Conditions of the Stomach (II)

R2 Kanyarat Olarachin, M.D.
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Gastric volvulus
Introduction

- Rare
- Potentially life-threatening condition
- 21% of cases are neonates in recent studies
- Associated with diaphragmatic defect
- Older children: associated with neurodevelopmental delay and splenic abnormalities

- Infancy: acute complete volvulus
- Older children: chronic, partial volvulus
Introduction

- Primary gastric volvulus
  - Laxity of the gastric ligaments

- Secondary gastric volvulus
  - Paraesophageal hernia or diaphragmatic hernia
Etiology

- Abnormal rotation of one part of the stomach
- 180 – 360 degree
- Anterior direction

- Organoaxial (54%)
- Mesenteroaxial (41%)
- Combined (2%)
Pathogenesis

- About **two-third** of children with gastric volvulus has **diaphragm** eventration or herniation (paraesophageal, posterolateral, Morgagni)
  - Upward displacement of the transverse colon
  - Greater curve of the stomach was pulled up into the expanded left upper quadrant

- Other rare causes of gastric volvulus
  - Abnormal band or adhesion
  - Rectal atresia -> overdistention of the transverse colon
  - Congenital absence or left lobe liver resection
  - Congenital deficiency of the gastrocolic omentum
  - Asplenic syndrome
  - Post-operative complication in older children
Clinical features

- Adults and older children: Borchardt triad
  - Unproductive retching
  - Acute localized epigastric distention
  - Inability to pass nasogastric tube

- Neonates
  - Persistent regurgitation
  - Hematemesis, anemia
  - Failure to thrive
  - Chest infection, wheeze
  - Successful passing NG tube cannot ruled out
Diagnosis

- Plain abdominal radiograph
- Distend stomach in abnormal position
Treatment

- Acute gastric volvulus need appropriate resuscitation and urgent surgery
- Gastric decompression pre-operatively
  - Nasogastric suction
  - Needle aspiration might perform before manipulating a tensely dilated stomach and reducing the volvulus
- Abdominal approach is recommended
  - Can identify associated gastrointestinal tract anomaly
  - Accurate diaphragmatic repair
Treatment

- Gastrostomy
  - Fixation
  - Route for feeding
- Stamm gastrostomy
  - 10- or 12- French gauge Malecot catheter
  - Secured by a double-purse string absorbable sutures
Treatment

- Anterior gastropexy
  - Added if patients don’t have diaphragmatic defect
- Suture the greater curve of the omentum to the parietal peritoneum and the undersurface of the diaphragm with non-absorbable sutures
Treatment

- Endoscopic assisted percutaneous anterior gastropexy:
  Chronic mesenteroaxial volvulus in older children

- Laparoscopic anterior gastropexy:
  Older children with isolated gastric volvulus

- Fundoplication:
  - Might be needed if patients have gross gastroesophageal reflux
  - Crural repair alone might be sufficient

- Gastric volvulus due to a wandering spleen
  Splenopexy alone might be sufficient
Complication

- Prolonged gastric ileus
- Pyloric ischemia
- Gastric outlet obstruction
- Gastric necrosis
- Gastric perforation

- Mortality rate
  - 7.1% in acute gastric volvulus
  - 27% in chronic gastric volvulus
  - Untreated: mortality rate up to 80%
Acute and chronic peptic ulcer
Epidemiology

- Primary peptic ulcer disease
  - *H. pylori* infection
- Secondary peptic ulcer disease
  - Excessive acid production
  - Stress
  - Other conditions: e.g. Eosinophilic gastroenteritis, etc.
  - Drug-related: NSAIDS, Aspirin, Ethanol
Primary peptic ulcer disease

- Incidence: 5.4 : 100,000
- **Boys : Girls** 2-3X : X
- Infants and very young children: X : X
- Hyperacid secretion: Duodenal and pre-pyloric ulcer
- Strong familial tendency -> *H. pylori* cluster in family
- Incidence of *H. pylori* infection
  - Industrialized countries: 0.5% per year
  - Developing countries: 3%-10% per year
- **Risk factor** associated with *H. pylori* infection
  - Crowded living, low socioeconomic level, immigrants, infected family member, ethnicity
- Pathologic condition associated with *H. pylori* infection
  - Nodular gastritis, primary duodenal ulcer, gastric ulcer, Barett esophagus, gastric cancer, MALT lymphoma
Secondary peptic ulcer disease

- Stress ulcers 80%
- Associated with critical illness, major trauma
- Multiple superficial mucosal erosions at fundus of stomach
- Causative factors
  - Decrease mucosal blood flow
  - Disruption of the protective mucosal barrier
  - Intraluminal acidity
- Drug- and chemical-induced ulcer: resemble stress ulcers
- Cushing ulcer: overstimulation of the Vagus nerve due to increase intracranial pressure -> increase acid output
  - single, deep ulcer -> prone to perforation
Gastric physiology

- 19th week: acid secretion
- 34th week: pepsin secretion

- Term infant
  - Gastric hyposcretion: 5 – 48 hr after birth
  - pH 3

- Preterm infant
  - Diminished amount of acid and pepsin secretion
  - 33%: alkaline gastric pH
  - 20%: no acid production for 10 days

- Maternal gastrin secretion:
  - Infant high acid secretion rate d7-10
Pathophysiology

**Aggressive factors**
- Vascular injury: decrease microcirculation
- Cancer chemotherapeutic agents
  - Aspirin
  - NSAIDs
- Infectious agent: CMV, Herpes virus
- Increase systemic stress
- Increase pepsin secretion
- H. pylori

**Defensive factors**
- Mucosal circulation: adequate microcirculation
- Epithelial cell turnover
- Increase bicarbonate secretion
- Inhibit gastric acid secretion
- Anti-inflammatory drugs
- Preserve vascular CMV flow/microcirculation
- Restore epithelial cell surface catecholamines
- Mucous layer: glycoprotein, glycocalyx
- Bicarbonate layer: pH gradient
- Immunoglobulins: IgG, IgA
Clinical presentation

Primary peptic ulcer

- Infant
  - Refusal of feeding
  - Persistent crying
  - Vomiting

- Preschool-aged and school-aged
  - Vomiting

- Older children
  - Abdominal pain (vague, related to meals)
Clinical presentation

Secondary peptic ulcer

- Acute onset
- Upper gastrointestinal hemorrhage (92%)
- Vomiting
- Perforation
Diagnosis

- Clinical presentation
  - GI bleeding
  - Dysphagia
  - Persistent vomiting
  - Abdominal pain

- Gold standard: EGD (85%)
- Angiography: useful in locating a bleeding ulcer if rate of bleeding is at least 0.5 mL/min
Diagnosis

- *H. pylori* infection
- Invasive
  - EGD + biopsy:
    - nodularity in the antrum (specific but not sensitive)
    - Urease activity
- Non-invasive
  - *H. pylori* specific IgG
  - Urea breath test (> 2 yrs old)
  - Stool antigen test
Treatment

- Medical treatment
  - Antacids
  - H2 receptor antagonists

- Other agents
  - Selective anticholinergic
  - Proton-pump inhibitors
  - Cytoprotective agents
  - Anti-infective agents
Antacid

- Neutralize acid secretion
- Heal peptic ulcer
- Dosage: 0.5 mL/kg
  - 1 hr ac, 3 hr pc, hs
- Side effects
  - Diarrhea
  - Constipation
**H₂ receptor antagonist**

- **Cimetidine**
  - Dosage 20-40 mg/kg/day
  - Antiandrogen side effects

- **Ranitidine**
  - Dosage 6-9 mg/kg/day oral, 2-4 mg/kg/day IV
Proton pump inhibitor

- Inhibit the stimulation of gastric acid secretion at the final common pathway

- Omeprazole
  - Dosage 1 mg/kg/day, max 20 mg/day
  - Side effects: headache, nausea, abdominal pain

- Lansoprazole
  - Dosage 0.5 mg/kg/day up to 30 kg
  - 30 mg daily if BW > 30 kg
Cytoprotective agent

- **Sucralfate**
  - Negative charge of sulfated disaccharide adheres to positive protein charge of the injured mucosa
  - Stimulated mucous production and prostaglandin synthesis
  - Dosage 40-80 mg/kg/day
  - Side effects: constipation

- **Prostaglandin E**: misoprostol, enprostil, arbaprostil
  - Blocking production of cyclic AMP, stimulation of HCO3-, increase mucosal blood flow
  - Few data in children use
# H. Pylori associated disease

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose</th>
<th>Duration</th>
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<tr>
<td>Amoxicillin</td>
<td>50 mg/kg/day</td>
<td>14 days bid</td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>15 mg/kg/day</td>
<td>14 days bid</td>
</tr>
<tr>
<td>Proton pump inhibitor</td>
<td>1 mg/kg/day</td>
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Surgical treatment

- Reserved for peptic ulcer disease with complication
  - Perforation
  - Bleeding
  - Obstruction
  - Intractable pain

- Prefer vagotomy and pyloroplasty > gastric resection
Surgical treatment

- Bleeding or perforated ulcer in the first 1-2 wk of life
  - Hypersecretion of acid caused by maternal gastrin
  - Often respond to OG decompression, lavage

- If surgical intervention is needed,
  - Perforation: simplest method
  - Bleeding: simple suture ligation of the ulcer bed

- Chronic partial gastric outlet obstruction secondary to a congenital problem can result in peptic ulcer -> correct obstruction
Zollinger-Ellison syndrome

- Hypergastrinemia
- Relatively rare in children
- Diagnosis
  - Large gastric rugal fold
  - Duodenal dilatation
  - Edema of the small bowel mucosa
  - Confirmed by elevated serum gastrin, calcium infusion test

- Treatment
  - Total gastrectomy if cannot completely resect primary tumor
  - MEN1 can be present in 25% of cases
Stress ulcer

- Prevention is the best
- Medication: H2 receptor antagonists and PPI

- Major UGIB or recurrent UGIB: EGD
  - Therapeutic injection: hypertonic saline, epinephrine, ethanol
  - Cauterization: heater probe, bipolar coagulation, laser
  - Stand-by surgery

- Massive hemorrhage: need operation
  - Blood loss in 24 hr
    - < 2 yr: = total estimate blood volume
    - > 2 yr: = half of total estimate blood volume (80mL/kg)
Stress ulcer

- Treat underlying cause
- Simple surgical procedure: plication at perforation site, oversewing of the bleeding point
- Vagotomy and pyloroplasty: not interfere growth and development
Gastric perforation in the newborn
Introduction

- Rare condition in newborn
- Incidence 1 in 2900 live births
- 10-15% of all GI perforation in neonates and children
- Male : Female -> inconclusive
- Mortality 25%-50% in most case series
- Can occur in full-term, premature, and SGA neonates
**History**

1926

**Siebold**
First demonstrated GI spontaneous perforation

1950

**Leger et al.**
First successful repair of a neonatal gastric perforation

1969

**Lloyd**
Multiple predisposing factors (selective circulatory ischemia)
Etiology

**Spontaneous**
- Theories
  - Congenital absence of gastric muscle
  - Forced exerted during vaginal delivery
    - Pneumatic distention: perforate at fundus, ischemic change
- Recent study
  - Deficiency of tyrosine kinase receptor C-KIT$^+$ mast cell and a lack of C-KIT$^+$ interstitial cell of Cajal
  - Impair immunity and abnormal motility

**Ischemic**
- Physiologic stress
  - Prematurity
  - Asphyxia
  - Sepsis
  - NEC
- Redistribution of blood flow
  - Causing microvascular injury

**Traumatic**
- Pneumatic distention: mask ventilation, PPV
- Gastric intubation
Causes of neonatal gastric perforation

- Idiopathic (Spontaneous)
- Perinatal stress
- Iatrogenic
- Medication
Causes of neonatal gastric perforation

- Perinatal stress
  - Hypoxia
  - Asphyxia
  - Anatomic defect
  - Distal obstruction
  - Tracheoesophageal fistula
  - Congenital deficiency of gastric muscle
Causes of neonatal gastric perforation

- Iatrogenic
  - Nasogastric tube
  - Aggressive bag ventilation
  - Cardiopulmonary resuscitation
  - Positive pressure ventilation
  - Unintentional perforation during surgery (VP shunt)
  - Vaginal delivery
Causes of neonatal gastric perforation

- Medication
  - Indomethacin
  - Corticosteroids
Clinical presentation

- Often occurs at DOL 3-5 (within the first 7 days)

- Presentation
  - Feeding intolerance
  - Emesis contains blood
  - Rapid abdominal distention
Signs and symptoms

- Respiratory distress
- Hemodynamic instability
- Signs of shock
  - Hypothermia
  - Cyanosis
  - Poor peripheral perfusion
  - Low urine output

- Physical examination
  - Abdomen rapidly tense and tender (peritoneal irritation)
  - Subcutaneous emphysema at the abdominal wall or pneumoscrotum
Signs and symptoms

- Common site of perforation
  - Greater curvature
  - Posterior perforation into the lesser sac: insidious cause
    (Difficult to diagnose)

- Increase risk in pregnancy with complication
  Abruptio placentae
  Placenta previa
  Amnionitis
  Delivered by emergency caesarian section
Diagnosis

- Clinical history + physical examination + radiographic studies

- Finding in plain abdominal radiographs
  - 90% non-visualized stomach
  - Pneumoperitoneum
  - Subcutaneous emphysema
  - Pneumoscrotum
  - OG/NG outside the confines of the stomach
  - Pneumatosis intestinalis : NEC (co-exist)

- Laboratory investigation
  - Complete blood count - Blood cultures
  - Arterial blood gas - Electrolyte profile
Differential diagnosis

- Vomiting and abdominal distention
  - Hirschsprung’s disease
  - Intestinal atresia
  - Meconium ileus
  - Meconium plug syndrome
  - Imperforate anus
  - Perforated viscus
  - NEC
  - Midgut volvulus
Differential diagnosis

- Cardiovascular collapse
  - Sepsis
  - Pneumothorax
  - Cardiac dysfunction
  - Intraventricular hemorrhage
  - NEC
  - Perforated hollow viscous organ
  - Malrotation with midgut volvulus
Perioperative care

- Early recognition and prompt treatment
- Respiratory distress from marked abdominal distention
  - Require intubation and ventilatory support
- Broad-spectrum antibiotics
- Fluid resuscitation, blood transfusion
- OG/NG carefully passed and placed on low intermittent suction for gastric decompression
- Paracentesis with IV canula: lifesaving when abdomen overly distend and interfere ventilation
Surgical technique

- Upper abdominal transverse incision
- Dissect through the rectus muscle layer by layer until the peritoneum is entered
- The umbilical vein is divided
- Peritoneal fluid and debris are evaluated and sent for cultures (aerobic, anaerobic, fungi)
Surgical technique

- Explore site of perforation
  - Mostly spontaneous gastric perforation: along greater curvature
  - Duodenal ulcer: anterior wall or near the pyloroduodenal junction
  - Gastric ulcer: along lesser curvature near the antral-fundic junction

- If cannot find the perforated site, carefully explore the EG junction, duodenum, small bowel and colon
- Open lesser sac and inspected for contamination and lesion at posterior wall of stomach
Surgical technique

- Debrided non-viable tissue around the perforated site
- Closed defect in one or two layers +/- omental patch
- Extensive perforation or necrosis may require sub-total or total gastrectomy
  - If the greater curve is extensively necrosed -> resection
  - If the antrum is extensively necrosed -> Billroth I
- Reconstruction could be performed in stable infants
Reconstruction technique

- In total gastrectomy cases
  - Transverse colonic interposition
  - Roux-en-Y esophago-jejunal anastomosis
  - Hunt-Lawrence pouch reconstruction

- Staged surgery in unstable patients
  - Performed several weeks later when the patient’s condition has improved
Surgical technique

- Lavage abdomen with warm NSS
- Peritoneal drainage is not needed for most primary repairs
- The fascia and skin are closed in standard fashion
Post-operative care

- Continue broad spectrum antibiotics until ...
  - WBC and PMN within normal range
  - Evidence of bowel and gastric function returns
    - Clear and low volume OG content
- Gastric acid suppression therapy
- TPN
- Continue gastric decompression
- NPO until the patient has stabilized
- May obtain a contrast study before starting enteral feeding
Outcomes

- Isolated gastric perforation survival 75-80%
- Poor outcome associated with multiple-organ dysfunction, sepsis, immature immunologic function
- High morbidity and mortality in infants with gastric necrosis with extensive NEC
Congenital microgastria
Rare congenital anomaly of the caudal part of foregut
- Small, tubular stomach, megaesophagus, incomplete gastric rotation, normal mucosa
- Associated anomalies: VACTERL association
  - GI: Non-rotation of midgut with duodenal band with asplenia, absence of gallbladder
  - Skeletal: micrognathia, radial and ulnar hypoplasia, vertebral anomalies, oligodactyly, and hypoplastic nail (rare: anophthalmia)
  - CVS: single atrium, single ventricle, total anomalous pulmonary venous return into the portal vein
Clinical presentation

- Prenatal ultrasonography
  - Polyhydramnios
  - Small stomach
- Dilated esophagus with ill-defined E-G junction
- Vomiting
- Aspiration and pneumonia
- Bacterial overgrowth and blind-loop-like syndrome
  - Failure to thrive
  - Diarrhea
- Normal Schilling test
Diagnosis

- Upper GI study
- EGD: confusing results
- If patients have GERD
  Manometry
  Esophageal pH
- If patients have diarrhea and malabsorption
  Intestinal absorption studies
Treatment

- Medical treatment
  - Continuous or night-time OG feeding
  - If GERD develops, start prokinetic agents and acid-reducing therapy
  - Complication of GERDS may require NJ feeding or surgical jejunostomy tube feeding

- If stomach fails to enlarge, several authors recommended create a double-row jejunal reservoir (Hunt-Lawrence pouch)
Thank you for your attention

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