

Diagnosis and Management of Barrett's Esophagus: An Updated ACG Guideline

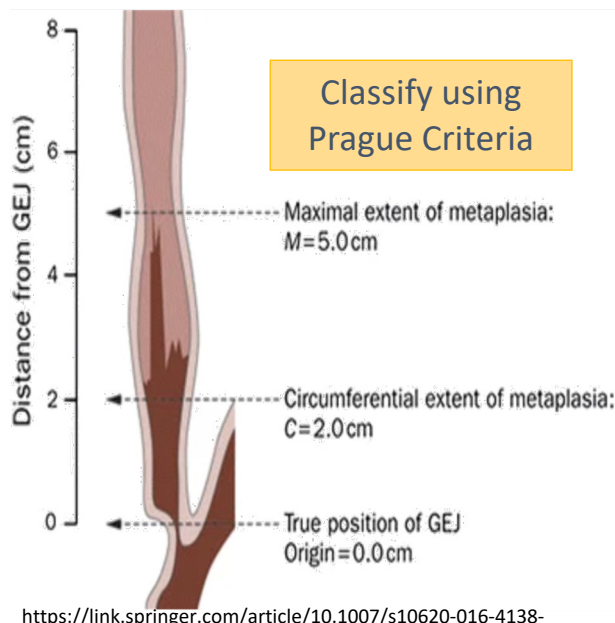
By Hima Veeramachaneni, MD

Background

- Barrett's esophagus (BE) = metaplastic change from squamous epithelium to columnar epithelium in the distal esophagus due to GERD
 - 5-12% of patients with GERD have BE
- BE is the **only known precursor** lesion to esophageal adenocarcinoma (EAC)
 - Diagnosis of EAC after symptom onset has poor survival (<20% at 5 years)
 - Screening & surveillance to identify early and treat endoscopically is key

Diagnosis

- Diagnosis = ≥ 1 cm length of columnar mucosa with **intestinal metaplasia (IM)**
- Do not biopsy:
 - Normal-appearing Z line
 - < 1cm displaced irregular Z line
 - <1 cm = low reliability of Prague criteria & low risk of progression
- Yield of IM \uparrow with \uparrow # of biopsies & \uparrow length of the BE segment
- All IM with dysplasia \rightarrow **confirmed by 2nd pathologist** with GI expertise



Screening

Candidates for Screening

- Chronic GERD + ≥ 3 additional risk factors**
 - Male
 - Age >50 years old
 - White race
 - Tobacco smoking
 - Obesity
 - 1st degree male relative with BE or EAC

Screening Modalities

- Endoscopy** = gold standard BUT invasive & expensive
- Alternative: Swallowable Capsule Device + Biomarker
- Other developing screening modalities:
 - Unsedated transnasal endoscopy
 - Exhaled volatile organic compounds
 - Risk prediction scores

If initial screen negative \rightarrow no repeat
 If LA grade B esophagitis or higher \rightarrow repeat EGD in 8-12 weeks to assess healing and r/o BE

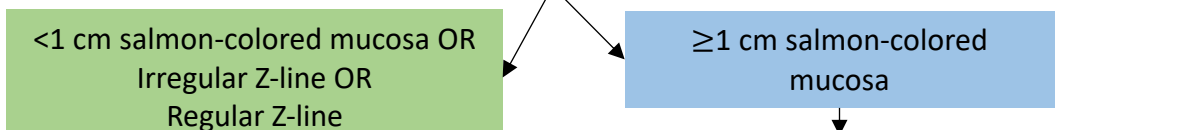
Surveillance

- White light endoscopy + chromoendoscopy** (electronic OR vital dyes) to better identify irregular mucosa or vascular pattern
- Structured biopsy protocol** to \downarrow detection bias
- Length + degree of dysplasia** = determines surveillance intervals

Cannot recommend (insufficient evidence): wide-area transepithelial sampling with computer-assisted 3-dimensional analysis (WATS-3D) or predictive tools/biomarkers

Screening & Surveillance

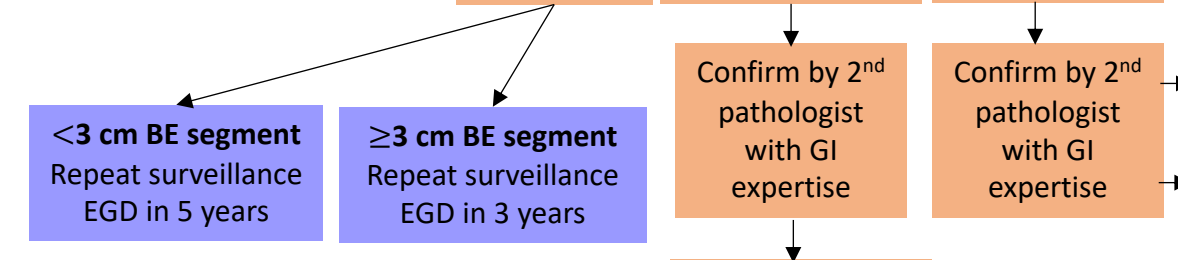
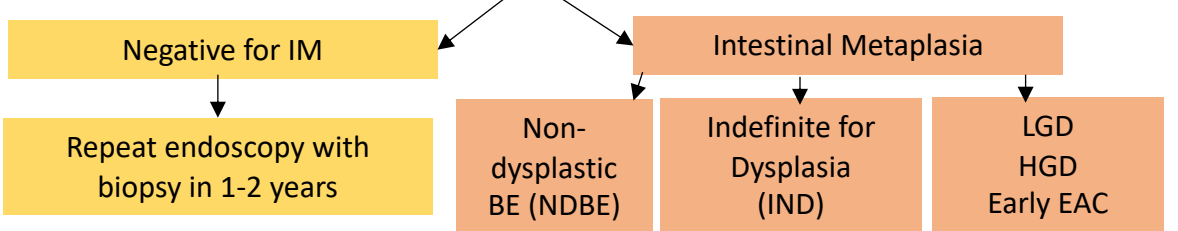
Salmon-colored mucosa extending proximally from the GEJ on endoscopy



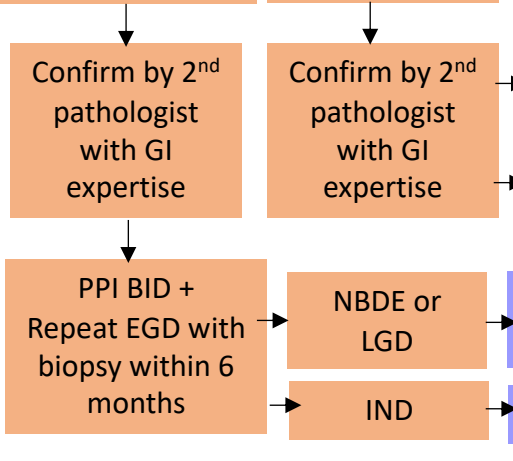
No biopsy

Biopsy

- ≥8 endoscopic biopsies should be obtained
- If only 1-2 cm segment or tongues and 8 biopsies not possible:
 - ≥4 biopsies per cm of circumferential BE
 - ≥1 biopsy per cm of tongue
- If >4 cm segment, then follow **Seattle protocol** = biopsy visible lesions + 4 quadrant biopsies of every 2 cm starting 1 cm above the gastroesophageal junction (GEJ)



****Consider cessation of surveillance if no longer a candidate for EET (Endoscopic Eradication Therapy) or life expectancy <5 years**



10 step approach to endoscopic exam of BE

Approach	Rationale
Identify esophageal landmarks: diaphragmatic hiatus, GEJ, squamocolumnar junction	Critical for further examinations
Consider distal attachment cap (especially if prior dysplasia)	Facilitate visualization
Clean mucosa well (water jet and careful suction)	Remove mucus or debris and limit mucosal trauma
Use insufflation & deflation	Fine adjustments can help detect subtle changes
Spend adequate time inspecting the Barrett's segment & gastric cardia on retroflexion	Increases dysplasia detection
Examine with chromoendoscopy	Enhances mucosa pattern and surface vasculature
Use Prague classification- circumferential and maximal length	Standardized reporting system
Use Paris classification- describe superficial neoplasia	Standardized reporting system
Use Seattle protocol with partially deflated esophagus to sample Barrett's segment	Increases dysplasia detection

Quality indicators for screening and surveillance

1. Documentation of landmarks + extent of BE
2. Not obtaining biopsies in the setting of normal-appearing junction
3. Sampling using Seattle protocol
4. Performing surveillance endoscopy in patients with NDBE no sooner than 3-5 years



Medical Treatment

- **PPI therapy** (at least once daily) if no allergy or contraindication
- No evidence for aspirin (ASA) with PPI therapy → no significant difference in cancer related outcomes
- Patients with BE also likely on ASA for cardio protection due to risk factors
- **DO NOT use antireflux surgery** as antineoplastic measure in BE → no change in progression to neoplasia risk compared to standard PPI therapy

Endoscopic Eradication Therapy (EET)

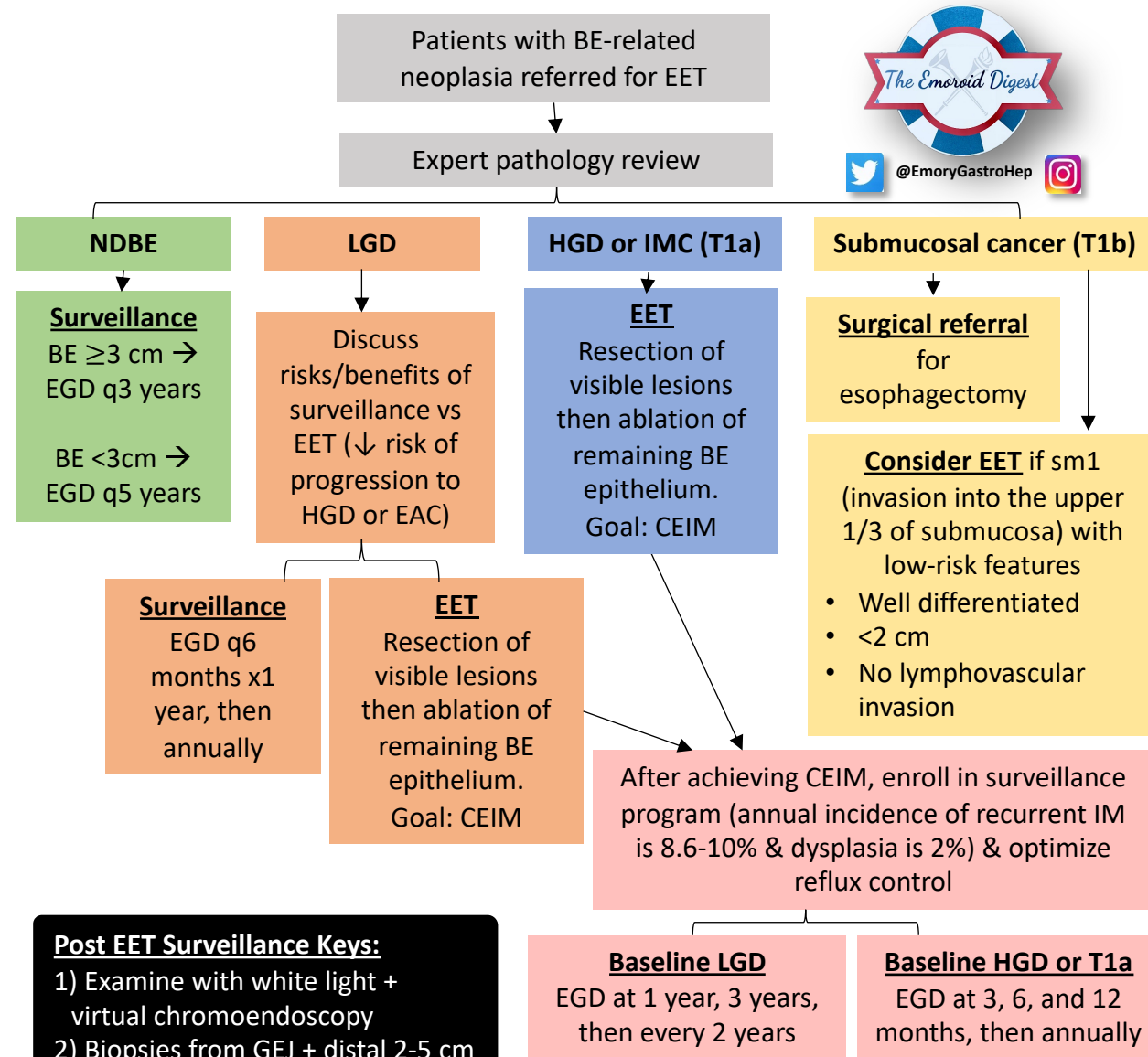
- Esophagectomy has 2% mortality & ↑ morbidity compared to EET
- **Goal of EET** = complete eradication of IM (**CEIM**) → defined as 1-2 surveillance endoscopies w/o visible BE or IM on biopsies
- Endoscopic resection of any visible lesions, THEN ablative therapy

Step 1: Endoscopic resection (ER) of any visible lesions

- Cap-assisted vs multiband endomucosal resection (EMR)
 - Bigger sample for accurate tumor staging & prognostication
- Endoscopic submucosal dissection (ESD)
 - Reserved for en bloc resection of larger lesions, submucosal invasion or post-ablation lesions

Step 2: Ablative therapy

- **Radiofrequency ablation (RFA)** → thermal ablation
 - Achieve CEIM in 3 sessions
 - Adverse events (AEs): esophageal stricture (4.2-7%) & perforation (0.4-0.9%)
- **Endoscopic cryotherapy** (spray vs balloon) → rapid freeze + slow thaw
 - Alternative modality in patients unresponsive to RFA
 - Adverse events: esophageal stricture (9-12%)



Quality indicators for EET in dysplastic BE

- The rate of confirmation by GI pathologist prior to EET
- Rate at which CEN (complete eradication of neoplasia) and CEIM is achieved by 18 mo in patients with dysplasia or IMC
- The rate at which AEs are being tracked and documented post EET