

are Brunner's gland hamartomas.⁵ They occur most frequently between the fourth and sixth decades of life and present as UGI bleeding in up to 45% of patients, usually as tarry stools, or as obstruction of the gastric outlet.^{3, 6, 7} In almost 25% of cases the tumor is an incidental finding.⁷ Tumors larger than 2 cm, as in our patient, are very rare.⁸ Disappearance of Brunner's gland hamartoma under treatment with antisecretory drugs was previously described.⁹ In our case, transient regression of the tumor occurred following HP eradication. The explanation for this phenomena may be a transient regression of the acute inflammatory infiltrate around the tumor in response to the triple-drug therapy. The histologic examination of multiple biopsies prior to surgical excision failed to define the correct diagnosis. The biopsy material obtained during upper endoscopy is often superficial and scanty and in our case was limited to the surface of the tumor, which was surrounded by an inflammatory process.

The features of the Brunner's gland hamartoma on EUS examination include (1) involvement of only the mucosa and submucosa and a smooth border, and (2) variable echogenicity, which represents the dilated glands seen within the tumor (Fig. 2B). This appearance on EUS excludes invasive tumors of the

duodenum and suggests the presence of Brunner's gland hamartoma. Such a diagnosis may even defer surgery in certain patients.

REFERENCES

1. Bostford TW, Crowne P, Crocker DW. Tumors of the small intestine. *Am J Surg* 1962;103:358-65.
2. Kouraklis G, Kostakis A, Delladetsima J. Hamartoma of Brunner's glands causing massive haematemesis. *Scand J Gastroenterol* 1994;29:841-3.
3. Kaplan EL, Dyson WL, Fitts WT. Hyperplasia of Brunner's glands of the duodenum. *Surg Gynecol Obstet* 1968;126:371-5.
4. Caletti GC, Ferrari A, Brocchi E, Bonora G, Carfagna L, Barbara L. Normal EUS anatomy: the gut wall. In: Sivak MV, Zuccaro G, editors. *Endoscopic ultrasonography course syllabus*. Cleveland: Cleveland Clinic Foundation; 1991. p. 35-42.
5. Attanoos R, Williams GT. Epithelial and neuroendocrine tumors of the duodenum. *Semin Diagn Pathol* 1991;8:149-62.
6. Schluger LK, Rotterdam H, Lebwohl O. Gastrointestinal hemorrhage from a Brunner's gland hamartoma. *Am J Gastroenterol* 1994;89:2088-9.
7. Levine JA, Burgait LJ, Batts KP, Wang KK. Brunner's gland hamartomas: clinical presentation and pathological features of 27 cases. *Am J Gastroenterol* 1995;90:290-4.
8. Khawaja HT, Deakin M, Colin-Jones DG. Endoscopic removal of a large ulcerated Brunner's gland adenoma. *Endoscopy* 1986;18:199-201.
9. De-Angelis G, Villanacci V, Lovotti D, Gianni E, Mazzi A, Buonocore M, et al. Hamartomatous polyps of Brunner's gland. Presentation of 2 cases. Review of the literature. *Minerva Chir* 1989;44:1761-6.

Dilation of a difficult benign pancreatic duct stricture using the Soehendra stent extractor

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Endoscopic therapy for pancreatic disease continues to evolve. Treatment of benign pancreatic strictures with dilation and stent placement can lead to improvement in abdominal pain in selected patients with chronic pancreatitis.¹⁻² Recently, difficult malignant biliary strictures that allowed passage of only a guidewire have been traversed and dilated using a Soehendra stent extractor.³⁻⁵ This report

describes its use for dilating a difficult benign pancreatic stricture.

CASE REPORT

A 59-year-old man with a long history of alcohol abuse presented with progressive abdominal pain. Sixteen years earlier he began experiencing acute recurrent pancreatitis related to alcohol abuse. Complications included development of a pancreatic pseudocyst that was surgically drained 15 years ago followed by percutaneous drainage of another pseudocyst 5 years ago. He discontinued alcohol use 6 months before this presentation of progressive abdominal pain similar to his pancreatitis type pain. An abdominal CT scan revealed a markedly dilated pancreatic duct from the neck of the pancreas to the tail without biliary ductal dilatation or significant pancreatic calcifications. Endoscopic retrograde pancreatography was performed with a diagnostic duodenoscope (Olympus JF-100, Olympus America Inc., Melville, N.Y.). A cholangiogram demonstrated no stricture formation or other biliary abnormalities. Cannulation of the pancreatic duct through the major papilla revealed a normal ventral pancreas, and cannulation of the dorsal pancreatic duct through the minor papilla confirmed pancreas divisum. The distal pancreatic duct was relatively normal with a tight focal stricture at the neck of the pancreas, accompanied by

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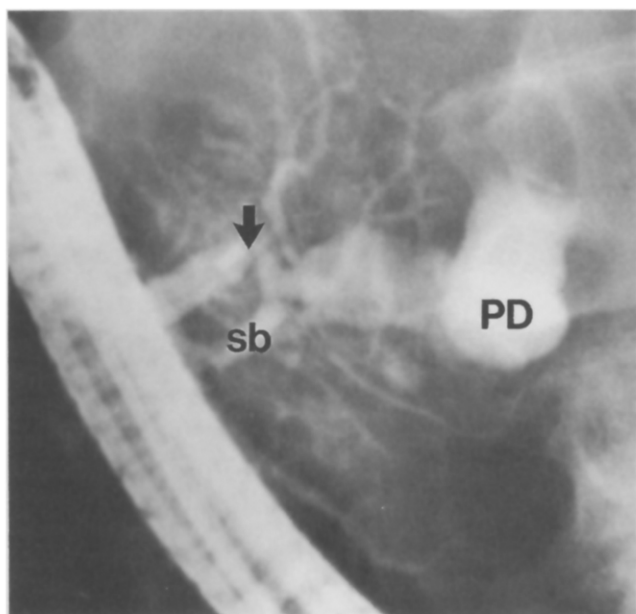


Figure 1. Benign pancreatic stricture. Endoscopic retrograde pancreatography demonstrates a nearly occlusive main pancreatic duct stricture (*small arrow*), inhibiting passage of a standard 5F catheter into the markedly dilated, more proximal dorsal duct (*PD*). A dilated side branch (*sb*) arising proximal to the stricture overlies the dominant stricture.

marked upstream pancreatic ductal dilatation (13 mm when corrected for magnification) (Fig. 1). Although a 0.035 inch angled hydrophilic guidewire (Microvasive Inc., Nadick, Mass) was advanced across the stricture after repeated attempts, a tapered 5F catheter (Glo-tip, Wilson-Cook Medical Inc., Winston-Salem, N.C.) would not pass across the stricture. The catheter was removed, and the hydrophilic wire was left in place. A 7F Soehendra stent extractor (Wilson-Cook Medical Inc.) was advanced over the wire to the minor papilla. Neither minor papilla sphincterotomy nor dilation had been performed because it was not anticipated to be necessary. The stent extractor required clockwise torque with pressure to advance through the minor papilla into the pancreatic duct (Fig. 2). The stricture was traversed by torquing the stent extractor clockwise while exerting continuous antegrade pressure for approximately 3 minutes as previously described for dilating malignant biliary strictures³⁻⁵ (Fig. 3). The hydrophilic wire was exchanged for a standard Teflon 0.035 inch guidewire. The stent extractor was removed and a 7F, 7 cm Geenen pancreatic stent (Wilson-Cook Medical Inc.) was inserted into the pancreatic duct proximal to the stricture. The patient did well and demonstrated no clinical signs or symptoms of acute pancreatitis, although results of a 24-hour postprocedure amylase test, done at the time of discharge, were 678 mg/dL (normal ≤ 110 mg/dL). The patient's abdominal pain resolved completely. A follow-up ERCP 1 month later showed improvement in the stricture; a 5F catheter easily passed. The stricture was then dilated with a 6 mm

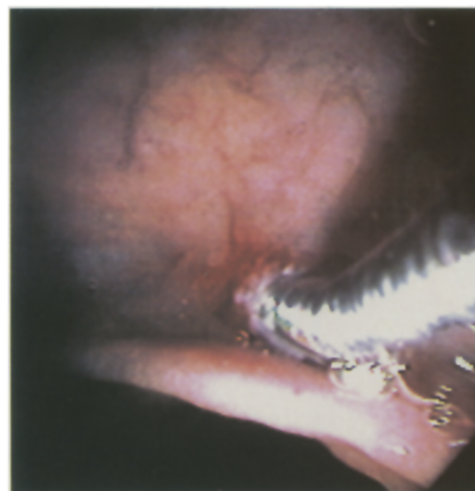


Figure 2. Endoscopic photo of Soehendra stent extractor passing through the minor papilla.

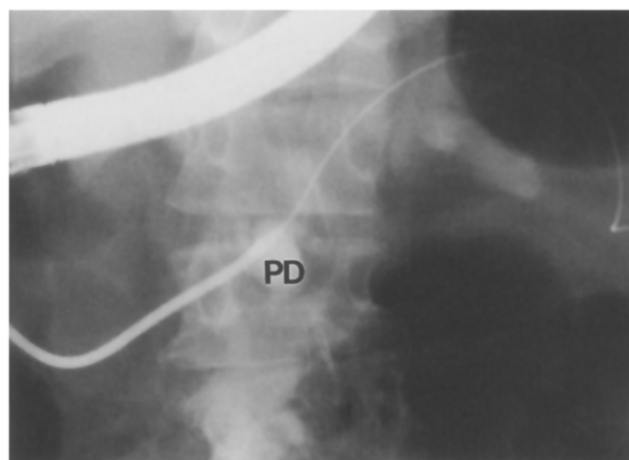


Figure 3. Following clockwise rotation, the Soehendra stent extractor has successfully traversed the stricture. *PD*, Dilated genu of pancreatic duct.

hydrostatic balloon and restented using side by side pancreatic stents with the proximal ends proximal to the stricture as per our own protocol (unpublished) because we have had poor results with short-term stenting using only one stent. The patient remains asymptomatic with indwelling stents 8 months after the initial dilation. The plan is to leave the stents for 12 months (Fig. 4).

DISCUSSION

Endoscopic therapy for pancreatic strictures is being increasingly performed. Sustained relief of abdominal pain is possible after stricture dilation and stenting in up to 65% of patients with a dominant stricture occurring in the setting of chronic pancreatitis.¹⁻² Successful endoscopic management of pancreatic strictures requires passage of a guidewire through the narrowed region, followed by place-

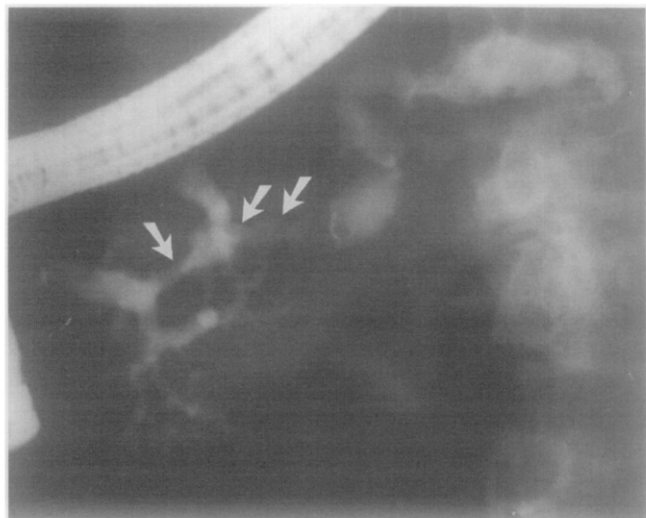


Figure 4. Follow-up pancreatogram 6 months after dilation and stenting reveals patent but slightly irregular dilated genu of pancreatic duct (PD) through the previously strictured region (arrows). Proximal side branch and residual main PD are seen.

ment of a stent after either rigid or balloon dilation. Currently, most dilators are not mounted on catheters smaller than 5F. Although a graduated dilator may have allowed passage across the stricture, it has been our experience that if a 0.035 inch guide-wire can be passed but a tapered tip 5F catheter will not, it is unlikely the stricture can be dilated using this method. Therefore, failure of a 5F catheter to pass the stricture equates with inability to successfully dilate and stent the stricture. Van Someron et al.³ described passage of a Soehendra stent extractor across difficult malignant biliary strictures, allowing successful stricture dilation and stent placement. The same group reported on their larger experience, using this technique successfully in 18 of 19 patients with malignant hilar biliary obstruction in which the strictures would initially allow passage

of only a 0.021 inch wire.⁴ No complications were seen. Similarly, Silverman and Slivka⁵ successfully used this technique to create an opening within a biliary Wallstent placed at the hepatic bifurcation to facilitate deployment of a second Wallstent into the contralateral intrahepatic system. Our case is the first describing the use of this technique in the pancreatic duct and its use in treating benign strictures. Because patients with established chronic pancreatitis are less susceptible to the development of severe acute pancreatitis than patients with relatively normal pancreatic parenchyma, dilation of pancreatic strictures with the Soehendra stent extractor should be performed only as a last resort in patients with chronic pancreatitis for nonoperative management of difficult strictures. This recommendation may change as more experience is gained using this technique. Additionally, the long-term outcome of benign strictures using this technique needs to be evaluated because alteration in the stricture (for better or worse) and removal of some tissue may occur as the extractor is screwed through the stricture.⁴

REFERENCES

1. Binmoller KF, Jue P, Seifert H, Nam WC, Izbicki J, Soehendra N. Endoscopic pancreatic stent drainage in chronic pancreatitis and a dominant stricture: long term results. *Endoscopy* 1995;27:638-44.
2. Ponchon T, Bory RM, Hedelius F, Roubein LD, Paliard P, Napoleon B, et al. Endoscopic stenting for pain relief in chronic pancreatitis: results of a standardized protocol. *Gastrointest Endosc* 1995;42:452-6.
3. van Someren RNM, Benson MJ, Aimley CC, Glynn MJ, Swain CP. No need to be defeated by the tight malignant biliary stricture at initial ERCP—a new technique [abstract]. *Gastroenterology* 1994;106:A364.
4. Niall R, van Someren RNM, Benson MJ, Glynn MJ, Ashraf W, Swain CP. A novel technique for dilating difficult malignant biliary strictures during therapeutic ERCP. *Gastrointest Endosc* 1996;43:495-8.
5. Silverman W, Slivka A. New technique for bilateral metal mesh stent insertion to treat hilar cholangiocarcinoma. *Gastrointest Endosc* 1996;43:61-3.

The whirlpool jet technique for removal of pancreatic duct ascaris

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Infestation with roundworms (*Ascaris lumbricoides*) is one of the most common parasitic diseases, involving more than a billion people worldwide.^{1,2} Ascariasis is most common in countries with tropical climates where the warm weather and wet soil create conditions that are conducive to the development of the larval stage of the parasite and the sustenance of the ascaris ova.³ The infection spreads by the fecal-oral route, which is promoted by conditions such as crowding, poor sanitation and hygiene, and unsafe water supply. Moreover, in many of the endemic countries, the human excreta is used as a