From Diagnosis to Treatment: A Literature Review on Hiatal Hernia Management

Darius Hartanto[*], Bradley Jimmy Waleleng[**], Luciana Rotty[**], Fandy Gosal[**], Jeanne Winarta[**], Andrew Waleleng[**]

*Department of Internal Medicine, Faculty of Medicine, Sam Ratulangi University, Manado, Indonesia

**Division of Gastroenterology-Hepatology, Department of Internal Medicine, Faculty of Medicine, Sam Ratulangi University, Manado, Indonesia

Corresponding author:
Darius Hartanto, Department of Internal Medicine, Faculty of Medicine, Sam Ratulangi University, Manado, email: dariushartanto11@gmail.com

ABSTRACT

A hiatal hernia is a medical disorder where the upper part of the stomach, or occasionally other intra-abdominal organs, abnormally protrudes through the diaphragmatic hiatus. The occurrence of this disorder ranges from asymptomatic to severe symptoms, depending on the extent of herniation and associated complications. The development of a hiatal hernia is associated with repeated acid exposure, shortening of the muscles, and proximal displacement of the esophagus. Based on the American Foregut Society (AFS) endoscopic classification, hiatal hernia is divided into types I–IV, which guide clinical evaluation and management. Moreover, management strategies vary according to symptom severity and progression, as treatment is usually not required in asymptomatic patients. For mild to moderate cases, non-pharmacological management is recommended, such as avoidance of smoking, weight reduction, and avoidance of trigger foods and beverages. Proton pump inhibitors (PPIs) are the first-line options in pharmacological treatment, while surgical intervention is the sole method capable of repositioning herniated organs back into the abdominal cavity and addressing the functional defects related to a hiatal hernia. Therefore, this review aimed to provide a comprehensive overview of hiatal hernia management, from initial diagnosis to therapeutic selection.

Keywords: *American Foregut Society (AFS), Endoscopic, Esophagus, Hiatal hernia, Proton pump inhibitors (PPIs)*

ABSTRAK

Hiatal hernia adalah kondisi medis yang ditandai dengan penonjolan abnormal bagian atas lambung atau organ intraabdomen lainnya melalui celah diafragma. Kejadian hiatal hernia dapat bersifat tidak bergejala sampai gejala berat. Paparan asam berulang, pemendekan dari otot dan berpindahnya esofagus kearah proksimal menyebabkan terjadinya hiatal hernia. Klasifikasi hiatal hernia berdasarkan endoskopi American Foregut Society (AFS) dibagi menjadi tipe I-IV. Pengobatan pada hiatal hernia biasanya tidak diberikan pada pasien yang tidak bergejala. Mencegah paparan rokok, penurunan berat badan, menghindari sumber makanan dan minuman pencetus menjadi pilihan dalam pengobatan non farmakologi. Proton pump inhibitor (PPIs) menjadi pilihan pertama dalam pengobatan farmakologi. Pembedahan merupakan satu-satunya cara untuk mengembalikan organ yang mengalami hernia ke dalam rongga intraabdomen dan untuk memperbaiki kelainan fungsional yang terkait dengan hernia hiatus. Review Article ini bertujuan untuk memberikan pengetahuan mengenai penangan secara komprehensif pada kasus hiatal hernia, dari awal diagnosis hingga pemilihan terapi.

Kata kunci: *American Foregut Society (AFS), Endoskopi, Esofagus, Hiatal hernia, Proton pump inhibitors (PPIs)*

INTRODUCTION

A hiatal hernia is a medical disorder where part of the abdominal cavity herniates through the esophageal hiatus into the mediastinal cavity, including the proximal portion of the stomach.[1] The diaphragm contains a small aperture that allows the esophagus to connect with the stomach at the gastroesophageal junction (GEJ).[2] However, when the stomach protrudes through the esophageal hiatus into the chest cavity, the lower esophageal sphincter (LES) is compromised. This shows that gastric contents and acid can reflux into the esophagus, causing gastroesophageal reflux disease (GERD).[2] Therefore, this review aims to provide comprehensive insight into the management of hiatal hernia, ranging from initial diagnosis to the selection of appropriate therapy.

METHODS

A literature search was conducted using PubMed/MEDLINE, Scopus, and Google Scholar. The search strategy included combinations of several keywords, namely "hiatal hernia," "paraesophageal hernia," "diagnosis," "classification," "management," "fundoplication," "Stretta," and "transoral incisionless fundoplication." Studies published between 2010 and 2026 were included when addressing the diagnosis or management of hiatal hernia and written in English. Eligible sources were clinical trials, observational studies, systematic reviews, and clinical guidelines. Case reports and expert opinions were not the primary focus but were included selectively when relevant. Subsequently, titles and abstracts were screened, followed by full-text review for eligibility. This narrative review was conducted without strictly following PRISMA guidelines, although a structured method was applied to literature selection.

EPIDEMIOLOGY

The prevalence of hiatal hernia is difficult to determine with certainty because most patients are asymptomatic or experience mild symptoms, leading to the estimation of occurrence frequency. The incidence rate often increases with age, affecting approximately 50–60% of patients aged 50 years. Approximately 9% of cases are symptomatic, with an overall incidence of 10–20% in Western countries.[3]

A study in the United States identified 316 cases of hiatal hernia from 3,179 non-contrast thoracic CT scans, with the highest prevalence observed in the 70–79-year age group, accounting for 45% of cases. The incidence was higher in women (66%) compared to men (34%), where the majority of cases were found in patients with central obesity, accounting for 88% of cases.[4] Another study conducted in Taiwan, including 255 patients who underwent laparoscopic vertical sleeve gastrectomy, reported type I hiatal hernia in 41 patients (16.1%).[5] Additionally, the report from Jakarta, Indonesia, identified this disorder in 16 of 51 patients (31.37%) who underwent endoscopic examination.[6]

PATHOPHYSIOLOGY

The majority of hiatal hernia cases are asymptomatic and are discovered incidentally, with chronic esophagitis due to acid reflux being considered a predisposing factor. This is because acid exposure causes contraction of the esophageal muscles, leading to shortening and proximal displacement of the esophagus. Increasing age also causes fibrosis of the longitudinal esophageal muscles, which predisposes to hiatal hernia. Dysfunction of the diaphragmatic sphincter, esophagitis due to repeated acid exposure, and hiatal hernia collectively contribute to enlargement of the hernia.[7]

Conditions associated with increased intra-abdominal pressure, such as obesity or multiple pregnancies, further exacerbate the disorder by causing deterioration of the pharyngoesophageal membrane, loss of elastic tissue integrity, expansion of the crural opening, and herniation. Consequently, a large hernia can develop, producing symptoms like early satiety, dysphagia, and regurgitation. In cases of incarceration or volvulus, compromised blood flow may lead to ischemia, necrosis, and sepsis.[8]

In terms of classification, type I hiatal hernia, which is commonly experienced, arises from the herniation of the GEJ into the thoracic cavity. The symptoms include decreased haemoglobin levels, regurgitation, acid reflux, dysphagia, chest discomfort, and a persistent cough. Type II occurs less frequently than type I but may cause conditions such as anemia due to ulcers on the gastric surface, postprandial acid reflux, and chest pain. Delayed esophageal and gastric emptying may result in digestive disturbances. Moreover, types II and III can cause regurgitation of food, and occasionally, blood may occur, leading to substernal chest pain. Aspiration of food particles and acid into the airway causes coughing and discomfort in the throat and chest. Type IV hiatal hernia is related to more serious and potentially fatal complications, including esophageal rupture, massive gastrointestinal hemorrhage, luminal obstruction, and strangulation of the intestines. [9]

DIAGNOSIS

Esophagogastroduodenoscopy (EGD)

Clinical indications for endoscopy include the presence of GERD symptoms refractory to treatment, alarm features such as anemia, gastrointestinal bleeding, dysphagia, and unexplained weight loss, as well as symptoms in patients older than 50 years. In asymptomatic cases, there is no indication to perform investigations for hiatal hernia detection.[10] To address this limitation, esophagogastroduodenoscopy offers the advantage of direct visualization of the esophageal, gastric, and duodenal mucosa. In addition to confirming the presence of a hiatal hernia, it can also detect abnormalities such as Barrett's esophagus, Cameron ulcers, and other lesions suggestive of malignancy.[11]

Esophageal Manometry

Esophageal manometry shows a separation of 2 cm or more between the crural diaphragm and the lower esophageal sphincter (LES). The limitation of this method is the difficulty in accurately positioning the manometry catheter, leading to application in fewer than 50% of cases.[11] Despite the limitation, manometry evaluates the adequacy of peristaltic function and is particularly valuable in the preoperative assessment of patients undergoing fundoplication.[10]

pH Test

A pH test is not often required for the diagnosis of a hiatal hernia, but it facilitates the analysis of reflux episodes in patients. Furthermore, pH is shown in patients with pathological GERD without high-grade reflux esophagitis.[11]

Computed Tomography (CT)

CT examination is not routinely performed, as a hiatal hernia is usually detected incidentally during CT scans conducted for other indications. Therefore, CT imaging is used to rule out complications such as perforation, pneumoperitoneum, and gastric volvulus in cases of paraesophageal hernia.[10,11]

CLASSIFICATION

Hiatal hernia can be classified into types I–IV based on the anatomical position of the GEJ and the extent of organ herniation into the intrathoracic cavity. Type I hiatal hernia, also known as a sliding-type hernia, is defined by the upward migration of the GEJ together with part of the gastric cardia through the diaphragmatic hiatus. It is the most common type, accounting for approximately 85–95% of cases. Meanwhile, types II–IV are collectively referred to as paraesophageal hernias. Type II is characterized by a normally positioned GEJ, with herniation of a portion of the gastric fundus into the intrathoracic cavity. Type III, also known as the mixed type, combines features of types I and II, comprising the protrusion of both the stomach and GEJ. Type IV is characterized by displacement of the GEJ higher into the thoracic cavity due to herniation of the stomach and other visceral organs, such as the pancreas, spleen, colon, and small bowel. The distinctions between types I-IV are shown in **Figure 1**.[12,13]

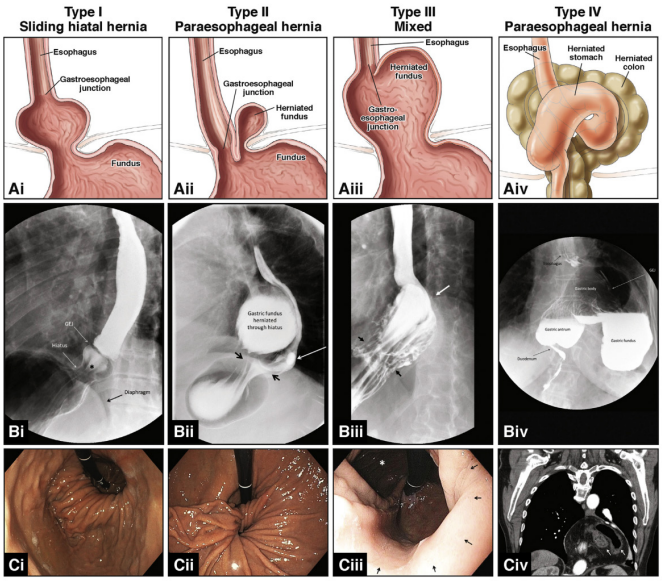
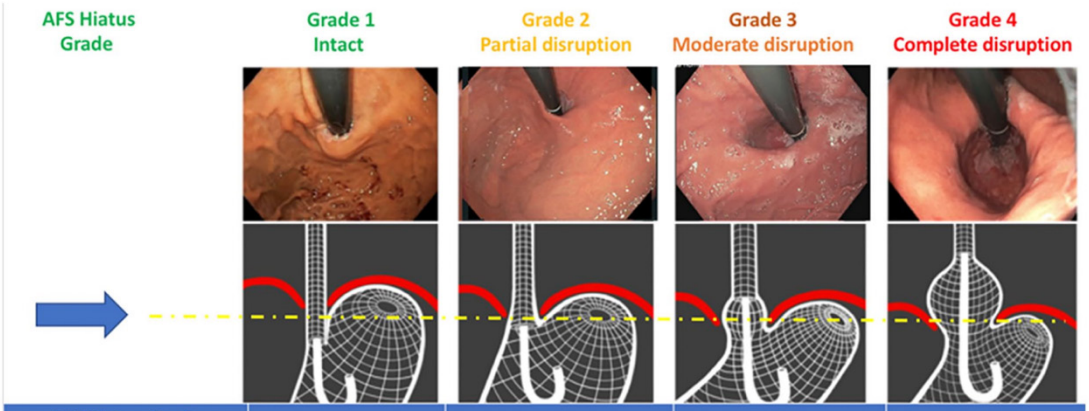


Figure 1. Comparison of Radiologic Appearances of Type I–IV Hiatal Hernias[13]



AFS Hiatus Grade	1	2	3	4
Hiatal axial Length, cm (L)	None (0 cm)	None (0 cm)	0-2 cm	>2 cm
Hiatal aperture, cm (D)	Snug to scope 1 cm	Loose 1-2 cm	Open 2-3 cm	Wide open >3 cm
Flap valve (F)	Present, full lip with Omega shape (F+)	Absent, thinning & flattening valve lip (F-)	Absent (F-)	Absent (F-)
LDF components	L0, D1, F+	L0, D1-2, F-	L0-2, D2-3, F-	L>2, D>3, F-

Figure 2. Hiatal Hernia Classification According to the American Foregut Society (AFS) Based on the LDF Components (Length/Diameter/Flap Valve)[14]

The American Foregut Society (AFS) classification system is determined by evaluating the extent of hiatal disruption as well as the presence or absence of the flap valve, using Length, Diameter, and Flap valve parameters. Length (L) refers to the hiatal axial length (cm), Diameter (D) is the hiatal aperture (cm), and Flap valve (F) describes the presence of the gastroesophageal flap valve. The AFS grading system starts with grade 1, which represents intact GEJ anatomy, a hiatal diameter of approximately 1 cm, as well as an intact, functional flap valve (L0, D1, F+). At this stage, there is no axial elongation of the GEJ, and the flap valve remains within normal limits. Endoscopically, this is characterized by the presence of thick, well-formed valve lips creating an omega configuration with an angle of approximately 270°. Grade 1 also has sufficient intra-abdominal esophageal length to maintain an effective flap valve[14]

In comparison, AFS grades 2 to 4 describe progressive severity of abnormalities at the GEJ. Grade 2 represents partial disruption and reduced intra-abdominal esophageal length, which is characterized by a hiatal diameter of 1–2 cm and loss of the gastresophageal flap valve. This loss is shown by thinning of the valve lips and lack of the omega shape (L0, D1–2, F−), which serves as a key feature distinguishing AFS grade 2 from grade 1.[14]

AFS grade 3 is characterized by an axial hiatal length of approximately 2 cm or a dilated hiatal aperture measuring 2–3 cm, together with the absence of the flap valve (L0–2, D2–3, F−). Grade 4 shows total disruption of the GEJ, defined by an axial hiatal length exceeding 2 cm or a significantly widened hiatus greater than 3 cm in diameter, with loss of the flap valve (L>2, D>3, F−). Hiatal hernia severity is assessed using the LDF criteria, and classification is based on the most severe parameter identified. To promote consistent use of this system, a color-coded model differentiates normal GEJ anatomy at AFS grade 1 (green) from increasing degrees of hiatal disruption at grade 2 (yellow), grade 3 (orange), and grade 4 (red), as shown in **Figure 2**.[14]

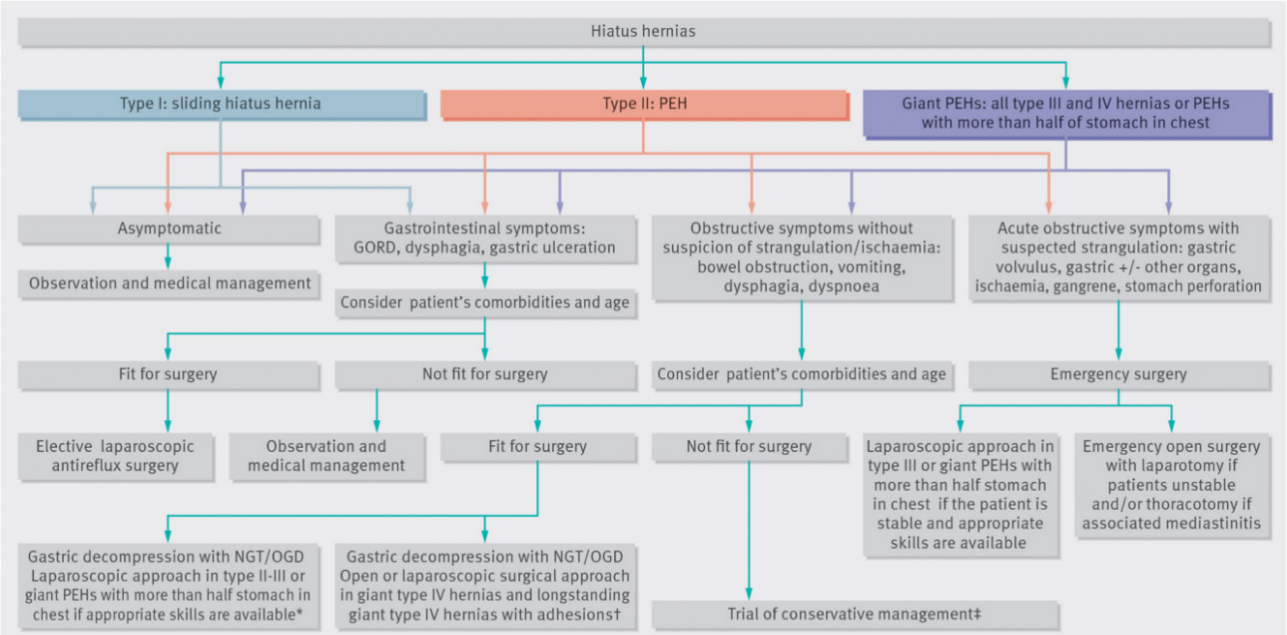
The AFS classification has been shown to identify the presence of GERD and stratify severity based on the degree of hiatal hernia. According to previous studies, AFS grades have been associated with a progressive rise in the prevalence of severe GERD, reported 3.5%, 5.3%, 37%, and 54% for grades 1, 2, 3, and 4, respectively.[15] In comparison, the HILL classification shows substantial overlap between grades 2 and 3, showing the limited ability to clearly discriminate between GERD and non-GERD patients. However, the classification appears to be less effective in evaluating severe GERD. This suggests that the AFS classification can provide superior diagnostic performance compared to the HILL classification.[15]

Studies comparing AFS and HILL classifications based on interobserver variability among endoscopists have also reported lower interobserver variability with the AFS system. This shows improved diagnostic consistency and reproducibility, thereby supporting the reliability of the AFS classification in clinical practice.[16]

TREATMENT

Treatment of a hiatal hernia is often not required in asymptomatic patients. However, paraesophageal hernias are considered for intervention because of the potential risk of complications. In symptomatic cases, management is primarily directed at alleviating GERD through the use of medications that suppress gastric acid production to reduce reflux-related symptoms and prevent esophageal complications. Sliding hiatal hernias can be managed with non-pharmacological measures, including smoking cessation, weight loss, and avoidance of trigger foods and beverages such as alcohol, coffee, mint, and fatty foods. When these measures are insufficient, proton pump inhibitors (PPIs) are recommended as the first-line treatment, due to their superior ability to suppress gastric acid and effectively manage reflux esophagitis and typical GERD symptoms. Histamine-2 receptor blockers and antacids are less effective and are generally used as alternative options when PPIs are not suitable. Therefore, the recommended method is to use the minimum effective PPI dose sufficient to control symptoms. Despite their efficacy, adverse effects are associated with PPIs, which include headache, diarrhea, and a higher risk of gastrointestinal infections, such as infectious gastroenteritis and *Clostridium difficile*–associated colitis. Rare but serious complications include acute interstitial nephritis and profound yet reversible hypomagnesemia. Prolonged use has also been related to osteopenia and small intestinal bacterial overgrowth, although there is limited supporting evidence. Administration of probiotics containing *Lactobacillus acidophilus* and *Bifidobacterium* strains can help reduce symptoms in hiatal hernia.[10,17] Due to the variability in clinical presentation and disorder severity, the treatment method for hiatal hernia should be patient-specific, as shown in **Figure 3**.[18]

Surgical intervention is the only method capable of repositioning herniated organs back into the abdominal cavity and addressing the functional defects related to a hiatal hernia. Currently, laparoscopic fundoplication is considered the standard operative method, comprising several key components. These include mobilization of the distal esophagus, reduction of the associated hiatal hernia, and either partial (Toupet 270°) or complete (Nissen 360°) fundoplication around the esophagus. Despite its effectiveness, current guidelines emphasize that surgical intervention is not



Type I hernias are sliding hiatus hernias, where the gastro-oesophageal junction migrates above the diaphragm. The stomach remains in its usual longitudinal alignment and the fundus remains below the gastro-oesophageal junction
Type II hernias are pure PEHs; the gastro-oesophageal junction remains in its normal anatomical position but a portion of the fundus herniates through the diaphragmatic hiatus adjacent to the oesophagus
Type III hernias are a combination of types I and II, with both the gastro-oesophageal junction and the fundus herniating through the hiatus. The fundus lies above the gastro-oesophageal junction
Type IV hiatus hernias are characterised by the presence of a structure other than the stomach, such as the the omentum, colon, or small bowel, within the hernia sac. More than 95% of hiatus hernias are type I. Type II–IV hernias as a group are referred to as PEHs, and are differentiated from type I hernias by the relative preservation of posterolateral phrenic oesophageal attachments around the gastro-oesophageal

* Reduction of stomach, with or without limited gastric resection if needed, hiatus hernia repair with Nissen funduplication or Nissen/Collis gastroplasty, +/- crural reinforcement with mesh
† Reduction of stomach +/- other organs and hiatal hernia repair with mesh closure and eventual association of Nissen funduplication or Nissen/Collis gastroplasty
‡ Gastric decompression with NGT, resuscitation fluid, OGD for decompression, water soluble contrast medium, laxatives, and medical treatment

Figure 3. Treatment Algorithm of Hiatal Hernia (GORD: Gastro-Oesophageal Reflux Disease; NGT: Nasogastric Tube; OGD: Oesophago-Gastro-Duodenoscopy; PEH: Paraoesophageal Hernia).[18]

necessary for asymptomatic hernia. When shown in paraesophageal hernia, laparoscopic correction can be technically demanding due to significant distortion of normal anatomy. The procedure typically includes full excision of the hernia sac from the mediastinum, mobilization of the esophagus, approximation of the hiatal defect (occasionally reinforced with mesh), and completion of a fundoplication.[10]

A study in Japan reported outcomes of surgical treatment in 25 elderly patients with hiatal hernia, showing that laparoscopic surgery led to faster postoperative recovery and a shorter average hospital stay of 11.9 days, compared with 73.8 days for laparotomy. However, there were no postoperative mortalities observed in any case.[19] Another study from Chile, comprising 121 patients with hiatal hernia who underwent laparoscopic surgery, found that postoperative complications were more common in elderly patients with a longer history of symptoms and type IV cases. There was no postoperative mortality, although one case required reoperation for a type III hiatal hernia with severe esophagitis.[20] A large study including 9,171 cases comparing laparoscopic, open, and robotic-assisted surgical methods showed that laparoscopic and robotic-assisted methods were associated with lower costs, shorter hospital stays, and lower rates of complications and mortality compared with open hiatal hernia repair.[21]

Toupet fundoplication (TF) and Nissen fundoplication (NF) are both effective surgical methods that increase LES pressure by approximately 10 and 12 mmHg, respectively. However, TF is superior in preserving esophageal motility and is associated with fewer early obstructive symptoms compared to NF. These methods significantly improve GERD Health-Related Quality-of-Life (GERD-HRQL) scores, although a greater reduction has been observed in the TF group.[22]

Other studies have shown that TF and NF remain equally effective in controlling reflux symptoms, swallowing function, and patient quality of life over long-term follow-up exceeding 15 years. Specifically, TF shows an advantage during the first postoperative year due to a lower incidence of dysphagia, but no significant differences are observed between the two methods after a decade.[23] Comparative analyses using the DeMeester index have also shown significant reductions at one year in both groups, decreasing from 60.51 ± 28.40 to 11.70 ± 9.65 in TF and from 55.23 ± 25.12 to 11.45 ± 10.20 in NF.[24]

A large comparative study, which comprises 24,488 patients undergoing laparoscopic hiatal hernia repair, evaluated outcomes at 30 days postoperatively and found no significant differences in mortality or morbidity between mesh and non-mesh groups. However, mesh use was associated with longer operative time, extended hospital stay, and higher readmission rates.[25] In a 13-year follow-up study, patients in the mesh group experienced a higher incidence of dysphagia to solid foods, without a reduction in recurrence rates.[26] The expert panel did not issue an evidence-based recommendation supporting or opposing the use of mesh in hiatal hernia repair, as sufficient results are currently unavailable. Therefore, the decision to use mesh should remain a shared decision-making process between the surgeon and the patient.[27]

Magnetic Sphincter Augmentation (MSA) is a minimally invasive procedure for controlling GERD symptoms, including the laparoscopic implantation of 10 or more magnetic beads (LINX™ device), as shown in **Figure 4.** The device is positioned circumferentially around the LES, enhancing sphincter closure and tightness, while allowing expansion of the beads during food passage through the esophagus. This procedure is used to prevent the progression of GERD and can be applied in cases of hiatal hernia.[28,29]

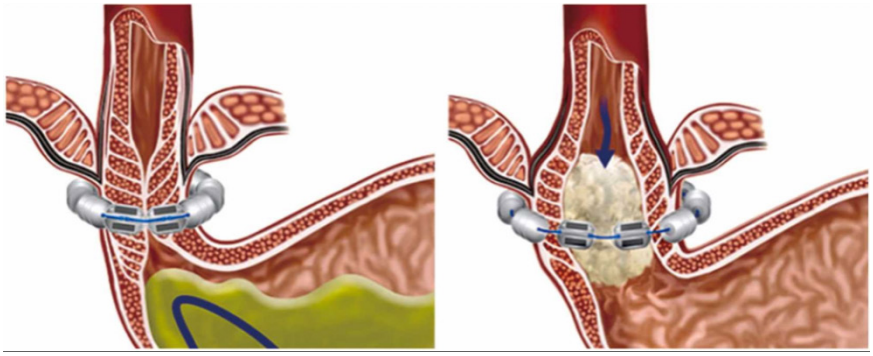


Figure 4. LINX™ Device in Magnetic Sphincter Augmentation[28]

Quality of life in patients with GERD improves after MSA, as shown by a reduction in GERD-HRQL scores from 20.5 to 3.6. In line with the improvement, the need for PPIs in patients with hiatal hernia decreases after undergoing MSA.[30] A previous study reported that the rate of reoperation was higher in patients undergoing MSA compared with those who received NF, at 10% and 4%, respectively. Recurrent hiatal hernia was also observed more frequently in the MSA than in the NF group, with rates of 18% and 7%, respectively.[31]

Transoral endoscopic treatments have been developed to fill the gap between invasive surgical fundoplication and long-term pharmacologic management of GERD. One of these treatments is Transoral Incisionless Fundoplication 2.0 (TIF 2.0), where the gastric fundus is mobilized and wrapped around the distal esophagus, then secured using polypropylene fasteners. The main objective of TIF 2.0 is to achieve endoscopic fundoplication aimed at reconstructing the angle of His. This is accomplished by placing stabilizing T fasteners approximately 2–3 cm above the GEJ, along with the stepwise creation of a 270-degree esophagogastric wrap.[29]

A comparative study evaluating TIF and NF in patients with hiatal hernia found no significant difference in PPI discontinuation rates or recurrence between the two groups. However, TIF was associated with a shorter length of hospital stay, fewer short- and long-term adverse events, and a lower incidence of bloating symptoms at 6-month follow-up compared with NF.[32]

Endoscopic radiofrequency anti-reflux therapy (Stretta) is another procedure that delivers controlled radiofrequency energy to the LES and the gastric cardia, leading to smooth muscle remodeling. This process increases the thickness of the musculature as well as the size and density of smooth muscle fibers within the treated area. Despite these effects, Stretta is not effective in patients with a hiatal hernia larger than 2 cm, significant dysphagia, severe esophagitis (Los Angeles grade C or D), or impaired esophageal peristalsis. Since the procedure does not strengthen the extrinsic barrier of the GEJ, the presence of a hiatal hernia remains an important factor that can reduce its overall efficacy.[33–35]

CONCLUSION

In conclusion, treatment of hiatal hernia is indicated only for symptomatic cases, with PPIs serving as the main pharmacological management. Although medical treatment focuses on symptom control, surgical intervention remains the only method to restore herniated organs to their normal anatomical position. Among various options, laparoscopic fundoplication is the preferred surgical method due to its association with lower postoperative complication rates.

CONFLICT OF INTEREST

The authors declare no conflicts of interest related to this study.

FUNDING

This review did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

AUTHOR CONTRIBUTIONS

DH, BJW, LR, FG, JW, and AW contributed to the conception, design, drafting of the manuscript, data acquisition, literature review, and manuscript revision. All authors approved the final version of the manuscript.

ACKNOWLEDGEMENT

The authors are grateful to the Division of Gastroenterology-Hepatology, Department of Internal Medicine, Faculty of Medicine, Sam Ratulangi University, Manado, Indonesia, for their institutional support during the preparation of this work.

REFERENCES

1. Fuchs KH, Kafetzis I, Hann A, Meining A. Hiatal hernias revisited—a systematic review of definitions, classifications, and applications. Life (Basel). 2024;14(9):1145. doi:10.3390/life14091145.
2. Smith RE, Sharma S, Shahjehan RD. Hiatal hernia [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2026 Jan 23]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK562200/
3. Pawluszewicz P, Wojciak P, Diemieszczyk I, Golaszewski P, Wozniewska P, Hady HR. Hiatal hernia—epidemiology, pathogenesis, diagnosis. Postepy Nauk Med. 2018;31(5):274–278. doi:10.25121/pnm.2018.31.5.274.
4. Kim J, Hiura GT, Oelsner EC, Yin X, Barr RG, Smith BM, et al. Hiatal hernia prevalence and natural history on non-contrast CT in the Multi-Ethnic Study of Atherosclerosis (MESA). BMJ Open Gastroenterol. 2021;8(1):e000565. doi:10.1136/bmjgast-2020-000565.
5. Lye TJY, Ng KR, Tan AWE, Nicholassy NS, Woo SM, Lim EKW, et al. Small hiatal hernia and postprandial reflux after

vertical sleeve gastrectomy: a multiethnic Asian cohort. PLoS One. 2020;15(11):e0241847. doi:10.1371/journal.pone.0241847.

6. Rustiasari UJ, H DR. Histopathological diagnostic criteria of Barrett esophagus and its association with endoscopy findings. J Kedokt Kesehat Indones. 2017;8(1):45–57. doi:10.20885/jkki.vol8.iss1.art7.
7. Hua L, Kohn GP. Minimally invasive surgery: hiatal hernia repair—a narrative review. Ann Esophagus. 2022;5:38. doi:10.21037/aoe-21-12.
8. Siegal SR, Dolan JP, Hunter JG. Modern diagnosis and treatment of hiatal hernias. Langenbecks Arch Surg. 2017;402(8):1145–1151. doi:10.1007/s00423-017-1606-5.
9. Yu HX, Han CS, Xue JR, Han ZF, Xin H. Esophageal hiatal hernia: risk, diagnosis, and management. Expert Rev Gastroenterol Hepatol. 2018;12(4):319–329. doi:10.1080/17474124.2018.1441711.
10. Roman S, Kahrilas PJ. The diagnosis and management of hiatus hernia. BMJ. 2014;349:g6154. doi:10.1136/bmj.g6154.
11. Sfara A, Dumitrascu DL. The management of hiatal hernia: an update on diagnosis and treatment. Med Pharm Rep. 2019;92(4):321–325. doi:10.15386/mpr-1323.
12. Ayyildiz VA, Ozgokce M, Turkoglu S, Dundar I, Durmaz F, Ozkacmaz S, et al. Radiological appearance of hiatal hernias on computed tomography. East J Med. 2022;27(1):11–15. doi:10.5505/EJM.2022.33341.
13. Callaway JP, Vaezi MF. Hiatal and paraesophageal hernias. Clin Gastroenterol Hepatol. 2018;16(6):810–813. doi:10.1016/j.cgh.2017.12.045.
14. Nguyen NT, Thosani NC, Canto MI, Chang K, Lipham J, Abu Dayyeh B, et al. The American Foregut Society white paper on the endoscopic classification of esophagogastric junction integrity. Foregut. 2022;2(4):339–348. doi:10.1177/26345161221126961.
15. Riva CG, Siboni S, De Maron R, Mema J, Milito P, Lovece A, et al. The American Foregut Society endoscopic classification outperforms Hill in phenotyping the esophagogastric junction to guide gastroesophageal reflux disease management. Dis Esophagus. 2026;39(1):doag004. doi:10.1093/dote/doag004.
16. Siboni S, Sozzi M, Lovece A, Milito P, Visaggi P, Ghisa M, et al. The AFS endoscopic classification of esophagogastric junction integrity is superior to the Hill classification in terms of interobserver variability. J Clin Gastroenterol. 2025 Nov 13. doi:10.1097/MCG.0000000000002288.
17. Goudoever V. Esophageal hiatal hernia: a cause of esophageal reflux disease. J Prob Health. 2023;11(1):313. doi:10.35248/2329-8901.23.11.313.
18. Di Saverio S, Lombardi R, Bianchi E, Tugnoli G, Jovine E. An elderly woman with chest pain and constipation. BMJ. 2015;350:h166. doi:10.1136/bmj.h166.
19. Horinishi Y, Shimizu K, Sano C, Ohta R. Surgical interventions in cases of esophageal hiatal hernias among older Japanese adults: a systematic review. Medicina (Kaunas). 2022;58(2):279. doi:10.3390/medicina58020279.
20. Braghetto I, Csendes A, Korn O, Musleh M, Lanzarini E, Saure A, et al. Hiatal hernias: why and how should they be surgically treated? Cir Esp. 2013;91(7):438–443.
21. Hosein S, Carlson T, Flores L, Armijo PR, Oleynikov D. Minimally invasive approach to hiatal hernia repair is superior to open, even in the emergent setting: a large national database analysis. Surg Endosc. 2021;35(1):423–428. doi:10.1007/s00464-020-07404-y.
22. Gadalla EAY, El Serafy ME, Abd El Monaem AH, El Sayed EA, Abdalla AKM. Comparative study between the effect of Nissen fundoplication and Toupet fundoplication on postoperative manometry findings: a randomized controlled trial. Surg Endosc. 2025;39(11):7281–7291. doi:10.1007/s00464-025-12112-6.
23. Analatos A, Hakanson BS, Ansorge C, Lindblad M, Lundell L, Thorell A. Clinical outcomes of a laparoscopic total vs a 270° posterior partial fundoplication in chronic gastroesophageal reflux disease. JAMA Surg. 2022;157(6):473. doi:10.1001/jamasurg.2022.0805.
24. Zhu Q, Sheng N, Wang Z, Xia Y. Comparison of Nissen vs Toupet fundoplication in laparoscopic hiatal hernia repair for gastroesophageal reflux disease with extra-esophageal symptoms. Front Med Technol. 2026;7. doi:10.3389/fmedt.2025.1678192.
25. Wyland C, Parievsky S, Madani R, Richter DM, Elian A, Shebrain S. Is mesh essential in laparoscopic hiatal hernia repair? Analysis of 30-day outcomes from the ACS-NSQIP database over eight years. Front Surg. 2025;12. doi:10.3389/fsurg.2025.1607633.
26. Analatos A, Hakanson BS, Ansorge C, Lindblad M, Lundell L, Thorell A. Hiatal hernia repair with tension-free mesh or crural sutures alone in antireflux surgery. JAMA Surg. 2024;159(1):11. doi:10.1001/jamasurg.2023.4976.
27. Daly S, Kumar SS, Collings AT, Hanna NM, Pandya YK, Kurtz J, et al. SAGES guidelines for the surgical treatment of hiatal hernias. Surg Endosc. 2024;38(9):4765–4775. doi:10.1007/s00464-024-11092-3.
28. Wang Q, Bonavina L. Magnetic surgery for lower esophageal sphincter augmentation: two decades of research and innovation. Magn Med. 2025;1(2):100025. doi:10.1016/j.magmed.2025.100025.
29. Bazerbachi F, Krishnan K, Abu Dayyeh BK. Endoscopic GERD therapy: a primer for the transoral incisionless fundoplication procedure. Gastrointest Endosc. 2019;90:370–383. doi:10.1016/j.gie.2019.05.028.
30. Rona KA, Reynolds J, Schwameis K, Zehetner J, Samakar K, Oh P, et al. Efficacy of magnetic sphincter augmentation in patients with large hiatal hernias. Surg Endosc. 2017;31(5):2096–2102. doi:10.1007/s00464-016-5204-3.
31. Daus KE, Farivar AS, Bograd AJ, White PT, Aye RW, Louie BE. Long-term outcomes of magnetic sphincter augmentation: a comparative study to Nissen fundoplication. Ann Thorac Surg. 2024;118(6):1207–1214. doi:10.1016/j.athoracsur.2024.09.010.
32. Jaruvongvanich VK, Matar R, Reisenauer J, Janu P, Mavrelis P, Ihde G, et al. Hiatal hernia repair with transoral incisionless fundoplication versus Nissen fundoplication for gastroesophageal reflux disease: a retrospective study. Endosc Int Open. 2023;11(1):E11–E18. doi:10.1055/a-1972-9190.
33. Sandhu DS, Fass R. Stretta therapy in the management of gastroesophageal reflux disease (GERD). Ann Esophagus. 2019. doi:10.21037/aoe.2019.01.01.
34. Jung HY. In which situation is endoscopic radiofrequency anti-reflux therapy (Stretta) effective for controlling gastroesophageal reflux symptoms? Clin Endosc. 2021;54:451–452. doi:10.5946/ce.2021.190.
35. Franciosa M, Triadafilopoulos G, Mashimo H. Stretta radiofrequency treatment for GERD: a safe and effective modality. Gastroenterol Res Pract. 2013;2013:783815. doi:10.1155/2013/783815.