Original Article

THE ANATOMICAL PERSPECTIVE OF GASTROESOPHAGEAL JUNCTION AND OCCURRENCE OF HIATUS HERNIA IN CADAVERS

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Abstract

One of the probable sites of herniation of the stomach into the thorax is the esophageal hiatus of the diaphragm. Lower esophageal sphincter mechanism is regarded as the major constituent guarding the aperture. Advancing age has its brunt on the diaphragmatic musculature, associated ligaments and the spine thereupon incapacitating the foramen producing upward pull of gastro-esophageal junction. Most of the anomalies were related to elderly women whose prevalence was reported incidentally. Despite the compression of the lung and heart in the mediastinum, the quality of life was not hampered and in most of the cases, kyphoscoliosis plays a paramount role in fostering the widening of the opening. PRISMA guidelines were followed for critical appraisal of the systematic review. Pubmed, Mendeley, Google Scholar, Google, Cochrane, ProQuest and Sci-Hub were the databases used to collect the literature review regarding anatomy of gastro-esophageal junction and its associated structures and hiatus hernia. This study documents various cadaveric reports of the thoracic stomach and details the anatomical features of the esophago-gastric intersection.

Keywords: Esophageal hiatus, gastroesophageal junction, hiatus hernia, kyphoscoliosis, lower esophageal sphincter.

INTRODUCTION

The esophageal hiatus (EH) is one of the major openings of diaphragm surrounded by right crural skeletal muscle fibers. The competency of the EH is maintained by the lower esophageal sphincter (LES). Till recently, most of the authors believed that LES was a physiological sphincter.[1] But later few authors observed that there is a thickening of circular smooth muscle fibers at the level of the distal esophagus.[2,3] Both the skeletal and smooth muscle components together form an effective sphincteric mechanism to prevent the occurrence of hernia. Apart from the muscular contributions for the sphincter, there are ligaments like gastro-phrenic ligament (GPL), gastro-hepatic ligament (GHL) and phreno-esophageal ligament (PEL). Among these, the PEL is considered to play a significant role as it possesses more elastic fibers and its close anatomical relation with gastroesophageal junction (GEJ).[4] Disrupted LES can be due to age-related changes in the crura and PEL, osteoporotic kyphoscoliosis, obesity and disorders of collagen synthesis thus causing widening of EH leading to herniation of the stomach into the mediastinum as hiatus hernia (HH).[5] This study describes the anatomical features of GEJ and documents various cadaveric reports of HH.

MATERIAL AND METHODS

PRISMA guidelines were adhered for critical appraisal of the systematic review. The databases used for the collection of literature review regarding anatomy of gastro-esophageal junction and its associated structures and hiatus hernia are Pubmed, Mendeley, Google Scholar, Google, Cochrane, ProQuest and Sci-Hub.

Search terms used were intrathoracic stomach, diaphragmatic hernia, hiatus hernia, anomalous diaphragm, esophageal hiatus, gastroesophageal junction, hiatus hernia, kyphoscoliosis and lower esophageal sphincter. Search phrases used were development of diaphragm, cadaveric reports of hiatal hernia, components of esophageal hiatus and blood supply of intrathoracic stomach. A total of thirty seven articles were obtained and using the following inclusion and exclusion criteria, thirty articles were selected.

Inclusion criteria

Full articles regarding the human anatomy and embryology of diaphragm, exclusive reports of cadaveric hiatal hernia were chosen as the inclusion criteria.

Exclusion criteria

Abstracts and case reports describing the hernia in patients were excluded in this study.

Outcome measures

Anatomy of GEJ, pathophysiology of HH and association of HH with scoliosis.

RESULTS

Nine full articles describing the anatomy of diaphragm and esophagogastric junction, seven articles explaining the etiology of hiatal hernia and pathological correlation of esophageal hiatus, six cadaveric reports of hiatal hernia and four articles describing the cases of hernia and its surgical management, two cases with scoliosis were collected and analyzed.

DISCUSSION

Anatomy of attachment of the diaphragm

The musculature of diaphragm arises from three sources namely the sternal part arising from the xiphoid process and adjacent transversus abdominis muscle, costal part arising from the posterior surface of the costal cartilages of seventh to ninth ribs, bony part of tenth to twelfth ribs as well and vertebral part originating from the body and transverse processes of the first and second lumbar vertebrae as lateral crura and arcuate ligaments.[6]

Esophageal hiatus

The EH is an elliptical opening in the muscular part of the diaphragm on the left side of the midline, surrounded by right crural fibers at the level of T10 vertebra. Its superior margin is located anterior to its inferior margin, about a length of 3 cm and a diameter of 4 cm. The mean surface area of the EH is measured to be 5.84 cm^2 in cadavers. It is directly proportional to thoracic girth and regardless of sex, weight, height and BMI.[7] The esophagus passes through this hiatus from posterior to anterior and from right to left. Below this opening, the abdominal part of the esophagus is about one inch in length and communicates with the stomach at its cardiac end.[8] The muscular arrangement of crura around the EH is classified into 11 types. The most frequent type is the Type I wherein, muscle fibers forming the right margin of EH is contributed by the right crus of the diaphragm which arises from the ventral aspect of L2 to L4 spine, running ventrally upward, to get inserted into the central tendon. The left margin of EH is formed by the muscle fibers arising from the homologous site, traversing dorsally upward with the right bundle which covers the anterior part of the distal esophagus. The second commonest type is the Type II which is almost similar to the Type I except few muscle fibers from left diaphragmatic crus passing posterior to esophagus and inferior to the muscle forming the left margin, eventually, get inserted to the muscle fibers contributing the right margin.[9] The majority of bundles of collagen fiber arise from the right diaphragmatic crus and form thicker right margin in contrast to the left thin margin of the

orifice.[10]

Gastroesophageal junction

The lower part of the thoracic esophagus is related to pericardium anteriorly, descending aorta posteriorly and pleura laterally. The esophagus is tethered two to three cm superior to the EH at its distal end by an attachment of circular elongation of fascia transversalis from the undersurface of the diaphragm and it is called as PEL.[11] The GEJ is kept at the normal anatomical position by the physiological function of the lower LES as an antireflux barrier. This sphincteric mechanism is also contributed by the PEL, GHL, GPL and the gastroesophageal mucosal flap.[4,11] This mucosal valve causes a pressure gradient around the EH thereby aiding the continence of GEJ. But the abovesaid function does not require the active contraction of LES.[1]

Miller et al. suggested that a substantial quantity of smooth muscle fibers at the distal part of the esophagus corresponding to the gastric-sling-clasp muscle group might be responsible for the normal location of GEJ.[1] The high-pressure zone of the lower esophagus is made by three separate sphincteric elements. The first element is the skeletal musculature of the crura of the diaphragm. The other two elements are made up of smooth muscle fibers. The proximal smooth muscle component is formed by the circularly arranged distal esophageal muscle fibers which move with crural element because of its strong attachment to the phreno-oesophageal ligament. The distal smooth muscle element is located below the diaphragm at the level of GEJ. This element is the primary factor that prevents gastro-esophageal reflux disease (GERD) and HH.

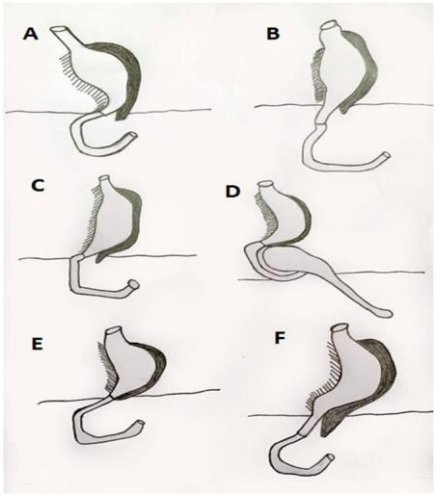
Apaydin et al. demonstrated a profound aggregation of circular muscle fibers of the gastroesophageal region in all the specimens of the study and called it as the internal anatomical sphincter.[2] The mean length of this muscular thickening was 3.1 cm and the densest part of this segment was in the middle, measuring 5.4 mm. The thickness of the esophagus immediately above the EH was 2.7 mm. The above-said internal sphincter and the external sphincter formed by diaphragmatic crura act together in maintaining a high- pressure zone at the GEJ. The diaphragmatic crural fibers exhibit a resting muscle tone that relaxes and contracts after distal esophageal distension and gastric distension respectively. Thickening of circular smooth muscle fibers at the distal esophagus was observed crossing at the angle of His and merging with the anterior and posterior oblique muscular bundles of the stomach. The fibers around the EH were arranged like a knot formed by the right crus of the diaphragm as a component of LES.[3] This sphincteric action of the crura is a reflex moderated by the inhibition of the esophagocrural fibers and excitation of the gastrocrural fibers to maintain the competency of GEJ. The distal esophageal sphincter or lower esophageal high- pressure zone is a 3–5 cm area in the abdominal esophagus acting as a sphincteric valve which prevents regurgitation of gastric contents into the esophagus.[13]

The PEL was first described by Galen in the year 1821, which connects the esophagus and the diaphragm and later it was called Treitz-Laimer membrane in the year 1883. Allison, a British surgeon identified the significant function of PEL in preventing HH. In 1973, Eliska described four types of PEL: fetal type, juvenile type, old age type and transitional type. In the fetal and infantile types, PEL consists of upper and lower phreno-esophageal membranes which arise from the fascia covering the thoracic and abdominal surface of diaphragm respectively.[14,15] The lower phreno-esophageal membrane had upper and lower limbs. Both the limbs were continuous with each other and connected margins of the hiatus with the adventitia of distal esophagus.[11] The elastic nature of both limbs allowed the normal physiological movement of the esophagus but in old age type, the upper limb degenerated and the lower limb became less elastic and allowed the free movement of GEJ. In the transitional type, the lower limb of the lower phreno-esophageal membrane was longer and passed through EH which was active than the other limb. If this type continues to persist, it can lead to HH with the replacement of connective tissue by fat in due course of time.[15]

The PEL is regarded as a significant component of LES in comparison with other ligaments because of its anatomical closeness with GEJ. The PEL accounts for the esophagogastric competency by acting against the upward displacement of the stomach into the mediastinum but allowing normal movement of the distal esophagus. Most of the studies infer that the PEL is principally formed by fascia transversalis and also by endo-thoracic fascia. On either side of the esophagus, the PEL is divided into two leaves. The upper broader leaf is attached to the esophagus above the EH and the lower narrower leaf is attached to the esophagus proximal to the GEJ extending one to two cm above it. Histologically, this ligament is composed of collagen and elastic fibers and richly vascularised.[16,11] Elastic recoiling and countertraction by PEL can be responsible for preventing the shift of GEJ during the longitudinal propulsive movement of the esophagus.

Herniation of stomach into the mediastinum can be due to enlarged EH. The types of congenital HH are (i) Sliding type (ii) Rolling type (iii) Mixed type and (iv) Associated with herniation of other abdominal organs like intestinal loops, spleen and colon.[17] Herniation of 30% or greater than 30% of the stomach is called a giant intrathoracic stomach which is a life-threatening condition causing symptoms like dyspnoea due to left atrial compression, intestinal obstruction, haemorrhage, volvulus complicating gangrene.[17,18,19,20,21] Acquired HH can be due to short esophagus, GERD, spinal disorders, increased BMI and age-related weakness of diaphragmatic musculature.[17,18,22]

Figure 1: showing different degrees of sliding hiatal hernia



A and E show intrathoracic stomach

B and F show most of the stomach herniating into the thorax while its pyloric part located in the abdomen

C shows entire herniation of stomach into the thorax with the initial part of the duodenum at the esophageal hiatus

D shows complete herination of stomach, duodenum and head of the pancreas

An 84-year-old female cadaver showed an intrathoracic stomach along with the herniation of lesser omentum and a part of the greater omentum [Figure 1A]. In this case, the EH was measured to be 4.3 cm in length at the level of T8-T12 vertebra because of the superior displacement of the diaphragm.[23] Similarly, Ishizawa et al. reported a 91-year-old Japanese cadaver with HH-associated with kyphosis where most of the stomach herniated into the thorax with axial torsion while the pyloric part of the stomach was seen in the abdominal cavity.[24] The stomach showed greater curvature ventrally and lesser curvature dorsally. The diameter of the EH was 4 cm and found at the level of the T12 vertebra. The histological aspect of the margin of the EH revealed muscle degeneration in the loose connective tissue beneath the peritoneum. Regarding the vasculature, the blood supply was found normal but with the longer celiac trunk [Figure 1B].

A female cadaver aged 97 years, showed the entire stomach inside the chest cavity and associated with kyphoscoliosis. Besides, the heart was enlarged and had a left-sided mediastinal shift compressing the left lung [Figure 1C]. The initial segment of the duodenum was found within the EH, near the pylorus along with the superior shift of head of the pancreas, but the remaining part of the pancreas and spleen were in the normal position.[22]

Dissection of a 66-year-old female cadaver revealed HH of Type IV along with herniation of stomach [Figure 1D], most of the duodenum and head of the pancreas into the posterior mediastinum. Further, the case presented with thoracic and lumbar scoliosis and kyphosis.[25]

Riefel et al. identified a giant HH in a 105-year-old female cadaver wherein almost entire stomach was observed in the thorax behind the normally-positioned heart without the compression of the left lung. The GEJ was identified at the level of the hilum

of the lung. The pylorus was located at the upper part of EH along with the presence of duodenum, a small gastric diverticulum and a part of the greater omentum [Figure 1E]. The blood supply was derived from the celiac trunk which had no variation.[18]

A 70-year-old female cadaver with kyphoscoliosis was dissected by the authors in the Department of Anatomy which revealed herniation of two-third of the stomach into the posterior mediastinum. On exploration, heart was found displaced anteriorly and the left lung smaller in size compared to the normal right lung. There was an upward displacement of gastro-esophageal junction through the wider esophageal opening at the level of T7 vertebra with major part of stomach along with a short lesser omentum accompanied by protrusion of greater omentum into the abdomen [Figure 1F]. The vertebral column showed right-sided scoliosis and the thoracic aorta followed the course of spinal column. Celiac trunk supplied the herniated organ via its gastric branches.[26]

Etiology of Hiatal hernia

The occurrence of HH can be due to any one or combination of the following two factors: GERD followed by cicatrization of the lower part of esophagus which leads to short esophagus that exerts upward traction on the stomach and a high positive-pressure at the level of EH resulting in HH and GERD.[17] Maziak et al. reported the presence of short esophagus in most of the patients out of 91 cases. Hence, the shortness of esophagus can be a cause and recurrence for HH.[27]

The reason for the incompetency of EH is poorly documented. Weakened intrinsic sphincter of distal esophagus, loosely arranged fibers looping around the cardiac part of stomach, increased intra-abdominal pressure were seen in cases of HH,[13] fibromuscular atrophy leading to loss of elasticity of the structures around the EH [28] and more derangement of LES is considered to be the potential factors in causing incompetent EH. An inborn error of collagen synthesis leads to HH rather than mechanical stress and a decrease in collagen Type I and Type III ratio can be the prime reason for the recurrence of HH. But, Brown et al.[29] studied the collagen content of PEL, GHL and GPL to rule out the etiology of Type I and Type III HH and he concluded that the defect in collagen synthesis may not be the cause for HH because the collagen Type I and Type III ratio was more or less equal to the control group. In a study by Curci et al. and Apaydin et al.[4,16] it was inferred that a reduction and derangement of elastic fibers in PEL and GHL was observed in cases of GERD and HH. Regarding the thoraco-abdominal pressure gradient, this difference was not observed across GEJ in cases with HH.[12]

KS is due to the result of wear and tear in the musculoskeletal system, long-standing abnormal body posture and asymmetry of para-vertebral musculature and its innervations. Other factors include an error in the distribution of collagen, depletion of serum melatonin, mutated expression of growth factor and genetic etiology.[30] In a study involving 320 patients, 93 of them had a giant intra-thoracic stomach associated with scoliosis. Scoliosis at the level of EH results in lateral and anteroposterior deflection of the vertebrae precipitating the distortion of LES mechanism and the curved spine causes upward displacement of lumbar vertebrae which in turn increases the intra-abdominal pressure which results in GERD and HH.[28,5]

The intensity of curvature spine has a well-established correlation with the size of the hernial sac in the case of old women.[6] Also, the KS generates increased intra-abdominal pressure and intra-abdominal volume which can provoke the formation of HH.[24,5,6] Apart from muscular, connective tissue, bony defect and advancement of age, the increased BMI is frequently associated with the occurrence of hernia, more commonly in males.[28]

Treatment

The surgical repair of HH is accomplished by the reduction and extirpation of hernia sac and repair of diaphragmatic crura.[17] In the case of the acquired short esophagus, the length of the esophagus has to be determined before the selection of the mode of surgical repair and to perform adequate mediastinal mobilization of the esophagus, anti-regurgitation management for GERD as well.[27,17] Valvuloplasty with the surgical fixation of GEJ to the fascia covering the ventral aspect of aorta would create a pressure gradient by enhancing the gastro-esophageal valve rather than simple valvuloplasty.[12]

Limitations

The incidence of hiatus hernia in cadavers and such cases with scoliosis were scarce. For this reason, the exact correlation between scoliosis and HH could not be inferred. Hence, there is a need for broader systematic review, in order to prove the relationship among HH, scoliosis and pathophysiology of GEJ.

CONCLUSION

After analyzing the cadaveric reports, it is concluded that senile muscular weakness can be a causative factor in distorting the EH. The incidence of HH is more in women of more than 65 years of age as a result of a decrease in serum estrogen and osteoporosis. Age-related physical changes of vertebral structures can cause KS, thereby the osteoporotic changes malform the vertebrae into wedge-shaped bones whose apices are stressed by the weight of the body leading to deviation of the spine or a congenital spinal anomaly in due course of time can result in HH and GERD. Few of the authors reported that HH was an incidental finding without marked signs and symptoms. It is presumed that a larger number of the reports might have had acquired HH rather than congenital because the vascularity was derived from the normal commencement of celiac trunk from the abdominal aorta, with the course of the gastric branches through EH from the abdomen into the thorax.

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CONFLICT OF INTEREST

None to declare.

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