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A New Horizon

Recommendations and Treatment Guidelines for Barrett's Esophagus

ABSTRACT

Barrett's esophagus is a premalignant metaplastic process that involves the distal esophagus and requires lifelong surveillance and treatment. One of the major risk factors identified in the development of Barrett's esophagus is gastroesophageal reflux disease, which is reported in up to 20% of the adult population in the United States. New mucosal ablative techniques have emerged to treat Barrett's esophagus with high-grade dysplasia in an effort to prevent the progression to esophageal adenocarcinoma. Esophageal cancer is the 7th most prevalent cancer in the world and has a very poor 5-year survival rate. This review focuses on the developments in the field of Barrett's esophagus including the epidemiology, presentation and progression, medical and surgical management, recommendations for treatment, guidelines for surveillance, and new therapeutic treatments.

The incidence of esophageal cancer in the United States has increased approximately 300%-500% in the last 40 years (Shaheen, 2005b). Currently, there are an estimated 15,560 new diagnoses of esophageal cancer and almost 14,000 deaths per year, with a poor curative success rate of 5%-10% (American Cancer Society [ACS], 2007; Koshy, Esiashvilli, Landry, Thomas, & Matthews, 2004). Men have a 3:1 greater incidence and death rate over women (ACS, 2007). Northern China and northern Iran have the highest rates of esophageal cancer, with an incidence of 100 in 100,000, whereas in the United States, the prevalence is much lower at 5 in 100,000 (Koshy et al., 2004).

Not only has the incidence of esophageal cancer risen, there has been a shift in the histological character and principal tumor location as well. Most esophageal tumors now originate in the distal esophagus, and histologically, adenocarcinoma has become more prevalent than squamous cell carcinoma in the United States and Western Europe (National Cancer Institute [NCI], 2007). Epidemiology of esophageal carcinoma and its presumed precursor, Barrett's esophagus, is poorly understood, with recent evidence showing Barrett's esophagus more prevalent in asymptomatic individuals than previously thought (Shaheen, 2005b). It is unclear why some patients develop severe recurrent erosive esophagitis and never develop Barrett's esophagus, whereas others with relatively few symptoms and little evidence of inflammation on esophagogastroduodenoscopy (EGD) develop long segments of severely dysplastic tissue changes.

Case Study

Mr. C. is a 52-year-old white man who comes into the endoscopy department for an EGD following admission to the hospital with chest pain and recurrent heartburn symptoms. An acute myocardial infarction was ruled out. Mr. C.'s medical and surgical history includes hypertension and hyperlipidemia, for which he takes hydrochlorothiazide and simvastatin, and an inguinal herniorrhaphy at age 27. He is married with two grown children, quit smoking 5 years prior to admission, drinks socially, denies illegal drug use, and works as a lineman with a local cable company. Upon

Received July 10, 2008; accepted November 6, 2008.

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The author has disclosed that she has no financial relationships related to this article.

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EGD, the gastroenterologist noted an irregular z-line in which the squamocolumnar junction was displaced upward with patches of salmon-colored, tongue-like mucosal projections extending into the normal squamous tissue. Circumferential biopsies were obtained at 2-cm increments and sent to pathology to rule out Barrett's esophagus and determine the degree of intestinal metaplasia to confirm this diagnosis.

Pathogenesis of Barrett's Esophagus and Cancer

Barrett's esophagus is a change in the lining of the distal esophagus that occurs when gastric secretions reflux into the esophagus. Injury and denudation occur through a metaplastic process in which an abnormal columnar epithelium replaces the injured squamous tissue (Spechler, 2002a). Because of increased exposure of the esophageal mucosa to acid and pepsin, damage and mucosal permeability occur into the submucosal layer causing pain, inflammation, and, in some cases, necrosis (Moss & Kelly, 2007). The proposed dysplasia to carcinoma sequence first involves intestinal metaplasia (Barrett's esophagus), evolving into low-grade dysplasia (LGD) to high-grade dysplasia (HGD) to adenocarcinoma; however, this progression has never been clearly defined or validated endoscopically (Figure 1) (Hur, Wittenberg, Nishioka, & Gazelle, 2005; Spechler, 2002a).

One of the strongest predictors of cancer risk in the Barrett's esophagus population is the degree of dysplasia. Research has not been able to predict the progression through the grades of dysplasia but has found that there is no orderly or discernable development. Those with HGD may experience a disease progression rate of about 10% a year; however, subjects with HGD may also undergo regression of the disease (Shaheen, 2005b). It is unclear if there is a genetic predisposition for Barrett's esophagus. Research has not found evidence of a "Barrett's gene" in first-degree relatives of those with Barrett's esophagus; however, in family cohort studies, Barrett's esophagus was found to be present in family groups (Shaheen, 2005b).

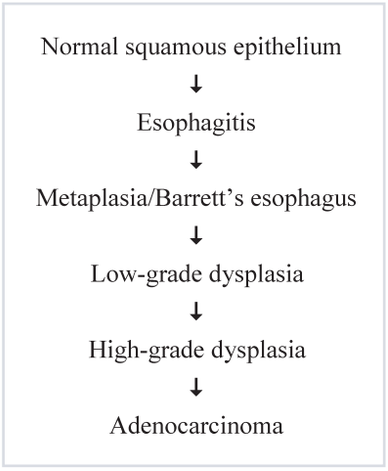

FIGURE 1. Progression of Barrett's esophagus.

Recent studies have demonstrated a close correlation between the duration of acid exposure and the length of Barrett's mucosa (Fass & Sampliner, 2003). Patients with prolonged acid exposure have been found to have a higher rate of dysphagia and a defective lower esophageal sphincter (LES) pressure, all leading to an increased prevalence of Barrett's esophagus (Fass & Sampliner, 2003). The diagnosis of Barrett's esophagus is based on tissue samples taken during an EGD. The prevalence of Barrett's esophagus in patients undergoing an upper endoscopy for any reason is 0.5%-4%; however, this increases to 12%-15% for patients with gastroesophageal reflux disease (GERD) symptoms (Fass & Sampliner, 2003).

Recurrent acid reflux and the duration of symptoms have been associated with a 7.7 times greater risk for developing esophageal adenocarcinoma, with more frequent, more severe, and longer lasting reflux resulting in approximately a 40% higher risk for cancer (Shaheen & Ransohoff, 2002; Tharalson et al., 2002). Predisposing risk factors for the progression of Barrett's esophagus to adenocarcinoma include the length of Barrett's esophagus, a hiatal hernia of at least 3 cm in length, duration of acid exposure, impaired mucosal defenses, and the presence of dysphagia (Koshy et al., 2004).

Cancers of the esophagus must involve 75% of the circumference before the sensation of dysphagia is experienced (Koshy et al., 2004). The most common presenting symptoms of esophageal cancer include dysphagia and weight loss, whereas odynophagia, cachexia, melana, retrosternal pain, and hoarseness are less commonly experienced. The most important prognostic indicators of survival include the extent of wall penetration and the degree of lymph node metastases (Koshy et al., 2004).

Classification of Barrett's Esophagus

Gastroesophageal reflux affects a large portion of today's population, with 20%-29% of Americans reporting weekly heartburn symptoms (Dent, El-Serag, Wallander, & Johansson, 2005). Up to 44% of the general population experience GERD, but only 10% of those who experience chronic reflux symptoms go on to develop Barrett's esophagus (Tharalson et al., 2002). Heartburn and regurgitation are the most common symptoms of GERD; however, it is also often associated with coughing, asthma, hoarseness, and chest pain (Fass, 2003). Independent predictors of

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erosive esophagitis and GERD have been directly linked to the male gender, a high body mass index (BMI), heavy alcohol use, and history of smoking (Table 1) (Hur et al., 2005; Koshy et al., 2004; Moss & Kelly, 2007). Anatomical factors that affect Barrett's esophagus include a reduced intra-abdominal LES length and the presence of a hiatal hernia (Fass & Sampliner, 2003). A recent population-based survey reported the prevalence of Barrett's esophagus in 1.6% of the population; however, up to 40% of these patients deny symptoms (Ronkainen et al., 2005).

The criteria for diagnosis of Barrett's esophagus are based on the visualization of the columnar epithelium lines (z-line) in the distal esophagus after identification of both squamocolumnar and gastroesophageal junctions during an EGD and the histological presentation of the biopsies that identify intestinal metaplasia taken from that columnar epithelium (Spechler, 2002b). Categorizations of the extent of metaplastic changes are identified as long segment (≥3 cm) and short segment (<3 cm). Although the pathogenesis for the difference between the two is unclear, it may represent a continuum of the same disease, and they are managed in a similar fashion (Spechler, 2002b; Tharalson et al., 2002).

Screening Guidelines

The goal of a surveillance program is to identify patients with Barrett's esophagus and to provide early treatment to reduce the mortality from esophageal cancer through early detection. The significance of developing a screening program for detecting Barrett's esophagus is based on current research demonstrating that 94%-98% of patients diagnosed with esophageal adenocarcinoma have no known history of Barrett's esophagus (Fass & Sampliner, 2003). The Practice Parameters Committee of the American College of Gastroenterology (ACG) concluded that any patient with long-standing or complicated GERD symptoms should have an initial endoscopy (DeVault & Castell, 2005; Sampliner, 2002).

Gastroesophageal reflux disease should be aggressively treated prior to an EGD to minimize confusion caused by inflammation in the biopsy tissue (Spechler, 2002b). Risk factors for Barrett's esophagus, such as obesity, male gender, and age greater than 50 years, should be taken into account when determining the necessity of an EGD to rule out intestinal metaplasia (Spechler, 2002b). This recommendation is based on the observation that patients with GERD who lack Barrett's tissue on endoscopy appear to be at minimal risk of developing Barrett's esophagus during their lifetime (Fass & Sampliner, 2003).

The risk of developing cancer in patients without dysplasia is 2%, whereas the risk jumps to 7% for patients with LGD and to 22% for those with HGD (Brunk, 2007). Less than 5% of patients with esophageal cancer were known to have had Barrett's esophagus before they sought help for symptoms of cancer, and up to 40% had no history of GERD (Dulai, Guha, Kahn, Gornbein, & Weinstein, 2002). Questionable pathology may sometimes require additional testing, such as an endoscopic ultrasound or a computed axial tomography scan, to help determine the appropriate treatment modality (Phan et al., 2005).

According to the ACG, the frequency of endoscopic surveillance programs for patients with Barrett's esophagus is dependant on the degree of dysplasia (Sampliner, 2002). Patients without dysplasia should have a surveillance endoscopy every 2-3 years. In the case of LGD, an EGD should be repeated after 6 and 12 months and can go to yearly if there has been no progression. The patient with HGD should have the diagnosis confirmed by an experienced pathologist and should undergo treatment to prevent the progression to cancer (Sampliner, 2003; Shaheen, 2005b). See Table 2 for guidelines for the management of Barrett's esophagus.

TABLE 1. Risk Factors Associated With Barrett's Esophagus

• Age > 50 years
• Caucasian
• Heavy alcohol use
• Long-standing history of gastroesophageal reflux disease
• Male
• Obesity
• Smoking

Treatment Modalities for GERD

Medical Treatment

Most patients with GERD have tried over-the-counter antacids, which improve the heartburn symptoms about 20% of the time. The prevalent medical therapy for Barrett's esophagus is proton pump inhibitors (PPIs) (Fass & Sampliner, 2003). Proton pump inhibitors decrease symptoms, heal esophagitis, and prevent Barrett's esophagus in up to 80% of patients, as compared with 60% of patients receiving histamine receptor antagonists (DeVault & Castell, 2005; Moss & Kelly, 2007). According to the ACG, treatment of GERD should be the same regardless of whether the patient is diagnosed with Barrett's esophagus or not (Sampliner, 2002).

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TABLE 2. Screening Guidelines for Barrett's Esophagus

Classification	Recommendation
Gastroesophageal reflux disease	• Esophagogastroduodenoscopy by age 50 for any signs or symptoms of heartburn • Treatment with proton pump inhibitors
Barrett's esophagus metaplasia	• Two surveillance endoscopies • Four quadrant biopsies, 2 cm apart, 1 year apart
No dysplasia	• Repeat endoscopy in 2-3 years • Low risk for developing cancer
Low-grade dysplasia	• Repeat endoscopy in 1 year • Low risk for developing cancer
High-grade dysplasia	• Intervention required • High risk for developing cancer

Adapted from "Updated Guidelines for the Diagnosis, Surveillance, and Therapy of Barrett's Esophagus," R. E. Sampliner, 2002, *The American Journal of Gastroenterology, 97*(8), pp. 1888-11895. Copyright 2002 by the American College of Gastroenterology.

Although most patients will respond to acid suppression therapy, there is a subgroup of patients (up to 30%) who do not respond to therapy, also called PPI failure. The most common reasons for PPI failure remain poor compliance, inadequate dosing, delayed gastric emptying, and esophageal visceral hypersensitivity (Moss & Kelly, 2007). Esophageal visceral hypersensitivity is an altered visceral pain perception threshold in patients with heartburn symptoms, which are triggered not only by acid exposure but also by other stimuli such as cold or carbonated beverages and saliva (Moss & Kelly, 2007).

Surgical Treatment

Patients with persistent reflux symptoms or esophagitis, despite medical therapy, can undergo surgery or other endoscopic antireflux procedures. Nissen fundoplication is a minimally invasive surgical procedure that involves tightening of the LES. It is the surgery of choice due to the minimally invasive technique involved and the short length of stay in the hospital (National Guideline Clearinghouse [NGC], 2004). This surgery is associated with good quality short-term results, with up to 90% of patients reporting noteworthy improvement in symptoms of GERD (Moss & Kelly, 2007). Unfortunately, over time, the symptoms do reoccur, with research showing a variation in the benefits without reduction in the cancer risk in patients after antireflux surgery (Moss & Kelly, 2007). One study found 62% of surgical patients repeatedly using antireflux medications 10 years later, whereas another study found 32% of the patients using antiflux medication within 20 months and only a 61% satisfaction rating for long-term control of reflux symptoms (Moss & Kelly, 2007; Tran, Spechler, Richardson, & El-Serag, 2005).

Endoscopic Antireflux Procedures

Endoscopic antireflux procedures including plication, radiofrequency energy, and polymer injection have been identified as safer, less-invasive, and cost-effective methods to treat GERD by mechanically tightening the LES (Moss & Kelly, 2007). Although somewhat promising, all of these techniques are still considered experimental, having been tested only in short-term unblinded trials with a small number of patients with mild disease (Shaheen, 2005a, 2005b). There was also a high placebo effect in this research patient population, with up to 40% no longer requiring daily antireflux medication (Moss & Kelly, 2007). Although endoscopic antireflux procedures remain experimental, they may be more appropriate in certain patient populations. These include patients who have failed medical therapy, yet are poor surgical candidates, and patients who present with a high risk for chronic aspiration with poor esophageal motility (Shaheen, 2005b).

Goals of Endoscopic Treatment of Barrett's Esophagus

The goals of screening for Barrett's esophagus are ultimately to identify high-risk patients, provide early intervention, and improve the survival of patients in whom the progression from Barrett's esophagus to adenocarcinoma occurs. Research shows that despite medical and surgical therapies for controlling reflux in Barrett's esophagus, neither treatment has demonstrated success in the progression from intestinal metaplasia to neoplasm (Fass & Sampliner, 2003). The goal of treatment of Barrett's esophagus is to control the symptoms of GERD, heal the mucosal inflammation, and prevent the sequence of dysplasia to malignancy (Fass & Sampliner, 2003; Lanas, 2005). This goal has not been fully achieved because there is little evidence that current screening practices or endoscopic treatments have decreased the number of deaths from esophageal cancer (Spechler, 2002a).

Treatment of HGD

Patients have several options for the management and treatment of Barrett's esophagus with HGD including surgery, ablative therapy, and endoscopic surveillance (Spechler, 2002a). Currently, the standard treatment is an esophagectomy because of the significant number of patients who were found to have esophageal carcinoma

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on the pathology report (Shaheen, 2005b). An esophagectomy clearly prevents the progression of HGD to carcinoma; however, there is significant mortality and morbidity associated with this surgery, especially in low-volume institutions (Shaheen, 2005b; Spechler, 2002b). The 30-day mortality rate in low-volume hospital settings is 18.7% for an esophagectomy, compared with 9.2% for patients in high-volume hospitals (Shaheen, 2005b). Serious postoperative complications have been reported in 30%-50% of patients who had undergone esophagectomy, which include pneumonia, atelectasis, myocardial infarction, arrhythmias, heart failure, wound infections, and anastomosis leaks (Spechler, 2002b). Following an esophagectomy, some patients were found to redevelop dysplastic mucosa in the remaining esophagus from persistent GERD (Spechler, 2002b). For this reason, new endoscopic therapies have been suggested in the management and treatment of Barrett's esophagus with HGD.

Endoscopic Ablation Therapy

There are three types of endoscopic ablation treatment of Barrett's esophagus: chemical, thermal, and mechanical (Table 3). Ablation therapy is an alternative treatment of Barrett's esophagus and is used to intentionally destroy the Barrett's mucosa and allow for reepithelialization in an "antacid or hypochlorhydric environment" (Johnston, 2005, p. 324). With a dismal 5-year survival rate of <10% for adenocarcinoma of the esophagus, and the mortality and complications rates identified following esophageal surgery, endoscopic ablation may provide a promising new treatment option for patients with HGD (Gopal, Reichelderfer, Gaumnitz, & Jobe, 2004; Shaheen, 2005b).

It is important to identify that ablative therapies are expensive and research has not demonstrated that they eradicate all of the dysplastic tissue or decrease the long-term risk of developing cancer; therefore, some specialists suggest that ablative therapy be considered experimental (Spechler, 2002a). Many of the ablative modalities require multiple treatments with endoscopic surveillance. Although more research is needed, certain patient populations may be better served utilizing an endoscopic ablative therapy, including those who are poor surgical candidates or too frail to tolerate an esophagectomy (Spechler, 2002b).

Chemical Ablative Techniques

Photodynamic Therapy

Photodynamic therapy (PDT) involves the administration of a chemical photosensitizing drug, administered intravenously, that accumulates in the specialized intestinal metaplastic mucosa (Johnston, 2005; Phan et al., 2005). Within 48 hours, a laser light source is introduced endoscopically that activates the photosensitive abnormal mucosa, resulting in the generation of singlet oxygen, which is toxic to those cells causing cellular and vascular destruction (Johnston, 2005; Phan et al., 2005). Porfimer sodium, the only photosensitizing medication approved for use in the United States by the Food and Drug Administration, remains in the body for up to 2 months (Phan et al., 2005). During PDT, patients remain very photosensitive; therefore, extra precautions should be taken to avoid any exposure to sunlight (Johnston, 2005).

Analysis of the effectiveness of PDT demonstrated a high success rate, with up to 78% eradication of the superficial malignancy following repeated treatments; however, approximately 4.6% of the subjects in the same study went on to develop subsquamous adenocarcinoma (Johnston, 2005). Adverse effects of PDT include strictures, chest pain, dysphagia, odynophagia, pleural effusions, and atrial fibrillation (Johnston, 2005). Esophageal strictures occurred in 34% of the patients, requiring one or more dilatations (Spechler, 2002b).

TABLE 3. Management and Treatment Options for Barrett's Esophagus

Medications
Proton pump inhibitors
Endoscopic mucosal ablation
Mechanical
Endoscopic mucosal resection
Thermal
Lasers
Argon plasma coagulator
Heater probe
Cryotherapy
Chemical
Photodynamic therapy
Surgery
Esophagectomy

From "What You Need to Know About Barrett's Esophagus," A. D. Rockey, 2002, *Gastroenterology Nursing, 25*(6), pp. 237-240. Copyright 2002 by the Society of Gastroenterology Nurses and Associates, Inc. Reprinted with permission.

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Thermal Ablative Techniques

Argon Plasma Coagulation

This method of ablative endoscopic therapy uses high-frequency monopolar current into the mucosa via ionized argon gas flowing through a catheter placed into the distal esophagus to burn the metaplastic tissue (Shaheen, 2005b). The depth of injury to the mucosa is less than that with PDT and complications include pneumatosis, pneumoperitoneum, subcutaneous emphysema, pain, ulceration, stricture, bleeding, perforation, and even death (Johnston, 2005). Success of this therapy has been reported in 38%-98% of the patients within a 12- to 51-month time range. Subsquamous specialized intestinal metaplasia (SIM) is reported in 0%-30% of the patients (Johnston, 2005; Shaheen, 2005b).

Multipolar Electrocoagulation

Multipolar electrocoagulation (MPEC) delivers thermal energy to the abnormal Barrett's mucosa through a probe that delivers electrical current between two or more electrodes. The probe is passed through the endoscope and half of the circumference of the esophagus is treated at a time, using the other side as an internal control (Shaheen, 2005b). The overall success rate is reported to be 75% when a concomitant PPI is given and multiple treatments are delivered (Johnston, 2005). The side effects of MPEC include dysphagia, odynophagia, and chest pain lasting up to 4 days. The success of ablation decreases significantly once the length of the Barrett's esophagus exceeds 4 cm (Johnston, 2005).

Laser Therapy

Laser therapy generates an intense beam of light that is directed at the abnormal mucosa and used to burn the dysplastic tissue (Shaheen, 2005b). The ablative depth of injury depends on the type of laser and ranges from 1 to 4 mm. Studies vary on the efficacy of this treatment, including a wide variation (18%-100%) for the successful treatment of Barrett's esophagus (Johnston, 2005). Complications are similar to those with MPEC except there is a higher esophageal stricture rate (Johnston, 2005).

Radiofrequency Ablation

This balloon-based, bipolar radiofrequency ablation technique is fairly new. Sizing balloons are used to determine the inner diameter of the Barrett's esophagus tissue. The actual balloon-based catheter contains an electrode with a 3-cm long treatment area that incorporates tightly spaced, bipolar electrodes that alternate in polarity. The electrode is attached to a radiofrequency generator and a preselected amount of energy is delivered in <1 second at 350 W. Research has shown full-thickness ablation of the epithelium without direct injury to the submucosa. There is no long-term data available on the effectiveness of this therapy. Side effects include throat pain, odynophagia, and dysphagia (Johnston, 2005).

Cryoablation

This experimental therapy utilizes a low-pressure spray with liquid nitrogen and is one of the newest endoscopic ablative therapies. An open-tipped 7-9 Fr catheter is used to spray supercooled nitrogen gas to freeze the abnormal intestinal metaplasia (Johnston, 2005). Cryoablation induces apoptosis and cryonecrosis at supercooled temperatures, −76 °C to −158 °C, which results in transient ischemia at the cryoablation site and immune stimulation. "Barrett's epithelium is resistant to apoptosis and therefore might be uniquely suited for treatment by cryoablation" (Johnston, 2005, p. 326). Preliminary results show that Barrett's esophagus was reversed in 9 of 11 patients, with varying lengths of Barrett's esophagus between 1 and 8 cm (mean length = 4.6 cm) (Johnston, 2005).

Mechanical Ablation

Endoscopic Mucosal Resection

Mucosectomy involves the removal of the damaged mucosa by the use of a blended electrosurgical current (Johnston, 2005). This procedure involves injection of a dilute solution of epinephrine or normal saline into the submucosa creating a pseudopolyp, and the engorged mucosa is then snared and removed by electrocaudery (Shaheen, 2005b). The advantage of endoscopic mucosal resection is the ability to obtain tissue for histology and define tissue margins (Johnston, 2005). The effectiveness of this ablative technique is not clearly defined. Endoscopic mucosal resection may be indicated for superficial, well-differentiated, or moderately differentiated squamous cell carcinoma; however, multiple endoscopic procedures may be necessary for complete removal of the Barrett's epithelium (Johnston, 2005; Shaheen, 2005b).

Multimodality Therapy

More research is needed to determine which endoscopic treatment regimen is the most successful in the abolishment of Barrett's esophagus. Clinical trials are currently taking place to compare and evaluate the safety and efficacy of current and experimental therapies. There is no gold standard for treatment, and little proven research regarding the effects of standard medical, surgical, and endoscopic treatments of Barrett's esophagus. Acid suppression therapy is a mainstay in the treatment of Barrett's esophagus, and when paired with mucosal ablation therapy, neosquamous reepitheliazation

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occurs (Dulai, Jensen, Cortina, Fontana, & Ippoliti, 2005).

The average thickness of Barrett's mucosa is about 1.5 mm and involves the epithelium, lamina propria, and the muscularis mucosa (Johnston, 2005). The depth of tissue injury reported in the literature varies for each ablative therapy yet is critical to eradicate the SIM, while trying to avoid complications such as strictures, perforation, and bleeding (Dulai et al., 2005; Johnston, 2005). The occurrence of residual subsquamous SIM after ablation therapy is significant in spite of the associated development of intramucosal adenocarcinoma arising under the new epithelium even with histological clearance (Johnston, 2005).

As part of the multimodality approach to the treatment of Barrett's esophagus, aspirin and other nonsteroidal anti-inflammatory drugs are being researched for implications in the reduction of esophageal cancer (Sampliner, 2002). The strong relationship between chronic inflammation and cancer development has provided new venues to explore the inhibitory effect of such medications in chemoprevention (Aldulaimi & Jankowski, 1999). The AspECT trial is a 10-year trial studying the effect of aspirin and esomeprazole in the role as chemoprotective agents to determine if it is possible to prevent the progression of Barrett's esophagus to adenocarcinoma (University Hospital of Leicester, 2005-2008).

Patient Preferences

Active participation of the patient in healthcare decisions is essential for optimal outcomes. A shared decision-making approach allows a bidirectional exchange of information with participation, active listening, and agreement on the course of treatment (Montori, Gafni, & Charles, 2006). In a pilot study by Hur et al. (2005), 26 patients with Barrett's esophagus were asked which treatment they would prefer: frequent surveillance endoscopy, PDT, or esophagectomy. All of the participants had a history of Barrett's esophagus, although none had HGD. The study, which used a "feeling thermometer," asked the patients to imagine if they had HGD, which management therapy would they pick. Of the 20 participants who responded, 70% chose the frequent surveillance endoscopy method, 15% chose the esophagectomy, and 15% chose PDT. The study results revealed that patients overwhelmingly choose endoscopic surveillance on the basis of knowledge of the procedure and comfort level. See Figure 2 for a Barrett's esophagus treatment algorithm.

Nursing Considerations

The goal of treatment of Barrett's esophagus includes acid suppression therapy with a PPI; however, patient education with regard to diet, lifestyle changes, and disease management is also significant. Patient education regarding dietary changes should include avoiding foods that increase acid secretion or decrease the LES pressure (NGC, 2007). For example, tomato, garlic, citrus fruits, spices, onions, peppermint, chocolate, caffeine, and alcohol all increase gastric acid. Patients should be encouraged to avoid large meals or lying down after eating for 2-3 hours (NGC, 2007). Smoking cessation and weight loss should be encouraged. Smoking results in air swallowing and an increase in acid exposure, whereas a high BMI increases intra-abdominal pressure (Rockey, 2002). Nurses should be aware of medications that lower the LES pressure, such as calcium channel blockers, barbiturates, and theophylline (NGC, 2007). Patients taking PPIs should take this medication half an hour before breakfast (Rockey, 2002).

In the case study of Mr. C., he was discharged from the hospital with a follow-up appointment with the gastroenterologist. During his appointment, the physician revealed that his biopsy results did show Barrett's esophagus with HGD. The treatment options were discussed, with identification of the advantages and disadvantages of frequent surveillance endoscopy, ablative therapy, and surgery. Mr. C. chose frequent surveillance endoscopy and follow-up with an oncologist for additional treatment recommendations. His nursing care included education regarding diet, lifestyle changes, and disease management including management of medications.

Conclusion

Research confirms that the significance of developing a screening program for detecting Barrett's esophagus is based on results demonstrating 94%-98% of patients diagnosed with esophageal adenocarcinoma have no known history of Barrett's esophagus (Fass & Sampliner, 2003). The goal of a surveillance program is to identify these patients with Barrett's esophagus to reduce the mortality through early detection. Endoscopic surveillance of patients with Barrett's esophagus requires a better risk stratification and diagnosis through endoscopic techniques. The use of endoscopic treatment modalities for patients with HGD is warranted in specialized cases, such as nonsurgical candidates, and unifocal disease (Shaheen, 2005b).

Much of the literature concludes that ablative therapy is expensive, technically demanding, and experimental, with varying results; therefore, it has not been proven to be superior for the general population (Dulai et al., 2005; Spechler, 2002a). Research has also not shown that these treatments decrease the long-term risk of cancer; therefore, more long-term randomized trials are needed to determine risk stratification strategies to aid in the process of screening and surveillance,

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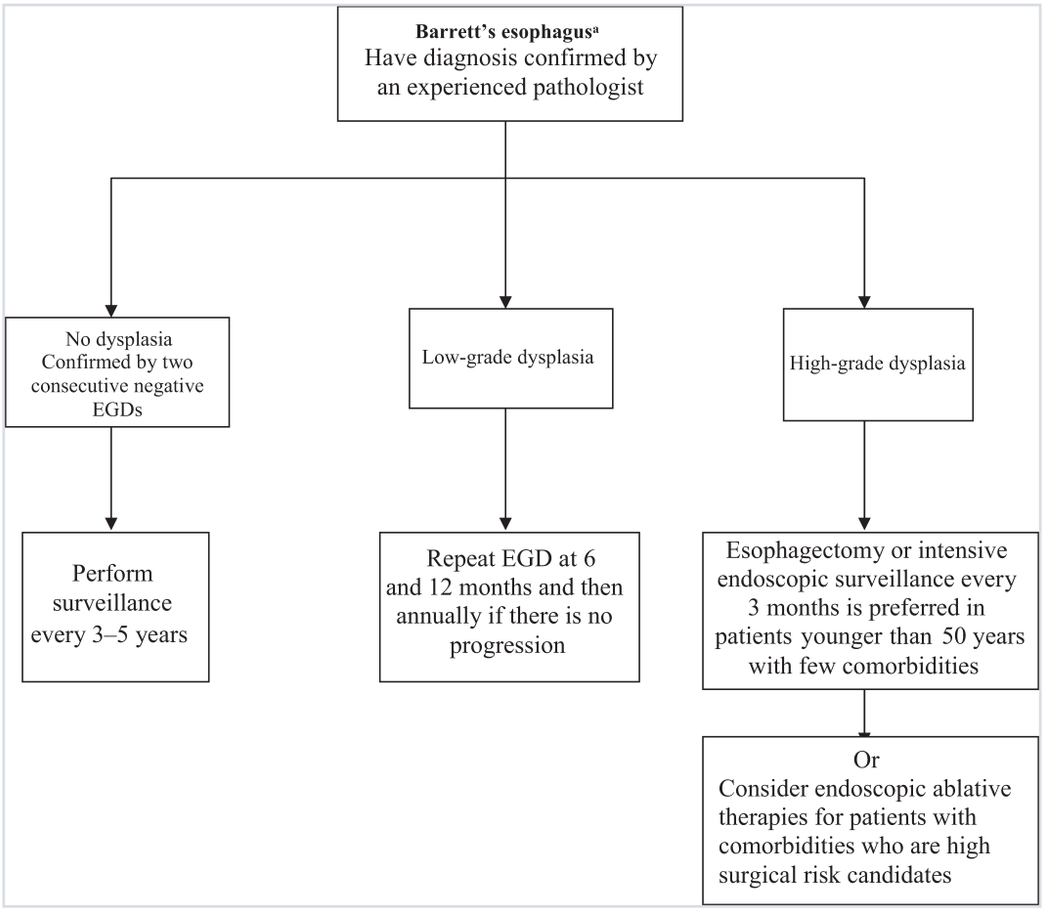

FIGURE 2. Barrett's esophagus treatment algorithm. EGD = esophagogastroduodenoscopy.
[a]All patients with Barrett's esophagus should be on high-dose proton pump inhibitor therapy. Adapted from "Updated Guidelines for the Diagnosis, Surveillance, and Therapy of Barrett's Esophagus," by R. E. Sampliner, 2002, *The American Journal of Gastroenterology, 97*(8), pp. 1888-1195. Copyright 2002 by the American College of Gastroenterology.

treatment guidelines designed to meet specific patient populations, and prevention techniques for the progression of Barrett's esophagus to neoplasm in the hope of providing better patient outcomes (Gopal et al., 2004; Spechler, 2002a).✪

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