Reflux Esophagitis and Barrett's Esophagus

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Jae Myung Park

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Diagnosis of reflux esophagitis can be made by endoscopic examination, when erosions or ulcerations are found in between the distal esophagus and the *Z* line. The most important role of endoscopic examination is to evaluate the severity and presence of complications by this chronic disease. Endoscopy can also differentiate other diagnoses with reflux-like symptoms, such as viral or eosinophilic esophagitis. Evaluation with endoscopy should especially be done for patients with weight loss, dysphagia, or vomiting. Barrett's esophagus is the replacement of squamous epithelium with columnar mucosa by chronic exposure of acid. Endoscopy is the principal method for the evaluation and diagnosis of Barrett's esophagus. Biopsy to confirm intestinal metaplasia in order to diagnose Barrett's esophagus should be done.

J.M. Park
Division of Gastroenterology and Hepatology,
Department of Internal Medicine,
The Catholic University of Korea, Seoul, Korea
e-mail: parkjerry@catholic.ac.kr

H.J. Chun et al. (eds.), *Clinical Gastrointestinal Endoscopy*,
DOI 10.1007/978-3-642-35626-1_4, © Springer-Verlag Berlin Heidelberg 2014

4.1 Pathophysiology

Pathologic reflux of gastric contents develops when the refluxate overwhelms the antireflux barriers of the gastroesophageal junction. The primary antireflux mechanism is the lower esophageal sphincter (LES) that is contracted to sustain a pressure above gastric pressure. Anatomic disruption of the gastroesophageal junction, commonly associated with a hiatal hernia (Fig. 4.1), contributes to the pathogenesis of reflux disease by impairing LES function [1]. Hiatal hernia is especially important in patients with severe esophagitis, peptic stricture, or Barrett's esophagus [2]. Chronic low LES pressure is the predominant mechanism in GERD patients with severe reflux, and impaired esophageal clearance also can be another pathophysiology as in patients with scleroderma (Fig. 4.2).

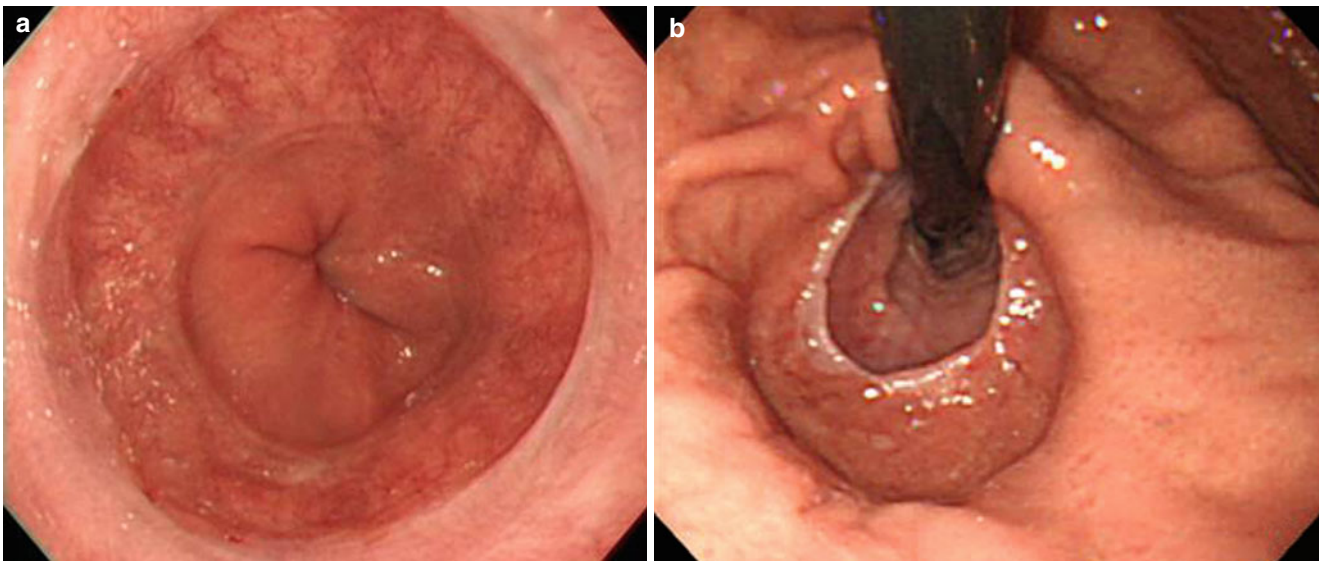

Fig. 4.1 Hiatal hernia. Hiatal hernia is defined as herniation of a portion of the stomach through the diaphragmatic esophageal hiatus. Lower esophageal sphincter pressure is lower due to the loss of the abdominal pressure and the crural diaphragm. (**a**) Forward and (**b**) retrospective view of the esophagogastric junction in a patients with a hiatal hernia

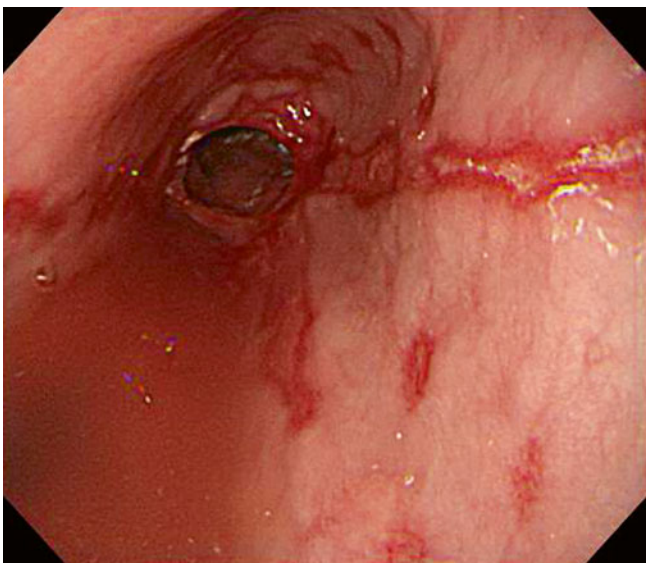

Fig. 4.2 Endoscopic finding in the patients with scleroderma. Esophageal clearance is impaired by the loss of peristalsis, which caused reflux esophagitis

4.2 Clinical Characteristics

Heartburn and regurgitation is the typical clinical picture of GERD (Table 4.1) [3]. The presence of "alarm signs"—dysphagia, odynophagia, weight loss, family history of upper gastrointestinal (GI) tract cancers, persistent nausea and emesis, long duration of symptoms (>10 years), and incomplete response to treatment—is warrant for the endoscopic evaluation.

Table 4.1 Symptoms of reflux disease

Typical	Atypical
Heartburn	Vomiting
Regurgitation	Chest pain
Odynophagia	Cough, "chronic bronchitis"
Dysphagia	Hoarseness

4.3 Indication of Endoscopic Evaluation

Upper endoscopy allows not only the diagnosis of reflux esophagitis but also detection of any complications such as strictures or Barrett esophagus. Patients who do not respond to appropriate antisecretory medical therapy or who have other clinical signs suggestive of complicated GERD should be evaluated with endoscopy. Other diagnostic modalities such as ambulatory pH monitoring, esophageal manometry, or multichannel impedance testing should also be considered. Endoscopy is also the test of choice in patients who are at risk for Barrett's esophagus. The indications for EGD in patients with GERD are listed in Table 4.2.

Table 4.2 Indications for endoscopy in patients with GERD

Reflux symptoms which are continuous or progressive after antisecretory medications
Unintentional weight loss
Dysphagia or odynophagia
Anemia
Suspicion of extraesophageal manifestations by GERD
Screening of Barrett's esophagus
Vomiting
Recurrent symptoms after endoscopic or surgical antireflux treatment

4.4 Diagnosis and Classification of Reflux Esophagitis

The endoscopic findings of reflux esophagitis are erosions or ulcerations involving the region from the distal esophagus to the *Z* line with a streaky pattern of spread, which are the result of esophageal mucosal injury and inflammation by acid exposure (Fig. 4.3). The presence of these typical endoscopic findings is diagnostic of GERD with a specificity of 90–95 %. At least 50 % of patients with reflux symptoms have normal esophageal endoscopic findings, which is named as nonerosive reflux disease.

The extent and severity of mucosal injury can be assessed endoscopically. The Los Angeles classification quantifies the length and circumference of mucosal breaks in the reflux esophagitis.

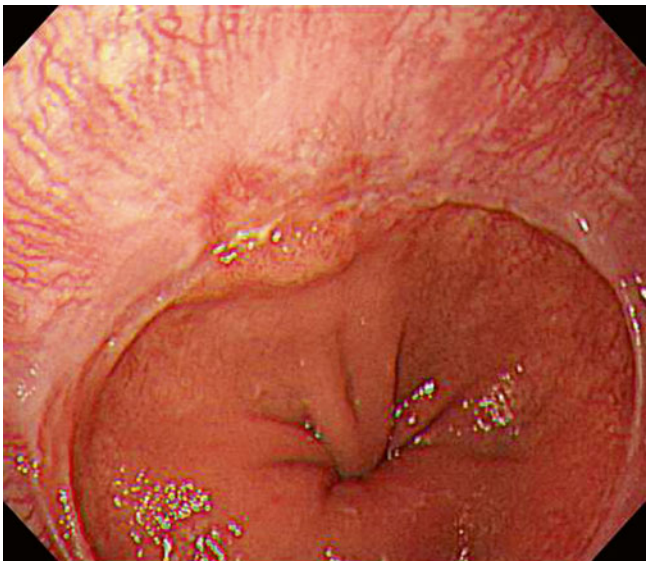

Fig. 4.3 Typical finding of reflux esophagitis. A streaky ulcer with erythema from the *Z* line

There are several classification systems for grading the endoscopic severity of erosive reflux esophagitis and associated complications. These classification systems have been primarily used in clinical trials to study the efficacy of medical therapy as treatment of reflux esophagitis. However, these systems are also useful in clinical practice for documenting disease severity. The most commonly used system is the Los Angeles classification (Table 4.3 and Fig. 4.4), which has good intra- and interobserver agreement as well as high correlation with the extent of esophageal acid exposure determined by 24-h pH monitoring. Description of the extent of endoscopic abnormalities can be used with an accepted grading system. Esophageal biopsy should be taken to exclude other diagnoses, including infectious etiologies and malignancy under the following conditions: immunocompromised patients, irregular or deep ulceration, presence of a mass lesion or nodularity, or an irregular or malignant-appearing stricture.

Table 4.3 The modified Los Angeles classification of GERD

Grade	Description
A	≥1 mucosal break no longer than 5 mm without continuation between mucosal folds
B	≥1 mucosal break longer than 5 mm without continuation between mucosal folds
C	≥1 mucosal break that is continuous between the tops of two or more mucosal folds but that involves less than 75 % of the circumference
D	≥1 mucosal break that involves at least 75 % of the esophageal circumference

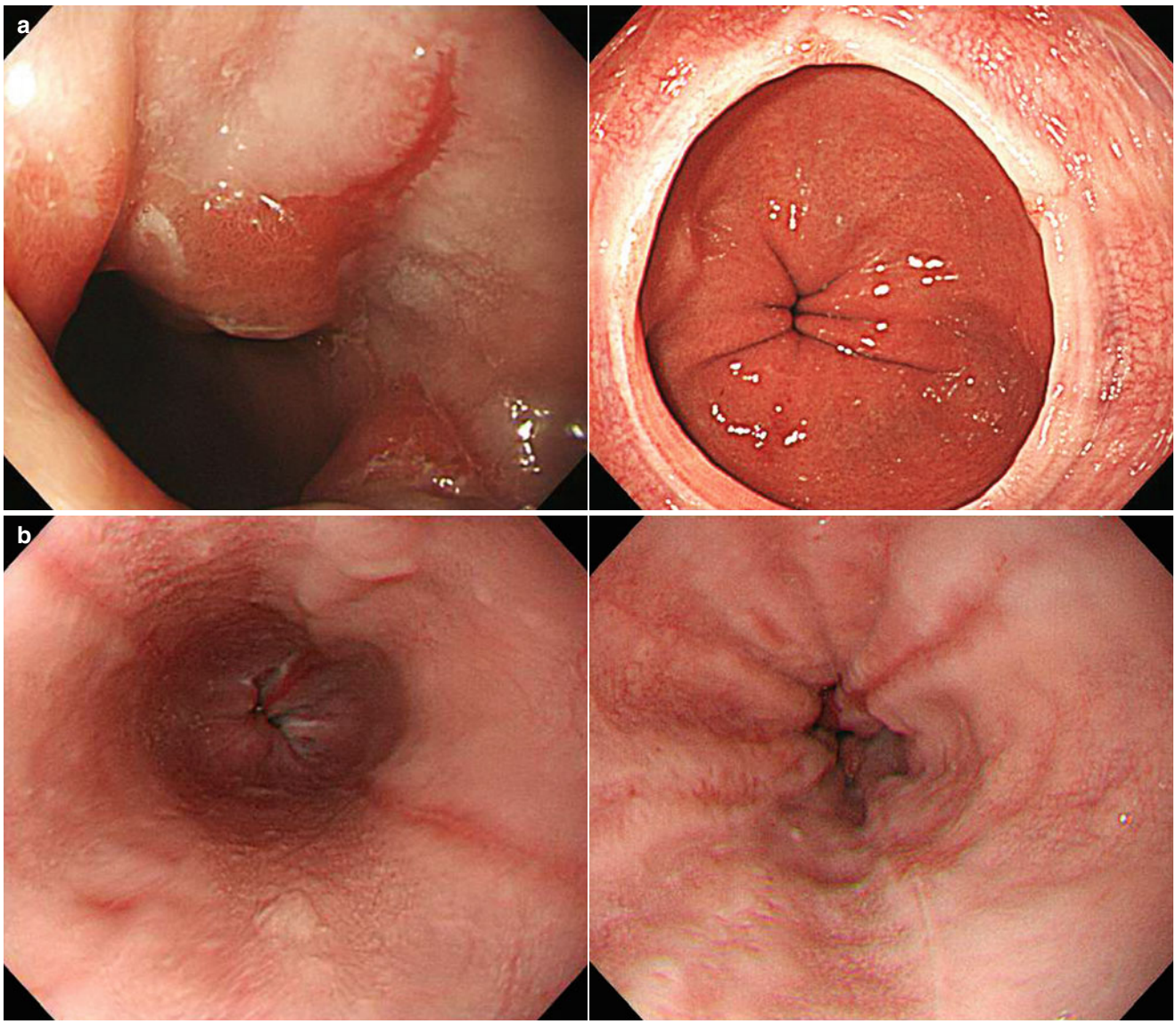

Fig. 4.4 Los Angeles classifications of reflux esophagitis. (**a**) Grade A, single-linear erosion (<5 mm in length) in the distal esophagus, (**b**) Grade B, multiple linear erosions and erythematous streaks (>5 mm in length), (**c**) Grade C, linear ulcers are becoming circumferential, (**d**) Grade D, severe disease with circumferential deep ulceration at the gastroesophageal junction above a patulous sphincter

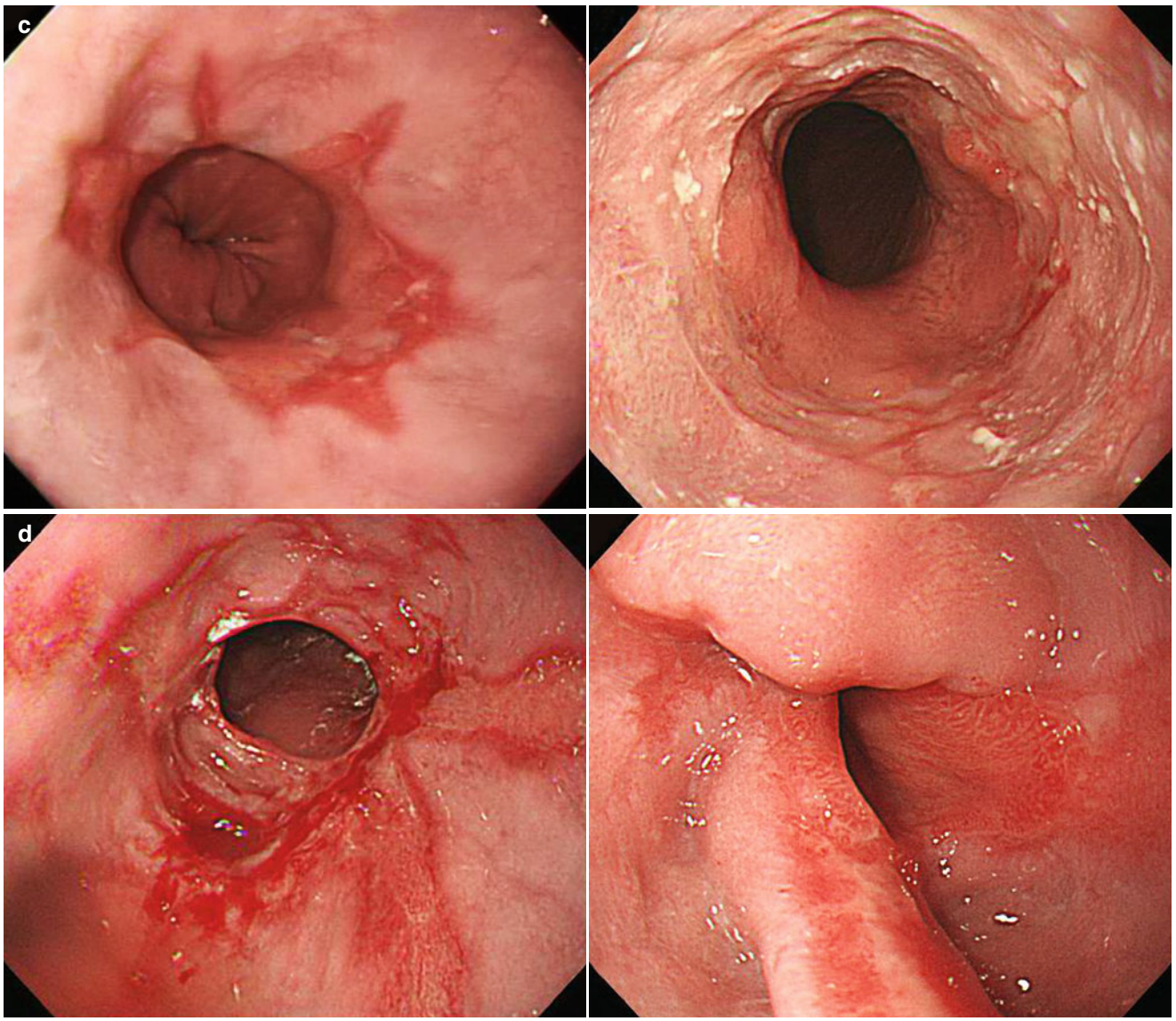

Fig. 4.4 (continued)

4.5 Minimal Change

There is another classification of GERD that the so-called minimal changes of mucosal edema, friability and erythema, whitish turbidity, fine granular change, exudates, mucosal friability, and invisibility of vessels are indicative of nonerosive reflux esophagitis. However, interobserver agreement of these findings is too low. To overcome the low agreement, new modalities have been evaluated, such as narrow band image (NBI) and magnification endoscopy. NBI system enhances visualization of microvasculature and mucosal patterns [4]. When it is combined with magnification, endoscopic abnormalities can be observed in the patients with NERD. Villous/ridge pit pattern, increased vascularity, microerosion, increased number of intrapapillary capillary loop, or tortuosity can be observed as a pattern of NERD (Fig. 4.5).

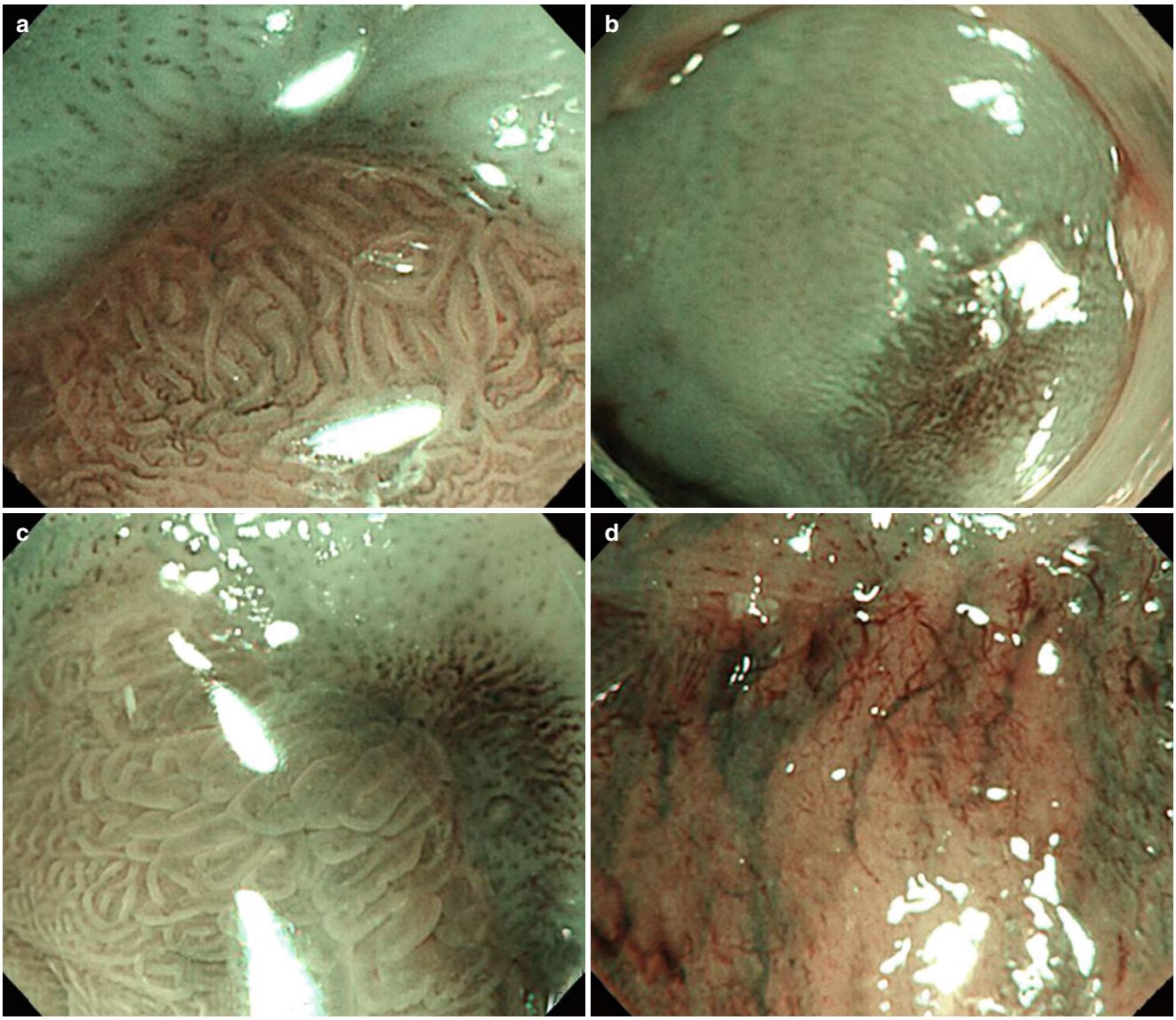

Fig. 4.5 Findings of gastroesophageal junction by narrow band imaging system with magnification in the patients with nonerosive reflux disease. (**a**) Villous/ridge pit pattern, (**b**) increased vascularity, (**c**) microerosion, (**d**) increased number of intrapapillary capillary loop, (**e**) tortuosity of microvessels

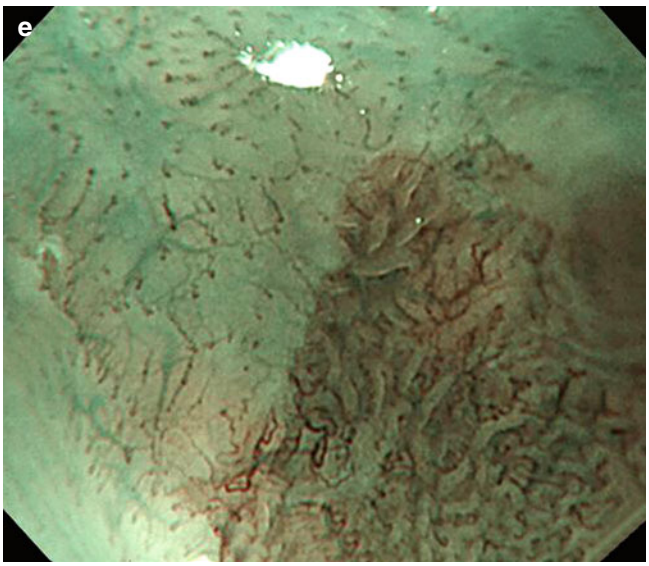

Fig. 4.5 (continued)

4.6 Treatment

4.6.1 Acid Suppressive Treatment

Proton pump inhibitors (PPIs) are the mainstay of both acute and maintenance treatment regimens for GERD. PPIs markedly diminish gastric acid secretion by inhibiting the final common pathway of the acid secretion pump (Fig. 4.6).

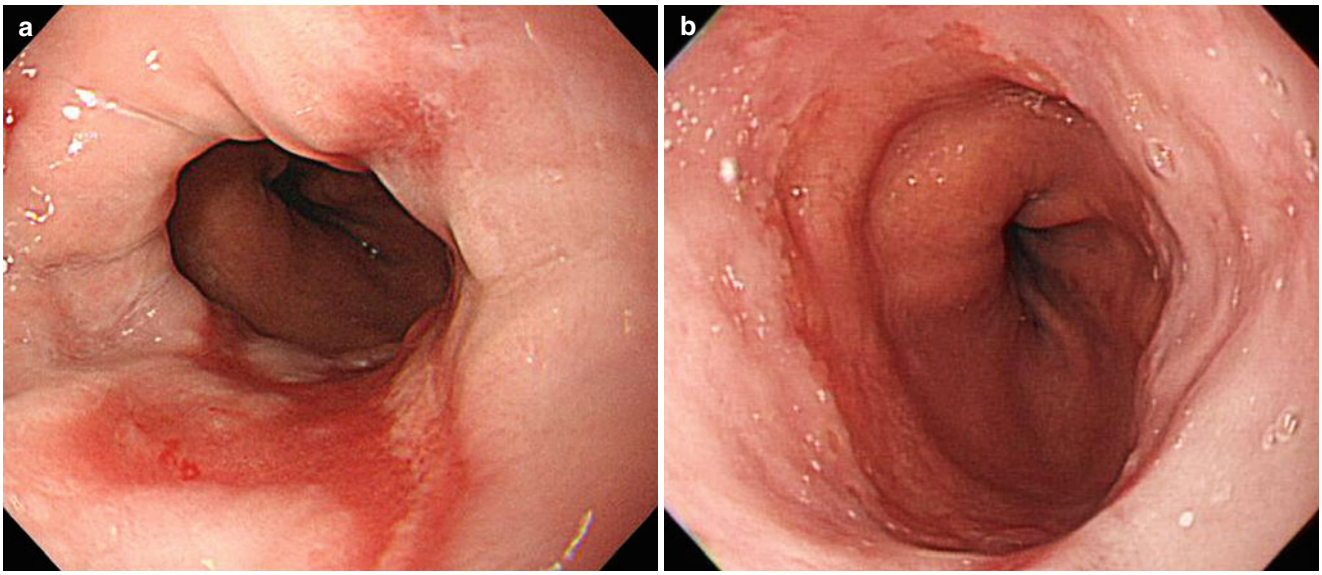

Fig. 4.6 An example of healed reflux esophagitis after treatment with proton pump inhibitor (PPI). (**a**) Before treatment, (**b**) 3 months after PPI treatment

4.6.2 Fundoplication

Laparoscopic fundoplication is a commonly performed surgery for the treatment of reflux esophagitis. The appropriately performed fundoplication should be short, straight, parallel to the diaphragm, and at the top of the stomach (Fig. 4.7).

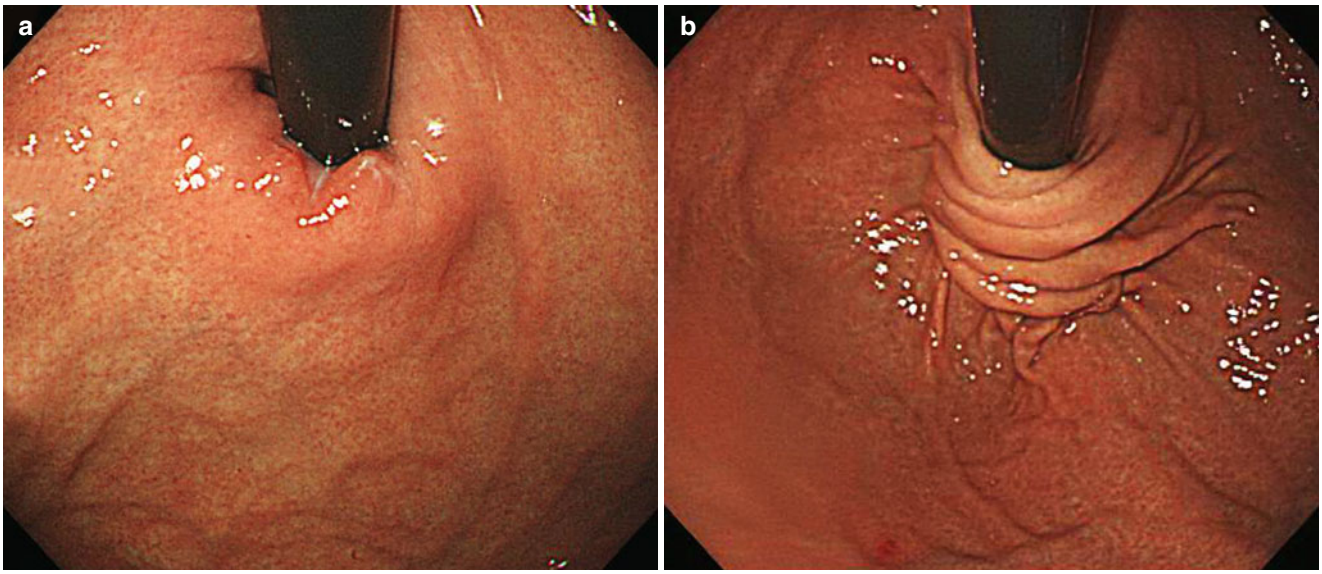

Fig. 4.7 Comparison of the retroflexed view of the esophagogastric junction before (**a**) and after Nissen fundoplication (**b**) in a patient

4.7 Sentinel Fold

The sentinel fold or polyp is a polypoid fold just distal to the esophagogastric junction (Fig. 4.8). Endoscopically, the sentinel fold is usually seen in an area of focal severe erosions or ulcerations. Biopsy of the fold reveals normal columnar epithelium with underlying acute and chronic inflammation. After aggressive antireflux therapy, the fold disappears or is significantly reduced in size.

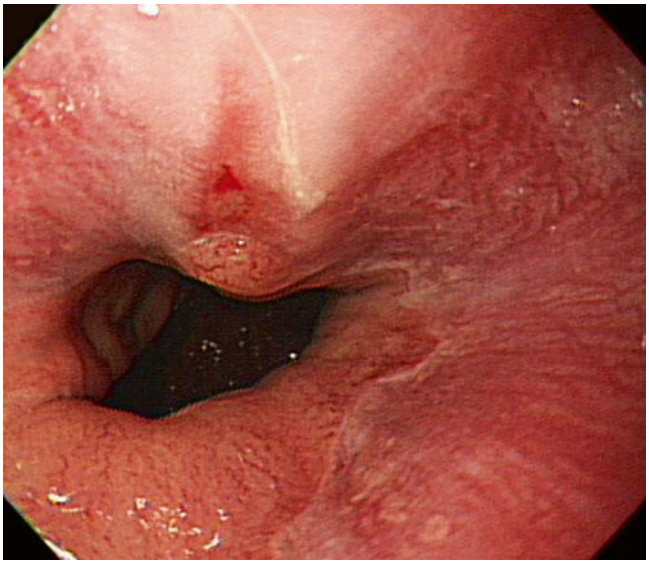

Fig. 4.8 Sentinel fold just below the esophagogastric junction with associated linear erosion

4.8 Peptic Strictures

Peptic strictures develop in the region of gastroesophageal junction as a result of long-standing GERD and inflammation with fibrosis and scarring. Most strictures are short, but some may extend for several centimeters in the distal esophagus (Fig. 4.9). The earliest change is usually a thickening of the *Z* line, followed by concentric luminal narrowing.

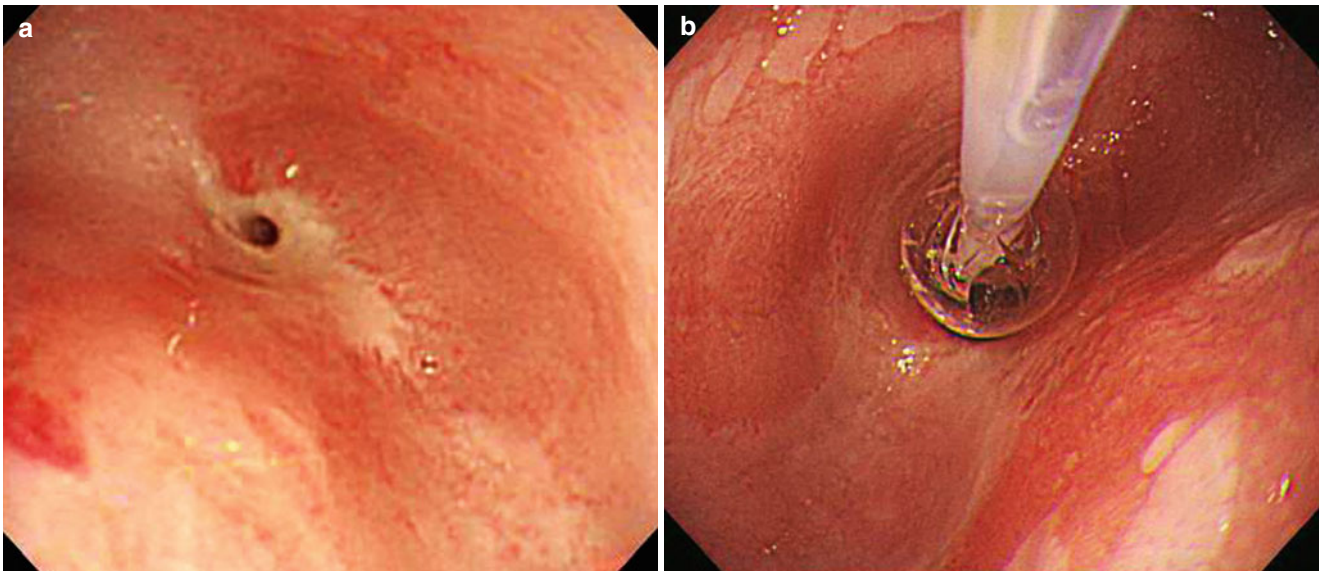

Fig. 4.9 Reflux esophagitis-associated stricture. (**a**) Tight and pinpoint structure of the distal esophagus associated with long-segment Barrett's esophagus. (**b**) Stricture was dilated with endoscopic balloon dilatation

4.9 Barrett's Esophagus

Barrett's esophagus is a condition in which the squamous epithelium of the distal esophagus is substituted with an intestinal-type columnar epithelium (specialized intestinal metaplasia, Fig. 4.10). This change occurs when the esophageal squamous epithelium which has been damaged by chronic reflux is replaced by metaplastic columnar epithelium. The importance of the finding and thus the necessity of identifying it, confirming it by biopsy, and monitoring its progression lie in the approximately 10 % risk of adenocarcinoma formation in the columnar-lined esophagus.

Endoscopy is the most accurate tool for the detection and diagnosis of Barrett's esophagus. To endoscopically diagnose the presence of Barrett's esophagus, the squamocolumnar junction and the gastroesophageal junction must be clearly identified. While proximal displacement of the squamocolumnar junction relative to the gastroesophageal junction is suggestive of Barrett's esophagus, this endoscopic appearance of salmon-colored mucosa or an irregular *Z* line, either alone or in combination, is not sufficient to make the diagnosis. Biopsy specimens should always be obtained for histologic confirmation of columnar epithelium.

In patients with Barrett's esophagus with no evidence of dysplasia on initial endoscopy, a repeated endoscopy should be performed within the next year. If no dysplasia is confirmed, these patients are considered to be at low risk to have their condition progress or develop cancer. Therefore, the interval for additional surveillance has been recommended to be every 3 years. If high-grade dysplasia is confirmed, the Barrett's epithelium should be removed. Recently, alternative endoscopic treatment, such as endoscopic mucosal resection, thermal coagulation, or photodynamic therapy, has been successfully tried to cure the dysplastic Barrett's esophagus.

The Prague classification was developed to standardize the classification of Barrett's esophagus. In this classification, both the maximal length (M) (including tongues) of Barrett's esophagus and the length of the circumferential Barrett's segment (C) are measured during endoscopic examination (Fig. 4.11). These numbers can be used to follow-up the Barrett's segment over time. This system has a high degree of overall validity for the endoscopic assessment of the visualized Barrett's esophagus segment when it is >1 cm in length.

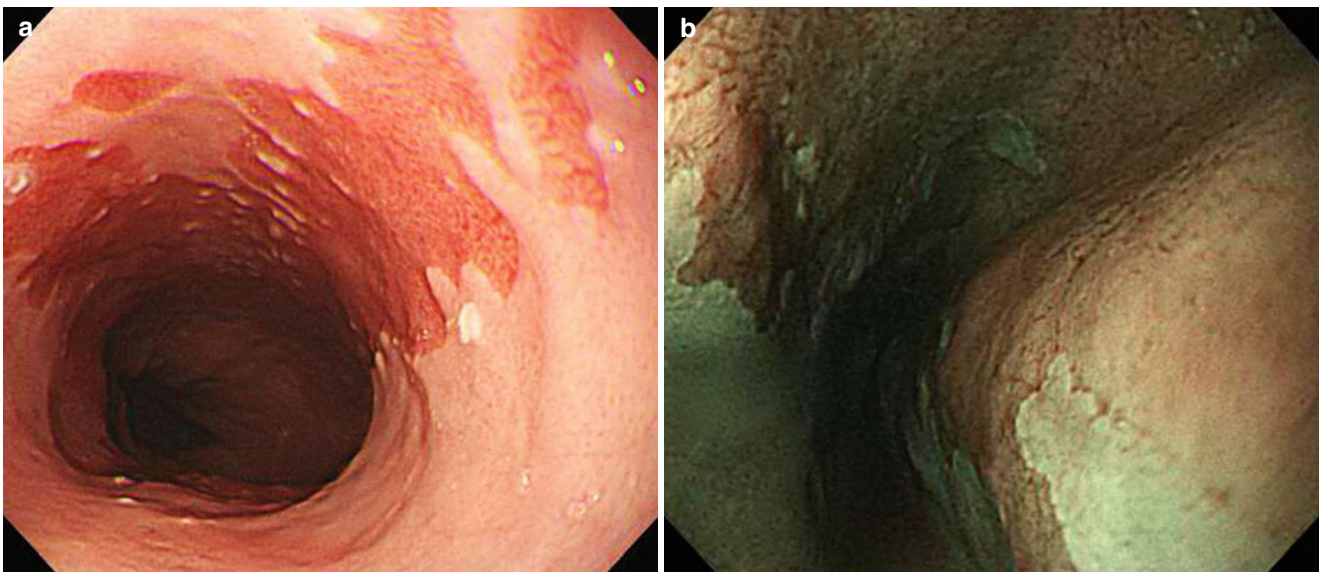

Fig. 4.10 Barrett's esophagus. The distal esophagus is lined with metaplastic columnar epithelium. The squamocolumnar junction migrated to a level of 34 cm from the incisor. (**a**) conventional white light endoscopy image. (**b**) narrow band image of Barret's epithelium

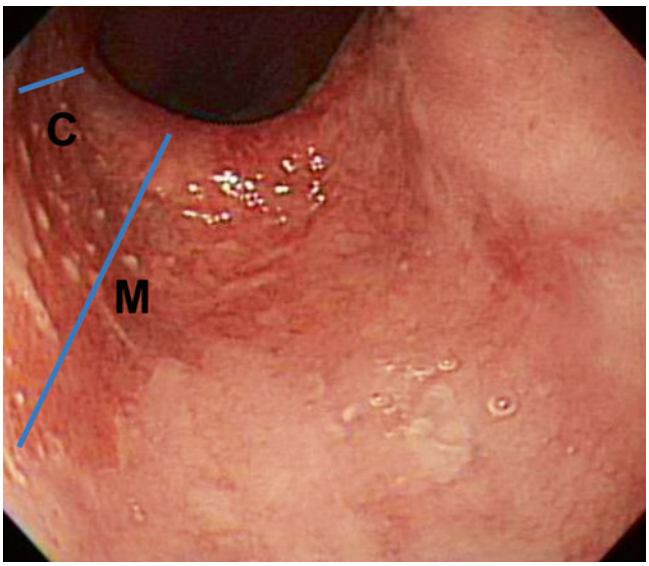

Fig. 4.11 The Prague classification of Barrett's esophagus. In this classification, both the maximal length (*M*) (including tongues) of Barrett's esophagus and the length of the circumferential Barrett's segment (*C*) are measured during endoscopy. These numbers can then be used to track the length of the Barrett's segment over time

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