Gastrointestinal Disorders (1)

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• I have no relevant financial relationships with the manufacturer(s) of any commercial product(s) and/or provider of commercial services discussed in this CME activity.

• I do not intend to discuss an unapproved/investigative use of a commercial product/device in this presentation.
• GI Disorders 1 and 2 cover most of the intestinal and nutritional objectives for PREP The Course
• Hepatic diseases are discussed in the concurrent GI case presentations
Objectives

- Understand the difference between GER and GERD
- Understand appropriate diagnosis of celiac disease
- Understand surgical causes of abdominal pain in children
Diseases of the Esophagus

• Gastroesophageal Reflux Disease
• Eosinophilic Esophagitis
• Motility Disorders (supplementary material)
• Structural Anomalies (supplementary material)
Gastroesophageal Reflux Disease

• Important to understand differences between GER and GERD
  – GER = GastroEsophageal Reflux
    • Passage of gastric contents into esophagus with OR without regurgitation/vomiting
    • NORMAL physiologic process, usually after meals, cause few or no symptoms
    • Occurs several times a day in infants, children, and adults
  – GERD = GastroEsophageal Reflux Disease
    • Troublesome symptoms and/or complications when reflux of gastric contents occurs

Vandenplas et al, JPGN 2009
Gastroesophageal Reflux (Disease)

- GER is most frequent in infants aged 1-6 months
  - Peak at 3-4 months
  - In infants, episodes may occur 100x/day!
  - Most GER in infants resolves by 12 months of age (95%)**
- Clinical features of GERD may include:**
  - Regurgitation/vomiting, weight loss/poor weight gain, irritability, heartburn/chest pain (older children), hematemesis, dysphagia, wheezing, stridor, cough, hoarseness

Vandenplas et al, JPGN 2009
Gastroesophageal Reflux Disease

- Complications of GERD can include:
  - Esophagitis, esophageal stricture, Barrett’s esophagus, recurrent pneumonia, anemia, dental erosions, feeding refusal, apnea, ALTE

- Evaluation of GERD:
  - GERD is a clinical diagnosis
    - History/exam is usually enough in older children (>8 years) and adults
      - Heartburn, regurgitation, epigastric pain

Vandenplas et al, JPGN 2009
Gastroesophageal Reflux Disease

• Diagnosis
  – History and physical
  – Testing to evaluate for other causes of reflux symptoms
    • e.g. Upper GI Series to evaluate for malrotation
  – Other tests
    • Endoscopy
      – Histology can confirm GERD
    • pH/impedance probes
Gastroesophageal Reflux (Disease)

• Management**
  – Infants:
    • “Happy spitter” = reassurance; if symptoms persist >18 months, referral to pediatric GI; endoscopy may be considered
    • FTT = (negative workup) +/- trial of hypoallergenic formula/thickened formula, consider acid suppression therapy (H2 receptor antagonist or proton pump inhibitor), refer to pediatric GI
  – Older child with heartburn
    • PPI for 8-12 weeks; if no improvement after 2-4 weeks, consult GI
      – If relapse occurs, consult GI

Vandenplas et al, JPGN 2009
Gastroesophageal Reflux Disease

• Pearls:
  – When deciding to trial an acid suppressing medications:
    • 1. **Follow symptoms.** (Have parents/patients log symptoms before and after medication/intervention is prescribed.)
    • 2. **It can take several weeks for medications/interventions to have maximal effect.** (Give medications at least 2-4 weeks to show an impact; one week of therapy is not enough!)
    • 3. **Treat for a specific period of time and then stop the medication.** (Trial a medication for 2-3 months, then stop. If there are no improvement in symptoms after this period, either the problem is not GERD or the medication dose/class is not appropriate.)
Eosinophilic Esophagitis (Allergic Esophagitis)

• Definition: a chronic, immune/antigen-mediated esophageal disease
  – Prevalence: ~50-100 per 100,000 population
  – Antigens are typically foods but environmental antigens may trigger inflammation as well
  – Most common foods: milk, soy, wheat, eggs, nuts, and fish
  – 50% of patients have an atopic history
• Symptoms can be similar to GERD (heartburn, abdominal pain) but dysphagia is often seen**
  – Food impaction can be the first presentation
Eosinophilic Esophagitis

- Dysphagia (difficulty swallowing)
  - Poor motility
  - Increased fibrosis (scarring) from eosinophilic inflammation

Esophageal Stricture
Eosinophilic Esophagitis (Allergic Esophagitis)

• Diagnosis:
  – Clinico-pathologic diagnosis
    • Classic endoscopic findings: white plaques, linear ridging, “trachealization” of the esophagus
    • Presence of > 15 eos/hpf in the esophagus of patients ON proton-pump therapy
  – Treatment:
    • Medications: swallowed steroids, acid suppressing medications
    • Exclusion of dietary antigens
Endoscopic Views of Esophageal Diseases

- Erosive esophagitis (GERD)
- Eosinophilic esophagitis
- Candidal esophagitis
Diseases of the Stomach

- *Helicobacter pylori* Infection
- Peptic Ulcer Disease
- Pyloric Stenosis
**H. pylori Infection**

- **Prevalence in children ranges from 10-90% throughout the world**
  - Highest prevalence with lower socioeconomic status and those living in poor sanitary conditions**
  - Children with first degree relatives with gastric cancer are at higher risk for *H. pylori* infection**
- **Gastrointestinal manifestations include:**
  - Gastritis, ulcer disease, gastric cancer
  - (extra intestinal) Iron deficiency anemia


**Diagnosis of *H. pylori* infection**

- Upper endoscopy with gastric biopsies are gold standard
  - Grossly can see nodularity in the stomach or pan gastritis
  - Histology can visualize *H. pylori*
- Serum IgA testing is not recommended as only 20-50% of *H. pylori*-infected patients will test positive
  - In addition, positive IgA testing does not discern between current and past infection

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*Nodularity in antrum*

Koletzo et al JPGN 2011
**H. pylori Infection**

- **Treatment**
  - Indicated when *H. pylori* is present histologically AND when gastritis or ulcer disease is present
  - Indicated with children who are infected with *H. pylori* and have a first-degree relative with a history of gastric cancer
  - First line treatment: “triple therapy” for 14 days
    - Proton-pump inhibitor + amoxicillin + clarithromycin
    - Other options available for resistant h. pylori or patients with allergies to first line medications
    - Noninvasive follow-up testing in 4-8 weeks (breath test or stool antigen test)
Peptic Ulcer Disease (PUD)

- Rare in children (incidence of ~2% of children undergoing endoscopic evaluation)**
  - Usually associated with stress (trauma/sepsis), use of medications including nonsteroidal anti-inflammatory drugs, or hypersecretory state like Zollinger-Ellison syndrome
- Presentation can vary
  - In younger children: irritability, abdominal pain
  - Older children: epigastric abdominal pain, vomiting
  - Hematemesis should prompt consideration of PUD
Peptic Ulcer Disease (PUD)

• Evaluation:
  – If history/physical examination is suspicious for PUD, treatment may be initiated
  – Endoscopy can be an important tool for evaluation when history/physical is less revealing
    • Therapeutic interventions
  – Imaging, including fluoroscopy, is not accurate nor reliable for the diagnosis of PUD
Peptic Ulcer Disease (PUD)

• Treatment
  – Acid-suppressing medications (treat for 2-3 months)
    • H2 Receptor Antagonists**
      – Raise gastric pH by blocking histamine receptor on gastric parietal cells
      – Pros: Generally well-tolerated with few side effects
      – Cons: Tachyphylaxis is common and acid secretion can still occur through the proton pump
    • Proton Pump Inhibitors
  – Antacids
Peptic Ulcer Disease (PUD)

• Treatment
  – Acid-suppressing medications (treat for 2-3 months)
    • H2 Receptor Antagonists
    • Proton Pump Inhibitors**
      – Raise gastric pH by blocking H+/K+ ATPase (“proton”) pump
      – Pros: more effective than H2RAs at protecting gastric/duodenal mucosa
      – Cons: possible increased risk of C. difficile diarrhea, increased risk of pneumonia in neonates, and effects on calcium/magnesium metabolism with long term use
  – Antacids
Peptic Ulcer Disease (PUD)

• Treatment
  – Acid-suppressing medications (treat for 2-3 months)
    • H2 Receptor Antagonists
    • Proton Pump Inhibitors
  – Antacids
    – Neutralizes gastric acid
    – Pros: improved symptomatic relief (quickly)
    – Cons: do not inhibit acid production
Pyloric Stenosis

- Most common surgical disorder in neonates
- Prevalence = 1.5-4:1000 births, more prevalent in Caucasians and males.
- Presentation: usually between 3-6 months of age
  - Projectile vomiting (can contain blood), weight loss
  - Hypochloremic, hypokalemic metabolic alkalosis

Hypertrophied pylorus
Pyloric Stenosis

• Treatment:
  – Correct dehydration and alkalosis**
    • Fluid resuscitation with 0.9% normal saline (NS) boluses and then infusion of 5% dextrose in 0.45% normal saline at 1.5x maintenance
      – K can be added once good urine output is established
  – Surgical correction with pyloromyotomy
    • Significant complications are rare
    • Continued vomiting may be a sign of incomplete surgical repair
Diseases of the Small Intestine

- Celiac Disease
- Disaccharidase deficiency
- Duodenal Atresia
- Malrotation
- Intussusception
- Volvulus
- Meckel’s Diverticulum
- Appendicitis
Celiac Disease

• Definition: Immune-mediated enteropathy caused by gluten in genetically susceptible individuals.

• Clinical Presentation**
  – Can vary significantly: severe FTT, vomiting, diarrhea, short stature, anemia, transaminitis, dental abnormalities, dermatitis herpetiformis, and no symptoms!

Celiac Disease Guideline Committee, NASPGHAN, JPGN 2005;40:1-19
Celiac Disease

• Diagnosis
  – Must be on a gluten-containing diet at the time of diagnosis!
  – Serology: TTG IgA (Tissue Transglutaminase)—autoantibody
    • **Pearl**: always check a total IgA when screening for celiac disease; IgA deficiency not only results in a false-negative screen but increases risk of celiac disease 10 fold
      – If IgA deficient, TTG IgG should be ordered
    • **Pearl**: TTG IgA is less reliable in patients <2 years of age
    • TTG IgA is 95-99% sensitive and specific for celiac in >2 yo
  – Diagnosis is confirmed by endoscopy
    • Villous atrophy + intraepithelial lymphocytes
Celiac Disease

• **Treatment**
  - Gluten free diet (lifelong)
  - Translation: exclude wheat, rye, barley, triticale, spelt, and kamut +/- oats
  - Dietician consultation
  - It is important to stress that GFD is recommended for anyone diagnosed with celiac
    • If untreated, patients with celiac have a 80 fold increased risk for small bowel adenocarcinoma (in addition to experiencing clinical manifestations of celiac)
Disaccharidase Deficiency

• Quick review on carbohydrate digestion:
  – Starch breakdown begins in mouth (salivary amylase)
  – Bulk of CHO digestion occurs in duodenum (pancreatic amylase)
    • Results in disaccharide production
    • Brush border enzymes in small bowel contain disaccharidases
      which degrade disaccharides to monosaccharides
    • Monosaccharides (glucose, galactose, and fructose) are absorbed
      into the enterocyte through specific transporters

• CHO malabsorption = flatulence, bloating, diarrhea, FTT in infants
Disaccharidase Deficiency

- **Lactase deficiency**
  - Most common type of CHO maldigestion
  - Primary: Congenital form is very rare; more common in adolescents/adults
    - Highest incidence in Native Americans and Asians
  - Secondary**: common after infectious gastroenteritis and can take weeks to months to recover; can occur with any enteritis
  - Treatment: lactaid; avoidance of lactose

- **Sucrase-Isomaltase deficiency**
  - Occurs in <1% of babies; symptoms occur once sucrose and starch is introduced into the diet = diarrhea/flatulence
  - Higher incidence in Alaskans, Canadians, and Greenlanders
  - Treatment: sucraild, avoidance of sucrose/maltose
Duodenal Atresia**

- Incidence is ~ 1:6000 births in the US
- Many babies with duodenal atresia have comorbidities:
  - Down syndrome
  - Malrotation
  - Congenital heart disease
- Presentation: usually bilious emesis within first few hours of life
Duodenal Atresia**

- Imaging: “Double bubble” (dilation of stomach and proximal duodenum)
- Treatment: stabilization, gastric decompression, evaluate for associated anomalies, surgery (duodenoduodenostomy)
Malrotation**

- **Definition:** abnormal intestinal rotation and fixation
- **Occurs in ~ 1-2% of the population**
- **Presentation**:  
  - **Symptomatic:** bilious emesis, developing to abdominal distention and peritonitis (if untreated)  
    - small bowel obstruction with volvulus  
    - Require emergent diagnosis and operative repair: Ladd’s procedure  
    - Can also have chronic symptoms (recurrent emesis, diarrhea)  
  - **Asymptomatic:** may be diagnosed based on upper GI series for other reasons  
    - May also require surgical repair
Volvulus**

- Life-threatening condition associated with malrotation and twisting of the intestine on the mesenteric axis
- Needs to be considered in an infant/child with bilious emesis
Volvulus

- Diagnosis may be clinical, based on history and physical exam
  - Upper GI series can demonstrate a “corkscrew” appearance of the small bowel
  - Negative imaging does not rule out volvulus
- Surgical consultation and operative intervention are essential

UGI series: volvulus ("corkscrew")
Intussusception**

- Affects 1:2000 infants and children
- Characterized by “telescoping” of the intestines
  - Advancing section of proximal intestine = “intusussceptum”
  - Receiving section of distal intestine = “intussuscipiens”
- Can involve any section of the bowel, however is usually limited to ileocolic distribution
- Can be life-threatening as bowel ischemia can occur as intestine is completely obstructed
Intussusception**

- Can occur at any age but incidence is highest in children younger than 2 years
  - Cause is idiopathic in this age group (3 months to 3 years)
    - Lymphoid hyperplasia
  - Need to consider pathologic lead point in children >3
    - Meckel’s Diverticulum
    - Polyp
    - Vasculitis from HSP (Henoch-Schonlein Purpura)
    - Lymphoma
Intussusception

- **Clinical presentation**
  - Classic Triad: abdominal pain, vomiting, “currant jelly” stools
  - Usually previously healthy, although some have had a preceding GI infection
  - Severe and colicky pain, inconsolable when in distress
  - Altered mental status
  - Vomiting, fever, anorexia
  - PE: hypotonia, “sausage-like” mass in RLQ or RUQ

- **Diagnosis**: ultrasound, CT, air-contrast enema

- **Treatment**: fluid resuscitation, antibiotics, surgical consultation
  - Options include air-contrast enema or exploration
Meckel’s Diverticulum  
(Rules of 2!)

• Most common congenital anomaly of the GI tract 
  – Prevalence of ~2% of the general population

• Contains rests of ectopic tissue (2 types: gastric or pancreatic) 
  – Believed to result from the incomplete obliteration of the omphalomesenteric (vitelline) duct 
  – Usually within 2 feet from the ileocecal valve 
  – Most commonly 2 inches long (but can range from 1-26 cm!)

• Children < 2 years have highest risk of a symptomatic Meckel’s 
  – Can also be clinically silent 
  – Presentation: small bowel obstruction, lower GI bleeding, intussusception
Meckel’s Diverticulum

• Diagnosis:
  – Meckel’s scan
    • Technetium-99 pertechnetate is taken up by gastric parietal cells (H2RA can accentuate this uptake)
    • High specificity (99-100%) but low sensitivity (60-80%)

• Treatment:
  – stabilization (may need pRBCs if significantly bleeding)
  – Surgical consult
    • Operative management may be warranted even if Meckel’s scan is negative
Appendicitis

- Initiated by an obstruction to the appendix, flow of secretions out of the appendix is limited
  - Causes increased intraluminal pressure and vascular compromise
    - Luminal bacteria traverse mucosal wall → gangrene, infection
    - Can progress to perforation
- 250,000 cases/year in the US
  - Highest incidence in teenagers
  - Perforation highest in children <2 (95% of appendicitis presents with perforation!)
Appendicitis

• Classic presentation in older children
  – Periumbilical pain that migrates to RLQ (McBurney’s point)**
    • Example of referred abdominal pain**
      – Begins in the periumbilical area (distention of appendix triggers transmission of pain through visceral afferent fibers which causes referred pain in periumbilical area)
      – As inflammation becomes peritoneal, peritoneal somatic afferent pain fibers transmit pain that localizes near the appendix (RLQ in most people)
    • Vomiting, anorexia, fever, elevated WBC
Appendicitis

• Presentation in infants/children
  – Irritability, fever, lethargy, anorexia, vomiting
  – Perforation is very common

• Diagnosis
  – Exam with RLQ tenderness, RLQ pain with palpation of LLQ, psoas sign (RLQ pain with R hip extension), Obturator sign (RLQ pain with internal rotation of flexed R thigh)
  – Elevated WBC; imaging consistent with appendicitis or perforated appendicitis

• Treatment: nonoperative management vs. operative management
  – If perforated: IV antibiotics, nonoperative management, and interval appendectomy after resolution of infection
Changes You May Wish to Make in Practice

• Carefully consider whether acid suppression therapy is necessary in otherwise healthy infants with vomiting
• Refer early to pediatric GI when there are concerns that GERD symptoms are:
  – Not responding to standard treatment
  – Refractory to standard treatment
• Use TTG IgA and total IgA tests when screening for Celiac
2. Nutrition and Nutritional Disorders
E. Nutritional problems associated with specific diseases/conditions
   1. Gastrointestinal disorders
      b. Lactose intolerance management involves either using lactose-free products or supplementing lactose with lactase enzyme at the time of PO intake. For secondary causes of lactose intolerance, patients may be educated to reintroduce lactose back into their diet once intestinal healing has occurred. Examples of intestinal diseases that may result in secondary lactose intolerance include: acute viral gastroenteritis, celiac disease, eosinophilic (allergic) GI disease, and giardia.
      c. Early refeeding in acute gastroenteritis is beneficial and in fact reduces stool output faster than oral rehydration alone. Despite secondary lactose intolerance being reported in acute gastroenteritis, 80% of children after gastroenteritis can tolerate lactose-containing milk. Provisional Committee on Quality Improvement, Subcommittee on Acute Gastroenteritis, Practice Parameter: The Management of Acute Gastroenteritis in Young Children. Pediatrics 1996;97(3): 424-34.
      d. Nutritional deficiencies may be common in untreated gastrointestinal disease. Iron deficiency is common in many GI diseases, including celiac disease and inflammatory bowel disease. With many GI diseases, anorexia and poor PO intake are recorded, thus undernutrition is common. In intestinal failure/short gut syndrome, micronutrient deficiencies may be present but will depend on the area of the intestine affected. Ileitis or loss of the ileum may result in vitamin B12 deficiency. Fat soluble vitamins (A, D, E, and K) can be deficient in diseases that result in fat maldigestion or fat malabsorption. Diarrheal losses can result in zinc deficiency.
      e. The nutritional deficiencies listed above need to be considered as part of the therapy for individual patients and their intestinal diseases.
   2. Hepatic disease
      a and b. Chronic cholestasis can result in failure to thrive as a result of chronic fat maldigestion and fat soluble vitamin deficiency. Formulas and supplements high in MCT oil may be beneficial in these patients, as MCT oil does not require bile for absorption. Supplementation with fat-soluble vitamins is recommended with chronic cholestasis.
      c. Rickets can occur secondary to vitamin D deficiency from chronic cholestasis.
6. Fluid and Electrolyte Metabolism

D. Disease states, specific therapy

2. Gastroenteritis: Children in shock or with severe dehydration (>10%) should receive IV fluid therapy. Electrolytes must be carefully monitored and multiple boluses of IVF may be required.

9. Infectious Diseases

C. Specific Viral Pathogens


a and b: Hepatitis C screening is indicated in:
- children born to HCV infected mothers (HCV ab after 18 months of age; RNA for younger ages)
- children with chronically elevated transaminases (HCV AB)
- children from a region with high prevalence of HCV infection
- Referral should be made to a pediatric gastroenterologist if testing is positive.

C. Regular screening evaluations are important in children with Hepatitis C infection. Children with chronic Hepatitis C infection (while usually asymptomatic) may be eligible for therapy, and particularly as newer therapies are being developed, it is important to have gastroenterology follow up to determine whether a child is eligible for treatment. Annual screening for hepatocellular cancer (serum alpha-fetoprotein, abdominal u/s) is recommended for children with significant liver disease.

d. Long term outcomes of hepatitis C can vary but include: spontaneous resolution in infants, young children with chronic hepatitis who achieve spontaneous resolution (generally by age 7 or earlier), children with mild chronic hepatitis (are otherwise well, with mild ALT/AST elevation), and more severe liver disease. Cirrhosis has been reported in ~ 1-2% of children with chronic Hep C infection.

D. Bacterial Pathogens


14. Helicobacter pylori--NASPGHAN and ESPGHAN H. pylori working groups published evidence-based guidelines for the management of H. pylori infection in children:

12. Gastrointestinal Disorders

A. Acute and chronic abdominal pain
B. Abdominal Masses: Age-based differential diagnosis of an abdominal mass

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<thead>
<tr>
<th>Organ</th>
<th>Malignant Diseases</th>
<th>Nonmalignant Diseases</th>
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<td>Adrenal</td>
<td>Adrenal carcinoma</td>
<td>Adrenal adenoma</td>
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<td>Neuroblastoma</td>
<td>Adrenal hemorrhage</td>
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<td>Pheochromocytoma</td>
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<td>Gall bladder</td>
<td>Leiomyosarcoma</td>
<td>Choledochal cyst</td>
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<td>Gall bladder obstruction</td>
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<td>Hydrops (eg, leptospirosis)</td>
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<td>Gastrointestinal</td>
<td>Leiomyosarcoma</td>
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<td>Non-Hodgkin lymphoma</td>
<td>Intestinal duplication</td>
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<td>Intussusception</td>
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<td>Kidney</td>
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<td>Hydronephrosis</td>
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<td>Renal neuroblastoma</td>
<td>Polycystic kidney</td>
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<td>Wilms tumor</td>
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<td>Focal nodular hyperplasia</td>
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<td>Liver metastases</td>
<td>Liver abscess</td>
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<td>Mesenchymoma</td>
<td>Storage disease</td>
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<td>Lower genitourinary</td>
<td>Ovarian germ cell tumor</td>
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<td>Rhabdomyosarcoma of bladder</td>
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<td>Rhabdomyosarcoma of prostate</td>
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<td>Spine</td>
<td>Acute or chronic leukemia</td>
<td>Congestive splenomegaly</td>
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<td>Histiocytosis X (possibly non-</td>
<td>Mononucleosis</td>
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<td>malignant)</td>
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<td>Retroperitoneal rhabdomyosarcoma</td>
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<td>Retroperitoneal germ cell tumor</td>
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C. Vomiting

1b. The presence of bilious emesis in early infancy nearly always necessitates surgical evaluation. The differential includes malrotation with volvulus, intestinal atresia, meconium ileus, meconium plug, and Hirschsprung’s Disease. Appropriate management includes stabilization of the infant, which could include surgical consultation, NG decompression, NPO, IV fluid resuscitation, and antibiotics. Further imaging, including an acute abdominal series as well as fluoroscopic evaluation including upper GI series or barium enema may be indicated depending on clinical presentation.

1c. While the differential diagnosis for projectile vomiting in an infant can be broad and possibly benign, gastric outlet obstruction (specifically pyloric stenosis) should be considered. Diagnosis of pyloric stenosis is generally made by history, physical, and labs, and is confirmed by pyloric ultrasound (which would demonstrate thickening of the pylorus).

1d. Selective serotonin receptor antagonists may be beneficial in both prevention and treatment of vomiting. Ondansetron, a 5-HT3 acts in the chemotrigger zone and vagal afferents in the intestine.

D. Esophageal Disorders

1b. Rumination can occur at any age and is characterized by the effortless regurgitation of gastric contents that are then expelled, re-chewed, or re-swallowed. Initially described in children with neurologic impairment, it can occur in developmentally normal children. Rumination occurs as a voluntary (but usually unintentional) abdominal wall contraction, which results in relaxation of the lower and upper esophageal sphincters and passage of stomach contents into the mouth. Regurgitation is passive passage of gastric contents into the esophagus, which may or may not end up in the mouth.

1c. Structural anomalies can interfere with normal esophageal function including esophageal strictures and webs, achalasia, and duplication cysts. These are usually diagnosed via fluoroscopy study (esophagram or upper gastrointestinal series). The use of fluoroscopic studies in the evaluation of reflux is limited to evaluating for anatomic anomalies that can mimic GER(D) symptoms.

2c. NASPGHAN (North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition) and ESPGHAN (European Society for Pediatric Gastroenterology, Hepatology, and Nutrition) published updated clinical practice recommendations for pediatric gastroesophageal reflux. This is a good reference with algorithms for several different scenarios.


H. Gastrointestinal bleeding

J. Hepatomegaly: Causes of Hepatomegaly (with or without splenomegaly)

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<thead>
<tr>
<th>A. NEONATE</th>
<th>B. CHILD</th>
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<tr>
<td><strong>COMMON</strong></td>
<td><strong>UNCOMMON</strong></td>
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<td>Biliary tract obstruction</td>
<td>Hepatoblastoma/hemangiomatosis Anemias</td>
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<td>Congestive heart failure</td>
<td>Hemophagocytic syndrome/histiocytosis</td>
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<td>Maternal diabetes</td>
<td>Isoimmunization</td>
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<td>Malnutrition</td>
<td>Neuroblastoma</td>
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<td>Pseudohepatomegaly</td>
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<td>Sepsis</td>
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(Table from Wolf AD et al, Hepatomegaly in Neonates and Children. Pediatrics in Rev 2000;21(9) 303-10.)
K. Malabsorption
  2. Mucosal disease (celiac disease): NASPGHAN published a guideline for the
diagnosis and treatment of celiac disease in children—good reference

  Celiac Disease Guideline Committee of the North American Society for
  Pediatric Gastroenterology, Hepatology, and Nutrition, Guideline for the
  Diagnosis and Treatment of Celiac Disease in Children: Recommendations
  of the North American Society for Pediatric Gastroenterology, Hepatology, and

L. Inflammatory Bowel Disease: An excellent review of Crohn's Disease and
Ulcerative Colitis for general pediatricians: Rufo PA et al, Health Supervision in the
Management of Children and Adolescents with IBD: NASPGHAN Recommendations.

29. Psychosocial Issues
B. Specific Problems and conditions
  2. Encopresis—in addition to overflow/constipation, need to consider
  postsurgical causes (i.e. post anorectal malformation repair/Hirschsprung's
  repair, spinal cord abnormalities including trauma and tumors)
  4. Rumination can occur at any age and is characterized by the effortless
  regurgitation of gastric contents that are then expelled, re-chewed, or re-
  swallowed. Initially described in children with neurologic impairment, it can
  occur in developmentally normal children. Behavioral intervention is the key
  to management of rumination syndrome.