Enhancing the Detection of Barrett Esophagus

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KEYWORDS

• Barrett esophagus • Neoplasia • Endoscopic imaging • Narrow band imaging
• Wide area transepithelial sampling with 3-dimensional analysis • Transnasal endoscopy

KEY POINTS

- The incidence of esophageal adenocarcinoma and its premalignant condition, Barrett esophagus (BE), has increased.
- Screening for BE is not recommended in general population. Surveillance endoscopy is recommended in patients with BE.
- Endoscopy with the Seattle protocol is the standard approach for BE surveillance.
- Advanced imaging techniques may enable targeted biopsies to improve the accuracy of BE surveillance.

INTRODUCTION

Barrett esophagus (BE) is defined as the replacement of normal squamous epithelium with metaplastic columnar epithelium with goblet cells.[1,2] This inflammatory condition is a result of chronic exposure of the esophageal epithelium to refluxed gastric contents. Gastroesophageal reflux disease (GERD) is an increasingly prevalent disease, especially in Western countries. BE is a known major risk factor for the development of esophageal adenocarcinoma (EAC), the incidence of which has increased dramatically since the 1970s.[3–5] The risk of developing EAC among patients with BE is 30- to 125-fold higher compared with the general population.[6] Ultimately, few patients with BE develop EAC, with an annual risk of 0.1% to 0.5%.[7,8] Individuals with BE are often asymptomatic and therefore are not selected for screening, leading to uncertain prevalence and incidence. Based on current studies, the prevalence of BE has been estimated to be about 2% among patients who have undergone upper endoscopy for any reason. The incidence of BE among patients with GERD symptoms varies between 5% and 20%.[1,9–11] The increased incidence of BE is thought to be caused by increasing availability and frequency of endoscopy.[12]

The endoscopist has a critical role of suspecting the presence of BE and obtaining tissue to confirm a diagnosis. A key component of this skill is considering risk factors such as GERD, obesity, family history, and tobacco use.

Generally, BE is suspected when columnar epithelium (ie, pink salmon-colored epithelium) is observed endoscopically to extend proximal to the gastroesophageal junction (GEJ) into the esophagus. Currently, there are 2 different landmarks to identify the GEJ. The most proximal extent of the gastric folds is used as the landmark in Western countries, while the most distal extent of palisade vessels is used as the landmark in Asian countries. Most published data on BE use the proximal extent of gastric folds as the

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landmark of GEJ. The location of the most proximal extent of gastric folds may be affected by distension of the esophagus and stomach, respiration, and gastric motility. The GEJ should be measured when the stomach is decompressed so that the gastric folds are most visible.

Once BE has been identified, it can be divided into short segment or long segment based on the extent of changes. BE that is less than 3 cm is considered short segment, while BE that is longer than 3 cm is considered long segment.[13] The risk of cancer in BE increases with the length of esophageal metaplasia.[14] Currently, the Prague circumferential (C) and maximal extent (M) classification often is used to objectively describe the extent of BE.[15] This classification is valuable in objectively describing a patient's BE as well as directing treatment and follow-up.

The definitive diagnosis of BE is made pathologically by the presence of intestinal metaplasia on an esophageal biopsy. Endoscopic surveillance has been proposed for patients with BE in order to detect dysplasia and neoplasia in an early stage. Although it remains uncertain if endoscopic surveillance of patients with BE reduces mortality from esophageal adenocarcinoma, current guidelines recommend endoscopic surveillance at 3 to 5 year intervals for BE without dysplasia, 6 to 12 months for those with low-grade dysplasia, and every 3 months for those with high-grade dysplasia who do not undergo intervention.[16]

SEATTLE PROTOCOL

The goal of surveillance of patients with BE is to prevent the evolution or progression of adenocarcinoma. The Seattle protocol is used to detect dysplasia and neoplasia by obtaining 4-quadrant biopsy sampling at 1 to 2 cm intervals throughout the area of suspected BE. The intention of the Seattle protocol is to increase the chance of identifying dysplasia and neoplasia that may be randomly distributed throughout the area of BE. In addition to Seattle protocol sampling, targeted biopsies should be performed of mucosal irregularities, such as nodules, masses, and ulcerations. The sensitivity of this protocol is diminished because of sampling error, especially for long segment disease where dysplastic and neoplastic lesions tend to have patchy and focal distribution[17–20] (**Fig. 1**). Early studies demonstrated foci of unsuspected carcinoma in up to 73% of resected esophagectomy specimens for high-grade dysplasia.[19] It has been reported that random biopsies obtained with white light endoscopy sample only 4% to 5% of Barrett

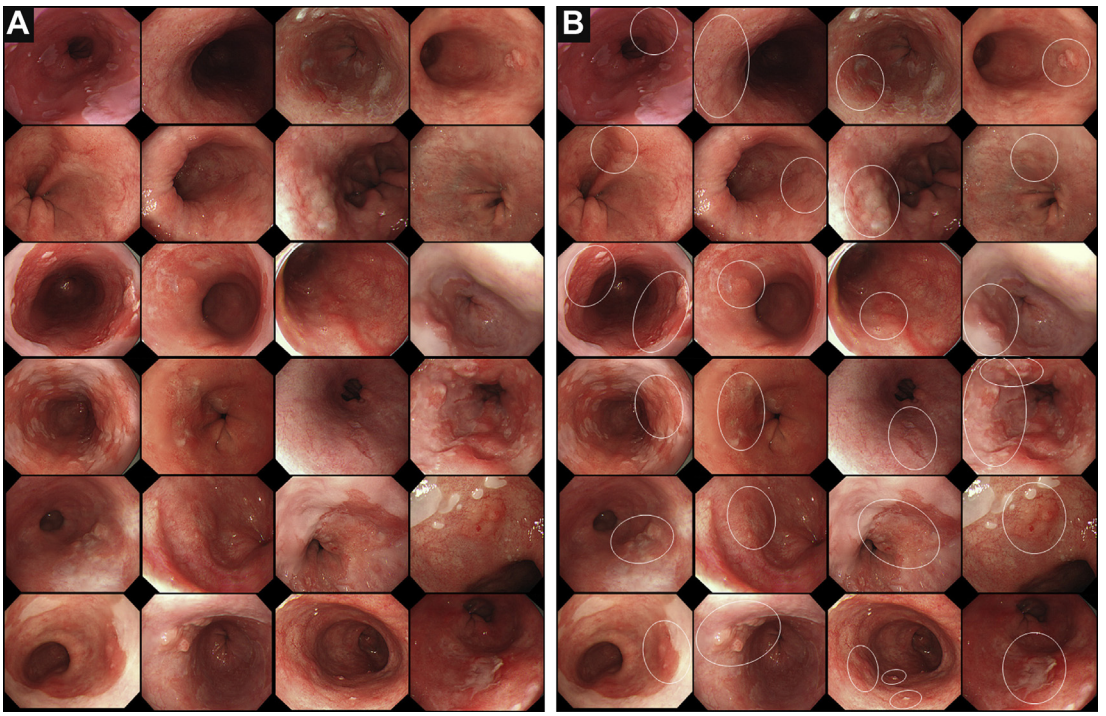

Fig. 1. (*A*) Examples of subtle neoplastic lesions in BE. (*B*) The neoplastic lesions are indicated with circles. (*From* Boerwinkel DF, Swager AF, Curvers WL, et al. The clinical consequences of advanced imaging techniques in Barrett esophagus. Gastroenterology 2014;146(3):623; with permission.)