Classification and Evaluation of Diaphragmatic Hernias

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Introduction

Patients with diaphragmatic hernias are referred to surgeons to determine the necessity for intervention. Often these hernias are incidental findings from diagnostic evaluations for reflux or ulcer disease, but they can also present in dire need of urgent surgery. Understanding the evaluation and treatment of diaphragmatic hernias is essential to determining the appropriate treatment. It is important to know the classification of the different types of diaphragmatic hernias and how to adequately evaluate them so that patients get proper treatment with the appropriate urgency. The large majority of these cases are not emergencies, and it is imperative that the patient gets a complete workup prior to surgical intervention to prevent debilitating postoperative complications. Through a discussion of the anatomy of the diaphragm and the esophageal hiatus, the pathophysiology of these hernias is more clear. In addition, the different types of hernias, their classification, and their appropriate evaluation will be discussed.

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Anatomic Considerations

Embryology

The diaphragm develops between the 3rd and 10th weeks of gestation and is derived from four precursors: (1) the septum transversum, (2) two pleuroperitoneal folds, (3) cervical myotomes, and (4) the dorsal mesentery [1] (Fig. 18.1). The septum tranversum forms the central tendon, and the two pleuroperitoneal folds grow medially to join this tendon. The posteromedial portion is formed from the dorsal mesentery which contains the aorta, inferior vena cava, and esophagus. The crura are created from migration of myoblasts to this dorsal mesentery. The third, fourth, and fifth cervical myotomes provide the diaphragmatic musculature [2]. Congenital defects occur with failure of the pleuroperitoneal folds to develop, as there is no scaffolding for the musculature to develop upon [1].

Diaphragm

The diaphragm has three muscle groups: sternal, costal, and lumbar, which all join at the central tendon and create a dome-shaped membrane separating the thoracic and abdominal cavities [2]. There are three distinct foramina: aortic, esophageal, and caval. The aortic hiatus at T12 is the most posterior and contains the aorta, the thoracic duct, and azygous veins. It is bordered posteriorly by the vertebral bodies, anteriorly by the median arcuate ligament, and laterally by the crural origins.

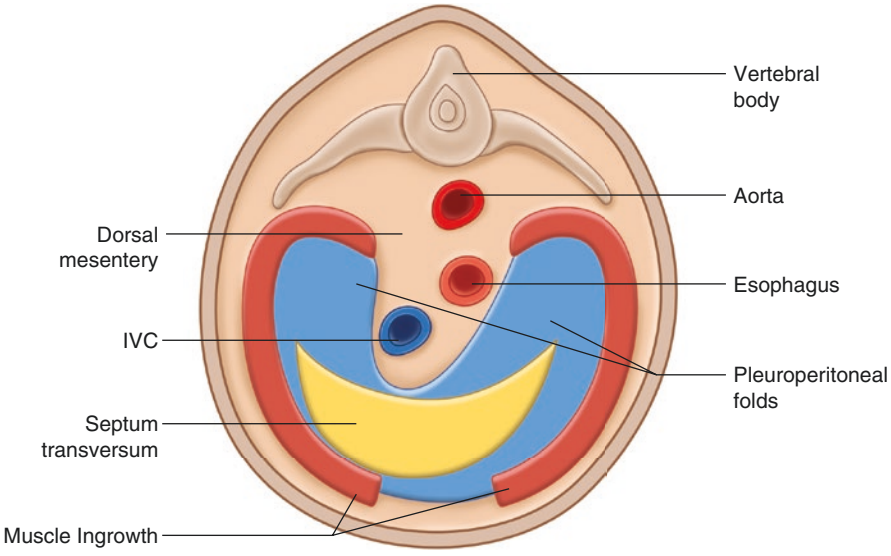

Fig. 18.1 Embryology of the diaphragm

At T10, the esophageal hiatus is the only foramen surrounded completely by muscle. The caval opening is the most anterior lying between T8 and T9 [1] and is completely surrounded by the central tendon of the diaphragm.

Blood is supplied by the right and left phrenic arteries, the intercostal arteries, and musculophrenic branches of the internal thoracic arteries [1]. Muscular and sensory innervation is provided by the left and right phrenic nerves which arise from the C3, C4, and C5 rami [2].

Hiatal Anatomy

The majority of diaphragmatic hernias occur through the esophageal hiatus, therefore it is essential to understand this anatomy (Fig. 18.2). The right and left diaphragmatic crus are muscular fibers that arise from the anterior longitudinal ligaments and are anchored at the lumbar vertebrae. As the right crus emerges from the anterior longitudinal ligament, it splits into two arms, one coursing medially and wrapping posterior to the esophagus and the other wrapping anteriorly. These arms decussate anterior to the esophagus where, along with the left crus, they attach at the central tendon of the diaphragm [3].

The esophagus is anchored to the crus by the phrenoesophageal membrane, which is formed from fused endothoracic and endoabdominal fascias. Additional posterior support is provided by the vagus nerves and radicles of the left gastric artery and vein. The phrenoesophageal membrane is attached circumferentially on the esophagus at the squamocolumnar junction. An intact membrane prevents herniation through the hiatus [4].

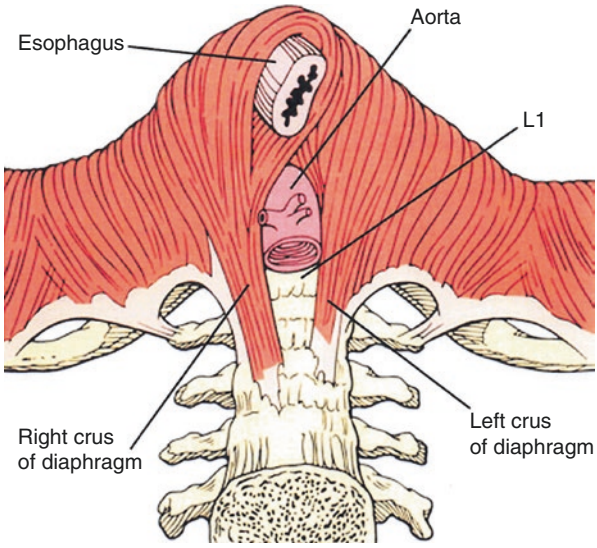

Fig. 18.2 Anatomy of the hiatus. (From Kahrilas et al. [4])

Esophagogastric Junction

The esophagogastric junction can be difficult to clinically define because of its intrinsic mobility (Fig. 18.3). Defining a true hiatal hernia depends on the relative positioning of the esophagus, the stomach, and the hiatus which is constantly variable [3]. Esophageal shortening occurs with contraction of the longitudinal muscles which elevates the distal esophagus [5]. With lifting of the distal esophagus, the esophagogastric junction is elevated above the hiatus, and there is physiological herniation [6]. It is known that this normal herniation occurs during primary and secondary peristalsis, esophageal distension, and transient relaxations of the lower esophageal sphincter [7]. Once the inciting stimulus for physiologic herniation ceases, the intrinsic elastic recoil of the phrenoesophageal membrane returns the

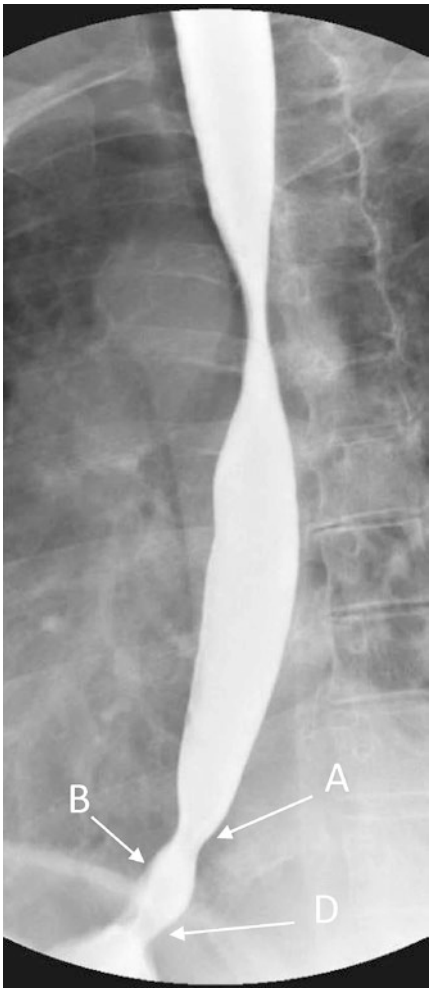

Fig. 18.3 Normal barium swallow. (A) "A" ring, (B) "B" ring or Z-line, and (D) diaphragmatic impression

esophagogastric junction to the abdomen [3]. This variability in relative anatomy at the hiatus creates unique challenges in the diagnosis and treatment of hiatal hernias. It is helpful to localize the esophagogastric junction with endoscopy although it can be difficult to get an accurate picture of its location relative to other relevant structures.

Pathophysiology

Risk Factors

Obesity causes gradual increases in intra-abdominal pressure which creates forces that encourage hiatal herniation [8]. Body mass index (BMI) has a direct relationship with increasing risk of hiatal hernia [9]. Clinically, many surgeons hesitate to offer hiatal hernia repair for patients with elevated body mass index because of increased risk of re-herniation. There is some controversy as to whether obesity increases the likelihood of recurrence after surgery at the hiatus. In a study comparing antireflux surgery outcomes in obese patients (BMI > 30), overweight patients (BMI 25–29.9), and nonobese (BMI < 25), obese patients had significantly higher recurrence rates (31%) versus the overweight (8%) and the nonobese (4.5%) [10]. More recent studies make the case that obesity has no effect on outcomes after antireflux surgery with or without concurrent hiatal hernia repair. Winslow et al. [11] showed that although surgery in the obese population is more difficult with significantly greater operative times, there was no difference in recurrence rates, symptoms, and patient satisfaction. In a prospective analysis of both clinical and objective outcomes, there were no differences in quality of life measures and recurrence rates between the obese and nonobese groups [12]. This controversy supports a frank discussion between obese patients and physicians as to the ideal timing for repair.

Age is also associated with increased risk for hiatal herniation. As the elasticity and recoil of the phrenoesophageal membrane decrease with age, the risk of herniation increases [9]. This is discussed in more detail in the pathophysiology section.

Previous surgery at the hiatus is another known risk factor for hiatal herniation. In a prospective study, radiologic recurrence after hiatal hernia repair has been reported as 57% (median follow-up = 58 months), although the majority of these were not clinically relevant and only 3% required reoperation [13]. Another study showed a radiologic recurrence rate of 27% at 1 year follow-up with no clinically relevant recurrences and no identifiable risk factors for recurrence [14]. It is assumed that each subsequent repair would have an increased likelihood of recurrence.

Other known risk factors include thoracoabdominal trauma most commonly from motor vehicle collisions [15]. Skeletal deformities such as scoliosis that change the anatomy of the diaphragm also increase risk [16]. Finally, congenital deformities are the most common cause of diaphragmatic hernia in children [17].

Causal Theories

As was well described in a review article by Weber et al., there are three theories of causation of hiatal hernia: (1) increased intra-abdominal pressure forcing the gastroesophageal junction into the chest, (2) displacement of the gastroesophageal junction into the chest due to esophageal shortening from fibrosis or excess vagal stimulation, and (3) gastroesophageal junction migration due to enlargement of the hiatus from congenital defects or acquired molecular/cellular changes. Through their review, they conclude that none of these theories are definitive and that causation is likely multifactorial [18].

Increased Intra-Abdominal Pressure

As previously discussed, obesity is a known risk factor for development of hiatal hernia which supports this causal theory. It has been shown that with increases of BMI of one point, intragastric pressure increases by 0.3 mmHg and intraesophageal pressure rose by 0.17 mmHg; waist circumference increases of 1 cm increased intragastric and intraesophageal pressures by 0.16 mmHg and 0.1 mmHg, respectively [6]. These gradients would theoretically transfer forces leading to hiatal herniation.

Esophageal Shortening/Vagal Stimulation

With contraction of the longitudinal muscles of the esophagus, physiological herniation is proposed to occur during swallowing [6]. It has been shown that with inhibitory vagal innervation anterior to the stomach cardia, this physiological herniation is not allowed to occur. Therefore, it is theorized that damage to the vagal nerve at the esophagogastric junction can cause either a decrease in this inhibitory function or an increase in stimulation of the longitudinal muscles resulting in chronic herniation over time [19].

Hiatal Enlargement

Through a combination of changes in the molecular makeup of the tissues that create the hiatus, progressive weakening can lead to physical weakening that can allow herniation. On analysis of the phrenoesophageal, gastrohepatic, and gastrophrenic ligaments at the time of fundoplication for gastroesophageal reflux disease, those patients with concurrent hiatal hernia were found to have 50% less elastin than those without hiatal hernias [20]. Impairments in collagen have been shown in both inguinal and incisional hernias, which would beg the question as to collagen involvement in hiatal hernia [21, 22]. Although this question is yet to be answered, it is not unreasonable to theorize that collagen also plays a role at the hiatus. Crural muscle fibers also seem to be involved in weakening of the hiatus. At the microscopic level, when comparing crural muscle in patients with and without hiatal hernia, there appears to be degradation of the myofibrils and degeneration of the muscular architecture in the patients with hiatal hernia [23]. Therefore, hiatal enlargement through a combination of tissue factors at the supportive ligaments and impairments of crural muscle is highly supported.

Types of Diaphragmatic Hernias

Hiatal Hernias

Hiatal hernias are categorized into sliding or paraesophageal hernias with four recognized types (Fig. 18.4).

Type I Hiatal Hernia

Type I hiatal hernia is commonly described as a sliding hiatal hernia and occurs when the esophageal hiatus is dilated enough to allow herniation of the gastric cardia and bringing the gastroesophageal junction above the diaphragm. While they are the most common of the diaphragmatic hernias, they are also the most difficult to

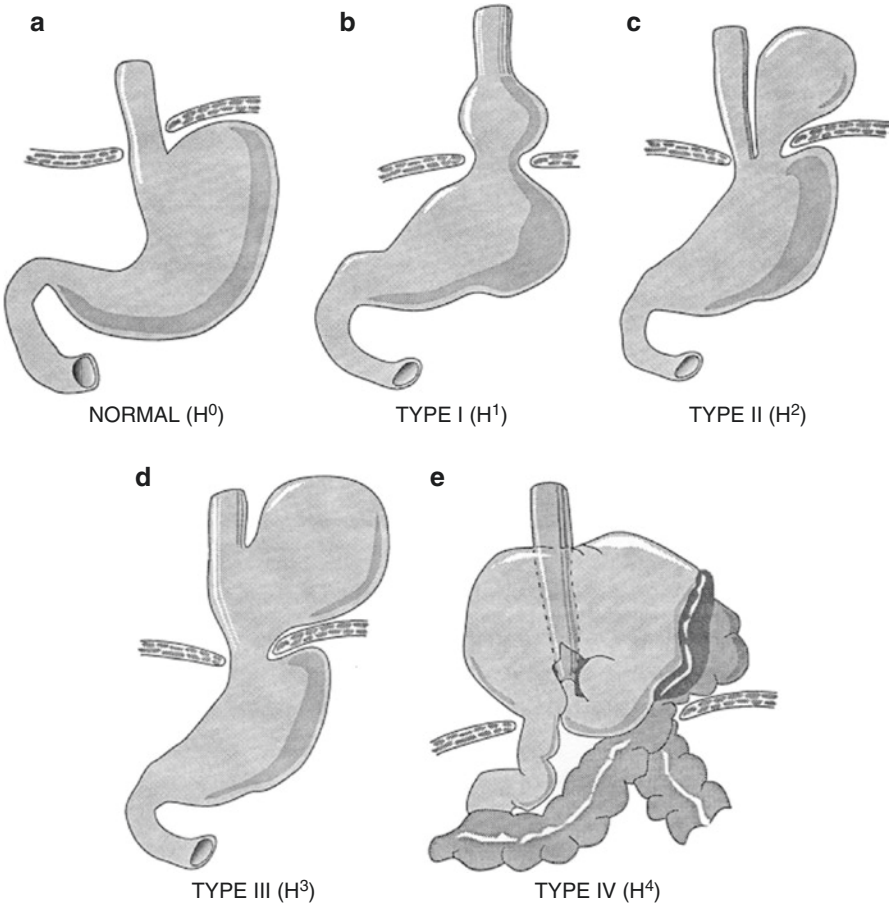

Fig. 18.4 (**a**) Normal hiatus, (**b**) Type I sliding hernia, (**c**) Type II paraesophageal, (**d**) Type III paraesophageal, and (**e**) Type IV. (From Zaman and Lidor [14])

define [3] because of the previously described phenomenon of "physiologic herniation" [6]. With these hernias, the phrenoesophageal ligament is weakened and thinned but remains intact. Therefore, there is a widening of the hiatus that allows the gastric cardia to herniate into the mediastinum. These hernias become clinically significant because of their association with gastroesophageal reflux [3]. This is likely due to the significantly larger cross-sectional opening at the esophagogastric junction in patients with hiatal hernias versus patients without hiatal hernia at different intragastric pressures, as was eloquently described in a study by Pandolfino et al. [24]. The hiatus becomes circular in dimension as opposed to elliptical [24].

Paraesophageal

These hernias are less common than the sliding type hiatal hernia and correspond to approximately 5–15% of all hiatal hernias [25]. Although these hernias are also associated with gastroesophageal reflux, their clinical significance is rooted in the mechanical complications [3] to include obstruction, dysphagia, and organ strangulation.

Type II

These hernias result when there is an actual defect in the phrenoesophageal membrane that allows herniation of the gastric fundus while the gastroesophageal junction remains tethered at the hiatus [26].

Type III

Type III hernias are viewed as a progression of a Type I or II hiatal hernia (Fig. 18.5). As the Type II hernia enlarges, there is continued weakening of the phrenoesophageal membrane that allows the gastroesophageal junction to slide into the mediastinum [3]. Therefore, the Type III hernia is by definition a combination of the Type I and Type II hiatal hernias.

Type IV

These are large hernias where the hiatus has enlarged enough to accommodate herniation of other organs in addition to the stomach. These can be associated with a large variety of symptom profiles.

Congenital

Morgagni

This hernia was first described by anatomist Giovanni Morgagni in 1769 as an "anterior retrosternal diaphragmatic defect that occurs between the xiphoid process of the sternum and costochondral attachments of the diaphragm" [2] (Fig. 18.6). These hernias result from a failure of the complete migration of muscle fibers to cover a triangular space between the sternum and bilateral costal margins, and herniation of abdominal contents usually results from trauma, obesity, or pregnancy [27]. Although these hernias are congenital, they are often not diagnosed until adulthood when they become symptomatic or as an incidental finding [28]. These hernias should always be

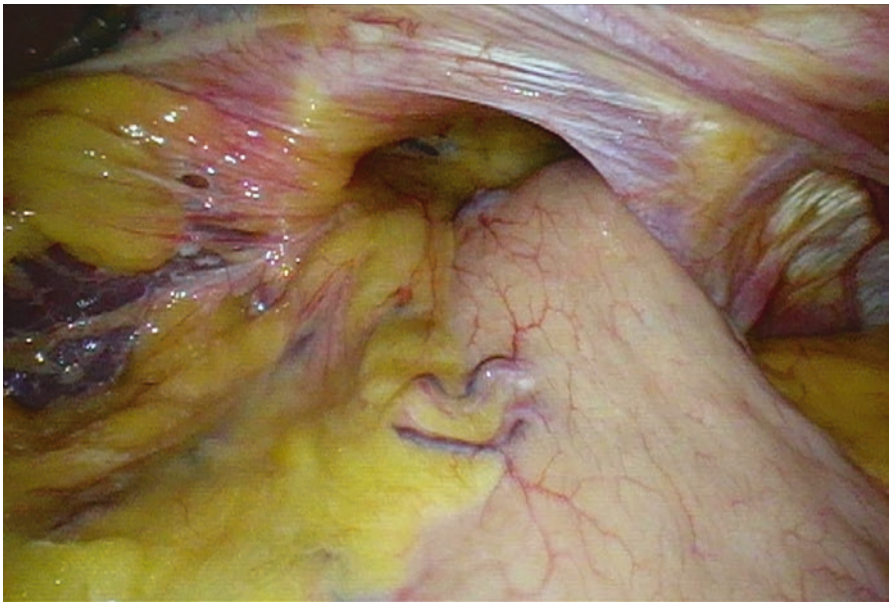

Fig. 18.5 Intraoperative view of sliding hiatal hernia with cardia and gastroesophageal junction above the diaphragm

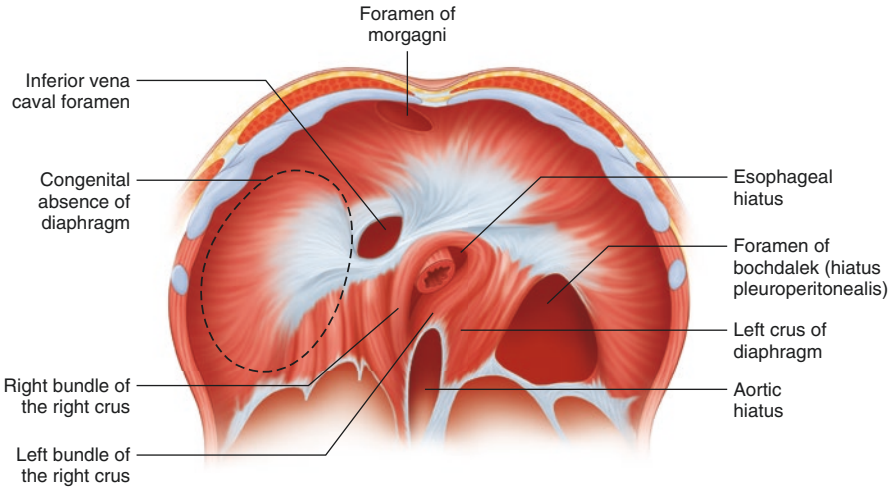

Fig. 18.6 Anatomy of Morgagni and Bochdalek hernias. (From http://www.continentalhospitals.com/blog/diaphragmatic-hernia/)

repaired upon diagnosis for fear of complications of obstruction or strangulation. Repair is usually completed transabdominally, although a transthoracic approach may be required for herniation above the carina with the use of mesh for larger defects [2]. Minimally invasive techniques have been an accepted approach for many years [27].

Bochdalek

Bochdalek hernias comprise 90% of congenital diaphragmatic hernias [29]. The diagnosis is often made with antenatal scans or at the time of delivery with respiratory distress [29]. As first described by anatomist Vincenz Bochdalek, these are congenital diaphragmatic hernias that occur during early embryologic development (Fig. 18.6). During this phase of development, the gastrointestinal tract is formed, and due to abnormal development of the pleuroperitoneal canal, the viscera become contained in the chest which prevents normal development of the lung [2]. Therefore, this pulmonary hypoplasia as well as associated vascular and cardiac abnormalities can result in a neonatal mortality of almost 50% [29]. For these patients, intensive cardiac and respiratory support are required for whatever period is necessary to obtain clinical stability with surgical intervention as a secondary goal [29]. Primary repair is often achievable, although more complex repairs may require a patch for a large defect and/or silo placement to allow return of the viscera to the abdomen [2].

Evaluation

Clinical Presentation

Chronic Symptoms

Most chronic symptoms result from the anatomic changes at the esophagogastric junction [30]. Most commonly patients will complain of reflux symptoms to include both heartburn and regurgitation. The hernia causes separation of the lower esophageal sphincter from the diaphragmatic crus which leads to acid exposure at the esophagus [25]. Concurrently, once this acid refluxes into the esophagus, the hiatal hernia compounds the exposure by also preventing acid clearance [31]. Increased intragastric pressures caused by the hernia also impair gastric emptying which complicates the reflux mechanism further [30].

Dysphagia may be the presenting symptom when the herniated portion compresses the distal esophagus. Stasis within the herniated stomach can also lead to symptoms of dysphagia [25]. Simple discoordination at the distal esophagus caused by the separation of the lower esophageal sphincter and the crura is often experienced as dysphagia by the patient [25].

Bleeding or anemia can be the presenting sign in patients with Cameron lesions, although other sites of gastrointestinal hemorrhage must be excluded [30]. Chest pain is a non-specific symptom associated with hiatal hernia, although, again, cardiopulmonary etiologies must be ruled out. Progressive dyspnea can be a presenting symptom that is often assigned to a cardiopulmonary or age-related source [32]. If

hiatal hernia is identified as the source of dyspnea, repair can result in improvements in pulmonary function which correlate to the size of the hernia [33].

Acute Symptoms

Symptoms in the acute setting are primarily associated with paraesophageal hernias and are related to obstruction, ischemia, or volvulus [25]. Patients who present with obstruction usually have non-distended abdomens, can usually be managed with nasogastric decompression, and often resolve spontaneously [30]. For those who are unable to be managed nonoperatively due to a deteriorating clinical picture, the concern is strangulation and eventual necrosis of the stomach. Although necrosis is rare, it is the leading cause of mortality from hiatal hernia [34]. Patients in whom necrosis is possible, emergency diagnostic upper endoscopy or surgical intervention are essential.

Radiography

The primary role of both chest radiography and computed tomography is initial identification of the hernia either incidentally or in the acute setting. For adequate radiologic evaluation of a hiatal hernia, the primary study is a barium swallow because it identifies the anatomy of the hernia, the relative orientation of the hernia contents, and localizes the gastroesophageal junction (Fig. 18.7). In addition, this

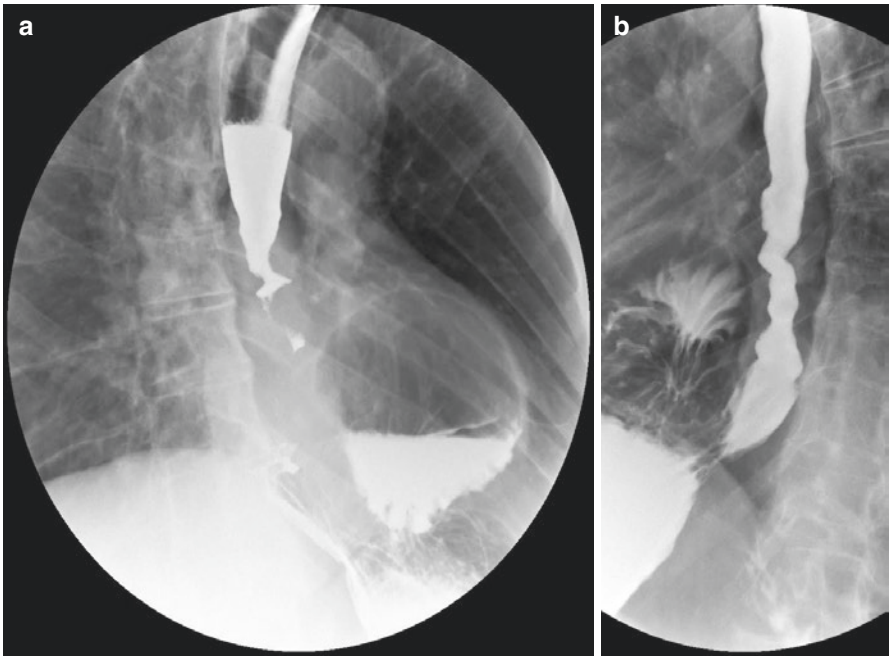

Fig. 18.7 Barium swallow. (**a**) Type III paraesophageal hernia depicting herniation of the gastric cardia and fundus above the diaphragmatic hiatus. (**b**) Type II paraesophageal hernia with herniation of the gastric fundus adjacent to esophagus with the esophagogastric junction still tethered at the hiatus

study is conducted in real time, so it allows the radiographer to identify the anatomy as it relates to other key structures and how these essential elements interact. For paraesophageal hernias, the imaging and diagnosis is fairly straightforward, whereas the diagnosis of Type I hiatal hernias can be more difficult.

Because of variations in protocol and radiographic criteria for defining hiatal hernias, there can be significant differences in interpretations of barium swallows. In order to visualize the key structures, they have to be distended, which intrinsically changes their relative positions. This distension causes shortening of the esophagus and displacement of the esophagogastric junction which is the basis of the 2 cm rule [3]. The 2 cm rule states that there must be more than 2 cm between the diaphragmatic hiatus and the squamocolumnar junction (or B ring) for diagnosis of a Type I hiatal hernia [35]. Without visualization of the B ring, three rugal folds above the diaphragm are necessary for diagnosis. Additionally, the timing of measurements during the peristaltic sequence can have significant effects on the results. If measurements are taken early in the peristaltic sequence, the size of the hernia will appear significantly larger than if the measurements are taken at the end of the sequence [3]. These variations can make the sizing and identification of small sliding hernias especially difficult and intrinsically erratic. Barium swallow can also help identify issues with esophageal motility which can be further elucidated with high-resolution manometry.

Endoscopy

Upper endoscopy is an important part of the evaluation of hiatal hernias. It allows for accurate diagnosis of hiatal herniation and is important in evaluating potential complications such as bleeding and dysphagia (Fig. 18.8). A Type I hiatal hernia is defined on endoscopy as a 2 cm separation of the squamocolumnar junction and the

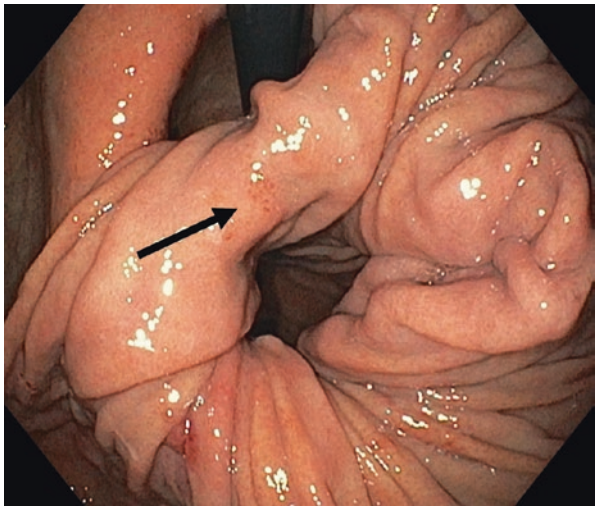

Fig. 18.8 Retroflexed endoscopic view of paraesophageal hernia. Arrow indicates early Cameron lesions which are a common etiology of anemia in patients with hiatal hernia

diaphragmatic pinch as the stomach encounters the crura. This diagnostic criterion can be limited in settings such as Barrett's metaplasia or esophagitis where the squamocolumnar junction is obscured. The mobile nature of the esophagogastric junction can also make measurements quite difficult. Paraesophageal hernias are a more straightforward diagnosis although endoscopy can be difficult due to the tortuous path.

Just as with barium swallow, there can be significant variations between endoscopic technique and interpretation. Bytzer showed that patient history alone can bias interpretation of endoscopic images where only 23% of endoscopists showing the same video case interpreted the same diagnosis [36]. This inherent variability in endoscopy and its interpretation can limit the value of information drawn from endoscopy, especially in hiatal hernias less than 3 cm in size [3]. Retrograde view can provide some extra information about the integrity of the hiatus and displacement of the squamocolumnar junction relative to the hiatus. Additionally, variations in the extent of gastric distention with insufflation may cause an inherent 2 cm error in size measurement [3].

Manometry

The esophagogastric junction can be identified with high-resolution manometry because of three physiologic phenomenon: (1) intragastric pressure is greater than intraesophageal pressure, most notably during inspiration, (2_ the pressure wave seen at the esophagogastric junction has both tonic (representing the lower esophageal sphincter) and phasic (representing the crura) elements, and (3) there is relative movement and intraluminal pressure changes at the esophagogastric junction during respiration [3]. High-resolution manometry with pressure plotting helps locate the upper esophageal sphincter, lower esophageal sphincter, and crural diaphragm in real time as the three high-pressure zones. A separation of greater than 2 cm between the lower esophageal sphincter and crural diaphragm is defined as a hiatal hernia [25]. There can be great variation in how well defined these high-pressure zones are in individuals which can create some interpretive variability in diagnosis [3]. Even with this variability, high-resolution manometry has dramatically improved diagnostic capabilities because of its ability to localize the lower esophageal sphincter and crural diaphragm in real time without swallow or distension-related distortions seen in barium swallow and endoscopy [3].

Diagnostic Summary

Although all the diagnostic modalities are plagued with some inadequacies, it is important for the clinician to take all the information provided to formulate a reasonable approach to the care of the patient. It is important for the gastroenterologists, surgeons, and radiologists to discuss the more complex cases where the diagnosis is not so apparent. It is imperative for each institution to create protocols and diagnostic standardization to promote consistency between different

practitioners. Precise discussion of patient-specific symptoms can provide invaluable clues as to the diagnosis and predict which symptoms can be alleviated with surgical intervention to maintain patient satisfaction.

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