Malabsorption

→ Impaired absorption of digested food caused by alterations in the intestinal mucosa.

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Pediatric gastroenterology, hepatology and nutrition

→ If fecal elastase was normal and α-1 antitrypsin clearance test was abnormal, this tells me that the pancreas is functioning but we can't absorb proteins.
If fecal elastase was abnormal and α-1 antitrypsin clearance test was normal, then the problem is with the secretions of the pancreas.

Q1. Mention six signes and symptoms of malabsorption

Q2. Name two screening tests for protein loosing enteropathy → child failure to thrive and edematous (hypoproteinemia)
- Fecal elastase
- α 1 antitrypsin clearanee test

Q3. Malabsorption of fat-soluble vitamins
- Vitamin A deficiency..Night blindness/keratomalacia/immunosuppression/xerosis cutis/keratomalacia
- Vitamin E deficiency leads to..~~Rbc~~ Ms weakness/Hemolytic anemia/demyelination of posterior columns and spinocerebellar tracts
- Malabsorption of vitamin D leads to ~~Rickets~~/Osteomalacia/ hypocalcemic tetany → neurologic manifestations
- Malabsorption of vitamin K is associated with...Coagulopathy..

Q4. Most common causes of malabsorption in children (name 5)
① Celiac disease
② Giardia infx
③ Cystic Fibrosis (pancreatic insufficiency)
④ IBD
⑤ Cow's protein milk allergy
⑥ Short bowel syndrome

* Signs of Rickets:
- Bow legs
- Rachitic rosary (rosary beads)
- Wide anterior frontanelle
- swelling of wrist and ankle joints

Malabsorption

- The primary function of the small intestine is digestion and absorption of ingested nutrients. The term malabsorption refers to impairment in the absorption of one or more substances by the small intestine.

Malabsorption

Signs and Symptoms of Malabsorption

- – Weight loss
- – Failure to thrive
- Diarrhea → In small intestine
  - –Loose and watery due to carbohydrate, bile acids, or fatty acids malabsorption
  - –Bulky and foul-smelling due to fat malabsorption → in large intestine
- Abdominal pain
- Abdominal distention
- Anemia
- Increased flatulence
- Edema → due to hypoproteinemia
- Osteomalacia
- Bleeding tendencies

Diagnostic Investigations

Initial Evaluation of Malabsorption

- Detailed history
- Complete physical examination
- Serial growth and anthropometric measurements → Growth charts
- Screening laboratory tests: عشان نتوقع وجود الـ malabsorption أو FTT عند الـ
- Blood:
  - Complete blood count, complete metabolic panel, erythrocyte sedimentation rate, tissue transglutaminase immunoglobulin A (IgA) antibody, total IgA → HG and IgA for celiac disease
- Stool:
  - Culture, ova and parasites, Clostridium difficile testing, occult blood, pH, reducing substances, fecal hydrolysis for detection of nonreducing carbohydrates, ***elastase***, alpha-1- antitrypsin, stain for fat globules
- Sweat chloride test
  - ↳ for cystic fibrosis

*Reducing substances to rule out Carbohydrate malabsorption

→ CBC : MCV و الـ Hb بغيرنا بانه نشوف الـ

ex. If we have Macrocytic hypochromic anemia → we think of folate or Vit B12 deficiency
and if we have an element of malabsorption → Then we know that the problem is in the terminal illeum

We also look at the level of ESR
*If it's high we think of an inflammatory process ... ex. IBD

→ Pt w/ diarrhea, failure to thrive, macrocytic hypochromic anemia and illieitis ...
Could this be crohn's disease? yes

*Note Celiac disease doesn't come w/ high ESR. However IBD does.

→ We also look at the lymphocytes → ex. Pt w/ FTT, edematous and lymphopenia we think of Intestinal Lymphangiectasia

Diagnostic Investigations

Second-phase Evaluation

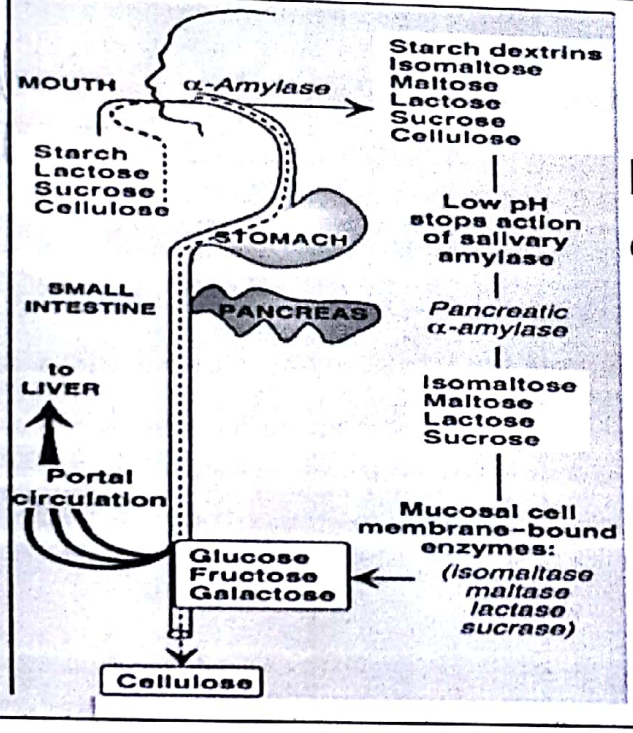
- 72-hour quantitative fecal fat → عشان نتوقع إذا steatorrhea أو لا
- Breath hydrogen test → for carbohydrate malabsorption
- Vitamins A, D, E, and B12; prothrombin time; folate, zinc, iron, ferritin
- Radiolabeled Tc albumin lymphatic scan → for intestinal lymphangectasia
- Endoscopy with biopsy for histology and disaccharidase analysis → for Celiac disease and other pathologies as well.
- Pancreatic enzyme analysis

→ We give the pts radio-labeled carbohydrates → it is then metabolized → produces labeled CO_2 excreted through the lungs.

احنا منعمل measurement ، قديش دخلت كربوهيدرات و قديش رجع بطلعلك CO_2 ، إذا طلعوا نفس الاشي معناته ما عنا مشكلة ، إذا ما طلع ، معناته كله راح بال feces و ما صار له absorption و عنا مشكلة بال Carbohydrate absorption

Carbohydrates

PHYSIOLOGY AND PATHOPHYSIOLOGY OF DIGESTION AND ABSORPTION



Digestion of carbohydrate

Carbohydrates

In malabsorption, maldigested oligosaccharides and unabsorbed monosaccharides are emptied into the colon

- Osmotic effect → Diarrhea
- Gases → Abdominal distention
- Acids → usually stool is very acidic due to the presence of reducing substances.
- Unabsorbed reducing sugars

The hydrogen breath test

Carbohydrates

- **Carbohydrate malabsorption may be due to:** → Absence of the enzyme w/ intact mucosa OR → Destruction of the mucosa (so we are using the enzyme which is present on the brush border).
  - Mucosal damage
    - Brush border enzyme deficiencies can follow injury to the small intestinal mucosa caused by disorders such as
      - infectious gastroenteritis
      - gluten-induced enteropathy ≡ gluten hypersensitivity disease / celiac disease
      - cow milk protein sensitivity
  - Short bowel syndrome
  - Congenital intestinal transport or enzyme deficiencies
  - Excessive ingestion of juices → causes osmotic dietary diarrhea ≡ Sorbitol diarrhea
  - "Adult-onset" lactase deficiency. → switch off
  - Sucrase-isomaltase deficiency
  - Glucose-galactose malabsorption
    - a problem of infancy... these babies can't even absorb breast milk, so they are put on milk which it's sugar is only fructose.

موات بتكون في جين مسؤول عن هاد الانزيم وطارله switch off أو موات بتكون الواحد و هو صغير مثلاً متلاقوا عنده الـ lactose أو يعني قعد مدة طويلة ما بوكل اشي فيه lactose and we need lactose to stimulate lactase, and absence of lactose for a very long time will switch off the gene so they be lactose intolerant.

carbohydrate malab ممكن يعمل mucosal damage ← أي اشي يعمل

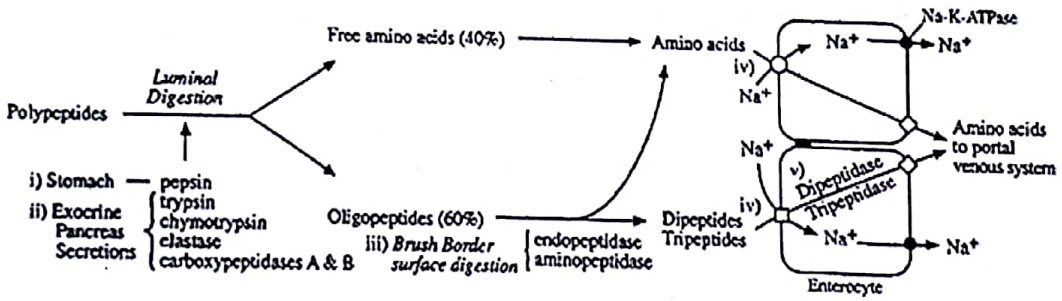
* Rota virus can cause Carb malabsorption → but the idea here is that it's an infectious acute problem.

فبس يجي مريض هيك، وحدة من الـ indications بهاي الحالة هي rota virus. الـ بعطوها بعد الـ lactose free formulas

Proteins

PHYSIOLOGY AND PATHOPHYSIOLOGY OF DIGESTION AND ABSORPTION

Proteins



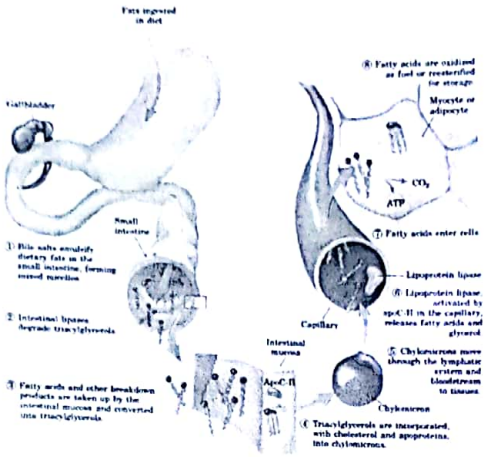
Proteins

- Protein malabsorption leads to failure to thrive, hypoproteinemia, and edema; and can be seen in:
  - Pancreatic insufficiency
  - enterocyte deficiency → ↓ enterocyte due to enterocyte destruction (we lose enterocytes)
  - impaired AA or peptide transport by the enterocyte. (rare)
- A fecal elastase test is a good screening test for....pancreatic insufficiency
- Measuring fecal clearance of alpha-1-antitrypsin in.....
- Other features of protein deficiency include
  - recurrent or severe infections → because we lose our immunoglobulins in the GIT (they are proteins)
  - muscle atrophy
  - Weakness
  - hair loss
  - irritability

Lipids

PHYSIOLOGY AND PATHOPHYSIOLOGY OF DIGESTION AND ABSORPTION

Lipids



Lipids

Fat maldigestion or malabsorption results in a variety of manifestations due not only to malassimilation but also to

- weight loss and malnutrition
- fat-soluble vitamin (A, D, E, and K) deficiency
- Diarrhea
- Steatorrhea
- Increase risk for oxaluria and calcium oxalate kidney stones.

1 gm of fat → 9 calories
1 gm of sugar → 4 calories

*So when we have lipid/fat malabsorption the deficiency of calories is very high فبكون الـ FTT واضح هنا

→absorption الـ GIT ملياان مش بعبرو الـ lipids و هاي الـ lipids بتمسك بالكالسيوم و بعمل الـ oxalate free و بروح عالـ kidneys و بعمل stones

(احنا عادة منخفف من الـ oxalate بوجود الكالسيوم)

فإذا اجاك طفل محمول من الـ nephro load عنده calcium oxalate stones و failure to thrive و عنده إسهال دهني يخطر على بالنا إنه هاد ممكن عنده lipid malabsorption

Fat malabsorption occurs in:

- Pancreatic insufficiency → secretes enzymes that absorbs fat
  - Congenital, such as in cystic fibrosis and Shwachman-Diamond syndrome
  - Acquired, as in chronic pancreatitis.
- In diseases that impair bile production or excretion (ex. cholestasis)
- Abetalipoproteinemia.....(rare).....
  ↳ an autosomal ressecive disease in which there is deficiency of apolipoproteins (ApoB-48, ApoB-100)

* Insufficient ApoB-48 leads to defective chylomicron formation which then causes resorbed lipids to be stuck in intestinal epithelial cells.

Vitamins and Minerals

PHYSIOLOGY AND PATHOPHYSIOLOGY OF DIGESTION AND ABSORPTION

Vitamins and Minerals

- malabsorption of fat-soluble vitamins
  - Vitamin A deficiency.....
  - Vitamin E deficiency leads to.....
  - Malabsorption of vitamin D leads to .....
  - Malabsorption of vitamin K is associated with .....
- vitamin B12 deficiency.
  - lack of intrinsic factor
  - Ileal resection or inflammation (crohn's disease, illeal TB)
  - pancreatic insufficiency

if severe, vit B12 deficiency can lead to: Macrocytic hypochromic megaloblastic anemia
- Neurological symptoms (ex. parasthesias
- Memory problems
- Hypersigmented polymorphonuclear cells

- Zinc malabsorption..can present as.....
  Acrodermatitis enteropathica (so diarrhea and skin manifestations)

Pancreatic Insufficiency

SPECIFIC DISORDERS LEADING TO MALABSORPTION

Pancreatic Insufficiency

Pancreatic Causes of Malabsorption

- Cystic fibrosis
- Shwachman-Diamond syndrome
- Johanson-Blizzard syndrome
- Pearson syndrome
- Chronic pancreatitis
- Trypsinogen deficiency
- Amylase deficiency
- Lipase deficiency

بس اعرفي اسمائهم الوحيدة اللي بتهمنا تفاصيلها هي ال cystic fibrosis

Defects in Bile Acid Micellar Solubilization

SPECIFIC DISORDERS LEADING TO MALABSORPTION

Defects in Bile Acid Micellar Solubilization

Moderate steatorrhea can occur in any hepatobiliary disorder leading to bile acid deficiency, which can result from impaired hepatic synthesis or impaired bile flow.

Conditions Leading to Bile Acid Deficiency

- Chronic cholestasis → ما في bile يوصل الأمعاء عشان fat لا يمتص
- Bile acid pool depletion → This occurs when the enterohepatic circulation is interrupted.
- Ileal resection → site of the enterohepatic circulation
- Bile acid deconjugation by bacteria → occurs when there is bacterial overgrowth (ex. w/ excessive use of Abx)
  - * other causes of bacterial overgrowth:
  - Short bowel syndrome
  - Abnormal motility of the GIT (we need normal motility to keep the balance)

interruption of the entero-hepatic circulation يصير كمان فيصير bacterial overgrowth عنا يصير فيصير stasis عنا يكون لما

Intestinal Brush Border Disorders

SPECIFIC DISORDERS LEADING TO MALABSORPTION

Intestinal Brush Border Disorders

Brush Border Disorders
- Congenital Causes
- Microvillus inclusion disease
- Tufting disease
- Primary lactase deficiency
- Sucrase-isomaltase deficiency
- Glucose/galactose malabsorption

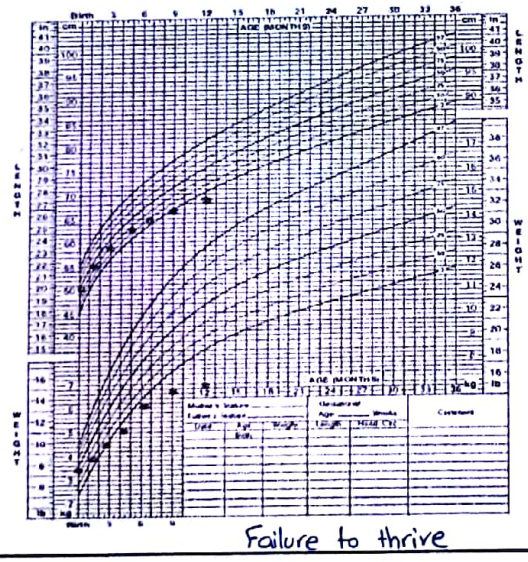
Reduced Mucosal Surface Area
- Short bowel syndrome
- Ileal resection (such as necrotizing enterocolitis or Crohn disease)

Inflammatory Causes
- Celiac disease
- Crohn disease
- Postinfectious diarrhea
- Allergic enteropathy
- Autoimmune enteropathy

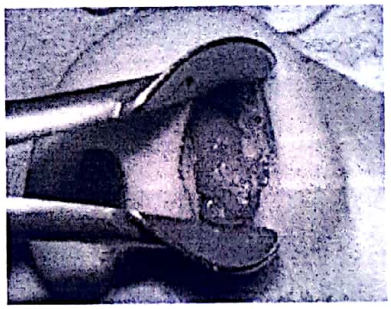
☆ Very Important for the mini OSCE

CLINICAL CASES

1.5 year old presented with



Failure to thrive

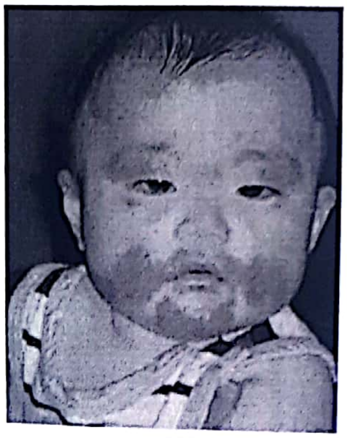


nasal polyp

Cystic Fibrosis

8 months old boy presented with

Bloody Diarrhea

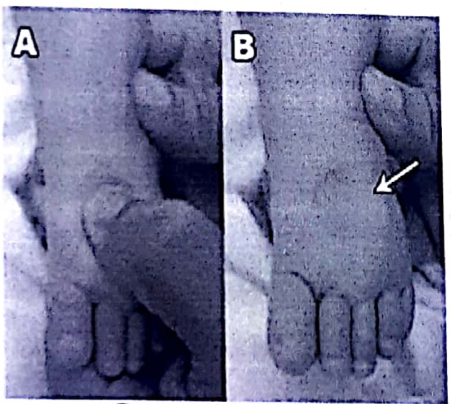


Eczema
↳ Allergy

*IBD comes w/ bloody diarrhea but the presentation is mostly at 2 or 3 years of age (not this small as in this case)
إذا بدك تشخص IBD بطفل أقل من سنتين
you have to rule out immunodeficiency)
+ IBD doesn't cause eczema.

Answer : Cow's protein milk allergy

4 month presented with



Pitting edema
(so protein losing enteropathy)

Table 1: Complete blood count (CBC) before treatment

CBC Report

Parameters	Results	Reference values
Red blood cells count (10^{12}/l)	12.2	8-18
Packed cell volume %	29	22-38
Hemoglobin Conc. (g/dl)	12.5	8-12
Mean corpuscular volume (fl)	21.3	16-25
Mean corpuscular hemoglobin (pg)	10.9	5.2-8
Mean corpuscular hemoglobin concentration (g/dl)	39	30-36
White blood cells (10^9/l)	10.5	4-13
Neutrophils %	66	30-48
Lymphocytes %	29	50-70
Monocytes %	03	0-4
Eosinophilia %	02	1-8
Basophils %	Nil	0-1

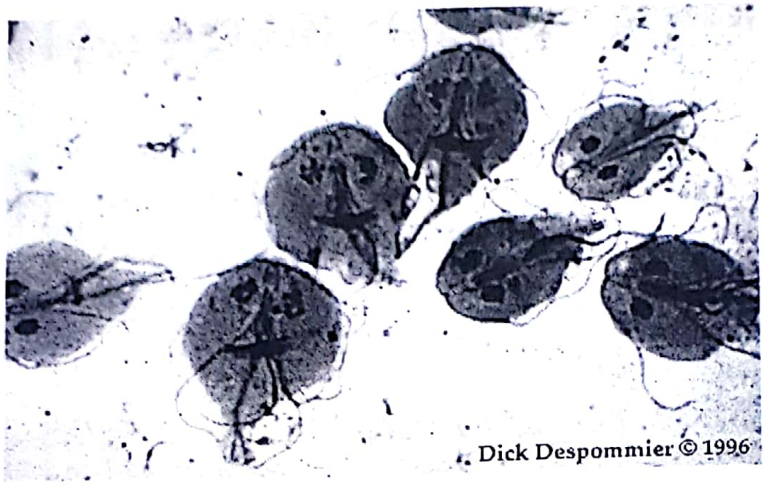
Lymphopenia

Intestinal lymphangectasia

(lymph is being poured into the GIT)

لما نعملهم حدول endoscopy منشوف white mucosa مش pink ، من الlymph

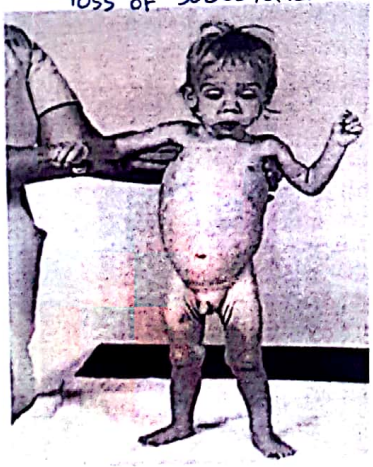
A 4 year old girl presented with diarrhea for 20 days; stool cx showed this parasite that lives in swimming pools



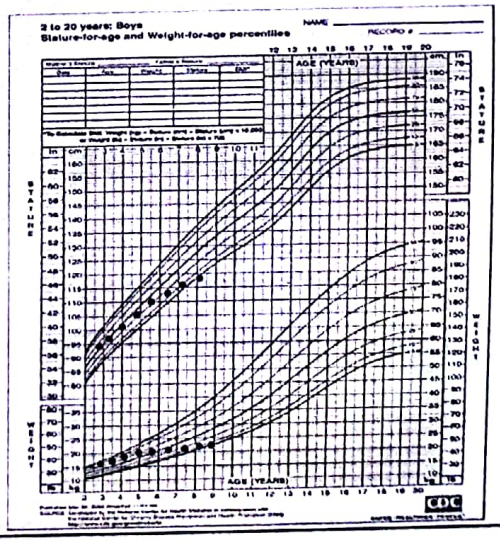
Giardia Lamblia

48 months old child with failure to thrive, chronic diarrhea, severe rickets and iron deficiency anemia

Abd. distention
Cachexic
loss of subcutaneus fat



Failure to thrive.



Typical case of Celiac Disease

٠٥/٠٢/١٤٤١

Celiac disease

- Celiac disease is an immune-mediated enteropathy caused by permanent sensitivity to gluten in genetically susceptible individuals.
- Its prevalence is estimated to be 1 in 300 to 1 in 80 children.
- Gluten protein is derived from a group of cereal grains that includes wheat, rye, and barley. Pure oats are not considered an offending agent.

It doesn't go away, it doesn't get cured

↳ I need to prove genetics-mediated by immunoglobulins

↳ in the small intestine

قمح / شعير / جاودر

They can eat corn, gluten free oatmeal, rice, potato

(النخوفان العادي منصنوع عنه كونه بنطحن بنفس مطاحن الشعير)

Risk groups of CD

- First-degree relatives
- Dermatitis herpetiformis
- Unexplained iron-deficiency anemia
- Autoimmune thyroiditis
- Type 1 diabetes
- Dental enamel hypoplasia
- Autoimmune liver disease
- Short stature
- Delayed puberty
- Down, Williams, and Turner syndromes
- Irritable bowel syndrome
- Sjögren syndrome
- Epilepsy (poorly controlled) with occipital calcifications
- Selective immunoglobulin A deficiency
- Autoimmune endocrinopathies
- Addison disease
- Aphthous stomatitis
- Ataxia
- Alopecia
- Polyneuropathy

→ Ppl screened anually for celiac disease (anually because it can present at any age)

* First degree relatives
* Type 1 DM
* Selective IgA deficiency
* Other autoimmune diseases
* Down, williams, Turner syndromes

Clinical manifestations in C.D

- **Gastrointestinal tract** (Atrophy of the small bowel mucosa /Malabsorption)
  - Diarrhea
  - Distended abdomen
  - Vomiting
  - Anorexia
  - Weight loss
  - Failure to thrive
  - Rectal prolapse
  - Aphthous stomatitis
  - Intussusception
- **Endocrinologic** (Malnutrition, Calcium/vitamin D malabsorption)
  - Short stature
  - Pubertas tarda
  - Secondary hyperparathyroidism
- **Dermatologic** (Autoimmunity)
  - Dermatitis herpetiformis
  - Alopecia areata
  - Erythema nodosum

Clinical manifestations in C.D

- Hematologic (Iron malabsorption)
  - Anemia
- **Skeletal** (Calcium/vitamin D malabsorption)
  - Rickets
  - Osteoporosis
  - Enamel hypoplasia of the teeth
- Respiratory
  - Idiopathic pulmonary hemosiderosis
- **Muscular** (Malnutrition)
  - Atrophy
- **Neurologic** (Thiamine/vitamin B12 deficiency)
  - Peripheral neuropathy
  - Epilepsy
  - Irritability
  - Cerebral calcifications } we don't know why
  - Cerebellar ataxia

* Some pts w/ uncontrolled seizures, and on MRI occipital calcifications were found, and are put on a gluten-free diet we find that they have very drastic improvement for the seizures.

Clinical spectrum of CD

- SYMPTOMATIC
  - With symptoms mentioned above. Htg +ve , histology +ve
- SILENT
  - No apparent symptoms in spite of histologic evidence of villous atrophy In most cases identified by serologic screening in at-risk groups

  Htg +ve , histology +ve , no signs and symptoms

  على gluten-free diet because if not they will go into complications of CD. (منعشيرهم)
- LATENT
  - Subjects who have a normal histology, but at some other time, before or after, have shown a gluten-dependent enteropathy , so these might have CD in the future (we don't put them on gluten free diet)

  Htg normal
- POTENTIAL
  - Subjects with positive celiac disease serology but without evidence of altered jejunal histology It might or might not be symptomatic

  + Htg , normal IgA

  normal histology

  *This pt has CD but it still hasn't affected the GIT

symptoms CD لحد ما يصير gluten-free diet في اشي بحكي ما نحطه على برأي الحالة
وفي اشي بحكي انه نحطه على gluten-free diet إذا symptomatic أو لا

Diagnosis of CD

Htg +ve and total IgA level
upper endoscopy and biopsy
genetics , DQ2 and DQ8

- 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.
  - Patients with positive anti-TG2 antibody levels <10 × upper limits of normal should undergo upper endoscopy with multiple biopsies.
  - 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

Antibodies :

Anti-gliadin antibodies
Anti-endomysial antibodies
Anti-Htg antibodies → the most specific and sensitive

Other causes of flat mucosa (w/ normal Htg these could be the causes)

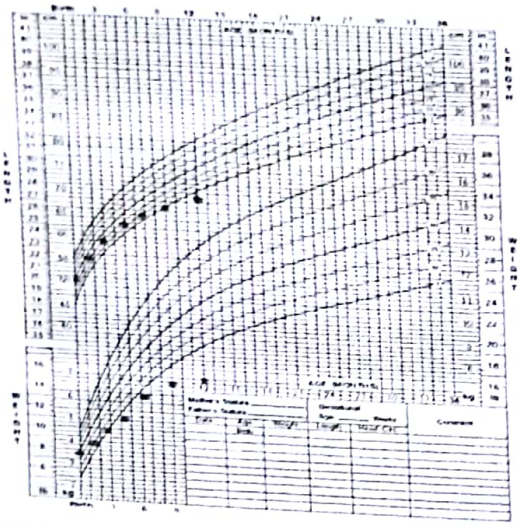
- Autoimmune enteropathy
- Tropical sprue
- Giardiasis
- HIV enteropathy
- Bacterial overgrowth
- Crohn disease
- Eosinophilic gastroenteritis
- Cow's milk enteropathy
- Soy protein enteropathy
- Primary immunodeficiency
- Graft-versus-host disease
- Chemotherapy and radiation
- Protein energy malnutrition
- Tuberculosis
- Lymphoma
- Nongluten food intolerances

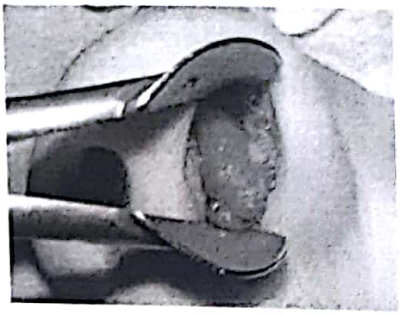
→ gluten free diet
→ high caloric intake (if FTT)
→ Vitamin supplementation

Management

- The only treatment for celiac disease is lifelong strict adherence to a gluten-free diet. This requires a wheat-, barley-, and rye-free diet.
- 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 reductionin antibody titers can be helpful as indirect evidence of adherence to a gluten-free diet

1.5 year old presented with





Cystic Fibrosis CF

- Cystic fibrosis (CF) is a major cause of pancreatic exocrine failure in children.
- Autosomal recessive disorder caused by a mutation in the CFTR gene on chromosome 7.
- Commonest mutation is Delta F508
- Up to 90% of patients with CF have loss of exocrine pancreatic function as well as inadequate digestion and absorption of fats and proteins.

CF

- Even though pulmonary disease is the major cause of morbidity and mortality, most patients (85%) have pancreatic insufficiency
- Clinical signs of pancreatic insufficiency develop when less than 10% of normal pancreatic enzyme activity is present in the duodenum.
- Patients usually present before 6 months of age with
  - failure to thrive.
  - hypoalbuminemia,
  - edema
  - anemia.

Complications of CF

- GASTROINTESTINAL
  - Meconium ileus, meconium plug (neonate)
  - Meconium peritonitis (neonate)
  - Distal intestinal obstruction syndrome (non-neonatal obstruction)
  - Rectal prolapse
  - Intussusception
  - Volvulus
  - Fibrosing colonopathy (strictures)
  - Appendicitis
  - Intestinal atresia
  - Pancreatitis
  - Biliary cirrhosis (portal hypertension: esophageal varices, hypersplenism)
  - Hepatic steatosis
  - Gastroesophageal reflux
  - Cholelithiasis
  - Inguinal hernia
  - Growth failure (malabsorption)
  - Vitamin deficiency states (vitamins A, K, E, D)
  - Insulin deficiency, symptomatic hyperglycemia, diabetes
  - Malignancy (rare)

→ due to meconium illeus and very rigid meconium, it causes perforation of the small intestines → and meconium enters peritoneum causing meconium peritonitis

Complications of CF

- RESPIRATORY
  - Bronchiectasis, bronchitis, bronchiolitis, pneumonia
  - Atelectasis
  - Hemoptysis
  - Pneumothorax
  - Nasal polyps
  - Sinusitis
  - Reactive airway disease
  - Cor pulmonale
  - Respiratory failure
  - Mucoid impaction of the bronchi
  - Allergic bronchopulmonary aspergillosis
- OTHER
  - Infertility
  - Hypochloremic hypokalemic metabolic alkalosis
  - Delayed puberty
  - Edema-hypoproteinemia
  - Dehydration–heat exhaustion
  - Hypertrophic osteoarthropathy-arthritis
  - Clubbing
  - Amyloidosis
  - Diabetes mellitus
  - Aquagenic palmoplantar keratoderma (skin wrinkling)

Diagnosis of CF

- Presence of typical clinical features (respiratory, gastrointestinal, or genitourinary)

or

- A history of CF in a sibling

or

- A positive newborn screening test

plus

- Laboratory evidence for CFTR dysfunction:
  - Two elevated sweat chloride concentrations obtained on separate days

or

  - Identification of two CF mutations

or

  - An abnormal nasal potential difference measurement

Maagement

- High caloric diet
- Pancratic enzymes replacement (Creon)
- Daily supplements of the fat-soluble vitamins.

Pancreatic Insufficiency

Shwachman-Diamond syndrome
- autosomal recessive disorder
- exocrine pancreatic failure due to fatty deposition
- skeletal abnormalities, and
- bone marrow dysfunction, primarily cyclic neutropenia.

Johanson-Blizzard syndrome is characterized by
- hypoplasia of the alae nasi
- deafness,
- imperforate anus or urogenital malformations
- dental anomalies.
- exocrine pancreatic failure due to fatty deposition

Pearson syndrome,
- deletions in mitochondrial DNA. Patients have
- pancreatic insufficiency and
- refractory sideroblastic anemia.

8 months old boy presented with

Cow's protein milk allergy

- earlier presentation
  - IgE mediated
  - non-IgE mediated
    - later presentation

- The prevalence of CMA in children living in the developed world is approximately 2 to 3 %, making it the most common cause of food allergy in the pediatric population.
- There is some cross-reactivity with soy protein, particularly in non-IgE mediated allergy.
- CMA is mostly a disease of infancy and early childhood. Affected infants present usually within the first 6 months of life, and one review reported that the majority of infants develop symptoms before 1month of age, often within 1 week after the introduction of cow's milk proteins to their diet.

Cow's protein milk allergy

- However, breastfed infants can also be affected by dairy products ingested by the mother and eliminated in her breast milk.
- The majority of affected children have one or more symptoms involving one or more organ systems, mainly the gastrointestinal tract and/or skin
- In addition to the detailed medical history and physical examination, diagnostic elimination diets, skin prick tests (SPTs), specific IgE (sIgE) measurements, and oral food challenges are part of the routine work-up

→ this type is IgE-mediated
↳ severe, earlier onset

symptoms include
- bloody diarrhea
- eczema
- can be associated with reflux

(50-90% of children) usually when children reach 1 year of age they recover → بطلع بيولوجي

95% of children at 2 yrs and 5% of children do not recover and it stays with them

Cow's protein milk allergy

- Avoidance of cow's milk protein in any form is the only available treatment.
- In the case of breastfed infants,
- Calcium supplements should be added to the mother's diet to replace milk intake .
- For infants 6 months old or younger, the recommended formulas for treatment of CMA are extensively hydrolyzed protein or amino acid-based formula

↳ so we decrease the antigenicity

hydrolyzation could be → Partial, extensive, free amino acids

→ Soy protein should NOT be given to babies less than 6 months of age because it is a plant source for estrogen

فممكن يتدخل بهرمونات الأطفال ، فخافوا يعمل males عند infertility

4 month presented with

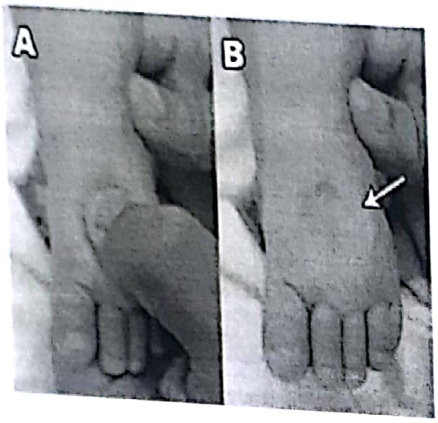


Table 1 Complete blood count (CBC) before treatment

CBC Report

Parameters	Results	Reference values
Red blood cells count (10^{12}/l)	12.2	8–18
Packed cell volume %	29	22–38
Hemoglobin Conc. (g/dl)	12.5	8–12
Mean corpuscular volume (fl)	21.3	16–25
Mean corpuscular hemoglobin (pg)	10.9	5.2–8
Mean corpuscular hemoglobin concentration (g/dl)	39	30–36
White blood cells (10^{9}/l)	10.5	4–13
Neutrophils %	66	30–48
Lymphocytes %	29	50–70
Monocytes %	03	0–4
Eosinophilia %	02	1–8
Basophils %	Nil	0–1

Protein loosing enteropathy

- Protein-losing enteropathy (PLE) is a rare condition characterized by protein loss through the gastrointestinal tract, leading to reduced serum protein levels, mainly albumin.
- Main laboratory findings are reduced serum concentration of albumin, gammaglobulins, and ceruloplasmin. Diminished oncotic pressure due to hypoalbuminemia may lead not only to edema, but also to ascites and pleural or pericardial effusions. PLE can also be associated with fat malabsorption and deficiencies of fat-soluble vitamins due to small bowel involvement

TABLE.

Causes of protein-losing enteropathy in children

Infectious	CMV, *Helicobacter pylori*, *Clostridium difficile*, *Giardia lamblia*, measles, bacterial overgrowth
Noninfectious	Inflammatory bowel disease, celiac disease, Ménétrier's disease, allergic gastroenteropathy, eosinophilic gastroenteritis, Henoch-Schonlein purpura, system lupus erythematosus
Metabolic	Congenital enterocyte heparin sulphate deficiency, congenital disorders of glycosylation
Lymphatic obstruction	Thoracic duct damage, intestinal lymphangiectasia
Cardiac	Heart failure, pericarditis, post-Fontan procedure
Others	Post-chemotherapy, graft-versus-host disease

Source: Mohanty PH, Karjoo M, Beg M. Reprinted with permission.

pericarditis → GIT بال lymph الـ stagnation بعمل

post-Fontan procedure → as a side effect of the procedure (lympatic drainage الـ بتسكر)

Post-chemotherapy → and radiotherapy, cause obstruction and destruction to the lymphatic system.

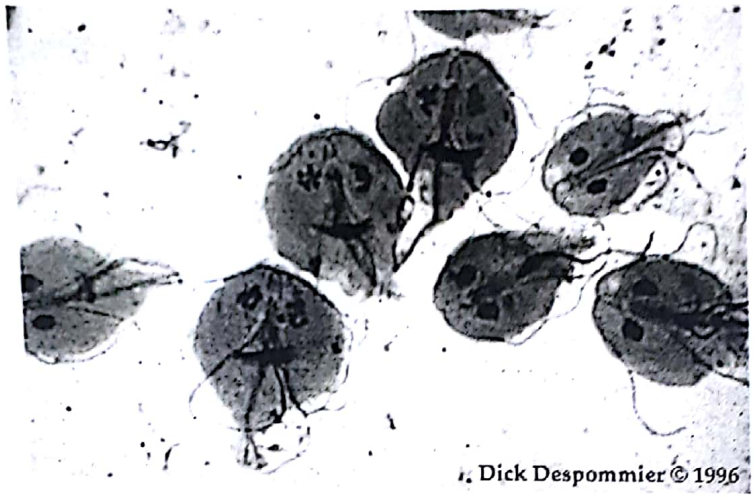
Intestinal lymphangiectasia

- Intestinal lymphangiectasia is an uncommon disorder andan important cause of protein-losing enteropathy.
- The major symptoms were edema and hypoproteinemia, low serum albumin and gammaglobulin levels.
- Biopsies of the small intestine showed variable degrees of dilatation of lymph vessels in the mucosa and submucosa
- Treatment of PIL consists of lifelong dietary modification with high protein and low fat substituted with MCT

MCT → medium chain triglyceride (they get into the circulation by passive diffusion)
→ so they don't lymph to get absorbed like short and long chain fatty acids do.

*MCT don't stimulate more lymph into the GIT → so it decreases lymph loss via the GIT

A 4 year old girl presented with diarrhea for 20 days; stool cx showed this parasite that lives in swimming pools



Giardia lamblia

- Giardia lamblia is a flagellated protozoan that is a major cause of diarrhea, especially in patients who travel to endemic areas.
- The life cycle consists of 2 stages: the trophozoite (motile form), and the cyst.
- IgA deficiency and hypogammaglobulinemia predispose patients to symptomatic infection.
- The clinical manifestations are foul-smelling diarrhea, with nausea, anorexia, abdominal cramps, bloating, belching, flatulence, and weight loss. Abdominal distention and cramps can last for weeks to months. تطول
- The illness is usually self-limited, lasting 2 to 6 weeks, but may become chronic.

Giardia lamblia

- Chronic symptoms can include fatigue, nervousness, weight loss, steatorrhea, lactose intolerance, and growth retardation.
- The easiest way to diagnose Giardia is by identifying cysts in a stool specimen. However, these specimens are frequently falsely negative. The diagnosis can also be made by antigen detection tests, endoscopic examination of the upper small intestine, by mucosal biopsy or by collection of jejunal contents.
- The treatment of choice for both symptomatic and asymptomatic patients is furazolidone or metronidazole. An alternative drug is quinacrine

Glucose-Galactose malabsorption

- Autosomal recessive
- Neonatal presentation
- Diarrhea persistes on breast feeding as well as on lactose free infant formula, he was admitted so far 3 times with
- Hyper-natremic dehydration is often present
- Glucose/galactose free diet, fructose is well absorped (fructose based formula)
- Intestinal adaptation to glucose and galactose with age

* حدول لما نخطرم NPO بتوقف الـ diarrhea ، وانه بطل في حليب فما في سكر بيطلع diarrhea.

فوقتيا بفكر بـ Cow's protein allergy ← و بتكون عادة IgE-mediated كونه الـ presentation بهي الـ neonate

أو Carbohydrate malabs... فبعمل reducing substances in stool ← test for إذا +ve معناته عنده carbohydrate malabsorption
↳ and the most common is glucose-galactose