Case Report

Recurrent upper gastrointestinal bleeding in chronic pancreatitis since childhood: a case report

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ABSTRACT

Chronic pancreatitis is a progressive inflammatory disease of the pancreas. Causes of gastrointestinal (GI) bleeding in chronic pancreatitis are bleeding into the pseudocyst, pseudoaneurysm of peripancreatic vessels, and thrombosis of portal vein and splenic vein. We are presenting a rare case presentation of recurrent GI bleed in chronic pancreatitis since childhood and its management. A seventeen-year-old female patient presented with recurrent attacks of upper abdominal pain, hematemesis, bleeding per rectum, and melena since 1 month of age. She was diagnosed with a case of chronic calcific pancreatitis with pseudoaneurysm of common hepatic artery, and portal vein thrombosis with splenomegaly at the age of 2 years. She underwent angioembolization of the common hepatic artery pseudoaneurysm at the age of 2 years. However, she continued to have recurrent episodes of upper GI bleeding which required multiple hospital admissions and blood transfusions in the past. Contrast-enhanced computed tomography of abdomen was suggestive of chronic calcific pancreatitis with status post coil embolization of common hepatic, gastroduodenal, and proper hepatic arteries. She underwent modified Frey’s procedure in view of recurrent symptoms. Currently, the patient is doing well after 18 months of follow-up, with no further episodes of GI bleed. Angioembolization or surgical ligation of pseudoaneurysm of involved vessels in chronic pancreatitis can control GI bleed. However, in cases where the source of bleeding is not localized by imaging/endoscopy, drainage of pseudocyst or local resection of pancreatic tissue can reduce inflammation in and around the pancreas and prevent further GI bleed in chronic pancreatitis.

Keywords: Chronic pancreatitis, Hemosuccus pancreaticus, Common hepatic artery, Pseudoaneurysm

INTRODUCTION

Chronic pancreatitis is a chronic inflammatory condition, which can lead to a variety of life-threatening long-term complications. Vascular complications from chronic pancreatitis are not infrequent and include venous thrombosis and arterial pseudoaneurysm.1 Hemosuccus pancreaticus is described as a hemorrhage that enters the pancreatic duct and travels via the ampulla of Vater to the gastrointestinal tract.2 Hemosuccus pancreaticus is one of the rare causes of intermittent upper gastrointestinal (GI) hemorrhage. Causes of GI bleed in chronic pancreatitis are bleeding into the pseudocyst- as the cyst enlarges in size, rupture of pseudoaneurysm from peripancreatic vessels, and thrombosis of the splenic vein and portal vein. We are presenting a rare case presentation of recurrent GI bleed in chronic pancreatitis since childhood and its management.

CASE REPORT

A seventeen-year-old female patient presented with recurrent attacks of upper abdominal pain, hematemesis, bleeding per rectum, and melena since 1 month of age. The patient continued to have these symptoms of pain in the upper abdomen followed by hematemesis and melena at a frequency of once in 2 to 3 months. At the age of two years, she was diagnosed to have chronic calcific pancreatitis with pseudoaneurysm of the common hepatic artery.
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(CHA), portal vein thrombosis, and splenomegaly by contrast-enhanced computed tomography (CECT) abdomen. In view of recurrent episodes of GI bleeding, coil embolization of CHA, gastroduodenal artery (GDA), and proper hepatic artery was done to exclude the pseudoaneurysm of CHA. She underwent a CT-guided celiac plexus block for her recurrent intractable abdominal pain. However, her symptoms of hematemesis and abdominal pain continued to recur. There were no bleeding sources identified in spite of repeated upper gastrointestinal (UGI) endoscopy till the last episode of hematemesis. She required multiple hospital admissions and received multiple blood transfusions in the past. Her serum calcium, parathormone, and sweat chloride test were normal. Pancreatic divisum was ruled out with magnetic resonance cholangiopancreatography. She then presented to us for further management. She had no features of exocrine or endocrine pancreatic insufficiency. On examination, she was hemodynamically stable and had pallor. Her abdomen examination was unremarkable. Her hemoglobin was 2.9, serum amylase - 129, serum lipase - 186, and C-reactive protein – negative. CECT abdomen with abdominal angiography showed non-visualization of CHA (status post coil embolization), the right hepatic artery was reformatted from an inferior phrenic artery, the left hepatic artery was reformatted from GDA and GDA was reformatted from left gastroepiploic artery (Figure 1). Also, there were calcifications in the head and uncinate process of the pancreas, pseudocyst in the region of aortic hiatus, and thrombosis of the portal vein and splenic vein (Figure 2). She was optimized and taken up for surgery in view of her long-standing, recurrent symptoms of abdominal pain and upper GI bleeding. Intra-operatively there were multiple, large perigastric and peripancreatic collaterals present (Figure 3).

The head and body of the pancreas were hard in consistency with intra-ductal calculi. The main pancreatic duct (MPD) was identified using intra-op ultrasound and was laid open up to the level of the body of the pancreas in view of hostile vascular anatomy around the pancreas and to the liver. She underwent a modified Frey’s procedure with limited pancreatic head coring and laid open MPD (Figure 4). Her immediate post-op period was uneventful. She has been asymptomatic on regular follow-ups for the last 18 months post-surgery.

Figure 1: CT abdominal angiogram showed non-visualization of common hepatic artery (status post coil embolization).
SPL ART, splenic artery; SMA, superior mesenteric artery; IMA, inferior mesenteric artery; RT RA, LT RA, right and left renal arteries

Figure 2: Abdominal CT scan showed calcifications (arrow) in the head and uncinate process of the pancreas.

Figure 3: Intra-operative image of multiple, large perigastric (arrow) and peripancreatic collaterals.

Figure 4: Intra-operative image of limited coring of tissue in the head of pancreas (arrow) and limited laid-open main pancreatic duct to the level of the body of pancreas.
DISCUSSION

Chronic pancreatitis is a chronic inflammatory condition that causes permanent damage to the pancreatic parenchyma leading to exocrine and endocrine insufficiency. Epidemiology, clinical presentation, and natural course of chronic pancreatitis are well described in the adult population. In our case, the patient had been experiencing symptoms of chronic pancreatitis and its complications since infancy—a rare case presentation in children. The most common cause of chronic pancreatitis in children is idiopathic pancreatitis. There is no identifiable cause of pancreatitis in our case. Patients with chronic pancreatitis will present with abdominal pain, exocrine pancreatic insufficiency, malnutrition, and diabetes mellitus. Vascular complications are common in patients with chronic pancreatitis. Venous thrombosis is more frequently seen than arterial pseudoaneurysm. In pancreatitis, the leaked pancreatic enzymes with background inflammation can cause erosion of peripancreatic vessels leading to the formation of pseudoaneurysm of peripancreatic vessels. A pseudoaneurysm can erode and bleed into a pseudocyst, pancreatic duct, peritoneal cavity and gastrointestinal tract. Rupture of pseudoaneurysm into pseudocyst or pancreatic duct causes hemosuccus pancreaticus. Arteries most commonly involved in pancreatitis are the splenic artery, gastroduodenal artery, and pancreaticoduodenal arteries, which account for up to 90% of visceral arterial pseudoaneurysm due to pancreatitis. Common hepatic artery (CHA) pseudoaneurysm due to pancreatitis is very rare. Our patient was diagnosed with chronic pancreatitis with CHA pseudoaneurysm and underwent angioembolization of CHA at the age of two years in view of recurrent episodes of endoscopy-negative upper gastrointestinal (UGI) bleeding.

Hemosuccus pancreaticus is an obscure cause of intermittent UGI bleeding, which can be a potentially life-threatening condition. UGI endoscopy is the initial investigation of choice. Endoscopy detects bleeding from the ampulla only in 30% of cases, however, a negative endoscopy does not rule out hemosuccus pancreaticus. In the index case endoscopy was done on multiple occasions which were of normal study, no bleeding sources were identified. Contrast-enhanced computed tomography (CT) scan of the abdomen can demonstrate the features of chronic pancreatitis. CT abdominal angiography has a sensitivity of greater than 90% in delineating the involved artery by pseudoaneurysm. In our case, CT abdominal angiogram demonstrated status post coil embolization of CHA but there was no identifiable pseudoaneurysm.

The management options for hemosuccus pancreaticus are interventional radiology procedures and surgery. Interventional endovascular procedures such as angioembolization, stenting across the pseudoaneurysm, and balloon tamponade are the initial treatment options of choice if the patient becomes hemodynamically stable after resuscitation. Endovascular therapy’s successful control of bleeding is seen in more than 65% of cases and with a reported risk of rebleeding in 20% of cases. Angiographic control of bleeding in hemosuccus pancreaticus can be done to stabilize the patient or as a definitive treatment. In our case, the CT abdominal angiography showed no pseudoaneurysm or contrast extravasation hence interventional radiology procedures were not contemplated.

Surgical intervention is needed in patients with uncontrolled, persistent bleeding leading to shock and in patients with failed endovascular therapy. Also, surgery is considered when the angiography shows no abnormal findings despite recurrent GI bleeding in chronic pancreatitis. Surgical procedures described include drainage of the pseudocyst with ligation of the bleeding vessel within the cyst, ligation of arterial pseudoaneurysm, drainage procedures (Frey’s procedure or lateral pancreaticojejunostomy), and pancreatectomy. Studies have reported success rates of surgical management of up to 80% and mortality rates of 23% to 50%. In our case surgical procedure (modified Frey’s procedure) was carried out because of long-standing recurrent episodes of endoscopy-negative GI bleeding with no identifiable pseudoaneurysm on angiogram. Surgical drainage of the pancreas in chronic pancreatitis reduces inflammation in and around the pancreas, reducing the risk of pseudoaneurysm formation and further bleeding.

CONCLUSION

Recurrent upper gastrointestinal bleeding in a patient with chronic pancreatitis could be due to hemosuccus pancreaticus. In patients with pancreatitis and GI bleeding, there should be a high index of suspicion for hemosuccus pancreaticus. Investigations such as upper GI endoscopy, CECT abdomen, and CT abdominal angiography performed at the time of bleeding will help in diagnosis. Though radiological intervention is the primary modality of treatment in hemosuccus pancreaticus, in the absence of demonstrable pseudoaneurysm, surgical drainage of pseudocyst or pancreatic duct reduces the inflammation in and around the pancreas which can reduce the risk of pseudoaneurysm formation and further bleeding.

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REFERENCES


