J Neurogastroenterol Motil, Vol. 29 No. 1 January, 2023
pISSN: 2093-0879 eISSN: 2093-0887
https://doi.org/10.5056/jnm22215
Journal of Neurogastroenterology and Motility

Editorial

Length of the Barrett's Esophagus Is Proportional to the Abnormality of Esophageal Motility and Anti-reflux Barrier

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Article: Esophageal hypocontractile disorders and hiatal hernia size are predictors for long segment Barrett's esophagus
Shibli F, Fass OZ, Teramoto OM, et al
(J Neurogastroenterol Motil 2023;29:31-37)

The Barrett's esophagus (BE) is the premalignant condition of esophageal adenocarcinoma. BE is characterized by the replacement of normal esophageal squamous cell epithelium with columnar metaplasia. BE results from reflux of gastric contents into the esophagus, resulting in inflammation and chronic tissue injury. Diagnosis of BE requires both endoscopic and histologic documentation of columnar epithelium with intestinal metaplasia extending into the tubular esophagus ≥ 1 cm proximal to the esophagogastric junction.[1] The progression from BE to the esophageal adenocarcinoma is believed to be a continuous sequence, from nondysplastic BE, followed by low grade dysplasia, then high grade dysplasia, and eventually esophageal adenocarcinoma. BE segment length is potentially correlated with risk of progression from BE to esophageal adenocarcinoma.[2-4] Meta-analysis and systematic review of the BE comparing progression rates to esophageal adenocarcinoma or to high grade dysplasia combined demonstrate significantly lower rates in patients with short segment BE (SSBE) compared with long segment BE (LSBE).[5] Current American College of Gastroenterology guideline recommends that the length of the nondysplastic BE segment be considered when assigning surveillance intervals such that LSBE (≥ 3 cm) are evaluated with endoscopy every 3 years, while SSBE (< 3 cm) are evaluated with endoscopy every 5 years.[6]

Esophageal dysmotility contributes to worsening reflux and thus the development of BE. Main pathophysiologies of gastroesophageal reflux disease (GERD) are the disruption of antireflux barrier and failure of esophageal clearance of refluxate. Anti-reflux barrier of esophagogastric junction (EGJ) is mainly composed of lower esophageal sphincter (LES) and crural diaphragm. High-resolution esophageal manometry (HRM) is not definite diagnostic test for GERD, however, it provides complementary information for diagnosis by evaluating the motility abnormalities usually accompanying GERD.[7] The function of the anti-reflux barrier can be identified by the HRM with EGJ morphology and EGJ contraction integral.[8] The follow variables of HRM may be used to qualify the motility abnormalities which predispose to the development of GERD;

1. The anti-reflux barrier function: contraction vigor the form

Received: December 15, 2022 Revised: None Accepted: December 26, 2022

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of an EGJ contractile integral, and LES baseline pressure
2. The anti-reflux barrier morphology: degree of hiatal hernia
3. Esophageal body contraction; ineffective, hypotensive, failed peristalsis or transition zone defect: esophageal movement is directly related to esophageal cleaning of refluxate.
4. The esophageal peristaltic reserve: evaluated with a multiple rapid swallow[9]

Shibli et al[10] reported the abnormal HRM findings in the SSBE and LSBE patients. The findings of HRM in BE are predictable, because which may be observed in GERD patients.[10] The most frequent motor disorders observed in GERD were ineffective esophageal motility, fragmented peristalsis, absent contractility and hypotensive LES. Hiatal hernia is common. The abnormal findings of HRM in LSBE are basically the same as in SSBE, but differ in grade and severity. LES residual pressure, mean LES resting pressure were lower and failed swallows or hypotensive contractions are more frequent in those with LSBE as compared those with SSBE. This is the first study to report that the more severe the BE, the greater the likelihood patients will demonstrate an esophageal motor abnormality.[10]

Shibli et al[10] reported hypocontractile motility disorder increased the odds of LSBE by 242.0%, as compared to SSBE. This suggests the possibility of causing more and longer esophageal mucosal damage because the refluxed gastric contents in the esophagus are not effectively cleaned and remains for a long time. The 38.0% of increase in the odds for LSBE, for every 1 cm increase in hernia size means the length of the Barrett's esophagus increases as the antireflux barrier is broken.[10] The objective HRM findings of this study supports that the disruption of antireflux barrier and failure of esophageal acid clearance make prolonged gastric acid exposure to the esophagus, causing mucosal damage, and eventually worsening to the columnar epithelial metaplasia. This risk stratification using HRM is possible to predict the progression of BE.

Financial support: None.

Conflicts of interest: None.

Author contributions: Yu Kyung Cho reviewed and wrote the editorial.

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