Pancreas divisum: correlation between anatomical abnormalities and bile precipitation in the gallbladder in seven patients

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SUMMARY: Pancreas divisum: correlation between anatomical abnormalities and bile precipitation in the gallbladder in seven patients.


Pancreas divisum is a genetic defect associated with recurrent acute pancreatitis due to insufficient drainage of the accessory pancreatic duct. Seven young patients diagnosed with pancreatic divisum and thickening of the gallbladder bile as shown on magnetic resonance cholangiopancreatography without pancreatic ductal changes underwent laparoscopic cholecystectomy. During the mean follow-up of 32 months no episode of pancreatitis was reported. There is an association between PD and higher concentration of bile in the gallbladder. Cholecystectomy can be considered curative in patients with PD in the absence of indications for major surgery.

KEY WORDS: Pancreas divisum - Laparoscopic cholecystectomy - Pancreatitis.

Introduction

The pancreas divisum (PD) is the most common congenital anatomical anomaly of the pancreas. It is found in 7% of autopsy series (1, 2). PD is characterized by a lack of fusion between the embryological dorsal and ventral ducts of the pancreas. The dorsal pancreatic bud is composed of the head, body and tail and is drained into the duodenum through the papilla minor, the ventral pancreatic bud is composed of part of the head and uncinate process and is drained together with the bile duct through the papilla major. There is often association between PD and recurrent acute and chronic pancreatitis, which appears to be caused in most cases by inadequate drainage of the dorsal pancreas. Instead, an incomplete PD is characterized by a connection between the two ductal systems, which are present, but are insufficient (4). The diagnosis of PD is based on Endoscopic retrograde cholangiopancreatography (ERCP) and magnetic resonance cholangiopancreatography (MRCP) imaging with secretin stimulation. PD is asymptomatic in most patients, but in about 5% of cases patients develop symptoms of pancreatitis, potentially as a result of insufficient drainage through the papilla minor (7). Surgical treatment aims to increase the drainage of the dorsal pancreas (8) by ERCP with papillotomy or stent, causing a regression of symptoms in two-thirds of patients (3, 5, 6, 8-10). In cases of persistent symptoms, reinsertion of the papilla, ”duodenum-preserving” resection of the head of the pancreas or pancreatic resection according to Whipple can be done (11-13, 19).

Patients and methods

Seven patients (4 males and 3 females) with a mean age of 34 years (range 28-36), were prospectively observed between 2006 and 2011. All patients were hospitalized for recurrent pancreatitis (at least 2 episodes), with an elevated amylase and lipase blood levels (above what threshold?). The MRCP Imaging demonstrated the presence of complete pancreas divisum (4 patients) in association with mild ectasia of the pancreatic ducts in only 2 patients (Fig. 1) and incomplete pancreas divisum (3 patients) with normal calibre of the pancreatic ducts (Fig. 2). In all patients examined, we observed the presence of thickening of the bile in the gallbladder.

In the absence of clear indications to surgery on the
pancreatic ducts, all patients underwent laparoscopic cholecystectomy in our Department of surgery. The mean follow-up period of 32 months (range 28-36) demonstrated the complete absence of further episodes of postoperative pancreatitis.

Discussion

Over 95% of patients with PD are asymptomatic, but the remaining 5% develop symptoms of acute pancreatitis. The causative factor for this appears to be an insufficient drainage of pancreatic fluids through the papilla minor. The origin of the symptoms, whether from the sphincter of Oddi dysfunction or structural stenosis of the papilla, is still debated. The reason why only some patients develop symptoms is even less clear (8, 14-17). It should be noted that 22% of patients with clinical symptoms associated with PD have mutations in the “cystic fibrosis trans-membrane conductance regulator gene” (CFTR). The clinical symptoms seem to be the only factor influencing the decision of surgical treatment (18).

In 7 patients treated in our department, the patients had no dilation of the accessory pancreatic duct, but showed symptoms of recurrent, acute pancreatitis. In all cases the MRCP Imaging identified thickening of the bile in the gallbladder, which we consider the aetiology of their symptoms. The patients underwent laparoscopic cholecystectomy with resolution of pain, and absence of further episodes of acute pancreatitis during a mean follow-up period of 32 months. Therefore, it is possible that there is an association between the primitive pancreatic anatomical malformations and increased precipitation and concentration of bile in the gallbladder that causes poor drainage of the accessory pancreatic-duct.

Conclusions

It is possible that laparoscopic cholecystectomy may be curative in the presence of bile thickening within the gallbladder, associated with PD - in the absence of dilation of the accessory pancreatic duct. It may also be beneficial in patients possessing clinical symptoms, when interventional ERCP contraindicated to avoid duodenal preserving resection of the pancreas carries with its high associated morbidity and mortality (19-23).

References

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