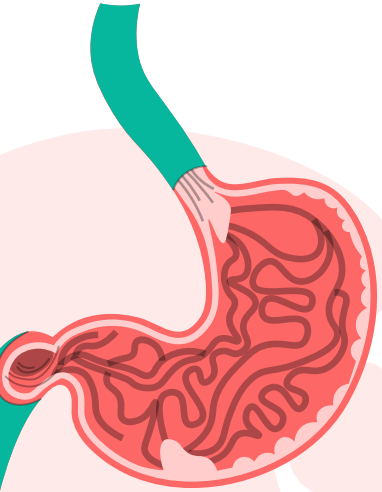
Upper Gastrointestinal Pathologies



Done By:
Mohammad Mousa
Bashar Alhardan
Haya Hindiyeh
Hala Hindiyeh
Rafi Kakish

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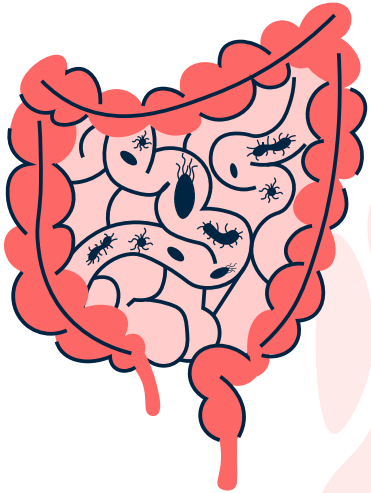

CLINICAL PRESENTATION

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b. Vomiting
c. Diarrhea
d. Constipation
e. Failure to Thrive
f. GI Bleeding

ESOPHAGUS

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c. Achalasia
d. Eosinophilic Esophagitis

STOMACH

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b. Gastropathy
c. Peptic Ulcer Disease

1) Abdominal Pain

- There are multiple causes of abdominal pain, some might be related to injury of any intraabdominal organs or overlying structures within the abdominal wall, other causes include various extraabdominal diseases.

- Patients present with different types of abdominal pain, they include:

1. **Visceral pain**: results when autonomic nerves within the gut detect injury, transmitting sensation by nonmyelinated fibers. The pain is vague, dull, slow in onset, and poorly localized.

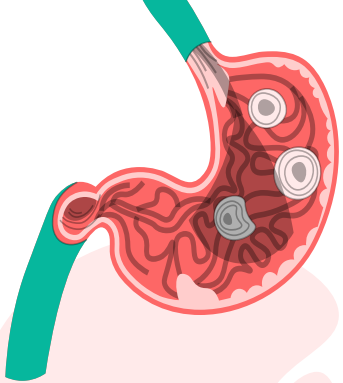
2. **Somatic pain:** results when overlying body structures are injured. Somatic structures include the parietal peritoneum, fascia, muscles, and skin of the abdominal wall. In contrast to visceral pain, sensation here is transmitted through somatic nociceptive fibers than are myelinated.

3. **Referred pain:** is a painful sensation in a body region distant from the true source of pain. The location of referred pain is predictable based on the locus of visceral injury.

Abdominal Pain

- Abdominal pain can also be divided into acute abdominal pain and recurrent abdominal pain.
- **Acute abdominal pain** can be an indicator of a serious intraabdominal process, such as appendicitis or bowel obstruction. It may also arise from extraintestinal sources, such as lower lobe pneumonia or urinary tract stone.
- Not every case of acute abdominal pain is considered as an emergency. Volvulus and appendicitis, for example, must be ruled out as they require immediate surgical intervention. The majority of patients who experience acute abdominal pain do not have a condition that requires surgical intervention. However, it is important to distinguish them from cases that can be addressed through conservative management.

Acute Abdominal Pain

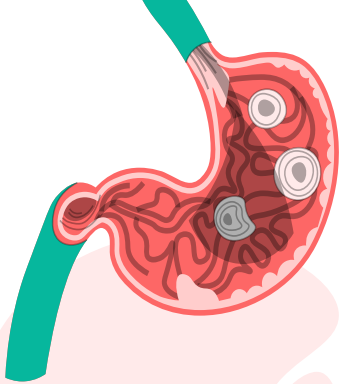


- A proper approach to acute abdominal pain should include the following:

1. **History:**

- Onset: Sudden or gradual, prior episodes, association with meals, history of injury.
- Nature: Sharp versus dull, colicky or constant, burning.
- Location: Epigastric, periumbilical, generalized, right or left lower quadrant, change in location over time.
- Associated symptoms: Fever, vomiting (bilious?), diarrhea (bloody?), abdominal distention.
- Extraintestinal symptoms: Cough, dyspnea, dysuria, urinary frequency, flank pain.
- Course of symptoms: Worsening or improving, change in nature or location of pain, aggravating and alleviating factors, last menstrual period.

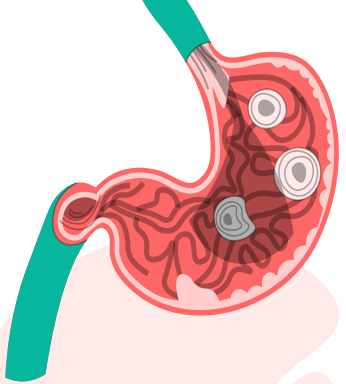
Acute Abdominal Pain



2. Physical examination:

- General position: Growth and nutrition, general appearance, hydration, degree of discomfort, body position
- Abdominal: Tenderness, distention, bowel sounds, rigidity, guarding, mass
- Genitalia: Testicular torsion, hernia, pelvic inflammatory disease, ectopic pregnancy
- Surrounding structures: Breath sounds, rales, rhonchi, wheezing, flank tenderness, tenderness of abdominal wall structures, ribs, costochondral joints
- Rectal examination: Perianal lesions, stricture, tenderness, fecal impaction, blood

Acute Abdominal Pain



Multiple investigations can be done to rule out causes of acute abdominal pain, they include the following:

Laboratory	Radiology
CBC, C-reactive protein, ESR	Plain flat and upright abdominal films
AST, ALT, GGT, bilirubin, Amylase, lipase	CT scan
Urinalysis	Barium enema
Pregnancy test (older females)	Ultrasound
	Endoscopy
	Upper Endoscopy

Differential Diagnosis of Abdominal Pain

Functional	Ruptured spleen, Perforated viscus, Traumatic pancreatitis
Functional	Constipation, Irritable bowel syndrome, Dysmenorrhea
Infectious	Appendicitis, Gastroentiritis, UTI, Cholecystitis, Pneumonia, Hepatitis
Genital	Testicular torsion, Ovarian torsion, Ruptured Ovarian cyst, Ectopic pregnancy
Genetic	Sickle cell crisis, FMF
Metabolic	Diabetic ketoacidosis
Inflammatory	IBD, Vasculitis, HSP, Pancreatitis
Obstructive	Intussuseption, Ileus, Malrotation with volvulus
Biliary	Gallstone
Peptic	Peptic ulcer disease, Gastritis, Esophagitis
Renal	Kidney stones, Hydronephrosis

Recurrent Abdominal Pain

- The majority of children rarely have a significant or identifiable underlying illness contributing to their pain.
- Differential diagnosis of recurrent abdominal pain include:

1. Functional abdominal pain
2. Irritable bowel syndrome
3. Chronic pancreatitis
4. Gallstones
5. Peptic ulcer
6. Celiac disease
7. Inflammatory bowel disease
8. Constipation

Recurrent Abdominal Pain

- Functional abdominal pain is a type of recurrent abdominal pain, children experiencing it have pain almost daily. The pain is not related to meals nor is relieved by defecation, it is usually worst in the morning.
- IBS falls under the category of functional abdominal pain, distinguishing features of it include pain at the time of a change in stool frequency/ consistency, fluctuating pattern of diarrhea and constipation with pain alleviated upon defecation.

Diagnosis	Rome III criteria*
Functional abdominal pain	■ Episodic or continuous abdominal pain ■ Insufficient criteria for other functional gastrointestinal disorders
Functional abdominal pain syndrome	■ Functional abdominal pain for at least 25% of the time and one or more of the following: 1. Some loss of daily functioning 2. Additional somatic symptoms such as headache, limb pain, or difficulty sleeping
Functional dyspepsia	■ Persistent or recurrent pain or discomfort centred in the upper abdomen ■ Not relieved by defecation or associated with a change in stool frequency or form
Irritable bowel syndrome	■ Abdominal discomfort or pain associated with two or more of the following at least 25% of the time: 1. Improved with defecation 2. Onset associated with a change in frequency of stool 3. Onset associated with a change in form of stool
Abdominal migraine	■ Paroxysmal episodes of intense, acute peri-umbilical pain that last for one or more

Warning Signs of Underlying Illness in Recurrent Abdominal Pain

- If any of the following warning signs are present, further evaluation should be done:

- Vomiting
- Abnormal screening laboratory study (anemia, hypoalbuminemia, inflammatory markers)
- Fever
- Bilious emesis
- Growth failure
- Pain awakening child from sleep
- Weight loss Location away from periumbilical region
- Blood in stools or emesis
- Delayed puberty
- Nocturnal diarrhea
- Dysphagia Arthritis
- Perianal disease
- Family history of inflammatory bowel disease, celiac disease, or peptic ulcer disease

Warning Signs of Underlying Illness in Recurrent Abdominal Pain

Initial evaluation	Follow-up evaluation
• Complete history and physical examination Ask about “warning signs” (see Table 126.6) Determine degree of functional impairment (e.g., missing school) • CBC • ESR • Amylase, lipase • Urinalysis • Abdominal ultrasound—examine liver, bile ducts, gallbladder, pancreas, kidneys, ureters Trial of 3-day lactose-free diet	• CT scan of the abdomen and pelvis with oral, rectal, and intravenous contrast • Celiac disease serology—endomysial antibody or tissue transglutaminase antibody • Barium upper GI series with small bowel followthrough • Endoscopy of the esophagus, stomach, and duodenum • Colonoscopy

Treatment of Recurrent Abdominal Pain

- Recurrent abdominal pain usually limits the functioning of the child, this leads to anxiety and elongation of the course of pain. One line of management is assisting the child in returning to normal activities, this is done by allowing the child to take a short break from whatever they are doing until the symptoms are relieved.

- Fiber supplements, probiotics and peppermint oil can be of aid in managing the symptoms of IBS.

- In persistent cases, cognitive behavioural therapy, amitriptyline or SSRIs may be helpful.

2) Vomiting

- Vomiting is a coordinated, sequential series of events that leads to forceful expulsion of gastric contents through the mouth. It should be differentiated from regurgitation, referred to as gastroesophageal reflux.

- While vomiting and regurgitation have a similar outcome, they have distinct characteristics. Vomiting typically occurs after experiencing nausea and involves forceful gagging and retching. In contrast, regurgitation is effortless and not preceded by nausea.

Differential Diagnosis of Vomiting

Differential diagnosis	Historical clues
Viral gastroenteritis	Fever, diarrhea, sudden onset, absence of pain
Gastroesophageal reflux	Effortless, not preceded by nausea, chronic
Hepatitis	Jaundice, history of exposure
Otitis media	Fever, ear pain
Urinary tract infection	Dysuria, unusual urine odor, frequency, incontinence
Pneumonia	Cough, fever, chest discomfort
Milk or soy protein intolerance	Associated with particular formula or food, blood in stools
Peptic ulcer or gastritis	Epigastric pain, blood or coffee-ground material in emesis, pain relieved by acid blockade
Appendicitis	Fever, abdominal pain migrating to the right lower quadrant, tenderness
Pyloric stenosis	Nonbilious vomiting, postprandial, hunger, progressive weight loss
Cyclic vomiting syndrome	Similar to migraine, usually no headache
Meningitis	Fever, stiff neck

Vomiting

- Physical examination must include assessing the child's hydration status (Capillary refill, moistness of mucous membranes, skin turgor). The chest should be auscultated to look for signs of pulmonary involvement. Distention, organomegaly, bowel sounds, tenderness and guarding should all be checked in thorough abdominal examination. Rectal examination and testing stool for occult blood may be conducted.
- Investigations must include serum electrolytes, kidney function test, complete blood count, amylase, lipase and liver function test. In situations where the history and physical examination indicate a particular cause, additional testing should be done. Additional testing include ultrasound (rule out gallstones, renal stones, hydronephrosis,...), CT scan to rule out appendicitis or structures not visible by ultrasound, Barium studies to locate obstructive lesions along the gut.
- Dehydration should first be treated with fluid resuscitation, either using oral fluid-electrolyte solutions or IV fluids, the choice of fluids should be based on the type of electrolyte imbalance.
- Underlying causes should be treated when possible.
- Antiemitic medications should only be used when the cause of vomiting is determined and only for severe symptoms.

3) Diarrhea

- Acute diarrhea is a major problem when it occurs with malnutrition or in the absence of basic medical care.

- The majority of cases of acute diarrhea are viral in nature and resolve on their own without the need for diagnostic testing or specific treatment. Bacterial infections, on the other hand, often result in more severe illness and are commonly associated with food-related outbreaks or areas with inadequate public sanitation. Suspecting bacterial enteritis is appropriate when dysentery (bloody diarrhea with fever) is present or when the symptoms are severe.

- Chronic diarrhea, lasting beyond a period of 2 weeks, has a wide range of potential causes, ranging from serious to benign conditions that are challenging to diagnose.

- Diarrhea may be described as loose or watery stool, excessively frequent stools, or stools that are large in volume. A more exact definition of diarrhea is excessive daily stool liquid volume (>10 mL stool/kg body weight/day).

Classification of Diarrhea

- Diarrhea can be categorized based on etiology or physiological mechanisms (osmotic or secretory).

- Secretory diarrhea occurs when the intestinal mucosa directly secretes fluid and electrolytes into the stool. This type of diarrhea is often associated with inflammation, such as in inflammatory bowel disease (IBD), or exposure to certain chemicals. Inflammation mediators and hormones, such as vasoactive intestinal peptide produced by neuroendocrine tumors, can also stimulate secretion. Cholera is an example of secretory diarrhea caused by the enterotoxin of Vibrio cholerae, which triggers increased levels of cyclic adenosine monophosphate (cAMP) within enterocytes, leading to excessive secretion into the small intestine.

- Osmotic diarrhea occurs after malabsorption of an ingested substance, such as lactose in lactose intolerance or nonabsorbable laxatives (polyethylene glycol). Absorption of these substances draws water into the intestinal lumen.

- Diarrhea can also be classified into acute and chronic. Common causes of acute diarrhea include gastroenteritis, food poisoning and systemic infections. Common causes of chronic diarrhea include Cow's milk intolerance, toddler's diarrhea, IBS, IBD and celiac disease.

Management of Diarrhea

- The first step in the management of diarrhea is determining whether it's osmotic or secretory, this is done by calculating the osmotic gap. Secretory diarrhea has an osmotic gap of less than 50, a number higher than 50 indicates osmotic diarrhea.

- History should include asking about the onset, number and character of stools, volume of stools and whether the diarrhea is associated with other symptoms such as blood in the stool, fever and weight loss. Recent trave, dietary factors and medications taken recently should all be inquired about.

- It is important to do thorough physical examination by checking for signs of abdominal distention, tenderness, quality of bowel sounds, presence of blood in the stool. Rectal examination could show a large fecal mass, anal tone sphincter must be checked.

- Stool culture and complete blood count are done if bacterial enteritis is suspected. Additional tests are done when certain diagnoses are suspected, C. difficle toxin is ordered if diarrhea didn't resolve after a course of antibiotics, fecal fat content if stools are oily to rule out pancreatic insufficiency, ...

4) Constipation

- Pediatric constipation refers to the condition where children experience difficulty or infrequent bowel movements. It is a common gastrointestinal problem in childhood that can cause discomfort and distress.

- Constipation also defined as two or fewer stools per week or passage of hard, pellet-like stools for at least 2 weeks.

Constipation

- Functional constipation, a frequently observed form of constipation, is characterized by infrequent bowel movements, typically less than two per week. Individuals experiencing functional constipation often intentionally hold back stool and experience sporadic passage of large-sized stools that are often uncomfortable or painful. In children, this condition is often accompanied by a behavior known as "retentive posturing," where they stand or sit with their legs extended and stiff or crossed. Additionally, children with functional fecal retention may also have fecal incontinence due to leakage of retained stool, a condition referred to as encopresis.

Constipation

Causes:

- Several factors contribute to pediatric constipation, including:
- Diet: Low fiber intake, inadequate fluid intake, and excessive consumption of processed foods.
- Toilet training: Stressful or delayed toilet training can lead to withholding of stool.
- Medical conditions: Certain medical conditions, such as hypothyroidism or Hirschsprung's disease, can cause constipation.
- Emotional factors: Emotional stress, anxiety, or fear of using unfamiliar bathrooms can contribute to constipation.

Constipation

Symptoms of constipation may vary but commonly include:

- Infrequent bowel movements (less than three times per week).
- Hard, dry, and painful stools.
- Abdominal pain or discomfort.
- Straining during bowel movements.
- Fecal incontinence.
- Loss of appetite.

Differential Diagnosis of Constipation

1. **Hirschsprung disease:**

Failure to pass stool within the first 24 hours, abdominal distention, vomiting, symptoms of enterocolitis (fever, foul-smelling diarrhea, megacolon).

1. Examination: Snug anal sphincter, empty, contracted rectum. May exhibit explosive release of stool when the examiner's finger is withdrawn.
2. Laboratory: Absence of ganglion cells on rectal suction biopsy specimen, absent relaxation of the internal sphincter, "transition zone" from narrow distal bowel to dilated proximal bowel on barium enema.

2. **Functional constipation:**

1. History: No significant neonatal constipation, onset during potty training, passage of large-caliber stools, retentive posturing. May have encopresis (fecal soiling).
2. Examination: Normal or reduced sphincter tone, dilated rectal vault, presence of fecal impaction, soiled underwear, palpable fecal mass in the left lower quadrant.
3. Laboratory: No abnormalities. Barium enema would show a dilated distal bowel.

Differential Diagnosis of Constipation

3. Anorectal and colonic malformations:

Anal stenosis, anteriorly displaced anus, imperforate anus, colonic stricture. Constipation present from birth due to abnormal anatomy.

1. Examination: Anorectal abnormalities easily identified on physical examination. Anteriorly displaced anus mostly seen in females, with a normal-appearing anus located close to the posterior fourchette of the vagina.
2. Laboratory: Barium enema can reveal the anomaly.

4. Multisystem disease:

Muscular dystrophy, cystic fibrosis, diabetes mellitus, developmental delay, celiac disease.

1. Examination: Specific abnormalities related to the underlying diagnosis may be present.
2. Laboratory: Tests directed at the suspected disorder confirm the diagnosis.

Differential Diagnosis of Constipation

5. Spinal cord abnormalities:

Meningomyelocele, tethered cord, sacral teratoma or lipoma.History of swelling or exposed neural tissue in the lower back, urinary incontinence.

1. Examination: Lax sphincter tone due to impaired innervation, visible or palpable abnormality of the lower back (usually) present.
2. Laboratory: Plain x-ray often shows bony abnormalities. Magnetic resonance imaging (MRI) of the spinal cord reveals characteristic abnormalities.

6. Drugs:

Narcotics, psychotropics.
Recent use of drugs known to cause constipation (lead poisoning, opiates).
Examination: Features suggestive of functional constipation.

5) Gastrointestinal Bleeding

- Gastrointestinal bleeding in pediatrics refers to the presence of blood in the gastrointestinal tract in children. It is characterized by the passage of blood through the digestive system, which can manifest as hematemesis (vomiting of blood), melena (black, tarry stools), or hematochezia (bright red blood in stools).

- Gastrointestinal bleeding in children can be caused by various underlying conditions such as peptic ulcers, esophagitis, gastritis, vascular anomalies, inflammatory bowel disease, intestinal polyps, and coagulation disorders.

- Evaluation of bleeding should include confirmation that blood truly is present, estimation of the amount of bleeding, stabilization of the patient's intravascular blood volume, localization of the source of bleeding, and appropriate treatment of the underlying cause.

- When bleeding is massive, it is crucial that the patient receive adequate resuscitation with fluid and blood products before moving ahead with diagnostic testing.

Causes of Gastrointestinal Bleeding According to Age :

1. Newborn:

a) Ingested maternal blood:
Characterized by hematemesis (vomiting blood) or rectal bleeding.
Cracked maternal nipples may be a contributing factor.

b) Peptic disease:
Hematemesis with the amount of blood varying.
Blood found in the stomach on lavage.

c) Coagulopathy:
Hematemesis or rectal bleeding.
Bruising may be present in other sites.
Associated with a history of home birth and lack of vitamin K administration.

d) Allergic colitis:
Streaks of bloody mucus in the stool.
Presence of eosinophils in feces and rectal mucosa.

e) Necrotizing enterocolitis:
Rectal bleeding.
Sick infant with a tender and distended abdomen.

f) Volvulus:
Hematemesis and hematochezia (bright red blood in the stool).
Acute presentation with a tender and distended abdomen.

Causes of Gastrointestinal Bleeding According to Age :

2. Infancy to 2 Years Old:

a) Peptic disease:
Usually presents with hematemesis, but rectal bleeding is also possible.
Epigastric pain and coffee-ground emesis may be observed.

b) Esophageal varices:
Hematemesis.
Associated with a history or evidence of liver disease.

c) Intussusception:
Rectal bleeding.
Presents with crampy pain, distention, and a palpable mass.

d) Meckel diverticulum:
Rectal bleeding that is massive and bright red, usually without pain.

e) Bacterial enteritis:
Rectal bleeding with bloody diarrhea and fever.

Causes of Gastrointestinal Bleeding According to Age :

3. Older than 2 years :

a) **Inflammatory bowel disease:**
Usually presents with rectal bleeding.
Symptoms include crampy abdominal pain, poor weight gain, and diarrhea.

b) **Juvenile polyp:**
Rectal bleeding, painless, and bright red blood in the stool (not massive).

c) **Meckel diverticulum:**
Similar characteristics as mentioned earlier.

d) **Nodular lymphoid hyperplasia:**
Rectal bleeding with streaks of blood in the stool and no other significant symptoms.

e) **Mallory-Weiss syndrome:**
Hematemesis with bright red or coffee-ground appearance, typically follows retching.

f) **Hemolytic uremic syndrome:**
Rectal bleeding along with thrombocytopenia, anemia, and uremia.

g) **Hemorrhoids:**
Rectal bleeding with dilated external veins and blood noticed during wiping.

Gastrointestinal Bleeding

Diagnosis

- The location and severity of the bleeding can be determined through a thorough review of the patient's medical history and physical examination. It is necessary to ask the parents to provide specific information regarding the amount of bleeding, and to inquire about any related symptoms. Evaluating vital signs, including checking for changes in blood pressure, assessing pulses, capillary refill, and examining the color of the mucous membranes for signs of paleness can offer valuable insights. Laboratory tests and imaging studies should be conducted as needed based on the clinical situation.

Gastrointestinal Bleeding

Laboratory Investigation:

- CBC (Complete Blood Count) and platelet count.
- Coagulation tests: Prothrombin time (PT) and partial thromboplastin time (PTT).
- Liver function tests: AST (Aspartate Aminotransferase), ALT (Alanine Aminotransferase), GGT (Gamma-Glutamyl Transferase), and bilirubin levels.
- Occult blood test of stool or vomitus to detect hidden blood.
- Stool culture to identify bacterial pathogens

Gastrointestinal Bleeding

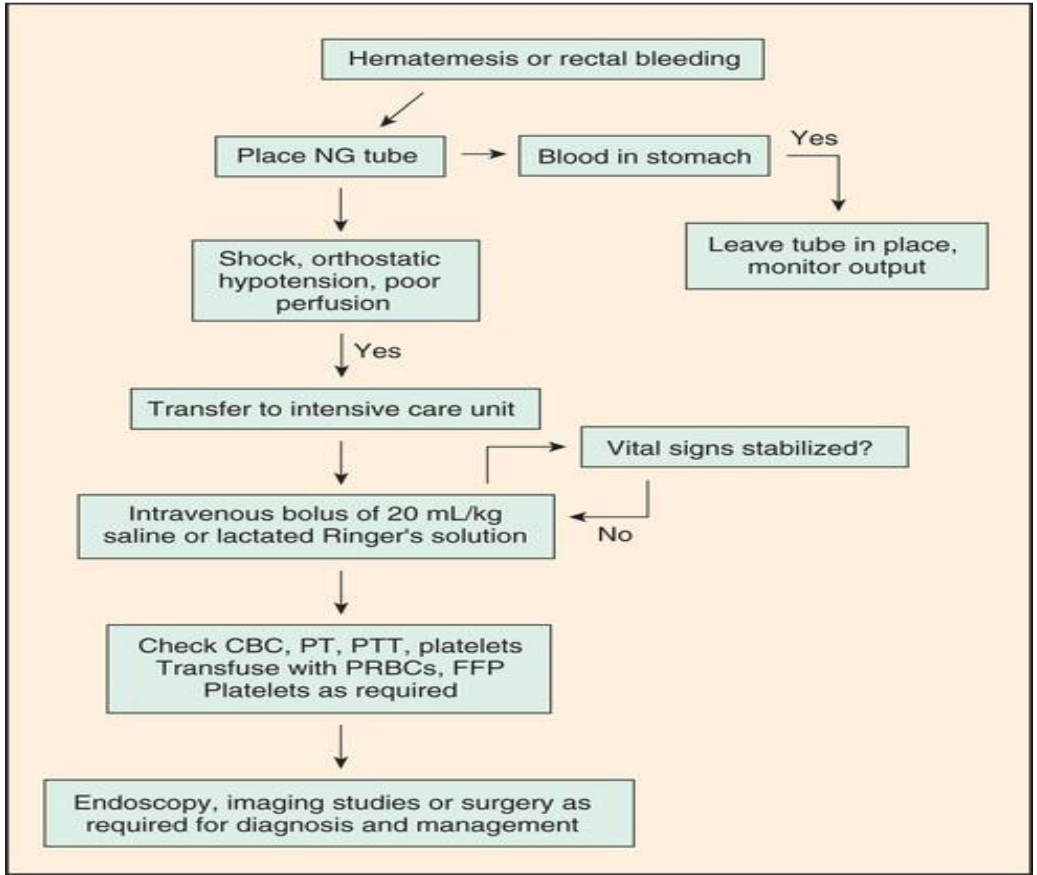
Radiologic Evaluation:

- Abdominal x-ray series to assess for any obvious abnormalities.
- CT (Computed Tomography) scan with contrast to provide detailed images of the gastrointestinal tract.
- Endoscopy.

Management of Gastrointestinal Bleeding

- The management of gastrointestinal bleeding should commence with an initial evaluation, prompt stabilization, and a systematic series of diagnostic examinations. Once an identifiable and treatable cause is identified, targeted treatment should be initiated. In situations where the bleeding is minimal and does not warrant resuscitation, no immediate intervention may be necessary. However, for pediatric patients experiencing significant bleeding, it is crucial to prioritize the ABCs of resuscitation (airway, breathing, circulation) as the primary focus.

Gastrointestinal Bleeding



6) Failure to thrive

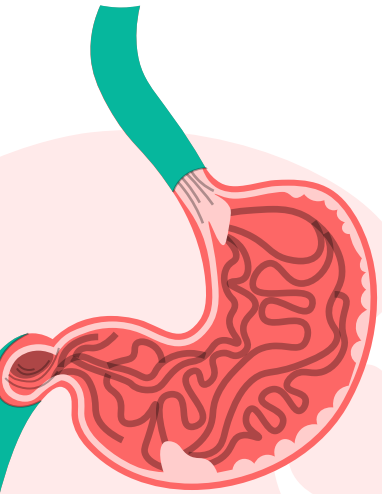
- Failure to thrive (FTT) is a descriptive term given to infants and young children with malnourishment resulting in inadequate growth.
- A condition in which a child's growth and development significantly deviate from the expected patterns.
- It typically manifests as a deceleration or lack of weight gain, height growth, or both, in comparison to other children of the same age and sex.
- Commonly used criteria include weight below the 3rd or 5th percentile for age; weight decreasing, crossing two major percentile lines on the growth chart over time; or weight less than 80% of the median weight for the height of the child.

Failure to thrive

Category	Causes
Environmental (Common)	Emotional deprivation
	Child maltreatment
	Maternal depression
	Poverty
	Improper formula preparation
Gastrointestinal	• Cystic fibrosis and other causes of pancreatic insufficiency. • Celiac disease. • Gastrointestinal reflux.
Congenital	• Chromosomal abnormalities and genetic syndromes. • Congenital heart disease. • Congenital immunodeficiency syndromes. • Gastrointestinal abnormalities (e.g., pyloric stenosis, malrotation).

Failure to thrive

Category	Causes
Metabolic	• Thyroid disease. • Adrenal or pituitary disease. • Galactosemia.
Renal	• Chronic renal failure. • Urinary tract infection.
Hematologic	• Sickle cell disease. • Iron deficiency anemia.



The Esophagus

1) Congenital malformation of the esophagus:

Esophageal Atresia and Tracheoesophageal Fistula

Definition

- Esophageal atresia is a congenital defect in which the upper esophagus is not connected to the lower esophagus, ending blindly instead. It is caused by the abnormal development of the tracheoesophageal septum. Esophageal atresia with a fistula connected distally to the trachea is the most common kind of esophageal malformation (classified as Gross type C).

- Tracheoesophageal fistula is an abnormal connection between the trachea and esophagus that may be connected to the proximal and/or distal esophageal segment.

Etiology and Epidemiology

- The esophagus and trachea develop in close proximity to each other during weeks 4-6 of fetal life.
- Defects in the mesenchyme separating these two structures result in a tracheoesophageal fistula (TEF), often in association with other anomalies (renal, heart, spine, limbs).
- This defect occurs in about 1 in 3,000 live births.
- TEF is not thought to be a genetic defect.

Clinical Manifestations

- **Prenatal**
  - Polyhydramnios: the fetus is unable to swallow amniotic fluid, this increases the risk of premature birth
- **Postnatal**
  - Exhibit drooling due to pooling of secretions
  - Mucus and saliva bubbling from the nose and mouth.
  - Patients with a TEF are vulnerable to aspiration pneumonia and present with:
  - Coughing spells
  - Rales
  - Cyanotic attacks due to reflex laryngospasms that prevent reflux aspiration.
- When TEF is suspected, the first feeding should be delayed until a diagnostic study is performed.

Clinical Manifestations

- The most common forms of TEF occur with esophageal atresia; the “H-type” TEF without atresia is uncommon, as is esophageal atresia without TEF (Fig. 128.3).

- Associated defects include the VACTERL association

  - Vertebral anomalies (70%),
  - Anal atresia (imperforate anus) (50%),
  - Cardiac anomalies (30%),
  - Tracheoesophageal fistula (70%),
  - Renal anomalies (50%),
  - and Limb anomalies (polydactyly, forearm defects, absent thumbs, syndactyly) (70%).

- A single-artery umbilical cord is often present.

Various types of tracheoesophageal fistulas (TEF) with relative frequency (%)

- Esophageal atresia with distal TEF (85%), Gross type C
- Esophageal atresia with no TEF (8%), Gross type A
- H-type TEF (4%), Gross type E
- Esophageal atresia with proximal TEF (2%), Gross type B
- Esophageal atresia with proximal and distal TEF (1%), Gross type D

Various types of tracheoesophageal fistulas (TEF) with relative frequency (%)

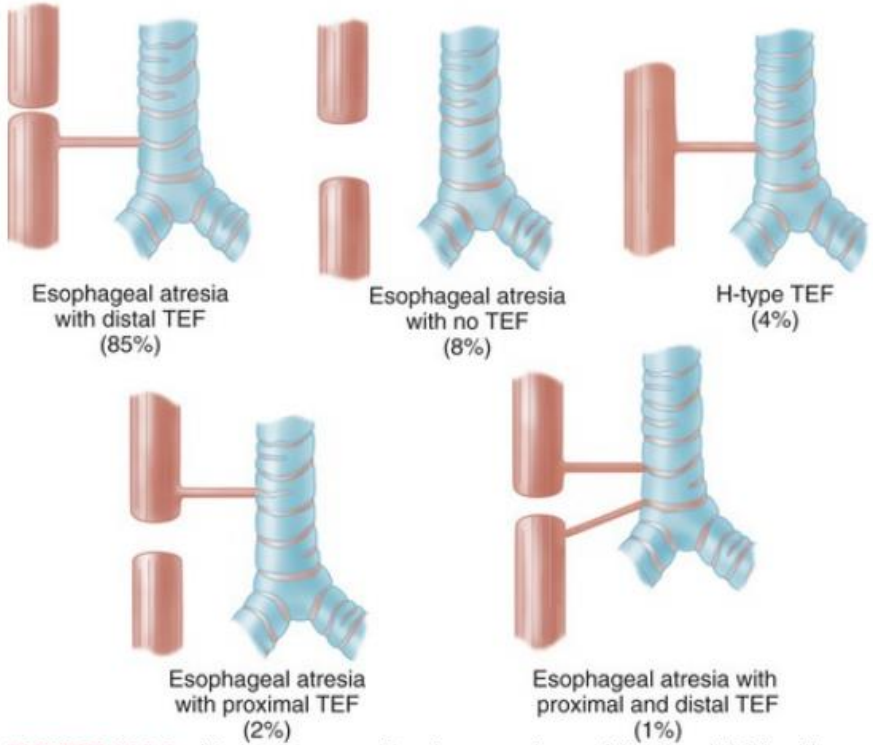


FIGURE 128.3 Various types of tracheoesophageal fistulas (TEF) with relative frequency (%).

Laboratory and Imaging Studies

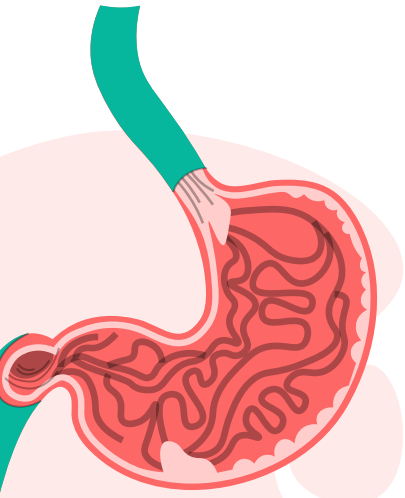
- The simplest test for TEF is to gently attempt to place a 10F or larger tube via the mouth into the stomach. The passage of the tube is blocked at the level of the atresia.
- A chest x-ray reveals the tube coiled in the esophageal pouch. Air can be injected through the tube to outline the atretic pouch. Barium should not be used because of extreme risk of aspiration, but a tiny amount of dilute water-soluble contrast agent can be given carefully, then suctioned when the defect is clearly shown.
- The chest x-ray may also reveal a large gastric bubble (air in the stomach), gross types A and B present with a gasless abdomen.

Treatment and Prognosis

- The treatment of TEF is surgical. The fistula is divided and ligated. The esophageal ends are approximated and anastomosed. In some cases, primary anastomosis cannot be performed because of a long gap between the proximal and distal esophagus. Various techniques have been described to treat this problem, including pulling up the stomach, elongating the esophagus by myotomy, and simply delaying esophageal anastomosis and providing continuous suction to the upper pouch while allowing for growth.

Complications

- The surgically reconstructed esophagus is not normal and is prone to poor motility, GER, anastomotic stricture, recurrent fistula, and leakage. The trachea also is malformed; tracheomalacia and wheezing are common.



The Esophagus

2) Gastroesophageal Reflux Disease

Etiology and Epidemiology

- Gastroesophageal reflux (GER) is defined as the effortless retrograde movement of gastric contents upward into the esophagus or oropharynx.

- In infancy, GER is not always an abnormality. Physiological GER (“spitting up”) is normal in infants younger than 8-12 months old. Nearly half of all infants are reported to spit up between 2 and 4 months of age. Infants who regurgitate meet the criteria for physiological GER so long as they maintain adequate nutrition and have no signs of respiratory complications or esophagitis.

- Contributing factors of infantile GER include liquid diet; horizontal body position; short, narrow esophagus; small, noncompliant stomach; frequent, relatively large volume feedings; and an immature lower esophageal sphincter (LES).

- As infants grow, they spend more time upright, eat more solid foods, develop a longer and larger diameter esophagus, have a larger and more compliant stomach, and experience lower caloric needs per unit of body weight. As a result, most infants stop spitting up by 9-12 months of age.

Etiology and Epidemiology

- Gastroesophageal reflux disease (GERD) occurs when GER leads to troublesome symptoms or complications such as poor growth, pain, or breathing difficulties.

- GERD occurs in a minority of infants but is often implicated as the cause of fussiness. GERD is seen in fewer than 5% of older children. In older children, normal protective mechanisms against GER include antegrade esophageal motility, tonic contraction of the LES, and the geometry of the gastroesophageal junction.

- Abnormalities that cause GER in older children and adults include reduced tone of the LES, transient relaxations of the LES, esophagitis (which impairs esophageal motility), increased intraabdominal pressure, cough, respiratory difficulty (asthma or cystic fibrosis), and hiatal hernia.

Clinical Manifestations

- The presence of GER is easy to observe in an infant who spits up. In older children, the refluxate is usually kept down by reswallowing, but GER may be suspected by associated symptoms, such as heartburn, cough, epigastric abdominal pain, dysphagia, wheezing, aspiration pneumonia, hoarse voice, failure to thrive, and recurrent hiccoughs or belching.

- In severe cases of esophagitis, there may be laboratory evidence of anemia and hypoalbuminemia secondary to esophageal bleeding and inflammation. When esophagitis develops as a result of acid reflux, esophageal motility and LES function are impaired further, creating a cycle of reflux and esophageal injury.

Laboratory and Imaging Studies

- A clinical diagnosis is often sufficient in children with classic effortless regurgitation and no complications. Diagnostic studies are indicated if there are persistent symptoms or complications or if other symptoms suggest the possibility of GER in the absence of regurgitation. A child with recurrent pneumonia, chronic cough, or apneic spells without overt emesis may have occult GER.

- A barium upper gastrointestinal (GI) series helps to rule out gastric outlet obstruction, malrotation, or other anatomical contributors to GER. Because of the brief nature of the examination, a negative barium study does not rule out GER, nor does it rule it in as it is normal to have some reflux into the esophagus many times per day.

- A 24-hour esophageal pH probe monitoring uses a pH electrode placed transnasally into the distal esophagus, with continuous recording of esophageal pH. Data typically are gathered for 24 hours and analyzed for the number and temporal pattern of acid reflux events. Esophageal impedance monitoring records the migration of electrolyte-rich gastric fluid in the esophagus.

- Endoscopy is useful to evaluate for esophagitis, esophageal stricture, and anatomical abnormalities.

Treatment

- In otherwise healthy young infants, no treatment is necessary.
- For infants with complications of GER, an H2 blocker or proton-pump inhibitor may be offered (Table 128.1), but these have shown little benefit in infants with uncomplicated GER and/or fussiness.
- Prokinetic drugs, such as metoclopramide, occasionally are helpful by enhancing gastric emptying and increasing LES tone, but they are seldom effective and may lead to complications.
- When severe symptoms persist despite medication, or if life-threatening aspiration is present, surgical intervention may be required. Fundoplication procedures, such as the Nissen operation, are designed to enhance the antireflux anatomy of the LES. In children with a severe neurological defect who cannot tolerate oral or gastric tube feedings, placement of a feeding jejunostomy may be considered as an alternative to fundoplication.
- In older children, lifestyle changes should be discussed, including cessation of smoking, weight loss, not eating before bed or exercise, and limiting intake of caffeine, carbonation, and high-fat foods. However, proton pump inhibitor therapy is more effective in reducing symptoms and supports healing.

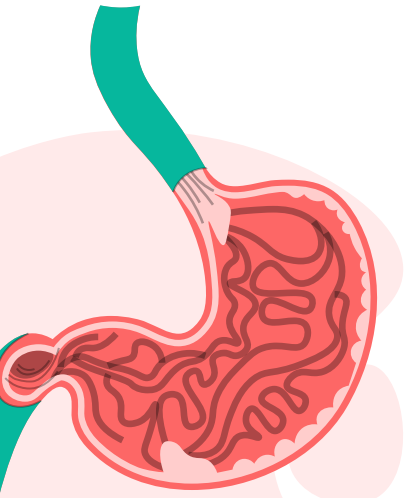
TABLE 128.1

Treatment of Gastroesophageal Reflux

THERAPIES	COMMENTS
CONSERVATIVE THERAPIES	
Towel on caregiver's shoulder	Cheap, effective; useful only for physiological reflux
Thickened feedings	Reduces number of episodes, enhances nutrition
Smaller, more frequent feedings	Can help some; be careful not to *underfeed* child
Avoidance of tobacco smoke and alcohol	Always a good idea; may help reflux symptoms
Abstaining from caffeine	Inexpensive, offers some benefit
Positional therapy—upright in seat, elevate	Prone positioning with head of crib or bed up is helpful, but *not* for young infants because of risk of SIDS
Weight loss when indicated	Increased weight (especially abdominal) increases intraabdominal pressure, leading to reflux
MEDICAL THERAPY	
Proton pump inhibitor	Effective medical therapy for heartburn and esophagitis
H_2-receptor antagonist	Reduces heartburn, less effective for healing esophagitis
Metoclopramide	Enhances stomach emptying and LES tone; real benefit is often minimal
SURGICAL THERAPY	
Nissen or other fundoplication procedure	For life-threatening or medically unresponsive cases
Feeding jejunostomy	Useful in child requiring tube feeds; delivering feeds downstream eliminates GERD

GERD, Gastroesophageal reflux disease; *LES,* lower esophageal sphincter; *SIDS,* sudden infant death syndrome.

The Esophagus



3) Achalasia

Overview and Cause

Achalasia is an uncommon swallowing disorder that affects about 1 in every 100,000 people. The major symptom of achalasia is usually difficulty with swallowing. Most people are diagnosed between the ages of 25 and 60 years. Although the condition cannot be cured, the symptoms can usually be controlled with treatment.

In achalasia, nerve cells in the esophagus degenerate for unknown reasons. The loss of nerve cells in the esophagus causes two major problems that interfere with swallowing:

- The muscles that line the esophagus do not contract normally, so that swallowed food is not propelled through the esophagus and into the stomach properly.
- The lower esophageal sphincter (LES), a band of muscle that encircles the lower portion of the esophagus, does not function correctly.

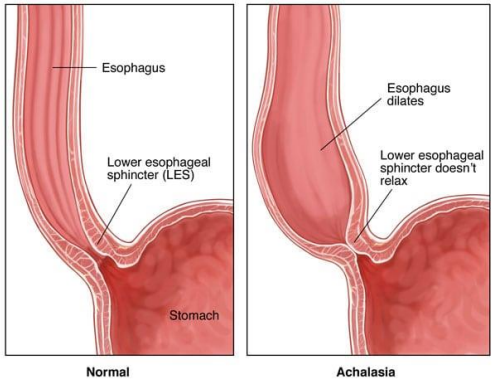
Normally, the LES relaxes when we swallow to allow swallowed food to enter the stomach. When the food has moved through the esophagus into the stomach, the LES muscle contracts to squeeze the end of the esophagus closed, thus preventing the stomach contents from flowing backwards (refluxing) into the esophagus.

In people with achalasia, the LES fails to relax normally with swallowing. Instead, the LES muscle continues to squeeze the end of the esophagus, creating a barrier that prevents food and liquids from passing into the stomach. Over time, the esophagus above the persistently contracted LES dilates, and large volumes of food and saliva can accumulate in the dilated esophagus.

Clinical Manifestations

- The most common symptom of achalasia is difficulty swallowing. Patients often experience the sensation that swallowed material, both solids and liquids, gets stuck in the chest. This problem often begins slowly and progresses gradually. Many people do not seek help until symptoms are advanced. Some people compensate by eating more slowly and by using maneuvers, such as lifting the neck or throwing the shoulders back, to improve emptying of the esophagus.

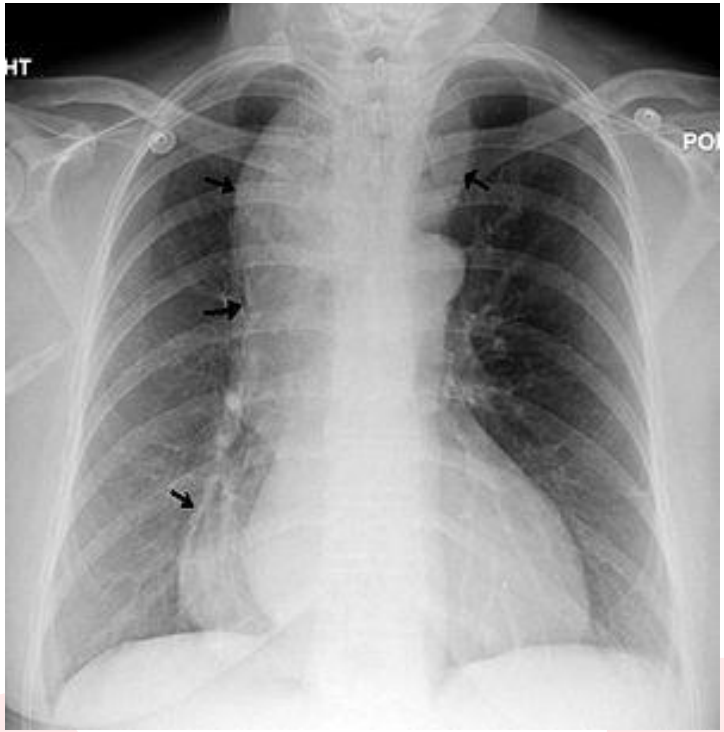
- Other symptoms can include chest pain, regurgitation of swallowed food and liquid, heartburn, difficulty burping, a sensation of fullness or a lump in the throat, hiccups, and weight loss.

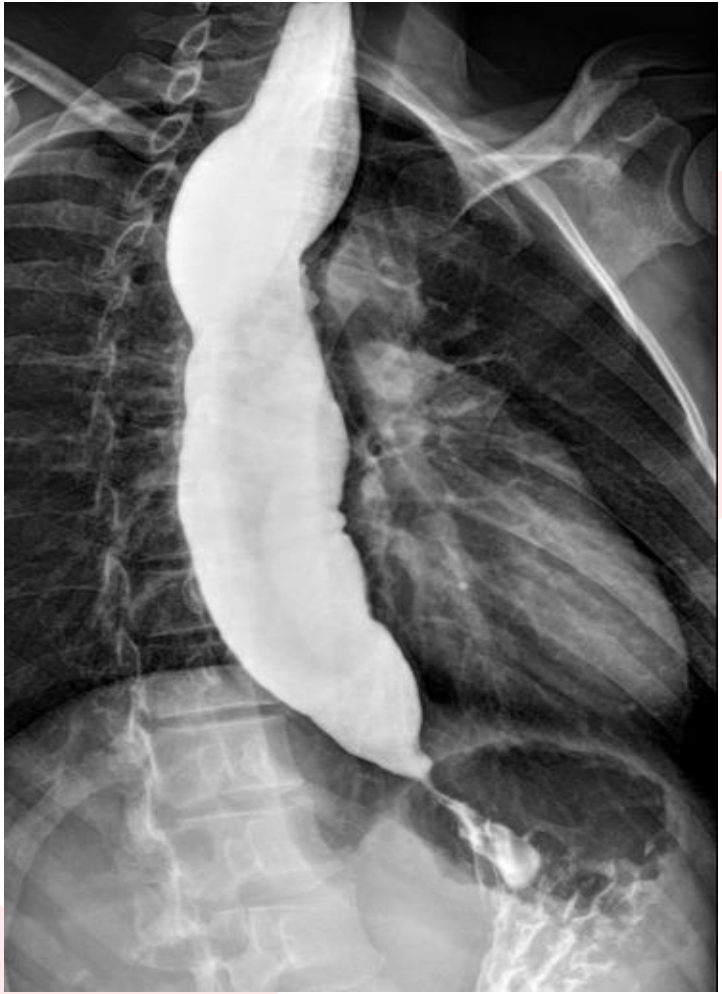


Diagnosis

- Achalasia may be suspected based upon symptoms, but tests are needed to confirm the diagnosis.
- Chest X-rays: may reveal a dilated esophagus and absence of air in the stomach. However, a chest X-ray is not adequate for a diagnosis of achalasia and further testing is required.
- Barium swallow test: The barium swallow test is a common screening test for achalasia. Characteristic findings include a persistently narrowed region at the end of the esophagus (the LES), with a dilated esophagus above the narrowed region. The tapering of the inferior esophagus in achalasia is refered to as the bird's beak sign or rat's tail sign. The barium swallow may also show spastic contractions in the esophagus above the LES, a condition called "vigorous achalasia"
- Esophageal manometry (also called esophageal motility study): The test typically reveals three abnormalities in people with achalasia: high pressure in the LES at rest, failure of the LES to relax after swallowing, and an absence of useful (peristaltic) contractions in the lower esophagus. Esophageal manometry is the gold standard to diagnose achalasia.
- Endoscopy: it often reveals a dilated esophagus that contains retained food ; it may also reveal inflammation, small ulcers caused by residual food or pills, and candida (yeast) infection. This test is usually recommended for people with suspected achalasia and is especially useful for detecting other conditions that can mimic achalasia such as cancer of the upper portion of the stomach.

Diagnosis





Treatment

- Several options are available for the treatment of achalasia. Unfortunately, none can stop or reverse the underlying loss of nerve cells in the esophagus of patients with achalasia. However, the treatments are usually effective for improving symptoms.

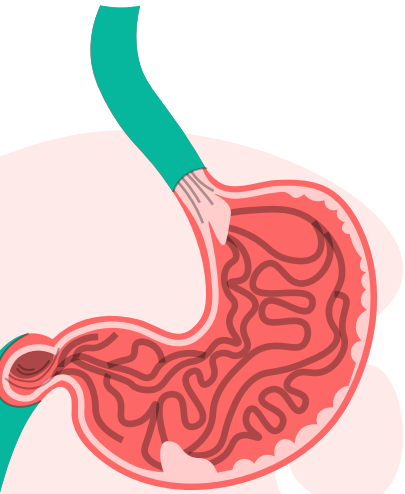
- Drug therapy — Two classes of drugs, nitrates and calcium channel blockers, have LES muscle-relaxing effects. These drugs can decrease symptoms in people with achalasia. The drugs are usually taken by placing a pill under the tongue 10 to 30 minutes before meals. However, because of safety concerns about short-acting nifedipine (a calcium channel blocker), we no longer use it for treating achalasia.

- Balloon dilation (pneumatic dilation) — Balloon dilation is typically performed during endoscopy, during which the physician positions a deflated balloon in the LES and inflates it abruptly to a large size in order to tear the muscle of the LES. This procedure is effective for relieving the swallowing difficulty in patients with achalasia in approximately two-thirds of people, although chest pain persists in some. Patients frequently require more than one balloon dilation treatment for adequate relief.

Treatment

- Surgery (myotomy) — Myotomy is an operation that is used to weaken the LES by cutting its muscle fibers. The most common surgical technique used to treat achalasia is called the Heller myotomy, in which the surgeon cuts the muscles at the end of the esophagus and at the top of the stomach.

- Botulinum toxin injection — Botulinum toxin injections temporarily paralyze the nerves that signal the LES to contract, thereby helping to relieve the obstruction. Botulinum toxin injection also is used occasionally as a diagnostic test for people who appear to have achalasia but who have inconclusive test results.

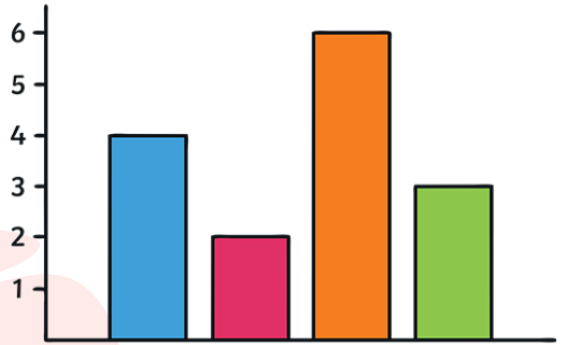


The Esophagus

4) Eosinophilic Esophagitis

Etiology and Epidemiology

- This chronic immune-mediated disorder is characterized by infiltration of eosinophils into the mucosa of the esophagus.
- It is thought to be triggered by non–immunoglobulin (Ig)E-mediated allergic reactions to ingested foods or aeroallergens.
- Eosinophilic esophagitis (EoE) may have a familial component; no specific gene has yet been identified. Incidence appears to be increasing with estimated prevalence of more than 4 per 10,000 children. It may be more common in males than females and in patients with history of atopy.



Clinical Manifestations

- The presentation of EoE often varies with age.
- In young children: it may present with oral aversion, vomiting, and failure to thrive.
- In school-age children: it may present with vague abdominal pain or vomiting.
- In adolescents and adults: it presents with dysphagia and food impactions.
- These symptoms are attributed to the inflammatory response in the esophagus leading to edema and poor esophageal motility.

Laboratory and Imaging Studies

- Diagnosis requires multilevel esophageal biopsies via flexible endoscopy with the finding of more than 15 eosinophils per high-power field.
- Treatment with high-dose proton pump inhibitor therapy is recommended to exclude the possibility that findings are secondary to severe acidic esophageal injury or by evaluating for acid reflux by pH probe testing.
- Gross findings at endoscopy may include normal appearance, or esophageal furrowing, trachealization, and eosinophilic abscesses.
- A barium study may reveal a food impaction in an acutely symptomatic patient or esophageal stricture in someone with chronic disease.

Laboratory and Imaging Studies

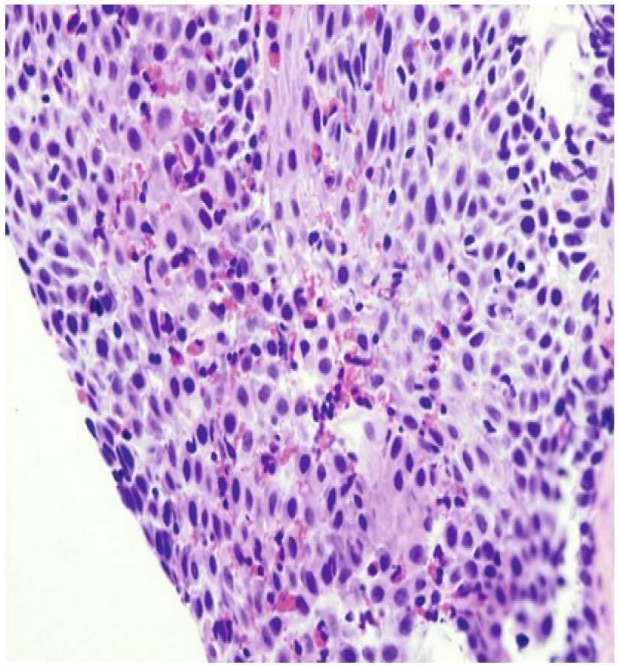


FIGURE 128.1 Histological image of eosinophilic esophagitis. Note the large number of eosinophils within the lamina propria.

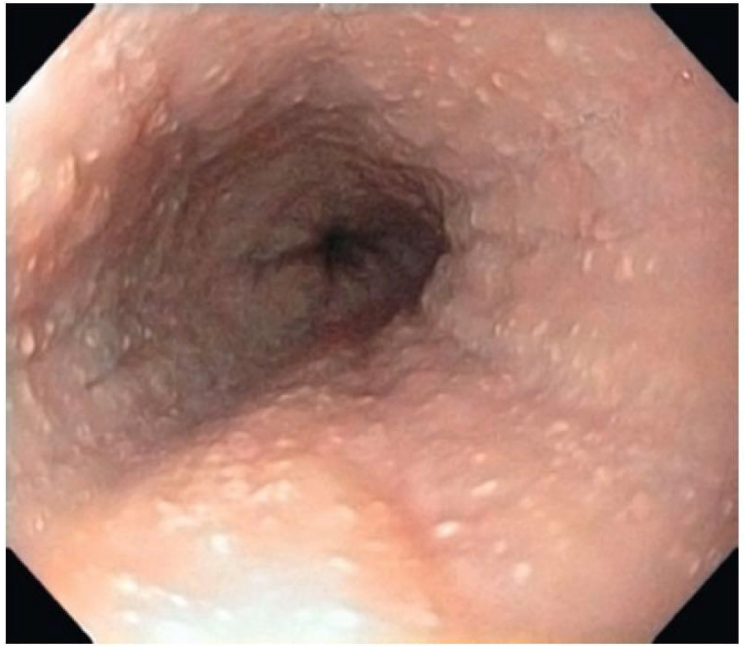


FIGURE 128.2 Endoscopic picture of eosinophilic esophagitis. White plaques on the surface are collections of eosinophils (eosinophilic abscesses). Linear furrowing is also seen.

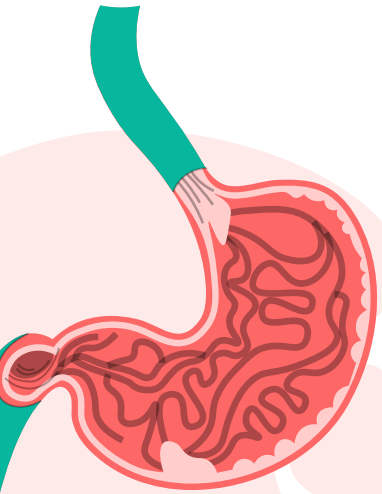
Treatment and Prognosis

- Exposure to identified causative antigens needs to be eliminated. Identification can be difficult, as typical allergy testing (skin prick, RAST, and immunocap assays) only identifies IgE-mediated antigens.
- Atopic patch testing may be more reliable but is not standardized and can be difficult to perform.
- One approach is to eliminate cow's milk, soy, wheat, eggs, peanuts, and fish/shellfish from the diet (“Six food elimination diet”), as these are the most common causative dietary antigens. Repeat endoscopies are often necessary to document efficacy of these eliminations. An elemental diet can also be used and is very effective, but often requires either nasogastric or gastrotomy administration because of its poor palatability.
- Systemic glucocorticoids can decrease symptoms, but long-term use is discouraged due to concerns for potential complications. Swallowed “topical” glucocorticoids administered via a metered-dose inhaler (fluticasone) or mixed as a slurry (budesonide) have shown benefit. Candida esophagitis or oral thrush are the most common side effects.
- Endoscopy can be used to relieve food impactions and to dilate esophageal strictures secondary to EoE.
- The prognosis for EoE is largely unknown. Symptoms tend to wax and wane over time.

Complications

- Failure to thrive or weight loss may be seen due to difficulty in eating.
- Food impactions are a common complication in the older child and may require endoscopic removal.
- Chronic inflammation of the esophagus can predispose to esophageal strictures and possibly dysplasia.

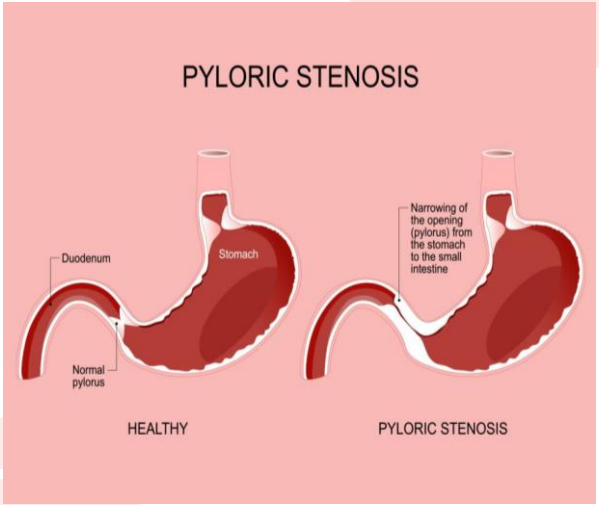
The Stomach



1) Pyloric Stenosis

Etiology and Epidemiology

- Pyloric stenosis is an acquired condition caused by hypertrophy and spasm of the pyloric muscle, resulting in gastric outlet obstruction. It occurs in 6-8 per 1,000 live births, has a 5-to-1 male predominance, and is more common in firstborn children. Its cause is unknown.



Clinical Manifestations

- Infants with pyloric stenosis typically begin vomiting during the first weeks of life, but onset may be delayed.
- The emesis becomes increasingly frequent and forceful as time passes. Vomiting differs from spitting up because of its extremely forceful and often projectile nature.
- The vomited material never contains bile, because the gastric outlet obstruction is proximal to the duodenum. This feature differentiates pyloric stenosis from most other obstructive lesions of early childhood.
- Affected infants are ravenously hungry early in the course of the illness but become more lethargic with increasing malnutrition and dehydration.
- The stomach becomes massively enlarged with retained food and secretions, and gastric peristaltic waves are often visible in the left upper quadrant. A hypertrophied pylorus (the “olive”) may be palpated.
- As the illness progresses, very little of each feeding is able to pass through the pylorus, and the child becomes progressively thinner and more dehydrated.

Laboratory and Imaging Studies

- Repetitive vomiting of purely gastric contents results in loss of hydrochloric acid; the classic laboratory finding is a hypochloremic, hypokalemic metabolic alkalosis with elevated blood urea nitrogen (BUN) secondary to dehydration.
- Jaundice with unconjugated hyperbilirubinemia may also occur.
- Plain abdominal x-rays typically show a huge stomach and diminished or absent gas in the intestine.
- Ultrasound examination shows marked elongation and thickening of the pylorus.
- A barium upper GI series also may be obtained whenever doubt about the diagnosis exists; this shows a “string sign” caused by barium moving through an elongated, constricted pyloric channel.

Laboratory and Imaging Studies

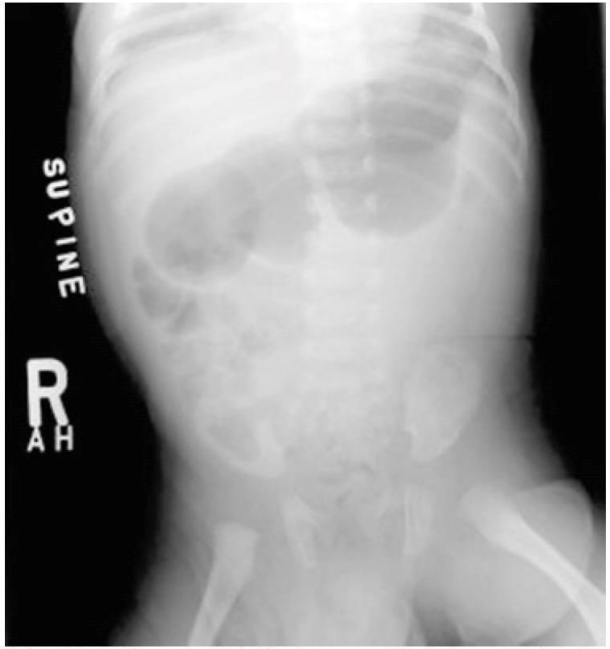


FIGURE 128.4 **Pyloric Stenosis. Note the huge, gas-filled stomach extending across the midline, with minimal air in the intestine downstream.** (Courtesy Warren Bishop, MD.)

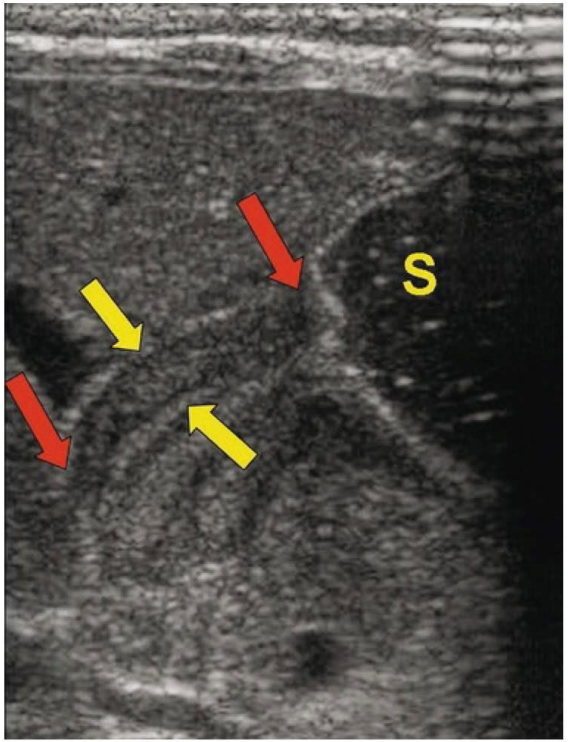
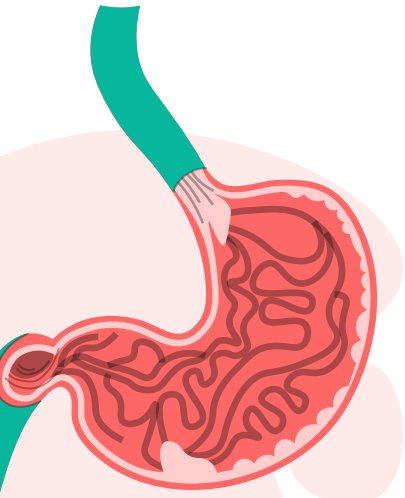


FIGURE 128.5 Ultrasound image of infant with pyloric stenosis. Large, fluid-filled stomach *(S)* is seen at right, with an elongated, thickened pylorus. The length of the pylorus is marked by the *red arrows*; the wall thickness is marked by the *yellow arrows*.

Treatment

- Treatment of pyloric stenosis includes IV fluid and electrolyte resuscitation followed by surgical pyloromyotomy.
- Before surgery, dehydration and hypochloremic alkalosis must be corrected, generally with an initial normal saline fluid bolus followed by infusions of half-normal saline containing 5% dextrose and potassium chloride when urine output is observed.
- In pyloromyotomy (often by laparoscope), the pyloric muscle is incised longitudinally to release the constriction.



The Stomach

2) Gastritis and Gastropathy

Introduction

- Gastritis, gastropathy, and peptic ulcer disease (PUD), collectively known as acid peptic disease, are often described as a spectrum of the same disease.

- Left untreated, gastritis can progress to PUD, which can result in serious complications such as perforation, bleeding, bowel strictures, and obstruction.

  - **Gastritis** is when the lining of your stomach becomes irritated (inflamed), leading to the recruitment of inflammatory cells. Can be classified into acute or chronic gastritis.

  - **Gastropathy** occurs when there is gastric mucosal damage with no inflammatory cells. Agents that cause gastropathy include nonsteroidal antiinflammatory drugs, alcohol, bile, and stress-induced injury.

Etiology

- Infection with bacteria, such as Helicobacter pylori (H. pylori)
- Prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs)
- Traumatic injury
- Ingestion of corrosive substances
- Extreme stress as result of sepsis, shock ....
- Certain diseases, such as megaloblastic (pernicious) anemia and autoimmune disorders.
- Rarely, hypersecretory states (Zollinger-Ellison syndrome)

H.Pylori Gastritis

- H pylori is a **gram-negative bacillus** described microscopically as spiral shaped.
- The bacteria is transmitted via the **fecal-to-oral** or **oral-to-oral route.**
- Most people are asymptomatic from infection, but the bacteria are also implicated as the cause for some gastric and duodenal ulcers as well as gastric adenocarcinoma and lymphomas.
- H pylori organisms have certain features that contribute to their virulence, including flagella, urease, adhesins, and toxin production. These features allow the organism to overcome the mucosal defenses and cause chronic gastritis, which can eventually lead to ulcer formation.

Clinical Manifestations

- Most children have the H. pylori bacteria for years without knowing it because they don't have any symptoms.

- When symptoms do occur, they may include belly pain, which can:

- Be a dull, gnawing pain
- Happen 2 to 3 hours after a meal
- Come and go for a few days or weeks
- Occur in the middle of the night when the child's stomach is empty

- Other symptoms may include:

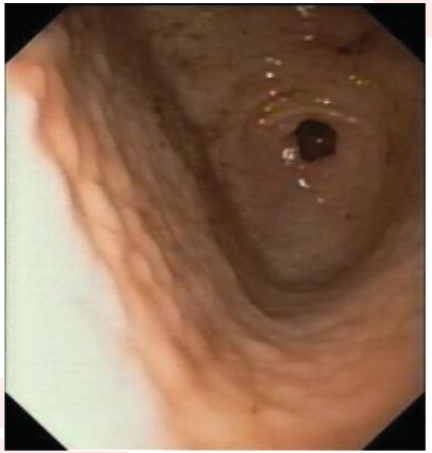
- Loss of weight and appetite
- Swelling or bloating
- Burping
- Nausea and Vomiting
- Blood in vomit or stool (a sign that the stomach lining may be bleeding).
- Refractory IDA
- ITP

Laboratory and Imaging Studies

1) **Stool culture**

2) **C- Urea breath tests:** These tests check if there is any carbon present. If carbon is found, that means that H. pylori is present.

3) **Esophagogastroduodenoscopy** (also called EGD or upper endoscopy): to detect underlying pathologies. A small tissue sample or biopsy is taken if needed. The tissue sample can be checked for signs of infection or of H. pylori bacteria.



Lymphoid Nodular Hyperplasia

Table 2. Diagnostic tests in *Helicobacter pylori* infection in children.

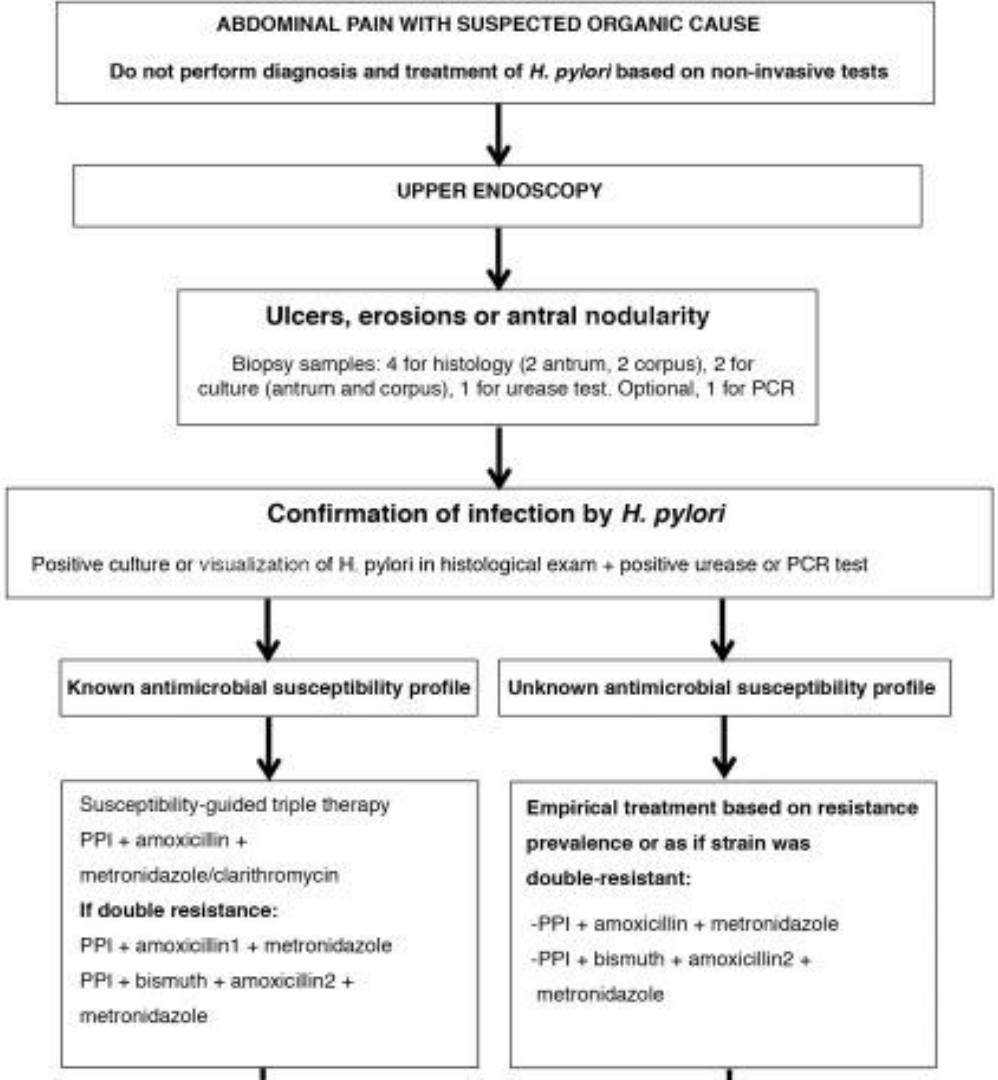
Noninvasive test	Remarks
Serology: anti *Helicobacter pylori* IgG antibody serology	Not recommended for clinical diagnosis in children Very low sensitivity [17] Recent serology incorporating current infection marker may detect active infection [18]
Stool *Helicobacter pylori* antigen assay [20,21]	Several commercial assay kits available Monocolonal assays are more sensitive than polyclonal tests High sensitivity and specificity shown in both diagnosis and after treatment testing
^{13}C urea breath test [6,22]	Well accepted test to confirm eradication. Recent studies have shown accuracy in children below 6 years of age
Diagnosis at endoscopy	
Rapid urease test	Acceptable specificity and sensitivity False-negative in atrophic gastritis, low-density infection Testing with two or more tissue samples increases detection rate [32–34]
Histology	Standard method of diagnosis for *Helicobacter pylori* gastritis
Microbial culture	Not routinely used Useful for susceptibility testing Recent studies report success with quicker culture method [37]

Treatment

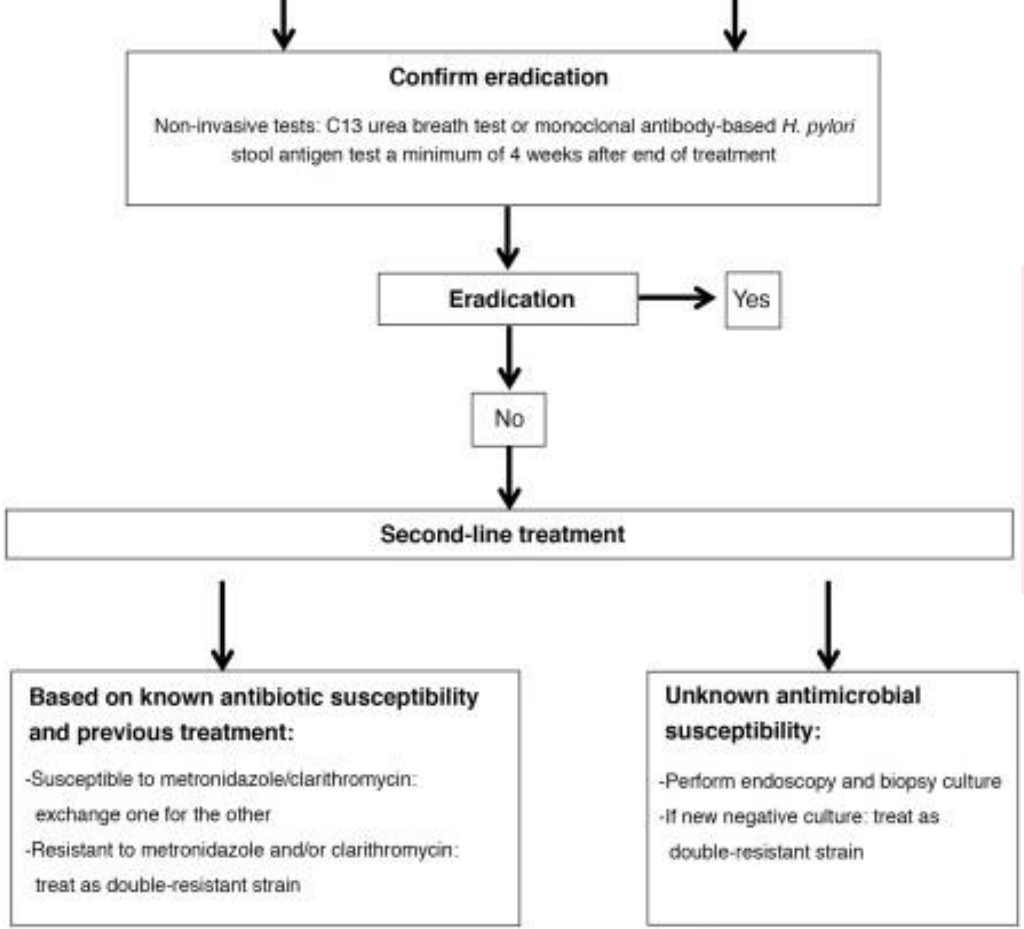
- Proton Pump Inhibitor (PPI)
- High-dose amoxicillin: 75mg/kg/day to a maximum of 3g/day.
- In children aged more than 8 years, tetracycline may be substituted for amoxicillin

→ **Multidrug Regimen:** such as omeprazole, clarithromycin-metronidazole (or amoxicillin)

Management


ABDOMINAL PAIN WITH SUSPECTED ORGANIC CAUSE
Do not perform diagnosis and treatment of H. pylori based on non-invasive tests
UPPER ENDOSCOPY
Ulcers, erosions or antral nodularity
Biopsy samples: 4 for histology (2 antrum, 2 corpus), 2 for culture (antrum and corpus), 1 for urease test. Optional, 1 for PCR
Confirmation of infection by H. pylori
Positive culture or visualization of H. pylori in histological exam + positive urease or PCR test
Known antimicrobial susceptibility profile
Unknown antimicrobial susceptibility profile
Susceptibility-guided triple therapy
PPI + amoxicillin + metronidazole/clarithromycin
If double resistance:
PPI + amoxicillin1 + metronidazole
PPI + bismuth + amoxicillin2 + metronidazole
Empirical treatment based on resistance prevalence or as if strain was double-resistant:
-PPI + amoxicillin + metronidazole
-PPI + bismuth + amoxicillin2 + metronidazole

Management



PPI: Proton pump inhibitor

[1] High-dose amoxicillin: 75 mg/kg/day to a maximum of 3 g/day.

[2] In children aged more than 8 years, tetracycline may be substituted for amoxicillin.

Other Types of Gastritis

1) **NSAIDs**, like aspirin, reduce the production of the hormone prostaglandin as well as inhibit cyclooxygenase (COX). One of the things prostaglandin does is increase the production of gastric (stomach) mucus and substances that neutralize stomach acid. If there is too little prostaglandin, the stomach lining becomes more susceptible to damage from stomach acid.

2) Portal Hypertensive Gastropathy

- Refers to changes in the stomach lining, which includes gastric mucosa becoming congested with dilated and distended capillaries, caused by portal hypertension. PHG can cause changes to the entire gastrointestinal tract and lead to internal bleeding.

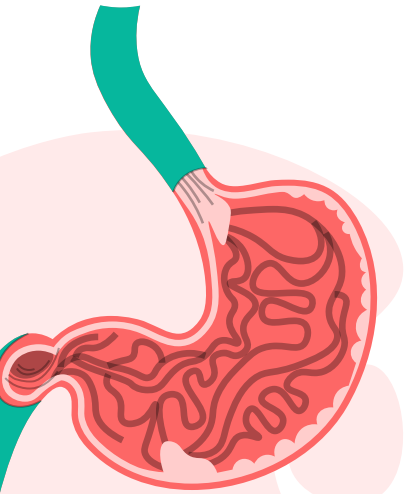
- Most patients with PHG do not have immediate symptoms. However, if PHG worsens, it can lead to later-stage symptoms, including **bleeding** in the stomach, **anemia**, **poor wound healing**, increased susceptibility to side effects from ingested toxins, such as medications and food.

- There are two categories of portal hypertensive gastropathy classified based on the degree of changes to the stomach lining as follows:

1- Mild PHG: In mild PHG, a snakeskin mosaic pattern appears on the lining of the stomach.

2- Severe PHG: In severe PHG, the snakeskin pattern will appear along with other changes to the lining. These changes can consist of flat or bulging red or black-brown spots, an irregular shape of the lining. In this stage there may also be bleeding inside the stomach.

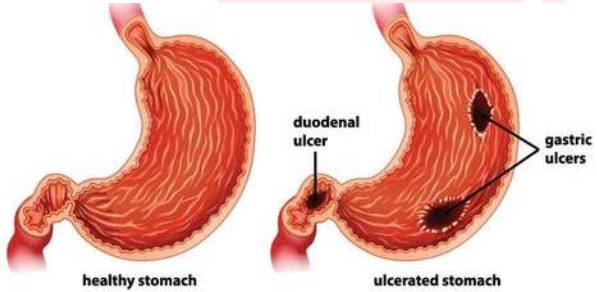
The Stomach



3) Peptic Ulcer Disease

Introduction

- Ulcers develop when the normal defense and repair mechanisms of the lining of the stomach or duodenum are weakened, making the lining more likely to be damaged by stomach acid.
- A peptic ulcer located in the stomach is known as a gastric ulcer.
- A peptic ulcer located in the duodenum is called a duodenal ulcer.
- Risk Factors include:



a) Helicobacter pylori infection
b) Drugs like NSAIDs, Tobacco use, Bisphosphonates, Potassium supplements
c) Family history
d) Sepsis
e) Head trauma
f) Burn injury
g) Hypotension

Clinical Manifestations

- Symptoms—epigastric pain, fullness, bloating, nausea, retrosternal and epigastric pain, sensation of regurgitation, dysphagia, odynophagia

- With duodenal ulcers, pain typically occurs several hours after meals and often awakens patients at night. Eating tends to relieve the pain.

- Gastric ulcers differ in that pain is commonly aggravated by eating, resulting in weight loss.

- Alarm symptoms:

1) Weight loss
2) Failure to thrive
3) Hematemesis
4) Melena
5) Chronic vomiting
6) Iron deficiency anemia
7) Nocturnal pain

Clinical Manifestations

- Patients with perforated ulcer may present with shock, anemia, peritonitis and pancreatitis
- If inflammation and edema are extensive, acute or chronic gastric outlet obstruction can occur.
- In a child with a normal diet for age, iron deficiency anemia may suggest peptic ulceration.

Laboratory and Imaging Studies

- Endoscopy can be used to diagnose the underlying condition.
- Empiric therapy with H2 blockers or proton pump inhibitors can be used but may delay diagnosis of conditions such as H. pylori, that's why we stop therapy 2 weeks prior to testing.
- For patients with **chronic epigastric pain**, the possibilities of inflammatory bowel disease, anatomic abnormality such as malrotation, pancreatitis, and biliary disease should be ruled out by appropriate testing when suspected.
- Testing for H. pylori can be performed by biopsy during endoscopy with use of a urease test or presence histologically on tissue.
- If endoscopy is not done, noninvasive tests for infection can be done with reasonable accuracy with H. pylori fecal antigen and 13C urea breath tests

Treatment

1) **Proton Pump Inhibitors**

2) **H2-blockers**

- If H. pylori is present in association with ulcers, it should be treated with a multidrug regimen, such as omeprazole clarithromycin-metronidazole (or amoxicillin). Other proton pump inhibitors may be substituted when necessary.

- **Bismuth compounds** are effective against H. pylori and can be considered in addition to the above mentioned therapies.

- In most cases, anti-ulcer medicines heal ulcers quickly and effectively, and eradication of H. pylori prevents most ulcers from recurring.

Thank You!