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Anomalous Pancreatico-Biliary Junction – A Non-Dilated Biliary System and Gallbladder Carcinoma

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ABSTRACT

Anomalous pancreatico-biliary junction (APBJ) is commonly associated with cystic dilatation of the bile ducts but recently, several cases without the cystic dilatation have been reported. We treated a young female patient with intractable back and epigastric pain of three months duration. The spine was normal on magnetic resonance imaging (MRI), but several lymph nodes were seen around the coeliac axis. An APBJ, a non-cystic biliary system, non-filling of the gallbladder and an irregular right margin of the bile duct were evident on endoscopic retrograde pancreatography (ERCP), which was highly suggestive of gallbladder (GB) malignancy. At surgery, the GB was hard with local infiltration of the bile duct. Numerous large para-aortic and supraduodenal lymph nodes were present and only a biopsy was possible. Details of the case are presented and the growing etiological importance of an APBJ, especially without cystic biliary dilatation in gallbladder carcinogenesis is discussed.

Keywords: long common channel; acalculous gallbladder; non-cystic ducts; malignant potential

CASE REPORT

A 39-year-old female first experienced sudden onset of severe pain in her back in October 1993, after lifting some heavy objects. She did not respond to analgesics but developed severe epigastric pain. Erosive gastritis was seen on gastroscopy, while upper abdominal ultrasonography was considered normal. A magnetic resonance imaging (MRI) was done to determine the cause of the intractable back pain. This showed a normal spine but multiple matted lymph nodes were seen in the region of the pancreas and coeliac axis (Fig 1). An APBJ, non-cystic bile ducts, indentation of the right border of the CBD below the confluence and non-filling of the gallbladder were documented on ERCP, which was suggestive of GB malignancy (Fig 2).

At laparotomy, the gallbladder was small, hard and puckered and the porta hepatis was frozen with multiple matted nodes in the hepatoduodenal ligament along the hepatic artery and the entire peripancreatic area. The GB lumen was full of obvious tumour and only a biopsy was posssible. This proved to be a well-differentiated adenocarcinoma on histology. Jaundice developed in 3 weeks and endoscopic stenting was unsuccessful. A week later, she succumbed to the disease (4 weeks after surgery and 5 months after the first symptom).

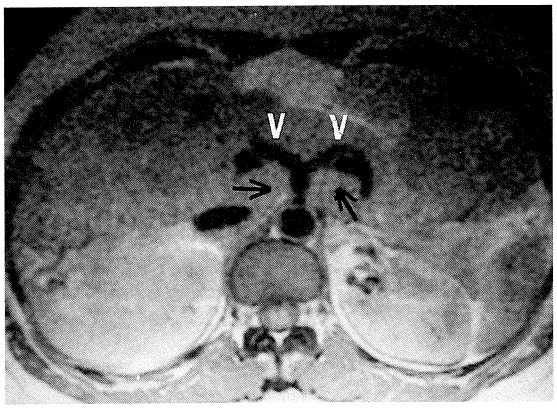

Fig 1 - Magnetic resonance imaging (MRI), showing numerous large lymph nodes around the coeliac axis (arrows) causing splaying with the sea gull sign (arrowheads).

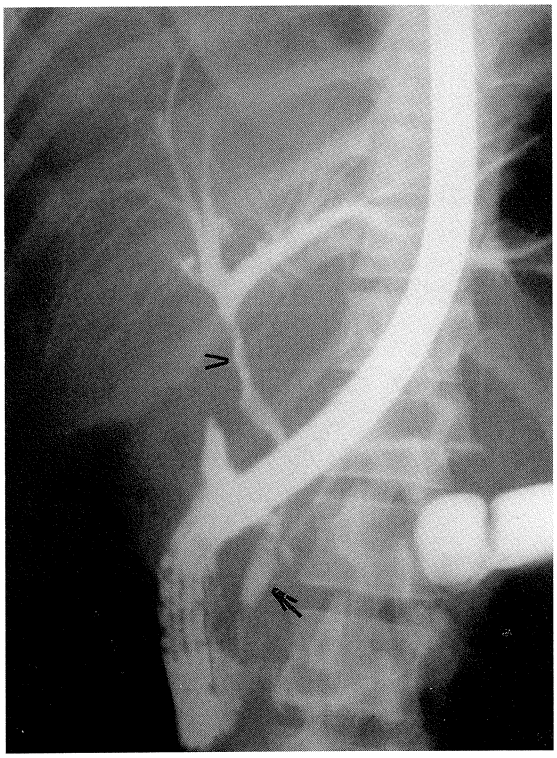

Fig 2 - ERCP showing the irregular right margin of the proximal common bile duct (arrowhead) and an APBJ with a long common channel (arrow). The intrahepatic ducts are normal and the gallbladder is not outlined.

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DISCUSSION

Anomalous pancreaticobiliary junction (APBJ) has been associated with cystic biliary dilatation or choledochal cysts since Babbitt first proposed the common channel theory[1]. Being independent of sphincteric control in patients with APBJ, pancreatic enzymes continuously reflux into the biliary system, leading to subclinical inflammation. This long standing inflammation within the bile ducts is now accepted as being potentially malignant[2]. Recently, APBJ has also been documented in patients without the cystic component and interestingly, has a higher incidence of GB cancer as compared to cases with choledochal cystic dilatation[3]. Mucosal dysplasia is believed to be the most common pathway to malignant change[4], and tumours excised at this stage have a better outcome[5] than after malignancy is established, as in our patient. It is being increasingly reported that APBJ with choledochal cysts is associated with cancer in the cyst wall, and those without the cysts tend to be associated with gallbladder cancer[2,3]. Although well-differentiated, this tumour behaves aggressively as evidenced in our patient who succumbed only 5 months after developing symptoms. Generally, gallbladder cancer affects the elderly and over 75% are associated with calculi causing chronic irritation with mucosal change. In contrast, our patient was young and no stones were found in the gallbladder. The reason for presentation at a younger age may be because the pathological mucosal changes with APBJ begins soon after birth and have at least a 20 – 30-year head start on calculi. A raised biliary amylase level would confirm a functioning APBJ with pancreatic reflux but was not possible in our patient since there was no bile in the gallbladder. The discovery of an anomalous junction appears sufficient to warrant a prophylactic cholecystectomy and common bile duct excision, irrespective of ductal status. This procedure separates the pancreatic and biliary systems and also removes the common sites of dysplastic and malignant change[2,3,5]. This anomaly must be recognised when present and once documented, must be treated adequately, in an effort to prevent malignant change.

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CORRIGENDUM

The Editor of the SMJ wished to apologise for the inadvertent errors made in the following article: SMJ 1997; Vol 38 (12): 535-539.

1. The second author should be S Simarak
2. Fig 4c has been mislabelled as Fig 4b and vice versa
3. Table II: Comments, 3rd line should be "cavitate" and not "caviate"
4. Page 538, 2nd paragraph, line 15: "soil saphrophyte" and not "soils saphrophyte"
5. Page 538, 2nd paragraph, last line: "trimethoprim-sulfamethoxazole"
6. Page 538, 4th paragraph, line 17: correct reference is 4, 17-20 and not 4, 7-20.