Case Report

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Radiology Section

Haemosuccus Pancreaticus as an Uncommon Cause of Upper Gastrointestinal Bleeding: A Case Report

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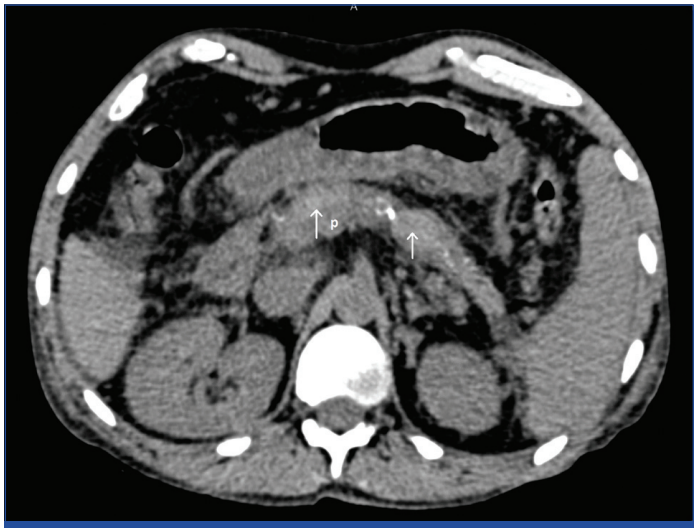
ABSTRACT

Haemosuccus Pancreaticus (HP) is a rare cause of upper Gastrointestinal (GI) bleeding. Despite being a rarity, HP should be considered in the differential diagnosis of patients with upper GI bleeding and pancreatitis. This condition commonly occurs secondary to pseudoaneurysm formation in the setting of acute or chronic pancreatitis. Angiographic embolisation remains the gold standard for treatment, and the condition is associated with a high mortality rate, if left untreated. The authors discuss the case of a 40-year-old male who presented with upper GI bleeding secondary to HP. The imaging findings of HP are presented in the present case, including the "sentinel clot sign," which is seldom seen but establishes the diagnosis of HP. Although HP is a rare and potentially life-threatening cause of upper GI bleeding, prompt diagnosis and effective treatment can reduce the mortality associated with HP.

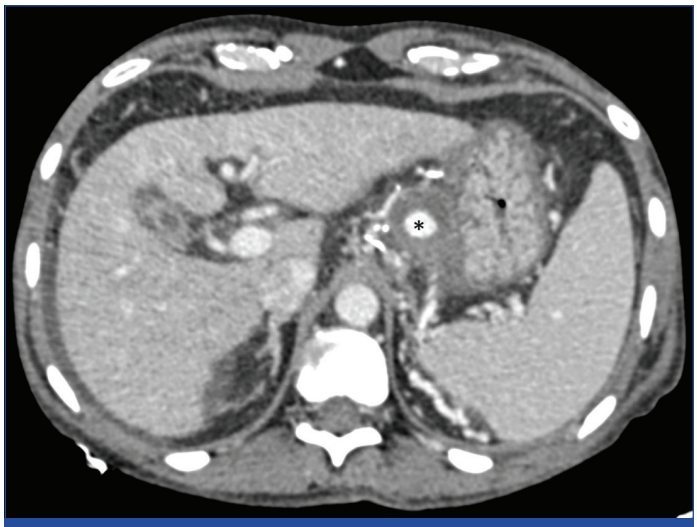
Keywords: Angiographic embolisation, Pancreatitis, Pseudoaneurysm

CASE REPORT

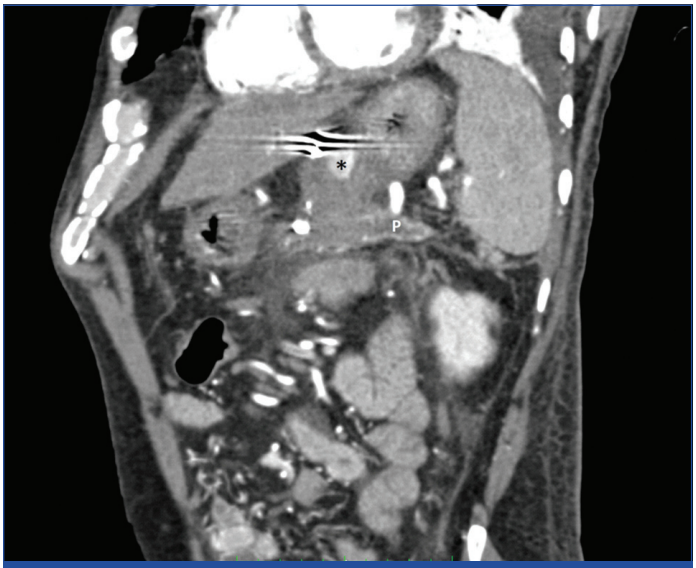
A 40-year-old male presented to the Emergency Department with complaints of blood in vomit (haematemesis), loose stool, chest pain, and difficulty swallowing solid foods for four days. There was no history of bleeding per rectum, fever, or abdominal pain. The patient was a known case of alcoholic liver disease-cirrhosis and diabetes mellitus and was on medication (Lasilactone 50 mg and metformin 500 mg) for the same. The patient gave a history of undergoing left gastric artery embolisation two months back because of upper GI bleed. On examination, the patient had pallor, a blood pressure of 120/90 mmHg, a pulse rate of 98/min, and a soft, non tender abdomen. Per rectal examination revealed no abnormalities. The initial laboratory workup revealed a haemoglobin level of 4.21 g/dL, Prothrombin time of 17.2 seconds, and International Normalised Ratio (INR) of 1.67 seconds. His lipase and amylase levels were within the normal range. The patient was subsequently admitted to the surgical ward and started on Fresh Frozen Plasma (FFP) transfusion, intravenous fluids, proton pump inhibitor, and antibiotics. An upper GI endoscopy showed features of antral gastritis and portal hypertensive gastropathy in the stomach. No ulcer or blood staining was seen in the D1 segment of the duodenum; however, the D2 segment of the duodenum showed blood staining without the presence of an ulcer. Ultrasound of the abdomen showed a coarse liver echotexture and mild ascites. An urgent Computed Tomography (CT) angiogram of the abdomen was requested to detect any active source of bleeding in the GI tract. On the non enhanced CT scan, the pancreatic duct was dilated (6.5 mm) and showed hyperdense contents within (average attenuation of +70 Hounsfield units), suggestive of clots-known as the "sentinel clot sign" [Table/Fig-1]. The pancreatic parenchyma was atrophic and showed few parenchymal calcifications, with the largest measuring 14 mm. The CT angiogram revealed a pseudoaneurysm measuring 10×12×14 mm {Anterior-Posterior (AP)×Transverse Measurement (TR)×Craniocaudal (CC)} adjacent to the lesser curvature of the stomach [Table/Fig-2]. A surrounding hyperdense haematoma was noted (thickness of 12.3 mm). Hyperdense sutures and surgical clips were seen along the course of the left gastric artery. The surrounding haematoma was seen to extend inferiorly and erode into the pancreatic body and the main pancreatic duct [Table/Fig-3]. No obvious enhancement of the pancreatic parenchyma was noted. The right gastric artery was seen branching from the hepatic artery proper and supplying the pseudoaneurysm [Table/Fig-4]. No active extravasation of contrast was noted at the site of the pseudoaneurysm. The splenic vein was thrombosed with multiple



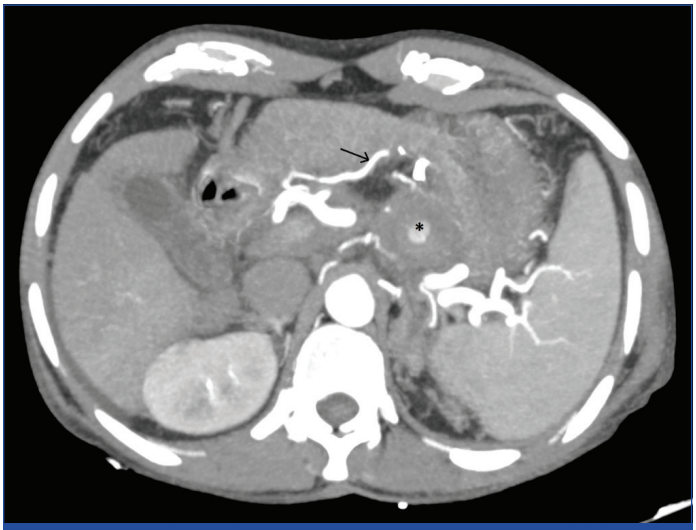
[Table/Fig-1]: Axial non enhanced CT scan shows hyperdense clots (arrows) within the pancreatic duct. P: Pancreas.



[Table/Fig-2]: Axial contrast CT scan shows pseudoaneurysm (asterisk) adjacent to the lesser curvature of stomach.



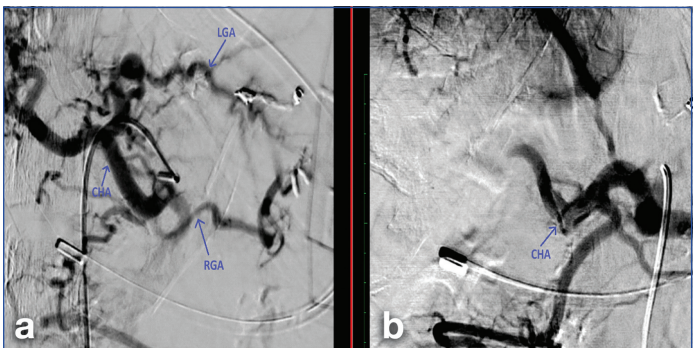
[Table/Fig-3]: Coronal contrast CT scan shows communication between the pseudoaneurysm (asterisk) and pancreas (P).



[Table/Fig-4]: Axial contrast CT scan shows the right gastric artery (arrow) supplying the pseudoaneurysm (asterisk).

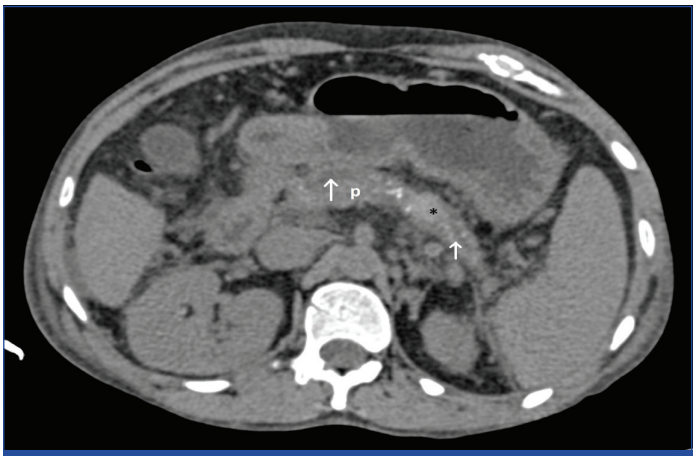
collaterals in the perisplenic and perigastric regions. Other findings on the CT angiogram included enlargement of the caudate lobe of the liver and mild ascites.

The patient was taken to the cath laboratory the following day for right gastric artery embolisation. The approach was made through the right femoral artery, and multiple attempts were made to hook the right gastric artery, with the final Digital Subtraction Angiography (DSA) run showing non opacification of the right gastric artery, likely due to a dissection caused intraprocedure [Table/Fig-5a,b].



[Table/Fig-5]: Initial run of digital subtraction angiogram: (a) Shows the right gastric artery coursing towards the site of pseudoaneurysm. Late run of digital subtraction angiogram; (b) Shows non opacification of right gastric artery likely due to dissection. CHA: Common hepatic artery; RGA: Right gastric artery; LGA: Left gastric artery.

A follow-up ultrasound the next day showed no colour flow within the pseudoaneurysm, likely thrombosed. The hepatic artery was seen to show good colour flow. A plain CT scan of the abdomen revealed resolution of haemorrhage in the main pancreatic duct [Table/Fig-6]. There was a rise in the haemoglobin level post-procedure (9.6 g/dL). At a follow-up three months after discharge from the hospital, the patient did not have haematemesis or melena.



[Table/Fig-6]: Follow-up plain CT scan of the abdomen shows resolution of haemorrhage in the pancreatic duct in the region of the head and tail (arrows). Residual haemorrhage is seen at the site of communication between the pseudoaneurysm and pancreas (asterisk). P: Pancreas.

DISCUSSION

HP, also known as haemoductal pancreatitis, is a rare cause of upper GI bleeding with a reported incidence of 1 in 1500 cases [1]. The term "Haemosuccus Pancreaticus" was coined by a Swedish surgeon, Philip Sandblom, in 1970, who reported three patients with GI bleeding from the pancreatic duct. It is defined as bleeding from the ampulla of Vater via the pancreatic duct into the duodenum [1]. The bleeding may originate from the pancreatic parenchyma, pancreatic duct, or structures near the pancreas such as arterial aneurysms [1]. It is more common in males, especially in the setting of alcohol intake [3,4]. Patients with this entity can present with melena, haematemesis, abdominal pain, anaemia, or haemoptysis [1,2]. The most commonly identified cause of HP is the rupture of a pseudoaneurysm secondary to pancreatitis. The causes of HP are listed in [Table/Fig-7] [5-7].

Clinical condition	Mechanism
Arterial aneurysms	Most commonly occurs in the setting of pancreatitis leading to gradual arterial wall necrosis. Vessels that can be involved are the splenic artery, common hepatic, gastroduodenal, pancreaticoduodenal, and gastric arteries [1,5].
Inflammatory/pancreatitis (acute or chronic)	Inflammatory reactions within the pancreatic duct due to pancreatic calculi and pseudocyst can activate lytic enzymes causing erosion of vessel walls [6].
Pancreatic tumours	Pancreatic neoplasms including pancreatic carcinoma, serous/mucinous cystic neoplasm, and neuroendocrine tumours have been associated with HP. Tumour bleeding into the pancreatic duct may lead to HP [1].
Developmental (Heterotopic pancreas, Pancreatic divisum)	Recurrent bouts of pancreatitis leading to HP [7].
Mechanical	Pancreatic trauma and iatrogenic causes (ERCP, EUS-guided FNA, pancreatic stenting) can lead to peripancreatic arterial damage [3].
Infectious (Brucellosis, Syphilis)	Leads to formation of aneurysm which can bleed into the pancreatic duct [3].

[Table/Fig-7]: Potential causes of Haemosuccus Pancreaticus (HP) [5-7].
ERCP: Endoscopic retrograde cholangiopancreatography; EUS: Endoscopic ultrasound; FNA: Fine-needle aspiration

The diagnosis of HP requires a multidisciplinary and integrative approach. An esophagogastroduodenoscopy is usually performed first in cases of upper GI bleed to rule out more common causes of GI bleeding such as peptic ulcer and erosive gastritis. It can also detect bleeding at the duodenal papilla, but only in 30% of cases

because of the intermittent nature of bleeding and suboptimal view of the duodenal papilla in a forward-viewing endoscope [8]. Ultrasound helps in the detection of aneurysms, pancreatic pseudocysts, and other features of acute/chronic pancreatitis [1]. A CT angiography is performed to detect the source of bleeding as well as to rule out any active bleed. On the precontrast scan, the characteristic finding in HP is the presence of high-attenuating blood clots in the pancreatic duct, referred to as "sentinel clot sign" [3]. The post-contrast CT images help in detecting pseudoaneurysms as well as features of acute and chronic pancreatitis such as pseudocysts and pancreatic necrosis. It can also identify the source artery and define its anatomy for therapeutic intervention [9]. Ultimately, the gold standard investigation in upper GI bleed is angiography.

There are two treatment modalities for HP: Angiographic procedures and surgery. The preferred treatment method in HP is angiographic embolisation. Overall, this is an effective treatment option; however, rebleeding can occur in 37% of cases [3].

Cui HY et al., reported a case of HP caused by gastroduodenal artery pseudoaneurysm associated with chronic pancreatitis. Ayala D et al., also reported a case of gastroduodenal artery pseudoaneurysm caused by a pancreatic head mass leading to HP. In both cases, the patients were managed with endovascular coil embolisation of pseudoaneurysm with no recurrence of haematemesis or melena after the procedure [6,9].

Surgery is indicated in haemodynamically unstable patients, bleeding from a large vessel, failed angiographic treatment, and in cases when another co-existent condition requires surgery such as pancreatic pseudocyst or pancreatic tumour [10]. The various surgical procedures used in HP include intracystic ligation, pancreatectomy, external vessel ligation, and pseudocyst drainage. The rebleeding rate after surgery is comparatively low at 0 to 5% [3,11].

CONCLUSION(S)

HP is a rare and potentially life-threatening cause of upper GI bleeding. It should be included in the differential diagnosis of patients with upper GI bleed and a history of pancreatitis. The diagnosis of HP requires a multidisciplinary and integrative approach, with endoscopy and various imaging modalities aiding in the diagnosis. Angiographic embolisation is the preferred treatment modality, with surgery being reserved for selected cases. Prompt diagnosis and effective treatment are indispensable in reducing the mortality associated with HP.

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