Acute Non-Variceal Upper GI Bleeding

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Objectives of this talk

- Learn the **etiology and pathogenesis** of non-variceal upper gastrointestinal bleeding (UGIB)
- Learn the current **management of UGIB: non-variceal**
- Learn the **prognostic indicators of UGIB: non-variceal**
Definitions

- **Acute** GI bleed
  - < 3 days duration
  - hemodynamic instability
  - requires blood transfusion

- Overt vs occult
  - overt = visible blood (melena, bright red blood, burgundy colored stool, coffee grounds)
  - occult = only detected by lab tests (FOBT, FIT)
Upper vs. Lower GI bleed

- UGIB = proximal to ligament of Treitz
- LGIB = distal to ligament of Treitz
Epidemiology

- Upper GI bleed: non-variceal (UGIB-NV)
  - 100,000 admissions/year to US hospitals
  - 10% mortality

- Upper GI bleed: variceal (UGIB-V)
  - 30% of identified varices will bleed in 1 year
  - 33% mortality with each bleed

- Lower GI bleed (LGIB)
  - Less common than UGIB
  - 3% mortality

Key Point: Mortality
LGIB < UGIB < Variceal bleeds
Upper GI Bleeding - Epidemiology

- Incidence 150/100,000 population per year
- Increases 20-30 folds from 3rd to 9th decade of life
- Stops spontaneously in 80% of cases
- Overall mortality 5-15% in those admitted to hospital (depending on severity)
- Mortality increases in case of re-bleed
- In 2% of cases surgery is needed to arrest bleeding
- Mortality 30% in the elderly
Trend in Peptic Ulcer Disease Epidemiology

Chapter 32, Textbook of Clinical Gastroenterology and Hepatology by Hawkey CJ, Bosch JE, Richter J, Garcia-Tsao G, and Chan FKL
Time Trends in Upper GI Bleeding

Minimum Basic Data Set of 30,498 patients from 10 Spanish General Hospitals from 1996-2005

Lanas A and Garcia S et al; Am J Gastro, 2009
Etiology of Upper GI Bleeding (UGIB)

Causes of UGIB

**Major causes**
- Peptic ulcer disease
- Esophageal and gastric varices
- Hemorrhagic gastritis
- Esophagitis
- Duodenitis
- Mallory-Weiss tear
- Angiodysplasia
- Upper gastrointestinal malignancy
- Anastomotic ulcers (after PUD surgery or bariatric surgery)
- Dieulafoy lesion

**Minor causes**
- Cameron lesion
- Gastric antral vascular ectasia (watermelon stomach)
- Portal hypertensive gastropathy
- Post chemotherapy or radiation sequelae
- Gastric polyps
- Aortoenteric fistula
- Submucosal lesion/mass (e.g., leiomyoma)
- Connective tissue disease
- Hemobilia
- Hemosuccus pancreaticus
- Kaposi sarcoma
- Foreign bodies
- Postprocedural: nasogastric tube erosions, endoscopic biopsy, endoscopic polypectomy, EMR, endoscopic sphincterotomy

**Abbreviations**: EMR, endoscopic mucosal resection; PUD, peptic ulcer disease.
Etiology of Upper GI Bleeding (UGIB): Changing Epidemiology

Peptic Ulcer Disease
- Gastric ulcer: 21%
- Varices: 13%
- Mallory-Weiss tear: 4%
- Esophagitis: 12%
- Erosive duodenitis: 10%
- Neoplasm: 10%
- Miscellaneous: 3%
- Mucosal erosive disease: 3%
- Duodenal ulcers: 3%
- Gastric erosions: 3%

Varices
- No diagnosis or >1 type of lesion: 39%
- Ulcers: 33%
- Gastroesophageal varices: 17%
- Other: 11%

Boonpongmanee S, Fleischer DE, and Benjamin SB et al; GI Endoscopy, 2004
Jutabha R and Jensen D; UpToDate, 2013
<table>
<thead>
<tr>
<th>Bleeding etiology</th>
<th>Historical clues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mallory-Weiss tear</td>
<td>Emesis before hematemesis, alcoholism</td>
</tr>
<tr>
<td>Esophageal ulcer</td>
<td>Odynophagia, GERD, esophagotoxic pill ingestion</td>
</tr>
<tr>
<td>Peptic ulcer</td>
<td>Epigastric/RUQ pain, NSAID or aspirin use</td>
</tr>
<tr>
<td>Stress gastritis</td>
<td>Patient in an ICU, gastrointestinal bleeding occurring after admission, respiratory failure, multiorgan failure</td>
</tr>
<tr>
<td>Varices, portal gastropathy</td>
<td>Alcoholism, cirrhosis</td>
</tr>
<tr>
<td>Gastric antral vascular ectasia</td>
<td>Renal failure, cirrhosis</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Recent involuntary weight loss, dysphagia, cachexia, early satiety</td>
</tr>
<tr>
<td>Angiodysplasia</td>
<td>Chronic renal failure, hereditary hemorrhagic telangiectasia</td>
</tr>
<tr>
<td>Aortoenteric fistula</td>
<td>Known aortic aneurysm, prior abdominal aortic aneurysm repair</td>
</tr>
</tbody>
</table>

Abbreviations: GERD, gastroesophageal reflux disease; NSAID, nonsteroidal anti-inflammatory drug; RUQ, right upper quadrant.
Pathogenesis of UGIB: Non-Variceal

<table>
<thead>
<tr>
<th>Cause</th>
<th>Comments</th>
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</thead>
<tbody>
<tr>
<td>Common</td>
<td></td>
</tr>
<tr>
<td><em>H. pylori</em> infection</td>
<td>Found in 48% of patients with PUD</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>5–20% of patients who use NSAIDs over an extended period develop PUD</td>
</tr>
<tr>
<td>Other medications</td>
<td>Corticosteroids, bisphosphonates, potassium chloride, chemotherapeutic agents</td>
</tr>
<tr>
<td>Rare</td>
<td></td>
</tr>
<tr>
<td>Zollinger–Ellison syndrome</td>
<td>Hypersecretory state causing multiple gastroduodenal, jejunal, or esophageal ulcers</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Gastric cancer, lymphoma, lung cancer</td>
</tr>
<tr>
<td>Stress</td>
<td>After acute illness, multi-organ failure, ventilator support, extensive burns (Curling’s ulcer), or head injury (Cushing’s ulcer)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type</th>
<th>Location</th>
<th>Acid hypersecretion</th>
<th>Complications</th>
<th>Incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Gastric body, lesser curvature</td>
<td>No</td>
<td>Bleeding uncommon</td>
<td>55</td>
</tr>
<tr>
<td>II</td>
<td>Body of stomach + duodenal ulcer</td>
<td>Yes</td>
<td>Bleeding, perforation, obstruction</td>
<td>20</td>
</tr>
<tr>
<td>III</td>
<td>Prepyloric</td>
<td>Yes</td>
<td>Bleeding, perforation</td>
<td>20</td>
</tr>
<tr>
<td>IV</td>
<td>High on lesser curvature</td>
<td>No</td>
<td>Bleeding</td>
<td>&lt;5</td>
</tr>
<tr>
<td>V</td>
<td>Anywhere (medication induced)</td>
<td>No</td>
<td>Bleeding, perforation</td>
<td>&lt;5</td>
</tr>
</tbody>
</table>
Pathogenesis of UGIB: H. pylori

Duodenal Ulcer

H. pylori-induced central predominant gastritis

Inflammation

↓Somatostatin ↑Gastrin

↑Acid

↑Histamine

Gastric Ulcer

H. pylori-induced pan gastritis

↓Acid

↓Histamine

Inflammation

↑Gastrin

↓Somatostatin

D cell
G cell
ECL cell
Parietal cell

Chapter 32, Textbook of Clinical Gastroenterology and Hepatology by Hawkey CJ, Bosch JE, Richter J, Garcia-Tsao G, and Chan FKL
Pathogenesis of UGIB: NSAIDs

deFonesca A and Kaunitz JD; Current Opinion in Gastroenterology, 2010
Akiba Y and Kaunitz JD; APT, 2006
Management of UGIB: Non-Variceal

Goals of in-hospital care

- Resuscitation, risk assessment, and pre-endoscopy management
- Endoscopic management
- Pharmacologic management
- Treat underlying cause (in-hospital care)
- Prevent recurrence (post-discharge ASA and NSAIDs)
Resuscitation - I

- Initiate ABC’s of Emergency Care
- Establish IV access:
  - 2 large bores (ideally at least 18-gauge peripheral IVs)
  - in MICU, may place triple-lumen or Cordis catheter
- Replace intravascular volume
  - if hypotensive and/or orthostatic, give NS boluses
  - if anemic (Hb ≤7 g/dL), give PRBCs
  - may need FFP (for coagulopathy) and/or platelets (for thrombocytopenia/<50K or dysfunction from chronic antiplatelet agents usage) if massive GI bleed
In summary, we found that a restrictive transfusion strategy, as compared with a liberal transfusion strategy, improved the outcomes among patients with acute upper gastrointestinal bleeding. The risk of further bleeding, the need for rescue therapy, and the rate of complications were all significantly reduced, and the rate of survival was increased, with the restrictive transfusion strategy. Our results suggest that in patients with acute gastrointestinal bleeding, a strategy of not performing transfusion until the hemoglobin concentration falls below 7 g per deciliter is a safe and effective approach.
Resuscitation - III

Key Points

Keep Hb ≤7 g/dL for low-risk patients
Keep Hb ≤10 g/dL for high-risk patients

Villanueva C and Guarner C et al; NEJM, 2013
## Table 3. Study Outcomes.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Restrictive Strategy (N = 444)</th>
<th>Liberal Strategy (N = 445)</th>
<th>Hazard Ratio with Restrictive Strategy (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death from any cause within 45 days — no. (%)</td>
<td>23 (5)</td>
<td>41 (9)</td>
<td>0.55 (0.33–0.92)</td>
<td>0.02</td>
</tr>
<tr>
<td>Further bleeding — no. of patients/total no. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>45/444 (10)</td>
<td>71/445 (16)</td>
<td>0.62 (0.43–0.91)</td>
<td>0.01</td>
</tr>
<tr>
<td>Patients with cirrhosis</td>
<td>16/139 (12)</td>
<td>31/138 (22)</td>
<td>0.49 (0.27–0.90)</td>
<td>0.02</td>
</tr>
<tr>
<td>Child–Pugh class A or B</td>
<td>12/113 (11)</td>
<td>23/109 (21)</td>
<td>0.53 (0.27–0.94)</td>
<td>0.04</td>
</tr>
<tr>
<td>Child–Pugh class C</td>
<td>4/26 (15)</td>
<td>8/29 (28)</td>
<td>0.58 (0.15–1.95)</td>
<td>0.33</td>
</tr>
<tr>
<td>Bleeding from esophageal varices</td>
<td>10/93 (11)</td>
<td>21/97 (22)</td>
<td>0.50 (0.23–0.99)</td>
<td>0.05</td>
</tr>
<tr>
<td>Rescue therapies</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Balloon tamponade</td>
<td>3/139 (2)</td>
<td>11/138 (8)</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>TIPS</td>
<td>6/139 (4)</td>
<td>15/138 (11)</td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>Patients with bleeding from peptic ulcer</td>
<td>23/228 (10)</td>
<td>33/209 (16)</td>
<td>0.63 (0.37–1.07)</td>
<td>0.09</td>
</tr>
<tr>
<td>Rescue therapies</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second endoscopic therapy</td>
<td>20/228 (9)</td>
<td>26/209 (12)</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>Emergency surgery</td>
<td>4/228 (2)</td>
<td>12/209 (6)</td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>No. of days in hospital</td>
<td>9.6±8.7</td>
<td>11.5±12.8</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Adverse events — no. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any†</td>
<td>179 (40)</td>
<td>214 (48)</td>
<td>0.73 (0.56–0.95)</td>
<td>0.02</td>
</tr>
<tr>
<td>Transfusion reactions</td>
<td>14 (3)</td>
<td>38 (9)</td>
<td>0.35 (0.19–0.65)</td>
<td>0.001</td>
</tr>
<tr>
<td>Fever</td>
<td>12 (3)</td>
<td>16 (4)</td>
<td>0.74 (0.35–1.59)</td>
<td>0.56</td>
</tr>
<tr>
<td>Transfusion-associated circulatory overload</td>
<td>2 (&lt;1)</td>
<td>16 (4)</td>
<td>0.06 (0.01–0.45)</td>
<td>0.001</td>
</tr>
<tr>
<td>Allergic reactions</td>
<td>1 (&lt;1)</td>
<td>6 (1)</td>
<td>0.16 (0.02–1.37)</td>
<td>0.12</td>
</tr>
<tr>
<td>Cardiac complications‡</td>
<td>49 (11)</td>
<td>70 (16)</td>
<td>0.64 (0.43–0.97)</td>
<td>0.04</td>
</tr>
<tr>
<td>Acute coronary syndrome‡</td>
<td>8 (2)</td>
<td>13 (3)</td>
<td>0.61 (0.25–0.49)</td>
<td>0.27</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>12 (3)</td>
<td>21 (5)</td>
<td>0.56 (0.27–1.12)</td>
<td>0.07</td>
</tr>
<tr>
<td>Pulmonary complications</td>
<td>48 (11)</td>
<td>53 (12)</td>
<td>0.89 (0.59–1.36)</td>
<td>0.67</td>
</tr>
<tr>
<td>Acute kidney injury</td>
<td>78 (18)</td>
<td>97 (22)</td>
<td>0.78 (0.56–1.08)</td>
<td>0.13</td>
</tr>
<tr>
<td>Stroke or transient ischemic attack</td>
<td>3 (1)</td>
<td>6 (1)</td>
<td>0.49 (0.12–2.01)</td>
<td>0.33</td>
</tr>
<tr>
<td>Bacterial infections</td>
<td>119 (27)</td>
<td>135 (30)</td>
<td>0.87 (0.63–1.21)</td>
<td>0.41</td>
</tr>
</tbody>
</table>
Nasogastric intubation and NG lavage

No role of occult blood testing of NG aspirate

Interpretation of aspirate:
- bright red, clots = active UGIB
- coffee grounds = slow bleeding, may have stopped, localizes to upper GI source
- clear = indeterminate (NOT a guarantee that the bleeding has stopped); ~18% of patients with UGIB source
- bilious = bleeding has stopped; ~18% of patients with UGIB source

Contraindications
- Facial trauma, nasal bone fracture
- Known esophageal abnormalities (strictures, diverticuli)
- Ingestion of caustic substances, esophageal burns
- In general, esophageal varices are NOT a contraindication to NG tube placement
Pre-endoscopy Management - II

- IV Erythromycin 250 mg bolus 30-60 min before EGD
- IV Reglan 10 mg bolus 60 min before EGD (if erythromycin not available)
- Initiate PPI drip: 80 mg bolus followed by 8 mg/h infusion
- No role for H₂-receptor antagonists
- Initiate Octreotide drip (if suspecting variceal bleeding): 50 µg bolus followed by 50 µg/h infusion
- Initiate Somatostatin drip (if octreotide not available): 250 µg bolus followed by 500 µg/h infusion
- Consider EGD within 6-12 h (or at least before 24 h)
## Glasgow-Blatchford Score Assessment Criteria

<table>
<thead>
<tr>
<th>Risk factors at presentation</th>
<th>Threshold</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood urea nitrogen (mmol/l)</td>
<td>6.5–7.9</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>8.0–9.9</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>10.0–24.9</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>≥25.0</td>
<td>6</td>
</tr>
<tr>
<td>Hemoglobin for men (g/l)</td>
<td>120–130</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>100–119</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>&lt;100</td>
<td>6</td>
</tr>
<tr>
<td>Hemoglobin for women (g/l)</td>
<td>100–120</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&lt;100</td>
<td>6</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>100–109</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>90–99</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>&lt;90</td>
<td>3</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>&gt;100</td>
<td>1</td>
</tr>
<tr>
<td>Melena</td>
<td>Present</td>
<td>1</td>
</tr>
<tr>
<td>Syncope</td>
<td>Present</td>
<td>2</td>
</tr>
<tr>
<td>Hepatic disease</td>
<td>Present</td>
<td>2</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>Present</td>
<td>2</td>
</tr>
</tbody>
</table>

Total Score = 0-23. Patients with scores > 0 are at higher risk.

Blatchford O et al; Lancet, 2000
Risk Assessment - II

**Complete Rockall Score for UGIB**

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>0 POINTS</th>
<th>1 POINT</th>
<th>2 POINTS</th>
<th>3 POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (yr)</strong></td>
<td>&lt;60</td>
<td>60-79</td>
<td>&gt;80</td>
<td>—</td>
</tr>
<tr>
<td><strong>Shock</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse rate (beats/min)</td>
<td>&lt;100</td>
<td>&gt;100</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>&gt;100</td>
<td>&gt;100</td>
<td>&lt;100</td>
<td>—</td>
</tr>
<tr>
<td><strong>Comorbidity</strong></td>
<td>None</td>
<td>—</td>
<td>Ischemic heart disease, cardiac failure, other major illness</td>
<td>Renal failure, hepatic failure, metastatic cancer</td>
</tr>
<tr>
<td><strong>Endoscopic stigmata of recent hemorrhage</strong></td>
<td>No stigmata or dark spot in ulcer base</td>
<td>—</td>
<td>Blood in upper GI tract, adherent clot, visible vessel, active bleeding</td>
<td>—</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Mallory-Weiss tear or no lesion seen</td>
<td>All other diagnoses</td>
<td>Malignant lesions</td>
<td>—</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PRE-ENDOSCOPY SCORE</th>
<th>MORTALITY (%)</th>
<th>POSTENDOSCOPY SCORE</th>
<th>REBLEED RATE (%)</th>
<th>MORTALITY (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.2</td>
<td>0</td>
<td>4.9</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>2.4</td>
<td>1</td>
<td>3.4</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>5.6</td>
<td>2</td>
<td>5.3</td>
<td>0.2</td>
</tr>
<tr>
<td>3</td>
<td>11</td>
<td>3</td>
<td>11.2</td>
<td>2.9</td>
</tr>
<tr>
<td>4</td>
<td>24.6</td>
<td>4</td>
<td>14.1</td>
<td>5.3</td>
</tr>
<tr>
<td>5</td>
<td>39.6</td>
<td>5</td>
<td>24.1</td>
<td>10.8</td>
</tr>
<tr>
<td>6</td>
<td>48.9</td>
<td>6</td>
<td>32.9</td>
<td>17.3</td>
</tr>
<tr>
<td>7</td>
<td>50</td>
<td>7</td>
<td>43.8</td>
<td>27</td>
</tr>
<tr>
<td>—</td>
<td>—</td>
<td>8+</td>
<td>41.8</td>
<td>41.1</td>
</tr>
</tbody>
</table>

Total Score = 0-11. Based on score, patients are divided into 3 categories:

a) Low-risk: ≤2
b) Moderate-risk: 3-5
c) High-risk: ≥6

Rockall TA et al; Gut, 1996
ALIMS65 bed-side score

- Albumin <3 g/dL
- INR >1.5
- Altered Mental Status (GCS <14)
- SBP <90 mm of Hg
- Age >65 years

Total Score = 0-5. Patient with score of 0 has 0.3 % mortality vs score of 5 has 31.8% mortality.

Zero risk factors: 0.3%
One risk factor: 1%
Two risk factors: 3%
Three risk factors: 9%
Four risk factors: 15%
Five risk factors: 32%

Saltzman JR and Johannes RS et al; GIE, 2011
Initial Assessment - I

- Age
  - risk, mortality increase with age
- Previous bleeding
- Comorbidities
  - CAD
  - heart failure
  - AAA repair
  - Cirrhosis
- Previous endoscopies (look at reports!)
- Associated symptoms
  - pain
  - retching
  - anorexia, weight loss
  - nausea/vomiting
  - early satiety
  - dysphagia
  - epistaxis, hemoptysis
- Medication history – NSAIDs, warfarin, ASA, Plavix
Symptoms and Signs

**Upper GI Bleed**

- Lightheadedness/Syncope
- Diarrhea
- Anemia
- Hematemesis
- Melena
- Stigmata of cirrhosis
- Heartburn
Physical Exam

- Vital signs
- Dry mucus membranes
- Stigmata of cirrhosis
- Fetid breath
- Digital rectal exam (DRE)
- Weak pulses
- Cool skin
- Encephalopathy
Diagnostic Testing

- EGD
- Enteroscopy: Push, Single- or Double-balloon
- Capsule Endoscopy
- Angiogram
- Tagged red blood cell scans
Acute UGIB: Differential Diagnosis

- Peptic ulcer disease
  - Gastric ulcer
  - Duodenal ulcer

- Mallory-Weiss tear

- Portal hypertension
  - Esophagogastric varices
  - Gastropathy

- Esophagitis

- Dieulafoy’s lesion

- Vascular anomalies

- Hemobilia

- Hemorrhagic gastropathy

- Aortoenteric fistula

- Neoplasms
  - Gastric cancer
  - Kaposi’s sarcoma
Erosive Esophagitis
Reflux Esophagitis and Barrett’s Metaplasia

Developed by the International Working Group for the Classification of Reflux Oesophagitis (IWGCO)

**LA Grade A**
One (or more) mucosal break no longer than 5mm, that does not extend between the tops of two mucosal folds

**LA Grade B**
One (or more) mucosal break more than 5 mm long, that does not extend between the tops of two mucosal folds

**LA Grade C**
One (or more) mucosal break that is continuous between the tops of two or more mucosal folds, but which involves less than 75% of the circumference

**LA Grade D**
One (or more) mucosal break which involves at least 75% of the esophageal circumference

Developed by the Barrett’s Oesophagus Subgroup of the International Working Group for the Classification of Reflux Oesophagitis (IWGCO)

1. **Ensure Hiatus Hernia**
   - Is recognised by distinguishing diaphragmatic hiatal impression from gastroesophageal junction

2. **Locate Gastroesophageal Junction**
   - By depth of endoscope insertion at level of:
     - Tops of gastric mucosal folds
     - Sphincter “pinch”
     - = 36 cm

3. **Look for Displacement of Squamocolumnar Junction Above Gastroesophageal Junction**

4. **Measure Depth of Endoscope Insertion**
   - At the most proximal circumferential extent of suspected columnar metaplasia
   - = 29 cm

5. **Measure Depth of Endoscope Insertion**
   - At the maximum extent of suspected columnar metaplasia
   - = 33 cm

6. **Subtract the Depth of Insertion for Circumferential and Maximum Extents from the Depth of Endoscope Insertion at the Gastroesophageal Junction**
   - 36 cm - 29 cm = C3
   - 36 cm - 33 cm = C7
   - Prague C3 and M7

Lundell L et al; Gut, 1999

Sharma P et al; Gastroenterology, 2006

* To the nearest centimeter
* Squamous and columnar islands do NOT contribute to measures of extent
* To the nearest centimeter, except when areas of columnar metaplasia are estimated to be less than 1 cm report this as < 1 cm
Mallory-Weiss Tears and Cameron’s Lesions
Upper GI Cancers: Esophageal and Gastric

Esophageal Adenocarcinoma

Gastric Adenocarcinoma
Other Causes of UGIB

- Vascular Ectasia
- Hemobilia
- Dieulafoy’s lesions
Forrest Classification in UGIB

A  Spurting Blood

B  Oozing Blood

Grade IA  Grade IB

Gralnek IM et al; NEJM, 2008
Forrest Classification in UGIB

C  Nonbleeding Visible Vessel

D  Adherent Clot

Grade IIA

Grade IIB

Gralnek IM et al; NEJM, 2008
Forrest Classification in UGIB

E  Flat, Pigmented Spot

F  Clean Base

Grade IIC  Grade III

Gralnek IM et al; NEJM, 2008
Endoscopic Management of Acute UGIB

UCLA CURE Hemostasis Group

Forrest Grade IB-IIA

Forrest Grade II A

Endoscopic Management of Acute UGIB

UCLA CURE Hemostasis Group

Forrest Grade IIB

## Endoscopic Management of Acute UGIB

### UCLA CURE Hemostasis Group’s Technical Parameters for Using Multipolar Electrocoagulation

<table>
<thead>
<tr>
<th></th>
<th>Peptic Ulcer</th>
<th>MALLORY-WEISS TEAR</th>
<th>DIEULAFOY’S LESION</th>
<th>GASTRIC ANGIOECTASIA</th>
<th>COLON DIVERTICULUM WITH VISIBLE VESSEL</th>
<th>COLON ANGIOECTASIA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACTIVE BLEEDING</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epinephrine Injection</td>
<td>Yes†</td>
<td>No</td>
<td>Yes‡</td>
<td>Yes</td>
<td>No</td>
<td>Maybe§</td>
</tr>
<tr>
<td><strong>NONBLEEDING VISIBLE VESSEL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Probe size III</td>
<td>Large</td>
<td>Large</td>
<td>Large or small</td>
<td>Large</td>
<td>Large or small</td>
<td>Large or small</td>
</tr>
<tr>
<td><strong>ADHERENT CLOT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure¶</td>
<td>Firm</td>
<td>Firm</td>
<td>Firm</td>
<td>Moderate</td>
<td>Firm</td>
<td>Light</td>
</tr>
<tr>
<td>Pulse duration (sec)</td>
<td>8-10</td>
<td>8-10</td>
<td>8-10</td>
<td>4</td>
<td>8-10</td>
<td>2</td>
</tr>
<tr>
<td><strong>ENDPOINT</strong></td>
<td>Bleeding stops</td>
<td>Flat vessel</td>
<td>Flat stigma</td>
<td>Bleeding stops</td>
<td>Flat vessel</td>
<td>White</td>
</tr>
</tbody>
</table>

### Notes
- † Epinephrine (1 : 20,000) injected in 1-mL aliquots into each of 4 quadrants should be used to control bleeding initially, followed by coagulation.
- ‡ Epinephrine (1 : 20,000) injected in 1-mL aliquots into each of 4 quadrants should be injected around clot initially, followed by piecemeal snare resection and treatment of underlying stigmata.
- § Colonic diverticulum with active bleeding can be treated with epinephrine (1 : 20,000) injected into the neck or base. If a visible vessel is seen at the neck, it can be treated with multipolar electrocoagulation.
- III Large probe is 10 Fr (3.2-mm diameter) and fits through a 3.8-mm endoscope channel. Small probe is 7 Fr (2.4 mm) and fits through a 2.8-mm endoscope channel.
- ¶ Pressure is the tamponade pressure exerted en face or tangentially via the contact probe directly on the lesion.
- ** Power setting using BICAP II generator. Power settings are general guidelines and may vary based on the generator used.
### Endoscopic Management of Acute UGIB

- Second Look Endoscopy not routinely indicated
- Only recommended in the absence of high-dose PPI

*(El Ouali S and Martel M et al; GI Endoscopy, 2012)*

#### Outpatient versus Inpatient Onset of UGIB

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>OUTPATIENT</th>
<th>INPATIENT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Frequency (%)</strong></td>
<td>80-90</td>
<td>10-20</td>
</tr>
<tr>
<td><strong>American Society of Anesthesiologists comorbidity score[†]</strong></td>
<td>≤3</td>
<td>&gt;3</td>
</tr>
<tr>
<td><strong>Time to re-bleeding (%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤72 hr</td>
<td>70-80</td>
<td>40-50</td>
</tr>
<tr>
<td>4-7 days</td>
<td>10-15</td>
<td>15-20</td>
</tr>
<tr>
<td>8-30 days</td>
<td>1-5</td>
<td>15-20</td>
</tr>
<tr>
<td>&gt;30 days</td>
<td>0</td>
<td>5-10</td>
</tr>
</tbody>
</table>

† 1 point signifies a healthy person; 5 points signifies high likelihood of mortality within 24 h

Pharmacologic Management: Acid Suppression

- Applies to UGIB from ulcers

### Table A: Rebleeding and Surgery

<table>
<thead>
<tr>
<th>Group</th>
<th>Proton-Pump Inhibitor</th>
<th>Control</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no. of patients/no. of events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rebleeding</td>
<td>1026/111</td>
<td>1031/189</td>
<td></td>
</tr>
<tr>
<td>Surgery</td>
<td>1081/38</td>
<td>1103/71</td>
<td></td>
</tr>
</tbody>
</table>

### Table B: Death

<table>
<thead>
<tr>
<th>Group</th>
<th>Proton-Pump Inhibitor</th>
<th>Control</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no. of patients/no. of events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>1040/20</td>
<td>1062/38</td>
<td></td>
</tr>
<tr>
<td>With endoscopic hemostasis</td>
<td>954/17</td>
<td>969/32</td>
<td></td>
</tr>
<tr>
<td>Without endoscopic hemostasis</td>
<td>86/3</td>
<td>93/6</td>
<td></td>
</tr>
</tbody>
</table>

**Key Point:** PPIs improve mortality

Gralnek IM et al; NEJM, 2008
Management of UGIB: Non-Variceal

- Syncope, shock, severe comorbidity, hematochezia, bright red blood per nasogastric tube, or inpatient start of upper gastrointestinal bleed

  **Yes**: Resuscitation and treatment in an intensive care unit

  **No**: Evaluate and resuscitate in step-down unit or ward

- Urgent upper endoscopy

  - Arterial bleed, nonbleeding visible vessel, or clot
    - Combination endoscopic hemostasis
    - IV bolus and infusion PPI for 72 hours
    - Discharge on oral PPI twice daily

  - Oozing without other stigmata
    - Hemoclip or thermal coagulation hemostasis
    - Oral PPI twice daily

  - Flat spot or clean base ulcer
    - Oral PPI and early discharge
Criteria for Short Hospital Stay in Acute UGIB: Non-Variceal

Criteria

Age, <60 yr

Absence of hemodynamic instability, which is defined as resting tachycardia (pulse, ≥100 beats per minute), hypotension (systolic blood pressure, <100 mm Hg), or postural changes (increase in pulse of ≥20 beats per minute or a drop in systolic blood pressure of ≥20 mm Hg on standing), or hemodynamic stability within 3 hours after initial evaluation

Absence of a severe coexisting illness (e.g., heart failure, chronic obstructive pulmonary disease, hepatic cirrhosis, hematologic cancer, chronic renal failure, and cerebrovascular accident)

A hemoglobin level of more than 8 to 10 g per deciliter after adequate intra-vascular volume expansion and no need for blood transfusion

Normal coagulation studies

Onset of bleeding outside the hospital

Presence of a clean-base ulcer or no obvious endoscopic finding on early endoscopy (performed within 24 hours after presentation)

Adequate social support at home with the ability to return promptly to a hospital

Gralnek IM et al; NEJM, 2008
Management of UGIB: Non-Variceal
## Risk of Re-bleeding in UGIB: Non-Variceal

<table>
<thead>
<tr>
<th>Endoscopic Appearance</th>
<th>Frequency (%)</th>
<th>Risk of Rebleeding (%)</th>
<th>Risk of Rebleeding after Endoscopic Hemostasis (%)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active arterial bleeding</td>
<td>12</td>
<td>90</td>
<td>15-30</td>
</tr>
<tr>
<td>Visible vessel</td>
<td>22</td>
<td>50</td>
<td>15-30</td>
</tr>
<tr>
<td>Adherent clot</td>
<td>10</td>
<td>33</td>
<td>0-5</td>
</tr>
<tr>
<td>Oozing without stigmata</td>
<td>14</td>
<td>10</td>
<td>0-5</td>
</tr>
<tr>
<td>Flat spot</td>
<td>10</td>
<td>7</td>
<td>NA</td>
</tr>
<tr>
<td>Clean ulcer base</td>
<td>32</td>
<td>3</td>
<td>NA</td>
</tr>
</tbody>
</table>

## Independent Risk Factors for Persistent or Recurrent GIB

<table>
<thead>
<tr>
<th>RISK FACTORS</th>
<th>RANGE OF ODDS RATIOS FOR INCREASED RISK</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical Factors</strong></td>
<td></td>
</tr>
<tr>
<td>Health status (ASA class 1 vs. 2-5)</td>
<td>1.94-7.63</td>
</tr>
<tr>
<td>Comorbid illness</td>
<td>1.6-7.63</td>
</tr>
<tr>
<td>Shock (systolic blood pressure &lt; 100 mm Hg)</td>
<td>1.2-3.65</td>
</tr>
<tr>
<td>Erratic mental status</td>
<td>3.21</td>
</tr>
<tr>
<td>Ongoing bleeding</td>
<td>3.14</td>
</tr>
<tr>
<td>Age ≥ 70 yr</td>
<td>2.23</td>
</tr>
<tr>
<td>Age &gt; 65 yr</td>
<td>1.3</td>
</tr>
<tr>
<td>Transfusion requirement</td>
<td>NA</td>
</tr>
<tr>
<td>Presentation of Bleeding</td>
<td></td>
</tr>
<tr>
<td>Hematemesis</td>
<td>1.2-5.7</td>
</tr>
<tr>
<td>Red blood on rectal examination</td>
<td>3.76</td>
</tr>
<tr>
<td>Melena</td>
<td>1.6</td>
</tr>
<tr>
<td><strong>Laboratory Factors</strong></td>
<td></td>
</tr>
<tr>
<td>Coagulopathy</td>
<td>1.96</td>
</tr>
<tr>
<td>Initial hemoglobin ≤ 10 g/dL</td>
<td>0.8-2.99</td>
</tr>
<tr>
<td><strong>Endoscopic Factors</strong></td>
<td></td>
</tr>
<tr>
<td>Ulcer location on superior wall of duodenum</td>
<td>13.9</td>
</tr>
<tr>
<td>Ulcer location on posterior wall of duodenum</td>
<td>9.2</td>
</tr>
<tr>
<td>Active bleeding</td>
<td>2.5-6.48</td>
</tr>
<tr>
<td>High-risk stigmata</td>
<td>1.91-4.81</td>
</tr>
<tr>
<td>Ulcer size ≥ 2 cm</td>
<td>2.29-3.54</td>
</tr>
<tr>
<td>Ulcer location high on lesser curve</td>
<td>2.79</td>
</tr>
<tr>
<td>Diagnosis of gastric or duodenal ulcer</td>
<td>2.7</td>
</tr>
<tr>
<td>Clot over ulcer</td>
<td>1.72-1.9</td>
</tr>
</tbody>
</table>

Factors Predictive of Poor Prognosis after Acute UGIB from Peptic Ulcer

- Age > 60 years
- Bleeding onset in hospital
- Comorbid medical illness
- Shock or orthostatic hypotension
- Fresh blood in nasogastric tube
- Coagulopathy
- Multiple transfusions required
- Higher lesser curve gastric ulcer (adjacent to left gastric artery)
- Posterior duodenal bulb ulcer (adjacent to gastroduodenal artery)
- Endoscopic finding of arterial bleeding or visible vessel

Contraindications of Urgent Endoscopy in Acute UGIB

- When the risks to patient health or life are judged to outweigh the most favorable benefits of the procedure.
- When adequate patient cooperation or consent cannot be obtained.
- When a perforated viscus is known or suspected.
Management of UGIB: Non-Variceal

- Initial Assessment and Risk Stratification
- Pre-endoscopic medical therapy
- Gastric Lavage
- Timing of endoscopy
- Endoscopic diagnosis
- Endoscopic therapy
- Medical therapy after endoscopy
- Repeat endoscopy
- Hospitalization
- Long-term prevention of recurrent bleeding ulcers
Management of UGIB: Non-Variceal

Established institutional protocols in place
- Develop institution-specific protocols for management
- Have available support staff trained to assist in endoscopy

ABC’s and adequate resuscitation
- Evaluate and resuscitate
- Transfuse blood if hemoglobin ≤ 70g/L
- Correct coagulopathy but do not delay endoscopy

Early risk stratification / initial management

Pre-endoscopy
- Consider placement of nasogastric tube
- Determine the Blatchford or pre-endoscopic (clinical) Rockall score to stratify into low- and high-risk categories
- Do not use somatostatin or octreotide
- Consider promotility agents in patients likely to have blood clots in the stomach
- Consider pre-endoscopic PPI therapy

At early endoscopy
- Determine the complete Rockall score (using the additional endoscopic information)

Greenspoon J, Barkun AN and Sung J et al; Clin Gastro Hepat, 2012
Management of UGIB: Non-Variceal

Discharge very low-risk patients pre-endoscopically if Blatchford score is 0

Admit all other patients

Low-risk patients (without high-risk endoscopic lesions)
- Initiate daily dose oral PPI
- Consider early discharge same day or next day

High-risk patients (exhibiting high-risk endoscopic lesions)

Endoscopic therapy
- Endoscopic hemostasis as clips, thermocoagulation or sclerosant injection alone or in combination with epinephrine for high-risk lesions
- Clot in ulcer bed requires irrigation to determine the presence of an adherent clot
- Adherent clots - consider endoscopic therapy or sole PPI use

Pharmacologic therapy
- High-dose IV bolus + continuous infusion of PPI (initial bolus equivalent to 80 mg of omeprazole followed by infusion equivalent to 8 mg/hour of omeprazole for 72 hrs)
- H2RA are not recommended

Management issues
- High-risk stigmata patients hospitalized for 72 hrs
- Stable patients after endoscopy can be fed within 24 hrs

Greenspoon J, Barkun AN and Sung J et al; Clin Gastro Hepat, 2012
Management of UGIB: Non-Variceal

If rebleeding occurs
- Second attempt at endoscopic therapy recommended
- Seek surgical consultation
- Percutaneous embolization can be considered as an alternative to surgery

Upon discharge
- Discharge patients with prescription for daily oral PPI for a duration determined by the cause of the bleed
- Test for *H. pylori* and eradicate accordingly with subsequent confirmation of eradication
- Repeat negative *H. pylori* tests outside the acute setting
- Adding a PPI to a traditional NSAID or switching to COX-2 inhibitor alone are strategies associated with increased risk for recurrent ulcer bleeding; recommend COX-2 + PPI instead for patients having bled on NSAID or COX-2, if cardiovascular status allows it
- Restart ASA therapy when cardiovascular risks outweigh risk of rebleeding, aiming for <7 days when safe; add a PPI as secondary prophylaxis since clopidogrel alone has increased risk for rebleeding
- Add PPI to patients having bled on clopidogrel

Greenspoon J, Barkun AN and Sung J et al; Clin Gastro Hepat, 2012
Clopidogrel and Omeprazole in Coronary Artery Disease (COGENT trial)

**Figure 1.** Kaplan–Meier Estimates of the Probability of Remaining Free of Primary Gastrointestinal Events, According to Study Group.

The event rate for the primary gastrointestinal end point at day 180 was 1.1% in the omeprazole group and 2.9% in the placebo group.

**Figure 2.** Kaplan–Meier Estimates of the Probability of Remaining Free of Primary Cardiovascular Events, According to Study Group.

The event rate for the primary cardiovascular end point at day 180 was 4.9% in the omeprazole group and 5.7% in the placebo group.

Bhatt DL and Cannon CP et al; NEJM, 2010
Consensus Recommendations for Management of UGIB: Non-Variceal

A. Resuscitation, risk assessment, and preendoscopy management
   A1. Immediately evaluate and initiate appropriate resuscitation.*
   A2. Prognostic scales are recommended for early stratification of patients into low- and high-risk categories for rebleeding and mortality.†
   A3. Consider placement of a nasogastric tube in selected patients because the findings may have prognostic value.*
   A4. Blood transfusions should be administered to a patient with a hemoglobin level ≤70 g/L.
   A5. In patients receiving anticoagulants, correction of coagulopathy is recommended but should not delay endoscopy.
   A6. Promotility agents should not be used routinely before endoscopy to increase the diagnostic yield.
   A7. Selected patients with acute ulcer bleeding who are at low risk for rebleeding on the basis of clinical and endoscopic criteria may be discharged promptly after endoscopy.†
   A8. Preendoscopic PPI therapy may be considered to downstage the endoscopic lesion and decrease the need for endoscopic intervention but should not delay endoscopy.†

B. Endoscopic management
   B1. Develop institution-specific protocols for multidisciplinary management.* Include access to an endoscopist trained in endoscopic hemostasis.*
   B2. Have available on an urgent basis support staff trained to assist in endoscopy.*
   B3. Early endoscopy (within 24 hours of presentation) is recommended for most patients with acute upper gastrointestinal bleeding.†
   B4. Endoscopic hemostatic therapy is not indicated for patients with low-risk stigmata (a clean-based ulcer or a nonprotuberant pigmented dot in an ulcer bed).*
   B5. A finding of a clot in an ulcer bed warrants targeted irrigation in an attempt at dislodgement, with appropriate treatment of the underlying lesion.†
   B6. The role of endoscopic therapy for ulcers with adherent clots is controversial. Endoscopic therapy may be considered, although intensive PPI therapy alone may be sufficient.†
   B7. Endoscopic hemostatic therapy is indicated for patients with high-risk stigmata (active bleeding or a visible vessel in an ulcer bed).*
   B8. Epinephrine injection alone provides suboptimal efficacy and should be used in combination with another method.†
   B9. No single method of endoscopic thermal coaptive therapy is superior to another.*
   B10. Clips, thermocoagulation, or sclerosant injection should be used in patients with high-risk lesions, alone or in combination with epinephrine injection.†
   B11. Routine second-look endoscopy is not recommended.†
   B12. A second attempt at endoscopic therapy is generally recommended in cases of rebleeding.*
C. Pharmacologic management
C1. Histamine-2 receptor antagonists are not recommended for patients with acute ulcer bleeding.*
C2. Somatostatin and octreotide are not routinely recommended for patients with acute ulcer bleeding.*
C3. An intravenous bolus followed by continuous-infusion PPI therapy should be used to decrease rebleeding and mortality in patients with high-risk stigmata who have undergone successful endoscopic therapy.†
C4. Patients should be discharged with a prescription for a single daily-dose oral PPI for a duration as dictated by the underlying etiology.

D. Nonendoscopic and nonpharmacologic in-hospital management
D1. Patients at low risk after endoscopy can be fed within 24 hours.*
D2. Most patients who have undergone endoscopic hemostasis for high-risk stigmata should be hospitalized for at least 72 hours thereafter.
D3. Seek surgical consultation for patients for whom endoscopic therapy has failed.*
D4. Where available, percutaneous embolization can be considered as an alternative to surgery for patients for whom endoscopic therapy has failed.
D5. Patients with bleeding peptic ulcers should be tested for H. pylori and receive eradication therapy if it is present, with confirmation of eradication.†
D6. Negative H. pylori diagnostic tests obtained in the acute setting should be repeated.

E. Postdischarge, ASA, and NSAIDs
E1. In patients with previous ulcer bleeding who require an NSAID, it should be recognized that treatment with a traditional NSAID plus PPI or a COX-2 inhibitor alone is still associated with a clinically important risk for recurrent ulcer bleeding.
E2. In patients with previous ulcer bleeding who require an NSAID, the combination of a PPI and a COX-2 inhibitor is recommended to reduce the risk for recurrent bleeding from that of COX-2 inhibitors alone.
E3. In patients who receive low-dose ASA and develop acute ulcer bleeding, ASA therapy should be restarted as soon as the risk for cardiovascular complication is thought to outweigh the risk for bleeding.
E4. In patients with previous ulcer bleeding who require cardiovascular prophylaxis, it should be recognized that clopidogrel alone has a higher risk for rebleeding than ASA combined with a PPI.
Acknowledgements

- Funding agency: NIH, MDACC Physician Scientist Award
  Mayo Clinic IRG, and UTH
- American Gastroenterological Association (AGA)
- UCLA CURE Hemostasis Group (PI: DM Jensen)

Thank You