



GI

Pathology

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Intestinal pathology, part 1

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We will talk in this lecture about intestinal pathology (when we talk intestine ,we mean small and large intestines)

The small intestine has three parts(duodenum ,jejunum and ileum is at the end)

The large intestine is formed of caecum ,ascending colon ,transverse colon ,descending colon ,sigmoid and rectum .

Diseases of the intestines

- Intestinal obstruction
- Vascular disorders
- Malabsorptive diseases and infections
- Inflammatory intestinal disease.
- Polyps and neoplastic diseases

Intestinal obstruction

The obstruction happens when something interferes with the flow of substances in small bowel or large bowel.
Most commonly site is small intestine because it is small and narrow canal compared to large intestine .

Mechanical obstruction:

Intussusception
Hernias.
Adhesions.
Volvulus

80% of
intestinal
obstruction

Tumors.
Diverticulitis
Infarction

Non-mechanical obstruction (functional)

Hirschsprung disease
Neurological disorders.
Drugs....etc

Clinical picture of intestinal obstruction.

The same symptoms occur in any intestinal obstruction with some differences according to the severity

- Abdominal pain
- Distention
- Vomiting
- Constipation.

- Acute or chronic.

Case can be acute emergency or chronic

-(acute is more severe symptoms with suddenly onset)

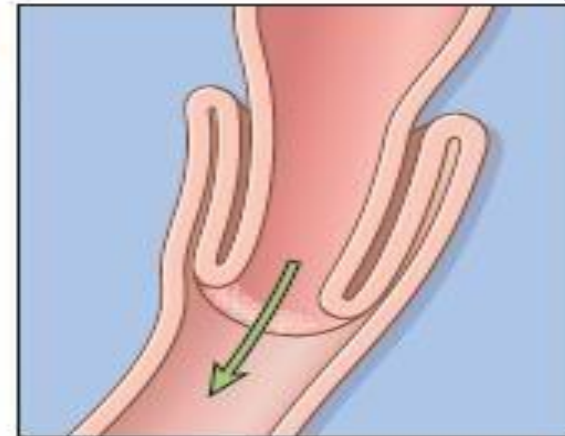
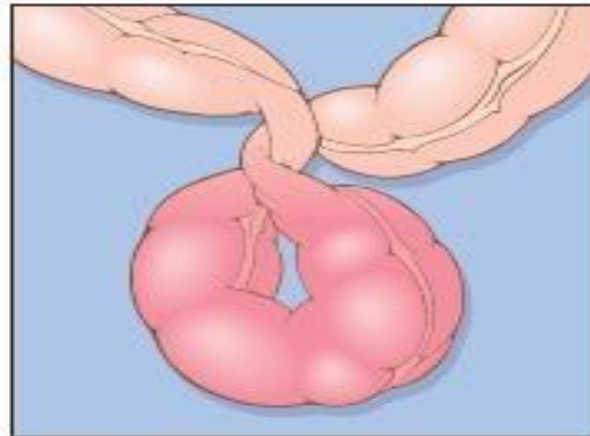
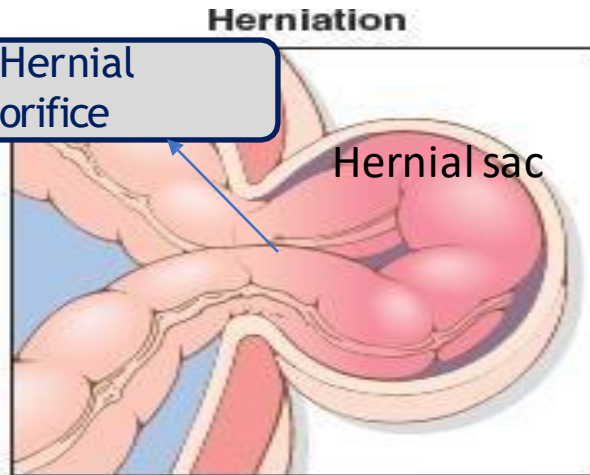
-(chronic is less severe symptoms and extend for long period of time)

80% of mechanical obstruction

Adhesions

Previous surgery or previous inflammation >>> fibrosis >>> adhesion between bowel loops or inflammatory loop with healthy loop (adjacent loops or distant loops)

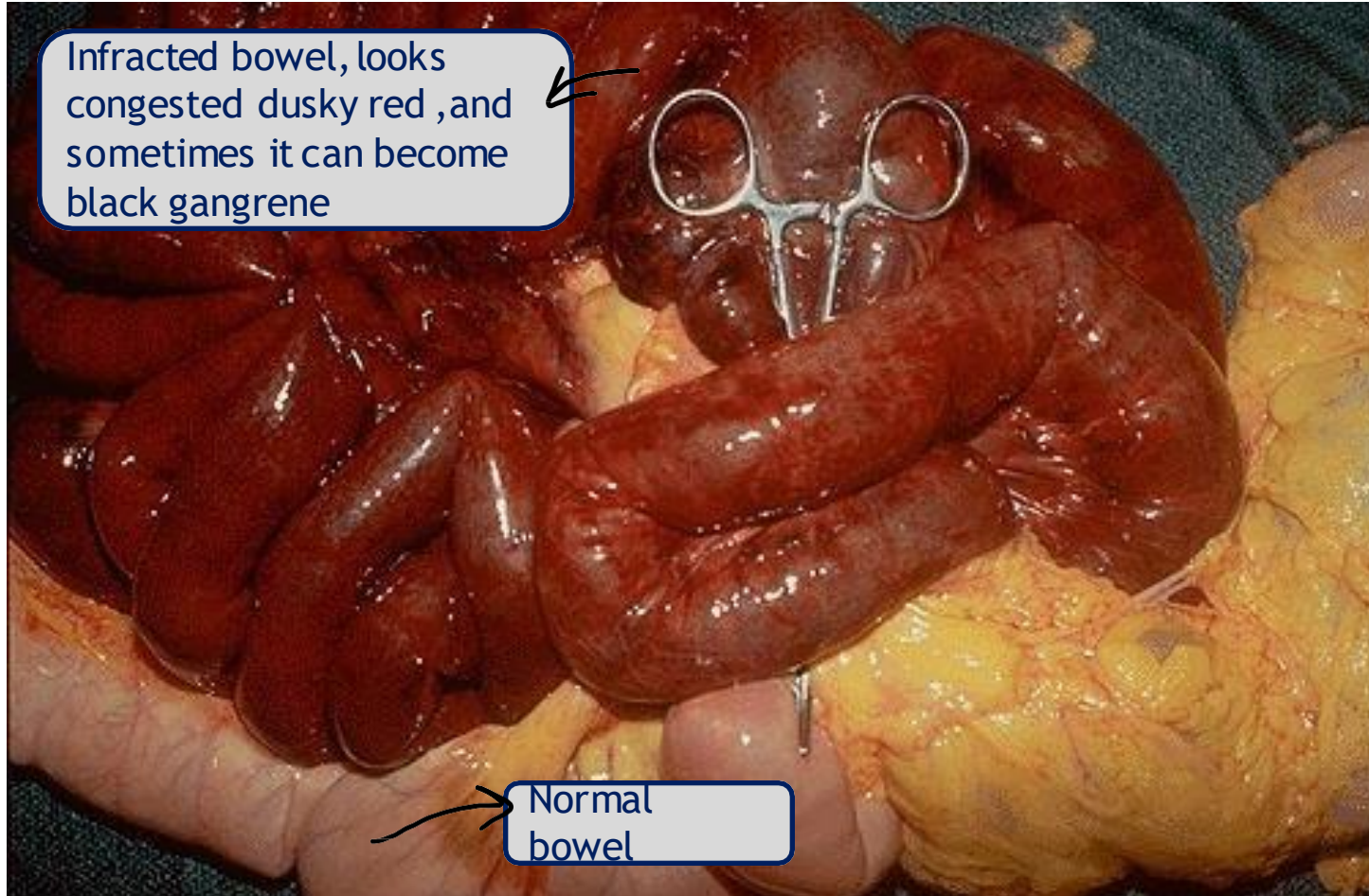
Herniation: when part of the bowel enters to defect (weak) area in the abdominal wall it becomes incarcerated there, which will decrease venous drainage and blood supply, this may lead to ischemia, in severe cases it will lead to gangrene >>>>> surgical management to release hernia or remove the affected bowel part.
- If the hernial orifice is wide, the bowel will enter and exit the hernial sac.



Intussusception occurs when a part of the intestine folds into itself, similar to how parts of a collapsible telescope slide into one another.

Volvulus (twisting the loop on itself) >>> decrease in blood flow >>> ischemia >>> gangrene necrosis
It's emergent as u are losing part of the bowel

Bowel infarction



Intussusception

- Segment of the intestine constricted by peristalsis, telescopes into the immediately distal segment.
- Once trapped, invaginated segment is propelled by peristalsis, and pulls mesentery with it.
- **Most common cause of intestinal obstruction in children younger than 2 years of age.**
- Untreated progresses to obstruction and infarction.



Mortality rate is high

Causes of intussusception

- **Idiopathic in most cases. (it means there's no certain cause, it just happens with this age group)**

The causes:


- **Other causes:**

- Peyer patches hyperplasia (rotavirus vaccine, viral infections)
- Meckles diverticulum (ileum) ▪

- Old children & adults: Intraluminal mass or tumors (Major of intussusception cases are to children less than 2 yrs, so if the patient is older, u should think of tumor!!)

Clinical features:

- **Abdominal** swelling
- **Vomiting**
- Passing stools mixed with blood and **mucus (currant jelly stool)**
- **Pain.**



It is a characteristic feature for intussusception

Management

By injecting a colored fluid or material in the anal canal for diagnostic and therapeutic reasons .

Diagnostic : the colored material appears on x-ray

Therapeutic : give high pressure in order to reduce intussusception

- Contrast enemas (diagnostic and therapeutic) in uncomplicated idiopathic cases.
- Surgery if complicated by infarction or if masses are the leading point.

Meckel's diverticulum

- The Cause*
- The most common congenital anomaly of the GI tract
 - Incomplete obliteration of omphalomesenteric duct (related to embryology, an embryonic structure that usually disappears as the fetus develops.. But here in this case it does not)
 - True diverticulum. →

It appears like a bulge(out pouching) that raises from bowel lumen and contains all the layers (mucosa , submucosa ,muscular ...) >>> true diverticulum.
 - **Remember (rule of 2):**
 - About 2% of people have them;
 - Located 2 feet from the ileocecal valve.
 - 2 inches in length.
 - 2 types of heterotopic mucosa (gastric or pancreatic).
 - Most common cause of lower GI bleeding before age of 2.

Meckel's diverticulum



Bulge (out pouching) = it secretes acid similar to stomach lining

Clinical presentation

Lower GI bleeding is featuring with fresh blood . Upper GI bleeding is featuring with melena (black stool)

- Can be asymptomatic and discovered incidentally.
- Ulceration, lower GI bleeding or perforation from ectopic gastric mucosa.
- Bowel obstruction due to the intussusception, volvulus or adhesive band.

- Can be confused with acute appendicitis.(As it is near to it and the symptoms caused by complications of Meckel's diverticulum can closely mimic those of appendicitis.)

Hirschsprung Disease

ADDITIONAL: It occurs when ganglion cells, are missing from a part of the colon. These cells are part of the enteric nervous system, which controls (peristalsis). Without these cells, the affected segment of the colon can't properly relax, leading to a blockage of the intestine and difficulty passing stool.

- Congenital defect in colonic innervations (**common in pediatric**)
- Congenital aganglionic megacolon(mega= it becomes larger due to obstruction)
- More common in males, but More severe in females
- Risk increase in siblings.
- **Typical presentation:**
- Neonatal failure to pass meconium meconium =(the first stool) within the first 48 hours after birth.
Later: Obstructive constipation.

Pathogenesis

- **During embryogenesis:** disrupted migration of neural crest cells from cecum to rectum.
- **Aganglionosis: Distal intestinal segment lacks both: Meissner submucosal plexus and the Auerbach (myenteric) plexus.**
- Failure of coordinated peristaltic contractions.

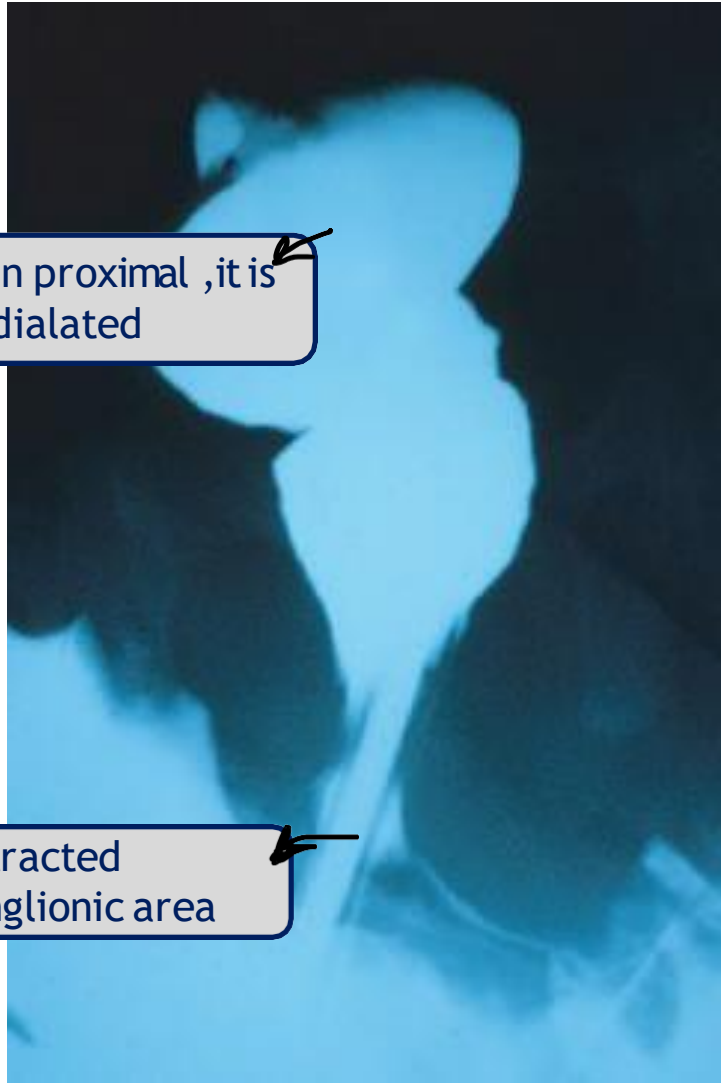
- RET Mutations: in familial cases and 15% of sporadic.
- Other genes and environmental factors play role.
- More in Down syndrome.

In severe cases
,it affects all
colon

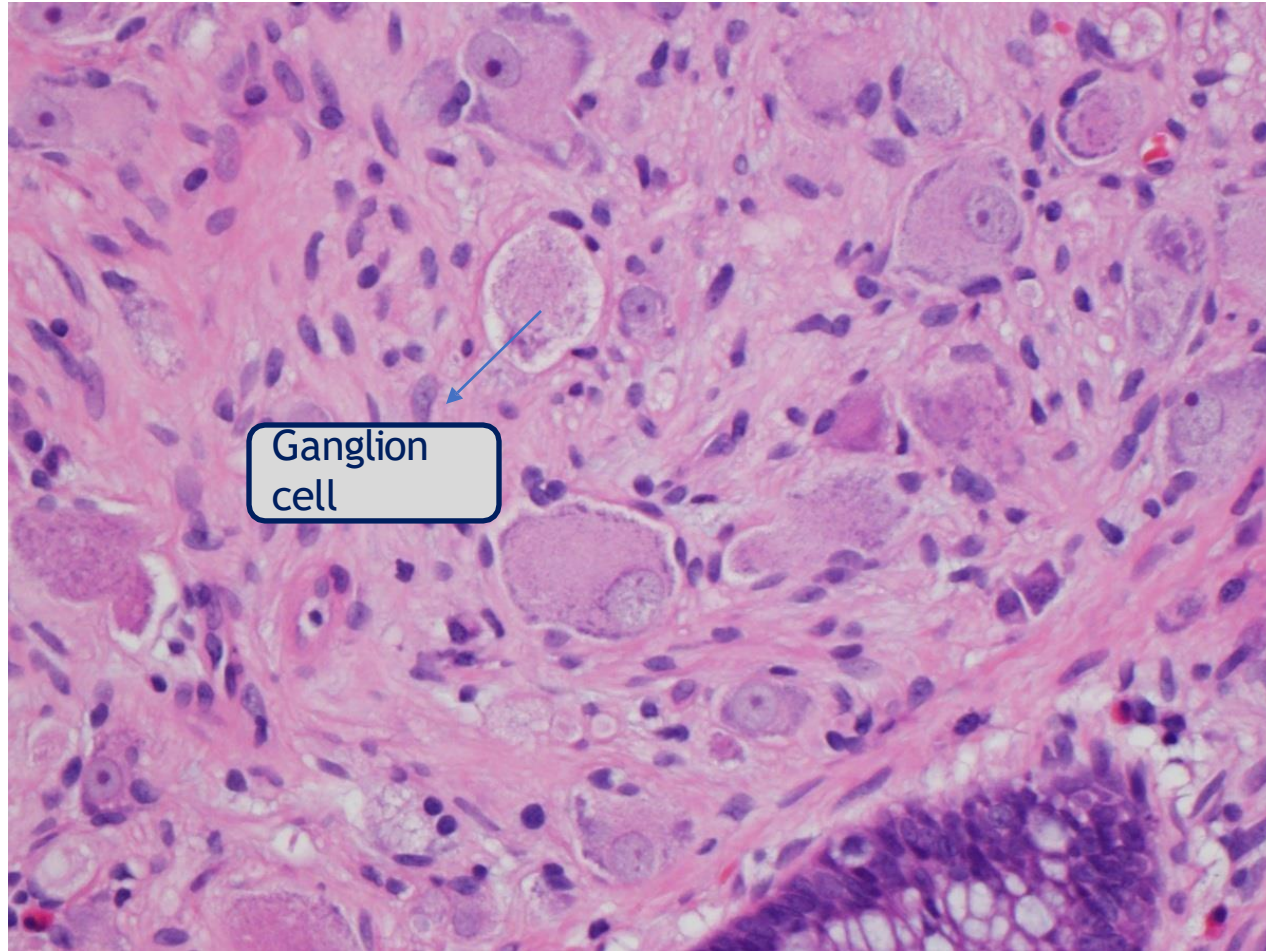
Morphology

- Rectum always involved, Most cases in rectosigmoid
- Extent is variable.
- Aganglionic region normal or contracted
- Proximal normal segment progressively dilated.
- **BIOPSY to confirm absence of ganglion cells.**
This is the gold standard method
- **Diagnostic workup: barium enema, biopsy.**

To confirm the absence of ganglion cells, the biopsy should be full thickness to contain all layers (mucosa, submucosa, muscular)



ganglion cells



Complications

- Enterocolitis
- Fluid and electrolyte disturbances
- Perforation
- Peritonitis

Rupture

- **Treatment:**
- Surgical resection of aganglionic segment and anastomosis of normal segments.

VASCULAR DISORDERS OF BOWEL

- **Ischemic Bowel Disease**
- **Angiodysplasia.**
- **Hemorrhoids**

It comes with older age groups which already have IHD (ischemic heart disease) or atherosclerosis, we will not talk about it now anyways

Let's talk about these TWO!

Angiodysplasia.

Rare disease

- Malformed submucosal and mucosal blood vessels.
- Most often in cecum and right colon.
- 6th decade of life.
- Less than 1% of adult population.
- 20% of cases of lower GI bleeding.
- Blood is bright red in color.

It is similar
to varices

Hemorrhoids

- Dilated anal and perianal collateral vessels that connect the portal and caval venous systems.

- **Predisposing factors:**

- Constipation and straining
- Venous stasis of pregnancy,
- Portal hypertension.

The type of diet

- External (below anorectal line, inferior hemorrhoidal plexus) and internal (above anorectal line, superior hemorrhoidal plexus).

they are classified to external and internal according to their location to anorectal line ,

Note that both of them can go to outside and be viewable

- **Morphology:**

- Thin -walled, dilated, submucosal vessels beneath anal or rectal mucosa.

- **Symptoms:**

- Bleeding (bright red), pain due to thrombosis and inflammation

Usually ,it is painless ,but can cause pain in the condition with thrombosis or inflammation

- **Treatment:**

- Sclerotherapy, rubber band ligation, infrared coagulation.
Hemorrhoidectomy.

Extra info :

Sclerotherapy: It's a treatment where a special solution is injected into hemorrhoids, causing them to shrink and eventually disappear.

Rubber band ligation: This involves placing a small rubber band at the base of the hemorrhoid to cut off its blood supply. The hemorrhoid then shrivels up and falls off within a few days.

Infrared coagulation: This method uses infrared light to create scar tissue, which cuts off the blood supply to the hemorrhoid, causing it to shrink and eventually disappear.

Hemorrhoidectomy: It's a surgical procedure to remove severe or recurring hemorrhoids. It involves cutting out the hemorrhoidal tissue.

DIARRHEAL DISEASE

- Diarrhea: increase in stool mass, frequency or fluidity.
- Dysentery: painful , bloody, small volume diarrhea.
- Secretory, osmotic, malabsorptive, exudative.

- **Malabsorptive Diarrhea**

- Pancreatic insufficiency.
- **Celiac disease**
- Crohn disease
- **Cystic Fibrosis**
- **Lactase (Disaccharidase) Deficiency**
- **Abetalipoproteinemia**

- **Infectious Enterocolitis**

- **Ischemia.**

- **Inflammatory bowel diseases.....**

Secretory ;secretion more fluid ,regardless of whether food or drink is taken
Osmotic ;accumulation of food content in bowel which is leading to pull the water to intestinal lumen (like lactose intolerance)
Exudative ; mucus and discharge are seen in diarrhea
.this type is seen more with infections and inflammation

Malabsorptive Diarrhea

- **Chronic.**

defective absorption happens in all diseases in this type, but they vary according to the specific matter which is malabsorbed (fat, carbs, etc..)

- Defective absorption of fats, fat- and water-soluble vitamins, proteins, carbohydrates, electrolytes, minerals and water
- **Hallmark is : steatorrhea.** (excessive fat, bulky, frothy, yellow, greasy stool)

Malabsorptive diarrhea

Defect in one of the following:

- **Intraluminal digestion.**
- **Terminal digestion.**
- **Transepithelial transport.**
- **Lymphatic transport.**

These are not for exact memorization but you should link each deficiency to its manifestations

Manifestations:


- Weight loss, anorexia,
- Flatus, abdominal distention, Due to the bacterial reactions against the accumulated materials (food), especially in carbs malabsorption
- Borborygmi (intestinal noise), Muscle wasting
- Anemia and mucositis (iron, pyridoxine (VB6), folate, or vitamin B12 deficiency)
- Bleeding (vitamin K deficiency)
- Osteopenia and tetany (calcium, magnesium, or vitamin D deficiency)
- Neuropathy (vitamin A or B12 deficiency)
- Skin and endocrine disorders.

Cystic Fibrosis

- Mutations in cystic fibrosis transmembrane conductance regulator (CFTR)
- Defects in ion transport across intestinal and pancreatic epithelium.
- Thick viscous secretions.
- Mucus plugs in pancreatic ducts >>> pancreatic insufficiency (80% of patients)
- Meconium ileus in neonates.
- Defect in intraluminal digestion.

Treatment(to the symptoms not the disease itself) :oral supplements of pancreatic enzymes

Celiac Disease

- *Gluten sensitive enteropathy* 
- Immune mediated enteropathy
- Wheat, rye or barley.
- Genetically predisposition, HLA-DQ2 or HLA-DQ8.
- Treatment: gluten free diet.

- Association with: type 1 diabetes, thyroiditis, and Sjogren syndrome

It is associated with other autoimmune diseases ,like (DM1,...)

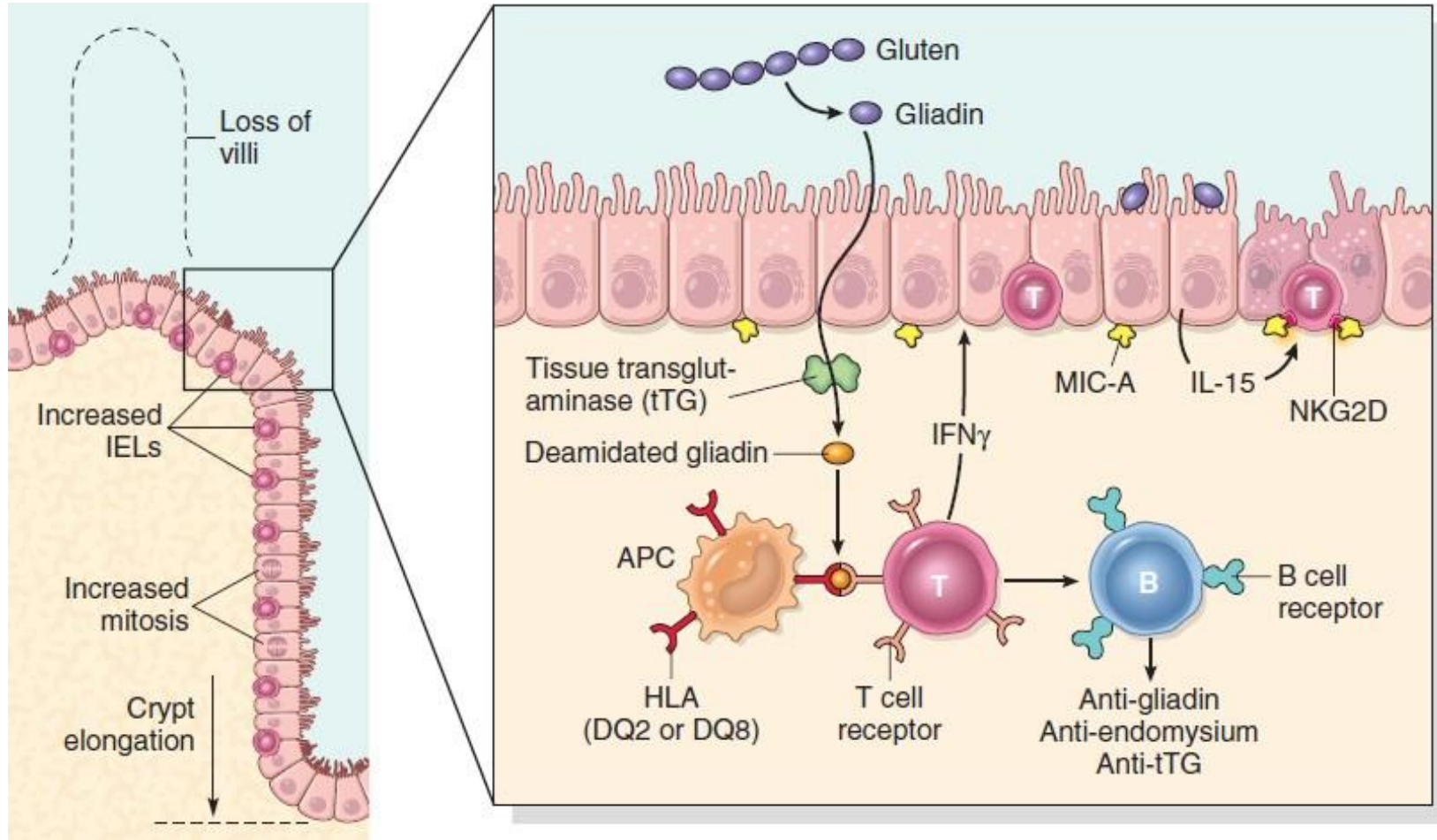
Pathogenesis

- Gluten >>> gliadin >>> react with HLA-DQ2 or HLA-DQ8 on antigen-presenting cells >>> CD4+ T cells activation >>> cytokines >>> tissue damage.

It also leads to B-cells activation and antibodies secretion

- Serology:
- Anti- tissue transglutaminase antibodies
- Anti-gliadin antibodies.
- Anti -endomysial antibodies

so to diagnose the patient we can do that clinically , serology and biopsy(especially by observing the difference in villi shape)



Robbins Basic Pathology 10th edition

MORPHOLOGY

- Second portion of the duodenum or proximal jejunum.
- **Triad**: intraepithelial lymphocytosis (CD8+ T cells), crypt hyperplasia, and villous atrophy.
- Lamina propria: lymphocytes, plasma cells, eosinophils.....
- IEL & villous atrophy are not pathognomonic, seen in viral enteritis.

- Diagnosis: Clinical, histologic and serologic correlation.

Normal intestine

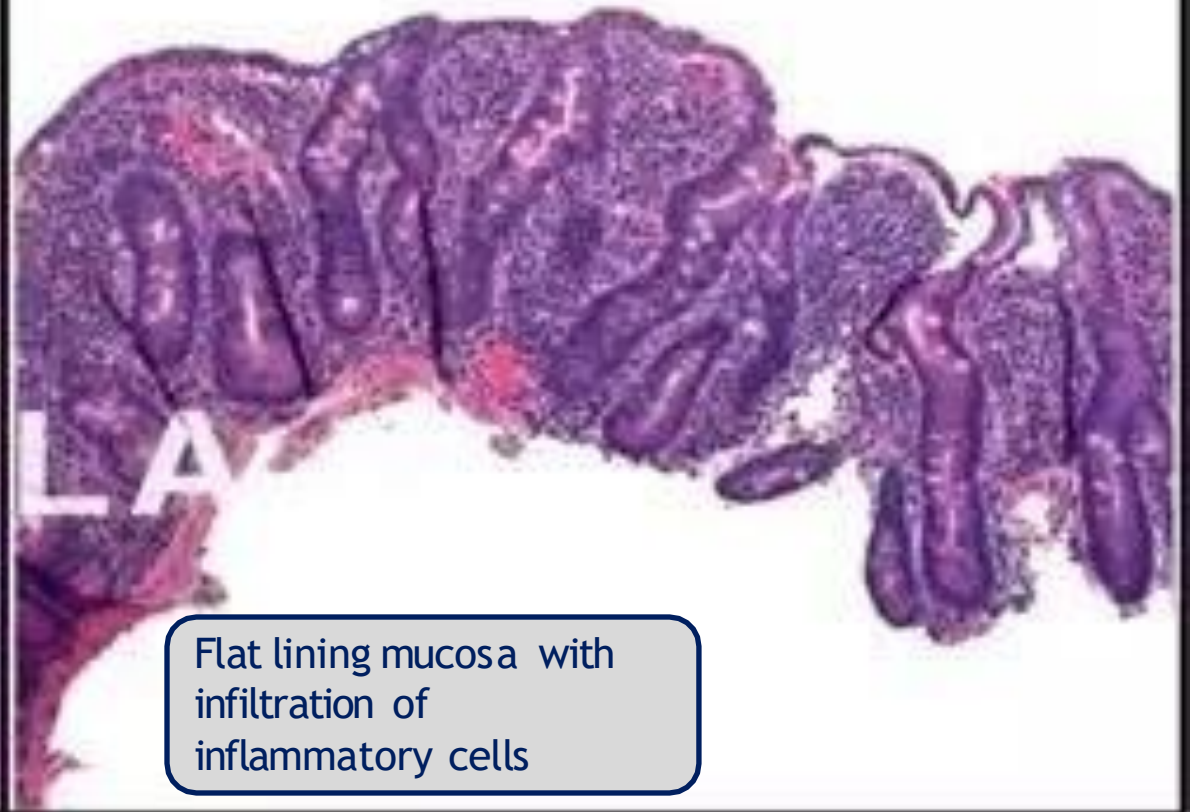
Finger-like projections



Normal



Celiac Disease



UC
LAY

Flat lining mucosa with
infiltration of
inflammatory cells

V2

more info has been added Highlighted in yellow