References


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Ischemic stricture of Roux-en-Y intestinal loop and recurrent cholangitis

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The most common complication of hepaticojejunostomy for the management of biliary strictures is recurrent cholangitis. We report a 54-year-old man who underwent choledochojejunostomy after choledochal cyst excision, and later developed ischemic stricture of the Roux-en-Y loop intestinal loop and recurrent cholangitis. The strictureal intestinal loop was excised with re-anastomosis with new Roux-en-Y loop, with uneventful recovery. [Indian J Gastroenterol 2005;24:76-77]

**H**epaticojejunostomy is a common curative and palliative procedure carried out for benign and malignant biliary obstruction. In spite of being a major procedure, it is tolerated well, with very few complications. Intestinal loop stricture after choledochojejunostomy is an uncommon cause for recurrent cholangitis.

A 54-year-old man underwent choledochojejunostomy six months prior to presentation to our unit. Two weeks after surgery he started complaining of colicky pain in the right hypochondrium and paraumbilical region that increased after meals. There was history of intermittent fever with rigors and chills for three months, but no history of associated jaundice.

On presentation the patient was mild icteric and afebrile; per abdomen examination was normal.

**Investigations:** Serum bilirubin 2.3 mg/dL, AST/ALT 34/25 IU/dL, serum protein 6.8 g/dL (albumin 3.9), prothrombin time 13.8 s (control 13) and alkaline phosphatase 765 units/dL (normal 15-117). MR cholangio-pancreatography revealed thickening at the site of anastomosis between the common bile duct and jejunum, with intrahepatic biliary radical dilatation. There was no focal hepatic lesion or intra-abdominal collection. Percutaneous transhepatic cholangiogram showed narrowing of the intestinal loop with thickening at the site of anastomosis with intrahepatic biliary radical dilatation. Bile culture was positive for *E. coli* and *Klebsiella pneumoniae*, which was treated with appropriate antibiotics.

The patient was re-explored; the afferent jejunal loop of Roux-en-Y till the antecolic window was fibrotic, while the loop distal to the window was normal. There was fibrosis at the previous anastomotic site with dense adhesions. The fibrotic afferent loop was excised and distal loop was closed primarily. Fresh Roux-en-Y loop was constructed with re-freshening of the edge of the common hepatic duct. Postoperative recovery was uneventful. Postoperative percutaneous cholangiogram showed normal anastomotic site with free spill of dye into the afferent loop and no narrowing or leakage of the contrast. The patient is asymptomatic and non-icteric for the last two months.

The resected specimen showed a stricture 3 cm long (Fig). Cut section of the corresponding area showed necrosis. The rest of the intestine was narrowed and thickened. Microscopy revealed extensive mucosal ulceration, marked submucosal fibrosis, and granulation tissue with sprinkling of inflammatory cells (lymphocytes, plasma cells and neutrophils), patchy destruction of muscularis propria, with fibrosis, granulation tissue and mixed inflammatory infiltrate, organized exudates and granulation tissue in the serosa. No lymphoid aggregates/granulomas were seen. There was vascular proliferation. However no vascular occlusion or thrombosis was seen. Features were suggestive of ischemic stricture.

Aside from cholangitis secondary to anastomotic stricture, reconstruction of biliary tract with intestinal tract using either hepatico-duodenostomy or Roux-en-Y
hepatico-jejunostomy is generally regarded as innocuous. The development of cholangitis is usually assumed to be due to obstruction of the stoma. Other causes include primary and coexistent pathogenic factors like intrahepatic stricture, intrahepatic calculi, improperly constructed enteric conduits, and rare causes like volvulus of the afferent loop.

Though we could not demonstrate a vascular block, the histology in our patient suggested ischemic etiology. This could have been due to vascular occlusion or ischemia due to kink in vessels. This resulted in biliary stasis and recurrent cholangitis. Percutaneous dilatation could not be done as the intestinal loop stricture was long and irregular. Hence, it was treated with surgical excision. Another cause of obstruction is anisoperistaltic loop causing recurrent cholangitis but this was not the case in our course. Intestinal stricture following choledochojejunostomy has not been reported.

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Cholestatic liver injury due to ibuprofen

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Ibuprofen is a member of the propionic acid class of NSAID. We report a 35-year-old man with ibuprofen-induced acute severe cholestatic liver injury. He recovered after seven months. [Indian J Gastroenterol 2005;24:77-78]

Almost all the nonsteroidal anti-inflammatory drugs (NSAID) currently available have been reported to cause liver damage that can range from minor transient abnormalities in liver function tests to fulminant hepatic failure. Ibuprofen is a member of the propionic acid class of NSAID, which is widely used for treatment of inflammatory conditions and musculoskeletal pain. Only a few cases of ibuprofen injury have been reported.

A 35-year-old man presented with history of road accident leading to minor abrasions and spasm of the left ankle. The patient was treated with injection tetanus toxoid and oral ibuprofen. After ingesting the first dose of 400 mg ibuprofen, the patient developed maculopapular skin rash with itching on the whole body within two hours. He was treated with antihistaminics and hydrocortisone; ibuprofen was withdrawn. The skin rash disappeared within five days, with residual hyperpigmented spots. Seven days later, the patient developed jaundice and pruritus, which were progressive. There was no associated abdominal pain, fever or anorexia. Physical examination revealed icterus, hyperpigmented spots on the whole body, and scratch marks. Examination of the abdomen did not reveal any abnormality.

Investigations: hemoglobin 11.5 g/dL, total leukocyte count 8600/cmm (P 76%, L 20%, M 4%); prothrombin time was 14 s (control 14). Serum was negative for IgM anti-HAV, IgM anti-HEV, HBsAg, anti-HCV antibodies, anti-nuclear antibodies, anti-smooth muscle antibodies, and anti-mitochondrial antibody. Blood lipid profile, X-ray chest, ultrasonography of the abdomen, and upper gastrointestinal endoscopy were normal. ERCP done to exclude sclerosing cholangitis revealed normal bile duct and intrahepatic biliary radicals. Liver biopsy after nine weeks of icterus revealed maintained liver architecture, inflammation, marked cholestasis, and spotty necrosis of hepatocytes with focal areas of ballooning and fatty changes (Fig).

The jaundice, pruritus, serum bilirubin and alkaline phosphatase increased gradually for three months after the onset of icterus. The biochemical abnormalities remained static for two months and then started decreasing gradually over the next two months. The patient was treated with antihistaminics and ursodeoxycholic acid (25 mg/Kg/day) till his jaundice and pruritus subsided and the liver function tests became normal. Seven months after the onset of hepatotoxicity there was complete improvement in jaundice, pruritus and tests of liver function.

Fig: Liver histology showing maintained liver architecture, inflammation, marked cholestasis, and spotty necrosis of hepatocytes with focal areas of ballooning and fatty changes (H&E, 40X)