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Chapter
The Role of Proton Pump Inhibitors in the Treatment of Barrett’s Esophagus
Zaim Gashi, Arjeta Gashi and Fadil Sherifi

Abstract

Barrett’s esophagus (BE), as a more frequent complication of gastroesophageal reflux disease, is a metaplastic condition in which the normal squamous epithelium of the esophagus is replaced by specialized intestinal metaplastic epithelium, and that, in about 10% of patients with gastroesophageal reflux disease (GERD) and the main condition for dysplasia and adenocarcinoma. The incidence of adenocarcinoma of the cardia is rapidly increasing at a rate that exceeds that of any other cancer. Recently, acid suppression with proton pump inhibitors (PPIs) has become the cornerstone of treatment for patients with BE. Many worldwide investigations showed that PPI is effective in the regression of BE with low-grade dysplasia and especially for the regression of intestinal metaplasia, incomplete or complete, for long-term use of these medicaments. This chapter reviews the specific endpoints of such treatment, included and our results for this dilemma.

Keywords: Barrett’s esophagus, low-grade dysplasia, proton pump inhibitor, regression, gastroesophageal reflux disease (GERD)

1. Introduction

Gastroesophageal reflux disease (GERD) is accepted as a cornerstone etiological factor for Barrett’s esophagus (BE), which is a major predisposition to esophageal adenocarcinoma.

GERD is a precursor to BE, which represents intestinal metaplasia (IM), [1] and the latter is most likely a precursor to esophageal cancer. Progression from Barrett’s to dysplasia is estimated to be in about 20% of cases [2]. Chronic heartburn can progress to Barrett’s, so EGD (esophagogastroduodenoscopy) is recommended every 5 years for these cases, but also for cases taking medication for chronic GERD [3].

BE is a condition in which there is an abnormal change (metaplastic tissue) with the replacement of multilayered epithelial cells, under the long-term influence of gastroesophageal reflux, with specialized intestinal cells that are present only in the small and large intestines. This change is considered to be a precursor of distal malignancy of the esophagus as it is associated with a high incidence of further transition to adenocarcinoma of the esophagus, with a highly malignant nature [1, 2].

BE is diagnosed with endoscopy: we encounter inflammatory, erosive, ulcerative changes up to narrowing of the distal lumen of the esophagus, classified according to
Los Angeles A-D, followed by microscopic examination of the tissue from the affected area from the biopsies obtained. BE cells are classified into four categories: nondysplastic (such as incomplete and complete intestinal metaplasia), low-grade dysplasia, high-grade dysplasia, and carcinoma.

Up to the level of low dysplasia, the changes can be treated with PPI, including the fundoplication according to Nissen. High-grade dysplasia and early stages of adenocarcinoma can be treated with endoscopic resection or radiofrequency ablation [1]. Later stages of adenocarcinoma can be treated by surgical resection. Nondysplastic or low-grade (LGD) cases are managed by annual surveillance with endoscopy or treatment with radiofrequency ablation. It should be borne in mind that in cases with high-grade dysplasia (HGD), the risk of developing cancer can be 10% per patient-year or more, so treatment is needed as soon as possible [4].

A greater extent of dysplasia has a significantly higher risk of cancer as well as the presence of an endoscopic abnormality [5].

BE is thought to be an adaptation to the chronic exposure of acid reflux, but also of another nature, in the esophagus for a long time [6].

2. Pathophysiology

BE reflects chronic chemical inflammation, as a consequence of persistent gastroesophageal reflux. Basically, it is the acidic content of the stomach, bile and small intestines, and pancreas as a potential cause of reflux changes. From this reflux, different cells react, including stem cells that express HOXA13, which are characterized by distal (intestinal) characteristics and compete with normal squamous cells [7].

Figure 1. Histopathology of Barrett's esophagus, showing intestinalized epithelium with goblet cells, as opposed to normal stratified squamous epithelium of the esophagus, and pseudostratified columnar epithelium of the fundus of the stomach. The submucosa displays an infiltrate including lymphocytes and plasma cells, constituting an underlying chronic inflammation. The area between the stratified and the intestinalized epithelium displays reactive changes, but there is no secondary dysplasia in this case. H&E stain [11].
This explains the participation of HER2/neu (also called ERBB2) and overexpressed (lineage-dependent) cancer cells during the process of carcinogenesis, and the efficacy of targeted therapy against the Her-2 receptor with Trastuzumab (Herceptin) in the treatment of adenocarcinomas in the gastroesophageal junction.

It cannot be determined which of the patients with reflux will develop BE later. While chronic heartburn affects the development of BE, researchers have not observed a strong association between the severity of reflux and the development of BE. But there was also the phenomenon that people with BE have no symptoms of heartburn at all.

Patients with bulimia, an eating disorder, are more likely to develop BE because bulimia can cause severe acid reflux and because it damages the epithelial cells in the esophagus to a large extent, disrupting the so-called “tight junction.” between the mucous cells [8, 9].

The very act of bile acids entering the esophagus can be an important factor in carcinogenesis [10]. Chronic patients with GERD and BE are exposed to high concentrations of deoxycholic acid, which has cytotoxic effects and can cause DNA damage (Figure 1) [12, 13].

3. Diagnosis

For the diagnosis of GERD and BE, in addition to the relevant clinical data, the macroscopic view during the endoscopy and the microscopic examination after biopsies have been taken are also necessary. In non-dysplastic Barrett’s, goblet cells and specialized intestinal cells are characteristic, which have replaced the previous multilayered epithelium. Of course, this is the body’s initial protective reaction to the reflux content, but it does not withstand time, following BE with a tendency to fail to turn into adenocarcinoma (Figures 2 and 3).
4. Management

BE is not always associated with dysplasia. According to the latest recommendations, if a patient with BE is diagnosed and if the last two endoscopic examinations with biopsy have confirmed the absence of dysplasia, then the patient should have the next endoscopy within 3 years [3, 10, 14].

The risk of malignancy is highest in the United States in Caucasian men over 50 years of age with more than 5 years of symptoms. Although watchful waiting is preferred in cases of BE, for cases with dysplasia, balloon-based radiofrequency ablation, invented by Ganz, Stern, and Zelickson in 1999, is a new treatment modality for the treatment of BE and dysplasia and has been the subject of numerous published clinical trials. The findings demonstrate radiofrequency ablation has an efficacy of 90% or greater with respect to complete clearance of BE and dysplasia with the durability of up to 5 years and a favorable safety profile [15–18].

The results of antireflux surgery, specifically fundoplication, have not been proven to prevent esophageal cancer. Proton pump inhibitors have been shown to be effective in limiting the progression of esophageal cancer. Laser treatment is used in severe dysplasia, while open malignancy may require surgery, radiation therapy, or systemic chemotherapy. A recent 5-year study randomly showed that photodynamic therapy using photofrin is statistically more effective in eliminating dysplastic foci than the use of a proton pump inhibitor alone [19].

The heterogeneous nature of Barrett’s explains the wide spectrum of the degree of mutational overlap between adjacent BE and esophageal adenocarcinoma [20].

Anti-reflux surgery (ARS), namely laparoscopic fundoplication, is the last step in GERD management. Its objectives are LES, basal pressure increase and hiatal repair [21].
Recent studies show that nonsteroidal anti-inflammatory drugs (NSAIDS), such as aspirin, have shown evidence of preventing esophageal cancer in people with BE [22, 23]. However, none of these studies have provided reliable evidence for the effect of these drugs in the prevention of esophageal adenocarcinoma in the field of BE.

BE is thought to be the result of esophageal epithelium in response to damage. The development of BE is a consequence of long-term GERD. Barrett’s epithelium, due to its specific histological features, can be expected to be more resistant to aggressive acidic stomach contents. Diagnosis of Barrett’s patients always requires histological confirmation to allow better monitoring of patients, according to the degree of change in Barrett’s patients.

<table>
<thead>
<tr>
<th>HP type</th>
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<tr>
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<tr>
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<td>HGD</td>
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<tr>
<td>Total</td>
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Table 1.
Structure of patients with BE at the beginning of the study.

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>SSBE</th>
<th>LSBE</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression</td>
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<td>%</td>
<td>N</td>
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<tr>
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<tr>
<td>Progression</td>
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</tr>
<tr>
<td>Total</td>
<td>39</td>
<td>100.0</td>
<td>50</td>
</tr>
</tbody>
</table>

$Progression / Other: Z = -2.66, P = 0.0078$

$SSBE: D_{\text{max}} = 0.32 > D (39;0.05) = 0.22$ and $P < 0.05$.  
$D_{\text{max}} = 0.32 > D (39;0.01) = 0.26$ and $P < 0.01$.  
$LSBE: D_{\text{max}} = 0.06 < D (11;0.05) = 0.39$ and $P > 0.05$.  

Table 2.
Evaluation of patients with BE after 2 years of treatment.

$Evaluation$ $SSBE$ $LSBE$ $TOTAL$

<table>
<thead>
<tr>
<th>N</th>
<th>%</th>
<th>N</th>
<th>%</th>
<th>N</th>
<th>%</th>
</tr>
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<td>2.6</td>
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<td>100.0</td>
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</tr>
</tbody>
</table>

$Table 3.$
Evaluation of patients with BE by endoscopic type.
The PPI class is the most potent type of acid suppression therapy. PPIs are replaced by benzimidazoles that continuously bind H⁺K⁺ATP as the final step in gastric acid secretion. Group members include omeprazole, lansoprazole, pantoprazole, rabeprazole, and esomeprazole. Standard doses of each type of drug may have a similar inhibitory effect. Omeprazole is the longest documented agent, while newer agents, rabeprazole and pantoprazole, have less interaction than both with the cytochrome P450 metabolism. Several studies have shown the superiority of PPI over H2RAs in the treatment of reflux esophagitis.

Of the 50 patients with BE according to histopathological type, 40 or 80.0% were IM, 10 or 20% were LGD and there was no case of HGD (Table 1).

Out of the 40 IM patients included in our study, after 2 years of treatment, only 20 patients or 50.0% had IM, 4 or 10.0% had LGD and 16 patients or 40.0% had NERD. Of the 10 patients with LGD after 2 years of treatment, only 5 patients or 50.0% had LGD, 2 patients or 20.0% had IM, and 3 patients or 30.0% had NERD (non-erosive reflux disease) (Table 2).

Patients of the endoscopic type Long segment of Barrett’s esophagus (LSBE) have a more frequent progression of 27.3% compared with 2.6% to patients of the endoscopic type Short Segment of Barrett’s esophagus (SSBE) 2.6% difference with significant statistical significance ($Z = -2.66, P = 0.0078$), (Table 3).

With the Kolmogor-Smirn test, we confirmed a statistically significant difference in the regressions of changes in BE, when we have to do with SSBE ($P < 0.05$ and 0.01), but also not a significant difference in the evolution of changes in BE, progression, or regression, when we have to do with LSBE ($P > 0.05$) (Figure 4).

The correlation of histopathological type and disease regression in patients with BE, in our study, did not result in a significant difference (Fisher Exact test, $P = 0.487$). From the group with intestinal metaplasia, 40.0% of patients and 50.0% of patients in the LGD (low-grade dysplasia) group had regression (Figure 5, Table 4).

Of the 70 patients with GERD regression, we had 40 or 57.1%, and of the 50 patients with BE regression we had 21 or 42.0% difference without any statistically significant value ($P = 0.138$), 34.3% of patients with GERD was stable and 50.0% of patients with BE without significant difference ($P = 0.05$), and we had progression in

![Figure 4](https://www.intechopen.com)

**Figure 4.** Regression in patients with BE by endoscopic type [24].
8.6% of patients with GERD and 8.0% of patients with BE without significant difference (P = 1.00).

Also with the Fisher test, we did not get a significant difference between the groups (P = 1.00) according to the degree of progression.

However, there is a significant intra-group difference between the two groups, where patients with regression and stable changes after IPP treatment visibly dominate over those with progression. The test was performed with the Colmogar–Smirn test for one sample.

After treatment of patients with PPI, there was more regression of the disease in patients with GERD than in those with BE (Table 5).

### 4.1 Progression of erosive esophagitis to BE

Eighty-three patients (54% male, median age 59 years) with mild esophagitis were treated with continuous PPIs and cisapride at doses sufficient to control symptoms (Table 3) [25]. After 2 years, during the second “follow-up” endoscopy, 12 (15%) had developed BE histologically confirmed in the biopsies taken. Of these patients, nine
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had short-segment disease (SSBE<3 cm) and the other three had long-segment BE (LSBE>3 cm).

Of great importance was the development of lower esophageal sphincter pressures (LOSPs) patients who developed BE, had significantly reduced LOSP compared to those who had not progressed, but their age and gender were not stated.

In another study by Isolauri et al. [26] 6 (or 12%) of 50 medically treated patients with GERD symptoms and abnormal pH values developed BE, defined as the presence of epithelial specialized columnar-intestinal histologically confirmed at least 3 cm. over the most proximal gastric fold, during a follow-up period of 17–22 years. Four of these patients had grade I esophagitis and two grade II esophagitis at index endoscopy (Savary-Miller classification). In this study, the distinguishing characteristics between those who developed and those who did not develop BE were not given. In an international, multicenter study of the use of maintenance omeprazole in patients with reflux esophagitis who were refractory to long-term histamine 2 receptor antagonist (H2RA) therapy, 20 of 166 patients (12%) developed BE during a median follow-up of 6.5 years (range: 1.4–11.2) [27]. All patients were taking omeprazole at all times, but dosage and demographic characteristics were not stated.

The study by McDougall et al. reported that 3 of 33 (9%) patients presenting with esophagitis developed BE, during a follow-up period of up to 4.5 years [28]. In terms of gender, all three patients were male. It was reported that a patient with minimal esophagitis and a small hiatus hernia at index endoscopy developed a 5-cm length of BE. But, from the anamnestic data, this patient was taking ranitidine 150 mg twice a day. Another patient had grade III esophagitis initially, which reverted to grade I at 2 months of H2RA but then developed a 5-cm segment of BE. This patient was also taking ranitidine 150 mg twice daily. The third patient with grade III esophagitis initially developed a 6 cm BE at his fourth endoscopy 18 months later, although this patient was started on omeprazole 20 mg daily after 12 months but despite this, BE was diagnosed.

In another study, patients with reflux esophagitis cured after PPI treatment continued with the maintenance dose for 14.6 months. Repeated endoscopy was repeated in those patients who had repeated symptoms [1]. Two patients developed BE; one of class II (Savary-Miller classification) after 24 months of follow-up and one of class III after 8 months. In this study, the length of BE and the fact which criteria were used for the diagnosis of BE are not given. These patients were part of a study of 692 patients with GERD, where more than half of the patients had esophageal reflux, but it is not known

<table>
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<th>Evaluation</th>
<th>GERD</th>
<th>BE</th>
<th>Fisher test</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
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<td>N</td>
<td>%</td>
</tr>
<tr>
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<td>Progression</td>
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<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>100.0%</td>
<td>50</td>
</tr>
</tbody>
</table>

| Fisher test Progression/Other | $P = 1.00$ |

Table 5. Comparison of the effect after PPI treatment of patients with BE and GERD.
which ones underwent endoscopy, nor the demographic details of those with BE and those without this pathology. The authors in a retrospective analysis of 582 patients with grade I-III esophagitis (Savary-Monnier classification) diagnosed at endoscopy during a 27-year period, in the follow-up for 6.5 years, 45 (8%) developed BE [29]. But the study was reported only in abstract form and neither the diagnostic criteria, the demographic characteristics of the patients, nor the drug history were described.

In an interesting study investigating the progression of NERD in BE, Bajbouj et al. [30] described that two of a group of 34 patients (6%) with typical GERD symptoms and a normal endoscopy had developed BE after a 35-month follow-up. No data are given for the treatment. As in many other works, the length of the BE segment, as well as the demographic data for the patients, were not given. As stated above, from 1–13% of patients with erosive esophagitis develop BE each year.

4.2 Indirect evidence of progression

El-Serag and Sonnenberg reported the relationship between the middle forms of erosive oesophagitis, esophageal ulcers, and strictures, in 194,527 hospitalized veterans over a 14-year period, using computerized hospital records [12]. Although patients with esophageal ulcers or strictures were older than those with uncomplicated esophagitis, no particular temporal pattern could be established consistently.

In a large multicenter study including 1253 centers from Germany, Austria, and Switzerland, designed to look at the outcome for patients with GERD. Risk factor analysis was performed for 5289 patients with erosive reflux disease or NERD [28, 31]. Small number of erosive diseases are in the population with a higher level of education and the presence of *Helicobacter pylori*.

The association of a longer duration of symptoms with erosive esophagitis compared to some cases of NERD may complicate erosive disease.

Another study reported results for 51,311 patients over 15 years [32]. In most cases, Barrett's esophagus peak arrived at the seventh to ninth decades. In 101 patients, there was no change in the length of BE. The authors showed that this happens more often in male patients aged 60, who had a follow-up endoscopy in a follow-up of 7.4 years.

NERD and erosive disease are part of the dilatation of intracellular spaces of esophageal epithelium confirmed on pH monitoring with results as follow: 38 patients with GERD and 22 with NERD. Early pathophysiological marker of esophageal damage is dilatation of intracellular spaces. Therapy with 40 mg omeprazole resulted in 97% complete recovery after 6 months in NERD and GERD [33]. High-grade dysplasia or esophageal adenocarcinoma can be prevented after using PPI therapy in patients with low-grade dysplasia. PPI therapy should be started after stratification by year within 2 years of definitive diagnosis (our results). LGD can be developed in case of delayed therapy with PPI.

This study confirms our observation that fewer patients with BE developed dysplasia after the introduction of PPI therapy in Australia in 1989. We postulated that the incidence of dysplasia was influenced by powerful acid suppression that reduced esophageal acid exposure.

El-Serag and Sonnenberg reported the relationship between the middle forms of erosive oesophagitis, esophageal ulcers and strictures, in 194527 hospitalized veterans over a 14-year period, using computerized hospital records [34]. In patients with Barrett's esophagus increased epithelial proliferation is step from dysplasia to adenocarcinoma.
Barrett’s esophagus (BE) was found in 11% of our GERD patients [35]. No evidence of completely reverses the length of Barrett’s esophagus [36, 37].

It is very important to emphasize that anticecretory therapy and using cyclooxygenase 2 (COX-2) inhibitors can prevent development of adenocarcinoma [36]. Overexpression of COX-2 inhibits apoptosis, allowing cancer to grow, and COX-2 inhibitors can help ensure that cancer cells die [38]. At the gene level, COX-2 inhibitors can reduce inflammatory factors [39].

In another study was reported that from 350 patients, only 111 patients developed HGD or adenocarcinoma. It should be noted that study didn’t have randomised controlled trial [29, 40]. Low-grade of dysplasia has atipical cells and active inflammation influenced in it [5, 35].

High-grade dysplasia was associated with macroscopic markers: severe esophagitis, nodularity, Barrett’s ulcer or stricture [41].

In another study, the time of the start of PPI use in patients with BE was recorded. The degree of acid reduction was not measured. Also, the doses were not reduced, and this therapy was used for a long time, even though the symptoms of the disease were controlled [2]. Cancer risk for a given patient with BE is lower than previously estimated [23]. Risk factors for the progression of BE to EAC include the increasing degree of dysplasia, increasing age, increasing BE segment length, male sex, and smoking, among others [42].

The degree of dysplasia has been directly related to segment length. The greater the length of the BE segment, the more dysplasia we have [25].

However, when the BE develops, its length generally does not change, so the short-segment BE normally remains short even in the context of continuous exposure of the esophagus to acid. Actually, when we have BE with a short segment, its length does not change much even though it is under the influence of acid [18].

Dysplasia and adenocarcinoma are complications of long and short BE, and are treated similarly [27]. For that more, 20% have an improvement in intestinal metaplasia, but more than 50% had an improvement in patients with low-grade dysplasia. These findings are of great importance in the clinical management of patients with BE, especially given the widespread use of experimental ablative therapies aimed at achieving a similar goal. When we have treated gastroesophageal reflux, we have permission from the EU. Many errors have been minimized through biopsies according to the protocols, the sessions of two biopsies with an output of about 6 months, systematization of regression, so that the biopsy sample was the mucosa of the cardia. It is possible that IM will spontaneously regress to normal tissue without treatment. Additionally, these findings have importance in the clinical management of BE using ablative therapies.

Based on the literature, IM or dysplasia was known after PPI therapy. Intestinal metaplasia was lost in 39% of SSBE patients and 10% of LSBE. Female gender, absence of hiatal hernia, and shorter Barrett’s length associated with loss of IM.

But Sampliner and others [43] suggested that follow up every 2 to 3 years in BE if no dysplasia after two endoscopies. If no change endoscopy should be done every year; in patients with LGD, they recommended every 6 months for the first year.

Author Sharma et al. reported in a multicenter study of LGD history; 35% had intermittent LGD; from the total of 1376 patients incidence of dysplasia was 4.3% every year; 7.3% was prevalence at presentation [44].

After medical therapy LGD had a regression. Our advice is that patients with intestinal dysplasia to follow up with proximal endoscopy every year. Patients with dysplasia should have every 3-month gastroscopic examination.

The goals for treating patients with BE are as follows:
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1. Reduce gastroesophageal reflux

2. Regression or elimination of intestinal metaplasia

3. Reduce progression from dysplasia to cancer.

Shaheen et al. [45] Surgical antireflux procedures are highly effective at reducing gastroesophageal reflux episodes, healing esophagitis, and decreasing the symptoms associated with reflux. It is logical, therefore, to consider their application in the setting of BE to reduce the risk of progression to cancer.

To achieve the first goal of treatment for patients with BE the therapy had not guided by symptoms. Patients should have 24-hour PH monitoring. The author Castell et al. [46] reported an evening dose of H2 receptor antagonist in addition to the twice-daily dose of PPI. Better no therapy compared with incomplete therapy. Finally, gastric PH should be PH =7 with therapy.

Second and third goals therapy are to eliminate IM, to prevent dysplasia and cancer. Despite regular therapy, this did not cause IM regression [47, 48].

Langergren et al. reported the patients who have had symptoms like heartburn and regurgitation have been at risk for adenocarcinoma of the esophagus 8-fold more compared with patients without symptoms [49].

They concluded that treatment did not prevent dysplasia. Some studies had emphasized this issue. There was no reduction in the length of BE despite therapy with 60 mg lansoprazole once a day, almost 3 years [18].

Malesci et al. have shown reducing from length from 4.5 to 2.1 cm with therapy for acid suppression [50]. These studies have demonstrated the difficulties to replicate the impressive decreased length of BE. With PPI therapy twice daily arrived total control of esophageal acid but just in a series of 9 patients. The length of BE was from 7.2 to 5.2 cm (with P < 0.0001) [5]. The use of ranitidine 150 mg twice daily compared with omeprazole 40 mg showed a minimal decrease in segment length in BE. Histamine blockers are not effective in decreasing in the length of BE compared with omeprazole [12].

The conclusion are as follow:

1. The course of BE and mainly of GERD patients may be improved by therapy.
2. Improving appears to be higher in cases with SSBE and in absence of a hiatal hernia.
3. The effect of PPI in decreasing cases with LGD shows that this microscopic evidence was not irreversible.

4. We found that PPI therapy is very beneficial in preventing the development of low-grade dysplasia in BE.

Conflict of interest

The authors declare no conflict of interest.
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