

Pseudoaneurysm of the celiac trunk following acute pancreatitis. Case report

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Background. Visceral artery aneurysms (VAA) are well-known complication of pancreatitis. Splenic artery is the most common localisation, but other peripancreatic vessels may also be affected. Although VAA may develop palpable epigastric mass, bleeding and pain, they are often fully asymptomatic, being incidentally picked up on abdominal US, CT or angiography for other reasons.

Case report. The authors report a case of a 38-year-old male with pseudoaneurysm of celiac trunk following an acute pancreatitis. The complex cystic-solid epigastric mass was initially detected by grey-scale US, and its vascular nature was suspected on colour-Doppler US scan. Precise localisation was determined by angiography.

Conclusions. Colour-Doppler US is a reliable diagnostic method for detection of VAA, but hardly identifies the vessel of origin in many patients. Angiography is fundamental for the final diagnosis, followed by immobilisation in selected cases. Celiac axis always has to be kept in mind as a rare possible localisation of VAA.

Key words: pancreatitis - complications; celiac trunk; aneurysm, false diagnosis

Introduction

The most common visceral artery aneurysm (VAA) is the splenic artery aneurysm (SAA), but other splanchnic vessels also may be affected by dilation.^{1,2} The dilation which does not affect all layers of the vessel wall is called pseudoaneurysm (PSAN). Blunt trauma of

the upper abdomen and enzymatic injury to the vessel wall in pancreatitis are the most common causes of PSAN. Vessel rupture causes the formation of haematoma, consecutively surrounded by fibrous capsule.³ Bleeding into the pseudocysts can also result in PSAN formation. The incidence of PSAN in patients with severe acute pancreatitis is up to 10%, and the affection of the celiac trunk is relatively rare.³⁻⁵

Although it may be asymptomatic, the patients with VAA often suffer from the pain in the upper abdomen, or have gastrointestinal or peritoneal bleeding.⁴ Rupture is a serious complication seen in up to 37% of SAA, re-

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sulting in high mortality. Celiac trunk aneurysms (CTA) have the lowest rupture incidence among all localisations of VAA.²

The presence of VAA is mostly suspected on grey-scale ultrasound (US) scan, while colour- Doppler ultrasound (CDUS) successfully indicates the vascular nature of the mass. Angiography is fundamental for confirming the diagnosis and exact localisation of VAA. CT and MRI allow good analysis of localisation and morphology of the aneurysm. Pseudocyst and other peripancreatic fluid collections, renal artery aneurysm and abdominal aortic aneurysm must be considered in differential diagnosis of VAA.

Transcatheter embolisation of VAA has to be performed immediately after diagnostic selective angiography. It may obviate the need for surgery in high-operative-risk-patients or be lifesaving in the case of aneurysm rupture.⁶

Case report

A 38-year-old male, without the history of alcohol abuse, was admitted on 1st October 2001, because of constant, localised epigastric pain that increased on palpation. He suffered from nausea and vomited black content. Ten days earlier he experienced similar symptoms and had melaena. On admittance he was afebrile, with *laboratory tests* indicating acute pancreatitis, slight anaemia and marked hypertriglyceridaemia. *Chest X-ray* and *plain abdominal film* were normal. *Abdominal US* demonstrated inhomogeneous, hypoechoic, well-defined oval fluid-filled mass, 20 mm in diameter, near the neck of the pancreas. A jet of blood entering the mass was clearly visible on CDUS; hence, the presumptive diagnosis of SAA was established. The pancreas was moderately enlarged, inhomogeneous and hypoechoic. The enlarged hyperechoic liver (diffuse lesion) with the slightly dilated hepatic veins, widened portal vein

(15 mm), slightly enlarged spleen (130 mm) and normal appearance of other abdominal organs were shown.

Five days later, *abdominal US* showed 53×51 mm cystic-solid oval mass near the pancreatic head, left to the midline. The central part of the lesion was anechoic (30×22 mm) with posterior sound enhancement (Figure 1), while the periphery was hypoechoic. Turbulent flow within the central part of the mass was seen at CDUS (Figure 2a). *Pulsed-Doppler* demonstrated bi-directional («to-an-for») flow in the central part (Figure 2b). The communication with the splenic artery was thought to exist; hence, the diagnosis of SAA with mural thrombus was presumed. US examinations were performed using Hewlett-Packard Image Point scanner (Andover, Massachusetts, USA) with a convex, 3.5 MHz probe. The conservative treatment of acute pancreatitis was recommended, and the *immobilisation* of suspected SAA was to be performed. *Liver biopsy* done in pre-interventional work-up revealed advanced cirrhosis. The patient had not alcohol abuse in his history, but was anti-HAV positive.

Celiac trunk angiography was undertaken on 7th November 2000, with the intention of



Figure 1. Grey-scale US of the upper abdomen (left parasagittal oblique scan) reveals a well-defined, 53×51 mm inhomogeneous (complex) mass, near the pancreatic head, posterior to the stomach, with anechoic central part, and crescent-like hypoechoic part, peripherally. Posterior sound enhancement was remarkable. (Presumptive diagnosis at this moment seen in comment).

aneurysm immobilisation. Film series revealed the dilatation of celiac trunk, with its greatest diameter three times larger than at its aortic orifice. Extraluminal contrast deposit at the left side of celiac axis, with 20 mm in diameter, was observed. Faint crescent-like contrast filling was also seen caudally (Figure 3), corresponding to anechoic clefts at the periphery of the mass seen at grey-scale US (Figure 1). Slight compression and dislocation of the proximal splenic artery from below indicated the presence of a mass greater than that delineated with contrast filling. In the celiac trunk-splenic artery corner, partially thrombosed CTA was suspected. The embolisation was not performed because of the risk for rupture of the dilated celiac trunk.



Figure 2a. CDUS consistently showed turbulent flow in both directions within the central part of the mass, coded *blue* and *red*.

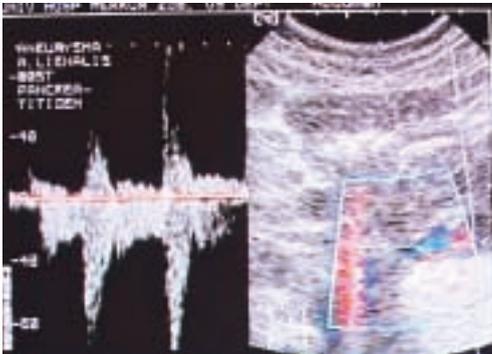


Figure 2b. Pulsed-Doppler demonstrated bi-directional flow in aneurysmal neck and cavity; the communication with splenic artery was initially thought to exist.



Figure 3a. Celiac angiogram revealed extraluminal contrast deposit, 2 cm in diameter, left to markedly dilated celiac trunk; the splenic artery was tortuous and dilated with extrinsic compression of proximal part from below; the diagnosis of partially thrombosed CTA was presumed.

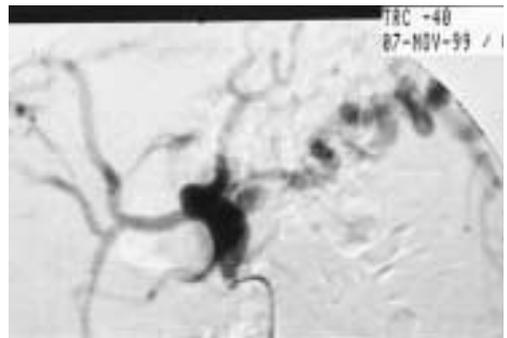


Figure 3b. Later film showed clearly that aneurysm was thrombosed in its greater part; the central part filled with blood exhibited colour signal at CDUS (s. Figure 2), peripheral crescent-like blood filling in the caudal part corresponded to anechoic clefts at the periphery (s. Figure 1), and caused no Doppler signal because of slow flow.

The findings were additionally confirmed by *magnetic resonance imaging (MRI)*, which showed partial thrombosis of CTA, and tortuous and dilated splenic artery compressed with CTA.

Operative findings

Five months after the onset of pancreatitis the patient was operated on. A firm mass in the upper abdomen, on the left of the mid-

line, was found at surgery. Partially thrombosed PSAN, measuring 70×63 mm, originating from celiac trunk, was impressing into the lesser sac, being intimately adherent to the pancreatic head. The splenic artery was elongated, tortuous and coiled, which was considered as congenital variant. The resection of the aneurysm with ligation of the celiac axis ostium and splenic artery were performed. Splenectomy was also done; the spleen was slightly enlarged and congested. After ten postoperative days of recovery, the patient was dismissed from hospital. Unfortunately, the patient died of injuries of car accident 3 months later.

Discussion

Splenic artery aneurysms (SAA) account for up to 60% of all VAA, but hepatic artery (20%), pancreaticoduodenal arcade (17%), superior mesenteric artery (5%), inferior mesenteric artery (3%), gastroepiploic and gastric artery (4%) and celiac trunk (4%) may also be affected.^{1,2} The most common cause of PSAN formation is pancreatitis. Activated and released pancreatic enzymes cause the rupture of membrana elastica interna of the splanchnic vessels, followed by segmental thrombosis of the vessel. Thrombosis of the vasa vasorum causes ischemia and nutrient deficit to the arterial wall with its necrosis. Massive bleeding occurs if weakened vessel wall suddenly ruptures, most probably with catastrophic outcome. PSAN develops when the rupture contains the haematoma that is surrounded by reactive fibrous capsule.⁷

The patients with VAA are often (72%) asymptomatic, or may suffer from the pain in the upper abdomen, or have signs of gastrointestinal bleeding and anaemia⁴ as was also the case in our patient who had sparse symptoms, relatively unspecific for PSAN. Haematemesis and melaena in his history might be caused also by associated peptic ul-

ceration or portal hypertension. In spite of a large mass growing near the head of the pancreas, our patient did not have jaundice or dilatation of biliary tree or pancreatic duct.

Diagnosis - imaging modalities: Typical grey-scale US features of PSAN include anechoic or hypoechoic, heterogeneous mass with distal sound enhancement, possibly with hyper-echoic margins.^{8,9} This presentation lacks specificity and mismatch with pancreatic pseudocyst has to be avoided. Pulsations of the mass may indicate the correct diagnosis, even when CDUS is not available. In our case, US morphology was less typical because the lesion was predominantly solid, with thrombosed lumen, and relatively small cavity filled with blood. Rapid enlargement of the mass was, however, suggestive of a vascular lesion.

Blood flow in VAA is usually easily detectable,^{10,11} but even scrutinise Doppler analysis may be unreliable in obese individuals with deeply located lesions, in patients having pains on probe contact or in thin patients with marked aortic pulsations. A faint blood jet is sometimes detectable only with power Doppler, but one has to beware of false positive results due to motion-artefacts. In patients with pseudocyst, normal flow in one of peripancreatic arteries may occasionally be mistaken for extravasation into the pseudocyst or peritoneal space. A swirling jet of blood entering the aneurysm (Figure 2) was clearly visible with CDUS in our case. As the lesion was very close to the splenic artery, it was initially falsely diagnosed as SAA. In spite of careful topographic analysis, we were unable to determine exactly the origin of aneurysm without coeliacography. This, however, was not a great shortage because the patient management in that moment would not be significantly influenced with this finding.

CT was not done in our case as we considered that no additional valuable information would be acquired from this modality that carries the risk of contrast medium administration and radiation. As the patient was ex-

amed by US under good conditions (no bowel gas, thin patient), it was considered reliable. Typical CT finding of VAA includes well-defined mass with hyperdense centre that shows contrast enhancement, and less dense periphery corresponding to mural clot and fibrous wall. If CT reveals high density within peripancreatic collection of near to water density, the finding should raise the suspicion of haemorrhage into the pseudocyst that may be of similar CT appearance as PSAN.

Due to the possibilities of multiplanar imaging, signal void phenomenon and contrast medium use, MRI is of great value in diagnostic work-up of unclear abdominal vascular masses. Flow in the lesion can be detected even without contrast medium.¹² In our case, the diagnosis of CTA was established prior to MRI examination, which was done on patient's insistence in a private clinic. Partial thrombosis of CTA and compression of the splenic artery were additionally confirmed, but no additional data were yielded that were not known prior to MRI.

Angiography remains the most fundamental modality in the diagnosis of VAA. It can exactly determine the origin of aneurysm, but may lack to predict its real size when partial thrombosis is present. We did not consider the possibility of CTA prior to angiography as we were impressed by the compressive effect of the lesion onto the splenic artery; at this moment, we concluded to deal with SAA. The analysis of several projections in different angles led to better visualisation of celiac axis, and establishing the correct diagnosis. It can never be overemphasised how the precise preoperative detection of bleeding source is desirable in the case of VAA, as its identification on laparotomy may be exceedingly difficult. In selected cases the risk of urgent surgery to control haemorrhage may be obviated by immobilisation.

Unfortunately, in many cases aneurysms remain undiagnosed and the patients' initial

presentation will be acute haemorrhage. Endoscopy is of value to eliminate other causes of gastrointestinal bleeding, especially in patients with alcohol abuse. In rare cases of rupture of an aneurysm in pancreatic duct, presented with recurrent haematemesis or melaena, endoscopy may reveal »haemosuccus pancreaticus« or wirsungorrhagia.¹³

We conclude that PSAN of peripancreatic vessels have to be taken into account as possible complication in each patient with pancreatitis or pseudocyst, and although splenic artery is the most common site of PSAN, other peripancreatic vessels including *celiac trunk* have to be considered. VAA of the upper abdomen may be easily visualised by grey-scale US, and the vascular aetiology confirmed with CDUS as non-invasive and cheap modality. Angiography has to be done to determine precisely the vessel involved. CT and MRI are not obligatory adjunct to US and angiography in patients with VAA, but may help in topographic analysis or detection of concomitant pathology or complications.

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