L: 27
Diarrhea and Malabsorption

objectives

1. To recognize the definition and different classifications of chronic diarrhea.

2. To understand the mechanism of chronic diarrhea.

3. To learn systematic approach of patient with chronic diarrhea.

4. To understand the different mechanisms and causes of malabsorption.

5. To be able to recognize the clinical manifestation of malabsorption and approach to patient with malabsorption.

Color index: Step up to medicine , slide , Doctor's note , Davidson , Extra Explanation

Definitions

1- Diarrhea: decrease in fecal consistency (weight of stool and frequency are not reliable)
Chronic diarrhea: > 4 weeks
Diarrhea is a symptom, not a disease and may occur in many different conditions.

2- Malabsorption: abnormality in absorption of food nutrients across the gastrointestinal (GI) tract.

3- Acute diarrhea is a common and usually transient, self- limited, Infection related

4 - Chromic diarrhea: usually requires work up, non-infectious cause.

Acute diarrhea is usually due to infection (virus, bacteria, and parasite) or medications.
• If nausea and vomiting are present, suspect viral gastroenteritis or food poisoning.
• If food poisoning is the cause, diarrhea appears within hours of the meal.
• Remember that occult blood in the stool may be present in all types of acute infectious diarrhea, but it is much less common to have gross blood.
• A finding of fever and blood together is typical of infection with Shigella, Campylobacter, Salmonella (may also be without blood), enterohemorrhagic Escherichia coli.
• No fever and no blood is typical of infection with viruses (rotavirus, Norwalk virus), enterotoxic E. coli, and food poisoning (Staphylococcus aureus, Clostridium perfringens)

Diarrhea
General characteristics

- Most cases of diarrhea are :<u>acute</u> , <u>benign</u> and <u>self-limited</u>.
- Some cases are chronic and may be associated with underlying disease.
- Acute diarrhea is diarrhea that lasts less than 2 to 3 weeks while chronic diarrhea lasts
more than 4 weeks.
- Most common cause of acute diarrhea is viral infection (rotavirus and the Norwalk virus are the most common).
- Most severe forms of acute diarrhea are due to bacterial infections (*Shigella , Escherichia coli, Salmonella, Campylobacter, Clostridium perfringens, Clostridium difficile).*
- Protozoa that may cause diarrhea Include: *Giardia lamblia, Entamoeba histolytica, and Cryptosporidium*.
- Elderly and immunocompromised patients (e.g., with HIV, transplantation patients) are vulnerable to diarrheal illnesses due to impaired immunity.
- In patients with HIV, diarrhea can be caused by Mycobacterium aviumintracellulare,Cryptosporidium, Cyclospora, or CMV.

Causes of diarrhea

First :Acute diarrhea. Caused by :

1- Infection:	2-Medications:	3-Malabsorption	4- Ischemic bowel	5-Intestinal tumors
viruses most common (viral gastroenteritis), followed by bacteria, then parasites.	Antibiotics (most common cause)— Antibiotic-associated diarrhea is caused by _C. difficile_ toxin in 25% of cases. Others include: laxatives, prokinetic agents (cisapride), antacids, digitalis, colchicine, antibiotics, alcohol, magnesium-containing antacids,and chemotherapeutic Agents.	(e.g., lactose intolerance)	in elderly patients with history of PVD and bloody diarrhea, along with abdominal pain	(very rare)

Important Parts of the History in a Patient with Diarrhea
• Is the stool bloody or melanotic?
• Are there any other symptoms (e.g., fever, abdominal pain, vomiting)?
• Is there anyone in the family or group with a similar illness?
• Has there been any recent travel outside the United States, or any hiking trips? (parasitic infections)
• Are symptoms linked to ingestion of certain foods (e.g., milk)?
• Are there any medical problems (e.g., AIDS, hyperthyroidism)?
• Have there been recent changes in medications (e.g., antibiotics within the past few weeks)?

Causes of diarrhea

Second :chronic diarrhea. Caused by:

1- IBS	**(most common cause, but is a diagnosis of exclusion)**
2- Inflammatory bowel disease (IBD)	
3-Medications	Like in acute
4- Infection	bacterial enterocolitis (Shigella, Salmonella, Campylobacter, enteroinvasive E. coli)
5- Colon cancer	
6-Diverticulitis	
7-Malabsorption syndromes	pancreatic insufficiency, celiac disease, short bowel syndrome, ischemic bowel, bacterial overgrowth
8-Postsurgical	(e.g., gastrectomy, vagotomy)
9-Endocrine causes	(hyperthyroidism,Addison's disease, diabetes,gastrinoma,VIPoma).
10-Fecal impaction	because only liquid stool can pass around the impaction. (factitious diarrhea).
11-Laxative abuse	
12-Immunocompromised patients	with acute infectious diarrhea.

History and physical examination

Main Symptom	Consistency or frequency of stools, the presence of urgency or fecal soiling)
Duration	Acute or chronic (weeks)
Stool characteristics	Greasy stools that float and are malodorous may suggest fat malabsorption while the presence of visible blood may suggest inflammatory bowel disease
Volume of the diarrhea	voluminous watery diarrhea is more likely to be due to a disorder in the small bowel while small-volume frequent diarrhea is more likely to be due to disorders of the colon
Specific food	Fatty , diary, allergy to certain food
Change with fasting or night	Secretory , IBS
Specific food	Fatty , diary, allergy to certain food
History of travel	Travelers diarrhea, parasitic etc…
Associated symptoms	ABD PAIN, Weight loss, appetite change Extra-intestinal manifestation (rash, arthritis. Mouth ulcers etc..)
Drug hx	Antibiotic use, others
F Hx	Of GI illnesses

History and physical examination cont.

Physical examination:

➢Rarely provides a specific diagnosis.

➢Findings suggestive of IBD (eg, mouth ulcers, a skin rash, episcleritis, an anal fissure or fistula, the presence of visible or occult blood on digital examination, abdominal masses or abdominal pain)

➢Evidence of malabsorption (wasting, physical signs of anemia, scars indicating prior abdominal surgery)

➢Lymphadenopathy (possibly suggesting, lymphoma, HIV infection)

➢Palpation of the thyroid and examination for exophthalmos and lid retraction may provide support for a diagnosis of hyperthyroidism

Specific investigations:
The history and physical examination may point toward a specific diagnosis for which testing may be indicated

Mechanism of Diarrhea

- **Change in:**
  - Absorption.
  - Secretion.
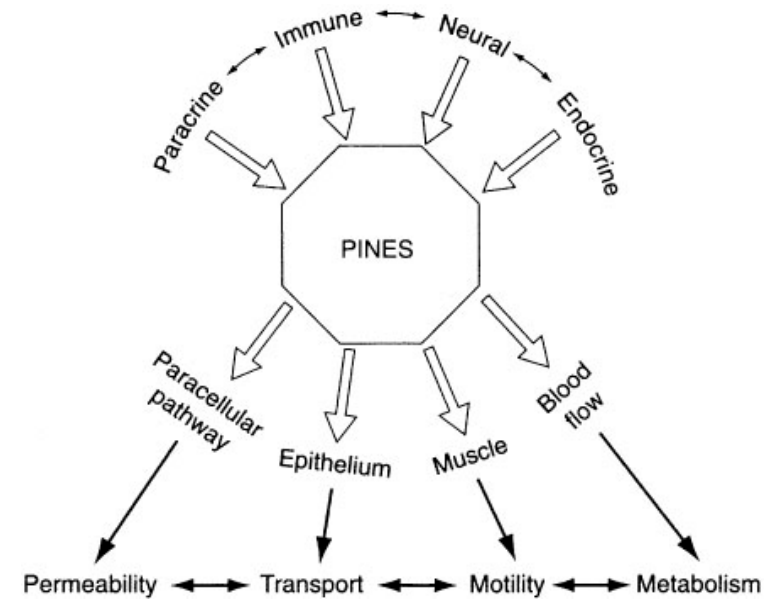 - Motility of the gut.

  in response to various etiology.

There two Classifications of Diarrhea:
1/
➢Time course (acute vs. chronic).
➢Volume (large vs. small).
➢Pathophysiology (secretory vs. osmotic).
➢Stool characteristics (watery vs. fatty vs. inflammatory).
➢Epidemiology (epidemic vs. travel-related vs. immunosuppression-related).

2/
✓Fatty diarrhea
✓Inflammatory diarrhea

Classification of Diarrhea:

➢ *According to mechanism:*

	Osmotic Diarrhea	Secretory Diarrhea	Motility Diarrhea
DESCRIPTION	Non – absorbable solute pulls excess water into intestine tract.	Intestinal wall is damaged resulting in increased secretion rather than absorption of electrolytes into intestinal tract.	Motility disorder results in decreased contact time of fecal mass with intestinal wall so decrease water absorption from feces.
EXAMPLE	Lactase deficiency		Motility disorders: •IBS •Gastric/intestinal resection.

Diagnosis

1-Laboratory tests are usually unnecessary in acute diarrhea.

2. <u>Some indications for diagnostic studies:</u>

a. Chronic diarrhea or diarrhea that is prolonged

b. Severe illness or high fever

c. Presence of blood in the stool/high suspicion for IBD

d. Severe abdominal pain

e. Immunodeficiency

f. Signs of volume depletion

3. <u>Laboratory tests to order</u>

a. CBC :look for anemia, WBC elevation.

b. Stool sample -for presence of fecal leukocytes:

- If fecal leukocytes are absent, there is no need to order stool cultures because they are unlikely to grow pathogenic organisms (unless invasive bacterial enteritis is suspected or the patient has bloody diarrhea).

- If fecal leukocytes are present and the patient has moderate to severe diarrhea

send stool for culture <u>or</u> C. difficile toxin assay <u>or</u> treat empirically with antibiotic.

-Fecal leukocytes are present in *Campylobacter, Salmonella, Shigella, enteroinvasiveE. Coli infection, and C. difficile which is absent in staphylococcal or clostridial* food poisoning; and absent in viral gastroenteritis.

> ❗ Assess volume status (dehydration is a concern), perform an abdominal examination, and check stool for occult blood in patients with diarrhea. In mild to moderate cases of acute diarrhea, further workup is unnecessary.

Diagnosis

c. Stool sample— Test three samples for presence of ova and parasites. Order this

if a parasite is suspected. For *Giardia*, order <u>enzyme-linked immunosorbent assay test</u> for antigen.

d. Bacterial stool culture:

• This has low sensitivity (and is an expensive test), and usually does not affect treatment or outcome.

• It should not be ordered routinely. <u>Some indications for bacterial stool culture include</u>:

1- if invasive bacterial enteritis is suspected. 2- if the patient has moderate to severe illness or fever.

3-if the patient requires hospitalization 4-if the stool sample is positive for fecal leukocytes.

• Most laboratory tests examine stool culture for only three organisms: *Shigella, Salmonella, and Campylobacter*.

e. Stool sample—Measure for *C. difficile* toxin if the patient has been treated with antibiotics recently. Note that this test has a false-negative rate of 10%. Treat the patient empirically even before laboratory results are back if the suspicion is high.

f. Colonoscopy/flexible sigmoidoscopy—may be considered for patients with blood in the stool or for patients with chronic diarrhea for which a cause cannot be identified.

Laboratory tests to consider:
• Stool WBCs
• Stool for ova and parasites
• Stool culture
• Stool for *C. difficile* culture
• Stool for *C. difficile* toxin assay
• Stool for *Giardia* antigen

Salmonella, Campylobacter, Shigella, and enteroinvasive E. coli cause diarrhea with fecal leukocytes and often blood.

The most common electrolyte/acid–base abnormality seen with severe diarrhea is **metabolic acidosis and hypokalemia.**

Treatment

1- Acute diarrhea :is typically self-limited and does not require hospitalization.

However, <u>consider hospitalization for any of the following reasons</u>:

<u>A-Dehydration</u> (especially in elderly patients)

 B- Patients initially <u>unable to tolerate or hold down PO fluids</u> .

 <u>C-Bloody diarrhea</u> (with profuse or brisk bleeding)

 D- <u>High fever, toxic appearance</u>.

2-The identification of the specific agent responsible for acute infectious diarrhea is not critical with regard to treatment. Treat the diarrhea <u>according to the patient's medical history and clinical condition</u>.

3. Specific therapy:

a. <u>Rehydrate;</u> monitor electrolytes and replace if necessary

b. <u>Treat the underlying cause</u> (e.g., stop or change medication, advise a lactosefree diet). Consider a trial of NPO status to see if diarrhea stops.
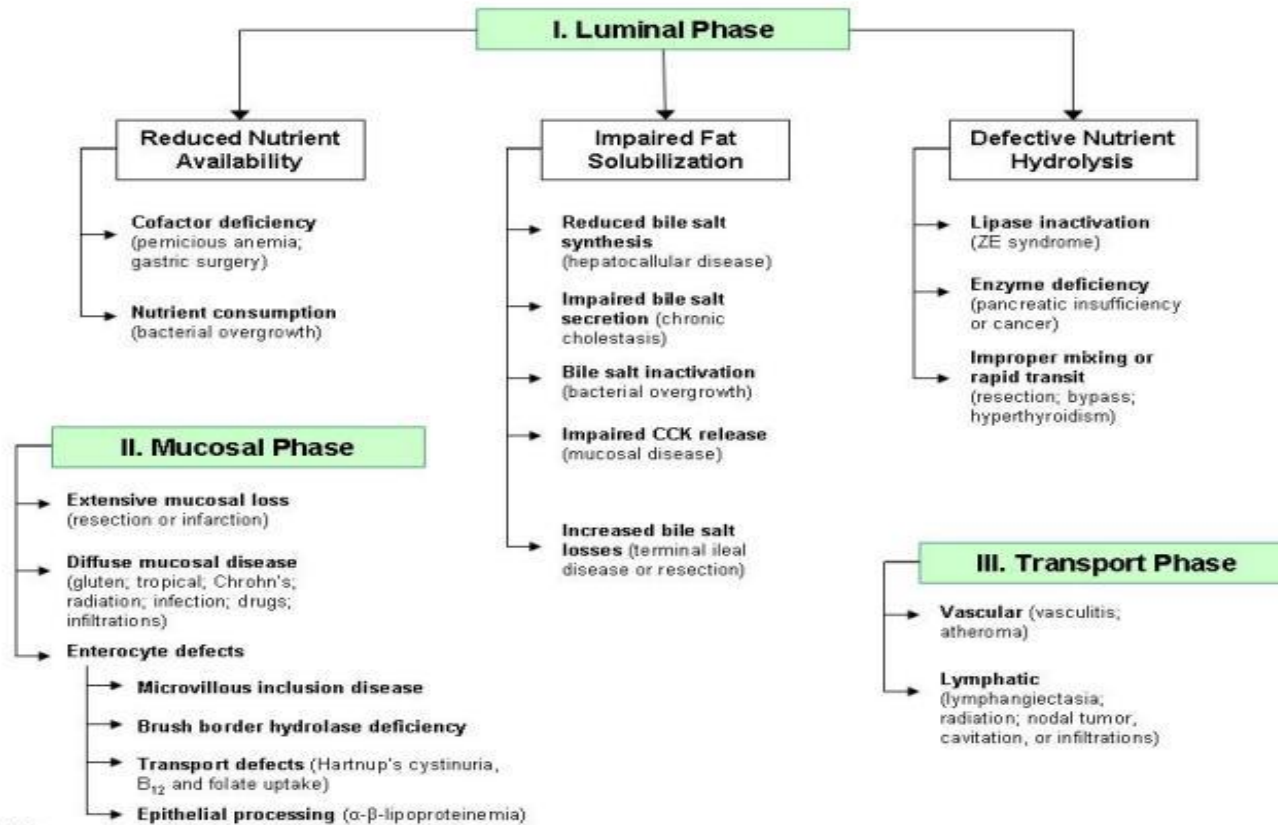
c. <u>Consider antibiotics</u>. Use of antibiotics in **infectious diarrhea has been shown** to decrease the duration of illness by 24 hours (regardless of the etiologic agent). Therefore, consider a 5-day course of <u>ciprofloxacin</u> in patients who have moderate to severe disease. Antibiotics are definitely recommended in the following situations:

• Patient has high fever, bloody stools, or severe diarrhea— quinolones are ppropriate

• Stool culture grows one of the pathogenic organisms

• Patient has traveler's diarrhea.

• C. difficile infection—metronidazole.

4. Loperamide (Imodium) :is an antidiarrheal agent that should only be given if diarrhea is mild to moderate and is not recommended in patients with <u>fever</u> or with <u>blood in their stool</u>.

(The second part of the lecture) Malabsorption

I. Luminal Phase

Reduced Nutrient Availability
- Cofactor deficiency (pernicious anemia; gastric surgery)
- Nutrient consumption (bacterial overgrowth)

Impaired Fat Solubilization
- Reduced bile salt synthesis (hepatocallular disease)
- Impaired bile salt secretion (chronic cholestasis)
- Bile salt inactivation (bacterial overgrowth)
- Impaired CCK release (mucosal disease)
- Increased bile salt losses (terminal ileal disease or resection)

Defective Nutrient Hydrolysis
- Lipase inactivation (ZE syndrome)
- Enzyme deficiency (pancreatic insufficiency or cancer)
- Improper mixing or rapid transit (resection; bypass; hyperthyroidism)

II. Mucosal Phase
- Extensive mucosal loss (resection or infarction)
- Diffuse mucosal disease (gluten; tropical; Chrohn's; radiation; infection; drugs; infiltrations)
- Enterocyte defects
  - Microvillous inclusion disease
  - Brush border hydrolase deficiency
  - Transport defects (Hartnup's cystinuria, B_{12} and folate uptake)
  - Epithelial processing (α-β-lipoproteinemia)

III. Transport Phase
- Vascular (vasculitis; atheroma)
- Lymphatic (lymphangiectasia; radiation; nodal tumor, cavitation, or infiltrations)

M.N.

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Malabsorption results from abnormalities of the three processes which are essential to normal digestion:

1. Intraluminal maldigestion occurs when deficiency of bile or pancreatic enzymes results in inadequate solubilisation and hydrolysis of nutrients. Fat and protein malabsorption results. This may also occur with small bowel bacterial overgrowth.

2. Mucosal malabsorption results from small bowel resection or conditions which damage the small intestinal epithelium, thereby diminishing the surface area for absorption and depleting brush border enzyme activity.

3. 'Post-mucosal' lymphatic obstruction prevents the uptake and transport of absorbed lipids into lymphatic vessels. Increased pressure in these vessels results in leakage into the intestinal lumen, leading to protein-losing enteropathy.

Clinical features

- Depend upon the **cause** and **severity** of the disease
- Malabsorption may either be **global** or **partial (isolated).**

☐ **Global malabsorption:** results from diseases associated with either diffuse mucosal involvement or a reduced absorptive surface
  – An example is **celiac disease** in which diffuse mucosal disease can lead to impaired absorption of almost all nutrients

☐ **Partial or isolated malabsorption:** results from diseases that interfere with the absorption of specific nutrients.
  – Defective cobalamin absorption, for example, can be seen in patients with pernicious anemia or those with disease (or resection) of the terminal ileum such as patients with Crohn's disease.

Global malabsorption: more than one nutrient
Partial malabsorption: just one nutrient component

Signs and symptoms

Malabsorption of	Clinical features	Laboratory findings
1-Calories	Weight loss with normal appetite	
2- Fat	Pale and voluminous stool, diarrhea without flatulence, steatorrhea	Stool fat >6 g/day
3- Protein	Edema, muscle atrophy, amenorrhea	Hypoalbuminemia, hypoproteinemia
4- Carbohydrates	Watery diarrhea, flatulence, acidic stool pH, milk intolerance, stool osmotic gap	Increased breath hydrogen
5- Vitamin B12	Anemia, subacute combined degeneration of the spinal cord (early symptoms are paresthesias and ataxia associated with loss of vibration and position sense)	Macrocytic anemia, vitamin B12 decreased, abnormal Schilling test, serum methylmalonic acid and homocysteine increased

Malabsorption of	Clinical features	Laboratory findings
6- Folic acid	Anemia	Macrocytic anemia, serum and RBC folate decreased, serum homocysteine increased
7- Vitamin B, general	Cheilosis, painless glossitis, acrodermatitis, angular stomatitis	
8- Iron	Microcytic anemia, glossitis, pagophagia	Serum iron and ferritin decreased, total iron binding capacity increased
9- Calcium and vitamin D	Paresthesia, tetany, pathologic fractures due to osteomalacia, positive Chvostek and Trousseau signs	Hypocalcemia, serum alkaline phosphatase increased, abnormal bone densitometry
10- Vitamin A	Follicular hyperkeratosis, night blindness	Serum retinol decreased
11- Vitamin K	Hematoma, bleeding disorders	Prolonged prothrombin time, vitamin K-dependent coagulation factors decreased

Signs and symptoms

From davidson's

Lethargy
Depression

Distension, steatorrhoea,
watery diarrhoea

Poor wound healing
(vitamin C, protein, zinc)

Purpura and bruising
(vitamins C, K)

Night blindness
(vitamin A)
Anaemia
(iron, folate, B_{12})

Angular stomatitis, glossitis
(iron, folate, B_{12})
Bleeding gums
(vitamin C)

Follicular
hyperkeratosis
(vitamin A)

Acrodermatitis enteropathica
(zinc)
Koilonychia
(iron)
Paraesthesia, tetany
(calcium, magnesium)
Clubbing

Osteomalacia, rickets
(calcium, vitamin D)

Muscle-wasting
(protein)
Proximal myopathy
(vitamin D)

Peripheral neuropathy
(B_{12})
Peripheral oedema
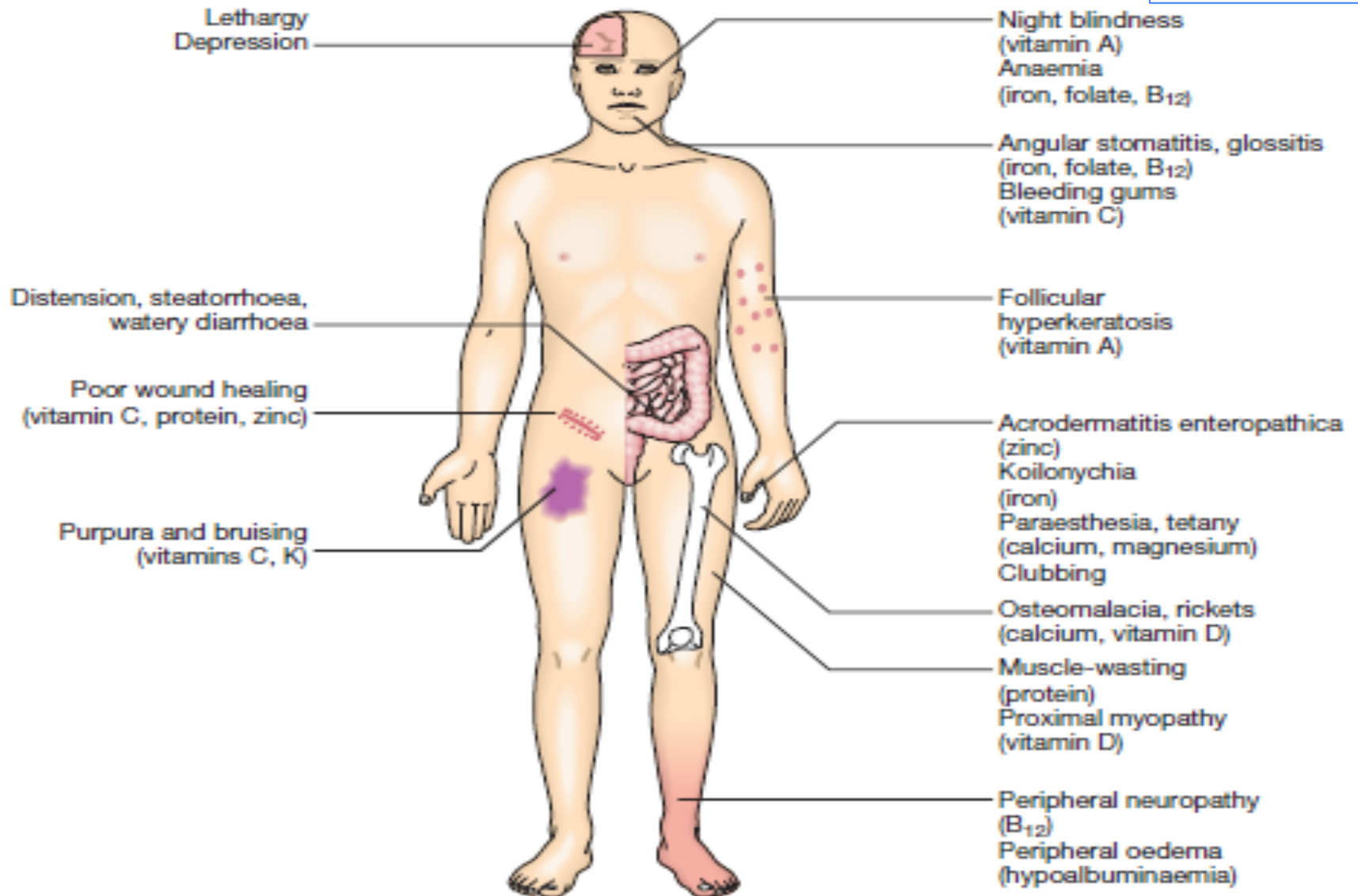(hypoalbuminaemia)

Fig. 22.21 Possible physical consequences of malabsorption.

Examples of Manifestations associated with celiac disease

Dermatological manifestations
Pale skin

Neurological examination
Motor weakness, peripheral neuropathy, or ataxia may be present.
The Chvosteks sign or the Trousseau sign may be evident due to hypocalcemia .

Investigations for malabsorption

✓ Investigations for nutrients deficiency.

✓ Specific investigations to define the etiology and severity.

✓ Common lab: CBC , ESR, CRP, PT, INR, LFT.

✓ Serology: Anti TTG (anti endomysial ab). *anti tissue transglutaminase* test for celiac disease

✓ Endoscopy. next slide
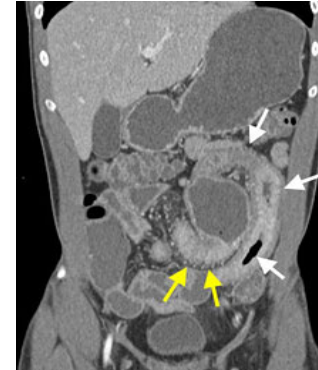
✓ Imaging:

X-ray

A- Small bowel barium studies .
Strictures
Mucosal changes.
Diverticula.

B- CT scan of the abdomen
Strictures.
Mucosal changes.
Diverticula.
Wall thickness.
Massesss, lymph nodes.

C- ERCP: pancreatitis (duct changes) ,biliary doseases.

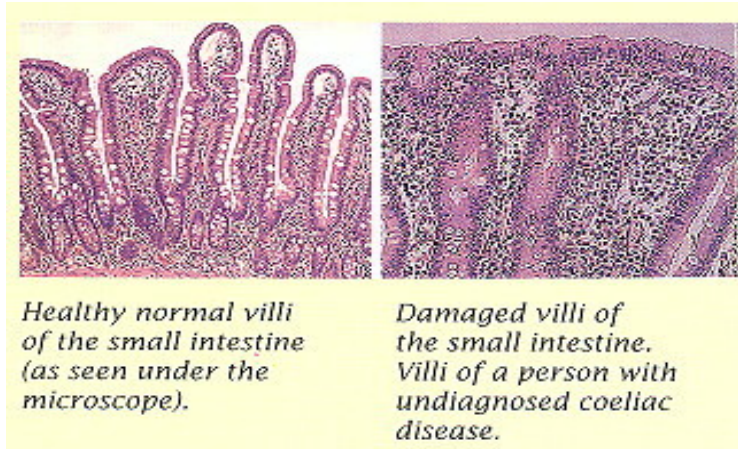D- Plain abdominal x-ray film: Pancreatic calcifications are indicative of chronic pancreatitis.

!

•No specific serology tests for all causes of malabsorption

•Serum Anti-TTG and antiendomysial antibodies can be used to help diagnose celiac sprue.

•Determination of fecal elastase and chymotrypsin can be used to try to distinguish between pancreatic causes and intestinal causes of malabsorption.

Investigations for malabsorption

Endoscopy:

❖ **Upper endoscopy with small bowel mucosal biopsy…. Examples**

- Celiac sprue
- Giardiasis
- Crohn disease
- Whipple disease
- Amyloidosis
- Lymphoma.

Healthy normal villi of the small intestine (as seen under the microscope).

Damaged villi of the small intestine. Villi of a person with undiagnosed coeliac disease.

❖ **Lower GI endoscopy: colonic and terminal ileal pathology (e.g Chrons disease)**

Routine blood test abnormalities in malabsorption

Haematology
- Microcytic anaemia (iron deficiency)
- Macrocytic anaemia (folate or B12 deficiency)
- Increased prothrombin time (vitamin K deficiency)

Biochemistry
- Hypoalbuminaemia
- Hypocalcaemia (p. 768)
- Hypomagnesaemia
- Hypophosphataemia
- Low serum zinc

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? Malabsorption

Clinical features of steatorrhoea
- Blood tests (urea and electrolytes, immunoglobulins, Ca^{++}, Mg^{++}, full blood count, clotting, albumin, folate, B_{12}, coeliac antibodies)

Investigate small intestine
- Duodenal biopsy
- Barium studies or small bowel MRI
- Faecal calprotectin
- Lactulose/glucose hydrogen breath test

Normal

Investigate pancreas
- Pancreatic function tests, e.g. faecal elastase
- Ultrasound scan/CT
- MRCP

Normal

Consider bile salt malabsorption
- SeHCAT scan
- Serum 7α-hydroxycholestenone

Fig. 22.22 Investigation for suspected malabsorption.

Treatment

A- Treatment of causative diseases :

❖A gluten-free diet helps treat celiac disease.

❖Similarly, a lactose-free diet

❖Protease and lipase supplements are the therapy for pancreatic insufficiency.

❖Antibiotics are the therapy for bacterial overgrowth.

❖Corticosteroids, anti-inflammatory agents, such as mesalamine, and other therapies are used to treat CD.

B- Nutritional support:

❖Supplementing various minerals calcium, magnesium, iron, and vitamins

❖Caloric and protein replacement also is essential.

❖Medium-chain triglycerides can be used for lymphatic obstruction .

❖In severe intestinal disease, such as massive resection and extensive regional enteritis, parenteral nutrition may become necessary.

From davidson's

22.21 Chronic or relapsing diarrhoea

	Colonic	Malabsorption	Small bowel
Clinical features	Blood and mucus in stool Cramping lower abdominal pain	Steatorrhoea Undigested food in the stool Weight loss and nutritional disturbances	Large-volume, watery stool Abdominal bloating Cramping mid-abdominal pain

Some causes	Inflammatory bowel disease
	Microscopic colitis
	Neoplasia
	Ischaemia
	Irritable bowel syndrome
		Pancreatic
		  Chronic pancreatitis
		  Cancer of pancreas
		  Cystic fibrosis
		Enteropathy
		  Coeliac disease
		  Tropical sprue
		  Lymphoma
		Lymphangiectasia
			Crohn's disease
			VIPoma
			Drug-induced
			  NSAIDs
			  Aminosalicylates
			  Selective serotonin re-uptake
			  inhibitors (SSRIs)

Investigations	Faecal calprotectin Ileocolonoscopy with biopsies	Faecal elastase Ultrasound, CT and MRCP Small-bowel biopsy Barium follow-through or small-bowel MRI	Faecal calprotectin Stool volume Gut hormone profile Barium follow-through or small-bowel MRI

Doctor's cases

- Sarah is 22 Y F c/o non bloody diarrhea for 7 months
- 6-10 times/ Day
- Mild LIF pain
- Weight loss 10 kg (49 kg -> 39)
- O/E cachexia, LIF tenderness (mild)
- Lab (low hb 8 ,low mcv) High ESR and CRP **What is the most likely next STEP?** Crohn's disease

- Ahmed is 18 years old with fatigue, abdominal bloating and diarrhea for 3 months. Itchy skin rash
- Unremarkable physical examination, except pallor and skin rash over thighs
- Has low Hb (low mcv)
- Serology : anti TTG ab positive
- **Most likely diagnosis and second step?** celiac disease

- Nada is 27 years old F, with intermittent diffuse abd pain and diarrhea for 3 years.
- No blood in stool
- Diarrhea is triggered by fatty food, no nocturnal diarrhea
- Symptoms improved with defecation.
- Symptoms free for weeks
- Weight and appetite stable
- Examination: N
- CBC, ESR: N
- **Most apropriate next step and diagnosis?** IBS

1. Chronic diarrhea results in the following acid base imbalance:

A. Normal anion gap metabolic acidosis

B. Metabolic alkalosis

C. Increased anion gap metabolic acidosis

D. Respiratory acidosis

2. A patient presents with chronic small bowel diarrhea, duodenal biopsy shows villous atrophy. Anti endomysial antibodies and IgA TTG antibodies are positive. What is the treatment of choice?

A. Gluten free diet

B. Antibiotics

C. Loperamide

D. 5—ASA

3. Diarrhea with visible blood in stools is called _____ .

A. Diphtheria

B. Dysentry

C. Chronic diarrhea

D. Hemorrhoids

4. A 30 y/o Irish woman is referred to you for evaluation of diarrhea (3-4 loose bowel movements/day) of four months duration with a wt loss of 5 kg. She has also noted easy bruiseability and fatigability. Milk products cause abdominal bloating and cramps. Beer causes increased diarrhea. She does have two cats at home. Physical examination reveals pallor. The pulmonary, cardiac and abdominal examinations are normal. Rectal examination reveals hemoccult negative stool.

Laboratory Findings:

Hct/hgb   9/26

MCV   69 microns

Platelets   200,000

AST/ALT   60/68

Albumin/Globulin   32/30 gm/L

Prothrombin time   16 second (INR = 1.6)

ESR   20

C-Reactive Protein   3.0

Serum Carotenes   25

Serum Calcium   7.0 mg%

Serum Magnesium   1.6 mg%

Stool fat   3(+) split fat

The Most likely diagnosis is:

A. Regional Enteritis
B. Lactose Intolerance
C. Pancreatic Exocrine Insufficiency
D. Camphylobacter enteritis

5. This type of diarrhea occurs when a stimulating substance either increases secretion or decreases absorption of water and electrolytes.

A)Osmotic

B) Exudative

C) Secretory

D) Transitory

E) Hydrostatic

6. Which of the following drugs or measures are not advocated for the prevention of traveler's diarrhea?

A) special care with drinking water

B) Bismuth subsalicylate (BSS)

C) special care with fresh vegetables

D) avoidance of meat products

E) antibiotic prophylaxis

7. If diarrhea occurs, therapeutic goals include all the following except

A. prevent excessive water and electrolyte loss

B. provide symptomatic relief

C. manage the diet

D. treat curable causes.

E. Stop the diarrhea at all costs.

8. Which of the following antisecretory agents used to treat diarrhea may interact with anticoagulants, interfere with tetracycline absorption, and interfere with some gastrointestinal radiographic studies?

A)Polycarbophil
B)Bismuth subsalicylate
C)Loperamide
D)Paregoric
E)Diphenoxylate with atropine

Answers : 1-C 2-A   3-B   4-B   5-C 6-D   7-E   8-B

DONE BY

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Medicine is a science of uncertainty and an art of probability