Acute & Chronic Abdominal Pain
Pediatric Urgent Care Knowledge Series 2021

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Acute Abdominal pain

- Careful History and Exam
- Only 5% actually “emergent”
  - Emesis? Bloody, coffee ground, bilious
  - Stools? Watery, hard, melena, bloody, acholic.
- Visceral abdominal pain poorly localized.
- Once the parietal peritoneum becomes irritated (appendicitis), pain more localized.
- Referred pain usually is located in cutaneous dermatomes sharing same spinal cord level as visceral inputs
Life threatening causes

- Hemorrhage, obstruction, perforation
  - Trauma
  - Appendicitis
  - Intussusception
  - Malrotation with midgut volvulus
  - Incarcerated inguinal hernia
  - Adhesions with intestinal obstruction
  - Necrotizing enterocolitis
  - Peptic ulcer disease
  - Ectopic pregnancy
Common Causes

• Constipation
• Gastrointestinal infection
• Other infections
  • - Urinary tract infections
  • - Streptococcal pharyngitis
  • - Pneumonia
  • - Viral illnesses
  • - Pelvic inflammatory disease
  • - Mesenteric lymphadenitis
• Ruptured ovarian cyst
• Foreign body ingestion
• Colic
Other GI Causes

- IBD
- Pancreatitis
- Acute cholecystitis
- Abscess
- Abdominal Migraines
Non GI Causes

- HSP
- Hepatitis
- Sickle cell crisis
- Testicular/ ovarian torsion
- Acute porphyrias
- FMF
Evaluation

• Labs: CBC, UA, pregnancy test
• KUB: obstruction / perforation/ constipation
• USS / CT when no improvement
• PAIN CONTROL!
Appendicitis
• Appendicitis most common surgically treated source of abdominal pain in children
• Peak age: 12 years
• 1/3 rupture before surgery (within 24 – 48 hours)
  • Rupture in infants 70%; fecalith 50%
• S/S:
  • Anorexia, fatigue, indigestion
  • Periumbilical pain → inflammation involves parietal peritoneum → localized RLQ pain
  • Malposition appendix- different pain locations! Hip pain
  • Fever
  • N/V
• Sudden pain relief – RUPTURE!
• Perform Rectal → “pelvic appendicitis”
• Diagnosis
  • KUB not helpful (? Fecalith)
  • USS –useful in adolescent females to r/o adnexal pathology
  • CT > 90% sensitivity

• Complications post-op:
  • Infection
  • Abscess

• Chronic appendicular pain:
  • Chronic appendicitis
  • Recurrent acute appendicitis

• D/D – Typhlitis – leukemic with neutropenia
  • Yersinia, constipation, UTI, Crohn’s, PID, ovarian cyst, etc..
Cholecystitis, cholelithiasis, choledochololithiasis
Cholecystitis

- Gallbladder inflammation – gallbladder stasis
  - With/without stones (acalculous- after burn/trauma/illness)
  - Rare in children
  - RUQ pain, nausea, fever, anorexia.
- D/D:
  - Hepatitis
  - Hepatic abscess
  - Fitz-Hugh-Curtis syndrome (gonococcal peri-hepatitis)
  - Pancreatitis
  - Appendicitis
  - Pneumonia
  - Pyelonephritis / Kidney stones
• Diagnosis:
  • WBC
  • ? Elevated AST/ALT/bili
  • Amylase (elevated in 8%)
  • USS
  • HIDA – normal uptake, BUT with reduced concentration in GB bile
• Complications – 30% in children:
  • Perforation
  • Abscess
  • Empyema
• Rx: gut rest, IVF – prophy antibiotics not routinely recommended
• Pain control- can use morphine
Cholelithiasis / Choledocholithiasis

1. **Cholesterol stones:**
   - >50% cholesterol content (yellow / white)
   - Females / pregnancy
   - **Not seen on X-ray**

2. **Black pigment stones:**
   - Occur when there is increased direct bilirubin
   - Bile acid malabsorption (e.g. ileal resection)
   - Hemolysis
   - TPN
   - No gender predominance
   - **Seen on X-ray**

3. **Brown pigment stones:**
   - Infections predispose (e.g. E. coli, ascaris)
   - Asian countries
   - Seen with bile duct stricture- bile stasis
   - Common in bile duct
   - **Not seen on X-ray**
• Symptoms:
  • 90% no symptoms

• Choledocholithiasis:
  • CBD stone
  • Jaundice / pancreatitis
  • Symptoms: RUQ or epigastric pain, nausea, and vomiting.
Figure 2. Illustrations of cholesterol and pigment stones.
• Risk Factors:
  • Obesity
  • Females, after puberty
  • Hemolysis
  • Reduced bile salt pool (e.g. ileal resection / short gut, crohn’s, CF pancreatic insufficiency)
  • TPN $\rightarrow$ bile stasis
  • Medications: ceftriaxone, furosemide, octreotide, cyclosporines
  • Down’s Syndrome
• Evaluation:
  • USS
  • ERCP / MRCP if stones in duct / CBD
  • Choledocholithiasis:
    • Elevated bili / GGT, AST / ALT

• Management:
  • Infancy – spontaneous resolution
  • Intra-uterine USS – normal finding
  • Surgical
  • Ursodiol for cholesterol stone dissolution.
  • ERCP stone removal

• Complications:
  • Pancreatitis
  • Cholangitis
Pancreatitis
The Normal Pancreas

• Exocrine Function (80%)
  • Pancreatic secretion has 2 functions:
    1) **Neutralizes** acidic chyme
       • By HCO3-
    2) **Digests** carbs, proteins and lipids
       • By enzymes: amylase, proteases, and lipase

• Endocrine Function (20%)
  • Pancreatic hormones (insulin and glucagon) regulate nutrient metabolism.
Protein Digestion

• The pancreas has 2 proteases: trypsin and chymotrypsin
• Kept in **inactive form** (trypsinogen, chymotrypsinogen) in zymogen granules.
So, why doesn’t the pancreas autodigest itself?

• **3 Lines of Defense:**
  
  • *1st line of defense*: inactive precursors (**PRSS1** codes for trypsinogen)

  • *2nd line of defense*: zymogens contain pancreatic secretory trypsin inhibitor (**PSTI/SPINK1**)

  • *3rd line of defense*: autolysis of prematurely activated trypsin
What happens in pancreatitis?

This ultimately leads to an autodigestive injury to the gland.
Acute Pancreatitis

Top 5 causes:

• Biliary
• Medications
• idiopathic
• systemic disease
• Trauma

Followed by:
• infectious
• metabolic
• hereditary
Acute Pancreatitis

Inflammation of the pancreas:

• **Symptoms (no specific pathognomonic s/s)**
  • Epigastric /RUQ pain
    • **Constant, eating makes worse (CCK), supine**
    • Referred pain
  • N/V
  • Anorexia
  • Occasionally jaundice
Signs

• Abdominal Distention
• Decreased Bowel Sounds
• Fever, leukocytosis
• Ascites
• Respiratory Distress
• Grey-Turner Sign (blue flanks)
• Cullen Sign (blue umbilicus)
Grey Turner’s sign (ecchymoses of the flanks) and Cullen’s sign (ecchymoses of the umbilical region) indicate extravasation of hemorrhagic exudate.
Diagnostic Laboratory Tests

- Amylase
- Lipase

3 times the upper limit of normal

The degree of elevation of amylase or lipase is not a marker of disease severity.
Common Laboratory Findings

- Elevated WBC, Increased % bands
- Hypocalcemia (Acute pancreatitis precipitates calcium as a soap in the abdomen)
- Hyperglycemia
- Elevated liver enzymes
- Increased LDH
Acute Pancreatitis: Imaging

• **U/S** can confirm presence of acute pancreatitis:
  • An enlarged, edematous-appearing pancreas suggests pancreatitis
  • Rules out obstructive anomalies
  • Identifies gallstones
  • A dilated main pancreatic duct indicates obstruction

• **CT:**
  • Would **not** get at initial presentation
  • However, it is useful several days into the diagnosis when pancreatic necrosis is suspected clinically.
  • Pseudocyst
Initial Management

- IVFs
  - Volume expansion early on may prevent pancreatic necrosis
- Pain control
- Monitoring vitals:
  - Fever?
  - Tachycardia/hypotension?
  - Tachypnea?
Nutritional Management

• NPO vs early feeding (<72hrs)?
  • No harm to early feeds
  • Enteral feedings help with gut integrity-promoting recovery
  • Fewer complications versus TPN

• NG vs NJ?
  • No difference.

• Versus PO?
  • Safe to do.

• What type of diet to start?
  • One study showed no difference between clear liquid diet vs low-fat solid diet
  • low-fat diet x 1 week

• Follow amylase/lipase?
  • No—labs are poor determinant of feeding success.
Recurrent and Chronic Pancreatitis
Causes of Recurrent Pancreatitis

• Drugs

• Anatomic Variant - Pancreatic divisum (single **pancreatic** duct is not formed- remains as two ), long common channel

• Systemic Disease
  • *CF, Hyperlipidemia, Hypercalcemia*

• Autoimmune

• Trauma (usually acute)

• Hereditary
  • *CFTR, SPINK1, PRSS1*
Hereditary pancreatitis: to screen or not to screen..?

Screening raises more questions than answers. Would only do it in limited cases with the guidance of GI, genetics or CF center.

<table>
<thead>
<tr>
<th>Gene</th>
<th>Key features</th>
<th>Diagnostic testing</th>
<th>Predictive testing/screening</th>
</tr>
</thead>
</table>
| PRSS1 | - Autosomal dominant  
   - High penetrance                                                                                                                                                                                    | Yes                | Yes/no*                      |
| SPINK1 | - High frequency in general population but low penetrance (NIAS mutation)  
   - Disease modifying rather than disease causing                                                                                                                                                    | No                 | No                           |
| CFTR  | - May be associated with CF disease  
   - Majority of the >1000 CFTR mutations have unknown functional and clinical significance, only a minority are disease-causing mutations                                                        | Sweat chloride test as primary test | Only in the context of CF disease |
Chronic Pancreatitis

• Irreversible
• Abdominal Pain
  • especially after high-fat/protein meals
• Exocrine Insufficiency
  • Steatorrhea
  • Fat-soluble vitamin deficiency (ADEK)
  • Poor growth
• Endocrine Insufficiency (Diabetes Mellitus)
Diagnosis-Pancreatic Insufficiency

• Non-invasive
  • Decreased amylase/lipase
  • 72-hour fecal fat
  • Fecal Elastase - unaffected by exogenous pancreatic enzyme treatment
  • Imaging: CT or MRCP

• Invasive
  • Pancreatic Stimulation Test - “gold standard” giving iv CCK and measuring bicarb/pancreatic enzymes in EGD duodenal aspirate
Schwachmann-Diamond Syndrome: a cause of Pancreatic Insufficiency

• Autosomal recessive
• The phenotypic features:
  1. Exocrine pancreatic insufficiency
  2. Skeletal abnormalities
  3. Bone marrow dysfunction (pancytopenia and cyclic neutropenia)
     • recurrent infections
Intussusception, volvulus, malrotation, obstruction
Intussusception

- Part of intestine telescopes into lumen of adjoining bowel.
- Mesentery trapped
- Venous obstruction, edema of bowel wall
- Arterial obstruction & ischemia
- Perforation
Intussusception

• Anatomic location:
  • Ileo-colic, colo-colic, small bowel
• Peak incidence 4-14 months (between 2 months – 5 years)
• Mostly idiopathic
• **90% ileo-colic**
• 5% lead point
  • Enlarged peyer patches in infancy
  • Meckel
  • Polyps
  • Duplication cyst
  • Lymphoma
  • HSP hemmorhage
  • Post-op
• **Presentation:**
  • Obstructive: colicky abdominal pain, bilious emesis
  • “sausage shaped” abdominal mass
  • Later: passing blood/mucus PR
  • Only 1/3: Classic Triad
    • Paroxysmal pain
    • Vomiting
    • Currant jelly stool
• Evaluation:
  • KUB – perforation / obstruction
  • USS “donut sign”

• Treatment:
  • Hydrostatic reduction: saline enema
    • Success rate 75-90%
  • Air enema – safer & faster
    • Success 75-95%
  • Enemas contraindicated with shock /peritonitis – surgery
  • Perforation risk: present for > 48 hours, Age < 6 months
  • Can recur 3-10%
Malrotation / volvulus

- 10-12 weeks gestation
- Can present any time
- Gastrochisis and omphalocele always have malrotation.
- “Non-rotation”:
  - Third part of duodenum / lig. Treitz lies to RIGHT of vertebral column
  - Cecum upper abdomen to LEFT of duodenum
- Presentation:
  - Acute obstruction/ volvulus, metabolic acidosis
  - 80% of those who are symptomatic- within 1st month of life.
- Diagnosis – UGI “bird’s beak” (volvulus of 3-4 part duodenum)
- Treatment – Ladd’s procedure
Functional Abdominal Pain/
Irritable Bowel Syndrome
Chronic Abdominal Pain - Epidemiology

• >2 months
• Chronic Pain significant medical problem in children.
• One of the most common pain syndromes
• 15% of all children
• Unexplained abdominal pain accounts for up to 25% of referrals to tertiary gastroenterology clinics
• Interferes with daily functioning
• Missed school days and impaired quality of life
• No diagnostic tests for FAP; diagnosis of exclusion / fulfills criteria
• No underlying identifiable disease process, either biochemical or structural.
• Recurrent pain → sensitization of the peripheral and/or CNS → hyperalgesia
• Children with FAP have been shown to have increased visceral sensitivity/ **visceral hyperalgesia**
• **Hyperalgesia** manifesting as decreased pain thresholds/ increased pain perception/ sensitivity
• Somatic response to “stress’?  
• Starting school common event
• Disappear over summer!
• Peripheral **5-HT (serotonin)** contributes to peripheral sensitization
Symptoms

• Periumbilical/generalized/mid-line
• Unlikely unilateral pain
• Unrelated to meals/activity/stool
• **Growth normal**
• No systemic s/s
Associations

• Depression and anxiety are common in FAP
• anxiety disorder in 79%
• depressive disorder in 43%
• Adults with a history of childhood FAP were significantly more likely than controls to experience:
  • anxiety,
  • hypochondriasis,
  • social dysfunction,
  • somatization
  • more likely to be taking psychoactive medication
Diagnosis

• r/o Organic causes: **H&P**
  • Meds use, e.g. chronic NSAID, doxy for acne
  • Sports, musculoskeletal
  • Pain- LIQR AAA
  • Bowel habits
  • Stressors
  • Growth
  • Thorough physical
  • **RULE OUT CONSTIPATION**
    • Hemeoccult
Diagnosis Criteria

• **Childhood Functional Abdominal Pain**
  
  *Diagnostic criteria* *Must include all of the following:*
  
  • Episodic or continuous **abdominal pain**
  
  • Insufficient criteria for other FGIDs
  
  • **No evidence** of an inflammatory, anatomic, metabolic, or neoplastic process that explains the subject’s symptoms

• *Criteria fulfilled at least once per week for at least 2 months prior to diagnosis*
Diagnostic tests?

- NASPGHAN guidelines:
  - No yield for routine:
    - Labs
    - USS, imaging,
    - endoscopy
  - Unless “alarm symptoms”
- Let symptoms guide you:
  - Comp/CBC/ESR/CRP
  - Celiac titers: TTG IgA, Total IgA
  - Amylase/lipase
  - β-hcg
- Stool - If have diarrhea – stool cx/ giardia
- H pylor? Controversial
- Imaging? – e.g. RUQ pain, HSM on exam

- Organic and FAP CAN coexist – can have IBD AND FAP
Differential
Musculoskeletal: Lower Rib Pain Syndrome

• Rib-tip syndrome, slipping rib, twelfth rib, and clicking rib
  • pain in the lower chest or upper abdomen
  • tender spot on the costal margin
  • reproduction of the pain by pressing on the spot
• “hooking maneuver”
• Naprosyn, heat pad, physical therapy
• Check ribs
“Reflux”

• Esophagitis/gastritis/ulcers

• Trial PPI ~8 weeks
Functional Constipation

Must include **two or more (for >4y/o)**

- **Two or fewer** defecations in the toilet per week
- At least one episode of **fecal incontinence** per week
- History of **retentive posturing** or excessive volitional stool retention
- History of **painful or hard** bowel movements
- Presence of a **large fecal mass in the rectum**
- History of large diameter stools which may **obstruct the toilet**
Constipation

- **Do a rectal please!**
- **Miralax cleanout:**
  - 1.5 gm/Kg/day - BID
  - 2-3 capfuls daily x 2-3 days
- **Maintenance Miralax:**
  - Taper to achieve “pudding” /”applesauce”/”soft-serve ice cream” stools DAILY
- **Fluid & fiber**
- **Regular toilet sitting**
## Bristol Stool Chart

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>Separate hard lumps, like nuts (hard to pass)</td>
</tr>
<tr>
<td>Type 2</td>
<td>Sausage-shaped but lumpy</td>
</tr>
<tr>
<td>Type 3</td>
<td>Like a sausage but with cracks on its surface</td>
</tr>
<tr>
<td>Type 4</td>
<td>Like a sausage or snake, smooth and soft</td>
</tr>
<tr>
<td>Type 5</td>
<td>Soft blobs with clear-cut edges (passed easily)</td>
</tr>
<tr>
<td>Type 6</td>
<td>Fluffy pieces with ragged edges, a mushy stool</td>
</tr>
<tr>
<td>Type 7</td>
<td>Watery, no solid pieces. <strong>Entirely Liquid</strong></td>
</tr>
</tbody>
</table>
Functional Dyspepsia

• Persistent or recurrent pain or discomfort centered in the **upper abdomen (above the umbilicus)**

• Not relieved by defecation or associated with the onset of a change in stool frequency or stool form (*i.e., not irritable bowel syndrome*)

• **No evidence** of an inflammatory, anatomic, metabolic or neoplastic process that explains the subject’s symptoms

• Fullness, EARLY satiety, bloating, nausea, vomiting

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- D/D PUD, crohns, celiac
- Rx – trial PPI 6 weeks
- Periactin / Iberogast
Irritable Bowel Syndrome

- Abdominal discomfort:
  - Improvement with defecation
  - Onset associated with a **change in frequency/form** of stool
  - **No evidence** of an inflammatory, anatomic, metabolic, or neoplastic process that explains the subject’s symptoms

- **Altered bowel habit key**
  - Stool Cx – giardia, r/o celiac
  - Rx – rifaximin/flagyl/ probiotics/levsin/fiber
  - Low FODMAP diet (Fermentable Oligosaccharides, Disaccharides, Monosaccharides and Polyols.)
Abdominal Migraine

- Episodes of **acute periumbilical pain** that lasts for hour or more
- **Intervening** periods of usual health lasting weeks to months
- The pain **interferes** with normal activities
- The pain is associated with 2 of the following:
  - a. Anorexia
  - b. Nausea
  - c. Vomiting
  - d. Headache
  - e. Photophobia
  - f. Pallor
- No evidence of an inflammatory, anatomic, metabolic, or neoplastic process considered that explains the subject’s symptoms

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- Family Hx migraine
- Periactin/TCA
Treatment

• REASSURANCE – but acknowledge pain
  • NOT CANCER - Therapeutic relationship – regular follow up – pain real but no underlying serious disease – explain it is common (15%) in children
  • “pain real” – phantom limb/migraine headache examples
• Return to normal function
• BACK TO SCHOOL
• Don’t need a referral to GI to start treatment
• Regular Exercise
Treatment

• Dietary: Low FODMAP diet

• **fermentable oligo-, di-, mono-saccharides and polyols** (groups of carbs that are notorious for triggering digestive symptoms)

• The main dietary sources of the four groups of FODMAPs include:
  - **Oligosaccharides:** Wheat, rye, legumes and various fruits and vegetables, such as garlic and onions.
  - **Disaccharides:** Milk, yogurt and soft cheese. **Lactose** is the main carb.
  - **Monosaccharides:** Various fruit including figs and mangoes, and sweeteners such as honey/ agave nectar. **Fructose** is the main carb.
  - **Polyols:** Certain fruits and vegetables including blackberries and lychee, as well as some low-calorie sweeteners like those in sugar-free gum.
Treatment: Cyproheptadine “Periactin”

- Cyproheptadine:
  - antihistamine (H1 receptor antagonist),
  - Anticholinergic
  - serotonin receptor antagonist.
- Used for years in children
- **At least 2 weeks to act!**
- RCT: Cyproheptadine effective in FAP
- Cyproheptadine relatively safe with common side effects:
  - appetite stimulation - *Teenagers with weight gain issue*
  - Sedation (bedtime dosing).
- Cyproheptadine safer than tricyclic anti-depressants (TCA)
• Relative Contraindications to use:
  • Using other CNS depressants

• Dose:
  • 0.25 mg/kg/day in 2-3 divided doses or
  • 2-6 years: 2 mg every 8-12 hours (not to exceed 12 mg/day)
  • 7-14 years: 4 mg every 8-12 hours (not to exceed 16 mg/day)
  • Adults: 4-20 mg/day divided every 8 hours (not to exceed 0.5 mg/kg/day)

• Syrup: 2 mg/5 mL
• Tablet: 4 mg
Antidepressants
TCA: Amitriptyline

• Open label trial- 84% reported improvement
• Initial: 0.1 mg/kg at bedtime
• May advance as tolerated over 2-3 weeks to 0.5-2 mg/kg at bedtime
• SE: anticholinergic, sedation,
• CVS: **Baseline EKG/at dose increase**

*Monitor for depression, suicidality, and associated behaviors (especially at the beginning of therapy or when doses are increased or decreased).*

• SSRIs – not studied as adequately in pediatrics as TCAs
Anti-spasmodics

- Levsin (Hyoscyamine): 0.125mg sublingual q4-6 hours PRN
- SE: anticholinergic effects, urine output, GI symptoms, Constipation exacerbated
- More for cramping- but does help sometimes- especially at school- pop under tongue- acts quickly
Non-Pharmacological Therapies

• Psychology referral – “behavioral” - NOT crazy, deal/distract
• Self regulation therapies:
  • mind-body therapy,
  • Hypnosis – success rate 85% vs. 25% standard medical therapy
  • biofeedback,
  • guided imagery,
  • meditation
  • relaxation techniques.
• Studies have demonstrated physiologic responses to relaxation:
  • decreased oxygen consumption, blood pressure, heart rate, serum lactic acid levels and tonic muscle tensions.
Guided Imagery

• A state of deep relaxation is induced by guiding the subject to actively create images which aim to resolve specific problems.
• requires the subject to generate his/her own solution to the problem instead of relying on the therapist to form this plan.
• The use of guided imagery allows for *deep relaxation and reduces anxiety*
• DVDs to teach guided imagery and audio CDs to aid in the daily practice of guided imagery:
  • 63.1% response rate, compared to a 27% response rate in children standard medical care.
• App
Complementary

- Pain diary
- Herbal – e.g. peppermint
  - menthol in peppermint relaxes GI smooth muscles by blocking calcium channels
  - placebo RCT - 76% decreased s/s vs 19% placebo
- Massage therapy - reduce excitation of visceral afferent fibers
- Acupuncture
Reassurance

Labs/Trial PPI/Treat Constipation

Cyproheptadine/Psychology referral

TCA

GI referral
Alarm....

- Weight loss/poor weight gain
- Early satiety
  - Can see early satiety in functional dyspepsia too.......... Ask “full quickly”- what about favorite food?
- Bloody diarrhea
- Night time (AWAKENS vs difficulty falling asleep)
- Systemic s/s
- Vomiting/dysphagia
- Abnormal labs
- Strong family history
Long term....

• 25-50% of children continue to experience symptoms into adulthood
• many children reportedly have complete resolution of their symptoms within months of diagnosis and others within 2-5 years
• Secondary gain....
Lactose Intolerance

- Lactase → Lactose → glucose + galactose
- Adult hypolactasia 75% population – less among Caucasians.
- Develop in childhood AFTER 4 years of age
- “congenital” – RARE, autosomal recessive.
- Preterm- “relative” deficiency, corrects with age
- Adolescence – 90-95 % decrease in lactase activity of newborn!
• Presentation:
  • Most have no symptoms
  • Within 3-4 hours of lactose ingestion: Colicky abdominal pain, flatulence, diarrhea
  • Most can tolerate up to 12 grams lactose (1 cup milk)

• Diagnosis:
  • Breath hydrogen test (increase breath hydrogen from colon bacterial fermentation of unhydrolyzed/unabsorbed lactose)- rise of >10-20ppm
    • False + SBBO, rapid transit
    • False – decreased motility, antibiotics
  • Stool reducing substances +
  • Small bowel biopsy with direct lactase activity measurement
  • Clinical trial of lactase or lactose free diet

• Treatment:
  • Lactase – yeast derived
Poll Question

A 15-month-old boy has rectal prolapse. Stools sometimes contain blood. Physical examination reveals a child who appears well nourished. The mucosal prolapse is easily reducible; other findings on rectal examination are normal. Of the following, the MOST likely cause for this boy's rectal prolapse is:

a. chronic constipation
b. cystic fibrosis
c. rectal polyp
d. trichuriasis
Poll Question

Children with acute pancreatitis must only be fed via NJ:

a. True  
b. False
Poll Question

16 year old female has 6 months of abdominal pain, with maintained weight, worse with stress, normal labs. Next steps:

a. Endoscopy
b. Upper GI
c. Low fat diet
d. Reassurance
Poll Question

16 year old female has 6 months of abdominal pain, with 15 pound weight loss, diarrhea, and night time awakening Next steps:

a. Endoscopy
b. Upper GI
c. Low fat diet
d. Reassurance
References

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