

# **Pathology**

Sheet

OSlide

number

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# **Small & Large Intestines**

# **Intestinal obstruction:**

Obstruction occurs more frequently in the small intestine because it's *the* <u>small</u> intestine (relatively narrow lumen); it's easier to block a small tube than it is to block a large tube.

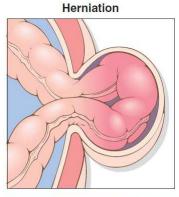
The **4 most common causes** of intestinal obstruction that you will see are:

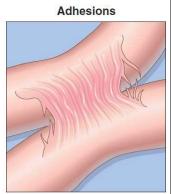
- Herniation
- Adhesions
- Volvulus
- Intussusception

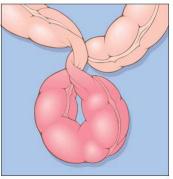
### 1. Herniation:

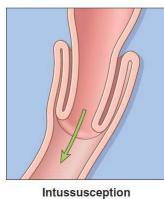
\*But what is a hernia??

Bits of the internal abdominal organs moving to a place where they shouldn't be through, typically, a narrow canal. Sometimes the canal is not so narrow but a narrow canal is what we worry about.









- \*What kind of places would you find abdominal organs herniating to?
- Hiatal hernia
- Lumbar hernia

Umbilical hernia

Volvulus

- Femoral hernia (rare)
- Inguinal hernia
- Surgical hernia: caused by doctors. When a doctor had operated on a
  patient in the past and the sutures opened or there was an infection (the
  area didn't come back to its full strength after repair) and there was too
  much abdominal pressure, it could herniate through the wound either
  covered or uncovered with skin.

The problem with hernias is when the opening that the hernia slights to is **narrow** like the inguinal canal.

\*What happens when it's narrow and it starts constricting on whatever slits through?

The first thing that is going to be compromised is **the vein** because it is thin walled. When you obstruct venous outflow it will end up with **edema**. You're going to have increased hydrostatic pressure pushing out to the extracellular fluid (the interstitial space) ending up with a heavier organ than it was before, raising the pressure inside the hernial sac and then obstructing the artery. When the artery gets obstructed you end up with **ischemia and necrosis**.

A lot of people with hernias can manipulate them back into the abdomen. When it doesn't go back in it is called an **incarcerated hernia**. When a hernia becomes obstructed and you start getting ischemia it is called a **strangulated hernia**. Incarcerated and strangulated hernias are problematic especially in the inguinal canal.

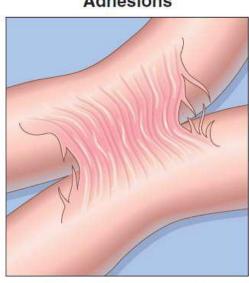
- \*When your doctor asks you to cough what is he looking for?
- A flow of peritoneal fluid into the hernial sac (increase in pressure).

#### 2. Adhesions

- \*What are adhesions?
- -Fibrous tissue.

Inflammation (e.g. from peritonitis, previous surgery, etc.) that results in repair not by regeneration <u>but by fibrosis</u>, can cause all sorts of adhesion bands (fibrous bands) that can encompass different loops of bowel and **obstruct** them.



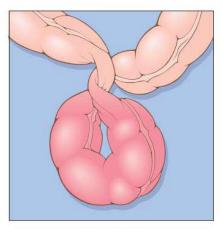


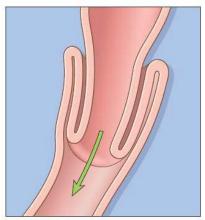
#### 3. Volvulus

If the area in your peritoneum is not very well packed (normally it is well packed) because of a surgery, etc. and you have much peritoneal fluid, loops of bowel can somehow twist on themselves cutting off their own lumina and potentially their blood supply.

## 4. Intussusception

When something inside the bowel is getting caught and material flows through, it pulls the upper part of the bowel into the lower part of the bowel. Generally, the thing that gets caught in the fecal flow is a large polyp or sometimes a tumor. As the feces or the food matter goes through, it pulls on that part from the inside pulling one bit of bowel into the other compromising the lumen and leading to obstruction.





Volvulus

Intussusception

Most of intestinal obstructions (more than 80% by western calculations) are caused by these four. The rest for the most part can be caused by <u>tumors</u> and <u>infarction</u>(Ischemia-reperfusion). In general, tumors have to get pretty large to cause obstruction. So, obstruction is a late presenting symptom of an intestinal tumor. (There are more but much rare causes)

Whatever the cause is (whether hernias, intussusception, volvulus, or ischemic bowel....), the general shared **signs and symptoms** of obstruction are going to be:

- Pain
- Distention
- Vomiting: If the obstruction is <u>high</u> in the GI tract.
- Constipation: If the obstruction is <u>low</u> in the GI tract.

If it is a particularly low obstruction (that causes <u>constipation</u>) and the patient ignores that he has constipation for a long period and keeps stuffing his intestine with food, that patient will eventually also present with <u>vomiting</u>.

## **Hirschsprung Disease**

Obstruction is not always acquired, it can be congenital. <u>Congenital</u> obstructions from **Hirschsprung Disease** occur in about 1 in 5000 births (western numbers). This congenital defect is in the <u>innervation</u> of the colon.

The colon has 2 plexuses: Auerbach's (myenteric) and Meissner's (submucosal) plexuses. These plexuses result from nerves travelling from the neural crest (in embryo); starting at the cecum, going up the ascending colon, across the transverse colon, down the descending colon all the way to the anorectal line. If there should be a problem affecting this embryological process preventing the nerves from reaching their final destination, you are going to end up with a region of colon that has no plexuses.

When a bit of intestine or the esophagus or any other part of the GI tract undergoes peristalsis, there is contraction and relaxation. When an area contracts, the <u>distal</u> one relaxes allowing the pressure wave to propagate distally towards the anus. *But* when the peristalsis (pressure wave) reaches the aganglionic region (where there is NO nerve plexuses), there won't be relaxation and it will cause **functional obstruction** (it is a small tube that is not relaxing, so pressure will accumulate leading to obstruction).

When you do a barium enema to the patient, you will find that the aganglionic diseased region is actually normal looking and the proximal non-diseased area is the one that is dilated. This is because peristalsis is reaching this functional obstruction and the pressure is accumulating there. To diagnose Hirschsprung disease you need to take a biopsy from the non-distended region (which is distal to the distended region).

This disease can occur on its own or with other congenital malformations. So, if you find one congenital malformation you look for the rest. It occurs more commonly in males but more severely in females.

The **presentation** for these patients is:

- Failure to pass meconium:
  - Meconium is the first feces after birth.
  - It contains amniotic fluid and some cells that are sloughed off.

- It is generally sticky, black, and shiny.
- If the mom was not educated and she didn't notice the baby hadn't pass meconium, the second symptom that is going to occur is obstructive constipation.

## Obstructive constipation:

- The child gets more and more distended, not feeding well, is constantly in pain, and essentially all the signs and symptoms we talked about in obstruction in adults will occur. But the thing is the child cannot tell you what's wrong.
- The problem is when it goes undetected for too long. If this happened it could result in fluid and electrolyte imbalances and if it goes for long enough it can result in perforation, peritonitis and death. So don't miss an **aganglionic megacolon**.

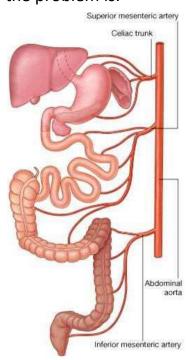
**Treatment:** Surgical resection. Take out the region that does not have any ganglionic cells or nerves and reconnect the proximal region to the anus. But here lies a problem!!

The colon absorbs water and electrolytes. So if you take a big chunk of the colon out, the stool will be watery. This makes it harder for these children to be potty-trained. The bigger the resected area, the bigger the problem is.

# **Ischemic Bowel Disease**

There are three major **arteries** that supply the stomach, small intestine, and large intestine which are:

- Celiac trunk: supplies the stomach and a bit of the duodenum.
- Superior mesenteric artery: supplies the rest of the small intestine and the large intestine up to the splenic flexure.
- Inferior mesenteric artery:from the splenic flexure downward.



Any time you have an overlap of the two major suppliers (celiac with sup. mesenteric or sup. mesenteric with inf. Mesenteric), this area is called a **watershed zone**. These areas need supply from BOTH major arteries to stay alive. They are the first areas to be affected if a patient is <u>hypotensive</u>; one supply is NOT enough. So if you are to have pain resulting from the splenic flexure specifically, the first thing you should worry about is ischemic bowel.

- \*What patients are typically hypotensive?
- Elderly people with coexisting cardiovascular disease, people who have heart failure because they are not providing enough blood to their organs.

Ischemia can result in several types of damages:

- **Mucosal:** only the mucosal membranes in the intestine are affected.
- Mural: the mucosa and submucosa are affected
- Transmural: all three layers are affected. It is the most severe because it could end up with spilling the intestinal content after necrosis into the peritoneum. The intestine contains enzymes like pancreatic lipases which cause <u>fat necrosis</u>, and more importantly bacteria that cause <u>peritonitis</u> which is a medical emergency. Results typically from an *acute obstruction* of vessels i.e. a thrombus/an embolus that came from cardiac vegetations (as in bacterial endocarditis) or a Marfan syndrome patient who has abdominal aortic aneurysm. Also, oral contraceptives in a small percentage of patients can cause increased thrombosis and ischemic bowel.

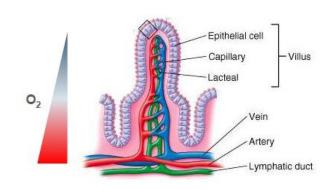
For mural and mucosal infarction we are talking about *long standing ischemia* i.e. elderly patients and cardiovascular disease (e.g. atherosclerosis).

The outcome mostly depends on severity of compromise, duration, and which vessel is involved.

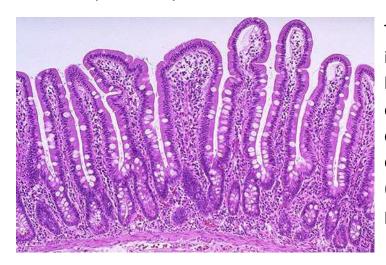
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\*Why is the mucosa affected first and not the outside of the intestine?

- The blood supply to your small or large intestine goes first to the crypt up to the villus and then turns back and comes back down. This happens because the stem cells are present in the stem cell niches at the bottom of the crypt and any time you lose cells you will get them from these stem

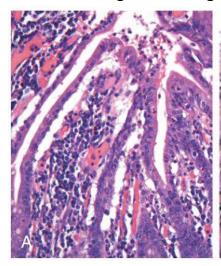


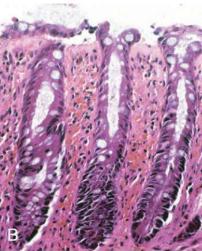
cells. Intestinal cells are labile cells (steady state population) and every time you lose a cell you are going to proliferate and replace that cell. So that is the most important area to maintain. Anytime you got ischemia you are going to have the stem cells to replenish but if you lose the stem cell compartment you can't.



This is how a normal small intestine should look like; having villi, different types of cells, and lots of stem cells at the bottom of the crypt that are proliferative (but not hyperproliferative).

## But looking at the image below:





- (A) acute ischemia
- (B) chronic ischemia

This is what an ischemic intestine looks like whether acute (A) or chronic (B).

\*How to differentiate between them?

- Chronic ischemia results in fibrosis and collagen stains eosinophilic under the microscope. In acute ischemia there is atrophy or sloughing of surface epithelium off of the villi.

There are inflammatory cells and sloughing of epithelium in both but the most important thing that is in chronic ischemia there is <u>eosinophilic</u> <u>staining</u>.

#### Clinical features of ischemic bowel disease:

- Older patient with co-existing cardiovascular disease.
- Severe pain and guarding (in a fetal position) because the abdominal wall muscles will start contracting uncontrollably and become rigid to quard you form anybody poking you.
- Nausea and vomiting especially if it is higher up.
- Bloody diarrhea if it is lower down.
- Reduced peristalsis: if you take a stethoscope and listen to the patient's stomach and you don't hear noises (from the peristaltic waves) this means the patient is in trouble.
- Blood loss → Shock and hypovolemia which means you are in further trouble because hypovolemia is going to extend to other places and bigger areas in the intestine are going to be compromised.
- Sepsis: if you reach transmural infarction and you have exposure of your blood supply to bacteria causing sepsis and death.

**DDx (differential diagnosis):** Acute appendicitis (AA), acute cholecystitis (AC), and perforated ulcer(PU). All three are common especially AA and AC. If you misdiagnose an ischemic bowel as an AA or AC patient you have condemned them to death. So if a patient presents with these symptoms and you suspect acute appendicitis and you do not do what you have to do to find whether it is ischemic bowel or not you are medically liable. So whenever you see these symptoms never miss ischemic bowel disease especially if it's an older patient.

## **Hemorrhoids**

Hemorrhoids are dilated anal and perianal collateral vessels.

Most patients who go into GI surgery are going to be hemorrhoid patients. It is almost limited to older males but it can also occur in pregnant females and old females (less common).

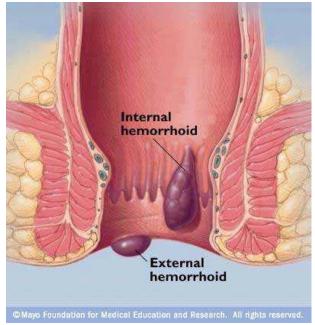
Hemorrhoids result from another portosystemic anastomosis (the first is in the esophagus and stomach). The esophageal varices are a medical emergency if they burst and most patients die especially if they are undiagnosed. On the other hand, hemorrhoids, which can result from portal hypertension, are not a medical emergency and are just painful.

Even though hemorrhoids can **result from** <u>portal hypertension</u> but it is <u>not</u> the most common cause. The most common cause is <u>increased intra-abdominal pressure</u>. That's why pregnant females get hemorrhoids. Old people get hemorrhoids because they stop eating proper amounts of fiber, so they are chronically constipated and straining cause increased abdominal pressure leading to hemorrhoids.

## Types of hemorrhoids:

- **1. Internal hemorrhoids:** occur above the anorectal line and are covered with intestinal lining.
- **2. External hemorrhoids:** occur below the anorectal line and are covered with skin.

We worry about hemorrhoids because they are so painful especially when trying to sit down.



Hemorrhoids themselves are not painful but they get injured, inflamed, and thrombosed which ends up causing pain. If internal hemorrhoids are particularly big, they can prolate outside the anorectal line and get strangulated and that can also cause ischemic pain.

Clinical Features: Pain and bleeding (bright red)

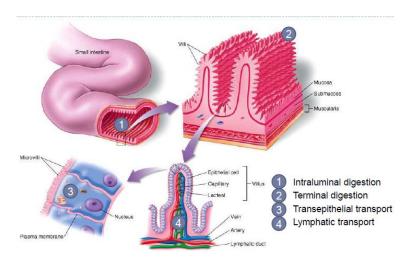
#### **Treatment:**

- **Sclerotherapy:** you inject something that thromboses the inside.
- **Banding:** you take a rough band and put it at the origin of the hemorrhoid strangulating it so it would die and fall off.
- **Infrared coagulation:** you inject something and then use infrared light (high energy) to coagulate blood.
- The last option is **surgery** because you are working very close to a particularly very important sphincter (the anus). You only go to surgery if it is problematic and large. First, you try to do something without surgery or risking the sphincter.

22:00-34:00

# **Digestion**

There are 4 phases of digestion:



**1. Intraluminal digestion:** done by pancreatic enzymes, some bacteria, and stomach acids.

#### 2. Terminal digestion:

Breaking disaccharides such as lactose. If you are missing the brush border lactase you are lactose intolerant, and if you drink milk you end up with pain and gases (because bacteria are breaking lactose by fermentation producing  $CO_2$ ).

- **3. Trans-epithelial transport:** After you have broken down proteins and sugars to their basic components you must get them from one side to the other side. Your epithelial cells in the intestine will take care of moving them from one side to the next.
- **4. Lymphatic transport:** You transport fat to the lymphatics because you can't move lipids into your blood stream (mostly water) unless you move them in the form of lipoproteins.

If someone has a problem with any one of these 4 digestion phases, he will end up with one disease or another.

#### Examples:

• If someone has **cystic fibrosis** causing a blockage of his exocrine pancreas\* and he ends up with decreased <u>intraluminal digestion</u>.

\* Chloride transport is broken and rather than producing fluidy mucus you start producing very viscous cloggy mucus that can block exocrine pancreas duct. If you have pancreatic insufficiency you will also end up with decreased intraluminal digestion.

- If someone has an infection of the lymphatic system with a particular microorganism that results in problems in transporting fat (Whipple disease), affecting <u>lymphatic transport</u>.
- You don't necessarily have to have one problem, you could have multiple problems. Celiac disease for example can cause terminal and transepithelial transport problems because of the resulting inflammation.
- Disaccharidase deficiency or lactose intolerance (altough other disaccharidases can be deficient). It is an example of terminal digestion problems.
- You could have all 3 involved like in inflammatory bowel disease where you have problems with intraluminal digestion, terminal digestion, and transepithelial transport.

*NOTE:* The table in the next page is not for memorization. However, try to understand the outliers and recognize the ones that are special and we are going to talk about.

				4
Disease	Intraluminal Digestion	Terminal Digestion	Transepithelial Transport	Lymphatic Transport
Celiac disease		+	+	
Tropical sprue		+	+	
Chronic pancreatitis	+			
Cystic fibrosis	+			
Primary bile acid malabsorption	+		+	
Carcinoid syndrome			+	
Autoimmune enteropathy		+	+	
Disaccharidase deficiency		+		
Whipple disease				+
Abetalipoproteinemia			+	
Viral gastroenteritis		+	+	
Bacterial gastroenteritis		+	+	
Parasitic gastroenteritis		+	+	
Inflammatory bowel disease	+	+	+	

# **Malabsorption Syndromes:**

All malabsorption syndromes are fairly similar; they share a lot of the same signs and symptoms and consequences of malabsorption. Some will have more of the consequences or symptoms highlighted above the others but essentially if you find the following symptoms your differential diagnosis is all of the malabsorption syndromes.

## The symptoms:

- -Diarrhea: is watery stools with increased mass (some diarrhea can reach 40L a day) and increased frequency of going to the bathroom.
- -Flatus
- -Abdominal pain
- Weight loss: if it is long-standing.

#### **Consequences:**

- -Anemia: pyridoxine deficiency.
- -Mucositis: folate and vitamin B12 deficiency.
- -Bleeding: vitamin K deficiency.
- -Osteopenia: Ca and Mg deficiency.
- -**Tetany:** vitamin D deficiency.
- Neuropathy: vitamin A or B12 deficiency.



