Laparoscopic Management of Duodenal Obstruction Resulting From Superior Mesenteric Artery Syndrome

Brian J. Pottorf, MD; Farah A. Husain, MD; H. Whitton Hollis Jr, MD; Edward Lin, DO, MBA

Importance Duodenal obstruction by compression from the superior mesenteric artery (SMA) can be managed using minimally invasive techniques initially developed for bariatric patients requiring gastric bypass.

Observations This retrospective review evaluates 12 patients with SMA syndrome who were treated with laparoscopic enteric bypass. Technical considerations are presented in detail. The study group comprised 5 men and 7 women, with ages ranging from 21 to 65 years (mean, 36.8 years). Operative times ranged from 53 to 126 minutes (mean, 72.4 minutes). Mean length of hospital stay was 4.2 days (range, 3-7 days). Obstructive symptoms were improved or eliminated in 11 patients (92%). One patient required readmission for inadequate control of generalized abdominal pain. No patients in this series developed postoperative bowel obstruction, wound complications, or anastomotic leaks or died.

Conclusions and Relevance Laparoscopic duodenojejunostomy is safe and effective and should be considered the optimal treatment for patients presenting with duodenal obstruction from SMA syndrome. Advances in minimally invasive surgery have demonstrated the safety and low morbidity of laparoscopically created enteric anastomoses. The shorter hospital stay, low morbidity, and high success of laparoscopic enteric bypass make this approach favorable to traditional open techniques.

Methods A retrospective review of 12 patients with a diagnosis of SMA syndrome treated with laparoscopic enteric bypass was performed from February 18, 2008, through March 3, 2014. All patients had failed nonoperative management and were referred after imaging studies confirmed proximal duodenal dilation in association with a narrowed aortomesenteric angle consistent with SMA syndrome. No attempt was made to classify the concomitant predisposing factors. All patients were offered duodenal decompression (laparoscopic duodenojejunostomy) as an alternative to continued nonoperative management. Duration of symptoms was not included in this analysis as the intent of the review focused primarily on the safety and efficacy of this minimally invasive alternative. This study received institutional review board approval by waiver of consent from Exempla Saint Joseph Hospital and Emory University.

In this series, 11 of 12 patients (92%) underwent isolated laparoscopic duodenojejunostomy while 1 patient (8%) had an adjunct pyloroplasty performed for delayed gastric emptying. The operation used included (1) placement of 4 functional laparoscopic ports, (2) inframesocolic exposure of the SMA, (3) confirmation of enteric compression resulting in proximal duodenal dilation, (4) creation of a transverse mesocolic window, (5) identification of ligament of Treitz, (6) isolation of 20 to 40 cm of jejunum to bring through the mesenteric defect, (7) careful construction of a duodenojejunostomy with endoscopic staplers (Figure 1), and (8) closure of the common enterotomy with an endoscopic linear cutting stapler or running whipstitch of absorbable suture. An accompanying video detailing the laparoscopic steps of this operation is included (Video).
Results

The study group included 5 men (42%) and 7 women (58%). Ages ranged from 21 to 65 years (mean, 36.8 years). Operative times ranged from 53 to 126 minutes (mean, 72.4 minutes). Mean length of hospital stay was 4.2 days (range, 3-7 days). Obstructive symptoms were improved or eliminated in 11 patients (92%). The patient requiring pyloroplasty for delayed gastric emptying and obstruction had persistent symptoms that required completion gastrectomy. One patient (8%) was readmitted for inadequate control of generalized abdominal pain. No patients developed postoperative bowel obstruction. No wound infections, anastomotic complications, or deaths were reported in this series.

Discussion

Rokitansky reported the first case of duodenal obstruction from a narrowed aortomesenteric angle by the SMA in 1861. In 1921, Wilkie published a detailed anatomical and clinical article describing the pathophysiological ramifications of SMA syndrome, which at the time he termed chronic duodenal ileus. Six years later, he reported a case series of 75 patients with SMA syndrome, which led to the eponym Wilkie’s Syndrome. Other names for SMA syndrome have included arteriomesenteric duodenal mesenteric compression syndrome, chronic duodenal ileus, and cast syndrome.

The incidence of SMA syndrome has been estimated between 0.013% and 0.3%. The disease occurs more often in females than males, at a ratio of 3:2, and often affects younger individuals between the ages of 10 and 39 years. Superior mesenteric artery syndrome can occur at any age, however. There appears to be no ethnic predisposition as monozygotic twins who spontaneously developed the syndrome within 1 year of each other.

Common symptoms include a sense of fullness in the epigastrium, postprandial abdominal pain, early satiety, and intermittent emesis. All patients in this series experienced 1 or more of the aforementioned symptoms. Symptoms are commonly aggravated by both eating and lying supine. Recumbency exacerbates symptoms as the angle between the SMA and spine is further decreased. All alleviation of symptoms occurs when there is release of the aortomesenteric angle, such as when a patient is positioned prone, knee to chest, or in the left lateral decubitus position. The Hayes maneuver (applying pressure to the infraumbilical region and lifting cephalad and dorsal to open the aortomesenteric angle via elevation of the mesenteric root) temporarily relieves the duodenal compression.

Decreases in the aortomesenteric angle can be classified into 2 broad categories: congenital and acquired. Congenital compression is associated with duodenal malrotation, low SMA takeoff, Ladd bands, idiopathic peritoneal adhesions, shortened and thickened mesenteric root, and more cephalad position of the ligament of Treitz. Acquired causes include (1) postoperative obstructions following restorative proctocolectomy with ileoanal anastomosis, spinal surgery, esophagectomy, bariatric surgery, abdominal aortic aneurysm repair, SMA stenting, or Nissen fundoplication; (2) dietary disorders such as malabsorption syndromes, bulimia, or anorexia; (3) severe debilitating conditions such as malignant neoplasm, human immunodeficiency virus/AIDS, paralysis, or congestive heart failure; (4) trauma such as multiple-trauma patients confined to extended bed rest, burn patients, spinal cord injuries, or SMA aneurysm; and (5) local conditions such as mesenteric root neoplasm, abdominal aortic aneurysm or SMA aneurysm compression, or peptic ulcer disease with resultant duodenal thickening. Other conditions occasionally as-
Associated with duodenal compression include Crohn disease, Henoch-Schönlein purpura, hypothalamic germinoma, pancreatitis, cholecystitis, connective tissue disorders, and pregnancy causing exaggerated lumbar lordosis.1,7,16-19

Diagnosis centers on clinical symptoms combined with radiographic images confirming duodenal obstruction. A wide array of imaging modalities can be used. Radiography can reveal an enlarged gastric bubble and proximal duodenum.1-3,5,6 Upper gastrointestinal series with contrast can reveal the exact location of luminal impingement from the SMA as it crosses the duodenum, preventing normal contrast passage.5,5,6 Ultrasonography has been useful in measuring the aortomesenteric angle and distance.1,2 Computed tomography has now replaced angiography as the standard for diagnosis of SMA syndrome.3,4 In this series, 11 of 12 patients were diagnosed with SMA syndrome using a combination of upper gastrointestinal series and computed tomography. One young patient underwent upper gastrointestinal series with magnetic resonance imaging for diagnosis to avoid gonadal radiation.

Treatment for SMA syndrome is largely medical and includes fluid resuscitation, total parenteral nutrition, passage of a nasoenteric tube past the obstruction for enteric feedings, small meals, and positional eating.2,13-16 Hyperalimentation can be attempted to increase the SMA mesentery and retroperitoneal fat, which results in widening of the aortomesenteric angle and distance.1 If nonoperative efforts fail, surgical intervention is indicated.

Gastrojejunostomy is a feasible alternative as it alleviates gastric obstruction. However, it does not relieve the proximal duodenal obstruction that can perpetuate symptoms.1,3 The Strong procedure (division of the ligament of Treitz) allows caudal duodenal mobilization outside the aortomesenteric angle.1,3 Although appealing because bowel integrity is not compromised by an anastomosis, the drawback of the Strong procedure is a 25% failure rate resulting from tethering of the inferior pancreaticoduodenal artery.1,3-4 Transabdominal gastrojejunostomy dates to 1908 and remains the standard of therapy.1,5 Significant morbidity can occur with this procedure. Laparoscopic duodenojjunostomy is under investigation as an alternative to laparotomy. Although no prospective trials are available, the benefits of laparoscopic surgery compared with open surgery are already well described in the literature.1,6

It is possible that the 1 patient in this series who required postoperative gastrectomy was misdiagnosed. However, there is no physiological reason explaining why gastric dysmotility alone would cause duodenal dilation and a narrowed aortomesenteric angle. We considered excluding this patient from the series, but this approach would corrupt the data.

To our knowledge, this case series is the largest reported number of patients with SMA syndrome being treated with laparoscopic duodenojjunostomy. A review of the literature demonstrates similar experiences and outcomes (Table).2,4-6,20-31 This large series corroborates and confirms that laparoscopic duo-

### Table. Literature Review Compiling Details Regarding Treatment of SMA Syndrome With Laparoscopic Duodenojejunostomy

<table>
<thead>
<tr>
<th>Source</th>
<th>Patients in Series, No.</th>
<th>Time, Mean, min</th>
<th>Adjunctive Procedure</th>
<th>Length of Stay, Mean, d</th>
<th>Complications</th>
<th>Mortality</th>
<th>Outcome/Duration of Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gersin and Heniford,20 1998</td>
<td>1</td>
<td>Not reported</td>
<td>None</td>
<td>4.0</td>
<td>None</td>
<td>None</td>
<td>Not reported</td>
</tr>
<tr>
<td>Richardson and Surowiec,21 2001</td>
<td>1</td>
<td>113.5</td>
<td>LOA</td>
<td>3.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 6 mo</td>
</tr>
<tr>
<td>Bermas and Fenoglio,22 2003</td>
<td>2</td>
<td>Not reported</td>
<td>None</td>
<td>3.5</td>
<td>Trocar site bleeding</td>
<td>None</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>Kim et al,23 2003</td>
<td>2</td>
<td>172.5</td>
<td>LA</td>
<td>5.5</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 12 and 18 mo</td>
</tr>
<tr>
<td>Goltstein et al,24 2004</td>
<td>2</td>
<td>Not reported</td>
<td>None</td>
<td>Not reported</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 18 mo</td>
</tr>
<tr>
<td>Kingham et al,25 2004</td>
<td>1</td>
<td>120</td>
<td>None</td>
<td>5.0</td>
<td>None</td>
<td>None</td>
<td>Not reported</td>
</tr>
<tr>
<td>Agarwalla et al,36 2006</td>
<td>1</td>
<td>Not reported</td>
<td>LC</td>
<td>7.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 3 mo</td>
</tr>
<tr>
<td>Palanivelu et al,27 2006</td>
<td>1</td>
<td>110</td>
<td>None</td>
<td>5.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 6 mo</td>
</tr>
<tr>
<td>Jo et al,28 2006</td>
<td>1</td>
<td>50</td>
<td>None</td>
<td>5.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 8 mo</td>
</tr>
<tr>
<td>Makam et al,29 2006</td>
<td>1</td>
<td>180</td>
<td>FJ</td>
<td>5.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 6 mo</td>
</tr>
<tr>
<td>Fraser et al,4 2009</td>
<td>1</td>
<td>Not reported</td>
<td>None</td>
<td>3.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>Singaporewalla et al,30 2009</td>
<td>1</td>
<td>130</td>
<td>None</td>
<td>7.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 6 mo</td>
</tr>
<tr>
<td>Munene et al,31 2010</td>
<td>1</td>
<td>110</td>
<td>None</td>
<td>4.0</td>
<td>None</td>
<td>None</td>
<td>Not reported</td>
</tr>
<tr>
<td>Wyten et al,2 2010</td>
<td>3</td>
<td>141.6</td>
<td>LOA</td>
<td>4.3</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic (n = 2) at 30 and 60 mo; 1 persistent bloating</td>
</tr>
<tr>
<td>Magee et al,2 2011</td>
<td>1</td>
<td>Not reported</td>
<td>None</td>
<td>5.0</td>
<td>None</td>
<td>None</td>
<td>Asymptomatic at 3 mo</td>
</tr>
<tr>
<td>Present study</td>
<td>12</td>
<td>72.4</td>
<td>HMP</td>
<td>4.2</td>
<td>Readmission for pain control</td>
<td>None</td>
<td>Asymptomatic (n = 11); 1 delayed gastric emptying</td>
</tr>
</tbody>
</table>

Abbreviations: FJ, feeding jejunostomy; HMP, Heineke-Mikulicz pyloroplasty; LA, laparoscopic appendectomy; LC, laparoscopic cholecystectomy; LOA, lysis of adhesions; SMA, superior mesenteric artery.
denojejunostomy can be efficacious in the treatment of SMA syndrome while avoiding the morbidity of the traditional transabdominal approach.

Conclusions

Superior mesenteric artery syndrome is a rare cause of chronic duodenal obstruction. The diagnosis is clinical and confirmed by contrast studies demonstrating obstruction of the duodenum. Initial treatment should include nonoperative management. If this modality fails, then surgical intervention can be performed. Currently, advances in minimal-access bariatric surgery have demonstrated the safety and low morbidity of laparoscopically created enteric anastomoses. The shorter hospital stay, low morbidity, and high success of laparoscopic enteric bypass make this approach favorable to traditional open techniques. Laparoscopic enteric bypass should be considered a primary treatment modality for patients with SMA syndrome.

ARTICLE INFORMATION

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Study concept and design: All authors. 
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Drafting of the manuscript: All authors. 
Critical revision of the manuscript for important intellectual content: Pottorf, Husain, Hollis. 
Administrative, technical, or material support: Pottorf, Hollis, Lin.
Study supervision: Pottorf, Husain, Hollis.

Conflict of Interest Disclosures: None reported.

REFERENCES