Part III: Gastroenterological Disorders

Chapter 31: Acute Gastrointestinal Bleeding

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Acute bleeding from the gastrointestinal tract is a serious and potentially lethal condition. The bleeding may be upper gastrointestinal (including variceal) or lower gastrointestinal. Bleeding from peptic ulceration is continuous or recurrent in 20% of patients, with a 10% mortality. The risk increases with advanced age or with associated cardiovascular or malignant disease. Variceal bleeding contributes to the death of over one-third of all patients with cirrhosis, half of whom die as a consequence of their first bleed. Continuous, massive colonic bleeding in the elderly has a mortality rate of over 15%.

Upper Gastrointestinal Bleeding

Clinical Presentation

Haematemesis, bloody gastric aspirate or melaena, usually indicate bleeding from the upper gastrointestinal tract. The absence of blood in gastric aspirate does not eliminate significant bleeding. Haematemesis without melaena may occur with oesophageal lesions. Melaena infrequently originates from right sided colonic bleeding. Massive upper gastrointestinal bleeding often gives "maroon" melaena. Rarely, bright blood alone may be passed.

The patient's history may suggest a possible source of bleeding (ie, forceful vomiting in Mallory Weiss syndrome, or previous gastric surgery in stomal ulceration) but it is unreliable. Bleeding may occur without prior dyspepsia in 10% of patients with peptic ulceration. Salicylate ingestion is as likely to be associated with bleeding from chronic ulceration as from erosions. Upper gastrointestinal bleeding in cirrhotics has a non variceal origin in over 50% of cases.

Management - Non Variceal Bleeding

Once the history of bleeding is clearly established, the severity of hypovolaemia must be determined. This assessment and subsequent resuscitation have immediate priority.

1. Resuscitation

Blood is taken for cross-matching. Plasma expanders are given until whole blood is available. Vital signs (pulse, blood, pressure, respiration and temperature) are recorded together with hourly urine output. Baseline haematological and biochemical parameters are measured. Serial haematocrit may be useful in assessing blood requirements once haemodilution is complete.

Central venous pressure (CVP) measurements are a helpful guide to the rate and magnitude of volume replacement, particularly in the elderly. Pulmonary capillary wedge pressure measurements may be indicated.

Following adequate resuscitation, management is directed towards identifying the high risk patient and the high risk lesion, ie, those most likely to benefit from early surgical or endoscopic control of bleeding.

2. The High Risk Patient

The mortality from gastrointestinal bleeding is significantly increased where the bleeding is massive or recurrent (usually indicative of an arterial or variceal source) or where the patient's ability to tolerate hypovolaemic shock or major surgery is severely compromised (Table 1).

Table 1. Poor Prognostic Factors in Gastrointestinal Bleeding

Age greater than 60 years.
Serious associated disease - ie, ischaemic heart disease, respiratory or renal insufficiency, hepatic failure, malignancy, and coagulopathy.
Shock.
High transfusion requirement (greater than 5 units whole blood).
Recurrent haematemesis and melaena.

3. The High Risk Lesion

Diagnosis

(a) Endoscopy

Early endoscopic examination allows precise identification of the site and nature of the bleeding, and provides valuable prognostic information. It should not be carried out until the patient is adequately resuscitated. The use of an endoscope overtube facilitates the removal of gastric contents and minimizes the risk of aspiration. Massive bleeding infrequently prevents adequate examination and routine gastric lavage prior to endoscopy is unnecessary. The value of ised saline lavage in controlling bleeding is unproven.

Endoscopy performed within 24-48 hours of the bleeding episode reveals an actual or potential site of bleeding in the majority of patients - 15-30% of all lesions are actively bleeding at the time of endoscopy. Chronic ulcers may have signs of recent haemorrhage (overlying clot, an exposed artery or a solitary black spot). The endoscopic features indicate the likely outcome. Active arterial bleeding is frequently associated with continued or recurrent bleeding and, without intervention, has a high mortality. The rebleeding rates from ulcers with a visible vessel is 50-65%; with a black spot 8-10%; and without any signs of recent haemorrhage less than 2%. Ulcers on the posterior duodenal wall and high on the lesser curve of the stomach are almost twice as likely to rebleed as ulcers elsewhere.

Endoscopy may induce serious hypoxaemia in patients with significant cardiorespiratory disease. Continuous monitoring of oxygen saturation with a pulse oximeter, and administration of oxygen by nasal cannulae may be prudent.

(b) Radiology

(i) Barium meal

The standard barium meal examination is significantly less accurate than endoscopy in the diagnosis of upper gastrointestinal lesions. Superficial mucosal lesions (ie, erosions) can be demonstrated by double contrast studies, but the exact site or nature of bleeding cannot be determined. Barium studies should not be carried out if early endoscopy or angiography are anticipated.

(ii) Angiography

Mesenteric angiography may be of value if the cause of bleeding has not been established by endoscopy. Selective catheterization of the coeliac axis and superior mesenteric artery are required. A bleeding point is identified by contrast extravasation. This occurs only when the bleeding rate exceed 0.5-1.0 mL/min. This technique is of value for arterial bleeding from solitary lesions such as chronic ulcers or mucosal tears. It does not exclude bleeding from diffuse mucosal lesions or varices.

4. Treatment

(a) H2 Antagonists

The H2 receptor antagonists (cimetidine, ranitidine, and famotidine) facilitate ulcer healing by reducing gastric acid output, and should be commenced once the diagnosis is established. They do not prevent early ulcer rebleeding. Oral administration is satisfactory in most patients. The intravenous route should be reserved for those patients with frequent vomiting, impaired gastric emptying (ie, pyloric stenosis) or an ileus.

The suppression of gastric acid secretion in the critically ill patient by antacids or H2 receptor antagonists may prevent the development of subsequent bleeding from acute mucosal lesions (erosions or ulcers). Both should be given in a dose sufficient to maintain gastric pH above 4.0. If antacid is given by nasogastric tube, care must be taken to prevent mucosal suction trauma.

(b) Control of Bleeding

Bleeding from chronic ulcers ceases spontaneously in 80% of patients and does not recur. Early intervention for control of active bleeding or prevention of rebleeding is indicated in high risk lesions (ie, arterial bleeding) or in the high risk patient with a visible vessel.

(i) Surgery

Surgical treatment is usually reserved for continued or recurrent bleeding from chronic lesions. With exsanguinating haemorrhage it should not be delayed by fruitless attempts at diagnostic endoscopy. Acute lesions sucha as a Mallory Weiss tear may bleed massively but only rarely require surgical intervention. The type of surgery (vagotomy and partial gastrectomy) depends on the nature and site of the lesion and the surgeon's preference.

Surgery based on the conventional indications - massive bleeding, recurrent bleeding in hospital or complications such as perforation, still has a substantial mortality rate, particularly in the elderly and frail. The decision to operate should be made early in the high risk patient with endoscopic evidence of a high risk lesion. Non-operative methods of haemostasis utilizing endoscopy are available and should be considered initially in the treatment of all high risk patients.

(ii) Endoscopic Haemostasis

1. Thermal Methods

Electrocoagulation: monopolar and bipolar diathermy (BICAP) with endoscopic probes can induce haemostasis in most cases. Control of brisk arterial bleeding is more difficult. Local tissue damage is unpredictable and perforation has been reported. The injection of water through the tip of the electrode prevents tissue adherence, and may limit the extent of coagulation.

Heater probe: an aluminium, non-stick, Teflon-coated probe, with the tip heated by an inner coil has been developed for endoscopic use. Early clinical experience indicates that it can control arterial bleeding from peptic ulceration with a significant reduction in rebleeding.

Laser photocoagulation: laser light can be transmitted endoscopically through a quartz fibre and directed at a bleeding lesion. Both argon-ion and neodymium YAG lasers are suitable for this purpose and will effectively stop ulcer bleeding in 80-90% of cases. Rebleeding does occur but the incidence of perforation is less than 1%. Controlled trials indicate that laser photocoagulation can significantly reduce the incidence of arterial rebleeding from ulcers and the need for emergency surgery.

2. Chemical Methods

The injection of sclerosants (adrenaline and alcohol) directly into the base of an ulcer adjacent to the visible vessel can control arterial bleeding in over 90% of cases. It significantly reduces the risk of rebleeding and the need for emergency surgery. It is a simple, safe and effective technique which is now widely available.

(iii) Angiographic Haemostasis

1. Vasoconstrictor Infusion

The selective infusion of vasopresin into a bleeding splanchnic artery reduces blood flow and promotes blood clot formation. Initial control of bleeding may be achieved in many cases. The hazards are minimal with short-term infusions but the rebleeding rate is high.

2. Embolization

A bleeding vessel may be occluded by embolization with gel foam or other materials through the angiographic catheter. Super-selective catheterization of the bleeding artery is

essential anhd there is a risk of tissue infarction. This procedure should be reserved for those patients who pose an unacceptably high surgical risk (ie, blood dyscrasia, or recent myocardial infarction).

Management - Variceal Bleeding

The outcome of variceal bleeding depends on the severity of the haemorrhage and the degree of liver dysfunction. Adverse prognostic factors include active bleeding at endoscopy, a transfusion requirements of two litres or more, ascites, encephalopathy and impaired liver function tests (Child's grade C).

1. Resuscitation and Diagnosis

Resuscitation with whole blood should be initiated as soon as possible. The administration of fresh frozen plasma or prothrombin concentrates is indicated if a significant clotting abnormality is demonstrated. Measures to minimize encephalopathy (ie, colonic lavage, oral neomycin and lactulose) are introduced once the bleeding has been controlled. Confirmation of the exact site of bleeding should be established by early upper gastrointestinal endoscopy. The bleeding is from fundal varices in 10% of cases. An alternative, non variceal source of bleeding (ie, erosion or chronic ulceration) may be found in half the patients with oesophageal varices.

2. Treatment

(a) Variceal Sclerosis

At oesophagoscopy, a sclerosant such as ethanolamine or sodium tetradecyl sulphate, can be injected into the varix or the adjacent submucosa. This can be carried out under light sedation at the initial endoscopy. When the varix is actively bleeding, the technique is facilitated by simultaneous balloon compression of varices at the cardia (ie, with a Linton-Nachlas tube). Control of bleeding is achieved in 90% of patients. Repeated sclerosant injections at fortnightly intervals leads to progressive variceal obliteration, with a significant reduction in rebleeding and improved long-term survival. Retrosternal pain and fever are common immediately after sclerosis. Early mucosal ulceration and late oesophageal strictures may occur after treatment.

(b) Vasopressin Infusion

The administration of vasopressin lowers portal venous pressure by splanchnic arterial constriction. An intravenous infusion of 0.2-04 units/min temporarily controls variceal bleeding in 60% of cases. Rebleeding is frequent and intra-arterial infusions confer no therapeutic advantage. Side effects include hypertension, colic and intestinal hurry. Severe ischaemic necrosis of skin, heart and intestine may occur. These effects may be significantly reduced by the simultaneous administration of nitroglycerine. Triglycyl-lysine vasopressin, a slow release depot form of vasopressin, may be just as effective and have fewer side effects.

Other drugs such as propranolol and somatostatin lower portal pressure, but a useful role in the emergency treatment of variceal bleeding has not been established.

(c) Balloon Tamponade

Variceal bleeding can be controlled by balloon compression either at the cardia or within the oesophageal lumen. A number of devices are available for this purpose. The Linton-Nachlas tube has a single balloon which is inflated in the gastric fundus. The Sengstaken-Blakemore tube incorporates an additional oesophageal balloon. A modification, the four lumen Minnesota tube allows aspiration of gastric and oesophageal contents.

Tamponade tubes are difficult to introduce and require meticulous supervision while inflated. The balloons are checked for patency and leaks before insertion. Use of a stiffening wire aids the passage of the tube into the stomach. The gastric balloon is inflated with 150-200 mL of air and is drawn up to the cardia by gentle traction. The tube is firmly secured by taping to the face, and the oesophageal balloon is inflated with air to 30-40 mmHg (4.5-5.4 kPa) using a sphygmomanometer. The lumen is clamped and the position of the balloon checked radiologically.

Balloon tamponade achieves initial control of bleeding in most cases, but rebleeding is common. The oesophageal balloon is deflated after 24 hours and variceal sclerosis may then be possible. Tamponade is poorly tolerated and should be reserved for bleeding not controlled by sclerosis or vasopressin. Complications are common and its use should be confined to Intensive Care areas. Oesophageal rupture can be avoided by careful monitoring of balloon position and pressure. Continuous oesophageal suction reduces the risk of pulmonary aspiration. Mucosal ulceration is minimized by limiting the duration of tamponade to 24 hours.

(d) Transhepatic Variceal Obliteration

Varices can be outlined by percutaneous transhepatic portography. Selective catheterization of the left and short gastric veins enables variceal embolization with a preparation such as gel foam, sclerosants or acrylate glue. Initial hemostasis is achieved in 80% of cases but early and late rebleeding is common. Complications such as portal vein thrombosis are potentially lethal.

(e) Surgery

Emergency surgical procedures can effectively control variceal bleeding. The two procedures most commonly used are trans-oesophageal ligation and a porto-caval shunt. Oesophageal trans-section (using a stapling gun) by the abdominal approach has a low operative morality (10-20%) and a low rebleeding rate. The incidence of portosystemic encephalopathy after emergency porto-caval shunt is high and long-term survival is not improved. Selective decompression procedures (ie, Warren shunts) reduce the risk of encephalopathy but are technically more difficult and have a high operative mortality.

Lower Gastrointestinal Bleeding

Clinical Presentation

Massive haemorrhage from the colon invariably presents with bright rectal bleeding. Caecal and small intestinal bleeding may result in "maroon" melaena. If there is any doubt about the lower intestinal origin of bleeding (ie, bright rectal bleeding and melaena), colonic investigation should be preceded by upper gastrointestinal endoscopy. Massive bleeding with a normal blood urea is unlikely to be from the upper gastrointestinal tract.

The commonest causes of massive colonic bleeding in the elderly, ie, angiodysplasia, diverticular disease (both often occurring on the right side) and polyps, frequently have no preceding symptoms. Life-threatening haemorrhage is an uncommon event in the natural history of colonic carcinoma and inflammatory bowel disease.

Management

Initial management is again directed to resuscitation, followed by diagnosis of the source, and specific therapy, as indicated.

1. Diagnosis

(a) Endoscopy

Sigmoidoscopy following phosphate enemas is carried out initially to exclude a low colonic lesion. If this is unrevealing, fibreoptic colonoscopy to the caecun is often possible. The use of intestinal saline lavage, mannitol or "Golytely" allows rapid preparation of the bowel. Dilute blood in the lumen is easily aspirated and visualization is usually adequate. An actual or potential bleeding site can be identified in over 60% of cases. Bleeding from vascular anomalies (ie, angiodysplasia), polyps, carcinoma or inflammatory bowel disease is easily recognized. An adherent clot or an exposed vessel may pinpoint diverticular bleeding.

(b) Isotope Scanning

If the source of bleeding is not demonstrated at colonoscopy, an abdominal scintiscan is carried out after the intravenous administration of radio-labelled technetium. Extravasated isotope can be recognized with a bleeding rate as low as 0.1 mL/min. A non-colonic site of bleeding may be suggested. Specific isotope uptake by parietal cells may outline a Meckel's diverticulum.

(c) Angiography

Selective mesenteric angiography is successful in identifying the source of massive lower gastrointestinal bleeding in up to 60% of cases. Bleeding can be detected at a rate of 0.5 mL/min or more. Failure is usually due to cessation of bleeding. Angiography is unlikely to be successful if the isotope scan is negative. Bleeding from a diverticulum gives rise to pooling of extravasated contrast. Angiodysplasia is recognized by an abnormal vascular

pattern. If bleeding has ceased, investigation should start with colonoscopy, and be followed by a barium enema and small bowel series before considering angiography.

2. Treatment

Spontaneous cessation of bleeding from all sources is common. However, bleeding from angiodysplasic lesions tend to be recurrent.

Methods for controlling bleeding include:

(a) Endoscopy

Bleeding colonic polyps can be readily removed by polypectomy. Angiodysplasic lesions can be coagulated using diathermy, laser photocoagulation or alcohol sclerosis with a low risk of rebleeding.

(b) Angiography

Low dose infusion of vasopressin (0.1-0.2 units/min) into the inferior mesenteric artery will effectively stop both diverticular and angiodysplasic bleeding in over 7-% of cases.

(c) Surgery

Early surgery is the preferred treatment for massive lower gastrointestinal bleeding due to colonic carcinoma, colitis, Meckel's diverticulum, or small bowel tumours.

Emergency subtotal colectomy for bleeding diverticular disease or angiodysplasia has a significant mortality in the elderly. It should be reserved for continuing or recurrent haemorrhage not controlled by more conservative methods. Since bleeding is not uncommon from right-sided diverticula, the success of segmental colectomy depends on accurate endoscopic or angiographic diagnosis. Right hemicolectomy for angiodysplasic lesions can often be carried out as an elective procedure. Subtotal colectomy may be necessary when massive colonic bleeding continues in spite of normal mesenteric angiography.