Laparoscopic Management Of Acquired Benign Duodenal Strictures In Adults

M Al-Rashedy, Y El-Dhuwaib, M Issa, P Ballester, B Ammori

Abstract

Background & Aims: Benign duodenal strictures in adults are a rare cause of gastric outlet obstruction. The application of laparoscopic surgery to the management of benign duodenal pathology has previously been scantily explored. Our experience with the laparoscopic approach is presented and discussed.

Methods: Six patients (4 men) with a median age of 46.5 years (range 31-74 years) underwent seven laparoscopic procedures for symptomatic duodenal (D) strictures/stenoses. The morphology of the duodenal disease included diaphragm-like strictures at the junction of D1/D2 in two patients, stenosis of D2 in two patients, a long post-bulbar D1 stricture in one patient, and long distal strictures (D3/D4) in two patients. The aetiology of the strictures was related to non-steroidal anti-inflammatory drugs (n=2), acute pancreatitis (n=1), chronic pancreatitis (n=1) and duodenal tuberculosis (n=1), and was idiopathic in one patient. Attempts at endoscopic and medical therapy were unsuccessful. Surgical treatment included laparoscopic pancreas-preserving distal duodenectomy with duodeno-jejunal anastomosis (n=1), loop gastroenterostomy (n=4), Roux-en-Y gastroenterostomy (n=1), and a loop gastroenterostomy combined with a Roux-en-Y hepaticojejunostomy and bilateral thoracoscopic splanchnotomy in one patient.

Results: All operations were completed laparoscopically. Re-laparoscopy for bleeding was required after duodenectomy in one patient. The median postoperative hospital stay was 4 days (range 3-19 days). The patient who underwent a duodenectomy developed a further diaphragm-like stricture at D1/D2 that was managed 3 months postoperatively with a laparoscopic Roux-en-Y gastroenterostomy, and a subsequent laparoscopic revision of this anastomosis 2 months later. No evidence of further recurrence of the gastric outlet obstruction was observed at a median follow up of 9 months (range 2-32 months).

Conclusions: In patients with benign duodenal obstruction, surgery should be reserved to those in whom medical or endoscopic therapy has failed and when suspicion of malignancy persists. Laparoscopic surgery (duodenal resection or gastroenterostomy) offers a safe and effective alternative to laparotomy in the management of benign duodenal strictures in adults.

INTRODUCTION

In the modern era of proton pump inhibitors (PPI), benign duodenal strictures are a rare cause of gastric outlet obstruction in adults, whilst malignancy is the main culprit. The causes of duodenal stenosis and obstruction may be classified as congenital and acquired, and the latter may be subdivided into benign and malignant causes (Table 1).
Laparoscopic Management of Acquired Benign Duodenal Strictures in Adults

We report our experience with the laparoscopic management (duodenal resection and bypass) of five patients who developed gastric outlet obstruction secondary to benign duodenal strictures. The applications of laparoscopic surgery in patients with benign duodenal pathology are discussed.

MATERIAL AND METHODS

PATIENTS

Between February 2001 and March 2003, we have treated six patients (4 men, 2 women) with a median age of 46.5 years (range 31-74 years) who presented with symptoms of gastric outlet obstruction secondary to duodenal strictures/stenoses. The patient details are summarised in Table 2. The median duration of preoperative symptoms was 4 months (range 1-42 months).

PREOPERATIVE ASSESSMENT OF THE DUODENAL PATHOLOGY

All patients underwent thorough investigations to assess the nature of the duodenal strictures including endoscopy, endoscopic biopsies, computed tomography (CT), and barium meal (Table 3). These demonstrated a diaphragm-like stricture at the junction between the first and second parts of the duodenum in one patient, a long post-bulbar proximal duodenal stricture in one patient, duodenal stenosis (second part) in two patients (Figure 1), and long distal duodenal strictures (third & fourth parts) in two patients (Figure 2).

Table 1: Aetiology of duodenal stenosis and obstruction

<table>
<thead>
<tr>
<th>Congenital causes</th>
<th>Acquired diseases</th>
<th>Malignant causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duodenal atresia [21]</td>
<td>Peptic ulcer disease [28], Zollinger-Ellison syndrome [39]</td>
<td>Cancer of the duodenum, pancreas, ampulla, bile duct or gallbladder [34]</td>
</tr>
<tr>
<td>Choledochal cyst [20]</td>
<td>Trauma [43], child abuse [42]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cystic Fibrosis [92]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Duodenal tuberculosis [51]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hyperplasia of the Brunner’s gland (Brunneroma) [22]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Superior mesenteric artery syndrome [19]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ingestion of corrosive agents [46]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anaphylactic purpura (purpura rheumatica) [19]</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1

Table 1: Aetiology of duodenal stenosis and obstruction

Table 2: Details of the patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>ASA score</th>
<th>Presentation</th>
<th>Preoperative duration of symptoms</th>
<th>Preoperative hospital stay</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>71</td>
<td>M</td>
<td>3</td>
<td>Progressively worsening vomiting &amp; a CABG procedure 2 months prior to admission</td>
<td>3 months</td>
<td>33 days</td>
</tr>
<tr>
<td>2A</td>
<td>40</td>
<td>F</td>
<td>2</td>
<td>Repeated vomiting, episodes of haematemesis and melena requiring blood transfusion, and weight loss</td>
<td>6 months</td>
<td>3 days</td>
</tr>
<tr>
<td>2B</td>
<td>40</td>
<td>F</td>
<td>2</td>
<td>Recurrence of vomiting 2 months after laparoscopic duodenectomy</td>
<td>1 month</td>
<td>6 days</td>
</tr>
<tr>
<td>3</td>
<td>31</td>
<td>F</td>
<td>2</td>
<td>Persistent vomiting following an attack of acute necrotising biliary pancreatitis 4 months earlier</td>
<td>3 months</td>
<td>92 days</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>M</td>
<td>3</td>
<td>Obstructive jaundice, episodes of cholangitis, repeated vomiting, and osteo-dependent chronic abdominal pain</td>
<td>42 months</td>
<td>1 day</td>
</tr>
<tr>
<td>5</td>
<td>74</td>
<td>M</td>
<td>2</td>
<td>Repeated vomiting and weight loss (symptoms started after discontinuation of steroid therapy for temporal arteritis)</td>
<td>6 months</td>
<td>42 days (admitting hospital) + 10 days</td>
</tr>
<tr>
<td>6</td>
<td>40</td>
<td>M</td>
<td>2</td>
<td>Repeated vomiting and weight loss</td>
<td>4 months</td>
<td>26 days</td>
</tr>
</tbody>
</table>
### Table 3: Diagnosis

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Extent &amp; location of the duodenal stricture</th>
<th>Aetiology</th>
<th>Investigations</th>
<th>Non-operative management of the duodenal stricture</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Diaphragm-like stricture at junction of D1/D1</td>
<td>NSAID (acetylsalicylic acid for more than 5 years)</td>
<td>Endoscopy &amp; Barium meal; tight stricture CT scan; no tumour mass</td>
<td>NSAID discontinued; High-dose PPI therapy; Attempted balloon dilatation → unsuccessful</td>
<td>Long stricture in D3/D4 (Figure 2) Repeated CT scans over 4 months: no tumour mass, marked proximal duodenal dilatation (Figure 3) Laparoscopy: no tumour mass; Hormonal assessment excluded a gastrinoma</td>
</tr>
<tr>
<td>2A</td>
<td>Long stricture in D3/D4</td>
<td>NSAID (ibuprofen for more than 2 years)</td>
<td>Endoscopy: superficial ulcers in antrum/D1; Barium meal; long stricture in D3/D4; CT scan: no tumour mass; Hormonal assessment excluded a gastrinoma</td>
<td>NSAID discontinued; A few months of high-dose PPI therapy → no benefit</td>
<td>Idiopathic</td>
</tr>
<tr>
<td>2B</td>
<td>Diaphragm-like stricture at junction of D1/D2</td>
<td>NSAID (ibuprofen for more than 2 years)</td>
<td>Endoscopy &amp; Barium meal; short diaphragm-like tight stricture at junction of D1/D2</td>
<td>Balloon dilatation was reamed by short-lived (2 weeks) symptomatic improvement</td>
<td>2-cm post-bulbar stricture in D1</td>
</tr>
<tr>
<td>3</td>
<td>Stenosis in D2</td>
<td>Acute necrotising biliary pancreatitis</td>
<td>Barium meal 16 weeks after onset of attack: hold up in D2 with a delayed partial emptying (Figure 1); Repeat CT: no pseudocyst or collections</td>
<td>Conservative treatment with intravenous nutrition for up to 3 months → duodenal stenosis failed to resolve either clinically or radiologically</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>4</td>
<td>Stenosis and rigidity in D2</td>
<td>Alcohol related calcific chronic pancreatitis</td>
<td>ERCP: tight and rigid duodenum; isotope studies: delayed gastric emptying</td>
<td>Previous balloon dilatation + prokinetics &amp; anti-emetics</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 4**

CT scan: small duct, calcific chronic pancreatitis; no tumour mass; MRCP: smooth 2-cm stricture in distal CBD; no detectable benefit; High-dose PPI therapy; Intravenous nutrition; Attempted balloon dilatation → failed to advance the balloon over the guide wire; Non-steroidal anti-inflammatory drugs; D1/D2: first and second parts of duodenum; D3/D4: third and fourth parts of duodenum; USS: abdominal ultrasonography; CT: computed tomography; ERCP: endoscopic retrograde cholangiopancreatography; MRCP: magnetic resonance cholangiopancreatography; CBD: common bile duct; PPI: proton pump inhibitors.
One of the patients (Patient No. 2) developed a further proximal diaphragm-like stricture at the junction between the first and second parts of duodenum two months after resection of a benign distal duodenal stricture. No tumour mass was shown on CT imaging of any of the patients. In addition to the investigations above, hormonal assessment in one of the patients with recurrent duodenal and gastro-enteric anastomotic stricture (Patient No. 2) ruled out a gastrinoma. The data available from these investigations, as well as from post-resection pathological evaluation (Patient No. 2), and from clinical, radiological and endoscopic follow up (Table 5) confirmed or supported their benign nature (Table 3).
Options for non-operative management of these strictures including prolonged intravenous feeding (in the patient with acute necrotising pancreatitis), high-dose PPI therapy, discontinuation of NSAID, and balloon dilatation as appropriate were explored and exhausted before embarking on surgery (Table 3).

**OPERATIVE PROCEDURES**

The six patients underwent seven laparoscopic operative procedures to treat their duodenal disease (Table 4). These included a gastroenterostomy that was fashioned as a loop (n=5) or in a Roux-en-Y configuration (n=1), and a pancreas-preserving distal duodenectomy[,] with a duodeno-jejunal anastomosis (n=1). In one of the patients (No. 4) who suffered with alcohol-related chronic pancreatitis, the laparoscopic loop gastroenterostomy was combined with a Roux-en-Y hepaticojejunostomy for a symptomatic benign distal bile duct stricture and a concomitant bilateral thoracoscopic splanchnotomy for an opiate-dependent intractable abdominal pain[].

**OPERATIVE TECHNIQUE**

All the laparoscopic gastroenterostomies were fashioned in an antecolic anterior arrangement. Every effort was made to ensure that the gastroenteric anastomosis was placed distally on the gastric/antral wall. The anastomoses were fashioned in a side-to-side manner using two or three firings of an endo-stapler (ATB45 45mm-articulating, Ethicon Endo-Surgery, Cincinnati, Ohio, USA), and the common gastro-jejunal side opening was closed with a continuous Vicryl 2-0 suture (Ethicon Inc., Somerville, New Jersey, USA). The Roux-en-Y entero-enteric anastomoses were fashioned in a similar manner. The Roux-en-Y hepaticojejunostomy was constructed in a retrocolic side-to-side manner (previous open cholecystectomy) to the common hepatic duct, and the anastomosis was fashioned with a single firing of an endo-stapler (TSW35 35mm, Ethicon Endo-Surgery, Cincinnati, Ohio, USA), and completed with interrupted absorbable
sutures (Vicryl 3-0).

The operative technique for the laparoscopic distal duodenectomy has been described previously [3].

RESULTS
OPERATION-RELATED OUTCOMES
The intraoperative course was uneventful in all cases and all procedures were completed laparoscopically. The operating time (median 160 minutes) ranged between 30 minutes for a loop gastroenterostomy to 355 minutes for the combined laparoscopic gastric and biliary bypass and bilateral thoracoscopic splanchnotomy (Table 4).

The recovery from surgery was uneventful in five of the seven procedures (Table 5). Intraoperative bleeding developed on the first postoperative day following the duodenectomy, and was managed by re-laparoscopy and peritoneal lavage successfully (no source of bleeding was identified). Central line sepsis presented on the second postoperative day in a patient who had a prolonged preoperative course and was fed parenterally.

The median duration of postoperative hospital stay (Table 5) was 4 days (range 3-19 days), and appeared to relate to the duration of preoperative hospital stay (Table 2). All patients were discharged on long-term PPI therapy.

AETIOLOGY OF DUODENAL DISEASE
Determination of the aetiology of the duodenal strictures was based on the preoperative data as well as the histological findings of operative specimens (Patients No. 2 and No. 6) and was supported by follow up information. The aetiology is shown in Table 3.

FOLLOW UP
The median duration of follow up was 9 months (range 2-32 months). One of the patients (Patient No. 2) developed a further NSAID-related diaphragm-like duodenal stricture at the junction of the first and second parts of duodenum 2 months after a distal duodenectomy and duodenonojejunal anastomosis [4] that was initially managed by endoscopic balloon dilatation with only a short-lived response. A month later she underwent a laparoscopic Roux-en-Y gastroenterostomy, which subsequently occluded (stricture) and was managed by laparoscopic revision gastroenterostomy with a successful outcome. At the time of writing, all patients remain symptom-free, but for occasional vomiting in one of the patients (Patient No. 5).

DISCUSSION
Patients presenting with benign duodenal strictures that fail to respond to medical or endoscopic therapy may be managed successfully with minimally invasive surgery without the need to resort to a laparotomy. Relief of the obstruction may be simply accomplished in the majority of patients with a laparoscopic gastroenterostomy. This procedure was employed by Nagy et al [30] in a patient with duodenal obstruction secondary to peptic ulcer disease and by Reissman et al [35] in three patients with duodenal Crohn's disease, and we have applied it successfully in five patients. However, laparoscopic resection of the diseased distal duodenum (with preservation of the pancreas) was necessary in one of our patients who also had clinical evidence of recurrent bleeding from the stricture [3]. In addition, uncertainty about the diagnosis of a 'benign' pathology in this good-risk 'young' patient supported a duodenal resection.

The role of laparoscopic surgery in the palliation of malignant duodenal and biliary obstruction is now well recognised, and is associated with significant reduction in hospital stay compared with open surgery [7,36,38]. In addition, laparoscopic surgery has its applications in the management of benign duodenal conditions in adults and children.

In adults, the laparoscopic approach to closure of perforated duodenal ulcers is well established [4]. In addition, laparoscopic transgastric or transduodenal resection of large duodenal villous adenomas [39], Brunner's gland hamartoma (Brunneroma) [3] or duodenal stormal tumour [40], resection of bleeding distal duodenal diverticulum [41] or a carcinoid tumour of the duodenal bulb [42], transgastric removal of an obstructing gallstone impacted in the duodenum (Bouveret's syndrome) [43], duodenotomy and under-running of actively bleeding duodenal ulcers [44], and duodenojejunostomy for the superior mesenteric artery syndrome [19] have all been described. In paediatric patients, laparoscopic duodenoduodenostomy for duodenal atresia [6] as well as laparoscopic division of congenital Ladd's bands that cause intermittent duodenal obstruction in neonates with malrotation of the gut [13] have been practiced.

It is essential to establish the diagnosis of a 'benign' disease and to explore non-surgical management options before embarking on a laparoscopic gastric bypass as a definitive treatment. Although the benign nature of the stricture may be readily evident as the case was in one of our patients who
developed the stricture a month into an acute attack of severe necrotising pancreatitis, the establishment of a benign aetiology often poses a diagnostic dilemma. Endoscopic evaluation with repeated biopsies, a barium study to examine the length, morphology and location of the stricture, and cross-sectional imaging to exclude a tumour mass are essential investigations. The detection of histologically benign, diarrhagm-like duodenal strictures in patients with a history of NSAID intake and absent tumour mass on computed tomography may suggest a NSAID-related disease \(^{(25)}\). Absence of a tumour mass on imaging in patients with an established history of chronic pancreatitis attests to the benign nature of the duodenal stenosis, although caution should be exercised in order to avoid missing a progression of disease to pancreatic cancer \(^{(25)}\). Other causes shown in Table 1 ought to be considered when a long distal duodenal stricture is detected.

In patients with benign duodenal strictures, it is worthwhile exploring the non-surgical treatment options in the first instance. Either medical treatment (PPI therapy) alone \(^{(17)}\) or in combination with endoscopic balloon dilatation \(^{(30)}\) may provide a long-lasting relief of gastric outlet obstruction secondary to peptic ulcer disease in some patients, though some 50 per cent of patients may still require further endoscopic or surgical intervention \(^{(30)}\). We employed the techniques of endoscopic balloon dilatation in four of the five patients and found its results disappointing. Duodenal obstruction secondary to Crohn's disease may respond to medical therapy \(^{(18)}\). Duodenal tuberculosis causing obstruction is a rare condition that is usually seen in the tropics; it is rarely diagnosed preoperatively by endoscopic biopsy and may then respond to drug therapy \(^{(19)}\).

Surgery for benign duodenal obstruction should therefore be reserved to patients in whom non-operative management options fail, and when the diagnosis remains in doubt. In the former scenario, relief of the obstruction may be accomplished by a laparoscopic gastroenterostomy; whilst in the latter a duodenal or pancreaticoduodenal resection is warranted in the good surgical-risk patient and may be safely accomplished laparoscopically.

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