



Whether the Role of Esophageal Baseline Impedance Is Complementary to or Alternative to the 24-Hour Esophageal pH Monitoring for the Diagnosis of Gastroesophageal Reflux Disease?

Yu Kyung Cho

Division of Gastroenterology, Department of Internal Medicine, The Catholic University of Korea, College of Medicine, Seoul, Korea

Article: The usefulness of esophageal baseline impedance levels for the diagnosis of nonerosive reflux disease and the proper time for measurement in endoscopy-negative Korean patients with esophageal or supraesophageal symptoms
Kim YG, Noh CK, Lee KJ
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Ambulatory reflux monitoring assesses reflux burden using acid exposure time and reflux-symptom association. Chronic esophageal acid exposure in the animal and human models induces changes in the mucosa, seen histologically by increased numbers of dilated intracellular spaces. Low baseline impedance (BI) in the distal esophagus is likely explained by reflux-mediated damage to the integrity of esophageal mucosa. Esophageal BI has been suggested as a surrogate marker for esophageal mucosal integrity which may reflect longitudinal reflux mucosal injury over time. Distal esophageal mucosal BI measured during the ambulatory 24-hour esophageal pH-impedance monitoring is considered to be a surrogated market of reflux burden.^{1,2}

Esophageal BI values can be acquired via several approaches; (1) from nocturnal periods without swallows during ambulatory pH-impedance tracings as mean nocturnal baseline impedance (MNBI), (2) from prototype balloon-mounted electrodes during sedated endoscopy (mucosal integrity or mucosal impedance [MI]),

or (3) from high-resolution impedance manometry (HRIM) studies during the resting landmark phase prior to administration of test swallows (BI-HRIM).³ MNBI, BI-HRIM, and MI are all considered adjunctive metrics supporting a gastroesophageal reflux disease (GERD) diagnosis. MNBI may be of particular value when acid exposure time is inconclusive in predicting symptomatic outcomes with antireflux therapy.⁴ Both MNBI and BI-HRIM may be compromised by inconsistent or incomplete contact between impedance sensors and the esophageal mucosa due to the presence of intraluminal air or liquid bolus or a dilated esophagus.³ The recent study revealed that esophageal contractile segment impedance from HRIM correlates with MNBI and acid exposure time from 24-hour pH-impedance monitoring.⁵

BI-HRIM can distinguish patients with GERD from controls.⁶ Because HRIM catheters incorporate esophageal impedance sensors, BI-HRIM acquisition has the potential to provide complementary GERD diagnostic data with a shorter procedure time than

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*Correspondence: Yu Kyung Cho, MD

Division of Gastroenterology, Department of Internal Medicine, The Catholic University of Korea, College of Medicine, 222 Banpo-daero, Seocho-gu, Seoul 06591, Korea
Fax: +82-1048297750, E-mail: ykcho@catholic.ac.kr

catheter-based ambulatory reflux monitoring, carrying lower risks than sedated endoscopy for MI acquisition. Standard BI-HRIM from the resting landmark phase has been shown to correlate well with MNBI, offering discriminatory value in distinguishing pathologic from physiologic esophageal acid exposure time and correlating with MNBI in nonerosive reflux states.⁷

Kim et al⁸ investigated factors related to esophageal BI level and the value of esophageal mean BI for the predicting pathological esophageal acid exposure and diagnosing nonerosive reflux disease. The results are published in *Journal of Neurogastroenterology and Motility* this month.⁸ They compared the BI levels of the both proximal and distal esophagus in 3 groups of the nonerosive reflux disease (NERD), reflux heartburn, and functional heartburn. The factors that influence impedance change are meals and sleep, especially in the distal esophagus. Esophageal BI values became lower shortly, at 5 minutes and 30 minutes after meal ingestion in both the proximal and distal esophagus. BI levels in the proximal esophagus became lower within 1 hour after sleeping compared with before sleeping. Lower BI in the distal esophagus represent abnormal acid exposure in the distal esophagus. The value of the area under the receiver operating characteristic curve was 0.774 (95% CI, 0.679-0.869; $P < 0.001$) for the diagnosis of NERD. The area under the curve under the receiver operating characteristic curve was 0.753 (95% CI, 0.630-0.876; $P < 0.001$) for the diagnosis pathological acid reflux. The BI of the distal esophagus were significantly lower in the NERD group, compared with the functional group. These results are similar to the previous studies. The advantage of this study is many patients complaining of atypical symptoms, globus, were included. It suggests short time measurement of BI can be adjustable for the diagnosis of NERD in atypical symptomatic patients.

The clinical significance of this study is to define how much the esophageal BI levels help to discriminate pathological acid reflux and NERD, if it is possible, and whether BI can represent the whole time 24-hour esophageal pH monitoring, especially in which patients?

The diagnostic accuracy is satisfactory, however, the considerable NERD patients may be missed or over diagnosed as GERD with only esophageal impedance. It can be explained by the complex

pathophysiology of GERD. Many factors besides the acid reflux can contribute the development of GERD. In addition, nonacid reflux, small amount reflux, or short time reflux episodes will not induce the mucosal change contributing to lowering BI. Therefore the clinical utility of BI in diagnosing NERD should be determined by additional further studies.

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