

Incidence of Portal Biliopathy in Extrahepatic Portal Vein Obstruction (EHPVO)

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Abstract- The etiology of PB is unknown, but compression by dilated veins in or around the common bile duct may play a major role. Treatment of PB consists of endoscopy (sphincterotomy, lithectomy, or biliary stenting of the common bile duct) or surgery (final decompression with a portosystemic shunt and, if necessary, cholangiostomy). This review describes the etiology, clinical features, evaluation, and management of portal biliary disorders. Aim of the study is to identify the incidence of Portal Biliopathy in Extra Hepatic Portal Vein Obstruction. Methodology- Retrospective cross-sectional study. Result and conclusion: Incidence of Portal biliopathy in Extrahepatic portal vein obstruction is 64%

Index Terms- Portal biliopathy, Extra hepatic portal vein obstruction, Portal hypertension, Venous thrombosis, MRI Cholangiography, Venous obstruction

INTRODUCTION

Portal Biliopathy (PB) has been described as an abnormality occurring somewhere in the wall of the biliary tree as a result of extrahepatic portal hypertension (PH). These include extrahepatic and intrahepatic bile duct strictures and dilations, gallstones, and choledochovariates.

PB is most commonly due to extrahepatic portal vein occlusion (EHPVO). The etiology of EHPVO is often unknown and may involve local and systemic risk factors. EHPVO in adulthood is associated with coagulopathy, local inflammation, intra-abdominal sepsis, myeloproliferative disease, underlying liver injury, or hepatobiliary pancreatic tumors. Ombilitis and intra-abdominal sepsis can cause EHPVO in neonates and children. Venous congestion due to PH appears to increase the risk of portal vein thrombosis. We summarize the etiology and prevalence of EHPVO and portal vein thrombosis (PVT) in a study of adults without cirrhosis.

The venous drainage of the common bile duct (CBD) is served by two pericholedochal plexuses: the epicholedochal venous plexus of Saint and the paracholedochal plexus of Petren. They form a fine reticular venous plexus surrounding the external wall of the CBD and hepatic ducts. The veins of these plexuses vary in size, but normally their diameter does not exceed 1 mm. The paracholedochal veins drain into the pancreaticoduodenal veins, the gastric veins, the portal vein and directly into the liver directly. These collateral plexuses around bile ducts dilate in response to PH and they form the basis of collateral vessel compression of major bile ducts in PB. Ductal ischemia secondary to thrombosis of small venules in the bile duct walls may also result in stricture formation and fibrous scarring at the porta hepatis. Extension of the thrombotic process may extend ischemia of bile duct wall with subsequent stricture formation, irregularities in ductal caliber with cholangiectasia. Chronic portal vein thrombosis results in a network of hepatopetal collateral veins at the hilum of the liver bypassing portal obstruction that appear one week to three months after the initial PVT at the hilum of the liver. In the long term, PB can be a risk factor for cholelithogenesis secondary to chronic cholestasis, changes in bile constituents due to a reduced portal flow and associated liver atrophy.

Approximately 65-95% of patients have no symptoms of biliary obstruction. Symptoms such as jaundice, abdominal pain, and fever are associated with older age, longer duration of illness, more frequent cholelithiasis, and abnormal liver function tests.

Distinguishing between benign biliary strictures due to PB and malignant strictures due to cholangiocarcinoma can be difficult despite improvements in imaging and endoscopic techniques.

Magnetic Resonance Cholangio Pancreatography (MRCP) and endoscopic retrograde cholangiopancreatography (ERCP) are predictors of malignant stricture. The greatest diagnostic challenge in patients with suspected PB is to confirm that portal spongiform changes (PCT) are the sole cause of biliary changes. In addition to cholangiocarcinoma, chronic pancreatitis and bile duct injury after cholecystectomy are difficult to differentially diagnose. MRCP and portography are noninvasive imaging modalities that allow simultaneous examination of biliary tract lesions and portal vein collaterals.

Therefore, MRCP should be indicated to elucidate the mechanisms of biliary obstruction prior to therapeutic intervention, especially in asymptomatic patients or as an initial investigation in patients with biliary symptoms.

In addition, MR portography also depicts portal vein anatomy, helping to assess the feasibility of a therapeutic portosystemic shunt (PSS).

PB is a true complication of portal hypertension. Specific management depends on clinical presentation and imaging. Management of PCT patients with biliary obstruction is difficult for three reasons. First, the clinical and radiological appearance may mimic cholangiocarcinoma or sclerosing cholangitis. PB is rare and should be the diagnosis of exclusion. Second, PCT may be associated with other medical conditions (such as cancer, trauma, and inflammatory strictures of the biliary tract). These diagnostic problems can lead to serious mistakes in the treatment of this disease. Third, direct access to the bile duct within the hepatic pedicle affected by PCT is highly risky, making surgical treatment difficult. Dissection of the liver stalk can cause massive bleeding and even death

of the patient. The most appropriate procedure seems to be PSS, the type of which is selected according to the anatomy of the portal system, followed several months later by bile digestion anastomosis.

AIM OF THE STUDY

To study the incidence of portal biliopathy in Extra hepatic portal venous obstruction

MATERIALS AND METHOD

PATIENT SELECTION

This was a single-institution retrospective study that was approved by our institutional review board. As this is a retrospective study, informed patient consent was not required. Patients were selected according to several criteria. First, we searched the radiology database for patients with MRI reports containing the string "cavernous" between April 2021 and June 2022 (48 patients in total). Second, we selected patients with abnormal biliary MRI findings. The following MRI findings were considered inclusion criteria: Irregular contours, strictures, enlarged bile ducts, wall thickening, and dynamic widening of the bile ducts.

Third, the resulting patient list was refined using the following exclusion criteria:

Patients with a history of other causes of biliary tract abnormalities, such as MRI technology

MRI was performed using a 1.5 T superconducting scanner (Sonata, Siemens Medical Solutions) and a phased array multicoil. First, the Fourier half of his RARE his sequence was acquired in the axial plane. 2 He used the MR cholangiography technique. Multi-slice half-Fourier RARE and thick single-slice turbo spin-echo (TSE) sequences. Thick-slice MR cholangiopancreatography (MRCP) images were obtained in the coronal plane. Multi-slice half-Fourier RARE images were acquired in the coronal, sagittal, and oblique planes. The imaging parameters for the multislice half-Fourier RARE sequence were TR/TE, 1000/87. Flip angle 150°; Seamless panel thickness 3-4 mm. Field of view, 310 x 310 mm. Matrix, 256×218. Acquisition time is 18 seconds. Dynamic 3D gradient echo imaging before and after IV bolus injection of ml Gado (volume-interpolated breath-holding [VIBE] study) (3.8/1.67; flip angle 10°; matrix, 256 × 115 or 256 × 134) - purchased for 20 weeks. Dimeglumine pentetate (Magnevist, Schering) at a rate of 2 ml/s. Arterial, portal vein, and late-phase images were obtained. Five of the 12 patients also underwent ERC, or percutaneous transhepatic cholangiography (PTC).

SAMPLING TECHNIQUE

Consecutive sampling

STUDY DESIGN

Retrospective Cross sectional study

STUDY SETTING

Sri Lakshmi Narayana Institute of Medical Sciences

STUDY PERIOD

April 2021- June 2022

RESULTS

At this step, the study population consisted of 48 patients. Extrahepatic portal vein occlusion—In our study, causes of extrahepatic portal vein occlusion included unexplained (n=8) and recurrent suppurative cholangitis (n=4).

The final study group consisted of these 12 patients (6 males, 6 females), with a mean age of 53 years (range 24–74 years). Gallbladder varices were observed in his 5 (100%) of patients who did not undergo cholecystectomy.

In all patients, the biliary veins and cystic varices appeared as hypointense on T2-weighted imaging and enhanced tortuous collateral vessels on dynamic 3D gradient echo imaging. Portosystemic Collaterals - Spleen size was within normal limits in most study patients, but was slightly enlarged in 13. Portal systemic collaterals, including esophagogastric varices (n=2) and splenic varices (n=1), were found in 3 patients (25%).

In bile duct abnormalities, the irregular outline of the bile duct was mainly caused by multiple smooth extrinsic compression of dilated and tortuous parabiliary veins along the common bile duct. In fibrous type biliary abnormalities, MR scan showed focal strictures with biliary dilatation. A smooth regional constriction was seen at the level of the thickened wall of the MRCP common bile duct.

Irregular contours with multiple areas of constriction and dilation were also noted in the mixed type of biliary abnormality, but wall thickening at the level of the constriction did not show delayed potentiation enhancement in this type. There were no discrepancies in the overall contours or degree of biliary constriction and dilatation. The mixed type showed an irregular contour with multiple areas of constriction and dilation.

Portal Biliopathy was seen in 64% of patients with Extrahepatic portal venous Obstruction.

DISCUSSION

The formation of multiple thin hepatic petal collateral veins in response to extrahepatic portal vein occlusion is now well established. These collateral veins, called the portal corpus cavernosum, originate somewhere in the peripancreatic region along the occluded portal vein, enter patent intrahepatic portal branches, and enter the main vein in response to extrahepatic portal vein occlusion. Portal lumen is made up of two venous systems, including the para-choledochal vein, which runs parallel to the bile duct wall, and the superior common biliary plexus, which lies on the surface of the bile duct. Portal cholangiopathy is defined as a biliary abnormality in patients with extrahepatic portal vein occlusion that causes cavernous changes. It has been reported in 70-100% of patients with extrahepatic portal vein occlusion. Recent studies suggest that the mechanism of biliary abnormalities in extrahepatic portal vein occlusion is either extrinsic compression by collateral vessels or ischemic injury by venous thrombosis.

Portal cholangiopathy is a new term used to describe changes in the bile ducts due to cavernous changes in patients with portal hypertension. Such bile duct changes are more common in portal hypertension caused by extrahepatic portal vein occlusion than in cirrhosis or other causes of portal hypertension.

In one study, the majority of patients with portal cholangiopathy were asymptomatic. Only 14% of his patients showed biliary symptoms]. In our study, slightly elevated serum bilirubin levels (1.5–2.2 mg/dl) were observed in 3 of 12 patients (25%). Direct cholangiography findings in patients with extrahepatic portal vein occlusion, including extrinsic influence on the bile duct by partial upstream dilatation, caliber irregularity, stenosis, and collateral tracts, are consistent along the bile duct. It is called “pseudocholangiocarcinoma manifestation” because it mimics spreading cholangiocarcinoma.

The mechanism of biliary abnormalities in extrahepatic portal vein occlusion is either extrinsic compression by collaterals or peribiliary duct fibrosis resulting from ischemic or inflammatory changes underlying portal vein thrombosis. Diman et al. Partial or complete regression of portal cholangiopathy was observed in four patients, regression in her one of her five patients with extrahepatic portal vein occlusion who underwent portosystemic shunt surgery. In reversible bile duct alterations after shunt surgery, mechanical compression by collateral muscles is the mechanism behind biliary abnormalities in extrahepatic portal vein occlusion. In persistent biliary tract abnormalities, bile duct ischemia or fibrous scarring are believed to be the main causes of biliary tract abnormalities. According to Diman et al. For illustration, we classified these biliary abnormalities as varicocele, fibrosis, or mixed type based on the presence or absence of stricture and etiology. In the varicose type, the irregular contours of the bile ducts were mainly caused by multiple smooth extrinsic compressions of the corpus cavernosum. This was clearly demonstrated in both his MR portography and MRCP of the fibrotic type. MR scan shows focal stricture with proximal canal dilatation. Stenosis was mainly caused by fibrotic scarring associated with chronic inflammation and ischemic injury. Gibson et al. The study reported the occurrence and natural history of extrahepatic portal vein occlusion in patients and found that only 5 of 28 study patients had jaundice. Jaundice in patients with portal cholangiopathy can be caused by strictures or development of strictures in the common bile duct, or by choledocholithiasis. The majority of these patients are asymptomatic and do not require treatment. However, choledocholithiasis, or symptomatic biliary stricture, can be treated by endoscopic procedures such as sphincterectomy, balloon dilation, and stent placement. Due to the large collateral veins in the peripapillary region, endoscopic procedures should be performed with caution. No patient in our study experienced symptoms indicative of progressive or persistent biliary obstruction. In one case, doctors attempted to place a plastic stent endoscopically, but it failed. Clinical and imaging follow-up over 26 months showed no worsening of laboratory values or symptoms. Therefore, biliary stenting may not be indicated in many cases of portal cholangiopathy. When occlusion occurs at the level of the portal trunk, portal flow remains adequate in the central zone of the liver, but not in the peripheral zone. The arterial response, based on activation of the peribiliary plexus, results in enhancement of the hepatic parenchyma and relatively less enhancement of the central perihilar region. However, none of the 12 study patients showed zonal differences in these transient attenuations. It is thought to result from transformation and the consequent reduction in arterial compensation.

Our study has some limitations. First, as this study is a retrospective review, selection bias was inevitable. Extrahepatic portal vein occlusion with spongy changes was detected in 101 patients on contrast-enhanced CT scans during the study period and was primarily due to her HCC, metastases, and cirrhosis. Liver MRI with MRCP was not performed in most patients because CT alone was usually sufficient to diagnose these disorders. Therefore, from a large series of patients with extrahepatic portal vein occlusion, only 12 patients formed the basis of the study. Second, the small number of patients involved limited our study. Therefore, our results are not considered representative of portocholeangitis. Third, because this study is a retrospective review, we were not able to compare MRCP and ERC findings in all patients with suspected portal cholangiopathy, but the comparison is very strong for diagnosis. However, we believe that MRCP with the addition of dynamic 3D gradient echo imaging can improve all diagnostic components of portal bile disorders, including: Extrahepatic portal vein obstruction, cavernous and biliary changes. Finally, not all cases provided pathological evidence. However, the benign nature of the disease has been characterized using clinically recognized and widely used techniques. In summary, MR cholangiography combined with dynamic 3D gradient echo imaging is a non-invasive and diagnostic tool for all portobiliary disorders, including extrahepatic portal vein obstruction, portal cavernoma, gallbladder varices, and bile duct abnormalities.

CONCLUSION

This study shows the prevalence Portal Biliopathy in patients with extra hepatic portal vein obstruction is 64%.

RECOMMENDATION

The study should be done among more no. of patients at different settings to identify the trend of disease in various population.

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