

Barrett's Esophagus: Signs, Progression and Treatments

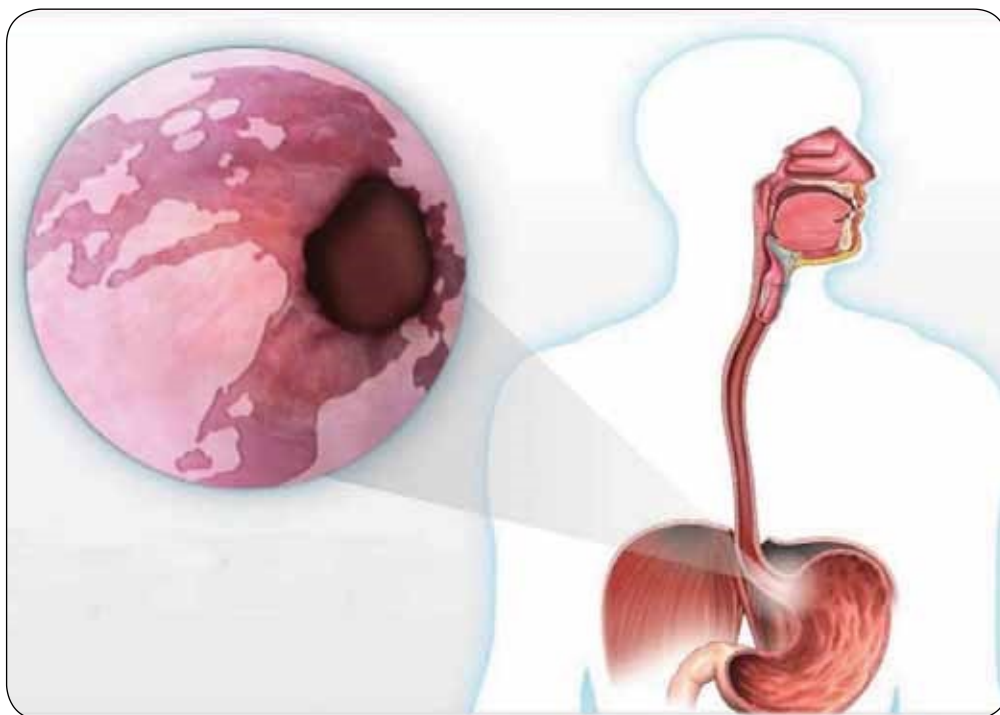
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Many of us are familiar with gastroesophageal reflux disease (GERD) and its uncomfortable symptoms. A number of sufferers deal with a more serious result of the acid reflux that is a hallmark of this condition. Chronic GERD can cause Barrett's esophagus (BE), a premalignant condition that can be difficult to manage, depending on the severity of the disease process. Barrett's esophagus is characterized by metaplastic changes in esophagus as the tissue heals. The cells change from squamous mucosa, normally found in the lining of the esophagus and on the surface of the skin, to columnar epithelium, a more absorptive type of cell commonly found in the stomach and intestines. The transformed cells are actually more resistant to damage from acid reflux, but they are also more susceptible to carcinogenesis, making Barrett's esophagus the most commonly identified risk factor for developing esophageal adenocarcinoma.

Barrett's esophagus is named for Norman Barrett, an Australian surgeon who first described the condition in 1950. The Library of Congress officially codes the condition for electronic searches as "Barrett esophagus" (likewise, in transcription the standard is to drop the possessive form for eponyms), but it's universally

termed "Barrett's esophagus" (with an apostrophe and "s" on the end), including in the ICD-10-CM codebook.

Since Barrett's esophagus has no outward presenting signs, the condition is often diagnosed during endoscopic evaluation performed for symptoms of GERD, usually in middle-aged and older adults. Barrett's esophagus is most commonly found in white men. Black and Asian populations have very low occurrences. Obesity is also a risk factor for the condition, especially when the weight is carried around the middle, possibly because of increased abdominal pressure worsening gastroesophageal reflux. Cigarette smoking is also associated with a modest increased risk for the condition, although alcohol consumption has been disproved as a factor. Interestingly, consumption of green leafy vegetables may also contribute to the increase in esophageal adenocarci-



noma. This may be related to nitrate-based fertilizers in Western countries (Spechler, 2016 December).

Although all researchers would agree that the findings of columnar epithelium in the esophagus is indicative of Barrett's esophagus, some authorities argue that a gastric cardia-type epithelium, which is also composed of mucus-secreting cells also indicates the condition. This variation is still a matter of debate. There are, however, two accepted categories of BE: long-segment and short-segment. These are simply categories of measurement, with long-segment BE extending for 3 cm or more above the esophagogastric junction (EGJ) and short-segment BE extending less than 3 cm above the EGJ. Short-segment BE is, by far, the most common type.

Diagnosis of Barrett's esophagus is difficult, even from a histologic point of view. Endoscopic tissue samples are usually taken randomly from four quadrants of the esophagus to provide the best possible representation of cell type. BE can exist with or without dysplasia, or early cancerous changes. Dysplasia is categorized as high-grade or low-grade, depending on the degree of abnormality found. However, reactive changes from reflux esophagitis are easy to mistake for the abnormal proliferation of cell changes that constitute dysplasia, and agreement by a second pathologist is often required to make a definitive low-grade dysplasia diagnosis. Metaplasia is the replacement of one mature type of cell with another, and that is the next step toward neoplasm. Because the changed cells in Barrett's esophagus have a higher risk of neoplastic changes, patients with Barrett's may have a 30- to 40-fold risk of developing esophageal adenocarcinoma than the general population (Lekakos, 2011 Oct 7).

Statistics for BE and its resulting effect on the rate of esophageal adenocarcinoma are sometimes confusing.

- Barrett's esophagus affects 1.3%–2% of the adult population (Martinucci & al, 2016 May 6).
- 3%–5% of patients who have endoscopy because of GERD symptoms are found to have long-segment BE, and 10%–20% are found to have short-segment BE (Spechler, 2016 December).
- The incidence of esophageal adenocarcinoma has increased seven-fold in Western populations over the past few decades (Spechler, 2016 December).
- Despite the increase in esophageal adenocarcinoma, the total occurrence of this condition in the population is 0.3% per year (Martinucci & al, 2016 May 6), and most patients with cancer do not die of esophageal cancer, rather of another cause.

As you can see, the risk of Barrett's esophagus is quite low, and the risk of it progressing to adenocarcinoma after treatment is very small, even though treatment options still have not been perfected. However, the incidence of esophageal adenocarcinoma is increasing in the United States and, although it is still a relatively rare cancer, five-year survival rates for those diagnosed fall between 4%–40% (American Cancer Society, 2016).

The most common form of esophageal cancer in the United States is adenocarcinoma, which is related to progression of Barrett's esophagus. For at least 20 years, researchers have noticed a significant increase in esophageal adenocarcinoma, particularly among white males. Because BE and, in turn, esophageal adenocarcinoma, have such a strong association with this one demographic group, there is thought to be a genetic component. Interestingly, the presence of *Helicobacter pylori* (*H. pylori*), a known risk factor for gastric cancer, appears to decrease the risk of developing Barrett's esophagus and esophageal carcinoma. One theory is that an *H. pylori* infection decreases gastric acid secretion and, therefore, limits any progression of GERD. When the infection is successfully treated, acid reflux may worsen, which contributes to Barrett's esophagus.

Treatment for Barrett's esophagus is rapidly changing as newer technology is developed. Treatment is determined by the degree of dysplasia and the extent of the condition. The gold standard was once an esophagectomy for patients with high grade dysplasia. Aspirin and NSAIDs have been studied to prevent progression of disease, but NSAIDs have not been sufficiently proven to have a significant effect, and aspirin comes with its own risks. In patients with heart disease as well as BE, the aspirin may provide treatment for both conditions and be worth the risk. Proton pump inhibitors (PPIs) are also used to reduce the amount of gastric reflux, which can lead to healing of the damage caused by the acid.

There are a number of endoscopic and other surgical techniques regularly used in treatment of Barrett's esophagus and adenocarcinoma. Because esophagogastroduodenoscopy (EGD) is the tool used to diagnose both conditions, regular endoscopic surveillance to monitor the results of medicinal treatments has been used. However, there are risks associated with endoscopy, as well, especially if esophageal tissue becomes friable from continued damage. Also, superficial layers of tissue may cover remaining disease, which would not be visible to the endoscope. Esophagectomy is, of course, guaranteed



to excise all diseased tissue, but it is a radical solution, with high morbidity and mortality and is now not justified if the condition is not very advanced and still amenable to less invasive techniques. Endoscopic ablative therapy with either heat, cold or photochemical means has become the treatment of choice for most patients. It can eradicate diseased tissue, but it doesn't allow for any pathology specimens to determine if the disease was completely removed. Endoscopic mucosal resection (EMR) does provide tissue for a pathologist to examine. In recent times, these two procedures have been combined and termed endoscopic eradication therapy. This technique allows the endoscopist to ablate obviously diseased segments and then take biopsies of remaining tissue to determine that all margins are clear. Following any of these procedures patients are normally treated with long-term PPIs to prevent future damage in hopes of preventing progression to cancer.

There are experimental treatments and diagnoses being considered in this field, including:

- Chromoendoscopy – using dyes/stains and light wavelengths to highlight diseased tissue
- Autofluorescence endoscopy – imaging technique using natural tissue fluorescence
- Magnification endoscopy – high-resolution magnified images more easily identify biopsy sites
- Narrow band imaging – optical image enhancement to more easily visualize tissue structures
- Optical coherence tomography – high-resolution cross-sectional imaging for mucosal tissue
- Raman spectroscopy – molecule identification by measurement of light wavelength
- Confocal laser endomicroscopy – endoscopic imaging that displays at 1000-fold magnification

Prevention and treatment of Barrett's esophagus and any possible resultant esophageal adenocarci-

noma is dependent on reliable diagnostic techniques, standardization of the criteria for diagnosis, and reliable methods of ablation and surveillance for recurrence. These are all procedures that continue to evolve. Patients with risk factors for these conditions should also avoid behaviors that contribute to overactive gastric acid production. Researchers also continue to look for genetic markers for these conditions so better screening and prevention techniques can be implemented. **P**

Jennifer has worked in the allied health care industry for more than 20 years in every capacity from receptionist to practice manager. Currently, she writes and edits courses and study guides about clinical subjects, medical coding, transcription, electronic health records and the use of technology in health care. She also regularly contributes online and print articles about these topics and has actively helped prepare industry professionals for the conversion to the ICD-10 coding system.

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