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REVIEW ARTICLE

Giant Hiatus Hernia and Association with Gastro-Oesophageal Reflux: A Review

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Abstract

The term "giant hiatus hernia" has been variably defined, but most authors refer to at least 30% of the stomach, with or without other abdominal viscera, herniating through the oesophageal hiatus of the diaphragm into the mediastinum. This causes dysfunction of the lower oesophageal sphincter complex and can lead to obstructive symptoms, atypical extra-oesophageal symptoms as well as medically difficult to control reflux. When indicated, operative repair involves complete reduction of the hernia and the associated peritoneal sac, and partial hiatal closure in combination with an anti-reflux procedure. Surgery for giant hiatal hernias is complex, but can be performed with low morbidity and mortality with effective, long-term symptom resolution and improvement in health related quality of life.

Keywords

Giant hiatus hernia, Para-oesophageal hernia, Gastro-oesophageal reflux, Reflux, GORD, GERD

Introduction

A Hiatus Hernia (HH) describes the protrusion of intra-abdominal contents, through the oesophageal hiatus of the diaphragm and into the mediastinum. The hernia typically contains the stomach to varying degrees but may include other organs such as the omentum, colon, liver, pancreas and spleen. The term "Giant Hiatus Hernia" (GHH) has been variably defined, but most authors refer to at least 30% of the stomach, with or without other abdominal viscera, protruding through the oesophageal hiatus of the diaphragm into the mediastinum [1-6]. Symptoms arise due to dysfunction of the lower oesophageal sphincter complex and due to the mechanical effects of the stomach or other viscera above the diaphragm.

Gastro-oesophageal reflux is a common problem worldwide with an estimated prevalence of 10-20% in the Western world [7,8]. The causes of GORD can include lifestyle choices (smoking, eating habits) as well as obesity and the presence of a hiatus hernia. The aim of this review is to focus on the association of GORD and its management in patients with GHH.

Methods

A literature search was performed on PubMed, MEDLINE and EMBASE and suitable clinical papers were selected for review. A systematic search was carried out with the following keywords: hiatus hernia, hiatal hernia, paraoesophageal hernia, giant, large, GORD, GERD and reflux. The last update of searches was performed on 19th December 2016.

Classification of HH

Four sub-types of HH are recognised [4,9]. The precise prevalence of each subtype is not known due to variations in techniques and definitions used for radiologic and endoscopic diagnosis, the fact that the majority are asymptomatic and also a lack of large prospective population studies [10]. Type I is the most common accounting for 80-85% of HH [10]. This is a sliding HH with axial migration of the stomach into the chest with the Gastro-Oesophageal Junction (GOJ) remaining the

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Table 1: Incidence of reflux in patients with giant HH.

Authors	N	Definition of Hiatus Hernia	Patients with reflux (%)
Carrott, et al. [35]	270	Para-oesophageal hernia 87% had > 50% in chest	176 (65%) heartburn, 76 (28%) oesophagitis
Carrott, et al. [2]	120	No definition 95 of patients had > 50% of their stomach in the chest	71 (59%) heartburn
Pallabazzer, et al. [58]	38	Type III or Type IV HH	33 (87%) heartburn
Chowbey, et al. [59]	73	> 5 cm hernial defect	42 (57.5%) heartburn
Leeder, et al. [3]	53	Symptomatic para-oesophageal HH All had > 50% of stomach in the chest on "barium swallow"	23 (43%) reflux symptoms 9 (17%) oesophagitis
Aly, et al. [1]	100	> 50% stomach in chest	79 (79%) heartburn, 55 (55%) oesophagitis
Geha, et al. [60]	100	> 50% stomach in chest	74 (74%) heartburn 2 (2%) oesophagitis
Maziak, et al. [61]	94	Incarcerated paraoesophageal HH. No definition	78 (83%) symptomatic reflux 34 (36%) oesophagitis

most cranial part of the stomach. Type II rolling HH or para-oesophageal hernias involve protrusion of part of the stomach into the chest with the GOJ retaining its normal position in the abdomen. These account for 3.5-10% of HH [11-13]. Type III HH account for 5% of cases and are mixed sliding and rolling HH. The GOJ and other parts of the stomach herniate into the chest and the GOJ is no longer the most cranial aspect of the stomach. Type IV HH are those incorporating the stomach and other abdominal viscera and occur in around 5% of cases. Overall GHH are reported to represent 0.3-15% of all HH [4] and are either Type III or IV, with Type III being the most common.

Pathophysiology of HH

The existence of HH has been recognized for centuries, but the precise pathophysiology that leads to this condition is not known [4,9,14]. Important factors include raised intra-abdominal pressure causing displacement of the GOJ into the thorax [15], weakening of the phreno-oesophageal ligament due to depletion of elastin fibres [16], oesophageal shortening secondary to longstanding reflux disease or vagal stimulation and age-related or congenital widening of the oesophageal hiatus [14,17,18].

GORD and HH

Symptomatic GORD is common in individuals with HH. The normal anatomical alignment of the GOJ has important functional implications and displacement from the diaphragmatic hiatus predisposes patients to reflux [19]. There is an established relationship between HH, reflux disease and oesophagitis [4,20-22] which is likely to persist for patients with *giant HH.* However, in this group, GORD may also be due to poor gastric emptying from relative gastric obstruction at the hiatus.

The precise incidence of GORD in patients with HH of any size is difficult to know and has been variably reported to be between 8-80% [20,21,23,24]. The incidence of GORD in patients with GHH is similarly divergent being reported between 43% and 87% (Table 1). The reasons for the divergence in incidence are unclear, but there are those patients with GHH who do not complain of GORD so that it may be the size of the HH is not relevant.

Pathophysiology of GORD in patients with HH

It is reasonable to extrapolate that some of the changes seen with small HH that predispose to GORD will also apply to GHH. The potential pathophysiology is outlined below.

Impairment of oesophageal acid clearance: Oesophageal dysmotility, seen in some patients with GHH, is associated with a reduced ability to clear acid contents, however, the data allowing analysis of the relationship between HH and oesophageal dysmotility is inconsistent [25-28].

- There is impaired oesophageal clearance of acid if HH is present [22,25].
- Patients with HH have higher rates of oesophageal dysmotility and abnormal oesophageal pH [25].
- It is not clear, whether dysmotility is a primary phenomenon, which results in poor clearance of acid or secondary to the reflux itself.
- GHH may impair the clearance of acid from within the oesophagus due to mechanical obstruction.
- The supra diaphragmatic gastric segment may act as a reservoir for acid containing material.
- Acid may be trapped in the HH during oesophageal clearance, refluxing into the oesophagus during Lower Oesophageal Sphincter (LOS) relaxation as the patient swallows [24,28,29].

Reduced basal LOS tone: Large HH have tenden-

cy towards a lower mean LOS pressure/tone, reduced LOS length and reduced intra-abdominal length of LOS contributing to the increased likelihood of GORD [22-25,28,30]. It is important to note that some patients with GHH have normal LOS pressures, which may explain why reflux is not always a major symptom in this group of patients [22,30].

Reduced gastric distension required for reflexive LOS relaxation: Prandial gastric distension results in reflex LOS relaxation. In patients with HH there is a reduced required gastric tension for receptive LOS relaxation, which increases the tendency towards GORD [23].

Interruption of the gastro oesophageal flap valve: HH disrupts this mechanism by: reducing the acuity of the angle between the fundus and the oesophagus and enlargement of the oesophageal hiatus with resultant widening of the oesophageal lumen [31,32]. This reduces the size of the mucosal fold and its effectiveness as a flap valve.

Dissociation of the LOS and the diaphragmatic pinchcock: Patients with HH have the high-pressure zone separated almost arithmetically between the diaphragmatic crura and the Squamo-Columnar Junction (SCJ) [33]. The following differences are noted.

- The high-pressure zone produced at the SCJ in these patients is cranial in relation to the diaphragmatic high-pressure zone.
- There is a radial and symmetrical distribution of pressure in patients without a HH. Most of the pressure from the crural fibres arises from an anterior and lateral direction. In patients with HH, only the pressure at the SCJ is radial. The pressure exerted by the diaphragmatic hiatus is irregular and this is disrupted as the size of the hiatus increases [30,33].
- The size of the diaphragmatic hiatus is negatively correlated to the LOS pressure [28,30].
- The lower pressure at the GOJ, seen with separation of the SCJ and the diaphragmatic crura, in combination with dis-coordinate contraction at the diaphragm, results in an increased risk for reflux in patients with HH, which will be magnified for a larger GHH.

Severity of reflux disease in patients with HH

The presence of any HH increases an individual's likelihood of GORD and if a HH is present, the severity of GORD is usually greater [20,22]. Given that the natural history of both HH and GORD is for a slow progression, it is difficult to elucidate if larger hernias are etiologically implicated in worsening reflux or if both disorders progress in parallel. A prospective study by Franzen, et al. [34] looked at the severity of GORD and HH size. 75 patients were assessed using 24-hr oesophageal pH testing in 3 groups of patients with HH < 3 cm, 3-5 cm and > 5 cm. They found the group with larger HH had significantly more GORD and acid symptoms compared to the groups with smaller HH.

The evidence for more significant GORD, when it is present in GHH is: endoscopically proven oesophagitis is more prevalent (Table 1); endoscopic grade of oesophagitis is higher [25]; larger HH are associated with higher number of reflux episodes lasting more than 5 minutes in 24-hour pH studies [28,29]; para-oesophageal hernias are associated with worse symptoms and endoscopic findings than fixed sliding HH [19].

Other symptoms from GHH

Non-reflux symptoms that arise from GHH occur due to the abnormal position of the stomach within the chest. This can lead to a gastric volvulus or complete obstruction of the stomach within a HH. Friction from the stomach moving in and out of the diaphragmatic hiatus can cause ulceration of the overlying gastric mucosa leading to upper gastrointestinal bleeding and chronic anaemia. The herniated stomach can also cause a mass effect in the posterior mediastinum leading to exertional dyspnea [35]. The annual risk of developing acute symptoms necessitating emergency surgery has been quoted as 1.1% per year [36].

Management of GORD in patients with GHH

General: The medical treatment of the GORD symptoms is acid suppression therapy with Proton Pump Inhibitors (PPI). In patients with GHH, there are reported short-term efficacy rates up to 90%, however, in the majority of patients, this effect wanes over time due to tachyphylaxis [8]. This group of patients are also at risk of specific problems related to the GHH. A careful history is important considering these symptoms may be subtle and slowly progressive over many years such that the causality of individual symptoms may not be recognised by the patient. In particular an assessment for the presence of mechanical symptoms is important as these represent an important indication for surgery compared with reflux symptoms alone [36]. Thus the standard indication for surgical intervention of a GHH includes mechanical symptoms arising from the GHH or an emergency presentation with obstruction [37,38]. There have been recommendations advocating repair of all GHH, if there is acceptable surgical risk, citing the natural history of a progressive increase in size and symptoms [39]. No comparative series are available to establish the optimal approach. In the presence of reflux symptoms alone without mechanical symptoms, the indication for surgery may be considered to be the same as for a patient without a GHH.

In a number of cohort studies where patients were selected to have a GHH repair it has been reported that: 80% to 92% of preoperative symptoms resolve [2,40]; there is an improved gastro-intestinal quality of life [37,40,41]; there is an objective improvement in pulmonary function [35] and there is an objective improve-

ment in cardiac function [42].

Prior to consideration of surgery the pre-operative assessment of patients being considered for GHH repair includes an upper gastrointestinal endoscopy to document the presence or absence of: oesophagitis; Barrett's metaplasia; Cameron's ulcers; oesophageal stricture and the size and nature of the hernia [43]. A barium swallow may give additional anatomic information regarding the type of hiatal herniation and the presence of any organo-axial volvulus highlighting the risk of future obstruction either progressive or acute. Patients will also require investigations appropriate for establishment of medical suitability for a major surgical intervention.

Laparoscopic repair is the standard approach in most centres, however advanced laparoscopic skills are required [1,41]. Operative mortality in series from centres with an interest in the disease is reported to be 0% to 2%, with operative morbidity between 13% to 16% [1,3,35,37]. Conversion to an open approach has been reported to be 0% to 7.5% [1,3,37]. The median hospital stay is 2 to 4 days [1,3,35]. Open repair is still advocated in some centres with one group suggesting a lower long-term recurrence rate [2,19,35,38]. A recent series of 270 patients who underwent open repair reported no post-operative mortality, but post-operative morbidity was 38% and the median length of stay 4 days [2].

The principles of operative treatment relate to the anatomy and the pathophysiology of GHH. These include: reduction of the contents of the hernia; complete dissection of the hernial sac from the posterior mediastinum; mobilization of the oesophagus from within the mediastinum, restoring a segment (1-3 cm) of intra-abdominal oesophagus; tension free anchorage of the intra-abdominal oesophagus; closure of the hiatal defect and construction of an anti-reflux mechanism.

These general considerations and indications for surgery are consistent with established guidelines for the management of hiatal hernias provided by the Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) in 2013 [16].

Surgical management; specific issues relating to GHH and GORD

The shortened oesophagus: The shortened oesophagus has been defined as "a relative shortening of the expected length of the oesophagus associated with intramural and peri-oesophageal scarring and fibrosis, which inhibits the easy re-establishment of normal length during oesophageal surgical procedures" [44]. In the modern era, the importance of the short oesophagus is debated. It has been suggested that due to the liberal use of proton pump inhibitors, the incidence of true fibrous shortening is rare and only occurs in the setting of severe oesophageal stricturing and/or a large fixed HH [38,40,41,45]. The incidence of a shortened oesophagus has been reported to be 7% to 19% in patients with reflux disease and a large HH [19,45].

The relevance of the short oesophagus relates only to patients having surgical intervention, however, there are no objective measures to assess oesophageal length using radiology, endoscopy or after surgery has commenced. In most cases adequate mobilisation of the mediastinal oesophagus is sufficient to restore a segment into the abdomen. For those proposing an oesophageal lengthening procedure, there are no objective means to determine the optimal intra-abdominal oesophageal length [45]. For patients having surgical intervention, where the focus has been the achievement and maintenance of a segment of intra-abdominal oesophagus.

When oesophageal shortening is considered present, there are surgeons who advocate a Collis gastroplasty which in principle creates a tubed gastric segment (neo-oesophagus) below the true GOJ to relocation of a new "GOJ" in the abdomen. There are series reporting the use of this technique in 4% to 86% of patients treated for GORD with the higher rates in patients with larger hernias [41,44]. This procedure adds to the technical challenge of repair of the GHH. There have been no randomised trials assessing the use of the Collis gastroplasty in any series of patients with GHH and thus the role for this procedure is not clear.

The need for an anti-reflux procedure: The present standard of care for most surgeons is to incorporate an anti-reflux procedure in all patients as part of the repair of a GHH [13,16]. This is clearly most important in patients where GORD was a preoperative symptom. Specific to GHH GORD is not consistently remedied by fixation of the hernia alone and the fundoplication with fixation of the oesophagus in the abdomen may bolster a GHH repair, and perhaps reduce rates of recurrence.

The risk of recurrent HH: The risk of a symptomatic recurrent HH after GHH repair has been reported to be 5% after ten years of follow-up [36,40,41]. These symptoms can include recurrent reflux, dysphagia, chest pain, nausea and bloating [46]. If thorough radiological assessment is performed, a recurrent HH has been defined in 15% to 42% of patients (median 22%) [1,13,36,37,41]. Wang, et al. reported a prospective study looking at the clinical significance of asymptomatic recurrences and found that 35.7% of patient had a radiological recurrence [47]. Whilst there were significantly higher rates of subjective reflux and PPI usage at longer-term follow-up, 94.6% reported satisfaction with their initial operation and the re-operation rate of only 1.7%. Radiological recurrence is typically not to the same extent as the originally repaired GHH. Mostly these are small axial asymptomatic, hernias of less than 2 cm in length, of which the clinical importance is likely to be trivial [1,41]. In patients who only have recurrent reflux following a HH repair, medical management provides good symptomatic relief in the majority without

the need for re-operation and should be considered first line management in these patients.

The role of mesh to repair the hiatal defect

The use of synthetic or bio-absorbable mesh to support a hiatal repair is controversial with authors reporting a lower the rate of recurrent HH when mesh was used to reinforce the hiatal repair when compared with direct primary closure [48-51]. The potential disadvantages include oesophageal stricture, oesophageal obstruction and mesh migration into the oesophageal or gastric lumen [13,36,40]. The presentation of mesh related complications may be delayed, with some occurring beyond 10 years from original surgery [40].

A recent multicentre RCT of 126 patients comparing suture repair vs absorbable mesh (Surgisis) vs. non-absorbable mesh (TiMesh) for large hiatus hernias [52] no statistically significant difference in the rate of recurrent hiatus hernia between the 3 groups measured by barium meal radiology and endoscopy at 12 months. Symptomatically the outcomes were similar in all 3 types of repair without evidence to suggest that one technique was superior to the other. Two recent systematic reviews [53,54] compared mesh herniorrhaphy to primary hiatal suture when repairing GHH found a higher rate of recurrence in the suture repair group that was not statistically significant. The rates of re-operation for symptomatic recurrence were comparable between the two groups. Based on current literature, it is not possible to be definitive with respect to role of mesh, synthetic or biological for hiatal closure [16,48-51].

Novel techniques

Given the controversies surrounding the use of mesh, some authors have reported the use of autologous tissue to aid in hiatal closure. A small study (15 patients) reported the use of the falciform ligament to bridge the posterior hiatal defect [55]. The left triangular ligament has been used as an autologous graft to close an anterior defect after posterior crural repair [56]. Both techniques have small numbers but offer an alternative to mesh but lack long term data.

Diaphragm relaxing incisions have also been reported to aid in the closure of giant hiatal defects with a relaxing incision in the diaphragm parallel to the IVC, to the left of the right crus. The defect is closed with a PTFE patch. In a small number of patients (14/15) the authors reported good symptom control without hernia recurrence at 15 months [57].

Conclusion

GHH are relatively uncommon. Patients may be asymptomatic, present with reflux alone but more likely will have a combination of reflux and obstructive symptoms. Reflux is more common and more severe, when present, compared to patients without hiatal herniation. Symptomatic patients with GHH who are medically fit, should be offered operative repair. Although a complex procedure, surgical repair is performed with low morbidity and mortality. The basic principles for repair are clear, though there are a number of different techniques relating to the repair that are considered by some surgeons to be important for improved long term outcomes. These techniques have not been proven in randomised trials. There is no one defined procedure that when performed in all patients an excellent clinical outcome is guaranteed.

Statement of Author's Contribution

All three authors have:

1. Made substantial contributions to the concept and design of the work, as well as the acquisition, analysis and interpretation of data for the work,
2. Assisted in drafting the work and revising it critically for important intellectual content,
3. Approve the final version to be published, and
4. Have agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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