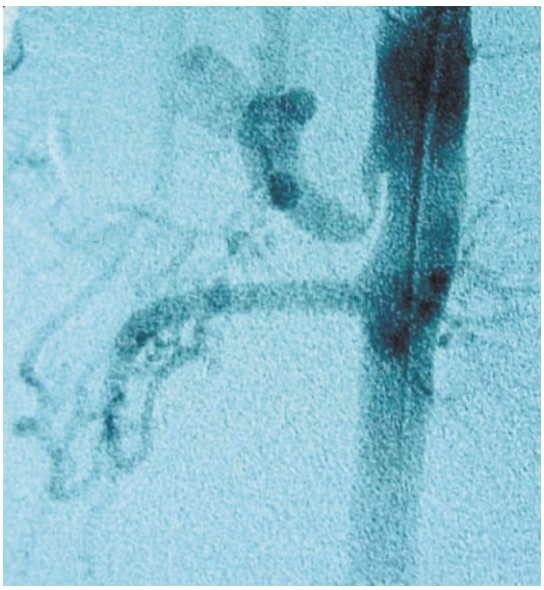

Fig. 1. Aortic flush arteriography showing stenosis of the cœliac trunk and a dense collateral arterial network connecting the superior mesenteric artery to the cœliac trunk.

was placed and a 4-F pig-tail catheter was inserted into the aorta. The first contrast injection revealed a tight stenosis involving the cœliac trunk (Fig. 1), and a dense network of collateral vessels connecting the superior mesenteric artery (SMA) to the cœliac trunk. Selective SMA catheterization showed the anterior and posterior pancreaticoduodenal arcades from the gastroduodenal artery. On the anterior arcade there was an aneurysm smaller than 6 mm. On the posterior arcade, there was an aneurysmal malformation (Fig. 2(a)) with a contrast leak (Fig. 2(b)). The distal part of this malformation was embolized with two coils (Cook-MREY Embolization coil®: IMWCE-35-5-8 and IMWCE 35-5-5). The proximal part of the malformation was then embolized with a single coil (Fig. 3). These maneuvers achieved complete thrombosis of the malformation and the posterior pancreaticoduodenal arcade while preserving the gastroduodenal artery. The patient had an uneventful postoperative course. A CT follow-up scan on day 6 showed a stable non-bleeding hematoma. Follow-up scans at 3 and 4 months showed that the hematoma had regressed. Six months after the original operation the patient underwent surgery to decompress the cœliac axis stenosis. Through a sub-umbilical laparotomy approach, the cœliac trunk was decompressed by sectioning the large left pillar of the arcuate ligament. Palpation showed normal blood flow into the cœliac axis with satisfactory pulsation. Arteriography on postoperative day 3 confirmed that the cœliac axis stenosis initially observed had regressed, and the aneurysmal malformation on the anterior pancreaticoduodenal arcade had disappeared (Fig. 4). No contrast leaks were visible nor were there signs of a recurrent pancreaticoduodenal artery aneurysm. Short-term and mid-term follow-up was uneventful.

Discussion

The first case of a pancreaticoduodenal artery

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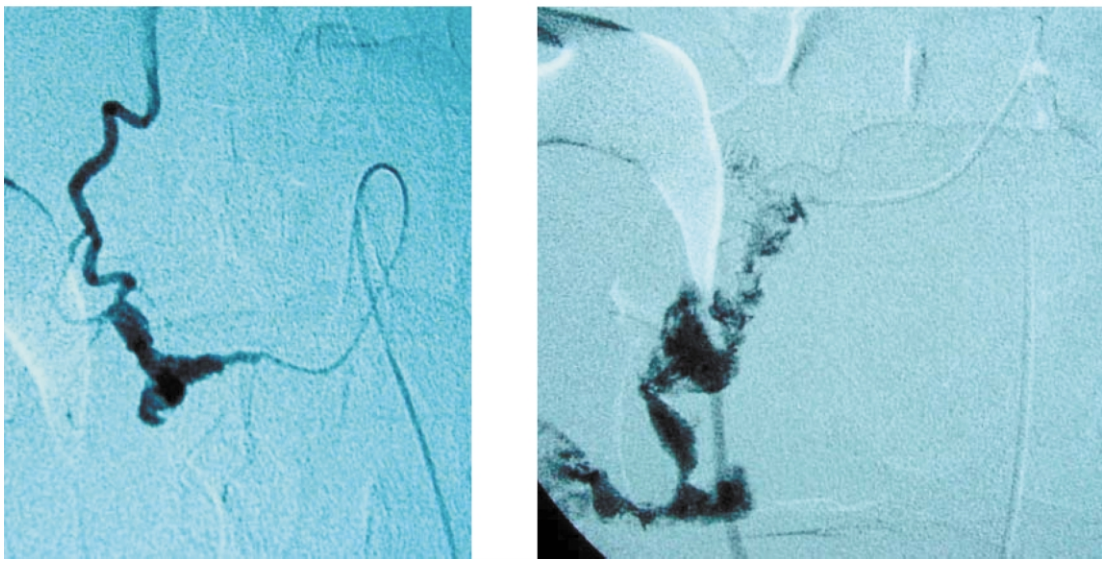

Fig. 2. Selective catheterization of the posterior arcade showing an arterial malformation (a) with contrast leak (b).

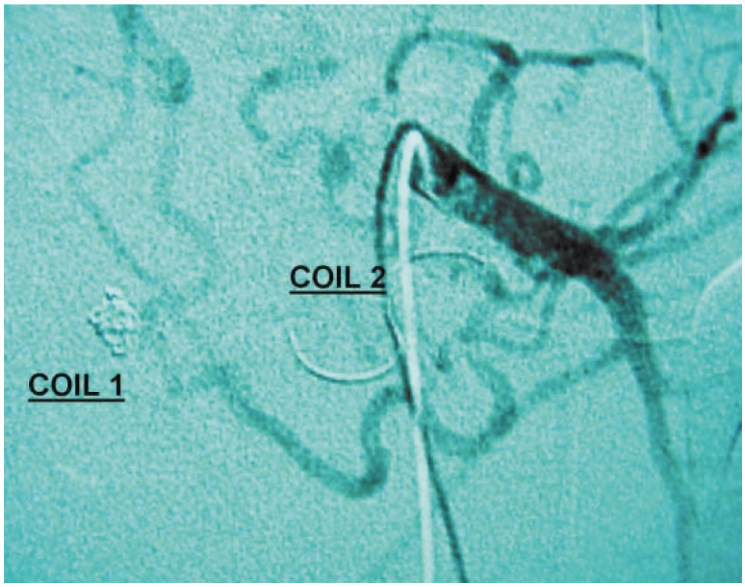

Fig. 3. Complete thrombosis of the malformation (coil 1: accumulation of two coils) and the posterior pancreaticoduodenal artery after embolization of the proximal part (coil 2) and preservation of the gastroduodenal artery.

aneurysm was reported in 1895 by Ferguson.[1] True aneurysms are especially rare and often hard to distinguish from false aneurysms (principally observed during acute or chronic pancreatitis). Since Sutton in 1973 described a patient with a true aneurysm of the pancreaticoduodenal artery associated with a cœliac trunk lesion, a cœliac lesion is acknowledged as a major cause for the development of an aneurysm of the pancreaticoduodenal artery.[2] This association varies from 68%[3] to 74%.[4] To explain the association of a pancreaticoduodenal artery aneurysm with a cœliac artery lesion, Sutton originally proposed that the increased blood flow in the peripancreatic arterial network provided collateral supply for revascularization of the cœliac trunk thus dilating the vascular walls until an aneurysm developed.[2] The frequency for rupture varies from 52%[3] to 69%.[4] Most ruptured aneurysms manifest clinically with non-specific abdominal pains and in a few cases an acute abdominal syndrome associated with bleeding into the peritoneal cavity, and ultimately hemorrhagic collapse. They usually rupture into the retroperitoneal space around the pancreas. More rarely, if treatment is delayed, as happened in our case, the aneurysm may ultimately rupture into the peritoneal cavity.[5,6]

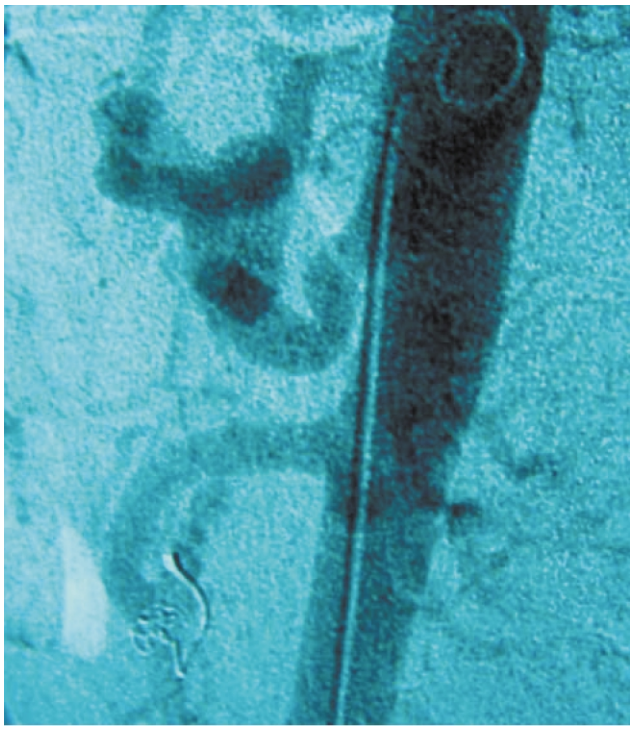

Fig. 4. Post procedural arteriography after section of the median arcuate ligament revealing the regression of the cœliac axis stenosis initially observed (Fig. 1).

As our case report shows, arteriography must be done without delay in a patient with a bleeding ruptured pancreaticoduodenal artery aneurysm. The investigation should begin with an aortic flush to identify the culprit lesion. Selective catheterization of the SMA will then reveal the collateral arterial network revascularizing the cœliac branches, locate the aneurysm and identify the number of lesions. This is followed by immediate radiological embolization of the aneurysm and its feeding artery. In our patient, these procedures (Figs. 1–3) confirmed the diagnosis and guided management avoiding recourse to surgery. In patients whose pancreaticoduodenal artery

aneurysms are caused by a lesion of the cœliac trunk, good management depends on resolving the lesion surgically and preventing recurrence. In our patient we did this by a simple section of the median arcuate ligament thus resolving the hemodynamic pressures responsible for the aneurysm.

Although a ruptured aneurysm of the pancreaticoduodenal arteries associated with a lesion of the cœliac trunk is a rare event, it still requires prompt management. Our case report suggests immediate arteriography to confirm the etiology, establish the diagnosis, and allow non-surgical treatment using embolization. Patients with stenosis of the cœliac trunk caused by median arcuate ligament compression must then undergo elective surgical decompression to prevent the risk of recurrent aneurysm.

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