GIT practical II

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1 - Shortening of villi → diarrhea

2 - Crypts hyperplasia (increase in crypts number and thickness to regenerate surface epithelia)

3 - Increase intraepithelial lymphocytes (CD8)

Normal Intestinal Villi
(Source: library.med.utah.edu)

Damaged Villi (Celiac Disease)
(Source: commons.wikimedia.org/wiki/User:Nephron)
Main manifestations of celiac disease:
- Malabsorption (iron deficiency anemia)
- Skin lesion (dermatitis herpetiformis)

If it appears, we diagnose it as celiac disease.

Diagnosis of celiac disease:
1. Serological test for anti tissue transaminases (tTG), anti endomysial in IgA form
   - If negative: ask for IgG test due to IgA deficiency in these patients
   - If positive: do upper GI endoscopy to take biopsy from duodenum to detect the severity (how much the villi are short)
Crohn disease

*Crohn disease → chronic inflammation with on and off symptoms (diarrhea, malabsorption, pain) → relapsing. Affect the GI from mouth to anus.

**Fistula formation:** connection of 2 loops

**Perforation**

**Cobblestone appearance:** elevated areas (normal), depressed areas (abnormal) → skip lesions

**Fatty creeping:** fatty accumulation on inflamed area that reach serosa

Figure 17-34 Gross pathology of Crohn disease. A, Small-intestinal stricture. B, Linear mucosal ulcers, which impart a cobblestone appearance to the mucosa, and thickened intestinal wall. C, Perforation and associated serositis. D, Creeping fat.
Cobblestone appearance – Diseased tissue is depressed below the level of normal mucosa
UC start in rectum to colon (in pan colitis → backwash ileitis) with bloody diarrhea but there is no fistula or perforation.

Superficial inflammation (mucosa, submucosa)

Pseudopolyps (inflamed areas with high edema → elevation)

Mucosal bridges between pseudopolyps
Toxic megacolon

Very thin wall (نفس فكرة البالون لما ننفخه بيعتى هواء و غلافه بينحف)
Due to disruption of neuromuscular control \(\rightarrow\) dilatation of wall \(\rightarrow\) bowel perforation (ex: trypanosome cruizi infection cause chagas disease)
Repeated pass of inflammation and resolution $\rightarrow$ altered mucosal architecture (normally flat surface).

Hyperplastic changes in crypts: inflated surface, bigger, branched.

*In some places there is loss of glands or crypts (drop of glands) $\rightarrow$ chronicity.


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Abnormal histology of colon
*In lamina propria of colon and small bowel  \(\rightarrow\) lymphocytes, plasma cell normally but no neutrophils
In stomach there are no inflammatory cells if any of them appears  \(\rightarrow\) gastritis

*If there are large number of neutrophils attack the gland  \(\rightarrow\) cryptitis

Non caseating granuloma (granulomatous inflammation)  \(\rightarrow\) chronic inflammation (TB infection  \(\rightarrow\) caseating granuloma  \(\rightarrow\) until we distinguish them

If neutrophils accumulate inside the crypt  \(\rightarrow\) crypt abscess (mean there is active disease) more dominant in UC.
Ischemic bowel disease

Emboli or thrombosis in major vessels that supply the small intestine → complete obstruction → transmural ischemia → necrosis → gangrene (dark color)
Ischemic colitis
Ishemic bowel disease

Ischemia and fibrosis in the bowel wall ..
commonly seen in heart failure patients → decreased blood supply ...
only involves the mucosa and submucosa.
- colon
  a pseudomembrane composed of inflammatory cells & fibrin
المهم إنه كيف صار هاد
crypt abscesses on surface epithelium (neutrophils accumulated inside the crypt) and it ruptured as a volcano!
Pseudomembranous Colitis

• generally caused by C. difficile, can also be referred to as antibiotic-associated colitis.

Mostly, when elderly patients take antibiotics for simple reasons! This changes the flora in GI especially C. difficile. when it undergoes overgrowth, it causes cryptitis.. abscesses and eventually this pseudomembrane..

Clinicians call it antibiotic associated colitis. While pathologists call it PseudomembranousColitis.

- Presentation: mainly watery diarrhea with a history of antibiotics intake.
- Treatment: antibiotic for C. difficile.. either metrodiazole or vancomycin.
Diverticula:
pockets in the muscle wall of the colon but not extended through the serosa.

Outpouching „
In elderly patients with constipation due to decreased fiber in diet → increased intraluminal pressure during defecation will lead to something like invasions through mucosa and submucosa in weakened areas.

Asymptomatic but if an inflammation happens "diverticulitis". it may lead to perforation.
Presentation: acute abdominal pain ...
Volvolus:
sigmoid has a redundant loop so it's more easy to have twisting.
It causes:
- ischemia by cutting blood supply.
- intestinal obstruction and risk of perforation.
Sigmoid volvulus
Polyps

A & B are taken by endoscope ..
polyps with a stalk - pedunculated .
without a stalk – sessile .

Fig. 1. (A) Pedunculated polyp. (B) Small sessile polyp.

types of polyps in colon :
- hyperplastic : elevated portion with hyperplastic changes in crypts , large sized crypts .
  #no risk of malignancy (completely benign) , while in stomach hyperplastic polyps increase the risk the malignancy according to its size.
- adenomatous polyps : adenomas are precancerous lesions .
- hamartomatous polyps : it has septations by muscle fibers , mainly it's benign.
ADENOMAS

Pedunculated Tubular

Sessile Villous

Basement Membrane

Mucosa
Submucosa
Muscularis propria
Hyperplastic polyp/colon
M.C pigmentation

perioral pigments and in the mucus membrane of the oral cavity, this patient came with lower GI bleeding, after endoscopy polyps were found in colon, histopathologist said that it’s a hamartomatous polyp!! then you'll understand that it’s a Peutz–Jeghers syndrome!!

Take this:
hamartomatous polyps in bowel + mucus membrane's pigmentation(oral/anal) = Peutz–Jeghers syndrome

Those patients on long term have higher risk of adenocarcinoma in small bowel / large bowel and extraintestinal malignancies like: thyroid tumors and cervical and endometrial cancers.
hamartomatous polyp
Adenomas:
Two types: tubular and villous. Tubular don’t have villi on surface while villous have villi.

Clinically there is no difference between them. It’s nothing but descriptive terms for pathologists!! Some villous secrete protein and potassium and patient may develop hypoproteinemia and hypokalemia. But it’s another cases not involved here with us.

Tubular adenoma
FIGURE 34.28  Villous adenoma. (A) Whole-mount view showing adenomatous epithelium growing in a villous or fingerlike arrangement. (B) High-power view showing elongated nuclei with some palisading. The nuclei do not reach the cell surface, and the apical portion of cells shows mucin production. This is low-grade dysplasia.
Villous adenoma
mutation in APC gene. There must be 100 polyps in colon at least!
Hereditary, happens at small ages 15-16
Lower GI bleeding
in family history, the parent had cancer at age of 30-50 years.
biopsy of polyps shows adenomas "this means dysplasia and higher percentage of malignancy, 100% at age of 30"
treatment: colectomy
extraintestinal malignancies may occur: brain tumors and others..
FAP
Colon adenocarcinoma
Colon mucinous adenocarcinoma

: abundant amount of mucin
Figure 18-7 Cirrhosis resulting from chronic viral hepatitis. Note the depressed areas of dense scar separating bulging regenerative nodules over the liver surface.
Liver cirrhosis:

Cirrhotic nodules, microscopically: fibrous bands—blue—separating the nodules, nodules are different in size.

Masson's trichrome stain
steatosis

normally fat droplets in liver <5%. If it's higher, then we call it steatosis
Hepatitis:

Apoptotic cells in liver: acidophiles.
Inflammation in liver is either portal or tubular according to its site.
Mainly by hepatotropic viruses (A, B, C, D, E).
A & E are acute not chronic, can make fulminant liver failure.
B & C: chronicity, liver cirrhosis & hepatocellular carcinoma.
Chronicity in C is higher: 80-90%
Microscopically:
B-> glass hepatocytes
C -> lymphoid follicles
To differentiate clearly between B & C use serological tests: B surface antigen (s-Ag) or C antibody
Hepatitis B vaccines are used usually, as you know HCV doesn’t have vaccine.
plasma cells secrete Antibodies, so when liver is infiltrated with plasma cells, we observe that it's an autoimmune disease. And here it's called autoimmune hepatitis.
inclusion inside the hepatocytes.. called mallory bodies (accumulation of intermediate keratin 8 & 18 ) mainly seen with alcoholic liver disease. it appears like pink granules.

ballooning: water accumulation due to alcoholic effect, appears white.
Figure 18-20  A, Alcoholic hepatitis with clustered inflammatory cells marking the site of a necrotic hepatocyte. A Mallory Denk body is present in another hepatocyte (arrow). B, Alcoholic steatohepatitis with many ballooned hepatocytes (arrowheads). Clusters of inflammatory cells are also present; inset shows immunostaining for keratins 8 and 18 (brown), with most hepatocytes, including those with fat vacuoles, showing normal cytoplasmic staining, but in the ballooned cell (arrow) the ubiquinated keratins are collapsed into the Mallory-Denk body, leaving the cytoplasm “empty.” (Courtesy Dr. Elizabeth Brunt, Washington University, St. Louis, Mo.)
Figure 18-21 Alcoholic cirrhosis. A, The characteristic diffuse nodularity of the surface is induced by the underlying fibrous scarring. The average nodule size is 3 mm in this close-up view, typical of the "micronodular" cirrhosis of alcoholic liver disease. The greenish tint is caused by cholestasis. B, Microscopically, this cirrhosis is marked by small nodules entrapped in blue-staining fibrous tissue; fatty accumulation is no longer seen in this "burned out" stage. (Masson trichrome stain.)
Figure 18-22 Nonalcoholic fatty liver disease. A, Liver with mixed small and large fat droplets. B, Steatosis and steatofibrosis extending along sinusoids in a chicken wire fence pattern in which individual and clustered hepatocytes are surrounded by thin scars (blue fibers). Note the resemblance to alcoholic steatohepatitis depicted in Fig. 18-19. (Masson trichrome stain.)
Hemochromatosis

**hemochromatosis**: primary causes that lead to iron accumulation in body by genetic defect.

iron in tissues → hemosiderin (yellow/golden particles)

**hemosiderosis**: any secondary cause of iron accumulation in body – (one cause mentioned was: iron overload in anemia patients who undergo blood transfusion)

*Prussian blue stain.*

![Image of hemochromatosis](https://example.com/image)
Figure 18-25 Hereditary hemochromatosis. In this Prussian blue-stained section, hepatocellular iron appears blue. The parenchymal architecture is normal.
Alpha-1-antitrypsin deficiency

Alpha-1-antitrypsin: an antiprotease produced by liver, inhibits proteases accompanied to inflammatory cells, its deficiency leads to emphysema by destruction of alveolar wall.

* autosomal recessive..

alpha-1-antitrypsin is produced normally but the problem is in excretion and transportation, so it accumulates in liver causing cell injury. At the same time it's not transported to lungs and thus it can't inhibit elastase activity there... so emphysema happens!

*special stain: PAS

Figure 18-26 α1-Antitrypsin deficiency. A, Periodic acid–Schiff (PAS) stain after diastase digestion of the liver, highlighting the characteristic magenta cytoplasmic granules. B, Electron micrograph showing endoplasmic reticulum dilated by aggregates of misfolded protein.
Cholestasis:
accumulation of bile in liver, bile contains bilirubin, cholesterol, water, bile acids, green-yellow pigments..
Figure 18-30 Ascending cholangitis. Individuals with large bile duct obstruction risk bacterial infections of the static bile within the biliary tree. Neutrophils are then seen within the bile duct epithelial lining and within the lumen.
Neonatal cholestasis

Figure 18-34 Neonatal hepatitis. Note the multinucleated giant hepatocytes.
tumor in liver, green in color (related to bile).
Hepatocellular carcinoma: main primary tumor in liver.
Most common tumors in liver are metastatic tumors!
Figure 18-60 Cholangiocarcinoma. A, Multifocal cholangiocarcinoma in a
Black stones in bladder, mainly in **hemolytic anemia patients** (the idea of increasing bilirubin levels)
cholesterol stones in bladder due to hypercholesterolemia, more common than black stones.
Histology of gall bladder:
Gall bladder doesn’t have submucosa, that’s why glands -somehow- reach the muscularis propria …. Called Rokitansky – Aschoff sinus.

Rokitansky-Aschoff sinuses are diverticula of the gallbladder wall which may be microscopic or macroscopic. Histologically, they are outpouchings of gallbladder mucosa that sit within the gallbladder muscle layer.

Related pathology: They are not of themselves considered abnormal, but may be associated with cholecystitis and adenomyomatosis. external information for better understanding.
# main risk factor for gall bladder carcinoma is gall stones