# Paraesophageal Hernia

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**KEYWORDS**

• Hiatal • Paraesophageal • Nissen fundoplication • Hernia • Laparoscopic

**KEY POINTS**

- A paraesophageal hernia is a common diagnosis with surgery as the mainstay of treatment.
- Accurate arrangement of ports for triangulation of the working space is important.
- The key steps in paraesophageal hernia repair are reduction of the hernia sac, complete dissection of both crura and the gastroesophageal junction, reapproximation of the hiatus, and esophageal lengthening to achieve at least 3 cm of intra-abdominal esophagus.
- On-lay mesh with tension-free reapproximation of the hiatus.
- Anti-reflux procedure is appropriate to restore lower esophageal sphincter (LES) competency.

## INTRODUCTION

Hiatal hernias were first described by Henry Ingersoll Bowditch in Boston in 1853 and then further classified into 3 types by the Swedish radiologist, Ake Akerlund, in 1926.[1,2] In general, a hiatal hernia is characterized by enlargement of the space between the diaphragmatic crura, allowing the stomach and other abdominal viscera to protrude into the mediastinum. The cause of hiatal defects is related to increased intra-abdominal pressure causing a transdiaphragmatic pressure gradient between the thoracic and abdominal cavities at the gastroesophageal junction (GEJ).[3] This pressure gradient results in weakening of the phrenoesophageal membrane and widening of the diaphragmatic hiatus aperture. Conditions that are associated with increased intra-abdominal pressure are those linked with all abdominal wall hernias, including obesity, pregnancy, chronic constipation, and chronic obstructive pulmonary disease with chronic cough. There has even been a potential genetic component discovered in the development of hiatal hernias. Specific familial clusters across generations have been identified, indicating a possible autosomal dominant mode of

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**Abbreviations**

| | |
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| EGD | Esophagogastroduodenoscopy |
| GEJ | Gastroesophageal junction |
| GERD | Gastroesophageal reflux disease |
| GI | Gastrointestinal |
| LES | Lower esophageal sphincter |
| LUQ | Left upper quadrant |
| PEH | Paraesophageal hernia |

inheritance. Certain evidence has linked a collagen-encoding COL3A1 gene and an altered collagen-remodeling mechanism in the formation of hiatal hernias.[4] This link indicates that there may be both genetic and acquired factors that contribute to the development of hiatal hernias.

Although hiatal hernias were originally only classified into 3 types, the current classification scheme defines 4 types of hiatal or paraesophageal hernias (PEHs).[1] These types are listed next:

Type 1: Sliding hernia, the GEJ migrates into the thorax
Type 2: True PEH or rolling hernia, the herniation of the gastric fundus through a weakness in the phrenoesophageal membrane, the GEJ remains in the normal anatomic location
Type 3: Combination of types 1 and 2, the herniation of the GEJ and stomach into the chest (occasionally they can be larger and are sometimes termed *giant PEHs*)
Type 4: Includes other intra-abdominal viscera, such as colon, small bowel, omentum, or spleen along with the stomach migrating into the chest

Type 1, or sliding hiatal hernias, are the most common type and account for approximately 95% of hiatal hernias. The other 3 types combine to make up the remaining 5% of hiatal hernias.[1] All can be approached with similar preoperative and operative strategies when patients are symptomatic.

## CLINICAL PRESENTATION

Although the true prevalence of these hernias is difficult to determine because they are often asymptomatic or poorly defined, more recent epidemiologic studies have shown them to be more common than previously recognized in the Western population.[4] Typical patients are female and elderly, more commonly in or beyond their sixth decade of life. They may present with vague symptoms of intermittent epigastric pain and postprandial fullness. Sliding hiatal hernias are most commonly associated with gastroesophageal reflux disease (GERD). Large hiatal defects tend to present with symptoms of progressive intolerance to solids/liquids with regurgitation, nausea, and vomiting. These defects can also present with symptoms related to the space they occupy, such as chest pain and respiratory problems caused by lung compression or aspiration. These respiratory issues may include shortness of breath, asthma, and bronchitis. Other unpredictable symptoms that may be revealed with a thorough history include hoarseness, cough, laryngitis, and pharyngitis.[4] An unusual cause of gastrointestinal (GI) bleeding and iron deficiency anemia is Cameron lesions related to hiatal hernias. These lesions are linear gastric ulcers or erosions located on the gastric mucosal folds at the diaphragmatic impression of large hiatal hernias.[5] Cameron lesions are prevalent in 5% of patients with a hiatal hernia discovered on upper endoscopy, and the risk of one existing increases with hernia size.[5] More acute complications of PEHs are mechanical problems, such as gastric obstruction,