Chronic diarrhea

Frequent loose bowel movements (4 to 6/day) may occur in normal infants; they are of no concern unless anorexia, vomiting, weight loss, failure to gain weight, or passage of blood also occurs. Breastfed infants tend to have frequent bowel movements, especially if they are not receiving solid food. Diarrhea is a condition characterized by increased frequency and liquidity of bowel movements.

Definition of diarrhea

–Excessive loss of fluid and electrolytes in the stool
– defined as acute and chronic diarrhea

- Acute diarrhea: Short in duration less than 2 weeks
- Chronic diarrhea :-Diarrhea lasting for 2 weeks or more.
  –Volume:- In infants stool volume > 10 g/kg
  In children older than 3 and adults stool volume > 200 g/day

Evaluation of Patients with Chronic Diarrhea

PHASE I
Clinical history focuses on the quality and frequency of stools as well as accompanying signs and symptoms. Reports of vomiting or fever suggest GI infection. An accurate dietary history is critical. Reports of diarrhea beginning with the introduction of wheat cereal suggest celiac disease. Reports of variation in the stool pattern with certain elements of the diet suggest dietary intolerance. The persistent presence of blood in the stool mandates a careful search for more serious infection or GI disorder including specific amounts of fluids ingested per day
Physical Examination focuses on overall appearance and signs of dehydration, growth parameters, and abdominal findings; poor growth suggests more serious disorders. Pulmonary status is also evaluated in children in whom cystic fibrosis is suspected
Stool exam (pH, reducing substances, smear for white blood count, fat, ova, and parasites)
Stool cultures
Stool for *Clostridium difficile* toxin
Blood studies (complete blood count, erythrocyte sedimentation rate, electrolytes, blood urea nitrogen, creatinine)
PHASE II
Sweat chloride
72 hr stool collection for fat determination

	Stool electrolytes, osmolality Stool for phenolphthalein, magnesium sulfate, phosphate Breath H_2 tests
Phase III	
	Endoscopic studies Small bowel biopsy Sigmoidoscopy or colonoscopy with biopsies Barium studies
Phase IV	
Hormonal studies vasoactive intestinal polypeptide, gastrin, secretin, 5-hydroxyindoleacetic assays	

Celiac disease

Clinical Manifestations of Celiac Disease in Children and Adolescents

❖ **System manifestation**

- Gastrointestinal
  - Diarrhea
  - Distended abdomen
  - Vomiting
  - Anorexia
  - Weight loss
  - Failure to thrive
  - Rectal prolapse
  - Aphthous stomatitis
  - Intussusception
- Hematologic -Anemia
- Skeletal -Rickets
  - -Osteoporosis
  - -Enamel hypoplasia of the teeth
- Muscular Atrophy Malnutrition
- Neurologic Peripheral neuropathy
  - Epilepsy
  - Irritability
  - Cerebral calcifications
  - Cerebellar ataxia
- Endocrinologic Short stature

Secondary hyperparathyroidism

- Dermatologic Dermatitis herpetiformis
  Alopecia areata
  Erythema nodosum
- Respiratory Idiopathic pulmonary hemosiderosis

Etiology: Celiac disease is an immune mediated systemic disorder elicited by gluten and related prolamines in genetically susceptible individuals and characterized by the presence of a variable combination of gluten dependent clinical manifestations, celiac disease–specific antibodies, HLA-DQ2 or DQ8 haplotypes, and enteropathy. Celiac disease–specific antibodies comprise autoantibodies against TG2,including endomysial antibodies (EMA), and antibodies against deamidated forms of gliadin peptides.

Celiac disease is triggered by the ingestion of wheat gluten and related prolamines from rye and barley. In most studies oats proved to be safe; however, a few celiac disease patients have oats prolamine–reactive mucosal T cells that can cause mucosal inflammation.

Infectious agents have been hypothesized to play a role because frequent rotavirus infections are associated with an increased risk. It is plausible that the contact with gliadin at a time when there is ongoing intestinal inflammation, altered intestinal permeability, and enhanced antigen presentation can increase the risk of developing celiac disease, at least in a subset of persons.

DIAGNOSIS

- The diagnosis of celiac disease is based on a combination of symptoms, antibodies, HLA, and duodenal histology. The initial approach to symptomatic patients is to test for anti-TG2 IgA antibodies and in addition for total IgA in serum to exclude IgA deficiency. If IgA anti-TG2 antibodies are negative and serum total IgA is normal for age celiac disease is unlikely to be the cause of the symptoms. If anti-TG2 antibody testing is positive the patients should be referred to a pediatric gastroenterologist for further diagnostic workup.
- In patients with positive anti-TG2 antibody levels at or >10 ×upper limits of normal, blood should be drawn for HLA and EMA testing. If the patient is positive for EMA antibodies and positive for DQ2 or DQ8 HLA testing, the diagnosis of celiac disease is confirmed, a gluten-free diet is started and the patient is followed for improvement of symptoms and decline of antibodies.
- In the rare case of negative results for HLA and/or anti- EMA in a child with TG2 antibody titers ≥10 ×upper limits of normal, the diagnostic workup should be extended, including repeated testing and duodenal biopsies. In totally asymptomatic persons belonging to high-risk groups, celiac disease should always be diagnosed using duodenal biopsies.
- **Mucosal biopsy** :When biopsies are indicated at least 4 fragments should be obtained from the descending part of the duodenum and at least 1 from the duodenal bulb. The classic features include villous atrophy, infiltration of the epithelium by cytotoxic intraepithelial T lymphocytes, and crypt hyperplasia. However, the spectrum can range from a simple intraepithelial lymphocytosis without villous blunting or crypt hyperplasia to total villous atrophy with severe crypt hyperplasia. The diagnosis is confirmed by an antibody decline and preferably a clinical response to a gluten-free diet. Gluten challenge and repetitive

biopsies will only be necessary in selected cases in which diagnostic uncertainty remains. (children less than 2 yr)

TREATMENT: The only treatment for celiac disease is lifelong strict adherence to a gluten-free diet . This requires a wheat-, barley-, and rye-free diet. Despite evidence that ***oats are safe*** for most patients with celiac disease, there is concern regarding the possibility of contamination of oats with gluten during harvesting, milling, and shipping.
Compliance with a gluten-free diet can be difficult, especially in adolescents. It is recommended that children with celiac disease be monitored with periodic visits for assessment of symptoms, growth, physical examination, and adherence to the gluten-free diet. Periodic measurements of TG2 antibody levels to document reduction, in antibody titers can be helpful as indirect evidence of adherence to a gluten-free diet

PROGNOSIS. The clinical response to a gluten-free diet of a child with celiac disease is gratifying. Improvement of mood and appetite is followed by lessening of diarrhea. In most cases, changes occur within 1 wk of starting therapy, but the response may occasionally be delayed.Teenagers often become noncompliant. Unfortunately, this is an age when the disorder tends to be symptomatically quiescent, and a teenager may believe that the disorder has resolved. Nevertheless, mucosal damage is present. Subtle manifestations of growth failure or delayed sexual maturation may take place when these patients ingest a gluten-containing diet. Appropriately diagnosed gluten-sensitive enteropathy is a lifelong condition requiring lifelong treatment. The development of malignancy secondary to long-standing celiac disease, especially with poor adherence to the strict gluten-free diet, is mostly a problem seen in the adult population. Malignancy affecting the esophagus, stomach, pharynx, and intestines is increased in patients with celiac disease. The observation that T-cell lymphoma can arise from long-standing celiac disease makes a gluten-free diet the best possible prophylaxis. **Strict adherence to a gluten-free diet reduced the risk of all disease-associated cancers**, including enteropathy-associated T-cell lymphoma. No complications from long-term gluten-free diet treatment are recognized.

Risk Groups for Celiac Disease

- First-degree relatives
- Dermatitis herpetiformis
- Unexplained iron-deficiency anemia
- Autoimmune thyroiditis
- Type 1 diabetes
- Unexplained infertility
- Recurrent abortion
- Dental enamel hypoplasia
- Cryptic hypertransaminasemia
- Autoimmune liver disease
- Short stature
- Delayed puberty

- Down, Williams, and Turner syndromes
- Irritable bowel syndrome
- Unexplained osteoporosis
- Sjögren syndrome
- Epilepsy (poorly controlled) with occipital calcifications
- Selective immunoglobulin A deficiency
- Autoimmune endocrinopathies
- Addison disease
- Aphthous stomatitis
- Ataxia
- Alopecia
- Polyneuropathy
- Irritable bowel syndrome.

lactose intolerance

presentation might be: loose watery diarrhea, flatulence, abdominal distention, bloating, gas, cramps and pain. Some children are asymptomatic unless milk and other lactose containing foods are consumed in large amounts.Types of lactose intolerance

- **Congenital lactase deficiency** is rare and is associated with symptoms occurring on exposure to lactose in milk. Fewer than 50 cases have been reported worldwide.
- **Primary adult type-hypolactasia** is caused by a physiologic decline in lactase actively that occurs following weaning in most mammals. The brush-border lactase is expressed at low levels during fetal life; activity increases in late fetal life and peaks from term to 3 yr, after which levels gradually decrease with age. This decline in lactase levels varies between ethnic groups. Lactase deficiency occurs in approximately 15% of white adults, 40% of Asian adults, and 85% of black adults in the United States.
- **Secondary lactose intolerance** follows small bowel mucosal damage.Partial or total villous atrophy due to disorders such as celiac disease, or following rotavirus gastroenteritis can result in secondary disaccharidase deficiency and transient lactose intolerance. The disaccharidase levels revert to normal after mucosal healing.

Diagnosis: Lactase deficiency can be diagnosed by H2-breath test or by measurement of lactase activity in mucosal tissue retrieved by small bowel biopsy. Diagnostic testing is not mandatory, and often simple dietary changes that reduce or eliminate lactose from the diet relieve symptoms.

Treatment of lactase deficiency consists of a milk-free diet.

- A lactose-free formula (based on either soy or cow’s milk) can be used in infants.
- In older children, low-lactose milk can be consumed.
- Addition of lactase to dairy products usually abbreviates the symptoms.
- Live-culture yogurt contains bacteria that produce lactase enzymes and is therefore tolerated in most patients with lactase deficiency.
- Hard cheeses have a small amount of lactose and are generally well tolerated