MANAGING GI BLEEDING IN A COMMUNITY HOSPITAL SETTING

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DISCLOSURES

Presenter: Dr Michele Brule
Relationships with commercial interests: None
OBJECTIVES

- Assess the severity of GI bleeding in patients being transferred from a community hospital to a tertiary care facility.
- Recognize the requirements for blood products based on estimating the amount of GI bleeding.
INTRODUCTION

- Evaluation of patients with GI bleeding involves assessment of hemodynamic stability and resuscitation if necessary.

- In order to manage these patients appropriately, it is important to differentiate between upper and lower GI bleeding and acute vs chronic bleeding.
Which statement is correct?

A. Melena is always due to upper GI bleeding.

B. If a patient is not tachycardic, they are hemodynamically stable.

C. FOBT should be used for patients presenting to the ER with melena but not hematochezia.

D. AIMS 65 score is based on age, albumin, mental status, blood pressure, and INR.

E. All patients presenting with GI bleeding require large caliber IVs and blood products on hand.
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Hemodynamic instability
- Syncope (or pre-syncope)
- Tachycardia (note: beta-blockers, postural in young patients)
- Hypotension

Presentation of bleed
- Hematemesis (vomiting blood or coffee-ground)
- Melena (black/tarry stool)
  - 90% are proximal to the ligament of Treitz
  - Others: oropharynx, small bowel, right colon
- Hematochezia (red/maroon)
  - Presence of clots: more likely lower GI bleed
  - Orthostatic hypotension + hematochezia: massive upper GI bleed
### APPROACH TO GI BLEEDING - HISTORY

- History of presenting illness
  - When it started
  - Frequency of blood
  - Colour of the blood
  - Amount of blood
  - Prior episodes of GI bleeding (including vomiting and black BM)
    - 60% of patients with a history of upper GI bleed are bleeding from the same lesion
  - Acute vs chronic blood loss
    - Symptoms of anemia prior to presentation (SOB, weak, tired, pre-syncope or syncope)
    - Black bowel movements before presentation
  - Signs and symptoms
    - Upper abdo pain, GERD, dysphagia, nausea, vomiting (Mallory-Weiss tear), jaundice, weakness, fatigue, anorexia, weight loss, early satiety, and others
Past medical and surgical history

- Comorbidities which may lead to bleeds
  - **Peptic ulcer disease** *(NSAIDs, *H. pylori*, smoking, alcohol, caffeine)*
  - **Varices or portal hypertensive gastropathy** *(liver disease, alcohol abuse)*
  - Aorto-enteric fistula *(AAA or aortic graft)*
  - Angiodysplasia *(renal disease, aortic stenosis, hereditary hemorrhagic telangiectasia)*
  - Inflammatory bowel disease and other colitis
  - **Diverticular disease** *(painless hematochezia)*
  - Malignancy *(especially with changes in bowel habits)*
  - Marginal ulcers at an anastomotic site *(bariatric surgery)*
Past medical and surgical history

- Comorbidities which may influence subsequent management
  - Susceptibility to hypoxemia: CAD, pulmonary disease
    - Should be maintained at higher hemoglobin levels
  - Predisposition to volume overload: renal disease, heart failure
    - Monitor closely in resuscitation
  - Difficulty to control bleeding: coagulopathy, thrombocytopenia, significant hepatic dysfunction
    - May require FFP or platelets
    - May require reversal agents
Medications

- Predispose to peptic ulcer formation
  - NSAIDs
  - Aspirin
  - Steroids

- Promote bleeding
  - Antiplatelet agents (clopidogrel, NSAIDs, aspirin)
  - Anticoagulants (warfarin, heparin, novel oral anticoagulants or NOACs)

- Drugs that may mimic melena
  - Bismuth (Peptobismol)
  - Iron

- Drugs: smoking and alcohol use
Approach to GI bleeding – Physical Examination

- Signs of hypovolemic
  - Resting tachycardia: mild/moderate hypovolemia
  - Orthostatic hypotension: blood volume loss of > 15%
    - Decrease in BP > 20 mmHg +/- increase in HR > 20 beats/min
  - Supine hypotension: blood volume loss of > 40%

- Digital rectal examination: red or maroon stool
  - In 80 patients with red/maroon blood in the stool: 74% had colonic lesion, 11% upper GI lesion, 9% presumed small bowel, 6% no site identified
  - Special note: there is no role for FOBT in acute GI bleeding

- NG lavage: rule out upper GI source

- Look for signs of liver disease, tenderness, and masses
APPROACH TO GI BLEEDING – INITIAL WORKUP

- CBC
  - Hemoglobin, MCV, platelets
- INR and PTT
- Cross and type
- Urea and Creatinine
  - Ratio of blood urea nitrogen to serum creatinine
- Liver tests
Mr N Said, a 70 year old male, takes coumadin for chronic atrial fibrillation. Presents to the emergency department with black BM for 3 days.

Vitals: HR 80 irregularly irregular, BP 110/70.

Hb 85, MCV 83, Plat 235, INR 2.3, Urea 12, Cr 79. What do you do?

A. Continue coumadin
B. Stop coumadin and reverse with vitamin K
C. Stop coumadin and reverse with plasma or Octaplex
D. Stop coumadin and start IV heparin
E. Transfuse 2 units packed RBCs
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MANAGEMENT OF GI BLEEDING

- Supplemental oxygen (if needed) and NPO
- 2 large caliber IVs with crystalloid bolus
  - Have blood products available
- Empiric PPI for all upper GI bleeds at presentation
  - Somatostatin (octreotide) and prophylactic antibiotics for suspected variceal bleeding
  - Tranexamic acid
- Stop all anticoagulants and antiplatelet agents (NSAIDs and aspirin)
  - May also require reversal if actively bleeding
Mrs Pale is a 65 year old woman with coffee-ground emesis. PMH: hypothyroidism.

Vitals: HR 95, BP 110/70.

Hb 76, MCV 73, Plat 115, INR 1.1, Urea 5, Cr 68. What do you do?

A. Start large bore IVs and cross and type 4 units.
B. Transfuse 2 units packed RBCs.
C. 3 views of the abdomen.
D. Send home and arrange outpatient upper endoscopy.
E. All of the above.
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MANAGEMENT OF GI BLEEDING

- Blood transfusion thresholds:
  - Restrictive strategy associated with better outcomes in most patients (including stable CAD)
  - Hg < 70 g/L with ongoing bleeding (Hg < 80 g/L in high risk patients)
- Transfuse platelets in patients with platelets < 50,000
  - May also transfuse in patients with life-threatening bleed who have received antiplatelet agents (aspirin or clopidogrel)
- Transfuse FFP in patients with coagulopathy (INR >1.5) not due to cirrhosis and active bleeding
  - Or after 4 units of blood with ongoing bleeding
Transfusion Strategies for Acute Upper Gastrointestinal Bleeding

Càndid Villanueva, M.D., Alan Colomo, M.D., Alba Bosch, M.D., Mar Concepción, M.D., Virginia Hernandez-Gea, M.D., Carles Aracil, M.D., Isabel Graupera, M.D., María Poca, M.D., Cristina Alvarez-Urturi, M.D., Jordi Gordillo, M.D., Carlos Guarner-Argente, M.D., Miquel Santaló, M.D., Eduardo Muñiz, M.D., and Carlos Guarner, M.D.
A randomized clinical trial shows that among patients with upper GI bleeding, withholding transfusion until the hemoglobin level falls below 70 g/L results in better outcomes than using 90 g/L as the trigger for transfusion.

- Inclusion criteria: hematemesis, bloody NG aspirate or melena
- Exclusion criteria: high risk patients
  - Massive exsanguinating bleeding, ACS, symptomatic PVD, stroke, TIA, transfusion within 90 days, recent trauma/surgery, or lower GI bleed
- All patients received PPIs and early endoscopy (within 6 hours)
RATE OF SURVIVAL, ACCORDING TO SUBGROUP

A Survival, According to Transfusion Strategy

B Death by 6 Weeks, According to Subgroup

In practice, FFP is frequently transfused to patients with cirrhosis and variceal hemorrhage during initial resuscitation, but the risk-benefit balance between haemostatic correction and potential hypovolemia is unclear.

Important details

- Plasma should not be used for warfarin reversal
  - Exception: rare cases of HIT – Octaplex/PCC contains heparin

- Blood products should only be used to reverse anticoagulation if there is a life-threatening bleed or an emergency surgical procedure in the next 6 hours
  - Prothrombin complex concentrates (Octaplex/PCC) last 6 hours and contain factors 2, 7, 9, 10
  - Must be given with IV vitamin K (starts working in 6 hours and prevents rebound)
  - PCC is associated with lower all-cause mortality than plasma

- Patients with liver disease are generally in a prothrombic state, even if INR is elevated, and giving plasma may be harmful in these patients
Almost half of patients with acute upper GI bleeds are taking NSAIDs or anti-platelets, which last for the duration of the platelet lifespan (7-10 days).

There is no evidence to support the use of platelet transfusion in patients taking anti-platelet agents presenting with major GI bleeding.

Current recommended practice is to transfuse platelets when the platelet count falls below 50.
Upper GI bleed
- When hemodynamically stable, early upper endoscopy
  - Safe to do in hemodynamically stable patients, even without normal hematocrit and with moderate anticoagulation (INR < 2.7)
  - Studies have reached variable conclusions regarding early endoscopy
- Therapeutic endoscopy may be used to achieve acute hemostasis and prevent recurrent bleeding in most patients
- If source is not identified, follow with colonoscopy
- Other investigations: RBC scan, CT angiography or standard angiography (only if actively bleeding), evaluations for small bowel bleeding

Unstable patient
- Resuscitate and prepare for emergency endoscopy
- If unable to stabilize, consult surgery and/or interventional radiology
Lower GI bleed

- Colonoscopy is the initial examination of choice for diagnosis and treatment of acute lower GI bleeding (once upper GI bleeding source is excluded)
- Other useful diagnostic procedures: radionuclide imaging, CT angiography, mesenteric angiography if actively bleeding at time of examination
Factors associated with re-bleeding in an upper GI bleed:
- Hemodynamic instability (systolic BP < 100 mmHg, tachycardia)
- Hemoglobin less than 100 g/L
- Active bleeding at the time of endoscopy
- Large ulcer size (greater than 1-3 cm)
- Ulcer location (posterior duodenal bulb or high lesser gastric curvature)

Low risk patients may be discharged early or treated as out-patients
- No comorbidities
- Stable vital signs
- Normal hemoglobin
- Identified bleeding source on endoscopy and have low risk of rebleeding (i.e. not variceal bleed, active bleed, Dieulafoy’s lesion, or ulcer with high-risk stigmata)
- I.e. Glasgow Blatchford score of 0 or AIMS 65 score of 0
RISK STRATIFICATION OF UPPER GI BLEED WITH THE FULL GLASGOW BLATCHFORD SCORE

<table>
<thead>
<tr>
<th>Admission risk marker</th>
<th>Score component value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood urea (mmol/L)</td>
<td></td>
</tr>
<tr>
<td>$\geq 6.5 &lt; 8.0$</td>
<td>2</td>
</tr>
<tr>
<td>$\geq 8.0 &lt; 10.0$</td>
<td>3</td>
</tr>
<tr>
<td>$\geq 10.0 &lt; 25.0$</td>
<td>4</td>
</tr>
<tr>
<td>$\geq 25$</td>
<td>6</td>
</tr>
<tr>
<td>Haemoglobin (g/L) for men</td>
<td></td>
</tr>
<tr>
<td>$\geq 120 &lt; 130$</td>
<td>1</td>
</tr>
<tr>
<td>$\geq 100 &lt; 120$</td>
<td>3</td>
</tr>
<tr>
<td>$&lt; 100$</td>
<td>6</td>
</tr>
<tr>
<td>Haemoglobin (g/L) for women</td>
<td></td>
</tr>
<tr>
<td>$\geq 100 &lt; 120$</td>
<td>1</td>
</tr>
<tr>
<td>$&lt; 100$</td>
<td>6</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td></td>
</tr>
<tr>
<td>100–109</td>
<td>1</td>
</tr>
<tr>
<td>90–99</td>
<td>2</td>
</tr>
<tr>
<td>$&lt; 90$</td>
<td>3</td>
</tr>
<tr>
<td>Other markers</td>
<td></td>
</tr>
<tr>
<td>Pulse $\geq 100$ (per min)</td>
<td>1</td>
</tr>
<tr>
<td>Presentation with melaena</td>
<td>1</td>
</tr>
<tr>
<td>Presentation with syncope</td>
<td>2</td>
</tr>
<tr>
<td>Hepatic disease</td>
<td>2</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 1: Admission risk markers and associated score component values

Blatchford et al. The Lancet 2000;356: 1318-1321
AIMS 65 scoring system: high accuracy for predicting inpatient mortality in upper GI bleeding patients

- Albumin < 30 g/L
- INR > 1.5
- Altered Mental status (GCS < 14, disorientation, lethargy, stupor, coma)
- Systolic BP < 90 mmHg
- Age > 65 years

Mortality rates

- Zero risk factors: 0.3%
- 1-2 risk factors: 1-3%
- 3-4 risk factors: 9-15%
- 5 risk factors: 25%

Increased score also associated with increased length of stay and increased cost
## Endoscopic predictors of recurrent peptic ulcer hemorrhage

<table>
<thead>
<tr>
<th>Endoscopic stigmata of recent hemorrhage</th>
<th>Prevalence, percent</th>
<th>Risk of rebleeding on medical management, percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active arterial bleeding (Forrest Ia)</td>
<td>10</td>
<td>90</td>
</tr>
<tr>
<td>Oozing without visible vessel (Forrest Ib)</td>
<td>10</td>
<td>10 to 20</td>
</tr>
<tr>
<td>Non-bleeding visible vessel (Forrest IIa)</td>
<td>25</td>
<td>50</td>
</tr>
<tr>
<td>Adherent clot (Forrest IIb)</td>
<td>10</td>
<td>25 to 30</td>
</tr>
<tr>
<td>Flat spot (Forrest IIc)</td>
<td>10</td>
<td>7 to 10</td>
</tr>
<tr>
<td>Clean ulcer base (Forrest (III))</td>
<td>35</td>
<td>3 to 5</td>
</tr>
</tbody>
</table>

Hematemesis or bright red blood per NG tube aspirate was identified as the **sole independent significant predictor of early re-bleeding among NVUGI** (more specifically, peptic ulcer bleeding) by Maggio et al. (2013)

- Early re-bleeding defined as within 72 hours
CONCLUSION

- Hemodynamic stability and hemoglobin levels are the main indicators of GI bleed severity and dictate the need for transfusion.
- The mainstay of resuscitation are fluids and blood, not inotropes.
- Restrictive blood thresholds have been shown to be safe and beneficial, but IV access and blood product availability is crucial.
- Early endoscopy once the patient is stable is beneficial for diagnosis and treatment.
REFERENCES


- Saltzman, J. R. Approach to acute upper gastrointestinal bleeding in adults. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed December 27, 2016).

- Strate, L. Approach to acute lower gastrointestinal bleeding in adults. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed December 27, 2016).
