REVIEW ARTICLE

CONTENTS

Surgical treatment strategy for Barrett's esophagus as a complication of hiatal hernia

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ABSTRACT

Aim: To assess current approaches to treatment of Barret`s esophagus in patient`s with hiatal hernia.

Materials and Methods: English language search restriction was used. The PubMed, Scopus, Embase, Cochrane, and Google Scholar databases were searched using syntaxes consisting of keywords ("Barret`s esophagus" OR "Hiatal Hernia" OR "Esophageal Adenocarcinoma" OR "Esophagitis").

Conclusions: Barrett's esophagus is a precancerous condition, prone to causing esophageal adenocarcinoma. Currently, approaches to treatment and monitoring in different European countries and worldwide vary for Barrett's Esophagus. Nevertheless, most current guidelines favour endoscopic therapy as a first line of treatment of Barrett`s esophagus in patients with hiatal hernia.

KEY WORDS: Barrett`s esophagus, hiatal hernia, endoscopy

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INTRODUCTION

Barrett's esophagus (BE) is a precancerous condition characterized by the replacement of normal squamous epithelium in the esophagus with specialized intestinal metaplasia. This transformation significantly increases the risk of developing esophageal adenocarcinoma. One of the key factors contributing to the development and progression of Barrett's esophagus is gastroesophageal reflux disease (GERD), which is often associated with hiatal hernia. Hiatal hernia can exacerbate acid reflux, leading to chronic esophageal irritation and an increased likelihood of Barrett's esophagus.

AIM

Evaluation of current approaches to the treatment of Barrett's esophagus in patients with hiatal hernia.

MATERIALS AND METHODS

The PubMed, Scopus, Embase, Cochrane, and Google Scholar databases were searched using syntaxes consisting of keywords ("Barret`s esophagus" OR "Hiatal Hernia" OR "Esophageal Adenocarcinoma" OR "Esophagitis"). English language search restriction was used.

REVIEW AND DISCUSSION

Barrett's Esophagus (BE) is a precancerous condition, prone to causing esophageal adenocarcinoma. Currently, approaches to treatment and monitoring in different European countries and worldwide vary for Barrett's Esophagus.

The diagnosis of Barrett's Esophagus is established when the distal part of the esophagus, which is normally lined with stratified squamous non-keratinized epithelium, is lined with columnar epithelium (CLE) more than 1 cm above the esophagogastric junction and may contain specialized intestinal metaplasia upon histological examination [1].

Barrett's Esophagus is named after the British surgeon Norman Barrett, who in 1950 published his foundational article 'Chronic Peptic Ulcer Disease of the Esophagus and Esophagitis', describing an esophagus with metaplasia of columnar cells. However, the first description of this pathology was made by Wilder Tileston, who reported three cases of "peptic ulcer disease of the esophagus" in 1906, where he described the histological structure of the esophageal ulcer and the adjacent epithelium resembling a gastric ulcer with adjacent columnar epithelium.

Over the next four decades, there were widespread discrepancies regarding the histological structure of

the mucosa of the distal third of the esophagus. Some authors argued that ulcers in the distal esophagus were gastric ulcers located intrathoracically in patients with a congenitally short esophagus. Barrett supported this theory in his work in 1950.

In 1953, Ellison and Johnston published a convincing article rejecting Barrett's hypothesis and disputing the possibility of intrathoracic gastric placement, since the latter:

1. Lacked an external serous coat;
2. The muscular structure was identical to the esophagus;
3. Its mucosa was composed of columnar epithelium with areas of squamous epithelium;
4. The epithelium did not include parietal cells characteristic of the stomach.
5. There were mucosal glands characteristic of the esophagus.

This paper led N. Barrett to revise his previous statements and publish an article in 1957, in which he described this area as "The lower esophagus lined with columnar epithelium." [2].

Between 1960 and the mid-1970s, there were various histological descriptions of subtypes of columnar tissue in the distal esophagus, including transitional epithelium cardio-gastric type, gastric-fundal type, and intestinal epithelium with goblet cells.

This histological issue was resolved in 1976 by Paul and co-authors, who performed biopsies on 11 patients with BE and identified a histological spectrum that could include: columnar epithelium containing villi and goblet cells which is now known as intestinal metaplasia, sometimes referred to as specialized intestinal metaplasia, with subsequent transitional epithelium, and atrophic fundic gastric epithelium with basal and parietal cells [2].

In the 1980s, it was established that gastroesophageal reflux disease (GERD) and the presence of a hiatal hernia were risk factors for BE.

To avoid errors, diagnostic criteria for BE were established by Skinner and co-authors, who proposed a diagnostic criterion—the presence of a metaplastic area of at least 3 cm in length.

By the mid-1980s, the link between BE and esophageal adenocarcinoma was firmly established, and it was proven that intestinal metaplasia has a mosaic distribution and a significant predisposition to the development of dysplasia, which led to the recognition of intestinal metaplasia as a defining feature of BE.

In the mid-1990s, Spechler and co-authors challenged the widely accepted practice of performing biopsies in BE over 3 cm, as they demonstrated that in 18% of patients with endoscopic signs of BE, with metaplasia less than 3 cm in length, goblet cells were still present in the mucosa. Moreover, reports of esophageal adenocarcinoma developing on the background of BE with segment sizes less than 3 cm were also noted. Currently, the classification of BE into short and long segments has proven important for diagnostic criteria and treatment strategy [2]. Histological criteria for the diagnosis of BE remain a contentious issue. The American College of Gastroenterology (ACG) considers biopsy to confirm intestinal metaplasia as a necessary condition for diagnosing Barrett's Esophagus [3]. However, the British Society of Gastroenterology (BSG) in its guidelines states that the diagnosis of Barrett's Esophagus can be made with the presence of visible columnar epithelium (gastric type) and confirmation with biopsy as an intestinal metaplasia is not a mandatory condition for the diagnosis of BE [4].

Japanese scientists consider the diagnosis of Barrett's Esophagus confirmed with the presence of gastric-type columnar epithelium, based on studies confirming the possibility of developing esophageal adenocarcinoma against the background of BE without intestinal metaplasia [5,6].

The International Group on BE and Esophageal Cancer (BOBCAT) defines BE as the presence of columnar epithelium in the lower third of the esophagus but specifies that it must be noted whether intestinal metaplasia is present above the esophagogastric junction [7].

The differences in recommendations depend on the differential risk of malignant transformation of columnar epithelium with and without intestinal metaplasia. Emphasis on intestinal metaplasia as a defining feature of Barrett's esophagus (BE) is based on an increasing number of studies that have shown a stronger association between BE with intestinal metaplasia and adenocarcinoma than BE without intestinal metaplasia.

A study of 8522 patients showed that the risk of malignant progression of intestinal metaplasia was higher compared to columnar cell metaplasia of the stomach (0.38% per year vs. 0.07% per year) [8]. A recent detailed genomic analysis comparing BE with intestinal metaplasia and BE without intestinal metaplasia in 45 patients reported higher mutation frequencies in cancer-related genes such as CDKN2A, WWOX, c-MYC, and GATA6 in patients with BE featuring intestinal metaplasia [9]. However, other studies did not confirm these findings. A retrospective analysis of 688 patients showed no significant difference in cancer risk between patients with BE and intestinal metaplasia and BE without intestinal metaplasia [10]. Several studies have also highlighted the detection of esophageal cancer in the context of columnar cell epithelium without the presence of goblet cells [11].

The biopsy collection protocol developed by Seattle, which includes 4-quadrant biopsies every 2 cm along the length of BE, is a reliable method for obtaining appropriate material for diagnosing BE. However, this protocol is not always strictly followed in clinical practice.

In a comparative study designed to determine the optimal number of biopsies for detecting intestinal metaplasia, researchers showed that the diagnostic value of detecting intestinal metaplasia increases with the number of biopsies. When the number of biopsies increased from 4 to 8 to 16, the diagnostic accuracy for intestinal metaplasia increased from 34.7% to 67.9% and to 100%, respectively [12]. These findings led to the latest recommendations from the American College of Gastroenterology (ACG) to obtain at least 8 random biopsies when BE is suspected during diagnostic endoscopy. Obtaining 16 biopsy samples achieves high accuracy, but this would not only take a lot of time for the procedure but could also increase the risk of bleeding after the biopsy and raise the cost of biopsy processing [13].

Dysplasia is a biomarker of cancer risk in BE, classified according to the Vienna classification [14]. Since the assessment of dysplasia affects treatment strategies, most recommendations require that the diagnosis of dysplasia be confirmed histologically by two pathologists.

The use of alternative biomarkers, particularly the expression of p53 protein, has become a valuable addition for improving BE risk stratification. Sikkema and colleagues showed that overexpression of p53 was a stronger predictor of progression to high-grade dysplasia (HGD) or esophageal adenocarcinoma, regardless of histology, compared to diagnosing low-grade dysplasia (LGD) [15].

In an analysis of over 12,000 biopsies in 635 BE patients, it was shown that abnormal p53 expression, either overexpression or loss of expression - increased the risk of cancer, and the risk was higher for BE with loss of p53 expression (14.0) compared to BE with overexpression of p53 (5.6) [16]. Furthermore, immunohistochemical studies for p53 detection have shown good interobserver reliability. Although immunohistochemistry for p53 has not yet been widely implemented in clinical practice, its use could allow for a more accurate risk group assessment for more intensive monitoring of these patients [13].

Barrett's esophagus is diagnosed in 7% to 10% of individuals with chronic gastroesophageal reflux disease (GERD), and it is estimated to be present in 1% to 2% of the general adult population [3, 4]. In chronic GERD, 5-15% of the esophageal mucosa may transform normal squamous epithelium to columnar mucous epithelium. The development of Barrett's esophagus leads to a tenfold increase in the risk of esophageal adenocarcinoma compared to the general population [17].

The incidence of esophageal adenocarcinoma in non-dysplastic Barrett's esophagus is approximately 1 case per 300 patients per year [18].

Esophageal adenocarcinoma continues to be one of the fastest-growing cancers in Western populations, and this correlates with the rising mortality from this disease [19, 20].

Survival of patients with esophageal adenocarcinoma correlates with the disease stage. The 5-year survival rate is around 20% in patients with locally advanced disease and less than 5% for those with distant involvement. The low survival rate for patients with advanced esophageal adenocarcinoma highlights the necessity of early detection. Endoscopic surveillance for BE has become the cornerstone for preventing esophageal adenocarcinoma, especially in Western countries, and this trend has accelerated with the advent of visualization technologies and endoscopic treatment methods [21, 22].

BE can be classified as short-segment or long-segment depending on the extent of metaplastic changes in the esophagus observed during endoscopic examination. If intestinal metaplasia extends less than 3 cm above the gastroesophageal junction, it is considered short-segment BE, while more than 3 cm indicates long-segment BE [23].

Risk factors for BE include white race, male gender, age over 50 years, obesity, and persistent gastroesophageal reflux [3]. The presence of a hiatal hernia is also associated with the development of BE [4].

Hiatal hernia (HH) is a common condition characterized by the displacement of the esophagogastric junction, stomach, or other abdominal organs through an enlarged diaphragmatic hiatus into the chest cavity. According to the widely accepted anatomical classification, HH is divided into four types. Type I hernias are the most common, with a frequency of up to 90% of all HH cases. Types II - IV are classified as paraesophageal hernias, with type III being the most common (about 90%) [24, 25, 26]. The prevalence of HH in the population can range from 3% to 30%, and in individuals over 50 years of age, it may reach up to 50%, according to Mittal's data.

The frequency of HH diagnosis depends on the quality of diagnostics, geographic features, and the ethnic composition of the population. The frequency of symptomatic cases of HH is linked to the diagnosis of GERD, as these conditions are closely correlated. GERD, which most commonly manifests as heartburn and acid regurgitation, affects 18-28% of the population. In Ukraine, statistical registration of GERD started in 2009,

Table 1. Endoscopic classifications for the diagnosis of lesions in patient`s with Barret`s esophagus

	BING classification	JES classification for BE
Non – dysplasia	Mucosal pattern: regular Vascular pattern: regular	Mucosal pattern: regular Vascular pattern: regular flat pattern
Dysplasia	Mucosal pattern: absent or irregular Vascular pattern: irregular	Mucosal pattern: irregular Vascular pattern: irregular
Diagnostic accuracy	Sensitivity 80% Specificity 88%	Sensitivity 87% Specificity 97%
Reproducibility	k = 0.68	k = 0.77

with the primary incidence rate being 10 cases per 1000 population and a tendency to increase. The prevalence of GERD is approximately 30% (25.1% in men and 39.1% in women) [4]. More than 80% of patients with HH have endoscopic signs of esophagitis.

Among patients who underwent endoscopy for various indications, the connection between HH and reflux esophagitis is significant in different countries, regardless of the prevalence of HH. HH statistics are not maintained in Ukraine, and data are aggregated with the total number of abdominal wall hernias. Untimely detection and treatment of HH can lead to chronic anemia, acute gastric bleeding, esophageal strictures, perforation, and subsequent development of ulcers and erosions of the gastric mucosa, acute gastric incarceration, and its necrosis [17]. The presence of HH leads to anatomical and functional disturbances at the esophagogastric junction, causing the reflux of gastric contents into the esophagus. This includes gastric secretions such as hydrochloric acid and pepsin, as well as pancreatic enzymes and bile. Chronic exposure to these substances is considered a factor in the development of BE [27,28]. Observational and experimental studies have demonstrated that acid and duodenogastroesophageal reflux have a synergistic effect and increase the risk of BE development [29,30]. A meta-analysis showed a strong association between HH and BE, which is linked to dysplastic changes. It is plausible that HH increases the risk of BE by increasing exposure of the esophageal mucosa to gastric contents such as acid and bile, making BE more common in individuals with HH. The role of gastric contents or gastric contents and bile in damaging the esophageal mucosa, leading to metaplasia, is supported by animal models [23].

In a study involving 118,750 BE patients, 24,030 of whom had HH, a connection was found between the size of the HH and its complications. Larger hernias were proportionally associated with an increased risk of BE development, its extent, and the occurrence of dysplasia and adenocarcinoma [31].

Endoscopic surveillance for Barrett's esophagus has become the foundation for the prevention and early detection of esophageal adenocarcinoma. Surveillance typically includes periodic endoscopy of the upper gastrointestinal tract with biopsies of suspicious areas and random biopsies from four quadrants. However, targeted biopsies using narrow-band imaging can detect more dysplastic areas, thereby reducing the number of necessary biopsies. Several specific pit structures and vascular patterns characteristic of Barrett's esophagus have been described, but the proposed criteria are complex and varied. Recently, simpler classifications have been developed focusing on differentiating between dysplasia and the absence of dysplasia. One such classification is the Japanese Society of Esophagus Classification, which identifies correct and incorrect structures in terms of mucosal and vascular patterns (Table 1).

The depth of cancer invasion is diagnosed using endoscopic ultrasound (EUS); however, a meta-analysis of EUS diagnostics for superficial esophageal adenocarcinoma showed favorable combined data for mucosal-stage cancer but unsatisfactory results for adenocarcinoma at the gastroesophageal junction. Endoscopic resection has recently been proposed as a more accurate method for assessing the depth of invasion compared to EUS. European guidelines describe endoscopic resection as therapeutic for well- or moderately differentiated mucosal cancer without lymphovascular invasion, and these criteria can be extended to lesions invading the submucosa (≤ 500 μm) and tumors smaller than 3 cm. These criteria were confirmed by a recent study in Japan [32].

The European Association of Endoscopists recommends changing surveillance intervals based on the length of the Barrett's esophagus (BE). For patients with an irregular Z-line or the presence of columnar metaplasia without dysplasia in the esophagus less than 1 cm, routine biopsies or endoscopic surveillance are not recommended. For BE ≥ 1 cm and <3 cm, surveillance should be conducted at intervals of 5 years. For BE ≥ 3 cm and <10 cm, the interval for endoscopic surveillance should be 3 years. Patients with BE of a maximum size ≥ 10 cm should be referred to a specialized BE center [33].

Endoscopic screening for BE is not recommended. However, screening can be considered for patients with

a family history of BE and prolonged GERD symptoms with risk factors**.**

High-definition endoscopy (endoscope, processor, and screen) is recommended for endoscopic surveillance of BE. It is recommended to use virtual chromoendoscopy (NBI mode) and dye spraying - acetic acid is the only dye-based chromoendoscopy method that meets ASGE PIVI thresholds [34].

Endoscopic examination of patients with BE should include:

1. The degree of BE using Prague criteria: circumferential extent (C), maximal extent (M), and any individual areas proximal to the maximal extent.
2. A description of the location: in cm from the teeth and clock face orientation of any visible abnormalities within the metaplasia.
3. Presence or absence of erosive esophagitis according to the Los Angeles classification.
4. The location of biopsies taken from the Barrett's segment: number of biopsies and location in cm from the teeth.

Biopsy sampling is performed according to the Seattle protocol and includes 4 points around the circumference of the esophagus every 2 cm, as well as a biopsy from the proximal point of metaplasia and material sampling from suspicious areas of the mucosa with disrupted pit and vascular patterns. If Barrett's esophagus with low-grade dysplasia (LGD) is found, endoscopic ablation should be proposed. In the presence of visible lesions (nodular lesions, suspicion of adenocarcinoma), the area should be removed by endoscopic mucosal resection (EMR) or endoscopic submucosal dissection (ESD) with further histological confirmation.

If high-grade dysplasia (HGD) is found during random biopsy, endoscopic ablation of BE or resection/dissection of the pathological area is recommended [35].

If drug control of gastroesophageal reflux disease (GERD) is not possible in patients, an anti-reflux surgery should be considered for patients with BE to prevent further neoplastic progression [3,33].

The American Society for Gastrointestinal Endoscopy (ASGE), the British Society of Gastroenterology (BSG), and the European Society of Gastrointestinal Endoscopy (ESGE) guidelines do not comment on the presence of GERD in patients with BE. [22,31,45] When it comes to choosing the optimal treatment strategy for patients with GERD, conservative treatment, particularly proton pump inhibitors (PPIs), provides only temporary symptomatic relief and cannot fully prevent complications. Clinical studies show a significant advantage of laparoscopic surgery over conservative treatment when compared for symptoms and quality of life after treatment.[17] Laparoscopic anti-reflux surgery is safe option with a low complication rate. The main aim of GERD treatment is symptom control, which improves quality of life. Results from laparoscopic Nissen fundoplication have proven effective in relieving typical GERD symptoms. According to a study of patients who underwent laparoscopic Nissen fundoplication (37% of them had BE) 87% considered themselves completely healthy, and another 11% reported noticeable improvement in symptoms with an average follow-up of 2 years. For patients with BE undergoing anti-reflux surgery, symptom control was similar. In a cohort of 85 patients with a follow-up period of 5 years, 77% considered themselves completely healthy, and another 22% showed significant improvement in symptoms. A third study with 215 patients followed for 8 years after fundoplication showed that 86% of patients had fully controlled heartburn and regurgitation. These studies show that laparoscopic anti-reflux surgery leads to effective symptom control of GERD in most patients with BE [37].

Using proton pump inhibitors (PPIs) in comparison to no therapy reduces the progression of BE to dysplasia or esophageal adenocarcinoma. Although there is no evidence in highly selective prospective studies, there is scientific plausibility to this claim; preventing injury is the main preventive factor for mutations and neoplasms. Cohort studies demonstrate that PPI use reduced the development of neoplasia. Systematic reviews report a strong negative correlation between PPI use and the risk of severe dysplasia or esophageal adenocarcinoma in patients with BE [7].

Anti-reflux surgical interventions offer an alternative to PPIs in the treatment of GERD, as abnormal gastroesophageal and duodeno-gastroesophageal reflux are prevented.

Performing anti-reflux surgery has advantages over PPI use, as it prevents the entry of duodenal content as well as non-acid gastric content such as pepsin into the esophagus, which are irritants not alleviated by PPIs[37].

The risk of dysplasia or adenocarcinoma progression in BE is similar when comparing medical therapy with fundoplication.

Surgical treatment of reflux in patients with GERD, with or without BE, can provide long-term symptom control and esophageal pH control. [38] Some cohort studies suggest that effective anti-reflux surgery may reduce the risk of BE progression. [39,40] However, a study comparing treatment and monitoring of 101 patients found no significant difference in the development of severe dysplasia in BE when comparing medical therapy and fundoplication after a median follow-up of 5 and 6 years, respectively [41]. A meta-analysis comparing anti-reflux surgery with PPI use in patients

with BE showed similar results in terms of progression to dysplasia or cancer [42]. Thus, performing anti-reflux surgery is not an anti-cancer measure.

In several studies, it has been proven that only surgical anti-reflux intervention without endoscopic ablation of BE does not prevent BE progression but eliminates the need for antisecretory drugs [7,43,44].

CONCLUSIONS

The approach to BE treatment has significantly evolved over the last twenty years. Esophagectomy and esophageal resection were the only options for the surgical treatment of high-grade dysplasia and esophageal adenocarcinoma; however, with significant technical advancements, endoscopic therapy has become the main method of BE treatment.

In patients with dysplastic BE or intramucosal carcinoma, endoscopic surgical interventions such as ablation and mucosal resection have become the standard of care, replacing esophagectomy as the best treatment option in most cases. These endoscopic methods are associated with reduced morbidity and mortality, fewer complications, and improved long-term quality of life compared to esophageal resection.

The choice of ablation method is also subject to discussion in patients with GERD, as the lower third of the esophagus is usually dilated, and the diameter of the esophagus may vary. Radiofrequency balloon ablation may produce poorer results and increase the risk of recurrence, requiring more sessions and increasing the risk of dysplasia.

Argon plasma ablation (APA) is a well-known technique in gastrointestinal endoscopy with various indications, such as thermal ablation of the mucosa in BE, treatment of vascular malformations, removal of BE segments after endoscopic resection, endoscopic hemostasis, and others.

A relatively newer technology, radiofrequency ablation (RFA), is now often the method of choice compared to traditional APA, especially for long-segment BE. However, there are not enough studies comparing APA and RFA for an accurate comparison of techniques.

Recent studies show several advantages of hybrid radiofrequency ablation. When performing ablation in BE using the traditional APA method, the risk of stricture formation may reach 12-15%, while with RFA, it is 5%. Therefore, there is still a need for technical improvements in BE ablation techniques. The ideal technique would result in complete ablation of BE while minimizing the risk of complications. One possible approach to reduce the number of strictures is submucosal injection of fluid before thermal ablation (hybrid ablation). This prevents damage to the deeper layers of the esophageal wall. This technique combines submucosal injection of isotonic saline with standard APA in one procedure. However, the thermal impact on the esophageal wall, uniformity of ablation, and penetration depth, depending on the solutions used, require further research. Preliminary data from studies using hybrid APA show significant advantages compared to APA and are comparable in effectiveness and safety to RFA.

The issue of treating Barrett's esophagus combined with a diaphragmatic hiatal hernia remains unresolved and requires further study. Based on current global literature, hybrid argon plasma coagulation is an effective method for treating Barrett's esophagus with low complication rates and good long-term outcomes.

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CONFLICT OF INTEREST

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