

Disease of the esophagus :-

انتبه على
الاعراض التنوير
كل مرض

1- obstruction

mechanical

Congenital

• **Atresia**: Thin, non colonized

Cord

- near oral the tracheal bifurcation

+/- fistula: abnormal passage

between 2 hollow

• **Clinical**: regurgitation

• Rejoin

• **Complication (with fistula)**

- Aspiration

- suffocation

- Pneumonia

- fluid imbalance

Acquired

• **Stenosis**: fibrous thickening of submucosa + atrophy of muscularis propria

• **Causes**: GERD, Irradiation, caustic agents

• **Clinical**: dysphagia, difficulty eating solids → liquids

Functional

• **discoordinated Peristalsis or Spasm**

Achalasia

Traid

incomplete LES relaxation

increased LES tone

aperistalsis



primary

most common

Secondary

• Failure of **distal** esophageal inhibitory neurons

• Idiopathic

• **Clinical**: X Swallowing

• Regurgitation

• Chest Pain

• degenerative in neural innervation

- Intrinsic
- Vagus nerve

• Chagas disease

2- Vascular (Varices)

- dilated veins within the submucosa of distal esophagus and proximal stomach

• **Diagnosis**: endoscopy or angiography



• **Portal hypertension**

- Cause: - Cirrhosis, Alcoholic liver, hepatic schistosomiasis 2nd

• **Clinical**: often asymptomatic

- massive hematemesis and death

3- Esophagitis:-

1) **Lacerations**: Tear from vomiting

linear, cross GEJ
superficial,
heat injury

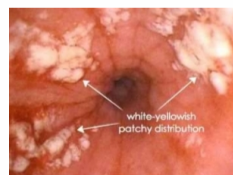
2) **Chemical**: damage by irritants

Clinical: odynophagia, hemorrhage

3- **Infectious**:

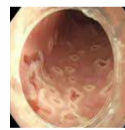
- **Candidiasis**: Adherent

, Gray-white



Pseudomembranes

- **Herpes**: Punched-out ulcers



• Multinucleated + giant cells

- **CMV**: Shallower ulcerations

4- **Reflux**: Lower esophagus

decreased sphincter tone
↑ abdominal pressure



Eosinophils → neutrophils → basal zone → elongation

Complications: melena, strictures

heartburn
dysphagia
Tx: PPT

5- **Eosinophilic**: chronic immune mediated disorder.

• upper and mid esophagus

• ↑ eosinophils

Tx: diet xpls
corticosteroids



4- Esophageal Tumors

AdenoCarcinoma

• background (Barrett esophagus)

Male >>

- genetic and epigenetic
- Chromosomal abnormalities and TP53 mutation

Distal third {
 early: Flat
 later: mass



Clinical: Pain, weight loss, vomiting

5y {
 early 80%
 advanced 25%

Squamous cell Carcinoma

• Male >> Alcohol, Tobacco

• Morphology:

Polypoid, ulcerated or infiltrative

• Wall thickening, lumen narrowing

• invade surrounding structure



Clinical:-

- dysphagia
- odynophagia
- weight loss

5y < 9%

• Barrett

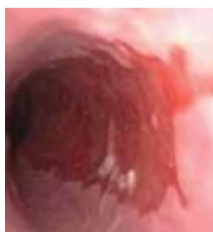
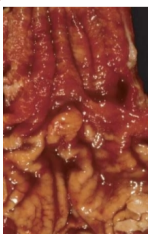
Esophagus:

Complication of chronic GERD

• metaplasia >> dysplasia >> adenocarcinoma

• Red tongues upward GEJ

• goblet cells



• lymph node

metastases:

- upper 1/3 : Cervical

- Middle 1/3 : mediastinal paratracheal + tracheobronchial

Lower 1/3 : gastric + celiac

* pathology of stomach :-

A-inflammatory Conditions

Gastropathy:
no inflammation

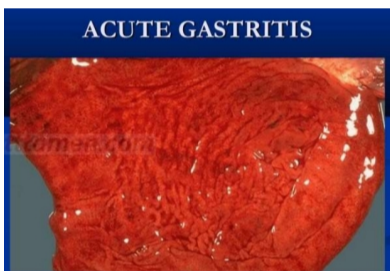
1. Acute gastritis:-

- mucosal injury
- Asymptomatic or epigastric Pain, nausea
- Vomiting
- imbalance bt protection and damage:

- Main Causes**
- Chemotherapy
 - NSAIDs, Alcoholic
 - H. Pylori
 - uremic P
 - elders
 - Hypoxia

• PGE1 + I2

Stimulate all defense mechanisms.



Morphology:

- Hyperemia
- Edema + Vascular Congestion
- intact surface
- neutrophils ...

2. acute gastric ulcers:



- physiological stress

Stress ulcers due to local ischemia {hypertension, systemic v.c, acidosis}

Curling ulcers

- Burn
- duodenum

Cushing ulcers

- intracranial lesion
- Stomach, duodenum, esophagus

Morphology:

- anywhere in stomach
- shallow to deep
- normal adjacent
- no scarring



Clinical: - Melena
- Coffee-ground hematemesis

Tx: PPI

• out comes depend on underlying cause

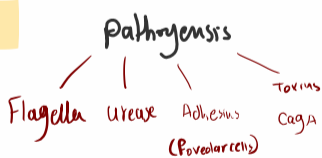
3- Chronic Gastritis

• Clinical Features: upper-abdominal discomfort

• Causes: ① H. pylori (-ve) 75%

• underlying cause → duodenal ulcers

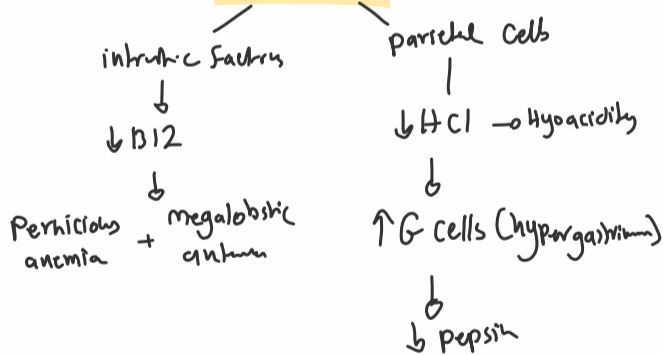
• non-invasive



Diagnosis → Serologic Test (Stool urea, gastric culture, biopsy, histologic culture, PCR)
Tx: 2 antibiotics + PPI

② Autoimmune atrophic 10%

Antibodies - Shnigwell



Feature	H. pylori-Associated	Autoimmune
Location	Antrum	Body
Inflammatory infiltrate	Neutrophils, subepithelial plasma cells	Lymphocytes, macrophages
Acid production	Increased to slightly decreased Hyperacidity	Decreased hypoacidity
Gastrin	Normal to markedly increased	Markedly increased
Other lesions	Hyperplastic/inflammatory polyps	Neuroendocrine hyperplasia
Serology	Antibodies to H. pylori	Antibodies to parietal cells (H ⁺ , K ⁺ -ATPase, intrinsic factor)
Sequelae	Peptic ulcer, adenocarcinoma, lymphoma	Atrophy, pernicious anemia, adenocarcinoma, carcinoid tumor
Associations	Low socioeconomic status, poverty, residence in rural areas adenocarcinoma MALToma	Autoimmune disease; thyroiditis, diabetes mellitus, Graves disease adenocarcinoma neuroendocrine tumor

4- Peptic Ulcer disease :- Any portion exposed to acidic gastric

- H. pylori + NSAID

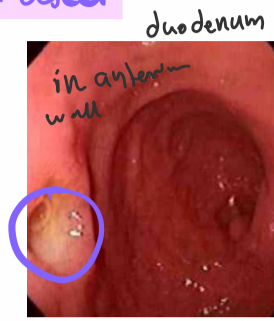
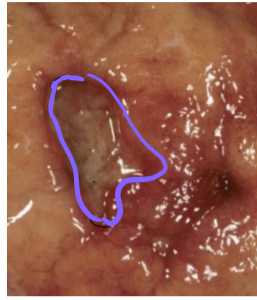
4 duodenum : 1 stomach

- imbalance between mucosal defenses and damaging forces

- clean base, punched out defect

Cause of hyperacidity

- H. pylori
- Parietal cell hyperplasia
- Excessive secretion
- Zollinger-Ellison Syndrome → Hypergastrinemia



* Clinical Features :

- burning + pain 1-3 after meal
- relieved by eating + worse at night
- iron deficiency, hemorrhage,

Perforation
surgery

B-Polyps and Tumors

1- Gastric Polyps :

- masses above mucosa
- epithelial or stromal cells hyperplasia
- 75% inflammatory and hyperplastic polyps
- background of chronic gastritis
- H. pylori → Regress



• Gastric adenoma

- 10% of Polyps
- background of chronic gastritis
- Dysplasia in all cases
- Risk adenocarcinoma
- 30% → carcinoma

2- Gastric Adenocarcinoma :

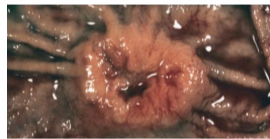
- mimic gastritis → late diagnosis

2 Types

intestinal

FAP: APC gene mutation

M: Bulky mass
- form glands



weight loss

diffuse

- Familial → CDH-1 (E-cadherin)
- sporadic → CDH-1, B catenin
- signet ring cells ☹️
- thick wall

bad prognosis

C: developed from precursor

M > F - 55y

- no precursor
M = F
younger

Tx: surgery, chemotherapy, anti HER2

3- lymphoma :

• common extranodal in stomach

- MALToma
- diffuse large B cell lymphoma

4- Neuroendocrine (Carcinoid) Tumor

- From G-cells
 - good prognosis
 - Carcinoid syndrome:
 - associated with metastasis
 - due to vasoactive substance
- amines production leads to:

flushing, sweating, bronchospasm,
colicky pain, right cardiac fibrosis

Disease of the intestines:

1 - obstruction

(Pain, distention, Vomiting, Constipation)

Mechanical

Intussusception:

- propelled by peristalsis
- < 2y . untreated → infarction

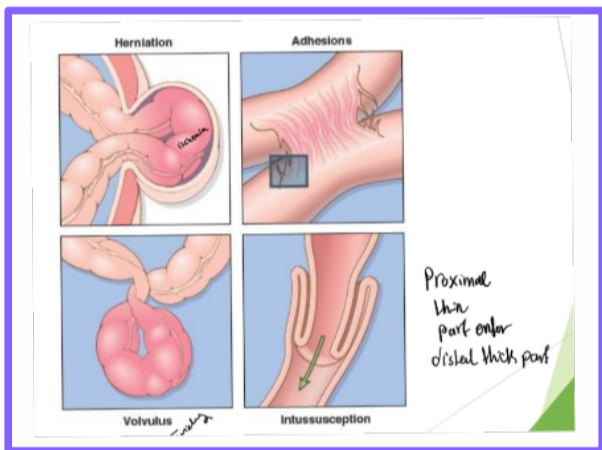
Causes {

- < 2 → idiopathic
- Peyer patches
- Meckles diverticulum (ileum)
- Tumors

Jelly stool (stool mixed with mucus)

Management {

- uncomplicated → Contrast enemas diagnostic + therapeutic
- complicated → surgery



non-mechanical functional

Hirschsprung disease:

- Congenital → colonic innervations
- absent of ganglionic cells → prevent peristalsis
- Neonatal failure to pass meconium
- later: obstructive constipation
- disrupted migration Cecum → rectum
- Mutation in RET

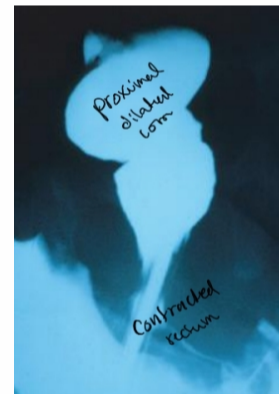
most → rectosigmoid

Macro: Aganglionic region → Normal or contracted
proximal → dilated

D: barium enema, Biopsy, microscopic

Complications: Enterocolitis

Tx: Surgical resection



2 - Vascular disorders of bowel

Ischemic bowel disease

Hemorrhoids:

dilated anal and perianal collateral vessels

Portal + Caval venous systems

Predisposing factors: venous stasis + portal hypertension

External below anorectal line

Internal above

(Fresh)

S: Bleeding, thrombosis

Tx: Sclerotherapy, rubberband, infrared coagulation, hemorrhoidectomy

Dysentery: small, bloody, painful diarrhea

3- diarrheal disease

a- Malabsorptive diarrhea:

• Steatorrhea

- Manifestations: anorexia, Borborygmi, Anemia, bleeding (Vit.K), Neuropathy, endocrine disorders

b- Cystic Fibrosis:

- Mutation CFTR
- defects in ion transport (intraluminal)
 - ↓
 - Thick viscous secretions
- Meconium ileus in neonates

c- Celiac disease:

• gluten sensitive

• HLA-DQ2 . HLA-DQ8

• **Gluten** → gliadin → APC

→ CD4+T → cytokines → Tissue damage

• 2nd portion of duodenum or proximal jejunum

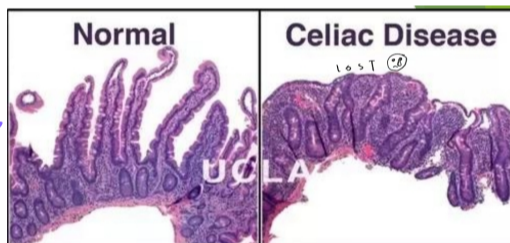
Triad: {

- CD8+ T cells
- crypt hyperplasia
- villous atrophy

D: Clinical, histologic + serologic correlation

C: Children — classical

• Blistering skin — non classical



→ Failure to thrive

D:

① Non-invasive serology

a) most sensitive: - IgA - IgA + IgG

b) most specific: Antiendomysial Ab

③ invasive: small bowel biopsy

• adults: Anemia / B12 + folate deficiency: less common

↑ risk of T cell lymphoma & S.I adenocarcinoma

D- Lactase deficiency:

• osmotic diarrhea

• lactose remain in the gut lumen

Congenital: rare, after milk ingestion

Acquired: follow viral or bacterial enteritis

2 Types

E- Abetalipoproteinemia:

• AR • rare • steatorrhea

• infant → Failure to thrive

• ↓ absorption of fat bc X synthesis of TG-rich LP X transepithelial

الصفحة التي بعدها
 sigmoid diverticulitis

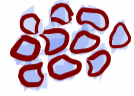
4 - inflammatory For life (chronic)

Chronic inflammatory bowel disease (CIBD) Hygiene hypothesis

a - Crohn disease: any area in GIT / transmural

most common region: terminal ileum, ileocecal valve and cecum

Cobblestone appearance



Fissure, fistulas, perforations

micro: Neutrophils, ^{in acute}

deep ulcer

Creeping Fat

metaplasia in left colon

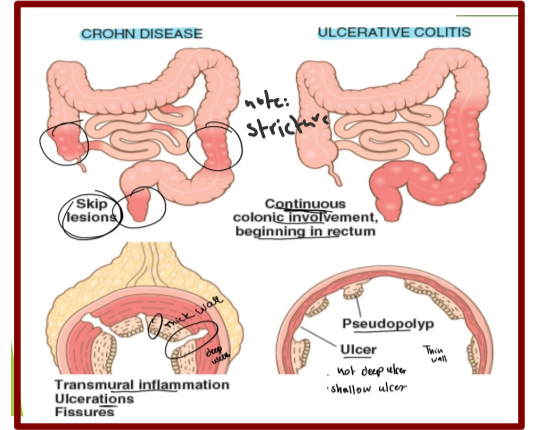
Non-caseating granulomas 35%, Crypt abscess, arranged

C: ① right lower-quadrant acute pain

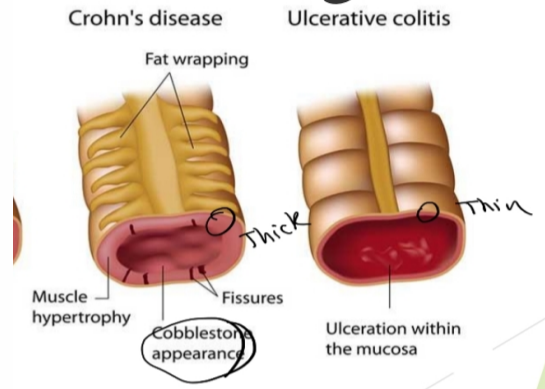
② Bloody diarrhea - متقطع

Complications: anemia, hypoproteinemia, fistulas, Peritoneal abscess, Strictures, adenocarcinoma

فقر حديد



الفروقات

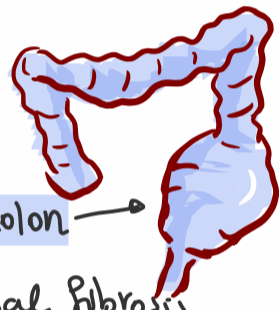


b - Ulcerative Colitis: colon & rectum / mucosa & submucosa

SI is normal (except in backwash ileitis)

Macro: Pseudopolyps, ^{superficial} broad-based ulcers, Toxic megacolon

Micro: inflammatory infiltration, E. metaplasia, Submucosal fibrosis (neutrophils)



C: bloody mucoid diarrhea + lower abdominal cramps, relieved by defecation

TX: Colectomy

Colitis-associated Neoplasia:

UC + CD

Risk:

- ① duration
- ② involvement
- ③ inflammation

Extra intestinal:

For both

- Uveitis
- Polyarthritides
- Sacroiliitis
- Ankylosing spondylitis
- Erythema nodosum
- Clubbing fingers
- Primary sclerosing cholangitis (UC more)

Feature	Crohn Disease	Ulcerative Colitis
Macroscopic		
Bowel region affected	Ileum ± colon	Colon only
Rectal involvement	Sometimes	Always
Distribution	Skip lesions	Diffuse
Stricture	Yes	Rare
Bowel wall appearance	Thick	Thin
Inflammation	Transmural	Limited to mucosa and submucosa
Pseudopolyps	Moderate	Marked
Ulcers	Deep, knifelike	Superficial, broad-based
Lymphoid reaction	Marked	Moderate
Fibrosis	Marked	Mild to none
Serositis	Marked	No
Granulomas	Yes (~35%)	No
Fistulas/sinuses	Yes	No

Clinical		
Perianal fistula	Yes (in colonic disease)	No
Fat/vitamin malabsorption	Yes	No
Malignant potential	With colonic involvement	Yes
Recurrence after surgery	Common	No
Toxic megacolon	No	Yes

• Sigmoid diverticulitis

• Acquired Mucosa + submucosa

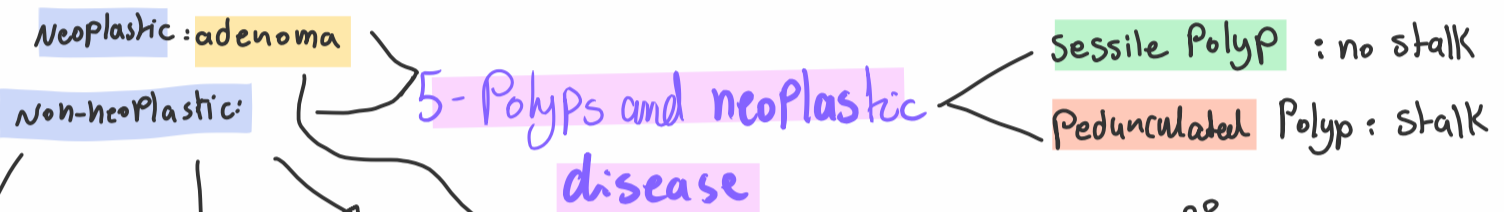
• Pseudodiverticular

Morphology: Sigmoid Colon, Thin wall, X muscularis, Perforation, strictures

• Can caused by diverticulitis

C: asymptomatic / Constipation or diarrhea

TX: Fiber diet (↓ constipation), Antibiotics (↓ infection), Surgery (↓ perforation)



• Inflammatory Polyps:

• Syndrome is rectum

• chronic cycle of injury and healing

• hyperplasia

• pileup of GC & Epithelial overcrowding

• no malignant / no atypia

• left colon / rectosigmoid

• Adenomas

• epithelial dysplasia

• Most don't → Carcinoma BUT Precursor for majority of adenocarcinoma

a- Colon adenoma:

• epithelial dysplasia

risk for malignancy { size, grade}

b- Villous adenoma:

more frequent invasive foci

3 Types { Tubular, Tubulovillous, Villous}

• hamartomas:

Disorganized

α- Juvenile Polyps: most common

• TGF-β mutation AD

• ↑ risk of adenocarcinoma

Sporadic → Solitary

Syndromic → Multiple

• Pedunculated • dilated gland

• Cystic + granulation

b- Peutz-Jeghers syndrome: 

• AD • Multiple Polyps • S.I

• ↑ risk of malignancies • LKB1 / STK11 mutation

• network of CT, Sm, lamina propria

• Mucocutaneous hyperpigmentation

normal S.E

Thank You