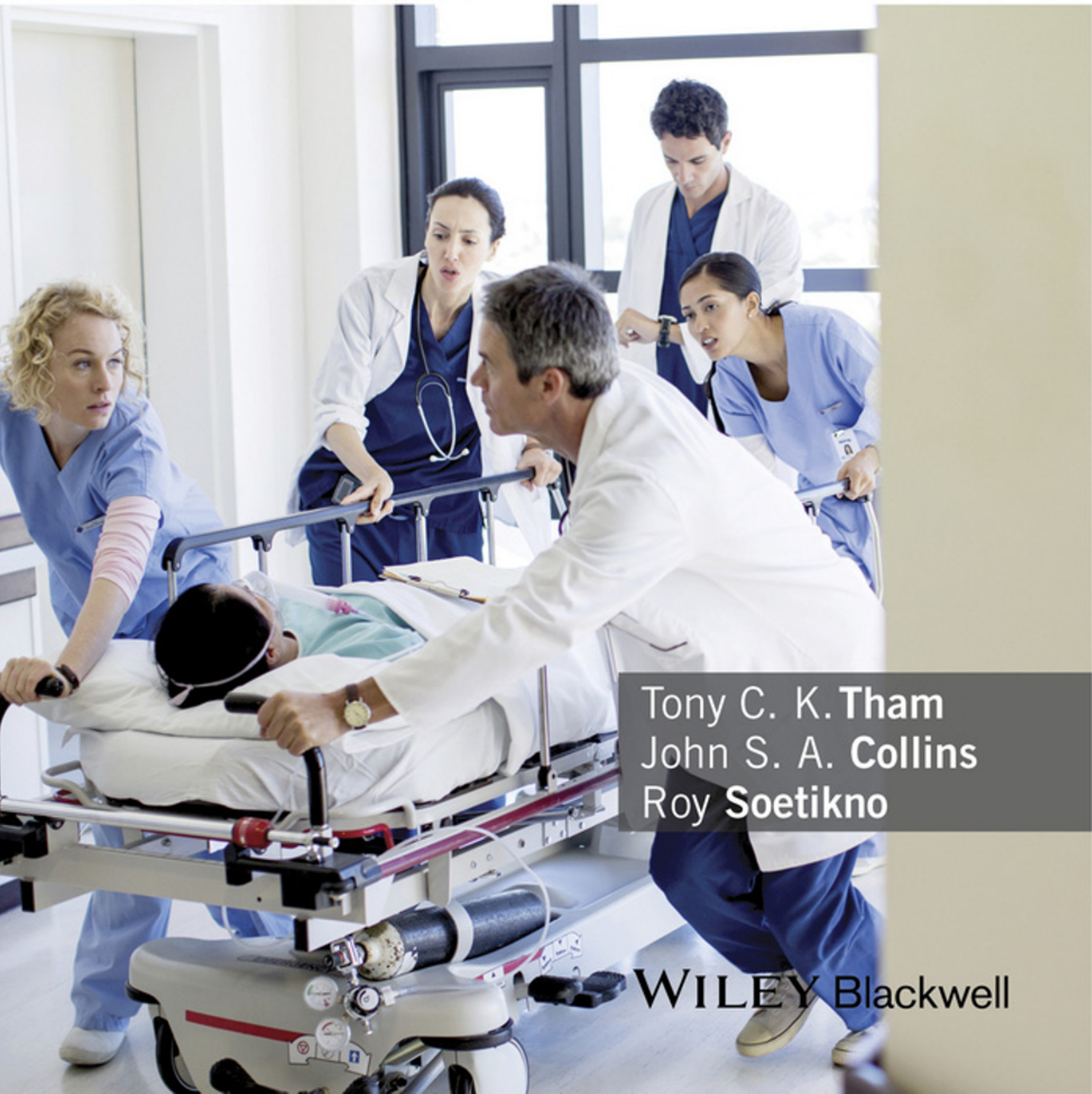
GASTROINTESTINAL EMERGENCIES

THIRD EDITION



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WILEY Blackwell

Gastrointestinal emergencies

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SECTION 1

Approach to specific presentations

CHAPTER 1

Approach to dysphagia

John S. A. Collins

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Definitions

Dysphagia refers to a subjective sensation of the obstruction of swallowed solids or liquids from mouth to stomach. Patients most frequently complain that food "sticks" in the retrosternal area or simply will "not go down." Patients may complain of a feeling of choking and chest discomfort. In some cases food material is rapidly regurgitated to relieve symptoms.

Dysphagia can be divided into two types:

- *oropharyngeal dysphagia*, where there is an inability to *initiate* the swallowing process and may involve disorders of striated muscle. There may be a sensation of solids or liquids left in the pharynx.
- *esophageal dysphagia*, which involves disorders of the smooth muscle of the esophagus and results in symptoms within seconds of the Initiation of swallowing.

Odynophagia is the sensation of pain on swallowing which is usually felt in the chest or throat. *Globus* is the sensation of a lump, fullness or tightness in the throat.

Differential diagnosis

The causes of the above types of dysphagia are shown in Tables 1.1 and 1.2.

History and examination

Acute dysphagia is a relatively uncommon, but dramatic, presenting symptom and constitutes a gastrointestinal emergency. The patient will complain of difficulty initiating swallowing or state that food is readily swallowed but results in the rapid onset of chest discomfort or pain, which is only relieved by passage or regurgitation of the swallowed food bolus. The latter sensation can result after swallowing a mouthful of liquid. In the acute case it is important to ask the patient about the presence of other neurological symptoms.

If oropharyngeal dysphagia is suspected, the following points are important:

- The patient may complain of nasal regurgitation of liquid, coughing or choking during swallowing or a change in voice character which may indicate nasal speech due to palatal weakness.
- Patients may describe repeated attempts at the initiation of swallowing.
- Symptoms are noticed within a second of swallowing.
- Patients with cerebrovascular disease may give a history of symptoms of transient ischemic attacks (TIA) – these would include visual disturbance, dysphasia, or transient facial or limb weakness.
- There may be progressive muscular weakness and dysphagia is only part of the symptom complex, in contrast to esophageal dysphagia where swallowing disorder is the most prominent symptom.
- Patients should have a careful neurological examination and evaluation of the pharynx and larynx including direct laryngoscopy.
- In cases of esophageal dysphagia, the following points are important:
- Is the sensation of dysphagia worse with liquids or solids? If a progressive obstructive lesion is the cause of symptoms, the patient will notice difficulty

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Table 1.1 Etiology of oropharyngeal dysphagia.

Neurological disorders
Cerebrovascular disease
Amyotrophic lateral sclerosis
Parkinson's disease
Multiple sclerosis
Bulbar poliomyelitis
Wilson's disease
Cranial nerve injury
Brainstem tumors
Striated muscle disorders
Polymyositis
Dermatomyositis
Muscular dystrophies
Myasthenia gravis
Structural lesions
Inflammatory – pharyngitis, tonsillar abscess
Head and neck tumors
Congenital webs
Plummer–Vinson syndrome
Cervical osteophytes
Surgical procedures to the oropharynx
Pharyngeal pouch (Zenker diverticulum)
Cricopharyngeal bar
Metabolic disorders
Hypothyroidism
Hyperthyroidism
Steroid myopathy

Table 1.2 Etiology of esophageal dyphagia.

Neuromuscular/dysmotility disorders
Achalasia
CRST syndrome
Diffuse esophageal spasm
Nutcracker esophagus
Hypertensive lower esophageal shincter
Nonspecific esophageal dysmotility
Chaga disease
Mixed connective tissue disease
Mechanical strictures – intrinsic
Peptic related to GERD
Carcinoma
Esophageal webs
Esophageal diverticula
Lower esophageal ring (Schatzki)
Benign tumors
Foreign bodies
Acute esophageal mucosal infections
Pemphigus/pemphigoid
Crohn's disease
Mechanical lesions – extrinsic
Bronchial carcinoma
Mediastinal nodes
Vascular compression
Mediastinal tumors
Cervical osteoarthritis/spondylosis

swallowing solids initially and liquids later. Difficulty with both solids and liquids suggests dysmotility.

- Is the dysphagia intermittent or progressive? Intermittent dysphagia may indicate a motility disorder such as diffuse esophageal spasm whereas a progressive course is more characteristic of an esophageal tumor.
- How long have symptoms been present? A long history usually greater than 12 months suggests a benign cause, whereas a short history less than 4 weeks suggests a malignant etiology.
- Has the patient a history of heartburn suggesting gastroesophageal reflux disease (GERD)? While a history of heartburn does not rule out gastroesophageal cancer as a cause of dysphagia, a long history in the presence of slow onset, non-progressive symptoms may point to a benign peptic stricture as the cause.

A diagnostic algorithm for the symptomatic assessment of the patient with dysphagia is shown in Fig. 1.1.

The etiology of esophageal dysphagia is summarized in Table 1.2.

While acute dysphagia may be painful, especially in relation to foreign body or food bolus impaction above an existing stricture, a history of odynophagia usually suggests an inflammatory condition or disruption of the esophageal mucosa leading to the irritation of pain receptors. The causes of odynophagia are:

- *Candida*
- herpes simplex
- cytomegalovirus
- pill-induced ulceration
- reflux disease/stricture
- radiation esophagitis
- caustic injury
- motility disorders stimulated by swallowing
- cancer
- graft-versus-host disease
- foreign body.

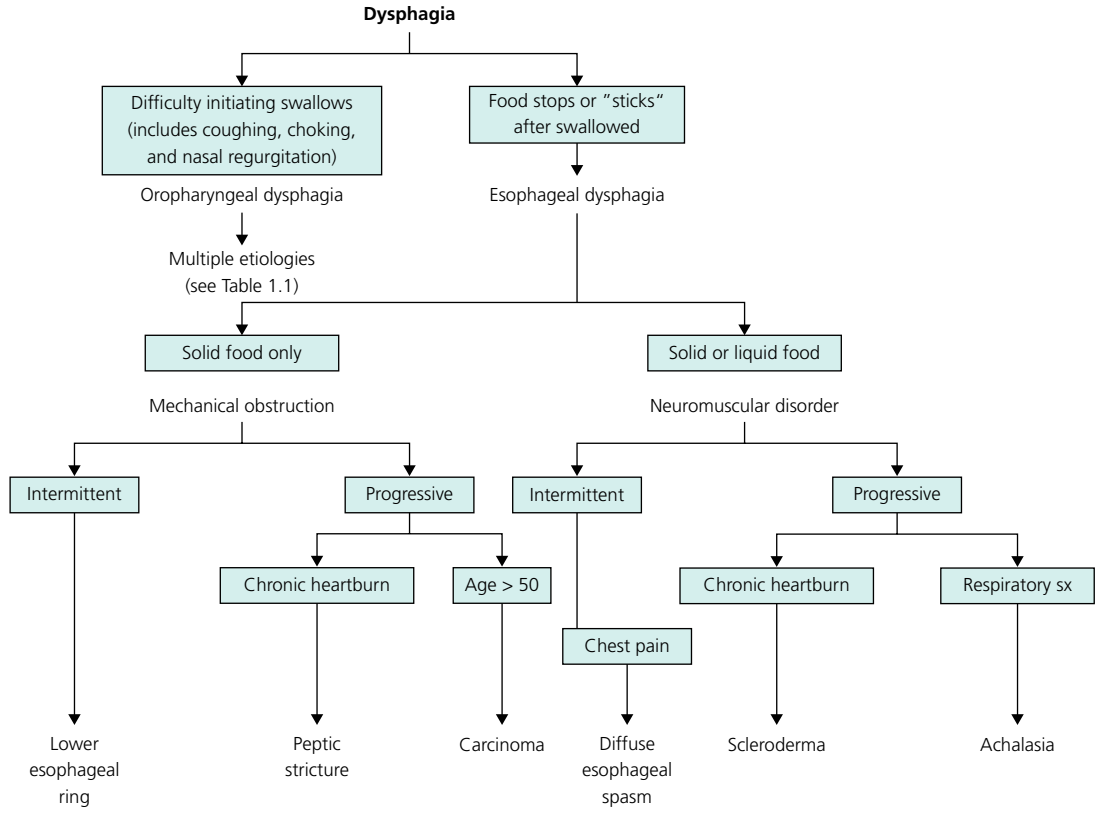

Fig. 1.1 Diagnostic algorithm for the symptomatic assessment of the patient with dysphagia. Source: Yamada 1995. Reproduced with permission of Wiley.

Clinical signs in patients who present with dysphagia are uncommon. On examination, the following signs should be noted:

- loss of weight
- signs of anemia
- cervical lymphadenopathy
- hoarseness
- concomitant neurological especially bulbar signs
- respiratory signs if history of cough/choking
- hepatomegaly
- oral ulcers or signs of *Candida*
- goiter.

Investigation

Dysphagia is considered to be an "alarm symptom" and should be investigated as a matter of urgency in all cases. Upper gastrointestinal endoscopy is a safe investigation in experienced hands provided the intubation is carried out under direct visualization of the oropharynx and upper esophageal sphincter. The endoscopist should be alert to the possibility of a high obstruction and the likelihood of retained food debris or saliva if dysphagia has been present for some time. If there is a history of choking, the patient should have a liquid-only diet for 24 hours followed by a 12-hour fast prior to the procedure. In some cases, the careful passage of a naso-esophageal tube to aspirate retained luminal contents may be necessary. At endoscopy, obstructing lesions can be biopsied and peptic strictures can be dilated with a balloon or bougie.

The presence of a dilated food and saliva-filled esophagus in the absence of a stricture raises the possibility of achalasia.

Barium studies are not a prerequisite for endoscopy but should be considered complementary in dysphagia. Barium swallow may give additional information in the following situations:

- in cases of suspected oropharyngeal dysphagia, especially if videofluoroscopy is employed;

- where a high esophageal obstruction is suspected prior to endoscopy;
- where a motility disorder is suspected as a method to assess lower esophageal relaxation.

In some cases, a barium swallow may be a useful investigation in certain circumstances:

- Where there is suspected proximal obstruction, e.g. laryngeal cancer, Zenker's diverticulum;
- Following a negative endoscopy or obstructive symptoms as lower esophageal rings may be more easily detected at fluoroscopy.

Esophageal manometry is indicated if both endoscopy and barium studies are inconclusive in the presence of persistent symptoms. Manometry requires intubation of the esophagus with a multilumen recording catheter attached to a polygraph. Pressure changes are recorded during water bolus swallows along the esophageal body and at the upper and lower esophageal sphincters.

Management of dysphagia

The management of dysphagia depends on the underlying cause. In a patient presenting with total dysphagia who is unable to swallow even small amounts of liquid or saliva, urgent treatment is indicated (Fig. 1.2).

The management of oropharyngeal dysphagia can be treated by control of the underlying neurological or metabolic disorder. Dietary modification under the supervision of a speech and language therapist may maintain oral swallowing and avoid gastrostomy tube placement in patients with stroke and pseudobulbar or bulbar palsy. Gastrostomy tube placement may be the only management option in patients with inoperable mouth or throat tumors, or in cases where recurrent pulmonary aspiration is life threatening.

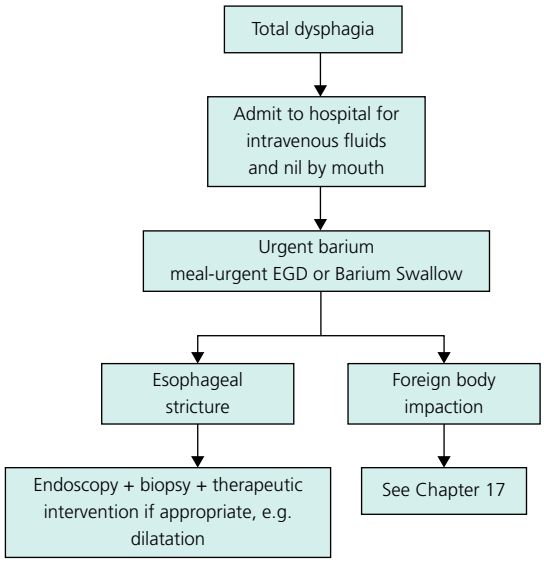

Fig. 1.2 Approach to management of total dysphagia.

Peptic stricture

When the endoscopic appearances are characteristic of a benign peptic stricture, dilatation can usually be carried out at the time of the procedure using either wire-guided bougies or a balloon. If the stricture is complex, very tight or associated with esophageal scarring, it may be safer to carry out wire-guided dilatation using graded bougies. The majority of patients will gain symptomatic relief and the risk of complications is low (see Chapter 21, Esophageal Perforation).

It is essential that all patients are treated with an adequate dose of a proton pump inhibitor to prevent recurrence. Repeat dilatations are necessary in some cases and repeat inspection and biopsy is advised if there is any concern about mucosal dysplasia or malignancy.

Esophageal carcinoma

Suspected carcinoma, which is detected at endoscopy, requires biopsy confirmation and subsequent staging so that a management plan can be formulated. The most accurate modality for staging is endoscopic ultrasound which can assess depth of local invasion and regional lymph node status. Chest and abdominal computerized tomography (CT) is a less accurate technique but CT/positron emission tomography (PET) scanning enhances staging accuracy, especially in adenocarcinomas.

Surgery offers the only chance of cure but only 30% of tumors are resectable and 5-year survival is 10% in European studies. Contraindications to surgery include invasion of vascular structures, metastatic disease and patients with comorbidity and high operative risk.

Palliative management will be indicated in 70% of patients following staging. Esophageal dilatation, followed by the endoscopic placement of a metal stent, gives adequate swallowing relief in the majority of cases. In situations where there is complete obstruction of the esophageal lumen by tumor, endoscopic laser therapy can provide adequate palliation of dysphagia. The prognosis is poor with a mean survival of 10 months

after diagnosis with a 5-year survival of 5%. Where surgical resection is completed after staging and selection, 5-year survival can be up to 25%.

Radiation injury

Following radiotherapy to the thorax or head and neck, some patients develop esophagitis, which may progress to stricturing and fibrosis. The diagnosis is confirmed by endoscopy and biopsy. Treatment consists of balloon or bougie dilatation and may have to be repeated in severe cases.

Esophageal webs and rings

Both of these esophageal lesions can result in dysphagia or lead to food bolus impaction. Webs are typically composed of thin mucosal tissue covered with squamous epithelium. They tend to occur proximally in the upper cervical esophagus and may be missed or ruptured at endoscopy, which is both diagnostic and therapeutic in these cases. They have been associated with chronic iron deficiency anemia. Rings are mucosal circular structures associated with dysmotility and seen at the esophagogastric junction. A Schatzki ring is a solitary thin rim of mucosa usually seen in the distended lower oesophagus. They are usually treated by dilatation and may be recurrent.

Food bolus impaction

See Chapter 20, Foreign Body Impaction in the Esophagus.

Eosinophilic esophagitis

This is an increasingly recognized cause of dysphagia and is diagnosed by characteristic endoscopic appearances and the finding of a dense eosinophilic infiltrate in esophageal mucosal biopsies (>15 per high power field). Dilatation is rarely required and the condition responds to low-dose swallowed topical steroids, administered by a metered inhaler.

Further reading

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CHAPTER 2

Approach to vomiting

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Definition

Acute nausea with or without vomiting is a common symptom. Nausea is described as an unpleasant sensation of imminent vomiting. Vomiting is defined as the forceful expulsion of gastric contents through the mouth and it should be differentiated from retching and regurgitation. Retching is the term that describes the labored, rhythmic respiratory activity and abdominal muscular contractions, which usually precede vomiting. Regurgitation is the effortless propulsion of gastric contents into the mouth without abdominal diaphragmatic muscular contractions.

The act of vomiting is initiated by the vomiting center in the medulla or the chemoreceptor trigger zone (CTZ) in the floor of the fourth ventricle, via a combination of motor and autonomic responses. Vomiting starts with salivation and then reverse peristalsis in the small intestines and a relaxed pyloric sphincter. Subsequent glottis closure (to prevent aspiration), abdominal and gastric muscular contractions and relaxation of the lower esophageal sphincter result in the final act of vomiting.

Etiology

The causes of acute nausea and vomiting are extensive and are summarized in Table 2.1.

- **Visceral (gut and peritoneum)** – visceral pain from a variety of intra-abdominal causes is often associated with an acute abdomen, including sepsis and mechanical obstruction. Gastric outlet obstruction leads to prolonged vomiting of the projectile nature.
- **CNS causes** – these include head injuries, intracranial infections/inflammation and raised intracranial pressure. Stimulation or disorders of the vestibular system such as motion sickness should not be overlooked.
- **Drugs** – nausea and vomiting are common side effects of chemotherapeutic agents, antibiotics, analgesics, and narcotics but the list of other offending drugs is endless. It is also important to enquire about recreational druguse, of which the commonest is alcohol abuse. Acetominophen/paracetamol and salicylate toxicity also result in nausea or vomiting, and this needs to be excluded.
- **Infections** – food poisoning (bacterial and viral) is the commonest. Others include epidemic viral infections, e.g. Norwalk agent and non-gastrointestinal infections such as otitis media and urinary tract infection.
- **Endocrine and metabolic** – commoner ones are hypercalcemia and uremia; less common cause includes acute intermittent porphyria.
- **Miscellaneous** – pregnancy (hyperemesis gravidarum), postoperation, cardiac causes (myocardial infarction, and congestive cardiac failure), psychogenic vomiting, and cyclical vomiting syndrome.
- **Functional nausea and vomiting.** This is a diagnosis of exclusion in some patients who present with chronic or episodic symptoms in the absence of a positive physical cause and despite full investigation.

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Table 2.1 Causes of acute vomiting.

Visceral stimuli	Peritonitis
	Small bowel obstruction
	Pseudo-obstruction
	Acute pancreatitis
	Acute cholecystitis
	Acute appendicitis
	Gastric outlet obstruction
	Mesenteric ischemia
CNS	Vestibular disorders
	CNS tumors
	Meningitis
	Cerebral abscess
	Subarachnoid hemorrhage
	Head injury
	Migraine
	Reye's syndrome
Drugs	Chemotherapeutic agents
	Antibiotics/antivirals
	Narcotics
	Analgesics
	Digoxin
Infections	Sporadic viral infections
	Gastroenteritis (bacterial/viral)
	Hepatitis viruses
	Non-gastrointestinal infections
Endocrine/metabolic	Diabetic ketoacidosis
	Adrenal insufficiency
	Hypercalcemia
	Uremia
	Acute intermittent porphyria
Miscellaneous	Psychogenic
Ethanol abuse	Radiotherapy
	Pregnancy
	Carcinomatosis
	Postoperation
	Cyclical (functional) vomiting

History

A detailed history is crucial in elucidating the cause of vomiting. The above causes should be considered. Constitutional symptoms of fever, myalgia, headache, or possible infectious contacts in the family, school, workplace, or institutions should alert the clinicians to an infectious etiology. Foreign travel and ingestion of inadequately cooked meat raise the suspicion of gastroenteritis. In these situations, a stool sample may reveal Norwalk agent, *Salmonella*, *Campylobacter*, *Staphylococcus aureus*, or *Bacillus cereus*.

Any associated abdominal pain with guarding points to an acute abdomen. Bilious vomiting suggests a proximal intestinal obstruction, while feculant vomiting is due to a more distal obstruction. Gastric outlet obstruction usually leads to postprandial projectile vomiting. When vomiting is associated with jaundice, anorexia and nausea, a hepatic etiology should be considered.

If there are no obvious symptoms of infection or acute abdomen, pregnancy should be excluded in female patients who are in their reproductive years. A thorough drug history, including over-the-counter medication and herbal remedies, may reveal the cause. Patients should also be asked about recent relevant CNS symptoms of vertigo, headache, blurred vision, or head injury. If no organic causes are obvious, then consider psychogenic or functional vomiting.

Examination

- Signs of dehydration – dry tongue, decreased skin turgor, postural hypotension.
- Smell of alcohol or ketones on the breath.
- Abdominal examination for signs of peritonism, gastric stasis, or acute intestinal obstruction. A succussion "splash" is suggestive of gastric outlet obstruction.
- CNS signs of meningism, nystagmus or papilledema.

Other important clinical features to look out for include:

- Signs of uremia – sallow appearance, pericardial rub.
- Signs of hypoadrenalism – pigmentation, postural hypotension.
- Characteristic skin blisters of acute intermittent porphyria.

Investigations

In all cases, basic laboratory tests such as full blood count, urea, electrolytes, and inflammatory markers are essential. A pregnancy test should be performed in any female of reproductive age, preferably before any radiographic studies are performed. Subsequent investigations will be directed towards the suspected cause elicited from the history.

If infection is suspected:

- Liver function tests.
- Viral hepatitis serology.

- Stool culture.
- Urinalysis and urine culture (particularly in elderly patients).

If a visceral cause is suspected:

- Serum pancreatic enzymes (amylase and lipase) – if acute epigastric tenderness suggests acute pancreatitis.
- Plain abdominal radiographs (erect and supine) – in the presence of peritonism, they may show an ileus, small bowel obstruction or free gas due to perforation.
- Abdominal ultrasound – may show gallstones and a thickened gallbladder wall if biliary signs are present.
- Upper endoscopy or barium meal – to confirm gastric outlet obstruction, preferably *after* the residual gastric contents have been emptied using a nasogastric tube.

If a central nervous system cause is suspected:

- Computerized tomography or magnetic resonance imaging of brain;
- Lumbar puncture – should be avoided until the presence of raised intracranial pressure has definitely been excluded.
- Vestibular testing.

Other tests to consider are synacthen test and urinary porphyrins.

Management of acute nausea and vomiting

A three-step approach is advocated:

1. Correction of any complications of vomiting such as dehydration and acid/electrolyte abnormalities.
2. Targeted therapy of identified cause of vomiting.
3. Symptomatic treatment if necessary.

Fluid replacement

If the patient is dehydrated and cannot tolerate oral fluids, intravenous fluid replacement using normal saline should be started. Potassium supplements may be required in patients with gastric outlet obstruction or if the vomiting has been associated with prolonged diarrhea. Management of diabetic ketoacidosis should be tailored according to local hospital guidelines.

Antiemetic drugs

These agents are useful in the acute phase in the majority of cases of acute vomiting where the underlying etiology is not clear but urgent symptomatic relief is necessary. In some cases, more than one agent may be required. The main types of antiemetic drugs are summarized in Table 2.2 and their clinical uses in Table 2.3.

Table 2.2 Classes of antiemetic drug.

Antimuscarinic	Scopalamine (hyoscine)
Antihistamine	Cyclizine
	Promethazine
	Meclozine
	Cinnarazine
Antidopaminergic	Prochlorperazine
	Domperidone
	Metoclopramide
Antiserotoninergic	Ondansetron
	Granisetron
	Tropisetron

Table 2.3 Clinical uses of different antiemetics.

Antimuscarinic	Motion sickness
Antihistamine	Motion sickness, vestibular causes
Antidopaminergic	
Extensive indications including gastroenteritis, postoperative, chemo/radiotherapy-induced vomiting, medication	
Antiserotoninergic	As above indications

- **Prochlorperazine (Stemetil®):** Particularly effective in vestibular vomiting. Its main side effects are extrapyramidal symptoms. It must be cautiously used in patients with Parkinson disease, narrow angle glaucoma and a history of phenothiazine sensitivity.

  Oral prochlorperazine – 20 mg initially followed by 10 mg after 2 hours. For prevention, give 5–10 mg 2–3 times daily.

  Sublingual (Buccastem®) – a 3 mg tablet can be placed high up between the upper lip and gums and left to dissolve. Recommended dosage is 1–2 tablets twice daily.

  Suppository – a 25 mg suppository can be placed rectally stat, followed by oral dose after 6 hours if necessary.

  Injection – give 12.5 mg stat by deep intramuscular injection, followed by oral dose after 6 hours if necessary.
- **Cyclizine (Valoid®):** This is a histamine H1 receptor antagonist and is effective in patients where there is a contraindication to the above. It can cause drowsiness and should be used with caution in the elderly. For

severe vomiting or in patients who cannot tolerate oral medication, give 50 mg stat either by intramuscular or intravenous injection and this can be repeated 8-hourly. For less severe vomiting, oral dose 50 mg 8-hourly can be given.

- **Domperidone (Motilium®) and metoclopramide (Maxolon®):** These drugs are dopamine receptor antagonists and also function as prokinetic agents. Domperidone has not been approved for use in the USA. Metoclopramide is contraindicated in gastrointestinal obstruction and perforation. Domperidone can be given orally (10–20 mg) or rectally (30–60 mg) every 4–8 hours. Alternatively, give metoclopramide 10 mg, either oral or injections (intramuscular/intravenous) every 8 hours.
- **Ondansetron (Zofran®):** If all above fail, this serotonin 5-HT3 receptor antagonist can be given as a 4 mg dose either by intramuscular or intravascular injection. It can also be given as 16 mg suppositories. If vomiting is controlled after the initial dose, the oral form can be given up to a daily maximum dose of 32 mg in the 4 or 8 mg tablet form. 5-HT3 receptor antagonists are regarded as first line antiemetics for chemotherapy-induced vomiting and they are generally well tolerated.
- **Erythromicin**: This antibiotic can enhance gastric emptying but may not improve nausea. It has been used as a prokinetic agent in cases of gastroparesis, but there is not a clear evidence base for prolonged benefit.
- **Antidepressants**: These drugs have been used in cases of functional nausea and vomiting where conventional antiemetics have been unsuccessful. They should be used in conjunction with psychological supportive therapy. There are few data or strong evidence base from trial to support their efficacy.

Further reading

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Management of specific causes of acute vomiting is described in detail in relevant chapters in this book.

CHAPTER 3

Approach to upper gastrointestinal bleeding

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Introduction

Upper gastrointestinal bleeding (UGIB) is a common presentation to emergency departments and associated with higher mortality with in-patients who experience UGIB. It commonly present with hematemesis (vomiting of blood) and/or melena (passage of black and tarry stools). The initial management of patients with UGIB involves an initial assessment, resuscitation if required, and then endoscopy to achieve hemostasis. A small number of patients may be discharged from the emergency department (ED) if they are deemed "low-risk" and an urgent endoscopy can be subsequently arranged; however, this practice varies throughout countries and institutions.

At endoscopy the major aim is to stratify the patients into low, medium, and high risk, depending on their initial findings and to perform therapeutic intervention if required to stop the bleeding and reduce the risk of recurrent bleeding post-endoscopy (rebleeding).

This chapter will summarize the approach and up-to-date management in patients with UGIB. (See also Chapter 22 on Acute Upper Non-variceal Gastrointestinal Hemorrhage and Chapter 25 on Variceal Hemorrhage.)

History

The symptoms and signs of patients presenting with upper gastrointestinal bleeding may include:

- dyspepsia
- epigastric pain
- heartburn
- weakness
- syncope
- hematemesis (e.g. coffee-ground vomitus, bright red vomitus, etc.)
- melena (black stools)
- hematochezia (bright red rectal bleeding)
- weight loss
- dysphagia.

Hematemesis suggests bleeding proximal to the ligament of Treitz. The character of vomitus can assist with the severity of bleeding – frank blood suggests active and more severe bleeding, whereas dark and coffee-ground vomitus suggests less active bleeding. The majority of patients with melena can have bleeding from the upper gastrointestinal tract; however, a small proportion may have bleeding from the small bowel or proximal right colon.

Causes of upper gastrointestinal bleeding

The causes of UGIB are summarized in Table 3.1 [1].

Past medical history

It is necessary to document the recent use of antiplatelet therapies and non-steroidal anti-inflammatory drugs (NSAIDs). Warfarin/low-molecular-weight heparins, and patients receiving newer anticoagulants such as

Gastrointestinal Emergencies, Third Edition. Edited by Tony C. K. Tham, John S. A. Collins, and Roy Soetikno.

Table 3.1 Causes of upper gastrointestinal bleeding.

Source of bleeding	Frequency (%)
Peptic ulcer	35–62%
Gastroesophageal varices	4–31%
Mallory–Weiss tear	4–13%
Gastroduodenal erosions	3–11%
Erosive esophagitis	2–8%
Malignancy	1–4%
Unidentified source	7–25%

Source: Laine 2001 (1). Reproduced with permission of McGraw-Hill Education.

Dabigatrin (Pradaxa®) and Rivaroxaban (Xarelto®), should be documented.

As peptic ulcers can recur, it is important to elicit a past medical history of peptic ulcer disease. A history of chronic alcohol abuse or likelihood of chronic viral hepatitis (B or C) increases the likelihood of variceal hemorrhage or portal gastropathy.

Examination and assessment

The initial evaluation of a patient with UGIB includes: a full history, physical examination, laboratory tests, and in some centers, nasogastric tube insertion (to estimate the volume and activity of the blood loss).

The benefit of a full initial evaluation is to assess the quantity of the bleed, the severity of the bleed, possible causes of the bleed, and any associated conditions that may be associated with a higher mortality (e.g. chronic liver disease).

In a recent meta-analysis on the indicators for UGIB, the specific indicators include: melena (likelihood ratio LR 5.1–5,9), melena identified on per-rectal exam (LR 25), blood or coffee ground-like vomitus detected during nasogastric lavage (LR 9.6), and a ratio of blood urea nitrogen to serum creatinine greater than 30 (LR 7.5) [2].

Specific factors associated with "severe bleeding" included red blood detected during nasogastric lavage (LR 3.1), tachycardia (LR 4.9), and or hemoglobin less than 8 g/dL (LR 4.5–6.2).

The majority of patients with melena can have bleeding from the upper gastrointestinal tract; however, a small proportion may have bleeding from the small bowel or proximal colon.

Table 3.2 Glasgow-Blatchford Score for upper gastrointestinal bleeding.

Admission risk marker	Score component value
Blood urea	
≥6·5 <8·0	2
≥8·0 <10·0	3
≥10·0 <25·0	4
≥25	6
Hemoglobin (g/L) for men	
12.0 <13.0	1
≥10.0 <12.0	3
<10.0	6
Hemoglobin for women	
≥10.0 <12.0	1
<10.0	6
Systolic blood pressure (mmHg)	
100–109	1
90–99	2
<90	3
Other markers	
Pulse ≥100 (per min)	1
Presentation with melena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

Scores of 6 or greater were associated with a greater than 50% risk of requiring an intervention (3,4).
Source: Blatchford 2013 (3) and Rockall 1996 (4). Reproduced with permission of Elsevier and the BMJ.

Initial assessment: risk stratification

See Chapter 22, Acute Upper Non-variceal Hemorrhage, for more details.

An urgent assessment of patients who present with UGIB should allow triage into low, medium-, and high-risk, and patients can be stratified according to risk with various risk calculators. The widely used calculators are the *Glasgow Blatchford Score (GBS)* (Table 3.2) and the *Rockall Score* (pre- and postendoscopy) (Table 3.3), and the *AIMS-65* scoring systems (Table 3.4) (3,4).

The Blatchford Score includes biochemical markers of blood loss and renal failure including: urea and hemoglobin level, whereas the Rockall Score includes age parameters, and also the postendoscopy diagnosis to ascertain the risk of rebleeding and mortality. The AIMS-65 scoring system includes albumin, INR, Glasgow Coma Scale, blood pressure, and age.

Table 3.3 Rockall Score for upper gastrointestinal bleeding.

Variable	Score 0	Score 1	Score 2	Score 3
Age	<60	60–79	>80	
Shock	No shock	Pulse > 100 BP > 100 systolic	Systolic < 100	
Co-morbidity	Nil		CCF, IHD, major morbidity	Renal failure, liver failure, metastatic cancer
Diagnosis	Mallory–Weiss tear	All other diagnoses	GI malignancy	
Evidence of bleeding	None		Blood, adherent clot, spurting vessel	

Source: Rockall 1996 (4). Reproduced with permission of Elsevier and the BMJ.

Table 3.4 Components of the pre-endoscopy AIMS 65 Score.

Albumin less than 30 g/L
INR greater than 1.5
Altered mental status (Glasgow Coma Scale score less than 14)
Systolic blood pressure of 90 mmHg or less
Age older than 65 years

Mortality associated with the number of risk factors in the AIMS 65 Score

- No risk factors 0.3%
- One risk factor 1%
- Two risk factors 3%
- Three risk factors 9%
- Four risk factors 15%
- Five risk factors 25%

Source: Saltzman 2011 (21). Reproduction with permission from Elsevier.

Can "low-risk' patients be discharged from the ED for early outpatient endoscopy?

A small proportion of patients with low GBS may be treated as outpatients with an early endoscopy [5]. In this study the GBS was better in predicting patients' need for hospital-based intervention, 30-day mortality and identifying low-risk patients than the Rockall Score, the Baylor Bleeding score, and the Cedars-Sinai Medical Center predictive index. An age-extended GBS (EGBS) identified a significantly higher proportion of low-risk patients than the GBS (p = 0.006); however, this requires further external validation. None of the scoring systems accurately predicted which patients would have rebleeding or mortality at 30 days.

This study is promising, but requires further validation and better scoring systems are required, which can accurately predict rebleeding and medium-term mortality, before they can be reliably utilized.

Medium- to high-risk patients with UGIB

All patients with hemodynamic instability (shock, orthostatic hypotension) or active bleeding should be admitted to a high dependency/intensive care unit for resuscitation and close observation. The majority of patients can be admitted to a gastrointestinal ward that is familiar with managing patients before and after endoscopic therapy.

AIMS 65 is a newly developed and validated score (Table 3.4), and in one US study appeared to be superior to GBS in predicting specifically inpatient mortality from UGIB; however, the GBS was superior for predicting the requirement for blood transfusion. Both risk scores were similar when predicting the composite clinical endpoint of: inpatient mortality, rebleeding, and need for endoscopic, radiological, or surgical intervention, blood transfusion, intensive care admission, rebleeding, length of stay, and timing of endoscopy [6]. The AIMS 65 score requires further validation in different countries, and racial mixes before it can be widely adopted.

Management of UGIB

Resuscitation

The initial resuscitation of patients should include administration of intravenous fluids, supplementary oxygen, and correction of any underlying coagulopathy. Routine blood transfusion in patients with UGIB is controversial, and a Cochrane Review meta-analysis suggested that red blood cell transfusion in patients with

UGIB may be associated with higher mortality and rebleeding rate. This meta-analysis was limited by the small studies included in the analysis and the large quantity of missing data. In addition, the most important confounder was the possibility that patients who presented with more severe bleeding received more rapid and aggressive transfusions [7]. In the recently published UK National Institute for Health and Care Excellence (NICE) guidance, there is agreement with blood transfusion in massive bleeding with blood, platelets, and clotting factors, if certain criteria are met. The guidance advised that platelet transfusions are not to be offered in patients who are "not actively bleeding and are hemodynamically stable," but only offered to patients "actively bleeding with a platelet count less than 50×10^9/L."

It was recommended that fresh frozen plasma should be offered to patients with either a fibrinogen level less than 1 g/L or a prothrombin time (international normalized ratio [INR]) or activated partial thromboplastin time greater than 1.5 times the upper limit of normal range. Those taking warfarin with therapeutic INRs and who are actively bleeding should be offered prothrombin complex concentrate [8].

Patients who receive early intensive resuscitation are more quickly stabilized hemodynamically and the hematocrit corrected quicker, resulting in a lower incidence of myocardial infarction and reduction in mortality.

The approach to a hemodynamically unstable patient begins initially with assessing the airway, breathing, and circulation (ABCs). Some patients who present with severe blood loss and hypovolemic shock can present with mental status changes; in these circumstances the patients are at increased risk for aspiration. This is a potentially preventable complication and one that if present may increase morbidity and mortality in these patients. This situation should be recognized early and patients electively intubated in a controlled setting using cricoid pressure.

When the airway has been secured it is important to obtain intravenous access. It is adequate to obtain bilateral 16-gauge upper extremity intravenous lines for volume resuscitative measures. An estimated guideline for fluid replacement to correct the hypovolemia is the *3-for-1 rule*. This rule aims to replace each milliliter of blood loss with 3 mL of crystalloid (or colloid) fluid. This regime is commonly the initial fluid replacement until cross-matched or type-specific blood becomes available. Patients with comorbidities (e.g. cardiovascular diseases) may require pulmonary artery catheter insertion to evaluate cardiac performance profiles and resuscitation adequacy in the early stages.

Are there risks with "over-transfusing" patients with UGIB?

It is important to avoid over-transfusing patients with variceal and non-variceal bleeding as it may be associated with worse outcomes. A recent randomized trial included 921 patients with UGIB were assigned to either a "restrictive transfusion strategy" (transfusion to only when the Hb fell to <7 g/dL) or a "liberal transfusion strategy" (transfusion when the Hb fell to less than 9 g/dL) [9]. Patients in the restrictive transfusion group were more likely than those in the liberal group to avoid transfusion (51% vs. 14%, respectively) and received fewer units of blood (mean 1.5 vs.3.8 units). Mortality was lower in the restrictive group (5% vs. 9%, adjusted hazard ratio 0.55). Patients in the restrictive group were also less likely to have further bleeding or complications. Among patients with cirrhosis, the risk of death and further bleeding were lower with the restrictive group for patients with Childs "A or B" cirrhosis, but were similar for patients with Childs "C" cirrhosis. All patients in this study underwent emergency endoscopy (mean within 5 hours) and endoscopic therapy was given to those with active bleeding, a non-bleeding visible vessel, an adherent clot, or bleeding esophageal varices. This prompt endoscopy cannot be replicated in all units, and therefore the restrictive strategy may not be better for these patients who do not have an early endoscopy within 5 hours of presentation to the ED.

A recent large retrospective study, which included 1677 patients with non–variceal UGIB, reported that if patients received a blood transfusion within 24 hours, this was associated with an increased risk of bleeding after adjusting for factors such as hemodynamic instability, endoscopic therapy, high risk stigmata, or recurrent hemorrhage, initial Hb value and the presence of blood on rectal examination or in the nasogastric tube aspirate (OR 1.8) [10].

How to manage anti-coagulated patients with UGIB?

Endoscopy also appears to be safe in patients with UGIB who are moderately anti-coagulated [11] and therefore our approach is to reverse the INR to less than 3 before

endoscopy, whilst continuing the reversal during endoscopy. This level of moderate anticoagulation should not delay the endoscopy.

How to manage patients with UGIB on aspirin or clopidrogel

Patients presenting with acute upper gastrointestinal bleeding whilst taking aspirin or clopidogrel present a clinical dilemma (see also Chapter 22, Acute Upper Non-variceal Hemorrhage). Stopping these drugs may reduce the risk of continuing bleeding but risks vascular events that can prove fatal. Both aspirin and clopidogrel bind irreversibly to platelet receptors, thereby impairing platelet function for 7–10 days. The rationale for discontinuing their use following the onset of bleeding is therefore ill founded, and for patients in whom there is a good indication for their use, it is wise to continue aspirin and clopidogrel despite upper gastrointestinal hemorrhage.

What is the role of proton pump inhibitors?

The use of proton pump inhibitors (PPI) before endoscopy in suspected non-variceal hemorrhage is controversial. The recently published NICE guidelines suggest that PPI should not be offered before endoscopy in patients with non-variceal bleeding [8]. However, in practice it is difficult to predict that patients do not have variceal bleeding, although with a reliable history and examination this accuracy may be improved.

One of the largest studies to evaluate the role of pre-endoscopy proton pump inhibitor therapy randomized 638 patients with UGIB to omeprazole or placebo before endoscopy. Patients randomized to omeprazole had significantly shorter lengths of stay, fewer actively bleeding ulcers (6% vs. 15%) and more ulcers with a clean base. There was no significant difference in the proportion needing surgery or with recurrent bleeding [12]. A Cochrane analysis of six randomized trials of pre-endoscopic PPI therapy found no significant difference between PPI groups and controls in mortality (OR1.12), rebleeding (OR 0.81), or surgery (OR 0.96). However, pre-endoscopic use of PPIs significantly reduced the number of patients with high risk stigmata (OR 0.67) and the need for endoscopic therapy (OR 0.68) compared with patients who received placebo or a histamine-2 receptor antagonist [13]. The 2010 international consensus guidelines on UGIB recommends the use of intravenous PPIs in all patient with high-risk lesions post-endoscopic therapy [14]. High-dose oral PPIs may be used in patients who do not have active bleeding or high risk stigmata for recurrent bleeding and therefore have a low risk for rebleeding.

The optimal prescribing of PPI therapy with dosing, route of administration, and the decision to prescribe prior to endoscopy remains controversial, and practice varies widely between units and countries.

Endoscopy for UGIB

Timing of endoscopy

It is recommended that all hospitals provide adequate resources for an endoscopy service that can provide interventional endoscopy within 24 hours after patient admission. In addition in high risk patients who present with unstable haemodynamic parameters and who present with UGIB, an urgent endoscopy should be performed when the patient has been resuscitated [8]. The benefit for early endoscopy (defined as endoscopy within 24 hours) was reported by a large retrospective study from the USA using a database of hospital inpatient admission (Nationwide Inpatient Sample) [15]. The study included 35 747 adults with acute variceal bleeding and 435 765 adults with non-variceal UGIB. Among patients with variceal hemorrhage, inpatient mortality was 8% for those who underwent upper endoscopy within one day of admission and was 15% who those who did not (OR 1.18). For patients with non-variceal bleeding the corresponding mortality rates were 3 and 7%, respectively (OR 1.32). Another study included 8222 patients with UGIB. In this study, patients who died had a significantly longer waiting time to endoscopy than those who survived (1.65 versus 0.95 days OR 1.10) [16].

Endoscopy for variceal and non-variceal UGIB will be discussed in Chapters 25 and 22, respectively.

What do in cases of rebleeding

In patients with rebleeding a second-look endoscopy is reasonable and has fewer complications than proceeding straight to surgery. However, if hemostasis is not achievable then these patients should be considered for interventional radiology, and if not available then emergency surgery. However, interventional radiology is not as widely available as emergency surgery and this should be delivered where local facilities and expertise exist; this approach was supported in the recently published NICE guidance [8]. Reduceed for surgery, and

had fewer complications without an increase in mortality [17][18].

Variceal hemorrhage

Prophylactic antibiotics

Multiple trials have demonstrated the effectiveness of prophylactic antibiotics in cirrhotic patients hospitalized for bleeding and suggest an overall decrease in the risk of infectious complications and mortality. Both the AASLD American Association for Study of Liver Diseases (AASLD) and NICE guidance have recommended antibiotic prophylaxis in any patient with liver cirrhosis and UGIB [8].

Intravenous vasopressin and its analogs

Intravenous vasopressin constricts mesenteric arterioles and decreases portal venous inflow, thereby reducing portal pressures. Terlipressin is a synthetic analogue of vasopressin (not available in the USA) and is widely used in many countries. A meta-analysis of patients prescribed terlipressin for variceal UGIB reported a significant reduction in all-cause mortality with terlipressin compared with placebo (RR 0.66) [19]. Terlipressin is usually administered at a dose of 2 mg every 4 hours and then reduced to 1 mg every 4 hours when haemostasis has been achieved, and the usual treatment period is over 72 hours.

Somatostatin inhibits the release of vasodilator hormones such as glucagon which results in decreased portal inflow and octeotride is a long acting analogue of somatostatin. Octeotride is usually administered as a 50 μg bolus followed by a continuous infusion of 50 μg per hour and is continued for 3–5 days. Somatostatin is more effective for controlling bleeding than placebo or vasopressin and has fewer side effects than vasopressin. Octeotride's effects are less well studied but remains the first line in the many centres in the USA for variceal haemorrhage.

Post-endoscopy management

Patients who have had UGIB in the presence of *Helicobacter pylori* should receive eradication therapy. A large systematic review demonstrated the benefit of *H. pylori* eradication on the rate of rebleeding was greater than antisecretory therapy [20].

If patients receive concomitant antiplatelet medications or NSAIDs the risks and need for therapy should be discussed with the patients and/or cardiologists (in the case of anti-platelets therapies if coronary stents are *in situ*). For patients with UGIB who require a NSAID, a PPI with a cyclo-oxygenase-2 inhibitor is preferred to reduce subsequent rebleeding. Patients with UGIB who require secondary cardiovascular prophylaxis should start receiving acetylsalicylic acid (ASA/aspirin) again as soon as cardiovascular risks outweigh gastrointestinal risks (usually within 7 days); ASA plus PPI therapy is preferred over clopidogrel alone to reduce rebleeding [14].

Summary

UGIB is a common presentation to EDs and in patients who are admitted to hospital. Patients who present to the ED with UGIB should be risk assessed with the GBS at presentation and the Rockall score after endoscopy.

In the initial resuscitation of patients it is important to avoid "over-transfusing" patients with blood products. Patient should receive endoscopy after resuscitation if severe acute upper gastrointestinal bleeding has occurred, and lower-risk patients should receive endoscopy within 24 hours after presentation. When required, therapeutic endoscopy for non-variceal UGIB a combination of techniques (clipping and/or thermocoagulation) is preferable to mono-therapy to achieve hemostasis.

Patients with suspected or confirmed variceal bleeding should receive antibiotics at presentation and those with high-risk/rebleeding after an episode of variceal bleeding should be considered for transjugular intrahepatic portosystemic shunt (TIPSS). After endoscopy in patients who have cardiovascular risk factors, aspirin and PPI therapy should be commenced when the risk of cardiovascular events are greater than the risk of rebleeding. If patients have had recent coronary intervention then discussion with cardiologists is advised and these patients may have a high risk of cardiovascular morbidity and mortality if the antiplatelet therapy is stopped abruptly.

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