GI Bleeding

Surgery Half Day
September 7, 2005

Joe Pham
Upper GI Bleed: Epidemiology

- 100 cases / 100,000 adults / year

- 150,000 hospital admissions / y (U.S. 1985)

- 15, 000 / year (Canada)

- bleeding ceases spontaneously 80%

- mortality rate 6%
Presentation

HISTORY:
Melena 69%
Hematochezia 15%
Hematemesis 30%
Coffee ground emesis 28%

Symptoms of anemia CP, SOB, presyncope fatigue, weakness

Risk Factors:
Previous PUD, UGIB
H pylori status

ASA 40%
NSAIDS 20%
COX-2 3.3%
Heparin 11%
Coumadin 11%
Steroids 6.7%

PHYSICAL EXAM:
Melena 25%
Bright red blood 5%
NSAID Ulcers: Prevalence

Laine L Gastro 2001: 120: 594
-endoscopic studies in pts using conventional NSAIDS

<table>
<thead>
<tr>
<th>Type</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>GU/DU</td>
<td>15-30%</td>
</tr>
<tr>
<td>GU</td>
<td>15-20%</td>
</tr>
<tr>
<td>DU</td>
<td>5-8%</td>
</tr>
</tbody>
</table>

Annual Risk of Complications 1-4%/yr
-POB (perforation, obstruction, bleeding)

Dyspepsia with NSAIDs 15-25%
10% NSAID users discontinue use due to dyspepsia
### NSAID Risk Factors for Ulcers / Bleeding

<table>
<thead>
<tr>
<th>Risk Factors for Adverse Events</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hx complicated PUD</td>
<td>13.5</td>
</tr>
<tr>
<td>Multiple NSAIDs</td>
<td>9.0</td>
</tr>
<tr>
<td>High Dose</td>
<td>7.0</td>
</tr>
<tr>
<td>anti-coagulation</td>
<td>6.4</td>
</tr>
<tr>
<td>PUD w/o cx</td>
<td>6.1</td>
</tr>
<tr>
<td>&gt; 70 yo</td>
<td>5.6</td>
</tr>
<tr>
<td>&gt; 60 yo</td>
<td>3.1</td>
</tr>
<tr>
<td>commitant steroids</td>
<td>2.2</td>
</tr>
<tr>
<td>hx of CAD</td>
<td>1.8</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>NSAID</th>
<th>Relative Risk hemorrhage / perforation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ibuprofen</td>
<td>2.9</td>
</tr>
<tr>
<td>Naproxen</td>
<td>3.1</td>
</tr>
<tr>
<td>Diclofenac</td>
<td>3.9</td>
</tr>
<tr>
<td>Ketoprofen</td>
<td>5.4</td>
</tr>
<tr>
<td>Indomethacin</td>
<td>6.3</td>
</tr>
<tr>
<td>Mulitple NSAIDs</td>
<td>8.9</td>
</tr>
<tr>
<td>Piroxicam</td>
<td>18.9</td>
</tr>
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</table>
## UGIB: Differential Diagnosis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>PUD</td>
<td>55%</td>
</tr>
<tr>
<td>Esophageal varices **</td>
<td>14%</td>
</tr>
<tr>
<td>AVM</td>
<td>6%</td>
</tr>
<tr>
<td>Mallory-Weiss tears</td>
<td>5%</td>
</tr>
<tr>
<td>Tumours</td>
<td>4%</td>
</tr>
<tr>
<td>Dieulafoy’s Lesion</td>
<td>1%</td>
</tr>
<tr>
<td>Other</td>
<td>11%</td>
</tr>
</tbody>
</table>

- **GAVE (Gastric Antral Vascular Ectasia)**
- **Portal Hypertensive Gastropathy**
- **Hemobilia**
- **Hemosuccus Pancreaticus**
- **Aortoenteric Fistula**
Initial Management
Initial Management

**ABCs**
- hemodynamic instability
- postural vitals (SBP↓ 20 mm Hg, HR↑ > 30 bpm)

**Resuscitation**
- O2, IV (x2 large bore), monitors
- Vitals & urine output (foley)
- IV NS
- EKG

**Hemoglobin & coagulation studies**
- cross & type
- FFP +/- vitamin K

**Nasogastric tube (in/out)**
- positive for blood rules in UGIB
- can NOT rule out Duodenal Bleed
  - ? Except when bile is present
- prognostic
Previous Treatment of PUD

- Truncal vagotomy prevents nerve stimulation of the stomach
- Pyloroplasty
- Oversewn ulcer
Then along came ENDOSCOPY
Endoscopic Stigmata of Bleeding


Data from 37 prospective trials (no endoscopic therapy)
Endoscopic Stigmata of Bleeding
## FORREST Classification of UGIB

### Acute hemorrhage

| Forrest I a | Arterial, spurting hemorrhage |
| Forrest I b | Oozing hemorrhage |

### Signs of recent hemorrhage

| Forrest II a | Visible vessel |
| Forrest II b | Adherent clot |
| Forrest II c | Hematin-covered lesion |

### Lesions without active bleeding

| Forrest III | No signs of recent hemorrhage |
Prevalence of Endoscopic Sigmata

- High Risk Stigmata: 44%
- Low Risk Stigmata: 56%
The role of endoscopy therapy in UGI bleeding is well established.

<table>
<thead>
<tr>
<th>30 RCTs (n=2,412)</th>
<th>All studies (30)</th>
<th>PUD (22/30)</th>
<th>High-risk Actively bleeding or NBVV (16-21/30)</th>
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</thead>
<tbody>
<tr>
<td>thermal contact (13)</td>
<td>0.38</td>
<td>0.57</td>
<td>0.23</td>
</tr>
<tr>
<td>laser (13)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>injection (7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rebleeding</td>
<td>0.36</td>
<td>0.37</td>
<td>0.26</td>
</tr>
<tr>
<td>Surgery</td>
<td>0.55</td>
<td>0.40</td>
<td>0.62</td>
</tr>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

No benefits with lower risk stigmata of bleeding
-adherent clots, flat pigmented spots

**Endoscopic Therapy**

Perform early (ideally within 24 h)

**Indications for haemostatic therapy:**
- Active bleeding (oozing, spurting)
- Nonbleeding visible vessel,

**Targeted Irrigation**
- Adherent clot

**Options**
1) Injection (NS, epinephrine, sclerosant)
2) thermocoagulation (heat probe, bicap, APC)
3) clips (bi, triclips)
**Endoscopy: Is Combination Therapy Better?**

- meta-analysis of 16 RCTs (1673 pts)
  - epinephrine injection alone vs epinephrine & second hemodynamic Tx

- primary end point: persistent hemorrhage & rebleeding
- high risk lesions: spurting, oozing, NBVV, adherent clot

<table>
<thead>
<tr>
<th></th>
<th>Epinephrine</th>
<th>Combination</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Further Bleeding</td>
<td>18.4%</td>
<td>10.6%</td>
<td>0.53</td>
</tr>
<tr>
<td>Failure Initial Hemostasis</td>
<td>2.8%</td>
<td>2.4%</td>
<td>0.84</td>
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<tr>
<td>Recurrent Bleeding**</td>
<td>15.8%</td>
<td>8.9%</td>
<td>0.53</td>
</tr>
<tr>
<td>Surgery</td>
<td>11.3%</td>
<td>7.6%</td>
<td>0.64</td>
</tr>
<tr>
<td>Mortality</td>
<td>5.1%</td>
<td>2.6%</td>
<td>0.51</td>
</tr>
</tbody>
</table>

CALVET et al. Gastroenterology 2004
<table>
<thead>
<tr>
<th>Studies</th>
<th>Group 1</th>
<th>Group 2</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Further Bleeding</td>
<td>Epinephrine</td>
<td>18.8%</td>
<td>Epinephrine &amp; Second Injected Agent</td>
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<tr>
<td>Surgery</td>
<td>11.1%</td>
<td>9.4%</td>
<td>0.82</td>
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<tr>
<td>Mortality</td>
<td>5.3%</td>
<td>2.1%</td>
<td>0.41</td>
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</table>

<table>
<thead>
<tr>
<th>Studies</th>
<th>Group 1</th>
<th>Group 2</th>
<th>OR</th>
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</thead>
<tbody>
<tr>
<td>Further Bleeding</td>
<td>Epinephrine</td>
<td>19.2%</td>
<td>Epinephrine &amp; Mechanical Method</td>
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<tr>
<td>Surgery</td>
<td>11.5%</td>
<td>2.4%</td>
<td>0.64</td>
</tr>
<tr>
<td>Mortality</td>
<td>3.8%</td>
<td>1.2%</td>
<td>0.34</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Studies</th>
<th>Group 1</th>
<th>Group 2</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Further Bleeding</td>
<td>Epinephrine</td>
<td>17.1%</td>
<td>Epinephrine &amp; Thermal Method</td>
</tr>
<tr>
<td>Surgery</td>
<td>11.8%</td>
<td>4.8%</td>
<td>0.40</td>
</tr>
<tr>
<td>Mortality</td>
<td>5.3%</td>
<td>4.8%</td>
<td>0.88</td>
</tr>
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</table>
Endoscopic Therapy: Adherent Clot

Treatment of adherent clots remains controversial:

Some advice to that the clot not be disturbed while others recommend endoscopic treatment.

The underlying stigmata will determine the risk for rebleeding.
Adherent Clot: Study 1

- 46 pts with clinical UGI bleed with clot found on endoscopy
- no medical therapy (PPI or H2 blocker)
- "targeted irrigation" using 3.2 mm bipolar probe
  - until clot was removed or for 5 minutes

- after irrigation:
  57% (26/46) adherent clots
  30% high risk lesion
    (spurt 2%, ooze 13%, NBVV 15%)
  13% low risk lesion
    (flat spot 11%, clean base 2%)

- only high risk lesions were treated with endoscopic therapy

Laine et al. Gastrointestinal Endoscopy. 1996
Adherent Clot: Study 2

- RCT 56 pts with UGI bleed
- adherent clot found on endoscopy done w/in 24 hrs
  - irrigated with 200 ml using 50 cc syringe
- Losec 20 mg PO bid x 1 month or famotidine 20 mg IV bid until tol. PO

Randomized to:
1) medical management (n=35)
2) endoscopic treatment (n=21)
   - injection epinephrine x 4 quadrants
   - removing clot (suction, snare, biopsy forceps)
   - heater probe (30 J) to all lesions

Bleau et al. Gastrointestinal Endoscopy. 2002

Rebleeding

\[ \begin{array}{c|c|c}
\text{Medical Mx} & \text{Endoscopic Tx} \\
\hline
34.5\% & 4.8\% \\
\end{array} \]

\( P < 0.02 \)
Adherent Clot: Study 3

-RCT 32 pts with UGI bleed
-adherent clot found on endoscopy after target irrigation with heater probe
-PPI bid

Randomized to:
1) medical management (n=17)
2) endoscopic treatment (n=15)
   -injection epinephrine x 4 quadrants
   -clot removing by snare guillotining
   -bipolar coaggulation 12-15 W

Jensen et al. Gastroenterology. 2002

% Patients
0%   10%   20%   30%   40%
Rebleeding

Medical Mx  Endoscopic Tx
35.3%  0%
p=0.011

0%
Early Risk of Rebleeding: First 72 hrs

Natural history of the sentinel clot

Day 0 - Adherent Clots (n=104)  

% of Endoscopic Stigmata

- Adherent clot
- Visible vessel
- Active bleeding

Day 0 - Visible Vessels (n=62)

- Adherent clot
- Visible vessel
- Active bleeding

Repeat Endoscopy vs Surgery

- 3473 pts with UGI bleeds (Tx with IV omeprazole 40mg bid, Tx for HP)
- 8.7% rebleeding after initial endoscopy (n = 92)
  - 48 pts re-treatment w endoscopic (injection & thermocoagulation)
  - 44 pts surgery with surgeon's choice of procedure

Surgery more likely to have complications

Normal Gastric Physiology

1. STS inhibits gastrin, ECL, parietal cell; gastrin, histamine, acid.
2. Gastrin stimulates parietal and ECL cells.
3. Acetylcholine stimulates parietal cell.

Role of Acid in Hemostasis

1) Impairs clot formation
   -impairs platelet aggregation & causes disaggregation

2) Accelerates clot lysis
   -predominantly acid-stimulated pepsin
Effect of Plasma pH on Platelet Aggregation


![Graph showing the effect of plasma pH on platelet aggregation.](image)
Effect of Gastric Juice on Clot Lysis

Lysis of a fibrin clot by gastric juice

Effect of H2 Receptor Antagonists on Gastric pH

- Increase intragastric pH
  - pH > 4.0 for 65-85% of day

- Tolerance develops rapidly (<24 h)
  - IV may have limited benefit in pts previously Tx with oral H₂RA
H2-Receptor-Antagonists

- meta-analysis 1984-2000
- IV H2-receptor antagonist vs placebo
- included 18-30 RCT

<table>
<thead>
<tr>
<th>Bleeding Peptic Ulcer</th>
<th>H2-RA</th>
<th>Placebo</th>
<th>RRR</th>
<th>ARR</th>
<th>NNT</th>
<th>OR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebleeding</td>
<td>20.4%</td>
<td>27.7%</td>
<td>26%</td>
<td>7.2%</td>
<td>14</td>
<td><strong>0.66</strong></td>
<td><strong>0.02</strong></td>
</tr>
<tr>
<td>Surgery</td>
<td>14.1%</td>
<td>20.8%</td>
<td>32%</td>
<td>6.7%</td>
<td>15</td>
<td><strong>0.61</strong></td>
<td><strong>0.02</strong></td>
</tr>
<tr>
<td>Mortality</td>
<td>5.8%</td>
<td>9.0%</td>
<td>35%</td>
<td>3.2%</td>
<td>32</td>
<td><strong>0.68</strong></td>
<td><strong>0.09</strong></td>
</tr>
</tbody>
</table>

**H2-Receptor-Antagonists**

<table>
<thead>
<tr>
<th>Bleeding Duodenal Ulcer</th>
<th>H2-RA</th>
<th>Placebo</th>
<th>RRR</th>
<th>ARR</th>
<th>OR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebleeding</td>
<td>23.9%</td>
<td>25.3%</td>
<td>6%</td>
<td>1.4%</td>
<td>0.90</td>
<td>0.48</td>
</tr>
<tr>
<td>Surgery</td>
<td>15.8%</td>
<td>19.8%</td>
<td>20%</td>
<td>4.0%</td>
<td>0.76</td>
<td>0.16</td>
</tr>
<tr>
<td>Mortality</td>
<td>5.0%</td>
<td>4.3%</td>
<td>-15.0%</td>
<td>-0.6%</td>
<td>1.15</td>
<td>0.57</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bleeding Gastric Ulcer</th>
<th>H2-RA</th>
<th>Placebo</th>
<th>RRR</th>
<th>ARR</th>
<th>OR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebleeding</td>
<td>20.4%</td>
<td>27.7%</td>
<td>26%</td>
<td>7.3%</td>
<td>0.66</td>
<td>0.02</td>
</tr>
<tr>
<td>Surgery</td>
<td>14.1%</td>
<td>20.8%</td>
<td>32%</td>
<td>6.7%</td>
<td>0.61</td>
<td>0.02</td>
</tr>
<tr>
<td>Mortality</td>
<td>5.8%</td>
<td>9.0%</td>
<td>35%</td>
<td>3.2%</td>
<td>0.68</td>
<td>0.09</td>
</tr>
</tbody>
</table>

- IV H2-RA provides **no benefit** for patients with bleeding DU
- limited role in decreasing rates of rebleeding & surgery in GU

- the authors suggest that PPI therapy has been shown to be more effective in the treatment of UGIB due to PUD as tolerance does not develop

- therefore they PPI therapy should be used as first line therapy
Effect of PPI on Gastric pH: Mechanism of Action

Covalently binds to **H+/K+ proton pump** of the parietal cell

Increase intragastric pH  
- pH > 6.0 for 84-99% of day

Proton pumps are continuously regenerated
- Bolus loading dose: inhibit activated pumps
- Continuous infusion: inactivates newly produced pumps

Decreased rebleeding and/or surgery with: **Bolus 80 mg + CI 8 mg/h**

No head-to-head trials of IV PPI vs PO

No reported tolerance
Effect of PPI on Gastric pH: Intermittent Bolus Vs. Continuous Infusion

Median % time 24-hour intragastric pH above indicated value after treatment with pantoprazole

- 80 mg + 8 mg/h
- 48 mg/h x 2 h + 8 mg/h
- 40 mg + 4 mg/h
- 40 mg q8h
- Placebo

Pantoprazole in Patients with Upper GI Bleeding after Endoscopic Hemostasis: 8 mg/h Infusion

80mg bolus then 8 mg/hour
N=14; 68% range

IV PPI bolus & CI: Decreases Rebleeding

240 pts successful endoscopic Tx w/in 24 hrs
- actively bleeding ulcers, NBVV, adherent clots
- epinephrine injection & thermocoagulation
- IV omeprazole (80-mg bolus & CI 8 mg/h) for 72 h v placebo


<table>
<thead>
<tr>
<th>Condition</th>
<th>OME IV CI (n=120)</th>
<th>Placebo (n=120)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebleeding</td>
<td>6.7%</td>
<td>22.5%</td>
</tr>
<tr>
<td>Surgery</td>
<td>2.5%</td>
<td>7.5%</td>
</tr>
<tr>
<td>Mortality</td>
<td>4.2%</td>
<td>10%</td>
</tr>
</tbody>
</table>

P<0.001

PO Omeprazole vs Placebo in Bleeding PUD


-NO therapeutic endoscopic was performed
-220 pts GU or DU
-stigmata of recent bleeding (arterial spurting, oozing, NBVW, adherent clot)

<table>
<thead>
<tr>
<th></th>
<th>OME PO 40 mg q12h x 5 d (n=110)</th>
<th>Placebo PO q12h x 5 d (n=110)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebleeding</td>
<td>10.9%</td>
<td>10%</td>
</tr>
<tr>
<td>Surgery</td>
<td>7.5%</td>
<td>23.6%</td>
</tr>
<tr>
<td>Mortality</td>
<td>1.8%</td>
<td>5.5%</td>
</tr>
</tbody>
</table>

P<0.001 P<0.001 P=NS
PO Omperazole vs Placebo in Bleeding PUD

-Javid G et al. AJM 2001 (India)

-therapeutic endoscopic (epinephrine & polidochanol)
-stigmata of recent bleeding (active bleeding, NBVV, adherent clot)

<table>
<thead>
<tr>
<th></th>
<th>OME PO 40 mg q12h x 5 d (n=82)</th>
<th>Placebo PO q12h x 5 d (n=84)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebleeding</td>
<td>7%</td>
<td>21%</td>
</tr>
<tr>
<td>Surgery</td>
<td>2%</td>
<td>9%</td>
</tr>
<tr>
<td>Mortality</td>
<td>1%</td>
<td>2%</td>
</tr>
</tbody>
</table>

p=0.02 p=0.17 p=0.98
RUBGE: Canadian Registry Novariceal UGI Bleeding and Endocopy

-18 sites community & tertiary hospitals
  -reflected “real life” setting

-observational study
-retrospective chart reviews on a sample of pts with non variceal UGIB
-1999-2002 with -30 day follow up
-1869 pts

Outcomes:
-continued bleeding, rebleeding
-surgery, mortality
-hospital stay
-endoscopy & medical therapy

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Age</td>
<td>66</td>
</tr>
<tr>
<td>Co-morbidities</td>
<td>2.5 / pts</td>
</tr>
<tr>
<td>Hemodynamic instability</td>
<td>30%</td>
</tr>
<tr>
<td>ASA score 4-5</td>
<td>14%</td>
</tr>
<tr>
<td>-systemic disease threat to life</td>
<td></td>
</tr>
<tr>
<td>-not expected to survive &gt; 24 hrs</td>
<td></td>
</tr>
<tr>
<td>ASA or NSAIDs</td>
<td>63.3%</td>
</tr>
<tr>
<td>Mean HBG</td>
<td>96</td>
</tr>
<tr>
<td>Mean INR</td>
<td>1.5</td>
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</table>
**RUGBE: Outcomes**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continued and/or rebleeding @ 30 days</td>
<td>14.1%</td>
</tr>
<tr>
<td>Surgery</td>
<td>6.5%</td>
</tr>
<tr>
<td>Mortality</td>
<td>5.4%</td>
</tr>
<tr>
<td>-mean age</td>
<td>72</td>
</tr>
<tr>
<td>-continued bleeding</td>
<td>38%</td>
</tr>
<tr>
<td>-rebleeding</td>
<td>13.3%</td>
</tr>
<tr>
<td>Mean length hospitalization</td>
<td>5.6 days</td>
</tr>
<tr>
<td>Discharged directly from ER</td>
<td>11.7%</td>
</tr>
</tbody>
</table>
RUBGE: Endoscopy

- endoscopy within 24 hrs 76%

- active bleeding 27%

Lesions:
- ulcers 56% (GU 47%, DU 42%, esophageal 11%)
- erosions 10%
- esophagitis 9%
- other

High risk endoscopic stigmata (active bleeding, NBVV)
- endoscopic therapy 74%

- injection alone 38%
- thermal therapy alone 23%
- injection & thermal therapy 34% **
- hemoclips 3%
- other 2%
<table>
<thead>
<tr>
<th>MEDICAL THERAPY</th>
<th>ENDOSCOPIC &amp; MEDICAL THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PPI</strong></td>
<td><strong>Endoscopy &amp; PPI</strong></td>
</tr>
<tr>
<td>IV or oral</td>
<td>ENDOSCOPY &amp; PPI 34%</td>
</tr>
<tr>
<td>IV</td>
<td><strong>Endoscopic Hemostasis</strong></td>
</tr>
<tr>
<td>oral</td>
<td><strong>94% PPI</strong></td>
</tr>
<tr>
<td><strong>H2-antagonist</strong></td>
<td><strong>Low risk stigmata</strong></td>
</tr>
<tr>
<td>24%</td>
<td>66.1% PPI</td>
</tr>
<tr>
<td><strong>PPI &amp; H2 antagonist</strong></td>
<td></td>
</tr>
<tr>
<td>7.1%</td>
<td></td>
</tr>
<tr>
<td>Octrotide or Somatostatin</td>
<td></td>
</tr>
<tr>
<td>7.1%</td>
<td></td>
</tr>
</tbody>
</table>
### RUBGE: Predictors of Rebleeding and Mortality

#### Rebleeding

<table>
<thead>
<tr>
<th>Health Status</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA 1/2 vs other</td>
<td>1.94</td>
</tr>
<tr>
<td>Rectal BRB</td>
<td>3.76</td>
</tr>
<tr>
<td>NGT BRB</td>
<td>2.55</td>
</tr>
<tr>
<td>Endoscopic High Risk Stigmata</td>
<td>4.81</td>
</tr>
<tr>
<td>PPI use</td>
<td>0.53</td>
</tr>
<tr>
<td>Endoscopic Tx high risk stigmata</td>
<td>0.39</td>
</tr>
</tbody>
</table>

#### Mortality

<table>
<thead>
<tr>
<th>Age by decade</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comorbidity &gt; 2 vs &lt; 2</td>
<td>2.92</td>
</tr>
<tr>
<td>Low SBP Per 10 mm Hg</td>
<td>1.46</td>
</tr>
<tr>
<td>PPI use</td>
<td>0.18</td>
</tr>
<tr>
<td>Endoscopic Tx high risk stigmata</td>
<td>0.31</td>
</tr>
</tbody>
</table>

**Conclusions:**
- this observation data suggests that PPI & endoscopic therapy have independent protective effects on rebleeding and mortality
- this needs to be explored in prospective studies
Risk Stratification

Goal: Determine if Patients are

- “LOW” or “HIGH” RISK for Rebleeding & Death

Based on Clinical & Endoscopic parameters

Multiple Risk Stratification Models (Rockwell)

Low Risk → discharged (young, healthy, no high risk stigmata)
   → admitted to floor

High Risk → ICU or Step down
### RISK STRATIFICATION: CLINICAL FACTORS

#### REBLEEDING
- > 65 years
- Shock
- Poor overall health status
- Comorbid illnesses
- Low initial hemoglobin level
- Melena
- Transfusion requirement - (> 6 u pRBC)
- Fresh red blood - rectal examination - emesis - nasogastric aspirate

#### MORTALITY
- > 60 years
- Shock
- Poor overall health status
- Comorbid illnesses
- Continued bleeding or rebleeding
- Fresh red blood - rectal examination - emesis - nasogastric aspirate
- Onset of bleeding while hospitalized for another reason
Risk Stratification: Endoscopic Predictors for Rebleeding

1) Stigmata of Bleeding

2) Ulcer Size  > 1 – 2 cm

3) Location  (posterior lesser gastric curve, posterior duodenal wall)
## Risk Stratification

**Table 1  The Rockall risk scoring system**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Score</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) Shock</td>
<td>&lt;60 “No shock”: pulse &lt;100 + systolic BP ≥100 mm Hg</td>
<td>60–79 “Tachycardia”: pulse ≥100 + systolic BP ≥100 mm Hg</td>
<td>≥80 “Hypotension”: systolic BP ≥100 mm Hg</td>
<td>Cardiac failure, ischaemic heart disease, any major comorbidity</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>No major comorbidity</td>
<td></td>
<td></td>
<td>Renal failure, liver failure, disseminated malignancy</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Mallory Weiss tear, no lesion identified and no SRH/blood</td>
<td>All other diagnoses</td>
<td></td>
<td>Malignancy of upper GI tract</td>
</tr>
<tr>
<td>Major SRH</td>
<td>None or dark spot only</td>
<td></td>
<td></td>
<td>Blood in upper GI tract, adherent clot, visible or spurting vessel</td>
</tr>
<tr>
<td>“Translation” of our comorbidity scale</td>
<td>No or mild coexisting illnesses (e.g. ECG abnormalities without symptoms)</td>
<td>Moderate coexisting illnesses (e.g. hypertension stable with medication)</td>
<td>Severe coexisting illnesses (diseases which need immediate treatment: e.g. cardiac failure)</td>
<td>Life threatening diseases (e.g. end stage malignancies, renal failure)</td>
</tr>
</tbody>
</table>

Major SRH, major stigmata of recent haemorrhage (active bleeding or visible vessel); GI, gastrointestinal; BP, blood pressure.
## Risk Stratification

### Table 2  Distribution of patients in the risk score groups, calculated with the Rockall risk score, for the Rockall validation sample and for our own patient group

<table>
<thead>
<tr>
<th>Risk score</th>
<th>Predicted probabilities*</th>
<th>Rockall’s validation sample</th>
<th>Vreeburg’s validation sample</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rebleeding (%)</td>
<td>Mortality (%)</td>
<td>Number of patients</td>
</tr>
<tr>
<td>0</td>
<td>4.9</td>
<td>0</td>
<td>48</td>
</tr>
<tr>
<td>1</td>
<td>3.4</td>
<td>0</td>
<td>131</td>
</tr>
<tr>
<td>2</td>
<td>5.3</td>
<td>0.2</td>
<td>142</td>
</tr>
<tr>
<td>3</td>
<td>11.2</td>
<td>2.9</td>
<td>162</td>
</tr>
<tr>
<td>4</td>
<td>14.1</td>
<td>5.3</td>
<td>176</td>
</tr>
<tr>
<td>5</td>
<td>24.1</td>
<td>10.8</td>
<td>199</td>
</tr>
<tr>
<td>6</td>
<td>32.9</td>
<td>17.3</td>
<td>137</td>
</tr>
<tr>
<td>7</td>
<td>43.8</td>
<td>27.0</td>
<td>96</td>
</tr>
<tr>
<td>8+</td>
<td>41.8</td>
<td>41.1</td>
<td>89</td>
</tr>
<tr>
<td>Total</td>
<td>18.9</td>
<td>10.0</td>
<td>1180</td>
</tr>
</tbody>
</table>

*Predicted probabilities based on observed percentages in original patient sample (Rockall, table V(B)\(^3\)).
Cost Effectiveness of IV PPI

It is current practice is to start IV PPI before endoscopy.

Decision analysis for cost-effectiveness of IV PPI.

**Asumptioins:**
all pts had endoscopy within 24 hrs

IV PPI only continued in those with high risk stigmata for rebleeding
-active bleeding, NBVV, adherent clot

costs were estimated from 2001 values.

Start IV PPI in all presenting to emergency department and endoscopy within 24 h

1000 Presenting to ER with UGI bleed

600 Peptic ulcer disease

216 High-risk stigmata

400 Other

384 Low risk

No benefit from IV PPI ∴ stop after ≤ 24 h

Evidence of reduced morbidity ∴ continue IV PPI (72 h)
Table 1. Values for health resource utilization in Canada

<table>
<thead>
<tr>
<th>Health resource utilization item</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital length of stay</td>
<td>5.7 days (s.d., 4.3)</td>
<td>CIHI hospital discharge data\textsuperscript{32}</td>
</tr>
<tr>
<td>for endoscopic treatment</td>
<td>N = 1114</td>
<td></td>
</tr>
<tr>
<td>Hospital length of stay</td>
<td>7.8 days (s.d., 3.4)</td>
<td>CIHI hospital discharge data\textsuperscript{32}</td>
</tr>
<tr>
<td>for suture ligation</td>
<td>N = 188</td>
<td></td>
</tr>
<tr>
<td>Hospital length of stay</td>
<td>12.5 days (s.d., 4.2)</td>
<td>CIHI hospital discharge data\textsuperscript{32}</td>
</tr>
<tr>
<td>for partial gastrectomy</td>
<td>N = 174</td>
<td></td>
</tr>
<tr>
<td>Requirements for anaesthesia units</td>
<td>8 units of 15 min</td>
<td>Expert opinion</td>
</tr>
<tr>
<td>for suture ligation</td>
<td>(min. 4; max. 12)</td>
<td></td>
</tr>
<tr>
<td>Requirements for anaesthesia units</td>
<td>10 units of 15 min</td>
<td>Expert opinion</td>
</tr>
<tr>
<td>for partial gastrectomy</td>
<td>(min. 5; max. 15)</td>
<td></td>
</tr>
<tr>
<td>Oral PPI — course of treatment</td>
<td>30 days</td>
<td>Product monograph</td>
</tr>
<tr>
<td></td>
<td>(min. 30; max. 45)</td>
<td></td>
</tr>
<tr>
<td>Unit cost</td>
<td>Canadian $</td>
<td>Source</td>
</tr>
<tr>
<td>--------------------------------------------------------------------------</td>
<td>------------</td>
<td>------------------------------------------------------------------------</td>
</tr>
<tr>
<td>IV PPI (first 24 h: 80-mg bolus + 8 mg/h continuous infusion)</td>
<td>128</td>
<td>St. Paul's Hospital, Vancouver, BC, Canada</td>
</tr>
<tr>
<td>IV PPI (2nd day: 8 mg/h continuous infusion)</td>
<td>92</td>
<td>St. Paul's Hospital, Vancouver, BC, Canada</td>
</tr>
<tr>
<td>IV PPI (3rd day: 8 mg/h continuous infusion)</td>
<td>92</td>
<td>St. Paul's Hospital, Vancouver, BC, Canada</td>
</tr>
<tr>
<td>Per diem hospitalization for endoscopic treatment</td>
<td>664</td>
<td>Derived using CIHI RIW™ and publication from IHE³³®</td>
</tr>
<tr>
<td>Per diem hospitalization for suture ligation</td>
<td>677</td>
<td>Derived using CIHI RIW™ and publication from IHE³³®</td>
</tr>
<tr>
<td>Per diem hospitalization for partial gastrectomy</td>
<td>989</td>
<td>Derived using CIHI RIW™ and publication from IHE³³®</td>
</tr>
<tr>
<td>Endoscopic treatment professional fee</td>
<td>140</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Suture ligation professional fee</td>
<td>511</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Partial gastrectomy professional fee</td>
<td>743</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Vagotomy professional fee</td>
<td>48</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Gastroenterologist initial consultation</td>
<td>125</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Gastroenterologist follow-up visit</td>
<td>21</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Surgical initial consultation</td>
<td>95</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Surgical follow-up consultation</td>
<td>16</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Anaesthesiologist initial consultation</td>
<td>65</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Anaesthesiologist follow-up visit</td>
<td>22</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Anaesthesiologist fee (per 15 min)</td>
<td>29</td>
<td>Provincial physician fees payment schedule³⁴®</td>
</tr>
<tr>
<td>Oral PPI (pantoprazole 20-mg tablet)</td>
<td>2.32</td>
<td>Publication from IMS HEALTH³⁵®</td>
</tr>
</tbody>
</table>
incremental saving favouring IV PPI with
- $37,840 saved with 40 re-bleeds prevented
- negative incremental ratio of ($946) per re-bleed averted

cost-effectiveness ratio become positive at $105 per re-bleed averted only when baseline probability of first re-bleed without IV PPI was lowered by 20%
Cost Effectiveness of IV PPI

several limitations
1) IV PPI discontinued in all patients without high-risk endoscopic stigmata

2) all patients received endoscopy on average 24 h after presentation
   - with mean time to endoscopy of 36 h IV PPI was cost neutral with better outcomes

2) all patients with lesions other than PUD with high-risk endoscopic stigmata would gain no benefit from acid suppression

Conclusion:
based on its cost-effectiveness authors suggested routine administration of IVPPI to all patients presenting to Canadian emergency rooms with UGIB should be considered as standard practice.
Cost Effectiveness of IV PPI vs High dose Oral PPI

To compare cost-effectiveness of
  IV PPI  (Pantoloc 80 mg bolus then 8 mg/hr x 72 hrs)
  oral high-dose PPI  (Pantoloc 40 mg PO bid x 5 days, then OD)
  placebo administration

-decision tree for high-risk stigmata of bleeding

Re-bleeding rates
  IV PPI  5.9%
  High dose oral PPI  11.8%
  Placebo  27%

Mean lengths of stay & cost
  pts with rebleeding  4.7 days  $11,802
  pts without rebleeding  3.0 days  $7993

Conclusion:
IV PPI more effective and less costly than high dose oral PPI with incremental savings of $136.40 per patient treated (US based costs)

Peptic Ulcer Bleeding : Summary

1) Resusitation / ABCs, Cardiac Workup
   Correct coagulopathies, Transfuse as required

2) IV Pantoloc 80 mg bolus, then 8 mg/hr

3) Endoscopy with 24 hrs (Ideally)

4) Treat High Risk Stigmata (Active Bleeding, NBV, +/- adherent clot)
   Use Bimodal Therapy (injection & coagulation)

5) High Risk Stigmata Continue with IV PPI x 3 days \(\rightarrow\) PO
   Low Risk Stigmata High dose Orally PPI (bid)

6) Rebleeding \(\rightarrow\) repeat Endoscopy

7) Uncontrolled bleeding \(\rightarrow\) Surgery

8) Treat H. pylori, d/c NSAIDs if possible

6) Risk Stratification \(\rightarrow\) Low or High Risk (Rebleeding, Death)
Variceal Bleeding

- 10-30% of all UGI bleeds

- cirrhotic pts have bleeding due may be due to other causes as well
  - portal hypertensive gastropathy
  - non-variceal mucosal abnormalities

- mortality index bleed 30-50% w/in 6 wks

- highest risk of rebleeding occurs in the first 6 weeks after index bleed
  - then the risk returns to baseline

- 70% re-bleed @ 1 yr

- 1 yr survival 32-80%
Risk Factors for Variceal Bleeding: 1) HVPG

1) **HVPG** hepatic vein pressure gradient
   
   = wedged Hepatic Portal Vein Pressure – Free Hepatic Portal Vein Pressure
   
   = WHPVP – FHVP

   >= 12 mm Hg is required for **variceal formation**
Risk Factors for Variceal Bleeding: 2) Size

“SIZE DOES MATTER!!”

Laplace's law:
Wall tension = \( (\text{radius} \times \text{transmural P}) \)

\[ \frac{\text{wall thickness}}{\text{radius}} \]

\[ \uparrow t_1 = \rho \cdot \frac{r_1}{W_1} \]
\[ \uparrow t_2 = \rho \cdot \frac{r_2}{W_2} \]

Varix
Mucosa
Muscularis mucosa
Lamina propria
Submucosa

\( W_1 \) (thinnest wall)
\( W_2 \) (thickest wall)
Risk Factors for Variceal Bleeding: 2) Size

I. Dilated veins (< 5mm) still @ level of surrounding tissue

II. Dilated, straight veins (> 5 mm) protruding into the esophageal lumen but not obstructing it

III. Large, tense & winding veins already obstructing the esophageal lumen considerably

IV. near complete obstruction of the esophageal lumen
## Risk Factors for Variceal Bleeding: 3) Childs-Pugh Score

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bilirubin</strong></td>
<td>34</td>
<td>35-51</td>
<td>&gt;51</td>
</tr>
<tr>
<td><strong>Albumin</strong></td>
<td>&lt;28</td>
<td>28-35</td>
<td>&gt;35</td>
</tr>
<tr>
<td><strong>INR</strong></td>
<td>&lt;1.7</td>
<td>1.7-2.3</td>
<td>&gt;2.3</td>
</tr>
<tr>
<td><strong>Encephalopathy</strong></td>
<td>None</td>
<td>Grade 1-2</td>
<td>Grade 3-4</td>
</tr>
<tr>
<td><strong>Ascites</strong></td>
<td>Absent</td>
<td>Slight</td>
<td>Moderate</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Survival</th>
<th>1 yr</th>
<th>2 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>A 5-6</td>
<td>100</td>
<td>85</td>
</tr>
<tr>
<td>B 7-9</td>
<td>80</td>
<td>60</td>
</tr>
<tr>
<td>C 10-15</td>
<td>45</td>
<td>35</td>
</tr>
</tbody>
</table>
Risk Factors for Variceal Bleeding: 4) Endoscopic Stigmata

**Red wale marks:**
- longitudinal red streaks

**Cherry red spots:**
- discrete spots flat & overlie varices

**Hemocystic spots:**
- raised discrete red spots,
  - "blood blisters"

**Diffuse erythema:**
- denotes diffuse red color
Management of Esophageal Varices

1) Acute Management

2) Primary Prophylaxis

3) Secondary Prophylaxis

**Treatment Options:**
- Medical
- Endoscopic
- Surgical
Acute Variceal Bleeding: Resuscitation

- emergency → usually hemodynamic compromise
- ?? 40% stop spontaneously

**Resuscitation:**

ABC  
Air way protection (++) Hematemesis  
Consider early intubation before endoscopy

- coagulopathy  
elevated INR → FFP
- thrombocytopenia  
transfuse platelets
- renal dysfunction  
consider DDAVP
- caution with over transfusion  
- can increase portal pressure  
- goal Hbg < 110
Acute Variceal Bleeding : Pharmacology

1) IV PPI 80 mg bolus, then 8 mg/hr x 3 days
   -can have causes of UGIB other than varices

2) IV octreotide 50 mcg bolus, then 50 mcg/hr x 5 days

   **Mechanism of action → Decreases Portal Pressure:**

   - decreases splanchnic blood flow

   - inhibits vasoactive peptides (i.e. substance P or glucagon)
Octreotide for Acute Esophageal Variceal Bleeding

-meta-analysis 13 studies (1077 pts)
-Octreotide had lower incidence of rebleeding after endoscopy vs:

-vasopressin or terlipressin \( \text{RR} 0.58 \)
-placebo or no therapy \( \text{RR} 0.46 \)

-Decrease in rebleeding by 37% \( \text{NNT} = 8 \)

-There was no effect on mortality \( \text{RR} = 0.89 \)
-Use for 5 days

Acute Variceal Bleeding: Endoscopy

-achieves initial hemostasis in 80-90% of variceal bleeds

1) **Sclerotherapy**
   - ethanol
   - polidocanol
   - sodium tetradecyl sulfate

2) **Variceal Band Ligation**
   - now the standard of care
   - easier to perform
   - less complications
Variceal Banding
Endoscopic Band Ligation (EBL) versus Sclerotherapy (EVS)

-Meta-analysis comparing endoscopic ligation vs sclerotherapy
-7 studies (n=547)
-rates of initial hemostasis were the same OR 1.14

<table>
<thead>
<tr>
<th></th>
<th>EBL (%)</th>
<th>EVS (%)</th>
<th>Odds Ratio</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>rates of rebleeding</td>
<td>31</td>
<td>46</td>
<td>0.52</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>rebleeding caused by varices</td>
<td>21</td>
<td>36</td>
<td>0.47</td>
<td>0.004</td>
</tr>
<tr>
<td>mortality</td>
<td>24</td>
<td>32</td>
<td>0.67</td>
<td>0.05</td>
</tr>
<tr>
<td>mortality caused by bleeding</td>
<td>7</td>
<td>14</td>
<td>0.49</td>
<td>0.05</td>
</tr>
<tr>
<td>esophageal stricture</td>
<td>0</td>
<td>10.9</td>
<td>0.10</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Endoscopic Band Ligation (EBL) versus Sclerotherapy (EVS)

**Variceal obliteration:**

- 2.2 fewer endoscopic treatment sessions to achieve variceal obliteration with EBL vs EVS (3-4 vs 5-6 sessions)

- complications:
  - bleeding caused by treatment-induced ulcerations
  - pulmonary infection, bacterial peritonitis
  - complications leading to death

**Conclusion:**

EBL is superior to EVS with respect to decreased:
  - rates rebleeding
  - mortality
  - sessions required for variceal obliteration

EBL versus EBL & Octreotide

- 94 pts with esophageal variceal bleeding
- EBL (n = 47) or
- EBL & octreotide (50 mug IV bolus then CI at 50 mug/h x 5 days (n = 47)

Combination EBL & Octreotide decreases recurrent bleeding and need for balloon tamponade.

Sung et al. Lancet. 1995
Sengstaken-Blakemore Tube

- Helmet or Catcher’s Mask
- Openings to Balloons and Tubes
- Balloon in Esophagus
- Balloon in Stomach
- Tube to remove fluid and air from Stomach
Acute Variceal Bleeding: Last Resorts

**TIPS (Transjugular Intrahepatic Portosystemic Shunt)**

- vascular placement of an expandable metal stent across a tract created between hepatic vein & major intrahepatic branch of the portal system
- decreases portal wedge pressure
- complicated by infection & encephalopathy
- usually a bridge to transplant
- controlled bleeding 93% → 12% early rebleeding (15 RCT)

**Surgery**
- portosystemic shunting
- esophageal staple transection
  - +/- esophageal-gastric devascularization
- morbidity is high with 30-day mortality associated with emergency surgery approaches 80%
Ascites Management: Surgical Shunts

- popularized in 1970’s

**Rarely used since advent of TIPS**
- poor long-term patency
- excessive complications
- makes liver transplantation difficult
  - shunt-related adhesions

**Examples:**

Fig A  Porto-caval Shunt

Fig B  Spleno-renal Shunt
UGI Bleeding & Cirrhosis: Antibiotic Prophylaxis

- Meta-analysis of 8 RCTs (864 pts)
- Cirrhosis with UGIB
- Antibiotics vs placebo or no treatment

<table>
<thead>
<tr>
<th>Condition</th>
<th>RR</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality from any cause</td>
<td>0.73</td>
<td>0.02</td>
</tr>
<tr>
<td>Mortality from bacterial infections</td>
<td>0.31</td>
<td>0.06</td>
</tr>
<tr>
<td>Prevention of bacterial infections</td>
<td>0.40</td>
<td>0.01</td>
</tr>
<tr>
<td>Prevention of bacteremia</td>
<td>0.26</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Acute Variceal Bleeding: Summary

1) Octreotide decreases rebleeding vs placebo.

2) EBL superior to EVS w/r rebleeding and mortality with less complications.

4) EBL in combination with Octreotide is better than EBL alone

5) Prophylactic antibiotics reduce mortality & rates of infection

6) Rerfractory Bleeding: Blakemore or TIPS

ALL CIRROHTICS WITH VARICEAL BLEEDING SHOULD RECEIVE:

- Octreotide IV (50 mug bolus then 50 mug /hr)
- urgent endoscopy (EBL or EVS if EBL is unsuccessful)
- prophylactic antibiotics
Acute Lower GI Bleeding

ABCs, Resuscitation

Rule out **significant UGIB**
- Hx melena, hematemesis
- NG tube
- if hemodynamically unstable, significant drop in HBG

**Causes of Brisk LGIB:**
1) Diverticulosis
2) Angiodysplasia (elderly, AS, CRF, Scleroderma, HHT)
3) Colon Cancer
4) Ischemic Colitis (LLQ pain → then bloody diarrhea)

>85% pts are stable → self limiting
15% ongoing significant bleeding
Acute Lower GI Bleeding

URGENT COLONOSCOPY FOR THE DIAGNOSIS AND TREATMENT OF SEVERE DIVERTICULAR HEMORRHAGE
JENSEN et al. NEJM 2000

121 pts acute LGIB
All received 4-6 L Golytely PO or NG over 5-6 hrs
Urgent Colonoscopy 6-12 hrs

Able to diagnosis a Definite Diverticular cause of bleeding 21-23%
(active bleeding, NBVV or clot)

Of 10 pts treated endoscopically → none rebled
Acute Lower GI Bleeding

**Treatment Options for SIGNIFICANT LGIB:**

1) Colonoscopy  usually only done electively
   acutely visualization is extremely poor
   multiple clots are seen throughout

2) Angiogram  (embolization → polyvinyl alcohol or microcoils)
   0.5 cc/min

3) RBC Scan  localization of bleeding, for obscure bleeding
   0.1-0.4 cc/ min

4) Colectomy  get your friendly neighbourhood general surgeon
   involved early