

How effective are we at understanding ineffective esophageal motility?

Ineffective esophageal motility has been with us since the development of perfused and, more recently, solid state esophageal motility testing. Previously described by various laboratories with a variety of terms, such as “failed peristalsis,” “ineffective peristalsis,” “dropped waves,” “nonspecific motility disorder,” etc, its definition and nomenclature were standardized by Spechler and Castell,¹ who defined the criteria for the diagnosis of ineffective esophageal motility as a disorder with low or normal esophageal sphincter pressure, normal lower esophageal sphincter (LES) relaxation, and greater than 30% low-amplitude waves, some of which may not be peristaltic. Although ineffective esophageal motility has prompted investigative work by laboratories on all continents, its pathophysiology and clinical significance are still being debated. Castell’s group linked ineffective esophageal motility with GERD and suggested that it was a highly specific (91%), albeit not sensitive (45%), indicator of supine GERD on proton pump inhibitors.² A more recent study, by Fornari et al,³ confirmed an association of GERD with ineffective esophageal motility, independent of other GERD risk factors, such as a low LES pressure, hiatus hernia, and male sex. Other investigators documented that GERD-associated ineffective esophageal motility improves after Nissen fundoplication and that its presence is not a manometric contraindication to antireflux surgery.⁴

In a study that used esophageal impedance to measure bolus transport, Tutuian and Castell⁵ showed, in a group of 70 patients with ineffective esophageal motility, that there was normal bolus transit by impedance in 68% of liquid swallows, and 59% of viscous swallows. Thus, they could not demonstrate by impedance evidence of impaired esophageal bolus transit in a significant number of those patients. Testing for esophageal reflux was not reported for that group. Mittal⁶ pioneered the use of small-diameter high-frequency intraluminal US (HFIUS) to study esophageal function. This provides dynamic real-time images of the esophageal wall during peristalsis and when at rest at a given chosen level, and can be done in conjunction with manometry.

In the current study, Kim et al,⁷ from Korea, reported the results of a prospective study of 16 controls and 46 patients with manometrically diagnosed ineffective esophageal motility. The subjects were first given a detailed standardized symptom questionnaire; they were then evaluated with an EGD, 24-hour dual-channel pH catheter testing, and also by HFIUS measurements of esophageal wall thickness at rest and during 5 wet swallows in the LES, and at 3 cm and 9 cm above the manometrically identified LES. They used Mittal’s methodology to measure changes in esophageal muscle thickness at baseline and at peak manometric

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contractions. The investigators divided the study patients with ineffective esophageal motility into a GERD-associated group of 26 patients (group I) and a second group of 20 patients with ineffective esophageal motility who did not have GERD (group II). Not surprisingly, significantly more patients in group I had typical reflux symptoms. There were no other statistical differences in the esophageal symptoms between the 2 groups. The study groups and controls were well matched for age, sex, body mass index, smoking, and alcohol use.

The investigators found a statistically significant increase in esophageal-wall thickness, by using HFIUS, in the group II patients with non-GERD-related ineffective esophageal motility, when compared with the controls, and with the group I patients, who had GERD and ineffective esophageal motility. This was present at all levels of the HFIUS measurement. Based on these data, the investigators postulated that the pathophysiologic mechanisms that underlie ineffective esophageal motility are different in group I patients with GERD, in whom the manometric abnormality may be induced by chronic acid-reflux exposure. In those group II patients without GERD, there may be a primary esophageal muscular disorder measured by HFIUS. This seems to

correlate with a recently published study by Mittal⁶ of increased esophageal wall thickness by HFIUS in patients with a number of esophageal motor disorders.⁸ In the current study by Kim et al,⁷ unlike other previous studies, testing for GERD was also done. I would suggest that this testing be a prerequisite for future studies when using HFIUS in this area.

In this study, as in others in this area, there were some methodologic issues. It was not clear to me how GERD was defined for study stratification. We know there is no criterion standard. Twenty-four of the 26 patients in the GERD group I had an abnormal distal pH profile. Presumably, the other 2 patients had endoscopic esophagitis, with a normal pH study, presumably, off acid-suppressive therapy. Is this GERD? Would removing those 2 patients from the study group affect the statistical calculations? Another thorny issue pertains to the manometric diagnosis of ineffective esophageal motility. Nayar et al,⁹ the group from Cleveland Clinic, published a study of intraobserver agreement for esophageal manometry. There was good agreement for the diagnosis of normal motility and of achalasia (overall $k = 0.68$) but much less agreement for the other motility disorders ($k = 0.27$) between 2 independent interpreters, who disagreed about this manometric diagnosis in 29% of the studies reviewed.

Another issue involves the symptoms of the 2 groups with ineffective esophageal motility. I was surprised not to see more differences in dysphagia and chest pain recorded between the 2 study groups, given the differences seen in the esophageal-wall thickness and the hypothesis of the investigators. In a recently published study of 94 patients, Mittal⁶ found that dysphagia was more common in patients with increased esophageal-wall thickness, including 24% of patients who had normal manometry but abnormal HFIUS.⁸ The current study by Kim et al⁷ was smaller. There were 2 patients with dysphagia in group I, and 6 in group II, but, because of the study size, this did not reach statistical significance. There was no difference in chest pain between the 2 groups.

Nevertheless, the study by Kim et al⁷ does advance our understanding of ineffective esophageal peristalsis and its association with GERD. It further suggests an increasing role of HFIUS in research in this area. Only time and more

studies will tell how these findings affect our clinical management of these patients.

DISCLOSURE

The author reports that there are no disclosures relevant to this publication.

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Abbreviations: HFIUS, high-frequency intraluminal US; LES, lower esophageal sphincter.

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