Pancreatic Pseudocyst Eroding Into the Splenoportal Venous Confluence and Mimicking an Arterial Aneurysm

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Abbreviations: CT, computed tomography; HU, Hounsfield unit; MRI, magnetic resonance imaging

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Abstract

We report the case of a 62-year-old man with chronic pancreatitis who presented with increasing abdominal pain. Sonography, magnetic resonance imaging, contrast-enhanced computed tomography, and ultimately catheter angiography demonstrated a pancreatic pseudocyst that had eroded into the splenoportal venous confluence, mimicking an arterial aneurysm. The diagnostic was confirmed at the time of surgical treatment. This case demonstrates the use of imaging to diagnose complications of pancreatitis, and the difficulty of distinguishing an eroding pseudocyst from an arterial aneurysm.

Introduction

Pancreatic pseudocysts are a well-known complication of both acute and chronic pancreatitis, with a higher incidence in the latter. Another serious consequence that may follow this inflammatory process is hemorrhagic pancreatitis, which may cause aneurysmal changes of the peripancreatic vessels. Many imaging modalities can be used to diagnose pancreatitis and its complications. However the differential diagnosis between pancreatic pseudocysts and vascular aneurysmal changes is mandatory as these conditions have different treatments. The diagnosis is challenging and difficult particularly when pseudocyst produces erosion of the nearby vessels. We present a case of pathologically proven pancreatic pseudocyst eroding the confluence between portal vein and splenic vein and associated with vascular leakage.

Case Report

A 62-year-old man presented with recurrent attacks of vague upper abdominal pain, which had increased in severity over a 2-week period. In addition, the he had suffered an acute attack of pancreatitis 15 years previous to this presentation, which was managed conservatively, but the condition then was complicated by insulin dependant type II diabetes mellitus. For the current episode, the patient sought medical advice, and was admitted to our hospital for further clinical assessment. The patient had a past medical history hypertension (medication controlled), and alcoholism with associated fatty liver disease. Laboratory investigations revealed serum amylase of subnormal level (were 18 U/L, Normal level: 23 to 85U/L). No definite diagnosis is reached. Radiological investigations were requested accordingly for further assessment.

US was the primary modality utilised for assessment of the patient. Scanning of the upper abdomen was carried out using a convex linear probe (3.5MHz frequency), focussing on the region of the pancreas and peripancreatic compartments. In particular, the lesser sac, anterior pararenal space and transverse mesocolon were investigated by scanning in the supine, longitudinal, transverse, semi-erect and coronal planes. The examination revealed well-defined oval, anechoic cystic lesion in the region of the pancreas, lying anteriorly and in direct contact with the confluence of the splenic and portal veins. Color Doppler scanning revealed incomplete color turbulent filling flow inside the lesion (Fig. 1). Biliary tract assessment revealed the absence of biliary stones in the gall bladder (extending to the intrapancreatic portion of the common bile duct) in addition to the absence of peripancreatic or intraperitoneal free fluid. The preliminary diagnosis entertained was a vascular aneurysm complicating pancreatitis. Vascular complications are known to arise in conjunction with pancreatitis due to the proteolytic nature of pancreatic enzymes which cause erosion of blood vessels. This often results in pseudoaneurysm formation or free rupture of a vessel. Hence, further assessment by MRI and CT was requested.

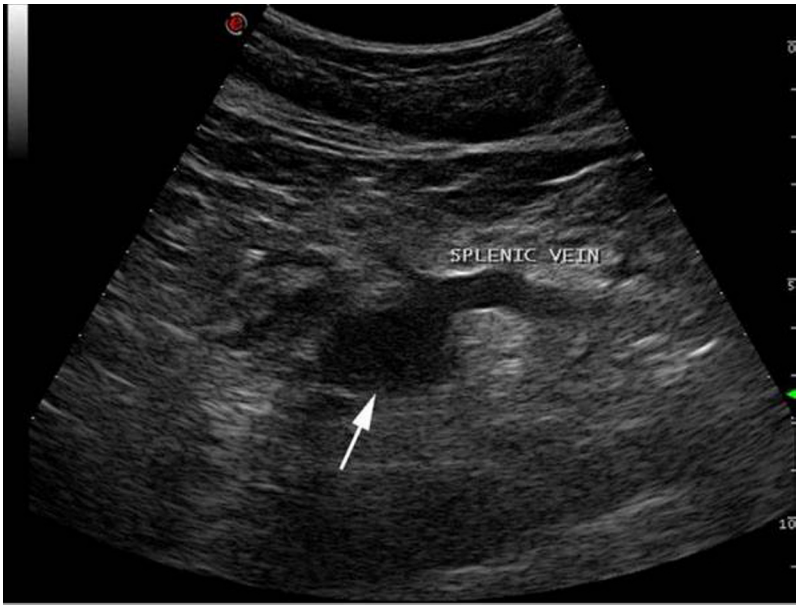

Figure 1A. 62-year-old man with eroding pancreatic pseudocyst. Transverse transabdominal sonogram shows an anechoic small cystic, round, lesion (arrow) that was 1.5 cm in diameter at the region of the pancreatic head.

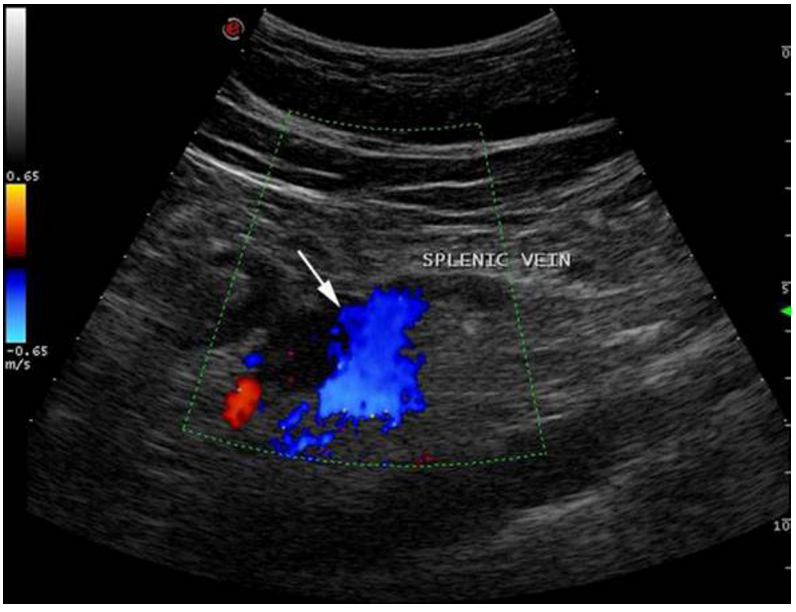

Figure 1B. 62-year-old man with eroding pancreatic pseudocyst. Color Doppler sonogram displayed turbulent partial color flow filling of the lesion (arrow) anteriorly located and in direct continuity with the junction between the splenic and portal vein.

T1-weighted and fast spin-echo, T2-weighted, out of phase chemical shift and fat-suppression sequences were carried out in addition to contrast enhanced MRA in order to further assess the lesion. The pancreas was found to be atrophic with no evidence of a focal lesion or an enhancement pattern abnormality. MRI revealed a well defined, thin walled, oval lesion which was mildly enhancing and inseparable from the anterior aspect of the confluence of portal and splenic veins. MRI revealed a low signal intensity in T1 and high signal intensity in T2 weighted images denoting the cystic nature of the lesion. An area of high signal intensity in contact with the porto-splenic junction in T1 and T2 and post Gadolinium-bawee injection was also observed, denoting a subacute blood collection (Fig. 2).

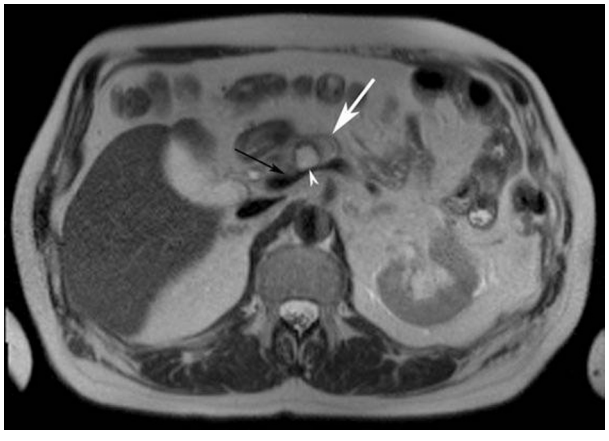

Figure 2A. 62-year-old man with eroding pancreatic pseudocyst. T2-weighted axial MRI shows the presence of intermediate signal intensity oval lesion with well defined margin (white arrow) that contains a rounded well defined area of high signal intensity (white arrow head). The legion appears inseparable from the anterior aspect of the terminal part of splenic vein and its confluence with portal vein (black arrow).

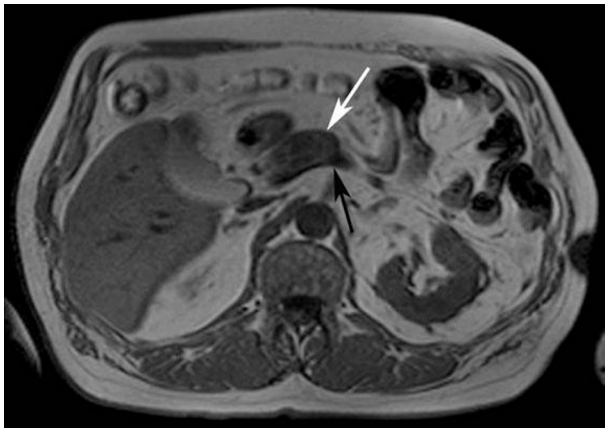

Figure 2B. 62-year-old man with eroding pancreatic pseudocyst. T1-weighted noncontrast-enhanced axial MRI shows low signal intensity of the lesion (white arrow) with absence of previously described central high signal area in T2.

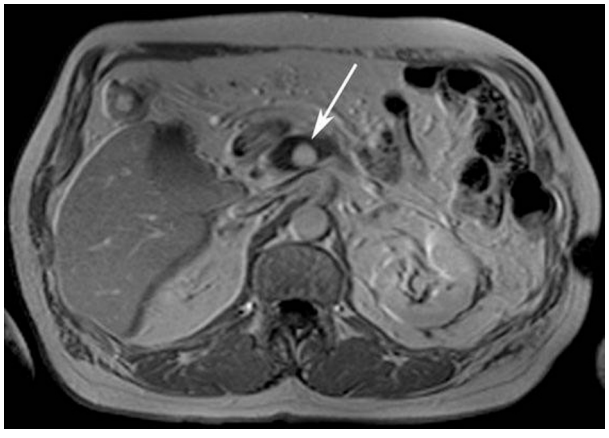

Figure 2C. 62-year-old man with eroding pancreatic pseudocyst. T1-weighted contrast-enhanced MRI shows low signal intensity of the lesion with high signal central area (arrow).

As contrast-enhanced CT imaging of the abdomen and pelvis remains the gold standard imaging modality in the evaluation of pancreatitis and its complications, this became the next step in the investigative process. Both intravenous and oral contrast were administered. Thin-section images were acquired during the peak of pancreatic arterial perfusion and venous phases. The protocol of scanning was the following: Tube Voltage: 120 KV, Tube current: 180 mAs, Pitch 1.2, slice collimation 0.6mm, Acquisition 64x 0.6 mm, slice width 0.75mm, reconstruction increment 0.4mm, Iodine concentration of contrast media 300 mg I/ml. CM flow rate 6.2 ml / sec; bolus timing determined by bolus tracking, bolus tracking threshold 180 HU, scan delay 7 sec for arterial phase and 15-20 sec for venous phase. Multiplanar reconstruction formats were employed for further assessment. The contrast-enhanced CT images revealed the presence of an oval shaped cystic lesion (40 HU) with anterior wall thickening which was mildly enhancing. The lesion was found to have a central collection of the contrast agent, and furthermore, a smooth anterior invagination of the splenic vein and its confluence with the portal vein was observed. The pancreas was atrophic with multiple, punctuate, calcific foci predominantly seen in the head and body of the pancreas. 3D reconstructed CT angiography confirmed that the contrast filling of the lesion seen during the portal venous phase was in direct continuity with the terminal course of splenic vein (Fig. 3).

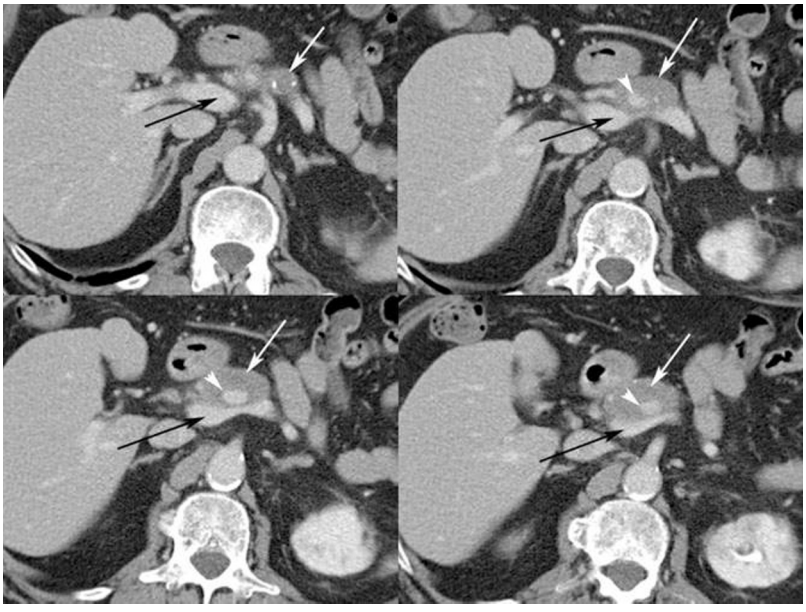

Figure 3A. 62-year-old man with eroding pancreatic pseudocyst. Serial axial contrast-enhanced CT images in portal venous phase show 40 HU lesion (arrow head) indenting the confluence between the portal vein and the splenic vein (white arrow) and central area of contrast extravasation having the same density of that of the portal vein (arrow head).

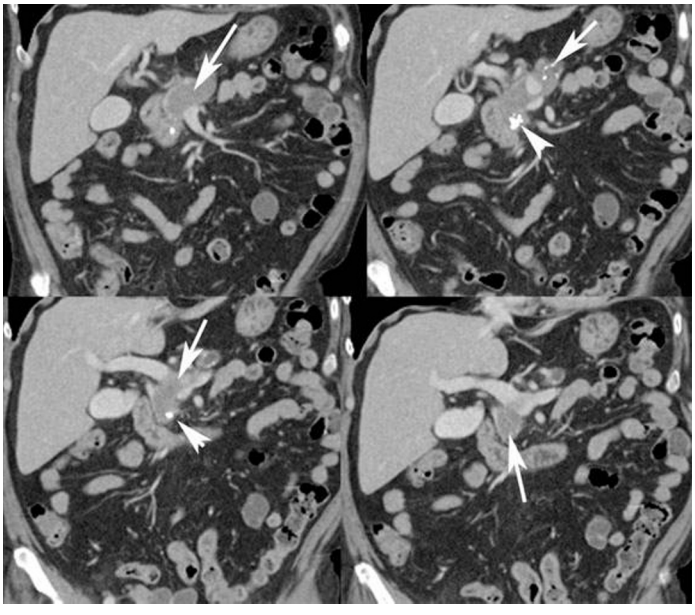

Figure 3B. 62-year-old man with eroding pancreatic pseudocyst. Serial coronal reformatted images in portal venous phase show the anatomical relations of the lesion (arrow) to the portal venous confluence with splenic vein. Notice the absence of pancreas which is extremely atrophic, with evidence of calcifications at the pancreatic head (arrow head).

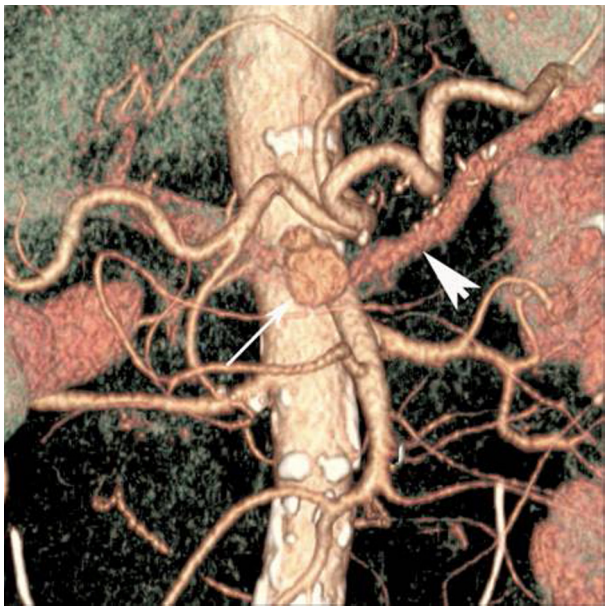

Figure 3C. 62-year-old man with eroding pancreatic pseudocyst. 3D reconstructed CT angiogram shows the contrast filling of the lesion (arrow) and its continuity with the splenic vein denoting vascular leakage (arrow head).

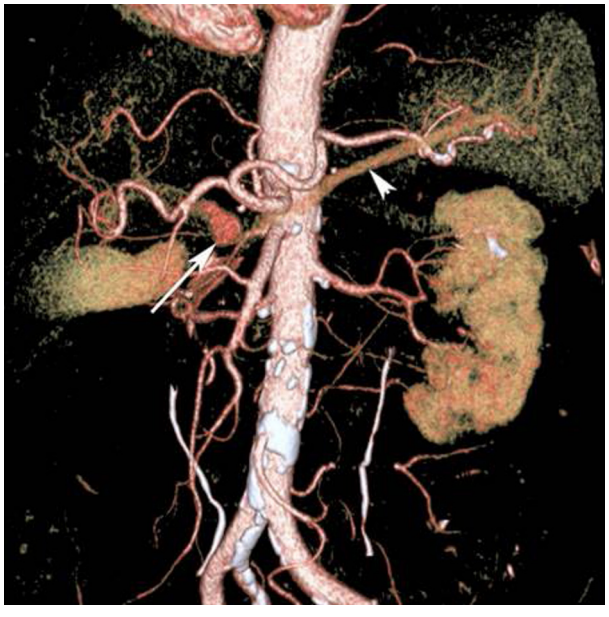

Figure 3D. 62-year-old man with eroding pancreatic pseudocyst. 3D reconstructed CT angiogram in different angle of obliquity.

As a final imaging tool prior to surgical exploration, celiac catheter angiography and indirect portography were carried out in order to further clarify the vascular nature of the lesion and definitively assess the extent of vascular involvement. These studies once more demonstrated the presence of contrast filling of the lesion during the portal venous phase (Fig. 4). No evidence of venous thrombosis of the splenic vein and/or collateral venous pathways was found.

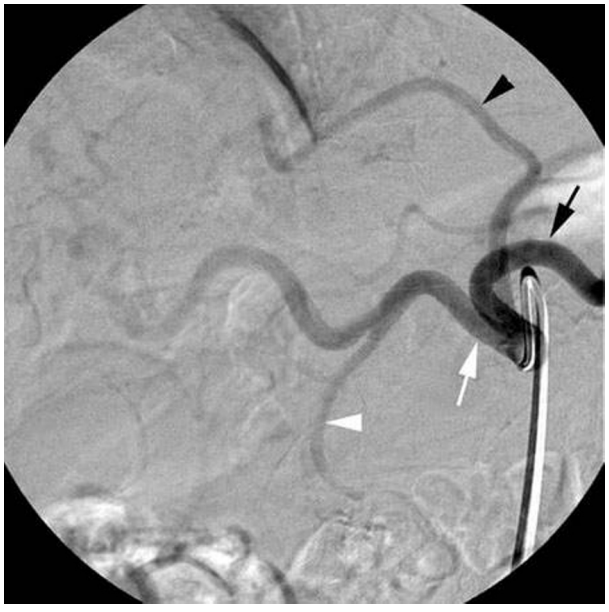

Figure 4A. 62-year-old man with eroding pancreatic pseudocyst. Celiac trunk catheter angiography in arterial phase shows normal angiographic appearance of common hepatic artery (white arrow), gastroduodenal artery (arrow head), left hepatic artery (black arrow head) and splenic artery (black arrow) with no evidence of aneurysmal change or vascular leakage.

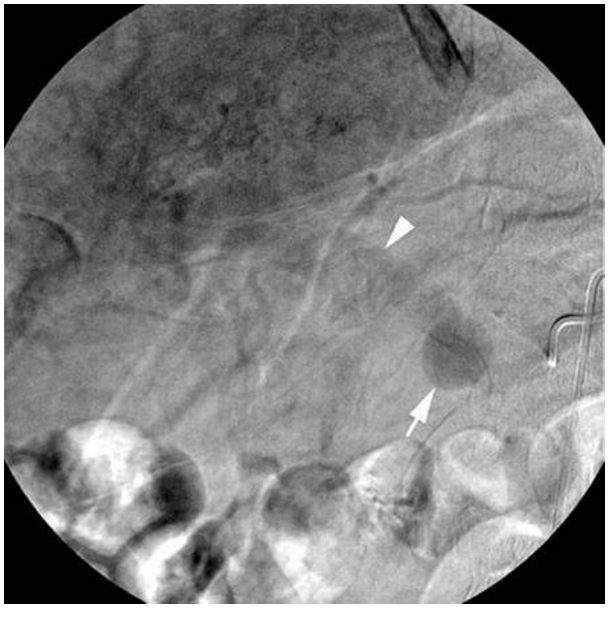

Figure 4B. 62-year-old man with eroding pancreatic pseudocyst. Celiac trunk catheter angiography in portal venous phase shows contrast filling the lesion (arrow) in continuity with the portal vein (arrow head).

During surgical exploration, the gross pathological change observed was the presence of a cystic lesion adherent to the anterior aspect of the splenic and porto-splenic venous confluence. The cyst was explored, revealing the presence of intracystic brownish serous fluid and clotted blood with erosion of the porto-splenic venous confluence. This cyst was resected vascular repair was carried out. Histopathological examination of the specimen revealed the diagnosis of a pancreatic pseudocyst, compressing and eroding the adjacent porto-splenic venous confluence.

Discussion

The majority of cystic masses of the pancreas encountered in clinical practice are post-inflammatory pseudocysts. Pancreatic pseudocysts are defined as localized, amylase-rich fluid collections located within the pancreatic tissue or adjacent to the pancreas and surrounded by a fibrous wall that does not possess an epithelial lining [1]. The CT findings of a pseudocyst include a round or oval fluid collection, with either a thin, barely perceptible wall, or a thick wall which shows evidence of contrast enhancement [2,3]. Peudocysts develop most often as a complication of acute or chronic pancreatitis, and may develop secondary to pancreatic trauma or surgery [4]. Although a prior history of pancreatitis cannot alone justify the diagnosis of pancreatic pseudocyst, careful evaluation of the patient's clinical history is important for the accurate diagnosis of a pseudocyst. Clinical scenarios include a pseudocyst developing after identifiable acute pancreatitis, a pseudocyst resulting from an acute incident superimposed on chronic pancreatitis and a pseudocyst with an uncertain or no known previous clinical history of pancreatitis. In the clinical setting of acute pancreatitis, the observation of peripancreatic inflammatory changes on initial CT scans and an evolving peripancreatic fluid collection that develops a wall or capsule, provides a sufficient diagnostic clue toward the diagnosis of pancreatic pseudocyst. Subsequent to an acute attack, the pseudocyst develops over a period of 4–6 weeks. As pseudocysts may resolve spontaneously, conservative management may suffice if they are smaller than 6 cm in diameter or the patient remains asymptomatic [5]. The recognition of a pancreatic pseudocyst resulting from chronic pancreatitis is facilitated by the presence of stigmata of chronic pancreatitis such as parenchymal calcifications, ductal stones, ductal dilatation, or atrophy of the parenchyma. Complications related to pseudocysts include infection, hemorrhage, rupture, obstruction of surrounding abdominal organs and vascular erosion of peripancreatic vasculature. Vascular involvement of vessels neighbouring the pancreas is not an uncommon complication of pancreatitis.

Intrapancreatic and peripancreatic arteries and veins may be eroded or thrombosed by the direct effect of pancreatic enzymes. Vascular involvement may be manifested by acute haemorrhage due to vessel erosion, rupture of oesophageal, gastric, or mesenteric varices or leakage from an arterial pseudoaneurysm [6]. Pancreatitis in combination with vascular complications is potentially lethal and therefore, early diagnosis utiliing various imaging modalities (including CT , MR imaging and Angiography) will play a vital role in evaluating the degree and extent of the condition and its differentiation from pseudoaneurysmal changes [7]. Aneurysms of the portal vein were once thought to be extremely rare, but nowadays they are well documented and not unusual. For perspective, they still represent only 3% of all aneurysms of the venous system. Although aneurysms of the portal venous system may be present in patients with liver disease, an overwhelming majority of patients do not have portal hypertension or chronic liver disease. Therefore, portal hypertension could be contributory, but is not essential to the development of portal venous system aneurysms. Both congenital and acquired causes have been proposed in the etiology. Acquired causes are due to portal hypertension, necrotizing pancreatitis, or abdominal trauma or surgery [8-9]. The most common aneurysm locations are the splenomesenteric venous confluence, main portal vein, and the intrahepatic portal vein branches at bifurcation sites. The rarest locations are the splenic, mesenteric, and umbilical veins. Given that there are variations in the diameters of both normal and cirrhotic portal veins, an aneurysm of the portal venous system is considered to be present if the vessel diameter is significantly larger at a certain point in comparison to the remainder of the vessel. The likelihood of aneurysm is higher if the morphology of the vessel is saccular or fusiform [9].

It is the major role of diagnostic imaging to clarify the nature of vascular complications following pancreatitis, as the way of management differs accordingly. Arterial aneurysms could be managed

either by: 1) surgical clipping and vascular grafting or 2) less invasively by endovascular stenting (using covered stents) or coiling particularly in patients at risk of surgery. In contrast, vascular erosions are more commonly operated upon surgically to secure the possibility of uncontrollable bleeding and when endovascular treatment is unfeasible. However, the differentiation can be challenging and hence the need of comprehensive utilisation of the diagnostic tools including: MRI, which involves contrast enhanced sequences, fat suppressed technique and MR angiography. CT and CT angiography with multiplanar reconstruction provide also an adequate clarification due to its high spatial resolution. Catheter angiography plays also an important role for proper anatomical localisation and can be used for the decision of the feasibility of endovascular intervention.

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