

Thrombosis of splenic artery pseudoaneurysm complicating pancreatitis

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Abstract

The natural history of pseudoaneurysms complicating pancreatitis is unknown. A patient with chronic pancreatitis is described in whom thrombosis of a splenic artery pseudoaneurysm occurred. Early diagnosis and radical treatment of a bleeding pseudoaneurysm are mandatory. When elective treatment is considered, however, contrast enhanced computed tomography may be useful just before surgery as thrombosis may occur.

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Arterial pseudoaneurysms are classic complications of pancreatitis, especially when pseudocysts are present.¹⁻⁵ Bleeding pseudoaneurysms may be life threatening and require early diagnosis and radical treatment.

We report on a case of thrombosis of a splenic artery pseudoaneurysm complicating chronic pancreatitis.

Case report

A 43 year old man with several years' history of alcohol abuse and with chronic pancreatitis confirmed at endoscopic retrograde pancreatography presented in October 1990 for recrudescient epigastralgia. Clinical examination was normal. Laboratory tests showed an inflamma-

tory syndrome/erythrocyte sedimentation rate: 91 mm/h (normal <15), fibrinogen 7.44 g/l (normal 1.80-4.00), and C reactive protein 120 mg/l (normal <7); a mild hyperleucocytosis was seen at $12.6 \times 10^9/l$ (normal 4-10). Pancreatic enzymes were of normal values.

A computed tomography examination showed a pseudocyst, 3 cm in diameter, in the pancreatic head and several pseudocysts located behind the body of the pancreas; a dilatation of the main pancreatic duct was seen in the tail. Endoscopic retrograde pancreatography showed irregular dilatation of the main pancreatic duct in the tail and opacification of three pseudocysts, one in the head and two in the body of the pancreas. The patient stopped any alcohol intake but his pain worsened despite analgesics.

Three weeks later, a second CT examination was performed. It showed an eccentric contrast enhancing mass, 1.5 cm in diameter, within one of the corporeal pseudocysts in which density was increased (Fig 1).

A coeliac and splenic angiography showed a postostial pseudoaneurysm of the splenic artery without active bleeding (Fig 2). Stable catheterisation of the splenic artery near the ostium was not possible, precluding transcatheter embolisation of the feeding artery. Selective catheterisation of the pseudoaneurysm itself was not attempted because of the risk of rupture. Somatostatin infusion (Somatostatine UCB 250 µg/h) was given to reduce pancreatic secretion and elective surgical intervention was decided on for the seventh day.

Computed tomography, performed the day before planned surgery, showed a spontaneous hyperdensity and a lack of contrast enhancement of the pseudoaneurysm, suggesting recent thrombosis (Fig 3). A coeliac angiography confirmed the thrombosis of the pseudoaneurysm and the permeability of the splenic artery (Fig 4).

Computed tomography performed two months and one year later showed regression of the clotted pseudoaneurysm and progressive disappearance of the pseudocysts.

Discussion

Arterial pseudoaneurysms are not uncommon with acute or, more often, chronic pancreatitis especially when pseudocysts are present.¹⁻⁵ Pseudoaneurysms are caused by enzymatic digestion^{6,7} or local compression of the vessel by the pseudocyst.³ Because of its proximity to the pancreas, the splenic artery is the most commonly affected vessel.^{3,4,6} Visceral pseudoaneurysms are difficult to diagnose. They may be detected with computed tomography and duplex sonography, but selective angiography is still the diagnostic examination of choice.^{8,9}

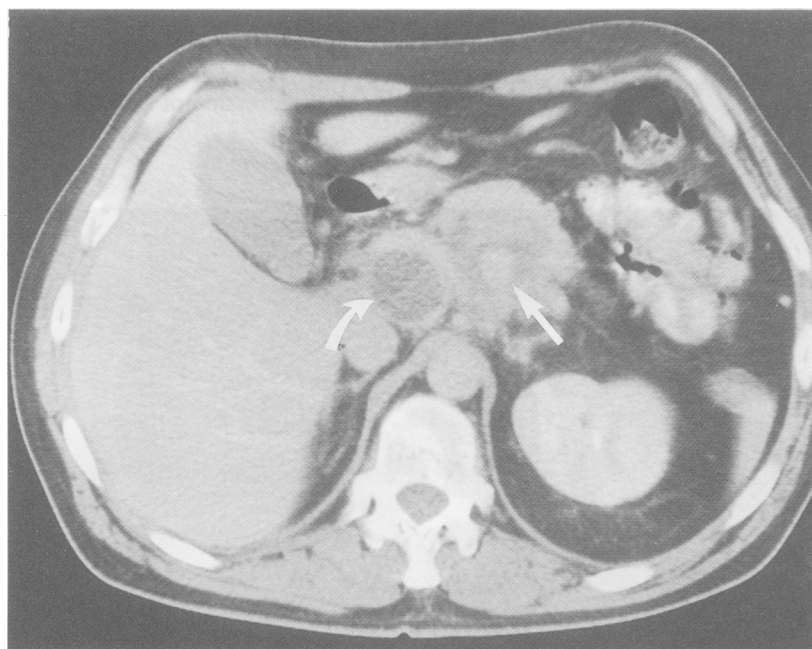


Figure 1: Computed tomography shows an eccentric contrast enhancing lesion, 1.5 cm in diameter (straight arrow) within a retrocorporeal pseudocyst where density is increased. Another pseudocyst is seen above the pancreatic head (curved arrow).

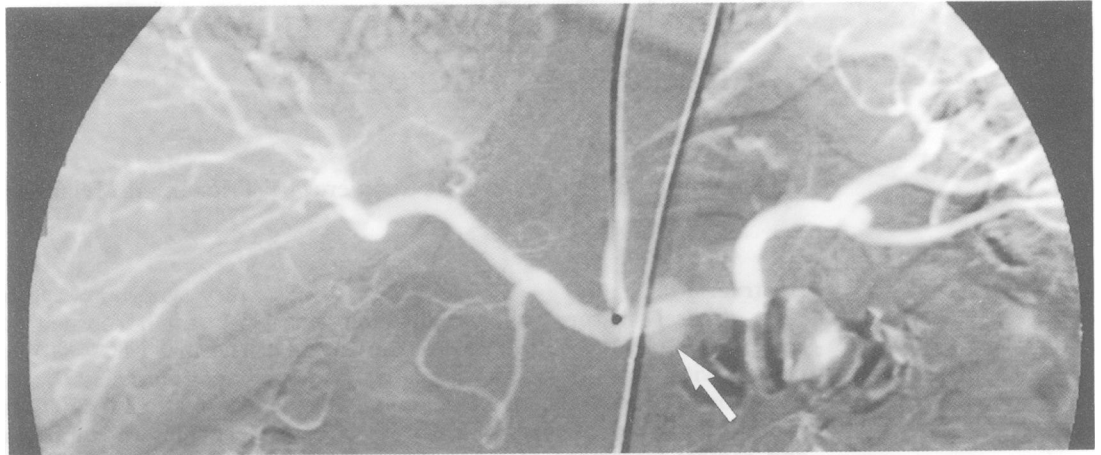


Figure 2: Coeliac arteriogram shows a postostial pseudoaneurysm of the splenic artery (arrow).

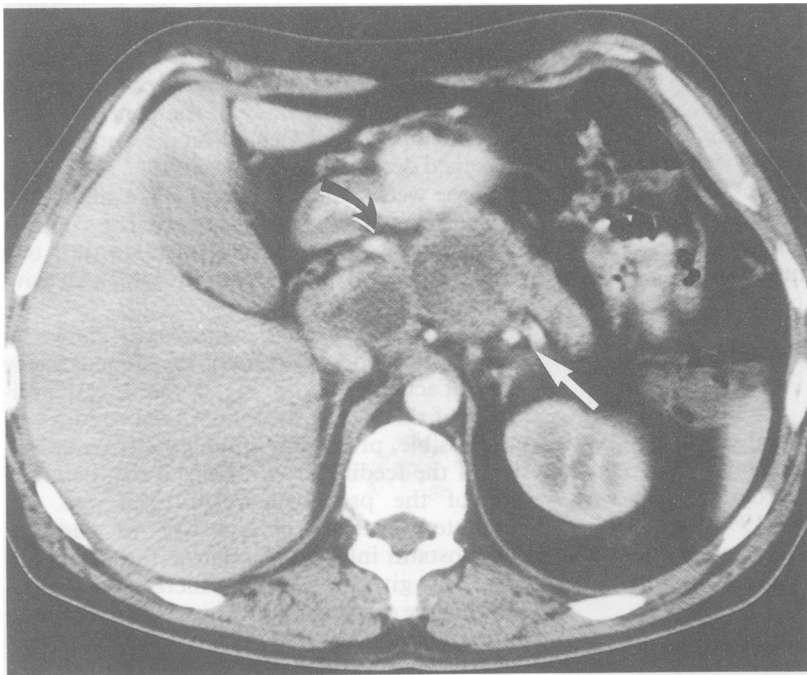


Figure 3: Computed tomography performed nine days later shows a lack of contrast enhancement of the pseudoaneurysm, suggesting thrombosis. Notice the high enhancement of the aorta, the splenic artery (white arrow), and the hepatic artery (black arrow).

The main risk of visceral pseudoaneurysm is rupture and consecutive bleeding. This risk is much higher in pseudoaneurysms secondary to pancreatitis.^{10,11} Bleeding can occur directly or through the pseudocyst, if present. This may cause secondary rupture in the main pancreatic duct, the peritoneum, the retroperitoneum or more often in an adjacent viscus such as duodenum, stomach or transverse colon.^{1,3,6,12,13} Early diagnosis and radical treatment are thus mandatory. Surgery is the classic treatment of pseudoaneurysm but transcatheter arterial embolisation seems to be a very attractive alternative either to permit further elective surgery or as a definitive treatment with good results and a lower mortality than surgery.^{3,5,7}

The incidence and natural history of pseudoaneurysms complicating chronic pancreatitis is unknown. At angiography performed in patients with chronic pancreatitis without gastrointestinal bleeding pseudoaneurysms have been seen in 10–21% of cases^{3,6,7,14} but only 1–8% of gastrointestinal bleedings in patients with pancreatic pseudocysts are thought to be pseudoaneurysm related.³ These data suggest that not all pseudoaneurysms will lead to gastrointestinal haemorrhage.³ Spontaneous regression of small visceral



Figure 4: Coeliac arteriogram confirms the lack of enhancement of the clotted pseudoaneurysm. (Arrows point to reflux of contrast material in the aorta.)

pseudoaneurysms has been reported, including pseudoaneurysms of the splenic artery.¹⁵ Several authors have thus recommended a conservative approach for asymptomatic patients with visceral pseudoaneurysms smaller than 2.5 cm in diameter, except for women of child bearing age.^{10,16}

Spontaneous early thrombosis of pseudoaneurysms secondary to pancreatitis has not been described, however, except in one case affecting the inferior pancreaticoduodenal artery.¹¹ Pseudoaneurysms complicating pancreatitis are thought to be the most life threatening of all splanchnic artery aneurysms with death rate greater than 50%.¹⁰

We have no clear explanation for the thrombosis of the pseudoaneurysm in our case. It may be related to the catheterisation of the coeliac trunk and the splenic artery. Trauma during catheterisation seems unlikely, however, because the splenic artery remained permeable on follow up studies and catheterisation of the pseudoaneurysm was not performed.

Somatostatin is known to reduce splanchnic blood flow without modifying systemic arterial blood pressure.¹⁷ The mechanism of action is still debated.¹⁸ Some authors have suggested that somatostatin may act by a direct vasoconstrictive effect on the splanchnic arterioles or may lower intestinal vasodilating hormones and glucagon.¹⁷ It has been shown that somatostatin has no effect on haemostasis, and particularly on platelet aggregation, with clinical doses of 250 and 500 µg/h in non-diabetic subjects; only with a higher dose (750 µg/h) were circulating platelet aggregates detected.¹⁹ The role of somatostatin, infused at a rate of 250 µg/h, in our patient remains thus hypothetical.

In conclusion, early diagnosis and radical treatment of symptomatic peripancreatic pseudoaneurysms are mandatory. When transcatheter embolisation is unsuccessful, however, and in the absence of acute bleeding that requires emergency surgery, a contrast enhanced computed tomography just before elective surgery may be useful as thrombosis may occur. Further

investigations about somatostatin in this field could also be of interest.

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