



Gastritis and Gastric Ulcers

8

Sang Kil Lee

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8.1 Classification of Gastritis

The gastritis was classified by Schindler in 1922. The gastritis was classified as acute and chronic gastritis based on gross findings and histologic findings. Chronic gastritis was classified as chronic surface gastritis and chronic atrophic gastritis. In 1991, the Sydney System was designed to simultaneously reflect pathologic findings and endoscopic findings as closely as possible while maintaining a correlation with the previously described classification. Gastritis can be diagnosed as one of categories of endoscopic gastritis including erythematous/exudative, flat erosive, raised erosive, atrophic, hemorrhagic, reflux, and rugal hyperplastic gastritis. In Japan, Kimura and Takemoto advocated the classification of atrophic gastritis. Recently the Kyoto classification of gastritis was proposed at 2015. The Kyoto classification attempted to standardize the endoscopic findings with the following descriptions such as atrophy; diffuse redness; foveolar hyperplastic polyp; map-like redness; xanthoma; hematin; red streak; intestinal metaplasia; mucosal swelling; patchy redness; depressive erosion; enlarged (or tortuous) folds; sticky mucus; fundic gland polyps; spotty redness; multiple white, flat, elevated lesions; regular arrangement of collecting venules (RAC); nodularity; and raised erosions.

S. K. Lee
Department of Internal Medicine, Yonsei University
College of Medicine, Seoul, South Korea
e-mail: sklee@yuhs.ac

8.2 Gastritis

Endoscopic findings in the antrum, body, and fundus were classified according to the Sydney classification Scheme [1, 2] as edema, erythema, friability, exudates, flat erosions, raised erosions, rugal hyperplasia, atrophy, visibility of a vascular pattern, intramural bleeding spots, or nodularity.

8.2.1 Acute Gastritis

Acute gastritis is a term covering a broad spectrum of entities that induce inflammatory changes in the gastric mucosa. Acute gastritis is commonly found in the antrum of the stomach and has the characteristic features of erythema and erosion. Furthermore, linear streaks can extend to the body of the stomach (Fig. 8.1a). Another feature of acute gastritis is erosion. Erosion refers to a raised lesion with mucosal edema (Fig. 8.1b) and flat and multiple hyperemia (Fig. 8.1c). Erosions can be multiple and combined with hyperemia

(Fig. 8.1d) and have the appearance of a linear feature. Most erosions on the greater curvature side of the body are linear (Fig. 8.1e, f). Sometimes erythema, exudate, hemorrhage, and friability of mucosa can be seen in combination in cases of severe acute gastritis or gastropathy (Fig. 8.1g). Bile or thick exudate with hyperemia is a feature of acute gastritis (Fig. 8.1h).

Intramural bleeding spots are distinct features of acute gastritis (Fig. 8.2a, b) and are sometimes seen in combination with intragastric hemorrhage (Fig. 8.2c). Acute gastric mucosal lesions can also be present in symptomatic patients (Fig. 8.2d).

Bleeding features of acute gastritis can be classified as fresh (Fig. 8.3a, b) or hematin (Fig. 8.3c). Bleeding can be diffuse or can occur in multiple locations in the body (Fig. 8.3c). Sometimes diffuse oozing and exudate can be seen (Fig. 8.3a, b). Acute gastric mucosal lesions can be resolved; however, they can also ulcerate (Fig. 8.3d). Similar findings can be observed in cases of portal hypertensive gastropathy (Fig. 8.3e) and radiation gastritis (Fig. 8.3f).

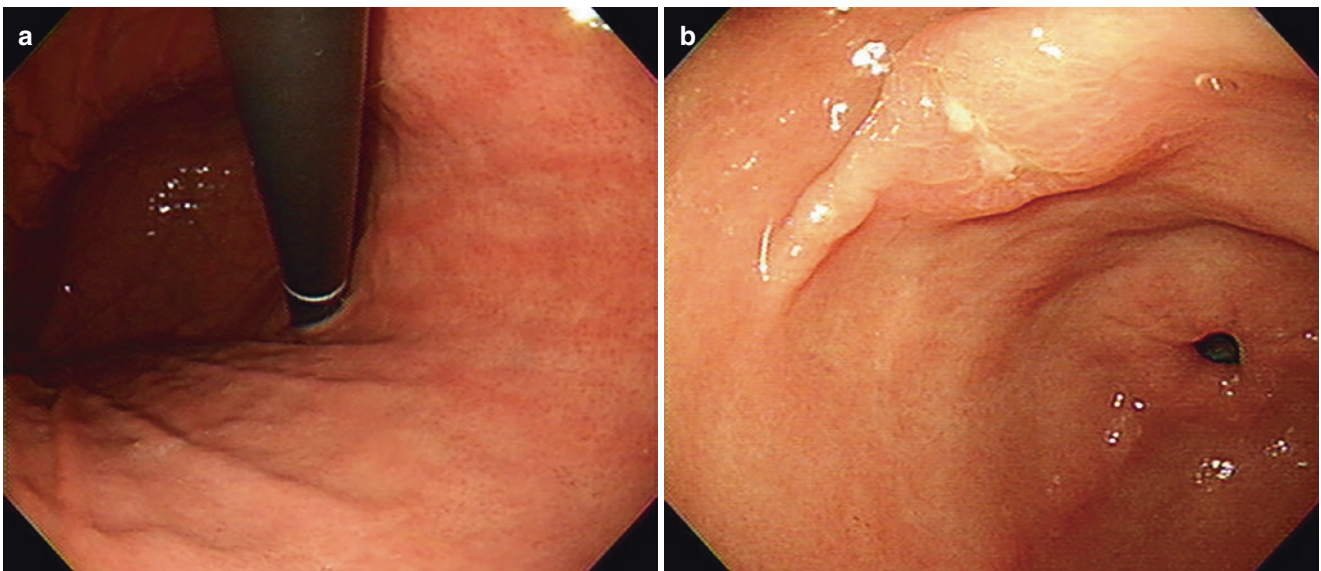


Fig. 8.1 Endoscopic features of acute gastritis. (a) Linear streak, (b) raised mucosal edema, (c) flat and multiple erosions, (d) erosions arranged in a linear pattern, (e, f) linear erosion on the greater curvature

side of the body, (g) severe hemorrhage features, (h) diffuse coating with bile juice and mucosal edema

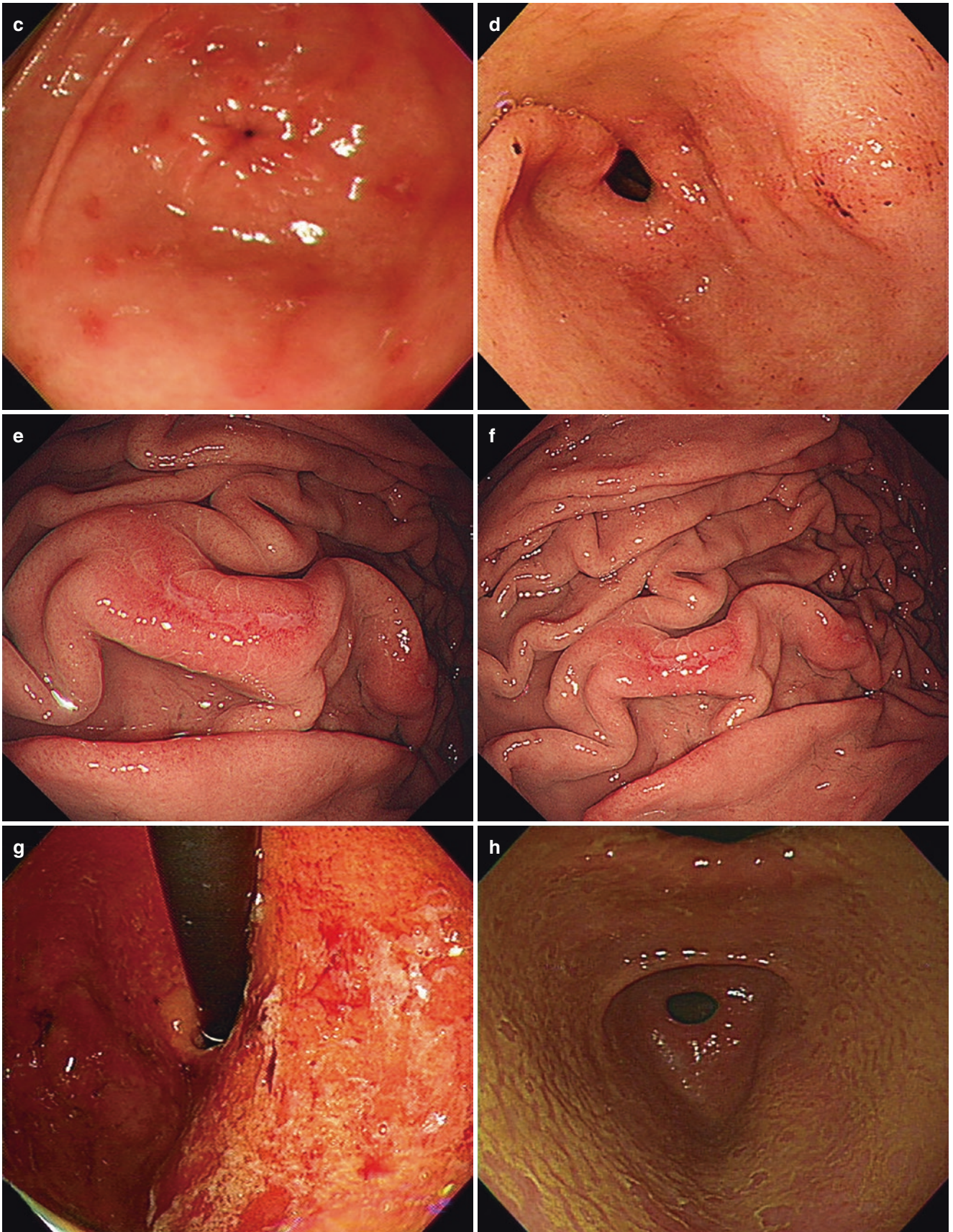


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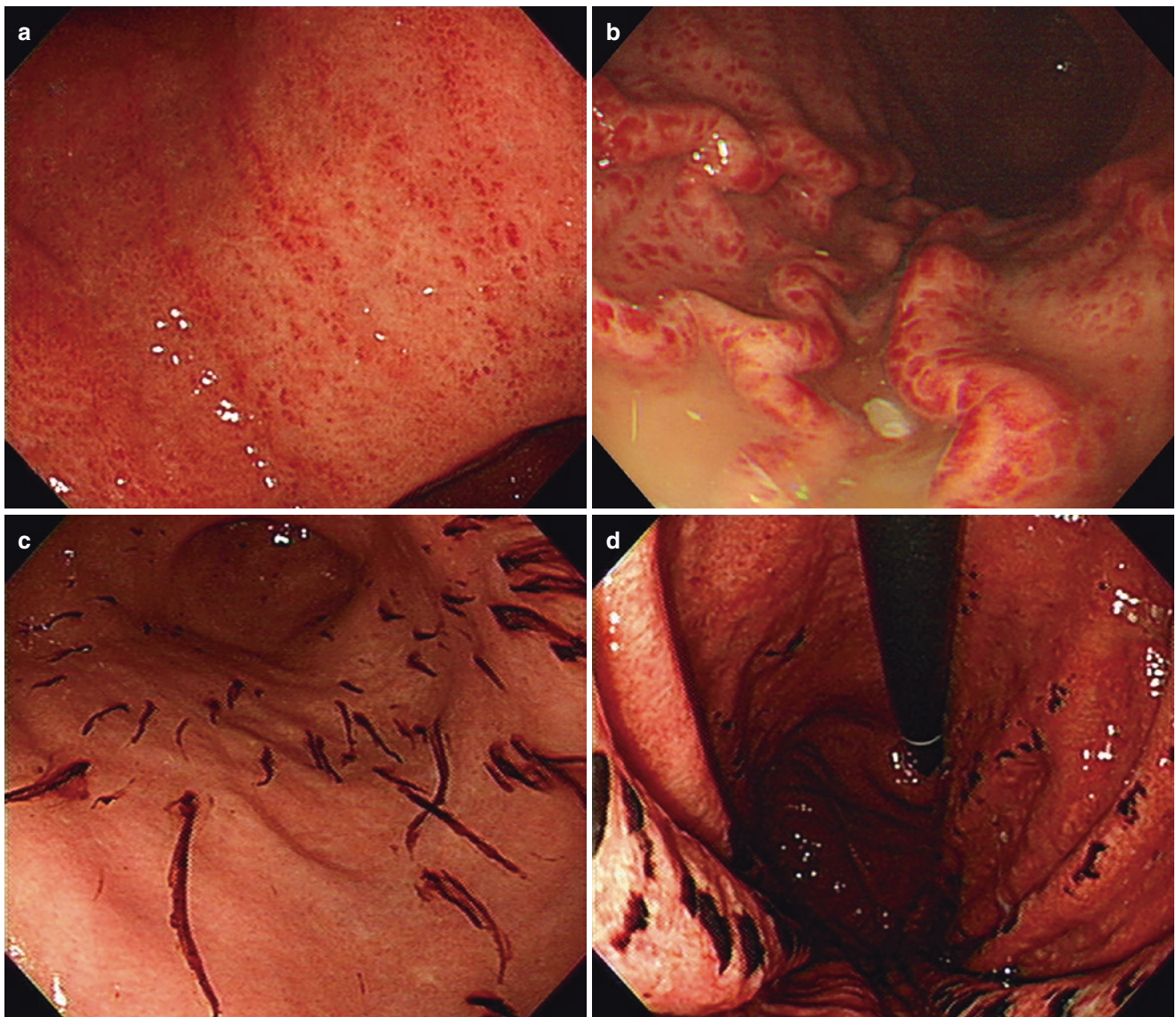


Fig. 8.2 Distinct hemorrhagic features of acute gastritis. (a) Intramucosal hemorrhagic spot scattered as millet, (b) diffuse intramucosal hemorrhagic spots on the body, (c) linear hematins scattered on the antrum, (d) mucosal bleeding from diffuse hemorrhagic lesion surrounded by hematin

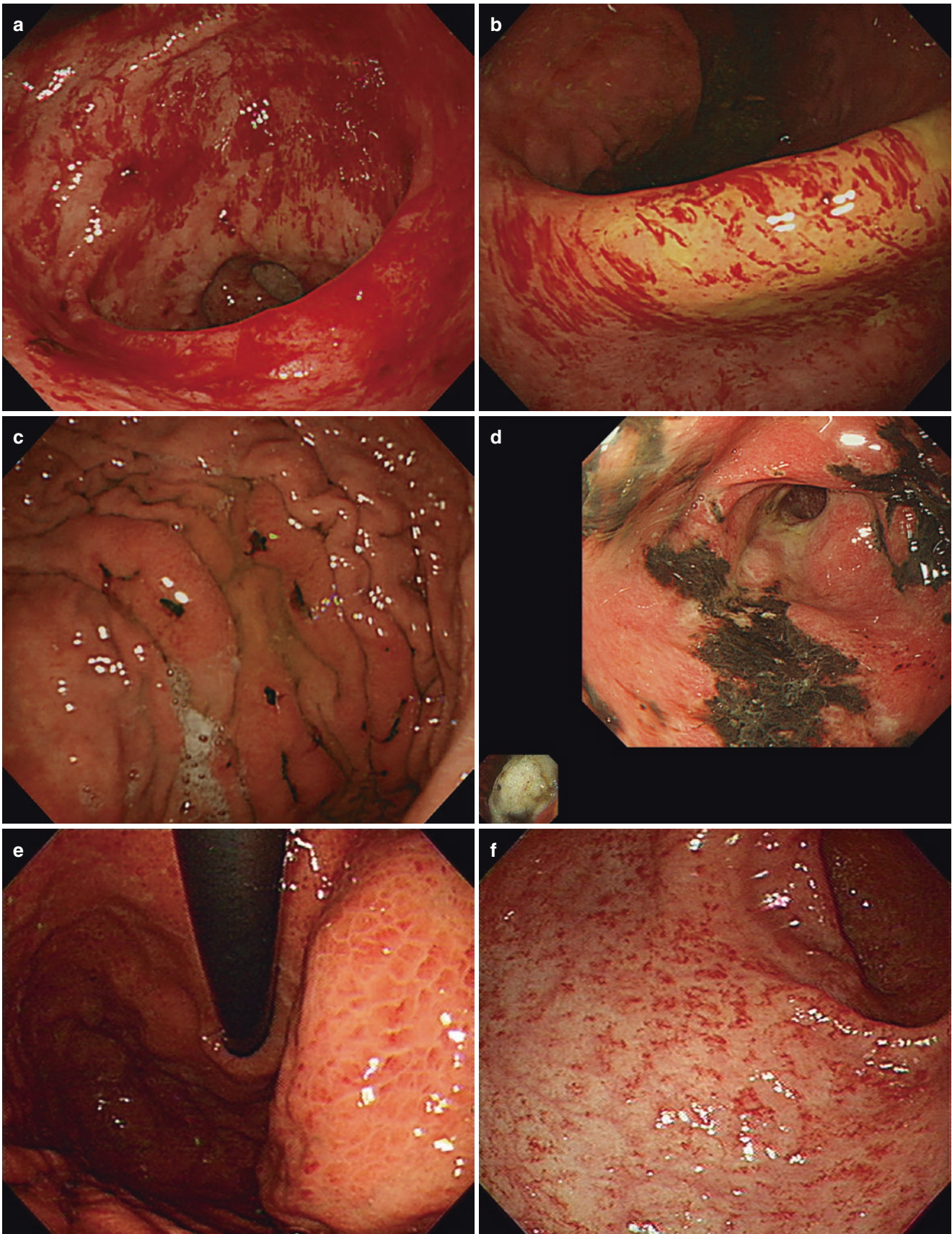


Fig. 8.3 Hemorrhagic gastritis caused by various factors. (a, b) Fresh bleeding was noted on the antrum and body. (c) Black pigmentation was noted on the greater curvature of the body. (d) Shallow and wide

ulcerations coated with hematin on the antrum. (e) Submucosal hemorrhagic on the body. (f) Diffuse intramucosal hemorrhage caused by radiation therapy

8.2.2 Chronic Gastritis

8.2.2.1 Chronic Non-atrophic Gastritis

Chronic gastritis can be roughly divided into chronic non-atrophic gastritis and chronic atrophic gastritis. In contrast to chronic non-atrophic gastritis, chronic atrophic gastritis is characterized by marked gastric atrophy with absent rugal folds and a prominent vascular pattern. Chronic superficial gastritis is a term often used to describe the initial stages of chronic gastritis. It is very difficult to differentiate acute gastritis from chronic

non-atrophic gastritis based on endoscopic finding alone. However, there is little clinical value in differentiating these two entities, because only chronic atrophic gastritis is associated with the risk of developing gastric cancer [3].

Chronic non-atrophic gastritis shares features with acute gastritis such as linear streaking and focal hyperemia (Fig. 8.4a, b). Relative unhealthy condition of the entire gastric mucosa distinguishes chronic non-atrophic gastritis from acute gastritis. Erosions were noted on the antral mucosa in cases with or without atrophic gastritis (Fig. 8.4c).

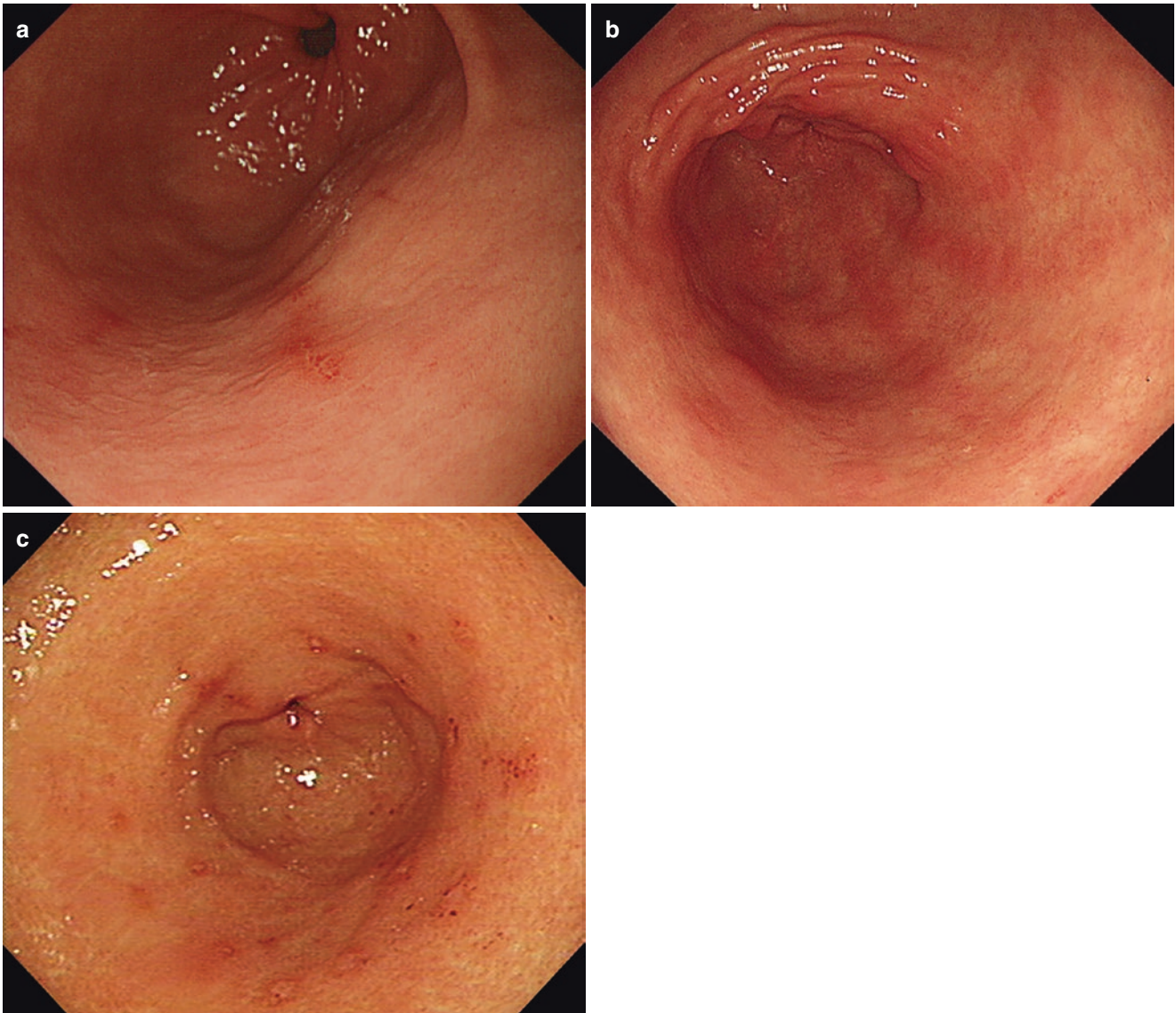


Fig. 8.4 Endoscopic features of chronic non-atrophic gastritis. (a, b) Flat and erythematous lesions on the antrum, (c) multiple erosions on the antrum

8.2.2.2 Chronic Atrophic Gastritis and Intestinal Metaplasia

Chronic atrophic gastritis is the most distinguishable pattern of chronic gastritis. It is easily detected with white light endoscopy, but there are often discrepancies between these findings and the pathologic diagnosis. Endoscopic findings of chronic atrophic gastritis are loss of mucosal glands and fibrosis of the submucosal layer induced by chronic inflammation. Endoscopic findings of chronic atrophic gastritis are antral mucosal thinning (Fig. 8.5a), color and context change of the mucosa to red and white (Fig. 8.5b), a white-colored mucosa (Fig. 8.5c, d), increased visibility of a vascular pattern (Fig. 8.5e), and loss of rugal folds with adequate air inspiration (Fig. 8.5f, g).

Atrophy caused by *Helicobacter pylori* (*H. pylori*) infection tends to start from the antrum and extends to the body, as Kimura suggested [4]. Transition line is noted in the closed type of chronic atrophic gastritis (Figs. 8.5f, g and 8.6).

Intestinal metaplasia is a premalignant condition like chronic atrophic gastritis. Intestinal metaplasia was noted as nodularity at the base of chronic atrophic gastritis (Fig. 8.7a, b). White granular plaques were also noted on closer observation (Fig. 8.7c–f). Methylene blue can differentiate intestinal metaplasia from the gastric mucosa; however, methylene blue is rarely used in current clinical practice. Indigo carmine can be used to examine the extension and severity of intestinal metaplasia (Fig. 8.7g, h). Intestinal metaplasia appears as an isolated glandular structure after staining with indigo carmine (Fig. 8.7i, j).

Confocal laser endomicroscopy (CLE) is an emerging endoscopic technology that permits high-resolution assessment of gastrointestinal mucosal histology at the cellular and subcellular levels. CLE allows optical biopsies of the stomach (Fig. 8.8a). Endoscopic criteria for intestinal metaplasia based on CLE findings are a villous-like gastric epithelium and dark (no fluorescein uptake) goblet cells in the gastric columnar epithelium (Fig. 8.8b).

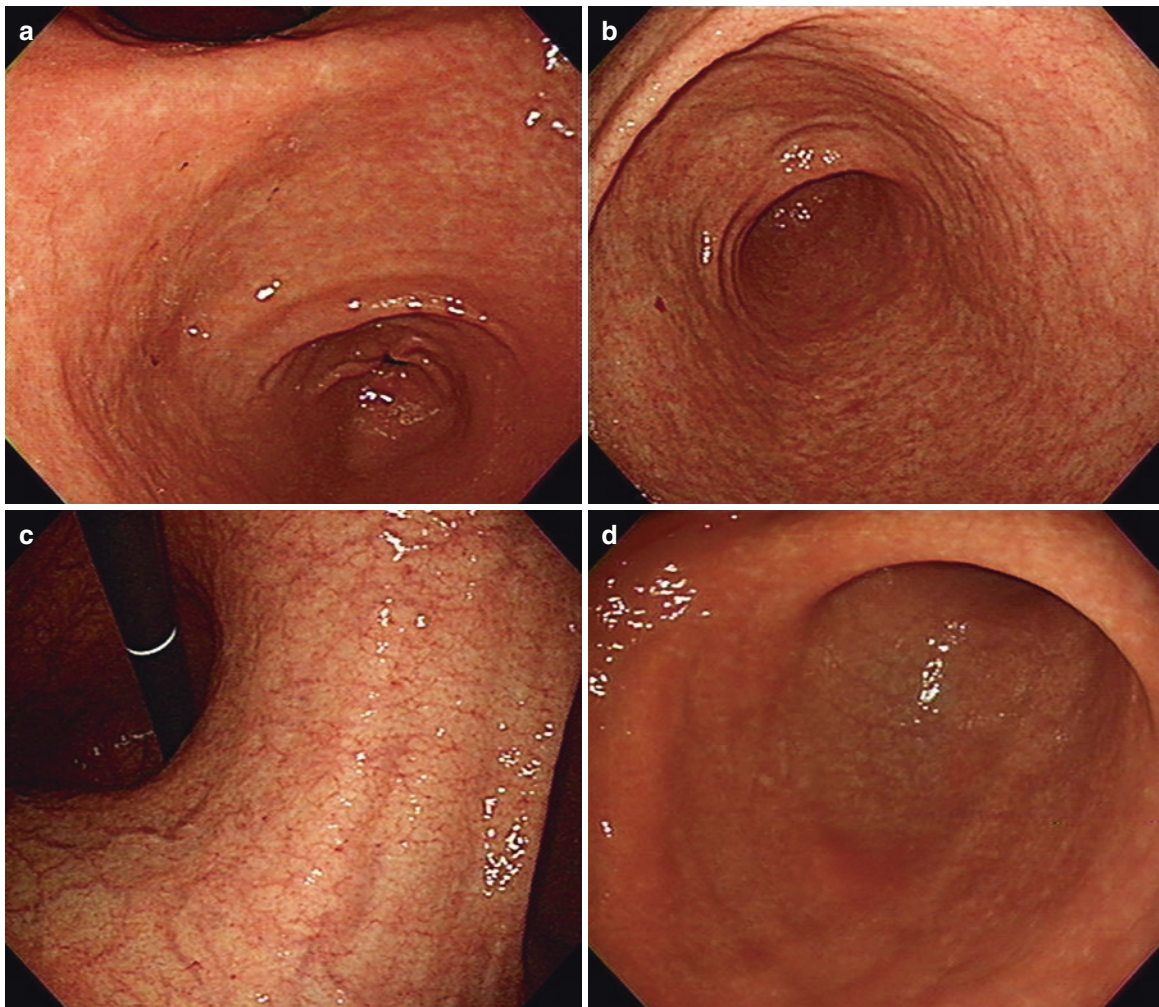


Fig. 8.5 Endoscopic features of chronic atrophic gastritis. (a) Thinning of the antral gastric mucosa, (b) loss of moisture and red and white color changes, (c) whitish color change of the mucosa, (d) loss of

mucosal moisture and change to a white color, (e–g) prominent vascularity and loss of rugal folds in the body

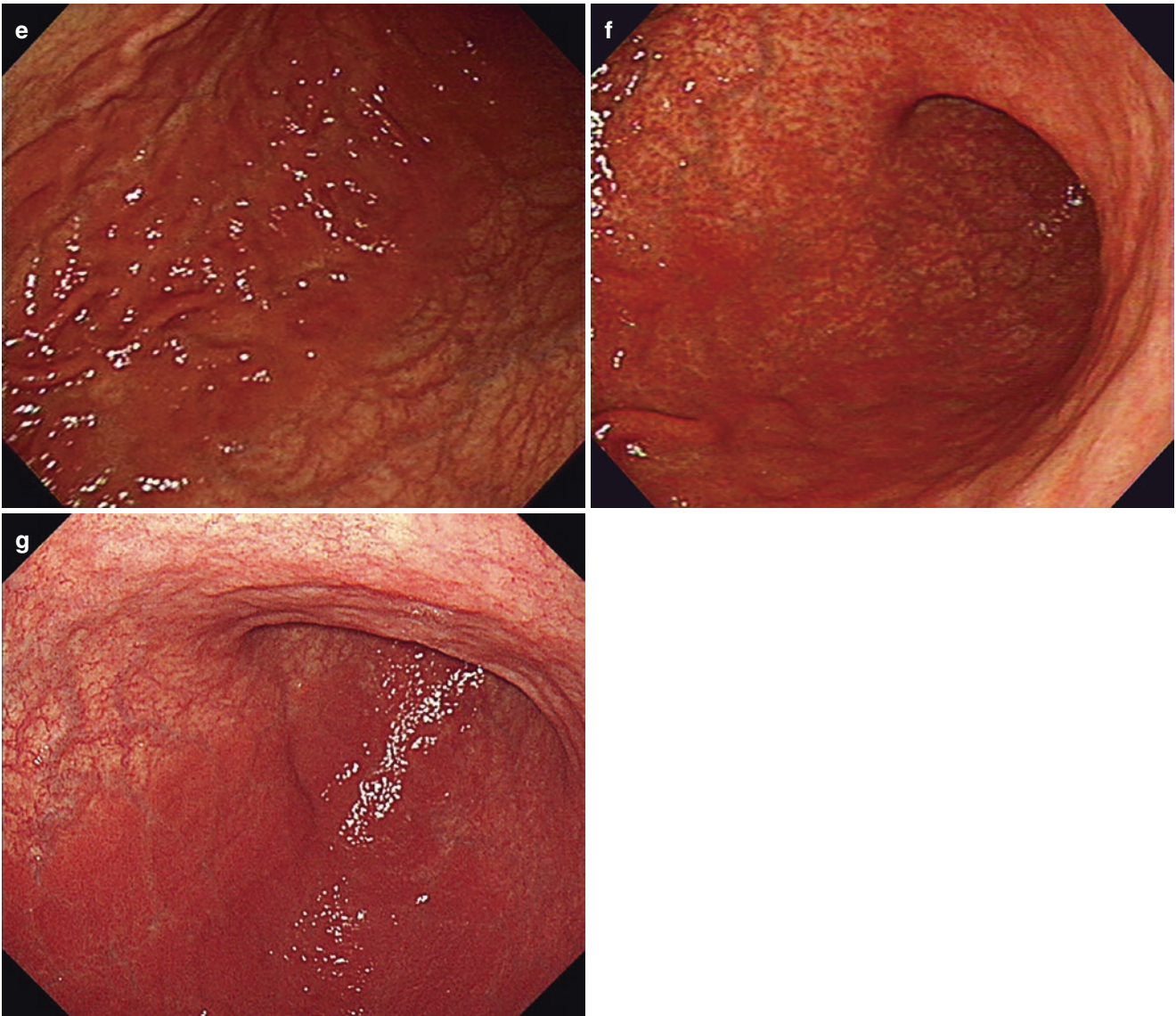


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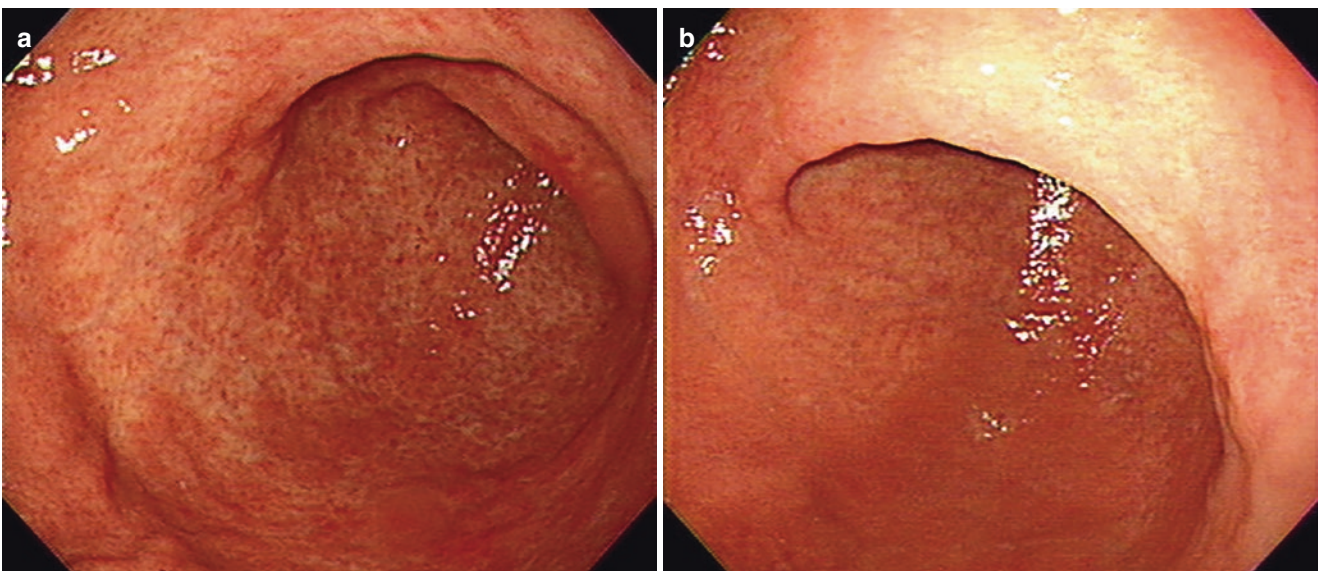


Fig. 8.6 Transition line of atrophy (a) hypothetical line separating the whitened part of the lesser curvature and the flaring part of the greater curvature, (b) more distinct line separating lesser curvature and the the greater curvature

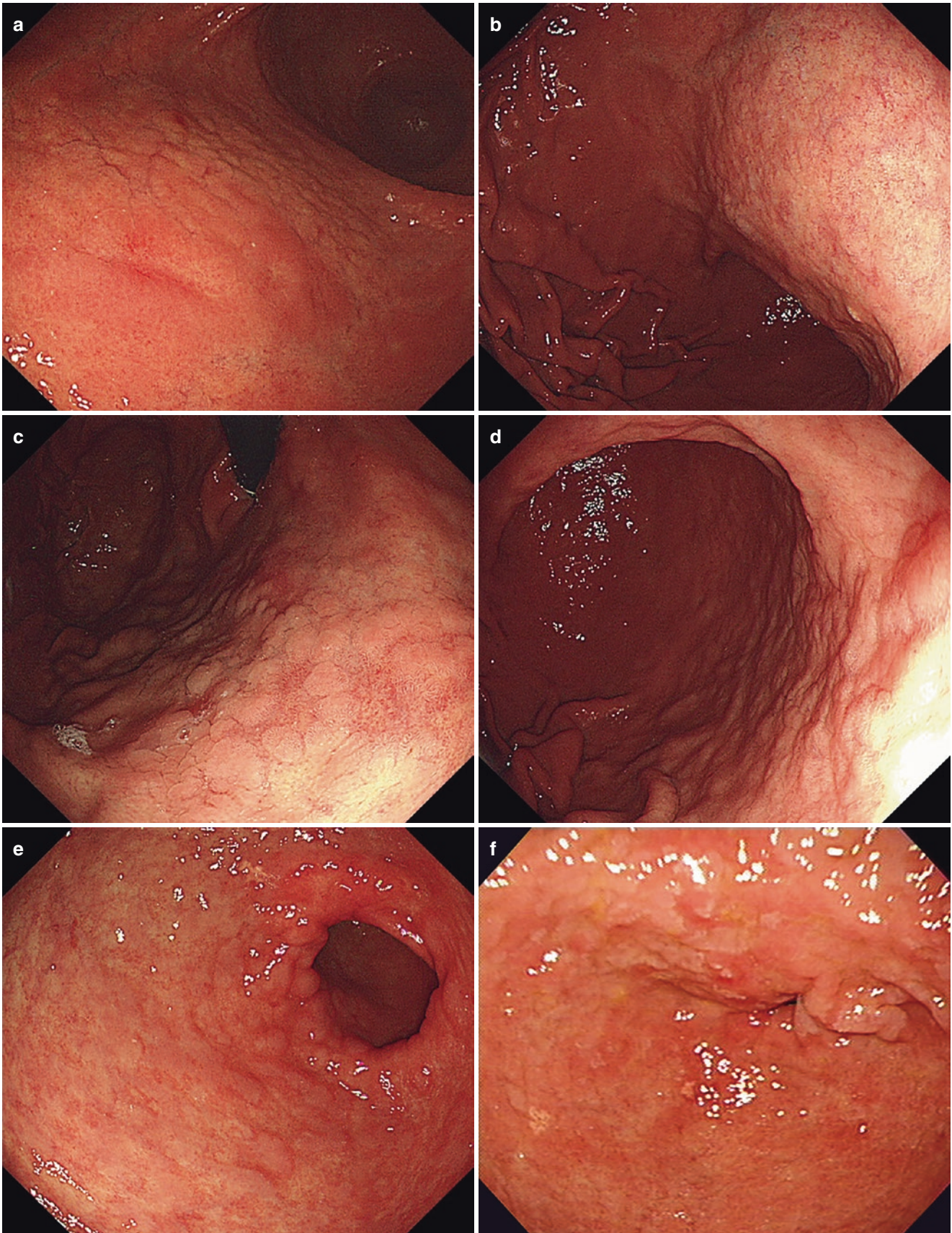


Fig. 8.7 Endoscopic findings of intestinal metaplasia. (a, b) Combined features of mucosal nodularity and atrophy on the body, (c, d) whitish granular plaques on the body, (e, f) whitish granular plaques on the

antrum, (g, h) endoscopic findings before and after indigo carmine staining, (i, j) distinct glandular structure of intestinal metaplasia after indigo carmine staining

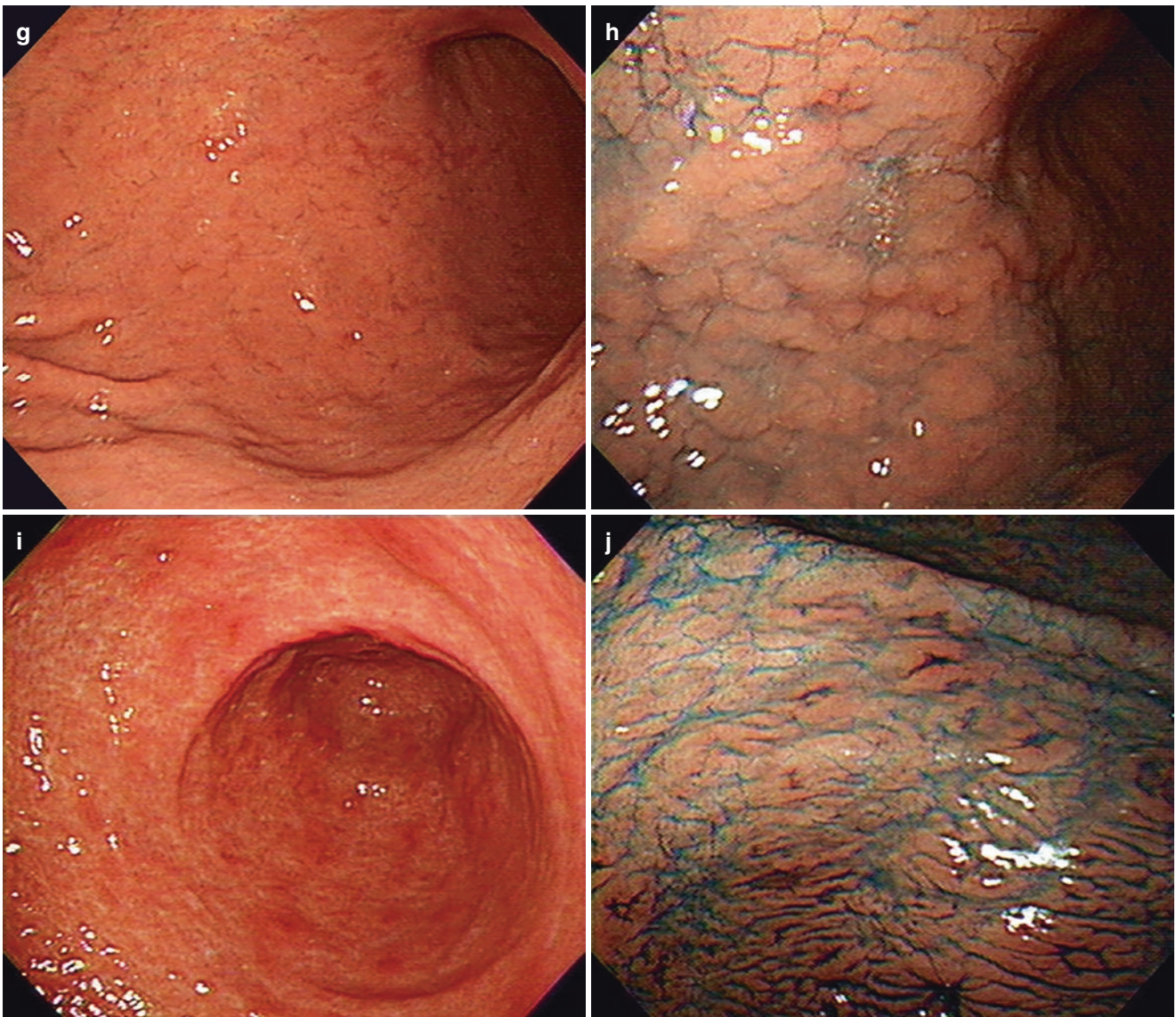


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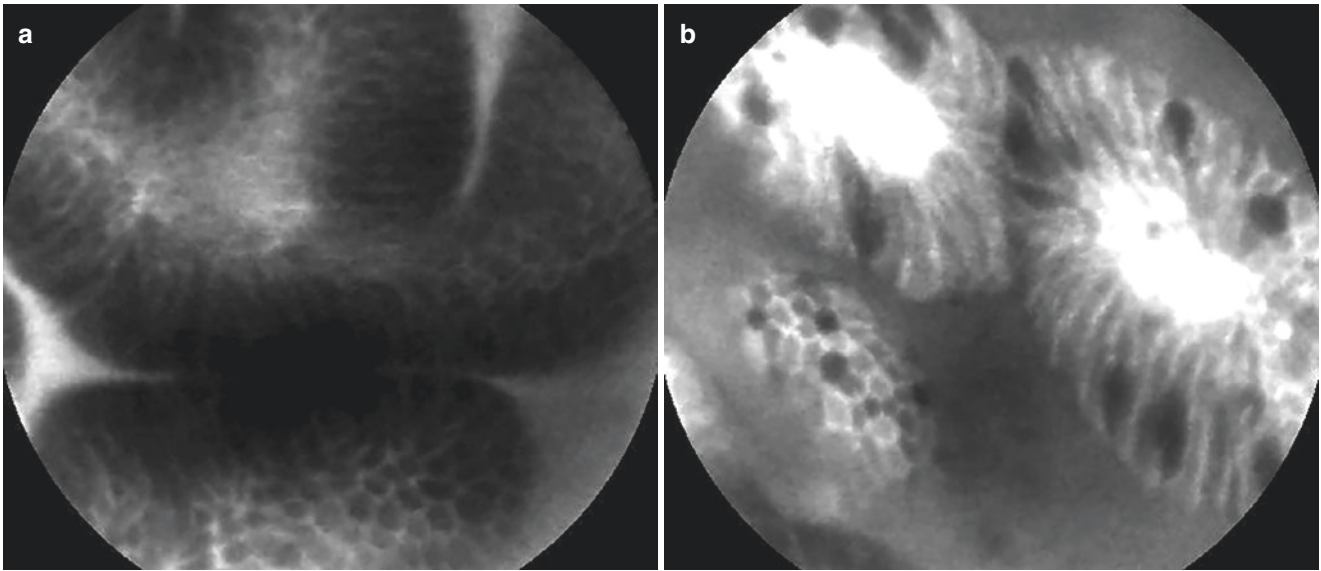


Fig. 8.8 Findings of intestinal metaplasia by confocal laser endomicroscopy. (a) Normal gastric mucosa showing a mosaic pattern, (b) villous columnar-lined epithelium and dark mucin in goblet cells in intestinal metaplasia

8.2.3 Other Gastritis

8.2.3.1 Lymphocytic Gastritis

Numerous tiny nodules were noted on the antrum to the body of the stomach (Fig. 8.9a–d). Indigo carmine staining can be

used to enhance visualization of small and regular nodularities on the lesions.

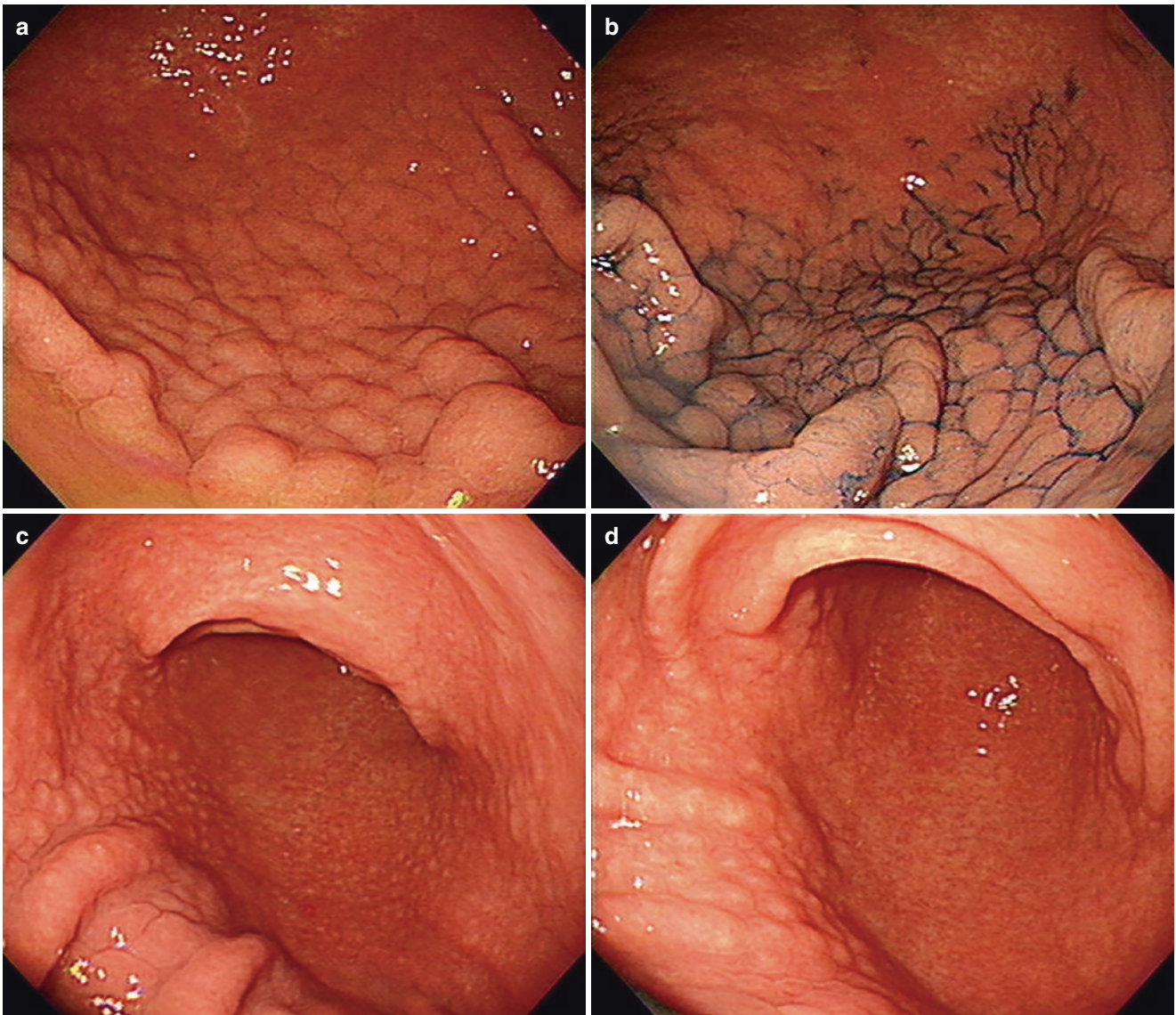


Fig. 8.9 Endoscopic finding of lymphocytic gastritis. (a) Large numbers of nodules covered with normal mucosa, (b) After indigo carmine spray, (c) Variable sizes of nodules in the stomach body, (d) Variable numbers of nodules in the stomach body

8.2.3.2 Eosinophilic Gastritis

Irregularly shaped and variable sized erythema were scattered from the antrum to the body (Fig. 8.10a, b). The biopsy showed ulceration with chronic superficial gastritis and many eosinophil infiltrations. There are no specific findings

for eosinophilic gastritis. Eosinophilic gastritis is sometime combined with ulceration of various degrees and shapes. Only pathology findings can confirm the diagnosis of eosinophilic gastritis.

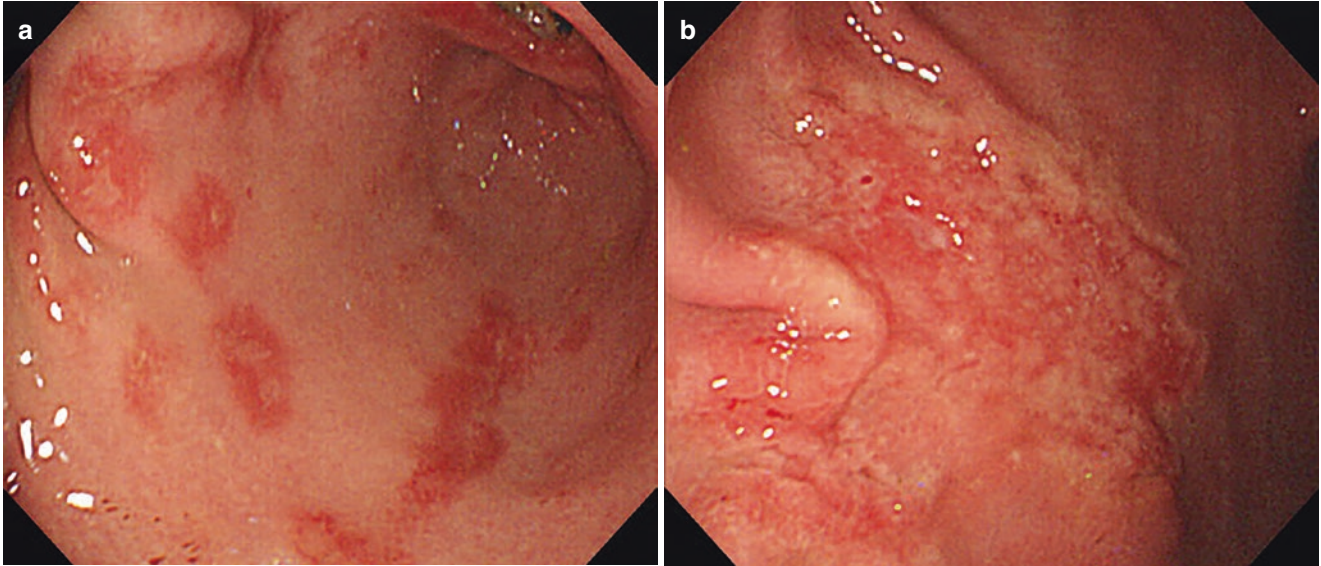


Fig. 8.10 Endoscopic finding of eosinophilic gastritis (a) Scattered erythema that looks like erosive gastritis, (b) Irregular lesion mixed with redness and discoloration

8.2.3.3 Syphilitic Gastritis

Diffuse hemorrhagic gastritis and ulceration were noted from the antrum to the body of the stomach (Fig. 8.11). This patient was referred to our hospital with suspicion of advanced gastric cancer. The biopsy showed plasma cell

infiltration to the lamina propria with an ill-defined granulomatous reaction. Syphilitic gastritis is difficult to diagnose without a history of syphilis, because endoscopic and microscopic findings are similar to those for gastric cancer or lymphoma [4].

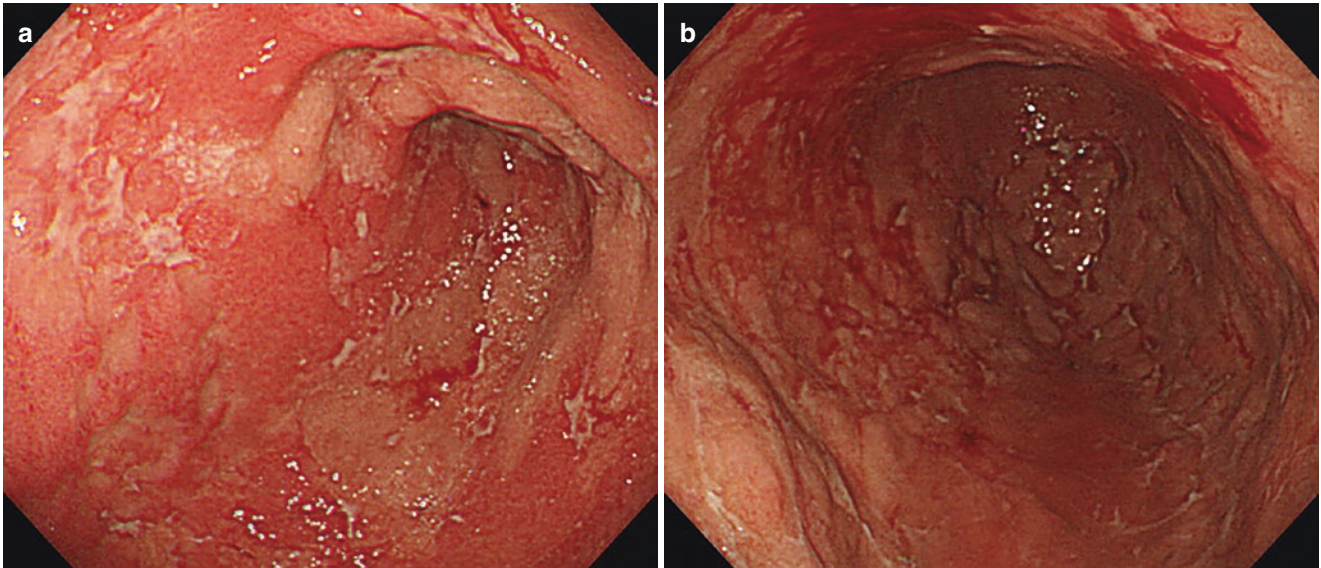


Fig. 8.11 Endoscopic finding of syphilitic gastritis. This patient was referred to our hospital with suspicion of advanced gastric cancer. (a, b) diffuse erythematous changes and ulceration are noted

8.2.3.4 Gastric Sarcoidosis

Endoscopic findings of gastric sarcoidosis vary from gastritis to benign- or malignant-appearing ulcers. Nodular mucosal irregularities are common, and flat erosions may occasionally be present (Fig. 8.12). H&E staining revealed

non-caseating granuloma in the mucosa. Endoscopic findings of gastric sarcoidosis include nodular changes, gastritis, thickened mucosa, greater or lesser curvature deformities, and benign- or malignant-appearing ulcers [5].

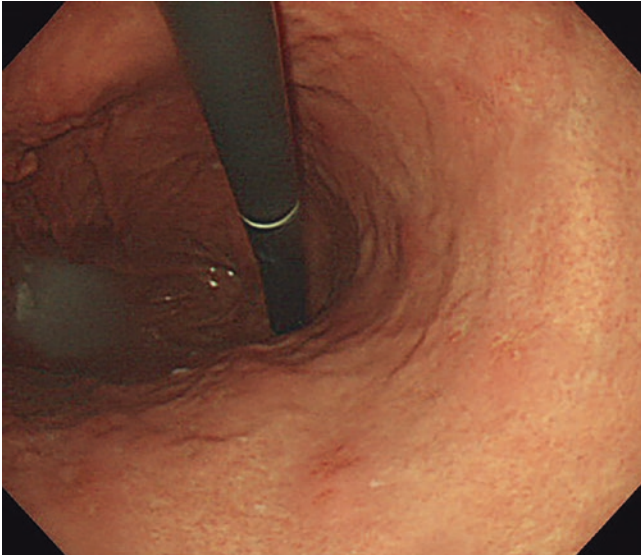


Fig. 8.12 Endoscopic findings of gastric sarcoidosis. Mucosal nodularities with erythema are shown in the gastric body, which was confirmed as sarcoidosis by histologic examination

8.2.3.5 Hypertrophic Gastritis (Giant Gastric Rugae)

Fold hypertrophy is usually detected on the greater curvature of the body. Thick mucosa coats the folds. A variety of proliferative, inflammatory, and infiltrative conditions are

associated with enlarged or giant mucosal folds in the stomach. The folds never disappear after full inspiration or the point when the patient can no longer tolerate air inflation (Fig. 8.13). This case was confirmed as infiltrative gastric cancer (signet ring carcinoma).

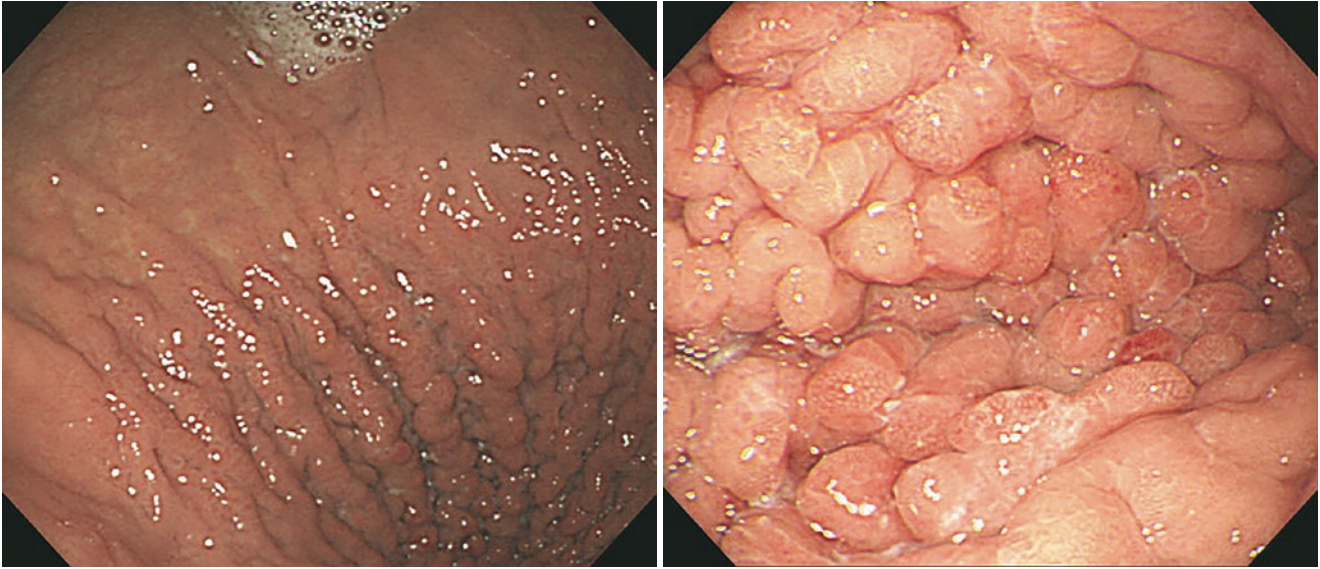


Fig. 8.13 Endoscopic findings of hypertrophic gastritis

8.2.3.6 Bile Reflux Gastritis

Bile reflux gastritis caused by an excessive reflux of duodenal contents into the stomach. The endoscopic findings

are erythema of the gastric mucosa, the presence of bile into the stomach, thickens of gastric folds, and erosions. (Fig. 8.14).

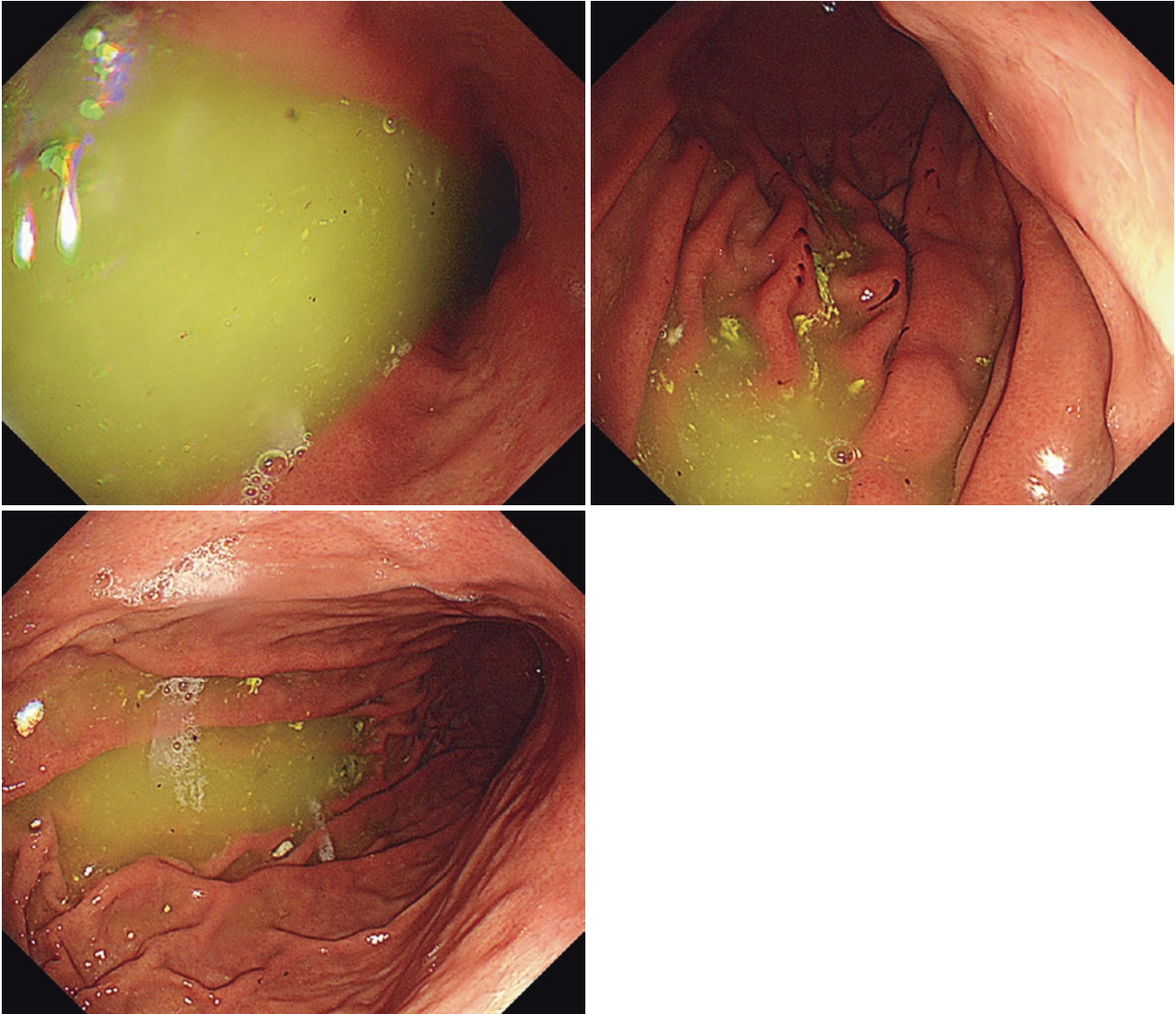


Fig. 8.14 Endoscopic findings of bile reflux gastritis

8.3 Gastric Ulcers

Gastric ulcers are defects or breaks in the gastric mucosa. Gastric ulcers penetrate through the muscularis mucosae in contrast to erosions. Gastric ulcers can vary in size from 5 mm to several centimeters and may lead to complications such as gastrointestinal (GI) bleeding, obstruction, penetration, and perforation. *H. pylori* infection, nonsteroidal anti-inflammatory drug use (NSAIDs), and aspirin use are the most common causes.

8.3.1 Benign Gastric Ulcer

8.3.1.1 Stages A1~S2

Gastric ulceration can be divided into active, healing, and scarring ulcerations according to the stage of healing

(Table 8.1). Deep, well-circumscribed ulcer is noted on the lesser curvature of the lower body (Fig. 8.15a). Black pigmented area was noted at the base of the ulcer, and there was no regenerating mucosa around the ulcer margin (Fig. 8.15b). This ulcer was classified as an A1 stage ulcer caused by NSAID use. The ulcer shown in Fig. 8.15c had a clean ulcer base and a regular shape and was therefore classified as an A2 stage ulcer

As the healing process continues, the margin of the ulcer becomes covered by hyperemic regenerating epithelium from the outer border; however, black pigmentation is still present at the ulcer base (Fig. 8.16a). This ulcer can be classified as an H1 stage ulcer. When the area of regenerating epithelium is larger than the ulcer base, the ulcer is classified as an H2 stage ulcer (Fig. 8.16b).

In the scarring stage, regenerating epithelium completely covers the ulcer base (Fig. 8.17a, b).

Table 8.1 Stages of gastric ulceration

| Stages | Endoscopic finding |
|----------------|--|
| Active stage | |
| A1 | Surrounding mucosa edematously swollen and no regenerating epithelium present |
| A2 | Decrease in surrounding edema, clear ulcer margin, and a slight amount of regenerating epithelium in the ulcer margin. Red halo in the marginal zone and a white slough circle in the ulcer margin are frequently seen. Usually, converging mucosal folds can be followed right up to the ulcer margin |
| Healing stage | |
| H1 | Thin white coating and regenerating epithelium extends into the ulcer base. The gradient between the ulcer margin and the ulcer floor becomes flat. An ulcer crater is still evident and the margin of the ulcer is sharp. The diameter of the mucosal defect is about one-half to two-thirds that of A1 |
| H2 | Defect is smaller than in H1, and the regenerating epithelium covers most of the ulcer floor. The area of white coating is about a quarter to one-third that of A1 |
| Scarring stage | |
| S1 | Regenerating epithelium completely covers the floor of the ulcer. White coating disappears. Initially, the regenerating region is markedly red. Upon close observation, many capillaries can be seen. This is called a “red scar” |
| S2 | In several months to a few years, the redness is reduced to the color of the surrounding mucosa. This is called a “white scar” |

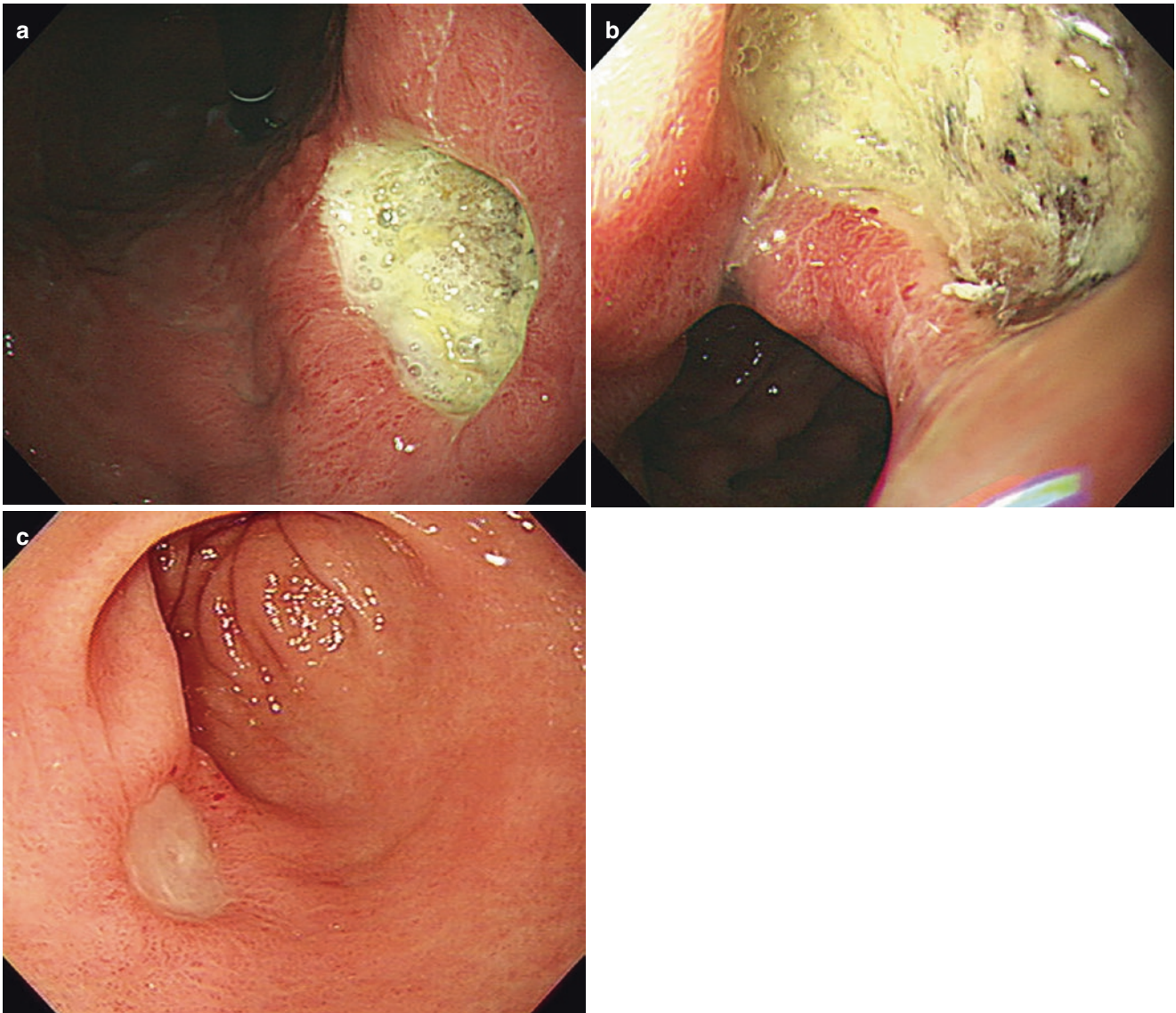


Fig. 8.15 Endoscopic features of active gastric ulcers. (a) well-circumscribed and deep penetrating active ulcer, (b) Black pigmented area shown on close observation, (c) clean ulcer base and a regular ulceration without exudate

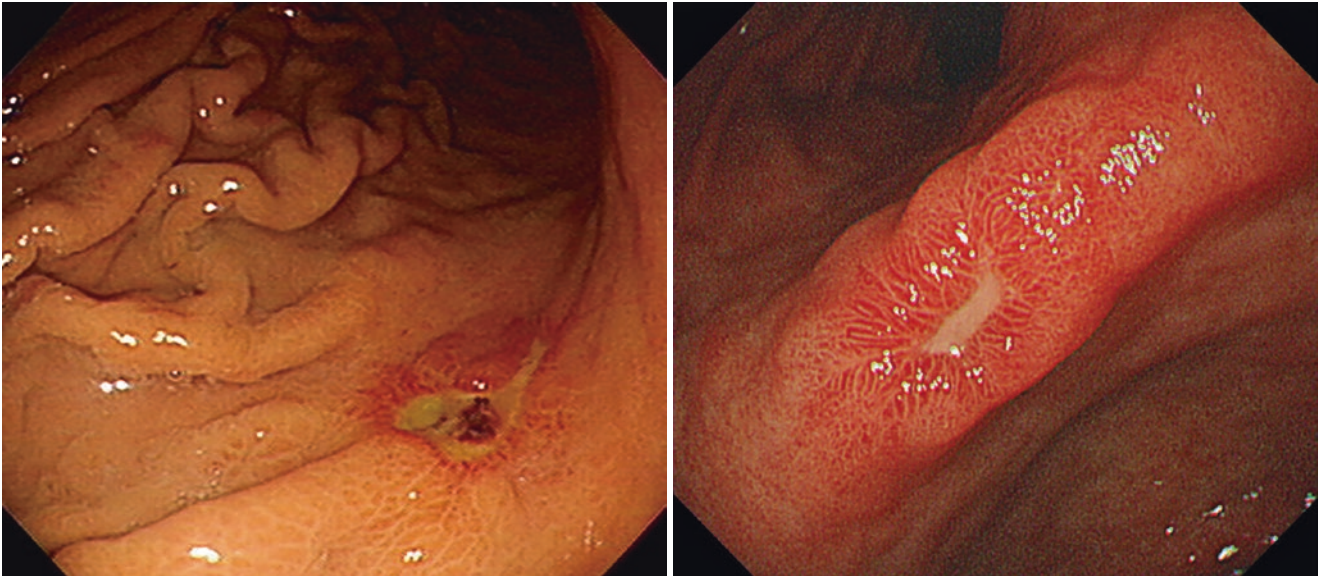


Fig. 8.16 Endoscopic features of healing gastric ulcers

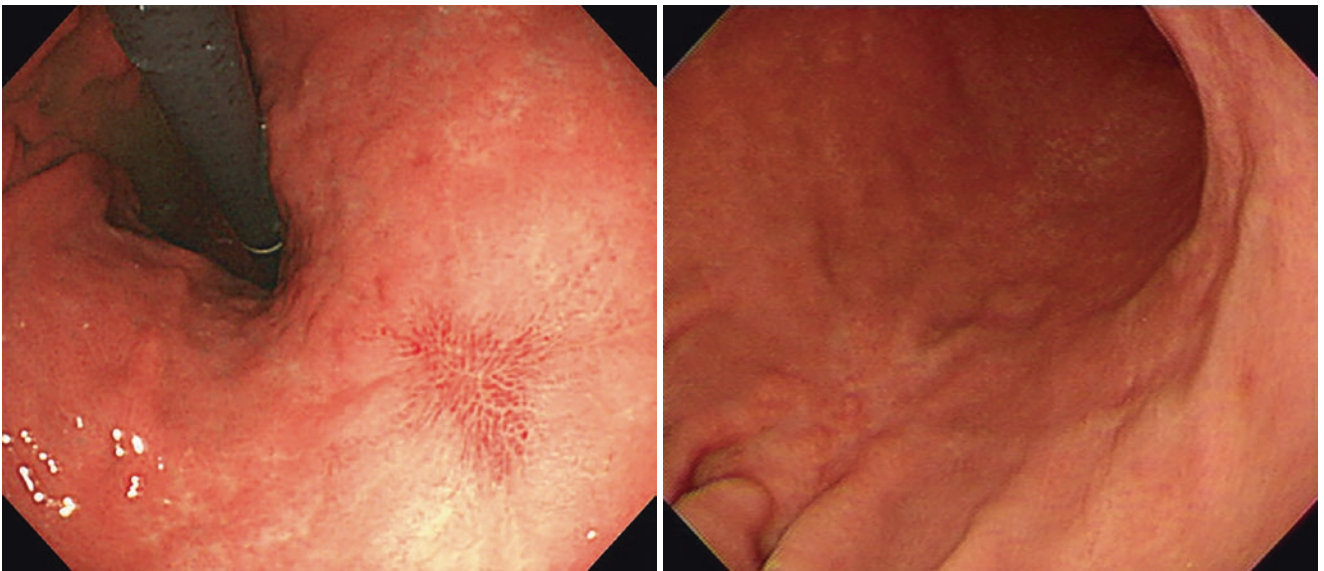


Fig. 8.17 Endoscopic features of scarring gastric ulcers

8.3.1.2 NSAID-Induced Ulceration

NSAID use can cause various types of ulceration, erosion, and gastropathy. Single (Fig. 8.18a) or multiple ulcers may be present (Fig. 8.18b, c). Differentiation from malignancy is

not difficult in most cases because most NSAID-induced ulcers have a regular shape with clear demarcations (Fig. 8.18c, d) and multiple ulcerations are present.

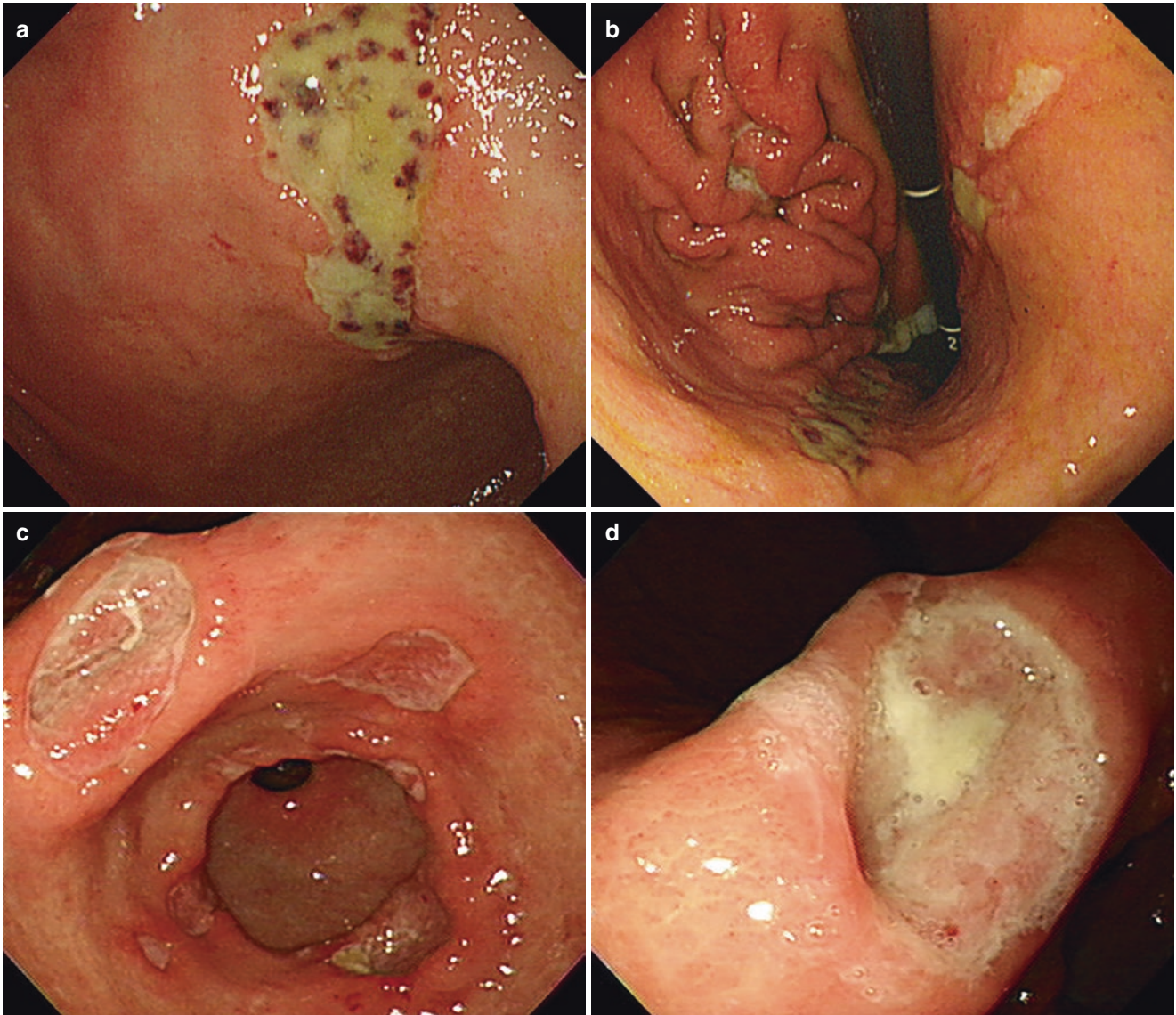


Fig. 8.18 Endoscopic features of gastric ulcers related to NSAID use. (a) longitudinal ulceration with multiple hematin on the base, (b) multiple and longitudinal ulcerations on the body of stomach, (c) multiple

round ulcerations on the antrum, (d) round and regular shaped ulceration covered by thin exudate on the angle

8.3.1.3 Other Ulcerations

Gastric Tuberculosis

Tuberculosis of the stomach is a rare disease. Tuberculosis may affect any part of the gastrointestinal tract, but gastric

involvement is very uncommon. Most reported cases were refractory ulcers that were sometimes misdiagnosed as submucosal tumors (Fig. 8.19).

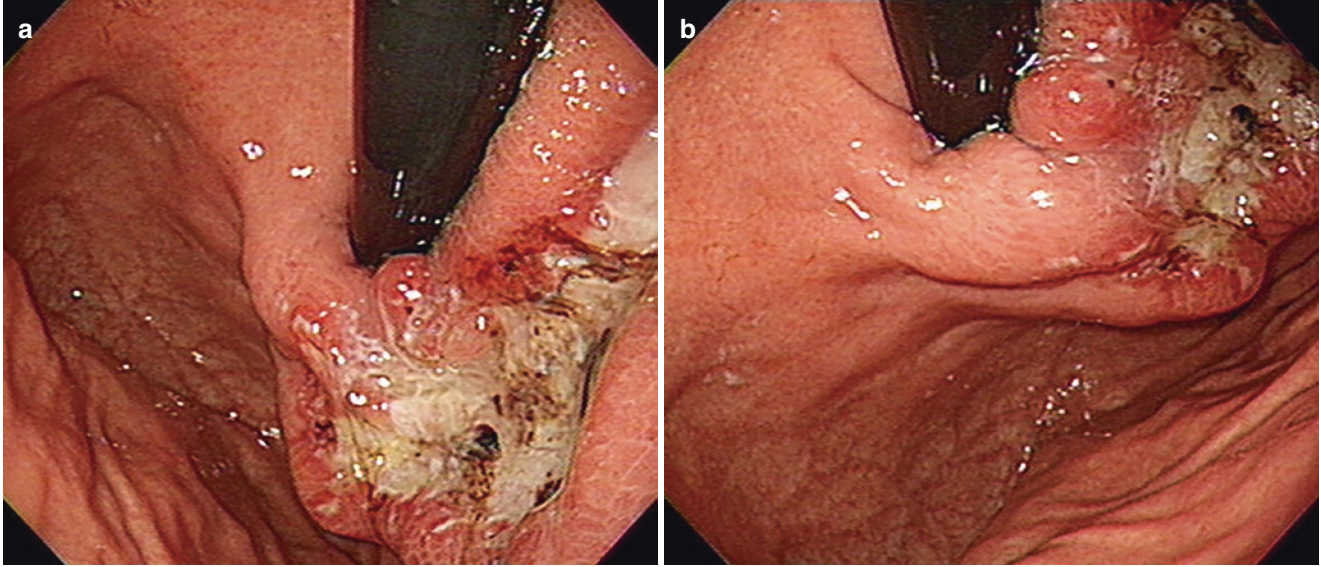


Fig. 8.19 Endoscopic finding of gastric tuberculosis. (a, b) Irregular shaped ulceration on the upper body

Amyloidosis

Shallow ulceration with thick and yellow exudate was noted on the fundus of the stomach (Fig. 8.20). Biopsy showed interstitial deposition of pinkish amorphous material with

apple green birefringence under a polarizing microscope; this finding is consistent with amyloidosis.

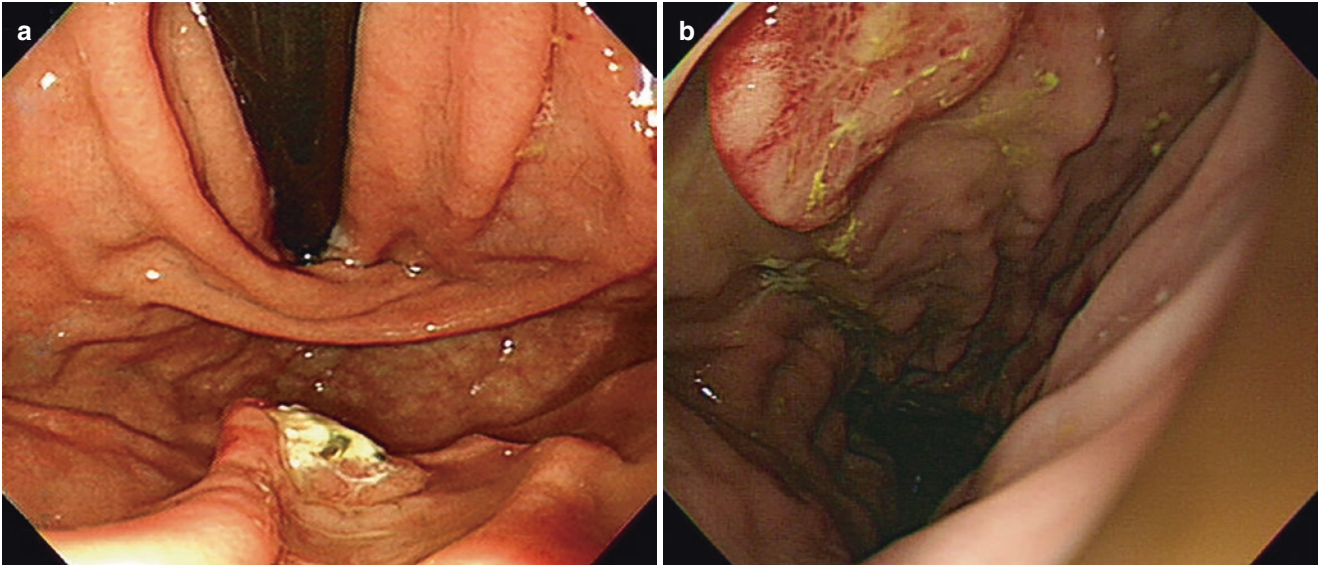


Fig. 8.20 Endoscopic finding of gastric amyloidosis. (a) Shallow ulceration with thick and yellow exudate on the fundus, (b) Flat elevated lesion with erythema and erosion on the surface in the same patient

CMV Gastritis-Induced Ulceration

The endoscopic appearance of cytomegalovirus (CMV) gastric infection is highly variable and includes normal mucosa, superficial or deep ulcers, mucosal erythema, and a discrete antral mass. Multiple ulcers with various shapes were noted

in the antrum (Fig. 8.21a) and body (Fig. 8.21b). Ulcers were small and punctate. Deep circular ulcer was noted at the pyloric channel (Fig. 8.21c). H&E staining revealed cytoplasmic and intranuclear inclusion bodies.

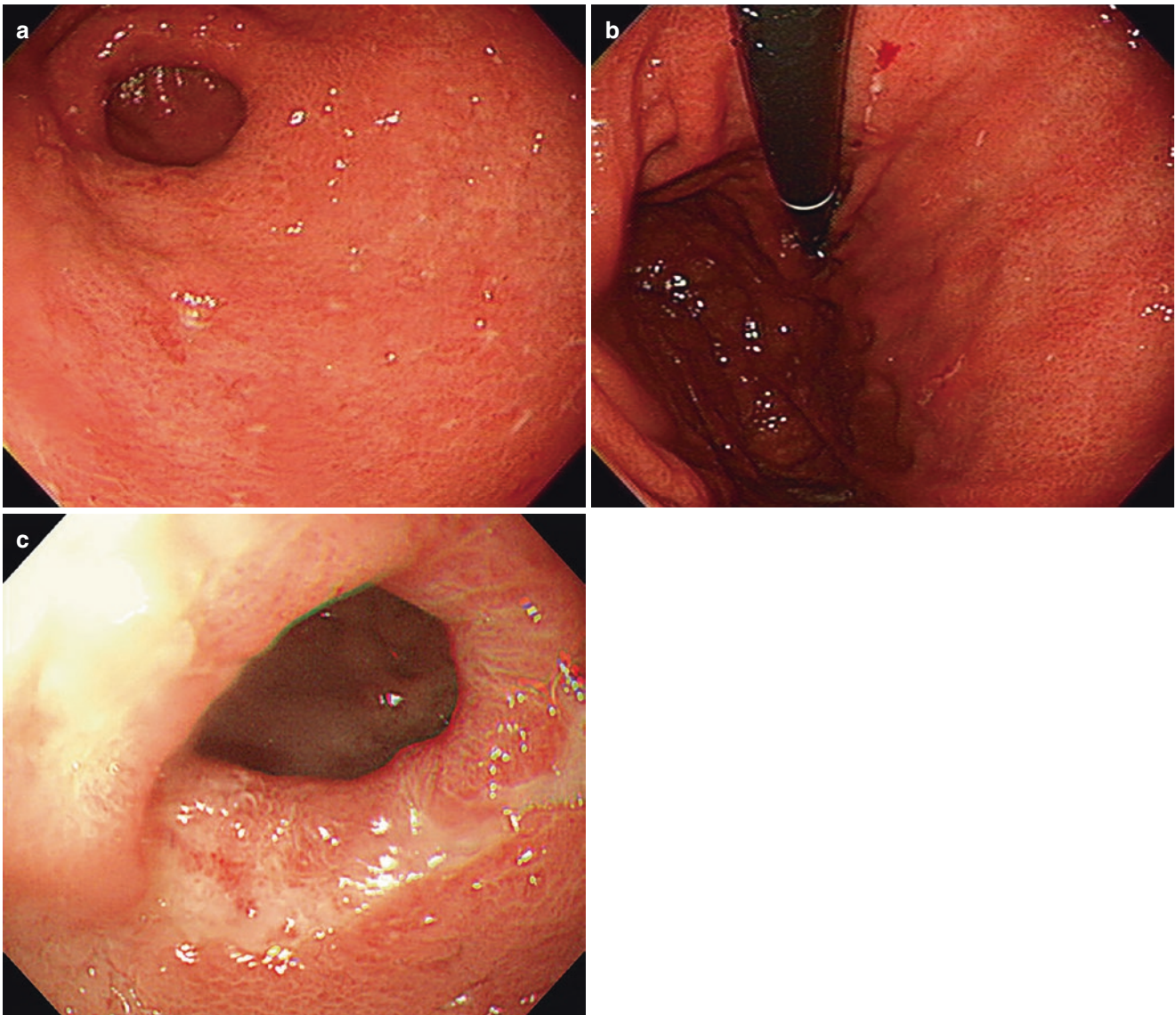


Fig. 8.21 Endoscopic findings of CMV-induced ulceration. (a) and (b) Features of CMV gastritis, (c) evolution of gastritis to ulceration

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