Adapted from/inspired by: *Atlas of Gastrointestinal Pathology*. Arnold et al. 2015
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Patterns of GI Tract Injury

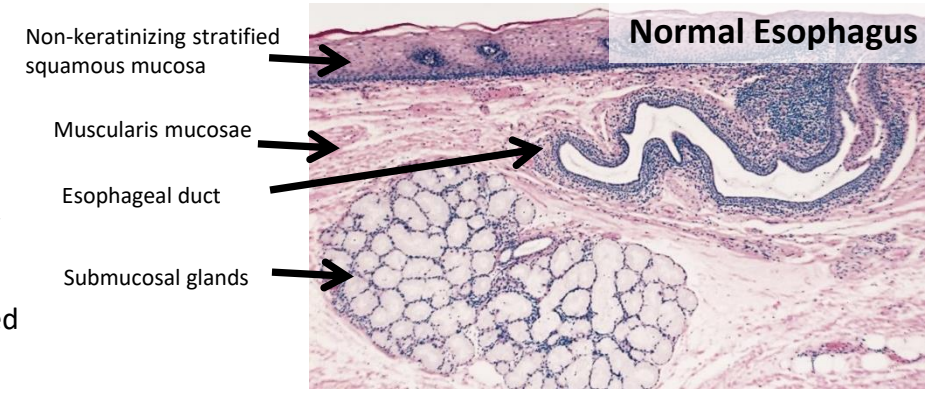
Esophagus

Benign Incidental Findings:

Gastric "Inlet Patch"- Heterotopic gastric mucosa in esophagus

Pancreatic Heterotopia/Metaplasia

Glycogenic Acanthosis – Epithelial hyperplasia with abundant, enlarged superficial glycogenated cells; clinically appears white

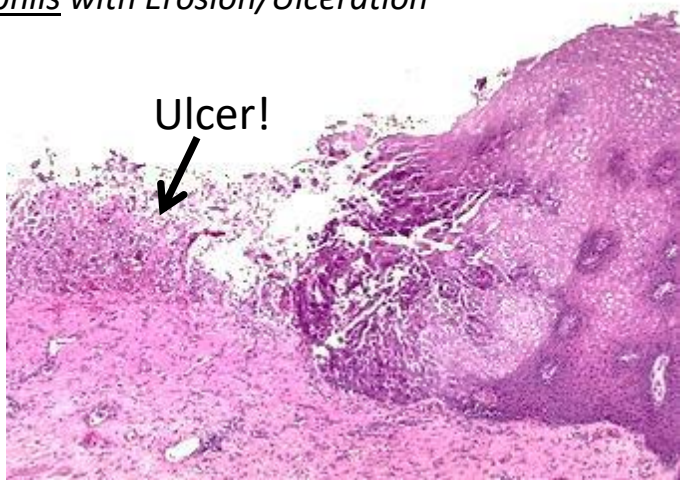


(Muscularis propria: distal = smooth muscle; proximal = skeletal muscle)

Acute Esophagitis

Intraepithelial Neutrophils with Erosion/Ulceration

GERD Often scattered Eos (usu. < 15/HPF), Intercellular edema, Basal cell hyperplasia, Elongation of vascular papilla. Worse distally (near GEJ).



Infections

Candida - Look for fungal hyphae, Get PAS-D/GMS

HSV - Look for Molding, Multinucleation, Margination in epithelial cells.

CMV – Look for inclusions in mesenchymal cells.

Medications *("Pill esophagitis")* Look for crystals, resins, and pill fragments; Polarize to help looking for foreign material.

Eosinophilic Esophagitis

Increased intraepithelial Eosinophils **(report per HPF)**

GERD

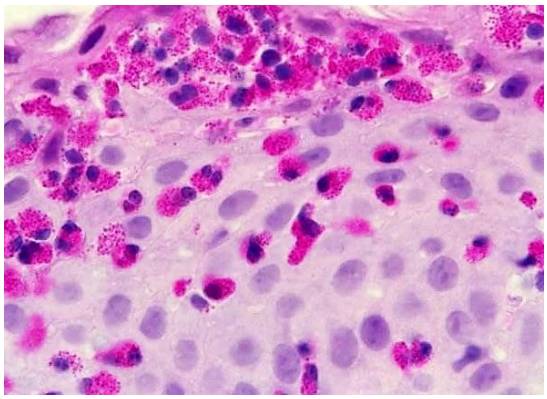
Eos typically < 15/HPF, Intraepithelial T lymphocytes ("squiggle cells"), Intercellular edema, Basal cell hyperplasia, Elongation of vascular papilla. Worse distally (near GEJ).

Eosinophilic Esophagitis

Typically, >20 Eos/HPF. Often eosinophilic microabscesses with degranulation. Often diffuse or worse proximally. Associated with "Atopic Triad" (Allergies, Asthma, Eczema). Presents with dysphagia, chest pain, food impaction, which may cause a food aversion. Endoscopically can appear as rings or furrows ("Trachealization/felinization")

Allergies/Systemic autoimmune disorders

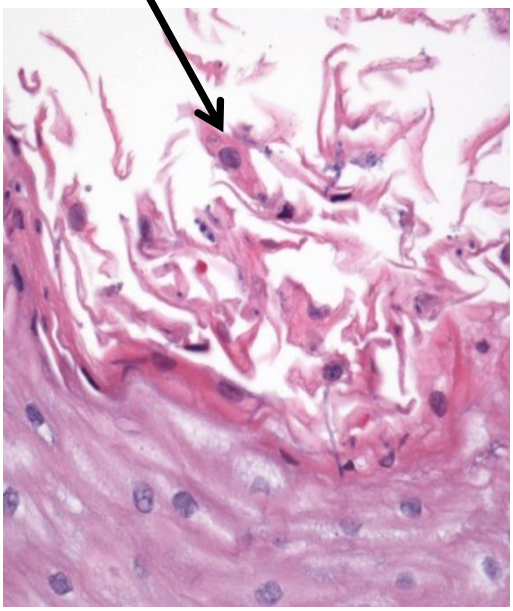
Medication Reaction



Note: *As EoE and GERD can appear identical on a single bx, close clinical and endoscopic correlation is often necessary to distinguish between the them!*

Parakeratosis Pattern

Superficial squamous cells with retained nuclei



GERD

Eos typically < 15/HPF, Intraepithelial T lymphocytes ("squiggle cells"), Intercellular edema, Basal cell hyperplasia, Elongation of vascular papilla. Worse distally (near GEJ).

Candida Esophagitis

Look for fungal hyphae at surface and get PAS-D or GMS, particularly in immunosuppressed individuals. Budding yeast are NOT good enough!

Esophagitis Dissecans Superficialis *("Sloughing Esophagitis")* Superficial "mummified" layer (with ghost nuclei) with variable necrosis and minimal inflammation. Clinically can be quite dramatic with extensive peeling and fissuring. Has been associated with thermal injury, medications, and some autoimmune conditions.

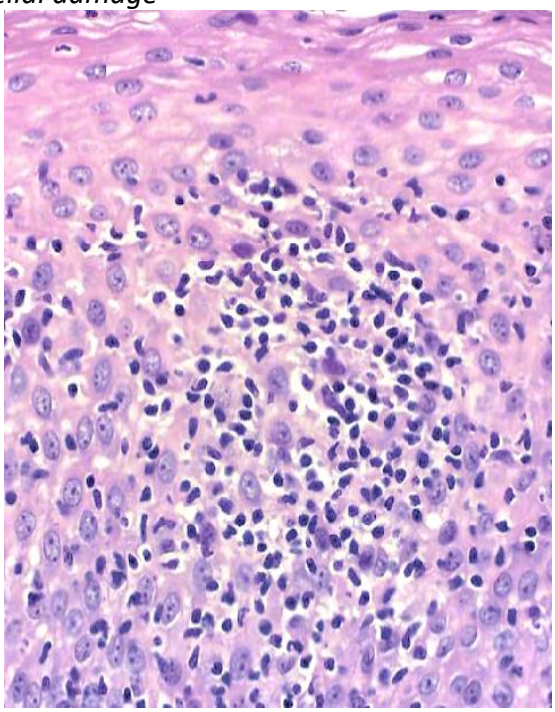
Esophageal Leukoplakia/Epidermoid Metaplasia

Looks like skin with keratinization and a granular layer. Associated with motility disorders and oral leukoplakia. Possible increased risk of squamous dysplasia/carcinoma.

Lymphocytic Pattern

Intraepithelial Lymphocytes (with few PMNs/Eos)

How many are too many? No strict cutoff, but >30/HPF is likely too many, esp. diffusely and with epithelial damage



GERD

Lichen Planus

Band-like ("lichenoid") infiltrate at junction between epithelium and submucosa with basal degeneration. Dyskeratotic keratinocytes ("Civatte bodies") are common. Associated with cutaneous LP, certain medications, and viral infections. Often older women. Can get strictures.
Risk of dysplasia → SCC

Graft Versus Host Disease

Donor T lymphocytes attack host tissue. Typically present with Rash, Diarrhea, elevated LFT's.
Intraepithelial lymphocytes with dyskeratotic keratinocytes and scattered apoptotic bodies. Make sure CMV IHC is neg.

Crohn's Disease

Esp. in kids. Look in lamina propria for granulomas

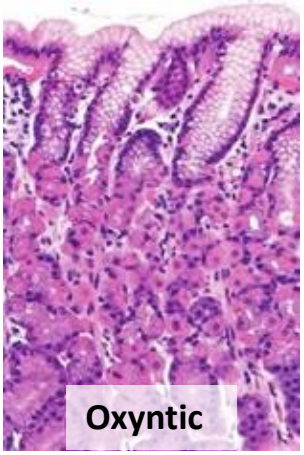
"Contact Mucositis"

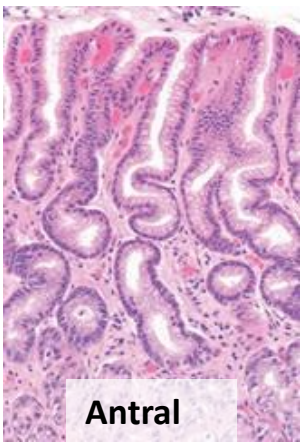
May be a generalized response to mucosal injury, for example to an allergy to a medication or food.

Other

CVID, Celiac disease, Dysmotility, connective tissue disease, Etc…

Stomach





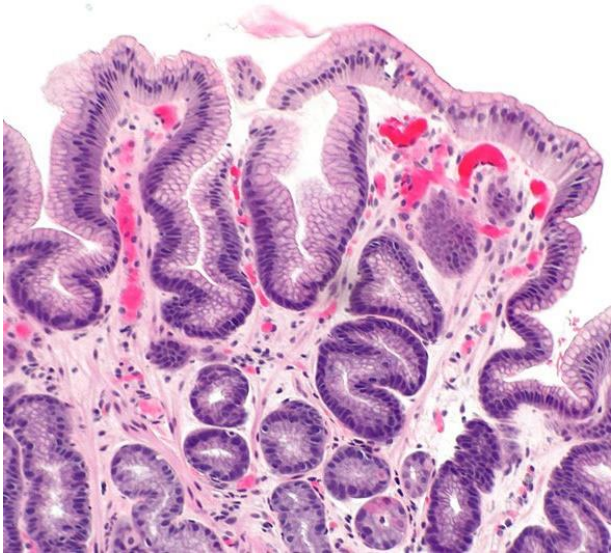
Oxyntic Mucosa (90% of stomach)
Present in body/fundus
Pink parietal cells make acid and intrinsic factor (B12 uptake)
Purple chief cells make pepsinogen

Antral Mucosa
Present in distal antrum and cardia

Gastrin-secreting G cells are found ONLY in antrum

Usu. extremely few inflammatory cells, except at the gastric cardia, which commonly has some chronic inflammation.

Reactive (Chemical) Gastritis/Gastropathy



Foveolar hyperplasia ("corkscrew glands"), Mucin depletion, Edema, Minimal inflammation, Extension of smooth muscle bands between glands

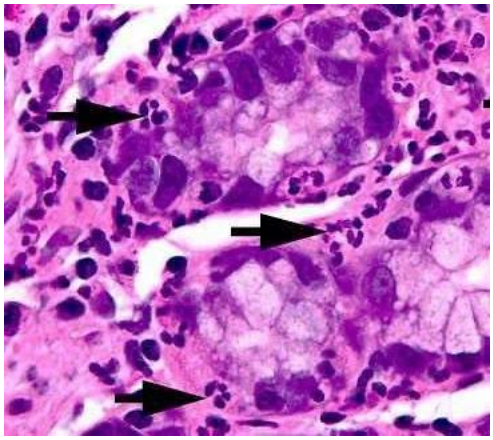
Often caused by **chemical irritation** by bile reflux, medications (particularly NSAIDs), or alcohol.

Portal Hypertensive Gastropathy
Above findings, plus dilated vessels in lamina propria. Seen in patients with portal hypertension.
Endoscopically like "snake skin"

Gastric Antral Vascular Ectasia ("GAVE")
Endoscopically looks like a watermelon. Above findings, plus fibrin thrombi present in lamina propria capillaries.

Acute Gastritis

Intraepithelial Neutrophils often with Erosion/Ulceration



Helicobacter pylori
Acute gastritis with characteristic superficial lymphoplasmacytic inflammation and prominent lymphoid aggregates. Most common in Antrum. Look hard in pits and consider getting Helicobacter IHC. Risk of MALT and dysplasia/carcinoma.

Helicobacter heilmannii
Less acute inflammation. More common in kids. Organisms are longer, more tightly spiraled, and less numerous

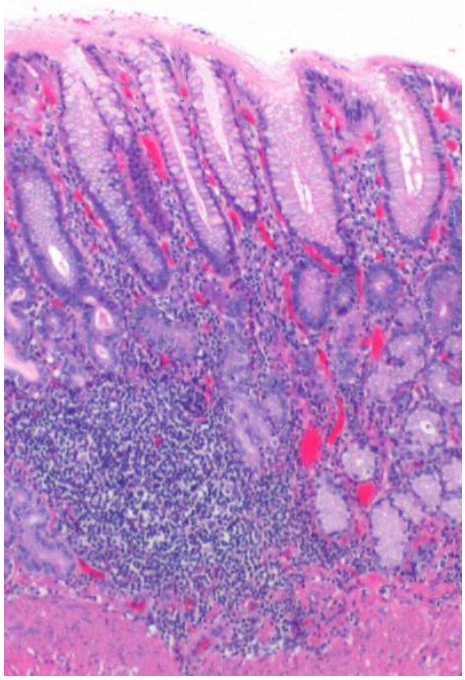
Medications
Esp. NSAIDs. Often associated ischemic or reactive changes.

"Focally Enhanced Gastritis"
Focally injured glands surrounded by inflammation. Associated in kids with IBD, particularly Crohn's disease.

CMV

Chronic Gastritis

Chronic inflammation in the Lamina propria



Helicobacter pylori/heilmannii

Acute gastritis with characteristic superficial lymphoplasmacytic inflammation and prominent lymphoid aggregates. Most common in Antrum. Get IHC stain if you don't see any organisms.

Autoimmune Metaplastic Atrophic Gastritis (AMAG)

Also known as autoimmune gastritis. Autoantibodies destroy parietal cells/oxyntic mucosa → No intrinsic factor → B12 deficiency → Pernicious anemia.

Body-predominant injury with loss of oxyntic mucosa and Deep chronic inflammation → Intestinal and pyloric metaplasia & ECL cell hyperplasia → Can make neuroendocrine tumors (type I)

Gastrin stain can help confirm sample came from body (negative) and not antrum (positive).

Medications

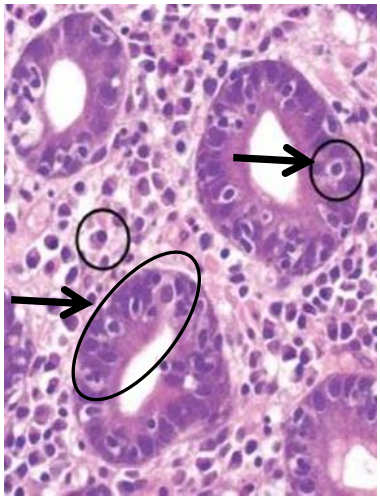
Esp. NSAIDs. Often associated ischemic or reactive changes.

Other

CVID, Celiac disease

Lymphocytic Gastritis

Intraepithelial Lymphocytes



Helicobacter

Celiac Disease

Medications (E.g., Ticlopidine, Olmesartan)

HIV

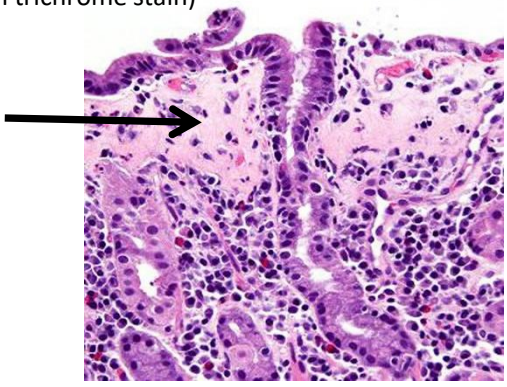
Other Immune-mediated Disorders

CVID, Crohn's Disease, Lymphocytic colitis, etc…

Lymphoma

Collagenous Gastritis

Increased subepithelial collagen band with Intraepithelial Lymphocytes (can highlight with trichrome stain)



Collagenous colitis/enteritis

Celiac Disease

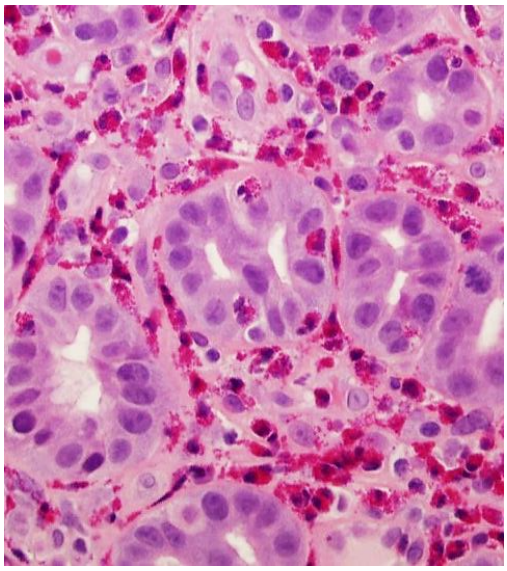
Medications (E.g., Olmesartan)

Helicobacter

Other Immune-mediated Disorders

Eosinophilic Gastritis

Increased Eosinophils



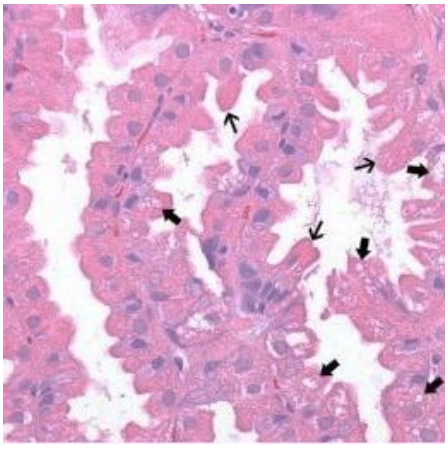
Although there is no strict cut-off, >30/HPF is likely too many and any in the epithelium, submucosa, or muscle is abnormal

Eosinophilic Gastritis/Gastroenteritis
Diagnosis of exclusion. Can be associated with Eosinophil-rich inflammation in other organs (e.g., esophagus and/or small bowel). Layer of bowel involved determines symptoms.

Helicobacter
Parasites
Connective tissue diseases/Vasculitis
Food Allergies
Medications
Inflammatory bowel disease (particularly Crohn's)

Oxyntic Gland Hyperplasia

Dilated oxyntic glands with hypertrophic parietal cells with "snouts"



Associated with **Proton Pump Inhibitor use** (increases gastrin levels through feedback, causing parietal cell hypertrophy).

Single/Sporadic Polyp → Fundic Gland Polyp
Extremely low risk of dysplasia/progression

Innumerable or Dysplastic? Consider a Syndrome:

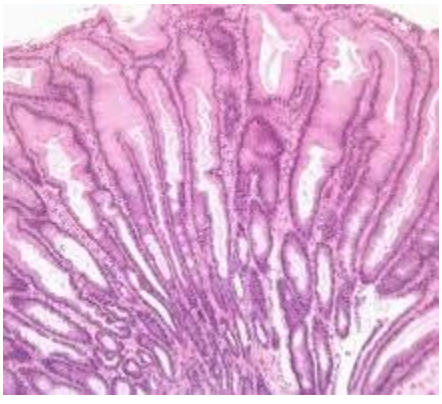
Familial Adenomatous Polyposis Can become dysplastic, but still low rate of progression to carcinoma

MutYH-Associated Polyposis

Zollinger-Ellison Syndrome
Gastrinoma (usu. in small bowel) causes increased acid secretion and ulcers. Associated with MEN1.

Foveolar Hyperplasia

"Corkscrew glands," Mucin depletion, Edema



Single/Sporadic Polyp → Hyperplastic Polyp
Associated with background inflammatory injury.
Extremely low risk of dysplasia/progression

Innumerable or Dysplastic? Consider a Syndrome:

Ménétrier's Disease Whole stomach. Rare, acquired.
Causes protein-losing enteropathy. In kids, associated with CMV.

PTEN Syndromes (Cowden's, etc…)
Cronkhite-Candada Syndrome
Juvenile Polyposis
Peutz-Jeghers Syndrome

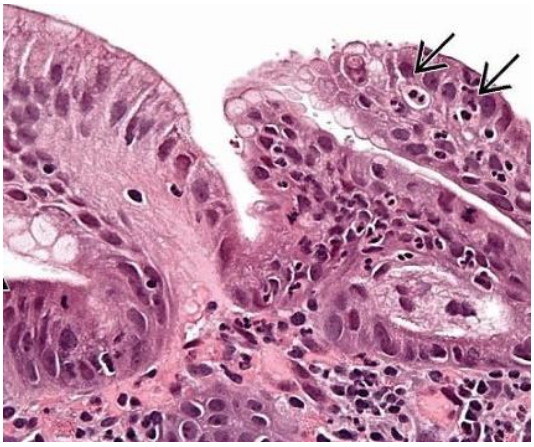
Small Intestine

Quick Checklist:

- Villi? Long and skinny? Go away or blunt with Celiac Disease
- Goblet Cells? Go away with autoimmune enteropathy
- Intraepithelial lymphocytes? Increased in Celiac (and others)
- Plasma cells? Go away with CVID
- Critters? Look between villi and on surface for Giardia, etc..
- Vessels ok? Look for amyloid and vasculitis
- Endocrine cells? Go away with endocrine dysgenesis

Acute Duodenitis

Neutrophils in duodenal epithelium



Peptic Duodenitis
Additionally see Gastric foveolar metaplasia and chronic inflammation. Associated with excess gastric acid and/or *Helicobacter*

Infection Most commonly *Helicobacter* (can lead to ulcers → Peptic Ulcer Disease). Sometimes Adenovirus, CMV, or other viruses.

Medications Most commonly NSAIDs

Inflammatory bowel disease (particularly Crohn's)

Acute Ileitis

Neutrophils in Ileal epithelium

Medications Most commonly NSAIDs

Infection Including common stool pathogens (bacterial and viral)

Inflammatory bowel disease Crohn's disease is more likely to impact TI (so look for granulomas, and signs of chronicity, including pyloric gland metaplasia). In UC, there is typically inflammation in the nearby cecum (that is thought to "backwash")

Eosinophilic Gastroenteritis

Increased Eosinophils

Although there is no strict cut-off, >60/HPF is likely too many and any in the epithelium, submucosa, or muscle is abnormal

Eosinophilic Gastroenteritis
Diagnosis of exclusion. Can be associated with Eosinophil-rich inflammation in other organs (e.g., stomach or colon).
Layer of bowel involved determines symptoms.

Parasites

Connective tissue diseases/Vasculitis

Food Allergies

Medications

Inflammatory bowel disease (particularly Crohn's)

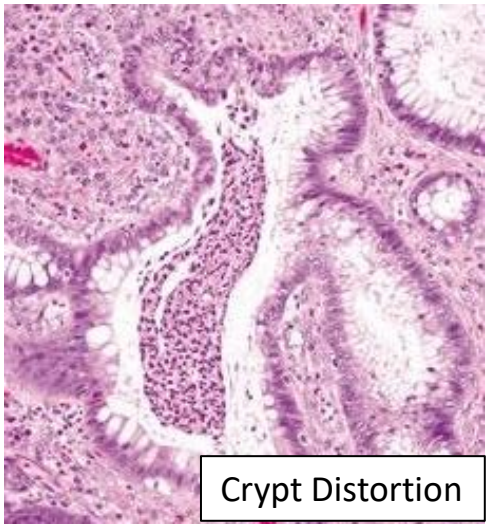
Eosinophilic Gastroenteritis Symptoms	
Layer	**Symptoms**
Mucosa	Diarrhea, malabsorption
Muscle	Ileus
Serosa	Ileus and ascites

Chronic Injury

Architectural distortion: crypt branching, dropout, and pyloric gland metaplasia often with villous blunting and a basal lymphoplasmacytosis

Often starts to look like colon with IBD!

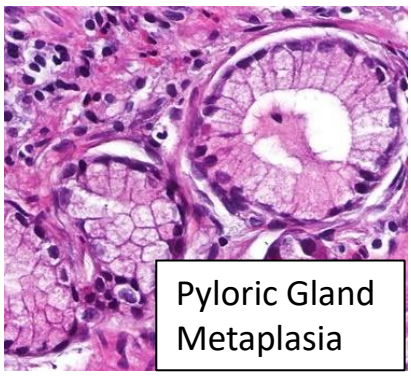
Inflammatory Bowel Disease
Chronic ACTIVE inflammation, with cryptitis and crypt abscesses.
Particularly Crohn's in the TI (and small bowel in general).
Look for granulomas and transmural inflammation in resections.


Crypt Distortion

"Diaphragm Disease" Due to NSAIDS
Mild → erosions with associated acute inflammation
Severe → multiple episodes can cause scarring → stenosis
Usu. Less chronic inflammation than Crohn's

Medications
Mycophenolate – Immunosuppressant (often given after transplantation) that can cause epithelial/crypt damage with increased apoptosis→ causes diarrhea

Ischemia Severe pain. Coagulative necrosis. Crypt withering. Lamina propria hyalinization and hemorrhage. Reperfusion brings acute inflammation.


Pyloric Gland Metaplasia

Radiation Most sensitive to damage. Endothelial injury → edema, fibrin, and ischemic changes with enlarged/bizarre nuclei.

Graft vs. Host Disease (GVHD) Donor T-lymphocytes attack host bowel. Hallmark finding: apoptotic bodies in crypts.
Severe damage shows crypt abscess, crypt distortion, and epithelial destruction. Some studies suggest minimum of 6 apoptotic bodies per 10 contiguous crypts to reliably diagnose grade 1 GVHD.

Graft Rejection Host T-lymphocytes attack donor bowel. Similar to GVHD: Inflammation (mostly lymphs) with crypt destruction and apoptosis.

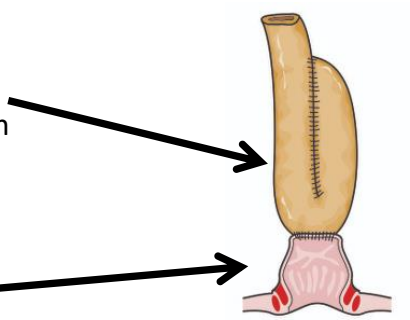
Lerner System for Grading GVHD	
Grade	**Findings**
1	Isolated apoptotic bodies
2	Loss or damage of isolated crypts, w/ or w/o crypt abscesses
3	Loss of 2 or more contiguous crypts
4	Extensive crypt loss with epithelial destruction

"IPAA" findings *Given to patients with UC or FAP after colectomy. Usu. NOT given to Crohn's patients as high risk of complications.*

Pouchitis Acute and chronic inflammation of ileal reservoir. Unclear etiology, but often treated with antibiotics/probiotics.
If refractory, consider Crohn's, but clinical Dx based on strictures/fistulas.

vs

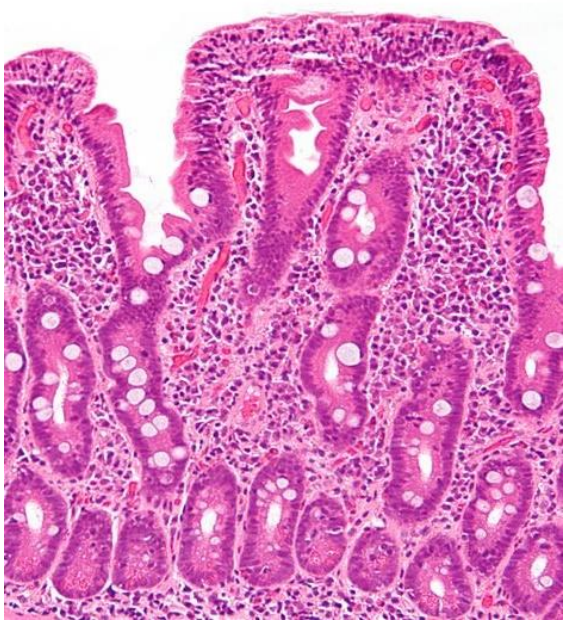
Cuffitis Chronic active inflammation of rectal cuff, attributed often to residual/recurrent UC.



Malabsorption

Villous atrophy with increased intraepithelial lymphocytes (IEL)

Causes Diarrhea clinically, often with weight loss.



Gluten Sensitive Enteropathy (Celiac Disease)
Gluten exposure triggers inflammation, primarily in duodenum. Positive serology for: Antigliaden, Tissue transglutaminase (TTG), and antiendomysial (EMA) (if not IgA deficient!). Associated with haplotypes HLA-DQ2 or DQ8 (absence both of these essentially excludes diagnosis). Number of IEL typically >20/100 enterocytes. "Crescendo" at tip of villi.

Other Protein Sensitives (e.g., cow milk, soy, eggs)
Often increased Eosinophils in mucosa

Peptic duodenitis

Medications (e.g., Olmesartan and NSAIDs)

Small Bowel Bacterial Overgrowth excess anaerobic bacteria (often caused by decreased acid and dysmotility) digest bile and carbohydrates→ variable local damage and bloating

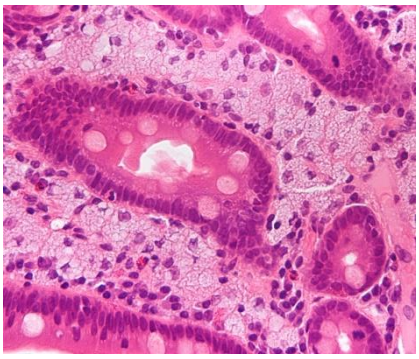
CVID Immunodeficiency with impaired B-cell differentiation. Usually plasma cells ABSENT→ low serum Ig's → recurrent infections.

Tropical Sprue Unknown etiology, but likely infectious. After travel to Africa, Asia, or South America. Must exclude other infections.

Autoimmune Enteropathy Gut autoantibodies → often absent goblet or Paneth cells. Most common in infants. Can see similar pattern associated with thymoma.

Foamy Macrophages

"Foamy" macrophages in mucosa



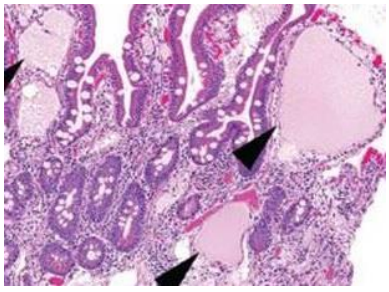
Mycobacterium avium intracellulare (MAI) Get a FITE stain! Immunocompromised/AIDS-defining opportunistic infection.

Whipple Disease Get a PAS/D stain! *Tropheryma whipplei* causes an infection often afflicting older white men→ arthralgias, weight loss, diarrhea. Treat with antibiotics.

Nonspecific Macrophages

Dilated Lacteal

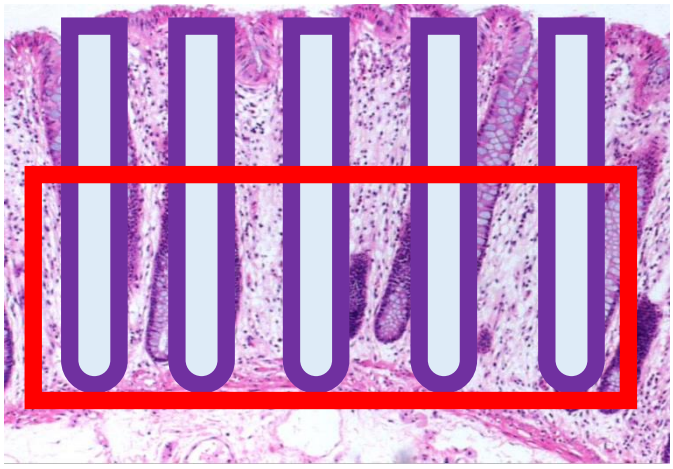
Engorged/dilated lymphatics



Primary Lymphangiectasia Poorly understood. Dilated lymphatics→ lymph/albumin leakage into gut → diarrhea and protein-losing enteropathy.

Secondary Lymphangiectasia Similar manifestations as primary, but secondary to obstruction, tumor, adhesions, stricture, prior surgery, etc….

Colon



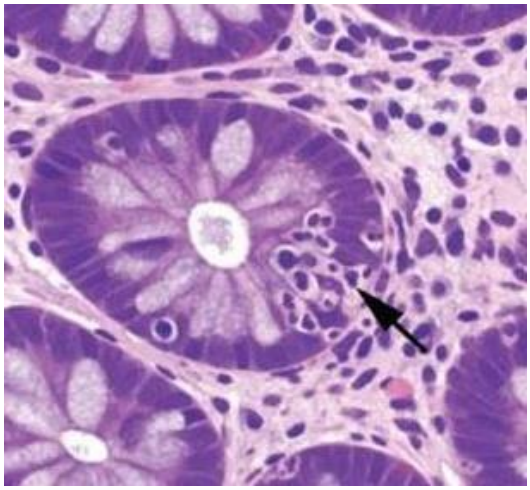
Crypts should be oriented parallel to one another, perpendicular to the surface (like test tubes in a rack), resting on the muscularis mucosae.

Regional Variation	
Right Colon	**Left Colon**
More lymphocytes	Less lymphocytes
Paneth cells normal	Paneth Cells abnormal
Fewer goblet cells	More goblet cells

Some architectural distortion and muciphages in the rectum is considered *normal*.

Focal Active Colitis

Rare collections of neutrophils in crypt epithelium (Otherwise normal)



Medications (esp. NSAIDs)

Acute Self-limited Colitis Resolves in <4 weeks. Usually infectious (e.g., *Campylobacter*, *Salmonella*, *Shigella*, or *Yersinia*) with abrupt onset and coinciding fever.

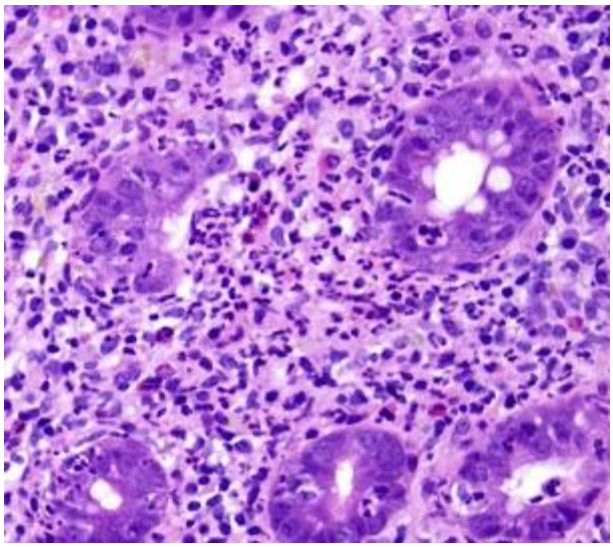
Bowel preparation artifact

Ischemic Colitis

IBD Usu. more dramatic findings though.

Acute Colitis

Extensive cryptitis and crypt abscesses, WITHOUT features of Chronicity



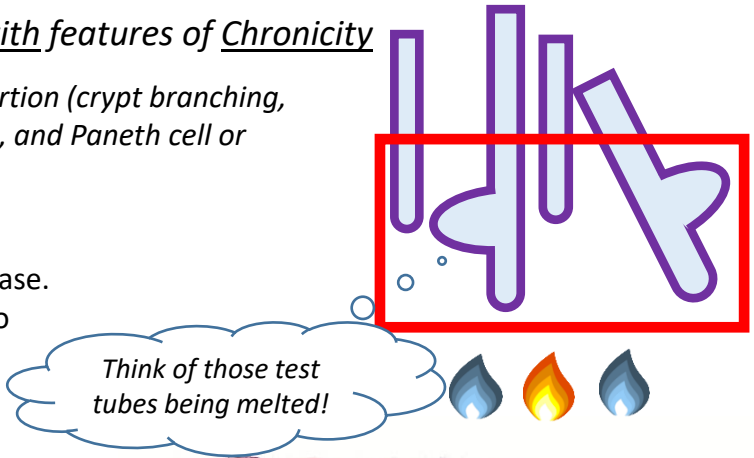
Infection Usu. acute bacterial or viral infections (e.g. CMV, *Salmonella*, *Shigella*, *Campylobacter*, etc…), so make sure this has been evaluated clinically. Often food contamination (fecal-oral). May see Pseudomembranes.

Medications Esp. NSAIDs. Also Resins (Kayexalate and Sevelamer) and Ipilimumab.

IBD Usually has features of chronicity, so would have to be emerging (very recent onset) or partially treated. Pediatric IBD may lack features of chronicity at diagnosis.

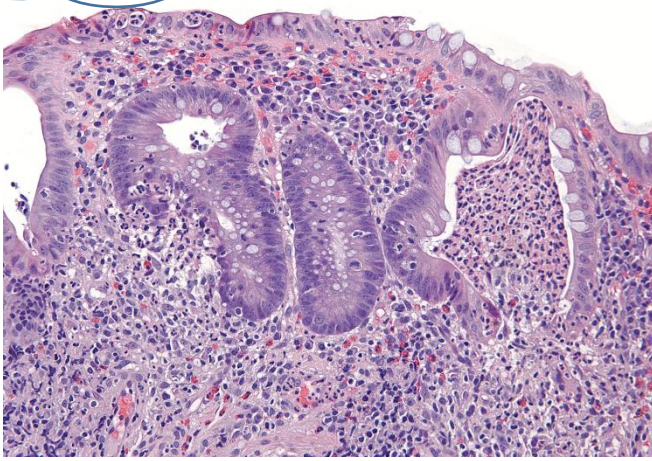
Chronic Active Colitis

Active colitis <u>with</u> features of <u>Chronicity</u>



Features of Chronicity include: Architectural distortion (crypt branching, loss, and shortening), basal lymphoplasmacytosis, and Paneth cell or pyloric metaplasia

<u>Inflammatory Bowel Disease (IBD)</u>
Chronic systemic autoimmune inflammatory disease. On a mucosal colonic biopsy, can be impossible to distinguish Crohn's from UC and must rely on clinical/endoscopic impression.
Look for Granulomas and Dysplasia.
(See separate IBD notes)



<u>Infection</u> Always rule out CMV in refractory IBD (along with other causes clinically)

<u>Diverticular Disease</u> Most common in older patients in sigmoid colon. Can mimic IBD with Diverticulitis and Segmental Colitis Associated with Diverticulosis (SCAD).

<u>Diversion-Associated Colitis</u> In bowel diverted from fecal stream (causes short chain fatty acid deficiency). Usu. see florid lymphoid hyperplasia with prominent germinal centers.

<u>STD Proctocolitis</u> Sexually Transmitted Diseases: Esp. Syphilis and lymphogranuloma venereum (Chlamydia). Often tons of plasma cells. Consider particularly with primarily rectal disease.

<u>Anastomotic site changes</u> Don't assume chronic active colitis at an anastomotic site is IBD!

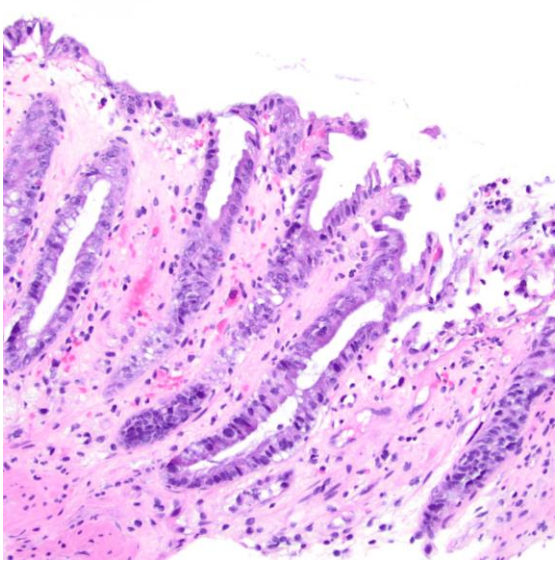
<u>Cord Colitis Syndrome</u> After Umbilical cord transplantation. Often see granulomas.

<u>Medications</u> NSAIDs, Ipilimumab, and resins

Ischemic Colitis

Superficial epithelial damage, Crypt withering, Lamina propria hyalinization and hemorrhage.

Occasional pseudomembranes and acute inflammation (with reperfusion)



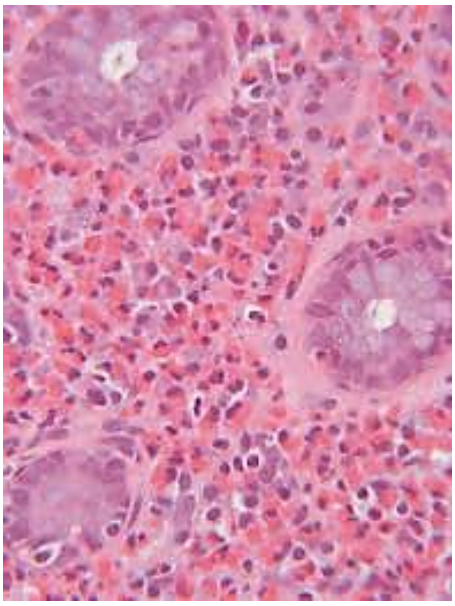
<u>Ischemia</u> Due to poor perfusion. Most common in "watershed" areas (splenic flexure, rectosigmoid, and ileocecal regions) in older patients with vascular occlusion or low-flow states.

<u>Infection</u>
E.coli 0157:H7 **(EHEC)**—Endothelial damage from toxin→ Fibrin thrombi often seen. Associated with Hemolytic Uremic Syndrome (Anemia, low platelets, renal failure)
C. Difficile—Pseudomembranes, less hyalinization and crypt withering

<u>Medications</u> Esp. NSAIDs. Also Resins (Kayexalate and Sevelamer) and Ipilimumab.

Eosinophilic Colitis

Increased Eosinophils



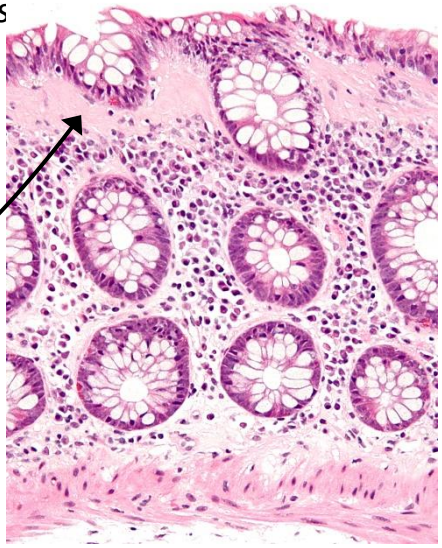
Although there is no strict cut-off, >60/HPF is likely too many and any in the epithelium, submucosa, or muscle is abnormal

Eosinophilic Colitis/Gastroenteritis
Diagnosis of exclusion. Can be associated with Eosinophil-rich inflammation in other organs (e.g., esophagus and/or small bowel). Layer of bowel involved determines symptoms.

Parasites
Connective tissue diseases/Vasculitis
Food Allergies
Medications
Systemic mastocytosis
Inflammatory bowel disease (particularly Crohn's)

Lymphocytic Colitis

Increased intraepithelial lymphocytes



Microscopic colitis: Normal endoscopic appearance with only *microscopic* findings. Includes lymphocytic colitis and collagenous colitis.

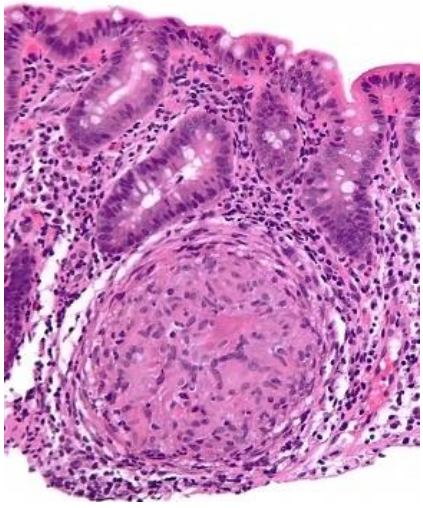
Lymphocytic Colitis Watery diarrhea. Increased intraepithelial lymphocytes (>20 lymphs/100 epithelial cells). Classically older women.

Collagenous Colitis Watery diarrhea. Increased intraepithelial lymphocytes with Increased subepithelial collagen layer (irregularly thickened, trapping inflammatory cells, vessels, and fibroblasts). Can highlight with a trichrome stain.

Medications (e.g., NSAIDs, Olmesartan, SSRIs, etc…
Viral Infections, Thymoma

Granulomatous Colitis

Granulomas! Rule out infection with FITE and GMS/PAS-D



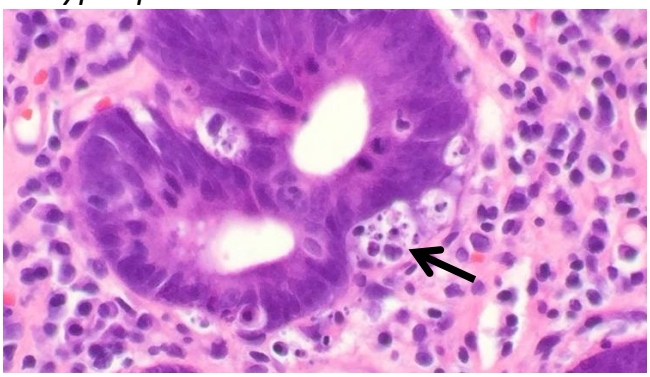
Crohn's Disease Loose, non-necrotizing. Seen in less than ½ of cases. *Note*: In UC can see granulomatous reaction to crypt rupture!

Infections Esp. if Necrotizing! Rule out fungi and mycobacteria. Look around for parasites (e.g., Schistosomiasis)

Nonspecific mucosal injury
Medications
Sarcoidosis
Cord Colitis Syndrome
Diverticular disease
CVID and Chronic Granulomatous Disease

Apoptotic Colitis

Increased apoptoses in crypt epithelium



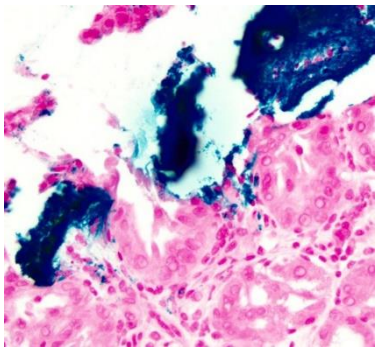
Graft vs. Host Disease (GVHD) Donor T-lymphocytes attack host bowel. First see apoptotic bodies in crypts. Severe damage shows crypt abscess, crypt distortion, and epithelial destruction.

Medication-effect Classic cause: Mycophenolate (MMF), which has a similar appearance to GVHD, but with more eosinophils

Pigments and Inorganic Material

Iron

Appears brown and granular on H&E; Blue on Iron Stain



Deposition Patterns:

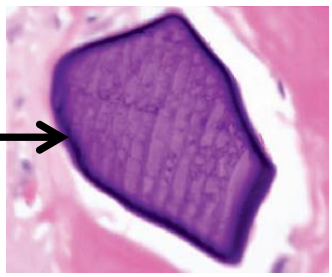
A: Deposition in lamina propria/macrophages → prior mucosal microhemorrhages

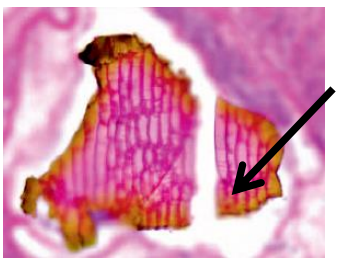
B: Coarse, crystals at surface → Iron pill

C: Subtle, uniform deposition in deep glands → Iron overload

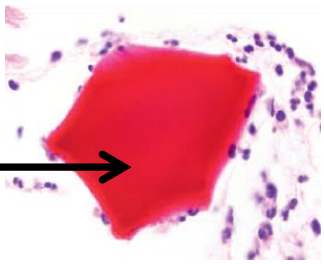
Resins

Kayexalate: Used to treat hyperkalemia in renal failure → causes ischemic and ulcerative changes. Linked to fatalities, so urgent dx. *Purple on H&E with narrow fish-scale pattern.*





Sevelamer: Used to treat hyperphosphatemia in renal failure → Associated with mucosal injury. *Bright pink to rusty yellow on H&E with irregular fish-scale pattern.*



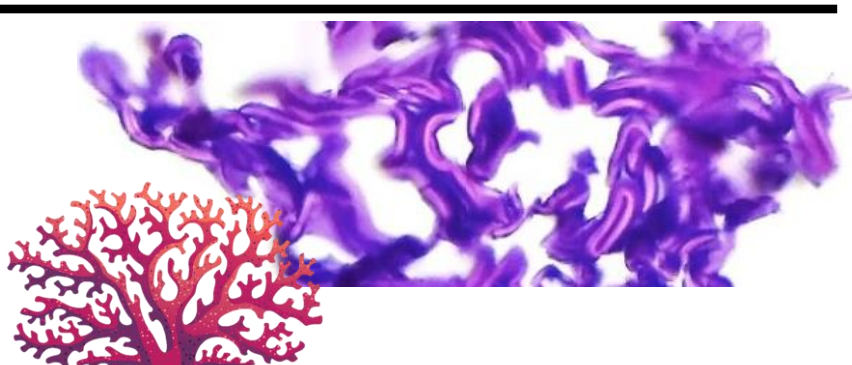
Bile Acid Sequestrants: (e.g., cholestyramine) Binds bile acids (lowers cholesterol). NOT associated with injury

Bright pink/orange on H&E with smooth, glassy texture.

Crospovidone

Coral shaped

"Pink center with a purple coat"



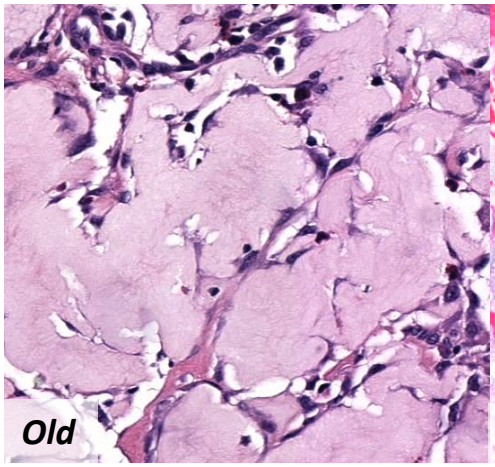
Incorporated into many medicines.
Biologically inert→ incidental finding
Non-birefringent

Lifting Agent

Appears homogeneous pink, often with a giant cell response (when fresh, more mucin-like)

Synthetic material injected during EMR/ESD. May resemble amyloidosis (but Congo Red negative) or Pulse granuloma.

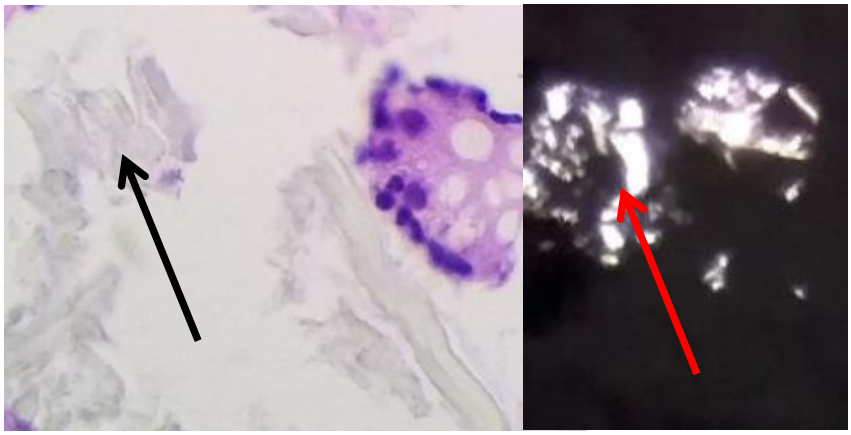
Common names: "ORISE" and "Eleview"



Microcrystalline cellulose

Appears transparent on H&E
Birefringent
Often rod-shaped

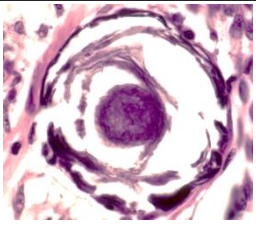
Incorporated into many medicines
Biologically inert→ incidental finding



Calcium

Appears dark purple and often cracked on H&E; Black on von Kossa

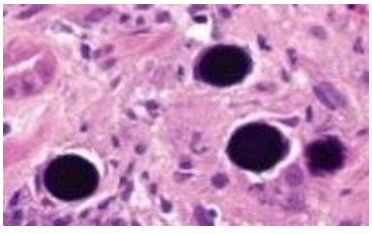
Can be: Metastatic (in normal tissue due to high serum calcium levels), dystrophic (in damaged tissue due to injury), or idiopathic



^{90}Yttrium-labeled Microspheres

Appear as uniform dark/opaque perfect circles.

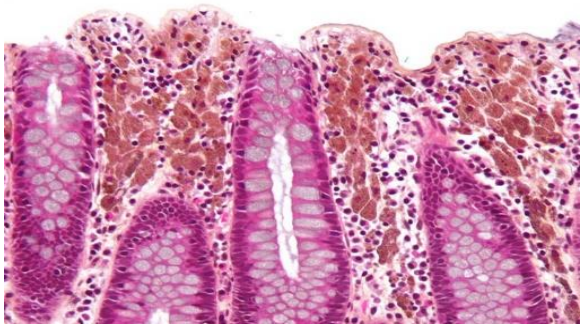
Given by interventional radiology as internal radiation therapy for hepatic malignancies. Often also see radiation injury.



Melanosis

Coarse, brownish black pigment in cytoplasm of macrophages.

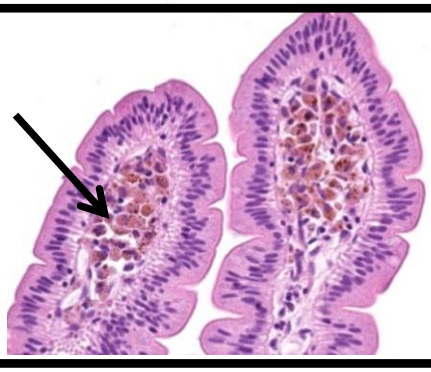
Consists of deposited Lipofuscin. Although classically associated with <u>laxative</u> use, can be seen in any disorder with increased epithelial cell turnover, including constipation.



Pseudomelanosis duodeni

Coarse, brownish black pigment in cytoplasm of macrophages, mostly at villous tips in duodenum.

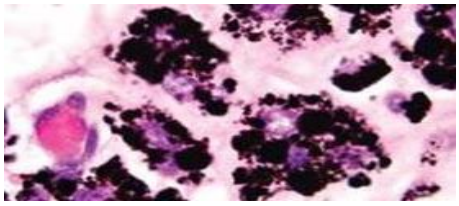
Consists of iron, calcium, and other elements. (Can highlight with Iron and/or calcium stains).
Associated with hypertension, gastrointestinal bleeding, renal failure, diabetes, and with particular medications,



Tattoo

Very black, coarse granules in macrophages, often with a foreign body giant cell reaction.

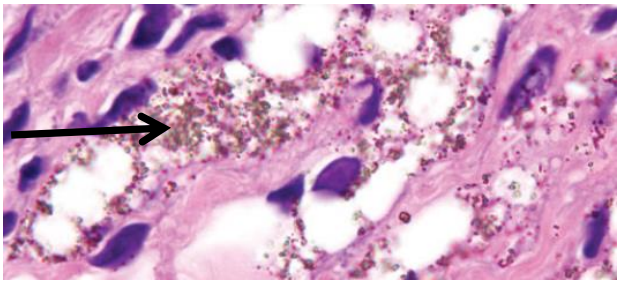
Used to mark lesions endoscopically for later identification.



Titanium

Fine, dark brown to black. Confined to macrophage cytoplasm

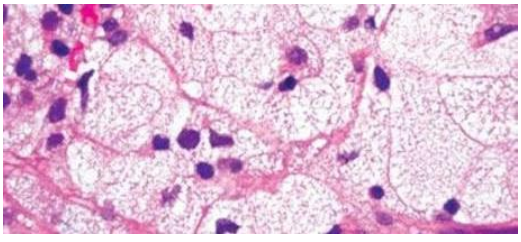
Unique to **Terminal ileum**.
represents titanium, aluminum, and silicon—but for simplicity's sake, referred to as titanium alone.
From food additives and Whitening agents (e.g. toothpaste)



Muciphages

Mucin-containing macrophages in lamina propria

Presumably cleaning up after epithelial injury and turnover. Very common, especially in rectum.

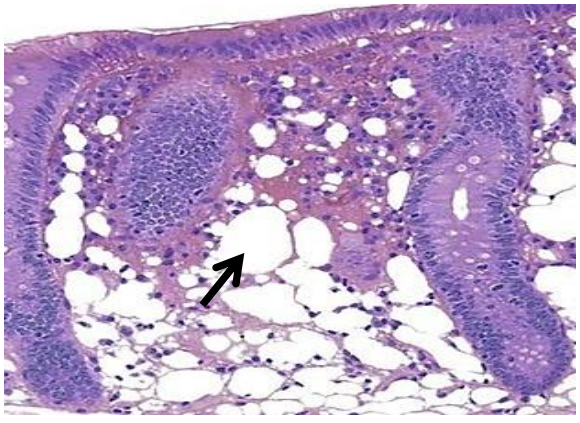


Air

"Pseudolipomatosis"

Empty spaces, irregular in size, <u>without</u> a foreign body reaction.

Attributed to insufflation artifact→ incidental.
No associated nuclei (not fat).



Pneumatosis Cystoides Intestinalis

Empty spaces, WITH a foreign body reaction.
(which means it happened *<u>in vivo</u>*!!)
Often iatrogenic or infectious cause→ Real!