Medicine

LARGE PANCREATIC PSEUDOCYST SPONTANEOUSLY FISTULIZED TO THE DUODENUM

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ABSTRACT:
ONE OF THE POSSIBLE COMPLICATIONS OF PANCREATITIS – BOTH ACUTE AND CHRONIC, THE PSEUDOCYST, MAY EVOLVE TO IMPRESSIVE SIZES AND POSSIBLE LIFE-THREATENING COMPLICATIONS (RUPTURE OF THE PANCREATIC PSEUDOCYST, INFECTION, PSEUDOANEURYSMS OF THE NEIGHBOURING ARTERIES AND INTRACYSTIC HEMORRHAGE).
THIS ARTICLE REPORTS THE CASE OF A 66 YEARS OLD MALE PATIENT WHO WAS ADMITTED WITH THE DIAGNOSIS OF PANCREATIC PSEUDOCYST LOCATED TO THE BODY AND TAIL, FOLLOWING RECURRENT BOUTS OF ACUTE PANCREATITIS, WHICH DURING THE ADMISSION EVOLVED TO SPONTANEOUS FISTULIZATION TO THE DUODENUM. THE SUBSEQUENT COURSE OF THE PATIENT WAS COMPLICATED BY AN INTRACYSTIC HEMORRHAGE, MANIFESTED BY THE PASSAGE OF MELENA. THE PATIENT WAS SUCCESSFULLY MANAGED CONSERVATIVELY AND WAS DISCHARGED FOLLOWING A 14 DAYS HOSPITAL STAY.

KEY WORDS: PANCREATIC PSEUDOCYST, FISTULA, INTRACYSTIC HEMORRHAGE, GASTRO-INTESTINAL BLEEDING

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INTRODUCTION

The pancreatic pseudocyst (PP) represents one of the long-term complications of acute or chronic pancreatitis, developing in about 25% of the patients with such a medical past history[6]. In its developing, PP may reach impressive sizes and can determine symptoms by compression to the adjacent organs. Among the possible complications, rupture of PP, infection, pseudoaneurysm and intracystic hemorrhage are noted. Spontaneous perforation and fistulization occurs in less than 3% of the cases of PP and may develop to the main peritoneal cavity, stomach, duodenum, colon, portal vein or the pleural cavity. Intracystic hemorrhage represents a severe complication and may manifest as an upper gastro-intestinal bleeding (UGIB)[7]. High mortality among patients with intracystic hemorrhage (13%-90%) requires immediate therapeutical actions.

CASE REPORT

A 66 years old male patient, with a medical background of controlled hypertension , diabetes mellitus (controlled by oral antidiabetic agents), chronic type-B hepatitis virus infection and a history of acute etanolic pancreatitis 3 years prior and multiple flare-up episodes, who was previously imagistically diagnosed with a pancreatic pseudocyst, reports to the Emergency Department complaining of emesis and abdominal pain (more severe to the upper qudrants), progressively getting worse over the past 3 to 4 days.

The findings on clinical examinations: alert and orientated patient; apyrexial; haemodinamically stable; no respiratory distress; intact and normally coloured and hydrated skin and mucosae; soft and mobile abdomen; large, firm on palpation pseudo-tumoral mass to the epigastrium (about 10 cm in diameter); spontaneus and on palpation tenderness to the epigastrium and right upper abdominal quadrant, but no guarding.

Blood tests on arrval were normal, except for mild anaemia (Hgb=10.5 g/dL) and mild hyperkalaemia ($[K^+]$= 5.6 mmol/L).

The computed tomography scan (CT) findings: 135mmX76mm pancreatic pseudocyst located to the body and tail; contrast uptake to the walls of the pseudocyst; 21mmX16mm splenic artery pseudo-aneurysm in close contact with the anterior aspect of the pseudosyst; enlarged (49mm antero-posterior diameter), ill-defined and homogenously enhanced pancreatic head; multiloculated pseudocystic image to the hepatoduodenal ligament; inflammatory circumferential thickening of the gastric wall to the antropyloric region; hepatosplenomegaly; splenorenal collateral vessels (Figure 1).

[6] KD Lillemoe, CJ Yeo. *Management of complications of pancreatitis.* Current Problems in Surgery 1998; 35:1-98.

[7] U Atsushi, T Tsukasa, K Tadahiko, et al. *Rupture of a bleeding pancreatic pseudocyst into the stomach.* Journal of Hepato-Biliary-Pancreatic Surgery 2002; 9: 383-5.

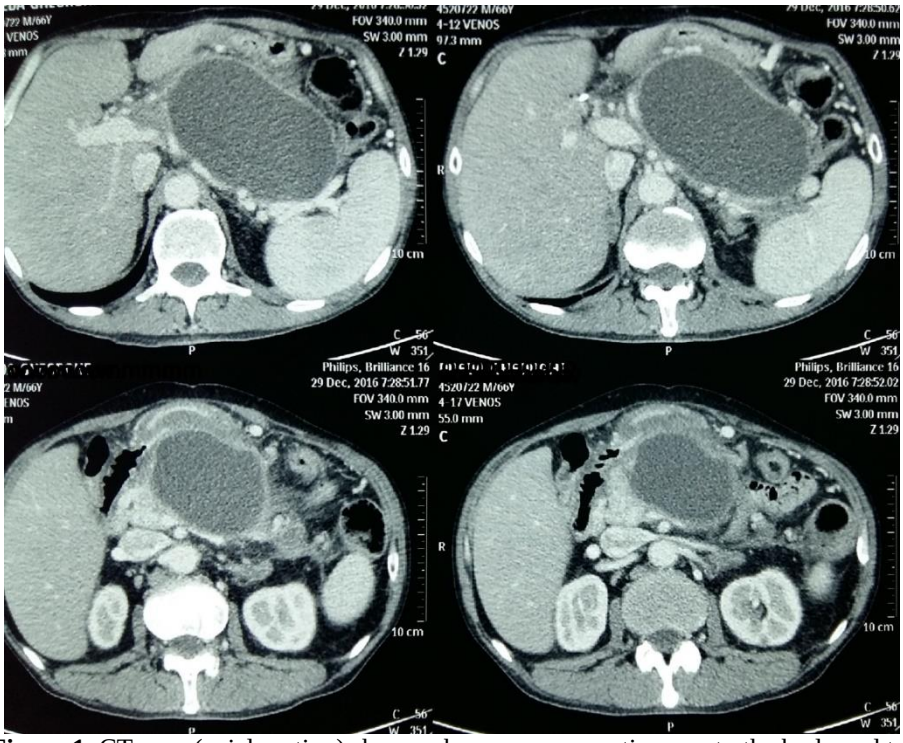

Figure 1. CT scan (axial section) shows a homogenous cystic mass to the body and tail of the pancreas, compressing the adjacent structures.

The patient received intravenous fluid repletion, analgesics and antispasmodics. Diabetology and cardiology consults were requested as part of the pre-operative workup.

On day 4 post-admission the blood tests show dropping Hgb level (9.1gt/dL), hyperglicemia (230mg/dL), altered liver function tests (ALT=111 U/L, AST=49 U/L), elevated pancreatic serum enzyme levels (Amylase=308U/L, Lipase=2848U/L) and hypocalcaemia (seric Ca=7.5mg/dL). On examination the abdomen was soft, non-tender; no changes were noted to the pseudo-tumoral mass described on admission.

On day 6 post-admission), on the pre-operative clinical review, the pseudo-tumoral mass was found to be barely observable; the patient described passing of black-coloured stools over the past 2 days; a digital rectal examination was performed showing melena.

An emergency upper-GI endoscopy was performed, finding a normal oesophagus and stomach, permeable pylorus; on passing to the duodenal bulb and further to the second part of the duodenum a significant amound of fresh blood was noted, originating from periampullary region on the medial wall of the descending duodenum (Figure 2).

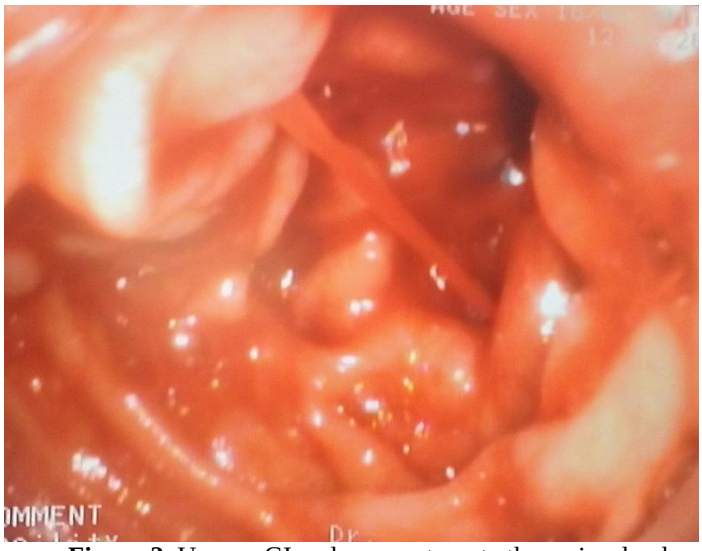

Figure 2. Upper –GI endoscopy: to note the major duodenal papilla (upper left) and the blood flow originating from the periampullary region.

The decision to perform an angiography was made. There was no spillage of the arterial contrast, but an anatomical variation of the splenic and common hepatic arteries was found, with both emerging from the aorta (Figure 3, Figure 4).

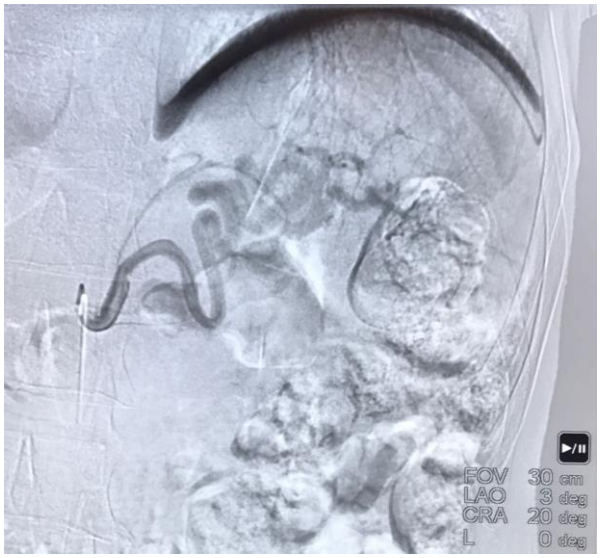

Figure 3. Angiography image – enhancement of the tortuous trajectory of the splenic artery.

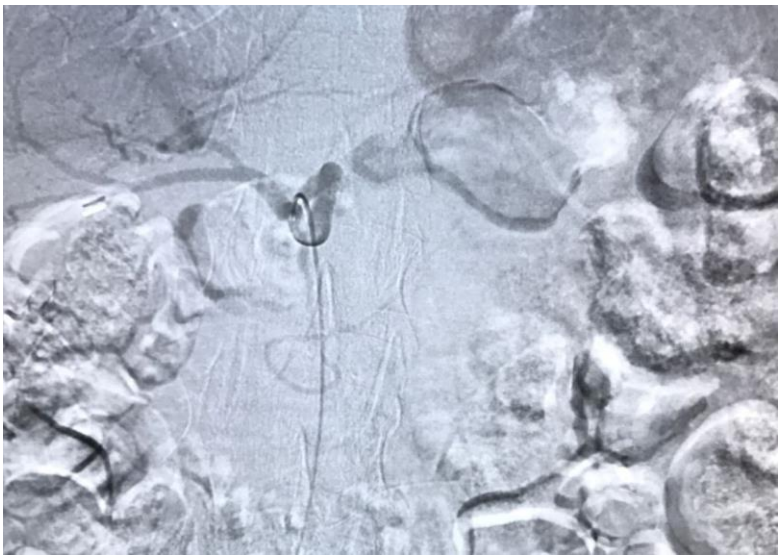

Figure 4. Angiography image – enhancement of the common hepatic artery and its branches.

A new CT scan of the abdomen was performed, showing spontaneous blood densities in an inhomogenous content of the pseudocyst in the pancreatic body and tail, which measured 12X6.5 cm. No spillage of the contrast agent in the arterial phase was seen, but an acute haematoma to the tail of the pancreas was noted in the venous phase (Figure 5, Figure 6, Figure 7).

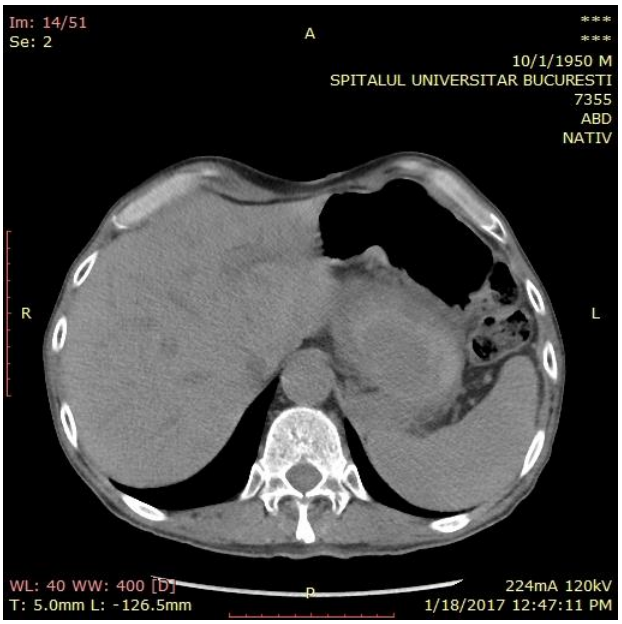

Figure 5. Native CT scan axial section – upper abdomen. 12x6.5cm spontaneously inhomogenous pseudocyst to the body and tail of the pancreas.

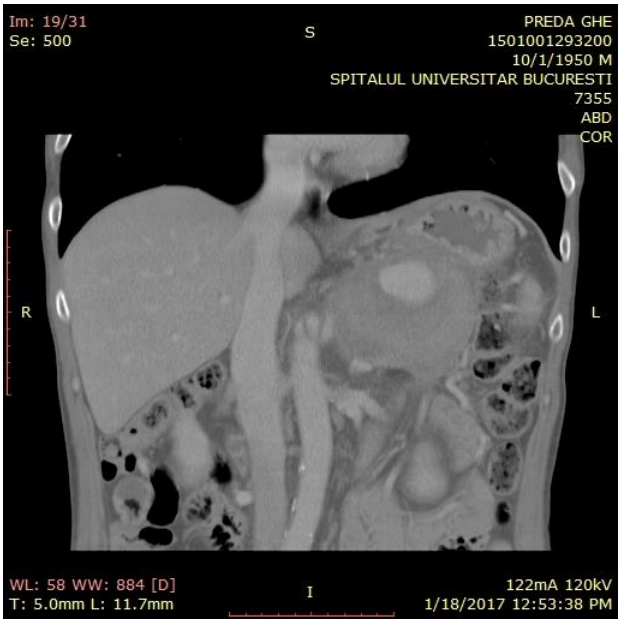

Figure 6. Contrast-enhanced CT scan coronal section – venous phase. Inhomogenous pancreatic pseudocyst, with intracystic contrast spillage to the postreo-superior area.

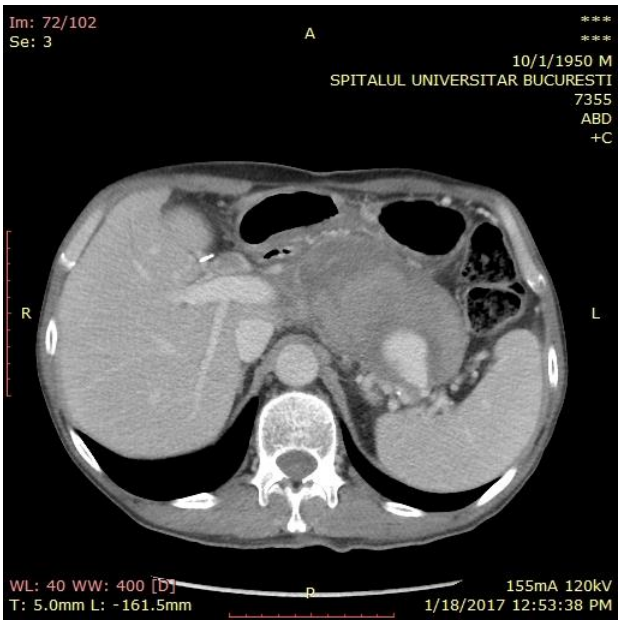

Figure 7. Contrast-enhanced CT scan axial section – venous phase. Inhomogenous pseudocyst to the body and tail of the pancreas; contrast is noted to the tail section of the pseudocyst

CT scan reconstruction images of the arterial phase confirm the separate origins of the splenic and common hepatic arteries from the aorta (Figure 8, Figure 9).

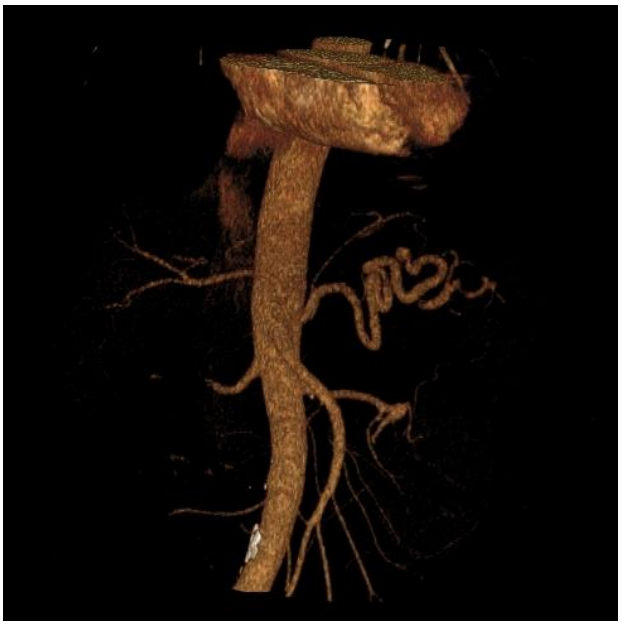

Figure 8. Angio-CT scan reconstruction. The main branches of the abdominal aorta are homogenously enhanced to the bifurcation, except for a wall calcification of the infrarenal aorta.

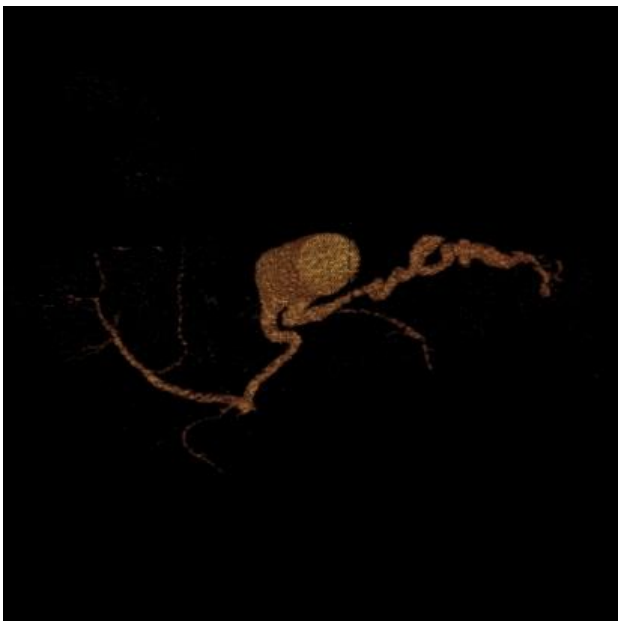

Figure 9. Angio-CT scan reconstruction showing the separate origins of the splenic and common hepatic arteries from the aorta (anatomical variation); the splenic artery pseudoaneurism described by the CT scan on admittance is no longer visible.

The surgical intervention was postponed. The patient received blood transfusion (RBC 1 unit, FFP 1 unit), unded close monitoring of the haemoglobin level. Hemostatic agents

(Phytomenadion, Etamsylate and Adrenostazin) and proton pump inhibitor (Controloc 80mg iv bolus) were also administered.

Following the administration of RBC, the Hb level dropped from 10.1g/dL to 9.9 g/dL after 2 hours and to 7.7g/dL after 6 hours. After 8 hours the Hb level appeared to be stable (7.6g/dL); further transfusion of 1 RBC unit and postponing the surgical intervention were decided.

As the Hb level maintained constant and subsequentely progressively rose over the following days and the patient did not pass further melena, we decided not to proceed with suplementary therapeutical interventions.

The patient was discharged on day 14 post-admission. At discharge he was in a good general condition, apyrexial and had a normal appetite; the abdomen was soft, mobile and non-tender.

DISCUSSION

The pancreatic pseudocyst occurs in about 25% of the chronic pancreatitis cases; it is more frequent in the set of alcoholic pancreatitis[8], but it may develop following any episode of acute pancreatitis or pancreatic trauma. The natural history of PP has not been fully defined yet, but it has been acknowlodged that 20-40% of the cases will evolve towards development of complications such as compression of the peripancreatic blood vessels, stomach or duodenum, infectoin, haemorrhage or spontaneous fistulization.

It is generally agreed that an asymptomatic and uncomplicated PP which is less than 6cm in size only requires follow-up. A larger but still asymptomatic PP may also be periodically monitored. Very large, fast-growing, symptomatic or complicated PP requires a form of treatment. Surgical, endoscopic and percutaneous techniques are to be considered. The surgical treatment aim is the drainage of the pseudocyst to the stomach or small bowell; supplimentary, stenting the Wirsung duct may be used in order to facilitate the pancreatic drainage[9].

Spontaneous resolving of PP is possible as the result of the spontaneous drainage to the duodenum through the Wirsung duct after the resolution of the local inflammation, the rupture of the pseudocyst to the greater peritoneal cavity or the developpment of a fistula between the pseudocyst and the gut (stomach, duodenum or colon); external fistulization through the abdominal wall is also possible.

Three pathogenical mechanisms were described for the bleeding, rupture or fistulization of PP. (1) The severe inflammation and activated enzymes (such as elastase and trypsin) can lead to enzymatic lysis of the elastic component of the vessel walls, eroding the adjacent blood vessels.(2) The mass effect and persistent compression of the neighbouring structures leads to ischaemia, increasing the enzymatic lysis. (3) The inflammation and the mass effect may lead to the compression of the splenic artery, generating venous thrombosis and limited secondary portal hypertension[10]. We support a complex mechanism involving local conditions increasing the enzymatic lysis secondary to persistent ischaemia and vascular thrombosis.

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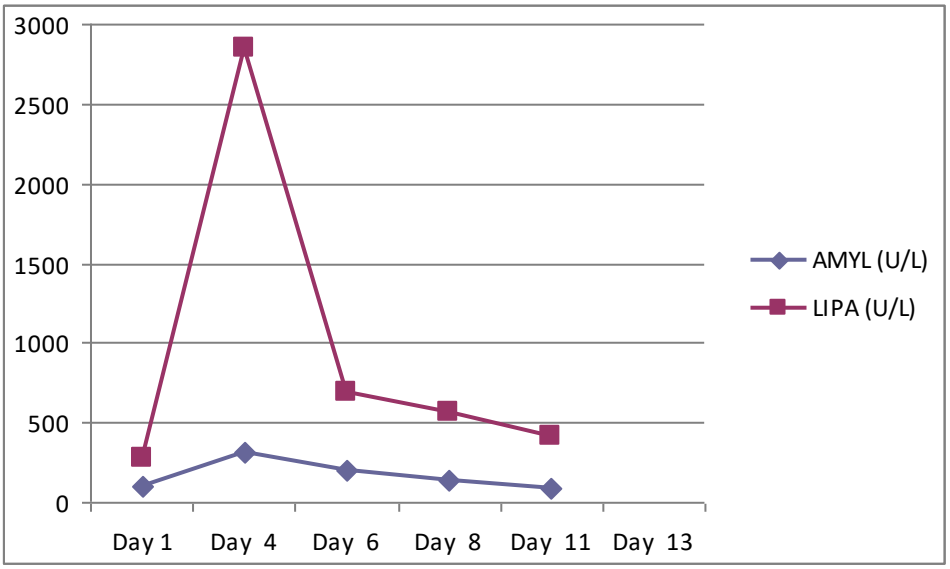
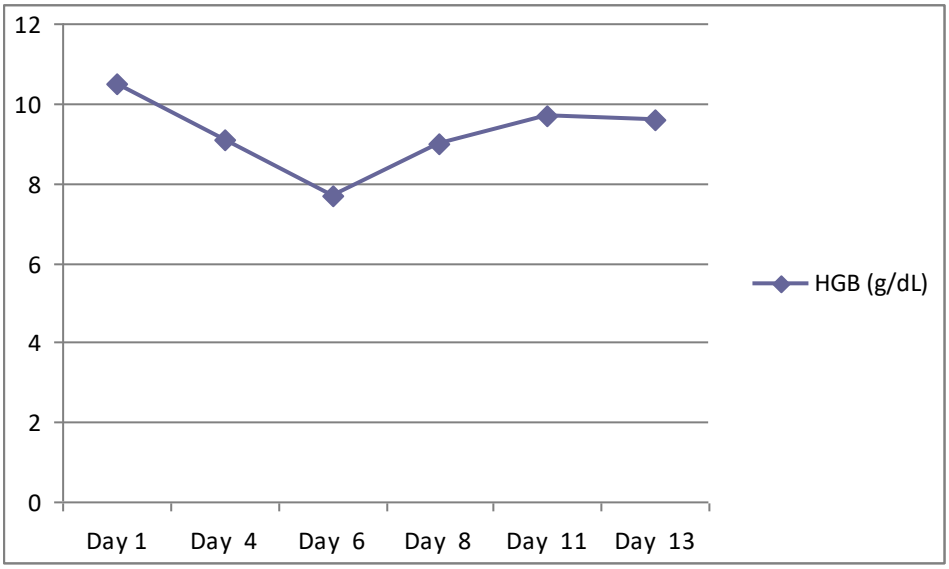
The fistulization of PP gives symptoms that point to its site. As an example, spontaneous fistulization to the stomach can present as a haematemesis or purulent vomitting; the fistulization of a haemorrhagic PP to the duodenum or tha small bowel can present as haemathemesis, diarrhoea, melena; the fistulization to the large bowel can lead to haematochezia.

In the discussed patient, the fistulization to the duodenum was initially asymptomatic, prior to the passing of melenic stools (on day 4 post-admission). Retrospective analisys of the laboratory results showed an abnormal serum level of the pancreatic enzymes (Amylase=308U/L, Lipase=2848U/L) on day 4 post-admission - the same moment the drop in the Hb level was noted (Graphic 1, Graphic 2). The data correlation led to our belief that the fistulization occurred on day 4 and that elevated serum pancreatic enzymes associated to a drop in the Hb level, in the absence of other clinical signs can suggest the fistulization of PP.



Graphic 1. The dynamics of serum amylase (AMYL) and serum Lipase (LIPA) during hospital admission. To note the spike of these levels on day 4 post-admission and the progression to normal levels to day 13.



Graphic 2. The dynamics of Hb level (HGB) during hospital admission. To note the descending trend starting from day 4 (from 10.5 to 9.1 g/dL), with a lowest on day 6 (7.7g/dL). The subsequent rise in the Hb level is due to blood transfusions (1 unit on day 6 and another 1 unit on day 8).

The angiography in a haemorrhagic PP can be negative if the bleeding is from a venous source, if the bleeding is intermitent or diffuse and also secondary to imaging artefacts.

Immediate diagnosis is mandatory for the proper management of a massive haemorrhage. The treatment depends on the source of the bleeding, the experience of the medical team and also the condition of the patient[11]. Transarterial embolization (TAE) is indicated for haemorrhages originating to the gastroduodenal and pancreaticoduodenal arteries or intrapancreatic arterial branches; it is not useful in haemorrhages originating to the splenic artery. Rebleeding is a possibility following TAE that should not be ignored. Some patients warrant an emergency surgical intervention as first therapeutical option[12].

If in a scheduled surgical intervention the perioperative mortality is 3%[13], in emergengy interventions it ranges between 25 and 47% [14].

The surgical treatment in order to stop the bleeding most oftenly requires a distal pancreatectomy and splenectomy. Some recommend in situ intracystic haemostasis or local compression by inflating a Foley catheter inside the cystic cavity. One should note that placing surgical ligatures on inflammated, necrotic or infected tissue carries the risk for rebleeding[15].

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The fistulae to the stomach or small bowell with no or limited bleeding do not require emergency surgical treatment. By comparison, the fistulae to the large bowel warrant surgical treatment in order to avoid the infection of the remaining cavity by the colonic bacterial flora[16].

In the particular case we have reported, the surgical treatment was not necessary, as the patient recovered under conservative treatment including haemostatic agents and fluid repletion.

CONCLUSION

In conclusion, PP is a possible complication of pancreatitis and can itself lead to complications such as rupture or fistulization to an adjacent viscus, haemorrhage or infection. The fistulization of a PP can be asymptomatic at the time of occurrence, but it can also present as an upper GI bleeding (hematemesis, melena, hematochezia). The clinical diagnosis should suffer no delays and the treatment should be tailored according to the anatomical site of the fistulization, the eventual complications, the local imagistic and surgical resources and the experience of the surgical team.

The clinical significance of measuring the levels of serum pancreatic enzymes in correlation with the level of haemoglobin as a predictive factor for fistulization in an asymptomatic patient requires further research.

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