Short Esophagus

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KEYWORDS

• Esophagus • Short esophagus • Gastroesophageal reflux disease • Hiatal hernia

KEY POINTS

- In the presence of long-standing and severe gastroesophageal reflux disease, patients can develop various complications, including a shortened esophagus.
- Standard preoperative testing in these patients should include endoscopy, esophagography, and manometry, whereas the objective diagnosis of a short esophagus must be made intraoperatively following adequate mediastinal mobilization.
- If left untreated, it is a contributing factor to the high recurrence rate following fundoplications or repair of large hiatal hernias.
- A laparoscopic Collis gastroplasty combined with an antireflux procedure offers safe and effective therapy.

INTRODUCTION

Hiatal hernias, paraesophageal hernias, and gastroesophageal reflux disease (GERD) are common problems.[1–4] Complications related to these disorders can occur in up to 40% of patients.[5] One of the most controversial complications is the acquired short esophagus. Although numerous studies have been published describing the short esophagus and its pathophysiology, diagnosis, and treatment,[6–10] various reputable investigators have challenged its existence.[11–14] Consequently, this dichotomy contributes to the confusion underlying this entity. This article considers the history, pathophysiology, diagnosis, and current treatment options for the shortened esophagus.

HISTORY

Some investigators have credited Dietlen and Knierim[15] with the first mention of a short esophagus in their description of a pregnant patient whose stomach was found to be intrathoracic on radiograph. However, others have attributed this description to

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Akerlund[16] or Findlay.[17,18] A shortened esophagus shown on esophagram was first reported by Fineman and Conner[19] in 1924,[18] whereas Woodburn Morison[20] provided the earliest endoscopic description in 1930.[18]

The grave operative challenges presented by a shortened esophagus was first shown in Plenk's description of a case of perforation and peritonitis in which the stomach could not be replaced into the abdomen, leading to a fatal situation.[18,21,22] This portrayal is consistent with the modern understanding of shortened esophagus in which esophageal length is insufficient to allow the esophagogastric junction to rest below the diaphragm, as first described by Barrett in 1950.[18,23,24] From this, the theory that chronic reflux disease was the underlying cause for acquired short esophagus was first proposed by Lortat-Jacob[25] a few years later.

INCIDENCE

Depending on the study, the incidence of shortened esophagus can vary widely. It has been reported to be as high as 60%[26] and as low as 0% in older open[11] and more recent laparoscopic series.[27] If the intraoperative requirement for extensive mediastinal dissection or the performance of a Collis gastroplasty is used to define the presence of shortened esophagus then the incidence in the open literature is between 8% and 10%.[28,29] Using the same definition, a wider range (7%–19%) has been documented in various laparoscopic series.[30–33] If the need for a Collis gastroplasty is used as the most restrictive definition, then retrospective review of the literature indicates that between 1% and 5% of patients undergoing GERD-related surgery meet criteria.[30,34–36]

PATHOPHYSIOLOGY

The most common entity associated with acquired short esophagus is chronic GERD, which results in a persistent inflammatory response.[7] In the setting of a dysfunctional lower esophageal sphincter the esophageal mucosa is exposed to either acidic or alkaline reflux and the normal esophageal squamous epithelium provides an inadequate barrier to the damaging effects of refluxed gastric juices. Animal models,[37,38] as well as in-vivo human[10] studies, have shown that refluxed gastric fluid leads to deeper penetration of hydrogen ions into the wall of the esophagus with localized cellular damage. The resulting inflammation causes tissue edema, migration of inflammatory cells into the damaged tissue, attempted healing, and ultimately tissue fibrosis. With repeated exposure over a period of time, the inflammatory process can extend transmurally. Transmural inflammation then leads to longitudinal contracture of the transmural scar and the clinical manifestation of esophageal shortening.[7]

PREOPERATIVE ASSESSMENT

Typical studies used in the preoperative evaluation of patients with GERD include dynamic esophagram, esophagogastroduodenoscopy, and esophageal manometry. Many prior attempts at identifying preoperative indicators of short esophagus have identified factors with low specificity.[31,39–41] Some investigators contend that evidence of a nonreducing type I hiatal hernia greater than 5 cm in length or a type III hiatal hernia on barium esophagram is predictive of short esophagus being present at surgery.[42] However, when a retrospective, blinded review of patients who had undergone Collis gastroplasty was performed, barium esophagram had a positive predictive value of only 50%.[7]