Patient’s Guide to Barrett’s Esophagus

**What is Barrett’s Esophagus?**

Barrett’s esophagus is a precancerous condition and refers to metaplastic conversion of normal squamous epithelium of tubular esophagus (see figure a) to columnar epithelium (figure b). Normally the gastroesophageal junction (GEJ) divides the esophagus and stomach and corresponds to squamocolumnar junction (SCJ “Z-lin”). The esophagus is lined by stratified squamous epithelium. The stomach is lined by columnar epithelium. In some chronic injury setting such as gastroesophageal reflux disease (GERD), the esophageal squamous epithelium goes through a metaplastic process and is converted to a columnar epithelium with goblet cells (which are usually found lower in the gastrointestinal tract and thus also called “intestinal metaplasia”).

The main cause of Barrett's esophagus is thought to be an adaptation to chronic acid exposure from GERD. GERD is associated with a 10–15% risk of Barrett’s esophagus. Other risk factors associated with the development of Barrett’s esophagus include male gender, central obesity and age over 50 years.





**What is the clinical complication of Barrett’s Esophagus?**

Barrett’s esophagus is the only recognized precursor of esophageal adenocarcinoma which incidence has been increasing in the United States since 1970s. Patients with Barrett's esophagus have a 30-125 fold higher risk of developing cancer of the esophagus than the general population.

**How is Barrett’s Esophagus diagnosed?**

Diagnosis of Barrett’s esophagus requires esophagogastroduodenoscopy (a procedure in which a fibreoptic cable is inserted through the mouth to examine the esophagus, stomach, and duodenum) and biopsy of esophageal lining. According to American College of Gastroenterology guideline, Barrett’s esophagus should be diagnosed when there is extension of salmoncolored mucosa into the tubular esophagus extending ≥1 cm proximal to the gastroesophageal junction (GEJ) with biopsy confirmation of intestinal metaplasia.

Barrett's esophagus, based on the histopathology, is classified into five categories: nondysplastic, indefinite for dysplasia, low-grade dysplasia, high-grade dysplasia, and adenocarcinoma.

**Who should be screened for Barrett’s Esophagus?**

Screening for Barrett's esophagus may be considered in men with chronic (>5 years) and/or frequent (weekly or more) symptoms of gastroesophageal reflux (heartburn or acid regurgitation) and two or more risk factors for Barrett's esophagus or esophageal adenocarcinoma. These risk factors include: age >50 years, Caucasian race, presence of central obesity (waist circumference >102 cm or waist–hip ratio (WHR) >0.9), current or past history of smoking, and a confirmed family history of Barrett's esophagus or esophageal adenocarcinoma (in a first-degree relative).

Given the substantially lower risk of esophageal adenocarcinoma in females with chronic GERD symptoms (when compared with males), screening for Barrett's esophagus in females is not recommended. However, screening could be considered in individual cases as determined by the presence of multiple risk factors for Barrett's esophagus or esophageal adenocarcinoma (age >50 years, Caucasian race, chronic and/or frequent GERD, central obesity: waist circumference >88 cm, WHR >0.8, current or past history of smoking, and a confirmed family history of Barrett's esophagus or esophageal adenocarcinoma (in a first-degree relative).

Screening of the general population is not recommended.

If initial endoscopic evaluation is negative for Barrett's esophagus, repeating endoscopic evaluation for the presence of Barrett's esophagus is not recommended. If endoscopy reveals esophagitis, repeat endoscopic assessment after proton pump inhibitor therapy for 8–12 weeks is recommended to ensure healing of esophagitis and exclude the presence of underlying Barrett's esophagus.

**What is the surveillance of patients with Barrett’s Esophagus**?

The goal of a screening and surveillance program for Barrett's esophagus is to identify individuals at risk for progression to esophageal adenocarcinoma.

For Barrett's esophagus patients without dysplasia, endoscopic surveillance should take place at intervals of 3 to 5 years.

Patients diagnosed with Barrett's esophagus on initial examination do not require a repeat endoscopy in 1 year for dysplasia surveillance.

For patients with indefinite for dysplasia, a repeat endoscopy after optimization of acid suppressive medications for 3–6 months should be performed.

If the indefinite for dysplasia reading is confirmed on this examination, a surveillance interval of 12 months is recommended.

For patients with confirmed low-grade dysplasia and without life-limiting comorbidity, endoscopic therapy is considered as the preferred treatment modality, although endoscopic surveillance every 12 months is an acceptable alternative.

Patients with Barrett's esophagus and confirmed high-grade dysplasia should be managed with endoscopic therapy unless they have life-limiting comorbidity.

**How is Barrett’s Esophagus treated?**

Treatments for Barrett's esophagus include chemoprevention, endoscopic therapy and surgical therapy.

***Chemoprevention***

Patients with Barrett's esophagus should receive once-daily proton pumper inhibitor (PPI) therapy.

Aspirin or NSAIDs should not be routinely prescribed to patients with Barrett's esophagus as an antineoplastic strategy. Similarly, other putative chemopreventive agents currently lack sufficient evidence and should not be administered routinely.

***Endoscopic therapy***

Patients with nodularity in the Barrett's esophagus segment should undergo endoscopic mucosal resection of the nodular lesion(s) as the initial diagnostic and therapeutic maneuver. Histologic assessment of the endomucosal resection (EMR) specimen should guide further therapy. In subjects with EMR specimens demonstrating high grade dysplasia, or intramucosal carcinoma, endoscopic ablative therapy of the remaining Barrett's esophagus should be performed.

In patients with EMR specimens demonstrating neoplasia at a deep margin, residual neoplasia should be assumed, and surgical, systemic, or additional endoscopic therapies should be considered.

Endoscopic ablative therapies should not be routinely applied to patients with nondysplastic Barrett's esophagus because of their low risk of progression to esophageal adenocarcinoma. Endoscopic eradication therapy is the procedure of choice for patients with confirmed low grade dysplasia, and confirmed high grade dysplasia.

In patients with T1a esophageal adenocarcinoma, endoscopic therapy is the preferred therapeutic approach, being both effective and well tolerated.

In patients with T1b esophageal adenocarcinoma, consultation with multidisciplinary surgical oncology team should occur before embarking on endoscopic therapy. In such patients, endoscopic therapy may be an alternative strategy to esophagectomy, especially in those with superficial (sm1) disease with a well-differentiated neoplasm lacking lymphovascular invasion, as well as those who are poor surgical candidates.

In patients with known T1b disease, EUS may have a role in assessing and sampling regional lymph nodes, given the increased prevalence of lymph node involvement in these patients compared with less advanced disease.

In patients with dysplastic Barrett's esophagus who are to undergo endoscopic ablative therapy for nonnodular disease, radiofrequency ablation is currently the preferred endoscopic ablative therapy.

***Surgical therapy***

Antirefleux surgery should not be pursued in patients with Barrett's esophagus as an antineoplastic measure. However, this surgery should be considered in those with incomplete control of reflux symptoms on optimized medical therapy.

In cases of Barrett's esophagus with invasion into the submucosa, especially those with invasion to the mid or deep submucosa (T1b, sm2–3), esophagectomy, with consideration of neoadjuvant therapy, is recommended in the surgical candidate.

In patients with T1a or T1b sm1 adenocarcinoma, poor differentiation, lymphovascular invasion, or incomplete endoscopic mucosal resection should prompt consideration of surgical and/or multimodality therapies.