Review Article

Endoscopic management of recurrent pancreatitis

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Abstract

Endoscopic therapy is a well-established, valuable alternative to surgery for the treatment of a variety of biliary tract diseases. Applications of similar techniques to the pancreas continue to develop. Encouraged by the recognition of the relative safety of ERCP and endoscopic sphincterotomy (ES) for gallstone pancreatitis, endoscopic therapy is now being applied in the setting of acute pancreatitis of other etiologies, chronic pancreatitis, complications of acute or chronic pancreatitis, and pancreas divisum.

Key words

Endoscopic sphincterotomy, pancreas divisum, recurrent pancreatitis

Introduction

Endoscopic therapy is a well-established, valuable alternative to surgery for the treatment of a variety of biliary tract diseases. Applications of similar techniques to the pancreas continue to develop. Progress in this area has been somewhat slow because of the concern for procedure-related complications. Encouraged by the recognition of the relative safety of ERCP and endoscopic sphincterotomy (ES) for gallstone pancreatitis, endoscopic therapy is now being applied in the setting of acute pancreatitis of other etiologies, chronic pancreatitis, complications of acute or chronic pancreatitis, and pancreas divisum [Table 1]. Since one third of "acute" pancreatitis patients already have chronic pancreatitis by more detailed evaluation, this approach applies to both groups. While the underlying cause of the pancreatitis (ethanol, high triglycerides etc) should be corrected if possible, most endoscopic therapies apply to all etiologies whether correctable or not. Pancreatic techniques are more technically demanding as noted by the Schutz Classification.

Management of Recurrent Pancreatitis

Management of recurrent pancreatitis can be categorized

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according to noninvasive imaging and serologic testing.

1. Specific findings such as biliary or pancreas stones (for which endoscopic, surgical or other therapy is appropriate).

2. Nonspecific abnormality such as ductal dilation. 3. Normal. The latter two groups are better managed at referral center with a full armamentarium of techniques available (eg. Manometry, ductoscopy, stentings, etc). Fischer reported the endoscopic finding on a large series of acute and acute recurrent idiopathic pancreatitis [Table 2].^[1]

Sphincter of Oddi Dysfunction (SOD) was the most common finding. Pancreatic basal sphincter pressure was abnormal in 57% of 888 manometrically studied patients while 36% had biliary sphincter basal pressure ≥40 mmHg. Biliary sphincterotomy alone improves 50-70% of such patients while combined pancreatobiliary is more efficacious. Residual pancreatic duct sphincter hypertension may account for the lack of improvement following biliary ES alone. Thorough evaluation of such patients includes bi-ductal manometry (both initially and if repeat study is needed). Recent studies show that rectal NSAIDS given immediately after ERCP decrease post ERCP pancreatitis by 50%. [2] Outcome studies are limited but SOD therapy gives pain/pancreatitis resolution in approximately 50% of chronic pancreatitis patients and 80% of patients without evidence of chronic pancreatitis.

Chronic pancreatitis changes were the 2nd most common finding (35%) in the Fischer series. [1] Management of pancreatic ductal strictures, pseudocysts, and stones is similar no matter what the etiology of pancreatitis. Simple management of pancreatic duct stricture is dilation and plastic stents. Duration and diameter of stenting needed to give stricture long-term patency is only partially known. Longer and bigger seems

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Table 1: Endoscopic therapy for recurrent and/or chronic pancreatitis

Clinical condition	Endoscopic therapy
Recurrent or chronic pancreatitis, smoldering pancreatitis	Endoscopic sphincterotomy (bile duct and/ or pancreatic), stricture dilation, stone extraction ± extracorporeal shockwave lithotripsy, bile duct and/or pancreatic duct stents
Pancreatic pseudocysts, PD disruption/ascites	Cystoenterostomy, transpapillary stenting (± EUS assistance)
Pancreas divisum	Minor papilla endoscopic sphincterotomy, stent and/or dilation
Pancreatic necrosis	Necrosectomy

Table 2: Post-procedure diagnosis*

Clinical condition	Number of patients	Percent of 1,241
Sphincter of oddi dysfunction	501	40.4
Pancreas divisum	233	18.8
Periampullary diverticulum	75	6.0
Intraductal mucinous tumors	52	4.2
Choledocholithiasis / ductal sludge	19	1.5
Cholelithiasis / gallbladder sludge	19	1.5
Choledochal cysts	10	0.8

^{*}Some of the patients had more than one diagnosis

better but this involves multiple endoscopic sessions and may require expandable metal stents (fully covered) to achieve full luminal patency. Further studies comparing plastic stents to fully coated metal stents are awaited.

Cahen^[3] reported that surgical bypass treatment of head main duct strictures gave superior pain relief at 5 years than endoscopic (incomplete) therapy. Thorough risk-benefit discussion should take place before initiating endoscopic or surgical treatment. Pseudocysts, stones and necrosis will be left to more specific chronic pancreatitis sessions.^[4]

Pancreas divisum is a congenital anomaly which causes relative obstruction to outflow of pancreatic juice via an always small diameter minor papilla. Recent studies show an association between genetic abnormalities (PRSS1, SPINK1, and cystic fibrosis) and pancreas divisum.^[5,6]

Similar to sphincter of Oddi dysfunction of the major papilla, approximately 80% of pancreas divisum patients without chronic pancreatitis will have pancreatitis resolution/improvement for 2-5 years after endoscopic sphincterotomy. Some studies report similar efficacy from only placement of 5-7 Fr stent χ 3-6 months. We find that such stent therapy

causes ductal strictures upstream to the minor papilla and is strongly discouraged.

Acute recurrent pancreatitis with associated LFT abnormality raises concern for biliary etiology. Noninvasive imaging (US, EUS, MRCP) will usually clarify the biliary (or not) etiology. This is an important categorization point as biliary stone patients have low risk from biliary sphincterotomy, whereas other etiologies (especially sphincter of Oddi dysfunction) are high risk for post ERCP complications. When ductal biliary stones are suspected but not found, factors to weigh in the final treatment plan are 1) current/past gall bladder stones; 2) lacerated papilla as from stone passage; 3) pancreatogram (preferably limited extent) evidence of an etiology such as mucus in duct; 4) sphincter manometry availability; 5) biliary sphincterotomy alone vs. additional pancreatic prophylactic stent. These factors are especially important in smaller hospitals without manometry or pancreatic stenting availability.

Conclusion

Endoscopic therapy of pancreatic disease is an evolving arena. Selection of appropriate candidates for the various treatment modalities appears important for optimal results of therapy. Controlled studies with long term follow up are awaited.

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