Stomach and Duodenum

2007-2008 Student Lecture Series
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Anatomy

- Appears in week 5.
- A pliable, saccular organ.
- Located in the LUQ and epigastrium.
- Separated from the GI tract (2 locations).
Gross Anatomy

- **Proximal** = Cardia (attaches to esophagus) attaches at the LES.
- **Fundus** = most superior portion, receives food.
- **Body** = largest portion, contains parietal, chief and ECL cells.
- **Distal** = antrum, contains the G cells.
Anatomy
• The stomach is almost entirely covered with peritoneum.
• The peritoneum forms the outer gastric serosa.
• Beneath the serosa is the muscularis propria.
• The MP is made up of 3 layers of smooth muscle.
  • The middle layer is the circular muscle and is the only “complete” layer of muscle
• As you progress distally the middle layer of muscle begins to thicken and form the __________? Which functions as a true sphincter.

• This and the GE junction form the gastric “borders” and are the two “fixed” points of the stomach.

• The outer muscle layer (longitudinal) is contiguous with the outer layer of the esophagus.
• Within the layers of the MP, there is a rich plexus of autonomic nerves and ganglia called__________________.

• The submucosa lies between the MP and the mucosa. It is a collagen rich layer of connective tissue and is the weakest/strongest layer of the gastric wall.

• The submucosa also contains the rich blood vessel network and the lymphatics as well as Meissner’s plexus.
• The mucosa consists of 3 layers:
  • Surface epithelium (columnar).
  • Lamina propria
    • Connective tissue layer that supports the surface epithelium.
  • Muscularis mucosae (probably the reason for rugal folds).
    • The MM is the boundary for invasive/noninvasive gastric cancer.
Anatomy/Morphology
Anatomy/Morphology

Gastric pit

Surface mucous cells

Isthmus

Parietal cells

Mucous neck cells

Neck

ECL cell

Base

Chief cells

Gastric gland

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Cell Types

- **Parietal:**
  - Location: Body
  - Function: secrete acid and intrinsic factor

- **Mucus:**
  - Location: Body, Antrum
  - Function: mucus production

- **Chief:**
  - Location: Body
  - Function: produce Pepsin
Cell Types

- **Surface epithelium:**
  - **Location:** Diffuse
  - **Function:** produce mucus, bicarb, prostaglandins (?)

- **ECL:**
  - **Location:** Body
  - **Function:** Histamine production

- **G cells:**
  - **Location:** Antrum
  - **Function:** Gastrin production
Cell Types

- **D cells:**
  - Location: Body, Antrum
  - Function: produce Somatostatin

- **Gastric mucosal interneurons:**
  - Location: Body, Antrum
  - Function: produce Gastrin-releasing peptide

- **Entric Neurons:**
  - Location: Diffuse
  - Function: CGRP, others production
Vascular Supply

- R&L gastrics
- R&L gastroepiploics
- Short gastrics
- Inferior phrenics
- Gastroduodenal

Venous drainage:
- R&L gastric veins drain to the portal, R gastroepiploic drains to the SMV, L ge drains to the splenic
Nerve supply
Gastric Physiology

• **Principle Function:**
  • Storage:
    • Receptive relaxation
  • Start digestion:
    • Separates meal into fat/protein/carbohydrates
Regulation of Function

- The stomach is under both neural and hormonal control.
**Gastric Hormones**

- **Gastric Hormones:**
  - Chemical messengers that regulate intestinal and pancreatic function.
  - The “gut” is the largest endocrine organ in the body.
  - The messengers can act as:
    - Endocrine: distant target
    - Paracrine: close target
    - Autocrine: self target
    - Neurocrine: neurotransmitter or stimulator.
Gastric Hormones

- Gastric Hormones:
  - Synthesized as inactive precursors
  - Converted to active form by post-translational modification
  - #1 stimulus for release is: FOOD
    - Composition of food dictates timing and specific hormone release.
Gastric Hormones

- Gastric Hormones:
  - Inhibition:
    - Removal of stimulus
    - Negative feed-back loops
    - Inhibitory peptides, ie. Somatostatin
Gastric Hormones

- Gastrin
- Somatostatin
- Gastrin-releasing peptide (GRP)
- Histamine
Gastrin

- **Synthesis:** G-cells in the antrum
- **Release:**
- **Inhibition:**
  - pH < 3.0, somatostatin, secretin, CCK, VIP, GIP, glucagon.
- **Target cells:**
  - Parietal and Chief cells
Gastrin

- **Action(s):**
  - Stimulates acid secretion
    - Direct action on parietal cells
    - Potentiating interaction with histamine
    - Possible: releasing of histamine
  - Increases release of lytes & water from stomach, pancreas, liver and Brunner’s glands
  - Stimulates motility in stomach, intestine, and gall bladder
  - Inhibits contraction of pylorus and sphincter of Oddi.
  - Stimulates GI mucosal growth.
Somatostatin

- Tetradecapeptide
- Synthesis:
  - CNS, antrum, fundus, sm. bowel, colon, and D-cells in pancreas.
- Release:
  - Antral acidification
  - Fats, protein, acid in duodenum
  - Pancreatic: glucose, amino acids, CCK
- Inhibition:
  - Release of acetyl-choline from vagal nerve fibers
Somatostatin

- **Action(s):**
  - The “master off switch”
  - Inhibits the release of most GI hormones
  - Inhibits pancreatic and GI secretion(s)
  - Inhibits intestinal motility.

- **Clinical:**
  - Octreotide- decrease fistula output
  - Treatment of esophageal variceal bleed
  - Can ameliorate symptoms of endocrine tumors
GRP

- Mammalian equivalent of Bombesin
- Synthesis:
  - Gastric antrum, small bowel mucosa
- Release: vagal stimulation
GRP

• **Action(s):**
  • The “master on switch”
  • Stimulates the release of all GI hormones (? Secretin).
  • Stimulates GI secretion
  • Stimulates GI motility
  • * most important: stimulates gastric acid secretion and release of antral gastrin
  • Stimulates bowel and pancreatic mucosal growth and stimulates various GI and pancreatic CA’s
Histamine

- Stimulates parietal cells
- Found in the acidic granules of ECL cells and resident Mast cells.
- Release is stimulated by:
  - Gastrin, acetyl-choline, epinephrine
- Inhibited by Somatostatin.
- ? A necessary intermediary of acid production.
Acid Secretion

- Two forms:
  - Basal Acid Secretion
  - Stimulated Acid Secretion
Stimulated Acid Secretion

- Three Phases:
  - Cephalic phase
  - Gastric phase
  - Intestinal Phase

- These phases occur *concurrently* NOT consecutively.
Cephalic Phase

- Originates with the sight, smell, thought or taste of food.
- Stimulates the cortex and hypothalamus.
- Signals cause Vagus to release Ach, Ach causes increase in parietal cell acid production.
- Accounts for 20-30% of acid production.
Gastric Phase

• Begins when food enters the gastric lumen (gastric distention).
• Digestion products stimulate the G cells, they release gastrin, parietal cells release acid.
• Distention alone can increase acid production.
• Accounts for 60-70% of acid production.
• Phase lasts until the stomach is empty.
Intestinal Phase

- Poorly understood.
- (?) initiated by chyme entering the small bowel.
- Accounts for ~10% of acid production.
Other functions

• Gastric acid suppression
• Mucosal protection
• $B_{12}$ absorption
Benign Gastric Disease(s) 

- Acute/Stress Gastritis 
- Gastric (peptic) Ulcer Disease 
- Hypertrophic Gastritis 
- Mallory-Weiss Syndrome 
- Gastric Polyps 
- Bezoars
Gastritis (acute or stress)

- Produces inflammation of the mucosa.
- Can be associated with erosions and bleeding.
- Causes:
Gastritis

• **S&S:**
  • Nausea, emesis, hematemeses, melena, hematochezia, etc.

• **Treatment:**
  • Prevention, removal of offending agent, acid suppression, occ gastric decompression and support.
Menetrier’s Disease (aka Hypertrophic Gastritis)
Menetrier’s Disease (aka Hypertrophic Gastritis)

- Rare.
- Characterized by massive hypertrophy of the gastric rugae.
- Etiology unknown.
- (?) autoimmune.
- (?) over-expression of TNF-β.
Menetrier’s Disease (aka Hypertrophic Gastritis)

- Patient’s usually present with:
  - Pain, N/V, occult hemorrhage, anorexia, wt loss and diarrhea.

- Disease progression is marked by protein-losing gastropathy.

- DX: UGI endoscopy w/ biopsy.

- Tx: typically medical, surgery is rare, Menetrier’s is a risk factor for gastric CA
Mallory-Weiss Syndrome

• UGI bleeding caused by linear tears at/near the GE junction.

• *Common Test Scenario:
  • Alcoholic, intense episode of emesis, now with UGI bleeding.
Mallory-Weiss Syndrome
Gastric Polyps
Gastric Polyps

- Rare, but frequency is increasing due to increasing numbers of UGI endoscopy.
- There are two types:
  - Hyperplastic
  - Adenomatous
- Hyperplastic polyps:
  - More common
  - Typically are benign (but can transform)
Gastric Polyps

- **Adenomatous polyps:**
  - Greater risk of malignancy.
  - "Size Matters"
    - < 0.5 cm in diameter = very low risk.
    - > 1.5 cm in diameter = very high risk.

- Once a polyp is diagnosed, one should evaluate for more.
Peutz-Jegher’s Syndrome

Figure 4 - Endoscopic view of sessile polyps in stomach.
Peutz-Jegher’s

• Characterized by:
  • Melanous spots on the lips and buccal mucosa.
  • Multiple benign gastric and small bowel polyps.
• Autosomal dominant w/ high degree of penetrance.
• Treatment is conservative, polyps are hamartomas and are infrequently malignant.
Bezoars
Bezoars

- Large masses of indigestible fiber(s) within the stomach.
- Phytobezoars = vegetable matter.
- Trichobezoars = hair.
Peptic (Gastric) Ulcer Disease
Gastric Ulcer Disease

- Most common in males and elderly.
- Peak incidence: 55-65 yoa
- Approx. 90,000 new cases/year.
- Approx. 35% will have complications
- Approx. 3,000 deaths/year due to complications.
- Approx. 10% of ulcers associated w/ CA.
Gastric Ulcer Disease

- 85%-95% of GU pts are colonized w/ H. pylori.
- Can occur anywhere in stomach.
- Most occur within 2 cm of the junction between the antral/fundic mucosa along lesser curve.
- 2/3 at incisura angularis
  - 20% distal, 10% proximal
  - Only 5% occur along greater curve.
The “Culprit”

- H. pylori
- Treatment:
  - Triple therapy
Gastric ulcers
Gastric Ulcers
Type I

- Occur at the incisura.
- Not associated with hyperacidity, most patients have low acid output.
- Associated with ABO group “A”.

[Diagram of the stomach showing Type I gastric ulcer]
Type II

- A combination of 2 ulcers, one in the body of the stomach, the other in the duodenum.
- Usually occur in hypersecretory states.
- Associated with ABO “O”.

[Diagram of the stomach with Type II gastric ulcer labeled]
Type III

- Located pre-pyloric.
- Associated with hypersecretion.
- Type “O” blood association.
- Typically 2-3 cm from pylorus, can be multi.
Type IV

- Csendes type ulcers.
- Occur high on lesser curve at/near the GE junction.
- Not associated with hypersecretion.
- Usually result from defective mucosal defense.
Type V

- Can occur anywhere in stomach.
- Result from chronic ASA/NSAID ingestion.
Malignant Gastric Disease

- Adenocarcinoma
- Lymphoma
- Gastrointestinal Stromal Tumor (GIST)
Gastric Adenocarcinoma
**Gastric Adenocarcinoma**

- Adenocarcinoma accounts for 95% of all gastric cancers.
- Worldwide is the leading cause of cancer death.
- US and Europe = low risk areas
- Asia (Japan/China), Russia, Chile = high risk areas.
Risk Factors

- H. pylori infection.
- Pernicious anemia.
- Achlorhydria.
- Chronic gastritis.
- H/o caustic injury.
- Presence of adenomatous polyps.
Classification

• In the US, there are 4 types:
  • Ulcerative, polypoid, scirrhous, superficial spreading.
  • Ulcerative is the most common.

• 2 distinct histologic types:
  • Intestinal
  • Diffuse
Intestinal

- Well differentiated with glandular elements.
- More common form in high incidence areas.
- Patients are usually older.
- Spread is hematogenous.
Intestinal
Diffuse

- Poorly differentiated with classic signet-ring cells.
- Patients are usually younger.
- Associated with ABO blood group “A”.
- Spreads via lymphatics and local extension.
Diffuse
Gastric Adenocarcinoma

- **Linitus plastica:**
  - Complete gastric infiltration with carcinoma.
  - Has “leathery” appearance.
  - Extremely poor prognosis.

- **Evaluation.**
- **Treatment.**
Linitus Plastica
Gastric Lymphoma
Gastric Lymphoma

- Stomach is the primary site for ~2/3 of all GI lymphomas.
- Patients tend to be older.
- Predominately non-Hodgkin’s variant.
- Symptoms are similar to Adeno-ca.
- Dx: via biopsy (endoscopic vs. open).
- If Dx is made prior to surgery, do lymphoma work-up.
- Treatment: chemotherapy vs. surgery.
GIST
GIST

- Formerly (incorrectly) called leiomyomas or leiomyosarcomas.
- Stomach is the most common site.
- Can be “benign” or “malignant”.
  - Malignant needs direct invasion.
  - Must count mitotic figures (>10 per 50 fields = malignant).
- Work-up.
- Treatment:
  - Surgical Resection w/ margins.
  - Gleevec
Duodenum

- 4 parts
- Metabolically active
  - Produces many enzymes
- D2: site of pacemaker
- D2: posterolateral insertion of ampulla.
- Becomes jejunum at the ______________?
Duodenum

- Brunner’s glands
- Blood supply:
  - GDA- superior pancreaticoduodenal
  - SMA- inferior pancreaticoduodenal
Benign Duodenal Disease

- Duodenal Ulcer Disease
  - Uncomplicated
  - Complicated
- Duodenal Polyps
Uncomplicated Ulcer Dz.

- PUD affects:
  - ~500,000 new cases yearly.
  - ~2,000,000 active adult cases in US.
- Most are duodenal ulcers.
- Typically these form in the bulb.
- In contrast to gastric ulcers, duodenal ulcers rarely harbor malignancy.
Uncomplicated Ulcer Dz.

- Important risk factors:
  - H. pylori, NSAIDs, (?) tobacco.
- Most duodenal ulcer disease is uncomplicated.
- Treatment has shifted to mainly medical instead of surgical.
Duodenal Ulcers
Presentation

- Both types:
  - Burning, gnawing pain in the epigastrium.
  - Can radiate to the back.
  - Pain usually occurs 1-3 hours after eating.
  - Exacerbated by fasting.
  - Relief gained from OTC meds and eating.
  - THESE CAN BLEED.
Evaluation/Testing

• (+) symptoms = non-invasive H. pylori testing.
  • Serum ab, breath urease, fecal antigen.
• (+) test = H. pylori treatment.
  • Triple therapy.
• If primary treatment fails then do upper endoscopy.
  • UE allows for visualization, biopsy, eval for alternate Dx.
Treatment

- Non-operative.
- Aim/Goal: promote healing of ulcer(s), prevent recurrence of ulcer(s).
  - Remove all ulcerogenic agents.
  - Start acid suppression.
  - Eradicate H. pylori.
Treatment

• H. pylori therapy:
  • First Line:
    • Amoxicillin and Clarithromycin –or-
    • Clarithromycin and Flagyl (+)
    • PPI (or H₂ blocker).
  • Treat for 7 – 14 days.
  • Recheck for H. pylori after treatment.
  • Continue acid supression until ulcer is healed.
Complicated Ulcer Dz.

- 4 major manifestations:
  - Perforation
  - Bleeding
  - Gastric outlet obstruction
  - Intractable
Evaluation

• Same as uncomplicated unless one of the four “bad things” is present.
Treatment: Perforation

• Typical presentation.
• Resuscitation.
• Operation:
  • Graham patch vs. Modified Graham patch.
  • +/- antrectomy/drainage (vagotomy).
Treatment: Bleeding

• Resuscitation.
• Upper endoscopy w/ local treatment.
  • (-) factors: active bleeding, visible vessel, fresh clot.
• (?) angiography w/ embolization.
• Surgical intervention.
Treatment: Outlet obstruction

- Decompression, NPO, rehydration, correct lytes.
- Some resolve (swelling).
- Surgery:
  - Remove obstruction.
  - Bypass obstruction.
- Antrectomy w/ drainage.
- Gastroenterostomy.
Treatment: Intractable dz.

- Medical failure.
- Need to r/o other issues:
  - ZE syndrome, non-compliance, etc.
- Surgery.
Duodenal polyps

- Typically arise as part of a familial disorder.
  - FAP.
    - Autosomal dominant
    - High malignant potential
  - Peutz-Jegher’s syndrome.
Malignant Duodenal Disease

• Zolliger-Ellison (ZE) Syndrome
• Adenocarcinoma
• Lymphoma
Z-E Syndrome

• Rare disease.
• Most well-known endocrine tumor disorder.
• Disease is a direct result of gastrinoma.
• 2/3 of gastrinomas are in the “triangle”
  • CBD/D2-D3/neck of Pancreas.
• Can be sporadic or hereditary.
  • Strong assoc. with MEN-I (3 p’s)
• Treatment is resection (if possible).
Gastrinoma
Adenocarcinoma

- Duodenum is the most common site for small bowel adenocarcinoma.
- ~ 2/3 are peri-ampullary (D2).
- Very rare, patients tend to present late in disease course.
- Only treatment is resection (if possible).
- Double bypass is unresectable.
- (+) LN’s = < 15%, 5 year survival.
Adenocarcinoma
Lymphoma

- Rare, most small bowel lymphomas are ileal.
- Similar presentation to adeno CA.
- Diagnose the same as gastric lymphoma.
- Resect if resectable.
- Chemotherapy and radiation.
Lymphoma