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Management for a Patient with Barret's Esophagus: A Case Report

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Abstract: Barrett's esophagus (BE) is a displacement of the squamocolumnar border (SCJ) site to proximal to the gastroesophageal junction (GEJ) accompanied by the presence of intestinal metaplasia. BE develops when reflux-induced stomach acid destroys the squamous epithelial layer of the esophagus and this lesion heals via a metaplasia process in which the damaged squamous epithelial layer is replaced by columnar colonic-type epithelium. BE prevalence in the general population is about 1.6-1.7%. Patients with gastroesophageal reflux disease (GERD) may progress to BE. This report concerns two cases of patients with Barrett's esophagus. In both these patients BE was found without dysplasia. A diagnosis was made on the basis of anamnesis, physical examination, laboratory, radiological, endoscopy and anatomical pathology. The management of BE is aimed at three main objectives: reduction of symptoms due to GERD, avoiding progression to strictures and ulcers, and preventing progression to adenocarcinoma. Both patients were given a PPI, a prokinetic and chemoprevention NSAID which achieved clinical improvement. Neither patient had ablation, photodynamic or mucosal resection. BE survival is much better than in groups without BE (5 years survival 61% vs. 28%, $P = 0.001$).

1 INTRODUCTION

The name Barrett's esophagus (BE) was coined when a London surgeon, R. Barrett N, described it in the 1950s. This definition continues to evolve, which initially included all of the three types of columnar epithelium, namely the fundal mucosa, cardia and intestinal metaplasia. However, there is currently a general consensus, namely the use of intestinal metaplasia and not using the other two types of mucosa, as markers for BE histology. The definition of BE according to the American Gastroenterological Association is the displacement of the squamocolumnar border (SCJ) site to proximal to the gastroesophageal junction (GEJ) accompanied by the presence of intestinal metaplasia (Bansal, 2007; Pascu, 2004).

BE prevalence in the general population is about 1.6-1.7%. Approximately 10-15% of patients with Gastroesophageal reflux disease (GERD) develop BE, where the incidence of BE developing into adenocarcinoma is 0.5% per year. Compared to

normal people, the risk of esophageal cancer is 30-50 times, with an average age of 55-65 years. More than 80% are white with a 2:1 male to female ratio (Zanten et al., 2006; Wang, 2008; Ponerros, 2009).

BE diagnosis is based on existing symptoms and risk factors, as well as the discovery of endoscopic layers of the columnar epithelium at the distal esophagus and is determined by the presence of intestinal metaplasia in biopsy specimens. BE is categorized based on how long the metaplasia layer limit is measured by endoscopy examination. It is called a long segment if its length is 3 cm or more, whereas if it is less than this it is called a short segment. If no columnar esophageal mucosa is found but intestinal metaplasia is via biopsy under the GEJ then it is called cardia intestinal metaplasia (CIM) (Bansal, 2007; Ponerros, 2009; Spechler, 2002a).

BE develops when reflux-induced stomach acid destroys the squamous epithelial layer of the esophagus and this lesion heals via a metaplasia process in which the damaged squamous epithelial

layer is replaced by columnar colon-type epithelium. Studies have shown the presence of incompetent mechanics in the lower esophageal sphincter, low clearance, frequency and duration of reflux episodes, and peristaltic wave amplitude in both BE types is significantly lower than in normal people (Zentilin et al., 2002; Lord, 2003).

2 CASE PRESENTATION

2.1 Case 1

We report a male patient, Mr. H, aged 30, unmarried, Chinese, bachelor's degree, working in a private company (marketing), living in Surabaya, who came for medication with a major complaint of heartburn in the chest.

The patient complained of heartburn in the chest for 14 years. It frequently recurs, and sometimes the burning sensation is up to the larynx. The complaints worsen if he lies flat, consumes fatty foods or coffee. In addition, the patient complained of nausea, vomiting, bloating and heartburn. There was also constipation that disappeared causing him to often consume traditional medicine. There was no swallowing pain or swallowing difficulty, black feces and black vomiting like coffee, weight loss, or fatigue.

He first had recurring shortness of breath for 7 years before and was diagnosed with bronchitis. He had a history of maxillary sinusitis for 7 years. There was a history of a diet low in vegetables and fruit and a history of treatment with ranitidine and antacids. There were no smoking history, history of drinking alcohol, history of frequent drinking of coffee, history of traditional medicine (ching sung), high blood pressure and diabetes.

Based on a physical examination, it was found that his general condition was moderate, good awareness and was compos mentis. Blood pressure (BP) 120/80 mmHg, pulse 84 x / min, axillary temperature 36.7°C, respiration 20 x / min and body weight 89 kg, Height of 173 cm and BMI 29.73. On examination of the head and neck no anemia, jaundice, cyanosis, dyspnea or enlarged lymph nodes (ELN) were found. Examination of the chest found symmetrical movement and no visible lagged chest breathing. First and second heart sounds is single, there was no noise in all ostia. Lung examination found no vesicular breathing sounds or rales and wheezing. On abdominal examination were found meteorismus, bowel sound increase, unpalpable liver and spleen. Examination of the warm acral

extremities, there was no edema or ELN. Laboratory results: Hb 13.6 g / dl, PCV 0.35, WBC 9,100 / ml, platelets (Pit) 404,000 / ml, blood sugar during 123 mg / dl, BUN 10 mg / dl, serum creatinine SK) 1.1 mg / dl, AST 15 U / L, ALT 13 U / L, Sodium (Na) 123 meq / L, Potassium (K) 3.29 meq / L and Albumin 4.1 g / dl, cholesterol total of 193 mg / dl, LDL 139 mg / dl, HDL 52 mg / dl, triglyceride 68 mg / dl, 5.9 mg / dl acids, HBsAg and non-reactive Anti-HCV.

Abdominal ultrasound found a lot of gas in the intestine. Hepar, lien, kidney, and gallbladder were within normal limits. Endoscopic examination found a mild patch of hyperemic spots with mucosal break of 7 mm and Barret's esophagus with a length of 2 cm. The gastrointestinal and corpus portions were within normal limits, but hyperemia and erythema were present on the antrum of the pylorus. Thus, it was concluded to be GERD grade B and BE and superficial chronic gastritis were suspected. A biopsy was performed.

An anatomical pathology examination report (PA) found a gastric gland under the esophageal epithelium and no dysplasia or any signs of malignancy with a conclusion of BE. A gastric biopsy found chronic superficial gastritis and helicobacter pylori infection did not appear.

From anamnesis, physical examination, laboratory, radiologic, endoscopy and PA, the patient was diagnosed with GERD grade B + BE short segment without dysplasia + chronic superficial gastritis. The patient was advised to change their lifestyle and to undergo Rabeprazole therapy 2x20 mg, domperidone 3x10 mg, aspirin 100 mg-0-0. After 2 weeks of treatment the patient's condition improved. The patient was advised to continue the therapy and undergo another endoscopy one year later by 2 endoscopists.

2.2 Case 2

A female patient, Mrs. P, 49 years old, married, Chinese, bachelor's degree, a teacher, living in Surabaya came for treatment with major complaints of frequent bloating and heartburn.

She complained of frequent bloating and heartburn for 8 years, frequent recurrences, which improved with antacid administration and rest. This complaint is accompanied by a burning sensation in the chest when consuming spicy foods, coffee or tea. The patient also complained of a frequent sore throat for the past 5 years up to twice a month and often had swallowing pain. There were no black feces or vomiting, decreased weight or fatigue.

There was a maxillary sinusitis 3 years ago. There was a history of coffee and tea consumption, omeprazole consumption (with a doctor's prescription) twice a day for 1 month, and no history of smoking, alcohol consumption, high blood pressure or diabetes.

A physical examination found that her general condition was moderate, with good awareness and was compos mentis. Blood pressure (BP) was 110/70 mmHg, pulse 82 x / min, axillary temperature 36.7°C, respiration 20 x / min and body weight 60 kg TB 153 cm BMI 25.63. Examination of the head and neck did not find anemia, jaundice, cyanosis, dyspnea or enlarged lymph nodes (ELN). Examination of the chest did not find asymmetrical movement and there was no visible lagged chest breathing. First and second heart sounds is single, no noise in all ostia. Lung examination found vesicular breathing sounds with no rhonchi and wheezing. Examination of the stomach found meteorismus, increased bowel sounds, and the liver and lien were not palpable. The examination of the warm acral extremities did not find edema and ELN.

Laboratory results: Hb 12.1 g / dl, PCV 0.36, WBC 6510 / ml, platelets (Plt) 316,000 / ml, fasting blood sugar 112 mg / dl, blood sugar 2 JPP 126 mg / dl serum creatinine (SK), LDL 91 mg / dl, HDL 38 mg / dl, Triglyceride 147 mg / dl, Acid vein 4.4 mg / dl.

Endoscopic examination found a mild patch of hyperemic spots with a mucosal break of 6 mm and a Barret esophageal picture with a length of 5 cm. The gastrointestinal and corpus segments were within normal limits, but hyperemia and erythema were detected on the antrum and angulus segment. Thus, it was concluded as GERD grade B and BE and superficial chronic gastritis were suspected. A biopsy was performed.

PA examination reported that under the esophageal epithelium there were gastric glands, but no dysplasia or any signs of malignancy with the conclusion of BE. In gastric biopsy, chronic superficial gastritis was found and helicobacter pylori infection did not appear.

From the results of the anamnesis, physical examination, laboratory, endoscopy and PA, the patient was diagnosed as GERD grade B + BE long segment without dysplasia + chronic superficial gastritis. The patient was advised to change her lifestyle and undergo 2x40 mg of esomeprazole therapy, 3x10 mg of domperidone, aspirin 100 mg-0-0, and ranitidine 150 mg before bed. The patient's condition got better after one month of therapy. The

patient was advised to continue the therapy and have another endoscopy one year later by 2 endoscopists.

3 DISCUSSION

For decades, BE was identified primarily in patients with severe GERD based on the long segmental findings of columnar epithelial extending more than 3 cm above the gastroesophageal junction (Spechler, 2002a). Clinically BE does not give symptoms; most people complain because of symptoms of GERD (Table 1). The most common symptoms of GERD are heartburn or pyrosis, regurgitation, and dysphagia. Heartburn, defined as heat in the retrosternal region, has a specificity of 89% and a positive predictive value of 81% for GERD. Regurgitation, defined as a reflux sensation from the stomach to the mouth or hypopharynx, has a 95% specificity and a 57% positive predictive value for GERD. The classic GERD symptoms rarely require confirmatory tests because they have a high positive predictive value and are sufficient to make a diagnosis in the majority of cases. In fact, the combination of heartburn and regurgitation has an accuracy greater than 90% for diagnosing GERD (Hirano, 2007).

Extraesophageal or atypical manifestations are quite common in patients with GERD. As many as 80% of patients with GERD symptoms may have at least one extra esophageal manifestation (Kamat, 2008). GERD causes coughing in 40% of patients and GERD is found in 34-89% of patients with asthma (Hirano, 2007). Sinusitis may be a manifestation of GERD either directly injuring the sinus mucosa or causes vagus-mediated neuro inflammatory changes or both (Jecker et al., 2006).

Table 1: Symptoms of Gastro esophagus reflux disease.

Esophageal Manifestation	An extraesophageal or atypical manifestation		
	Oral	ENT	Lung
Heartburn	Water-brash	Hoarseness	Asthma
Regurgitation	Burning sensation in mouth	Dysphonia	Bronchitis
Dysphagia	Tongue sensitivity	Sore throat	Pneumonia
	Halitosis	Chronic cough	Idiopathic pulmonary fibrosis

Source: (Kamat, 2008)

In patient I there were heartburn, nausea, vomiting, bloating and heartburn. There was a history of bronchitis and maxillary sinusitis for 7 years that may be associated with GERD. In patient II frequent bloating and heartburn, a frequent sore throat and pain on swallowing were found. There was a history of maxillary sinusitis for 3 years probably related to GERD.

Screening is recommended for patients who have a high risk of developing BE, i.e. male, white, aged > 40, duration of old GERD complaints of > 13 years, smokers, and obesity (Sharma, 2005; Wang, 2008). Increased BMI is a major contributor to the development of GERD, which in turn is a known risk factor for BE. Increased levels of proinflammatory cytokines (adipocytokines) including interleukin-6 and tumor necrosis factor- α , associated with visceral fat, have been shown to be expressed in patients with BE. Various biological mediators including leptin, insulin and insulin-like growth factors, which are closely related to obesity, are shown to stimulate cell proliferation and inhibit apoptosis. This may be similar to the mechanisms underlying the development of esophageal neoplasia (Kamat, 2008; Sharma, 2005).

In patient I screening was recommended because he was male, having the duration of GERD of 14 years and BMI of above normal. While in patient II the risk was the age of > 40 and BMI of above normal. The American College of Gastroenterology provides guidelines for the diagnosis of GERD, which provides empirical therapy of acid suppressant drugs in addition to lifestyle modification if the patient has symptoms of uncomplicated GERD. Empirical therapy is in the form of the provision of a proton pump inhibitor (PPI) such as omeprazole 20-40 mg twice daily for 1 week. One study reported that an assessment of 40 mg omeprazole response for 2 weeks had the same diagnostic efficacy value as gastric pH testing (Kamat, 2008). Uterine endoscopic examination (Upper Gastrointestinal) is a golden standard for the diagnosis of GERD with the presence of mucosal breaks in the esophagus (Table 2). It is included in grade B if the mucosal break is more than 5 mm but does not extend to the peak of the folds of two mucosa.

In both patients there was a mucosal break > 5 mm but did not extend to the peak of two mucosa folds, thus including grade B.

Measurement of the length of the BE segment requires the important participation of an endoscopist, because the presence of large hiatus hernia, ulcers / erosions, strictures etc., can reduce

the accuracy of the assessment leading to overdiagnosis. The method of measurement is to start by identifying the gastroesophageal junction (GEJ) and squamocolumnar junction (SCJ) accurately, then measure the distance between the proximal SCJ and GEJ measured by endoscopy and recorded as the length of the BE segment (Bansal, 2007). The length of the segment will confirm the high risk of progressive progression to adenocarcinoma. Long segments have an esophageal cancer risk 30 times greater than the general population.

Table 2: Modification of the Los Angeles classification for GERD.

Grade	Description
A	Break of mucosa of < 5 mm but does not extend to the peak of two mucosa folds.
B	Break of mucosa of > 5 mm but does not extend until peak folds of two mucosae.
C	The mucosal break extends to the peak of two mucosa folds but less than 75% of the esophageal periphery.
D	Mucosal breaks extend to the top of two mucosa folds over 75% of the esophageal periphery

Source (Lichtenstein et al., 2007)

Several new imaging modalities are proposed to help establish the diagnosis of BE. The currently commercially available technique is narrow band imaging (NBI), a method of filtering light into two main colors, blue and green, which means more is absorbed by the blood vessels in the mucosa and subepithelium. This difference helps the endoscopist to see the mucosa better, let alone combined with high-resolution endoscopy. The sensitivity of NBI for the detection of irregular mucosal patterns is 100% with a specificity of 98.7%. Another modality is autofluorescence imaging, a technology that uses blue lighting to detect fluorescence from cellular components in the esophagus. The dysplasia region does not have as intense autofluorescence as normal tissue and appears dark red. A study found that autofluorescence was 100% sensitive for high-class regions of dysplasia in 20 patients, but had a false-positive rate of 40%. Other contrast agents such as violet crystal, indigo carmine, and acetic acid have also been proposed to improve the detection of mucosal patterns in Barrett's esophagus in combination with high-resolution endoscopes. Although very promising, there is not enough current evidence to recommend the use of clinically routine imaging systems (Wang, 2008).

In Patient I, there was a long segment of BE of 2 cm, which included a short segment, whereas in patient II the segment length was 5 cm so it included a long segment, so theoretically had a higher risk of esophageal cancer.

Dysplasia is a histological diagnosis showing that one or more epithelial cell clones have undergone a genetic alteration that makes them neoplastic and prone to malignancy (Spechler, 2002a). The diagnosis of BE biopsy was classified with no dysplasia, low-grade dysplasia and high-grade dysplasia. Low-grade dysplasia is most commonly found and is a cause of cancer. In its development, low grade can turn into high grade then sometimes turns into cancer (Poneros, 2009).

In both patients there was no dysplasia, then diagnose as BE without dysplasia.

In 2008, the American College of Gastroenterology recommended that the class of dysplasia determines the interval of surveillance (Table 3).

Table 3: Grade of dysplasia, management and surveillance recommendations.

No	Grade of dysplasia	Management	Surveillance recommendation
1	No dysplasia	2 endoscopies with biopsy for confirmation within 1 year	No dysplasia, endoscopy every 3 years
2	Low-grade dysplasia	Confirmed by a PA expert, endoscopy with biopsy every 6 months	High-grade dysplasia -, endoscopy is performed / yr to no dysplasia obtained at 2 checks
3	High-grade dysplasia	Confirmed by a PA expert Irregular Mucosa: consider mucosal resection with endoscopy. Flat mucosa: repeat endoscopy and biopsy every 3 months	Confirms the presence of High-grade dysplasia, 3 months of intensive surveillance, esophagectomy, endoscopic therapy.

Source: (Kamat, 2008)

Endoscopic surveillance should be performed in chronic GERD patients. Ideally symptoms can be controlled with acid suppression therapy. This is

important because the inflammatory process associated with reflux esophagitis may interfere with the diagnosis and BE grading and may even be similar to dysplasia. Endoscopic BE monitoring involves 4 quadrants and a biopsy taken every 1 to 2 cm across the Barrett segment. For patients with intestinal metaplasia (without dysplasia), surveillance endoscopy should be performed twice in the first year to exclude dysplasia changes. Patients with low-grade dysplasia should undergo endoscopic follow up at 6 months from initial screening to exclude progression to high-grade dysplasia. Patients with high-grade dysplasia in the form of flat mucosa should be referred to a special center for further diagnostic evaluation and confirmation of the diagnosis by a gastrointestinal PA expert with endoscopies in the next 3 months (Kamat, 2008; Sharma, 2005).

In both patients BE was found without dysplasia, so endoscopic surveillance was planned again after 1 year by two endoscopists.

Peitz et al. detected *Helicobacter pylori* (HP) in 50% of subjects, independent of BE length (Tuncer et al., 2003). Some researchers have hypothesized that the presence of HP infections may have protective effects on the esophagus against esophageal cancer, as the gastric acid output is reduced (Spechler, 2002a; Laheij et al., 2002).

In both these patients there was no *Helicobacter pylori* infection.

Several studies have determined risk factors for progression to adenocarcinoma in BE patients (Table 4). Genetic abnormalities, developmental abnormalities of tumor suppressor genes, p16 and p53, as well as the development of tetraploid and aneuploid cell populations detected through flow cytometry and fluorescence are situ hybridization (FISH). Increased gastro-esophageal reflux is associated with increased abnormalities of biomarkers at all stages of molecular development, including p16, p53, tetraploidy and aneuploidy (Poneros, 2009; Souza et al., 2001; Jankowski, 2004).

In patient I risk factors for development into adenocarcinoma are a low-fat diet and fruit, increased BMI, male gender, heartburn and lack of a PPI. Patient II had risk factors of being > 40, BMI increase and BE long segment. Biomarkers were not performed on these two patients.

The management of BE is aimed at three main objectives: reduction of symptoms due to GERD, avoiding progression to strictures and ulcers, and preventing progression to adenocarcinoma. Changes in lifestyle that must be done are BB reduction,

sleeping with head elevation, and avoiding smoking, fatty foods, coffee, chocolate, alcohol and orange juice. Reduction of GERD symptoms by using suppression of gastric acid secretion by administration of a H2-Antagonist Receptor (H2RA) or PPI. The success of antisecretic therapy for GERD is directly related to the degree of acid suppression achieved and inversely related to the severity of the underlying esophageal reflux. The presence of incompetent mechanics in the lower esophageal sphincter, low clearance, frequency and duration of reflux episodes, peristaltic wave amplitude in both BE types significantly lower than that of normal people are the basic concepts of GERD pathogenesis. So prokinetic drugs such as metoclopramide, domperidone and cisapride are only used as an adjuvant PPI and not given as a monotherapy (Fitzgerald, 2004; Schuchert, 2007).

Table 4: Risk factors for development into adenocarcinoma in patients with Barret's Esophagus.

Clinical Factor	Endoscopy Factor	Histology factor
Diet low in vegetables and fruit	Working in hunchback position	Large hiatal hernia
Increase BMI	Lack of using PPI	BE long segment
Male	Symptoms of heartburn	Mucosal abnormalities
Old Age	Use of drugs that relax the esophageal sphincter	Nodularity Ulceration Strictureing
Smoker		Aneuploidy P53 loss of heterozygosity

Source: (Poneros, 2009)

Patients I and II were recommended lifestyle changes and were given a PPI and prokinetic.

All patients with BE should be given a PPI although there are no symptoms of GERD. A long-term retrospective study of 236 patients with BE who received a number of different anti-reflux therapies showed that in 10 years, the incidence of cumulative dysplasia was 21% in patients receiving a PPI compared with 58% in patients who did not receive a PPI or H2RA. Another study showed that the risk of developing low-grade dysplasia was 5.6 times higher in patients who started PPI therapy 2 years or more after the diagnosis than in patients starting PPI therapy in the first year. In addition,

delayed PPI therapy may increase the risk of progression to high-grade dysplasia or adenocarcinoma by 20.9%. Some researchers advise to add H2RA at night in addition to the use of a PPI twice daily to reduce acid relief (Chen et al., 2002; Sabel et al., 2000).

Currently there are five types of PPI with comparable efficacy of omeprazole (20 mg / day), lansoprazole (30 mg / day), Rabeprazole (20 mg / day), Pantoprazole (40 mg / day) and esomeprazole (40 mg / day). Research using two types of PPI approved by the Food and Drug Administration (FDA), giving once or twice daily oral esomeprazole shows the ability to control significantly the intragastric acidity compared to equivalent doses of lansoprazole in Patients with BE. Twice daily doses of the drug give better control of the intragastric pH than once-daily dosing, and both exhibit a relative advantage of controlling intragastric acid with increasing dosage in 75% of patients with BE (Jankowski, 2004; Spechler et al., 2002b). Ortiz et al. suggested monitoring gastric pH in the titration process of PPI dosing (Hirano, 2007).

Patient II was given 2 x 40 mg of esomeprazole PPI with 150 mg of ranitidine added in the evening. Patient I was given 2 x 20 mg of Rabeprazole.

Anti-reflux surgery is attempted by creating a barrier to gastroesophageal reflux through an esophagus packing by the fundus of the stomach (fundoplication). This is usually performed in patients with low comorbidity and the presence of symptoms of reflux despite being treated with a PPI. But the long-term results are disappointing with a 5-year failure rate of 20% (Wang, 2008). Initially, some researchers argued that the possibility of fundoplication is more effective than a PPI to prevent cancer deaths due to BE. Two recent studies provide little support for this opinion, but in one randomized trial of medical and surgical therapy in 247 complicated GERD patients, adenocarcinoma of the esophagus occurred in four of the 165 patients in the medical group (2.4 percent) and one of 82 in the surgical group (1.2 percent) during follow up 10 to 13 years with a statistically non-significant difference (Wang, 2008; Spechler, 2002a). Baulieux et al. reported complete or partial regression of 27% in patients undergoing fundoplication and 7.1% with PPI therapy. Ortiz et al. reported regression in 32% with fundoplication versus 7% with PPI on BE found at segment length (Lord, 2003). Medical and surgical therapy is very effective for correcting or relieving symptoms and signs of GERD, but has not been shown to reduce the risk of esophageal

adenocarcinoma (Fitzgerald, 2004; Cooper et al., 2006).

In both these patients anti-reflux surgery was not performed.

Chemoprevention in the prevention of adenocarcinoma of the esophagus represented by BE is considered quite rational. The best evidence of use of chemoprevention agents is Cyclooxygenase (COX) -2 inhibitors and 325-mg enteric-coated aspirin daily has been shown in several epidemiological studies and is associated with a significantly reduced cancer risk with a 0.57 odds ratio (95% CI 0.47- 0.71). This decrease in risk can be attributed to the effect on cyclo-oxygenase-2 mucosal expression (COX-2) and prostaglandin E2 production (PGE2), as both are associated with the development of BE into adenocarcinoma. Tsibouris et al. proved that BE patients with esophageal adenocarcinoma were rarely consumed NSAIDs (Poneros, 2009; Fitzgerald, 2004).

Both patients were given aspirin 100 mg-0-0. Ablation therapy, ablation with thermal energy (thermal ablation), photodynamics, and endoscopic mucosal resection have now been widely used for BE high-grade or early-stage care. However, the lack of data on the usefulness of these therapies for BE without dysplasia is therefore not recommended. In the future, biomarkers may be able to provide better risk stratification to justify endoscopic ablation therapy for non-displaced BE (Poneros, 2009; Wolfson et al., 2002; Schuchert, 2007).

In both patients there was no ablation, photodynamic or mucosal resection.

Survival in the BE group was much better than the group without BE (5 years survival 61% vs. 28%, $P = 0.001$). Adenocarcinoma of the esophagus is not a common cause of death in BE patients; the most common cause is bronchopneumonia and ischemic heart disease (Portale et al., 2005; Sabel et al., 2000).

4 CONCLUSION

This report concerns two cases of patients with Barrett's esophagus. In both these patients BE was found without dysplasia. Diagnosis was made on the basis of anamnesis, physical examination, laboratory, radiological, endoscopy and anatomical pathology. The management of BE is aimed at three main objectives: reduction of symptoms due to GERD, avoiding progression to strictures and ulcers, and preventing progression to adenocarcinoma. Both patients were given a PPI, Prokinetic and Chemoprevention NSAID which achieved clinical

improvement. Both patients had no ablation, photodynamic or mucosal resection. BE survival is much better than in groups without BE (5 years survival 61% vs. 28%, $P = 0.001$).

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