Pediatric GI Emergencies

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PEDiATRIC GI EMERGENCIES

I. MALROTATION

A. Normal bowel development prenatally involves counterclockwise rotation resulting in the C-curve of the duodenum with the ligament of Treitz on the left and the ascending colon on the right. The duodenum, ascending, and descending colon become relatively fixed to the posterior abdominal wall. The small bowel mesentery attachment extends from the left upper quadrant to the right lower quadrant. As a result, the bowel is relatively stationary within the peritoneal cavity.

B. Failure in part or all of the rotational processes or mesenteric attachments can lead to: the entire midgut hanging freely in the peritoneal cavity without its usual anchorage posteriorly.

II. WITH MIDGUT VOLVULUS

A. This occurs when there has been no rotation or fixation of the bowel. The entire midgut is hanging freely in the peritoneal cavity and completely twists around on its pedicle.

B. Midgut volvulus is the most urgent of GI emergencies

   1. There is upper bowel obstruction at the level of the duodenum. The midgut vasculature is in the twisted pedicle and may become strangulated leading to ischemic necrosis of the entire midgut.
   2. In most cases, midgut volvulus occurs in the 1st month of life. However, some cases occur in older children or even adults. There will often be a history of intermittent abdominal pain, vomiting, or constipation.

C. Clinical findings

   1. Initially, **bilious vomiting** occurs due to the complete duodenal obstruction. In a short time the infant becomes extremely ill.
   2. Obstruction of the arterial supply leads to bowel infarction. Shock, acidosis, fluid and electrolyte derangements quickly become severe *within hours*.
   3. Abdominal distension and tenderness may or may not be present. This is because the bowel obstruction is high at the level of the duodenum. Blood in the emesis or stool is an ominous sign denoting bowel ischemia. Infants can also appear pale or even jaundiced.
D. Diagnosis

1. It is imperative to make the diagnosis and operate on the patient as soon as possible.
2. Plain abdominal X-rays will often show signs of duodenal obstruction: gas in the stomach and gas in a dilated duodenum. This is called the modified double-bubble sign. If this is seen in association with a paucity of gas distally in the rest of the bowel, this is highly suspicious for malrotation with midgut volvulus, no further tests are needed.
3. Diagnostic imaging is completed with an UGI. This will show duodenal obstruction, an abnormal location of the ligament of Treitz, and most commonly a “corkscrew” appearance of the small bowel.
4. Sometimes the diagnosis can be made with a barium enema. The findings are a cecum too high in the RUQ, or even in the midline. Problems with this test are that normal infants may have a high cecum and volvulus can occur with a normally located cecum.

E. Differential diagnosis

1. Any other causes of obstruction: atresia, gastric volvulus, pyloric stenosis, or duplications.

III. HIRSCHSPRUNG’S DISEASE

A. Definition and description

1. Hirschsprung’s disease is caused by congenital absence of parasympathetic ganglion cells in the lower gut. Variable lengths of bowel are involved. Usually the rectum or rectum and sigmoid colon are affected but the entire colon may be abnormal. The aganglionic segment lacks peristaltic movements and so presents a functional obstruction to bowel contents. The affected segment is normal in caliber and thickness. The segment just proximal to the obstruction becomes dilated, hypertrophied and filled with stool.
2. Clinical findings
   a. Most cases are diagnosed in the neonatal period when the infant fails to pass meconium by the first 24 hours. Later abdominal distension or even frank bowel obstruction can occur.
   b. If the affected segment is short, the obstruction may be minimal and patients may present much later. Chronic constipation and abdominal distension are the usual findings. Patients have sometimes been managed for long periods with daily enemas. Malnutrition is not uncommon.
c. The large dilated proximal segment can sometimes obstruct the ureters causing hydronephrosis.

3. Diagnosis
   a. Definitive diagnosis requires rectal biopsy demonstrating absence of ganglion cells.
   b. Barium enema may show normal segment of colon with proximal dilatation. (Not useful if stool has been evacuated or if entire colon is involved.)

4. Differential diagnosis
   a. The first concern is differentiating the functional obstruction of Hirschsprung’s disease from other mechanical causes.
   b. A more common problem is differentiating the congenital obstruction of Hirschsprung’s from encopresis which is usually due to toilet-training difficulties. The most important aspects to use in differentiation are:
      i. Hirschsprung’s is associated with earlier onset in infancy, small ribbonlike stools, normal sized, empty rectal pulla, frequent explosive rush of flatus and feces after rectal exam or enema, and a clear transition zone on barium enema. On the other hand, encopresis is more frequently associated with age 2 or older, frequent straining at stool, stool incontinence, rectum distended with feces, and no transition zone on barium enema.

5. Complications
   a. Large bowel obstruction: The only difference is that the obstruction is functional rather than mechanical.
   b. **TOXIC MEGACOLON IS A SERIOUS COMPLICATION OF HIRSCHSPRUNG’S DISEASE**
      i. Progressive enlargement of the proximal segment of bowel can lead to edema, inflammation, and ischemia. Eventually breakdown of the mucosa allows invasion of luminal bacteria and gram negative sepsis.
      ii. Clinical findings: abdominal pain, fever, abdominal distension, profuse diarrhea, abdominal tenderness, variable degrees of fluid and electrolyte arrangements, acidosis, and shock.
   c. Renal insufficiency: The large megacolon lying on top of the ureters can cause obstruction.

6. Management
   b. Bowel obstruction: admit, correct metabolic abnormalities, NG tube, surgical consultation.
   c. Enterocolitis: admit, correct metabolic abnormalities, antibiotics for GN sepsis, decompress colon with rectal tube, NG tube. Surgery is usually performed later unless perforation has occurred.
   d. Chronic constipation: elective surgery to prevent future
development of above problems. Only 5% of patients are managed conservatively without surgery.

IV. HYPERTROPHIC PYLORIC STENOSIS (HPS)

A. Definition and description

B. Infantile Hypertrophic Pyloric Stenosis is when the pyloric muscle becomes thickened and edematous. This obstructs the gastric outlet. The typical patient is: male; males are 4X more frequent than females (first born).

C. 2-6 weeks old (extreme range 5 days - 5 months)

1. Otherwise healthy
2. It is seen in approximately 3-5/1000 births; there is a multifactorial genetic component. The highest incidence is in males born to mothers who had the disease. (20%)

D. Clinical findings

1. Affected infants have progressive, increasingly severe vomiting that is always NONBILIous and occurs just after feeding. The vomiting becomes increasingly frequent and forceful. Eventually, many infants develop projectile vomiting, weight loss, dehydration. Patients are otherwise healthy.
2. Important features on exam to note are:
   a. Baby usually feeds hungrily after vomiting.
   b. Evidence of dehydration: when extreme "old man facies."
   c. Gastric peristaltic waves in the upper abdomen moving L to R.
   d. Careful examination of the right upper abdomen reveals the pyloric "olive" in 70-85% of patients. To increase the likelihood of palpating an olive, the stomach should be empty either after vomiting or NG aspiration. The baby should be asleep or peacefully feeding on clear liquids. The olive can be found just to the right of the epigastrium, under the liver edge or around the umbilicus.
   e. Some infants may develop blood-streaked emesis from prolonged or forceful vomiting.
   f. There should NEVER be bile in the emesis.
   g. Prolonged vomiting leads to characteristic metabolic abnormalities: hypochloremic, hypokalemic, metabolic alkalosis. This occurs as a result of the loss of hydrochloric acid (H+Cl-) from vomiting, and the kidney wasting potassium in exchange for holding onto H+ ions to lessen the alkalosis.
E. Diagnosis

1. Palpation of a characteristic olive in a vomiting infant is sufficient to make the diagnosis.
2. Ultrasound is currently the gold standard imaging modality of choice.
   a. If an experienced pediatric ultrasonographer is not available, then an UGI will demonstrate gastric outlet obstruction and a long, thin channel within the pylorus (the so-called “string sign”).

F. Differential diagnosis

1. Overfeeding, gastroenteritis, gastroesophageal reflux, or other causes of obstruction such as atresia, volvulus, diaphragmatic hernia.

G. Management

1. Consult pediatric surgery
2. Correct severe dehydration with normal saline using 20 cc/kg boluses as needed to restore euvoeia.
3. Check and correct electrolyte abnormalities as needed. Correct potassium deficiencies at 3-5 mg/kg/day or 20-40 mg/liter.
4. IV fluids should be D5NS or D5 1/2NS at 1.5 times maintenance.
5. Keep NPO. NG tube usually not necessary.

V. INTUSSUSCEPTION

A. Definition and diagnosis

1. A proximal segment of bowel telescopes into the more distal end. Peristaltic movements can extend the process for variable distances of bowel. In greater than 80% of cases, the starting point is at the ileocecum. Only 5% are precipitated by a pathologic "lead point" such as Meckel's diverticulum, inverted appendix, tumors, polyps, duplication, and lymphoma. Henoch-Schönlein purpuras are known to cause intussusception. The bunching up of the bowel causes pain, bowel obstruction, and if left untreated too long: bowel necrosis and perforation occur.
2. Usual age: 4-10 months, 65% < 1 yr, 80% < 2 yrs
4. 50% preceded by viral illness
5. Siblings of an intussusception sufferer are more likely to have issues
6. Children over 2 yrs old are more likely to have a lead point.
B. Clinical findings

1. A previously well child suddenly screams in pain, draws up his or her legs and may even roll around in distress. This resolves after 15-30 minutes and the child seems well again. The episode may recur approx. 15-30 minutes later. Eventually, the child is vomiting, and appears pale. *Late* in the course the child becomes dehydrated, may pass bloody or *"currant jelly stool"* due to mucosal necrosis and appears clearly ill.

2. The appearance of the child depends on what stage he or she is at: the child may appear completely well between attacks, or be in shock.

3. Pain is seen in 80%, vomiting in 60% and bloody stools in only 30%. It is interesting to note is that over 10% of children may be obtunded (so-called “neurologic presentation”), resembling meningitis, sepsis, or other causes of altered mental status.

4. On exam look for: *(Dance’s Sign)*
   a. RUQ sausage shaped mass which is the intussusception
   b. Empty space in RLQ (cecum which is now in the RUQ)

5. Rectal exam may show bloody stool, or a mass which is due to a very distal intussusception.

6. Later findings:
   a. Bowel obstruction
   b. Dehydration and electrolyte imbalance,
   c. Strangulated, ischemic bowel

7. Perforated bowel with peritonitis

8. If untreated, intussusception is usually fatal in 2-5 days.

C. Diagnosis

1. Abdominal X-ray may show evidence of mechanical bowel obstruction, adipose rose sign, and free air from perforation, RUQ mass.

2. CONTRAST ENEMA IS BOTH DIAGNOSTIC AND THERAPEUTIC.

3. A surgical team should be pre-notified before the enema in case of bowel perforation, or the enema is unsuccessful.

4. The enema will make the diagnosis by showing bowel obstruction and the characteristic sharp cut-off of the intussuscepted bowel.

5. When performing the BE: only one to three attempts at 5-10 minutes each are recommended. No abdominal palpation should be done. If unsuccessful, the patient should go to surgery.

6. Success rates with BE are usually greater than 80%.

7. Contraindications to barium enema include the following:
   a. Evidence of perforated bowel
   b. Peritonitis
c. Frank bowel obstruction
d. Shock
e. Symptoms longer than 48 hours (relative)
f. These patients should instead be operated on.

8. Recurrence rates with either BE or surgery are similar (5-18%). More than one recurrence is often associated with a pathological lead point.

9. NEWER Enema Modality: a newer procedure using air insufflation rather than barium appears to have a higher success rate and may become the treatment of choice.

VI. NECROTIZING ENTEROCOLITIS (NEC)

A. Definition and description

1. Most common GI emergency in neonates
2. Most common cause of intestinal perforation
3. Age of development of NEC:
   a. Most common in premature infants, however not immediately after born, most often occurs at 32-36 wks post-gestational life.
   b. 24-28 wks gestation age → occurs 2-4 wks of life
   c. 29-32 wks → 1-3 weeks of life
   d. 36-40 wks → 1st week of life
   e. Usually occurs in the NICU, but 10% of cases are full term (FT) infants
4. Multiple risk factors
   a. Prematurity is most common (90%)
   b. Hypoxic-ischemic insult
   c. Rapid advancement/over-feeding
   d. Infectious/immunologic

B. Clinical findings

1. Feeding intolerance
2. Vomiting - may be bilious, nonbilious, or coffee ground
3. Abdominal distention
4. Ill-appearing
5. Bell Classification:
   a. I: Early, suspected – symptomatic, but nl X-rays
   b. II: Definite NEC – pneumatosis present
   c. III: Advanced disease - perforation

C. Diagnosis

1. Plain abdominal X-rays:
   a. 75% of pts w/NEC have pneumatosis
   b. 10-30% have portal venous gas
c. Only 50-75% w/perforation has free air detectable on initial plain films
2. Ultrasound: air in bowel wall

D. Differential diagnosis

1. Malrotation with midgut volvulus
2. Hirschsprung’s disease with obstruction
3. Hernia with obstruction/perforation
4. Sepsis

E. Complications

1. Ileus
2. Shock
3. Sepsis
4. DIC
5. Chronic complications/sequelae (10-20%):
   a. Short gut syndrome
   b. Strictures w/obstruction
   c. Fistulae

F. Management

1. IV, O2, monitor, NG tube
2. NS 20cc/kg over 15min, repeated as needed
3. Dexi, CBC, T&S, BMP, PT/PTT, BCx
4. Cath U/A, UCx
5. Flat and LLat Decub Abd Xray
6. IV ABx (Amp, Gent, and Flagyl)
7. Notify Peds Surg
8. Notify PICU/NICU

VII. MECKEL’S DIVERTICULUM

A. Definition and description

1. A Meckel’s diverticulum results from incomplete regression of the vitelline duct connecting the primitive gut to the yolk sac.
2. Usually found on the antimesenteric side of the ileum 20-100 cm (2 feet) proximal to the ileocecal valve.
3. Length: 1-5 cm (2 cm)
4. Gastric mucosa is found in 50-80% of those that are symptomatic. Others can be lined with pancreatic or endometrial mucosa.
5. 2% of the population has them. Most are asymptomatic. Patients are classically boys under 5 years old.
B. Manifestations seen in symptomatic Meckel's diverticula

1. Ulceration and lower GI bleeding occurs in 30-40%. The ulcer is at the border between the gastric and normal mucosa. The bleeding is usually painless and can be profuse. A clue to the diagnosis is the color of the blood which is brick-red.
2. Diverticulitis occurs in 5-20%. The mechanism is believed to be similar to that in appendicitis. In fact, Meckel's diverticulitis presents like appendicitis and is frequently mistaken for appendicitis.
3. Bowel obstruction occurs in 30%. This is often due to intussusception with the diverticulum acting as a lead point. Adhesions secondary to an inflamed Meckel's can cause obstruction. Volvulus of the small bowel can also occur around persistent bands connecting the Meckel's to the umbilicus.
4. Umbilical problems: most noteworthy is when the vitelline duct remnants remain connected with the umbilicus resulting in enteric-umbilical fistulas.
5. Strangulation can occur due to vitelline duct remnants attach to the umbilicus and perforation of an ulcer or diverticulitis is not uncommon.

C. Diagnosis

1. Meckel's scan: Technicum 99 sodium pertechnetate is taken up by gastric mucosa. Since many symptomatic Meckel's have gastric mucosa, the diverticula shows up on the scan at the same time as the stomach. Approximately 50% of Meckel's can be diagnosed this way.
2. The sensitivity of the Meckel's scan can be increased by administration of cimetidine, which increases uptake and inhibits intraluminal release of contrast material. Glucagon decreases peristalsis, decreasing isotope washout.
3. Occasionally a Meckel's diverticula is seen on BE. The rest are discovered at laparotomy.

VIII. APPENDICITIS

A. Definition and description

1. It is thought that obstruction of the narrow appendiceal lumen leads to swelling, edema, inflammation, infection, gangrene, and eventual rupture and peritonitis. If left untreated, mortality is up to 50%. Current treatment results in 0.1-1% mortality. Appendicitis occurs in any age group but most often teenagers. The very young and very old often have an atypical clinical picture leading to misdiagnosis and increased morbidity and
mortality.
2. **Perforation** rates are high in children:
   a. Age less than 2 years: > 90%
   b. Age less than 6 years: 55-75%
   c. All children: 35%
3. In addition, once perforation occurs, the short omentum in children is less capable of walling off the infection. Diffuse peritonitis rapidly ensues.

B. Clinical findings

1. The typical picture begins with pain poorly localized to the umbilicus. This is due to stretch of the appendix stimulating visceral pain fibers at the level of T10.
2. This is often followed by anorexia (2/3), nausea, vomiting.
3. As appendiceal inflammation progresses, the pain often changes in character (somatic) and position (localized to the RLQ).
4. Low grade fever, sometimes low volume diarrhea.
5. Patients become quiet, even lethargic, as movement increases the pain.
6. The following may be seen on exam:
   a. Fever: usually low grade initially
   b. Point tenderness, guarding, rebound over the RLQ. The tenderness may be located elsewhere if the appendix is in an atypical position. Tenderness may be in the lateral abdomen, RUQ, LLQ, or suprapubic area.
   c. A retrocecal appendix can present as mass and tenderness in the rectum, tenesmus, and frequent mucoid stools.

C. Tests

1. Mildly elevated WBC count with left shift (10-15,000)
2. Urinalysis shows a few WBC and RBC in up to 25% of cases due to the inflamed appendix sitting near the bladder or ureter.
3. Plain abdominal X-rays may be helpful, but are normal in greater than 50% of the cases. They may show fecalith, appendiceal air, deformity of the R psoas shadow, free air from perforation, abnormal gas pattern or sentinel loop, ileus, splinting to the R, RLQ mass, RLQ abscess.
4. CXR may show free air from perforation or another diagnosis such as RLL pneumonia.

D. Diagnosis

1. In many institutions, the decision to operate is still based on the history and physical exam. Fever and elevated WBC count are the usual helpful tests.
2. Diagnostic imaging:
a. The most popular diagnostic adjuncts currently being used are CT scan (with oral and rectal contrast) and ultrasound. Both have their advantages and disadvantages.

b. CT is believed to be greater than 95% sensitive, but involves ionizing radiation.

c. Ultrasound has slightly lower sensitivity (85-95%), does not involve ionizing radiation, but has significant operative dependence. It is particularly useful for young thin patients.

E. Differential diagnosis

1. Gastroenteritis, UTI, intussusception, bowel obstruction, Meckel's, mesenteric adenitis, pneumonia, testicular torsion, PID, ectopic pregnancy, ovarian cyst or torsion, Sickle cell crisis.

F. Problem patients

1. Some patients will have an inflamed appendix in an unusual location leading to unusual physical findings.

2. In very young patients, pain is difficult to detect. Sometimes the infant is simply "not his usual self," won't eat, or is lethargic. Unless a careful assessment is done, the infant can progress sepsis without being able to localize the source. They are often treated as meningitis or sepsis.

3. This same age group is prone to very rapid progression to perforation and peritonitis.

4. Adolescent and adult females are problematic because of multiple other diagnoses with similar presentations such as ectopic pregnancy, PID, ovarian cysts and torsion.

G. Management

1. NPO, IV fluids, correct deficits, surgical consultation ASAP antibiotics if a perforation is suspected.

IX. GASTROESOPHAGEAL REFLUX

A. Definition and description

1. GE reflux is when stomach contents reflux into the esophagus. This is due to decrease of pressure in the lower esophagus or rising of abdominal pressure or both. Regurgitation of the acidic stomach contents is responsible for the following problems:

2. Aspiration with chronic cough or night cough, tracheobronchitis, pneumonia, wheezing, APNEA

3. Esophagitis with altered motility, chest pain, fussiness, colic, vomiting or regurgitation

4. Ulceration, bleeding, even esophageal strictures
5. Fe deficient anemia due to occult bleeding
6. Malnutrition, failure to thrive
7. Sandifer's syndrome: “spasmatic torticollis” – GER episodes associated with arching accompanied by choking, gagging, and occasional high-pitched, shrill cry. Commonly mistaken for an ALTE, however is differentiated because is not associated with cyanosis, apnea, change in consciousness or tone. Resolves with treatment of GE reflux.

B. Diagnosis can be difficult

1. Barium swallow: good to rule out other structural causes
2. Esophagoscopy: good for detecting esophagitis, strictures, ulcers
3. Rectal manometer
4. Esophageal pH probe monitoring

C. Treatment

1. Maintain upright position at 60 degrees for up to 24 hours/day.
2. Thicken feedings: 1 tablespoon cereal/1 oz formula
3. Increase frequency of feedings in decreased amounts H2 inhibitors recommended if severe and accompanied by weight los
4. Treatment is usually successful in 80-90 %
5. Surgery may be needed for refractory cases and those who are having serious complications like aspiration (Nissen fundoplication).

X. INCARCERATED INGUINAL HERNIA

A. Incarcerated hernias occur as a result of incomplete obliteration of the processus vaginalis

1. Normal fusion of the layers of the processus occur by age 2
2. Autopsy reports show that the processus remains patent in 80-90% of newborns
3. Incidence is 10-20 per 100 live births

B. Diagnosis

1. Common symptoms are vomiting and irritability
2. With a thorough exam, a firm tender inguinal mass is palpated

C. Treatment

1. Should try manual reduction, as this is often successful

XI. FOREIGN BODIES IN THE GI TRACT

A. Most swallowed FB will traverse the entire GI tract. The most
common areas for them to become stuck are:

1. Esophagus: cricoid (thoracic inlet), aortic arch, esophagogastric junction
2. Pylorus
3. Duodenum at the ligament of Treitz
4. Distal ileum
5. Ileocecal valve (the most common area of perforation)
6. Sigmoid colon
7. Rectum. (These are often placed there by the patient)

B. Esophagus

1. Symptoms: history of choking while eating, vomiting, gagging, increased salivation, drooling, neck, throat or chest pain, FB sensation, inability to swallow. There is no history given of FB ingestion in 25% of cases.
2. If the FB is impinging on the airway, children can develop respiratory difficulty, wheezing or even stridor may occur.
3. If presentation is late: Patients may have wheezing, chronic cough, recurrent pulmonary infections, weight loss, malnutrition, hematemesis, or melena.
4. Complications: perforation, mediastinitis, abscess, pneumothorax, exsanguinating hemorrhage from erosion into aorta, airway compromise, or dislodging of the FB from esophagus to trachea.
5. Diagnosis: direct visualization, indirect or direct laryngoscopy, CXR (that includes pharynx to upper abdomen), barium swallow (radiolucent objects).
6. Esophageal coins are aligned in the frontal plane and seen best on AP CXR, tracheal coins are aligned in the sagittal plane.
7. If still not located, Barium swallow or cotton ball swallow may help locate the FB.

C. Endoscopy is performed if FB is still suspected.

D. Management of esophageal foreign bodies

1. ABCs
   a. Remove that FB which you can see directly; grasp firmly and pull out without injury to patient or dropping FB into airway.
2. Don’t try to wash the FB down with fluids – patients should remain NPO.
3. Food FBs (such as meat) are uncommon in children. Papain is not recommended in children. Associated with esophageal perforation in 3% of cases.
4. Glucagon has been used to relax the esophagus in adults, but is generally not recommended in children.
E. **Endoscopy** with conscious sedation or general anesthesia is considered by most to be the *best method* of removal. The esophagus should be inspected for perforation after the FB is removed.

F. Two other less common techniques

1. A Foley catheter technique has been advocated by some but the FB is not under control and could be lost into the airway. It involves passage of Foley catheter past the FB, filling the balloon, and then pulling the FB retrograde out (into the pharynx, mouth where it is then recovered.)
2. Bougiennage, in which a thick red rubber dilating catheter is inserted into the esophagus and the FB is “pushed” into the stomach.

G. Other GI FB

1. Objects which have passed the pylorus usually traverse the entire GI tract without problems. If the object is very small and smooth without sharp edges or points, the patient can be followed as an outpatient. Parents should NOT be instructed to strain stools looking for the FB (unless it is extremely valuable). Rather, they should be instructed to return immediately if problems occur such as pain, GI bleeding, vomiting, or abdominal distension.
2. Any object that may not pass warrants admission. Examples of objects which do not always pass are sharp or pointed: pins, needles, open safety pins, tacks, jacks, glass, toys, and objects **more than 2 cm in diameter or 5 cm long**. Certain objects such as knives, etc., may warrant surgical removal without a period of observation.
3. Many remedies have been tried but without success and can be hazardous: laxatives, cathartics, and papain.

H. Patients can be followed up with an X-ray in 1-2 weeks to document passage. Nothing further is needed unless symptoms develop.

**XII. BUTTON BATTERY INGESTION**

A. **If lodged in the esophagus, they should be immediately removed, as esophageal perforation may occur as early as 8 hours.** This is probably due to electrical current generated by the battery. Patients who have passed battery into the duodenum may be observed in the same fashion as any other FB. Indications for surgery are the same as in any other ingestion.

B. There has been concern about mercury poisoning from dissolution of
the battery case and GI absorption of mercury. Severe mercury poisoning has not been observed in clinical practice. However, some recommend getting mercury levels in patients who have retained decomposed button battery.

C. Cathartics and enemas are sometimes used to hasten evacuation of a battery which has disassembled to guard against this theoretical concern.

XIII. GASTROINTESTINAL BLEEDING IN PEDIATRIC PATIENTS

A. The presentation of GI bleeding in children is similar to that in adults. Hematemesis, coffee-ground emesis, hematochezia, melena carry the same significance.

B. Children who have had massive blood loss are a problem because they may not show the usual signs of hemodynamic compromise until very late. The blood pressure and pulse may remain normal until the patient abruptly decompensates. Skin color, mental status, and urine output are better indicators of hemodynamic stability.

C. Additionally, the hematocrit can remain normal for hours after hemorrhage until equilibrium between the vascular and intravascular space has occurred. Hematocrit is NOT a good indicator for the degree of blood loss.

D. Some patients with significant upper GI bleeding have no blood on NG aspiration.

E. Patients can bleed so briskly from upper GI sources that they pass frank blood per rectum. This may be mistaken for lower GI bleeding and mismanaged unless an NG tube is placed in all significant LOWER GI bleeding.

F. Patients may be bleeding so briskly that they decompensate before the blood has had time to present itself at the oral or rectal orifices.

G. GI bleeding can be divided into the following general categories:

1. **Mucosal irritation or abnormality**: i.e.: Meckel's, polyp, gastritis, ulcers, colitis, anal fissure, necrotizing enterocolitis (NEC), vascular malformations, Mallory-Weiss tear, foreign body.
2. **Ischemic bowel**: This is usually associated with bowel obstruction. i.e.: intussusception, midgut volvulus, gastric volvulus, strangulated hernia.
3. **Bleeding disorder**: hemorrhagic disease of the newborn: vitamin K deficiency liver failure thrombocytopenia: hypersplenism, etc., anticoagulant meds or accidental ingestion
XIV. CAUSES OF GI BLEEDING BY AGE GROUP

A. Neonatal
   1. Swallowed maternal blood
   2. Hemorrhagic disease
   3. Bleeding diathesis
   4. Necrotizing enterocolitis
   5. Esophagitis
   6. Gastritis
   7. Stress ulcer
   8. Anal fissure
   9. Midgut or gastric volvulus
  10. Vascular malformations

B. Infant, < 2 years old
   1. Intussusception
   2. Anal fissure
   3. Swallowed blood from ENT source
   4. Milk allergy
   5. Mallory-Weiss tear
   6. Esophagitis
   7. Gastritis
   8. Ulcer disease
   9. Colitis
  10. Meckel's diverticulum
  11. Pyloric stenosis
  12. Malformations
  13. Foreign body

C. Pre-school, 2-5 years old
   1. Colitis
   2. Stress ulcer
   3. Gastritis
   4. MW tear
   5. Juvenile polyps
   6. Anal fissure
   7. Varices
   8. Meckel's
   9. Vasculitis: Henoch-Schönlein purpura (HSP)
  10. Inflammatory bowel disease (IBD)
  11. Thrombocytopenia
  12. Foreign body
D. School age, > 5 years old

1. Colitis
2. PUD
3. Mallory Weiss tear (MW tear)
4. Varices
5. Inflammatory Bowel Disease (IBD)
6. Polyps
7. Gastritis
8. Hemorrhoids
9. Vasculitis
10. Thrombocytopenia

XV. APT-DOWNEY TEST

A. Neonates often present with "GI bleeding" secondary to swallowed maternal blood.

B. The Apt-Downey test can rapidly distinguish between neonatal and maternal blood.

C. 1 cc of the bloody stool or vomitus is mixed with 5 cc of H2O.

D. This is centrifuged and 5 ml of the pink hemolysate is mixed with 1 cc of NaOH.

E. After 2 minutes: if the fluid remains pink, it is fetal blood. If the fluid turns brown-yellow, it is maternal blood.

XVI. MANAGEMENT OF GI BLEEDING IN CHILDREN

A. Emergency management is similar to that in adults. The same labs are obtained and the same history and physical exam is done.

B. Resuscitate

1. Airway: intubate if needed
2. Breathing: all patients get oxygen, ventilate if needed
3. Circulation: Children in hemorrhagic shock should first be resuscitated with Ringer's lactate or normal saline with IV boluses at 20cc/Kg, repeated as needed. If necessary, further resuscitation is with PRBC transfusion at 10cc/Kg.

C. It is extremely important that blood and fluids be warmed to prevent infants from developing hypothermia.

D. All significantly bleeding patients should have an NG tube placed.
In lower GI bleeding an unsuspected upper GI source may be discovered.

E. Management: often depends on the particular clinical manifestation.

1. GI bleeding can be profuse but usually spontaneously resolves. Patients are managed similarly to any major GI bleed: Airway protection as needed, oxygen, warmed fluids, blood transfusion, and H2 blockers such as cimetidine.

2. Diverticulitis will nearly always be discovered at surgery for suspected appendicitis. If the diagnosis is correctly made pre-operatively, surgery is still the treatment of choice.

3. Bowel obstruction is also managed similarly to any other bowel obstruction: IV fluids, NG tube, electrolyte correction as needed, monitoring of hydration status and urine output. These children also need surgery.

4. Strangulation and perforation are managed similarly to bowel obstruction with the sole exception of earlier emergency surgery.

XVII. NOTABLE NON-GI CAUSES OF ABDOMINAL SYMPTOMS IN PEDIATRIC PATIENTS

A. Child abuse

B. Pharyngitis

C. Pneumonia

D. Urinary tract infection

E. Renal stone

F. CNS mass or infection

G. Cervicitis, PID

H. Ectopic pregnancy

I. Ovarian cyst or torsion

J. Testicular torsion

K. Epididymitis, urethritis, prostatitis

L. Diabetic ketoacidosis
M. Thyroid disease

N. Sickle cell crisis

O. Porphyria

P. Accidental poisoning
PEARLS

1. Hypertrophic pyloric stenosis - most common cause of infantile GI obstruction after the first month of life, presenting with nonbilious emesis that progressively becomes projectile. Classic electrolyte abnormality is hypochloremic, hypokalemic metabolic alkalosis.

2. Malrotation with midgut volvulus any bilious emesis is suspicious for this (seen in >75%) with classic presentation of sudden onset bilious emesis and abdominal distension, toxic appearing; procedure of choice is upper GI showing abnormal position of duodenal C-loop and small bowel with corkscrew appearance.

3. Necrotizing enterocolitis is the most common GI emergency in neonates, occurs more commonly in premies but 10% occur in full term infants. On plain film pneumatosis intestinalis (air in bowel wall) is pathognomonic for NEC and seen in 75% of cases.

4. Intussusception is the most common cause of intestinal obstruction in patients < 2 y.o., classic triad is abdominal pain, vomiting, bloody stools (currant jelly); may also present with profound lethargy.

5. Hirschsprung’s Disease usually presents with failure to pass meconium and may be complicated by enterocolitis (characterized by abdominal distension, bloody stools, fever and leukocytosis) and toxic megacolon.

6. Meckel’s Diverticulum follows the rule of 2s: It occurs in 2% of population, and only 2% of affected patients ever become symptomatic. Half of all patients become symptomatic by age 2; most present by age 20. It is usually located within 2 feet of ileocecal valve. Presents with massive, painless rectal bleeding (brick red) in males < 5y.o.

7. Henoch-Schönlein purpura most commonly occurs in spring after URI, presents with abdominal pain, nausea, vomiting, arthralgias, microscopic hematuria classic rash is palpable purpura on buttocks and lower legs; may also present with intussusception.

8. Foreign bodies 80-90% that make it into the stomach will pass, button batteries in esophagus must be rapidly removed to prevent erosions and mediastinitis, button batteries in the stomach must be followed with films to document passage beyond pylorus. Plain films of suspected coin ingestions will demonstrate the face of the coin in esophagus the edge of the coin in the trachea.
9. **Appendicitis** is the most common non-traumatic surgical emergency in peds; progressive symptoms 4-24 hours - abd pain, vomiting, fever, anorexia. Limited CT of appendix has 95-100% sensitivity/specificity -- has reduced negative lap rate from 20% to 7%.
REFERENCES


