Partial Biliary Obstruction Caused by Chronic Pancreatitis

An Appraisal of Indications for Surgical Biliary Drainage

THOMAS J. STAHL, M.D., MELODY O'CONNOR ALLEN, M.D., F.A.C.S., HOWARD J. ANSEL, M.D., and JACK A. VENNES, M.D.

This paper presents a retrospective review of 38 patients with intrapancreatic bile duct strictures secondary to chronic alcoholic pancreatitis. The strictures were identified by endoscopic retrograde cholangiopancreatography (ERCP). All patients with pancreatic cancer and gallstone pancreatitis were excluded. The mean alkaline phosphatase and total bilirubin values were 344 ± 57 IU/dl and 4.4 ± 0.7 mg/dl, respectively. The mean stricture length was 3.9 ± 0.5 cm, and the mean common bile duct (CBD) diameter was 1.8 ± 0.2 cm. The degree of bilirubin and alkaline phosphatase elevation did not correlate with stricture length or the severity of bile duct dilatation. Eighteen of the 38 patients received surgical biliary drainage (BD) as part of their initial therapy, and 20 patients did not. Liver function tests, intrapancreatic stricture length, and the degree of proximal CBD dilation were comparable in these two groups. Patients not undergoing BD did well clinically as only one patient required BD over an average follow-up period of 3.8 years. In conclusion, bypass of these strictures is usually unnecessary, and most patients may be safely treated without operation.

I NTRAPANCREATIC BILIARY STRICTURES have been estimated to occur in from 3 to 29% of patients with chronic alcoholic pancreatitis.¹⁻³ Opinions regarding the clinical significance and appropriate treatment for these lesions are variable. Strictures are often suspected in patients with clinical and laboratory signs of biliary stasis in conjunction with an acute exacerbation of chronic pancreatitis. Operative biliary drainage has often been recommended to prevent both the acute development of cholangitis and late development of biliary cirrhosis.^{4,5} The low incidence of cholangitis and From the Departments of Surgery, Radiology, and Gastroenterology, the University of Minnesota and Veterans Administration Medical Center, Minneapolis, Minnesota

biliary cirrhosis in association with biliary strictures has prompted others to advocate a less aggressive approach to the care of these patients.^{3,6–8} Since all prior published reports of patients with biliary strictures due to benign pancreatic disease report results of operative therapy, little is known about the natural history of patients who are not operated on. This study summarizes the clinical course of 38 patients with biliary strictures and chronic pancreatitis in an attempt to compare the risks and benefits of nonoperative therapy (20 patients) with those of traditional operative drainage of the biliary tree (18 patients).

Patients and Methods

Endoscopic retrograde cholangiopancreatography (ERCP) records from the Minneapolis Veterans Administration Hospital were reviewed from 1973 to 1985. All patients with clinical and radiologic evidence of chronic pancreatitis who were found to have an ERCPproven stricture of their intrapancreatic common bile duct (CBD) were included in this study. Inpatient and outpatient medical records of each patient were reviewed. The ERCPs were interpreted by a single staff radiologist (H.A.). Patients with either choledocholithiasis or pancreatic cancer as the possible cause of ductal abnormalities were excluded.

All results are presented as mean \pm SEM. Student's t-test and correlation coefficients were used for statistical comparison. A p value <0.05 was considered significant.⁹

Reprint requests and correspondence: Melody O'Connor Allen, M.D., F.A.C.S., Department of Surgery (112), Veterans Administration Medical Center, 54th Street and 48th Avenue South, Minneapolis, MN 55417.

Submitted for publication: July 20, 1987.

Results

Clinical Features

The mean age of the 38 patients was 49.5 years with a range from 23 to 66 years. Thirty-seven of the 38 patients were men. All patients had a long history of alcohol abuse, with over 60% having had one or more previous episodes of acute pancreatitis. The presenting signs and symptoms were abdominal pain (92%), weight loss (34%), jaundice (29%), nausea (24%), emesis (16%), fatigue (11%), gastrointestinal bleeding (8%), anorexia, fever, and ascites (3%).

Laboratory Values

The serum alkaline phosphatase was the laboratory test most frequently elevated at the time of admission (63% of patients) with an average value of 344 ± 57 IU/dl (normal range: 21–91 IU/dl). The total serum bilirubin level was elevated in 50% of patients, with an average value of 4.4 ± 0.7 mg% (normal range: 0.3–1.0 mg%). The SGOT was elevated in 54% of patients.

ERCP Findings

The intrapancreatic portion of the common bile duct (CBD) was strictured on ERCP in all patients. The mean length of the strictures was 3.9 ± 0.5 cm. The CBD proximal to the stricture was dilated in 64% of patients,



FIG. 1. A 48-year-old white man had pain and jaundice. Admission laboratory values were alkaline phosphatase of 750 IU/L and total bilirubin of 8.1 mg/dl. A 7.0-cm biliary stricture with a 1.4-cm diameter proximal CBD is seen on this cholangiogram. This patient was treated successfully with conservative medical therapy.



FIG. 2. A 36-year-old white man had recurrent pain and jaundice. Admission alkaline phosphatase level was 395 IU/L and total bilirubin level was 10.0 mg/dl. A 2.8-cm biliary stricture with 1.3-cm diameter proximal CBD was seen on this cholangiogram. This stricture was treated by choledochoduodenostomy. Follow-up for 1 year revealed no further problems.

with an average duct diameter of 1.8 ± 0.2 cm (normal diameter <1.0 cm), and a range from less than 1.0 to 4.0 cm. The configuration of the CBD was variable, as seen in Figures 1–4. The pancreatic ductal system routinely revealed a wide range of changes consistent with chronic pancreatitis. Findings varied from early side branch blunting to dilatation, tortuosity, and segmental stenosis of the main pancreatic duct. Pseudocysts were demonstrated in the pancreas in 18 of 38 patients (47%).

Therapy

The various treatments the 38 patients received are depicted in Figure 5. In 18 patients, surgical biliary drainage (BD) was part of the initial therapy, whereas the remaining 20 patients did not have BD. Of the 18 patients who had BD, 13 had a bilioenteric anastomosis, and five had T-tube drainage of the common bile duct. Of the 20 patients who did not have BD, 15 had other surgical procedures performed, and five were treated strictly with nonsurgical measures. Concomitant pan-



FIG. 3. A 54-year-old white man had weight loss and fatigue. His admission liver function tests were normal. ERCP revealed an intrapancreatic CBD stricture with no proximal CBD dilation. A pseudocyst was seen in the head of the pancreas displacing the CBD. Treatment consisted of a Roux-Y pancreaticojejunostomy (Puestow). Eight-year follow-up revealed no further biliary or pancreatic problems.

creatic procedures were performed in 23 of 38 patients, and consisted of pseudocyst drainage in 13, longitudinal pancreaticojejunostomy in nine (Puestow procedure), and pancreatic biopsy in one patient.

There were four treatment failures as defined by the need for a subsequent biliary procedure. Three occurred in the 18 patients initially undergoing drainage, and one occurred in the patients who did not have BD. One patient required conversion of a cholecystojejunostomy to a choledochojejunostomy subsequent to a severe episode of acute cholangitis, and two patients required choledochoduodenostomies after prolonged T-tube drainage. One patient in the group that did not have BD who experienced a treatment failure returned 4 years after conservative therapy with clinical sepsis, and underwent cholecystectomy, CBDE, and T-tube drainage of the common duct. However, his preoperative sepsis was found at operation to be due to an infected pancreatic pseudocyst, rather than CBD disease. All four of these patients who had treatment failure subsequently did well.

Postoperative Complications

There were no in-hospital deaths. Five major postoperative complications occurred including two subphrenic abscesses, both occurring after Puestow pancreaticojejunostomies; an anastomotic leak that occurred after a Puestow procedure; a peripancreatic abscess after a pancreatic pseudocystogastrostomy and cholecystectomy; and one severe episode of pneumonia.

Liver Histopathology

Intraoperative liver biopsies were obtained in 24 patients (Table 1). Three of 24 biopsy specimens revealed biliary cirrhosis (12%).

Comparison of Patients Who Had BD Versus No BD

Table 2 summarizes laboratory and clinical data for patients receiving BD as part of their initial therapy compared with those who did not. Although the mean serum bilirubin and alkaline phosphatase levels were higher in the group undergoing BD, the differences were not statistically significant. The biliary stricture lengths and CBD diameters proximal to the strictured area were nearly identical. The average length of follow-up was also comparable between groups.



FIG. 4. A 53-year-old white man had abdominal pain. His admission liver function tests were normal. ERCP revealed a 3.0-cm intrapancreatic CBD stricture with a proximal CBD dilated to 4.0 cm. Treatment consisted of a Puestow pancreaticojejunostomy and choledochoduodenostomy. No further problems developed during 26 months of follow-up.



Correlation of Laboratory Values with Cholangiographic Findings

To assess the potential predictive value of laboratory tests for cholangiographic findings, a simple linear regression analysis was performed, and correlation coefficients were determined between four variables (Table 3). There was no significant correlation of either the alkaline phosphatase or the total bilirubin levels with stricture length or proximal CBD diameter. Furthermore, there was no correlation of total bilirubin with alkaline phosphatase levels nor of stricture length with CBD diameter. The disparity between CBD configuration and laboratory values can be appreciated in Figures 1–4.

Hospital Course and Discharge Status (Table 4)

The average hospital stay was 41.7 days, with no difference between the patients who had BD compared with those who did not. The average alkaline phospha-

Interpretation	Ν
Normal	5
Biliary stasis	5
Biliary stasis + cholangitis	3
Fatty change only	3
Biliary cirrhosis	3
Portal fibrosis only	2
Pericholangitis	1
Early micronodular cirrhosis	1
Necrotizing granulomas	1
Total	24

tase value at discharge had fallen to approximately 1.5 times normal, and the average total bilirubin level was normal. There was no difference in average discharge laboratory values in the patients who did not have BD compared with those who did.

Follow-up (Table 5)

Six patients were lost to follow-up, leaving 32 patients with an average follow-up period of 4.0 ± 1.0 years. The mean lengths of follow-up for the patients who had BD and for those who did not were 4.4 ± 0.9 and 3.8 ± 1.1 years, respectively. One patient died of acute mesenteric infarction 4.5 years after a cholecystectomy and choledochoduodenostomy. Eight patients had repeated episodes of pancreatitis. A pancreatic abscess developed in one patient 2.5 years after drainage of a pseudocyst, and one patient required drainage of a second pseudocyst. The remaining 16 patients had no further pancreatic or

 TABLE 2. Comparison of Laboratory and Clinical Data in Patients with and without Operative Biliary Drainage*

	Drain	No Drainage		
Age (years)	48.0 ±	6.0	51.0 ±	10.0
Average alkaline phosphatase				
level (IU/L)	442.0 ±	121.0	260.0 ±	56.0
Average bilirubin level				
(mg/dl)	5.9 ±	1.6	3.1 ±	1.1
Stricture length (cm)	3.7 ±	0.7	4.1 ±	0.9
CBD diameter (cm)	1.8 ±	0.2	1.8 ±	0.4
Average length of follow-up				
(years)	4.4 ±	3.4	3.8 ±	4.4

* No statistically significant differences were noted.

	Alkaline Phosphatase Level	Total Bilirubin Level	Stricture Length	CBD Diameter
Alkaline phosphatase level	1.000			
Total bilirubin level	0.350	1.000		
Stricture length	0.081	0.050	1.000	
CBD diameter	0.237	0.023	0.101	1.000

TABLE 3. Correlation of Laboratory Values with Cholangiographic Findings*

* No statistically significant correlations were noted.

biliary problems. The frequency of problems was of no greater significance in the patients who did not have BD.

Discussion

Biliary strictures as a consequence of chronic pancreatitis have long been recognized,¹⁰ but until recently were considered unusual. With the increasing use of ERCP, strictures are being identified more frequently. A review of series published over the previous 10 years (Table 6) yields an incidence of biliary stricture in association with chronic pancreatitis in alcoholic patients of 5.7%, with a broad range from 2.7% to 45.6%.^{1-3,11-13} Differences in criteria used for patient selection accounts for this wide variation. Currently, controversy remains regarding the natural history of intrapancreatic biliary strictures, the accuracy of preoperative diagnosis, and the appropriate course of treatment. Our study, which contains the largest number of reported patients with biliary strictures treated without surgery, has helped clarify a number of these issues.

The pathologic characteristic of these strictures is one of encasement of the intra- or peripancreatic bile duct in a progressively fibrotic pancreas,¹⁴⁻¹⁷ with edema and inflammation of an acute pancreatitic episode often exacerbating the stricture. The clinical consequences of these strictures can in turn be variable. Many are asymptomatic, with transient or low-grade liver function test abnormalities. Acute episodes of pancreatitis can in turn prompt acute and sometimes sharp elevations of both the serum bilirubin and alkaline phosphatase levels.

The clinical diagnosis of an intrapancreatic common

 TABLE 4. Comparison of Hospital Course and Discharge Laboratory

 Values in Patients with and without Operative Biliary Drainage

	Drainage	No Drainage
Length of stay (days) Average discharge alkaline phosphatase level (IU/	42.0 ± 4.4	41.0 ± 4.6
L; normal: 21–91 IU/L) Average discharge total bilirubin level (mg/d):	150.0 ± 39.0	154.0 ± 19.0
normal: 0.1–1.0 mg/dl)	1.2 ± 0.4	0.9 ± 0.2

bile duct stricture in a patient with chronic alcoholic pancreatitis is often difficult. There are no definitive signs or symptoms, and liver function test abnormalities may be suggestive but nonspecific. An acute or chronic elevation of the serum alkaline phosphatase level is the most frequent laboratory abnormality, occurring in approximately 80% of patients (Table 6).^{3,5} The total serum bilirubin level is also frequently elevated during the acute episodes of pancreatitis, occurring in approximately 65% of patients (Table 6), returning to normal as the acute process subsides, and rarely remaining elevated on a chronic basis.^{3,7,14,16,18} Cholangiography, either endoscopic or transhepatic, is the definitive diagnostic tool. Cholangiographic findings, however, are variable, as has been shown in this study and by others.^{6,12,14} Previous publications have described these strictures as gradual taperings of the intrapancreatic duct over a distance of 3-5 cm, thereby distinguishing them from the abrupt, short obstructions seen in pancreatic cancer. The exceptions to this rule are sufficiently frequent to render cholangiographic findings dubious in the distinction of these two disease processes.¹²

The correlation of cholangiographic findings with liver function test (LFT) abnormalities was very low in this study, as others have also found.^{7,12} In the patients from our study, long strictures with dilated proximal common bile ducts were present despite normal LFTs, whereas short strictures with normal proximal biliary anatomy were seen with remarkably elevated LFTs.

Once a biliary stricture is discovered, the remaining

 TABLE 5. Comparison of Long-term Results in Patients with and without Operative Biliary Drainage

	Drainage	No Drainage
Lost to follow-up	3	3
Length of follow-up (years)	4.4 ± 0.9	3.8 ± 1.1
Deaths	1	0
Repeated episodes of pancreatitis	3	5
Initial treatment failures	3	1
Repeat pseudocyst drainage	0	1
Late pancreatic abscess	0	1
No further problems	7	9

First Author	No. of Patients	Elevated Bilirubin Level	Elevated Alkaline Phosphatase Level	Incidence of Cholangitis	Incidence of Biliary Cirrhosis	Incidence of CBD Stricture In All Cases Pancreatitis (%)
McCollum (1975) ²⁰	7	7/7	_	_		_
Warshaw (1976)4	6	6/6	6/6	3/6	3/4	
Scott $(1977)^2$	11	9/11	10/11	· _	1/11	11/38*
Schulte (1977) ⁵	13	8/13	13/13	4/13	· _	_
Sarles (1978)6		·	_	·	0/300	_
Bradley (1978) ¹⁸	13		_	2/13	·	_
Gremillion (1979) ¹⁴	7	5/7	7/7	0/7	_	
Yadegar (1980) ³	21	13/21	20/21	3/21	2/11	21/651†
Gregg (1981) ⁷	21	11/21	11/21	_	0/21	· · ·
Afroudakis (1981) ¹	24	· _	_	_	4/24	8/300‡
Creaghe (1981) ¹⁵	10		—	1/10	2/10	_
Wisloff (1982) ¹³	36	19/36	26/36	_	_	36/79§
Eckhauser (1983) ¹⁶	9	5/9	9/9	0/9	_	_
DaCunha (1984) ⁸	45	-		3/45		_
Aranha (1984) ¹¹	51	44/51	48/51	3/51	0/51	51/1262
Petrozza (1985) ¹²	15	—	14/15	—	—	15/151¶
Sugerman (1986) ¹⁹	16	—		1/16	_ `	
Current study	38	19/38	24/38	1/38	3/24	_
Total (%)	343	146/220 (66.3%)	188/228 (82.5%)	21/229 (9.2%)	15/456 (3.3%)	142/2481 (5.7%)

TABLE 6. Literature Review of CBD Stricture Characteristics in Patients with Chronic Pancreatitis

* Of 38 patients with chronic alcoholic pancreatitis, 11 were found to have intrapancreatic CBD strictures by ERCP.

† Of 651 admissions for chronic alcoholic pancreatitis from January 1968 through December 1979, 21 patients had surgery for intrapancreatic CBD strictures.

‡ Of 300 patients with chronic alcoholic pancreatitis from 1972

through 1979, 24 were found to have intrapancreatic CBD stricture.

§ Of 79 consecutive patients with moderately severe chronic pancre-

decisions involve the proper therapeutic choice(s). Concerns over the possible development of acute cholangitis and/or biliary cirrhosis have in the past prompted *early* surgical drainage of the proximal biliary tree.^{4,5} More recent recommendations are to delay drainage until problems manifest themselves.^{3,6,7,15,16} This latter approach is supported by low rates for cholangitis and biliary cirrhosis reported by ourselves and others (Table 6).

The decision to delay drainage of a CBD stricture due to pancreatitis must be based on an assessment of the risk in conservative or expectant management of this lesion. Although no study specifically addresses nonsurgical treatment of biliary strictures in chronic pancreatitis, there are small cohorts of patients who for various reasons did not have surgical therapy of their biliary strictures.¹⁻³ Scott et al.² reported on 11 patients with chronic alcoholic pancreatitis and intrapancreatic CBD strictures. Five of these patients had no surgery, and for a follow-up of 1 year or more, had no further problems. Yadegar et al.³ followed two patients with known strictures and chronic elevations of alkaline phosphatase levels for 5 years, obtaining several subsequent liver biopsies. No disease progression was discovered in either patient histologically or clinically, and there was no

atitis who had ERCP, 36 were found to have stenosis of the intrapancreatic CBD.

 \parallel Of 1262 patients with chronic pancreatitis admitted from 1970 through 1982, 51 of these patients were treated for distal CBD obstruction.

¶ Of 151 patients admitted for chronic alcoholic pancreatitis from 1979 through 1984, 15 were found to have a stricture of the intrapancreatic CBD by cholangiography.

mention of the occurrence of cholangitis. Afroudakis and Kaplowitz¹ reviewed liver histology from 24 patients with known CBD strictures due to chronic alcoholic pancreatitis, and found that three of these patients had disease progression to biliary cirrhosis over an unspecified period after the initial diagnosis. Our study contains the largest single collection of patients with known biliary strictures who did not have surgical therapy: These 20 patients were followed for an average of 3.8 years with no increased morbidity compared with those patients initially undergoing BD, despite clinical and cholangiographic disease of comparable severity.

Based on the above findings, we believe that the presence of a stricture should not be the sole indication for surgical intervention. Absolute indications for surgical intervention are acute cholangitis, biliary cirrhosis, and protracted jaundice.^{3,4,7,8,14–16,19,20} Based on previous and current findings, there are two relative indications for operative biliary drainage: in patients with diagnosed strictures in whom a pancreatic procedure is already planned,⁷ and in patients with known strictures and a previous history of cholangitis. The presence of a dilated CBD proximal to a stricture and/or a chronically elevated alkaline phosphatase level do not justify surgical intervention, but most certainly warrant long-term follow-up consisting of periodic liver function tests, cholangiography, and liver biopsies.^{3,16} Once operative biliary drainage is indicated, we recommend choledochoenterostomy over cholecystoenterostomy or T-tube drainage. Results of our study and of others suggest that cholecystoenterostomy does not offer sufficient or reliable long-term drainage.^{3,5,7,8,14} Prolonged T-tube drainage in two of five patients in our study necessitated revisions to choledochoenterostomies. This problem has also been reported by others.^{4,14}

In summary, we present 38 patients with chronic alcoholic pancreatitis and ERCP-proven intrapancreatic biliary strictures. Twenty of the 38 patients did not have surgical therapy, with no increase in morbidity compared with the 18 patients who had surgery, despite disease of equivalent severity. Based on these findings, we believe that asymptomatic biliary strictures in patients with chronic pancreatitis can be safely observed, with periodic evaluation using liver function tests, ERCP, and liver biopsies. Absolute indications for surgery are acute cholangitis, biliary cirrhosis, and persistent jaundice. Relative indications for biliary drainage include a planned pancreatic procedure and a previous history of cholangitis in a patient with a known stricture.

References

- Afroudakis A, Kaplowitz N. Liver histopathology in chronic common bile duct stenosis due to chronic alcoholic pancreatitis. Hepatology 1981; 1:65-72.
- Scott J, Summerfield JA, Elias E, et al. Chronic pancreatitis: a cause of cholestasis. Gut 1977; 18:196–201.
- Yadegar J, Williams RA, Passaro E, Jr, Wilson SE. Common duct stricture from chronic pancreatitis. Arch Surg 1980; 115:582– 586.
- 4. Warshaw AL, Schapiro RH, Ferrucci JT, Jr, Galdabini JJ. Persistent obstructive jaundice, cholangitis, and biliary cirrhosis due

- to common bile duct stenosis in chronic pancreatitis. Gastroenterology 1976; 70:562-567.
- Schulte WJ, LaPorta AJ, Condon RE, et al. Chronic pancreatitis: a cause of biliary stricture. Surgery 1977; 82:303-309.
- Sarles H, Sahel J. Cholestasis and lesions of the biliary tract in chronic pancreatitis. Gut 1978; 19:851–857.
- Gregg JA, Carr-Locke DL, Gallagher MM. Importance of common bile stricture associated with chronic pancreatitis: diagnosis by endoscopic retrograde cholangiopancreatography. Am J Surg 1981; 141:199–203.
- DaCunha JEM, Bacchella T, Mott C, et al. Surgical treatment of biliary complications from calcifying chronic pancreatitis. Int Surg 1984; 69:149-154.
- 9. Rosner B. Fundamentals of Biostatistics. Boston: Duxbury Press, 1982; 225-252, 344-400.
- Fraser J. The surgical treatment of obstructive jaundice in pancreatic disease. Br J Surg 1938; 26:393-411.
- Aranha GV, Prinz RA, Freeark RJ, Greenlee HB. The spectrum of biliary tract obstruction from chronic pancreatitis. Arch Surg 1984; 119:595-600.
- Petrozza JA, Dutta SK. The variable appearance of distal common bile duct stenosis in chronic pancreatitis. J Clin Gastroenterol 1985; 7:447-450.
- Wisloff F, Jakobsen J, Osnes M. Stenosis of the common bile duct in chronic pancreatitis. Br J Surg 1982; 69:52-54.
- Gremillion DE, Jr, Johnson LF, Cammerer RC, Guider B. Biliary obstruction: a complication of chronic pancreatitis diagnosed by endoscopic retrograde cholangiopancreatography (ERCP). Dig Dis Sci 1979; 24:145–149.
- 15. Creaghe SB, Roseman DM, Saik RP. Biliary obstruction in chronic pancreatitis: indications for surgical intervention. Am Surg 1983; 47:243-246.
- Eckhauser FE, Knol JA, Strodel WE, et al. Common bile duct strictures associated with chronic pancreatitis. Am Surg 1983; 49:350-358.
- Prinz RA, Aranha GV, Greenlee HB. Combined pancreatic duct and upper gastrointestinal and biliary tract drainage in chronic pancreatitis. Arch Surg 1985; 120:361–366.
- Bradley EL III, Salam AA. Hyperbilirubinemia in inflammatory pancreatic disease: natural history and management. Ann Surg 1978; 188:626–629.
- Sugerman HJ, Barnhart Gr, Newsome HH. Selective drainage for pancreatic, biliary, and duodenal obstruction secondary to chronic fibrosing pancreatitis. Ann Surg 1986; 203:558–567.
- McCollum WB, Jordan PH Jr. Obstructive jaundice in patients with pancreatitis without associated biliary tract disease. Ann Surg 1975; 182:116-120.