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Laparoscopic Heller myotomy for achalasia

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Abstract

Background: Achalasia is an uncommon illness affecting 1 per 100,000 patients yearly. There is evidence to suggest viral, autoimmune, and hereditary etiologies. There are many treatment options available including medications, botulinum toxin injection, pneumatic dilation, and surgical myotomy.

Methods: We present a retrospective review of patients undergoing laparoscopic-modified Heller myotomy at a large referral and surgical training center.

Results: There were 36 patients identified. Thirty patients had undergone prior treatment with botulinum toxin injection, pneumatic dilation, previous Heller myotomy, or esophageal stenting. Immediate complications included mucosal perforation (2), spleen injury (1), and trocar-site infection (1). There were no postoperative esophageal leaks. Three patients suffered reflux requiring the daily use of a proton pump inhibitor 9 months after surgery. Three patients suffered recurrent dysphagia.

Conclusions: Presently, there are little data to suggest an ideal management strategy in patients with achalasia. Our patient population consists predominantly of failures of other treatment methods submitted for laparoscopic myotomy. Our data suggest that laparoscopic Heller myotomy can be safely undertaken in this population, without a higher than expected rate of recurrent symptoms or reflux.

Keywords Achalasia; Laparoscopic; Heller myotomy; Botulinum toxin injection; Pneumatic dilation

Achalasia is an uncommon but not exceedingly rare problem. It affects 1 per 100,000 patients yearly. Its presentation has been described at all ages but seems to peak between the third and fifth decades of life. The disease was first described by Thomas Willis in 1674. Dilation by insertion of a whale bone was the mainstay of treatment. The fundamental principle of disruption of the muscular layers of the lower esophagus that was accomplished with seventeenth century methods still exists today in the form of pneumatic dilation and surgical myotomy.

There is increasing understanding of the pathophysiology of this disorder. Myenteric plexus degeneration occurs as a common final pathway, although the mechanism by which nerve cell loss occurs is not well described. Evidence exists to suggest viral [1], autoimmune [2], and hereditary etiologies [3]. There seems to be sparing, at least partially, of postganglionic cholinergic excitatory fibers and seemingly selective involvement of inhibitory nerves suggested by the loss of vasoactive intestinal peptide and nitric oxide synthase [4]. The functional impairment of the gastroesophageal junction in achalasia can be explained by these findings. Unopposed excitatory input leads to impaired lower esophageal relaxation, and loss of inhibitory fibers leads to aperistalsis.

Diagnosis

Late presentations of the disorder show classic symptoms and findings. These include dysphagia for solids and often liquids as well. There is frequently regurgitation of partially digested and undigested food and frequently halitosis. Chest pain is common with swallowing, as are nausea and heartburn. The patient who presents earlier in the course of the illness can have much more mild or vague symptoms, sometimes presenting with mild epigastric discomfort or dyspepsia that can be confused with gastritis or biliary colic.

Classic chest x-ray findings show an air fluid level in the mediastinum as a result of pooled secretions and food in the

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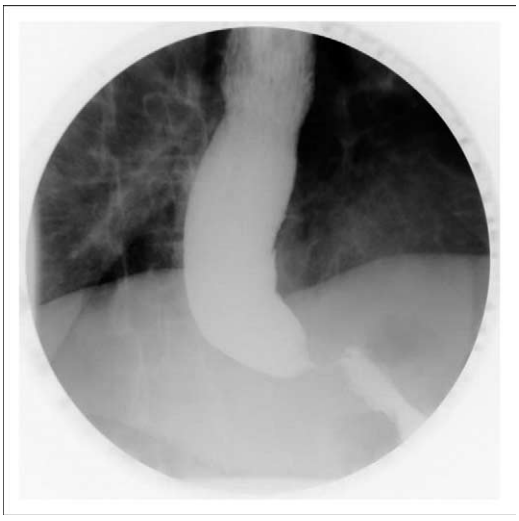

Fig. 1. Barium esophagram showing the dilated proximal esophagus and the distal tapering of the "bird's beak."

esophagus. Upper gastrointestinal contrast studies generally will show a smooth tapering of the distal esophagus frequently referred to as the "bird's beak." There is often proximal dilation of the esophagus, and in later stages the esophagus can assume a sigmoid appearance (Fig. 1). Occasionally, an epiphrenic diverticulum appears evident on contrast imaging. Recently, the timed barium swallow has gained popularity as a reliable method for following patients before and after therapy [5]. In the timed technique, the patient drinks 100 to 200 mL of a 45% barium sulfate solution. Upright anterior to posterior static images of the esophagus are obtained at times 0, 1, 2, and 5 minutes. The height and width of the barium column in the esophagus are measured.

Upper endoscopy is useful to rule out the presence of pseudoachalasia from an unrecognized distal esophageal tumor. Often the test is unremarkable or can reveal nonspecific findings such as distal esophagitis. In more advanced cases, esophageal dilation and tortuosity are evident. Sometimes pooled fluid and food is noted in the esophagus. The distal esophagus is visibly narrow, but with gentle insufflation and pressure, the endoscope can usually be passed beyond the lower esophageal sphincter and into the stomach.

The most helpful test in securing the diagnosis is esophageal manometry. Hypertension at the lower esophageal sphincter is common, often 30 to 50 mmHg. Some patients do not have marked elevations of the lower esophageal sphincter resting pressure, but to diagnose achalasia they should have a failure of appropriate relaxation of the lower esophageal sphincter. Diminished or absent peristalsis is also evident. The subset of patients with vigorous achalasia is characterized by the findings of high distal esophageal contraction amplitudes and the presence of tertiary contractions on manometry. Esophageal pH studies are often obtained over the course of evaluation in many of these patients but generally add little.

Surgical Technique

The patient is in the supine position. The operating surgeon stands on the patient's right and the assistant on the left. We use 2 monitors, positioned to either shoulder of the patient. The 4 operating ports are positioned on a horizontal line midway between the xyphoid and umbilicus. We use the Nathanson liver retractor (Cook Surgical, Bloomington, IN) inserted through a 5-mm subxyphoid incision, which is then in turn rigidly mounted to the operating table.

The gastroesophageal junction is completely dissected in the manner of a hiatus hernia repair. The diaphragmatic crura are identified. The esophagus is circumferentially dissected. The uppermost short gastric vessels are divided. The connective tissues within the mediastinum are carefully separated to expose the esophagus for several centimeters proximal to the narrowed lower esophageal sphincter. During most cases, a surgical resident or fellow is available to perform intraoperative flexible endoscopy. The transillumination afforded by the intraluminal endoscope aids in locating the necessary proximal extent of the esophageal dissection.

The myotomy is performed with laparoscopic scissors on the anterior surface of the esophagus without the use of any energy-delivering devices (Fig. 2). Most bleeding points along the edges of the myotomy are easily controlled by gentle grasping pressure. The intraluminal insufflation and transilluminaton is useful in identifying any missed muscle fibers. Furthermore, insufflation of the distal esophagus while submerging the region under saline helps to confirm the integrity of the mucosa.

The diaphragmatic crura are approximated posterior to

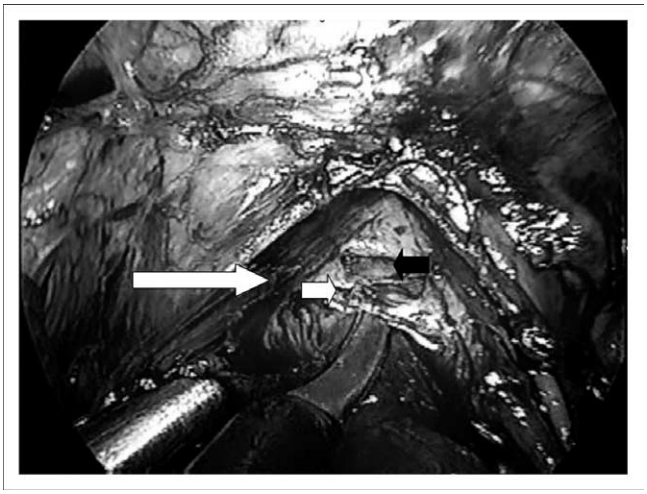

Fig. 2. Intraoperative view of the performance of the myotomy. Note the external longitudinal muscle fibers (large white arrow), internal circular muscle fibers (small white arrow), and mucosa (black arrow).