Diagnostic approach, pathophysiology, clinical consequences and treatment options of acute upper gastrointestinal bleeding.

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Introduction

Haematemesis and/or melaena are common symptoms of acute upper gastrointestinal bleeding (UGIB). Brighter rectal bleeding may be coupled with massive upper GI tract haemorrhage. A common medical emergency is upper gastrointestinal bleeding (UGIB). Patients who have been assessed as having a low risk of requiring an intervention or dying can be treated as outpatients. Intravenous fluids as needed for resuscitation and red cell transfusion at a haemoglobin threshold of 70-80 g/L are indicated for all other patients. Proton pump inhibitors (PPIs) and the prokinetic agent erythromycin may be given after resuscitation, with antibiotics and vasoactive medicines advised in cirrhotic patients. Gastrointestinal endoscopy should be performed within 24 hours of resuscitation, with earlier endoscopy recommended in high-risk patients, including those with hemodynamic instability.

Diagnostic approach

Preendoscopic management: The group recommends using a Glasgow Blatchford score of 1 or less to identify patients who are at very low risk of rebleeding and may not need to be admitted to the hospital. The recommended haemoglobin threshold for blood transfusion in people without cardiovascular disease is less than 80 g/L, with a higher barrier for those with cardiovascular illness. Patients with acute UGIB should have an endoscopy within 24 hours of presenting, according to the group. Endoscopic therapy for patients with high-risk stigmata should include thermocoagulation and sclerosant injection, as well as clips. In patients with actively bleeding ulcers, TC-325 (hemostatic powder) was recommended as a temporary treatment, but not as the only option [1].

Patients with bleeding ulcers with high-risk stigmata who have had successful endoscopic therapy should get high-dose proton-pump inhibitor (PPI) therapy (intravenous loading dose followed by continuous infusion) for 3 days, according to the researchers. Continued oral PPI therapy is recommended for these high-risk individuals twice daily for 14 days, then once daily for a total length that depends on the type of the bleeding lesion. Secondary prophylaxis: The group recommends PPI medication for patients who require antiplatelet or anticoagulant therapy for cardiovascular prophylaxis due to past ulcer bleeding [1].

Pathophysiology of feeding

Patients with upper gastrointestinal bleeding are commonly regarded to need to be fasted in a systematic manner. Oral and/or enteral feeding in patients with or at risk of upper gastrointestinal haemorrhage will be the focus of this evaluation. An endoscopy is always required in the case of upper gastrointestinal bleeding to diagnose the pathophysiology of the bleeding and, in some cases, to perform an endoscopic therapy. Enteral feeding is the most effective stress ulcer prevention in ICU patients. Concurrent use of histamine-2 receptor blockers or proton-pump inhibitors in individuals receiving enteral feeding may be hazardous. Enteral feeding can be restarted as soon as the patient tolerates it after bleeding due to stomach erosions. Nonbleeding oesophageal varices are not a contraindication for enteral feeding or a nasogastric tube in individuals with liver cirrhosis. It is recommended that patients who are admitted to the hospital with acute upper gastrointestinal bleeding due to an ulcer with a high risk of rebleeding (Forrest I-IIb) or variceal bleeding wait at least 48 hours after endoscopic therapy before starting oral or enteral feeding. There is no need to delay refeeding in patients with gastritis, Mallory-Weiss, oesophagitis, or angiodysplasia who have a low risk of rebleeding (Forrest IIc and III) or who have gastritis, Mallory-Weiss, oesophagitis, or angiodysplasia [2].

Clinical consequences

Protein digestion, as well as the absorption of iron, calcium, vitamin B(12), and several medicines, are aided by gastric acid (e.g. thyroxin). It also kills ingested bacteria and protects against bacterial overgrowth, intestinal illness, and maybe spontaneous bacterial peritonitis. Histamine, gastrin, acetylcholine, and ghrelin are all acid secretion stimulants. Somatostatin, nefstatin-1, interleukin-11, and calcitonin gene-related peptide are all inhibitors. Depending on the time course of infection and the location of the stomach that is primarily infected, Helicobacter pylori stimulates or inhibits acid secretion. Acute infection activates calcitonin gene-related peptide sensory neurons, inhibiting histamine and acid production at the same time. In patients using proton pump inhibitors, serum chromogranin A, a marker for neuroendocrine tumours, is raised [3].

Treatment options of acute upper gastrointestinal bleeding

Upper gastrointestinal (GI) bleeding is still a prevalent

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diagnosis for emergency room surgeons. Patients must be stabilised and resuscitated as soon as possible. Patients who are stable can begin medicinal therapy and have their bleeding localised, whereas those who are continuously unstable require urgent endoscopic or surgical surgery. For most upper GI bleeding, minimally invasive treatments have surpassed surgery as the treatment of choice [4].

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