Medical Science

pISSN 2321–7359; eISSN 2321–7367

Pseudohemobilia (Hemosuccus Pancreaticus) as a presenting feature of fibrocalcific chronic pancreatitis managed successfully with angioembolization and coiling – A case report

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To Cite:
Verma P, Phate N, Talwar D, Kumar S, Acharya S, Banode P. Pseudohemobilia (Hemosuccus Pancreaticus) as a presenting feature of fibrocalcific chronic pancreatitis managed successfully with angioembolization and coiling – A case report. Medical Science, 2022, 26, ms188e2224.
doi: https://doi.org/10.54905/disssi/v26i123/ms188e2224

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Peer-Review History
Received: 08 April 2022
Reviewed & Revised: 09/April/2022 to 17/May/2022
Accepted: 18 May 2022
Published: 23 May 2022

Peer-review Method
External peer-review was done through double-blind method.

URL: https://www.discoveryjournals.org/medicalscience

ABSTRACT

We present a case of a 28 years old young male who was admitted with complaints of pain in abdomen, black colored stools and hematemesis. His ultrasonography of abdomen and pelvis was suggestive of calcific pancreatitis. Digital subtraction angiography and gastroscopy revealed active pseudohemobilia from pseudoaneurysm of left gastric artery. The bleeder was uneventfully embolized with by coiling.

Keywords: Pseudohemobilia, Fibrocalcific chronic pancreatitis, young male, anemia, hematemesis, black stools, coiling.

1. INTRODUCTION

Classical Pseudohemobilia presents as a clinical manifestations of biliary colic, jaundice, and upper gastrointestinal tract bleed. The most common etiology is trauma, followed by gallstones, inflammatory diseases, vascular lesions, neoplasms and sinistral portal hypertension due to blockage of splenic vein as a result of pancreatic ailment (Shah et al., 2021). The proposed mechanism is that blood in the biliary system causes obstruction of the pancreatic and common bile duct, leading to obstructive jaundice and HP. The tell-tale signs of the fibrocalcific chronic pancreatic disease are the presence of abdominal pain in childhood and calculi in pancreas accompanying with dilation of the pancreatic duct and fibrosis of the gland in adolescence. Mostly patients have clinical trial of pain abdomen, steatorrhea and Diabetes. Pseudohemobilia as a vascular complication following laparoscopic cholecystectomy has been described (Han et al., 2012). Case reports are available about pseudohemobilia

from arterioportal fistula causing acute cholecystitis and pancreatitis as a complication of percutaneous liver proceedings (Sanyukta et al., 2020) on literature search not a single case has been reported about upper gastrointestinal bleeding secondary to HP in chronic calcific pancreatitis. Here we report a case of young male who presented with HP with chronic calcific pancreatitis and successfully treated with embolization and coiling.

2. CASE REPORT

A 28 years old male presented to the outpatient department with history of abdominal pain in epigastric region which used to get aggravated by meals and on resting and reclining, increased frequency of motions and frothy and oleaginous stools. He also gave a history of polyurea and polydipsia and was diagnosed with diabetes 4 months before for which he was treated with insulin but his blood sugar was uncontrolled due to inattention. There was no family history of diabetes, no history of consumption of tobacco and alcohol. However on further history taking it was found that patient suffered from recurrent bouts of black colored stools and also gave history of having episodes of vomiting which was ensanguined.

On examination, he was afebrile, His BP was 110/70 mmHg, pulse 78/min, respiratory rate was 18/min and SpO2 was 100%. Pallor and icterus were present, no cyanosis, no clubbing, lymphadenopathy or parotid gland enlargement. His BMI was 17.72kg/m2. On per abdomen examination there was tenderness in epigastric region with no organomegaly, other system were within normal limits. His laboratory investigations revealed Hemoglobin- 6.7 gm/dL, MCV- 69.8, WBC – 6000/cumm, Total iron Binding Capacity -365 microgram/dL, serum iron - 45 mcg/dL, ferritin – 5.97 ng/ml. FOBT- conclusive. HbA1c - 13 percent, Sr.calcium -9.3mg/dL. His kidney function, liver function, serum protein, lipid profile and other investigations were normal.

In view of pain in abdomen, Computed tomography (CT) was planned which showed atrophy of parenchyma of pancreas with multiple small calcifications within. Main pancreatic duct was dilated measuring 4 mm in diameter. Hence, on the basis of clinical features and investigations, a diagnosis of Fibrocalcific pancreatitis was made. The patient has received fixed-dose injection mixtard -35 units before breakfast and 24 units before dinner.

Upper GI Endoscopy of the patient was carried out that revealed – psudohemobilia/ Hemosuccus pancreaticus (fresh bleed coming from ampullary opening) (fig.1) and recommended emergency Digital subtraction angiography.

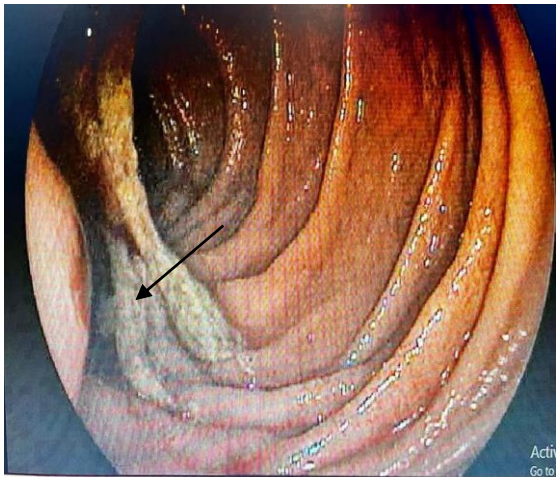

Figure 1 Endoscopy showing pseudohemobilia from ampulla of vater (black arrow)

DS angiography revealed bleeding pseudoaneurysm arising from left gastric artery (Fig 2A) which was treated with coiling and embolization (Fig 2B). Check angiogram revealed occlusion of left gastric artery pseudoaneurysm (Fig 2C). Sheath was removed and hemostasis was achieved (fig 3). Procedure went uneventful and was well tolerated by the patient.

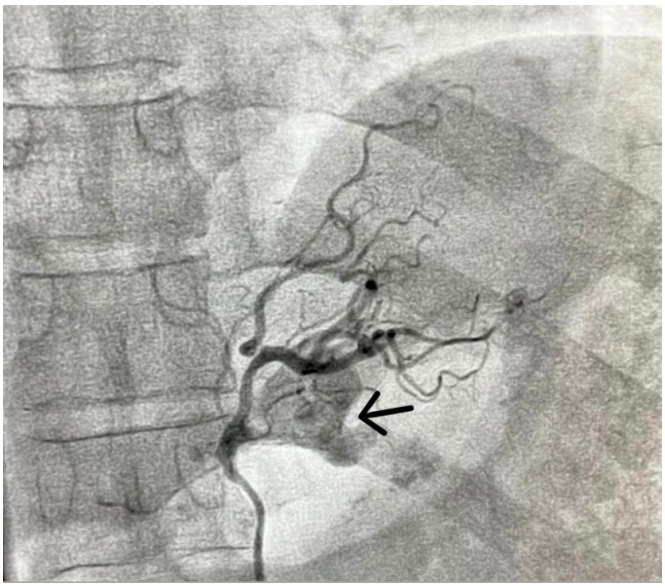

Figure 2A Showing pseudoaneurysm arising from left gastric artery with active bleed

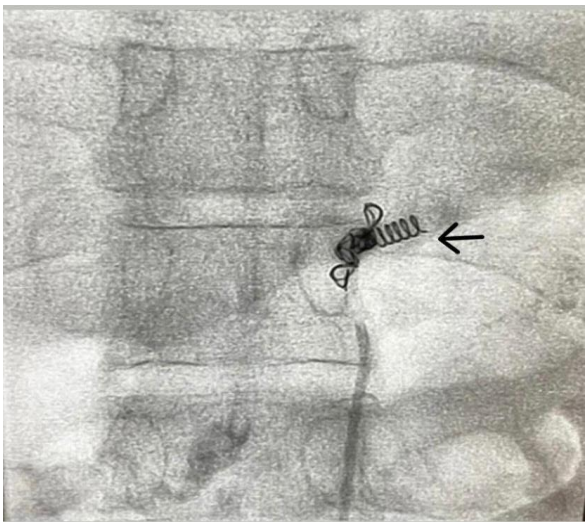

Figure 2B showing coil embolization of pseudoaneurysm.

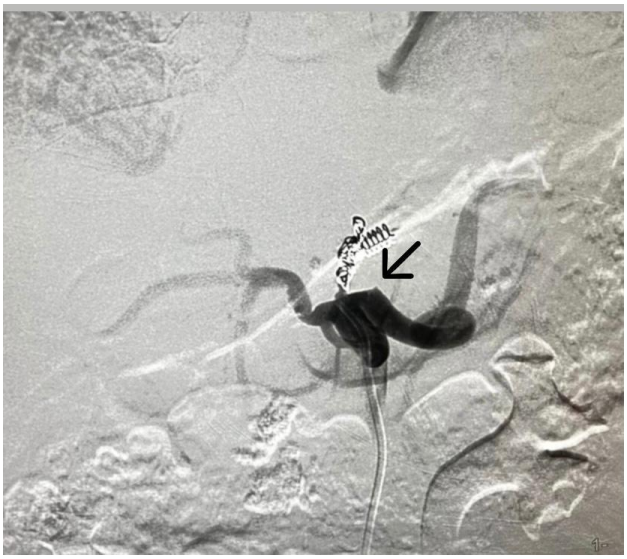

Figure 2C Check angiogram showing occlusion of pseudoaneurysm with achieved hemostasis.

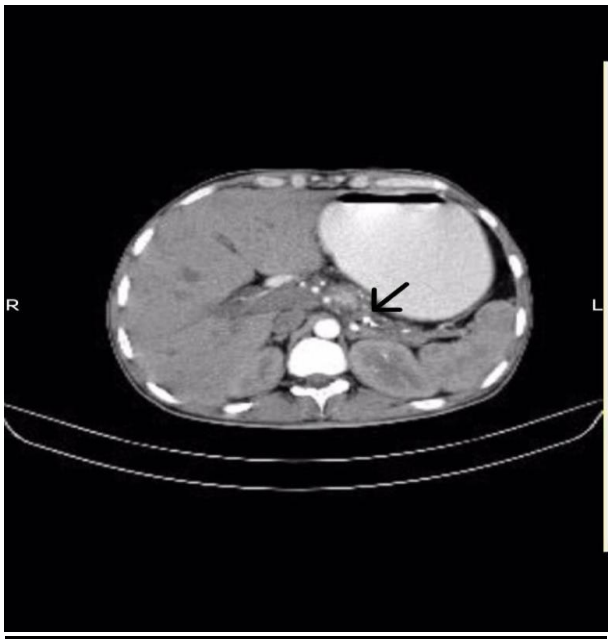

Figure 3 CECT abdomen showing chronic calcific pancreatitis

3. DISCUSSION

Pseudohemobila also known as Haemosuccus pancreaticus arises where a fistula or aneurysm has developed between a vessel of the splanchnic circulation, and the intrahepatic or extrahepatic biliary tree. It is a complication of chronic calcific pancreatitis and a potentially life-threatening obscure aftereffect of upper gastro-intestinal bleed, recounted as bleeding from ampulla of Vater via Pancreatic duct (Ferreira et al., 2015). It is one of the sporadic causes of upper gastrointestinal bleeding and is often invokes by chronic pancreatitis, pancreatic tumours and pseudocysts. This kind of chronic calcific pancreatitis is also prevalent in tropical regions of India seen in people with diet rich in tapioca (Kumar et al., 2012). Here pseudohemobilia occured as a consequence of chronic calcific pancreatitis as against most reported cases where chronic alcoholic pancreatitis was the pathophysiology. Pseudohemobilia is brought about by rupture of pseudoaneurysm of arteries liken splenic, gastroduodenal, pancreaticoduodenal, gastric and hepatic arteries. Breach of pseudoaneurysm can take place into the GI tract, peritoneum, pancreatic parenchyma or pseudocyst.

In our hospital patient the hemorrhage occurred from pseudoaneurysm of left gastric artery bursting into the pancreatic duct. Development of pseudoaneurysm secondary to calcified chronic pancreatitis and is rare but a significant phenomenon. Patients with gastrointestinal haemorrhage usually present with high amylase and lipase levels. However in our subject those were within normal range possibly due to the pancreatic exocrine damage correspondingly to chronic pancreatitis. Chronic local inflammation is thought to lead to an increased local release of elastase, with either autodigestion of peripancreatic vessels or erosion of a concurrent pseudocyst into the artery. Other factors are associated with accidental injury, gallstones and inflammation of gall bladder or pancreas, vascular malformations and tumors eg. Liver malignancy and bile duct cancers. Percutaneous liver interventions for liver such as liver biopsy, percutaneous cholangiography, and radiofrequency ablation are the major causes of pseudohemobilia (Mandaliya et al., 2014).

In our patient the pseudohemobilia developed within a span of 4 months after the patient underwent repeat gastroscopy in our set up. It was complicated by fibrocalcific pancreatitis and anemia. The incidence of pseudoaneurysm in patient with chronic calcific pancreatitis is quite a rare entity which made our case intriguing and a high index of speculation is necessary. Pseudohemobilia can be diagnosed by different modalities like contrast enhanced CT abdomen, Esophagogastroduodenoscopy, Visceral angiography, EUS and Endoscopic Retrograde Cholangiopancreatography. Ampullary hemorrhage could not always be recognized on gastroscopy owing to the intermittent and periodical nature of bleeding. Therefore it detects active bleed from ampulla only in a small number of cases and demonstrates bleeding from the ampulla on occasional instances. Thus, a negative endoscopy does not exclude the possibility of having pseudohemobilia.

If the source of hemorrhage is found by angiography then methods such as interventional radiological procedures & surgery should be the first line approach to initial management and stoppage of blood with immediate and successful positive results as perceived in our hospitalized patient.

4. CONCLUSION

Pseudohemobilia are usually obscure and requires high index of suspicion in the right clinical setting like chronic calcific pancreatitis along with appropriate investigations like Contrast Enhanced Computed Tomography of abdomen and DS angiography. Angioembolisation followed by coiling must be recommended for controlling bleeding, in the setting of chronic calcific pancreatitis. In a suitable patient who is stable, an upfront definitive procedure for chronic calcific pancreatitis may be considered.

Acknowledgement

We thank all the participants who have contributed in this Study.

Informed Consent

Informed Consent was obtained from the patient.

Author's Contribution

All the authors contributed equally to the case report.

Funding

This study has not received any external funding.

Conflicts of interest

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

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