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Chronic Pancreatitis Progressing to Duodenal Obstruction in the Absence of Classic Symptoms

John R. Condit, Jr, DO,* and Dominic K. H. Wong, MD†

We report the case of a 34-year-old alcoholic who was initially seen in March 1985 because of acute pancreatitis. A mass was demonstrated in the head of the pancreas. Serial sonogram and computed tomography scans over 4½ years revealed progressive encroachment of the duodenum without symptoms attributable to obstruction. In 1989, three separate endoscopies with multiple biopsies showed chronic inflammation and strictures. Hypotonic duodenography confirmed stricture and obstructed duodenum. Surgical intervention is being considered.

Duodenal obstruction secondary to chronic pancreatitis is rare. It may proceed subclinically for several years independent of continued alcohol use. Only when obstruction became severe in our patient did the classic symptoms of postprandial nausea, emesis, and weight loss become manifest. Obstructive jaundice from chronic pancreatitis due to stricture in the pancreatic portion of the common bile duct is uncommon. (Henry Ford Hosp Med J 1991;39:52-5)

Boerhaave is said to have reported in the 17th century the first case of duodenal obstruction (1). Since then, duodenal obstruction, stricture, or narrowing has been reported in a variety of disorders (Table). Chronic pancreatitis is a rare cause with a reported incidence of approximately 1% (2,3). Most reported cases appear in retrospective studies in the surgical literature (2-9). The preoperative diagnosis is usually suspected because of the classic symptoms of nausea, persistent emesis, and weight loss. The current case documents progression of chronic pancreatitis over a four-year period producing near total duodenal obstruction in the absence of classic symptoms of obstruction.

Case Report

A 34-year-old male alcoholic was hospitalized in March 1985 because of mid epigastric pain, nausea, vomiting, and loose stools. Serum amylase was elevated at 4.47 μ kat/L (268 U/L) (normal range 0 to 1.42 μ kat/L [0 to 85 IU/L]). Abdominal sonogram revealed a 5.5 cm mass close to the pancreatic head, which was thought to represent either a focal area of pancreatitis, a pseudocyst, or hemorrhage (Fig 1). The patient was treated conservatively and discharged six days later on a regular diet. Amylase levels had returned to normal.

One month later he remained asymptomatic. Repeat sonogram revealed that the pancreatic mass had shrunk slightly and become less homogeneous (Fig 2). As the mass caused no symptoms and appeared to be regressing, a conservative approach was taken and a follow-up sonogram was scheduled for six weeks later.

The patient did not return for this third study. In October 1985, he was hospitalized with acute exacerbation of pancreatitis. Serum amylase and lipase levels were elevated and symptoms were similar to those of the initial presentation in March. Alkaline phosphatase level

was 1.2 μ kat/L (72 U/L) (normal < 1.7 μ kat/L [$<$ 100 U/L]). Serum bilirubin was normal. Repeat sonogram showed reduction in the mass but more homogeneity (Fig 3). Computed tomography (CT) defined the

Table
Causes of Duodenal Obstruction

<i>Intrinsic</i>
Benign or malignant neoplasms
Regional enteritis
Postbulbar ulcers
Intramural hematoma
<i>Extrinsic</i>
Foreign bodies (e.g., gallstones)
Pancreatic:
Annular pancreas
Chronic pancreatitis
Acute pancreatitis
Hematoma
Pseudocyst
Carcinoma
Abscess
Congenital bands
Postsurgical strictures
Superior mesenteric syndrome
Surrounding structure neoplasms
Aortic aneurysm

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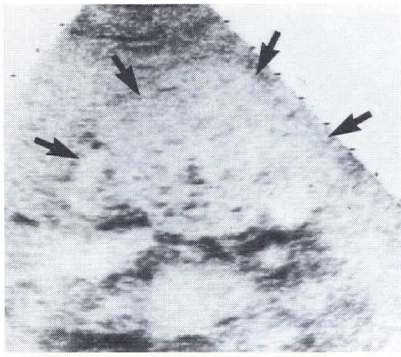


Fig 1—Abdominal sonogram (March 1985) demonstrating a 5.5 cm mass close to the head of the pancreas.

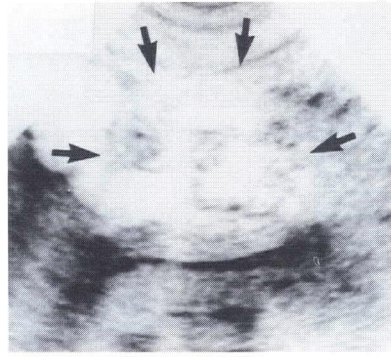


Fig 2—Follow-up sonogram one month later showing slight regression in the size of the mass.



Fig 3—Third sonogram (October 1985) revealing further mild regression.

lesion as inhomogeneous, measuring $4.5 \times 5.0 \times 4.0$ cm, contiguous with the head of the pancreas, and producing mass effect upon the duodenum (Fig 4). No other abnormalities were noted. Sputum and gastric aspirates for acid-fast bacilli were negative as was the PPD skin test. Conservative therapy with bowel rest, rehydration, analgesia, and gradual return to a regular diet was again successful. The patient was discharged on a regular diet and all laboratory values were normal.

He remained asymptomatic until July 1986, when four days of constant, sharp epigastric pain, nausea, and vomiting prompted him to seek emergency care. Rehydration and a temporary change to a clear-liquid diet avoided hospitalization. After this episode he abstained from alcohol for 37 months. However, in September 1989, he redeveloped symptoms after drinking wine. He self-initiated a clear-liquid diet for several days but had no relief and was hospitalized with his fourth acute exacerbation of pancreatitis.

His weight had not changed from the March 1985 admission. He denied abdominal pain, nausea, emesis, or change in bowel habits over the three-year interval he had abstained from alcohol. During this hospitalization persistent pain, nausea, and a rising serum amylase level despite conservative therapy prompted a second abdominal CT. Fullness of the pancreatic head was again noted with a new adjacent "apple-core" lesion of the descending duodenum (Fig 5). Air contrast upper gastrointestinal study revealed narrowing and nodular mucosal changes involving the apex of the duodenal bulb (Fig 6). To rule out a neoplastic

process, the patient underwent endoscopy with biopsy. At endoscopy a mass was encountered in the proximal portion of the second part of the duodenum (Fig 7). It was impossible to advance the endoscope into the third portion of the duodenum. Brushings and biopsies revealed severe chronic inflammation (Fig 8). Hypotonic duodenogram confirmed the presence of a stricture beginning in the apical region of the duodenal bulb and extending through the postbulbar area. The degree of obstruction and the suspicious visual appearance of the mass prompted a repeat endoscopy but the mass appeared unchanged. After several unsuccessful attempts, entry into the third portion of the duodenum showed it to be normal. Repeat biopsies were again consistent with chronic inflammation.

Amylase and lipase levels slowly returned to normal and on discharge the patient was able to tolerate a regular diet. He remained asymptomatic until December 1989 when his epigastric pain returned.

His pain has become constant in nature, exacerbated by eating. He has lost 6 kg (13.2 lb) and suffers from early satiety. Surgical intervention is being considered.

Discussion

Duodenal involvement by the inflammation of pancreatitis is not rare. Upper gastrointestinal radiographs disclose changes in 25% of cases (2). Duodenal obstruction can occur with either

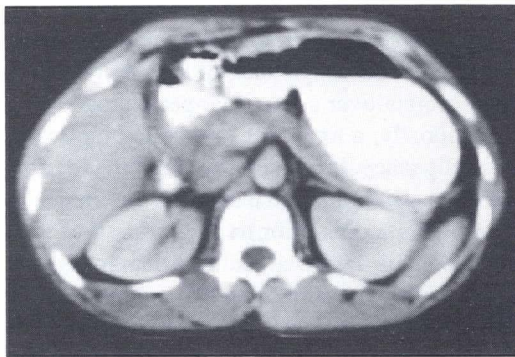


Fig 4—Computed tomography scan (October 1985) demonstrating mass effect on the duodenum.

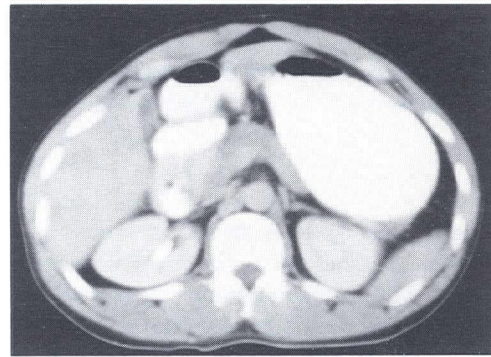


Fig 5—Computed tomography scan (October 1989) demonstrating an "apple-core" lesion of the descending duodenum.

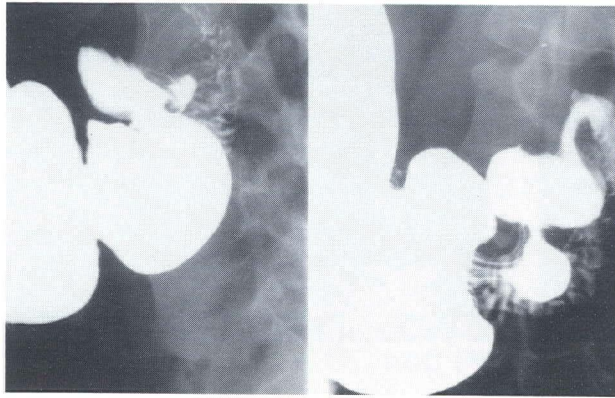


Fig 6—Air contrast upper gastrointestinal study showing nodular mucosal changes and narrowing of the duodenal bulb.

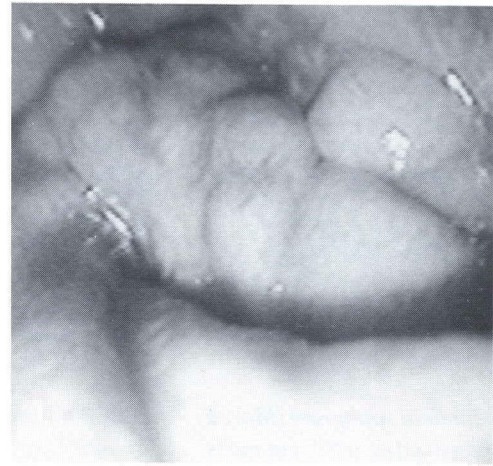


Fig 7—Endoscopic view of obstructing duodenal mass.

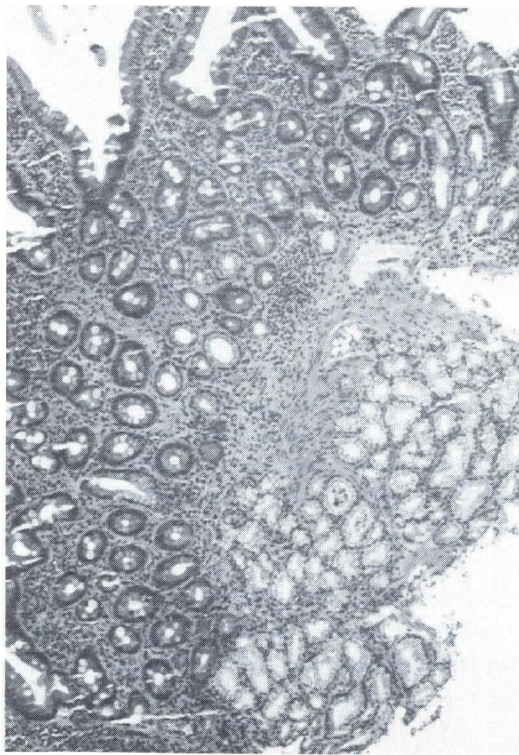


Fig 8—Endoscopic biopsy specimen revealing changes of chronic inflammation without evidence of malignancy (hematoxylin-eosin stain).

acute or chronic pancreatitis. In the former instance a functional obstruction occurs. Local edema, tissue swelling, and/or hematoma formation with resultant muscular spasm are believed responsible (2). Conservative therapy with nasogastric suction, bowel rest, and analgesia is generally all that is required.

Chronic pancreatitis produces a more anatomic obstruction which generally requires surgical intervention. Why the inflammation induced by pancreatitis becomes progressive in a minority of cases is not known. Duodenal obstruction is related to its

proximity to the pancreas, but this process is not unique to the duodenum as colonic stenosis secondary to pancreatitis has been reported as well (10). Bradley and Clements (3) speculate that pancreatitis-induced arterial narrowing and/or thrombosis within the pancreaticoduodenal circulation may produce local ischemia, which, superimposed on inflammatory edema, initiates the chronic fibrosis. If so, these changes would have to be on the microvascular level as Satake and Umeyama (4) have reported three cases with normal celiac and superior mesenteric arteriograms. Makrauer et al (11) detailed three cases of duodenal obstruction where intramural, nonmalignant pancreatic tissue was believed to play a role. Multiple biopsies in the present case revealed no evidence for this possible mechanism.

Was alcohol responsible? Alcohol is a well-known instigator of pancreatitis and most cases of duodenal obstruction related to chronic pancreatitis occur in chronic alcoholics. Poor outcome post pancreaticoduodenectomy has been associated with continued alcohol ingestion (12). Interestingly, our patient's pancreatic mass initially appeared to regress despite continued alcohol use. However, a subsequent prolonged period of abstinence did not prevent progression to obstruction. Clearly other factors are involved.

The chronic nature of the inflammatory process is well illustrated by the present case. The lack of symptoms and maintenance of body weight over a four-year period made carcinoma unlikely. Additionally, a total of 11 separate biopsies all were negative for malignancy. In a review by Gambill (13) of patients with both severe pancreatitis and pancreatic carcinoma, the median duration of symptoms prior to diagnosis of cancer was 8 months. Our patient has survived for more than five years. However, the radiographic appearance on CT and the visual inspection of the mass at endoscopy were suspicious for neoplasia, which emphasizes the need for biopsies as chronic pancreatitis has progressed to carcinoma in some cases (13).

Duodenal obstruction secondary to chronic pancreatitis is a rare complication which may proceed subclinically for a number of years, independent of continued alcohol use. Only when

obstruction becomes severe will the classic symptoms of post-prandial nausea, emesis, and weight loss ensue. Jaundice need not be present as significant common bile duct obstruction in chronic inflammatory pancreatic disease is uncommon (14,15). The possibility of coexistent or underlying carcinoma should always be considered.

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