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(CASE REPORT)

Pancreatic pseudocyst-portal vein fistula: Unveiling a rare and critical clinical challenge

Oumaima El Abd *, Samia Bentabet, Maria Lahlali, Dafr Allah Benajeh, Sidi Adil Ibrahimi and Nada Lahmidani

Department of Hepatogastroenterology, CHU Hassan II de Fès, Faculty of Medicine, Pharmacy and Dental Medicine of Fez, Sidi Mohamed Ben Abdellah University.

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Abstract

Pancreatic pseudocysts are common complications of acute and chronic pancreatitis, but fistulization into the portal vein is an exceptionally rare and life-threatening condition. We report the case of a 63-year-old woman with a history of severe pancreatitis complicated by portal vein thrombosis and a large pancreatic pseudocyst. Imaging studies, including contrast-enhanced computed tomography and magnetic resonance cholangiopancreatography, revealed a fistulous communication between the pseudocyst and the portal vein, associated with biliary compression and dilation. The patient initially improved under conservative management with antibiotics, analgesics, and anticoagulation. However, she later developed cholestatic jaundice requiring endoscopic drainage with placement of a metal stent, leading to favorable clinical and radiological evolution. Pancreatic pseudocyst–portal vein fistula remains a diagnostic and therapeutic challenge due to its rarity and nonspecific presentation. Imaging plays a key role, with MRCP being particularly useful for confirming the diagnosis. Management strategies vary depending on clinical presentation, ranging from conservative treatment to endoscopic or surgical intervention. This case highlights the importance of early recognition of this rare complication and supports a multidisciplinary approach, with minimally invasive management as a valid option in selected patients.

Keywords: Pancreatic pseudocyst; Portal vein fistula; Chronic pancreatitis; Portal vein thrombosis; MRCP; Endoscopic drainage

1. Introduction

Pancreatic pseudocysts are observed in both acute and chronic pancreatitis; these are fluid collections rich in amylases without their own wall. Their natural progression can lead to spontaneous resolution, persistence, or the development of complications such as infection, intracystic hemorrhage, intraperitoneal rupture, or rupture into a hollow organ, and fistulization. The diagnosis is primarily radiological, and the therapeutic management is multidisciplinary, using various methods ranging from simple monitoring to surgical treatment, including endoscopic and percutaneous drainage. [1,2]

2. Case Report

We report the case of a 63-year-old woman with a history of stage C pancreatitis with a non-lithiasic gallbladder three months before her admission. An MRCP performed outside the painful episode, as part of the etiological diagnosis, revealed: stage E pancreatitis with pancreatic necrosis estimated at less than one-third of the gland with a severity score of 6, interruption of the main pancreatic duct at the body by the necrotic collection, thrombosis of the portal vein trunk and right and left portal branches. Two months later, an abdominal CT scan was performed for control, showing: a bilobed fluid formation of 13 x 7 cm involving the corporocaudal region of the pancreas containing intracystic debris,

* Corresponding author: Oumaima El Abd

causing extrinsic compression of the lower bile duct with upstream dilation of the common bile duct (CBD) and intrahepatic bile ducts (IHBD), absence of opacification of the portal vein trunk and its dividing branches. The patient presented to the emergency department with sudden severe abdominal pain associated with nausea and vomiting. The biological assessment showed a lipase level of 368 (> 6 times normal), and a disturbed infectious assessment: white blood cells at 12,540, CRP at 296. An abdominal CT scan favored an increase in the size of the pancreatic cystic formation, well-defined, bilobed shape, with homogeneous hypodense fluid content, with an enhanced wall after contrast, currently measuring 9 x 13 cm versus 6 x 12 cm in axial diameter. This formation extends to the posterior cavity of the omentum and the hepatic hilum, where it is still fistulized to the portal vein trunk, with a defect measuring 10 mm, with a total lack of opacification of the latter as well as its branches. This formation also compresses the main bile duct, causing upstream dilation, measured at 12 mm in maximum diameter, associated with regular parietal enhancement of the latter. The pancreas is increased in size at the level of its cephalic portion, raised heterogeneously at this level. Increased infiltration of peripancreatic and percystic fat, with the appearance of a poorly defined subpancreatic collection, without its own wall, of heterogeneous fluid density, related to necrotic casting. We put the patient on antibiotics, analgesics, and anticoagulants, with good clinical and biological improvement. Our therapeutic approach was based on monitoring since the patient improved symptomatically. Six months later, the patient presented with cholestatic jaundice and fever. The biological assessment showed white blood cells at 9,200, a prothrombin rate of 67%, total bilirubin at 83 with a predominance of direct bilirubin at 71, gamma-GT at 1,674, and alkaline phosphatase at 1,583. MRI results favored a pancreatic pseudocyst measuring 62 mm in its largest dimension, with lobulated contours located in the body of the pancreas, accompanied by thrombosis of the portal vein and the right and left portal branches, as well as dilation of the intrahepatic bile ducts (IHBD) and the common bile duct (CBD) to 16 mm in its third part due to compression by the pancreatic pseudocyst. The patient underwent endoscopic drainage with the placement of a metal stent ensuring proper drainage. Follow-up imaging indicated a pancreatic cystic formation in the body and head of the pancreas measuring 33 x 22 mm, with a metal stent in place in the CBD, no dilation of the intrahepatic bile ducts, and the presence of some peri-hilar hepatic vascular tubular structures in favor of a portal cavernoma without opacification of the portal vein trunk and its dividing branches.

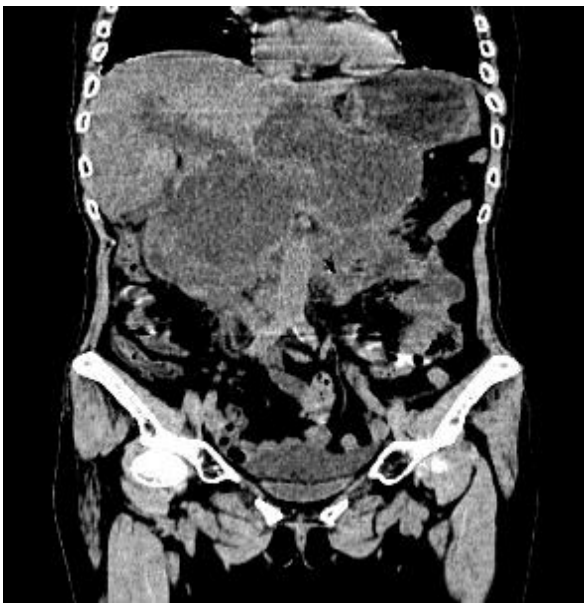

Figure 1 Coronal contrast-enhanced CT demonstrating a large bilobed pancreatic pseudocyst compressing the distal common bile duct with upstream dilation

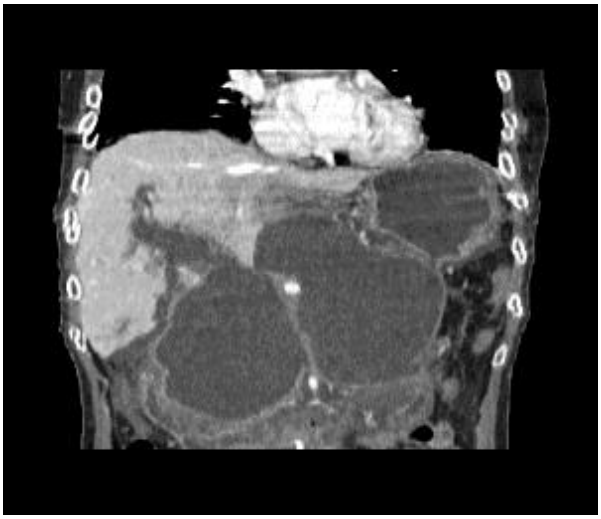

Figure 2 Coronal CT showing extension of the pseudocyst toward the hepatic hilum with communication to the portal vein trunk

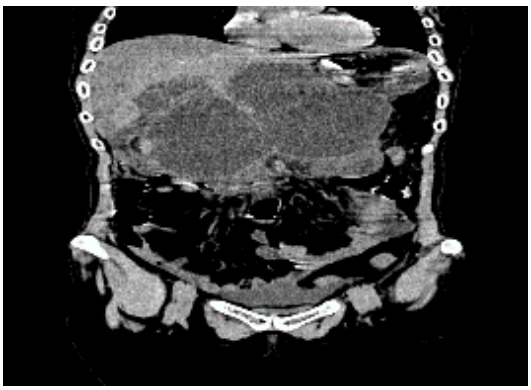

Figure 3 Coronal CT reconstruction illustrating the pseudocyst associated with portal vein thrombosis

3. Discussion

The formation of pancreatic pseudocysts (FKP) is a well-known complication of chronic pancreatitis, reported in up to 40% of cases. Vascular complications of chronic pancreatitis are also relatively common, occurring in up to 12% of patients. Although portal vein thrombosis and arterial pseudoaneurysms are well described in the literature, pancreatic pseudocyst-portal vein fistula (FPPVP) is an extremely rare complication of chronic pancreatitis, with only a few cases reported to date. [3]

The mechanism of fistulization remains poorly understood. Some authors suggest that the primary pathogenesis involves portal vein thrombosis resulting from mass effect and vessel compression by the pseudocyst, serving as a nidus for fistula formation. Others believe that high concentrations of pancreatic enzymes in the pseudocyst erode adjacent structures and directly cause intravascular thrombosis.

Several symptoms are observed in patients with FPPVP, with severe abdominal pain and elevated serum amylase/lipase levels being most common. Severe complications include septic shock, systemic lipolysis, hemorrhage, and death. Systemic lipolysis (Weber-Christian disease) is a feared complication of FPPVP due to pancreatic content release into the systemic circulation via the fistula, associated with high mortality rates. Chronic complications reported in the literature include portal hypertension and portal biliopathy secondary to cavernous transformation of the portal vein.

The definitive diagnosis of FPPVP in the context of chronic pancreatitis can be challenging. Endoscopic retrograde cholangiopancreatography (ERCP) was historically considered the most useful method by several authors before the availability of modern non-invasive imaging techniques such as high-resolution computed tomography (CT) and T2-weighted magnetic resonance imaging (MRI/MRCP). Recent retrospective studies have shown that all cases undergoing

initial MRCP imaging were correctly diagnosed with FPPVP, whereas cases initially evaluated by CT were diagnosed only with portal vein thrombosis, with correct diagnosis ultimately made by MRCP. Classic CT imaging findings of FPPVP include a portal vein attenuated by fluid with possible periportal inflammatory changes, collateral vessels, and associated pancreatic pseudocysts. These findings are particularly suspicious in patients with a history of chronic pancreatitis. On MRI/MRCP, a corresponding hyperintense central fluid signal is often observed in the portal vein on T2-weighted sequences. Direct visualization of a hyperintense T2 fistulous tract can be highlighted using three-dimensional rendering techniques. However, not all fistulous communications can be visualized on MRI. Endoscopic ultrasound can be useful for both diagnosis and intervention, demonstrating complex fluid in the portal vein with absent flow on color Doppler.

Treatment strategies for FPPVP vary widely in the literature, ranging from supportive medical management to surgical interventions, with variable outcomes. Most reported cases that survived required aggressive surgical treatment including portal venoplasty (for primary fistula closure) or pancreatectomy with pancreaticojejunostomy (to block pancreatic secretion flow) as definitive treatment. In one reported case, a pancreatic stent alone was successfully used to treat the fistula.

4. Conclusion

In summary, pancreatic pseudocyst-portal vein fistula is a rare but potentially life-threatening complication of chronic pancreatitis. Diagnosis in our case was established by contrast-enhanced CT and MRCP, and the patient responded well to medical management and anticoagulation. Findings of portal vein fluid attenuation on contrast-enhanced CT in a patient with chronic pancreatitis and pseudocysts should raise suspicion for pseudocyst-portal vein fistulization. Further imaging should be performed to definitively identify the fistula for potential surgical planning. MRCP is a reasonable confirmatory diagnostic study, which may be followed by ERCP if results remain equivocal. Treatment decisions are made on a case-by-case basis, with a conservative or minimally invasive approach favored in the acute phase due to the high risks of mortality and morbidity associated with surgery.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

Statement of ethical approval

Ethical approval was not required for this case report in accordance with institutional policies.

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