Percutaneous Transhepatic Approach for Recanalization of Superior Mesenteric and Portal Vein in a Patient With Pancreatic Neuroendocrine Tumor Presenting With Bleeding Duodenal Varices: A Brief Case Report

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Occlusion of Portal Vein (PV) and Superior Mesenteric Vein (SMV) is a known effect of local infiltration by pancreatic or mesenteric neuroendocrine tumors. Venous occlusion leads to formation of collateral pathways to restore hepatopetal flow in main PV and these collateral pathways can be seen in the form of ectopic (duodenal or jejunal) varices. We present a case of bleeding duodenal varices secondary to SMV occlusion by a locally infiltrating pancreatic neuroendocrine tumor which was successfully treated by coil embolization of varices and SMV stenting of the occluded venous segment after failure of endoscopic glue injection. Various endovascular minimally invasive approaches have been described in literature for recanalization of SMV in such clinical scenarios which maybe challenging to treat for surgical methods. We recommend use of the retrograde transhepatic technique for recanalization of occluded SMV and embolization of associated varices as an alternate treatment option in such scenarios. (J CLIN EXP HEPATOL 2018;8:318–320)

Pancreatic Neuroendocrine Tumors (PNET) comprise <2% of pancreatic tumors.¹ Non-functioning PNETs comprise the largest group and do not produce syndromes of hormone excess; rather, they cause morbidity and mortality by invading normal tissue and metastasizing.² Therapy for PNETs is primarily surgical resection for localized disease and selected patients with metastatic disease. However, options regarding systemic therapy for advanced disease are limited. Occlusion of Portal Vein (PV) and Superior Mesenteric Vein (SMV) is a known effect of local infiltration. Venous occlusion leads to formation of collateral pathways to restore hepatopetal flow in main PV which may present as ectopic varices. We present a case of bleeding duodenal varices secondary to SMV occlusion by locally infiltrating PNET which was successfully treated by coil embolization of varices and stenting of the occluded venous segment after failure of endoscopic glue injection.

CASE REPORT

An 82-year male presented to emergency with history of malena for 4 days. Clinical examination was suggestive of pallor. Hemoglobin measured 6.8 mg/dl. Liver function tests and renal function tests were within normal limits. Urgent blood transfusion was done followed by upper Gastrointestinal (GI) endoscopy which showed a compressible bulge on posterosuperior wall of duodenal bulb suggestive of duodenal varices. 2 ml glue was injected endoscopically. Subsequent CT angiography of abdomen revealed a large enhancing hyperdense pancreatic head mass with calcification within, encasing the SMV and superior mesenteric artery precluding tumor resection (Figure 1). Occlusion of SMV extended up to splenoportal confluence along with multiple venous varices draining from SMV to PV. Endoscopic ultrasound guided FNAC was suggestive of neuroendocrine tumor. In view of the

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Abbreviations: F: French; PNET: Pancreatic Neuroendocrine Tumor; PV: Portal Vein; SMV: Superior Mesenteric Vein

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Figure 1 Axial CT venous phase shows large lobulated heterogeneously enhancing mass lesion replacing pancreatic head with encasement of the superior mesenteric vein (straight arrow) and multiple collaterals along the duodenum (box arrow). The proximal main pancreatic duct is dilated with atrophy of the parenchyma around it.

Figure 2 Superior mesenteric venography shows the total occlusion of a segment of superior mesenteric vein extending up to the porto-splenic confluence (straight arrow) and re-filling of the main portal vein through extensive duodenal varices (arrowheads).

active bleeding, Interventional Radiology reference was sought.

The procedure was performed under general anesthesia in an angiographic suite. Transhepatic puncture of posterior branch of right PV was performed under ultrasound guidance and a 6 Fr vascular sheath (Cordis-Miami Lakes, Florida, USA) was inserted in PV lumen using Seldinger technique. Portal venogram was suggestive of main PV occlusion. A hydrophilic guide wire was used to cross the occluded segment and passed into the proximal SMV lumen. A 5 F kumpe catheter (Cook, Bjaverrkov, Denmark) was used for acquiring superior mesenteric venogram which revealed occlusion of a long segment of SMV extending into main PV, for a length of 3.5 cm. Multiple collaterals were also noted arising from the proximal SMV to continue with the main PV (Figure 2). PV, SMV and splenic vein pressures measured 12, 14 and 16 mmHg respectively. Two of the largest varices were selectively cannulated and embolized with 14 mm × 50 mm nester coils (Cook). Balloon venoplasty of SMV (6 mm × 40 mm—Evercross) was done followed by placement of uncovered self-expandable metallic stent (6 mm × 40 mm—Zilverflex, Cook) with distal end in PV. Post-stenting superior mesenteric venogram revealed patent stent with physiological hepatopetal contrast flow within the SMV and PV (Figure 3). The patient was chemotherapy intolerant and eventually died after 6 months due to progressive malignancy but there were no complaints of GI bleed in the period.

DISCUSSION

The rate of major vascular involvement in PNETs is 17%.³ The primary tumor or associated metastatic lymph nodes can result in stenosis/occlusion of mesenteric vessels.

Figure 3 Superior mesenteric venography after placement of the bare metallic stent (straight arrow) and embolization of the largest varices (arrowheads) shows the recanalized superior mesenteric vein with contrast opacifying the portal vein and intrahepatic portal branches. Near complete occlusion of the duodenal varices is noted with minimal contrast filling.

Carcinoids induce extensive desmoplastic reaction of adjacent mesentery resulting in retraction and shortening of fibrotic mesentery, leading to segmental occlusion, tortuosity and curved dislocation of mesenteric vasculature. Authors have documented different surgical strategies for resecting or debulking pancreatic/mesenteric carcinoid tumors. However, complete encasement of mesenteric vasculature has been considered a contraindication to surgery because of the risk of inducing prolonged

intestinal ischemia or uncontrollable bleeding. Few authors have reported a novel technique to resect extensive mesenteric root carcinoid disease using partial abdominal evisceration and intestinal auto transplantation.⁴ But the expertise for such an advanced technique is very limited.

Endovascular approaches to alleviate mesenteric venous obstruction are particularly valuable in such clinical scenarios. SMV invasion leads to intestinal congestion and may also present with upper GI bleeding secondary to ruptured ectopic varices. Duodenal variceal rupture can lead to severe hemorrhage, with mortality as high as 40%.⁵ The main goal in treating duodenal varices secondary to prehepatic PV obstruction is to relieve the mesenteric venous pressure. Restoration of physiological blood flow by endovascular means or by meso-Rex shunting is the treatment of choice.⁶ Other surgical shunts, such as mesocaval shunts have not proven to be equally effective and may be complicated by encephalopathy. Endoscopic techniques and interventional radiology are recommended first and second line therapy for bleeding ectopic varices respectively. Isolated transvenous embolization of varices and endoscopic cyanoacrylate injection are considered as limited options because of risk of complete occlusion of the only mesenteric venous outflow, which can result in catastrophic mesenteric ischemia. The pressure gradient across the occlusion in our case was not high. But gastric and ectopic varices are known to bleed at lower pressure gradients likely due to reduced portal collateral resistance. We used an uncovered stent in the scenario as an urgent palliative life saving measure for the GI bleeding since endoscopic intervention had failed and the patient was not fit for surgery. Patency rate of covered stent could have been better in case it was done in an elective setting with a better prognosis and median predicted survival. The other reason for choosing an uncovered stent was the long segment of the occlusion with few risk of trapping the inflow of few superior mesenteric venous tributaries post-stenting.

Literature search revealed several case reports and series of different endovascular approaches for mesenteric vein recanalization to palliate symptoms of intestinal congestion that often accompany mesenteric spread of midgut carcinoids. Kelay et al. described a case of retrograde stenting of an occluded SMV to treat life-threatening hemorrhage due to bleeding duodenal varices in a case of malignant carcinoid tumor.⁷ Percutaneous transjugular and trans-splenic recanalization of the PV occlusion have also shown promising results. Carnevale et al.⁸ achieved PV recanalization with access through an SMV tributary identified at minilaparotomy after unsuccessful transhepatic retrograde attempts. Sabri et al. also reported two

patients in whom bleeding of intractable duodenal varices was controlled with combined bidirectional transhepatic and transmesenteric technique.⁹ Hellman et al.¹⁰ published a case series of the insertion of self-expandable stents for the treatment of symptomatic SMV stenosis/obstruction in the cases of inoperable mesenteric masses (midgut carcinoid disease).

In conclusion, we suggest interventional transhepatic SMV/PV recanalization with ectopic variceal embolization as one of alternate treatment measures in advanced inoperable pancreatic/mesenteric malignancies with vascular stenosis/occlusion. If the retrograde route fails, then antegrade and combined routes can be attempted. However, long term patency rates of such porto-mesenteric venous stents depend on the biological behavior of the tumor and needs further research.

CONFLICTS OF INTEREST

The authors have none to declare.

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