The Barrett esophagus (BE) was first described in 1950 by a British surgeon whose name has been given to this condition. Barrett believed the condition to be present at birth but in 1953 Allison and Johnston postulated that in some instances the condition might be acquired. In 1961 Hayward considered the condition to be acquired and elegantly described the mechanism of its development as a consequence of destruction of the normal lining squamous cells (mucosa) of the esophagus by repetitive reflux of stomach acid over several years. The columnar cells are a variant of normal, so-called metaplastic type, which have the potential over time to become dysplastic (abnormal growth) and later evolve into adenocarcinoma (cancer). Knowledge of the stages of this sequence provides the opportunity for detecting those persons who develop dysplasia and have the highest risk to develop cancer in the BE.

BE has been recognized with increasing frequency over the past four decades and there is currently no explanation for this phenomenon. Obviously, some of this increase in prevalence is related to the wider use of endoscopy, as well as physician and public education. Likewise the esophageal cancer (adenocarcinoma) that occurs with BE has remarkably increased in frequency. Forty years ago less than 5% of esophageal cancers were of the adenocarcinoma type and 95% were squamous cell cancer. Today over 70% of esophageal cancers are of the adenocarcinoma type and nearly all of these are associated with BE. Both BE and adenocarcinoma are primarily diseases of white males between 40 and 80 years of age. We have seen BE also in young persons (teenagers) with a history of acid reflux symptoms (heartburn) for several years.

Pathogenesis (mechanisms occurring in development of disease)

Research in animals has shown that when acid injury results in destruction of the normal esophageal surface tissue (squamous mucosa), this tissue cannot regenerate if the exposure to acid reflux persists. The Barrett epithelium (intestinal metaplasia) represents the body’s ability to adapt by developing this type of acid resistant tissue to cover the acid-damaged segment. The presence of this tissue is therapeutic since it allows healing and is less sensitive to acid. Therefore, after BE develops these patients tend to have less severe heartburn than many patients without BE. The reduced severity of heartburn is deceptive since it does not correlate with the severity of the occult Barrett esophagus and the potential for development of dysplasia and cancer. Patients with the longer segments of BE (greater than 3 cm) tend to have a larger hiatal hernia, very low pressure in the lower esophageal sphincter, decreased esophageal motility (equates to reduced clearance of refluxed acid), and both recumbent and upright acid reflux. Patients with short segments of BE (less than 3 cm) have lesser degrees of these defects. Long segments of BE usually are associated with a significantly longer duration of heartburn than with short segments and have a higher risk of developing dysplasia and cancer.

Can Dysplasia and Cancer Be Prevented?

Currently there is no confirmation by appropriate research studies that acid suppression therapy or antireflux operations (surgical or endoscopic) can alter the presence of a segment of BE or reduce the risk of dysplasia and cancer. There is some suggestion based on retrospective studies that long-term acid suppression may reduce the risk of dysplasia and cancer. Research is ongoing but as yet no safe, effective and relatively inexpensive chemo-preventive drug has been developed. Research in animal models is ongoing to test for a possible chemo-preventive effect by difluoromethylornithine (DFMO), cyclo-oxygenase II (Cox-II) inhibitors and acetylsalicylic acid (aspirin) in BE. Clinical research in humans is underway to test whether the Cox-II inhibitor class of anti-inflammatory drugs (such as Celebrex, Vioxx) can prevent development of invasive cancer in patients with BE and high-grade dysplasia.
**Ablation of Barrett Esophagus**

Esophagectomy for high-grade dysplasia or epithelial (non-invasive) cancer associated with BE has been a standard therapy for many years. This surgical procedure carries a relatively high risk and when indicated should be done only by an experienced surgeon who regularly performs this operation. Clinical experience has shown in recent years that alternative therapies can eliminate high-grade dysplasia and focal, superficial cancers without the risks associated with esophagectomy. Photodynamic laser therapy (PDT) has proven safe and effective in a large multicenter study of carefully evaluated patients. There are some significant complications but these are less common when PDT is performed by experts. Esophageal strictures occur in about 25% of cases, are severe in some, but usually are managed simply by dilation. Most importantly, the esophagus remains intact and most of the BE segment is destroyed and replaced by a more normal squamous epithelium. Long-term endoscopic surveillance biopsy is considered essential after this therapy. In a series reported by Overholt, the incidence of developing cancer after PDT ablation in patients with high-grade dysplasia was 4% compared to the expected incidence of 25-50% in patients with untreated high-grade dysplasia.

Thermal ablation methods include multipolar electrical coagulation (MPEC), heater probe and argon plasma coagulation (APC) which have been used in some studies but require multiple sessions and are less likely to destroy as much BE and tissue dysplasia as PDT.

The most recent procedure used to improve therapy for BE with identifiable focal areas of high-grade dysplasia is endoscopic mucosal resection (EMR). This technique uses a snare through the endoscope with an electrical current to remove identifiable focal areas (nodules, polyps) that contain superficial cancer or high-grade dysplasia. The typical patient with acid reflux controls symptoms by using baking soda, over-the-counter antacids and H2 blockers (Tagamet, Pepcid, Zantac) and has never bothered to seek medical attention. This is probably why most patients with BE who develop adenocarcinoma are never seen by a gastroenterologist before the cancer develops. We can predict that with the greater potency of acid suppressing drugs that are becoming available over-the-counter, the higher the likelihood it will be to see patients with undiagnosed BE who will have heartburn controlled but ultimately present to a physician with symptomatic cancer. There is yet no proof that control of heartburn by medication or surgery reduces the risk of dysplasia and cancer in a patient with BE. Once the patient develops difficulty swallowing, anemia, weight loss or other so called alarm symptoms of cancer the chances for cure are significantly reduced. The current belief is that early diagnosis of BE and close follow-up by endoscopy and biopsy to detect the pre-cancerous tissue changes (dysplasia) offer the best chance for early curative treatment. Early diagnosis of GERD and adequate acid suppression therapy will prevent acid reflux complications as well as BE. In the absence of BE there is little chance of developing cancer (adenocarcinoma).

In patients with BE the gastroenterologist usually prescribes a proton pump inhibitor drug (Aciphex, Nexium, Prevacid, Prilosec, Protonix) to suppress stomach acid and thereby reduce the complications of GERD and hopefully, the progression of BE to dysplasia and cancer. Whether future development of cancer in BE can be prevented or delayed by maximum suppression of acid is unknown. Evidence to date indicates that as long as the abnormal tissue of BE is present in the esophagus the threat of dysplasia and cancer remains. Early diagnosis of BE by endoscopy and biopsy followed by proper therapy and surveillance will detect high-grade dysplasia and permit ablative therapy, or in some cases surgery, before invasive cancer develops.

**THE MYTH AND CHALLENGE OF THE HIATAL HERNIA**

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Many of us have heard others confess that they have been diagnosed or discovered to have a hiatal hernia and that it was likely causing them to feel sick. Hiatal hernia has been blamed for symptoms of fatigue, nausea, indigestion and abdominal distress after consuming large quantities of alcohol or food. Some individuals have even reported that their hiatal hernia causes them to experience pain when they move their body a certain way. Rare individuals report that their hiatal hernia may or may not be present at different times when barium x-rays are performed. A hiatal hernia remains with the individual throughout that person’s lifetime.

Sir Astley Cooper is credited with the definition of a hernia as “a protrusion of any viscus (soft tissue) from its proper cavity,” i.e., a protrusion of an organ through a wall of a cavity in which it is enclosed. The word hiatus is derived from Latin meaning a gap, cleft or opening. When a portion of the upper stomach protrudes or extends above the hiatus more than 2 centimeters (about 1 inch), which is normally located at the level the esophagus and the stomach join, a hiatal hernia is diagnosed. (Figure 1A & 1B) Hiatal hernias may be either congenital (present since birth) or acquired.

Congenital hernias of the diaphragm (thin breathing muscle that separates the chest cavity from the abdominal cavity) appear in 1 of 2200 births. They have been reported in 8% of autopsies of infants with other anomalies and in 1.4% of all newborn deaths. The complications of congenital diaphragmatic hernias are respiratory and intestinal. Congenital hernias should be surgically corrected.

The majority of hiatal hernias are of the acquired type and appear later in life. Possible causes of acquired hiatal hernias include: 1) esophageal contraction (shortening) secondary to gastroesophageal acid reflux or other esophageal injury with pulling-up of the stomach above the diaphragm; 2) increased intra-abdominal pressure pushing the stomach above the diaphragm (obesity with excessive abdominal fat, during pregnancy, straining during bowel movements or during periods of heavy lifting, pushing and pulling or abdominal trauma); 3) atrophy (breakdown or deterioration) or weakening of the
frequent increases in intra-abdominal pressure caused by strain-sexes with increasing age. Burkitt and James have proposed that hiatal hernias are more common in women and in both gastrointestinal tract in the Westen world. There is evidence finding of a widened or patulous hiatus is another reliable sign of the presence of a hiatal hernia. A hiatal hernia is present in most if not all individuals with complications of gastroesophageal reflux disease (GERD).

Dodd provided us with valuable evidence in his statement: “Although many patients with hiatal hernia do not exhibit reflux esophagitis, most patients with reflux esophagitis have an axial hiatal hernia”. In other words, most individuals that have a hiatal hernia have no symptoms of esophageal or stomach disease. On the other hand, the majority of those who have severe esophageal injury (esophagitis, ulceration, strictures, or Barrett esophagus) will have a hiatal hernia. The precise relationships between hiatal hernia, lower esophageal sphincter (valve) function and dysfunction, and gastroesophageal reflux disease have not been determined.

Typically every effort is undertaken to manage patients with hiatal hernia medically. In one report only thirty-one of the 786 patients studied with hiatal hernia (3.9 per cent) were specifically referred to the surgeon for treatment. All patients were informed that their hiatal hernia was an anatomic derangement that would persist throughout their life. For this reason, patients were advised that their initial management would be based simply on altering habits and living routines (i.e. lifestyle modifications). All patients with hiatal hernia, whether symptomatic or not, were strongly advised to lose weight if they were at all above a minimal normal weight. Surgical treatment should be elected only when certain complications of GERD recur or persist despite medical strategies and the addition of acid suppressing drug therapies.

In summary, the hiatal hernia alone rarely causes symptoms directly (e.g., incarceration with pain or bleeding); it is the anatomic companion of all the major complications of gastroesophageal reflux. Fortunately, proper medical (acid suppression) therapy with drugs controls acid reflux-related problems sufficiently well that surgical intervention is needed in fewer than 5% of patients. A significant part of the surgical procedure (fundoplication) includes replacement of the herniated segment of stomach as close as possible to its normal location below the diaphragmatic hiatus. Proton pump inhibitors (Aciphex, Nexium, Prevacid, Prilosec, and Protonix) provide the best medical acid suppression therapy for individuals with symptomatic GERD that is not controlled by lifestyle modification strategies.

It is important to remember the following: 1) the presence of a hiatal hernia may contribute to symptoms of GERD but is rarely a direct cause of symptoms; 2) hiatal hernia is a common occurrence in Western civilization during the aging process and with excessive weight gain; 3) surgical therapy specifically directed at the hiatal hernia is rarely needed to control uncomplicated symptoms; and 4) as we increase our knowledge concerning the significance of a hiatal hernia we can extinguish the myths surrounding its presence and appropriately withhold any unnecessary surgical operations.
Things To Remember

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