



Introduction

Portal venous and superior mesenteric artery injuries are rare pathologies with iatrogenic, traumatic, infectious/inflammatory, and neoplastic etiologies. Treatment possibilities include embolization and excision with a covered stent. Here we report a case of sequential stenting of the portal vein (PV) and superior mesenteric artery (SMA) after biliary stent and cancer related vascular injuries.

Case Presentation and Intervention

67-year-old male Jehovah's witness with unresectable pancreatic adenocarcinoma status post chemo and radiation who presented with bright red blood per rectum and drop in hemoglobin from 9.5 to 5.3 g/dL. Multiphasic CT did not initially identify bleeding source. At endoscopy, clot was seen in the stomach and duodenum without active hemorrhage. Repeat endoscopy one day later confirmed hemobilia with manipulation of the existing metallic biliary stent. IR review of the prior CT identified a PV pseudoaneurysm communicating directly with the stented common bile duct (Figure 1). Transhepatic portography confirmed PV pseudoaneurysm which was then excluded using a stent graft (Gore Viabahn VBX) (Figure 2). The patient's hemoglobin stabilized and he was discharged.

Two months later the patient re-presented with recurrent bright red blood per rectum. Multiphasic CT demonstrated occlusion of the stented PV with new fistula between proximal SMA and the previously excluded PV pseudoaneurysm (Figure 3); arteriography confirmed the finding (Figure 4A). A small branch arising from the proximal SMA opposite the fistulous connection was coil embolized (to prevent endoleak) and a stent graft (Gore Viabahn VBX) was then placed in the SMA across the fistula. Completion angiography confirmed exclusion of the arteriovenous fistula (Figure 4B). The patient was subsequently discharged without further bleeding. He expired 5 months later due to other causes.

Figures

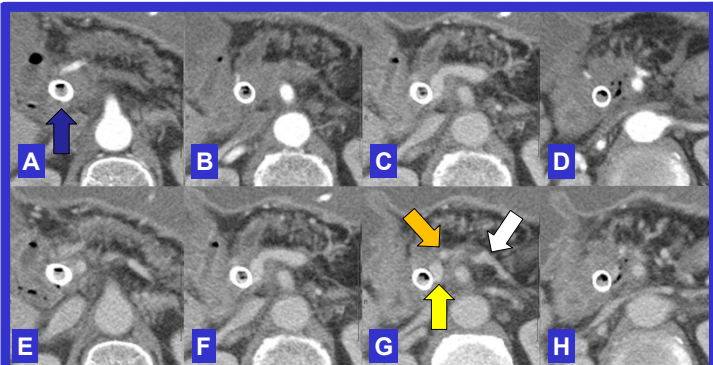


Figure 1: Axial images from superior to inferior in arterial phase (A-D) and venous phase (E-H) showing metallic biliary stent (blue arrow) with abutting portal venous pseudoaneurysm (yellow arrow). Note this extends away from the superior mesenteric vein and splenic vein (orange and white arrows respectively). Also note the subtle hyperdensity within the biliary stent.

Figures

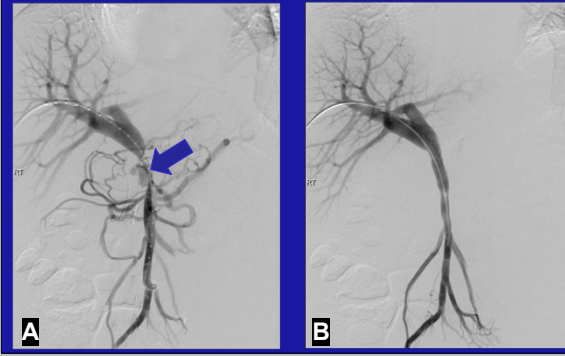


Figure 2: Digital subtraction venography of the superior mesenteric vein and main portal vein via transhepatic access (A). There is stenosis with pseudoaneurysm at the level of the metal common bile duct stent (blue arrow). (B) Portal venography after deployment of stent graft confirms exclusion of pseudoaneurysm with resolution of stenosis and elimination of collaterals.

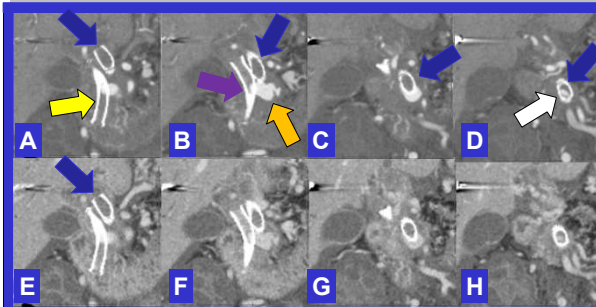


Figure 3: Coronal images from posterior to anterior in arterial phase (A-D) and venous phase (E-H) at the time of representation with GI bleed 2 months later. Thrombosed PV stent (blue arrow), biliary stent (yellow arrow), SMA pseudoaneurysm (orange arrow), extension of pseudoaneurysm into biliary stent (purple arrow), and arterial filling of SMV and inferior PV stent (white arrow)

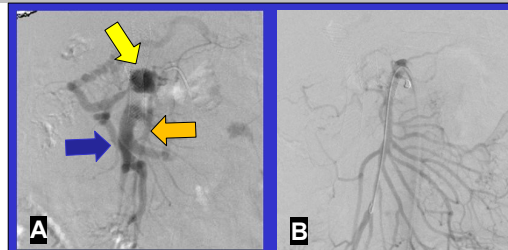


Figure 4: Superior mesenteric arteriogram before (A) and after (B) SMA stent graft deployment. Note preferential filling of the SMV (blue arrow) and the pseudoaneurysm (yellow arrow) with poor opacification of the SMA (orange arrow) before stent graft.

Discussion

Portal venous injuries in trauma have been reported to occur in only 0.08% of traumas (1). Historically many of these were treated with surgical repair or ligation, with mortality in nearly 50% of reported cases (2). Increasingly there have been reports of stent grafting for portal vein injuries. One relevant published report involved injury of the portal vein following endoscopic metallic biliary stent placement for treatment of biliary obstruction by pancreatic mass. This led to hemobilia and was treated with stent grafting over the fistula via transhepatic access, as was done in our case. Of note, the patient was not started on anticoagulation or antiplatelet medication due to bleeding risk and the stent remained patent up to 1 year follow-up (3). In our case, after stent deployment, anticoagulants were not consistently administered and, despite this being a potentially contaminated pseudoaneurysm, antibiotics were not continued; the PV stent graft was occluded at two months. Fistulous connections between the SMA and SMV related to trauma have been reported with delayed presentation related to portal hypertension due to arterioportal shunting (4). Endovascular treatment with coil or plug embolization has been reported (4, 5). Other reports of a three-way fistulous connection between portal vein, SMA, and the common bile duct were not found in the literature. The etiology of this complex fistulous connection is not certain, but we suspect it was due a combination of inflammation from an ascending infection of the space around the common bile duct stent combined with post radiation changes. One can speculate that PV stent patency may have been maintained and AV fistula may have been prevented if anticoagulation and antibiotics been continued.

Conclusion

Patients with pancreatic tumors and common bile duct stents have a small risk of visceral arterial and venous injuries. Recognition of these injuries on diagnostic scans by interventional radiologists is important as they are rare and sometimes subtle findings that can be overlooked by general radiologists, as was seen in our case. Overall understanding of the risk factors and outcomes are lacking due to the paucity of reported cases. We present a case where these injuries were effectively treated with the use of stent grafting.

References

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