PORTAL BILIOPATHY

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ABSTRACT

A 45-year old man with chronic abdominal pain due to chronic pancreatitis associated with pancreas divisum and obstructive jaundice requiring multiple ERCPs with biliary stenting presented with an episode of severe acute pancreatitis.

February 2015: Episode of severe acute pancreatitis (Baltazar E), the CT scan showed acute portal vein thrombosis (figure 1a).

April 2015: Follow-up CT abdomen demonstrates extensive venous collaterals surrounding the common bile duct (figure 1b).

Patient had multiple episodes of upper GI bleeding secondary to esophageal and gastric varices requiring repeated EGDs with endoscopic variceal ligation.

January 2018: Follow-up CT abdomen with cavernous transformation of the portal vein and associated enlargement of venous collaterals impinging on the CBD.

During **the next 2 years**, he presented two events of ascending cholangitis. In order to control the recurrence of variceal bleeding and cholangitis, in **October 2019**, he underwent **portal vein balloon angioplasty** by interventional radiology using a **combined transhepatic and trans-splenic approach**. The direct extrahepatic portal vein pressure decreased from **30 mmHg to 22 mmHg** immediately after the angioplasty. There were no further episodes of variceal hemorrhage after this procedure.

Immediately following the angioplasty he developed severe healthcare-associated cholangitis with acute pancreatitis and coagulopathy. He was treated successfully with broad-spectrum antibiotics and **ERCP with placement of a 10 mm x 8 cm self-expanding metal stent.**

September 2021: The patient achieved significant clinical improvement with resolution of portal hypertensive complications and has been free from cholangitis with normalization of liver synthetic function.

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INTRODUCTION

6 years of evolution with portal cavernomatosis and portal biliopathy, as well as its complications.

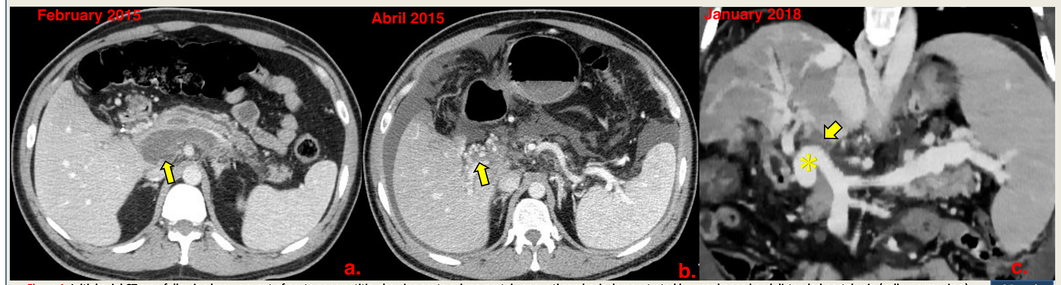

Figure 1. Initial axial CT scan following known event of acute pancreatitis, showing acute spleno-portal venous thrombosis demonstrated by unenhanced and distended portal vein (yellow arrow in a). Middle image three months later shows formation of small collateral vessels in the hepatic hilium, (yellow arrow in b) also note new onset ascites due to impaired liver function. Coronal reformat image two years later, points to focal area of chronic portal thrombosis, yellow arrow in (c) with more prominent appearance collateral vessels "cavernomatosis" (asterisk), several perfusion defects in liver parenchyma and marked splenomegaly.

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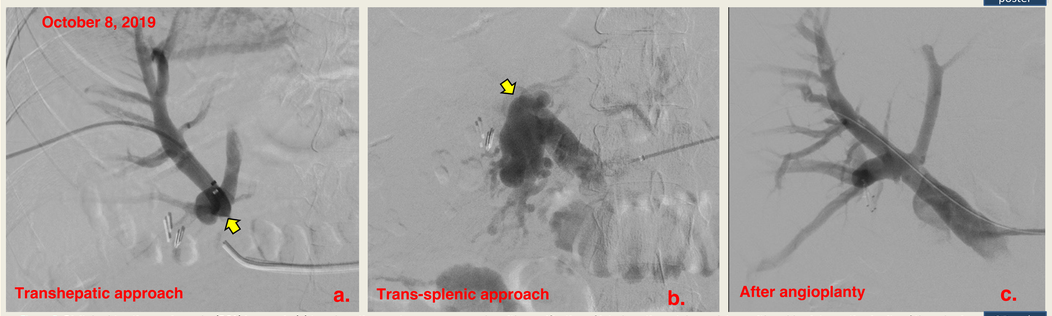

Figure 2. Digital subtraction angiography (DSA) images, in (a) transhepatic percutaneous venography with opacification of intrahepatic portal vasculature with sudden interruption in site of thrombosis (yellow arrow in a). (b) splenic approach with opacification of extrahepatic portal vein and aneurismatic collateral vessels due to cavernomatosis, and upstream thrombotic interruption (yellow arrow in b). (c) final imagen after portal vein angioplasty demonstrates adequate and complete opacification of portal vein and its branches.

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Figure 3. ERCP in (a) shows intrahepatic bile duct dilatation and impingement, and (b) after stent placement with diminished dilatation.

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For the follow-up of the patient´s laboratory tests use the code.

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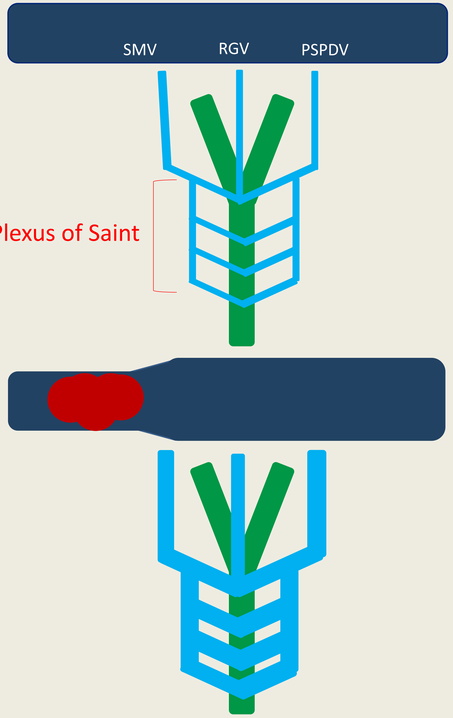
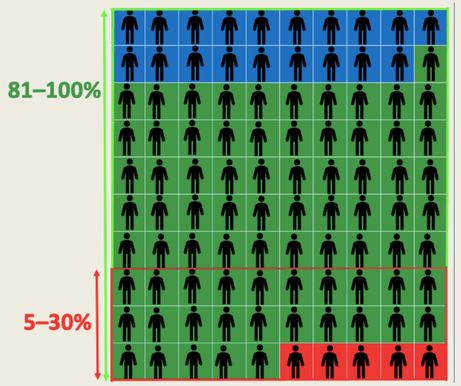
DISCUSSION



PVT global incidence of 0.05-0.5% in post-mortem studies (7). 5-year cumulative incidence of PVT in cirrhosis is 11% (6).



DISCUSSION

Biliary venous drainage goes through the epicholedochal venous plexus of Saint and the paracholedochal venous plexus of Petren. Portal biliopathy is due to the dilatation of the **plexus of Saint**, causing tortuous **collaterals and impingement** of theextrahepatic biliary ducts (1,5).

The high pressure leads to fibrous scarring of the porta hepatis, and ischemic injury to the bile ducts resulting in **stricture** formation and caliber irregularity (2).

Bile duct changes occur in 80–100% of patients with extrahepatic portal vein thrombosis. Yet, **only 5–30% of patients develop portal biliopathy** and present with clinically evident biliary obstruction (3, 4).

There should be a high index of suspicion for this diagnosis in patients who present with obstructive jaundice and have risk factors for thrombosis, including **surgery, sepsis, inflammation, and immobilization** (2).

CONCLUSIONS

Portal biliopathy is an infrequent, yet sometimes devastating complication of chronic extrahepatic portal vein obstruction and requires a multidisciplinary approach by interventional radiology and therapeutic endoscopy to **address the biliary obstruction and portal hypertension in tandem**, because the bile duct obstruction does not always turn back after the improvement in portal vein permeability.

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