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Pancreatic Arteriovenous Malformation Presenting with Upper Gastrointestinal Bleeding: Treatment with Transarterial Embolization—A Case Report

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Abstract

Pancreatic arteriovenous malformations (AVMs) are a rare entity among visceral AVMs and less common cause of gastrointestinal bleeding. We report a case of pancreatic AVM in a 45-year-old man who presented with upper gastrointestinal bleeding and duodenal ulcers, and was hemodynamically unstable. Ultrasound and computed tomography (CT) of the abdomen showed multiple dilated arterial channels in the head region of the pancreas, arising from the gastroduodenal artery, with early filling of the portal vein, suggestive of an AVM. Transarterial embolization was performed by selectively embolizing the arterial feeders using poly-vinyl alcohol (PVA) particles. Postembolization, obliteration of the AVM was seen. On follow-up 2 months later, the patient was clinically stable.

Keywords

- pancreatic arteriovenous malformation
- transarterial embolization
- upper gastrointestinal bleeding
- endovascular embolization

Introduction

Pancreatic arteriovenous malformation (AVM) is a rare condition with low incidence and only few cases being reported in the literature. It can present with varied clinical manifestations, such as vague abdominal pain, gastrointestinal bleeding, duodenal ulcers, pancreatitis, or even portal hypertension. However, the majority of the patients remain asymptomatic.[1]

Here, we report a case of pancreatic AVM in a 45-year-old man who presented with upper gastrointestinal bleeding and duodenal ulcers. The AVM was successfully treated endovascularly by transarterial embolization (TAE).

Case Report

A 45-year-old man presented with complaints of melena and hematemesis for the past 1 day, along with abdominal pain and generalized tiredness. The patient had similar episodes of vague abdominal pain and early satiety 1 year ago for which an endoscopy was performed and he was diagnosed with antral gastritis. On examination, the patient was hypotensive (blood pressure [BP]: 90/60 mm Hg; pulse rate [PR]: 106/min). Routine blood investigations showed anemia (hemoglobin [Hb]: 6.7 g/dL), leukocytosis (14,520 cells/mm^3), and elevated erythrocyte sedimentation rate (ESR). Endoscopy was performed, which showed duodenal ulcer with

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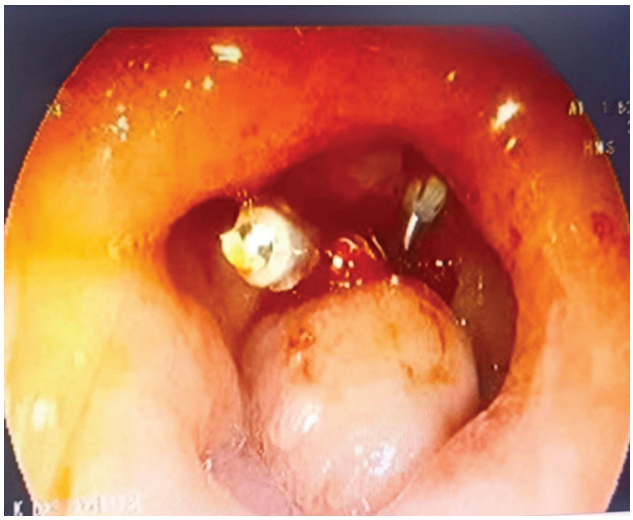

Fig. 1 Endoscopic image of a 45-year-old male patient who presented with upper gastrointestinal bleeding. Bleeding ulcer seen from the posterior duodenal wall for which two hemostatic clips were applied.

oozing blood for which clipping was done (►**Fig. 1**). The patient was managed conservatively with octreotide and pantoprazole infusion and blood transfusions.

Ultrasonography was done for the patient as part of routine workup, which showed a mildly bulky head of the pancreas with multiple intrapancreatic hypoechoic nodules, which on color Doppler showed vascularity (►**Fig. 2**). There were no findings of chronic liver disease or portal hypertension. In view of any mass lesion and to look for cause of collaterals, contrast computed tomography (CT) of the abdomen was planned. CT showed multiple dilated tortuous arterial channels in the head and proximal body of the pancreas, following similar enhancement to that of the aorta. The feeders were seen likely from the gastroduodenal artery (GDA) with venous drainage into the portal vein. Early

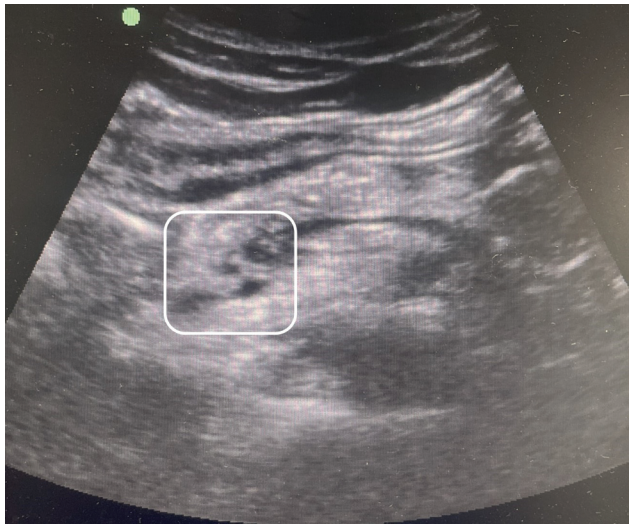

Fig. 2 Ultrasound image of a 45-year-old male patient with pancreatic arteriovenous malformation (AVM). Multiple hypoechoic nodules are seen in this image (*within the white box*), which showed vascularity on color Doppler (not shown here).

opacification of the portal vein was seen in the arterial phase. Based on these imaging findings, diagnosis of AVM of the pancreas was made (►**Fig. 3**). Once the patient's hemodynamic condition improved, he was advised TAE of the AVM.

Under local anesthesia, through the right transfemoral approach, celiac and superior mesenteric angiograms were taken. These showed multiple arterial feeders from the pancreaticoduodenal (PDA) branches of the GDA with early drainage and filling of the portal vein, suggestive of AVM. Selective cannulation of the PDA branches was done with a microcatheter and embolized with 350- to 500- and 500- to 700-µm poly-vinyl alcohol (PVA) particles. Postembolization, significant reduction of arterial feeders was noted with delayed (normal) filling of the portal vein (►**Fig. 4**). No significant feeder channels were seen from hepatic and splenic arteries or from the superior mesenteric artery (SMA). No periprocedural complications were seen.

At the time of discharge, the patient was clinically stable, with no further drop in hemoglobin values. Two weeks postprocedure, the patient came with complaints of vague abdominal pain with no episodes of melena or hematemesis. Blood investigations showed raised C-reactive protein (CRP) values; serum amylase and lipase were within normal limits. He was managed conservatively with intravenous fluids and antibiotics with no further complaints. Follow-up endoscopy 2 months later showed a healing ulcer in the duodenum. Plain CT showed a normal-appearing head of the pancreas with no obvious vascular channels. No features of pancreatitis or peripancreatic fluid collections were noted.

Discussion

Pancreatic AVM is a rare vascular anomaly with abnormal communication between the arterial and portal venous systems. Among visceral AVMs, less than 1% are seen in the pancreas.[2] About 90% of these AVMs are congenital, of which 10 to 30% can be associated with Osler–Weber–Rendu syndrome, an autosomal dominant disorder. The acquired causes of AVMs are usually secondary to pancreatitis, trauma, or rarely tumors. The majority of these pancreatic AVMs are seen in the head region.[3,4]

The clinical manifestations can range from vague abdominal pain and ulcers to bleeding and portal hypertension. Upper gastrointestinal bleeding, being the most common presentation, can be due to bleeding directly from the abnormal vessels into the duodenum, or from duodenal ulcer due to ischemic injury of the duodenal mucosa, or bleeding from the pancreatic duct, or from the abnormal varices due to portal hypertension.[5] Larger AVMs can cause shunting of blood away from mesenteric circulation resulting in chronic mesenteric ischemialike symptoms and vague abdominal pain, and long-standing increased flow into portal circulation can result in portal hypertension.[6]

Diagnosis is usually made with imaging and angiography. Ultrasound, which is usually the initial modality, can show multiple hypoechoic areas on B mode, which on color Doppler will show increased vascularity with a diffuse

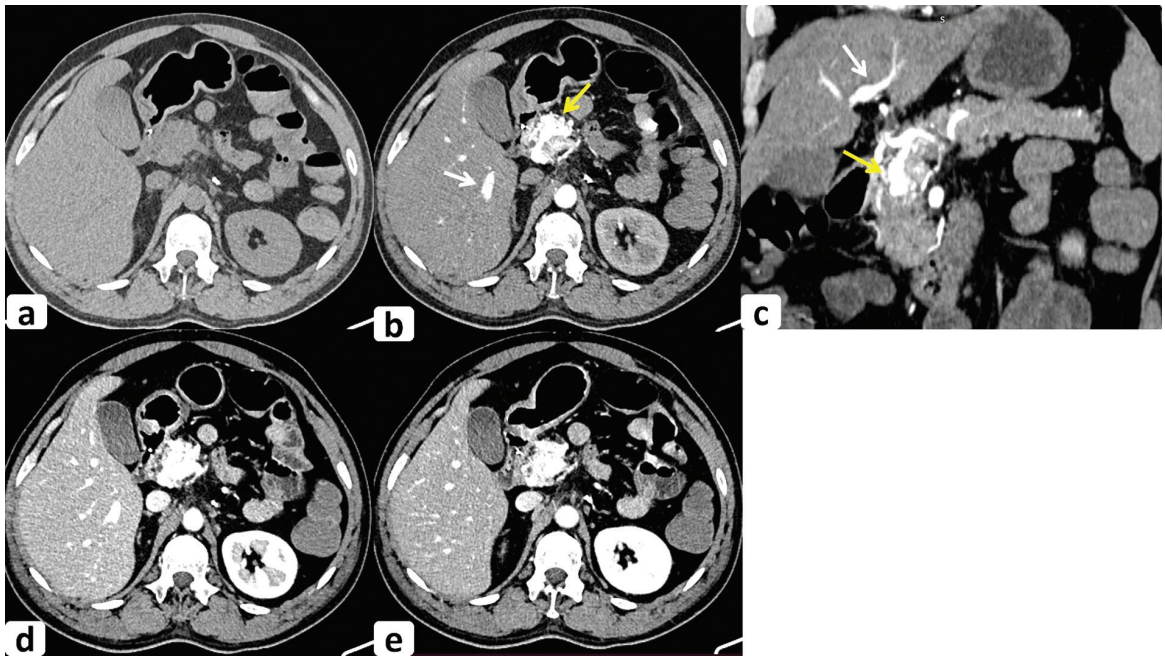

Fig. 3 Multiphasic computed tomography (CT) images of a 45-year-old male patient with pancreatic arteriovenous malformation (AVM). (**a**) Noncontrast axial image showing no obvious abnormality. (**b**) Arterial phase axial image and (**c**) arterial phase coronal oblique image, showing multiple arterially enhancing vessels in the region of the head of the pancreas (*yellow arrow*). Early opacification of the portal vein branches can be seen in this image (*white arrow*). Dilated feeder from the gastroduodenal artery can be seen in (**c**). No abnormality is seen in the rest of the pancreas. (**d**) Portal venous phase image and (**e**) hepatic venous phase image showing persistent enhancement of the vascular channels in the peripancreatic region, similar in enhancement to that of the aorta.

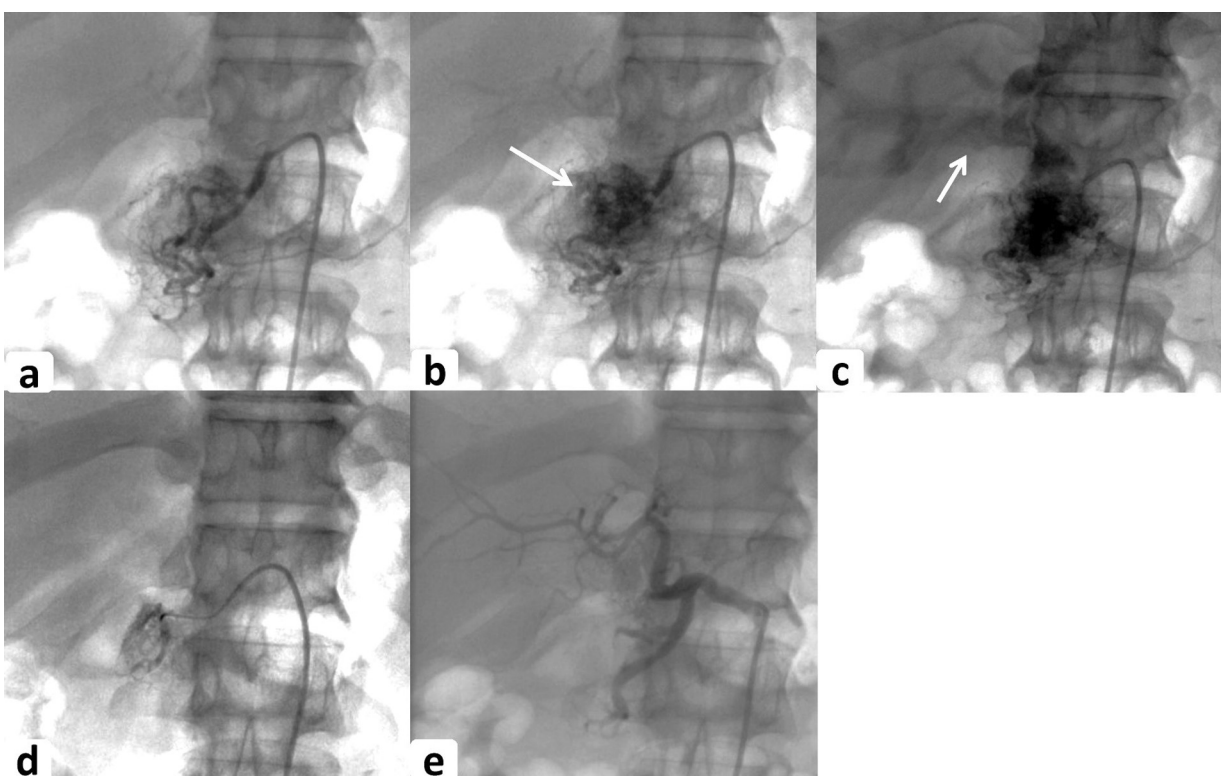

Fig. 4 Digital subtraction angiography (DSA) images of a 45-year-old male patient, who underwent transarterial embolization for pancreatic arteriovenous malformation (AVM). (**a–c**) Temporal images of a single angiographic run taken from the gastroduodenal artery (GDA) showing filling of a pancreatic AVM. (**a**) Dilated pancreaticoduodenal (PDA) branches of the GDA, with multiple small arterial feeders from these branches, are seen. (**b**) Complex nidus of AVM is seen (*white arrow*). (**c**) Early filling of the portal vein is seen (*white arrow*). (**d**) Selective cannulation of the PDA branches with a microcatheter, and angiogram showing dilated arterial feeders. (**e**) Postembolization with poly-vinyl alcohol (PVA) particles (300–500 and 500–700 µm), significant obliteration of the AVM nidus with normal filling of the GDA and hepatic arteries was observed.

mosaic color flow pattern and pulsatile flow within the portal vein. Multiphasic contrast CT will reveal the hypervascular lesion in the pancreas with enhancement pattern similar to that of the aorta. The arterial feeders can be well demonstrated on CT, with early filling of the portal vein on arterial phases. Additionally, CT will also show the extent and relation of the AVM to adjacent organs. Secondary complications of AVM, mainly portal hypertension, can also be assessed on CT based on the presence of abnormal portosystemic collaterals and gastroesophageal varices.[6,7] Angiography plays a crucial role in confirmation and also for planning the appropriate mode of treatment. It will depict the origin and extent of arterial feeders, complex nidal network, and the draining veins. Selective cannulation of hepatic artery, splenic artery, GDA, and SMA can show the presence of smaller feeders.

The main treatment options for pancreatic AVM include surgical resection such as pancreaticoduodenectomy or duodenum preserving pancreatic head resection, or TAE. Surgical resection is preferred as it can provide a complete cure and, if performed prior to the development of portal hypertension, offers a better prognosis for the patient.[1,3] TAE, on the other hand, offers a simpler yet effective method of treating AVMs without resorting to radical means. It is usually indicated in patients with acute bleeding symptoms, hemodynamic compromise, or larger complex AVMs with wide extent that cannot be completely removed or as a part of presurgical embolization.[5,8] The alternative options for patients with high surgical risk include transjugular portosystemic shunts or radiation therapies. Regarding asymptomatic cases of pancreatic AVMs, there is no general consensus of the timing or mode of treatment. However, it is postulated to treat such AVMs before the onset of portal hypertension, as it appears to be a major prognostic factor.

Conclusion

In conclusion, pancreatic AVMs are a rare entity among visceral AVMs and less common cause of gastrointestinal bleeding. The possibility of AVM should be kept in mind when encountering multiple vascular channels around the pancreas on ultrasonography or hypervascular lesions on contrast CT. The treatment options can be tailored according to the size and extent of AVM, number of arterial feeders, and hemodynamic condition of the patient. TAE offers an effective method for managing the bleeding episodes due to such AVMs, without undue morbidity for the patient.

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Conflict of Interest
None declared.

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