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A duodenal ulcer is disruption of the mucosal integrity of the duodenum leading to a submucosal exposure. The endoscopic findings of duodenal ulcers can be classified into active, healing, and scarring stage. Complications of duodenal ulcer include bleeding, perforation, and stricture. The most common complication is a bleeding ulcer which can be divided into type I, type II, and type III according to the Forrest classification. The second most common complication related to duodenal ulcer is perforation. A fixed obstruction can occur because of the scar and followed by distortion of structures. Duodenitis is inflammation of the duodenum. Endoscopic findings are erosions, erythema, and/or edema of duodenal mucosa.

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## 15.1 Duodenal Ulcers

### 15.1.1 Definition and Clinical Characteristics

A duodenal ulcer is defined as >5 mm disruption of the mucosal integrity of the duodenum leading to a submucosal exposure with perceptible depth at endoscopy. In contrast, erosions are mucosal breaks without perceptible depth [1, 2]. *Helicobacter pylori* (*H. pylori*) infection and nonsteroidal anti-inflammatory drugs (NSAIDs)-related injuries are the major causes of duodenal ulcers. Duodenal ulcers are usually located at

duodenal bulb. Malignant duodenal ulcers are extremely rare. Endoscopy is the most useful diagnostic method enabling direct mucosal visualization and tissue biopsy.

### 15.1.2 Endoscopic Stage

The endoscopic findings of duodenal ulcers can be classified into active (A1, A2), healing (H1, H2), and scarring (S1, S2) stage as described below (Sakita-Miwa classification) [1].

### 15.1.2.1 Active Stage

Stage A1: An ulcer containing thick mucus coating on the ulcer floor with edematous surrounding mucosa around the margin (Fig. 15.1a).

Stage A2: The surrounding edema has decreased, and the ulcer margin is clear. Usually, converging mucosal folds can be followed right up to the ulcer margin (Fig. 15.1b).

### 15.1.2.2 Healing Stage

Stage H1: The white coating is becoming thin, and the regenerating epithelium is extending into the ulcer base. The gradient between the ulcer margin and the ulcer floor is becoming

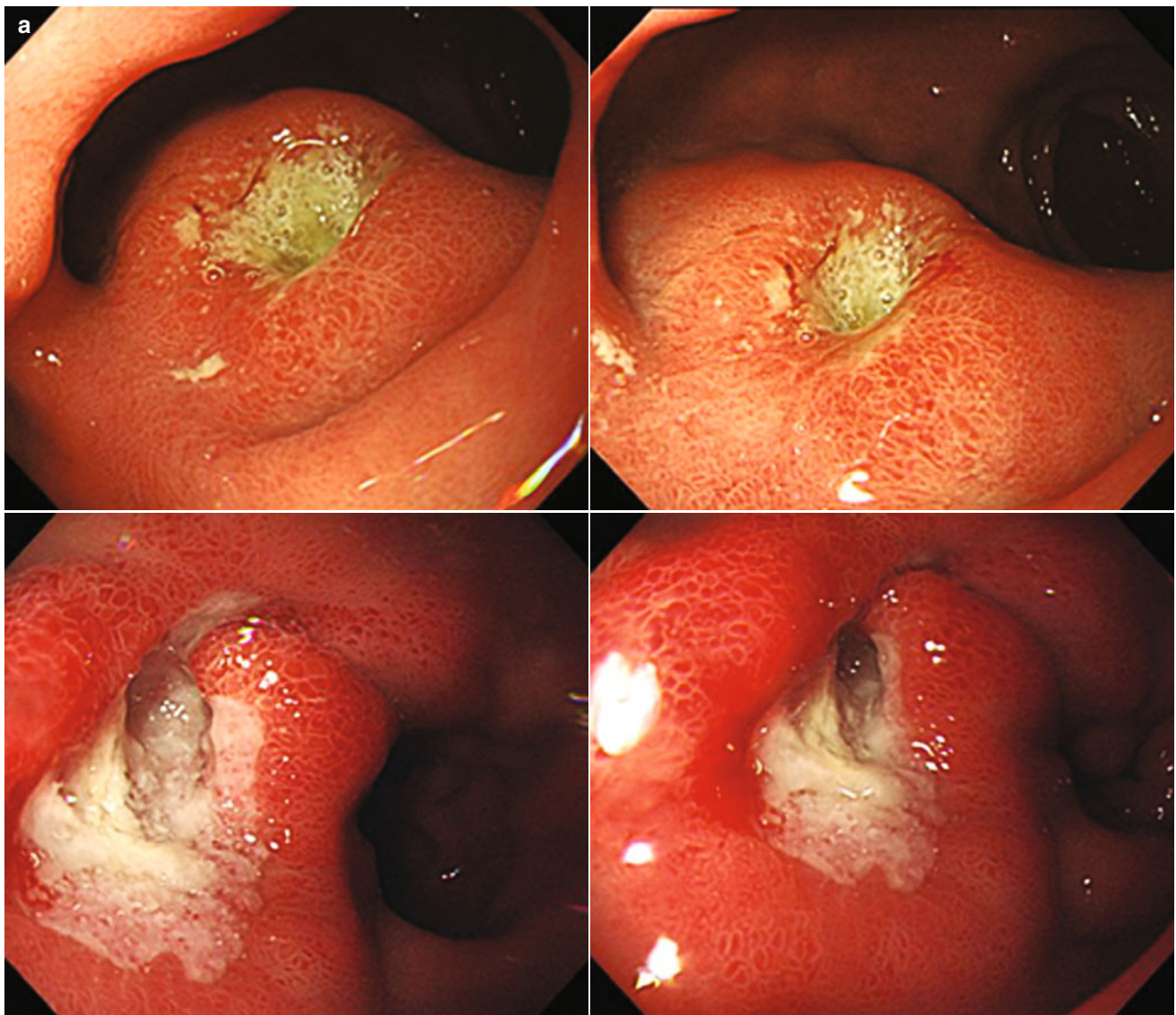
flat. The ulcer crater is still evident, and the margin of the ulcer is sharp (Fig. 15.1c).

Stage H2: The defect is smaller than in H1, and the regenerating epithelium covers most of the ulcer floor (Fig. 15.1d).

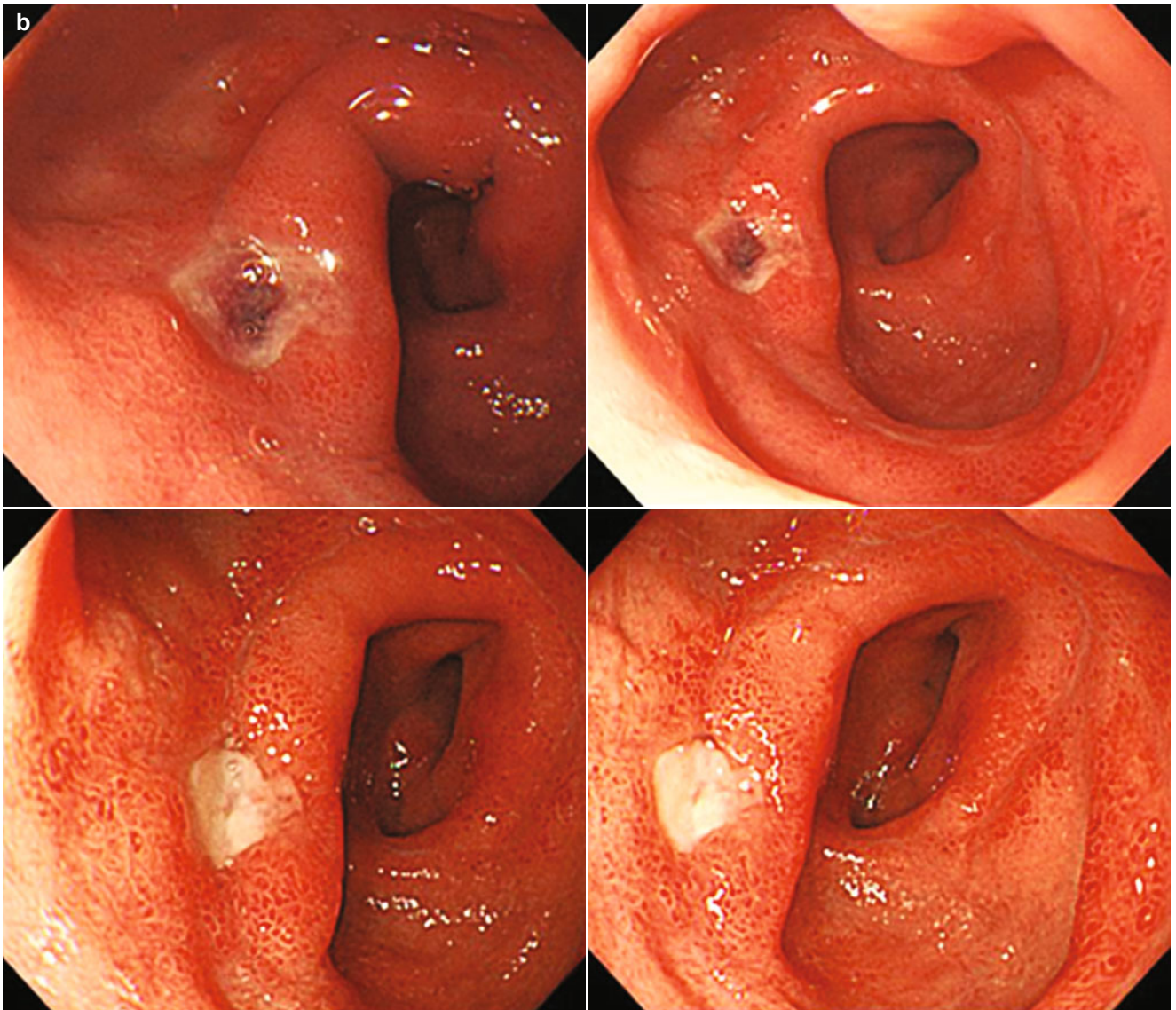
### 15.1.2.3 Scarring Stage

Stage S1: The regenerating epithelium completely covers the floor of ulcer. The white coating has disappeared. Initially, the regenerating region is markedly red. This is called “red scar” (Fig. 15.1e).

Stage S2: The redness is reduced to the color of the surrounding mucosa. This is called “white scar” (Fig. 15.1f).

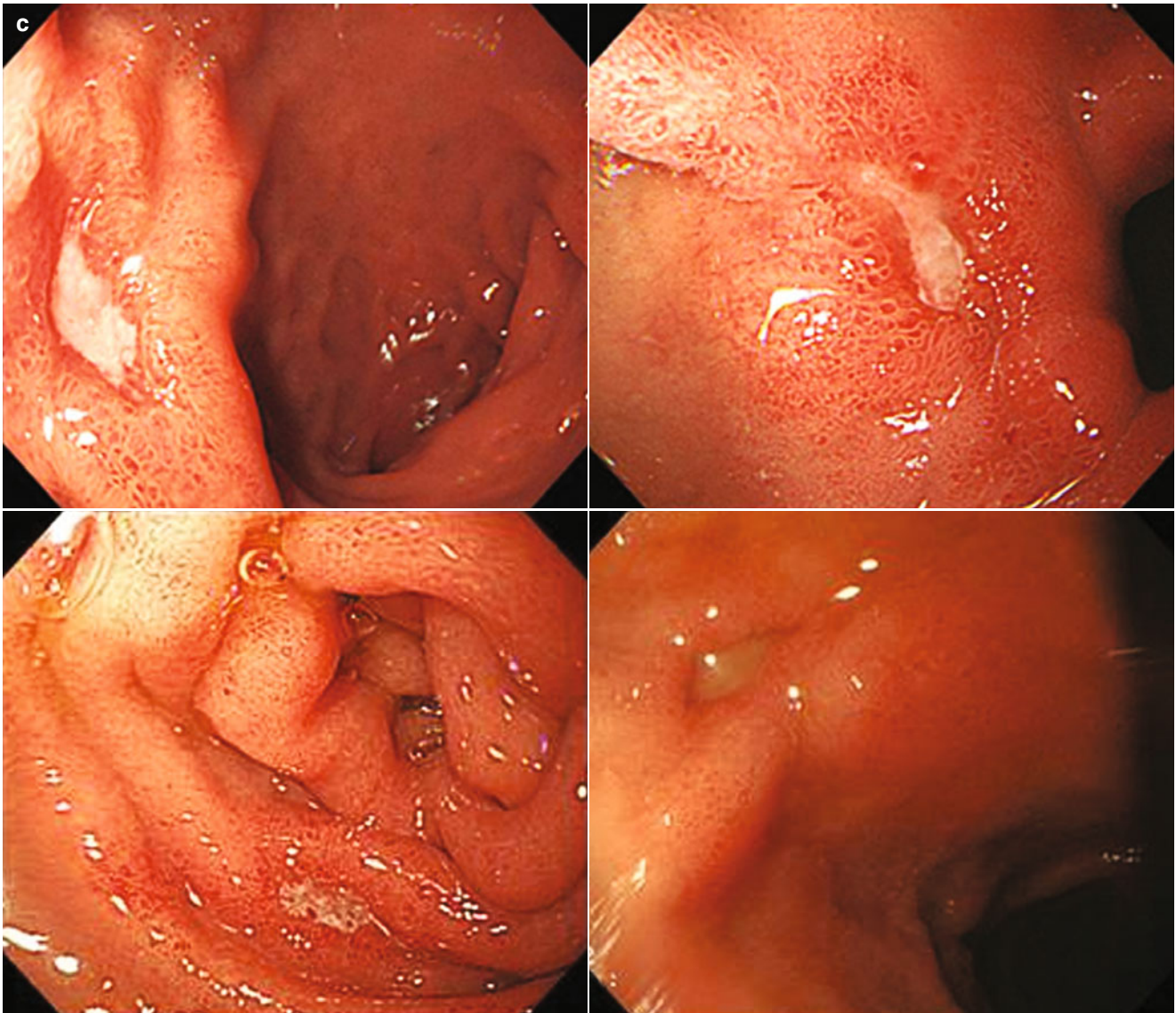


**Fig. 15.1** Endoscopic stages of duodenal ulcers. (a) Stage A1. (b) Stage A2. (c) Stage H1. (d) Stage H2. (e) Stage S1. (f) Stage S2

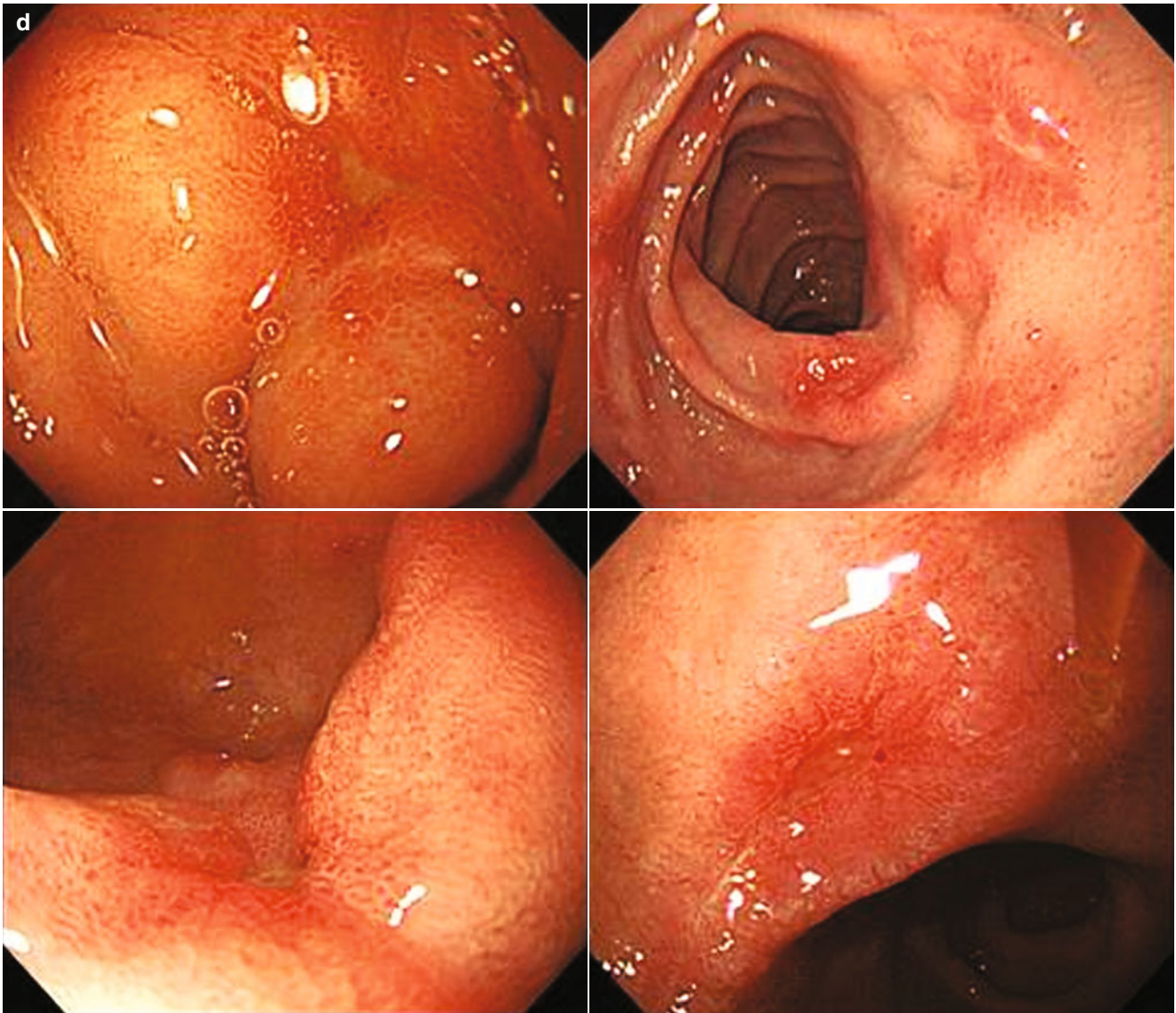


**Fig. 15.1** (continued)



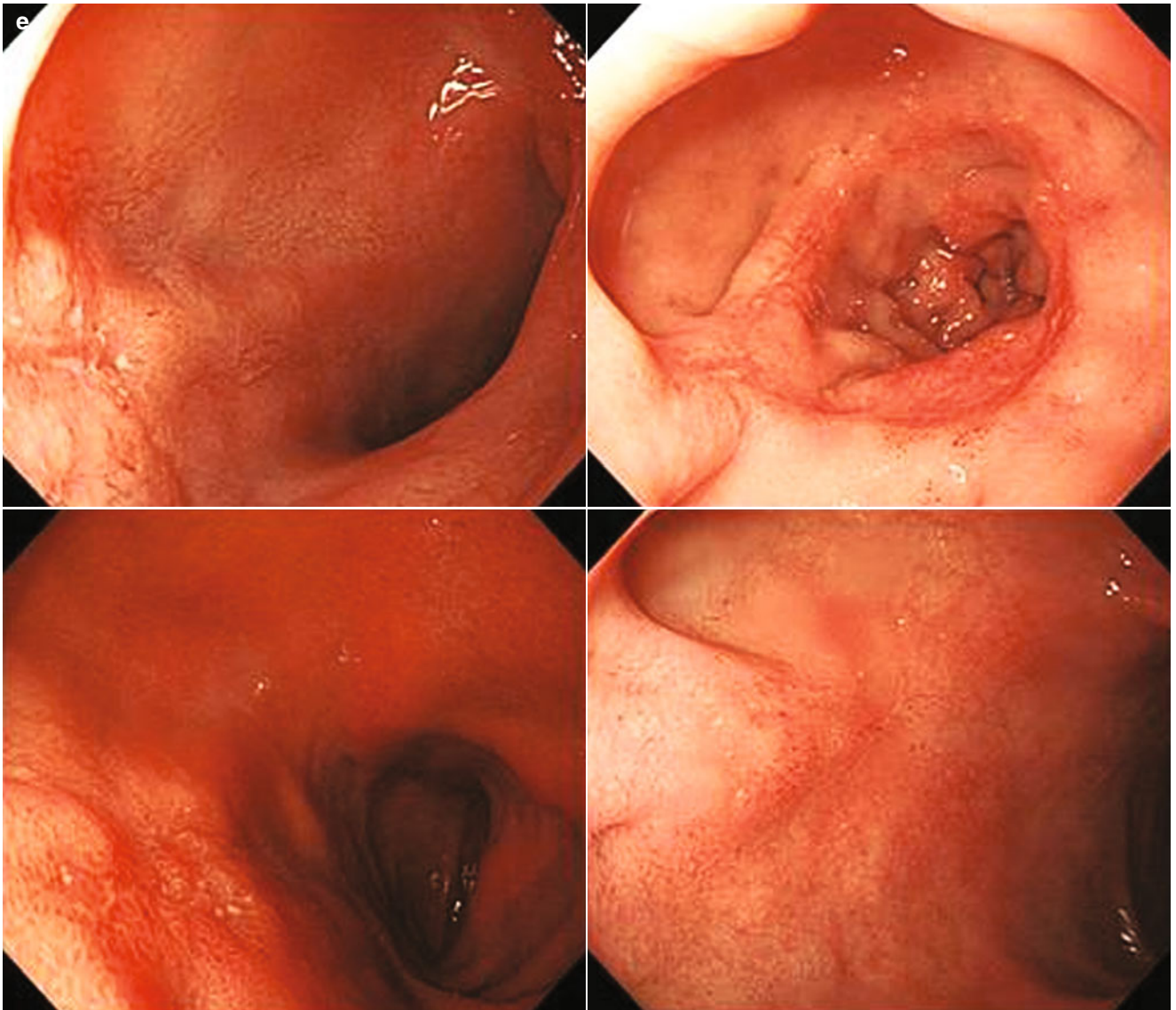


**Fig. 15.1** (continued)

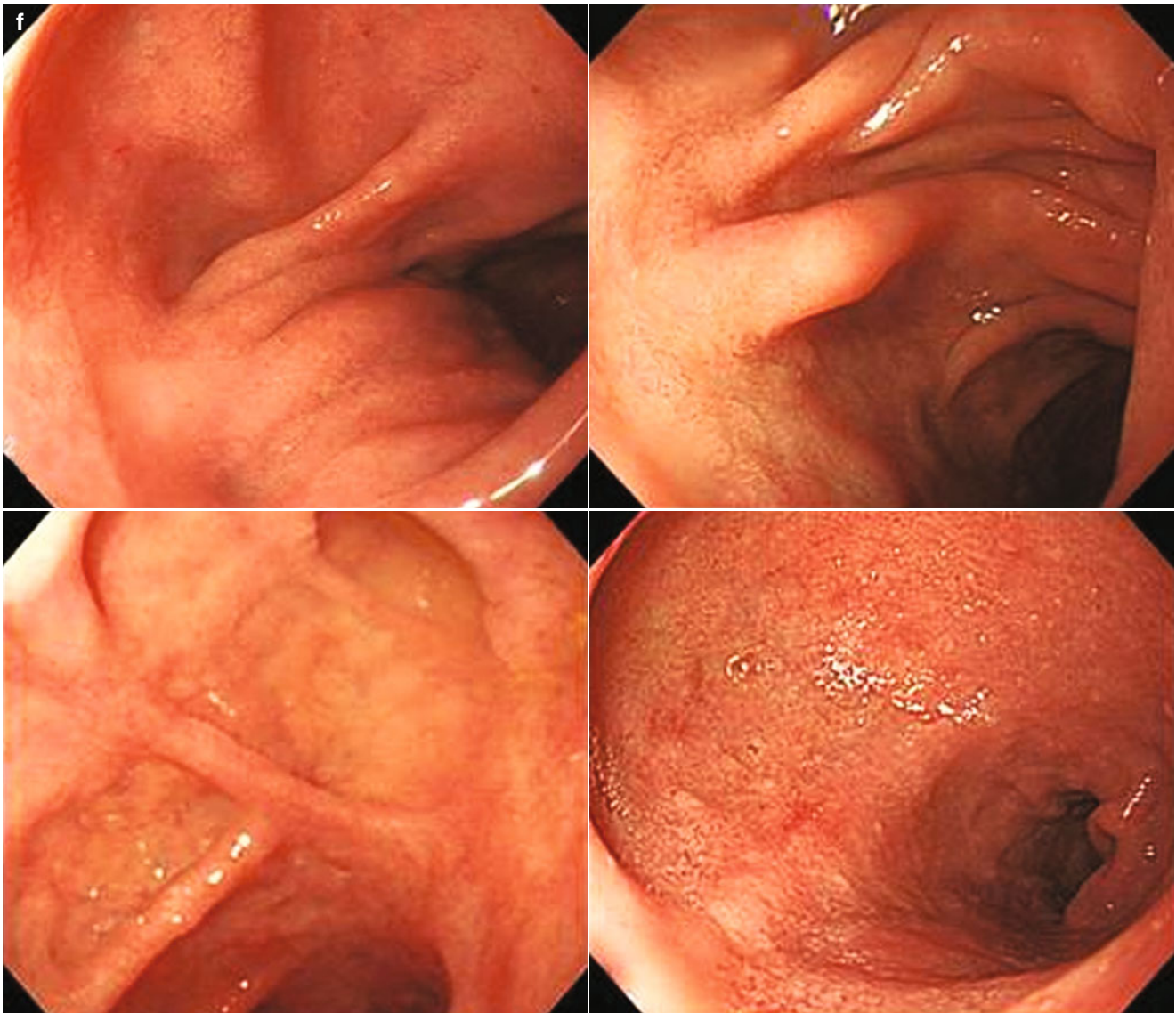


**Fig. 15.1** (continued)





**Fig. 15.1** (continued)



**Fig. 15.1** (continued)



### 15.1.3 Complications

#### 15.1.3.1 Bleeding

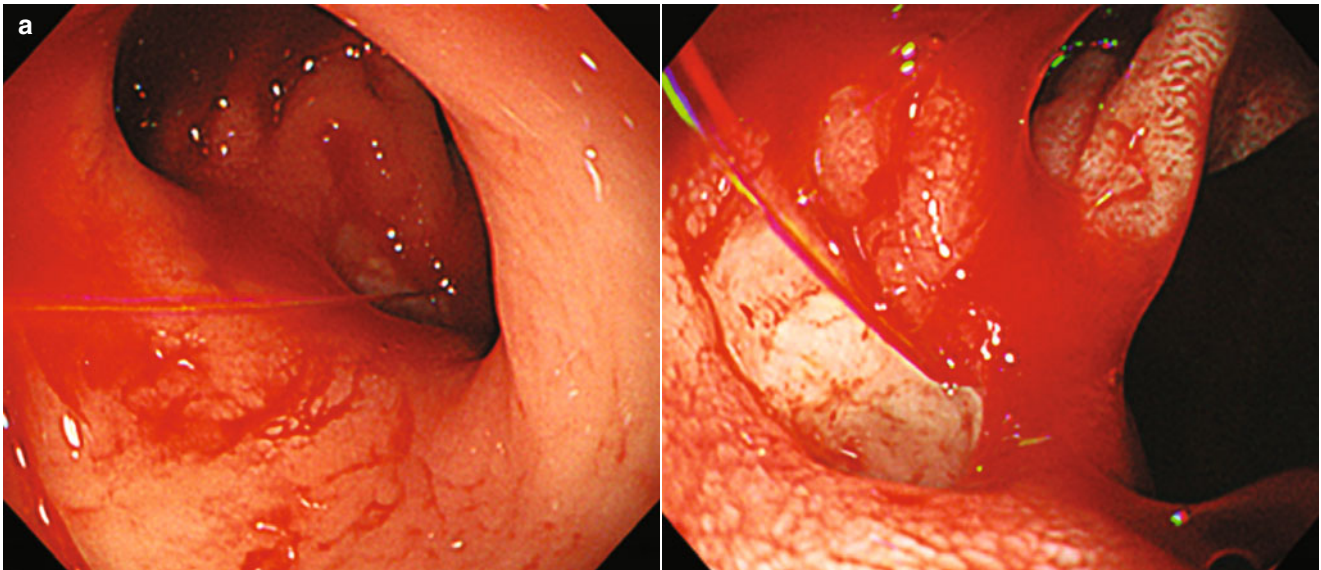
Hemorrhage from gastrointestinal tract is the most common complication in peptic ulcer disease such as gastric ulcer and duodenal ulcer [2, 3]. Hemorrhage from a duodenal ulcer can

occur when a blood vessel is exposed and eroded in the ulcer base. Bleeding ulcers can be divided into three types according to the Forrest classification (Table 15.1, Fig. 15.2) [1]. These endoscopic stigmata provide prognostic value. The ulcers with active bleeding or protruding visible vessels warrant endoscopic therapy as well as pharmacologic therapy.

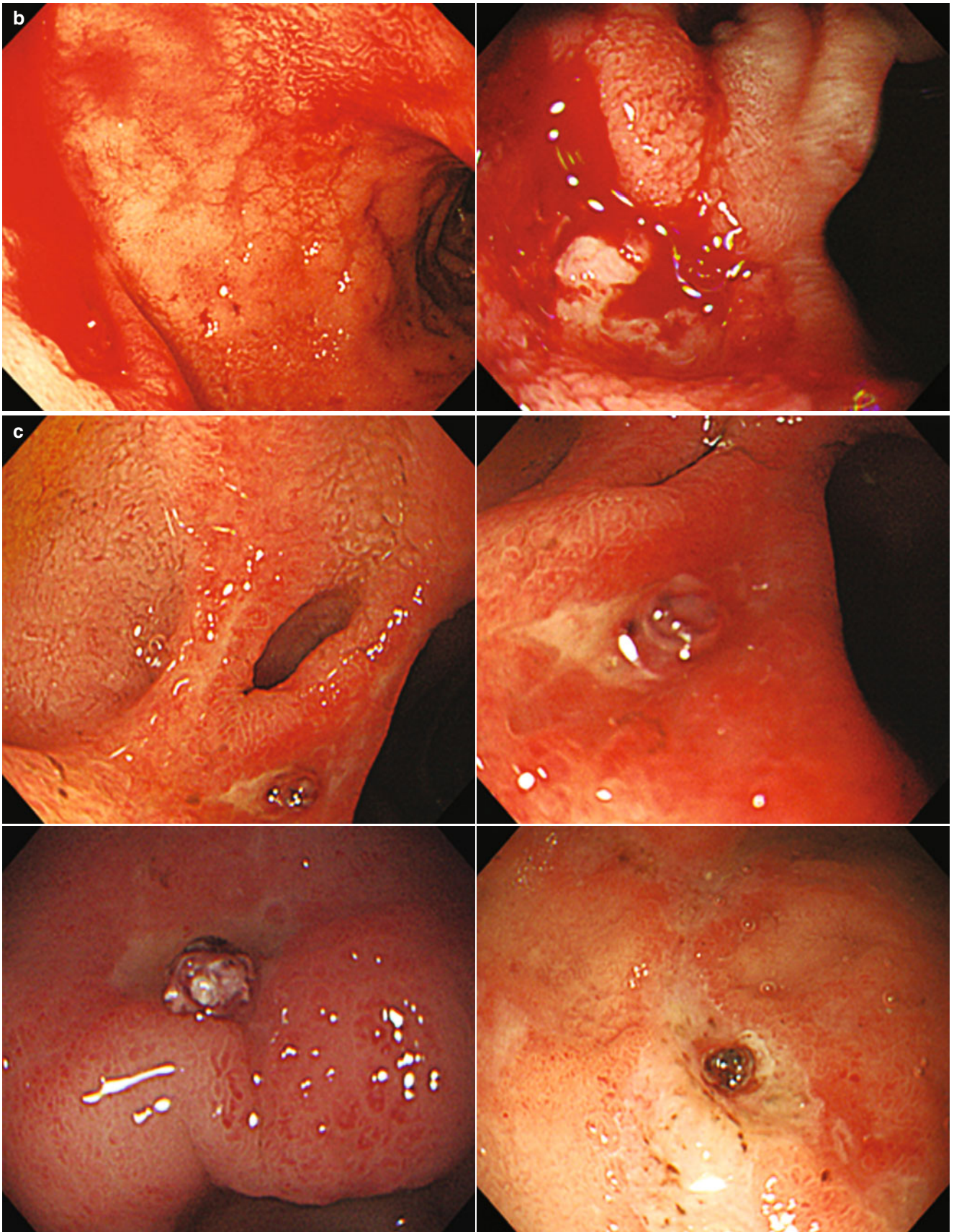
**Table 15.1** Forrest classification of bleeding duodenal ulcer

Type I	Active bleeding
Ia	Spurting bleeding
Ib	Oozing bleeding
Type II	Stigmata of recent bleeding
IIa	Nonbleeding protruding visible vessel
IIb	Adherent clot <sup>a</sup>
IIc	Flat red or black pigmentation
Type III	Clean-based ulcer

<sup>a</sup>Removal of adherent clot is usually recommended for precise classification and decision of therapeutic modality

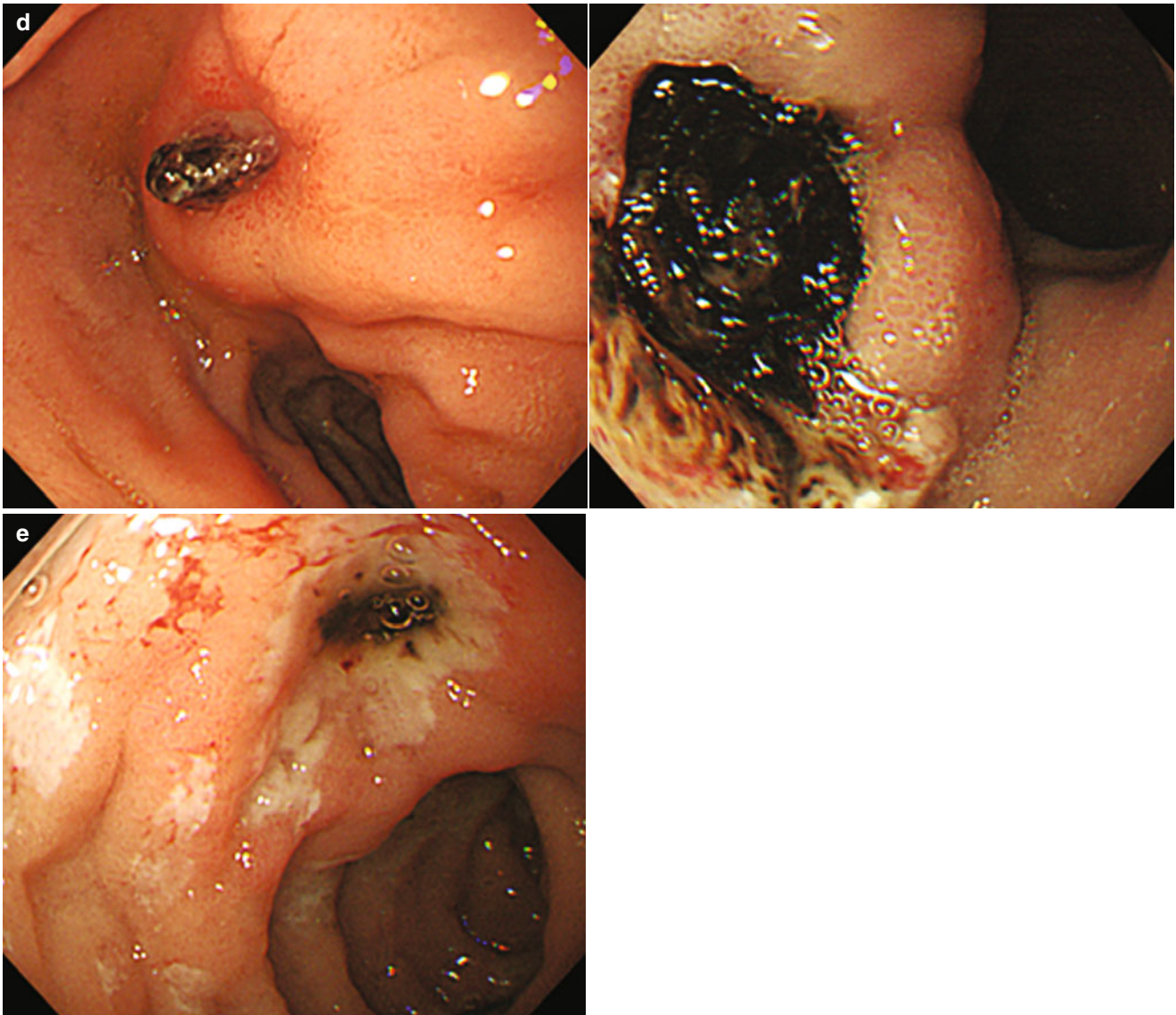


**Fig. 15.2** Bleeding duodenal ulcers. (a) Type Ia. (b) Type Ib. (c) Type IIa. (d) Type IIb. (e) Type IIc



**Fig. 15.2** (continued)





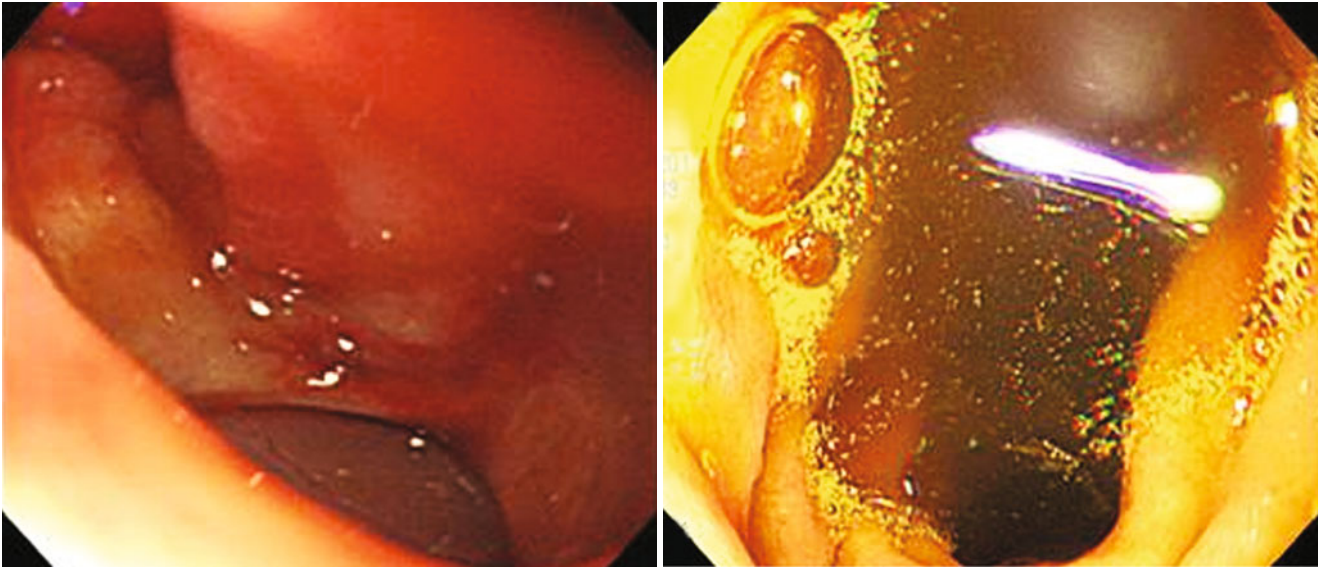
**Fig. 15.2** (continued)



### 15.1.3.2 Perforation

The second most common complication related to duodenal ulcer is perforation (Fig. 15.3) [2]. The peritoneal irritation

signs are usually developed, whereas the typical symptoms and signs cannot be noted in a posterior perforating ulcer. In this case, only radiating back pain can be presented.

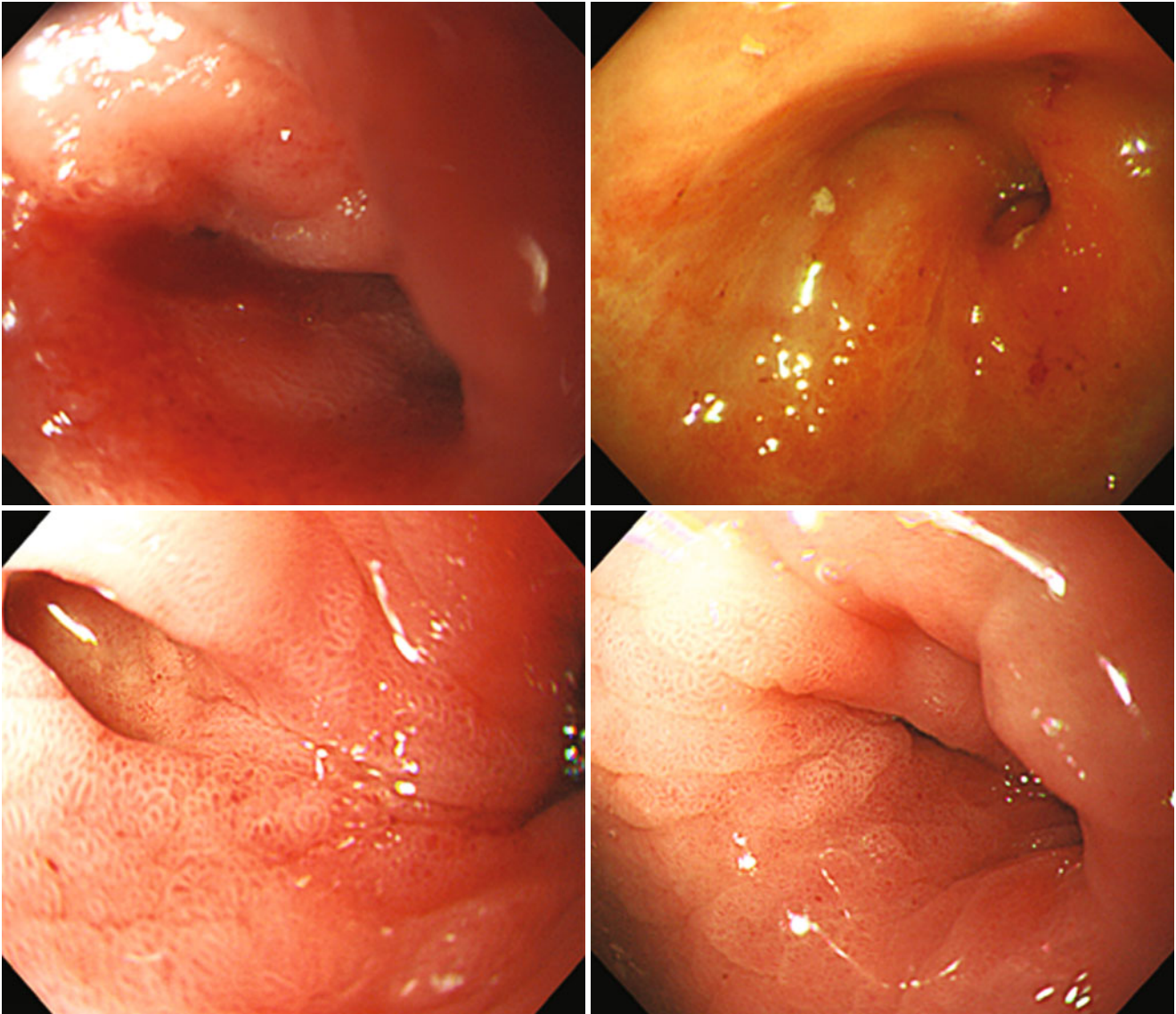


**Fig. 15.3** Perforated duodenal ulcers

### 15.1.3.3 Stricture

Active-stage duodenal ulcers with severe edema and inflammation can cause transient gastric outlet obstruction. This

relative obstruction usually resolves during ulcer healing. A fixed obstruction can occur because of the scar formation and followed by distortion of structures (Fig. 15.4) [3].



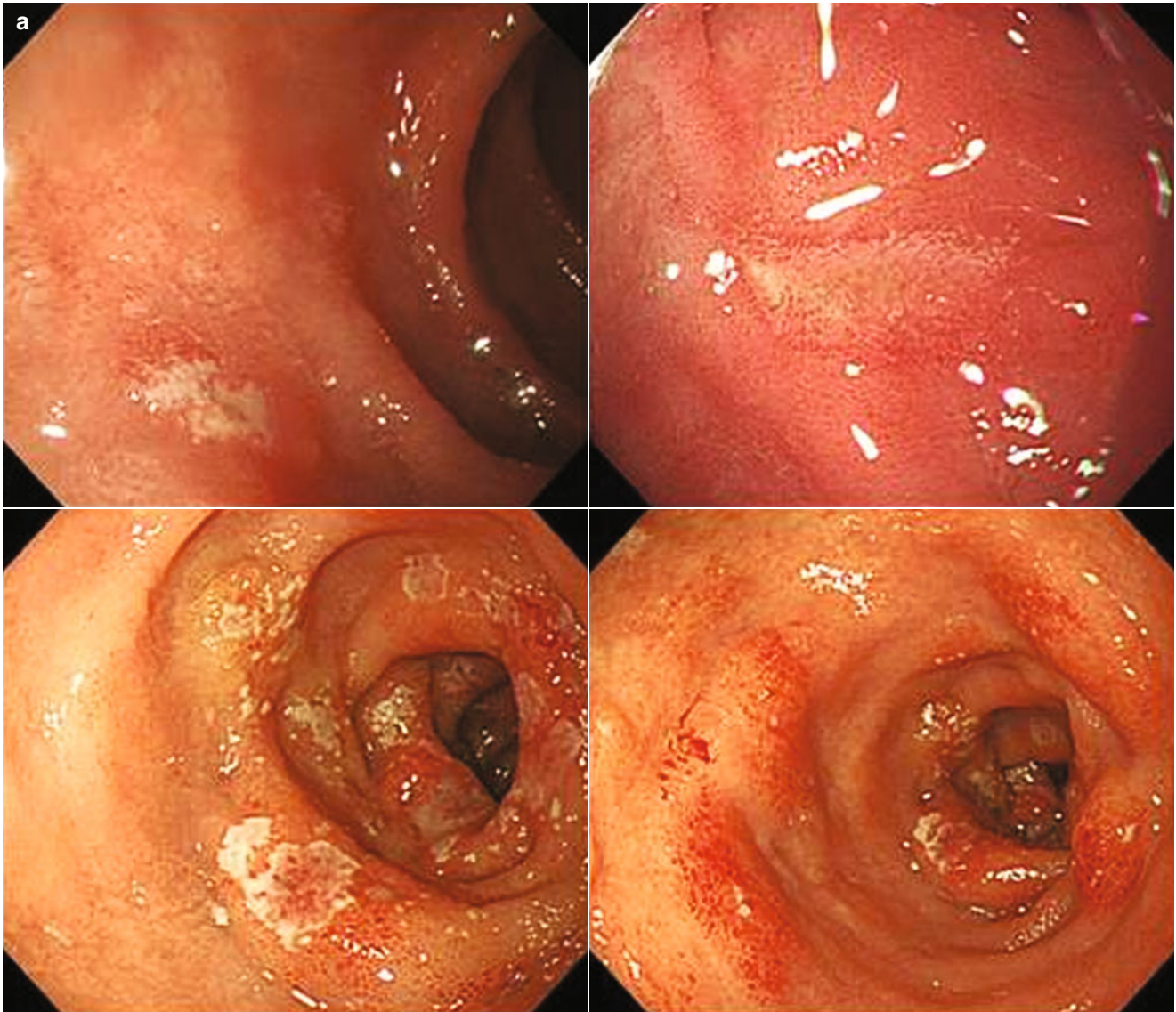
**Fig. 15.4** Duodenal ulcers with stricture



## 15.2 Duodenitis

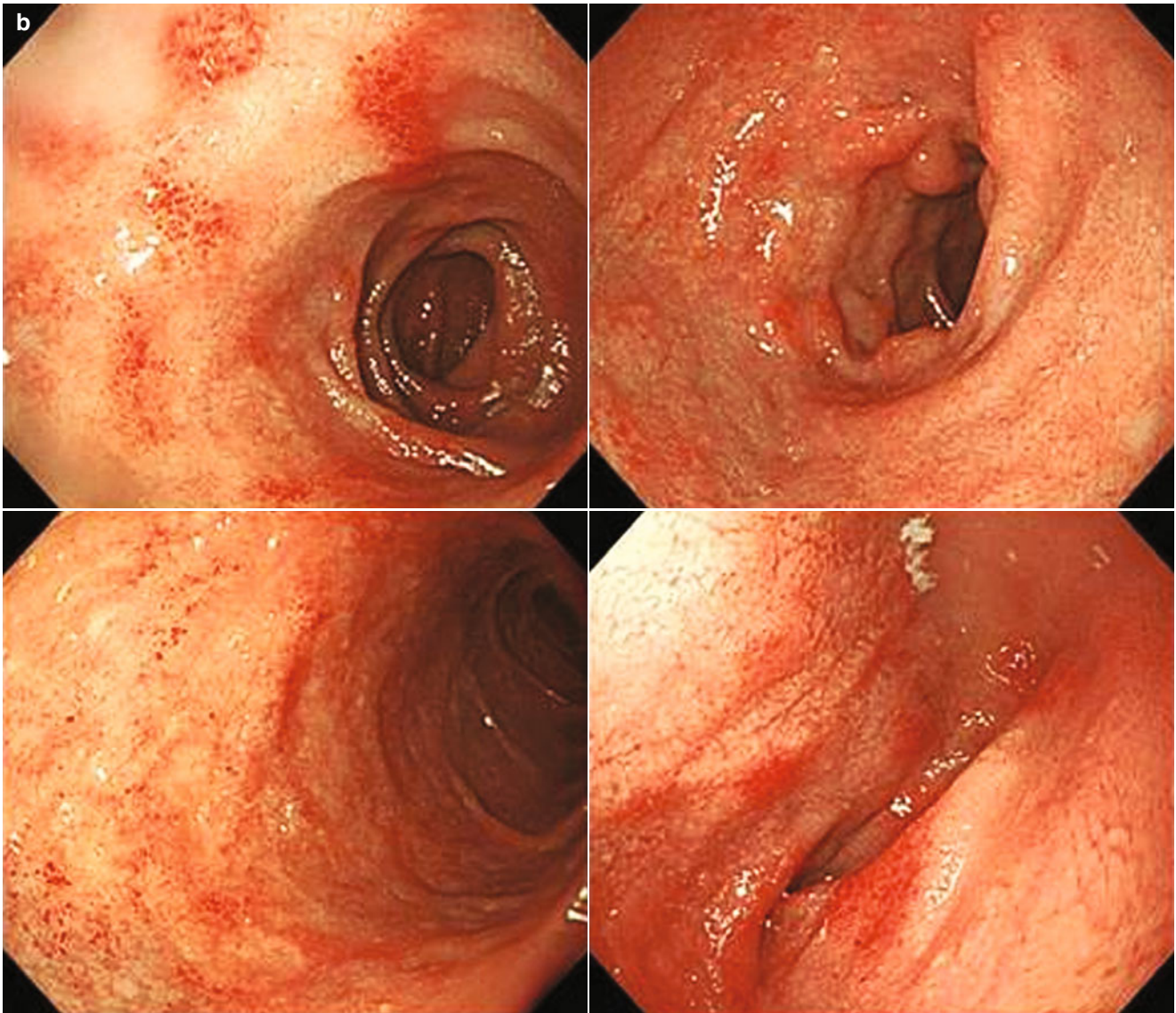
Duodenitis is inflammation of the duodenum. Endoscopic findings are erosions, erythema, and/or edema of duodenal

mucosa (Fig. 15.5). *H. pylori* infection, prolonged use of medications such as NSAIDs, alcohol, or tobacco may lead to duodenitis. Less commonly, severe illness, radiation therapy, or Crohn's disease are also associated with duodenitis [2].



**Fig. 15.5** Duodenitis. (a) Erosive. (b) Erythematous

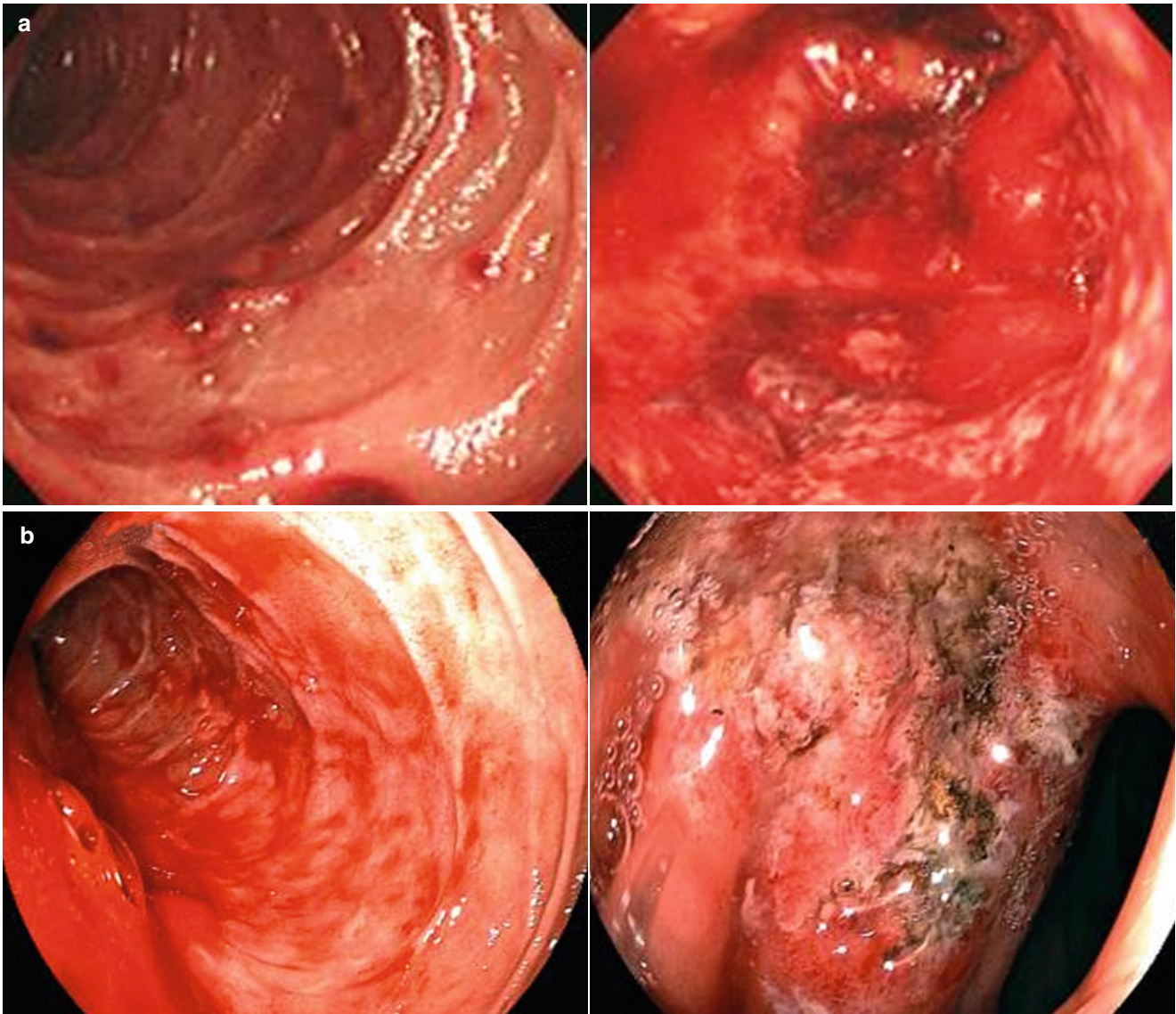




**Fig. 15.5** (continued)

### 15.3 Rare Causes of Duodenal Ulcer or Duodenitis

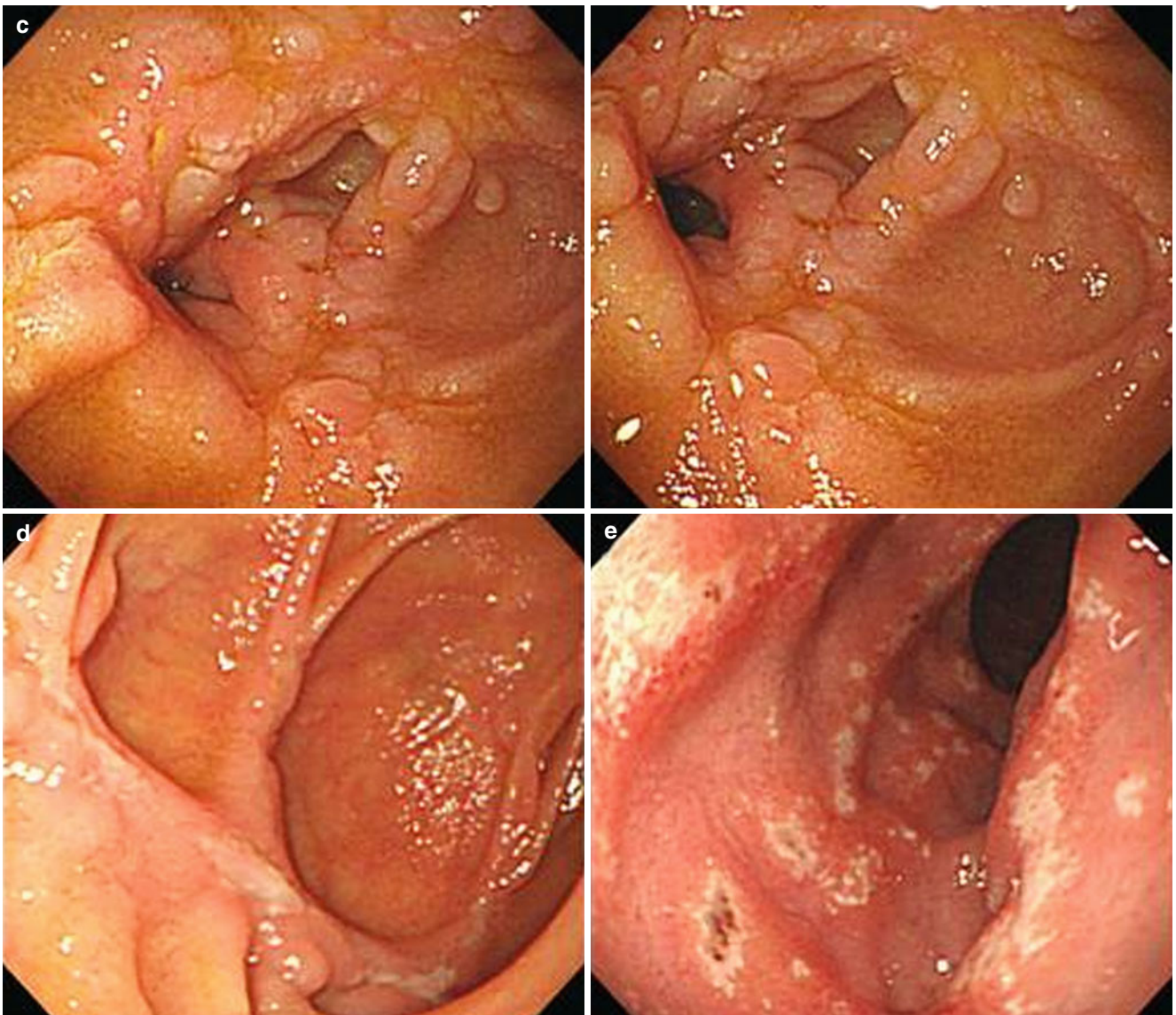
Rarely, severe illness, viral infection, tuberculosis, Crohn's disease, or Zollinger-Ellison syndrome can cause duodenal ulcers or duodenitis (Fig. 15.6) [2, 3].



**Fig. 15.6** Rare causes of duodenal ulcer or duodenitis. (a) Severe hemorrhagic duodenitis in a patient with aplastic anemia. (b) Severe hemorrhagic duodenitis in a patient with burn. (c) Duodenal scarring and

stricture in a patient with tuberculosis. (d) Longitudinal duodenal ulcer in a patient with Crohn's disease. (e) Multiple erosive duodenitis in a patient with Zollinger-Ellison syndrome





**Fig. 15.6** (continued)

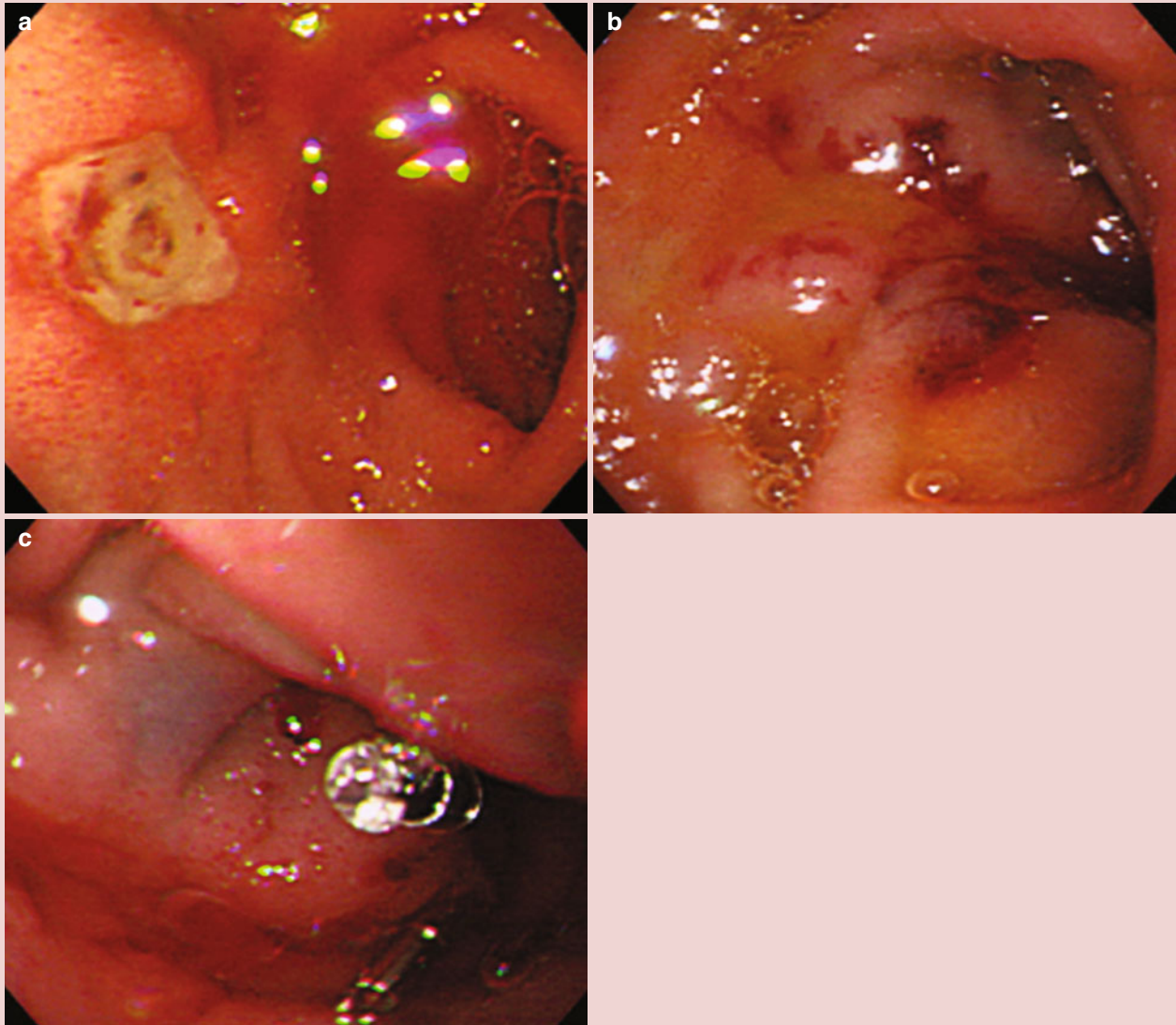


### Interesting Case

This case is an intramural duodenal hematoma, hemoperitoneum, and pancreatitis after endoscopic hemostasis for bleeding duodenal ulcer in a 54-year-old female end-stage renal failure patient undergoing maintenance hemodialysis (Fig. 15.7).

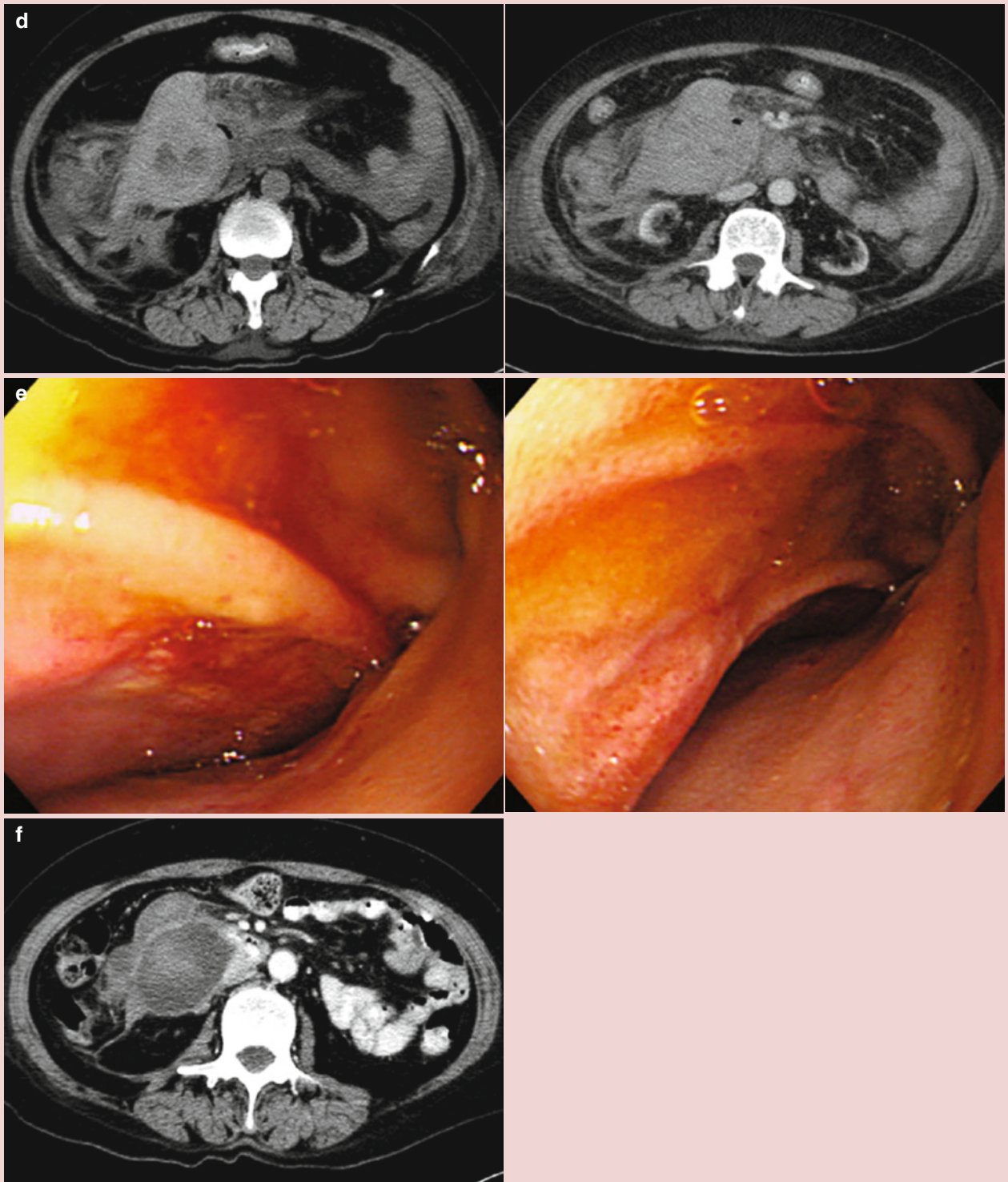
Duodenal intramural hematoma is mostly caused by blunt abdominal trauma. However, it can be developed as a complication of anticoagulation therapy, blood

dyscrasia, or a complication of diagnostic and therapeutic endoscopy. The symptoms and signs of these patients are abdominal pain, vomiting, fever, or hematochezia. This duodenal intramural hematoma is rarely accompanied with intestinal obstruction, severe pancreatitis, or acute peritonitis as its complications. Its diagnosis can be made clear by abdominal ultrasonography or abdominal computed tomography.



**Fig. 15.7** Intramural duodenal hematoma, hemoperitoneum, and pancreatitis in a patient with maintenance hemodialysis. (a) Duodenal ulcer (Forrest IIc) on anterior wall of bulb. (b) Dieulafoy lesion on superior descending angle. (c) Epinephrine injection and hemoclipping on Dieulafoy lesion. (d) Intramural

hematoma on duodenum and hematoma extending to the retroperitoneal space and abdominal cavity. (e) Luminal narrowing on the second portion of the duodenum due to hematoma. (f) Resolving hematoma after 1-month supportive treatment



**Fig. 15.7** (continued)

## References

1. Korean Society of Gastrointestinal Endoscopy. Atlas of gastrointestinal endoscopy. Seoul: Medbook; 2011.
2. Vakil N, et al. Peptic ulcer disease. In: Feldman M, editor. Sleisenger and Fordtran's gastrointestinal and liver disease. 9th ed. Philadelphia: Elsevier; 2010.
3. Valle JD, et al. Peptic ulcer disease and related disorders. In: Longo DL, editor. Harrison's principles of internal medicine. 18th ed. Seoul: McGraw-Hill; 2012.