CHAPTER 20

Hematemesis and melena

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ESSENTIAL FACTS ABOUT CAUSATION

Diagnosis	Number of patients (%) 2007[16]	Mortality (%)
Ulcer	1826 (27)	162 (8.9)
Erosive disease (gastric and duodenum)	1731 (26)	195 (14.1)
Esophagitis	1177 (17)	65 (5.5)
Varices and portal hypertensive gastropathy	819 (12)	87 (14)
Malignancy	187 (3)	31 (17)
Mallory-Weiss syndrome	213 (3)	10 (4.7)
Other diagnosis	797 (12)	125 (16)
Total	6750	675 (10)

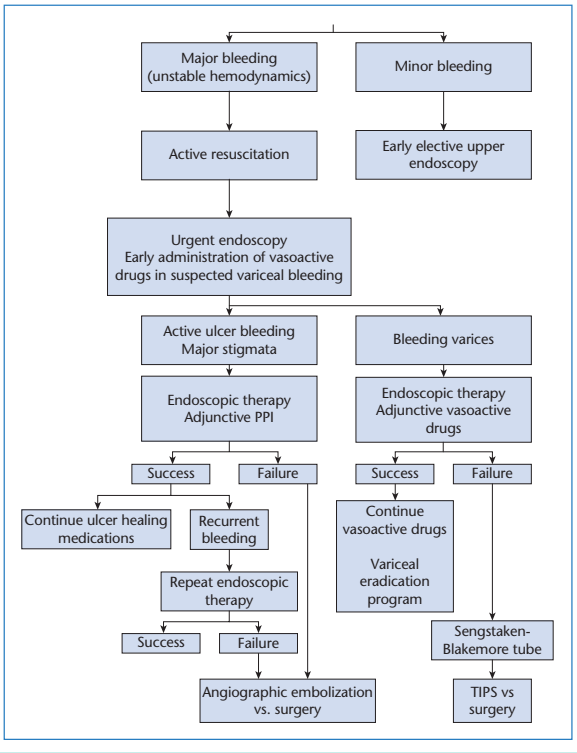
Data adapted from The United Kingdom National Audit in Upper Gastrointestinal Bleeding 2007 [16].

ESSENTIALS OF DIAGNOSIS

- Symptoms: Coffee ground vomiting, hematemesis, melena, hematochezia, anemic symptoms
- Past medical history: Liver cirrhosis, use of non-steroidal anti-inflammatory drugs
- Signs: Hypotension, tachycardia, pallor, altered mental status, melena or blood per rectum, decreased urine output
- Bloods: Anemia, raised urea, high urea to creatinine ratio
- Endoscopy: Ulcers, varices, Mallory-Weiss tear, erosive disease, neoplasms, vascular ectasia, and vascular malformations

ESSENTIALS OF TREATMENT

Algorithm for management of acute GI bleeding



Introduction

Upper gastrointestinal bleeding (UGIB) is a common medical emergency that carries substantial mortality. Despite advances in medical management, the mortality rate still ranges from 15% to 50% [1–3]. Upper endoscopy is the standard diagnostic and therapeutic tool in managing these patients. Stratification of the patients into low- or high-risk groups aids in formulating a clinical management plan and early endoscopy with aggressive post-hemostasis care should be provided in high-risk patients.

What are the signs and symptoms?

Hematemesis and melena are signs of upper gastrointestinal bleeding. Hematemesis is defined as vomiting of blood or blood clots, whereas melena is defined as passage of dark,

Textbook of Clinical Gastroenterology and Hepatology, Second Edition. Edited by C. J. Hawkey, Jaime Bosch, Joel E. Richter, Guadalupe Garcia-Tsao, Francis K. L. Chan.

tarry stool with a characteristic pungent smell. Fresh hematemesis is often a reliable sign signifying ongoing or active bleeding. Occasionally, vomiting of swallowed blood from hemoptysis or bleeding from the upper aero-digestive tract, e.g., nasopharynx, can confuse the diagnosis. Coffee ground vomiting is also a classic sign of upper gastrointestinal bleeding. The sign usually indicates a less severe bleed. The vomitus looks like ground coffee as a result of oxidized iron within heme molecules of red blood cells after their exposure to gastric acid. Melena occurs when hemoglobin is converted to hematin or other hemochromes by bacterial degradation. This can be produced experimentally by the ingestion of as little as 100–200 mL of blood. Blood is absorbed by the small intestine causing raised blood urea. A raised blood urea (BUN): creatinine ratio can therefore aid the diagnosis of UGIB. Some of this azotemia is probably secondary to hypovolemia. If the volume of an upper GI bleed is large, the patient may present with hematochezia (passage of fresh blood per rectum). Conversely, if the volume of bleeding is small but sufficient to supply enough hemoglobin for degradation, and if colonic motility is sufficiently slow, bleeding from the small bowel or proximal colon may cause melena. Small bowel bleeding is however uncommon. Bleeding from colonic sources, e.g. tumors, is either slow, leading to anemia or hemoccult positive stool; or rapid, such as in diverticular disease leading to hematochezia.

How common is upper gastrointestinal bleeding?

Upper gastrointestinal bleeding (UGIB) is a common medical emergency. The annual incidence in United Kingdom varies from 103 to 172 per 100 000 population [1,4]. In a recent United Kingdom national audit, the median age of patients who presented with upper gastrointestinal bleeding was 68 years [5]. Eighty-two percent of these patients were new admissions and 16% were patients who developed bleeding while hospitalized for other illnesses. The proportion of patients aged over 60 and 80 was 63% and 80% respectively. The overall mortality was 10%. Deaths occur mostly in elderly patients with co-morbid illnesses.

Initial management

It is mandatory that patients who develop acute gastrointestinal bleeding are assessed urgently. The aim of initial patient assessment is to stratify them into low- or high-risk groups for bleeding and mortality. This aids in formulating a clinical management plan [6,7]. In 80% of patients, bleeding has already stopped at their presentations. There are several risk scoring systems. The Rockall score is a composite score combining pre- and post-endoscopy clinical data with the aim to predict mortality. A score greater than 8 is associated with a 41% mortality and a rebleeding rate of 42.1%. The Glasgow-Blatchford Bleeding Score (GBS) is calculated using clinical and laboratory data on admission (Table 20.1 and Chapter 146) [8]. It has been found to accurately predict the need for intervention and also deaths. A score of 0 is classified as low risk for intervention or death. In these patients, outpatient management could be considered and this has been shown to be safe and may reduce hospital admissions [9]. Those judged to be of high risk should be admitted to high dependency areas and considered for urgent endoscopy.

Resuscitation is the first priority in the management of patients with UGIB. Table 20.2 provides a schema on the assessment of volume deficit. Large-bore intravenous cannulae should be inserted for rapid fluid administration. Circulating volume should be restored initially using crystalloid or

Table 20.1 Glasgow Blatchford bleeding score – admission risk markers and score values

Admission risk markers	Score value
Blood urea (mmol/L)	
6.5–7.9	2
8.0–9.9	3
10.0–24.9	4
≥25	6
Hemoglobin for men (g/L)	
120–129	1
100–119	3
<100	6
Hemoglobin for women (g/L)	
100–119	1
<100	6
Systolic blood pressure (mmHg)	
100–109	1
90–99	2
<90	3
Other markers	
Pulse ≥ 100/min	1
Presentation with melaena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

Table 20.2 Hypovolemic shock: symptoms, signs and fluid replacement

Blood loss (mL)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15	15–30	30–40	>40
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (mL)	>30	20–30	5–15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

colloid solutions. Red blood cells should be transfused when hemodynamics are unstable and/or hemoglobin is less than 10 g/dL at the time of presentation. In those with impaired mental status, their airways should be protected and often orotracheal intubation is required prior to endoscopy. A urinary catheter is useful, especially in those with hemodynamic compromise, to detect oliguria. A central venous catheter is useful for monitoring volume replacement, particularly in those with incipient heart failure. Physical examination may reveal stigmata of chronic liver disease that may call for specific treatments such as vasoactive drugs, antibiotics, and drugs to prevent encephalopathy.

Endoscopy

Endoscopy defines the cause of bleeding and provides prognostic information, and more importantly, therapies carried out during endoscopy can improve patients' outcomes. In pooled analyses of randomized controlled trials, endoscopic therapy reduces recurrent bleeding, hospitalization, transfusion, need for surgery, and deaths. Early endoscopy within 24 hours is recommended in most patients [10]. In selected patients with signs of ongoing bleeding and hypotension, urgent endoscopy with therapy may be lifesaving. Endoscopic procedures should be performed by medical or surgical gastroenterologists with expertise in therapeutic endoscopy. This should be performed in a unit supported by appropriate staff, monitoring and resuscitation equipment.

Causes of bleeding and their treatment

For practical management, UGIB can also be categorized into variceal and non-variceal bleeding. The Essential Facts about Causation box shows the endoscopic diagnoses in patients from the National Audit in 2007 [5]. Peptic ulcers remain the commonest cause accounting for 27% of cases. *Helicobacter pylori* infection is less prevalent in bleeding peptic ulcers when compared to uncomplicated peptic ulcers. Non-steroidal anti-inflammatory drugs or aspirin use are common in the elderly. Patients suffering from variceal bleeding have increased over the past decade accounting for 8% of those presenting with acute upper GIB.

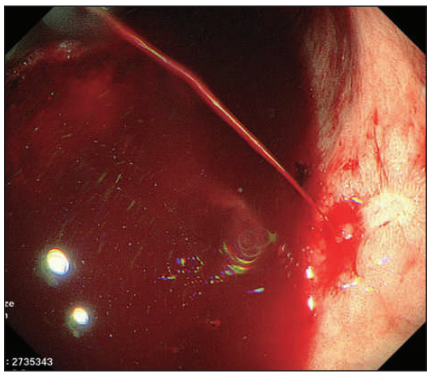


Figure 20.1 Ulcer with spurting hemorrhage.

Peptic ulcer bleeding

The endoscopic appearances of bleeding peptic ulcers can be categorized into those that are actively bleeding (Forrest I), ulcers that exhibit stigmata of recent bleeding (nonbleeding visible vessel defined as protuberant discoloration (II a), an adherent clot (II b), and flat pigmentations (II c)), and a clean base ulcer (Forrest III) (Figures 20.1–20.3). Their prevalence, risks of further bleeding, and need for surgery is summarized in Table 20.3 [11]. Endoscopic therapy is indicated in ulcers that are actively bleeding or with a non-bleeding visible vessel, which is defined as a protuberant discoloration in an ulcer base (Figures 20.4–20.5) (Videos 20.1 and 20.2). It is controversial whether clots overlying ulcer craters should be removed. Ulcers with flat pigmentations or a clean base are associated with small or negligible risk of recurrent bleeding. No

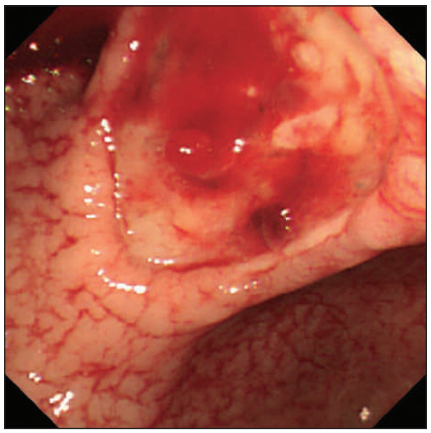


Figure 20.2 Blood oozing from a visible vessel.

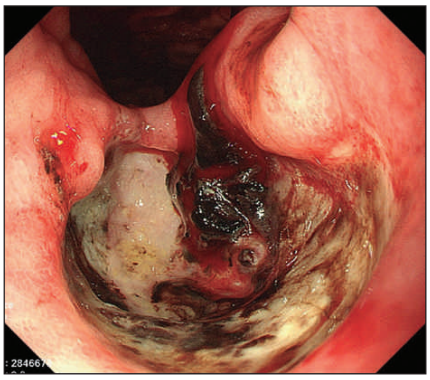


Figure 20.3 Ulcer with an adherent clot.

Table 20.3 Bleeding ulcers: prevalence, risk, and need for surgery

Endoscopic characteristics	Prevalence (%)	Further bleeding (%)	Surgery (%)	Mortality (%)
Clean base	42	5	0.5	2
Flat spot	20	10	6	3
Adherent clot	17	22	10	7
Nonbleeding visible vessel	17	43	34	11
Active bleeding	18	55	35	11

Data are from patients in prospective trials that did not receive endoscopic therapy. (Modified from Laine L and Peterson WL. *N Engl J Med.* 1994; 331:717–727.5. © 1994 Massachusetts Medical Society. All rights reserved.)

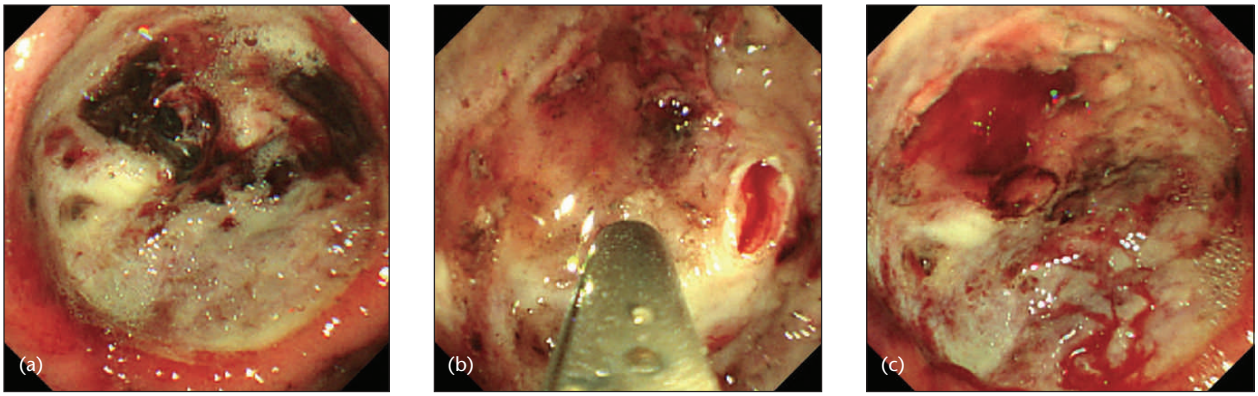


Figure 20.4 Heater probe application to a visible vessel.

endoscopic therapy is indicated. After endoscopic hemostasis to bleeding peptic ulcers, the use of high-dose proton pump inhibitor (PPI) up to 72 hours has been shown to reduce recurrent bleeding (Chapter 146).

Large ulcers in the bulbar duodenum and lesser curve of the stomach can erode into branches of the gastroduodenal artery complex and the left gastric artery. Endoscopic treatment often fails to stop bleeding from these arteries. Factors that have consistently been identified to predict failure of endoscopic therapy include: hemodynamic instability, comorbid illnesses, active bleeding, large ulcer size of greater than 2 cm, posterior bulbar or lesser curve ulcer [12].

In the management of patients with recurrent bleeding after initial endoscopic control, further endoscopic control can often

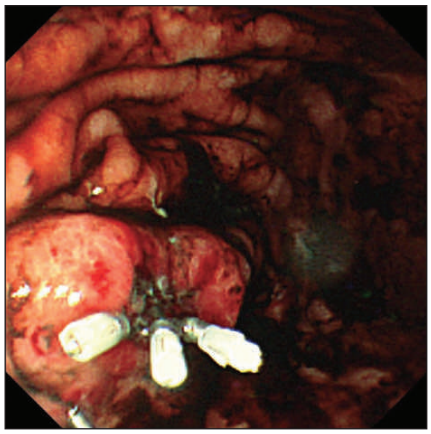


Figure 20.5 Hemoclip application to a visible vessel.

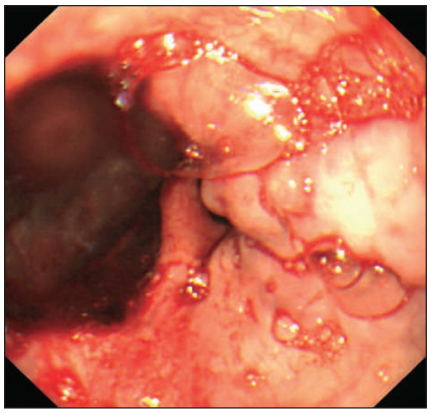


Figure 20.7 Bleeding esophageal varices.

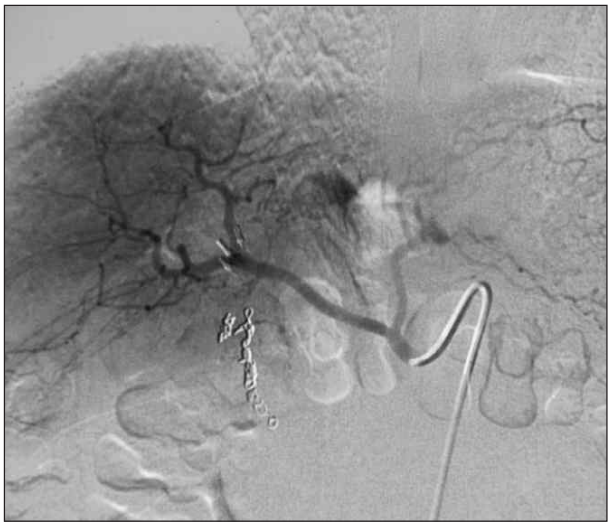


Figure 20.6 Transarterial embolization of the gastroduodenal artery. Hemoclips placed endoscopically are still visible.

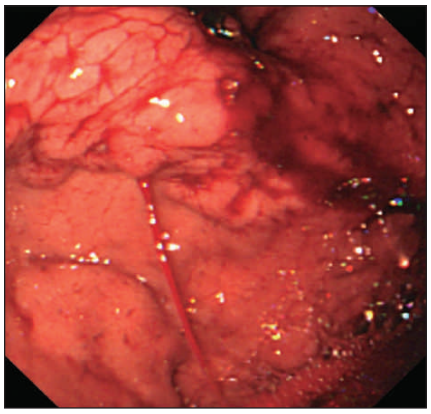


Figure 20.8 Bleeding gastric varices.

be successful. More definitive methods of ulcer hemostasis either in the form of surgery or angiographic embolization should however be offered to patients with episodes of hypotension and large ulcers [13]. Traditionally, surgery is considered the most definitive method of controlling bleeding. However, postoperative mortality occurs in around 20–25% of patients. When the expertise is available, angiographic embolization is an attractive alternative in those who fail endoscopic haemostasis [14] (Figure 20.6). Major ischemic complications are uncommon and ranged from 0% to 16%. A retrospective comparison between surgery and angiographic embolization favors the latter treatment in managing massive bleeding from ulcers that fail endoscopic treatment.

Variceal bleeding

Bleeding from esophago-gastric varices is often severe and mortality from the first bleeding is often high (Figures 20.7, 20.8). The overall prognosis is often dependent on the severity of liver disease as indicated by the Child-Pugh grading. The presence of features suggesting chronic liver disease e.g. alcohol use, ascites and jaundice should alert the possibility of a variceal bleed. It calls for specific treatments of prophylactic broad spectrum antibiotics and vascoactive drugs. Emergency endoscopic treatment is often associated with transient bacteremia. The administration of prophylactic antibiotics decreases the incidence of sepsis such as bacterial peritonitis and has been shown to improve survival [7]. Early administration of a vaso-active drug such as vasopressin or its analog or somatostatin or its analogs reduces portal venous blood flow and variceal pressure. This makes subsequent endoscopic treatment easier. Vasoactive drugs should be continued 2–5 days after endoscopic control to prevent recurrent bleeding [15]. Occasionally with massive bleeding, passage of a Sengstaken-Blakemore tube (with orotracheal protection of the airway) is

necessary. In up to 30% of cirrhotic patients presenting with UGIB, the bleeding source is non-variceal. In those with massive bleeding and often obtunded mental state from shock or encephalopathy, an orotracheal tube is required prior to endoscopy. Band ligation is the endoscopic treatment of choice for esophageal varices. When compared to endoscopic sclerotherapy, band ligation is associated with less local complications such as mediastinitis, esophageal strictures and leads to faster eradication of esophageal varices (Video 20.3).

Gastric varices can be divided into those in continuity with esophageal varices and those that occur in isolation. Isolated fundal varices are associated with high risk of recurrent bleeding and death. In the control of acute bleeding from isolated fundal varices, histoacryl glue injection appears to be the only effective method. Many would consider a transjugular intrahepatic portosystemic shunt (TIPS) or early shunt surgery as an alternative, especially after initial control with glue injection. In those patients where endoscopic therapy fails, a Sengstaken-Blakemore tube can tamponade bleeding in about 90% of cases; however, following deflation of the balloons, recurrent bleeding occurs in about 50% of cases.

Miscellaneous causes of upper GI bleeding

Other causes of upper GI bleeding are uncommon. Fresh hematemesis that occurs after emesis is typical of a Mallory-Weiss tear. The condition is often self-limiting and carries an excellent prognosis. Gastric vascular ectasia or "watermelon stomach" is characterized by the presence of linear red streaks radiating from the pylorus. Treatment is often difficult. The use of thermal ablative techniques appears to be the most effective option, although repeated sessions may be required [16]. Other causes include neoplasms and vascular malformations.

Prognosis

Despite advances in medical management, the mortality rate of patients suffering from upper gastrointestinal bleeding remains high and depends on the underlying pathology. In patients with non-variceal bleeding, a mortality rate up to 15% has been reported [1]. A number of studies have identified independent predictors for mortality and these include age, comorbidities, shock at presentation, in-hospital bleeders and presence of rebleeding [1,2]. In acute variceal hemorrhage, the risk of mortality can be up to 50% and the risk of mortality is strongly dependent on pre-existing liver function and the severity of cirrhosis [3]. The recognition of these predictors may help select patients who are most at risk and may benefit from intensive post-hemostasis care.

References

1. Blatchford O, Davidson LA, Murray WR, *et al.* Acute upper gastrointestinal hemorrhage in west of Scotland: case ascertainment study. *BMJ*. 1997;315:510–514.
2. Chiu PW, Ng EK, Cheung FK, *et al.* Predicting mortality in patients with bleeding peptic ulcers after therapeutic endoscopy. *Clin Gastroenterol Hepatol*. 2009;7:311–316.
3. Gatta A, Merket C, Amodio P, *et al.* Development and validation of a prognostic index predicting death after upper gastrointestinal bleeding in patients with liver cirrhosis: a multicenter study. *Am J Gastroenterol*. 1994;89:1528–1536.
4. Rockall TA, Logan RFA, Devlin HB, *et al.* Incidence of and mortality from acute upper gastrointestinal haemorrhage in the United Kingdom. *BMJ*.1995;311:222–226.
5. Hearnshaw SA, Logan RF, Lowe D, et al. Acute upper gastrointestinal bleeding in the UK: patient characteristics, diagnoses and outcomes in the 2007 UK audit. Gut. 2011 Jun 10. [Epub ahead of print]
6. British Society of Gastroenterology Endoscopy Committee. Non-variceal upper gastrointestinal haemorrhage: guidelines. *Gut*. 2002;51(Suppl 4):iv1–iv6.
7. Jalan R, Hayes PC. UK guidelines on the management of variceal haemorrhage in cirrhotic patients. *Gut*. 2000;46(Suppl 3): iii1–iii15.
8. Blatchford O, Murray WR, Blatchford M. A risk score to predict need for treatment for upper-gastrointestinal haemorrhage. *Lancet*. 2000;356:1318–1321.
9. Stanley AJ, Ashley D, Dalton HR, *et al.* Outpatient management of patients with low-risk upper-gastrointestinal haemorrhage: multicentre validation and prospective evaluation. *Lancet*. 2009;373:42–47.
10. Spiegel BM, Vakil NB, Ofman JJ. Endoscopy for acute non-variceal upper gastrointestinal tract hemorrhage: is sooner better? A systematic review. *Arch Intern Med*. 2001;161: 1393–1404.
11. Laine L, Peterson WL. Bleeding peptic ulcer. *N Engl J Med*. 1994;331:717–727.
12. Elmunzer BJ, Young SD, Inadomi JM, *et al.* Systematic review of the predictors of recurrent hemorrhage after endoscopic hemostatic therapy for bleeding peptic ulcers. *Am J Gastroenterol*. 2008; 103:2625–2632.
13. Lau JY, Sung JJ, Lam YH, *et al.* Endoscopic retreatment compared with surgery in patients with recurrent bleeding after initial endoscopic control of bleeding ulcers. *N Engl J Med*. 1999;340 :751–756.
14. Loffroy R, Guiu B, Cercueil JP, *et al.* Refractory bleeding from gastroduodenal ulcers: arterial embolization in high-operative-risk patients. *J Clin Gastroenterol*. 2008;42:361–367.
15. Ioannou G, Doust J, Rockey DC. Terlipressin for acute esophageal variceal hemorrhage. *Cochrane Database Syst Rev*. 2003;(1): CD002147.
16. Pavey DA, Craig PI. Endoscopic therapy for upper-GI vascular ectasias. *Gastrointest Endosc*. 2004;59:233–238.