Hiatal and Paraesophageal Hernias

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Anatomically, one of the primary defense mechanisms to prevent gastroesophageal reflux (GER) is an intact gastroesophageal junction (GEJ) that is composed of an overlapping lower esophageal sphincter and diaphragmatic crus (Figure 1). This barrier, if disrupted, can lead to increased GER and symptoms of gastroesophageal reflux disease (GERD) including heartburn, chest pain, regurgitation, and extraesophageal symptoms of cough or throat soreness. Prolonged or frequent GER can lead to complications including erosive esophagitis, Barrett’s esophagus, or stricture formation. The anatomic alignment of the lower esophageal sphincter and diaphragmatic crus is believed to contribute to reflux prevention primarily during dynamic situations such as post-deglutition or at times of increased intra-abdominal pressure.¹ The principal mechanism behind the development of a diaphragmatic hernia, either hiatal or paraesophageal, is a lax diaphragmatic hiatus and phrenoesophageal membrane. These defects allow for migration of the stomach cranially either in an anatomic (hiatal) or non-anatomic (paraesophageal) fashion and lead to disruption of the typical GER barrier. Hiatal hernias account for the majority of diaphragmatic hernias, whereas paraesophageal hernias account for approximately 5%–10%.² The overall U.S. prevalence varies widely from 14% to 84%, depending on the detection methods.²

Diaphragmatic hernias are classified as hiatal (type I or “sliding-type”), where the GEJ is displaced cranially above the diaphragmatic hiatus with a portion of the gastric cardia, or as paraesophageal (types II–IV), where defects in the phrenoesophageal membrane allow for migration of the stomach or other structures adjacent to the GEJ (Figure 2). In a type II hernia, the GEJ remains at the level of the diaphragm, but a portion of the gastric fundus migrates through the phrenoesophageal membrane into the mediastinum. Type III paraesophageal hernias, often referred to as “mixed-type”, have elements of both type I and type II where the GEJ is displaced cranially, but there also is a defect in the phrenoesophageal membrane with accompanying fundic displacement adjacent to the lower esophageal sphincter. A type IV paraesophageal hernia is diagnosed when non-gastric structures herniate through the phrenoesophageal membrane, including the spleen, colon, small bowel, or pancreas.

Clinically, hiatal hernias are often asymptomatic and are frequently encountered on routine endoscopy. In this instance, no clinical action other than noting presence of hernia is indicated. However, the hiatal hernia may lead to symptoms of GERD, including heartburn, regurgitation, and dysphagia. Hiatal hernias disrupt the normal GEJ reflux barrier, leading to delayed esophageal clearance and increases in acid exposure times³ and persistently abnormal pH/impedance monitoring despite proton pump inhibitor (PPI) therapy.¹ This is especially true in those whose heartburn may be resolved on PPI therapy but continue to have regurgitation as a sign of mechanical barrier dysfunction. Paraesophageal hernias may be asymptomatic but often present with symptoms of postprandial fullness, nausea, dysphagia, epigastric pain, and symptoms of GER. Iron deficiency anemia, caused by Cameron lesions or superficial linear ulcers related to sheer forces from the fundus moving in and out through the diaphragmatic hiatus, is a well-known presentation for paraesophageal hernias. Less well-known complications include exertional dyspnea from reduction of thoracic volume or compression on the left atrium.⁵ An ominous complication of paraesophageal hernias is its potential to become strangulated and lead to volvulus, ischemia, and potentially gangrene. This is an important clinical consideration in those with large hernias for whom surgical correction may be recommended to reduce the likelihood of this complication. The presence of a hernia should be sought if the appropriate clinical symptoms are present, especially if typical medical therapy for GERD does not resolve the presenting symptoms. A diagnosis can be made with numerous modalities, including upper endoscopy, contrasted fluoroscopy, computed tomography, and esophageal manometry. Each method has advantages and disadvantages. Barium esophagography has the advantage of providing bolus transit and anatomic information of the hernia, whereas endoscopy can better assess for mucosal changes including esophagitis, Barrett’s esophagus, and Cameron lesions. Computed tomography is useful in the assessment of type IV paraesophageal hernias.
hernias and should be obtained in suspected cases of volvulus or obstruction. Esophageal manometry provides a real-time assessment of the anatomic relationship between the esophagogastric junction and the diaphragmatic hiatus and can precisely characterize this relationship throughout the dynamic swallow. The clinical usefulness of this characterization continues to be investigated; however, if surgical intervention is considered, manometry should be performed in all patients to assess esophageal motility pattern.

The fundamental treatment of hiatal hernias with GER symptoms is acid suppression therapy with PPIs. PPIs are effective at controlling GER symptoms of heartburn and chest pain; however, PPIs are less effective for symptoms of regurgitation, especially when provoked by bending/stooping over or with increases in intra-abdominal pressure. Baclofen, a GABA-B receptor agonist and inhibitor of transient lower esophageal relaxations, is shown to be effective in patients with GERD and hiatal hernias; however, common side effects preclude its widespread utility. Surgical repair of a type I hernia in the absence of reflux disease is not necessary, and before any consideration for repair, an ambulatory pH assessment should be performed with either catheter-based or wireless modalities to document pathologic GER. If surgical repair is undertaken, an anti-reflux operation (fundoplication) should be offered by the performing surgeon because dissection of the phrenoesophageal membrane during hernia repair allows for hernia recurrence. For paraesophageal hernias, surgical repair has been historically advocated to potentially prevent the acute, emergent complications of volvulus and obstruction. As surgical technique has transitioned from open to laparoscopic approaches and the morbidity and mortality associated with emergent operations have decreased, routine elective repair of completely asymptomatic paraesophageal hernia is no longer the standard. A watchful waiting approach for asymptomatic patients is more likely to result in greater health outcomes compared with elective repair per 2 separate microsimulation models. Surgical repair is recommended for type IV hernias and all symptomatic type II or III paraesophageal hernias, including persistent iron deficiency anemia or pulmonary symptoms that are likely underappreciated. Recurrent hiatal hernia after surgical repair is common, occurring in up to 48% of patients with a large (>5 cm) initial hernia size. Hernias <5 cm at initial presentation were
associated with less than 25% chance of recurrence at 3 years. The majority of hiatal hernia recurrences are asymptomatic and found incidentally; however, recurrent symptoms can require repeat operation, depending on their severity.

Although the presence of a hiatal hernia predisposes patients to GERD, it can also result in delayed acid clearance and thus complicated reflux disease in the form of persistent GER symptoms, stricture formation, and Barrett’s esophagus. Treatment should be focused on symptom management with PPIs, and if symptoms do not respond to typical therapy, especially if regurgitation or dysphagia is the predominant presentation, surgical repair should be considered. Paraesophageal hernias are less common but can also cause symptoms and have the potential for volvulus or obstruction. Symptomatic type II and III hernias unresponsive to medical therapy should be repaired surgically as well as all type IV hernias. A watchful waiting approach can be considered for asymptomatic paraesophageal hernias. Recurrence of hiatal hernias is common after surgical repair, occurring in up to 50%, depending on the initial hernia size; thus,
the choice for surgical intervention must be approached cautiously, with special attention to ensure likelihood of surgical success for the refractory symptoms.

References

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Conflicts of interest
The authors disclose no conflicts.
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1. After surgical repair, hiatal hernia recurrence (with or without symptoms) occurs in:
   a. <10%
   b. 15%
   c. 65%
   d. 50%

**True or False**

2. The term paraesophageal hernia implies caudal displacement of a portion of the stomach adjacent to the gastroesophageal junction

3. Baclofen therapy may help in patients with hiatal hernia and persistent regurgitation despite PPI therapy who do not wish or cannot undergo surgical correction

4. Paraesophageal hernias may present as iron deficiency anemia

5. Paraesophageal hernias are always associated with sliding hiatal hernias

6. A type IV paraesophageal hernia includes herniation of non-gastric structures through the diaphragmatic defect

7. An asymptomatic patient who is found to have a type 2 paraesophageal hernia should undergo surgical repair ASAP.