A RARE CASE OF RECURRENT ACUTE PANCREATITIS-ANOMALOUS PAN-CREATO-BILIARY UNION

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Abstract:
Recurrent acute pancreatitis is defined as more than one episode of acute pancreatitis. These patients need a more extensive workup apart from the detailed history, laboratory evaluation and transabdominal ultrasound or computed tomography. The most common causes are due to alcohol abuse or gallstone disease. Recent developments in imaging techniques and endoscopic retrograde cholangio-pancreatography have improved the clinical diagnosis of rare etiologies like anomalous pancreatico-biliary union. In this case report, we report a adolescent female with recurrent abdominal pain due to recurrent acute pancreatitis since childhood with APBU and Choledochal cyst with a common hepatic duct membranestricture

Keyword: anomalous, pancreaticobiliary, recurrent, pancreatitis

INTRODUCTION:
Anomalies of the pancreaticobiliaryunion are the congenital variations. They are uncommonly symptomatic but may present with associated conditions ranging from benign acute abdominal pain to carcinomas. Recent developments improved the clinical diagnosis and management of APBU with their associated complications. Unpredictable onset of symptoms and remissions characterize the long-term course of APBJ

CASE REPORT:
A 14 years old female was admitted with recurrent episodes of upper abdominal pain over the epigastrium, since the age of five years and was on symptomatic treatment and was referred for further evaluation of abdominal pain. She had epigastric pain which was dull aching and non-radiating. The pain increased on food intake and was partly relieved after two hours. The pain increased on lying supine, during the episodes. It was not related to fatty meal. She also had early satiation. She had no other significant history or clinical examination findings. On evaluation she had an apparently normal complete hemogram, renal function and liver function test. Her serum lipase level was 136 IU/L.
Upper gastrointestinal endoscopy was normal. Ultrasonogram revealed a fusiform dilatation of proximal CBD from the level of portaehepatis for a length of 4.5cms, with a maximum diameter of 23mm. No evidence of calculi or mass visualized. MPD appears dilated -4.4mm in its entire length with a 3.3mm calculi at the level of head region. MRCP showed multiple CBD webs(CHD-CBD and distal 1/3 of CBD) with tubular dilatation of extrahepatic bile ducts(CHD-10.7mm and CBD-13mm) suggestive of choledochal cyst and smooth dilatation of dorsal pancreatic duct(5.4mm) due to narrowing of accessory duct suggestive of pancreatic divisum Fig1. She was taken-up for ERCP which showed a normal ampulla, through which cannulation was done. Cannula was seen entering the CBD and iohexol(water soluble) contrast was injected, which showed dilated CBD with web like membrane in proximal CBD at the level of junction of cystic duct and delayed filling of contrast into pancreatic duct which was dilated and a long common channel of nearly 2.5cms without any filling defect, suggestive of choledochal cyst Type Ic with Anomalous pancreatico-biliary union PB-type Fig.2. She was takenup for hepaticojejunostomy. Peroperatively, the presence of choledochal cyst typeI measuring 4x8cms was presentwithpericystic adhesions with short cystic duct and a normal gall bladder with abnormally long pancreatobiliary junction. Through a right subcostal incision, Kocherisation was done and choledochal cyst was dissected from hilum to pancreatic duct junction. Abnormally long pancreatobiliary junction of nearly 3cms was confirmed. Hepaticojejunostomy and jejunoojejunostomy was done.

**DISCUSSION:**
The main pancreatic duct and the common bile duct open into the second part of the duodenum either separately or after joining as a common channel. A common channel of >15 mm (an anomalous pancreaticobiliary duct) is associated with congenital cystic dilatation of the common bile duct and carcinoma of the gall bladder. In the present case the common channel was more than one vertebrae height, which is more than 2.5cms. Even a long common channel (8 mm) is associated with a higher frequency of carcinoma of the gall bladder. In the pre-endoscope era, a common channel was noted in 20-90% of the general population at necropsy and in 7-50% by cholangiography.\(^1\)
A common channel was found in 74% of specimens, 7% of which had interposed septum and 19% separate openings for the two ducts. The length of the common channel in normal people ranges from 1-12 mm, with a mean of about 4-5 mm. In APBD the connection between the common bile duct and the main pancreatic duct is located outside the duodenal wall and is therefore not under the influence of the sphincter of Boyden. The frequency of APBD varies from 1.5-3%. The highest incidence of 3-2% was reported by Kimura et al and Unozawa et al. If it appears that the pancreatic duct is joining the common bile duct it is denoted as P-B type and if the common bile duct appears to join the main pancreatic duct it is denoted as B-P type. In the present case the pancreatic duct was seen joining the common bile duct well outside the duodenal wall. Kimura et al have shown that the contractile segment of the common channel, in APBD, ended well below the common channel. APBD is a result of uneven proliferation of bile duct epithelium during fetal life. The union of the common bile duct and the main pancreatic duct is located lateral to the duodenum up to the eighth week of gestation and then it shifts medially to lie finally within the duodenal wall. Failure of this movement result in APBD. In 1906 Arnolds noted an association APBD and congenital dilatation of the bile duct. Ono et al found APBD in 15 (68%) of 22 patients with congenital cystic dilatation, while Sameshima et al found APBD in 47.5% of patients with congenital cystic dilatation. There was a imaging feature suggestive of choledochal cyst in the present case both in MRCP and ERCP. A study from Japan noted the P-B union in nine of 15 (60%) of cases. Arima and Akita noted the B-P type in 66% and P-B type in 34% of patients. The exact cause of congenital cystic dilatation is not known. Babbitt et al proposed that because of the abnormally long common channel maldevelopment of the sphincter of Oddi occurs, which results in a reversed pressure gradient between the common bile duct and the main pancreatic duct. This leads to regurgitation of the pancreatic juice into the common bile duct and repeated attacks of cholangitis. This results in thickening of the common bile duct wall, stenosis, and finally dilatation. Cylindrical congenital cystic dilatation may be accounted for by an APBD union, with resultant reflux of pancreatic juice into the common bile duct.

BIBLIOGRAPHY:


