Acute & Chronic Abdominal Pain

Pediatric Urgent Care Knowledge Series 2021

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Pediatric Gastroenterology

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

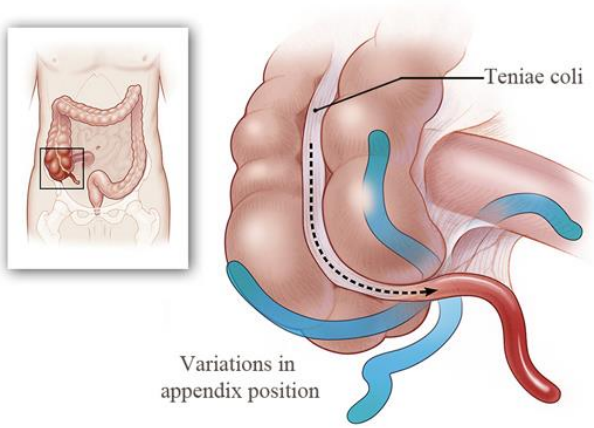
- HSP
- Hepatitis
- Sickle cell crisis
- Testicular/ ovarian torsion
- Acute porphyrias
- FMF

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, choledocholithiasis

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

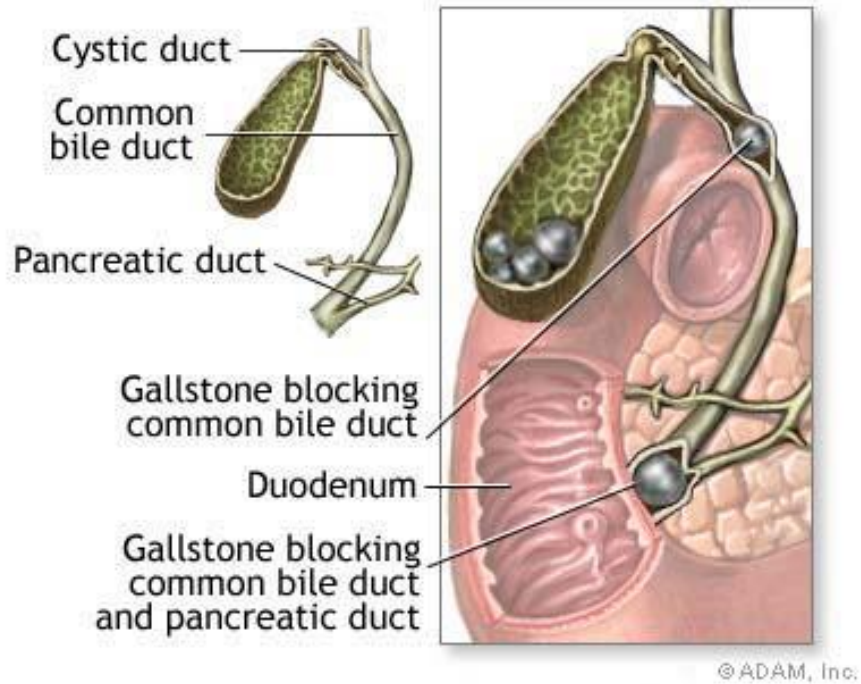
Cholelithiasis / Choledocholithiasis

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

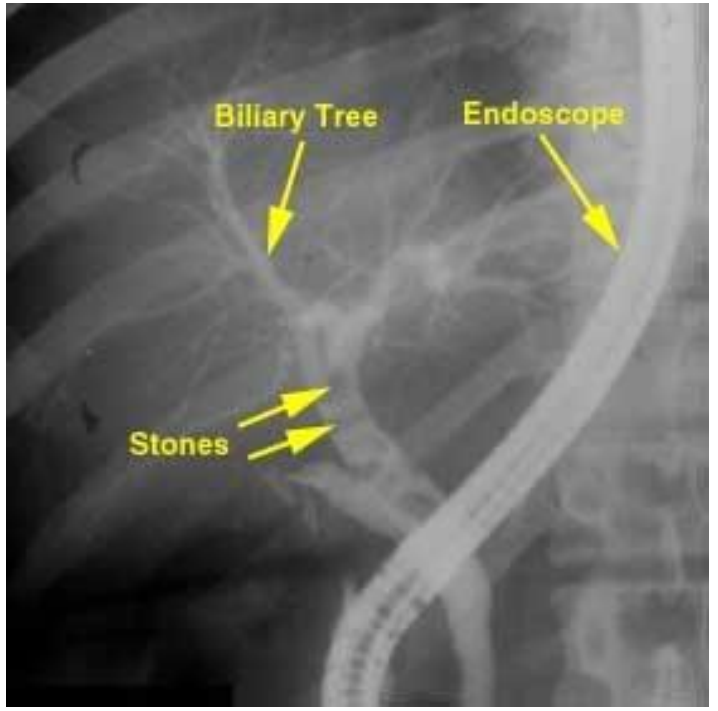
- Choledocholithiasis:
  - CBD stone
  - Jaundice / pancreatitis
  - Symptoms: RUQ or epigastric pain, nausea, and vomiting.

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 → 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

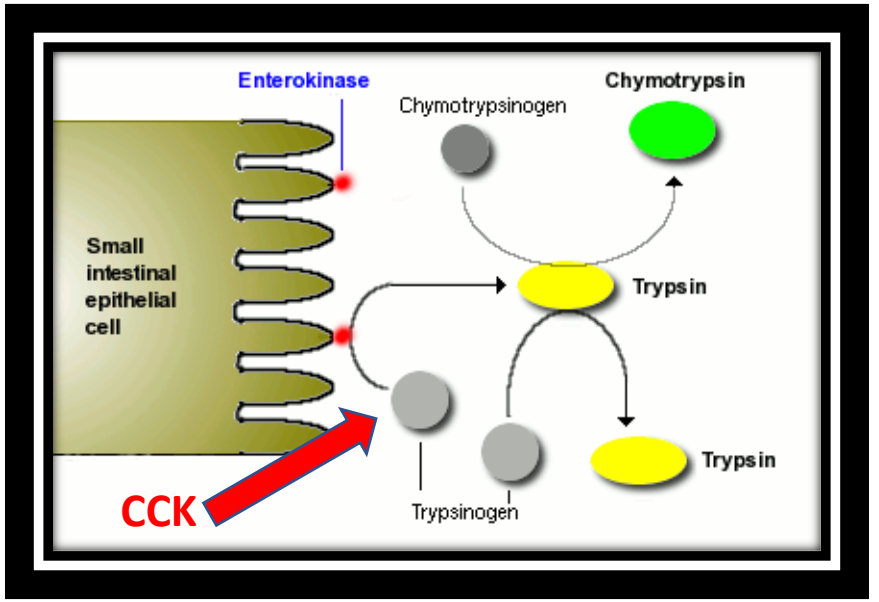
The Normal Pancreas

- **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

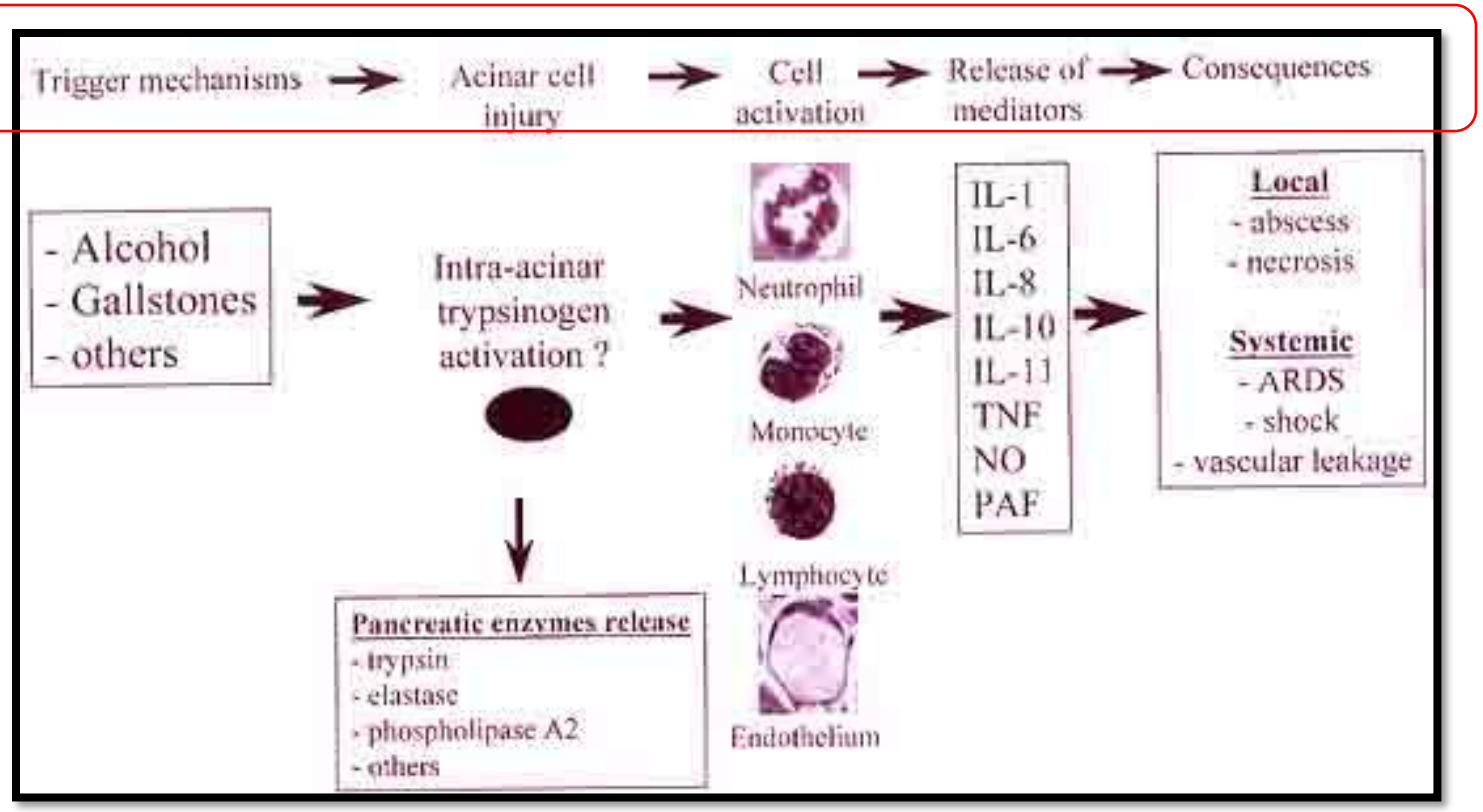
- The pancreas has 2 proteases: trypsin and chymotrypsin
- Kept in ***inactive form*** (trypsinogen, chymotrypsinogen) in zymogen granules.



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 Sign

Cullen Sign

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.*

TABLE 3. Reasons for false-positive amylase or lipase elevations

Amylase (29)	Metabolic
Abdominal causes	Diabetic ketoacidosis
Biliary tract disease	Drugs
Intestinal obstruction/ischemia	Opiates
Mesenteric infarction	Phenylbutazone
Peptic ulcer	Trauma
Appendicitis	Cerebral trauma
Pancreatic cancer	Burns
Ruptured ectopic pregnancy	Renal disease
Prostate disease	Renal insufficiency
Ovarian neoplasm	Renal transplantation
Afferent loop obstruction	Macroamylasemia
Dissecting aortic aneurysm	Lipase (30,31)
Nonabdominal causes	Pancreatic cancer
Salivary gland	Nonpancreatic abdominal pain
Salivary trauma	Macrolipasemia
Infection (mumps)	Renal insufficiency
Salivary duct obstruction	Acute cholecystitis
Irradiation	Esophagitis
Thoracic	Hypertriglyceridemia
Myocardial infarction	
Pulmonary embolism	
Pneumonia	
Metastatic lung cancer	
Cardiopulmonary bypass	

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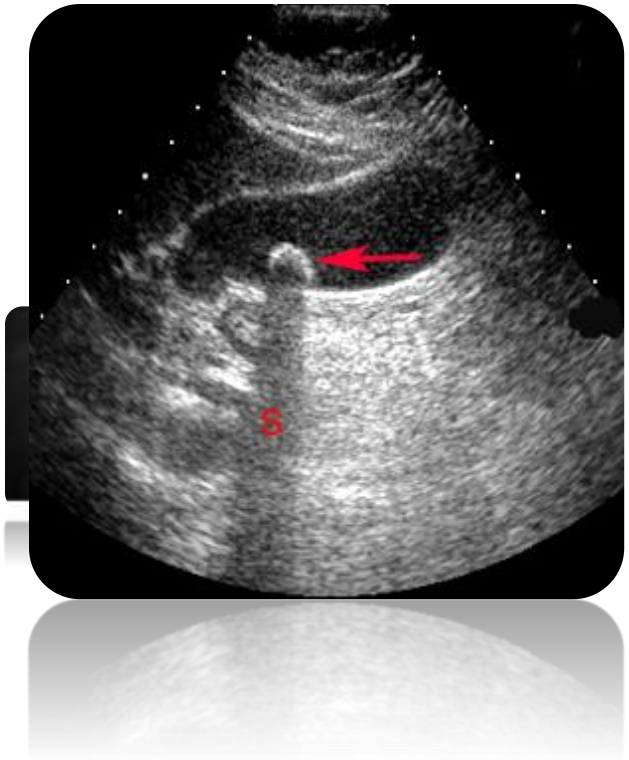
Common Laboratory Findings

- 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?

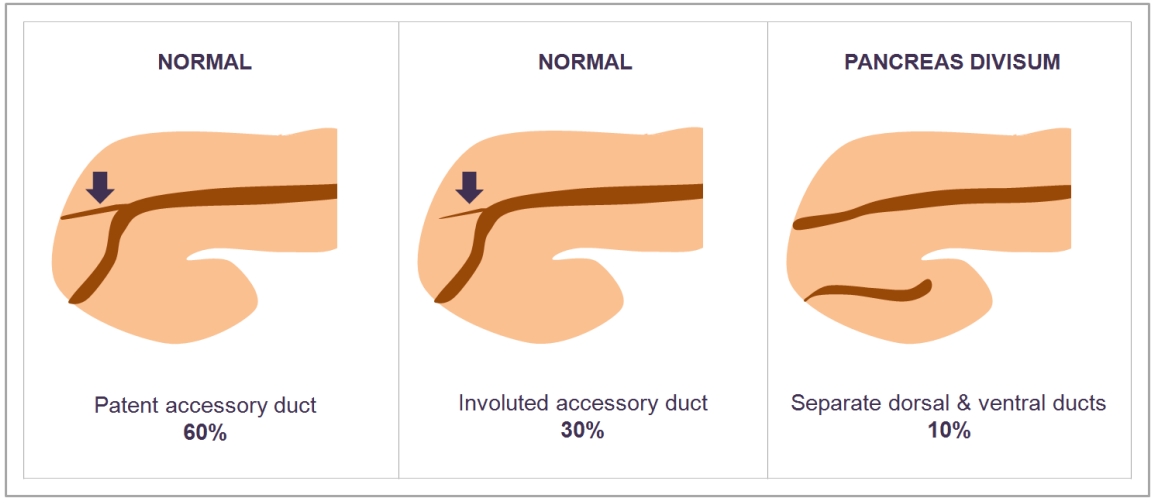
Nutritional Management

- ***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

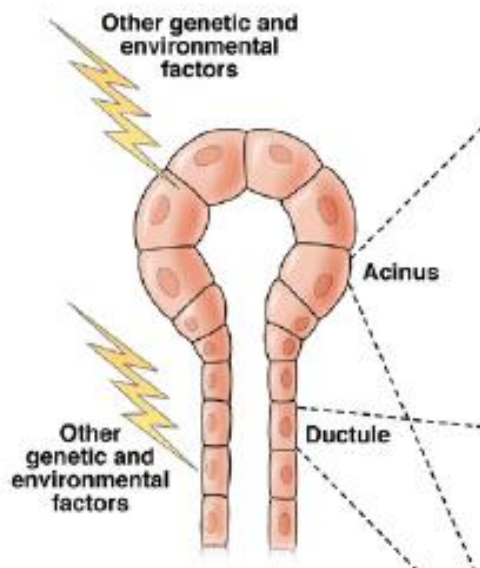
Causes of Recurrent Pancreatitis

- 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.



Gene	Key features	Diagnostic testing	Predictive testing/ screening
PRSS1	• Autosomal dominant • High penetrance	Yes	Yes/no*
SPINK1	• High frequency in general population but low penetrance (N34S mutation) • Disease modifying rather than disease causing	No	No
CFTR	• May be associated with CF disease • Majority of the >1600 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)

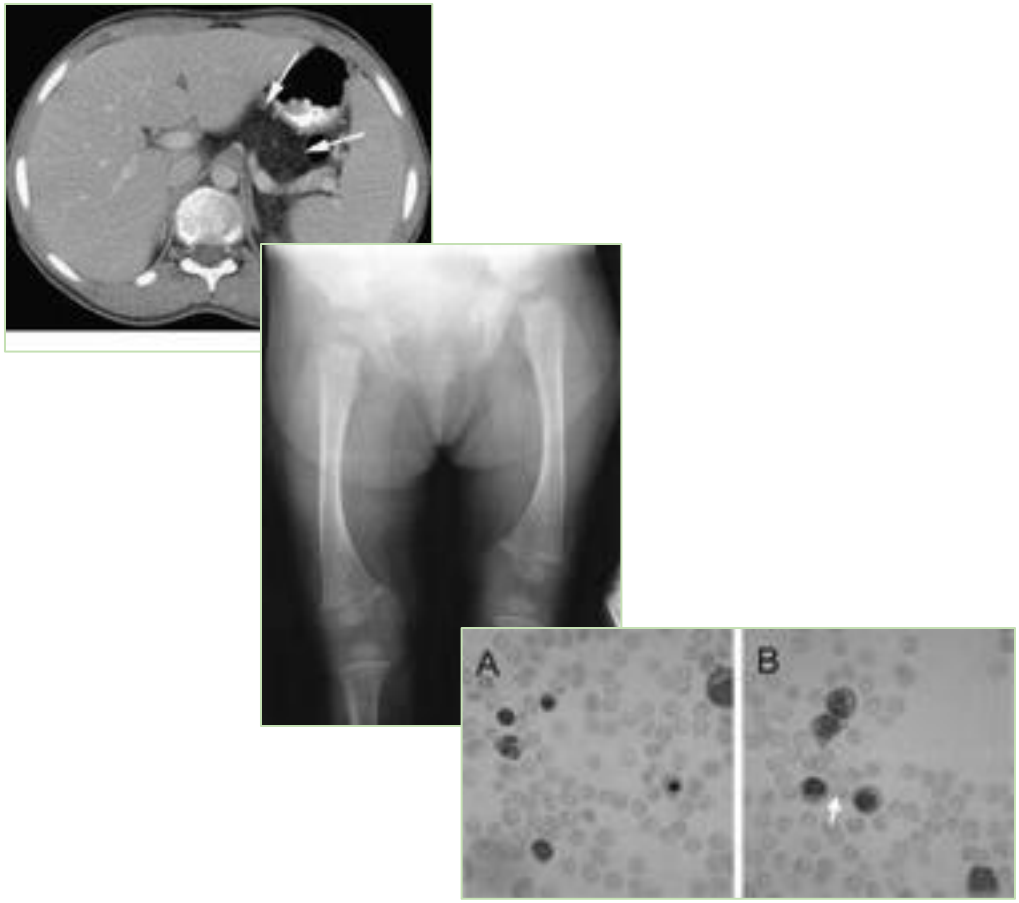
Diagnosis-Pancreatic Insufficiency

- 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

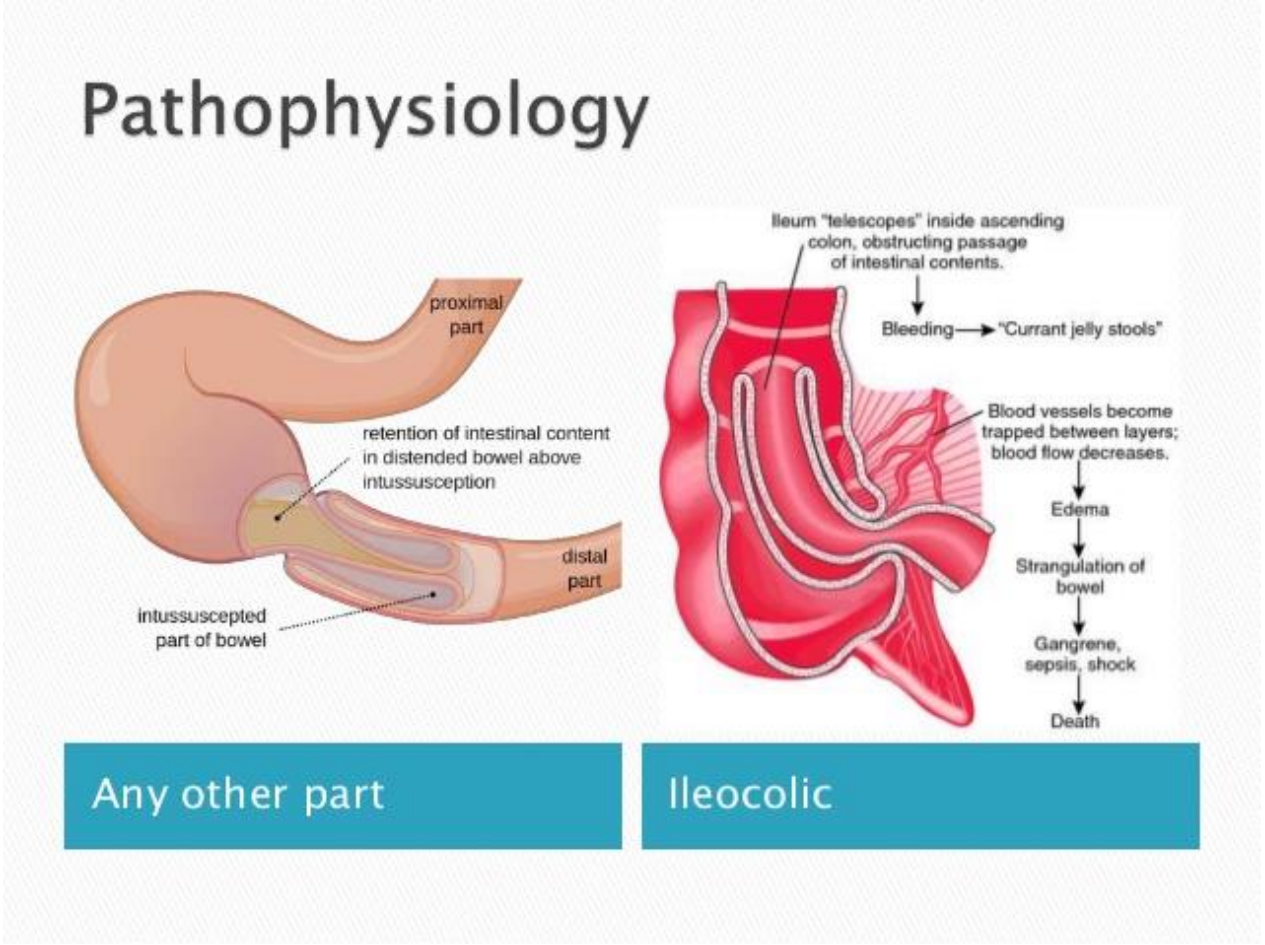
- Autosomal recessive
- **The phenotypic features:**
  1. Exocrine pancreatic insufficiency
  2. Skeletal abnormalities
  3. Bone marrow dysfunction (pancytopenia and cyclic neutropenia)
     - recurrent infections



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

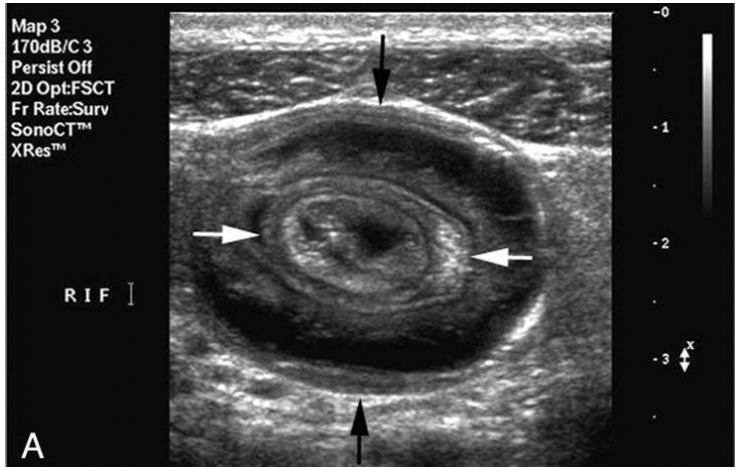


Intussusception

- 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

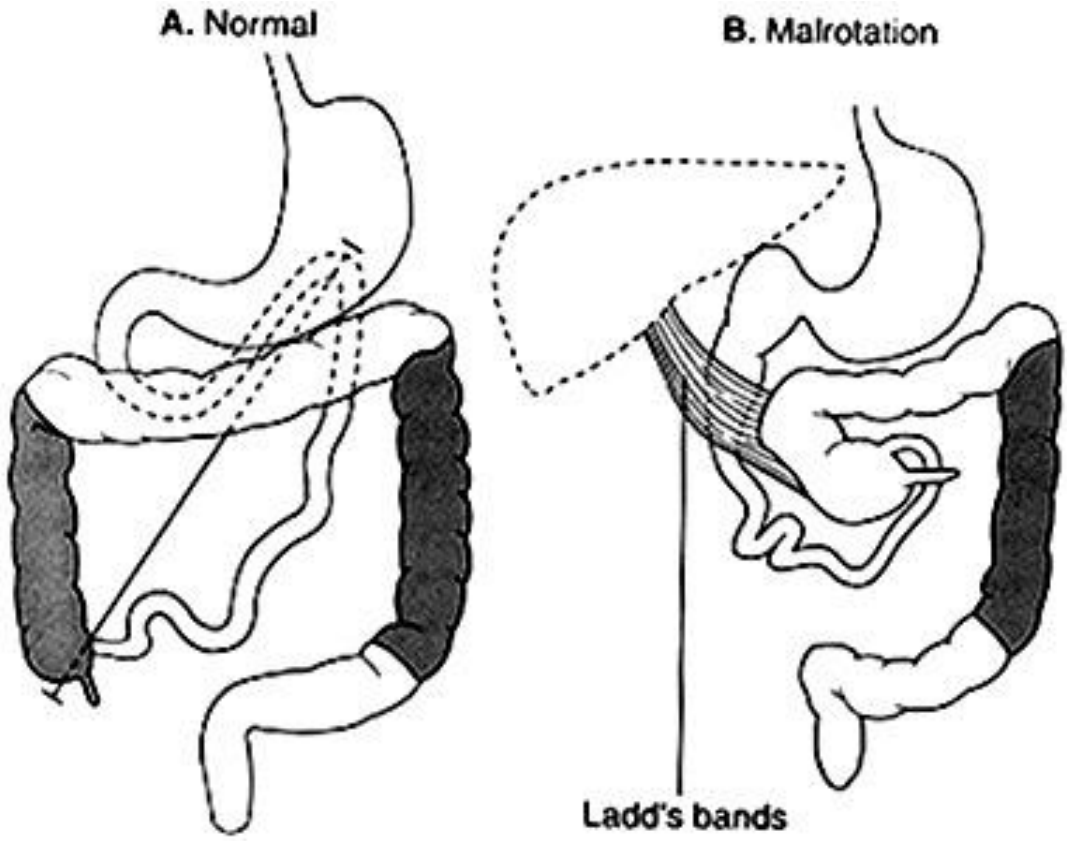
- Presentation:
  - Obstructive: colicky abdominal pain, bilious emesis
  - “sausage shaped” abdominal mass
  - Later: passing blood/mucus PR
  - Only 1/3: Classic Triad
    - Paroxysmal pain
    - Vomiting
    - Currant jelly stool

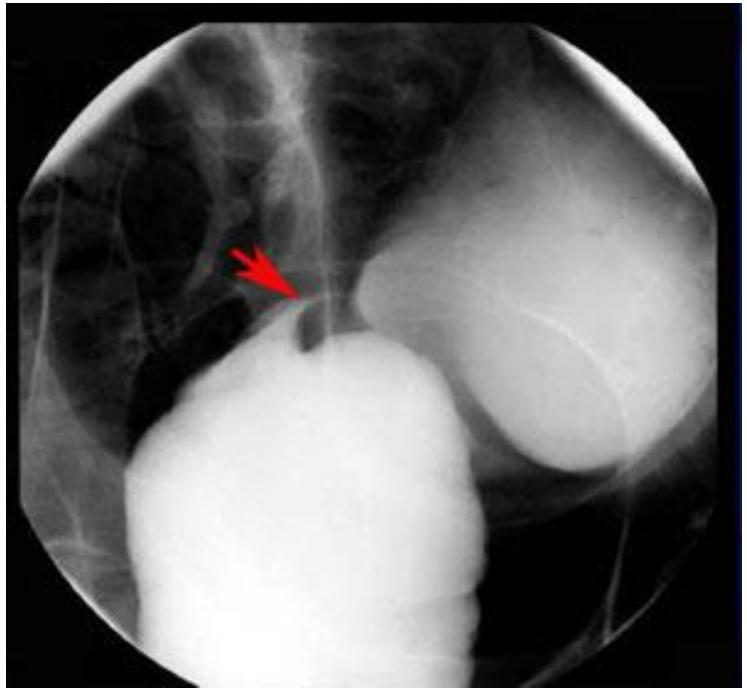
- Evaluation:
  - KUB – perforation / obstruction
  - **USS “donut sign”**
- Treatment:
  - Hydrostatic reduction: saline enema
    - Success rate 75-90%
  - Air enema – safer & faster
    - Success 75-95%
  - Enemas contraindicated with shock /peritonitis – surgery
  - Perforation risk: present for > 48 hours, Age < 6 months
  - Can recur 3-10%



Malrotation / volvulus

- 10-12 weeks gestation
- Can present any time
- Gastroschisis 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


A. Normal
B. Malrotation
Ladd's bands



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 2months prior to diagnosis

Diagnostic tests?

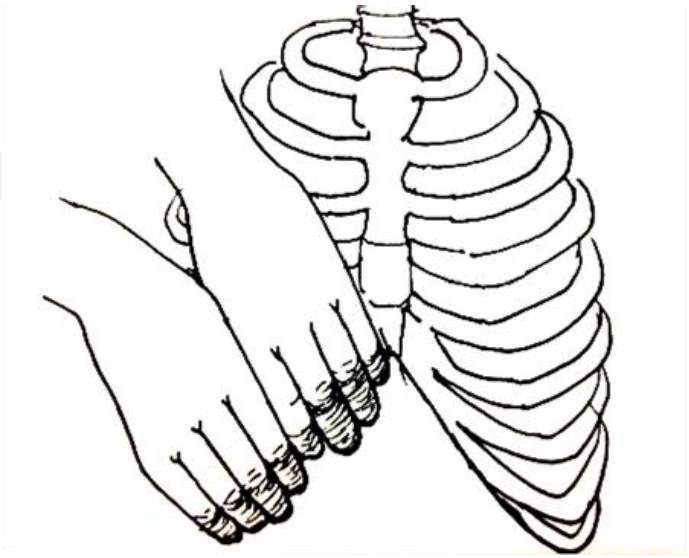
- NASPGHAN guidelines:
  - No yield for routine:
    - Labs
    - USS, imaging,
    - endoscopy
  - Unless “alarm symptoms”
- Let symptoms guide you:
  - Comp/CBC/ESR/CRP
  - Celiac titers: TTG IgA, Total IgA
  - Amylase/lipase
  - β-hcg
- Stool - If have diarrhea – stool cx/ giardia
- H pylor?Controversial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

Type 1		Separate hard lumps, like nuts (hard to pass)
Type 2		Sausage-shaped but lumpy
Type 3		Like a sausage but with cracks on its surface
Type 4		Like a sausage or snake, smooth and soft
Type 5		Soft blobs with clear-cut edges (passed easily)
Type 6		Fluffy pieces with ragged edges, a mushy stool
Type 7		Watery, no solid pieces. **Entirely Liquid**

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

- 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

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  - **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 a**cute 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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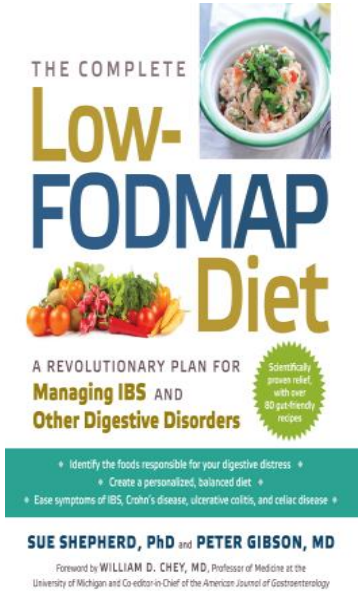
- Family Hx migraine
- Periactin/TCA

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 peds 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

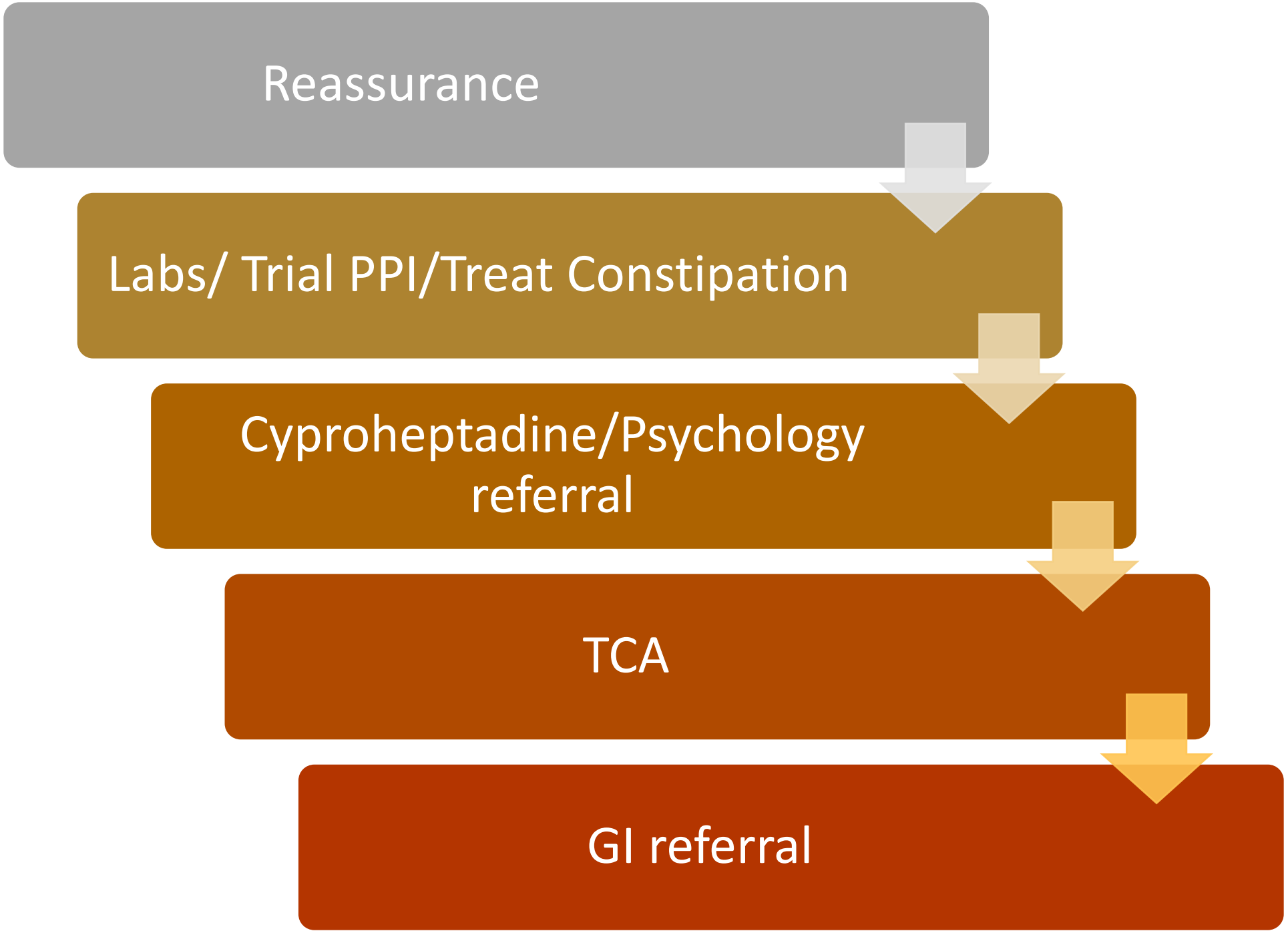
- **Psychology referral – "behavioral"** - NOT crazy, **deal/distract**
- Self regulation therapies:
  - mind-body therapy,
  - Hypnosis – success rate **85% vs. 25% standard** medical therapy
  - biofeedback,
  - guided imagery,
  - meditation
  - relaxation techniques.
- Studies have demonstrated physiologic 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

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