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Review

Role of Endoscopy in Motility Disorders of Upper Gastrointestinal Tract

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Gastrointestinal motility disorders have a wide range of symptoms and affect patients' quality of life. With the advancement of endoscopy, the diagnostic and therapeutic roles of endoscopy in motility disorders is becoming more significant. Endoscopy is necessary to rule out possible organic diseases in patients with suspected motility disorders and provide significant clues for their diagnosis. Moreover, interventional endoscopy may be a primary or alternative treatment option for selected patients with motility disorders, and it is becoming a promising field as new therapeutic applications are developed and utilized for various motility disorders. This review may provide suitable indications for the use of endoscopy in diagnosing and treating motility disorders of the upper gastrointestinal tract.
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Key Words
Endoscopy; Esophageal motility disorders; Upper gastrointestinal tract

Introduction

Gastrointestinal endoscopy is easy to perform and has a low risk of complications and high level of validity. It is usually performed as an initial diagnostic workup for patients who complain of gastrointestinal symptoms. Endoscopy has made a significant contribution to the diagnosis and treatment of organic diseases. However, understanding functional disorders remains challenging even with endoscopy, and the role of endoscopy in motility disorders has not been fully elucidated.

We recently reviewed past endoscopic images of patients with motility disorders and found several missing findings. Unintentionally excessive findings providing important clues for diagnosing motility disorders were overlooked. For example, early achalasia is characterized by a slightly dilated esophageal lumen, esophageal rosette, and gastroesophageal junction (GEJ) resistance to flow on endoscopy. Moreover, esophageal spasms may be shown as spastic contractions on endoscopy. As such, endoscopy can help clinicians understand, detect, and diagnose functional diseases more efficiently.

Furthermore, various interventional endoscopy approaches offer treatment and resolve the symptoms of several motility disorders. Novel endoscopic intervention techniques and devices are introduced in succession, and many studies regarding the efficacy and safety of these procedures have been reported. Interventional endoscopy is a promising field in the treatment of motility disorders. Accordingly, this review introduces the potential role of endoscopy from both diagnostic and therapeutic perspectives, especially in up-

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per gastrointestinal motility disorders.

Part I. Diagnostic Role in Motility Disorders —

Pathognomonic Endoscopic Findings

Gastroesophageal reflux disease

Gastroesophageal reflux disease (GERD) is a condition characterized by various clinical symptoms such as heartburn, acid regurgitation, and changes in the esophageal mucosa.[1] GERD is usually associated with transient lower esophageal sphincter (LES) relaxations or chronic LES hypotonicity below 10 mmHg.[2] It comprises non-erosive reflux disease (NERD) and erosive reflux disease (ERD) according to the presence of esophageal mucosal injury.[3] Endoscopy is the gold standard examination for verifying mucosal damage and distinguishing ERD from NERD or differentiating other esophagitis forms (such as viral, eosinophilic, and pill-induced).[4] NERD includes minimal-change esophagitis, such as mucosal hyperemia or edematous change, and no abnormality on endoscopy. ERD is established when there are patchy, mucosal breaks or circular and confluent mucosal defects in the distal esophagus. During endoscopy, the severity of mucosal damage is assessed using the Los Angeles (LA) grading system,[5] in which mucosal damage is classified as follows (with increasing mucosal defect): grade A, one or more mucosal breaks no longer than 5 mm; grade B, one or more mucosal breaks longer than 5 mm; grade C, one or more mucosal breaks that are continuous between the tops of 2 or more mucosal folds involving $< 75\%$ of the esophageal circumference; and grade D, one or more mucosal breaks involving $\geq 75\%$ of the esophageal circumference (Fig. 1). Patients with severe erosive esophagitis (LA grade C or D) may be expected to have other conditions predisposing to GERD, such as hiatal hernia or a loosened gastroesophageal flap valve (GEFV) that is also confirmed during endoscopy.[6] GEFV is considered a barrier to esophageal reflux at the GEJ, and endoscopic grading of GEFV offers useful information regarding reflux activity. Flap valves are graded from I through IV based on Hill's classification (Fig. 2).[7]

In addition to directly observing and grading the severity of esophagitis, the presence of accompanying complications like peptic

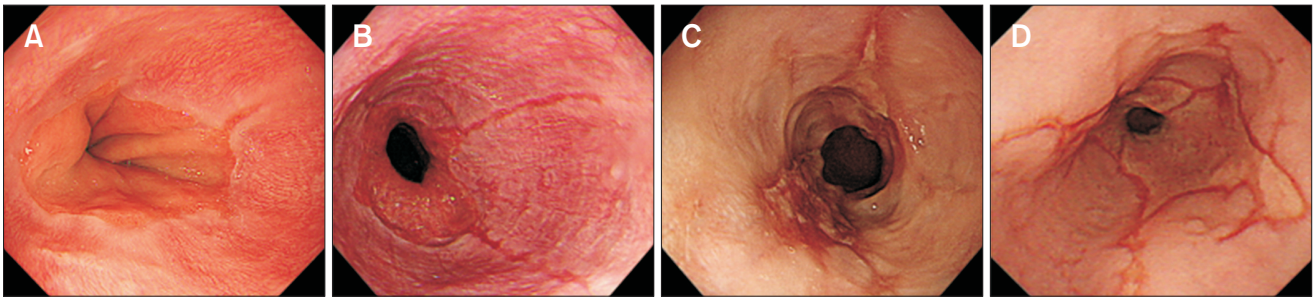

Figure 1. Los Angeles classification for grading erosive esophagitis. (A) Grade A: one or more mucosal breaks no longer than 5 mm. (B) Grade B: one or more mucosal breaks longer than 5 mm. (C) Grade C: one or more mucosal breaks that are continuous between the tops of 2 or more mucosal folds involving $< 75\%$ of the circumference. (D) Grade D: one or more mucosal breaks involving $\geq 75\%$ of the esophageal circumference.

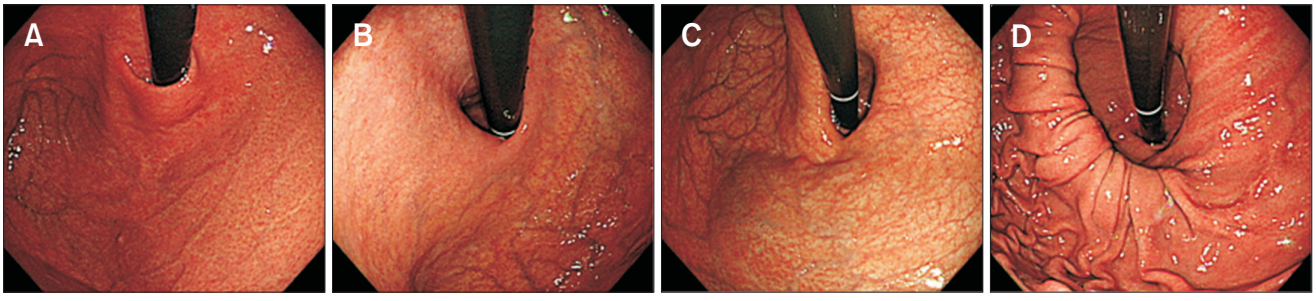

Figure 2. Hill's classification for grading gastroesophageal flap valves. (A) Grade I: a prominent fold of tissue along the lesser curvature and closely apposed to the endoscope. (B) Grade II: the fold is present, but there are periods of opening and rapid closing around the scope. (C) Grade III: the ridge is barely present, and there is often failure to close around the scope. (D) Grade IV: there is no muscular ridge, and the gastroesophageal area continuously remains open.

strictures or diseases such as gastric stasis and pyloric obstruction that may cause GERD by food stasis can be confirmed by endoscopy. Further, histological diagnosis of the esophageal mucosa can also differentiate infectious or eosinophilic esophagitis.[8] Previous studies have reported that 4% to 5% of patients with refractory reflux symptoms had endoscopy-verified eosinophilic esophagitis.[9,10] Barrett's esophagus, which can result from long-standing symptomatic GERD,[11] can progress to esophageal adenocarcinoma. The malignant progression risk factors of Barrett's esophagus such as the presence of long segments, specialized intestinal metaplasia can be verified by performing endoscopy. Therefore, regular surveillance endoscopy and esophageal biopsies are recommended for early detection of dysplasia or early neoplasia.[11-13]

Achalasia and esophageal spastic disorders

The importance of endoscopic assessments for esophageal motility disorders was emphasized for a long time, and the previous study reported that the observation of contractions at endoscopy can identify the motility disorders and it may represent an adjunctive diagnostic test to manometry.[14] According to a previous study, 64.4% of patients with esophageal motility disorders showed abnormal endoscopic findings.[15] The unusual resistance during passage of endoscope through the GEJ, retained food in the esophageal lumen, and spastic and nonocclusive contractions were significantly associated with esophageal motility disorders.[15]

Achalasia is a primary esophageal motility disorder character-

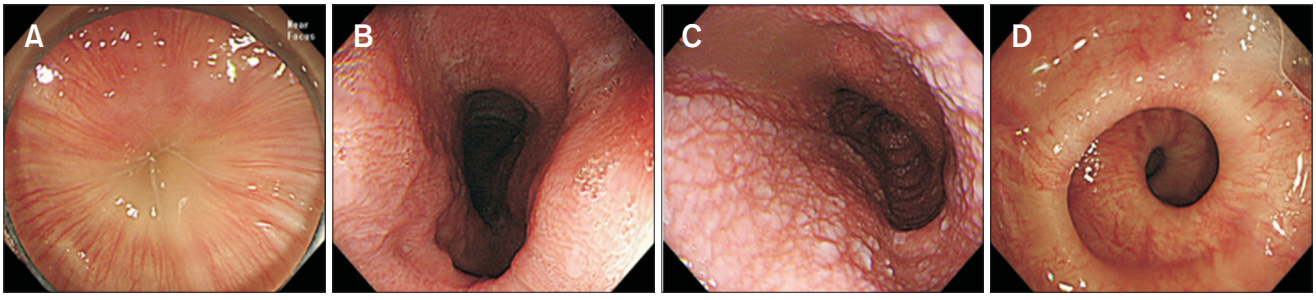

Figure 3. Endoscopic images suggestive of esophageal motility disorders. (A) Rosette sign with resistance when passing through the gastroesophageal junction in esophageal achalasia. (B) Esophageal luminal dilatation and (C) residual fluid with hyperkeratosis on esophageal squamous epithelium in advanced esophageal achalasia. (D) Spastic contraction in distal esophageal spasm.

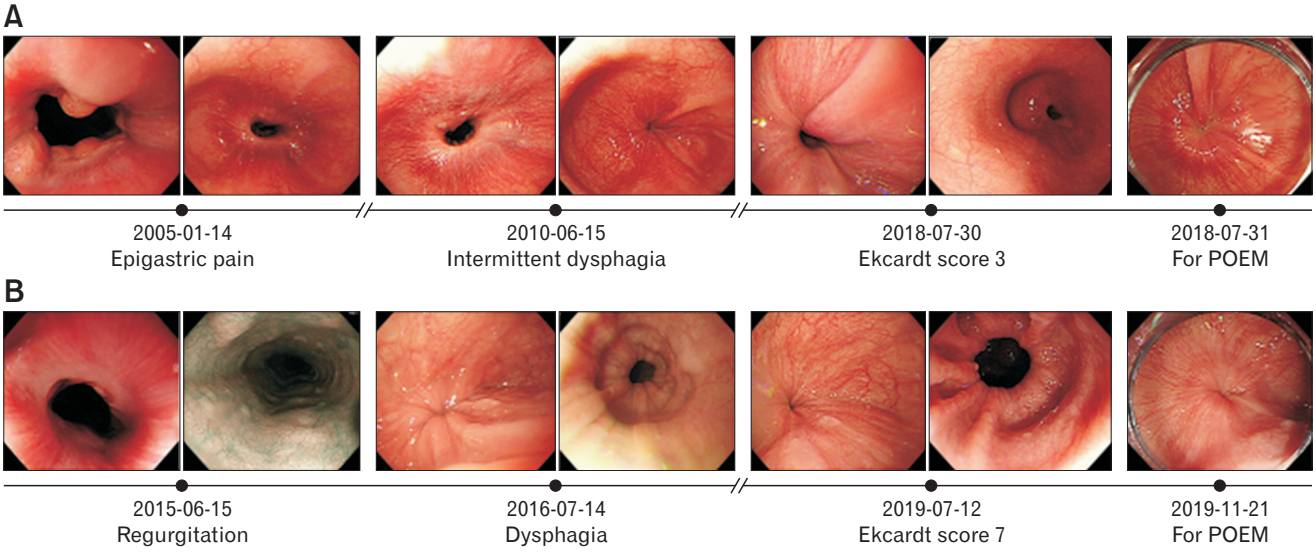

Figure 4. Serial endoscopic images of patients before diagnosing achalasia and performing peroral endoscopic myotomy (POEM). (A) The patient who was followed up for reflux esophagitis showed a narrowing gastroesophageal junction (GEJ) and slightly dilated lower esophagus from the time of dysphagia onset. (B) The patient who was followed up for regurgitation and intermittent dysphagia showed a narrowing GEJ and spastic contractions in the lower esophagus from the time dysphagia became more frequent and severe.

ized by the absence of esophageal peristalsis and impaired relaxation of the LES. It manifests as obstruction to the passage of food from the esophagus into the stomach, resulting in dysphagia, regurgitation, weight loss, and, rarely, aspiration pneumonia.[16] Achalasia is diagnosed by high-resolution manometry (HRM) or barium esophagography. Its common signs include resistance to endoscope passage through the GEJ and appearance of esophageal rosette in the lower esophagus after deep inspiration (ie, "rosette's sign"; Fig. 3A).[17] Some patients with achalasia without esophageal rosette demonstrate a gingko leaf-shaped morphology of the longitudinal section of the GEJ (ie, "gingko leaf sign").[18] These findings are mainly observed in early achalasia and are often found by reviewing past endoscopic images after disease diagnosis. Figure 4 shows serial endoscopic images, obtained over several years, of the lower esophagus of patients diagnosed with achalasia who underwent peroral endoscopic myotomy (POEM). From the time the patients complained of dysphagia, the GEJ was narrowed and spastic lower esophageal contractions were observed. Cimetropium bromide was not administered; therefore, a more prominent rosette sign was noted just before POEM.

Endoscopic findings for early achalasia may be normal; however, as the disease progresses, the esophagus may become dilated and sigmoid-shaped ("sigmoidal esophagus"; Fig. 3B), filled with secretions and food particles. At the time of achalasia diagnosis, these findings are often seen on endoscopy. In some cases, residues induce hyperkeratosis of the squamous cell epithelium (Fig. 3C) and, occasionally, esophageal candidiasis that causes worsening dysphagia or odynophagia. Additionally, spastic esophageal disorders manometrically identical to type III achalasia, diffuse esophageal spasm, and jackhammer esophagus can be shown as spastic and nonocclusive contractions at the time of endoscopy (Fig. 3D).[15]

It is very important to exclude pseudoachalasia due to a GEJ tumor, which accounts for up to 5% of manometrically defined cases of achalasia.[19] Imaging examination including computed tomography, endoscopic ultrasonography (EUS), or endoscopy with biopsy is strongly recommended to rule out a tumorous etiology of strictures mimicking idiopathic achalasia. Among patients complaining of dysphagia, endoscopic findings are normal, but increased muscle thickness near the GEJ is occasionally observed in EUS. Therefore, EUS may also be a useful tool in the differential diagnosis of motility disorders.

Esophageal diverticulum

Esophageal diverticulum is a rare disease presenting with an outpouching of the esophagus that causes symptoms such as dysphagia, regurgitation, and chest pain. There are 3 types, classified based on pathophysiology and location in the esophagus: Zenker's and Killian–Jamieson (upper third), Rokitansky (middle third), and epiphrenic (lower third).[20] Zenker's diverticulum presents between the oblique fibers of the inferior pharyngeal constrictor and cricopharyngeal muscles. Endoscopy must be performed very carefully due to the risk of entry of the endoscope into the pseudolumen and perforation of the parapharyngeal area. Sometimes, placing the patient from a left decubitus to a supine position may help if endoscopic insertion is impossible.[21] Rokitansky's diverticulum is a traction-type diverticulum formed by cicatrical contraction due to chronic inflammation, such as pulmonary tuberculosis. Cases of this type have decreased, and associated clinical problems are even rare.[20] An epiphrenic diverticulum is a pulsion-type diverticulum within 10 cm of the GEJ. As 75-100% of patients with an epiphrenic diverticulum have accompanying esophageal motility disorders, HRM is useful for diagnosis and establishment of a treatment plan.[22-24] Signs include resistance to scope passage through the GEJ during endoscopy due to accompanying esophageal motility disorders or a compression effect of the diverticulum itself.[25,26] Endoscopy can verify septal depth, diverticular sac size, and accompanying complications such as diverticular ulceration or bleeding.[27]

Functional dyspepsia

Functional dyspepsia is a symptomatic syndrome in which gastrointestinal symptoms appear chronically and repeatedly in the absence of organic diseases such as peptic ulcer, gastrointestinal ma-

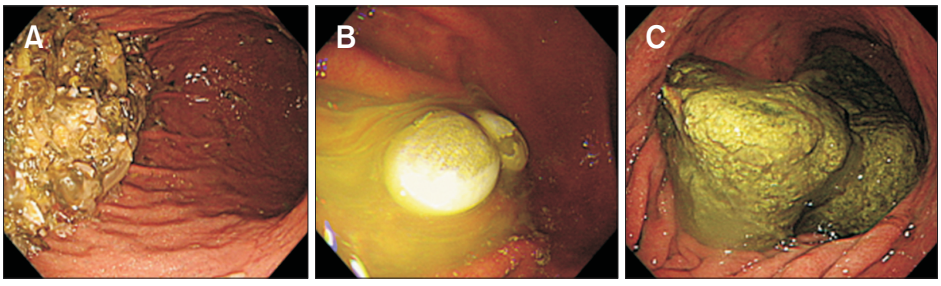

Figure 5. Clues for suspecting gastroparesis on endoscopy. (A) Food retention in the gastric body of a patient with diabetic gastropathy. (B) Undigested pills in the gastric body of a patient with diabetic gastropathy. (C) Bezoar in a patient with severe gastroparesis who complained of nausea and dyspepsia.

lignancy, GERD, or pancreaticobiliary disease.[28] The pathophysiology of functional dyspepsia has not been fully understood. This includes primary gastroparesis, duodenitis, psychosocial, genetic, and environmental factors.[29] Since there is no useful biomarker for the diagnosis of functional dyspepsia, excluding organic diseases that can cause dyspepsia is very important. Accordingly, endoscopic examination is important to differentiate other organic diseases, such as inflammation or malignancy, and the presence of *Helicobacter pylori* infection in suspected cases of functional dyspepsia.[21]

Gastroparesis

Gastroparesis is defined as delayed gastric emptying without mechanical obstruction of the stomach or small bowel. Although only a few population-based studies have accurately evaluated its prevalence, gastroparesis is generally reported to be higher in patients with diabetes.[30-32] Symptoms include nausea, vomiting, bloating, postprandial fullness, early satiety, and abdominal pain.[33] Gastroparesis is diagnosed by gastric emptying scans, electrogastrography, upper gastrointestinal series, and wireless motility capsule.[34-36] One of the presumed mechanisms is spasm- or fibrosis-induced pyloric dysfunction.[37] Endoscopy should be performed to exclude mechanical obstruction caused by gastric or duodenal ulcer or a tumor. Sometimes, reflux esophagitis induced by recurrent nausea and vomiting may be observed on endoscopy. In advanced gastroparesis cases, retained food or pills, or bezoars in severe cases, may be observed in the stomach (Fig. 5). However, previous studies have reported that only 27% of patients with gastroparesis experience food retention.[34,38] Since food retention can also happen as a result of insufficient fasting time before endoscopy, care should be taken when diagnosing gastroparesis based solely on food retention in the stomach.

Diagnostic Endoscopic Instruments and Tools

Catheter placement for high-resolution manometry

HRM is the standard test for diagnosing esophageal motility disorders in most motility centers.[39] An HRM catheter comprises 32 circumferential and 16 impedance sensors. It is positioned from the terminal portion of the proximal esophageal segment through the distal esophagus and into the proximal stomach.[40] It may be technically difficult to place the manometry catheter across the GEJ in following cases: anatomical difficulties from the nostril to the gastric lumen, impaired GEJ relaxation due to achalasia or a large epiphrenic diverticulum, impaired swallowing, or inability of the patient to tolerate the procedure. During this procedure, pressure is checked in real time as the manometry catheter is inserted and LES pressure is measured to confirm that the catheter has passed through the GEJ. If the LES pressure remains unchecked during catheter insertion, endoscopy is additionally performed to pass the catheter tip through the GEJ using accessories, such as a snare or forceps, if necessary (Figs. 6A and 6B).

Attachment of a wireless pH capsule

Esophageal pH recording using a wireless pH capsule, such as the Bravo capsule (Medtronic, Minneapolis, MN, USA), does not require maintenance of catheter and is performed by attaching the capsule to the esophageal wall and receiving the data through an external transmitter.[41,42] Since there is no discomfort by catheter, patients are more comfortable without sleep disturbances, and daily activities such as eating and showering are possible. The endoscopic procedures for attaching the capsule can be conducted on an outpatient basis. After confirming that there are no organic lesions such as malignant tumors, ulcers, varices, or strictures in the esophagus

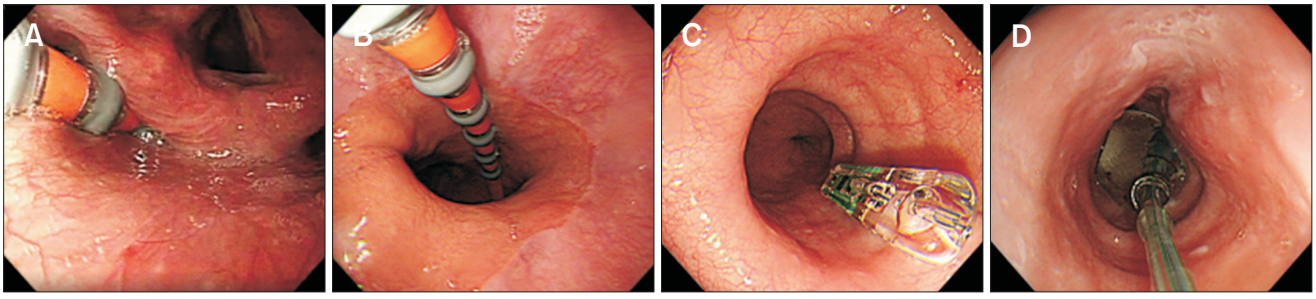

Figure 6. The use of endoscopy with additional instruments and tools in diagnosing upper gastrointestinal tract motility disorders. (A and B) Endoscopy may be used to ensure correct manometry catheter placement in patients with impaired swallowing. (C) Bravo capsule is placed 6 cm above the squamocolumnar junction via endoscopy. (D) Endoluminal functional lumen imaging probe is passed through the lower esophageal sphincter to measure the distensibility index.

on endoscopy, the pH capsule is placed approximately 6 cm above the GEJ (Fig. 6C).[43] Once the capsule is attached to the esophageal wall, the wrist or waist transmitter is activated to start measuring esophageal pH. From this point on, the patient will not feel any particular discomfort. In general, the test is performed for 24 hours to 48 hours. Once completed, the transmitter is recovered and data are analyzed.[44-46] The pH capsule spontaneously falls off within a few days; however, in rare cases, if detachment is delayed or patients complain of discomfort, endoscopic removal may be needed.[46]

Endoluminal functional lumen imaging probe

The recently introduced endoluminal functional lumen imaging probe (EndoFLIP) impedance planimetry system enables evaluation of the function of the GEJ through real-time assessment of its distensibility, compliance, diameter, and cross-sectional area during endoscopy (Fig. 6D).[47] This technology was introduced to screen for various esophageal motility disorders that are difficult to diagnose with esophageal manometry by accurately measuring GEJ distensibility.[48] The clinical usefulness of this test as both a diagnostic tool and a device for guiding and predicting treatment response is increasing.[49] EndoFLIP is a catheter with a total length of 240 cm and an outer diameter of 3 mm. It has an 8-cm- or 16-cm-long balloon mounted distally and 16 sensors spaced 5 mm or 10 mm apart in the balloon. To insert the catheter, an endoscope is placed in the hypopharyngeal area and then inserted into the pyriform sinus through the posterior pharyngeal wall while the catheter is visualized. Alternatively, the endoscope is positioned into the esophagus and the catheter is blindly inserted, similar to Levin tube insertion. The 8-cm catheter can measure the distensibility and cross-sectional area of the GEJ, and the 16-cm catheter can additionally measure secondary peristalsis of the esophageal lumen.[48] According to recent studies, based on the distensibility index (DI), 2 mm^2/mmHg or less is considered abnormal, and 3 mm^2/mmHg or more is considered normal.[48]

In addition to the LES, EndoFLIP is also used to evaluate the correlation between the dynamics and symptoms of the pyloric sphincter.[50-52] Endoscopy is first performed, and a catheter is placed through the pylorus. During endoscopy, the catheter is inserted into the stomach, with the end of the catheter (the part without the sensor) held using rat tooth forceps, and pushed into the duodenum. According to a previous study, pyloric elasticity, evaluated by EndoFLIP, was significantly reduced in patients with gastroparesis compared with that in healthy individuals.[52] A pylorus DI value of 10 mm^2/mmHg or higher is considered normal; lower DI values may be related to gastroparesis.[52-54]

Part II. Therapeutic Role in Motility Disorders

Botulinum Toxin Injection

Botulinum toxin injections (BTIs) block acetylcholine exocytosis from cholinergic nerve endings, inhibiting muscle contraction and finally causing muscle relaxation.[55] BTIs are a treatment option for several upper gastrointestinal tract motility disorders such as achalasia, diffuse esophageal spasm, and gastroparesis (Fig. 7A).[56] They are especially useful as a safe alternative treatment in patients with considerable risk factors for invasive procedures.[57,58] Symptom

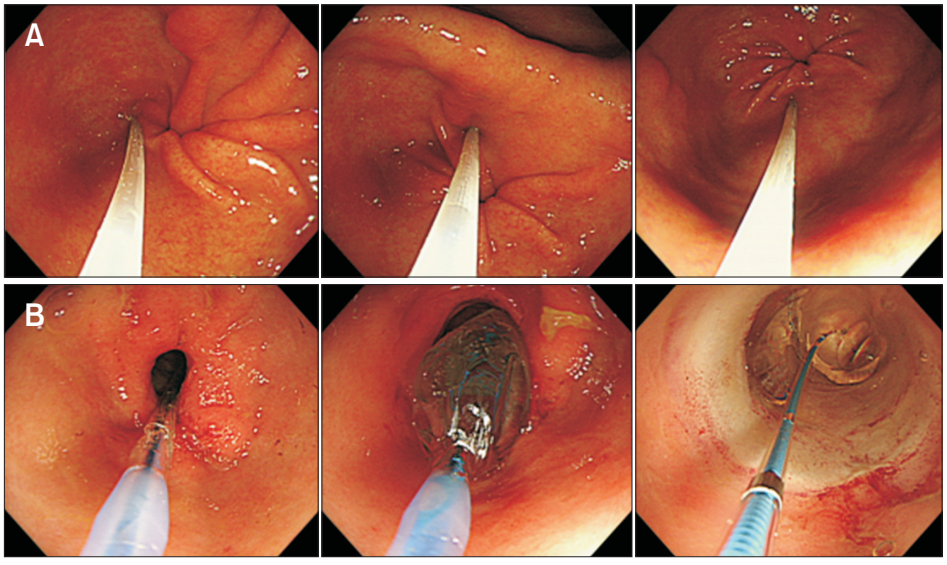

Figure 7. Endoscopic procedures for gastroparesis treatment. (A) Botulinum toxin is injected into the pyloric sphincter. (B) Balloon dilatation of the pylorus is performed to decrease pyloric resistance and improve gastric emptying.

improvement of gastroparesis after BTI has been reported in several previous studies; however, the effectiveness has not been clearly demonstrated in randomized controlled studies.[55,59] The problem with BTI treatment is that the effective area and depth of injection have not been fully established. The effects of BTI treatment are not permanent, lasting only 3 months on average.[55]

Pneumatic Balloon Dilatation

Pneumatic balloon dilatation (PBD) is one of the treatment modality for achalasia that is relatively noninvasive and relatively easy to perform using pneumatic balloons (30-40-mm diameter).[60] During endoscopy, a guidewire is placed in the stomach, the endoscope is withdrawn until the guidewire is caught outside the patient's mouth, and a balloon catheter is inserted along the guidewire and positioned over the GEJ. Then, the balloon is fully inflated and kept for about 1 minute to 3 minutes while the balloon waist is confirmed to be obliterated endoscopically.[61] In a previous study, there was no significant difference between PBD and laparoscopic Heller myotomy success rates; however, patients treated with balloon dilatation required more repetitive interventions to maintain therapeutic success.[62] Further, the recently introduced POEM resulted in a significantly higher therapeutic success rate compared with PBD among treatment-naive patients with achalasia.[63] Although POEM is considered a first-line treatment for achalasia, PBD remains a useful treatment option for these patients, especially those at risk of general anesthesia complications or those in a hospital that does not have facilities for POEM.

Pyloric balloon dilatation can be performed for gastroparesis treatment. Large-diameter balloons (15-18-mm diameter) are usually used in through-the-scope techniques to dilate the pylorus (Fig. 7B). A study has recently reported that pyloric balloon dilatation is effective at 2 months in 50% of patients with refractory gastroparesis.[64] Although research on pyloric balloon dilatation is lacking and the technique seems less effective than gastric POEM (G-POEM), this treatment should be considered in selected patients, because it is easy to perform, has fewer complications than G-POEM, and is accessible by many gastroenterologists practicing endoscopy.

Endoscopic Myotomy

POEM is a procedure that selectively excises the muscular layer of the lower esophagus and GEJ by approaching the esophageal submucosa by endoscopy. It has gradually expanded its boundaries to include treatment of esophageal spastic disorders and achalasia.[65] Many studies have recently reported the safety and efficacy of POEM for achalasia, with a short-term clinical success rate of 90-100% and a long-term clinical success rate of approximately 90%.[66,67] POEM can also be considered a safe and effective treatment option for patients whose initial endoscopic treatment for achalasia failed.[68,69] Further, POEM can be a feasible salvage treatment for patients with persistent symptoms after laparoscopic Heller myotomy.[70] To prove this, additional large-scale studies with longer follow-up periods are necessary.

Symptomatic esophageal diverticula require mechanical treatment to allow food materials to flow into the gastric lumen and improve the symptoms.[20] Surgical treatment, including diverticulectomy with esophageal myotomy, is a standard treatment strategy. Nowadays, diverticular POEM (D-POEM) is considered a novel and feasible alternative treatment option.[71,72] During this procedure, the diverticular septum can be precisely exposed and completely excised using the POEM technique. The recurrence rate was reported to be 10.5%, mostly due to incomplete septotomy.[73] Most symptomatic epiphrenic diverticula are associated with an esophageal motility disorder, and further cardiomyotomy may be needed to resolve the symptoms and prevent recurrence.[74] We investigated the clinical outcomes of D-POEM (including combined cardiomyotomy and septotomy) in patients with symptomatic epiphrenic diverticulum, and we showed favorable outcomes, including a decreased mean dysphagia score in all patients and absence of symptom recurrence, during a median follow-up period, post-procedure.[75]

Emerging novel POEM techniques have been increasingly applied successfully for gastroparesis treatment. One such example is G-POEM, a type of peroral pyloromyotomy (Fig. 8).[76] Pyloric dysfunction, including pylorospasm, can significantly contribute to the symptom pathogenesis observed in some patients with gastroparesis.[32] G-POEM may be considered as a potential localized therapy for this subset of patients. A recently published meta-analysis revealed that G-POEM is an effective and feasible treatment option for patients with refractory gastroparesis, with high technical and clinical success rates (100% and 82%, respectively).[76]

Endoscopic procedure for gastroesophageal reflux disease

Endoscopic GERD treatments are considered appropriate for patients who are in the early GERD spectrum and those whose standard surgical approaches are limited due to altered anatomy. A number of endoscopic treatment devices and procedures are currently employed for GERD treatment.

The Stretta system was approved by the FDA in 2000 as an anti-reflux endoscopic procedure for GERD. The Stretta instrument comprises a high-frequency module, 20-Fr balloon-shaped

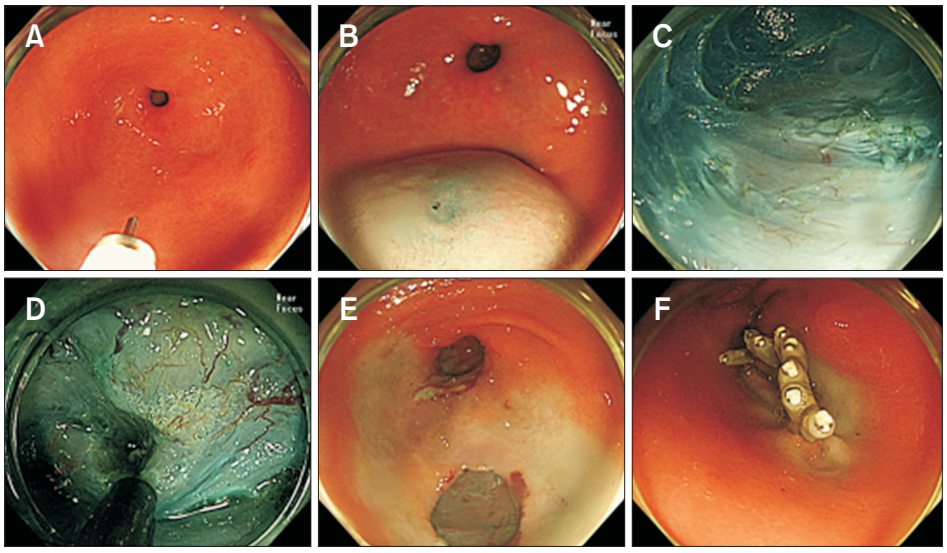

Figure 8. Gastric peroral endoscopic myotomy for gastroparesis. (A) The distal antrum and pylorus were observed. (B) A submucosal bleb and mucosal opening were created after submucosal injection 2-3 cm proximal to the pylorus. (C) Submucosal tunneling was started from the mucosal opening and continued toward the pyloric ring. (D) A complete myotomy was performed from the distal antrum to the pyloric ring. (E) After myotomy, the pyloric opening was significantly larger. (F) The mucosal opening was closed with hemoclips.

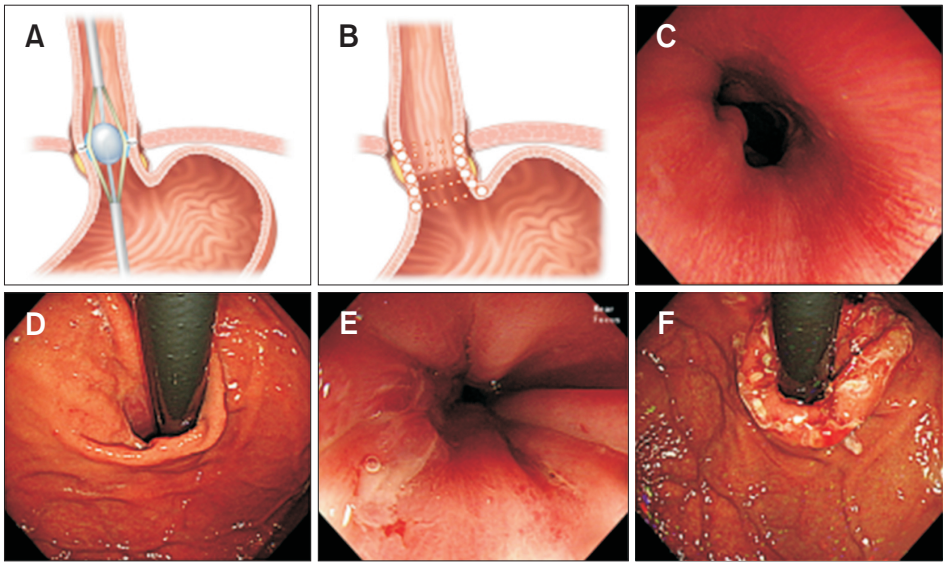

Figure 9. The Stretta system for gastroesophageal reflux disease treatment. (A) The Stretta catheter is delivered endoluminally and located at the gastroesophageal junction. (B) Temperature-controlled radiofrequency energy is delivered to the muscle at 6 levels from above the Z-line to the gastric cardia. (C) Pre-procedure endoscopic image of the lower esophagus, Z-line, and (D) gastric cardia. (E) Post-procedure endoscopic image of the lower esophagus, Z-line, and (F) gastric cardia.

bogey tip, and catheter with a maximum dilatation diameter of 3 cm.[77] The catheter is equipped with four needle-shaped electrodes. When the balloon is fully inflated, the 4 electrodes extend from the balloon catheter into the muscle and a radiofrequency energy of 60-300 J is delivered to the muscle at 6 levels across the GEJ (Fig. 9). The Stretta procedure is indicated for patients with GERD confirmed by pathologic esophageal acid exposure on pH monitoring, gross regurgitation, and reflux esophagitis. All patients should have adequate esophageal peristalsis and LES relaxation on HRM. Exclusion criteria for the Stretta procedure are as follows: hiatal hernia 2 cm or larger, LA grade C to D esophagitis, Barrett's esophagus, absence of response to proton pump inhibitors (PPI), negative pH impedance study results, and very low LES pressure (< 5 mmHg).[78] Stretta has been reported to be an effective endoscopic procedure in improving heartburn scores and quality of life in multiple randomized controlled trials.[79-81]

Anti-reflux mucosectomy (ARMS) is an effective procedure in which an anti-reflux mechanism is created by causing scar formation after healing of the mucosal defect and remodeling of a mucosal flap valve at the gastric cardia.[82,83] It is performed using a conventional endoscopic mucosal resection technique and does not require costly proprietary devices (Fig. 10). The best candidates for ARMS are patients with PPI-refractory GERD in the absence of hiatal hernia.[84] Several studies have reported that this technique

is safe and feasible; over two-thirds of patients in all studies had improved symptom scores, while some demonstrated improved esophageal acid exposure and decreased PPI use.[85-88] Despite its effectiveness, ARMS is difficult to standardize because the procedure entirely depends on each endoscopist's skill. Additionally, the appropriate size and depth of mucosa to be resected and the level of resection have yet to be established.

Another novel endoscopic treatment for GERD is concomitant endoscopic fundoplication following POEM (POEM-F). Previous studies have reported post-POEM GERD incidence rates as high as 15-88%, and since there are occasional case reports regarding post-POEM Barrett's esophagus and cancer, post-POEM GERD remains a significant problem.[89-91] POEM-F, which was introduced recently to reduce post-POEM GERD, involves reproducing surgical Dor fundoplication in which the gastric fundus is wrapped anteriorly 180° around the esophagus (Fig. 11).[92,93] This technique showed excellent short-term outcomes for controlling post-POEM GERD during a median of 12 months.[94] Further large-scale studies and long-term data are needed to demonstrate its clinical benefits.

Endoscopic Esophagoplasty for Megaesophagus

End-stage achalasia occurs in < 5% of all patients with achalasia and is characterized by a tortuous, massively dilated esophagus, greater than 6 cm in diameter.[95] It causes severe dysphagia, regurgitation, recurrent aspiration pneumonia, and weight loss.[96] The treatment option for these patients is limited, and they may require esophagectomy, which is associated with high mortality and morbidity. A recent case report has introduced a novel endoscopic approach of esophagoplasty for megaesophagus with sump stasis for end-stage achalasia.[97] The lateral margins of the esophageal sump were marked with coagulation; then, an endoscopic suturing device was used to apply full-thickness running sutures in a triangular fashion to crease the sump and straighten the esophagus. Positive outcomes included sump reduction and improved esophageal emptying over the GEJ. Endoscopic esophagoplasty is an alternative treatment option for patients with end-stage achalasia-associated megaesophagus who have high surgical risks due to underlying diseases.

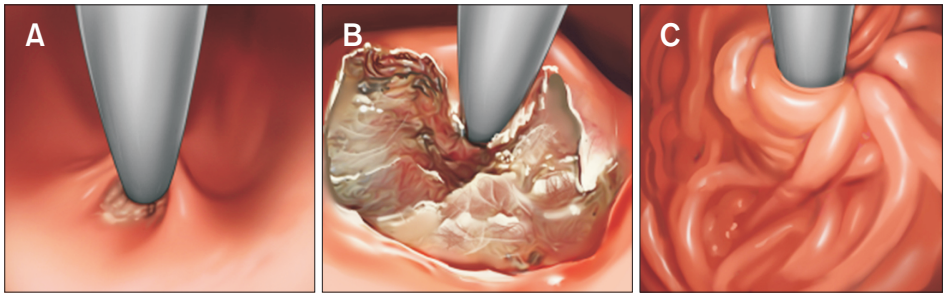

Figure 10. Anti-reflux mucosectomy (ARMS) for gastroesophageal reflux disease treatment. (A) Endoscopy in the J-turn maneuver demonstrated hiatal hernia without a sliding component. (B) After ARMS, two-thirds circumferential artificial ulcer, which was centered at the lesser curve, was observed, and the mucosal flap valve at the greater curve was preserved. (C) The mucosal valve was re-shaped and stitched to the lesser curve of the gastric cardia.

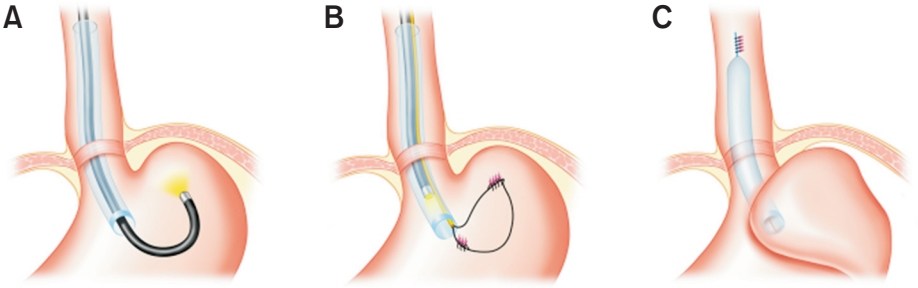

Figure 11. Peroral endoscopic myotomy (POEM) and fundoplication. (A) POEM is performed at the anterior wall of the esophagus. (B) A submucosal endoscope is advanced through the peritoneum into the abdominal cavity, distal anchoring is made at the anterior gastric wall, and a second stitch is applied at the distal end of the submucosal tunnel. (C) Suture is tightened up and fundoplication is created.

Conclusion

In this article, the diagnostic and therapeutic roles of endoscopy in various upper gastrointestinal tract motility disorders were reviewed. Endoscopy is associated with a low risk of complications, inexpensive, and relatively easy to access and perform. It can provide significant clues in diagnosing gastrointestinal motility disorders. With the development of endoscopic instruments and techniques, novel treatments for gastrointestinal motility disorders have been introduced. Further studies investigating their effectiveness and safety are needed.

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