SURGERY FOR BENIGN DISEASE OF THE UPPER GASTROINTESTINAL TRACT

H. DAVID REINES, M.D. FACS
VCU School of Medicine Inova Campus
Inova Fairfax Hospital

BENIGN GASTRIC DISEASE

• Anatomy  LUQ cross midline. Proximal=cardia mucous glands, Corpus(body) parietal and chief (pepsin) cells antrum Incisura (lesser curve) to gastroepiploic entrance (greater curve) G- cells (gastrin) pylorus distal, muscle

• Blood supply  L gastric= branch of celiac lesser curve becomes continuous with R gastric from common hepatic. Gastroduodenal gives off R gastroepiploic, Splenic  L Gastroepiploic
GASTRIC PHYSIOLOGY

- Acid production- body and fundus by parietal cells. ATP expending proton pump generates substantial concentration gradients. Concentrate HCL 10 million times blood!!
- Stomach has protective mechanisms tight junctions, luminal bicarbonate(minor), alkaline mucous next to epithelium
- Inverse relationship acid to pH. Higher pH, lower HCL vomit acid, get alkalotic (Hypochloremic, hypokalemic)

GASTRIC PHYSIOLOGY

- Signals for acid secretion- Vagal increase acetylcholine near parietal cells to increase calcium, increase acid. Also stimulates antral neurons to release gastrin releasing peptide (GRP=Bombesin), reaches parietal cells, stimulates acid. Gastrin release from antrum when pH<3. If cut vagus, leave antrum, gastrin secreted and stimulates acid; therefore, need both vagotomy and antrectomy

GASTRIC PHYSIOLOGY

- Appetite stimulants
- Ghrelin - in stomach, levels go up between meals, go down post gastric bypass - may be key to appetite

Peptic Ulcer Disease

- Interaction cell types on acid secretion

Peptic Ulcer-regulation acid

- Pathophysiology

Peptic Ulcer Disease

- Interaction cell types on acid secretion

Pathophysiology
Peptic Ulcer Disease

Pathophysiology

Gastric Ulcer - differ from duodenal lesions - more defect in protection than from acid. Not all gastric ulcers need acid (can arise in achlorhydric stomach).

- Type I ulcer - lesser curve
- Type II - midbody with duodenal or pre-pyloric ulcers (type III), High at GE junction (type IV).

GASTRIC PEPTIC DISEASE

- Defect unknown - prostaglandins play a role, NSAIDs cause ulcers, prevented by misoprostol, COX 2 drugs
- Malignant gastric ulcers look like benign - therefore all ulcers need biopsy
- Any ulcer not healing in 6 weeks = surgery
- Giant ulcers > 5 cm will not heal
- Surgical Rx = wedge resection for many ulcers

DUODENAL PEPTIC DISEASE

- Duodenal ulcer - "no acid, no ulcer"
- Thought to be overproduction of acid and failure of protective mechanisms
- Role of Helicobacter pylori - microaerophilic gram-negative rod adapted to acid. Infection rates > 50%. 95% duodenal ulcer patients, 50-80% gastric ulcer patients.
- Eradication of bacteria with acid reduction necessary

TABLE 22.1 PATHOGENESIS OF PEPTIC ULCER

- Endocrine consequences
- Increased basal serum gastrin
- Increased gastrin response to a meal
- Increased secretion of gastrin-releasing peptide
- Increased density of somatostatin cells
- Decreased mucosal vasoactive intestinal peptide
- Gastric acid secretion
- Increased acid secretory capacity
- Increased basal secretion
- Increased pentagastrin-stimulated output
- Increased renal impairment
- Abnormal gastric emptying
- Microcellular defense
- Decreased duodenal bicarbonate production
- Decreased gastric mucosal prostaglandin production

ENVIRONMENT
- Cigarette smoking
- Nonsteroidal anti-inflammatory drugs

PEPTIC ULCE DISEASE

- Diagnosis - Gastric Ulcer - differ from duodenal lesions - more defect in protection than from acid. Not all gastric ulcers need acid (can arise in achlorhydric stomach).
- Type I ulcer - lesser curve - type II - midbody with duodenal or pre-pyloric ulcers (type III), High at GE junction (type IV).
**Helicobacter pylori**

- Diagnosis: organisms on biopsy, culture, breath test for ammonia, Elisa for antibody (remains positive 50% time post Rx)
- Only 10-20% of patients with H.pylori gastritis develop ulcers
- Conclusion: important, not the only cause

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**Peptic Ulcer Disease**

- Conservative therapy
  - Treat the acid, treat the bacteria, stay off the wrong drugs
- Antacids
  - Work locally, quickly, non curative
- H2 blockers
  - Block histamine and decrease acid, but still other mechanisms. Control symptoms, not cure.
- Proton Pump inhibitors
  - Very effective
- H.pylori eradication
  - Amoxicillin and omeprazole x 2 weeks or bismuth/Flagyl!

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**Ulcer-treatment medical**

- Conservative
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**COMPLICATIONS-PEPTIC**

- Bleeding
  - DDX=Peptic, NSAID, Mallory Weiss (tear of proximal stomach), Dieulafoy (submucosal artery erodes), esophageal varices, gastritis, stress ulcer, aorto duodenal fistula, angiodysplasia of stomach/small bowel
- Perforation
- Gastric outlet obstruction

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**Peptic Ulcer Disease**

- Endoscopy necessary for bleeding-only 10-20% bleeding from ulcer, however 50% severe bleeding is peptic
- Endoscopic treatment of bleeding
- Bipolar Electrocoagulation - useful
- Injection therapy - Epinephrine - simple
- Laser - not as effective long term
ENDOSCOPY-BLEEDING

- NIH CONSENSUS: Predictors of outcome from bleeding
  - 60–70% stop spontaneously
  - Factors: Magnitude of bleeding, hemodynamic instability, bloody emesis, blood red stools
  - Host factors: anticoagulation
  - Endoscopic: visible vessel, arterial spurting, adherent clot on ulcer base
  - Flat pigment: low risk rebleed

GI BLEEDING-Forrest classify

- 1a: spurting blood
- 1b: non-spurting active bleeding
- 11a: visible vessel
- 11b: nonbleeding ulcer with clot
- 11c: ulcer with hematin-covered base
- 111: clean ulcer base

Peptic Ulcer Disease-Surgery

- Bleeding: greater than 6 units blood, shock, failure of endoscopy, bleeding vessel on endoscopy, unstable patient
- Diagnosis: clinical
  - Hematemesis, melena, hypotension
  - “The most common cause of lower GI bleeding is upper GI bleeding”
- No patient should die of bleeding in the endoscopy suite
Peptic Ulcer- Surgery

- Procedures
- Oversew (bleeding)
- Closure of perforation - Graham patch
- Pyloroplasty - render the pylorus incompetent
- Heineke-Muclicz
- Jaboulay
- Other
Peptic Ulcer-Surgery

- Vagotomy
- Truncal
- Selective
- Highly selective

Peptic Ulcer-Surgery

- Gastric resections
- Wedge resection
- Antrectomy/vagotomy (V/A)
- Subtotal gastrectomy
- Total gastrectomy

Table 22.4. CLINICAL RESULTS OF DUODENAL ULCEr SURGERY

<table>
<thead>
<tr>
<th></th>
<th>PGV (%)</th>
<th>TV + P (%)</th>
<th>TV + A (%)</th>
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</thead>
<tbody>
<tr>
<td>Mortality rate</td>
<td>0</td>
<td>0.5-1</td>
<td>1-2</td>
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<tr>
<td>Acid reduction</td>
<td>Basal</td>
<td>Basal</td>
<td>Basal</td>
</tr>
<tr>
<td>Stimulated</td>
<td>90</td>
<td>70</td>
<td>85</td>
</tr>
<tr>
<td>Ulcer recurrence</td>
<td>10</td>
<td>12</td>
<td>1-2</td>
</tr>
<tr>
<td>Gastric emptying</td>
<td>Liquids</td>
<td>Solids</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Accelerated</td>
<td>No change</td>
<td>Accelerated</td>
</tr>
<tr>
<td>Dumping</td>
<td>Milk</td>
<td>&lt;5</td>
<td>10</td>
</tr>
<tr>
<td>Disabling</td>
<td>Diarrhea</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Nausea</td>
<td>&lt;5</td>
<td>25</td>
</tr>
<tr>
<td>Nausea</td>
<td>Diarrhea</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

PGV: proximal gastric vagotomy; TV + P: truncal vagotomy and pyloroplasty; TV + A: truncal vagotomy and antrectomy.
COMPLICATIONS-SURGERY

- Postgastrectomy syndromes-1-3%
- Dumping - Clinical syndrome may be VIP mediated. Dizziness, palpitations, diarrhea, pain following a meal
- Alkaline Reflux gastritis - postprandial pain, evidence bile in stomach, endoscopic gastritis
- Gastric stasis

STRESS ULCER

- Etiology-probably ischemia and acid-seen in ICU patients on ventilators, septic, liver failure
- Small numerous superficial lesions
- Curlsings ulcer-stress ulcers and burns
- Cushings ulcer-stress ulcer and head injury or craniotomy
- Diagnosis-heme positive NG aspirate in an ICU patient-need endoscopy

STRESS ULCER

- Indications for prophylaxis older patient on ventilator, severe burns, head injury (spine also), hepatic failure, coagulopathy
- NOT EVERYONE IN THE ICU NEEDS PROPHYLAXIS.
- Enteral feeding appears protective
- Numerous studies prevention too much antacid may cause nosocomial pneumonia from aspiration. Sucralfate does not neutralize acid, coats ulcers. H2 blockers effective- incidence low

ZOLLINGER-ELLISON SYNDROME

Physiology in 1955 Zollinger and Ellison described 2 patients with severe peptic ulcer disease and pancreatic endocrine tumors.
- 75% sporadic, 25% MEN Most are benign and curable. Recurrent peptic ulcers 90% patients, diarrhea 10%
- Gastrinoma- fasting gastrin at least 200 pg/ml. Causes hyperchlorhydria or ulcers. Fasting gastrin up in states because gastrin normal from antral G cells. Need provocative tests secretin stimulation

ZOLLINGER-ELLISON SYNDROME

<table>
<thead>
<tr>
<th>Diagnosis</th>
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<tr>
<td><strong>Table 33.3. GASTRINOMA</strong></td>
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<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
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<tbody>
<tr>
<td>Symptoms</td>
<td>Peptic ulcer disease</td>
</tr>
<tr>
<td></td>
<td>Diarrhea</td>
</tr>
<tr>
<td></td>
<td>Esophagitis</td>
</tr>
<tr>
<td>Diagnostic tests</td>
<td>Serum gastrin measurement</td>
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<tr>
<td></td>
<td>Gastric acid analysis</td>
</tr>
<tr>
<td></td>
<td>Secretin stimulation test</td>
</tr>
<tr>
<td>Anatomic localization</td>
<td>Duodenum and head of pancreas (gastrinoma triangle)</td>
</tr>
</tbody>
</table>
ZOLLINGER-ELLISON SYNDROME

- Selective arterial secretin stimulation rise in hepatic vein after injection
- SMA, GDA, Splenic SPL

GASTRIN STIMULATION -
triangle gastritis
square gastric outlet
circle Z E
- positive increase in basal>200 pg/ml

WHAT IS THE MOST COMMON DISEASE IN THE USA?

It is not Pheochromocytoma.

Treatment used to be total gastrectomy to eliminate source of acid and gastrin, but leaves tumor intact. If localized, local resection of tumor. PPI reduced need for gastrectomy, especially if localization of tumor. Occasionally blind resection based on chemistry

- Results are 60-70% undergoing resection.
- Metastatic gastrinoma is still fatal
WHY BARIATRIC SURGERY?

- OBESITY is the most common disease in the US.
- What is the treatment for it?
- Why do we spend so much time on pheochromocytoma and so little on obesity?
- Are you prejudiced?

BARIATRIC SURGERY

- Definitions
  - BMI = Body Mass Index = Kg/M2
- Overweight -> 27 BMI
- Obese > 30 BMI
- Morbid (severe or Type II) obesity > 39 BMI
- What are alternatives? - diet, exercise, vomiting
- Failure rate diet exercise 90% at 5 years
- Surgical Indications - Morbid obesity or BMI > 35 with co-morbidities, fail diets

BARIATRIC SURGERY CO-MORBIDITIES

- Nonalcoholic steatohepatitis (NASH)
- Diabetes - Type II 30%
- GERD - 30-50%
- Hypertension, cardiac dysfunction
- Osteoarthritis - DJD
- Sleep Apnea, hypoventilation
- Stress Incontinence
- Venous stasis disease
BARIATRIC SURGERY

- Procedures - Restrictive, malabsorptive, combination: open or minimal invasive
- Gastroplasty, banded
- Gastroplasty with sleeve gastrectomy
- Gastric bypass - proximal, distal
- Biliary pancreatic diversion-duodenal switch
- Lap adjustable band

BARIATRIC SURGERY COMPLICATIONS

- Death 0.2%-bak, pulmonary emboli, bleeding
- Leak 0.6%-2%
- Wound infection 5%(open)
- Ulcers/strictures 5-10%
- Gall stones
- Hernia 10-20%(open)
- Vitamin deficiency, B12, iron?thiamin, Ca++
- Protein malnutrition
BARIATRIC SURGERY

- Results: WHAT OTHER OPERATION CAN CURE OR HELP SO MANY DISORDERS?
- Average weight loss: 60% excess weight (100+ lbs.). Stays off at 5 years in 85%
- Cures or helps: Diabetes 85-90%
- Sleep apnea, hypertension (50%)
- Stress incontinence, hypoventilation
- Pseudotumor cerebri

BARIATRIC SURGERY vs CABG

- CABG helps angina and prevents further cardiac damage from ischemia
- CABG-does not help diabetes, stress incontinence, hypertension, sleep apnea, body image, depression
- SO WHY DO WE SUPPORT CABG, BUT NOT GASTRIC BYPASS??

PREDJUDICE!!

"I would rather marry a felon than a fat person."

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