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The sphincter of Oddi and acute pancreatitis – revisited

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Background

One of the rare causes of recurrent acute pancreatitis is sphincter of Oddi dysfunction. This condition is objectively diagnosed by manometry of the sphincter of Oddi. An abnormally elevated sphincter of Oddi basal pressure has been shown to predict patients who have a successful outcome after transduodenal sphincteroplasty and pancreatic duct septoplasty.

Methods

Forty-nine patients presenting with recurrent pancreatitis and who had manometric stenosis of the sphincter of Oddi were treated by transduodenal division of the sphincter of Oddi. Clinical follow-up was conducted over a minimum of 2 years.

Results

In all, 43 patients were either cured or improved. None of these patients had any further episodes of pancreatitis. Three of these patients developed recurrent symptoms and were noted to have restenosis of the sphincter of Oddi. They were treated by insertion of an endoscopic stent into the pancreatic duct.

Conclusion

The results from this series of patients re-affirm the efficacy of transduodenal sphincteroplasty and septoplasty for treatment of sphincter of Oddi stenosis in patients presenting with recurrent acute pancreatitis.

Keywords

sphincter of Oddi, acute pancreatitis, sphincteroplasty, septectomy, manometry

Introduction

The association between sphincter of Oddi dysfunction and pancreatitis is now well established. An early study identified the manometric abnormalities of the sphincter of Oddi that were thought to lead to episodes of pancreatitis [1]. Furthermore, it was shown that total division of the sphincter of Oddi (i.e. division of the biliary as well as the pancreatic component of the sphincter) resulted in cure of recurrent episodes of pancreatitis in the majority of patients [2]. The manometric abnormality associated with best results was an abnormally elevated sphincter of Oddi basal pressure, indicative of sphincter of Oddi stenosis.

As a result of these findings, a treatment strategy evolved so that patients who had recurrent episodes of pancreatitis of unknown cause were investigated by sphincter of Oddi manometry. If a pancreatic duct stenosis was diagnosed, then operative division of the whole sphincter of Oddi was undertaken.

The aim of this report was to evaluate the long-term outcome of a cohort of patients who were treated for sphincter of Oddi dysfunction that was associated with recurrent episodes of pancreatitis.

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Patients and methods

Between January 1984 and December 1999, 49 patients with pancreatic sphincter of Oddi stenosis and recurrent pancreatitis were treated. In the same time period, over 1500 patients with pancreatitis were treated in the institution, the common causes of pancreatitis being gallstones and alcohol. The selected cohort of 49 patients had all other causes of pancreatitis eliminated by careful history and relevant investigations. All patients had at least two documented episodes of pancreatitis with abnormally elevated serum amylase and most had several documented episodes requiring inpatient hospital treatment.

Investigations

All patients underwent endoscopic retrograde cholangiopancreatography (ERCP) to eliminate a structural cause for pancreatitis or stones in the bile duct. Bile was not routinely examined for microlithiasis, as in a previous study this was not found to be useful in these patients [2]. Endoscopic ultrasound was not available for screening these patients for this condition.

All the patients then underwent sphincter of Oddi

manometry with the manometry catheter directed into the pancreatic duct so as to record the pancreatic duct sphincter pressures. The technique of sphincter of Oddi manometry has been described previously [1]. As in previous studies, diagnosis of pancreatic duct sphincter of Oddi stenosis is made when the basal sphincter of Oddi pressure is in excess of 40 mmHg.

Treatment

All patients had pancreatic duct sphincter of Oddi stenosis, hence total division of the sphincter of Oddi via a traditional approach was undertaken. The operation has been described in detail previously [2]. In brief, a transduodenal biliary sphincteroplasty was done and then the pancreatic duct opening was cannulated using fine probes. The septum between the pancreatic and bile ducts was divided until wide entry into the pancreatic duct was achieved.

Follow-up

All the patients were followed up by the author at 6–12-week intervals for the first year and then at 2 years; if they were well, there was no further follow-up. For patients who lived a long distance away, follow-up by contact through the referring doctor was undertaken after the initial 12-week interval. In order to avoid bias, the outcome results only used objective parameters.

On follow-up the patients were classified into one of three categories:

(1) *Completely cured* if there were no further episodes of pain or pancreatitis, no need for parenteral analgesia and no hospital admission for pancreatitis.

(2) *Improved* if there were occasional episodes of abdominal pain that were not associated with documented hyperamylasaemia and did not require hospital admission.

(3) *No change* if the symptoms and signs continued.

The disorder was classified as *recurrent* if the patient was cured or improved for 2 years and then the symptoms and signs recurred.

Results

Patients

Most patients (43 of 49) were female and the median age was 43 (range 20–70) years. None of them had a relevant alcohol intake history, with only four reporting occasional alcohol consumption. All but one patient had cholecystectomy before referral. Eight patients had a previous endoscopic sphincterotomy of the biliary sphincter without alteration of symptoms. No patient underwent endoscopic sphincterotomy of the pancreatic sphincter.

Investigations

All patients had documented rises in serum amylase associated with the clinical presentation of pancreatitis. In 27 patients the serum liver enzymes also demonstrated abnormalities, particularly in the serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT). ERCP did not reveal structural abnormalities in the pancreas or gallstones in the bile duct.

Sphincter of Oddi manometry revealed manometric stenosis (abnormal sphincter of Oddi basal pressure) in all 49 patients.

Postoperative Management

All patients had a T-tube inserted in the bile duct on conclusion of the operation. This tube was left open until a cholangiogram showed resolution of oedema at the choledochoduodenal junction. The outer limb was clamped (1 week after operation) and the patient was discharged home.

There was no incidence of pancreatitis in the postoperative period. In three patients, a bile leak occurred on removal of the T-tube 4 weeks after the operation. This event produced local peritoneal irritation requiring analgesics. None needed re-intervention to drain a collection.

Follow-up

The results of follow-up are shown in Table 1. In 87% of patients with manometric stenosis of the pancreatic sphincter of Oddi there was complete cure or improve-

Table 1. *Results of division of the sphincter of Oddi*

Patients	Number (n = 49)
Cured	35 (71%)
Improved	8 (16%)
No change	6 (12%)
Restenosis (>2 years)	3

ment after total division of the sphincter of Oddi. The successful outcome was sustained for a minimum of 2 years and, in some patients, over 10 years. As routine documented follow-up was not maintained beyond the first 2 years, a median length of follow-up that includes all patients cannot be recorded.

Recurrence of pancreatitis occurred in three patients after 2 years without symptoms. These patients have had repeat ERCP and sphincter of Oddi manometry, which showed a restenosis of the pancreatic duct opening. They were treated by inserting a pancreatic stent across the opening for 12 months. The symptoms were alleviated in all three patients; however, long-term follow-up is not available.

Discussion

This report describes a highly selected cohort of 49 patients who presented with recurrent episodes of pancreatitis and who, on sphincter of Oddi manometry, were shown to have stenosis of the pancreatic component of the sphincter of Oddi. As part of a prospective protocol, all patients underwent treatment by total division of the sphincter of Oddi via transduodenal septoplasty and pancreatic duct septectomy. In this highly selected group of patients, the results strongly support the conclusion that sphincter of Oddi dysfunction is associated with pancreatitis and that division of the sphincter of Oddi results in resolution of pancreatitis episodes.

Since the earlier reports of successful surgical division of the sphincter of Oddi for this condition [2], a number of endoscopic units have embarked on endoscopic treatment of both the biliary and pancreatic sphincters for this condition [3, 4]. Placement of a pancreatic stent is advocated to avoid pancreatitis following total division of the sphincter of Oddi [5, 6]. However, even with this approach, the best reports are associated with a 10% rate of post-procedure pancreatitis. Furthermore, repeat procedures are required in all patients to remove the pancreatic duct stent. As yet, long-term results are not available in these patients, but would need to match those of patients treated via the transduodenal approach if the technique is to be adopted. One major advantage of the endoscopic approach is the recovery of the patients, as there is no need for an abdominal incision. However, a 10% pancreatitis rate might adversely impact on this recovery. To date there has been no comparison of the two approaches.

An advantage of the operative approach is the control that the operator has in dividing the pancreatic sphincter. At operation, a biliary sphincteroplasty is done first. This initial procedure opens up the lower end of the bile duct, thus bringing the opening of the pancreatic duct into view. The part of the pancreatic sphincter that makes up the septum between the pancreatic and bile ducts can then be divided under vision and for a length that will ensure that the stenotic segment has been effectively divided. Subjectively, this measure of control may be the reason for the difference in early complication rates between the open and endoscopic approaches. Furthermore, this may translate to the sustainability of the long-term results

In this operative series, there was a 5% restenosis incidence on long-term follow-up. The incidence is low. The cause of restenosis is uncertain. Potentially it may be due to a recurrence of the original problem that led to stenosis in the first instance. Alternatively it may be a result of the healing process, with fibrosis at the sphincterotomy leading to narrowing of the pancreatic duct opening. What is encouraging for this series of patients is the subsequent prognosis after endoscopic re-intervention. All the patients had the restenosis successfully managed via this approach.

Sphincter of Oddi dysfunction leading to recurrent episodes of acute pancreatitis has been established as an identifiable entity. Manometry defines those patients who will respond to treatment of total division of the sphincter of Oddi. This report of a moderately sized cohort of patients with this condition emphasises the need for objective diagnosis via manometry, and treatment via open sphincteroplasty and pancreatic duct septoplasty.

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