anatomical relationship of the colonic segment to the pancreas and other associated complications of pancreatitis like abscess formation.[2,3,7] Colonic stenosis can either manifest early due to necrosis, ischemia and formation of an inflammatory mass or can present months to years after the inciting episode of pancreatitis consequent to retraction of the colon or by compression by a pancreatic pseudocyst or as a consequence of pericolic fibrosis.[3,5,7] Hunt and Mildenhall[7] have postulated that mesenteric ischaemia associated with a severe attack of pancreatitis may lead to fibrosis and stricture formation in the long term. Pancreatic carcinoma invading the colon has also been described in a patient of CP.[5] Enzymatic, inflammatory and ischemic processes are believed to be involved in the pathogenesis of colonic complications.[7,8]

The most common site of colonic stenosis is at or around the splenic flexure probably due to anatomical proximity to the tail of pancreas. However involvement of other sites is also reported.[3] Colonic stenosis frequently presents with intestinal obstruction. It can also mimic inflammatory bowel disease (IBD), malignancy, tuberculous stricture and nonsteroidal anti-inflammatory drugs (NSAIDS) induced CS.[6,9,10] Asymptomatic CP presenting as CS has been described in few case reports and poses a diagnostic challenge.[5,6] Barium enema is a good modality to diagnose CS however, in atypical presentation other imaging modalities and histopathology examination of stricture tissue is warranted. In recent years EUS has emerged as a more sensitive tool than conventional tests like ERCP, CT scan and MRCP in diagnosing CP.[11] In this case CT scan had failed however, EUS detected early changes of CP. Therefore, in cases of unexplained inflammatory CS with a high clinical suspicion of pancreatitis, undertaking EUS may prove rewarding. Patients of CS due to a complication of pancreatitis usually have good prognosis after surgery, as we seen in our patient.[3,6]

Our patient was a chronic alcoholic, and had had a prior episode of acute alcoholic pancreatitis. He presented with acute large bowel obstruction due to CS and on evaluation underlying asymptomatic CP was found. There was no other obvious causative factor which could have incited the colonic stenosis. Presence of calcification surrounding the stricture segment and histopathological evidence of chronic inflammatory cells with fibrosis in the resected specimen suggest a chronic inflammatory process as the possible mechanism for stricture formation. CP or prior episode of acute pancreatitis (acute on chronic pancreatitis) could be the cause of necrosis and subsequent chronic inflammatory reaction.

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Congenitally large gall bladder: a surgical surprise

Introduction

An anatomically normal and healthy gallbladder in humans measures 7-10 cm in size. Although some variation in its size is

common but massive congenital enlargement is rare. We report a congenitally large gallbladder measuring 17.5×3 cm, the second largest gall bladder reported so far.

Case report

A 33-year-old lady presented with history of episodic colicky pain in right upper abdomen of 1 year duration. There was no history of jaundice or fever. Clinical examination was unremarkable except for mild tenderness in right upper abdomen on deep palpation. Laboratory investigations including liver function tests were within normal limits. Ultrasonography of abdomen revealed a gallbladder (GB) filled with multiple calculi. The size of GB was not mentioned, common bile duct (CBD) and intra-hepatic biliary radicals were reported normal. A diagnosis of biliary colic secondary to cholelithiasis was made and the patient was planned for open cholecystectomy. During cholecystectomy, minimal pericholecystic adhesions and a GB measuring 17.5×3 cm with multiple stones was found (**Figure 1**). No stone was palpable at GB neck or cystic duct. CBD was not dilated and no stones were palpable in it. There was no mucocele or pyocele. The patient made an uneventful recovery. In the absence of any evidence of biliary tract obstruction, this massive enlargement of GB was considered congenital.

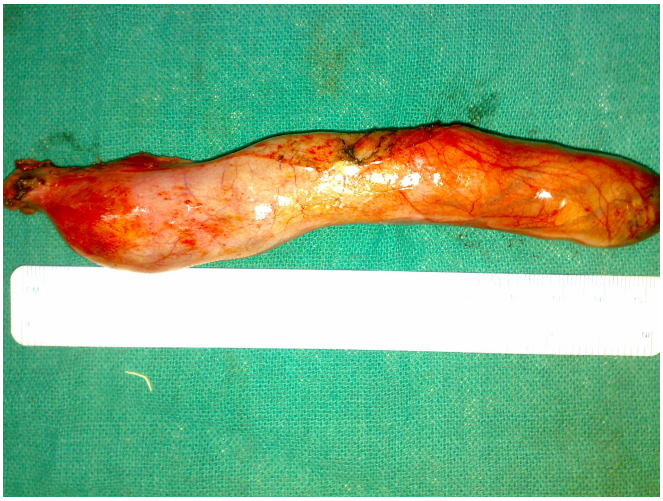

Figure 1: Long gall bladder

Discussion

Normal size of GB in humans is 7-10 cm with a volume of 30-50 cc.[1,2] Some variation in the size of GB is common but its massive congenital enlargement is rare. The gall bladder may enlarge in some pathological conditions like mucocele, empyema, carcinoma, acromegaly[3] (as a part of generalised visceromegaly), aneuploidy[4] and GB volvulus.[5] Congenitally large GB is extremely rare and only one such case has been reported so far.[6] We encountered a huge GB, in all probability a congenital one and hence, considered it worth reporting. During laparoscopic cholecystectomy, such enlarged GB may be a cause for potential misidentification of structures thereby predisposing to increased incidence of iatrogenic injuries. An enlarged GB may be mistaken for hepatic flexure of colon or duodenum making the task of the surgeon arduous. One may be compelled to place the sub-hepatic port more laterally or at times may have to place additional ports for handling and adequate traction of such large GB. During its dissection from the liver bed, the increased size and consequently more handling may predispose adjacent structures like duodenum, colon, and stomach to iatrogenic injuries. Identification of a large sized gall bladder pre-operatively may help the surgeon in making appropriate changes in the operative steps for cholecystectomy.

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