



Jae Young Jang

Contents

14.1	Vascular Abnormalities	283
14.1.1	Gastric Angiodysplasia.....	283
14.1.2	Portal Hypertensive Gastropathy.....	285
14.1.3	Dieulafoy's Lesion.....	287
14.2	Anatomic Abnormalities	288
14.2.1	Gastric Diverticulum.....	288
14.2.2	Mallory-Weiss Tear.....	289
14.3	Gastric Bezoar	290
14.4	Foreign Bodies	291
14.5	Corrosive Injury	293
	References	295

14.1 Vascular Abnormalities

14.1.1 Gastric Angiodysplasia

Vascular lesions have been described by many terms, such as angiodysplasia, arteriovenous malformations, telangiectasia, hemangioma, telangiopathy, and mucosal vascular abnormalities.

Endoscopically, angiodysplasias are bright cherry red, flat, or slightly elevated lesions with surrounding halo, varying in diameter from pinpoint to 10 mm (Fig. 14.1). They may be next to the mucosal layer. Larger, slightly elevated, or umbilicated lesions may have extensive submucosal or transmural anastomoses. An arteriovenous malformation may be localized to the fundus.

The differential diagnosis of small flat telangiectatic lesions includes clots or adherent blood, erosion, and an endoscopic suction artifact. Petechiae and submucosal hemorrhages are readily distinguished by their appearance.

“Watermelon stomach” or more extensive telangiectatic lesions, called gastric antral vascular ectasia (GAVE), is a term used for the typical endoscopic findings of red stripes, separated by normal mucosa, most commonly seen in the gastric antrum or proximal stomach, in patients with cirrhosis of the liver. GAVE also has been observed in a variety of autoimmune diseases and connective tissue diseases including atrophic gastritis, scleroderma, hypothyroidism, pernicious anemia, and primary biliary cirrhosis. Parallel longitudinal rugal folds are seen traversing the antrum and converging into the pylorus, each containing a visible convoluted column of vessels (Fig. 14.2). Unlike the abnormalities seen in hemorrhagic gastritis, the red linear streaks in the antrum blanch upon pressure with biopsy forceps. There is a peculiar tendency for the telangiectatic lesions to cluster along the crest of the longitudinal folds. Spontaneous bleeding and bleeding on contact is frequently noted. The lesion is thought to develop from intramural vascular shunts as a response to portal hypertension.

J. Y. Jang
Division of Gastroenterology, Department of Internal Medicine,
College of Medicine, Kyung Hee University, Seoul, South Korea
e-mail: jjjang@khu.ac.kr

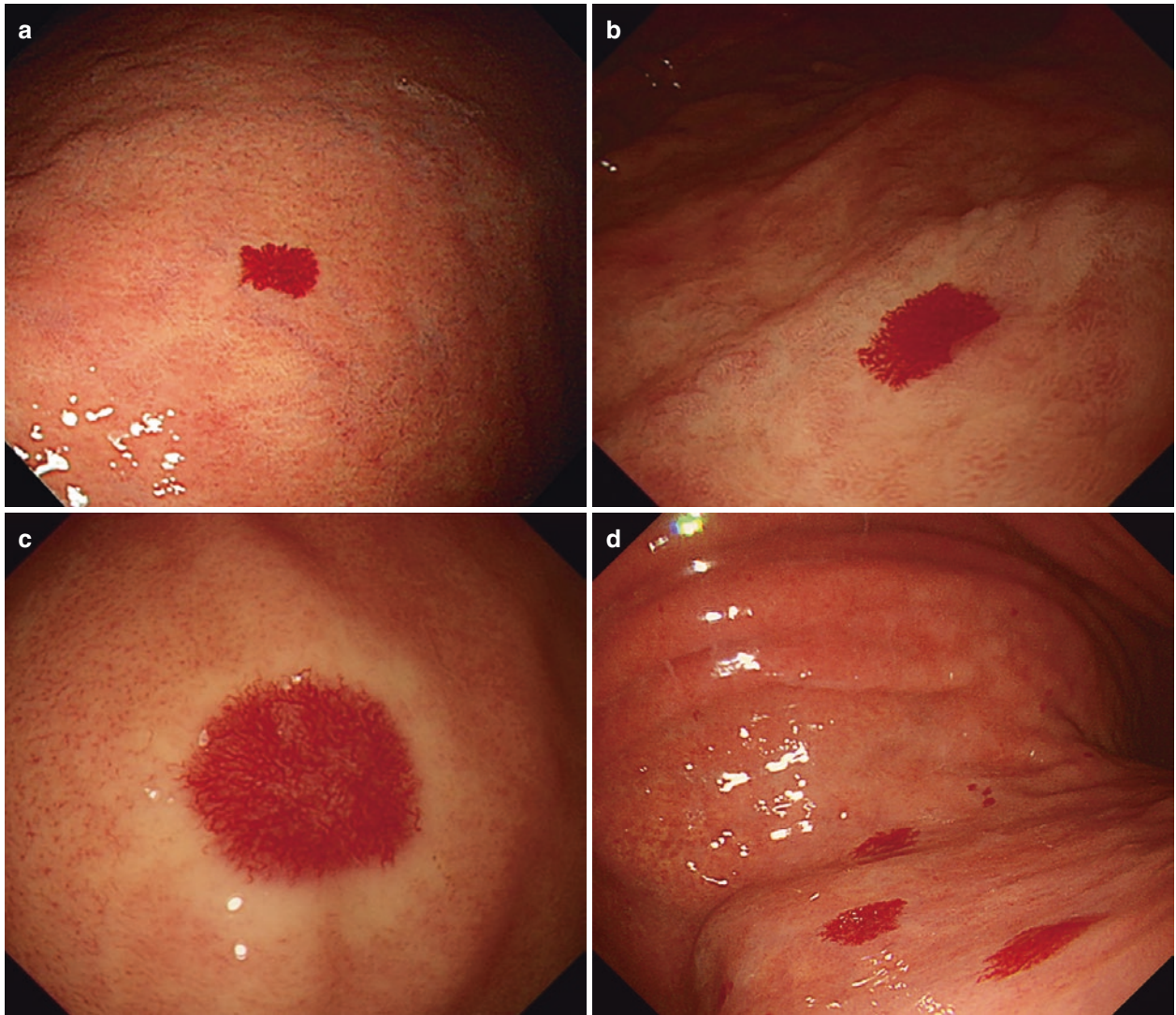


Fig. 14.1 Gastric angiodysplasia

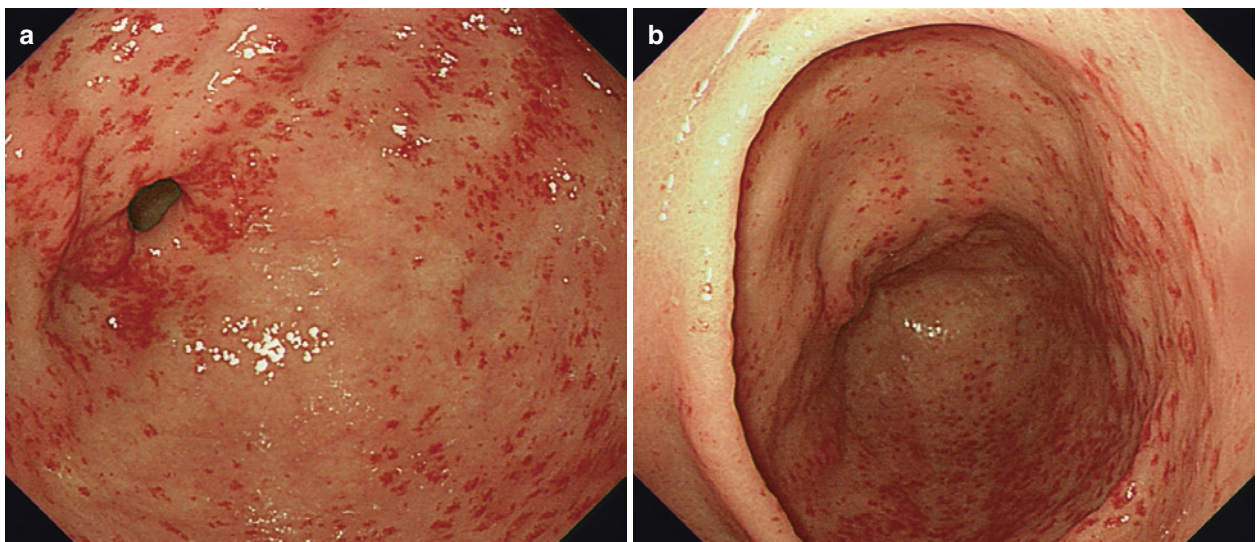


Fig. 14.2 Gastric antral vascular ectasia in cirrhotic patients

14.1.2 Portal Hypertensive Gastropathy

Portal hypertensive gastropathy is the term used to describe the endoscopic appearance of gastric mucosa, with a characteristic mosaic-like pattern with or without red spots, seen in patients with cirrhotic or noncirrhotic portal hypertension [1]. This type of lesion is thought to occur mainly in the body and the fundus of the stomach but is also seen rarely in the gastric antrum. The severity and the presence of portal hypertensive gastropathy do not have a linear correlation with the severity of portal hypertension. Endoscopically, mild portal hypertensive gastropathy appears as a fine reticular pattern, snake-skin appearance, or a mosaic pattern. Moderate types

are focally hemorrhagic and a few red spots in the body surface with a prominent mosaic pattern may be seen. The severe type shows severe diffuse hemorrhagic and red spots in the mucosal surface (Table 14.1, Fig. 14.3). Gastric varices may also be found in these patients.

Table 14.1 Classification of portal hypertensive gastropathy

Degree	Portal hypertensive gastropathy
Mild	Mosaic pattern without red spots
Moderate	Typical mosaic pattern and infrequent red spots
Severe	Numerous red spots

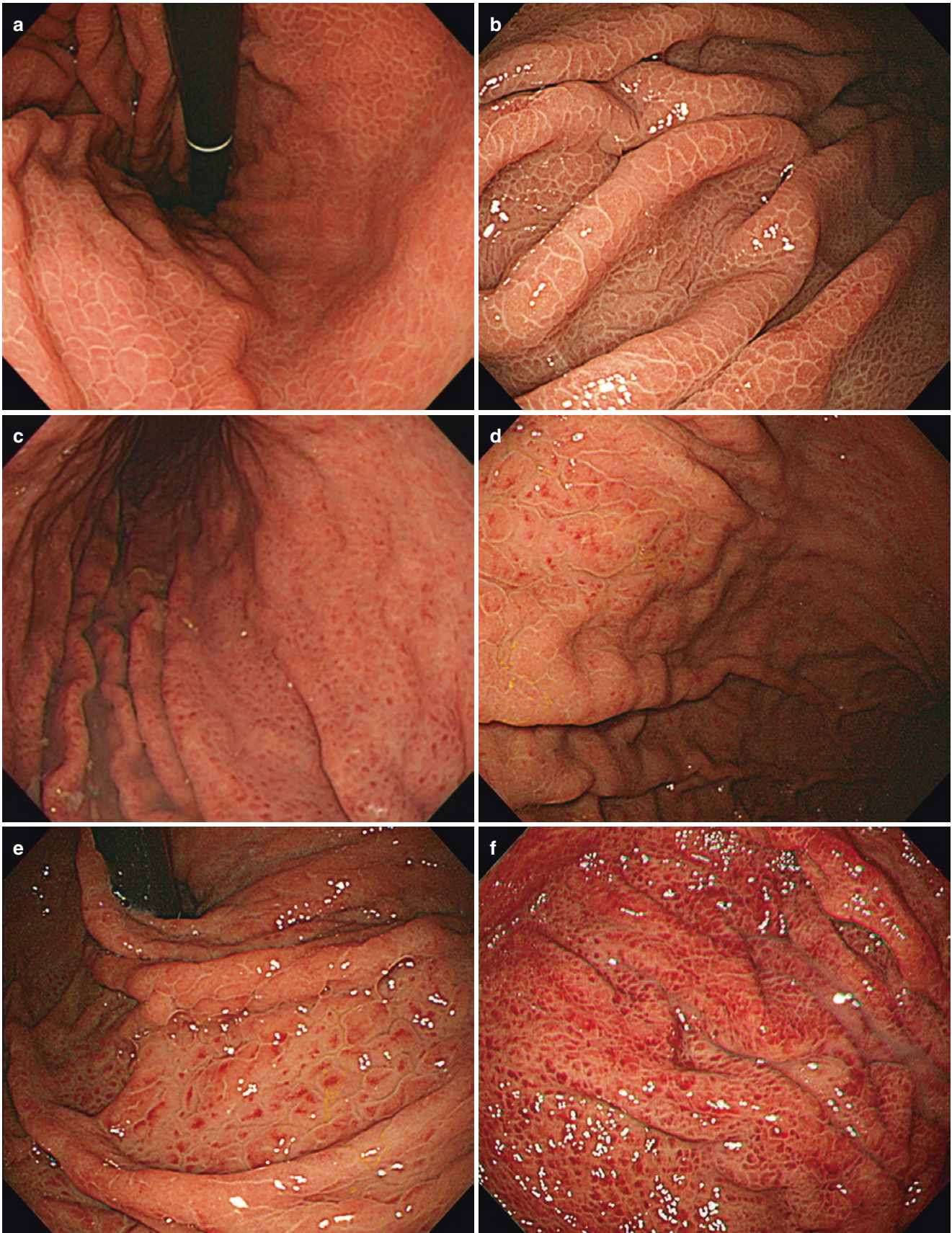


Fig. 14.3 Portal hypertensive gastropathy. (a, b) Mild type, (c, d) moderate type, (e, f) severe type

14.1.3 Dieulafoy's Lesion

Dieulafoy's lesion, also known as cirroid aneurysm and submucosal arterial malformation, consists of an abnormal, submucosal "caliber-persistent artery" that typically protrudes through a minute 2–5 mm mucosal defect. Dieulafoy's lesion accounts for 1–5.8% of cases of acute nonvariceal upper GI bleeding. Dieulafoy's lesion is considered to be a congenital lesion. However, there is a preponderance of men of advanced age. Comorbidities, particularly cardiovascular disease, hypertension, chronic renal failure, diabetes, and excessive use of alcohol have been described in almost 90% of the patients [2]. The endo-

scopic criteria for the diagnosis of Dieulafoy's lesion are the following: (1) active arterial spurting or micropulsatile streaming of blood from a minute (<3 mm) mucosal defect or through normal surrounding mucosa; (2) visualization of a protruding vessel, with or without active bleeding, within a minute mucosal defect or through normal surrounding mucosa; or (3) fresh, densely adherent clot with a narrow point of attachment to a minute mucosal defect or to normal appearing mucosa (Fig. 14.4). It may be difficult to diagnose this lesion endoscopically. Several endoscopies may be needed and the chance that surgery or angiographic embolization will be required to control bleeding is higher than with a bleeding gastric ulcer.

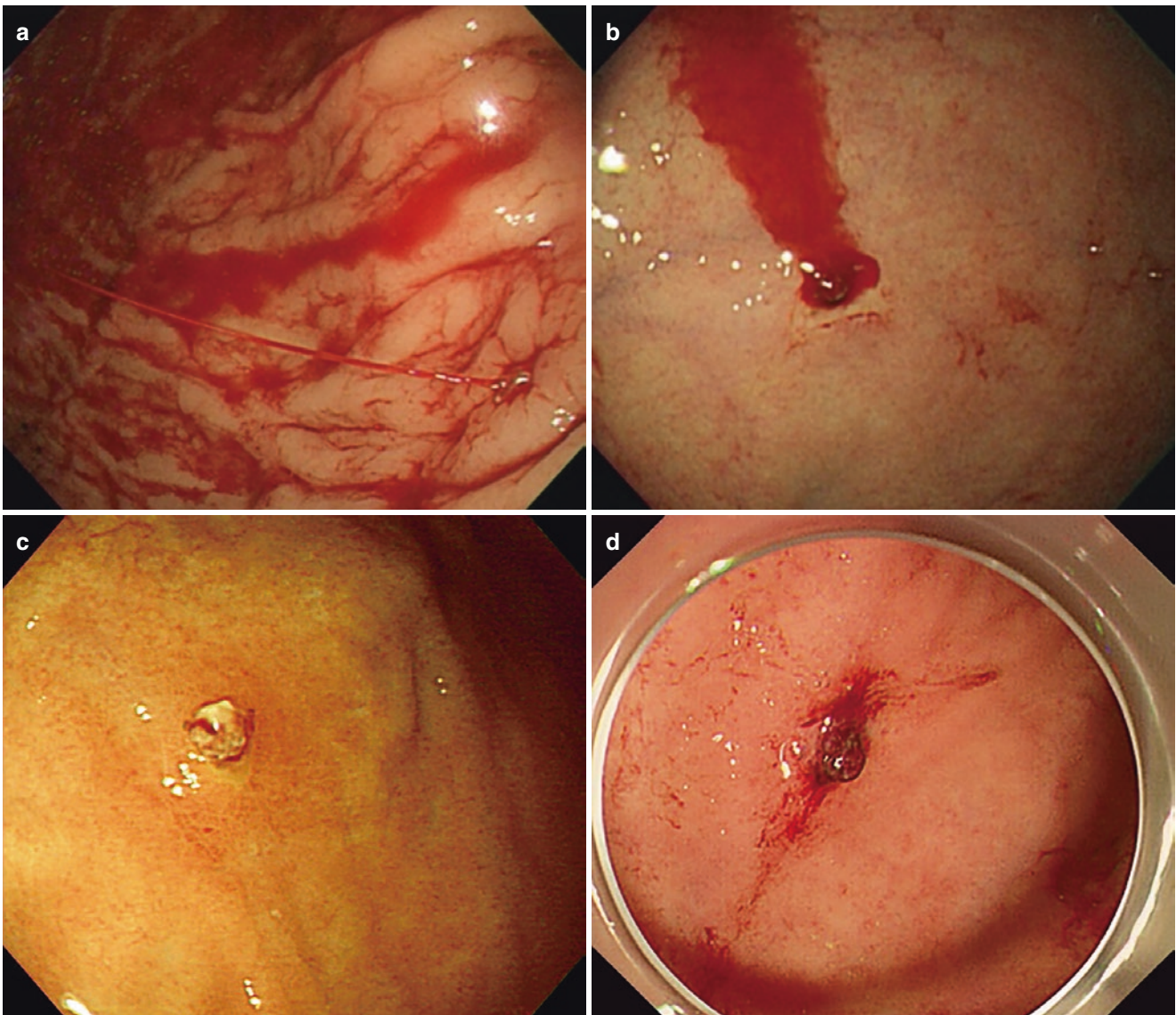


Fig. 14.4 Dieulafoy's lesions. (a) Spurting, (b) oozing, (c) exposed vessel, (d) adherent clot

14.2 Anatomic Abnormalities

14.2.1 Gastric Diverticulum

A gastric diverticulum is an outpouching of all of the wall layers. The subcardial region about 2 cm distal from the cardia and 3 cm dorsal to the lesser curvature is most

commonly affected. Less common sites include the prepyloric region and, rarely, the fundus or gastric greater curvature. The mouth of the diverticulum may be round, oval, or slit-like (Fig. 14.5). Radial folds frequently enter the diverticular outpouching. Potential complications are ulceration, hemorrhage, and food impaction, but these are rare.

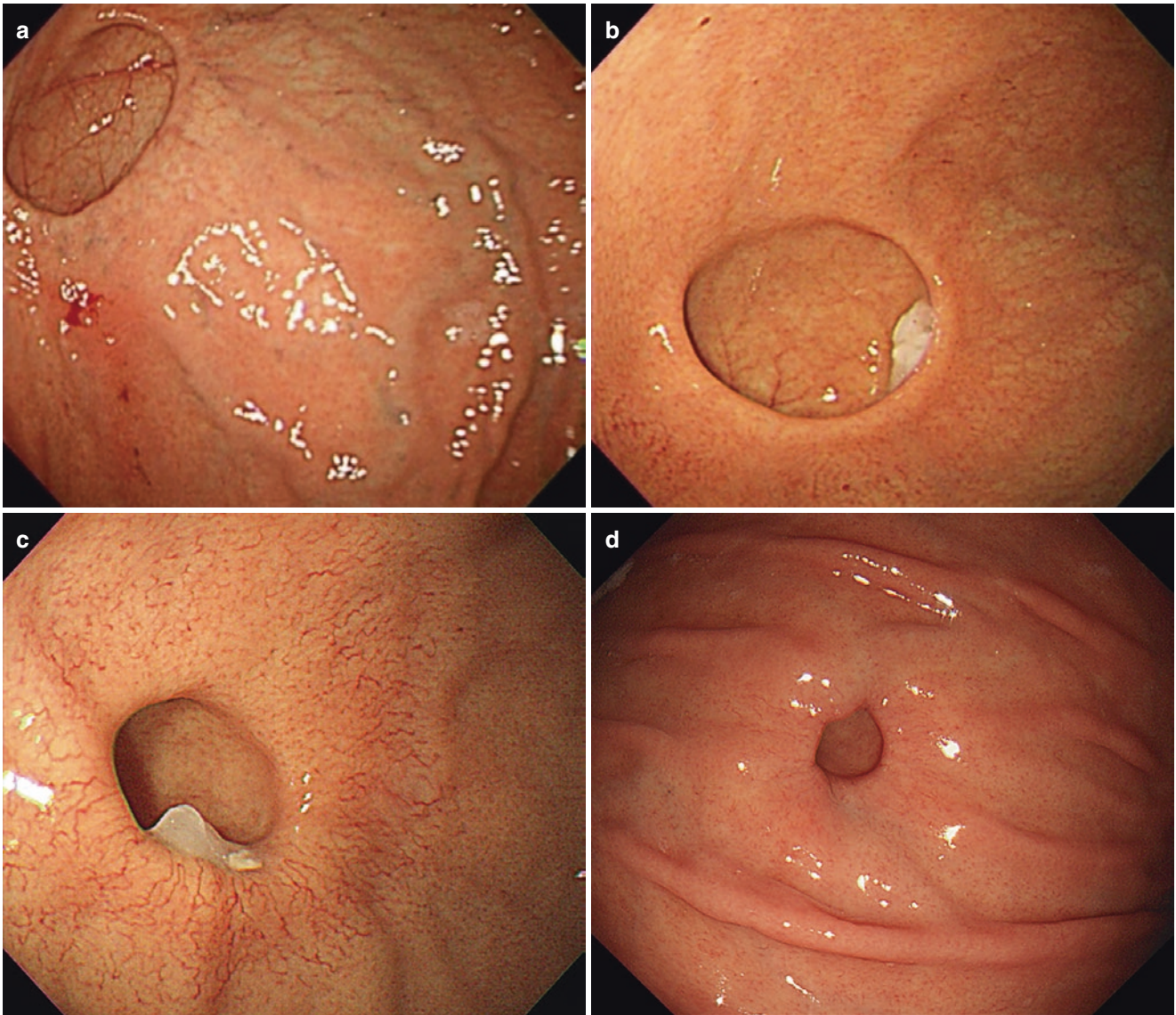


Fig. 14.5 Gastric diverticulum. (a–c) Fundus, (d) body

14.2.2 Mallory-Weiss Tear

Mechanical laceration in the area of the gastroesophageal junction, termed the Mallory-Weiss tear, is a common cause of upper gastrointestinal bleeding. A sudden increase in intra-abdominal pressure during violent retching of vomiting is considered the main cause. Alcoholics and patients on dialysis are most susceptible. The tears are usually linear, longitudinally oriented, and occasionally star-shaped. Tears are usually only

a few millimeters wide but sometimes up to several centimeters long. They often extend up into the squamous mucosa but more commonly involve the columnar cardiac-type mucosa. The tears may be superficial or extend into the submucosa. During the acute phase, oozing or even spurting bleeding may be seen, although most often the lesion is covered with an adherent clot (Fig. 14.6). A visible vessel may be seen in the lesion base. A tear may only be seen with the endoscope tip retroflexed.

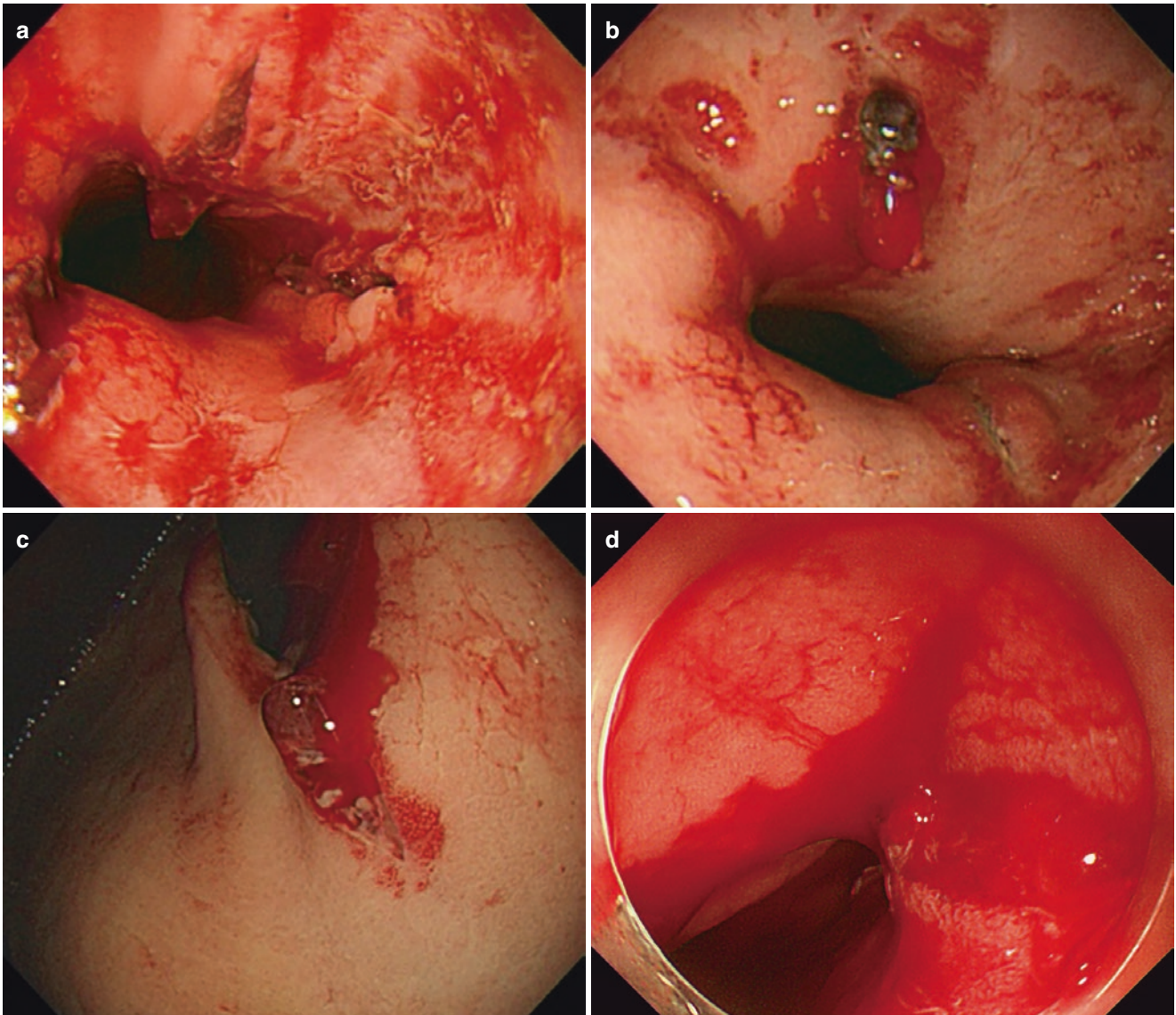


Fig. 14.6 Mallory-Weiss tear. Acutely oozing and adherent clots in the tear base

14.3 Gastric Bezoar

Bezoars are retained concretions of indigestible foreign material that accumulate and conglomerate in the gastrointestinal tract, most commonly in the stomach. It is a common complication after gastric surgery or altered gastrointestinal motility in which there is a loss of normal peristaltic activity, compromised pyloric function, or reduced gastric acidity. Bezoars can be classified into four types based on their origin and components: phytobezoars, trichobezoars, lactobezoars, and pharmacobezoars.

Phytobezoars are the most common type of bezoars. They are composed of food material nondigestible by humans. Usually, endoscopically, the phytobezoar will usually be visualized as a dark brown or green ball of amorphous material located in the fundus or antrum of the stomach (Fig. 14.7). Fragmentation may be attempted endoscopically using a biopsy forceps. An overtube can be inserted for repeated passage of the endoscope as pieces of the bezoar are removed. Endoscopic removal is followed by a combination of dietary restriction of fiber and instruction in proper mastication [3].



Fig. 14.7 Gastric phytobezoars

14.4 Foreign Bodies

Foreign bodies such as coins, balls, dental drills, denture, blades, mercury cells, press-through packs, toothbrushes, wires, and acupuncture needles may become lodged in the

stomach (Fig. 14.8). If the object is sharp, a plastic over-tube is recommended to protect the cardia and the esophagus.

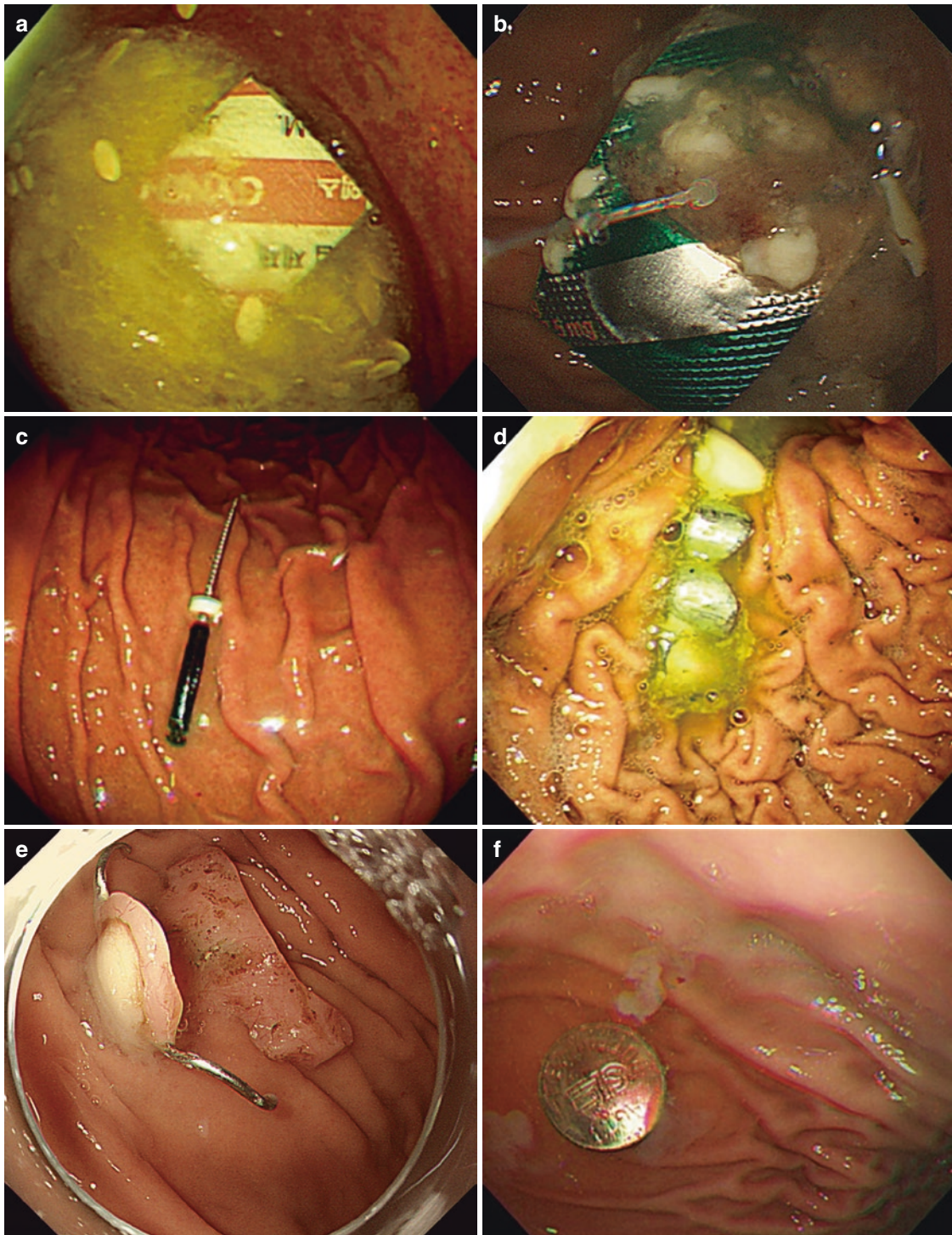


Fig. 14.8 Various gastric foreign bodies. (a, b) Press-through pack, (c) dental drill, (d, e) denture, (f) mercury cell, (g) acupuncture, (h) tooth brush, (i) thin wire, (j) razor, (k) migrated pyloric stent

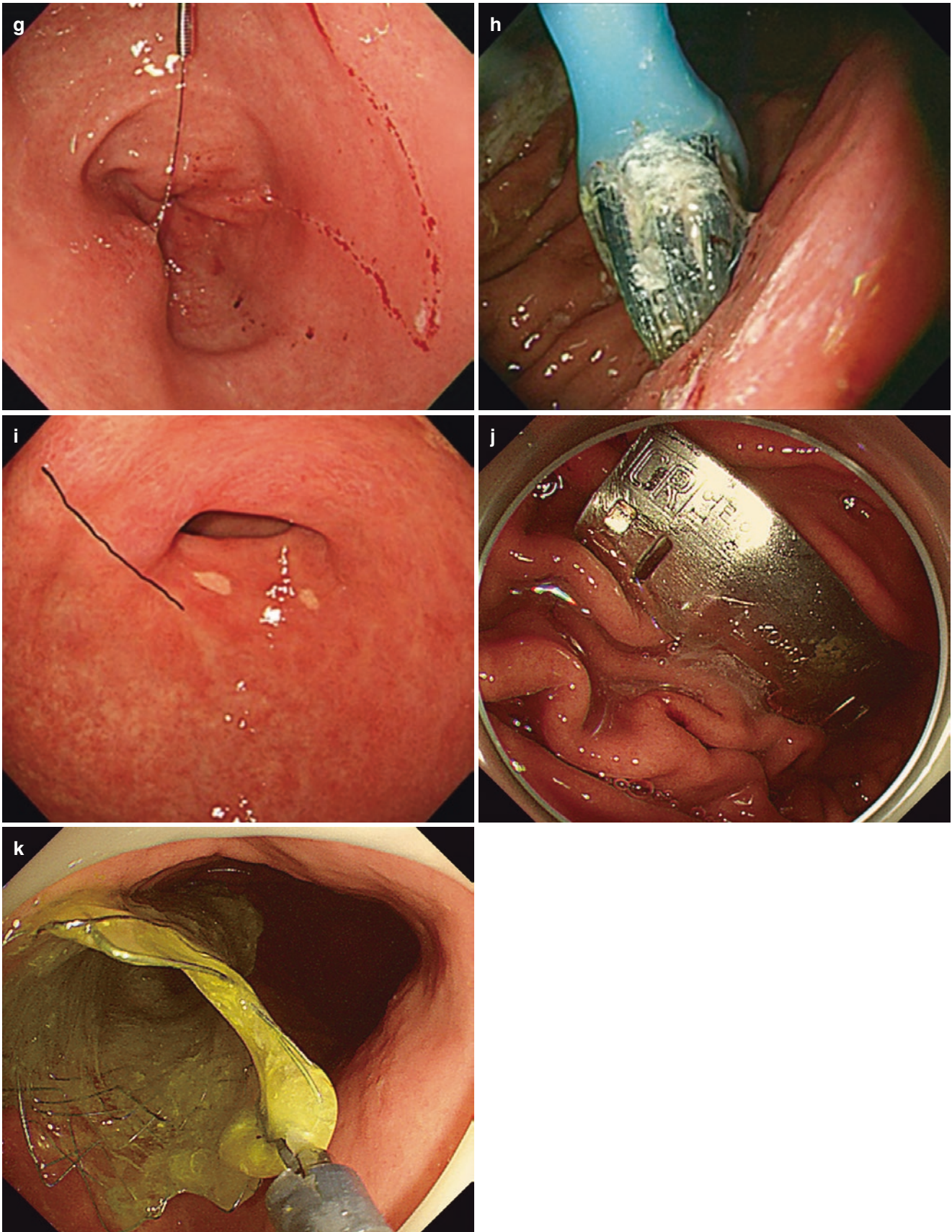


Fig. 14.8 (continued)

14.5 Corrosive Injury

Corrosive ingestion is a rare but potentially fatal event. Accidental ingestion by children accounts for 80% of cases worldwide, whereas in adults, most ingestions are intentional resulting from underlying psychiatric illness [4]. The corrosive agents involved include a wide range of chemicals that cause damage to tissue on contact. Early (<48 h) endoscopy assesses the extent and severity of caustic injuries from the luminal perspective. Several endoscopic classifications of upper digestive corrosive injuries have been proposed. The Zargar classifica-

tion has gained wide acceptance: grade 0 is normal; grade 1 is edema and hyperemia of the mucosa; grade 2a is superficial localized ulcerations, friability, and blisters; grade 2b is circumferential and deep ulcerations; grade 3a is multiple and deep ulcerations and small scattered areas of necrosis; grade 3b is extensive necrosis; and grade 4 is perforation (Fig. 14.9). Endoscopic grading predicts systemic complications and long-term survival. Initial endoscopy is reliable in predicting future stricture formation, with low-grade injuries (grades 1–2a) rarely causing strictures, but stricture can occur in as many as 80% of patients with severe burns (grade 3b).



Fig. 14.9 Endoscopic classification of caustic injuries. (a) grade 1, (b) grade 2a, (c) grade 2b, (d) grade 3a, (e) grade 3b

Acknowledgements Jae-Jun Shim, Division of Gastroenterology, Department of Internal Medicine, College of Medicine, Kyung Hee University, Seoul, Korea.

2. Lee YT, Walmsley RS, Leong RW, Sung JJ. Dieulafoy's lesion. *Gastrointest Endosc.* 2003;58:236–43.
3. Andrus CH, Ponsky JL. Bezoars: classification, pathophysiology, and treatment. *Am J Gastroenterol.* 1988;83:476–8.
4. Chirica M, Bonavina L, Kelly MD, Sarfati E, Cattani P. Caustic ingestion. *Lancet.* 2017;389:2041–52.

References

1. Thuluvath PJ, Yoo HY. Portal hypertensive gastropathy. *Am J Gastroenterol.* 2002;97:2973–8.