HANDBOOK OF GASTROINTESTINAL EMERGENCIES

SECOND EDITION

Edited by
Gary Gitnick, M.D.

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Preface

The management of gastrointestinal emergencies has become both complex and controversial. Better understanding of the pathophysiology of gastrointestinal disease and development of new endoscopic techniques for diagnosis and treatment have refined our approaches to the management of emergency conditions. Because much of our understanding is still evolving and therapeutic modalities are in transition, controversy continues in several areas.

This book was written to aid both the primary care physician and emergency room physician in providing rapid diagnostic and therapeutic maneuvers in emergency situations. As all medical facilities do not have the most refined diagnostic or therapeutic techniques available, the authors have endeavored to provide the reader with approaches to be used with and without the availability of these techniques, equipment, or technical expertise.

Because this text is a clinical manual, its aim is to instruct the reader in clinically applicable diagnostic and therapeutic approaches. Material necessary for the delivery of clinically relevant instruction is included, as well as aspects of pathophysiology that are important to the delivery of emergency medical care.

The authors are subspecialists who possess clinical expertise and knowledge of the management of emergency situations. They were instructed to confine their chapters to the essentials of emergency medical care. The editor is indebted to the contributors for their dedication and the time they devoted to the development of their chapters.

The field of gastrointestinal emergencies is an exciting one: much of the material is new; many of the approaches are transitional. These factors make gastroenterology a stimulating subspecialty and an interesting topic on which to write.

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Gary Gitnick, M.D.

The patient with a gastrointestinal emergency may present with unique, unusual, and unexpected symptoms and may be critically ill. It is therefore essential that the physician responsible for the care of such a patient be well versed in the assessment and management of gastrointestinal conditions.

Regardless of the type of emergency, the management of serious gastrointestinal disease often requires thoughtful consideration and the rapid application of diagnostic and therapeutic techniques. Although in many institutions personnel are at a premium and one or two people must provide all of the medical care, it is often optimal to utilize a health care team when delivering emergency treatment.

Historical details are frequently essential in understanding the problem and expediting care, and they usually provide the key to proper treatment. In the emergency situation it is often impossible and inappropriate for multiple members of the team to elicit historical details; therefore, one member of the team should be assigned to obtain a history from the patient, the relatives, the pharmacist, and any witnesses to the problem. Another member of the team should assess the urgency of the situation; obtain the appropriate tubes, wires, and instruments; pass appropriate catheters, nasogastric

tubes, and endoscopic tubes; and be in charge of ordering and obtaining appropriate blood studies and urine specimens.

Physical examination remains an essential ingredient in the diagnosis of gastrointestinal disease. A complete physical examination of all organ systems is especially important, as conditions involving the gastrointestinal tract frequently manifest at other sites. Shortcuts must be avoided. A compulsive, orderly physical examination should be routine in the assessment of the patient with gastrointestinal disease and should be performed by at least one and preferably more than one member of the treatment team.

Follow-up care is strongly influenced by the treatment offered in the emergency situation. After the history, physical examination, differential diagnosis, and treatment plan are recorded, one member of the team should take responsibility for maintaining the patient record. It is essential that all diagnostic procedures and their results be recorded to avoid unnecessary duplication. It is also important that all treatments given, drugs and doses administered, and responses observed be recorded to facilitate the rapid extension of emergency care once the patient leaves the initial treatment area.

Ideally, the treatment team consists of one or more physicians, one or more nurses, the pharmacist, and an aide or orderly. The responsibilities of each member should be clearly outlined, and an acknowledged team leader, usually a physician, should be designated. In the management of gastrointestinal emergencies there should be only one leader who has the responsibility of coordinating the efforts of and assigning responsibilities to each of the team members. In addition to the responsibilities already mentioned, one of the team physicians must maintain the flow of repeated communication with the family of the patient.

If the approach outlined here is used, and if care is taken to follow the details of the history and physical examination, most gastrointestinal emergencies can be efficiently and effectively handled. The combination of patients with unique symptoms, complex diagnostic and therapeutic techniques, and individual responsibilities of various health care team members makes gastrointestinal emergency medicine an exciting and unique experience.

ESOPHAGEAL EMERGENCIES

Andrew F. Ippoliti, M.D.,
and Barry J. Zamost, M.D.

INTRODUCTION

The esophagus is not often considered a likely source for an acute, catastrophic event. In the differential diagnosis of chest pain, for instance, esophageal diseases may be forgotten amid the myriad possible cardiac and pulmonary disorders. Similarly, the evaluation of bleeding or perforation generally proceeds in retrograde fashion from the duodenum to the stomach and then to the esophagus. Admittedly, esophageal problems are more frequently chronic than acute, and the acute conditions are less common causes of chest pain than ischemic heart disease or pulmonary emboli. Prompt recognition and appropriate management, however, are just as important for the injured esophagus as for its more notorious neighbors. This chapter considers esophageal emergencies under the following headings: the bleeding patient, the patient with an esophageal obstruction, the patient with an esophageal perforation, and the patient with caustic ingestion. In each section the clinical presentation, differential diagnosis, diagnostic steps, and management are reviewed. In addition, each section contains a summary of the important diagnostic and treatment features and an outline of the approach to the problem.

THE BLEEDING PATIENT

There are three principal esophageal causes of upper gastrointestinal (UGI) bleeding: esophageal varices, Mallory-Weiss tears, and esophagitis. In the widely quoted study of UGI bleeding by Palmer, the relative percentages of bleeding due to varices, esophagitis, and tears were 18%, 7%, and 5%, respectively. Thus, nearly a third of UGI hemorrhages emanated from the esophagus; however, the frequency of bleeding from varices or esophagitis is related to the frequency of alcoholism in the patient population, and these proportions may vary from hospital to hospital. We discuss the clinical presentation, differential diagnosis, and diagnostic tests for the bleeding patient in general, and then the specific treatment for each condition will follow. Of necessity, there is overlap with the discussion of bleeding in the following chapter on stomach emergencies.

CLINICAL PRESENTATION

An acute UGI hemorrhage is usually indicated by hematemesis and/or melena, and less often by hematochezia, which is the evacuation of bright red blood from the rectum. The black discoloration of the blood results principally, but not exclusively, from digestion of heme by gastric acid and pepsin. Large quantities of blood act as a cathartic and may pass rapidly through the bowel, resulting in hematochezia rather than melena. The amount and duration of bleeding can influence the presentation but do not predict the source. In addition, a prior history of bleeding, particularly in an alcoholic, does not aid in the diagnosis of the current hemorrhage.

The presence or absence of associated symptoms, such as pain or vomiting, offers no clear insights into the cause of bleeding. Although chest or abdominal pain per se is not a feature of variceal or Mallory-Weiss tear bleeding, unrelated factors that may contribute to bleeding in these conditions may also be causes of pain. For instance, the social habits of many variceal bleeders leave much to be desired. Consequently, an alcoholic binge may precipitate bleeding and chest pain from esophagitis or abdominal pain from gastritis, duodenitis, pancreatitis, or, in some unfortunate souls, all three. Similarly, a Mallory-Weiss tear does not occur de novo. The retching associated with the tear may be due to

an infectious, toxic, inflammatory, or obstructive gastric lesion. The underlying lesion may cause epigastric pain. In short, although varices and tears do not cause pain there may be an associated condition that accounts for that symptom.

Retching or vomiting is an important feature of Mallory-Weiss tears. Most lacerations are located on the right posterior wall of the esophagus and are associated with hiatal hernia. In fact, most of the tears begin in the hernia sac. It has been suggested that the lesser curvature aspect of the esophagogastric junction may be more immobile, and the shearing forces that develop during forceful vomiting and retching may result in tears at that location. Whatever the mechanism, the important clinical point is that hematemesis may accompany the initial vomiting episode. There was no antecedent history of vomiting or retching in 25% to 50% of patients with Mallory-Weiss tears.

It is useful to obtain an accurate history of drug ingestion. Prior alcohol and aspirin intake is often found in patients bleeding from esophagitis or Mallory-Weiss tears. There is a theory that severe esophagitis precipitates variceal hemorrhage (the so-called erosion theory, to be contrasted with the explosion theory, which attributes bleeding to rupture of the varix).

There are no specific physical findings associated with esophagitis or tears. Patients with variceal bleeding should have signs of portal hypertension, such as splenomegaly and ascites, and the usual cause of portal hypertension is acute or chronic liver disease. Thus, although the patient may have hepatomegaly, jaundice, spider angiomata, palmar erythema, or other signs of disease, it has been well documented that a substantial proportion of patients with these physical findings are bleeding from nonvariceal sources.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

In addition to the three esophageal causes of UGI bleeding one must also consider duodenal or gastric ulcer, duodenitis and/or gastritis, angiodysplasia (usually of the stomach), and, rarely, carcinoma. As mentioned above, an antecedent history of bleeding is of no help in an alcoholic; however, the patient with a history of prior ulcer bleeding, if unoperated, has a reasonable likelihood of bleeding from another ulcer. There is no clinical information that predicts the site of

UGI bleeding with certainty. Fortunately, the general therapeutic maneuvers and the diagnostic approach treat all potential lesions equally. One can rely on firm rather than circumstantial evidence for the diagnosis.

MANAGEMENT: GENERAL SUPPORTIVE MEASURES AND DIAGNOSTIC TESTS

Blood Pressure (Supine and Upright) and Pulse Rate

A resting tachycardia, hypotension, or an orthostatic blood pressure fall in excess of 15 mm Hg may be due to volume depletion. An intravenous infusion of either normal saline or a colloid-containing solution should be promptly started and the pulse rate and blood pressure monitored.

Blood Work

Initial blood work should include hemoglobin, hematocrit, blood type and crossmatch, and an assessment of coagulation status by means of platelet count and prothrombin and partial thromboplastin times. Because of hemoconcentration, the initial hematocrit may be in the normal range despite substantial blood loss, and it should be remeasured after initial rehydration. Transfusion requirements include packed red cells or whole blood, and fresh-frozen plasma. Fresh-frozen plasma is indicated in patients with prolonged prothrombin and/or partial thromboplastin times and in patients requiring multiple transfusions, as stored blood is deficient in clotting factors. This can be corrected by administration of fresh-frozen plasma. Phytonadione (AquaMEPHYTON), 10 mg IM, should be given in an attempt to correct the prothrombin time; a single injection is sufficient, as failure to respond is a reflection of impaired hepatic function and not of vitamin K deficiency.

Gastric Intubation

All patients with hematemesis, melena, or hematochezia should have gastric lavage (see chapter on stomach emergencies). In the case of hematochezia it is critical to determine whether a UGI source is the cause of the brisk hemorrhage. Failure to consider that possibility will

delay diagnosis in about 15% of patients presenting with hematochezia.

The stomach should be intubated with a 16 F or 18 F tube via the nares or the mouth. (One millimeter is roughly three French units.) Another dimension worth remembering is that the distance from the incisors to the esophagogastric junction is 40 cm. Generally, the nasal route is better tolerated for these relatively small diameter tubes. Intubation is facilitated by lubricating the tube and by having the patient sip water during passage of the tube through the nasopharynx past the upper esophageal sphincter. In some patients, repetitive shallow breaths (panting) rather than drinking may ease tube passage. The tube should be inserted a distance of 50 to 60 cm, which would place the tip along the greater curvature of the antrum. Passing it further may produce tube coiling and direct the tip of the tube away from the dependent portion of the stomach. To confirm that the tube is correctly positioned, insert and then immediately aspirate 50 mL of saline; if the tube is in the dependent part of the stomach, at least 40 mL should be aspirated. The tube should be pulled out until adequate return is achieved.

Gastric aspiration both confirms the diagnosis of UGI bleeding and indicates its activity. An attempt should be made to clear the stomach of blood by repeated lavage with saline. The efficacy of iced saline is more folklore than fact. In the presence of brisk bleeding or large clots, the nasogastric tube may be inadequate. An Ewald or rectal tube (30 F) should be passed through the mouth to lavage the stomach adequately. The standard rectal tube has a single orifice at the distal tip. Several side holes are often added by an eager physician, but large side holes with jagged edges convert the tube into a weapon: Impressive mucosal erosions of an iatrogenic nature can be found at endoscopy. It is recommended that some caution be used in the creation of additional aspiration ports.

Gastric lavage has two purposes: First, the stomach is emptied to reduce the likelihood of vomiting and aspiration. Second, the stomach is cleared of blood and clots to facilitate diagnostic tests such as endoscopy and barium x-ray.

Identification of the Bleeding Lesion

When it is necessary to determine the source of a UGI hemorrhage on an emergent basis, endoscopy is the preferred method. Endoscopy provides three levels of information: First, the actual source of bleeding may be identified, provided that some but not too much bleeding is occurring at the time of the endoscopy. Second, when the endoscopy is performed after cessation of bleeding, potential bleeding sites can be detected. Third, when bleeding is too heavy to determine the actual lesion, the area of involvement (i.e., the esophagus, the stomach, or the duodenum) can be noted; this information is useful if surgery is required.

MANAGEMENT: SPECIFIC TREATMENT

Esophageal Varices

There are two additional nonoperative measures to control bleeding from esophageal varices: intravenous Pitressin (vasopressin, a posterior pituitary extract) and balloon tamponade.

Intravenous Pitressin. Pitressin, a potent vasoconstrictor that reduces portal pressure by decreasing splanchnic blood flow, is administered by direct infusion into the superior mesenteric artery or into a peripheral vein. Recent evidence indicates that its efficacy is comparable by either route. Since the peripheral infusion is more practical, it is recommended that Pitressin be given by continuous intravenous infusion, beginning at a rate of 0.3 U/min. If bleeding persists, the dose may gradually be increased to 0.9 U/min, each time for 30 to 60 minutes. Some authors suggest that, once bleeding stops, Pitressin be tapered rather than abruptly discontinued. One method is to decrease the dosage by thirds every 24 hours until a rate of 0.1 U/min is reached.

Side effects of Pitressin include bradycardia, hyponatremia, and phlebitis at the infusion site. As with any vasoconstrictive agent, there is a potential for cardiac or cerebral ischemia. In two trials of Pitressin for variceal bleeding there was no evidence of myocardial or cerebral infarction. The reported efficacy in controlling hemorrhage with intravenous Pitressin was 50% to 60%; survival, however, may not be improved.