LETTERS TO THE EDITOR

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The Cystic Duct Remnant: An Unusual Case of a Biliary Intraluminal Filling Defect

To the Editor: Patients with recurrent biliary colic after cholecystectomy require evaluation to exclude choledocholithiasis. Endoscopic retrograde cholangiography (ERC) is the recommended examination because it provides a diagnostic cholangiogram and permits a therapeutic sphincterotomy to be performed, if indicated. The challenge to the cholangiographer is to distinguish common bile duct stones from other biliary intraluminal filling defects. We report a case of an intraluminal bile duct filling defect caused by an inverted cystic duct remnant found during ERC for choledocholithiasis.

CASE REPORT

At the time of admission, V.D. was a 59-yr-old woman with a 6-month history of sharp, episodic right upper quadrant pain. There was no history of jaundice, emesis, alcohol intake, trauma, or fevers. She had been hospitalized with biliary pancreatitis in 1979, at which time she underwent an elective cholecystectomy. The gallbladder was thickened and found to contain multiple stones. A 2-cm choledochotomy incision was made at the site of the cystic duct for common bile duct exploration and T-tube placement. The common bile duct was irrigated clean, and a subsequent T-tube cholangiogram showed no stones.

The patient has had an appendectomy, total abdominal hysterectomy, and right modified radical mastectomy for an intraductal breast carcinoma. She has type II diabetes mellitus and hypertension.

Physical examination revealed an obese female with normal vital signs. The general examination was unremarkable. The abdomen was obese and bowel sounds were present. There was mild tenderness in the epigastrium to deep palpation. Hepatosplenomegaly was absent.

Laboratory studies revealed a white blood cell count of 12,400 (61% polys, 20% bands, 16% lymphs, 2% monos, 1% eos), hemoglobin 14.4 g, hematocrit 42.7%, glucose 213 mg/dl, AST 84 IU/L, ALT 88 IU/L, LDH 224 IU/L, alkaline phosphatase 113 IU/L, bilirubin 1.7 mg/dl and amylase 59 IU/L. Coagulation parameters and other chemistries were normal.

A right upper quadrant abdominal ultrasound showed a dilated common bile duct with no stones appreciated. An ERC was obtained (Fig. 1). The common hepatic duct and common bile duct were dilated to approximately 2 cm. There were two stones in the distal common bile duct. A 1-cm fixed intraluminal


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FIG. I. Endoscopic retrograde cholangiogram demonstrating a discrete filling defect at the location of the cystic duct remnant. Biliary dilatation is evident, and two stones are visible in the distal common bile duct.

The filling defect was seen in the bile duct at the site of the cystic duct remnant. A 1.2-cm sphincterotomy cut was made without difficulty. A repeat ERC 2 wk later demonstrated interval disappearance of both stones, but persistence of the fixed intraluminal filling defect. The defect was pliable and easy to manipulate with the endoscopic cannula. The area was directly brushed and bile aspirated for cytology. No malignant cells were seen. Six months later, a third ERC was performed. The luminal filling defect was unchanged and direct brushings again revealed no evidence of malignancy. Presently, the patient feels well and liver tests are normal.

DISCUSSION

The persistent fixed filling defect located in the proximal common bile duct is likely an inverted cystic duct remnant. To our knowledge, this finding has not been previously reported on ERC (1, 2). Intraluminal bile duct filling defects are typically due to neoplasm or retained stones. Primary biliary carcinomas are rare, and benign neoplasms of the extrahepatic bile ducts are even more unusual. It is unlikely that this lesion is a primary carcinoma, because it remained unchanged after 6 months, and direct brushing on both occasions showed no evidence of neoplastic cells. It is also unlikely that it is a benign neoplasm since it lacked the pedunculated circumscribed appearance of an adenoma or papilloma. There were two mobile filling defects representing stones at the time of ERC which disappeared after sphincterotomy, raising the possibility that this lesion is an adherent stone. However, the soft deformable quality of the lesion during direct brushing was unlike that of an adherent common bile duct stone. The filling defect is located at the cystic duct site. Lesions that may cause filling defects in this region include a "reformed gallbladder," a cystic duct remnant calculus, and a cystic duct remnant neuroma (3). The T-tube cholangiogram (not shown) demonstrating the presence of the filling defect immediately after the original cholecystectomy argues against these possibilities. Of note, the barium enema appearance of an inverted appendiceal stump, somewhat analogous to an inverted cystic duct stump, resembles the bile duct filling defect in this patient (4).

One may also speculate that an inverted cystic duct remnant may predispose to choledocholithiasis. Other luminal lesions, such as bile duct carcinoma and, to a lesser extent, benign neoplasms, are associated with common duct stones (5). An intraluminal defect may attract bits of foreign matter that act as a nidus for stone formation (6). Subtle changes in bile flow may cause local stasis and subsequent stone formation. An association has been found between cholangiographic angulation of the common bile duct and choledocholithiasis, perhaps related to this phenomenon (7). The association between the fixed filling defect and the common bile duct stones found in this patient is speculative. In any case, we believe that the umbilicated appearance of this intraluminal bile duct lesion is most consistent with an inverted cystic duct stump, and that recognition of this lesion is important because it may be mistaken for other biliary tract pathology.

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Pseudotumor of the Lateral Duodenal Wall

To the Editor: Intramural lesions of the lateral wall of the duodenum almost always represent neoplastic growths. Inflammatory masses, on the other hand, are rare, and are usually related to benign diseases of the pancreas. When they occur, however, they must be differentiated from true neoplasms, since errors in judgment may result in inappropriate treatment.

An accurate tissue diagnosis revealed the true nature of the lesion in our patient and was the basis of appropriate surgical treatment.

CASE REPORT

A 46-yr-old black male with chronic alcoholism was hospitalized for mid-epigastric pain and intermittent vomiting of several months’ duration. He had noted a 14-kg weight loss during the previous 6 months. The physical examination was unremarkable and failed to reveal jaundice, abdominal masses, or tenderness. There was slight elevation of SGOT, alkaline phosphatase, and serum amylase; total bilirubin was in the upper normal range. Radiography of the abdomen revealed several small calcifications of the pancreas; the liver-spleen scan was normal. The upper gastrointestinal series showed a 5 x 3 cm filling defect extending from the post-bulbar region to the junction between the second and third parts of the lateral duodenum, producing near total occlusion of the lumen (Fig. 1).

On ultrasound examination, the head of the pancreas was slightly enlarged and contained a 5-cm fluid-filled mass. This was interpreted as either a pseudocyst or, possibly, a necrotic tumor. Our suspicion of a tumor was reinforced when fiberoptic duodenoscopy revealed a large, lobulated, soft tissue mass resembling a villous adenoma or carcinoma of the lateral wall of the duodenum. However, biopsies showed only normal duodenal mucosa.

At laparotomy, the first and second parts of the duodenum were enlarged to twice normal diameter and appeared to contain a rubbery, poorly defined mass involving the lateral duodenal wall. Transluminal biopsies through an antral gastrotomy were performed to avoid possible tumor spillage. Despite large biopsies, only normal duodenal mucosa was seen. With the probability of a neoplasm diminishing, trans-serosal needle biopsies revealed the true nature of an intramural duodenal pseudocyst of the pancreas. A formal duodenotomy and incision into the cyst demonstrated the communication of the intramural cyst with the substance of the pancreas.

A duodenal diverticulization procedure and a cyst-duodenostomy were performed. The patient recovered fully and was discharged on the 11th postoperative day.

DISCUSSION

We believe our case represents a stage in the evolution of an intramural pseudocyst just prior to its spontaneous internal drainage. This is a rare occurrence, since Littman et al. (1) found only three cases of pseudocyst
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