

EUS-guided pancreaticogastrostomy

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Background: The pain of chronic pancreatitis can be caused by pancreatic ductal hypertension, and endoscopic drainage of the main pancreatic duct can provide relief. When transpapillary access to a dilated portion of the main duct cannot be obtained, conventional endoscopic drainage is not possible. The use of interventional EUS to perform a pancreaticogastrostomy in such cases is described.

Methods: Four patients presented with pain and a dilated main pancreatic duct proximal to a complete obstruction. EUS was used to access the dilated duct and create a pancreaticogastrostomy. Patency of the latter was maintained by placement of a pancreaticogastric stent.

Observations: EUS-guided pancreaticogastrostomy was performed without major complication. Three of 4 patients had satisfactory relief of pain at a median follow-up of 1 year.

Conclusion: EUS-guided pancreaticogastrostomy may be a promising new technique for pancreatic drainage and pain relief when conventional transpapillary access to the pancreatic duct is not possible.

The pain associated with chronic pancreatitis (CP) is caused, at least in part, by ductal hypertension.¹ Both surgical and endoscopic treatments can relieve pain by improving ductal drainage.²⁻⁴ Endoscopic drainage requires transpapillary access to the pancreatic duct during ERCP. Techniques for improving pancreatic drainage include pancreatic sphincterotomy, dilation of strictures, extraction of stones, and placement of stents.^{2,5} One of the limitations of endoscopic therapy is that the pancreatic duct, or a portion of the duct, may not be accessible at ERCP. This is mostly encountered in obstructive

CP resulting from rupture of the main pancreatic duct (MPD) after acute pancreatitis or trauma. If the patient is symptomatic, surgery may be the only option, including resection or various types of pancreaticojejunostomy and pancreaticogastrostomy.^{3,4,6}

The development of interventional EUS has provided better access to the region of the pancreas. Just as pancreatic fluid collections, such as pseudocysts, can be successfully drained from the stomach or duodenum by endoscopic cystenterostomy or cystgastrostomy,^{7,8} the same technique could be used to access a dilated pancreatic duct in cases in which the duct cannot be drained by conventional ERCP because of complete obstruction.

The aims of this study were to determine the technical feasibility and short-term safety of EUS-guided pancreaticogastrostomy (EPG). The effectiveness of ductal decompression and patient response in terms of symptom relief were also evaluated during follow-up. EPG was performed in 4 patients with pain caused by CP.

PATIENTS AND METHODS

The EPG procedure was approved by the ethics committee of our hospital. All patients in the present report were first evaluated by a senior endoscopist and all gave informed consent for EPG. The technical aspects of the procedure are depicted in Figure 1.

Case 1

A 52-year-old man presented with a complaint of intense, episodic epigastric pain that suggested a pancreatic origin. Approximately 14 years earlier a diagnosis had been made of alcohol-induced CP. Four years subsequent to this diagnosis endoscopic pancreatic sphincterotomy and extracorporeal shock wave lithotripsy was performed to clear the MPD of stones and relieve pain; at that time a pancreatic stent was also inserted. As part of an experimental study protocol a metal expandable biliary stent (Wallstent, Boston Scientific, Natick, Mass.) was inserted to treat a biliary stenosis and persistent cholestasis.⁹ Cholestasis never recurred. During regular follow-up, the pancreatic stent was changed 4 times and removed without replacement after 4 years.

The patient remained well during the following 6 years, but then presented with abdominal pain and sepsis due to an infected pancreatic fluid collection. Endoscopic cystgastrostomy and placement of a gastrocystic stent resulted in

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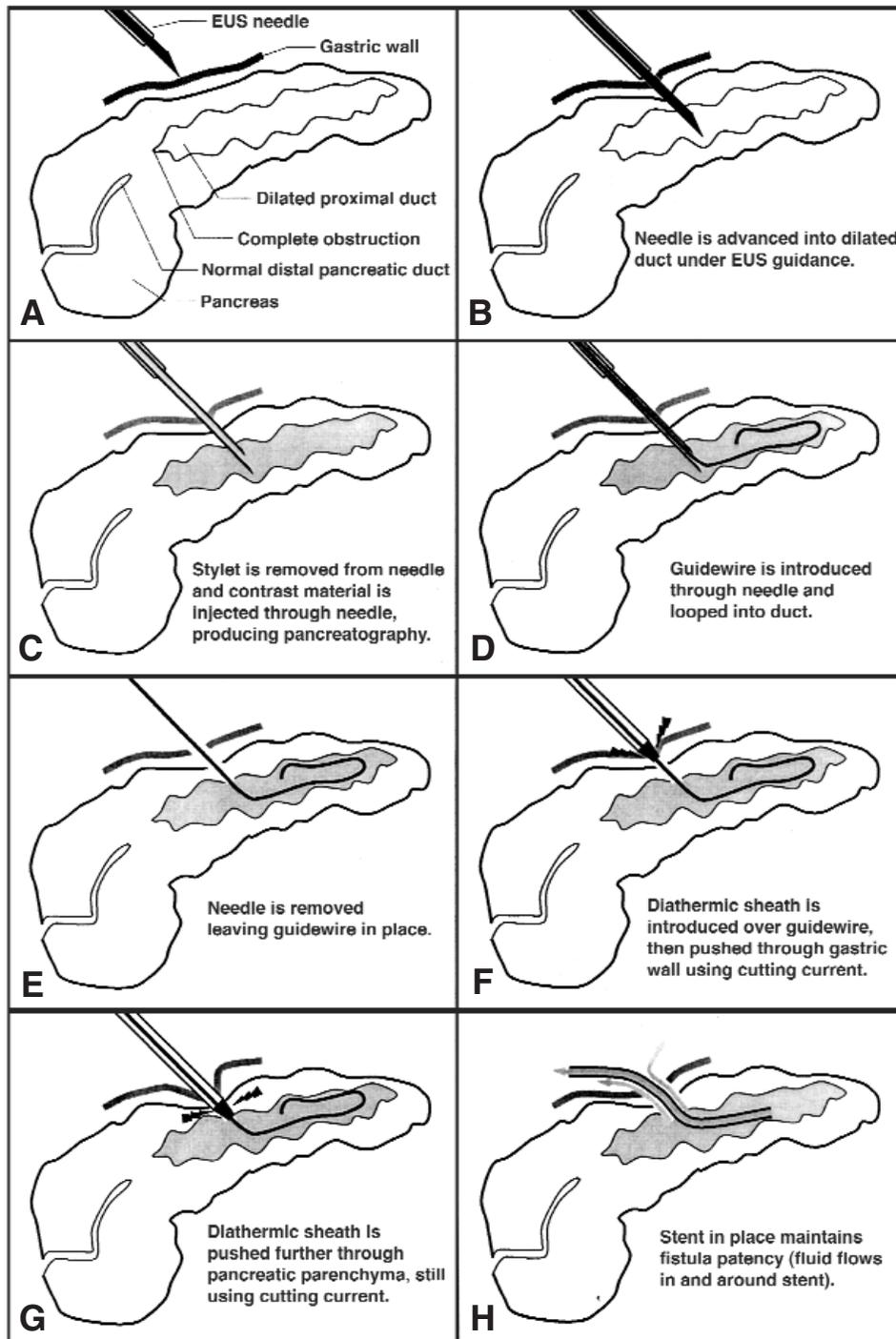


Figure 1. Schematic diagrams of EUS-guided pancreaticogastrostomy.

complete drainage of this collection. At that time, complete obstruction of the MPD at the prepapillary level was evident. Clinically, the patient remained well during the ensuing 7 months but then developed the present episodes of severe epigastric pain. The maximum intensity of the pain was 6/10 on a visual analog scale (VAS). Ingestion of food made the pain worse. Alcohol consumption was less than 20 g per week. CT demonstrated a dilated MPD (maximum diameter 13 mm) and showed the gastrocystic and biliary stents to be

still in place, but showed no residual fluid collection. The pain was assumed to be caused by ductal hypertension.

EPG was performed in the ERCP procedure room with the patient under general anesthesia (propofol), including endotracheal intubation, with an anesthesiologist in attendance. The patient was fully monitored throughout the procedure and afterward in the recovery room. At the beginning of the procedure, antibiotics were administered intravenously (amoxicillin 2 g, clavulante 200 mg) as a

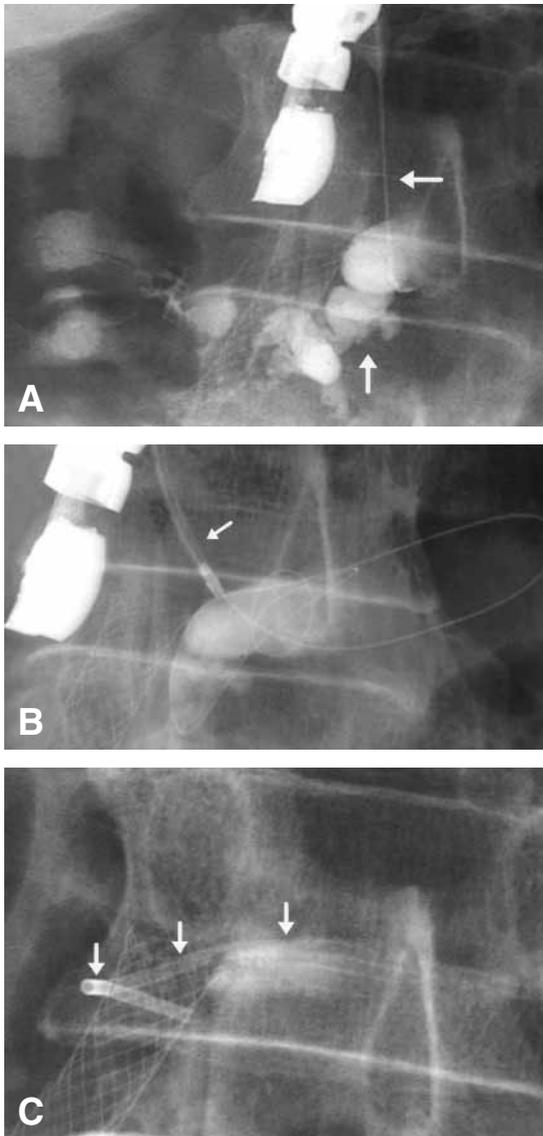


Figure 2. Patient 1. **A**, Radiograph showing echoendoscope with needle (*horizontal arrow*) entering pancreatic duct, which is partially opacified (*vertical arrow*). The metal biliary stent placed 9 years earlier is apparent. **B**, Radiograph showing diathermic sheath (*arrow*) introduced over a guidewire (looped in pancreatic duct). **C**, Radiograph showing pancreaticogastric stent (*arrows*).

prophylactic measure. Total procedure duration from induction of anesthesia to extubation was 50 minutes.

At ERCP, only the distal pancreatic duct could be opacified. By using an echoendoscope (FG 36UX, Pentax, Hamburg, Germany), the dilated MPD was well visualized. EPG was then performed under combined fluoroscopic and ultrasound guidance, with the tip of the echoendoscope positioned such that the inflated balloon was in the duodenal bulb while the accessory channel remained in the antrum. A needle (19 G, Echotip Ultrasound Needle, EUSN-19-T, Cook Ireland Ltd., Limerick, Ireland) was inserted transgastrically into the proximal pancreatic duct and contrast medium was

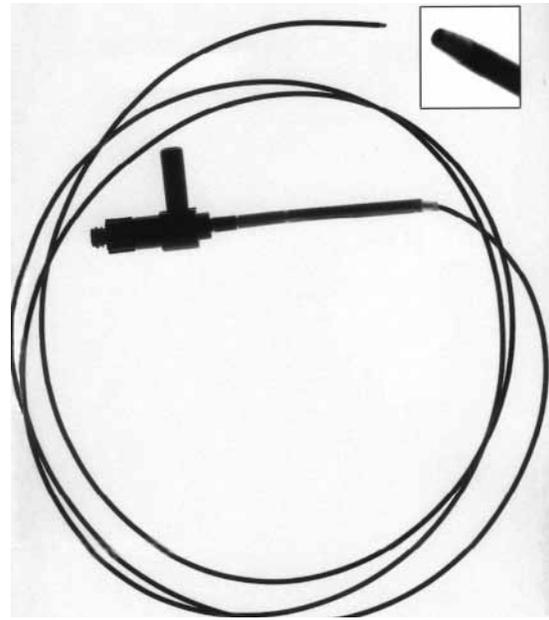


Figure 3. Prototype 6.5F diathermic sheath. *Inset*, detail of metal tip.

injected. Opacification demonstrated a dilated MPD proximal to the complete obstruction (Figs. 1A-C and 2A). The needle was exchanged over a guidewire (0.02 inch diameter, Terumo Europe, Leuven, Belgium) for a 6.5F diathermic sheath (prototype Cysto-Gastro set, EndoFlex, Voerde, Germany) (Figs. 1D-F and 3), which was then used to enlarge the channel between the stomach and MPD (Figs. 1G and 2B). The sheath was introduced by using cutting current. After exchange over a guidewire (TFE-coated 0.035 inch diameter, Cook Europe, Bjaeverskov, Denmark), a 6F, 8-cm-long pancreaticogastric stent (cut from a 6F nasobiliary catheter, Cook Europe) was positioned (Figs. 1H and 2C). There was no bleeding from the puncture site. As observed by fluoroscopy, contrast emptied from the stent into the stomach. The patient was hospitalized the day after and discharged the day after the procedure. There was no postprocedural pain, and he was asymptomatic until discharge. There was no elevation of pancreatic enzyme levels before or at 24 hours after the procedure.

Magnetic resonance cholangiopancreatography (MRCP) 6 months after EPG disclosed a significant reduction in the diameter of the MPD (maximum diameter 13 mm to 7 mm). Dynamic MRCP, with images taken after intravenous injection of secretin, demonstrated intensification of the pancreaticogastric stent and gastric filling, compatible with a patent fistula in a patient with residual pancreatic exocrine function. At 12.5 months after EPG the patient was asymptomatic.

Case 2

A 68-year-old woman presented with a history of 4 episodes of acute abdominal pain with associated elevation of serum amylase/lipase over a period of 2 months that suggested pancreatic disease. Approximately 1 year earlier, she had an episode of acute gallstone-induced pancreatitis that was complicated by the development of a retrogastric fluid collection, treated by endoscopic cystgastrostomy and cyst-

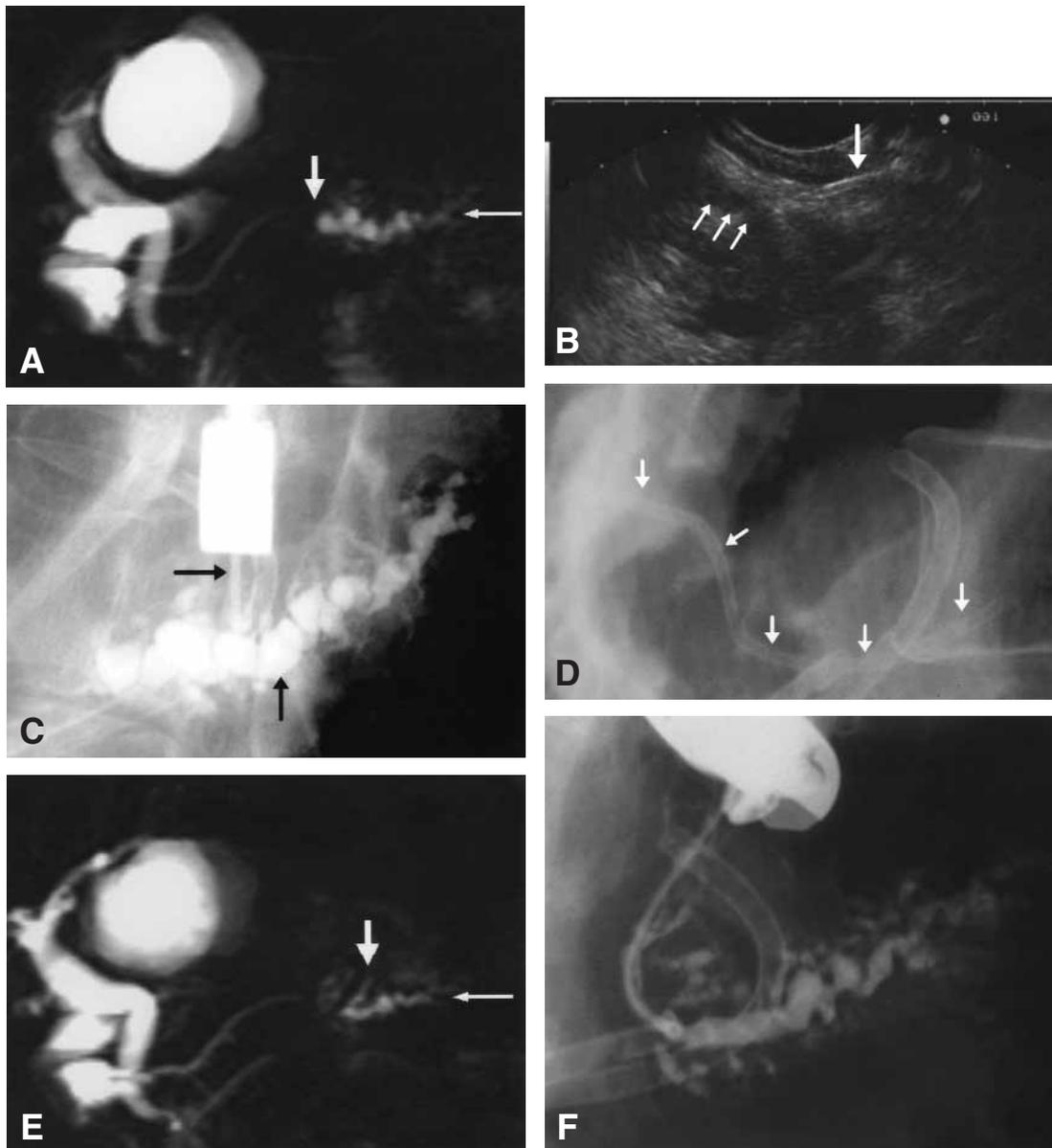


Figure 4. Patient 2. **A**, Secretin-enhanced MRCP image before EPG. The caudal pancreatic duct (*thin arrow*) is dilated proximal to a complete obstruction (*thick arrow*). **B**, EUS image of pancreatic duct (*small arrows*) and needle entering duct (*large arrow*). **C**, Radiograph showing opacification of dilated proximal pancreatic duct (*vertical arrow*) after EUS-guided puncture with needle catheter (*horizontal arrow*). **D**, Radiograph showing pancreaticogastric stent in place (*arrows*). Stents placed during previous cystenterostomies are also apparent. **E**, Secretin-enhanced MRCP after EPG. The caudal pancreatic duct (*thin arrow*) has decreased in diameter, and drainage through the pancreaticogastric fistula leading to the stomach is visible (*thick arrow*). **F**, Radiograph obtained 13 months after EPG. After removal of the stent, the fistula has been recannulated with a balltip catheter and the dilated duct has been opacified.

duodenostomy. MRCP at that time demonstrated pancreas divisum with a normal ventral duct and findings that suggested a leak from the dorsal duct. At ERCP, only the distal portion of the dorsal pancreatic duct could be opacified; the appearance was that of a complete rupture of the duct.

The pain, as evaluated with the VAS, ranged in intensity as high as 9 (scale of 10). Alcohol consumption was less than 20 g/week. MRCP showed dilation of the caudal portion of the dorsal duct, proximal to an obstruction that

had resulted from the earlier complete rupture of the dorsal pancreatic duct (Fig. 4A). There was no residual fluid collection. Pain was assumed to be caused by increased ductal pressure in this segment.

EPG with placement of a pancreaticogastric stent (Fig. 4B-D) were performed as described for patient 1, including general anesthesia and prophylactic administration of antibiotics. There was no complication. Procedure duration was 55 minutes. The patient was hospitalized the day

before EPG and discharged 2 days after the procedure. There was no postprocedural pain, and the patient was well until discharge. Pancreatic enzyme levels were not elevated either before or at 24 hours after the procedure.

MRCP 1 month after EPG disclosed a reduction in diameter of the caudal portion of the dorsal pancreatic duct (Fig. 4E). At 6 months after EPG, the patient was asymptomatic. However, at a scheduled follow-up evaluation 2 months after EPG she complained of weekly episodes of pain (intensity 4/10, VAS), evocative of the original bouts.

Endoscopy was performed with the patient under general anesthesia. The pancreaticogastric stent was visible in the stomach and appeared to have partially migrated from the pancreatic duct. It was removed and the fistula was cannulated with a balltip catheter (Huibregtse-Kalon ERCP catheter, Cook Ireland Ltd.) (Fig. 4F) and a guidewire (0.035-inch diameter, Terumo Europe). There appeared to be a stenosis of the fistula tract at the level of entry into the pancreas, but it was still possible to access the proximal duct. The tract was expanded with a 4-mm diameter, 4-cm long balloon (Maxforce, Boston Scientific International, La Garenne Colombes, France) and a 7F, 5-cm long stent was inserted. At follow-up 2 months after the latter procedure the patient was asymptomatic.

Case 3

A 39-year-old man was referred because of abdominal pain. A diagnosis of alcohol-induced CP was made 4 years earlier. Approximately 1 year before presentation the patient had undergone a pancreaticojejunostomy for relief of chronic pain. Postoperatively, pain relief was not adequate and a celiac plexus block was performed that provided satisfactory but only temporary pain relief. The patient had stopped drinking alcohol about 1 year earlier.

The patient was referred to our center again 9 months later because of frequent episodes of pain despite another celiac plexus block. At ERCP, deep cannulation of the pancreatic duct was not possible because of a tight stricture. However, opacification showed a dilated duct with no flow of contrast medium into the jejunum, indicating dysfunction of the pancreaticojejunal anastomosis. Because the pain was possibly linked to incomplete drainage caused by dysfunction of the surgical anastomosis, EPG was attempted as described for patient 1, including general anesthesia and prophylactic administration of antibiotics. Procedure duration was 40 minutes. The patient was admitted 13 days before EPG and discharged 7 days later, the extended length of hospitalization being due to his social situation rather than medical necessity. No complication was observed.

Pain was not exacerbated after the procedure, but was not significantly improved. There was no elevation of pancreatic enzyme levels before EPG or at 24 hours after the procedure. Although the procedure was technically successful and MRCP 6 days after EPG demonstrated a marked reduction in duct diameter (from 15 to 8 mm), the pain persisted. Another celiac plexus block provided adequate relief for several weeks. When last seen 10 months after EPG, the patient continued to have intermittent pain evocative of a pancreatic origin and was dependent on narcotic analgesics.

No further endoscopic or surgical treatment was performed.

Case 4

A 55-year-old man presented with 2 episodes of abdominal pain (intensity 9/10, VAS) associated with elevated serum levels of pancreatic enzymes. Nine months earlier he was hospitalized at another institution with necrotizing pancreatitis that was complicated by the development of a large abscess involving the head of the pancreas. Surgical drainage was performed and sepsis resolved, but this was followed by the development of a pancreaticocutaneous fistula. MRCP demonstrated probable complete rupture of the MPD, with drainage of the duct in the body and tail of the pancreas through the fistula by means of a pseudocyst cavity. Also noted was a normal-appearing short segment of the distal pancreatic duct that did not communicate with the proximal duct or the fluid collection. The patient was referred to our center 2 months later and was treated by endoscopic cystduodenostomy, which resulted in complete drainage of the pseudocyst into the duodenum and resolution of the fistula. Thereafter, the patient remained well for 4 months until the development of the presenting 2 episodes of abdominal pain. At this time, alcohol consumption was estimated at 40 g/week.

MRCP demonstrated a dilated proximal MPD. At ERCP, it was only possible to cannulate the distal MPD. Because the pain seemed linked to proximal ductal hypertension, EPG was attempted.

The duct was well visualized endosonographically from the antrum and was punctured under direct US and fluoroscopic guidance, as described for patient 1, with placement of a pancreaticogastric stent. No complications were observed. Procedure duration was 30 minutes for EPG; total procedure duration was 180 minutes including attempts at conventional drainage by ERCP. The patient was hospitalized the day before EPG and discharged the day after. There was no postprocedural pain.

At follow-up 7 months after EPG, the patient complained of mild upper abdominal discomfort (3/10, VAS). A fractured stent was noted on plain abdominal x-ray films. Endoscopy was performed with the aim of evaluating, and possibly changing, the pancreaticogastric stent. However, this showed that the stent had migrated from the stomach and was endoscopically inaccessible. Because the symptoms were mild and easily controlled, no further invasive procedures were attempted. At last follow-up 12 months after EPG, the patient was doing well with pain described as mild and intermittent (2 to 3/10, VAS).

RESULTS

Pain caused by CP is complex and multifactorial. Nevertheless, it is presumed to be associated with pancreatic intraductal hypertension in many cases and can be effectively treated by improving ductal drainage. Four patients are described who had pain thought to be caused by insufficient pancreatic duct drainage. Conventional ERCP was attempted but did not allow access to the dilated portions of pancreatic duct targeted for drainage. EUS was then

used to guide direct ductal puncture from the posterior stomach wall.

A relatively straightforward relationship seemed to exist between duct dilation and pain in cases 1, 2, and 4. EPG resulted in decompression, as confirmed by MRCP, and this was associated with pain relief. Case 3, however, was more difficult because the patient had undergone multiple prior treatments and was addicted to opiate analgesics. Pain relief was partial and, perhaps predictably, inadequate. Patients 1, 2, and 4 experienced complete relief of pain and were followed for, respectively, 12.5, 14.5, and 12 months. Patient 2 required a second endoscopic procedure at 11 months after EPG because of recurrence of pain; dilation of the fistula and replacement of the stent provided adequate pain relief during a follow-up of 3.5 months. Longer periods of follow-up are needed to confirm the effectiveness of EPG, evaluate long-term fistula patency, and determine appropriate measures in case of pain recurrence.

EPG was performed with the patient under general anesthesia and with the same conditions as for ERCP, thus allowing fluoroscopic guidance and pancreatography and providing the general infrastructure necessary for complex endoscopic procedures. This type of setting is not the only requirement for this complex procedure; an operator with extensive technical expertise in both therapeutic ERCP and interventional EUS is also essential. In addition, special devices are needed, such as a diathermic sheath, which provides the only means of entry into the pancreatic duct after passing through fibrotic pancreatic parenchyma. Specially designed stents (e.g., single pigtail with proximal side flaps) may avoid the technical pitfall of stent dislocation as observed in patient 4.

DISCUSSION

The results in the present series of patients are much too preliminary in nature to recommend wider use of EPG, which in any case should be restricted to tertiary centers specializing in biliopancreatic therapy. Nevertheless, the possibility of draining the MPD into the digestive tract through an endoscopically cre-

ated fistula, with patency maintained by stent placement, might be interesting as an alternative method of drainage without the complication of stent occlusion that is associated with transpapillary drainage. Indeed, it is clear that even though these small-diameter stents occlude rapidly, the flow of pancreatic juice to the stomach can be maintained alongside the catheter, as illustrated by dynamic MRCP.

In conclusion, pancreatic ductal hypertension, as reflected by duct dilation, is a cause of pain in some patients with CP. Although endoscopic ductal decompression is useful in such cases, conventional ERCP occasionally fails to obtain access to the targeted dilated duct. EUS-guided pancreaticogastrostomy in 4 patients is described here as a new method of ductal decompression in selected cases. Further evaluation of this technique including longer-term follow-up of patients is warranted.

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