

What looks like IBD is not always IBD: From cases to processes

SALVADOR J. DIAZ-CANO

ORCID 0000-0003-1245-2859

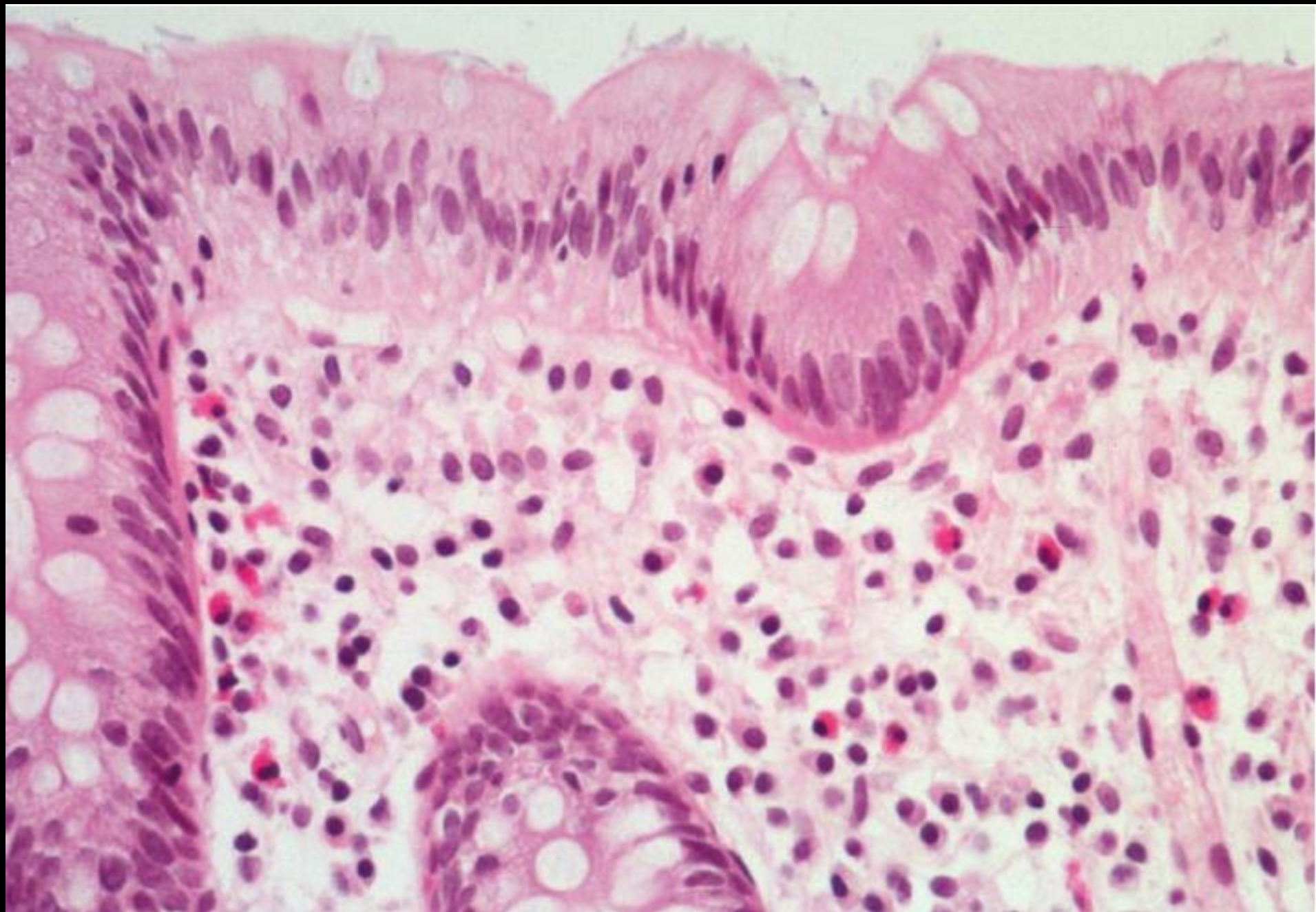
BAHRAIN, APRIL 2017



Approach to Non-Neoplastic Biopsies

- Is it normal? (Many/most GI biopsies are normal)
- If inflamed, is it acute or chronic disease?
- If acute, can I make a specific diagnosis?
- If chronic, can I make a specific diagnosis?

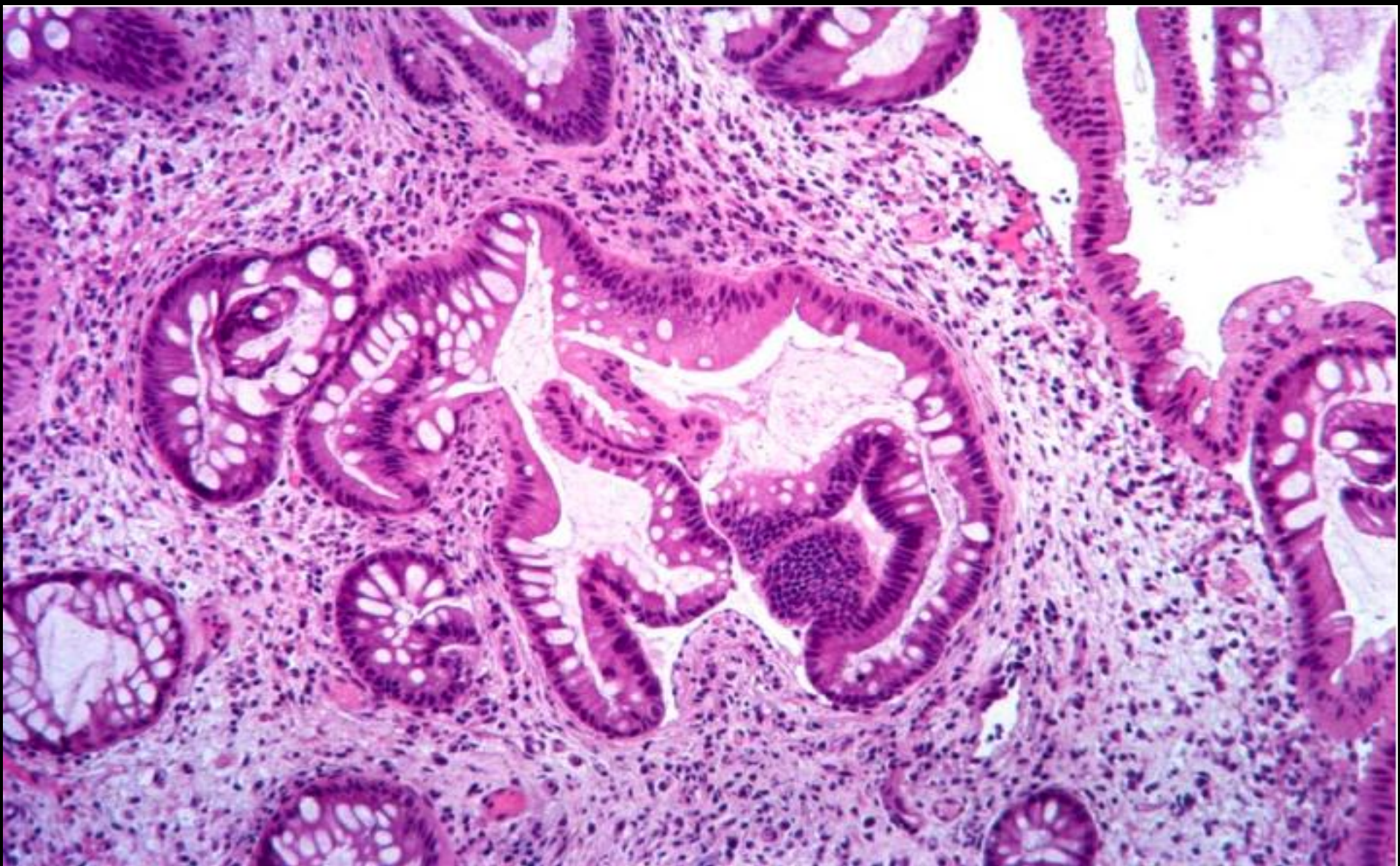


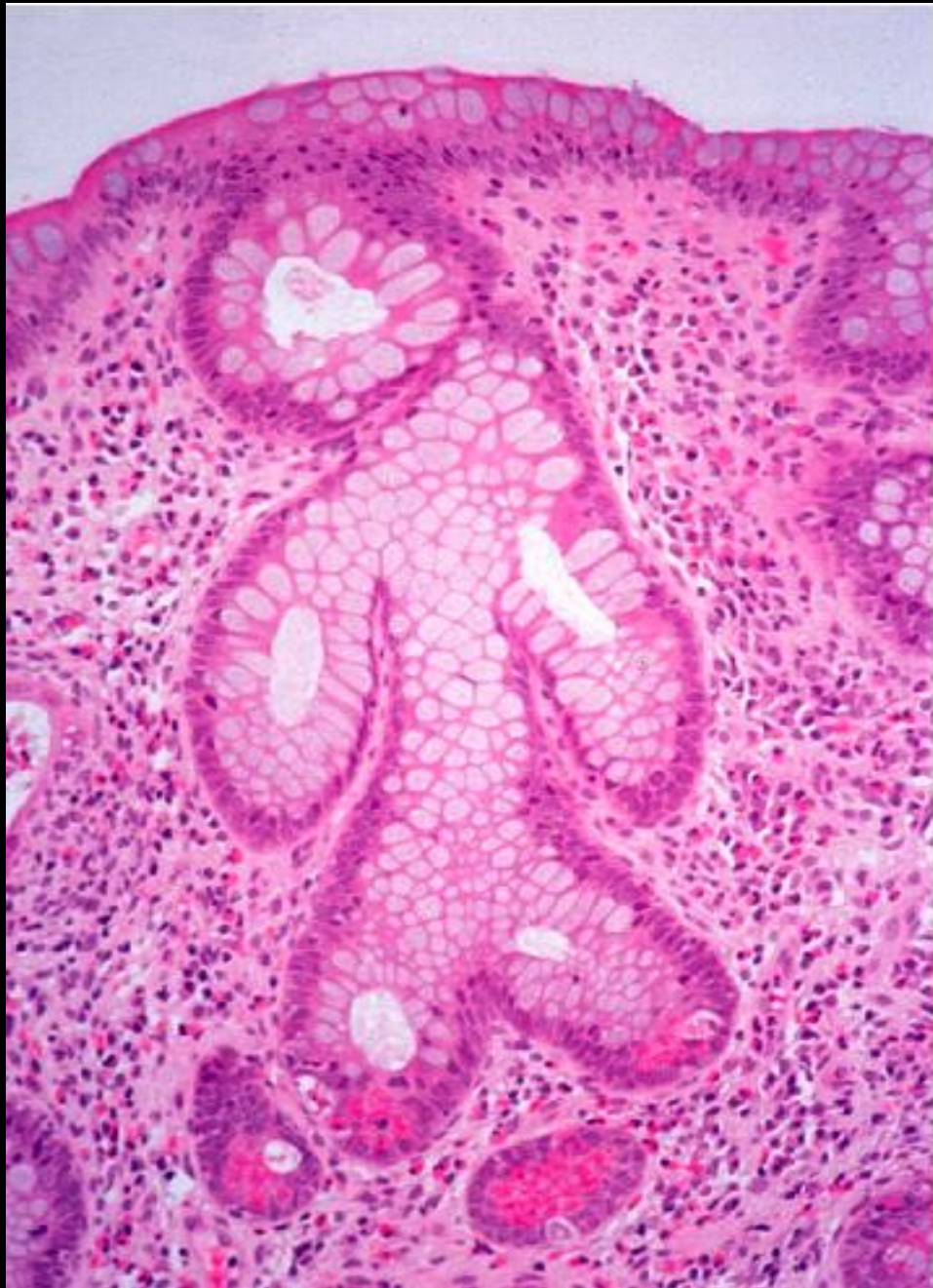


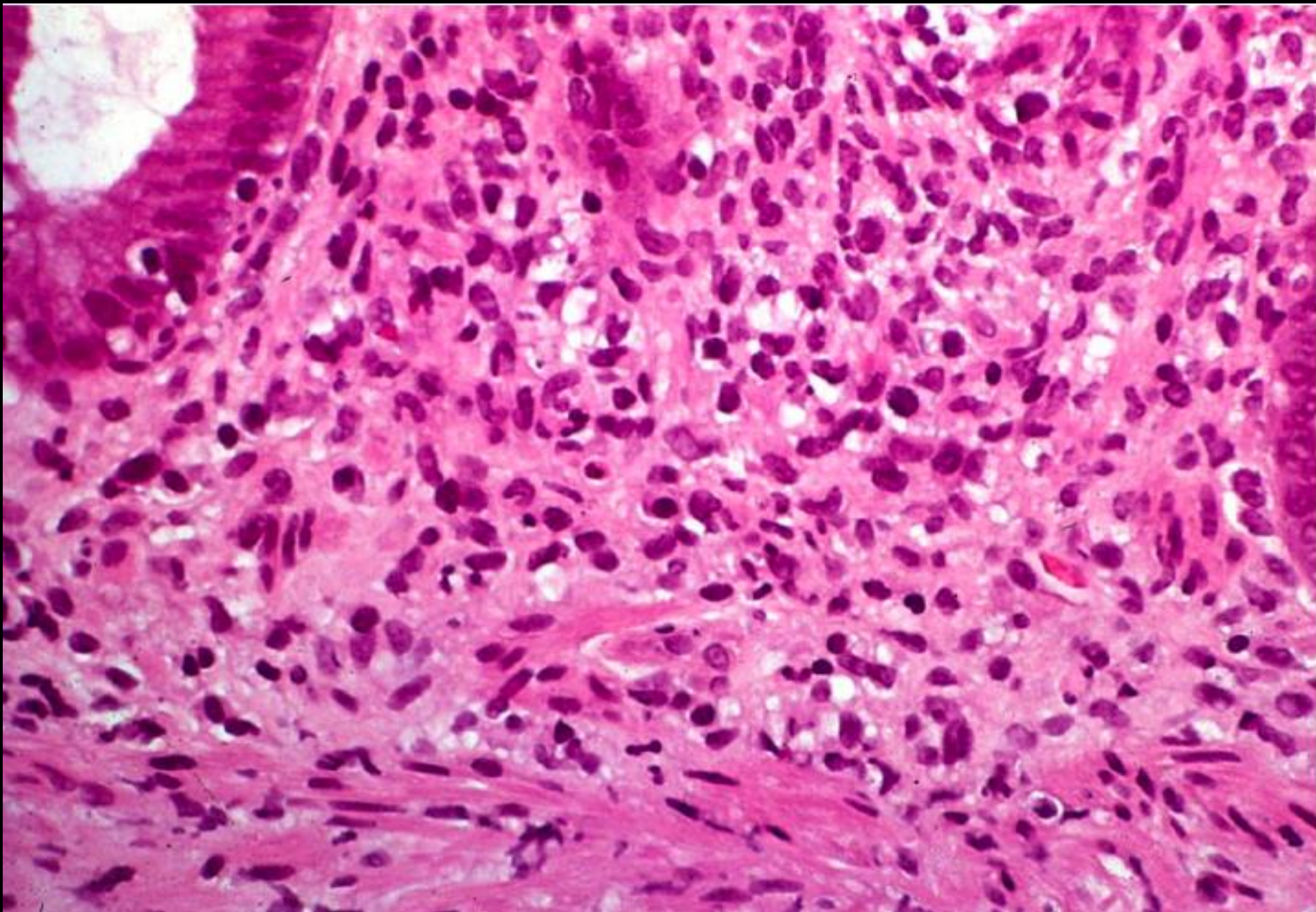


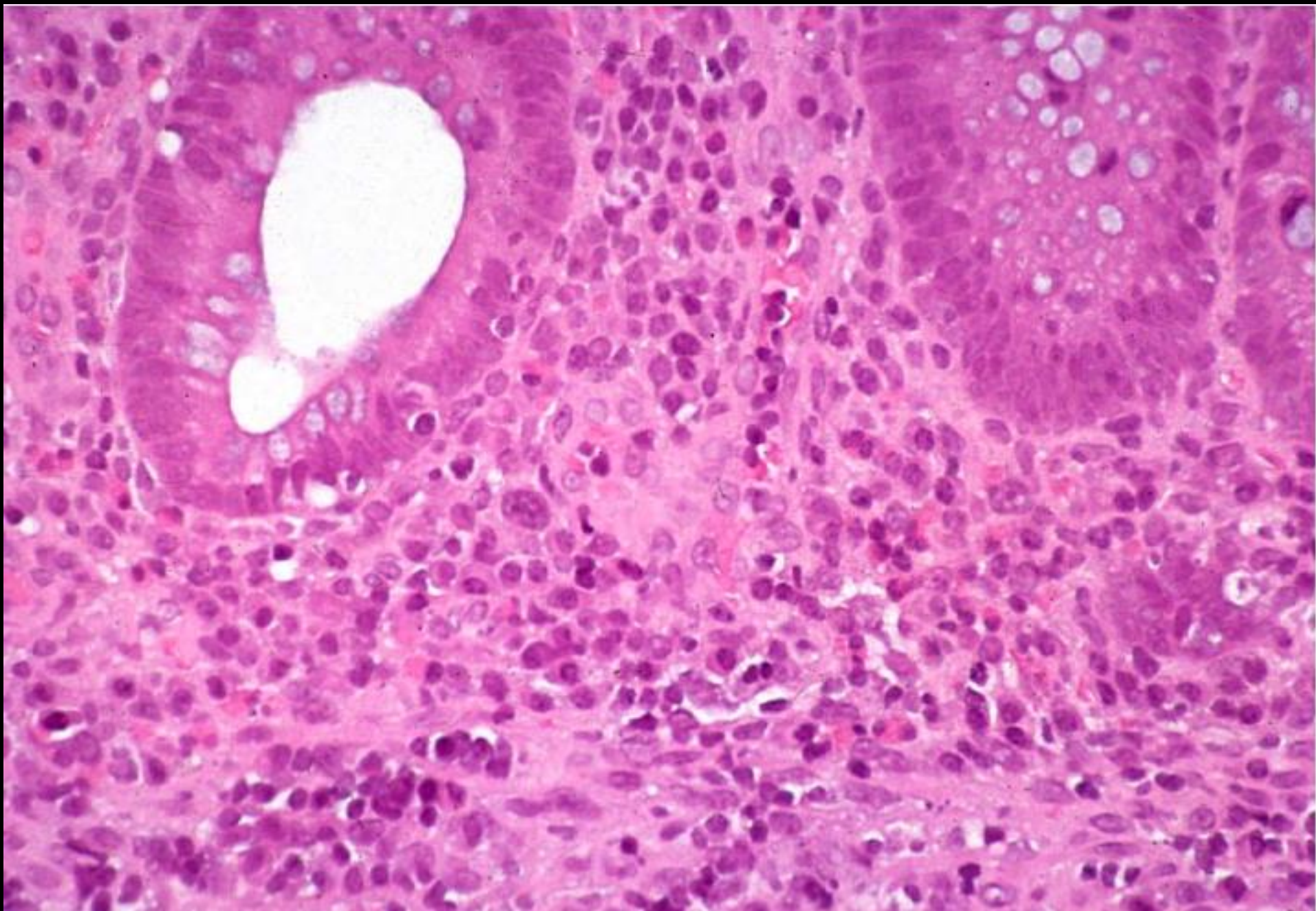
Markers of Chronic Injury

- **Forked or branched crypts**
- **Crypts shaped like animals, continents, or hebrew letters**
- **Paneth cells more distal than the right colon**
- **Basal plasma cells**





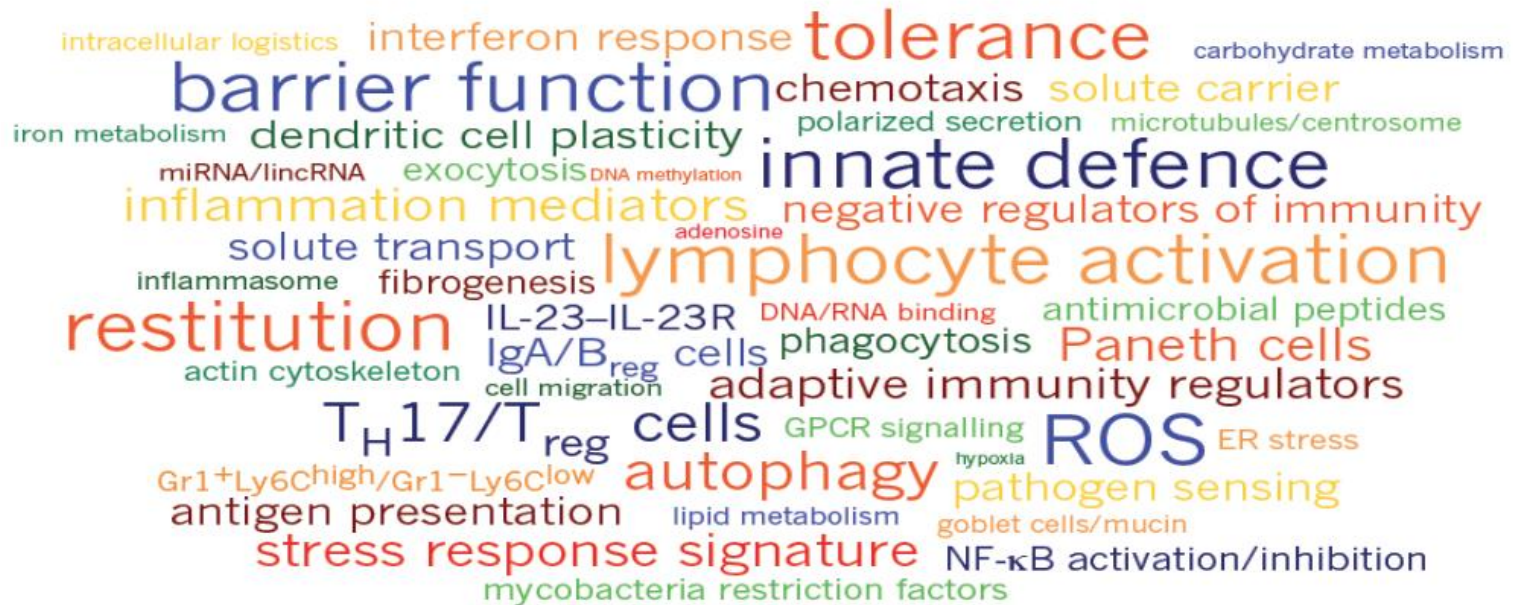




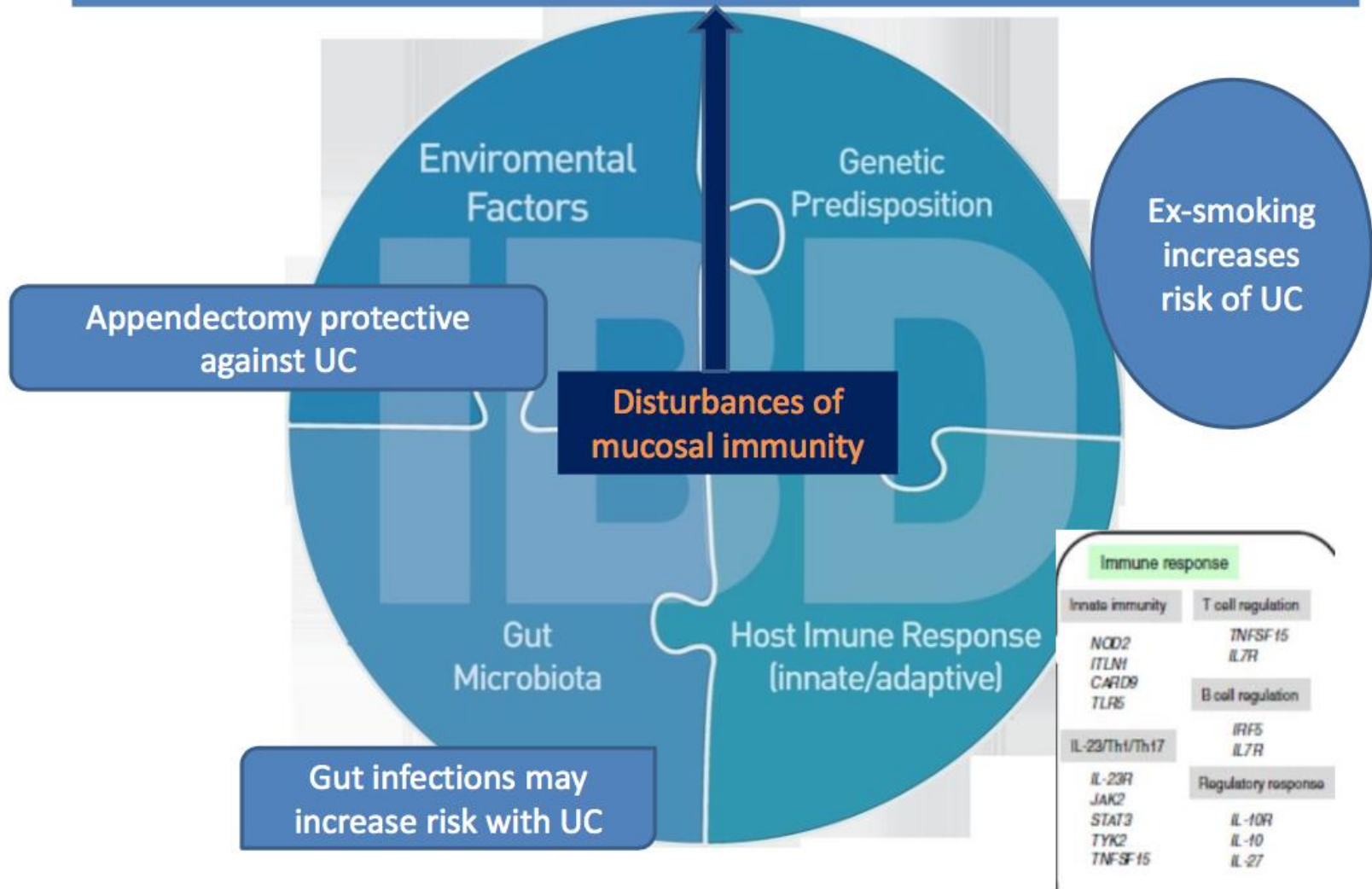
Inflammatory Bowel Disease (IBD)

- Refers to a group of inflammatory conditions which primarily affect the colon and small intestine, although the entire GI tract can be involved.
 - Ulcerative colitis
 - Crohn's disease
 - Indeterminate colitis
- They are identified and diagnosed by the appearance of a set of clinical, endoscopic, and histologic characteristics.

The Alphabet Soup of IBD



Pathogenesis of Inflammatory Bowel Disease



Diagnostic Approach

- **Clinical suspicion of the illness based upon history, examination and screening laboratory data**
- **Exclusion of other illnesses that have a similar presentation**
- **Establishment of the diagnosis of IBD, with differentiation between CD and UC**
- **Localization of the region of the disease**

Routine Diagnostic Testing

- **CBC**
 - Reasonable to look for anemia
 - May change therapy
- **ESR & CRP**
 - Not necessary for diagnosis
 - Possibly useful to know if elevated for assessment of recurrent symptoms
- **LFT's**
 - Little evidence that early diagnosis of IBD-related liver disease will change natural history
- **Fecal Calprotectin/lactoferrin**
 - Can detect active inflammation
- **Prometheus IBD sgi Diagnostic panel- NO**

Antibody Testing

- **Anti-Saccharomyces cerevisiae (ASCA)**
 - 40-80% CD patients
 - Identify those with TI and cecal disease
 - Unusual in UC patients
- **Atypical perinuclear antineutrophil cytoplasmic antibodies(P-ANCA)**
 - 60-80% UC patients
 - 10-27% CD patients
 - Patients with CD who have P-ANCA often exhibit UC-like features
- **Anti OmpC**
 - The addition of OmpC to ASCA and P-ANCA can increase sensitivity, but will decrease specificity in ASCA or P-ANCA negative patients
- **Anti CBir1**
 - Found in ~50% of CD patients
 - Associated with SB, internal-penetrating and fibrostenosing patterns

Antibody Testing

- P-ANCA combined with Anti-CBIR1 may be associated with the development of acute or chronic pouchitis
- Prometheus IBD Serology 7: Sensitivity IBD 74%, CD 71%, UC 92%
- Suboptimal sensitivity levels argue against the use for screening for IBD
- When combined into panels, the antibody tests have reasonably high sensitivities for detecting IBD (>90% in populations with sx), but ability to distinguish between CD and UC is only fair

GI Tract Pathology - Facts

- **Tubular GI tract is unique in many ways**
 - We can look inside and even get good samples of tissue
 - But, we are *absolutely dependent* on the endoscopist for gross description
- **There is a fairly limited number of ways the GI tract responds to injury**
 - But, we are fairly dependent on clinical information to sort out what we find on biopsies (and even resections)
- **There is a lot of normal stuff in the intestines that can resemble (or hide) pathology**
 - Lymphoid tissue, blunt-ish villi, the occasional bifid crypt, lamina propria fibrosis, focal cryptitis...

What is the meaning of this?

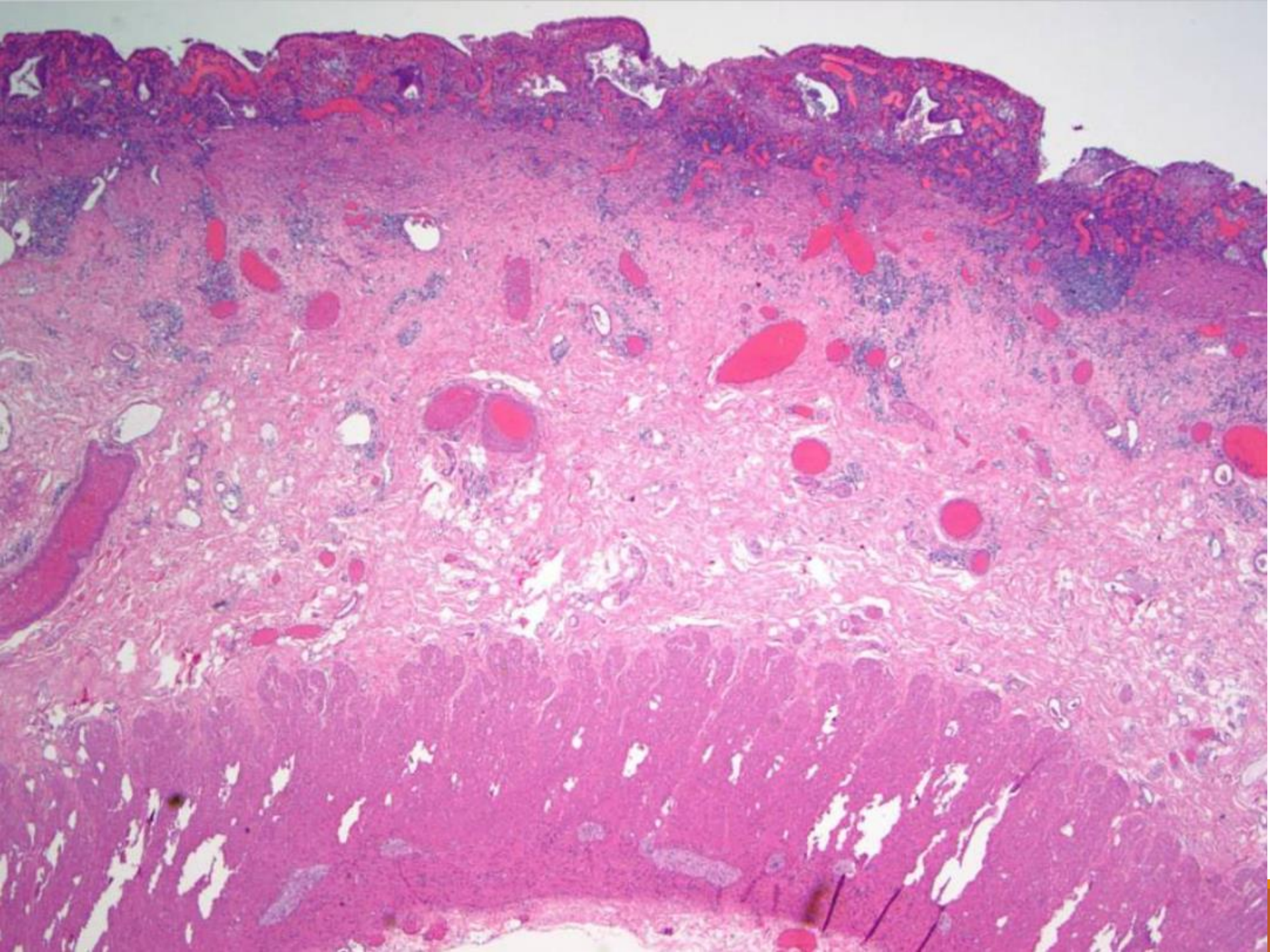
- Don't be afraid to say something is normal!
 - Or, at least *not significant*
- Know when you absolutely, positively need to have clinical information
 - Know where to find it and do so if you need it
 - If you can't, you may have to play the "if..., then..." game
- Think of any abnormalities you see (especially in biopsies) as samples of a *disease process*
 - As opposed to tiny, separate fragments that contain individual abnormalities

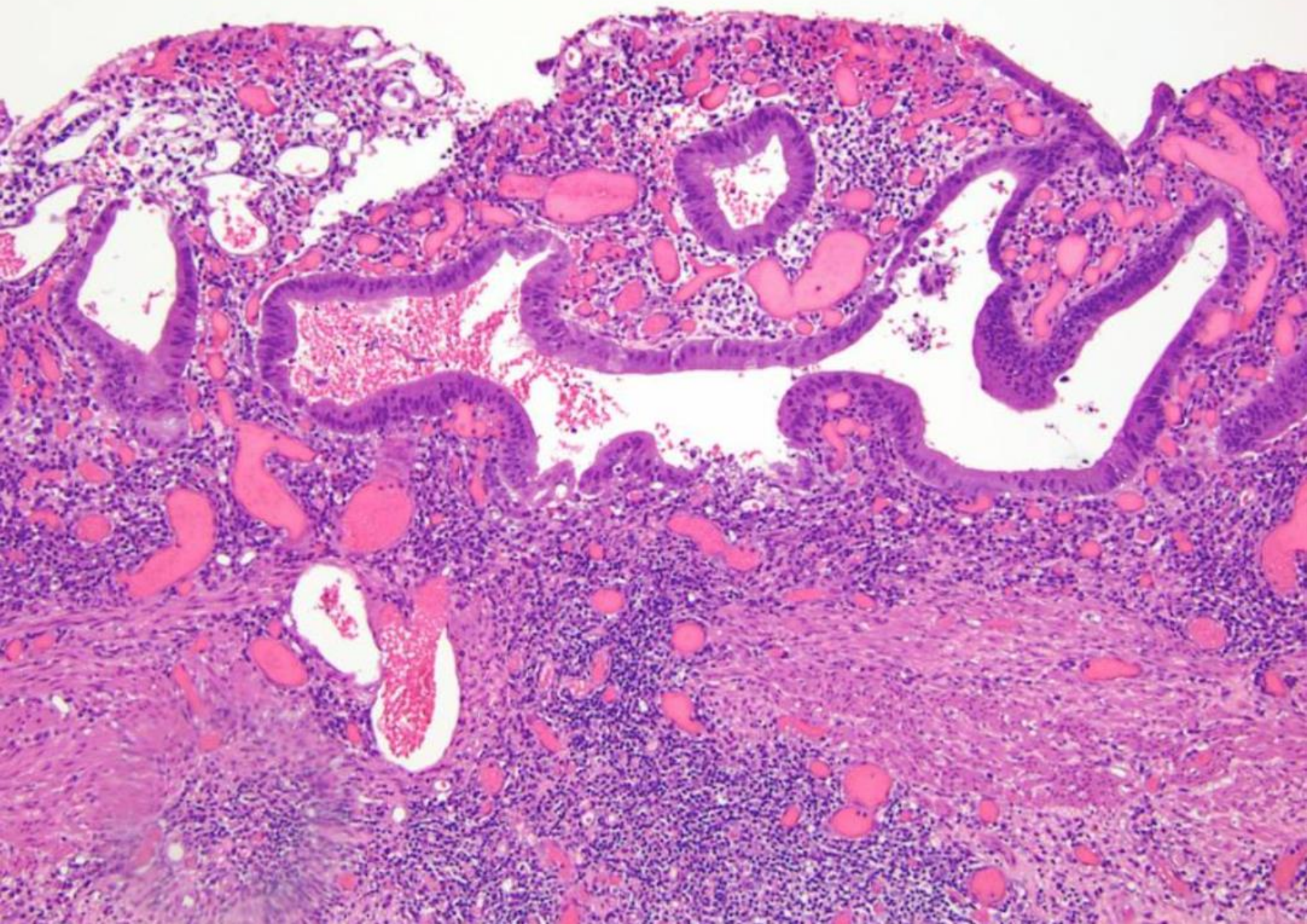
UC Pathology

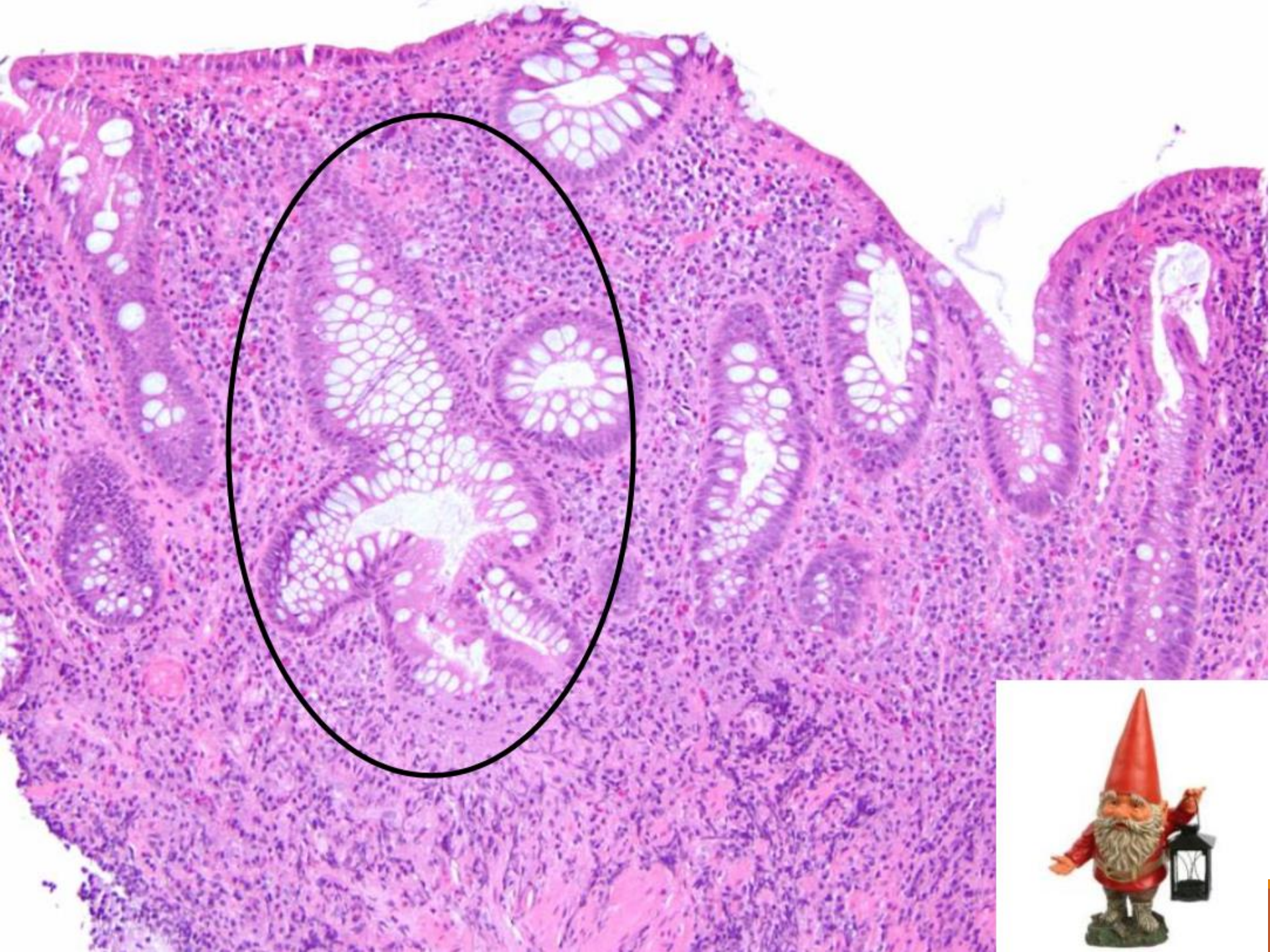
- **Mucosal inflammation**
 - ***Contiguous* active, chronic inflammation in colon**
 - Shallow ulcers; generally stop at muscularis mucosae
 - Crypt distortion (Dr. Seuss characters)
 - Not much (if any) small intestinal involvement
 - **(Pseudo)pyloric gland metaplasia unusual**
 - **Pseudopolyps fairly common**

UC Pathology

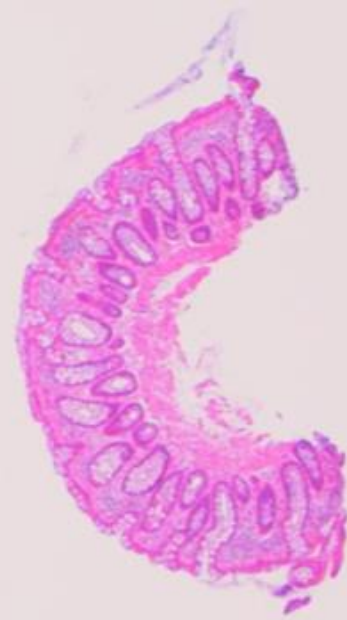
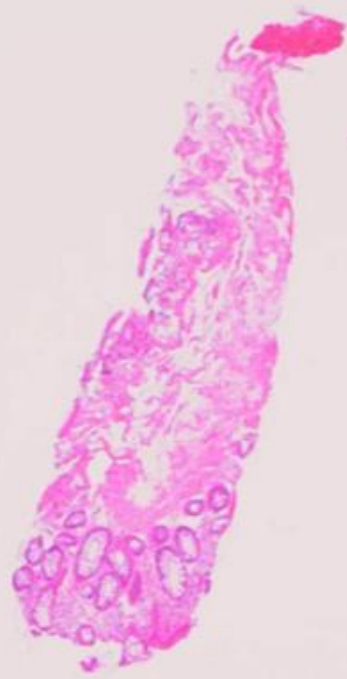
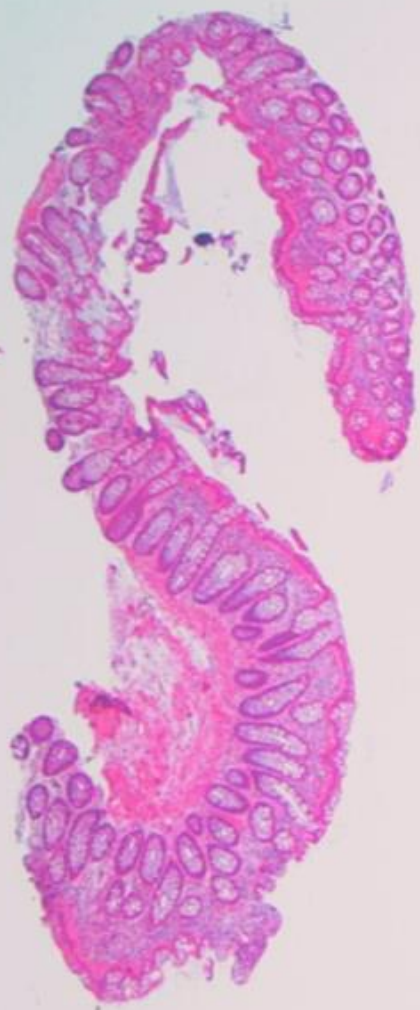
- **Unusual patterns**
 - **Relative rectal sparing**
 - **Cecal patch**
 - **Granulomas**
 - **Upper tract involvement(!)**











Crohn's Disease – Pathology

- **Mucosal changes**
 - ***Patchy* active, chronic inflammation**
 - Aphthous ulcers
 - Crypt distortion (Dr. Seuss characters again)
 - Blunted, thick, hypermucinous villi
 - **Gross ulcers (usually with clinical “activity”)**
 - **(Pseudo)pyloric gland metaplasia (“ulcer-associated cell lineage”)**

Crohn's Disease – Pathology

➤ Mural changes

➤ Transmural inflammation

➤ Deep, fissuring ulcers

➤ Fibrosis (including submucosal scar)

➤ Granulomas

➤ Granulomas don't necessarily mean Crohn's, nor does their absence mean it's NOT Crohn's

➤ Muscular and nerve hypertrophy

➤ Muscularis mucosa thickened, sometimes duplicated/splayed

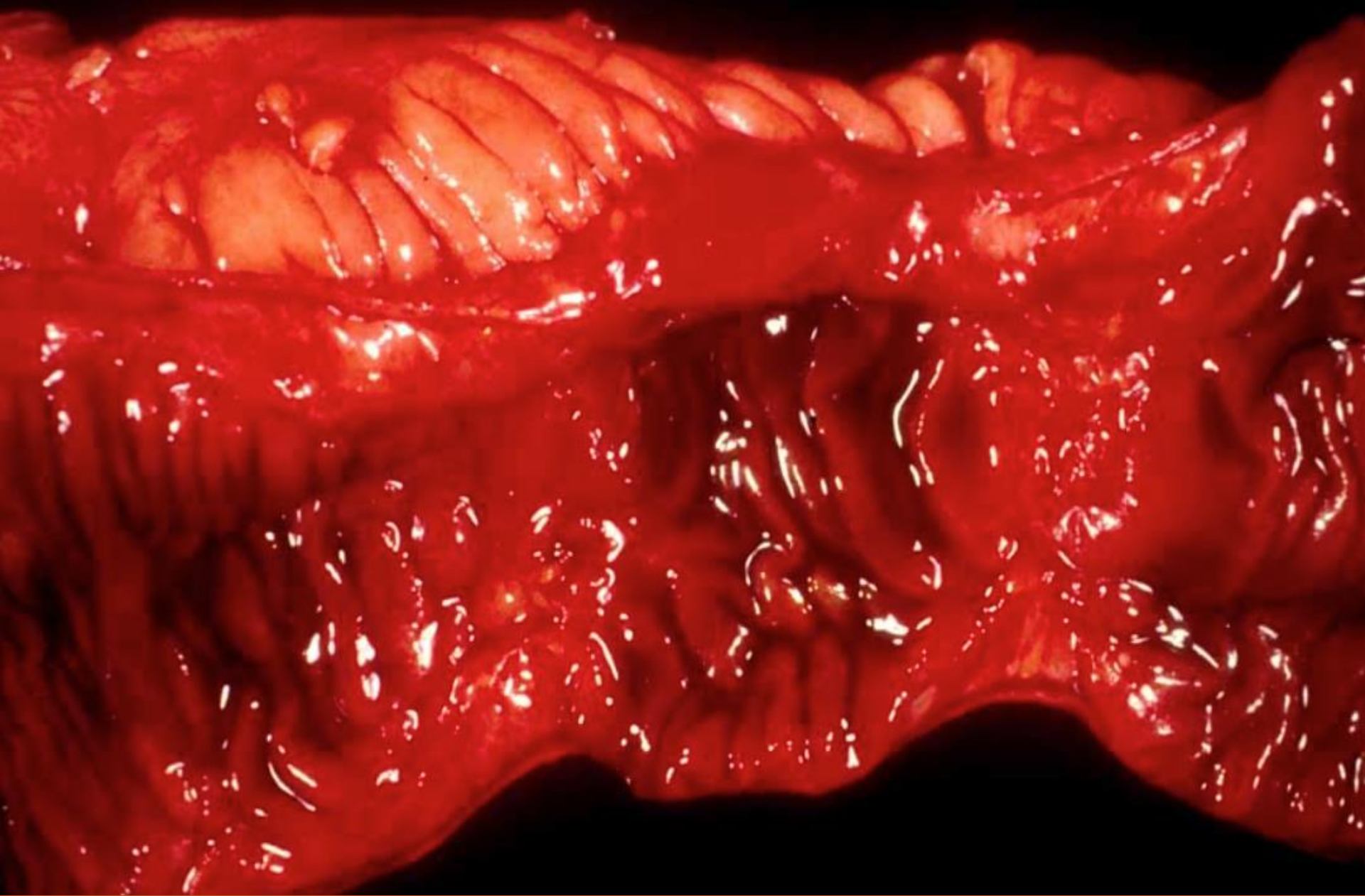
Fat wrapping



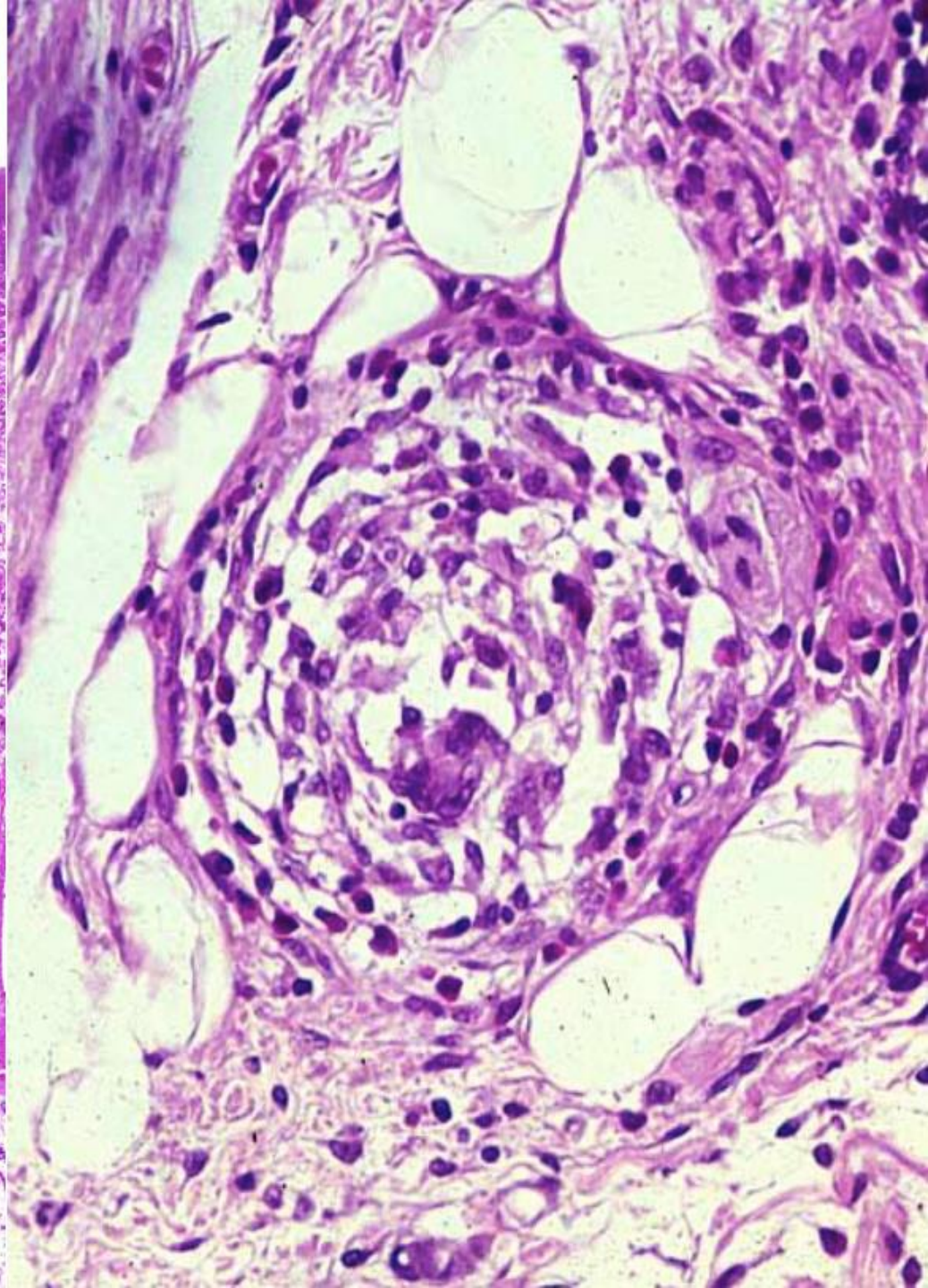
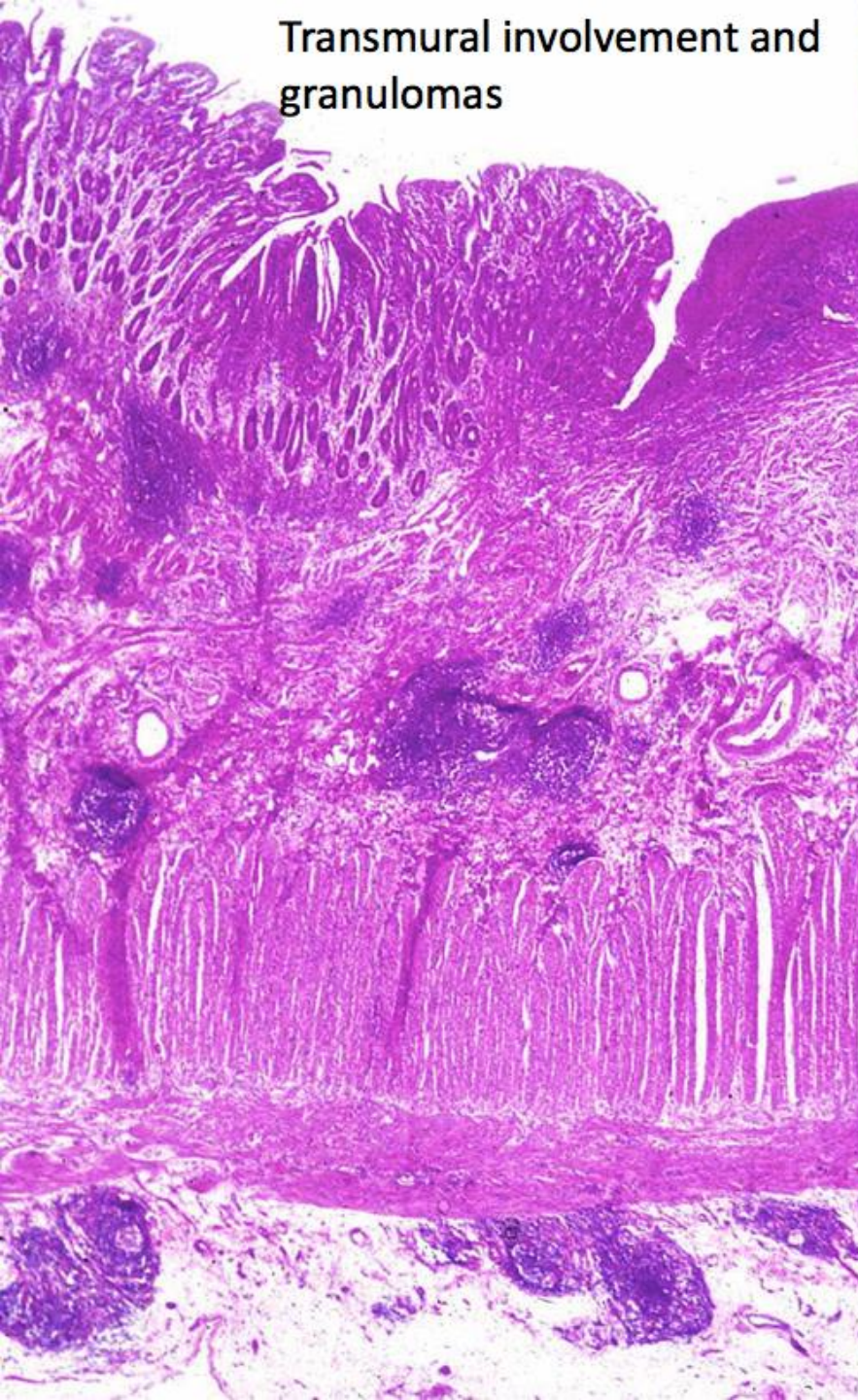
Mural thickening



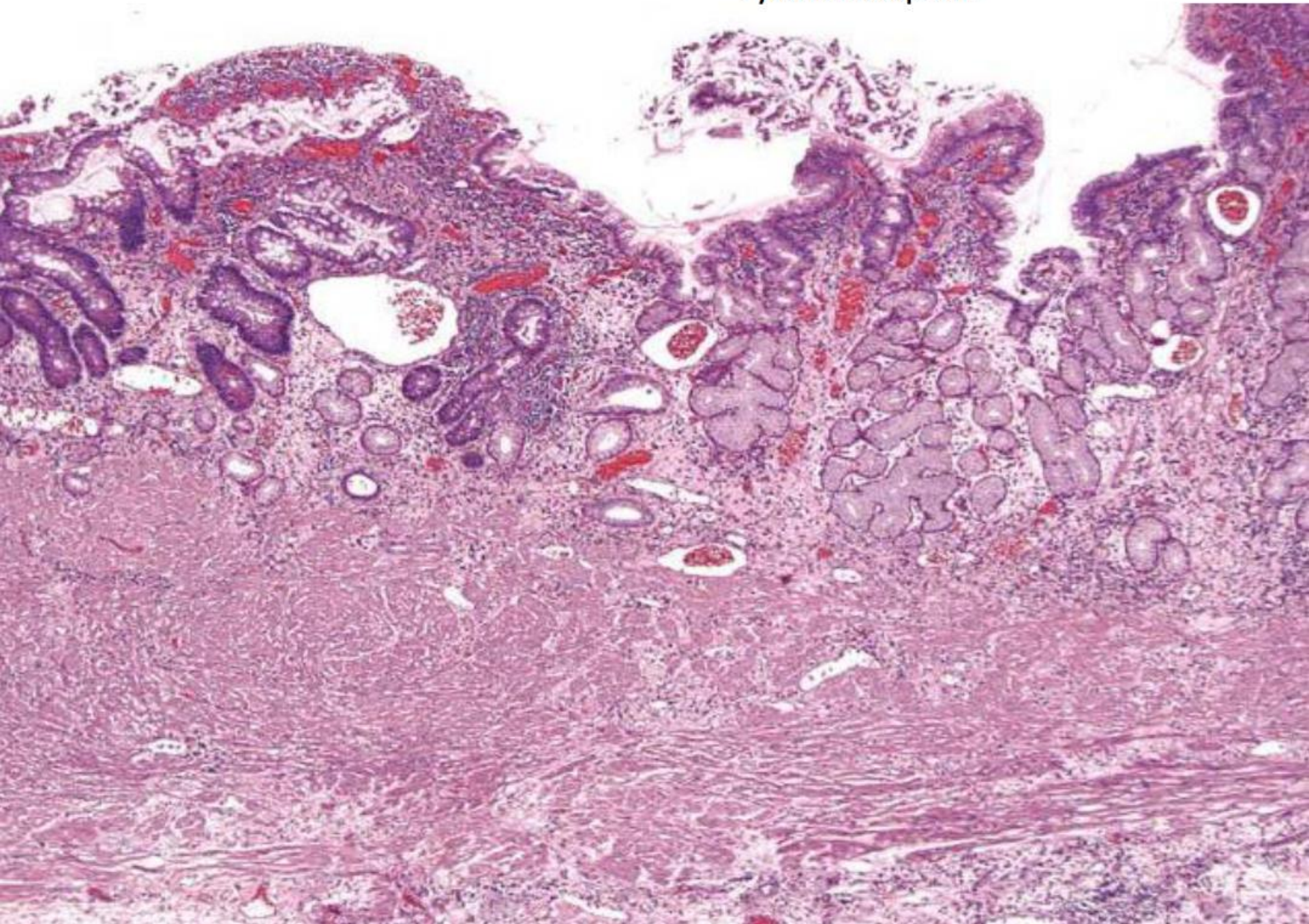
Skip lesions



Transmural involvement and
granulomas



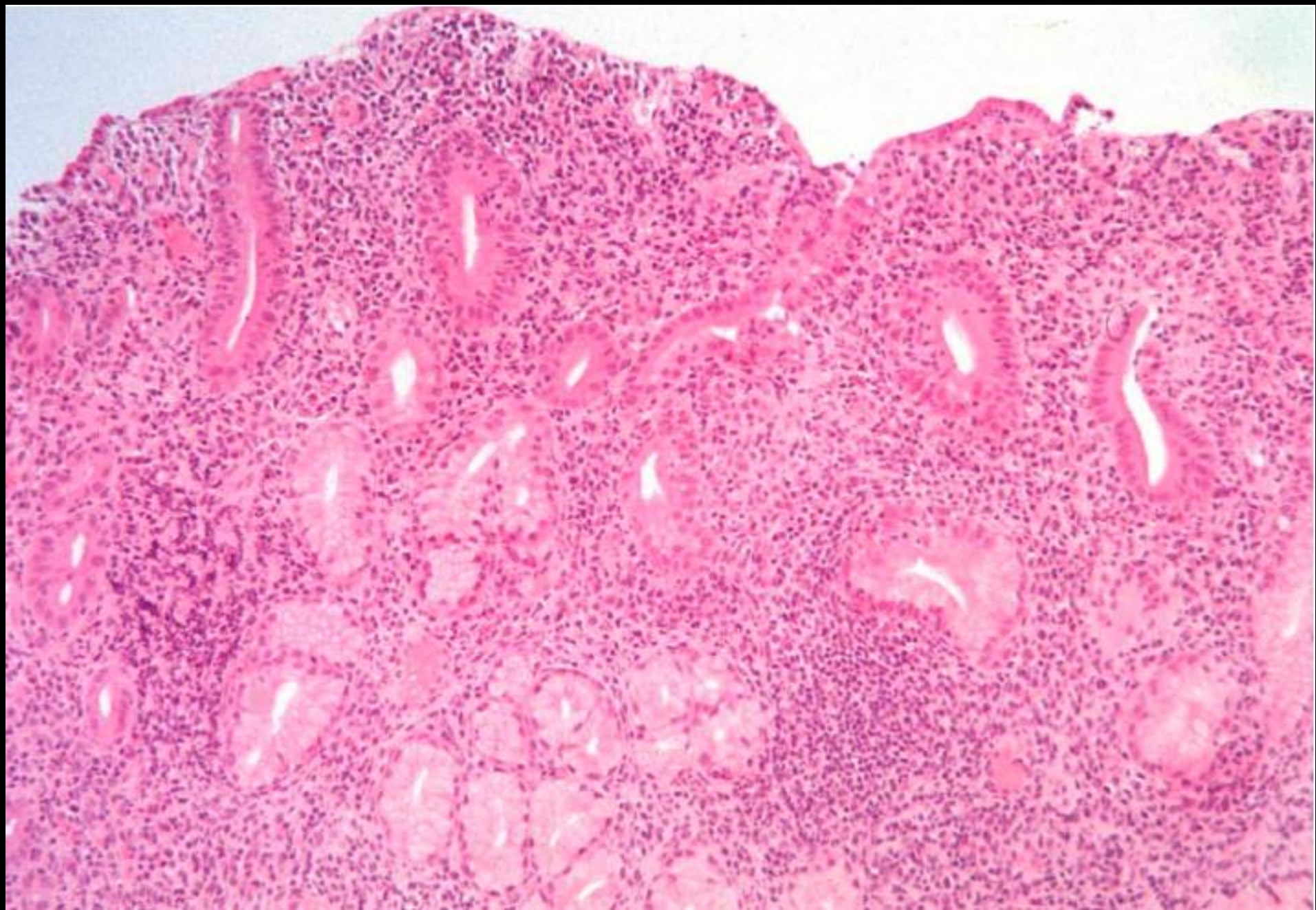
Pyloric metaplasia

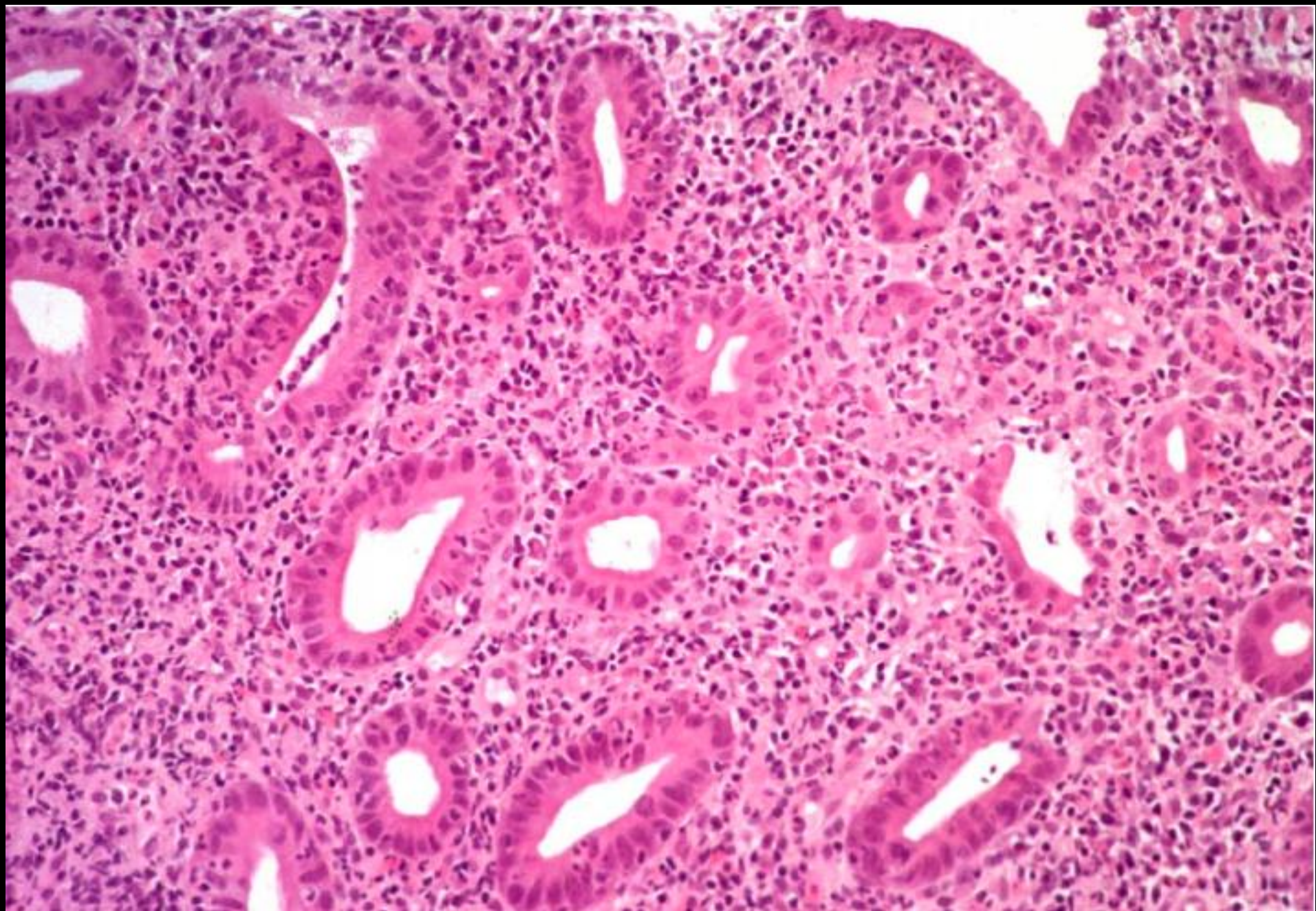


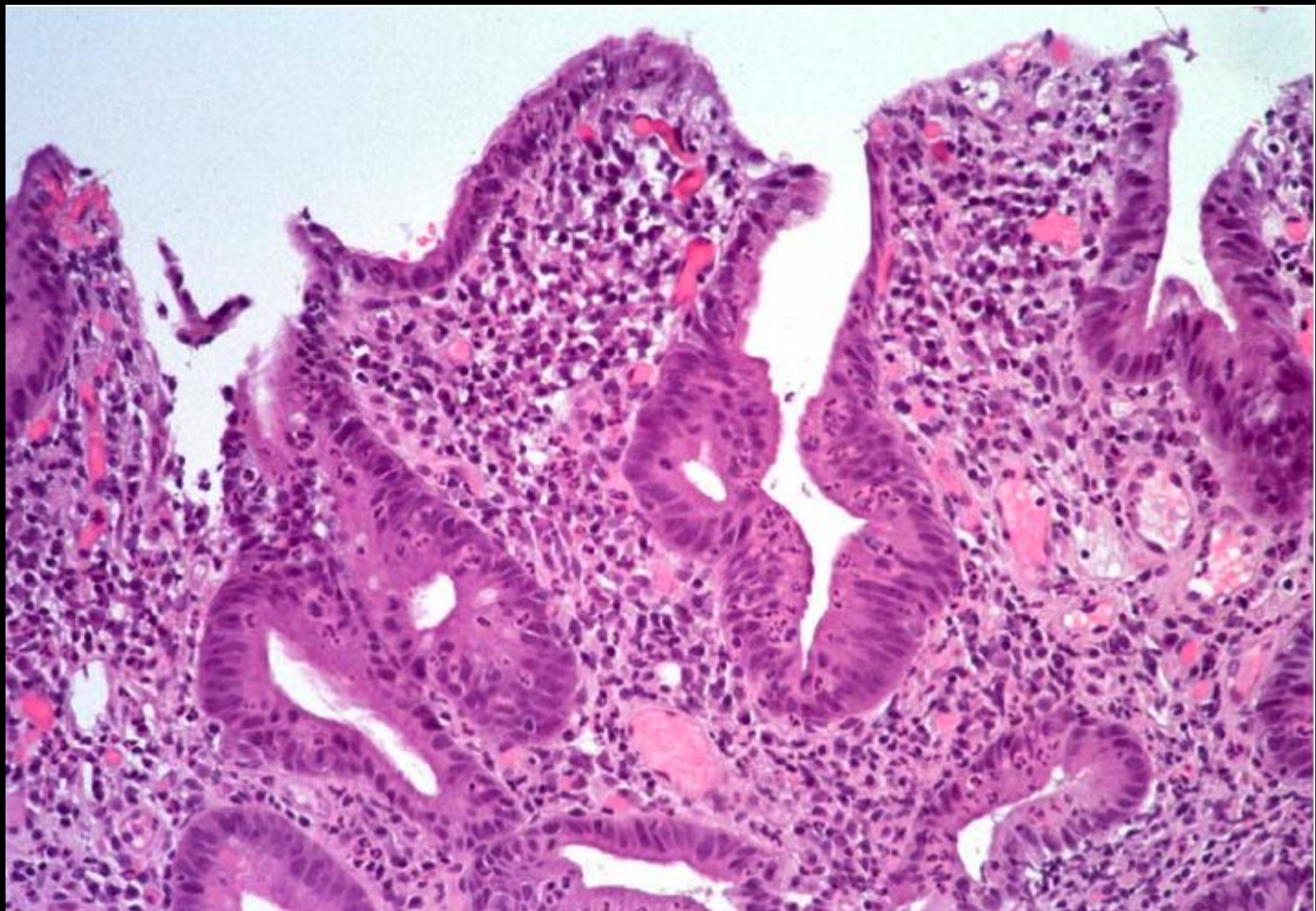
Changes in IBD Patterns

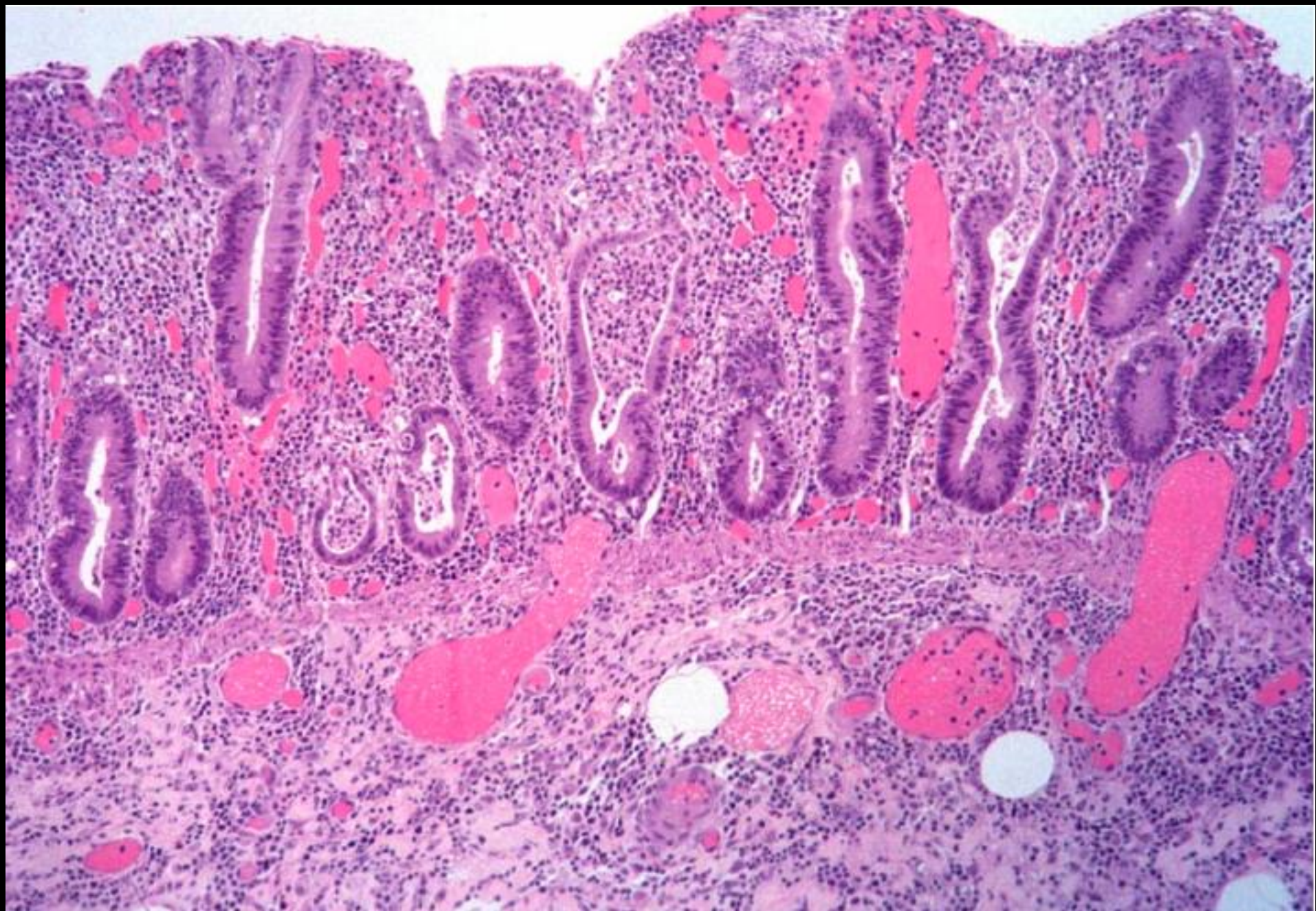
THESE DUODENAL BIOPSIES ARE FROM A 20 YEAR-OLD FEMALE WHO UNDERWENT A SUBTOTAL COLECTOMY WITH ILEOSTOMY FOR ULCERATIVE COLITIS THREE YEARS EARLIER. NINE MONTHS AFTER SURGERY, THE PATIENT BECAME SYMPTOMATIC AGAIN, PROMPTING BIOPSIES OF THE DUODENUM, STOMACH AND ILEUM. AT ENDOSCOPY THE DUODENUM SHOWED “DIFFUSE ENTERITIS”.











Variants of Ulcerative Colitis

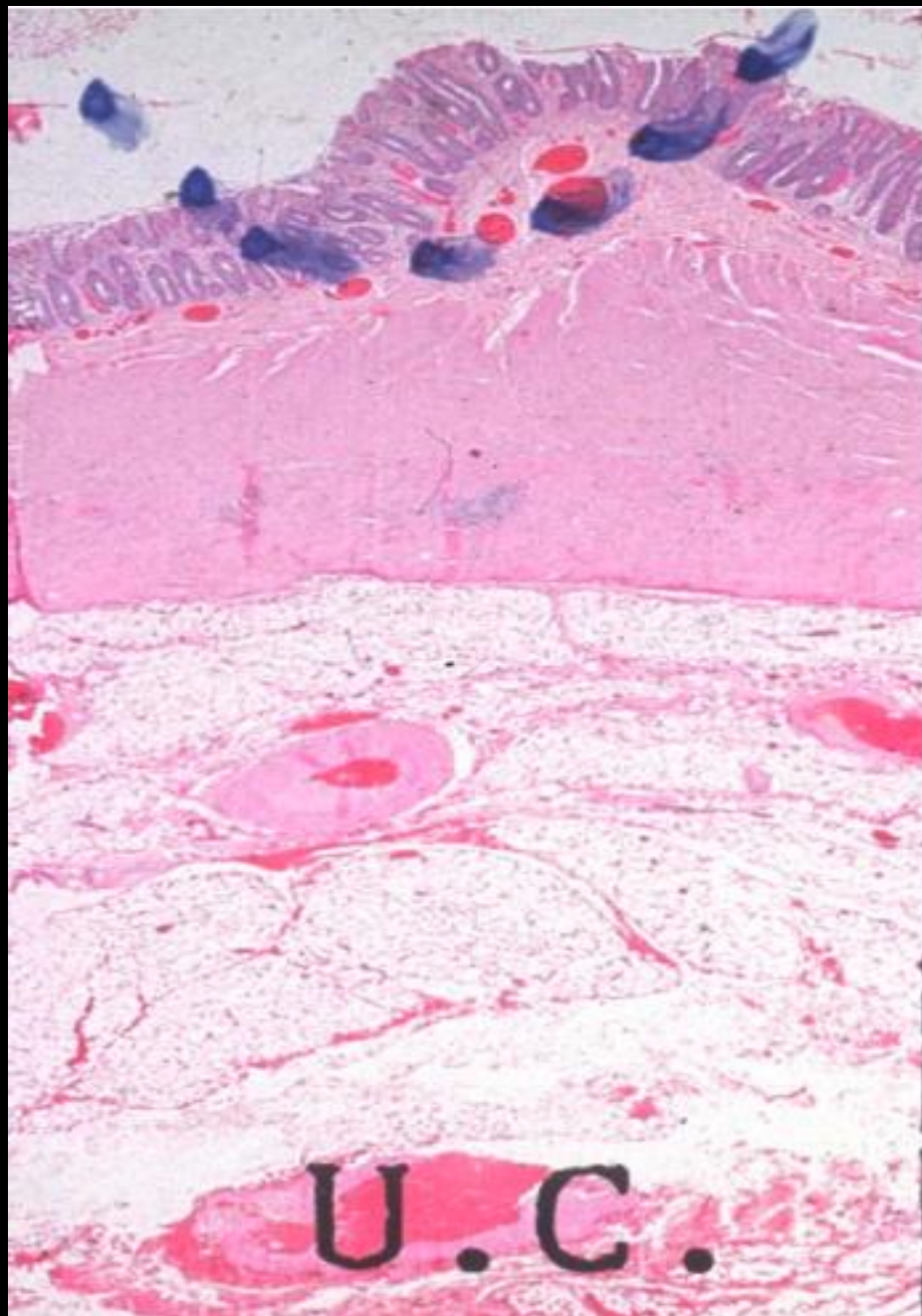
Things I used to call Crohn's Disease

➤ Patchy Distribution

- Left sided UC with peri-appendiceal disease (The cecal red patch)
- After therapy there is often uneven healing

➤ Rectal Sparing

- Steroid enemas
- Burnout in long-standing disease
- Rare cases can present with a normal rectum

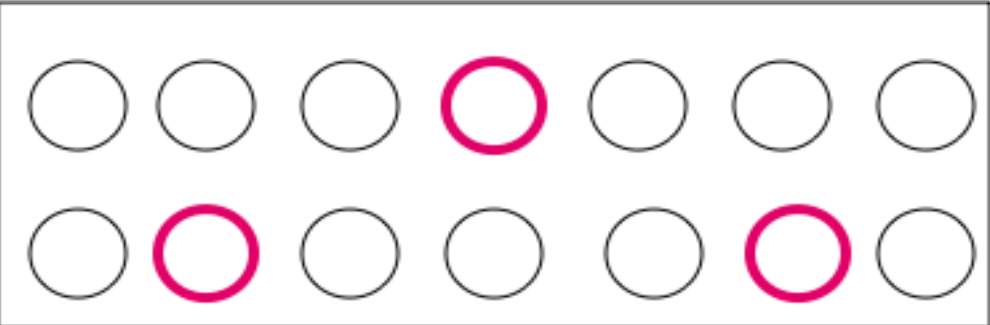


U.C.

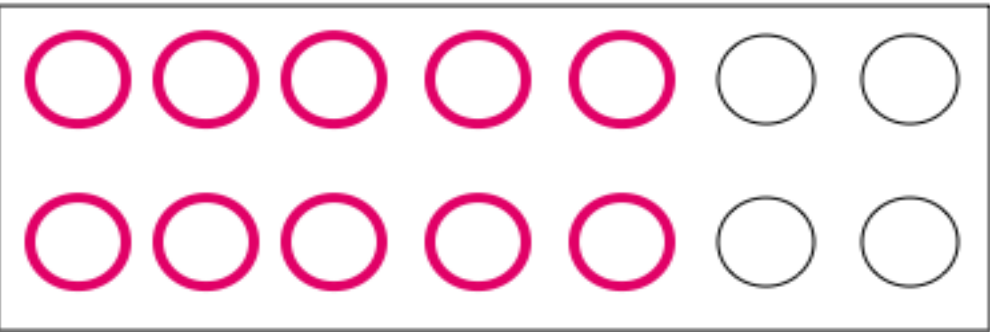


CRN'S

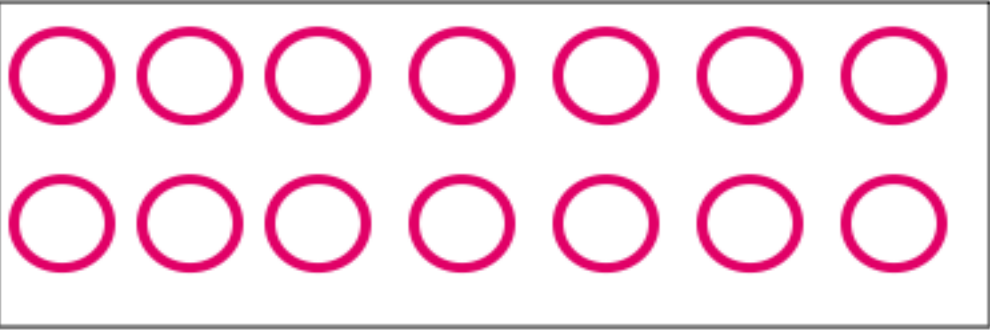
 = Cryptitis  = Normal



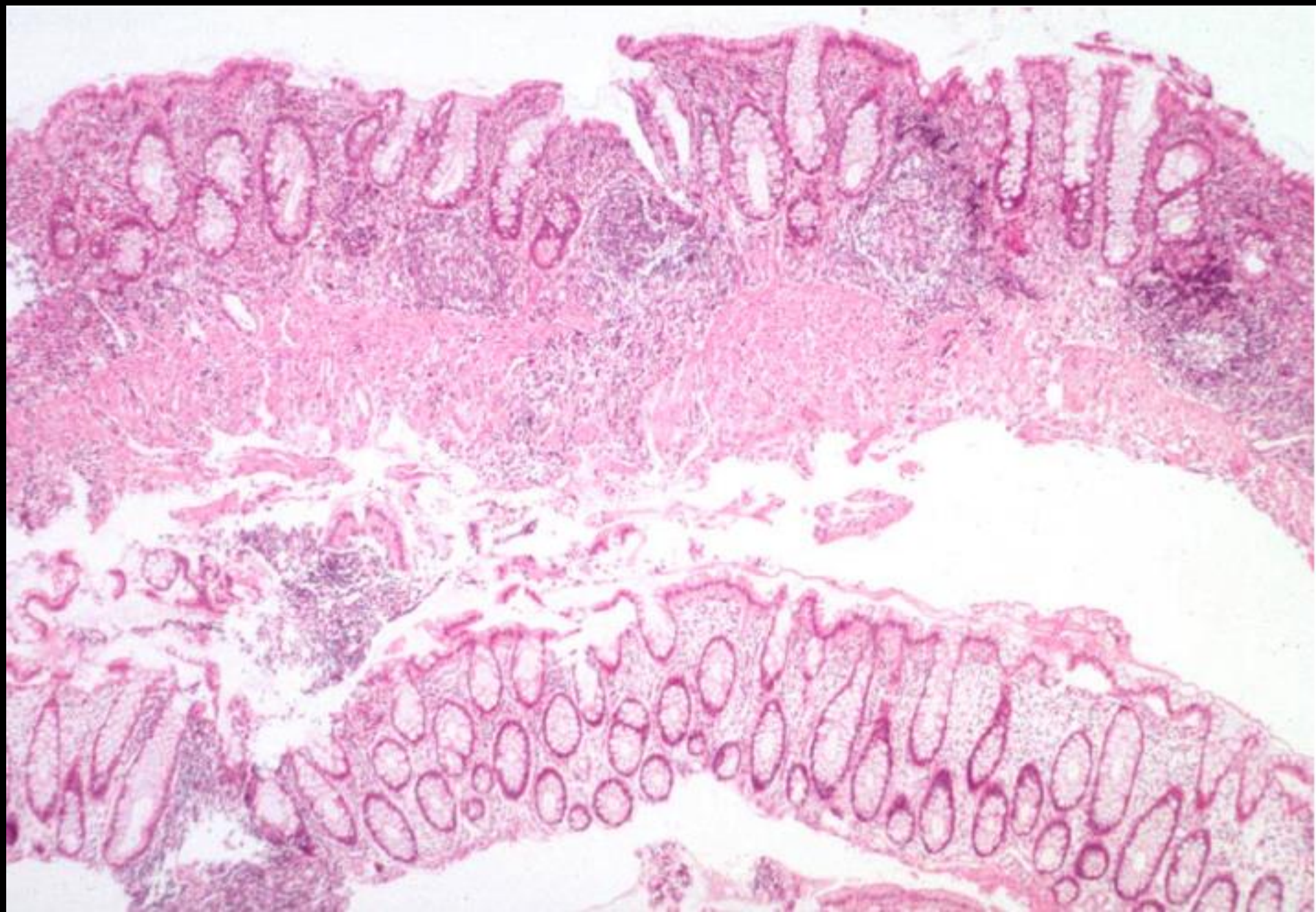
FOCAL



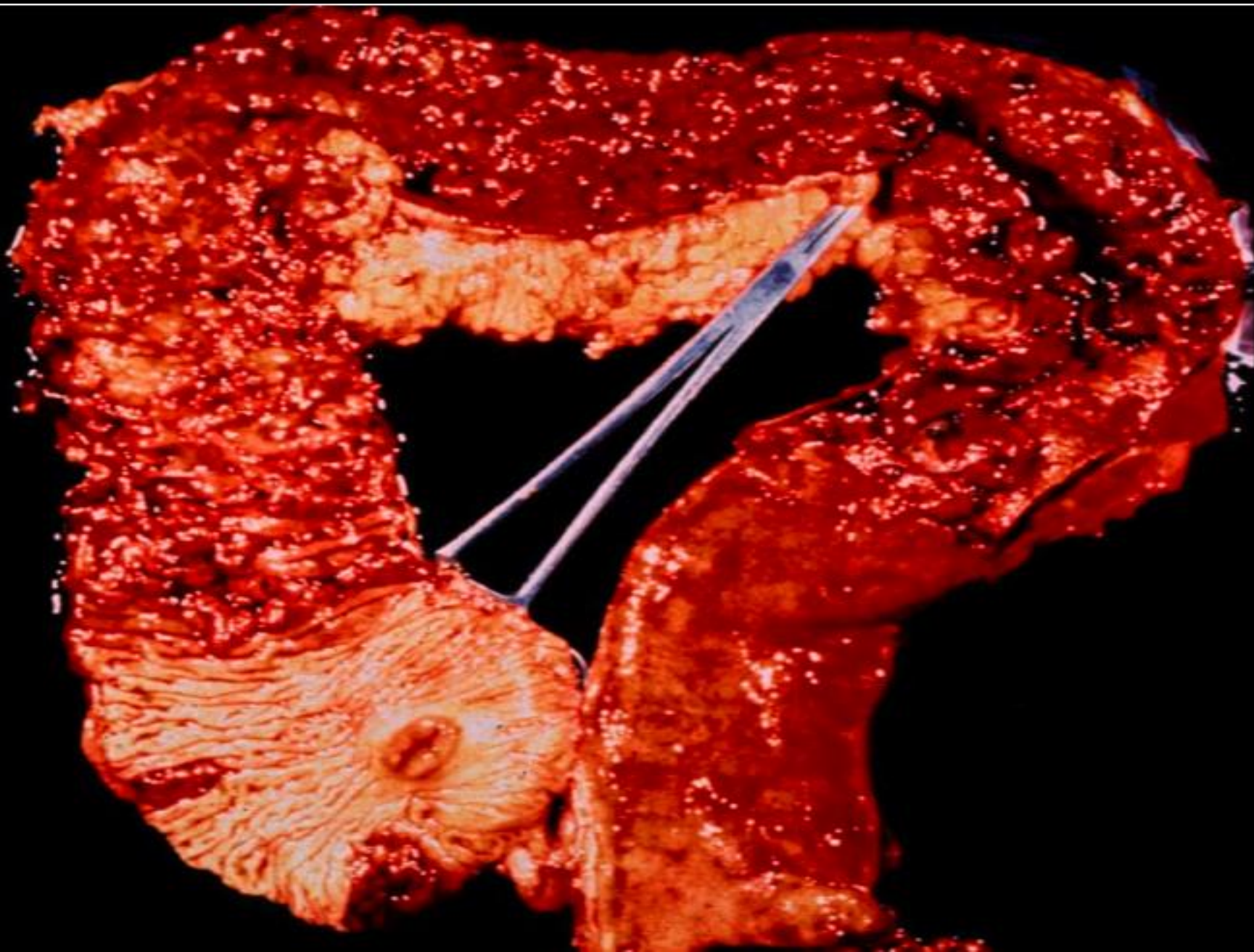
PATCHY



DIFFUSE







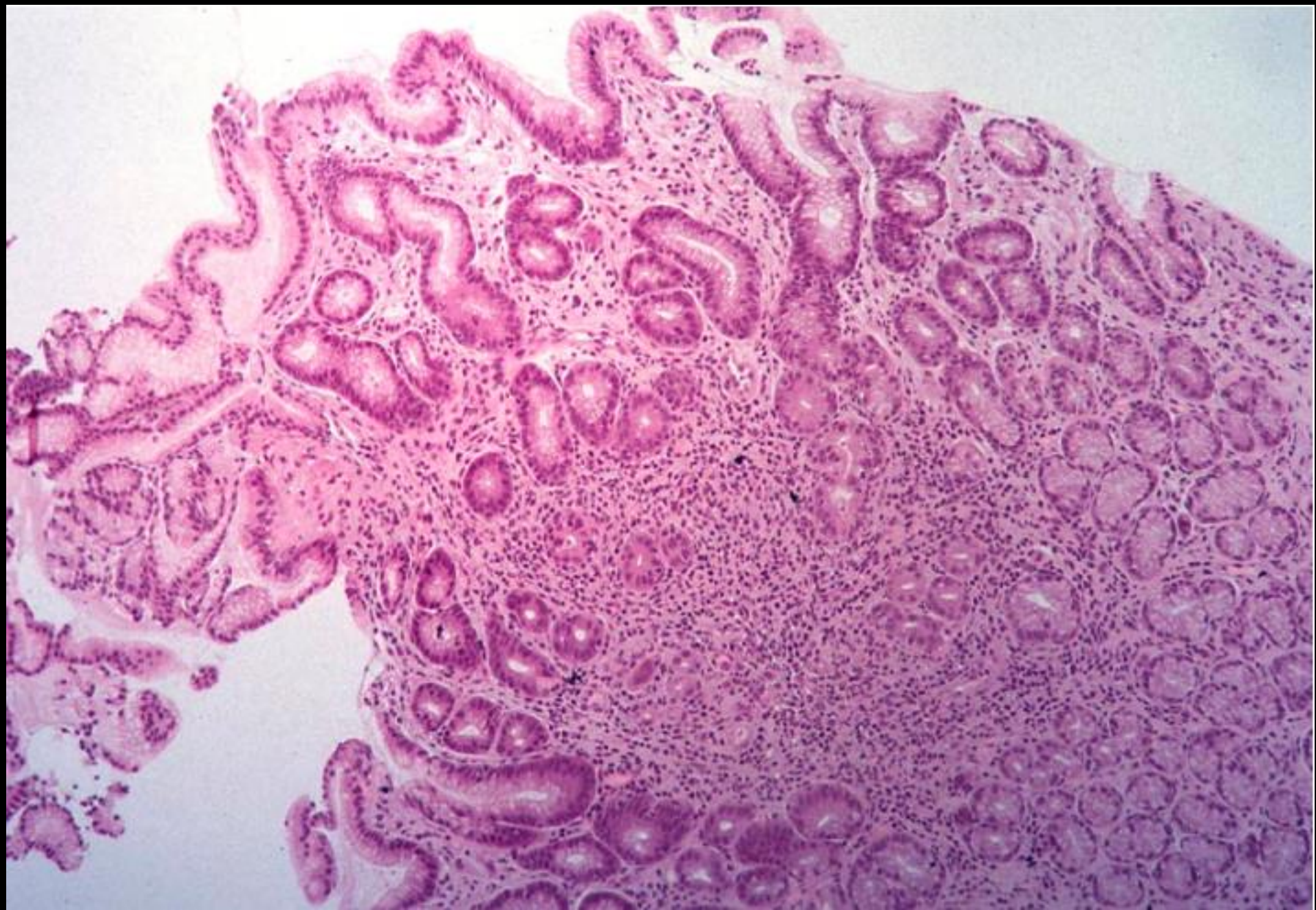
Ulcerative Colitis Extra-Colonic Disease?

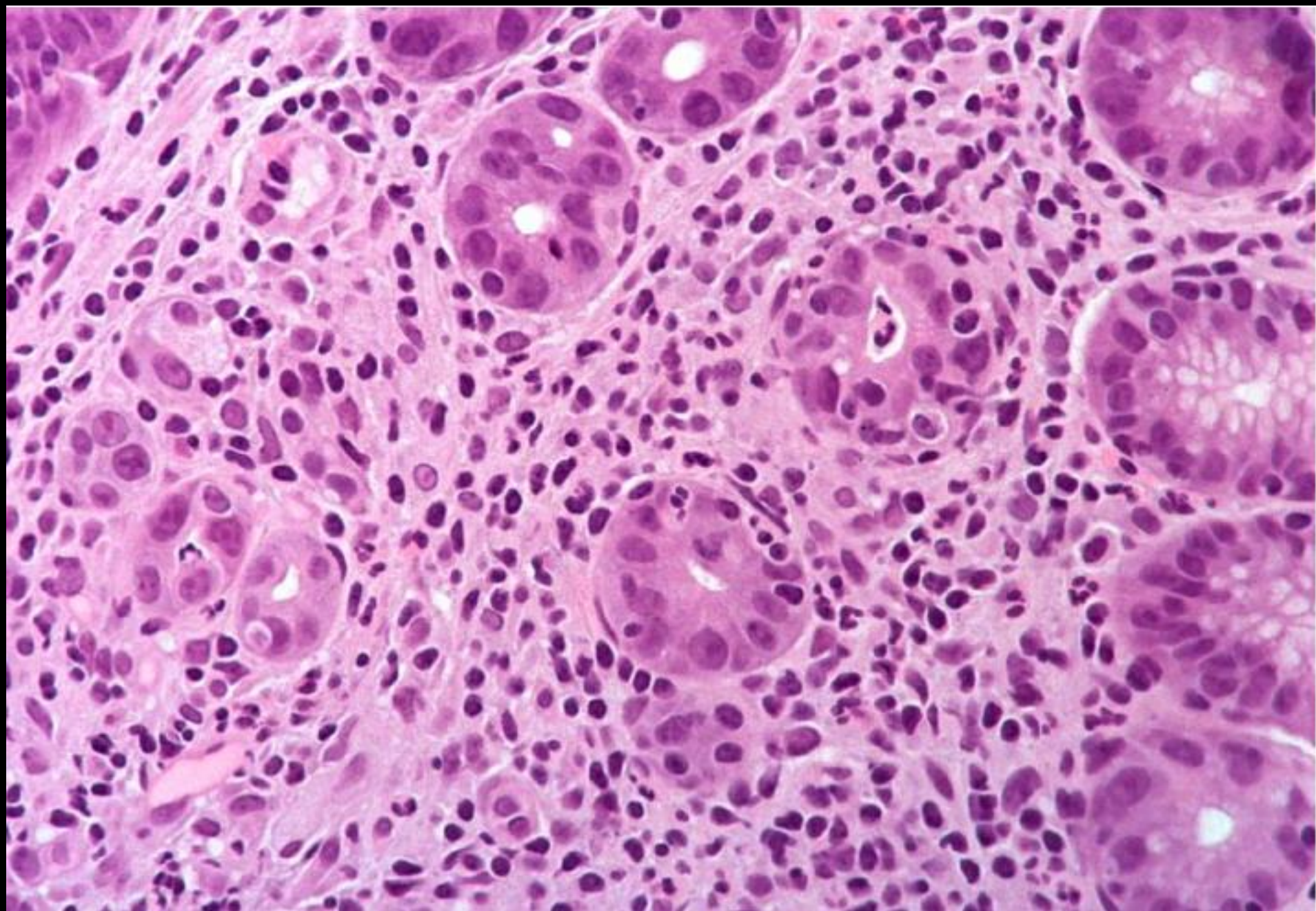
➤ Gastritis

- Focally enhanced gastritis (FEG) thought to be typical of Crohn's.
- 2 recent studies found 12% and 50% of UC patients had FEG compared to 43% and 35% of CD patients.

➤ Duodenitis

- Over the last 5 years many case reports have found diffuse duodenitis in patients with resection proven UC
- Several of these patients also had gastritis
- Pts tolerated endorectal pull-through procedures





Ulcerative Colitis

Histology in the new millennium

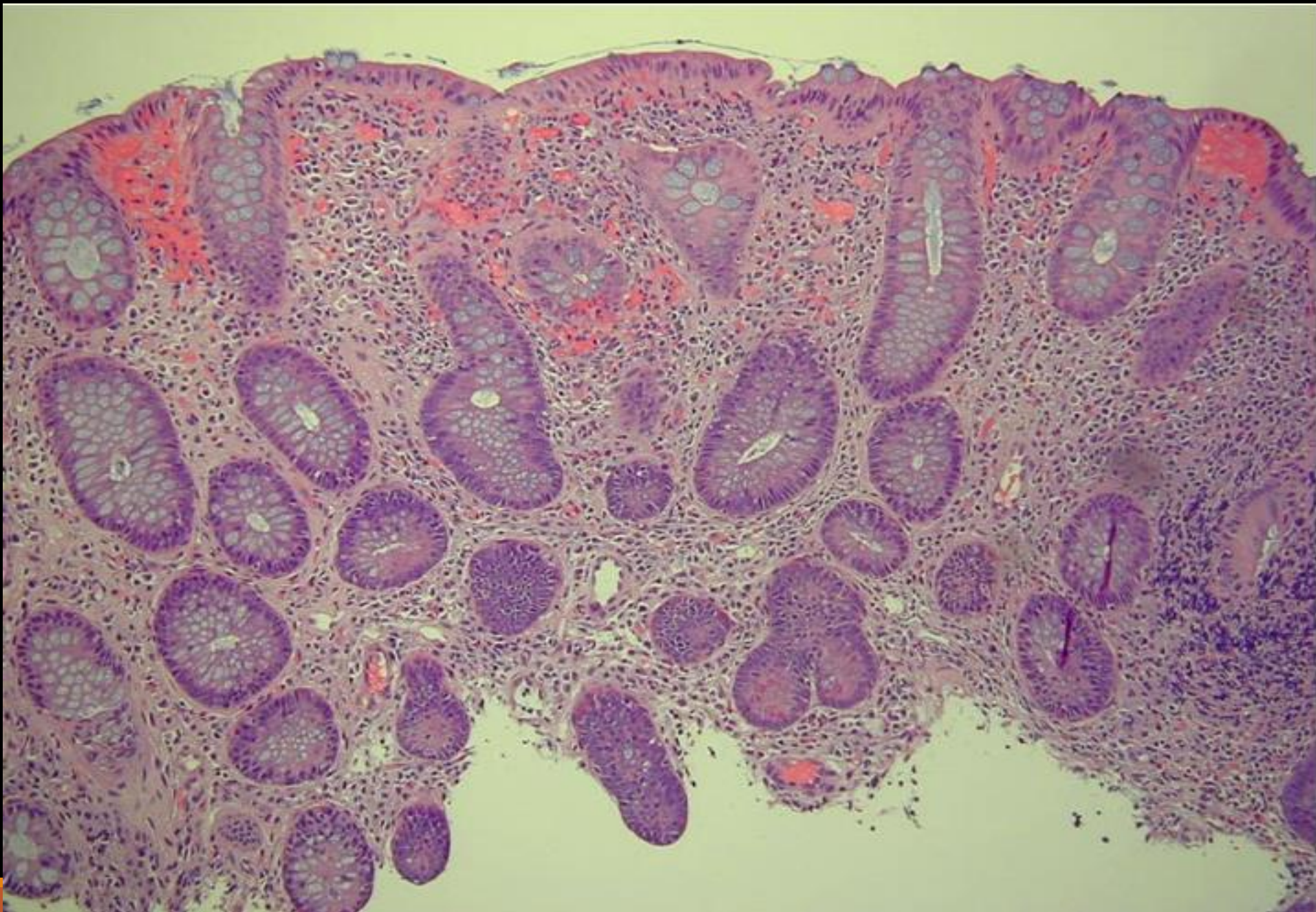
- Patchy distribution is often seen once the patient is on medical therapy.
- Rectal sparing can be seen in longstanding disease, in patients using steroid enemas, and rarely in de novo UC.
- Skip lesions (cecal patch) can be seen in UC.
- Focal gastritis and diffuse duodenitis can be seen in UC.

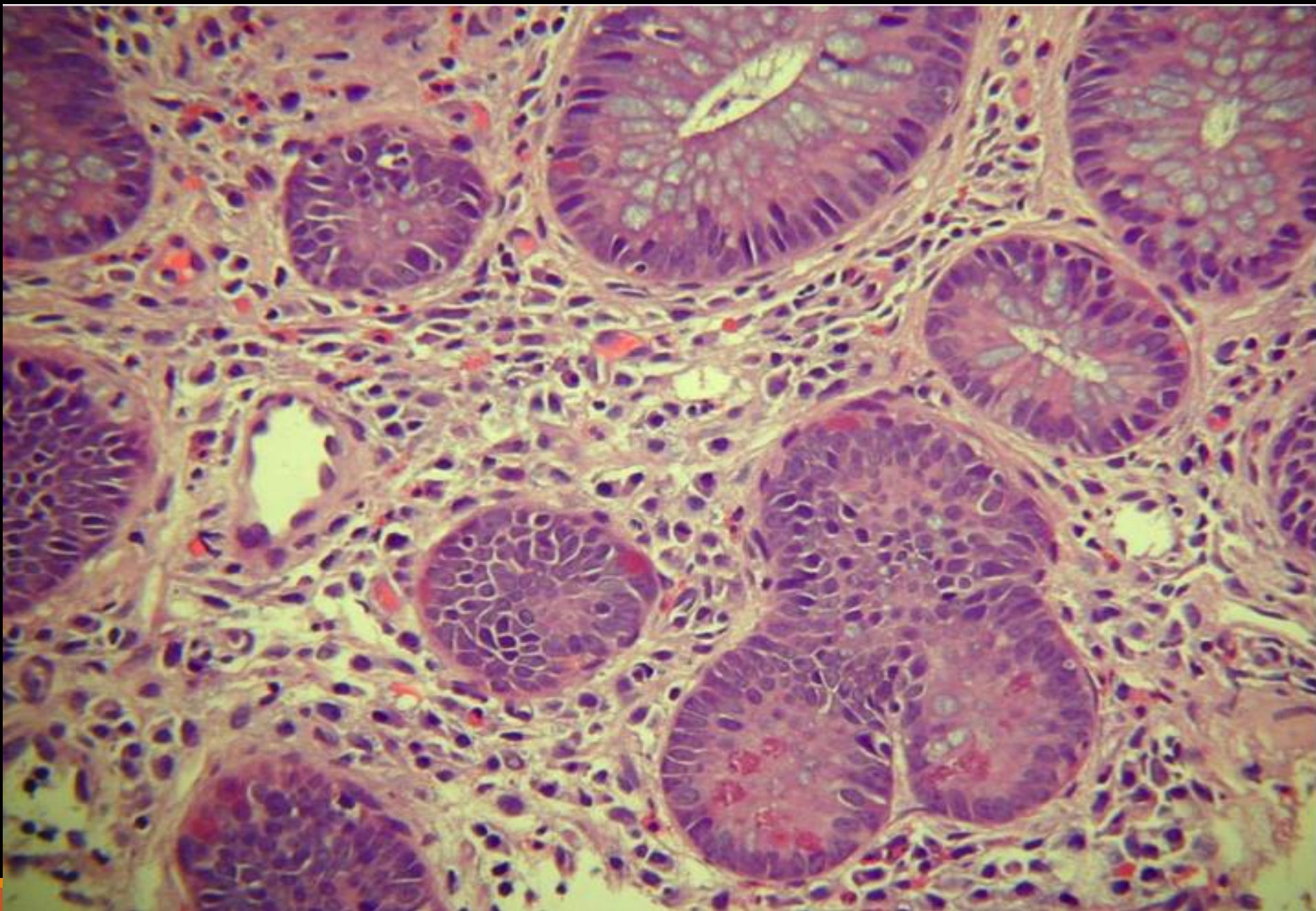
IBD Mimickers

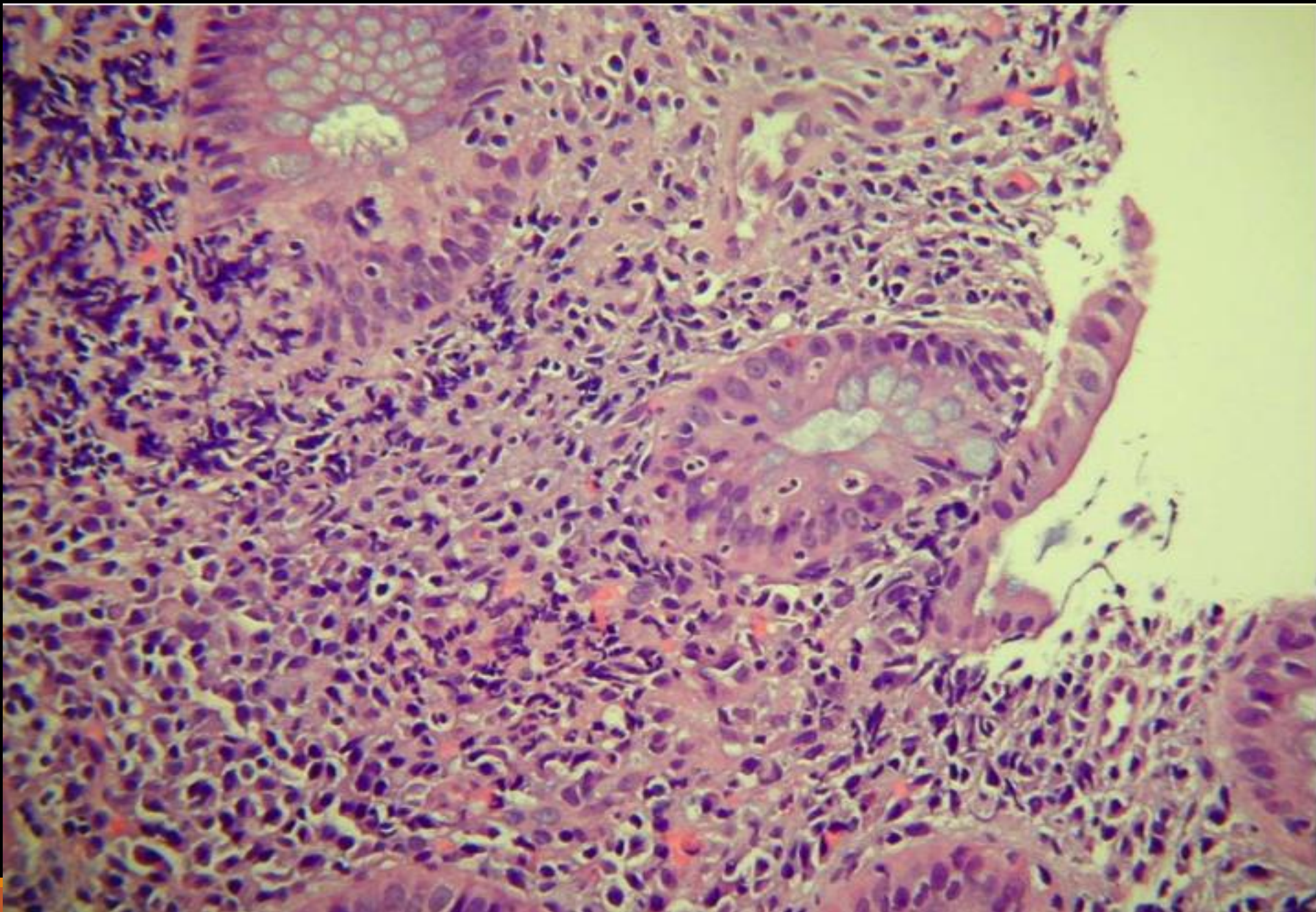
-
1. WITH SPECIFIC ANATOMICAL DISTRIBUTION
 2. INFLAMMATION WITH NO SIGNIFICANT ARCHITECTURAL DISORDER
 - a) Mostly active process
 - b) Mostly interstitial process
 3. MULTIFOCAL ULCERATIONS
 4. CHRONIC INTRAEPITHELIAL PROCESS

Case 1

THESE BIOPSIES ARE FROM THE SIGMOID COLON OF A 67 YEAR-OLD MAN WITH ABDOMINAL PAIN AND OCCASIONAL DIARRHEA. THE ENDOSCOPIST NOTED MILD ENANTHEMA IN THE SIGMOID COLON THAT SEEMED TO SPARE THE RECTUM. SCATTERED DIVERTICULA WERE ALSO NOTED.







Case 1

THESE BIOPSIES ARE FROM THE SIGMOID COLON OF A 67 YEAR-OLD MAN WITH ABDOMINAL PAIN AND OCCASIONAL DIARRHEA. THE ENDOSCOPIST NOTED MILD ENANTHEMA IN THE SIGMOID COLON THAT SEEMED TO SPARE THE RECTUM. SCATTERED DIVERTICULAE WERE ALSO NOTED.

Diverticular Disease Associated Colitis

- IBD-like inflammatory disease that mimics UC
- Mild chronic colitis in distribution of diverticula
- Segmental colitis in sigmoid colon of sixty year olds (the 3 S's)
- Spares the rectum (no tics here)
- This is not diverticulitis
- Pathogenesis is unknown

Makapugay et al. Am J Surg Pathol 20:94-102, 1996.

Diverticular Disease Associated Disease

- Patients typically present with hematochezia
- Endoscopy shows hyperemia, granularity, and/or exudates -spares the rectum
- Therapy is varied: Some respond to fiber and antibiotics but UC-type therapy often needed.
- Some patients are refractory – need resection.
- Some patients develop full-blown UC

Diverticular Disease Associated Colitis

- **Histology similar to mild UC**
 - Increased plasma cells in lamina propria
 - Mild crypt distortion
 - Paneth cell metaplasia
 - Cryptitis and crypt abscesses
 - Can be more intense and look like severe UC
- **Without endoscopic description of tics – tough DX to make!**

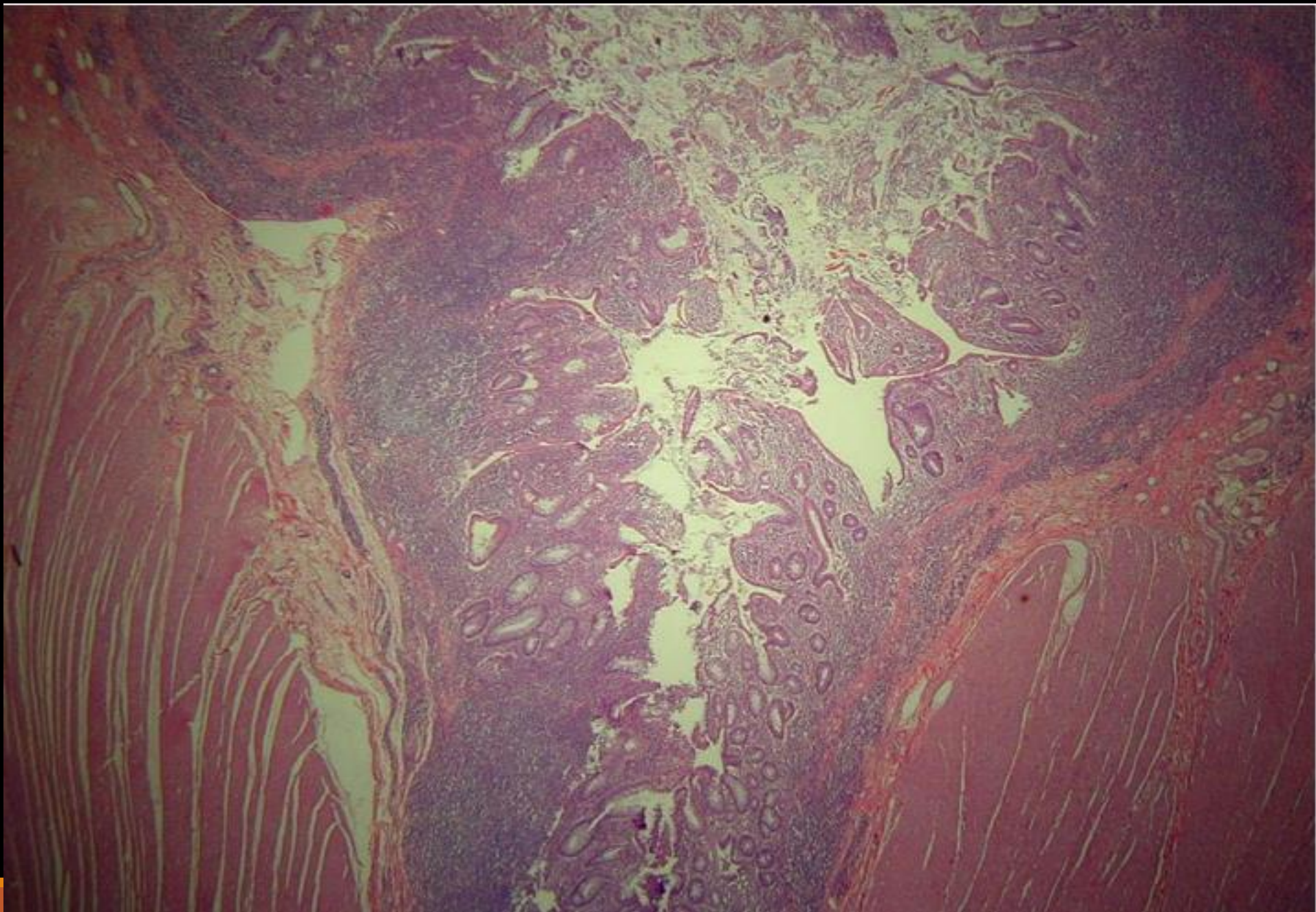
Diverticular Disease Associated Colitis

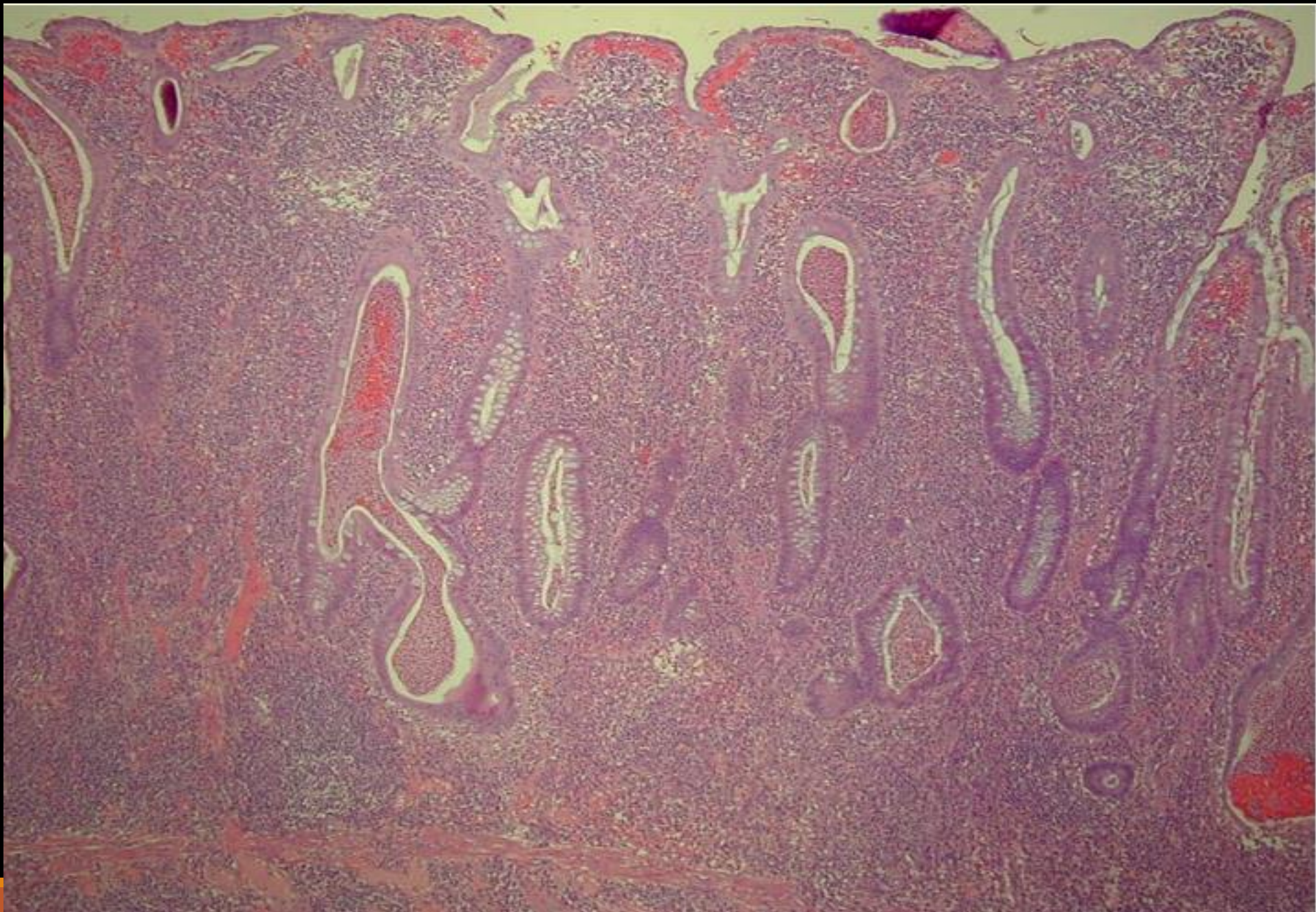
➤ Differential DX:

- UC with rectal sparing (seen in longstanding disease or with use of steroid enemas)
- Crohn's disease

➤ Keys to DX:

- Distribution limited to that of tics
- Age older than most *de novo* IBD patients





Crohn's-like Diverticulitis

- **A Crohn's-like colitis found in diverticulitis specimens.**
 - **Patients without any history of IBD**
 - **Grossly fat wrapping and bear claw ulcers**
 - **Granulomas, sinus tracts and transmural inflammation lacks neural hypertrophy, pyloric gland metaplasia and viliform surface change.**
 - **Treatment is simple resection**
- **Pathologist must be careful not to label the patient as having Crohn's disease**

Goldstein et al. Am J Surg Pathol 24:668-675;2000.

Case 2

THESE BIOPSIES ARE FROM THE SIGMOID COLON OF A 41 YEAR-OLD WOMAN WITH ABDOMINAL PAIN AND BLOODY DIARRHEA FOR THE LAST 3-4 WEEKS. THE ENDOSCOPIST NOTED DIFFUSE MILD ENANTHEMA IN THE COLON.

Acute Infectious-type Colitis

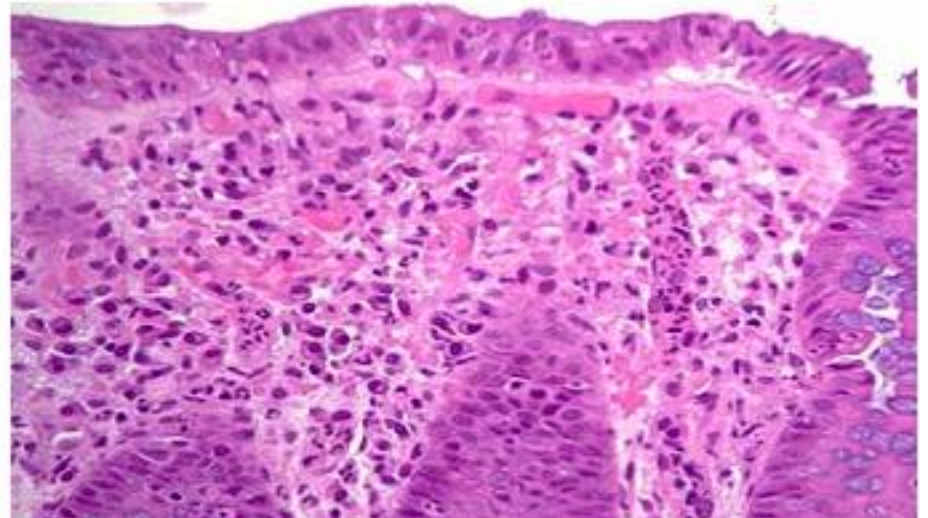
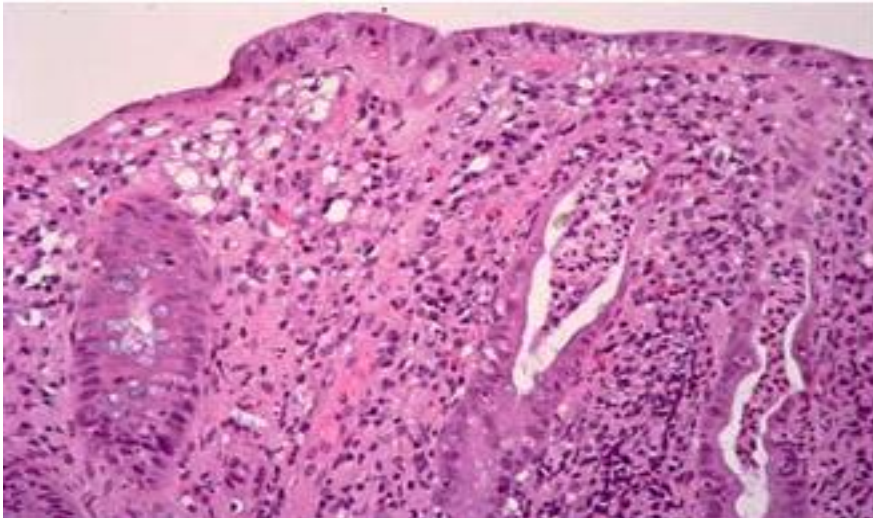
Clinical Presentation

- Acute onset bloody diarrhea
- Similar symptoms are seen in acute onset UC
- Colon biopsies may be required to distinguish between ASLC and new onset UC
- provided the patient's symptoms last long enough to get past their "gate keeper" and see a gastroenterologist

Acute Infectious-type Colitis

Histopathology

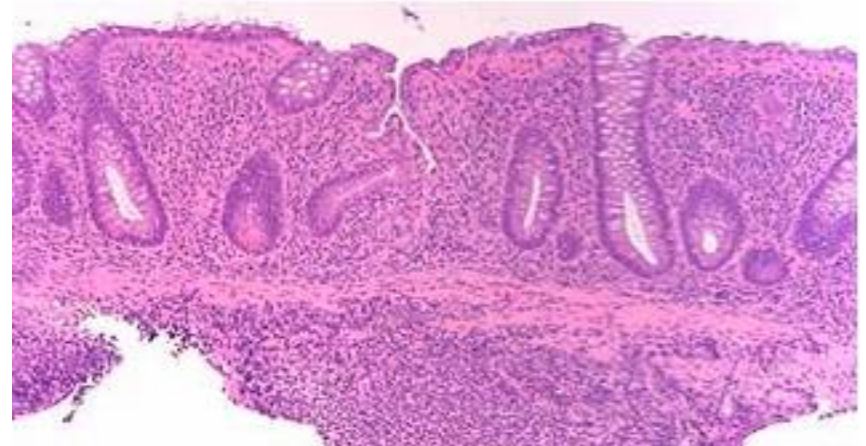
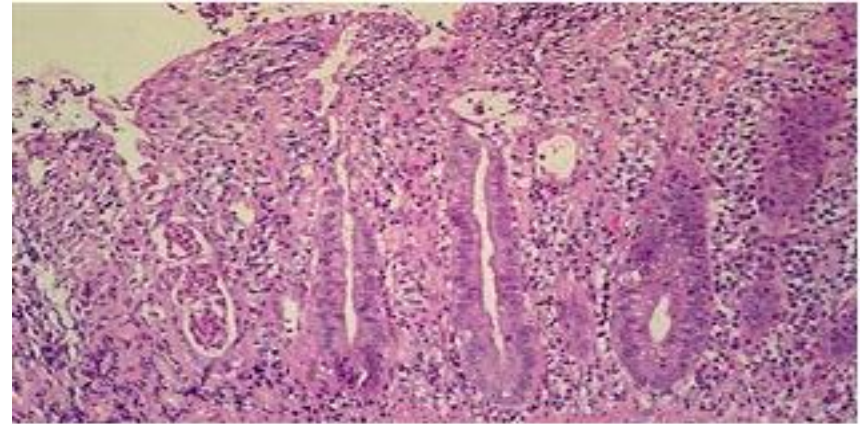
- At peak activity ASLC shows cryptitis, crypt abscesses, edema, and surface damage with erosions.



Acute Infectious-type Colitis

Histopathology

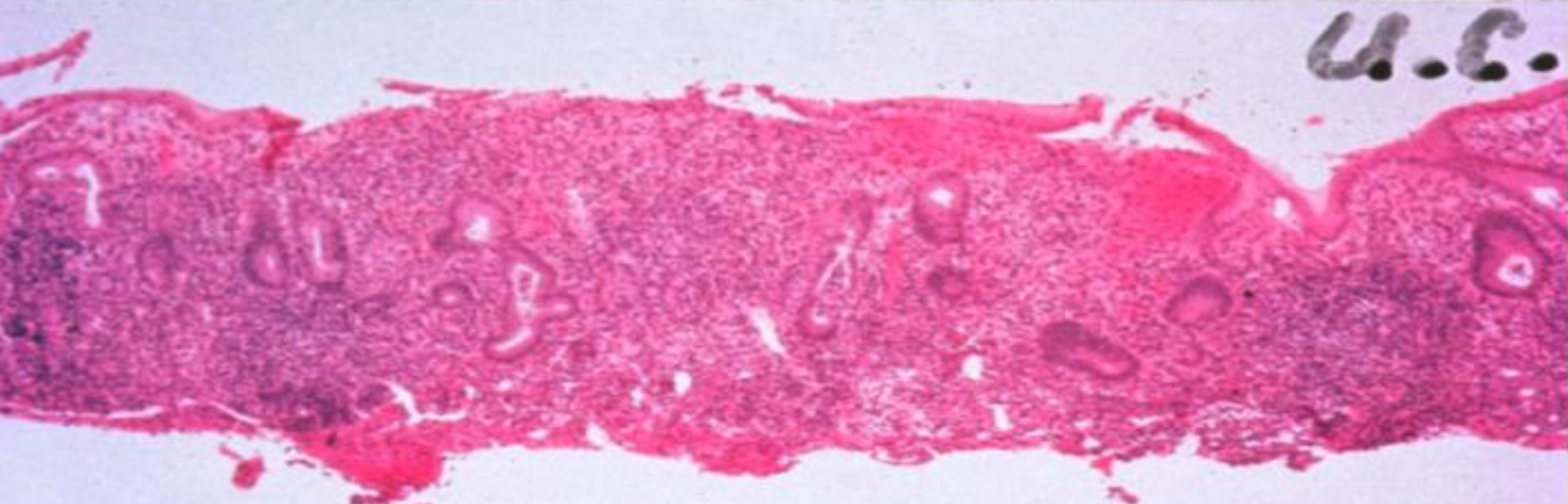
- ASLC does not have crypt distortion or basal plasma cells
- UC often has both crypt distortion and basal plasma cells even at first onset



SAL.



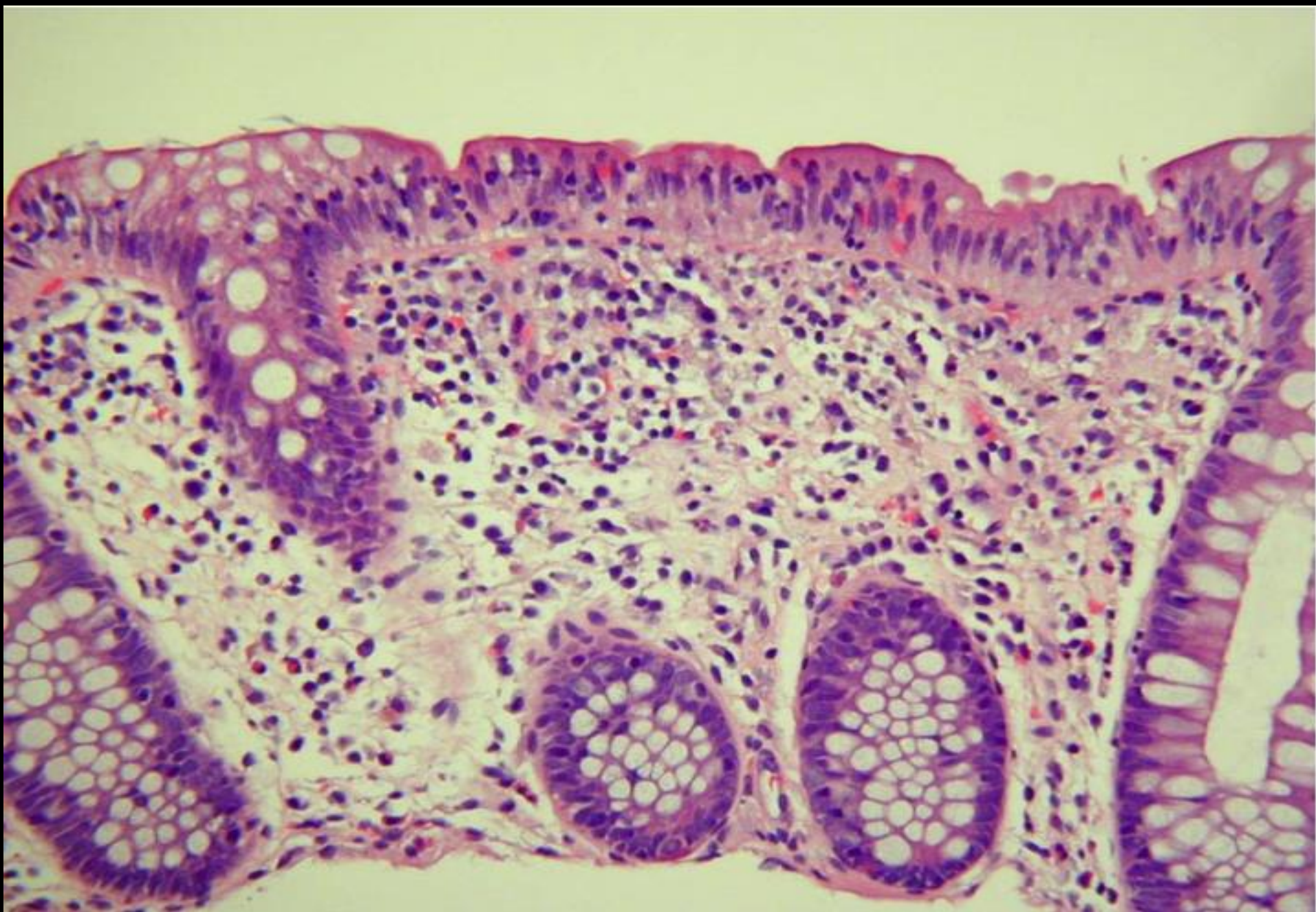
U.C.



Acute Infectious-type Colitis

Histopathology – Resolving ASLC

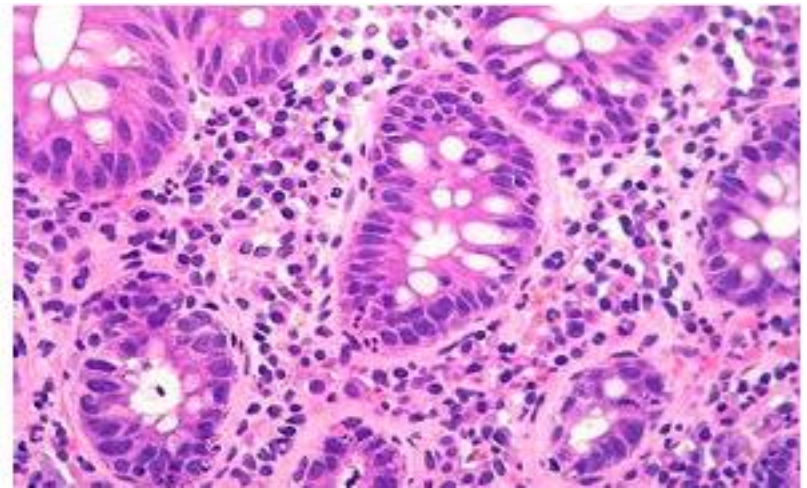
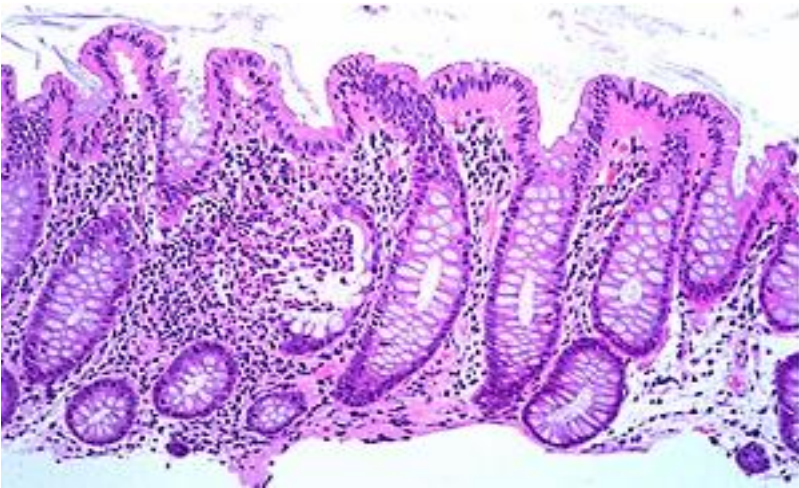
- Lamina propria may be hypercellular with increased lymphs, eos, polys, and a few plasma cells - Don't be fooled into calling this chronic colitis!
- There may be an increase in intraepithelial lymphocytes such that the changes mimic lymphocytic colitis - Don't be fooled, as the clinical history is not right for this!

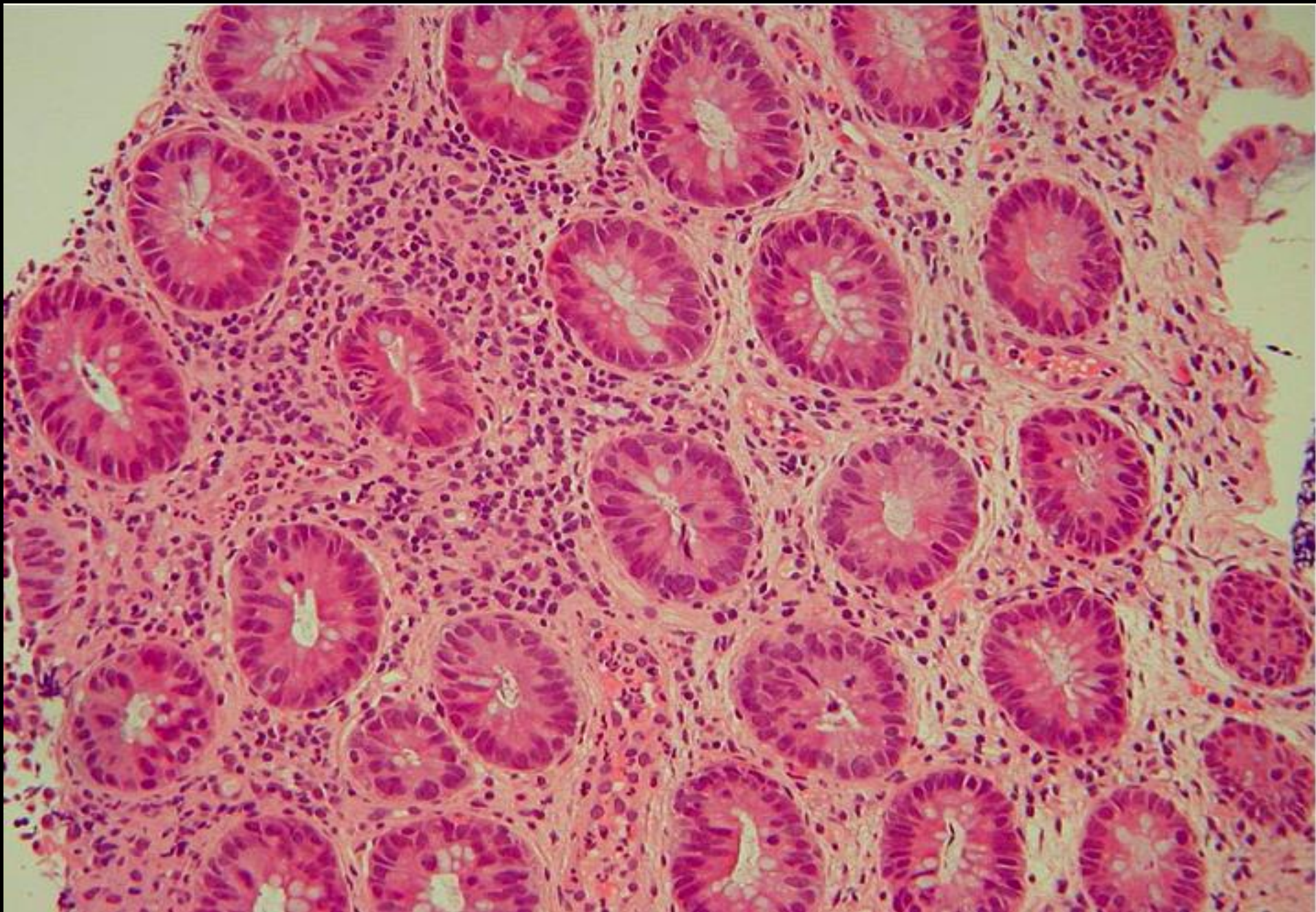


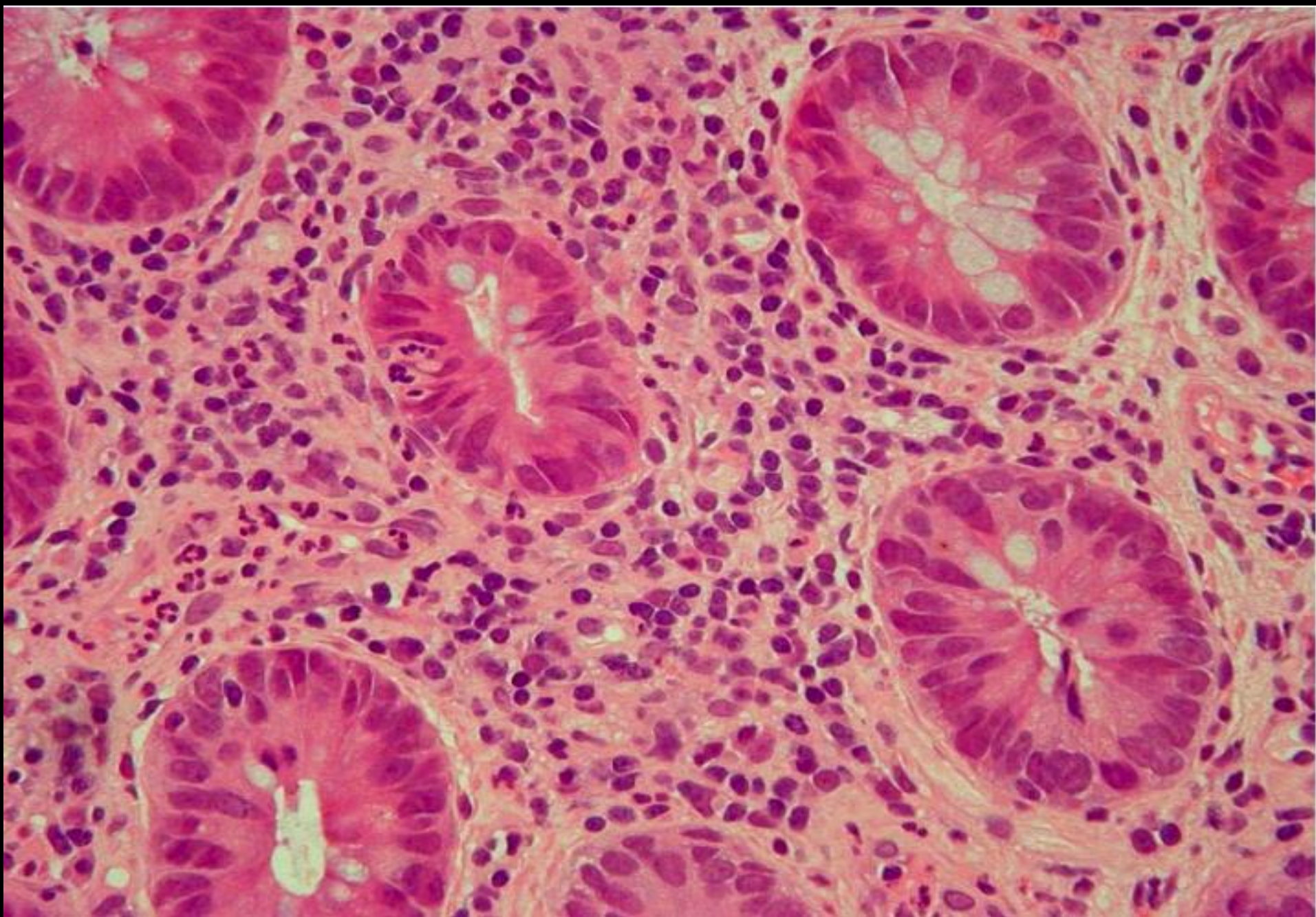
Acute Infectious-type Colitis

Histopathology – Resolving ASLC

- As ASLC resolves, there is mucus depletion with regenerative epithelial changes and a few residual foci of cryptitis or “focal active colitis”







Etiology of Focal Active Colitis

Diagnosis	Adult #1*	Adult #2**	Children
Infectious	55%	48%	31%
Incidental	40%	29%	28%
Ischemia	5%	10%	0%
Crohn's	0%	13%	28%
Allergic	0%	0%	7%
UC	0%	0%	3%
Hirschprung's	0%	0%	3%

* GreensonJK et al. Hum Pathol 28:729-733, 1997

** Volk EE et al. Mod Pathol 11:789-794, 1998

Etiology of Focal Active Colitis

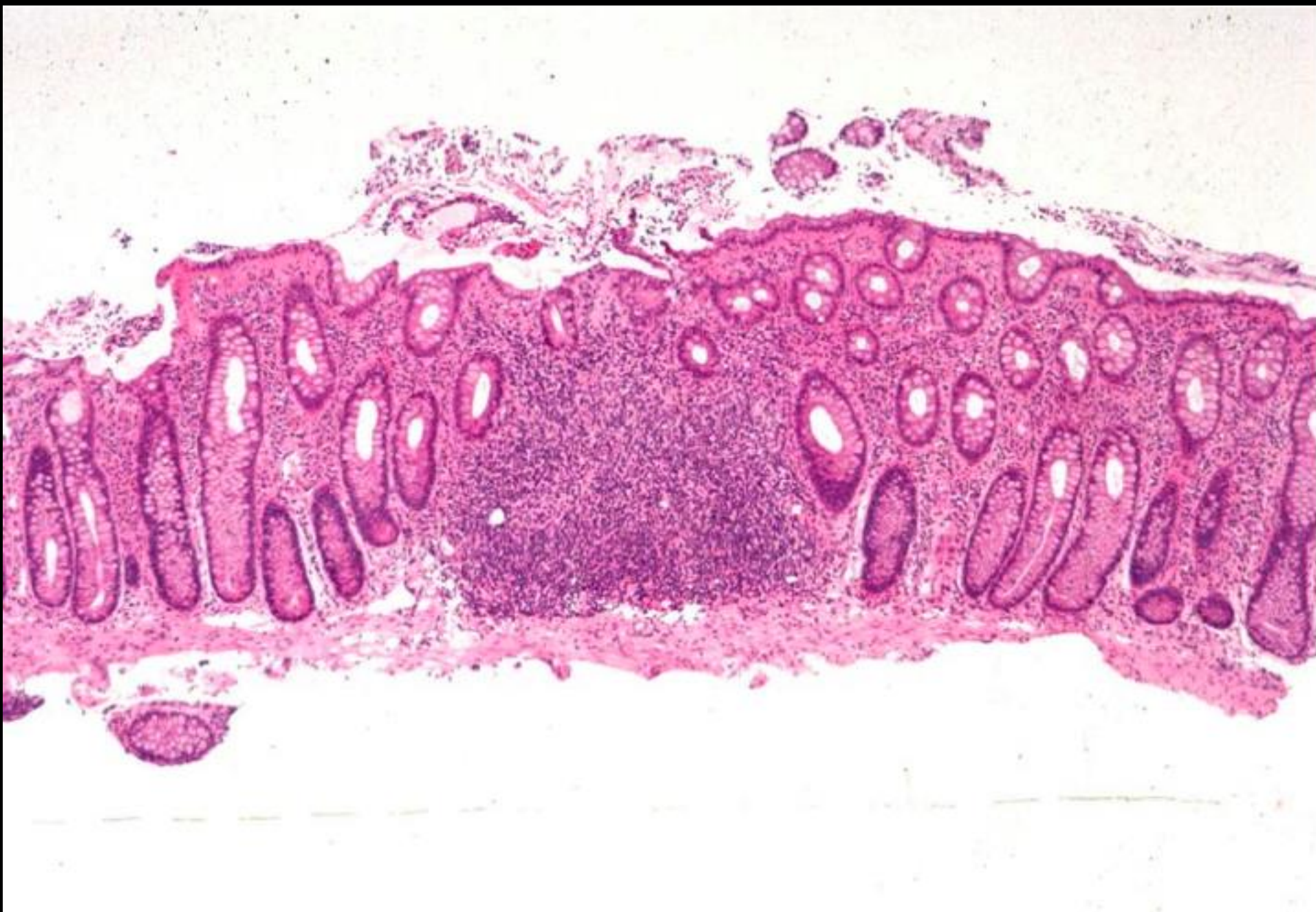
- Oral sodium phosphate bowel preparation caused FAC as well as aphthous lesions of the colon.
- These lesions were not present when patients were re-endoscoped without the same bowel prep 1 to 8 weeks later.

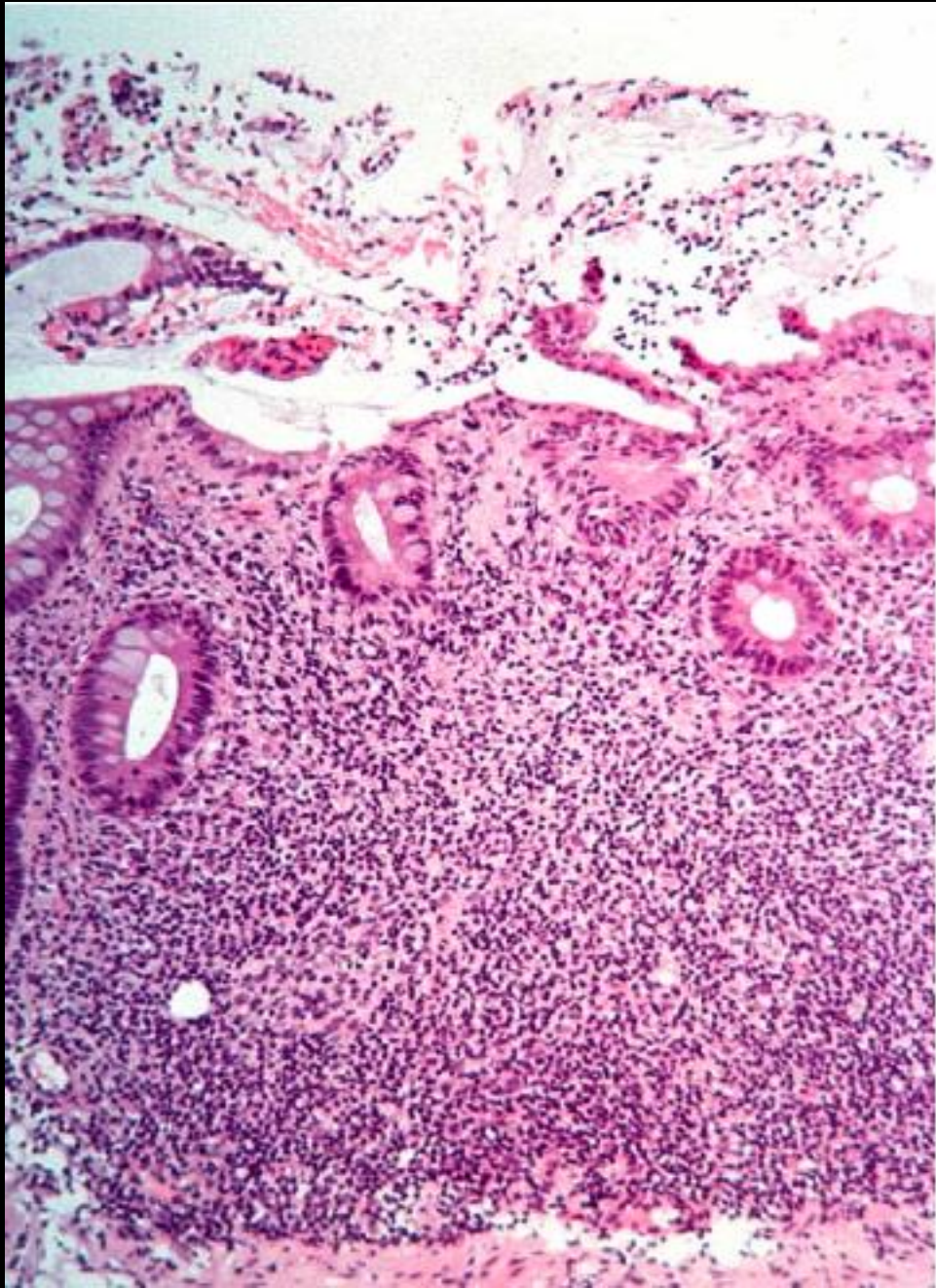
Driman and Preiksaitis. Hum Pathol 1998;29:972-978.

Case 3

THESE RECTAL BIOPSIES ARE FROM A 38 YEAR-OLD MAN WITH CROHN'S DISEASE WHO IS STATUS POST RESECTION OF HIS TERMINAL ILEUM AND RIGHT COLON. HE HAS AN ILEOSTOMY AND A HARTMANN'S POUCH. THE ENDOSCOPIST

DESCRIBED STREAKY ERYTHEMA WITHOUT ULCERS IN THE HARTMANN'S POUCH. (1A AND 1B)



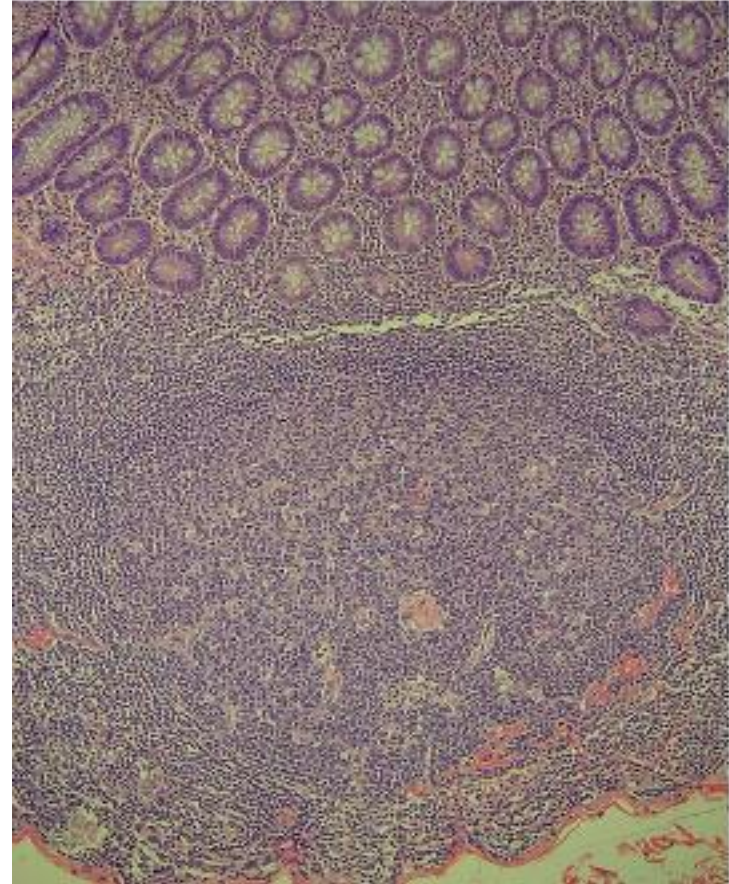


Diversion Colitis

- Occurs in a diverted segment of colon
 - Usually Hartmann's pouch
- Caused by lack of short chain fatty acids in fecal stream
 - Colonocyte malnutrition
- Symptoms: None, bloody or mucus discharge, pain
- Resolves if bowel hooked back up or with fatty acid enemas

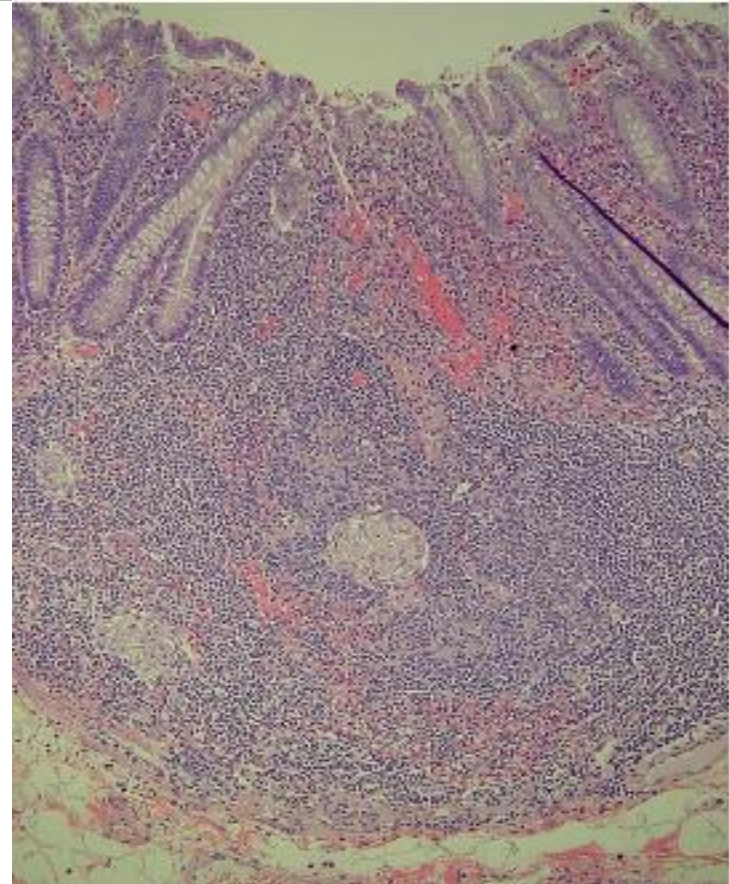
Diversion Colitis

- **Endoscopic:** Enanthema, friability, edema and nodularity ± aphthous erosions.
- **Histology:** Large lymphoid aggregates with prominent germinal centers (so-called diversion reaction)

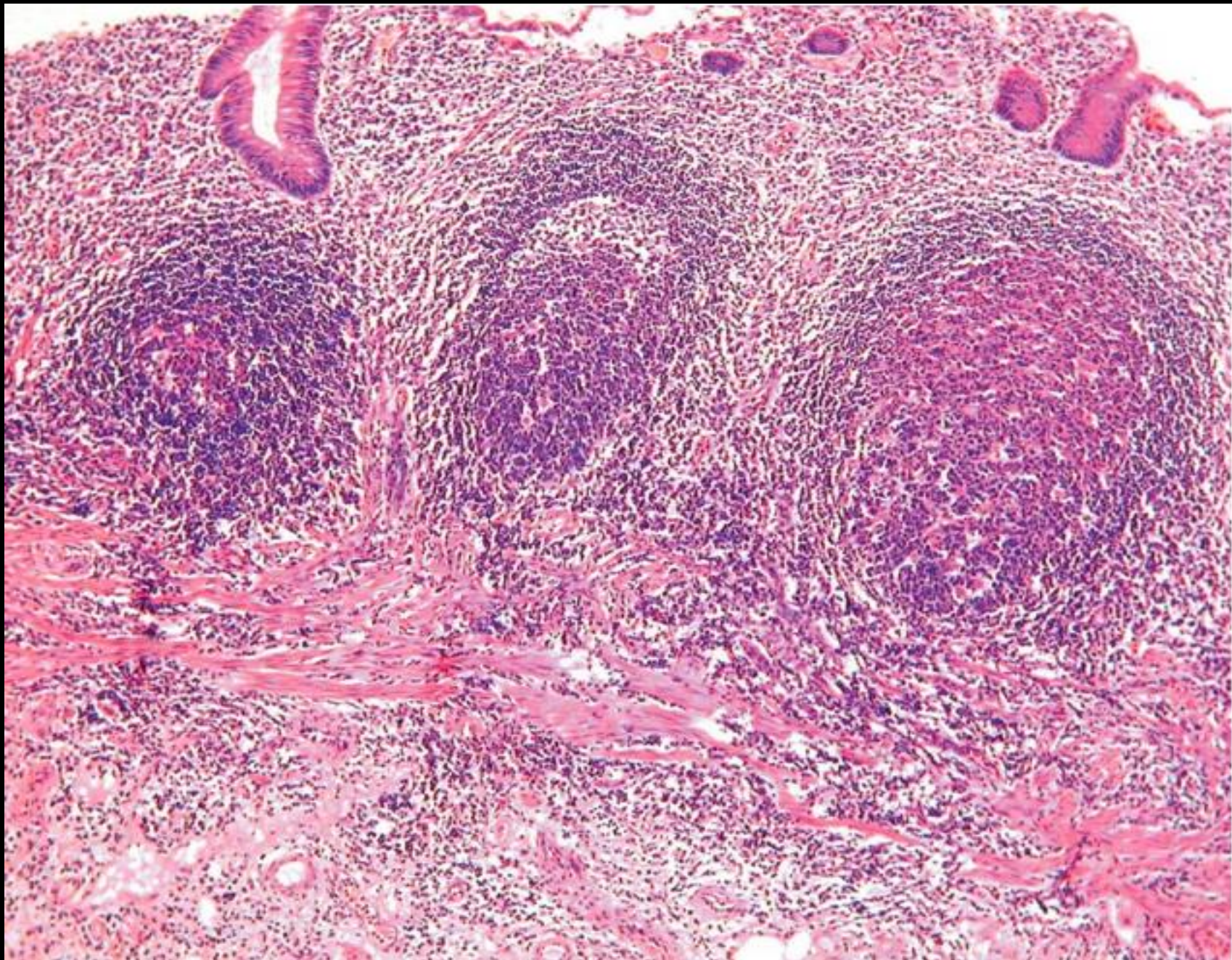


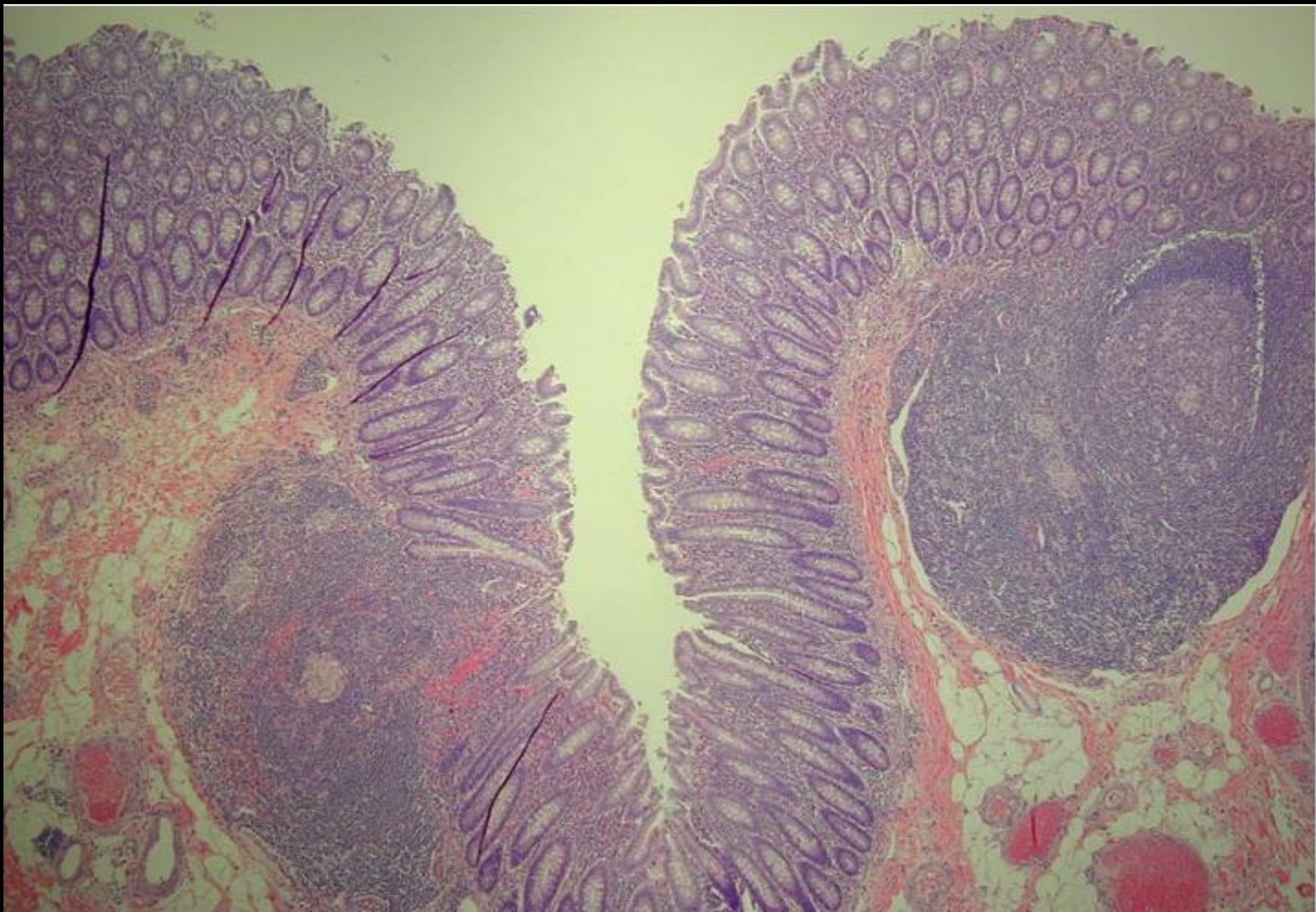
Diversion Colitis

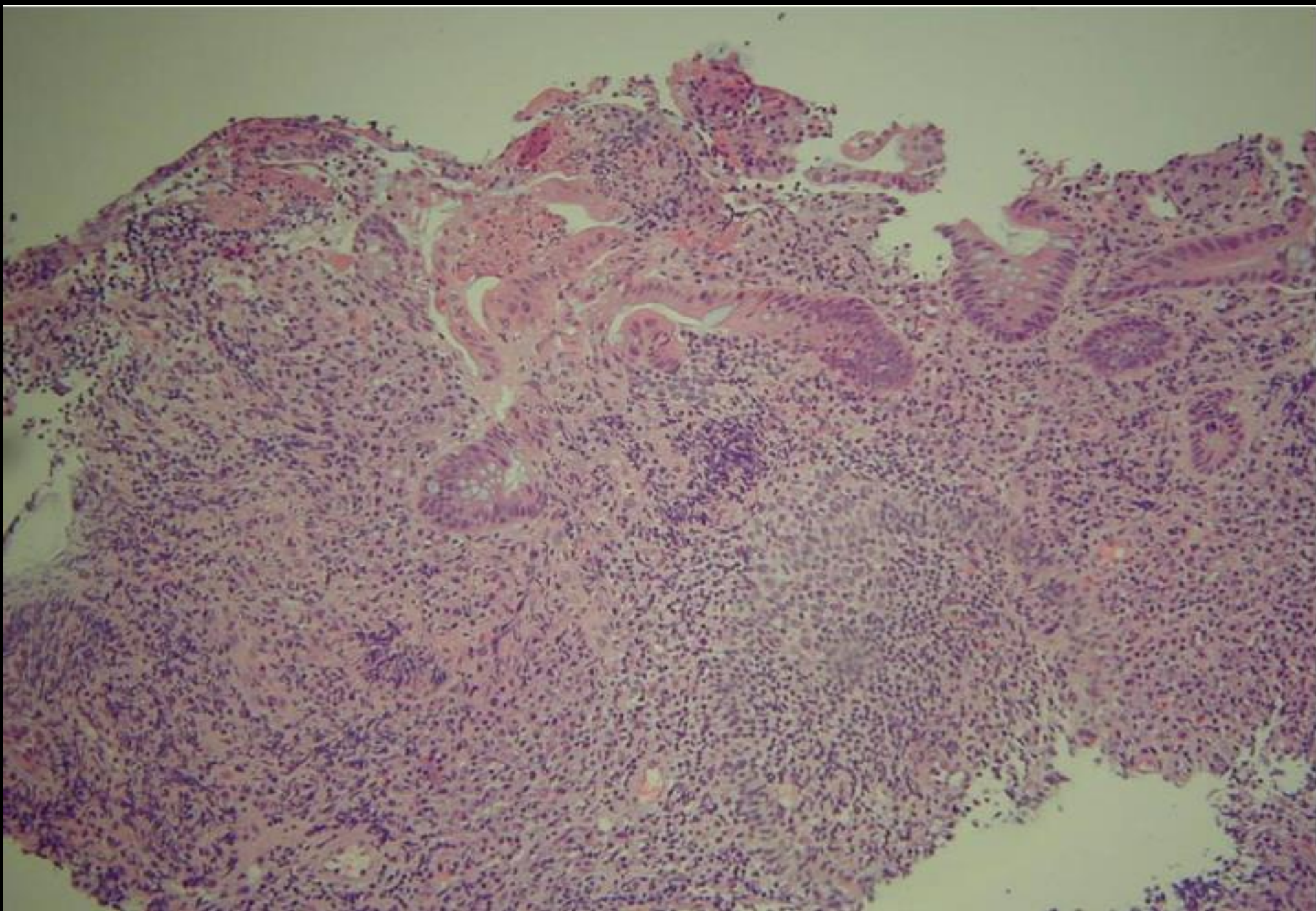
- Cryptitis, Crypt abscesses, polys in lamina propria.
- Aphthous lesions
- May have plasmacytosis and some crypt distortion (due to large lymphoid follicles)

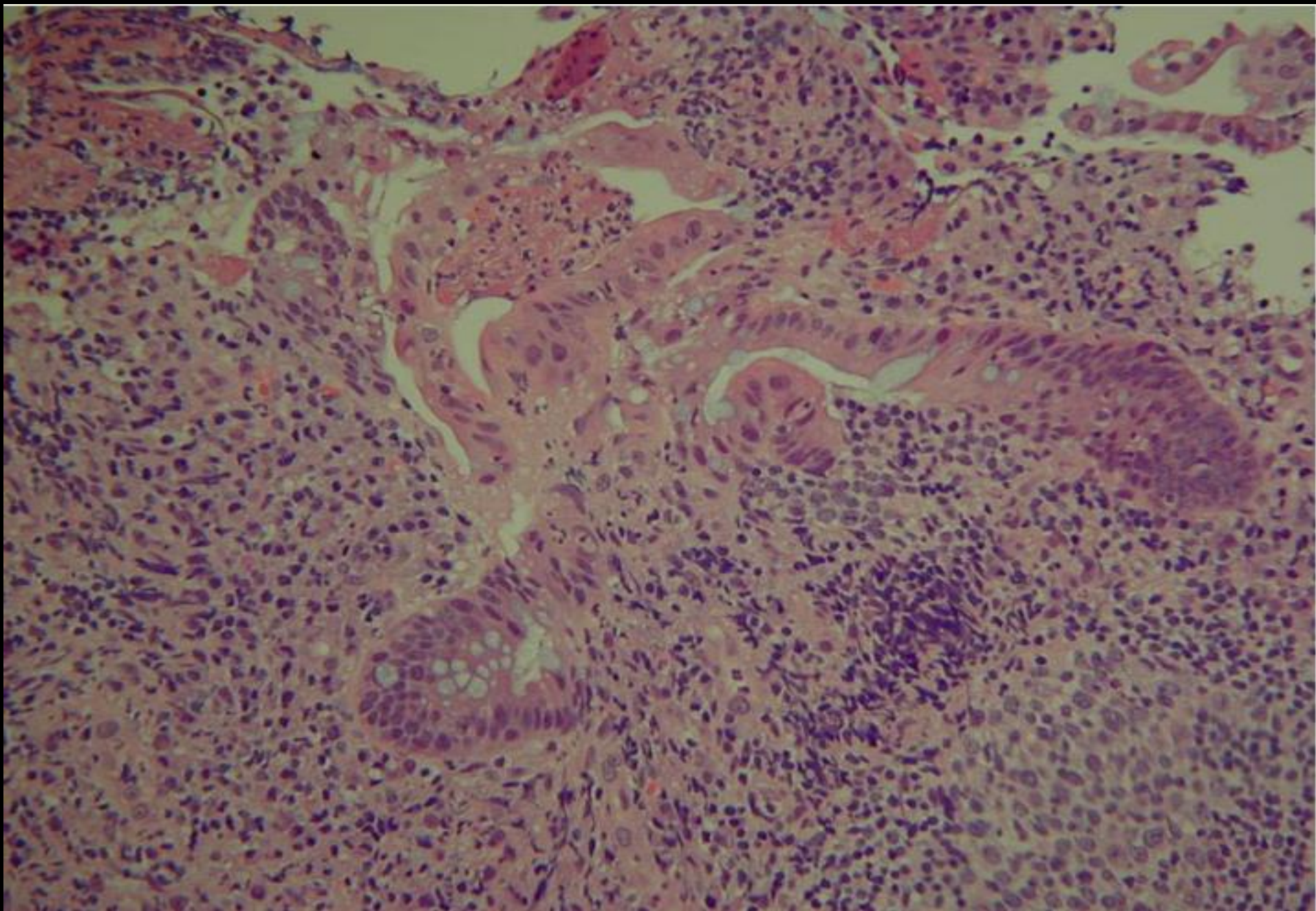










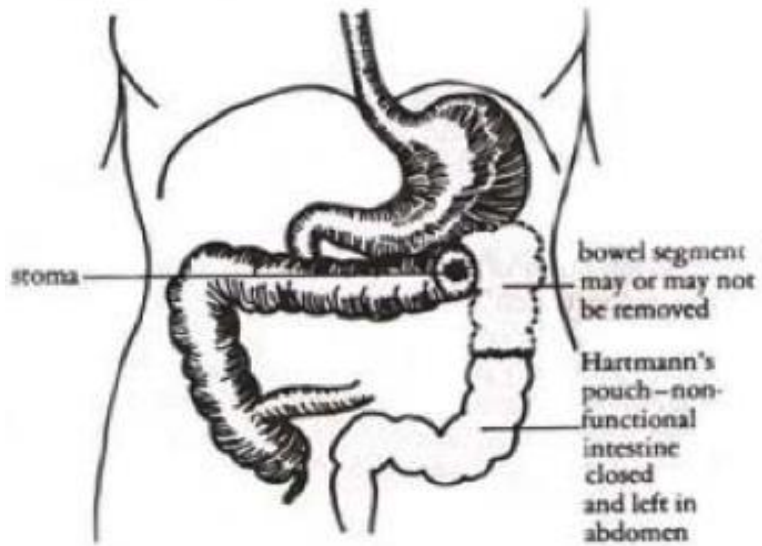




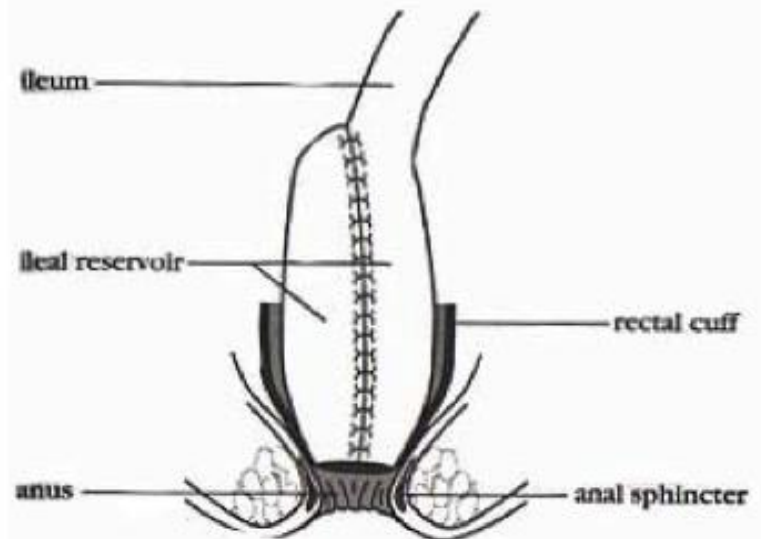
Diversion Colitis - DDX

- **Crohn's Disease**
 - **Common problem in Crohn's patients with a diverted segment: Is it Crohn's or diversion?**
- **Ulcerative Colitis**
- **Infectious Colitis**
- **Pouchitis: Remember a Hartmann's Pouch is diverted segment of colon – not a real pouch!**
- **History is paramount**

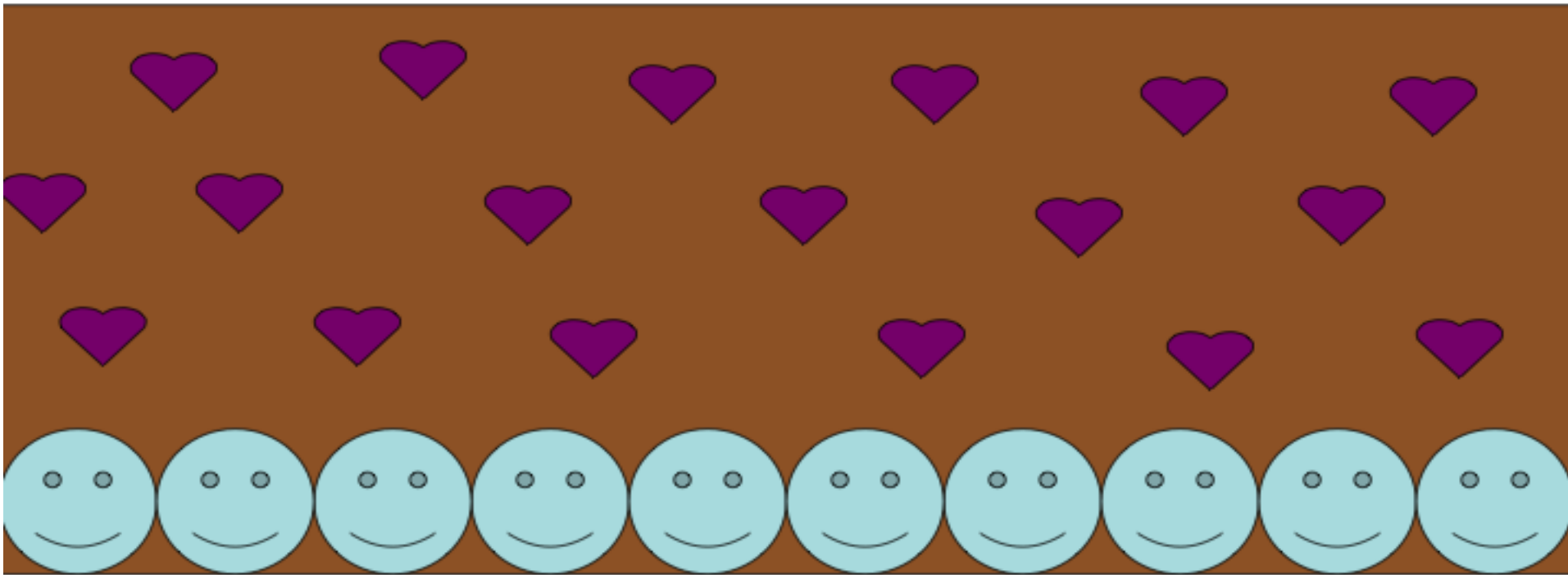
END OSTOMY AND HARTMANN'S POUCH — one stoma



Ileoanal anastomosis with reservoir

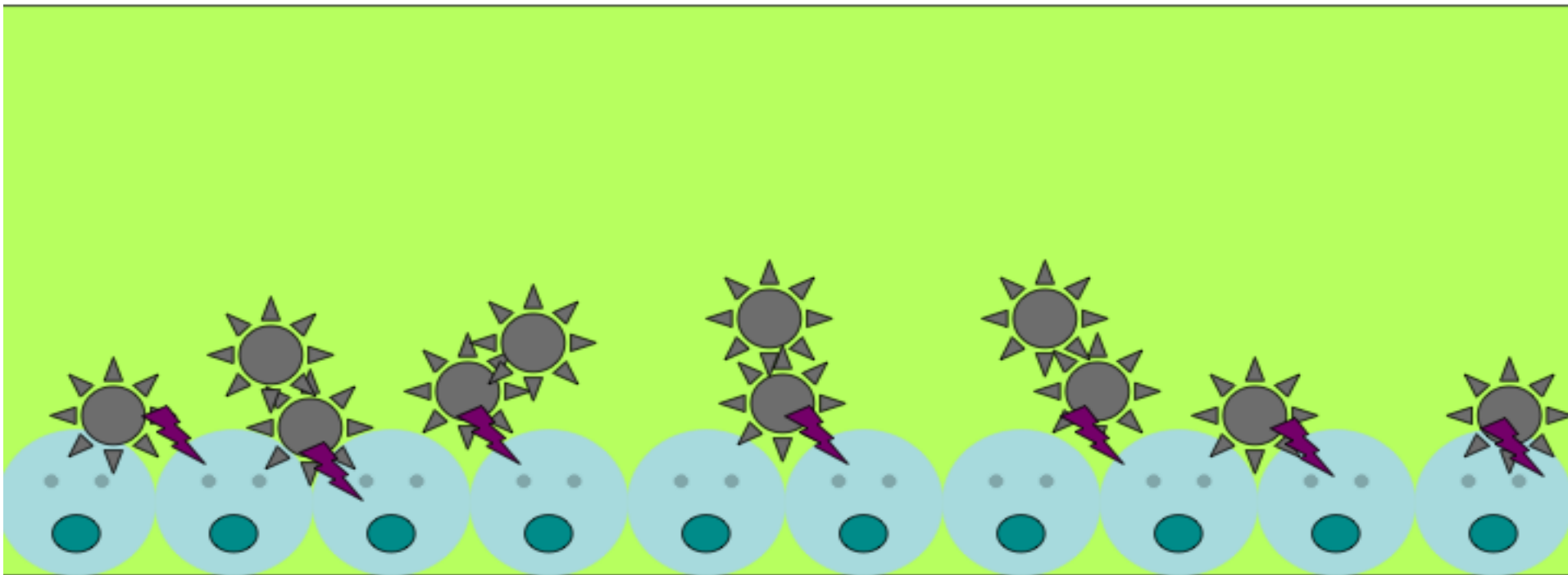


Life in the Fecal Stream



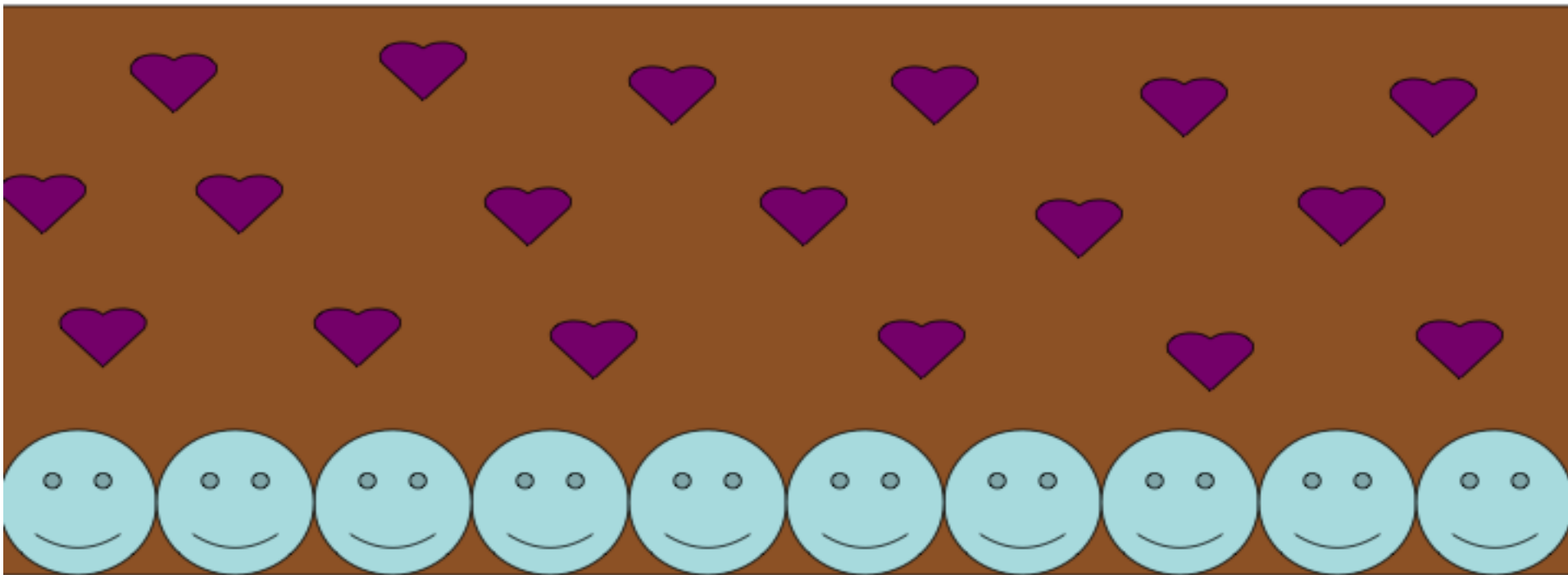
Happy colonocytes bathed in short chain fatty acids

Life without the Fecal Stream



Help, We're starving, please restore the fecal stream!

Restoration of the Fecal Stream (Life is good)



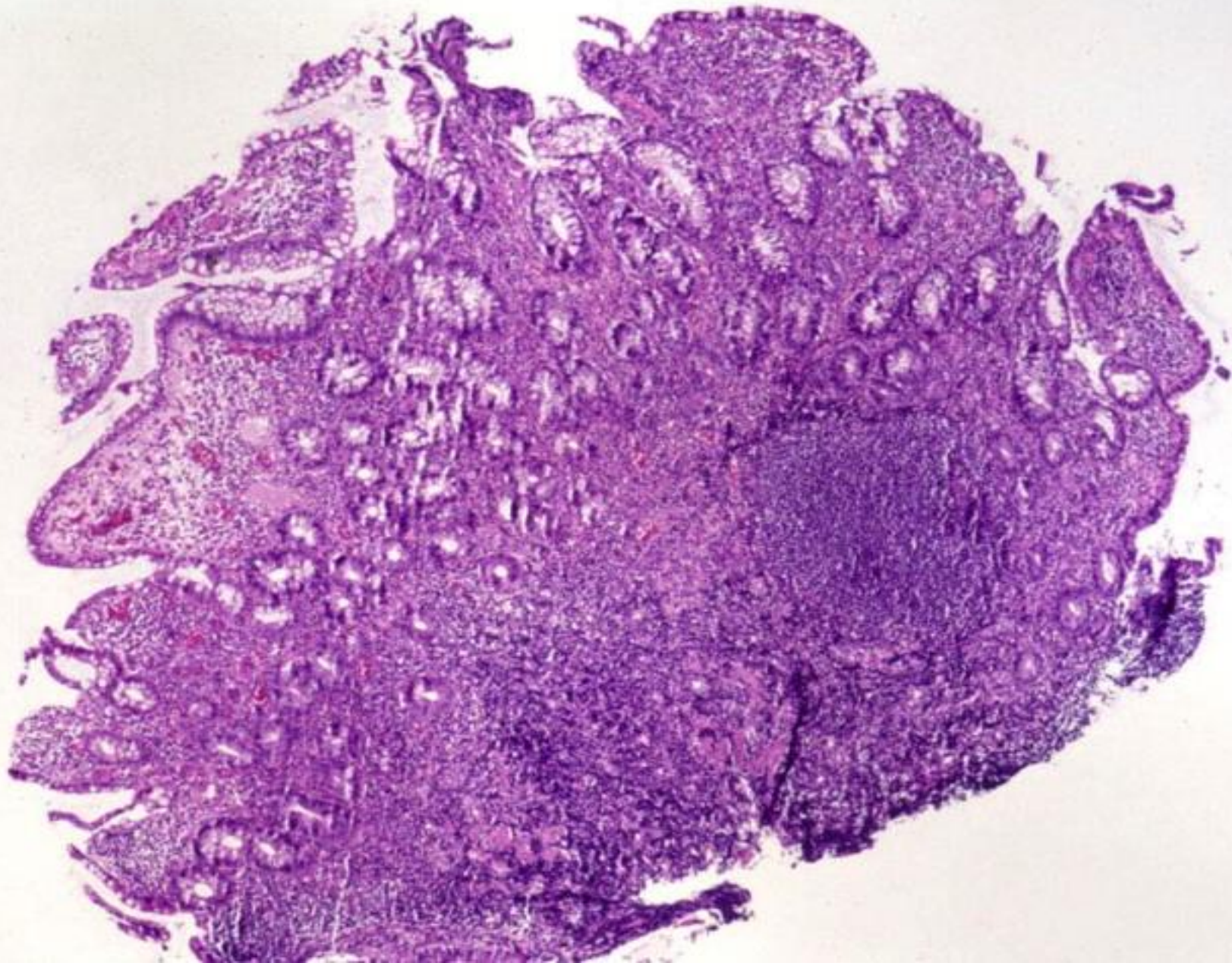
Colonocytes are happy again!

Diversion Colitis

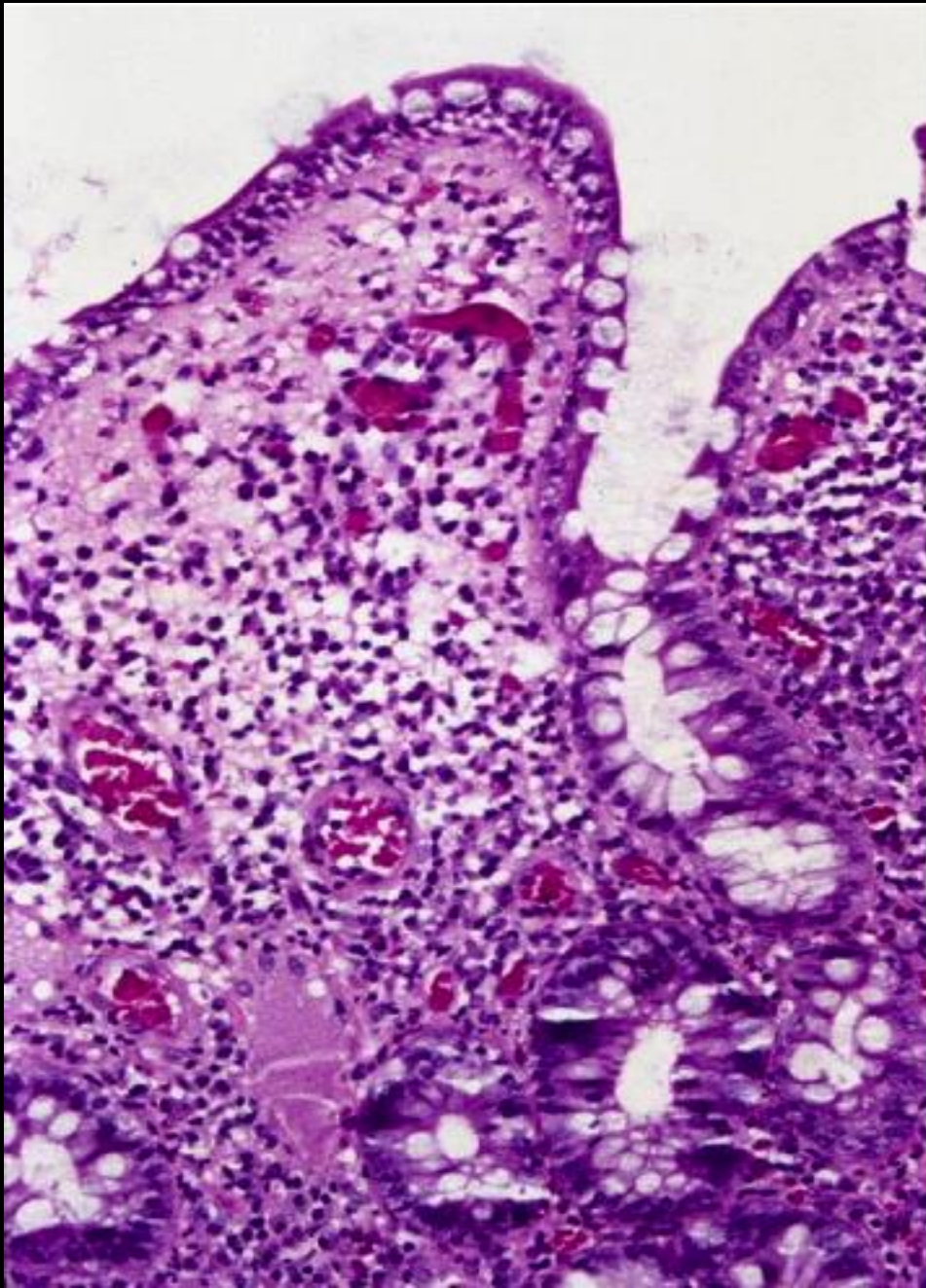
- **Enanthema, friability, ulcers, nodularity**
- **Diffuse nodular lymphoid hyperplasia**
- **Crypt atrophy/bending**
- **Increase lymph, plasma cells**
- **Neutrophils (mild, focal)**
- **+/- granulomas**

Case 4

54 YEAR-OLD JEWISH PHYSICIAN UNDERWENT SCREENING COLONOSCOPY AND WAS FOUND TO HAVE SEVERAL ULCERS IN THE TERMINAL ILEUM. CROHN'S DISEASE HAD PREVIOUSLY BEEN DIAGNOSED IN A COUSIN.







Drug-Induced Damage

Small Intestine

➤ Duodenum

➤ Same meds that affect stomach but lesser incidence (NSAIDs)

➤ Ulcers

➤ Ileum

➤ NSAIDs, KCl

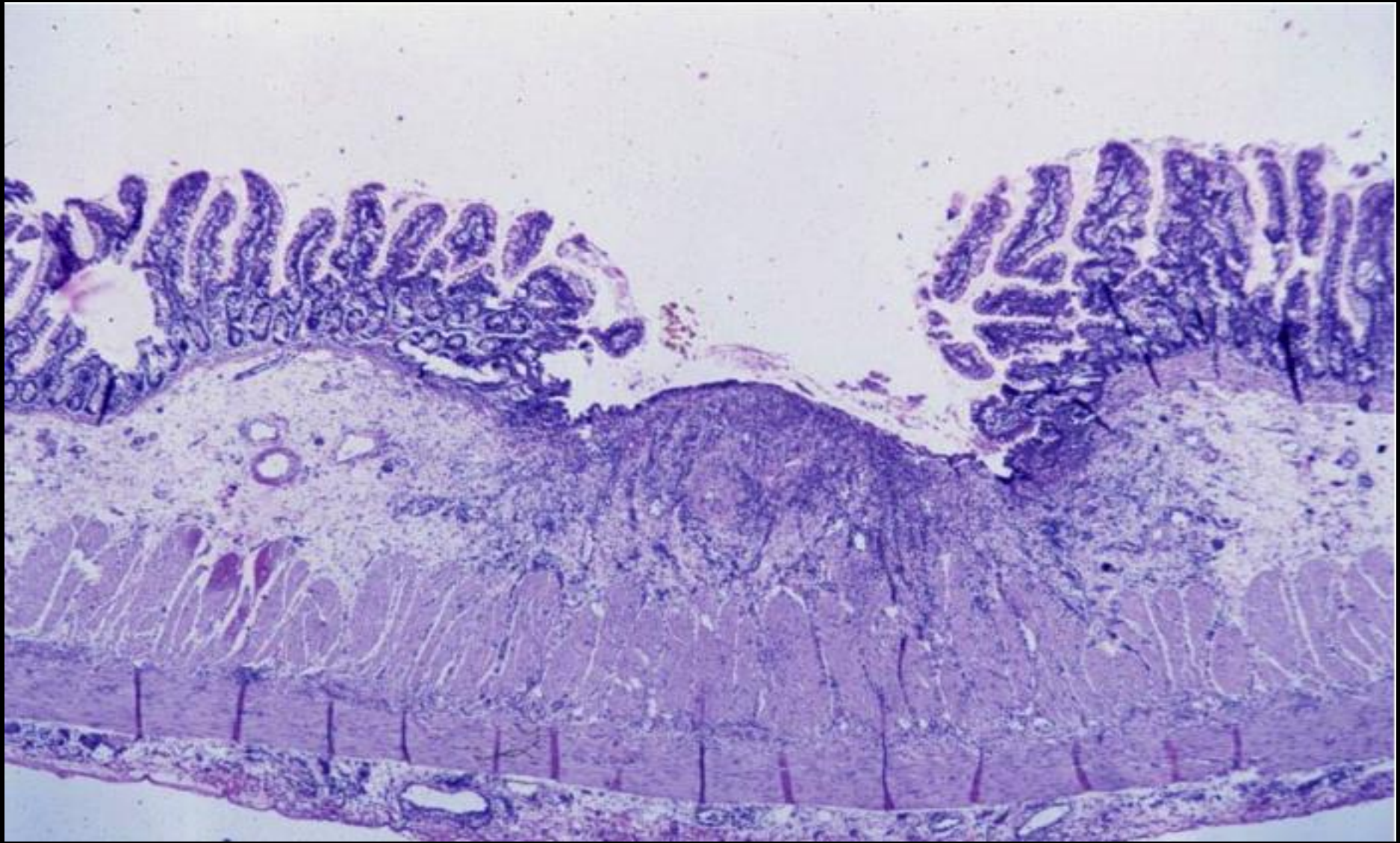
➤ Ulcers, Strictures, Diaphragm disease

NSAID Damage

Small Intestine

- **Ulcers – aphthoid, bland**
- **Strictures**
- **Diaphragm disease**
 - **Multiple, concentric luminal protrusions of fibrotic mucosa & submucosa**
 - **Distal ileum >> prox colon > jejunum**
 - **Slow release NSAIDs, piroxicam**
- **NSAID enteropathy**

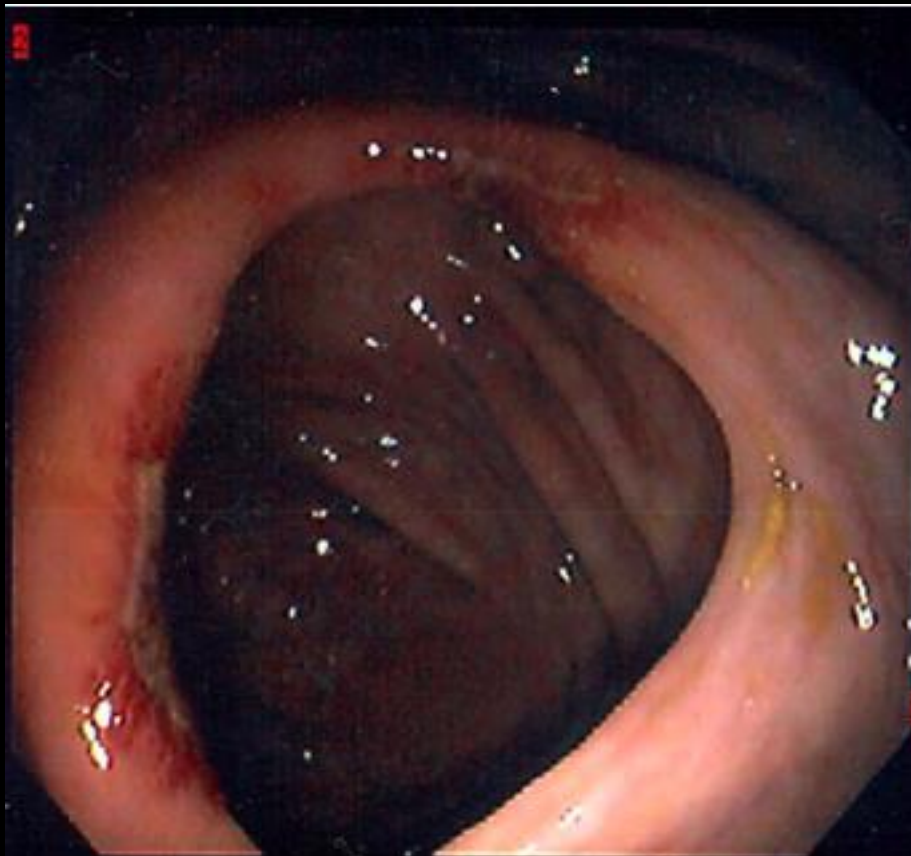




NSAID Damage


Ulcers in Small Bowel

- **Endoscopic study of TI (Midwest)**
 - Ulcers in 2% ileoscopies, screening
 - 84% Conventional NSAIDs, 16% COX2/other
- **Autopsy study (Scotland)**
 - Ulcers in 8.4% NSAID users, 0.6% non-users
 - Ulcers in 13.5% long term users
 - Ileum > jejunum
- **High end users (RA) by push enteroscopy**
 - Jejunal or ileal ulcers in 47%

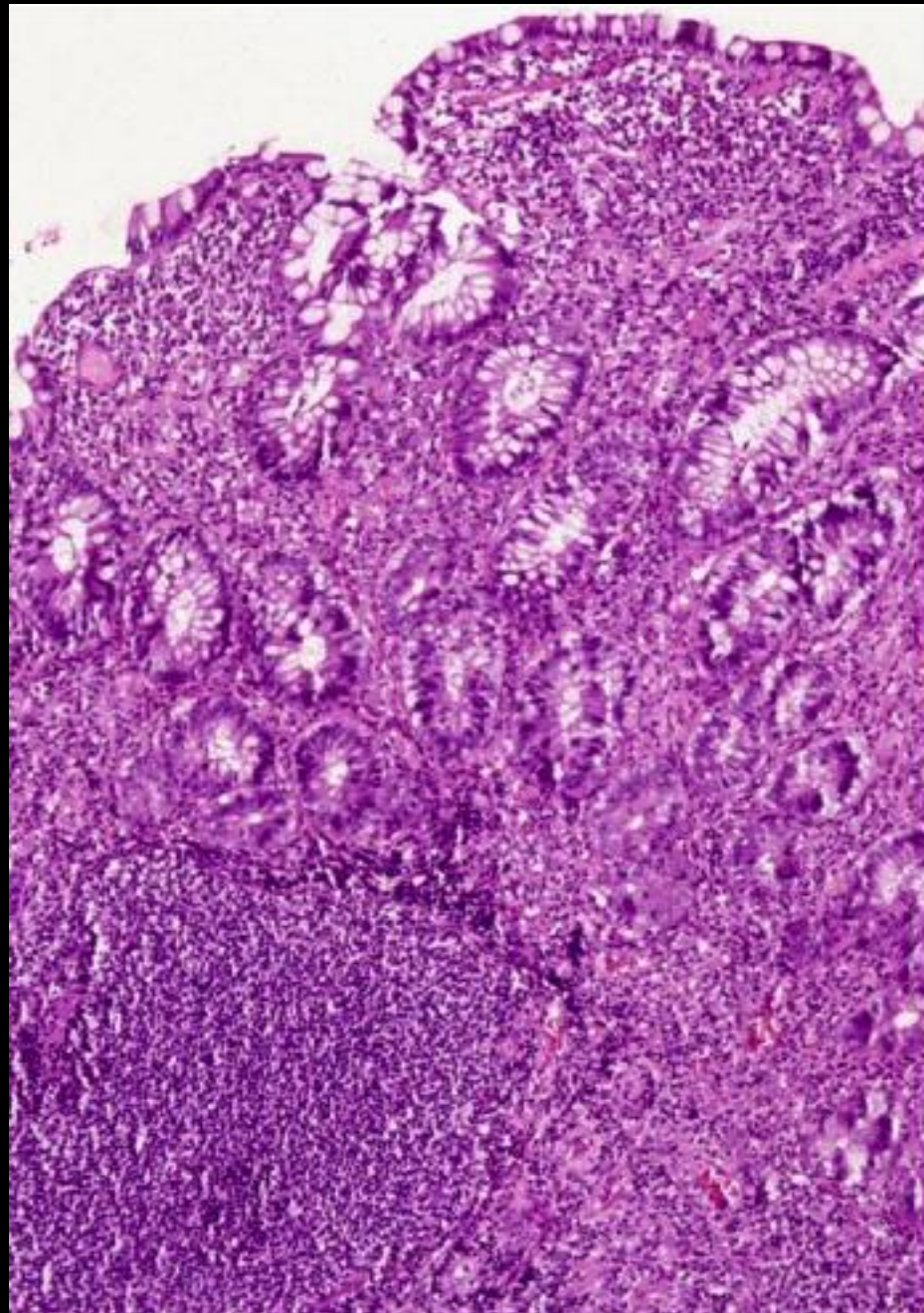


Morrison, Clifton
ID: 418722473
12/18/2002 COL



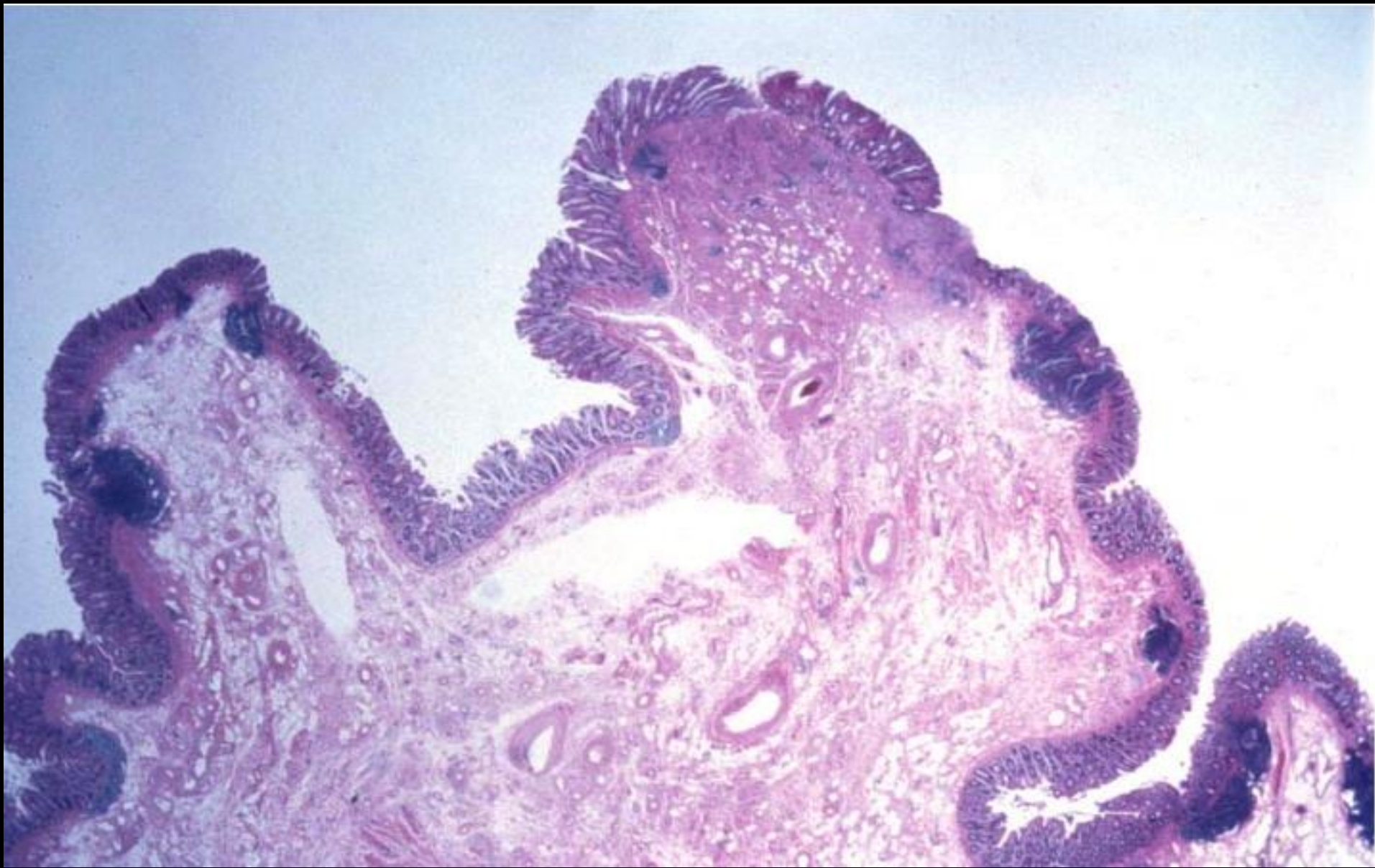
A large yellow inverted triangle pointing downwards, centered on the page. Inside the triangle, the text is written in a bold, black, serif font.

Caution
Ulcers in TI
not always
Crohn's





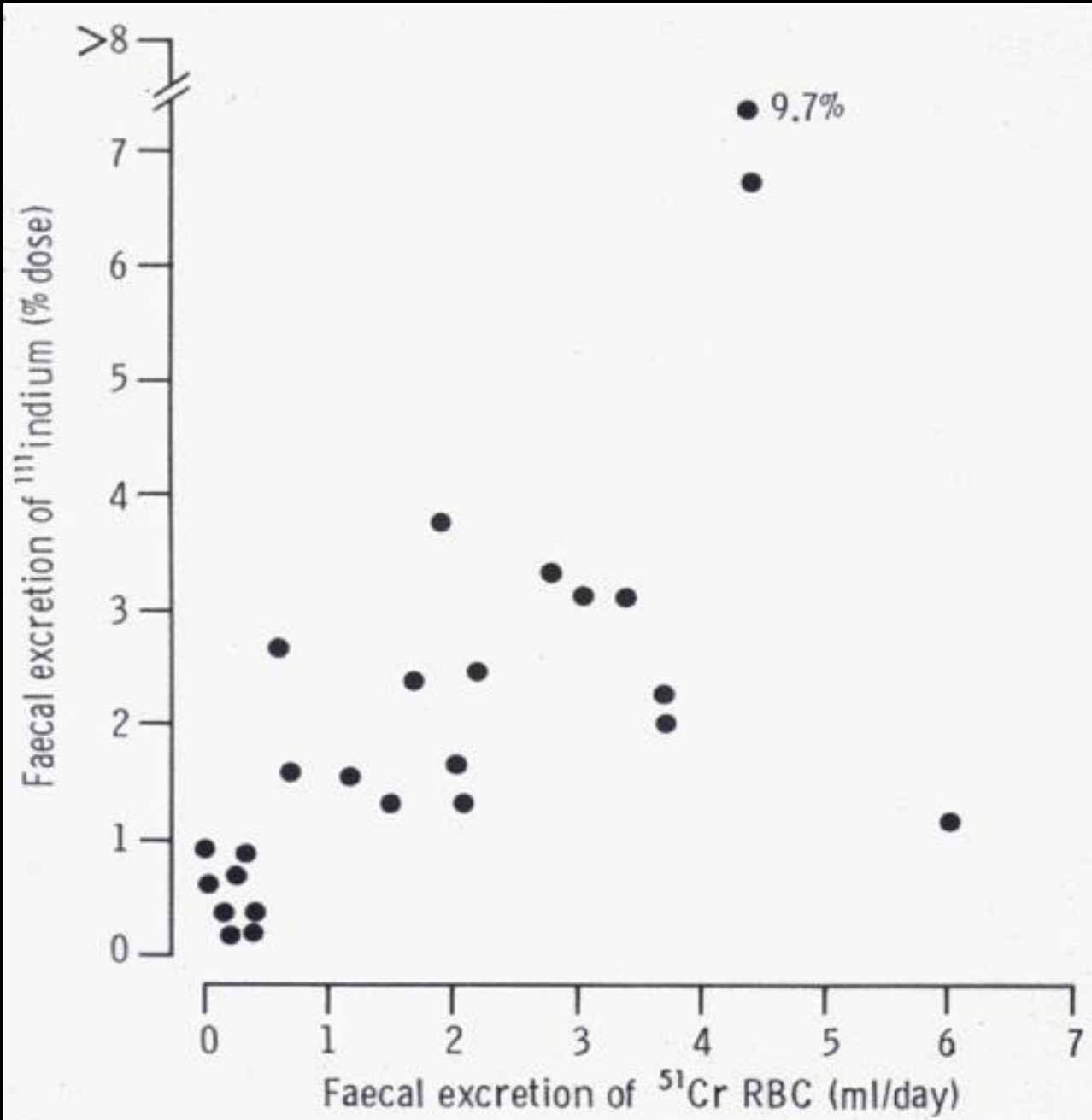




NSAID Enteropathy

- Sensitive detection studies using radiolabelled PMN's, RBC's, other molecules, scintigraphy
- Changes in permeability
- Accumulation & fecal loss PMN's
- Accumulation & fecal loss RBC's
- Changes in bile acid & B12 absorption (ileal)
- Mild protein loss



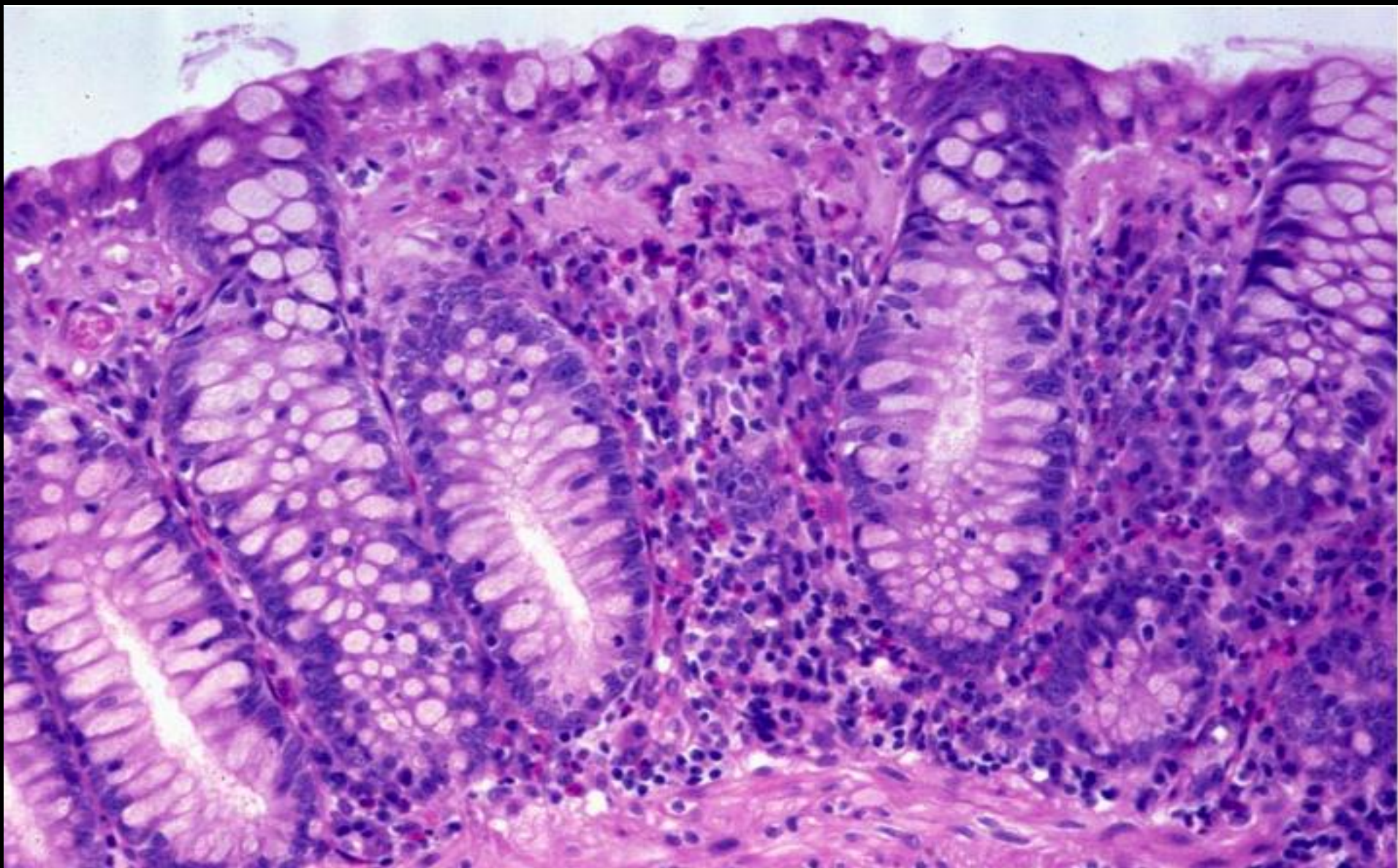


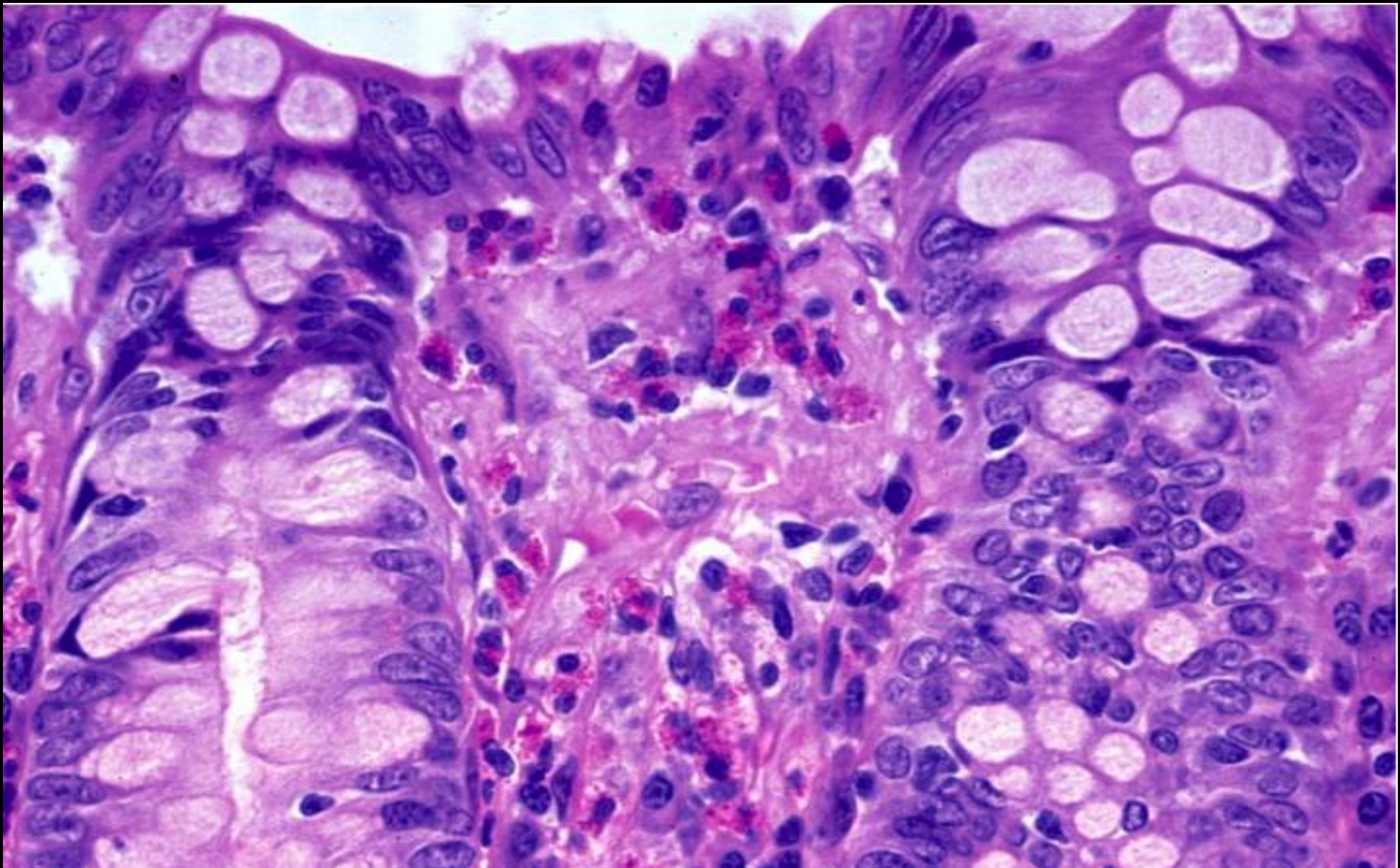
NSAID Enteropathy

- **Few pathologic studies**
 - **Mostly tagged cells & scans**
- **Bjarnason, 18 pts with most damage by radionuclide scans had barium studies**
 - **3 ulcers**
 - **2 strictures**
- **Enteropathy likely due to tiny erosions spread throughout length of small bowel**

Case 5

47 YEAR-OLD WOMAN HAD AN EIGHT-MONTH HISTORY OF DIARRHEA, NAUSEA, VOMITING AND PERIPHERAL EOSINOPHILIA OF 11%. PATIENT UNDERWENT COLONOSCOPY AND MUCOSA WAS FOUND TO BE NORMAL.





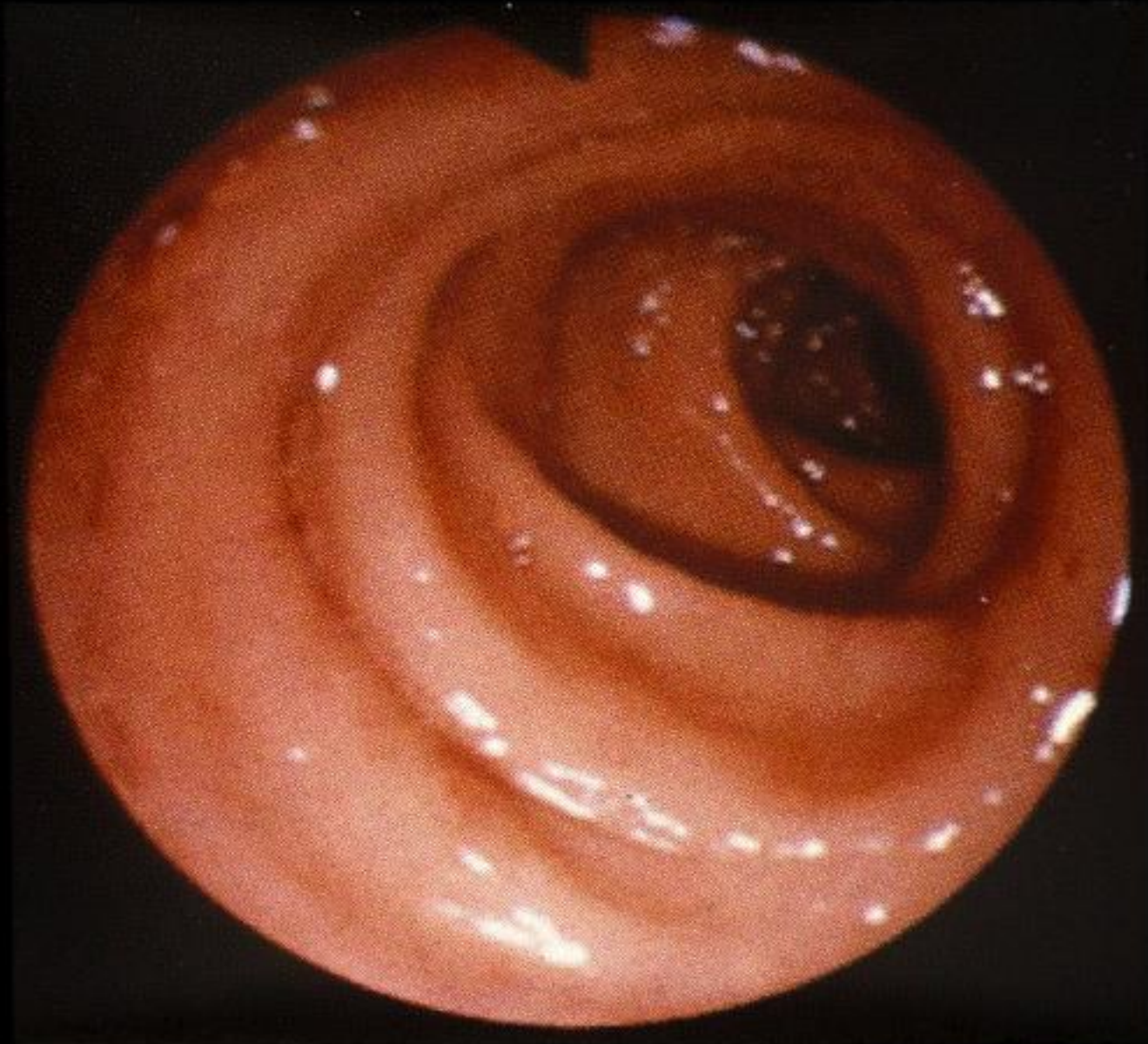
Collagenous Colitis

- **First described by Lindstrom in 1976**
- **Diagnosed primarily in Europe, North America and Australia (Western)**
- **Incidence 1.8 –5.2/100,000 general population, but 0.3 to 5% of chronic diarrhea patients**

Collagenous Colitis

Clinical Features

- **Chronic watery diarrhea. Mean 5.3 years duration (Range 0.5-18)**
- **Crampy abdominal pain**
- **Arthritis (7%), other autoimmune (17-40%)**
- **Middle-aged patients (mean 59 years)**
- **Female predominance**
- **Normal colonoscopy and barium enema**



Collagenous Colitis

Histopathology

- **Mucosal inflammatory process**
 - Increased intraepithelial lymphocytes
 - Surface epithelial damage
 - Increased plasma cells and eosin LP
 - Little crypt distortion or PMNs
- **Subepithelial collagen band**

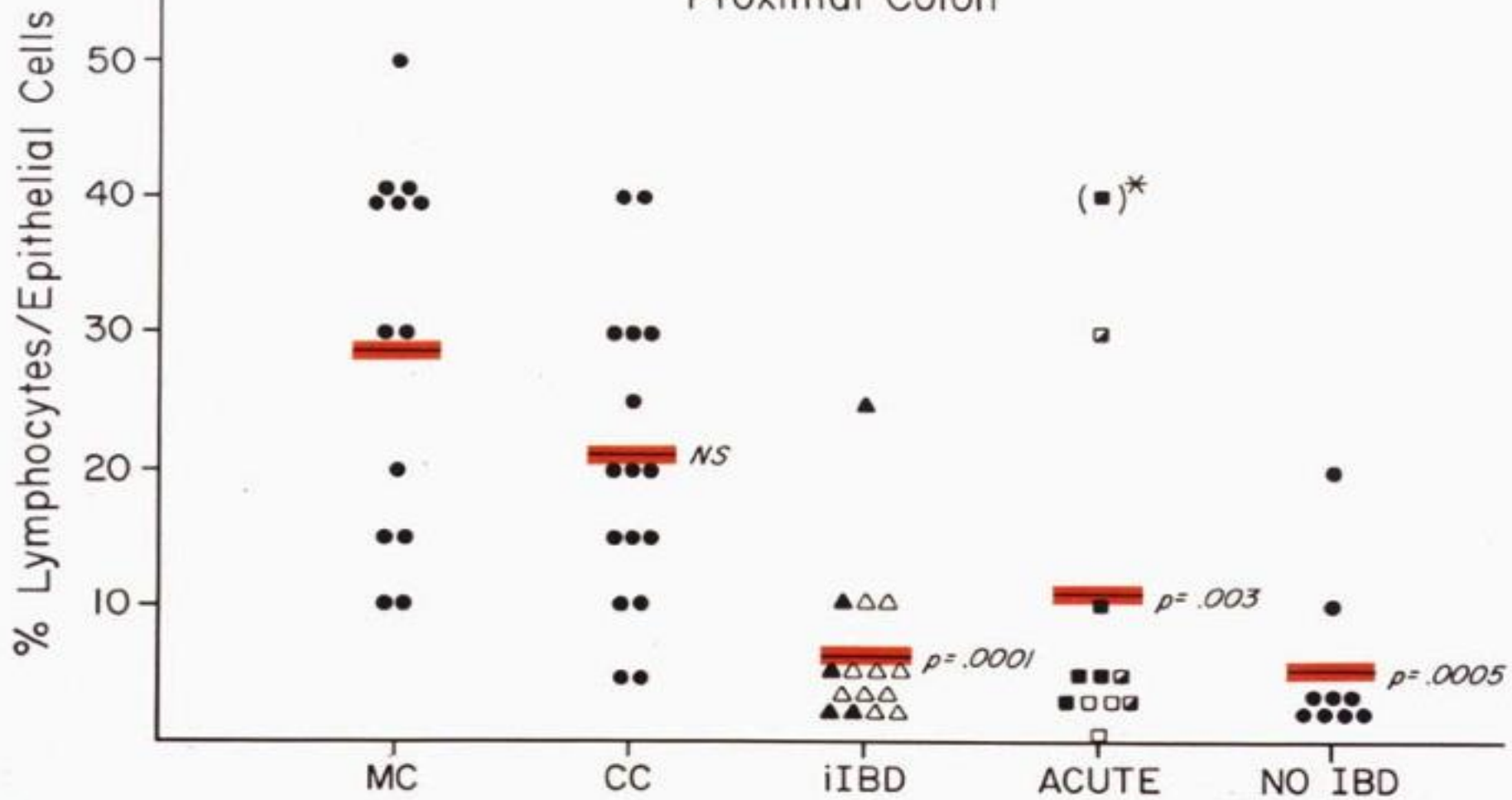
Collagenous Colitis

A Chronic Inflammatory Disorder

- Two words in the name
- Recognition of inflammation
 - Key to correct diagnosis
 - Pathogenesis
- Distinctive from other chronic colitis
 - CC -Increased intraepithelial lymphs
 - UC & Crohn's – Crypt distortion, PMNs

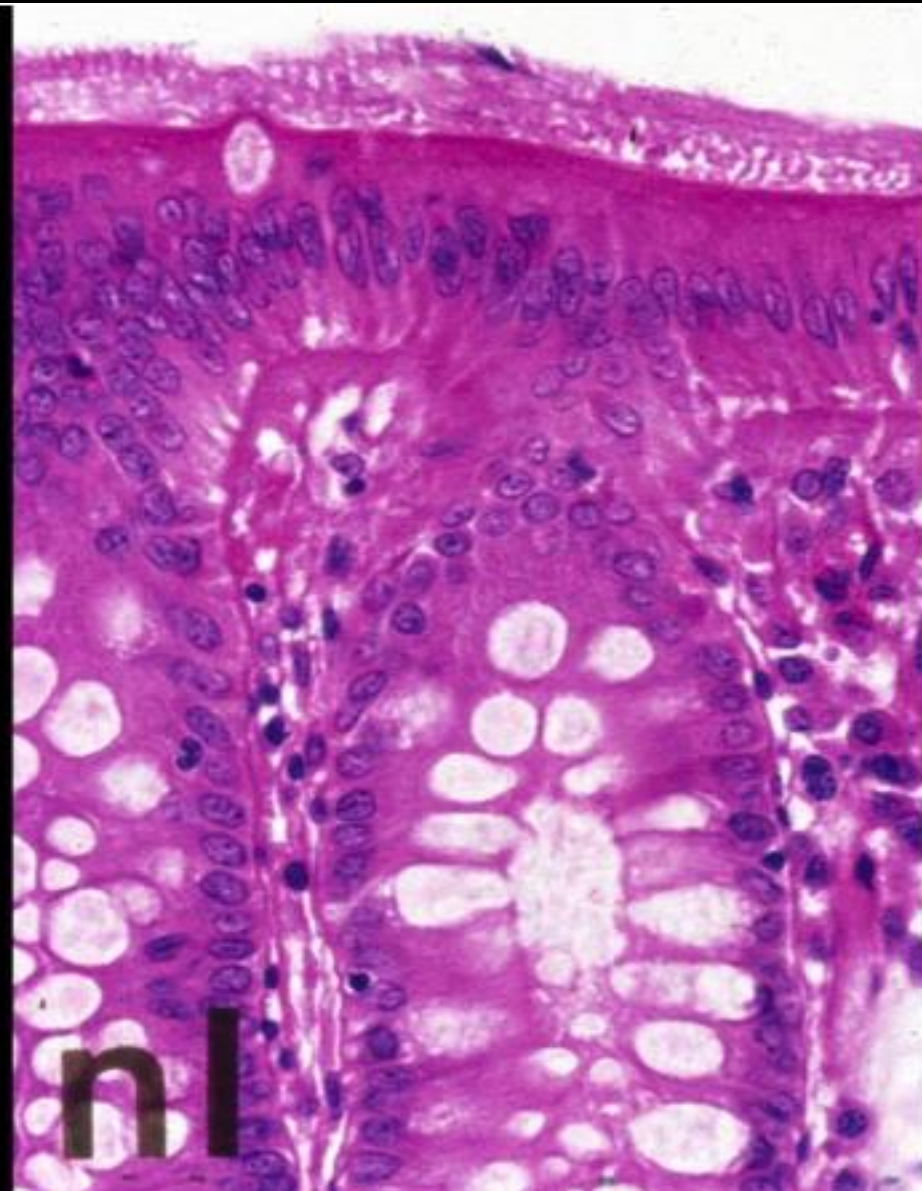
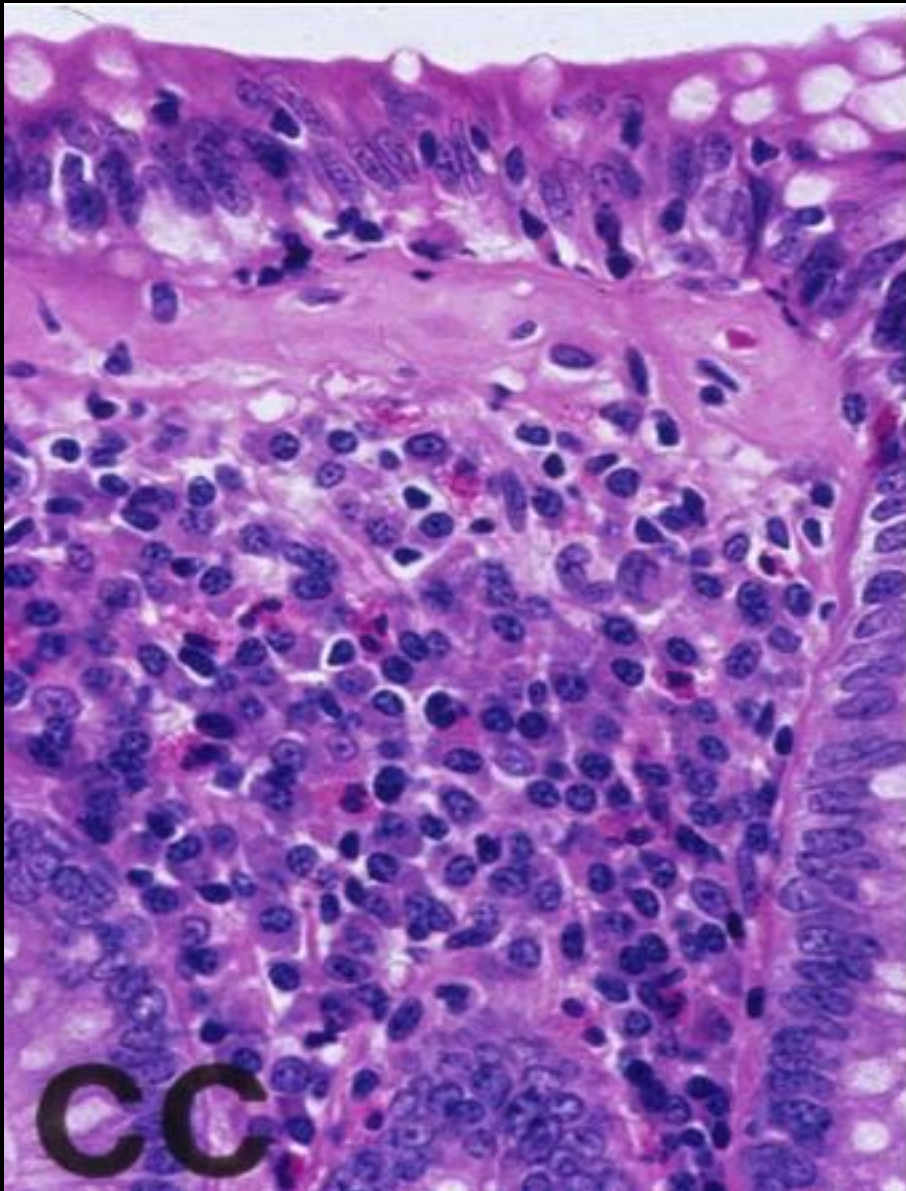


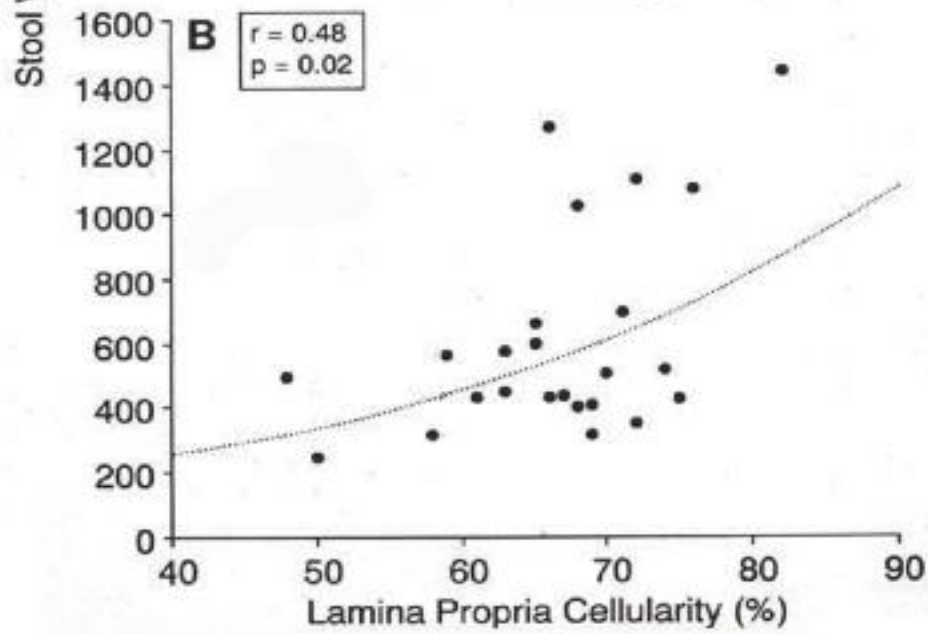
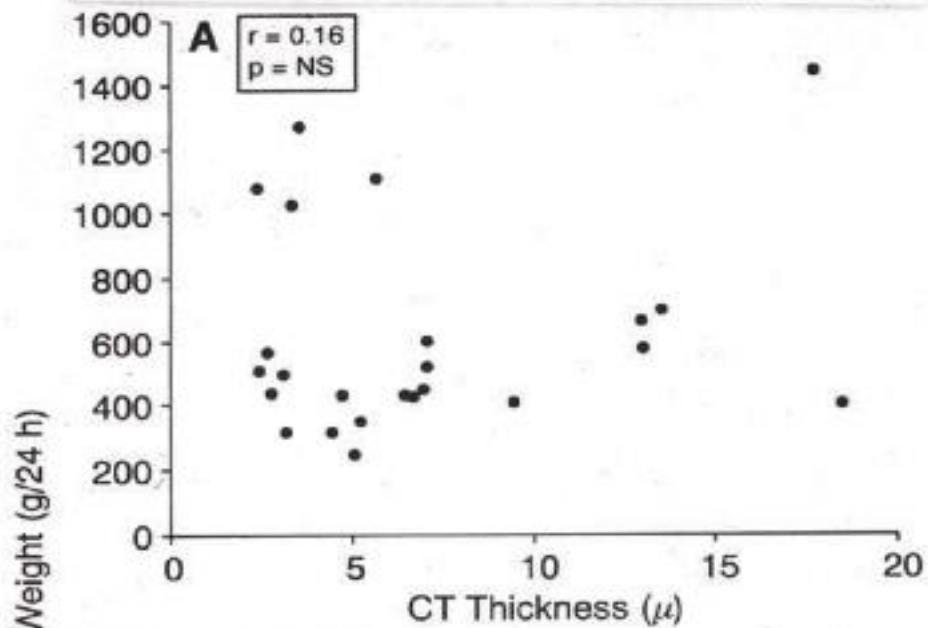
SURFACE LYMPHOCYTES Proximal Colon



*p values of means vs MC
(Wilcoxon Rank Sum)*

- ▲ Crohn's
- △ UC
- acute self limited
- ischemic
- ◻ antibiotic assoc.

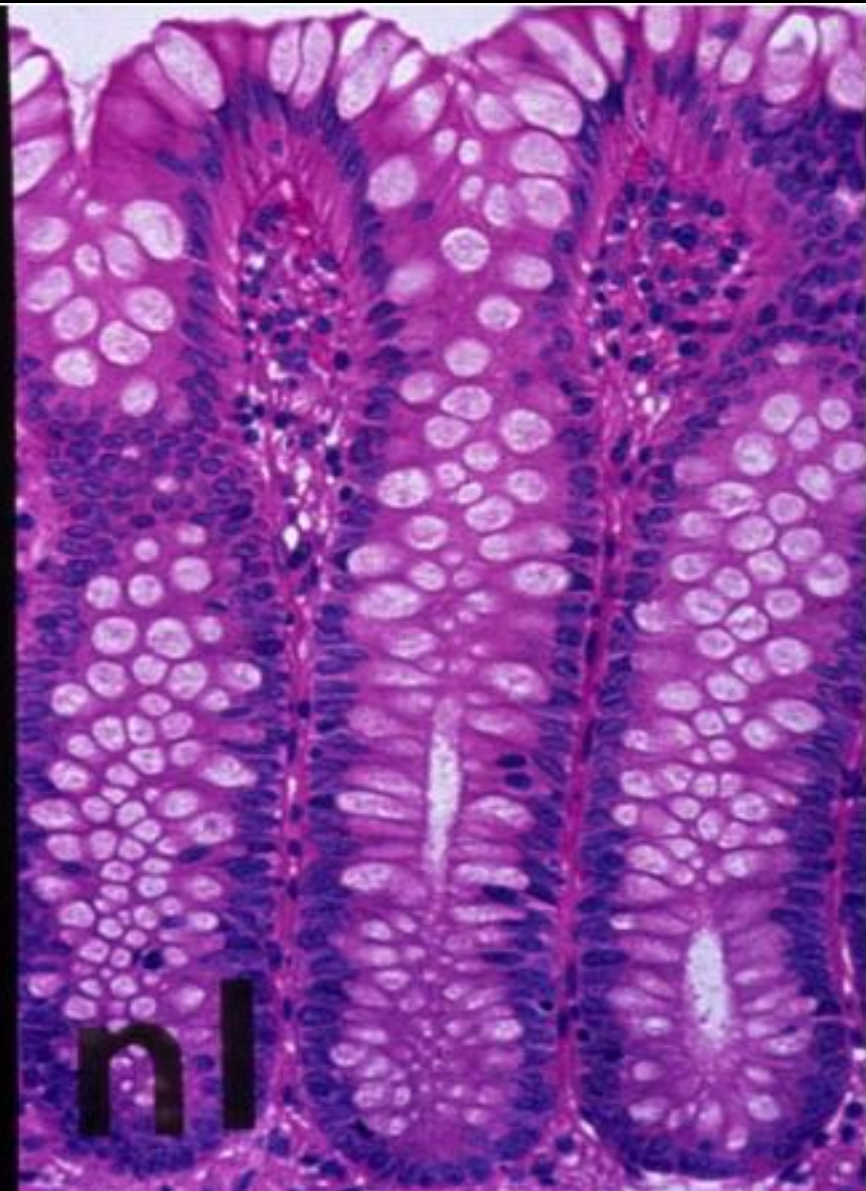
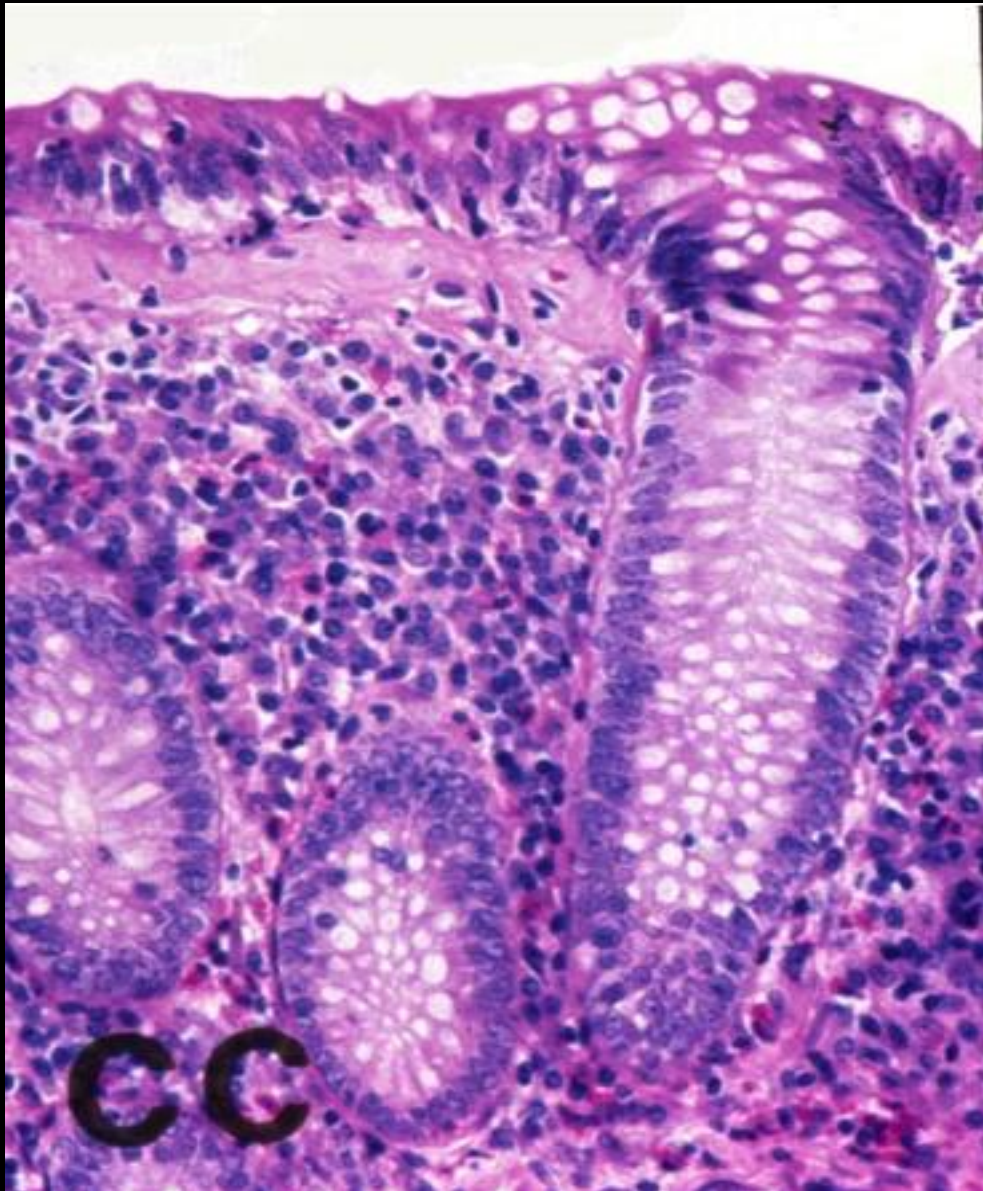


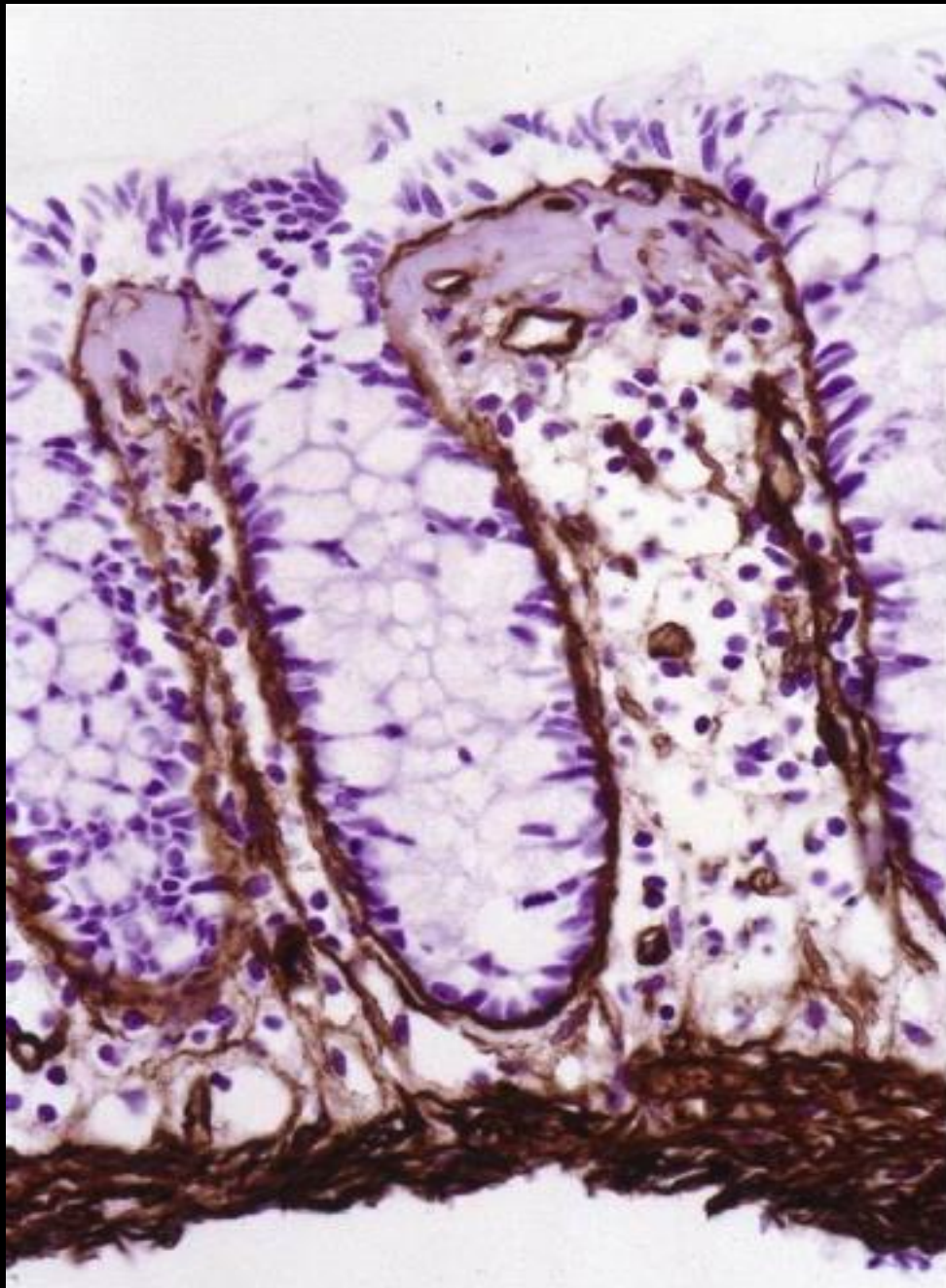


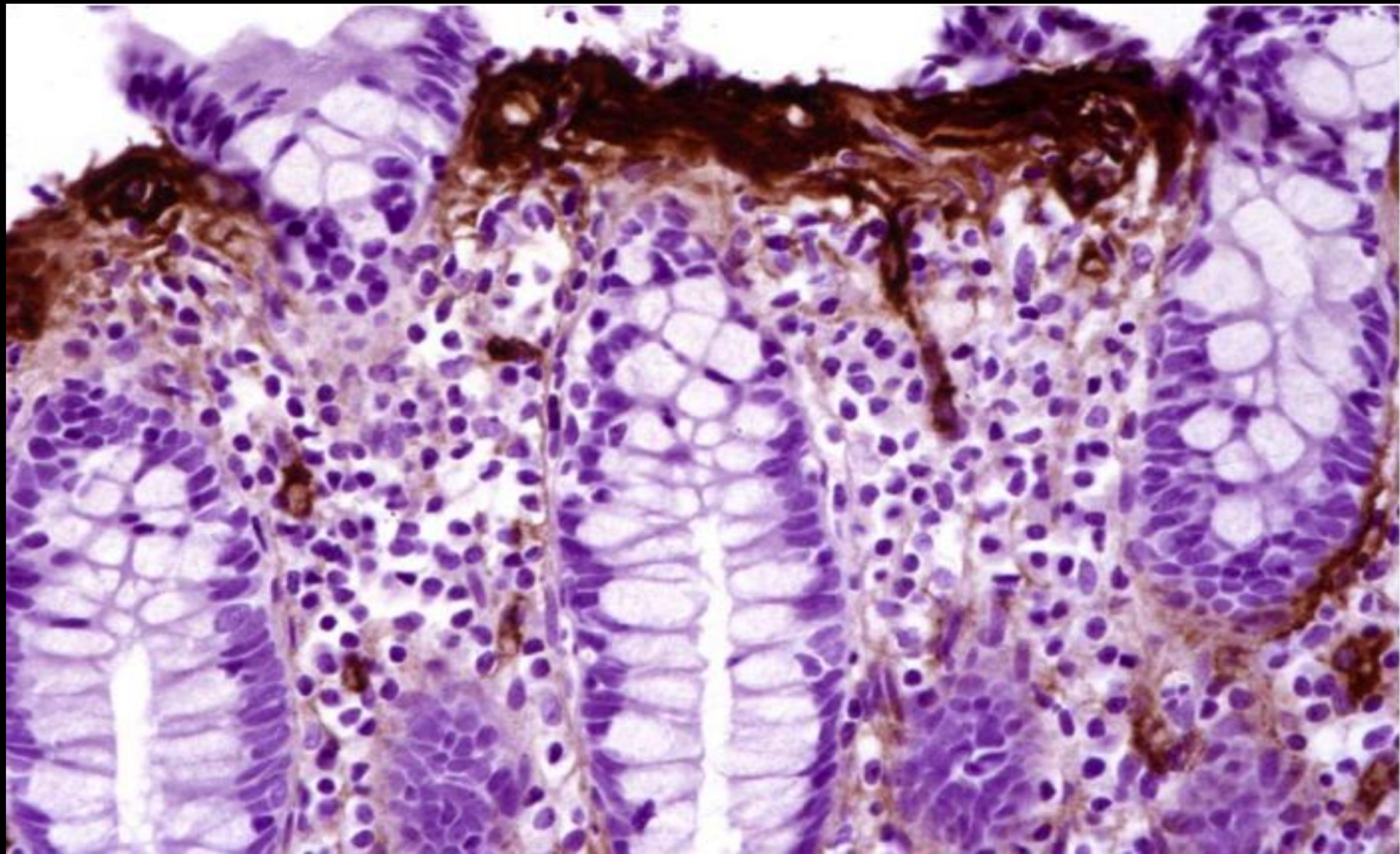
Collagenous Colitis

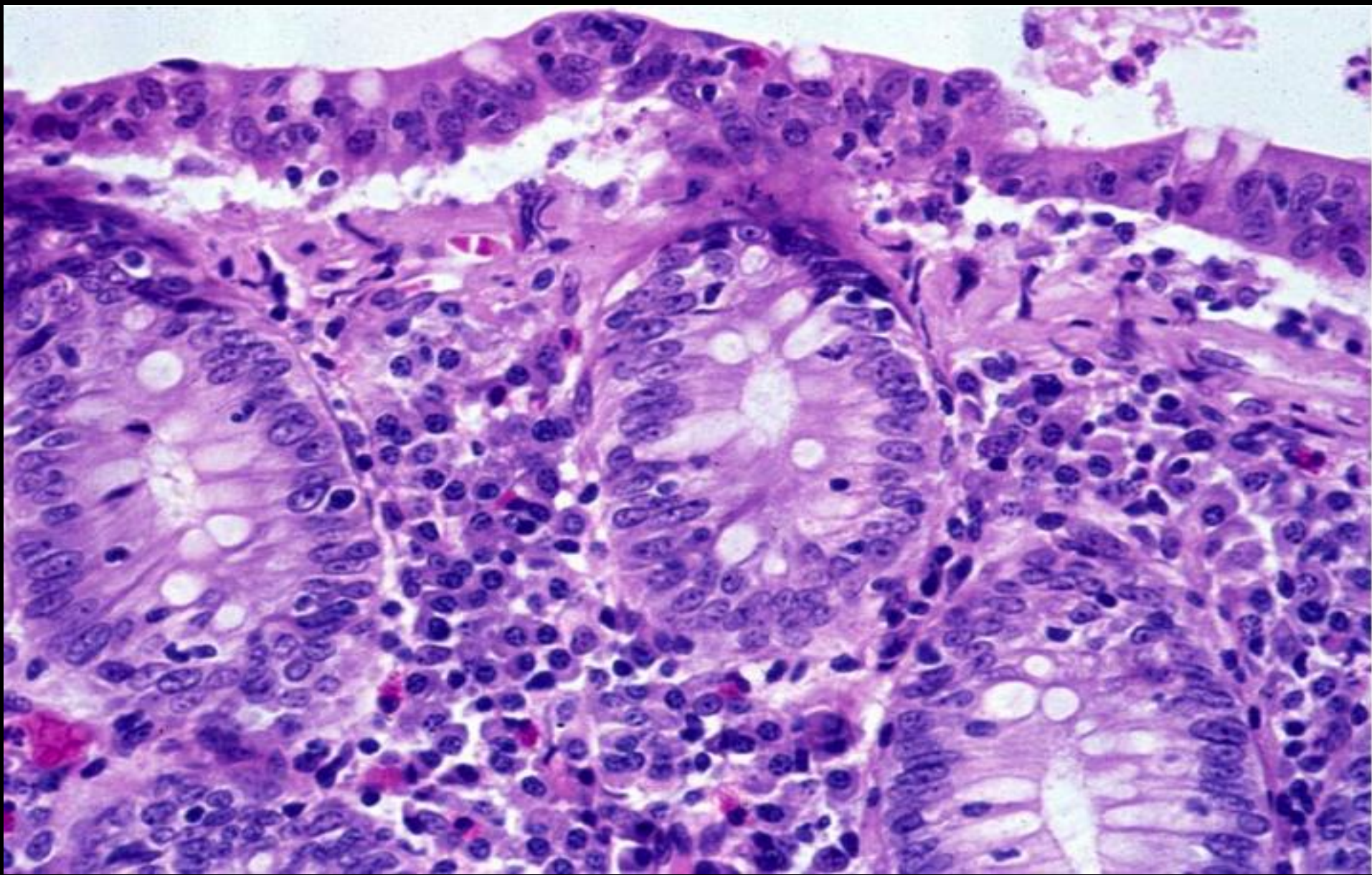
Subepithelial Collagen Band

- **Not a thick basement membrane**
 - **CIV, laminin negative**
- **Separate from and beneath BM**
 - **CIII, CVI, tenascin positive**





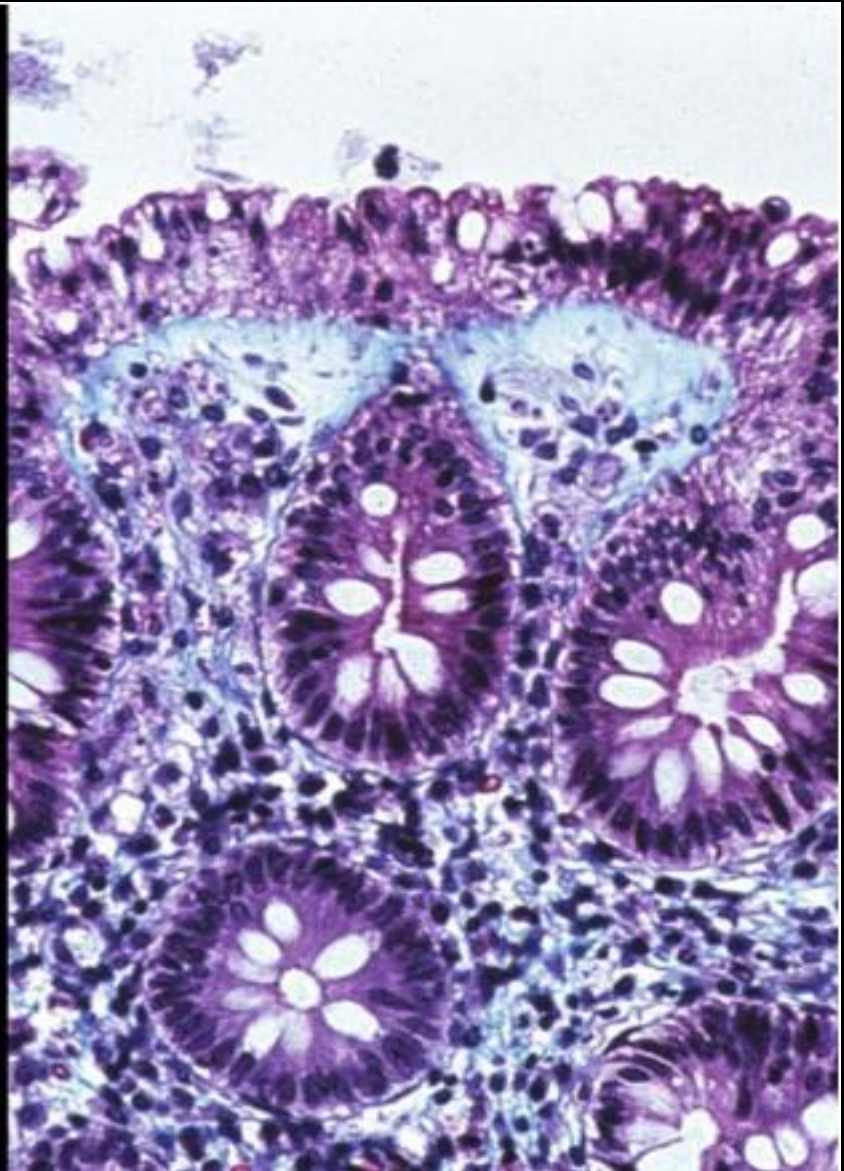


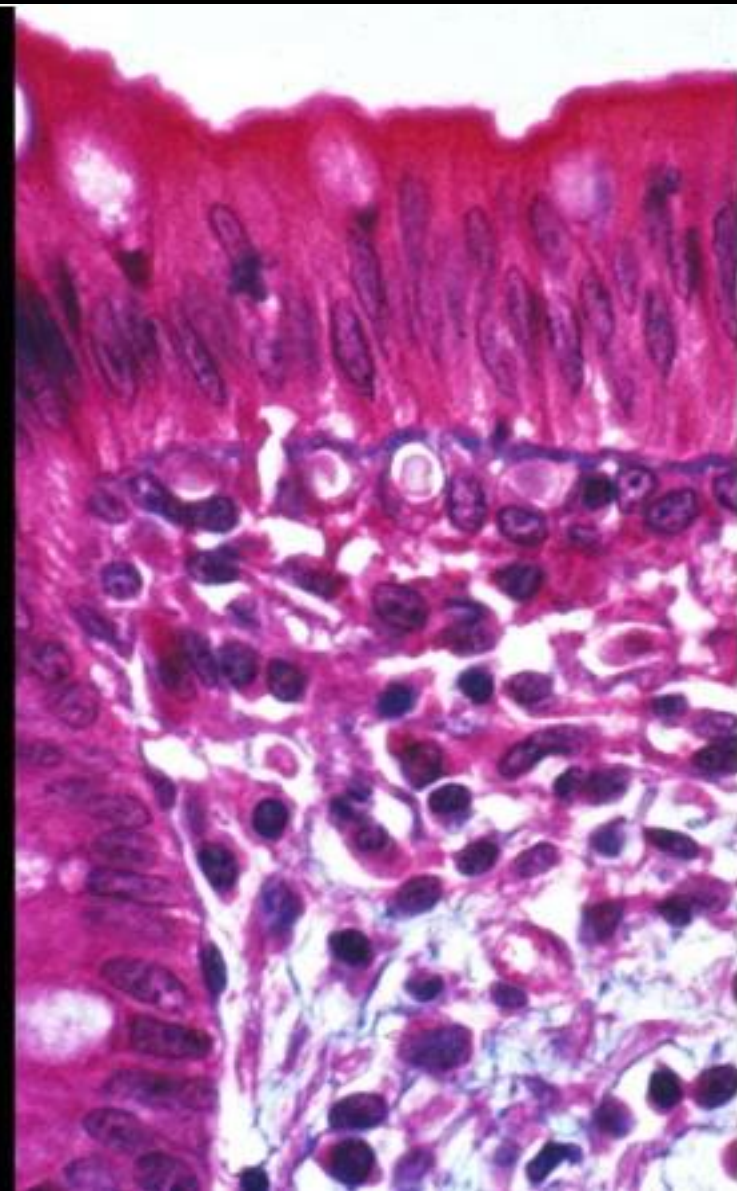
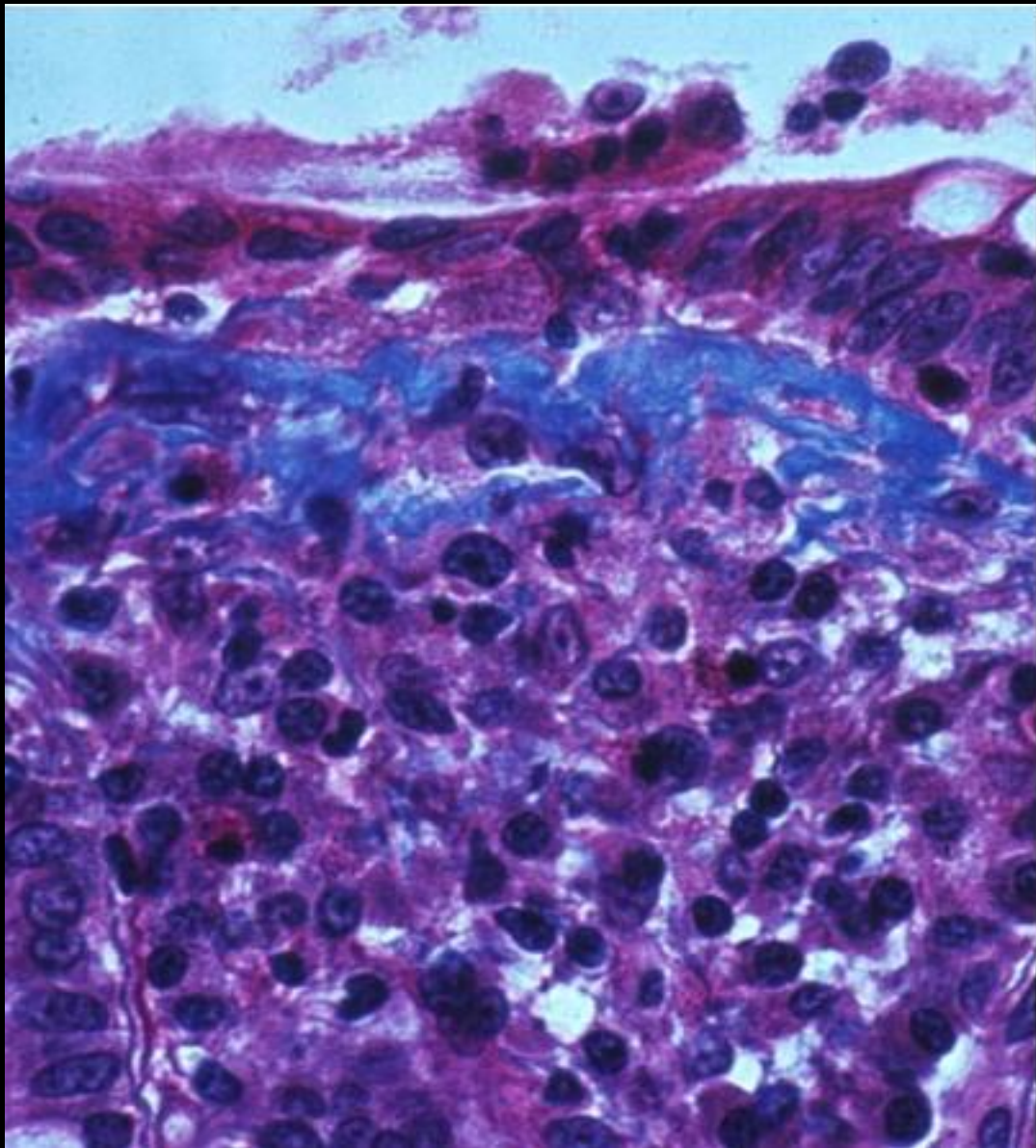


Collagenous Colitis

Subepithelial Collagen Band

- **Quantification of thickness**
 - Not necessary
 - Not adequate
 - Maybe misleading
- **Qualitative changes**
 - Tendrils extend into LP imparting “messy” edge to base -BM
 - Entraps superficial capillaries
 - Any increase in SCL in proper inflammatory context = CC

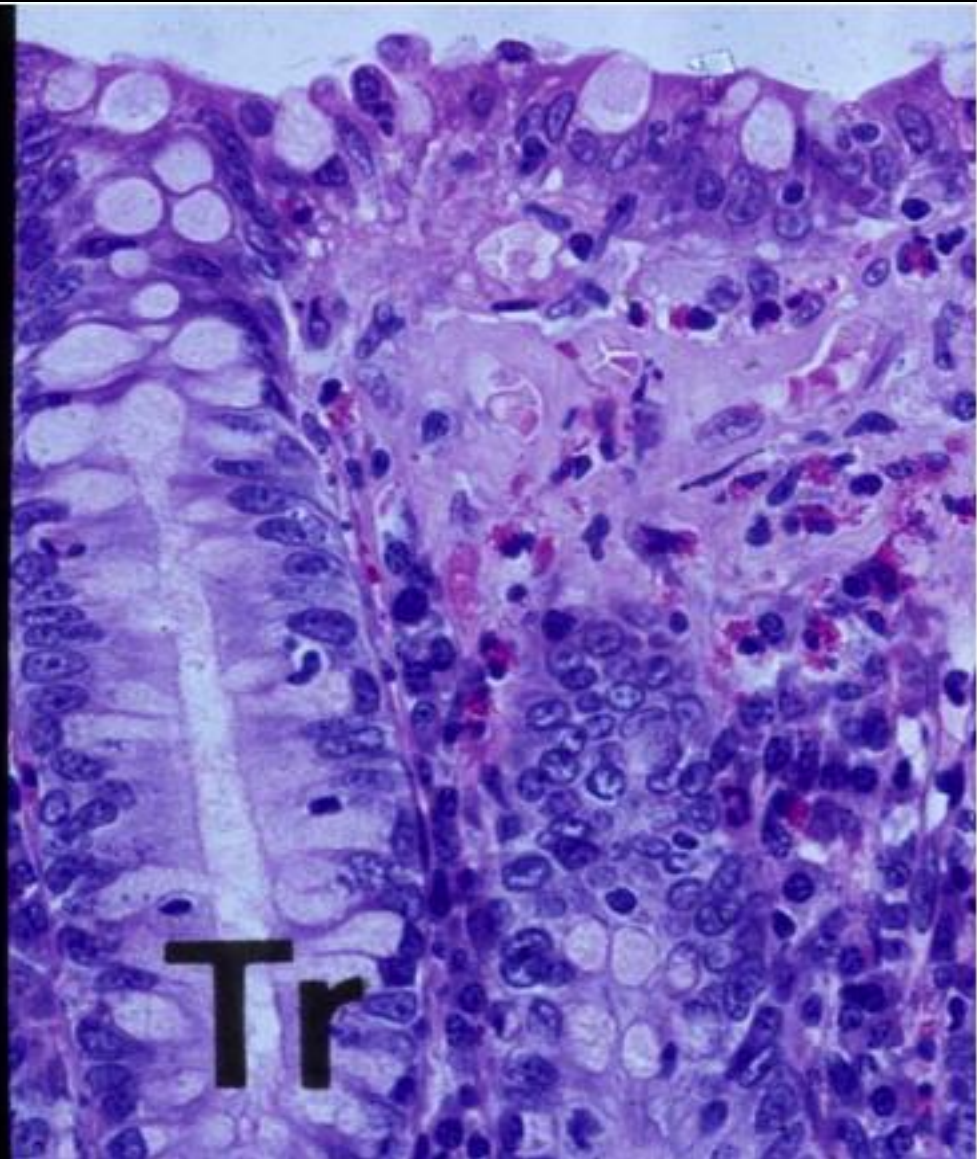
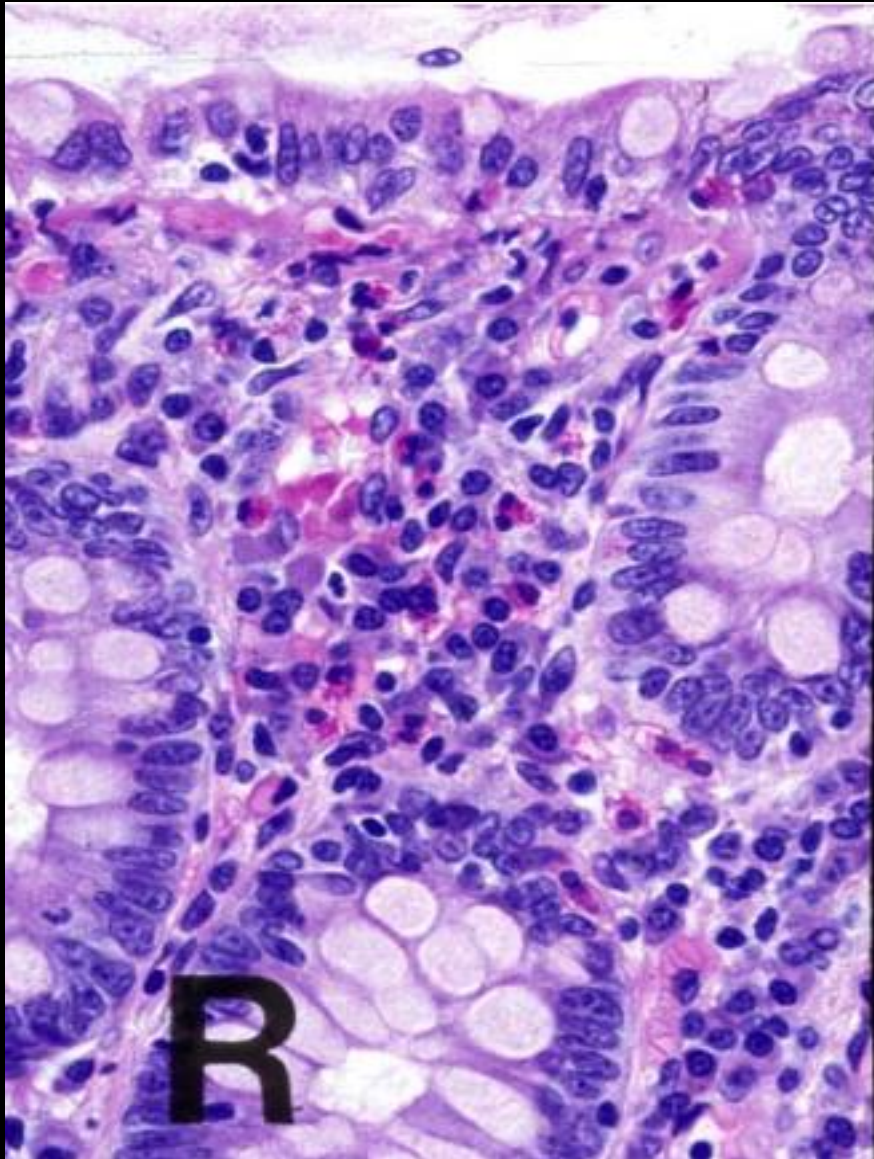


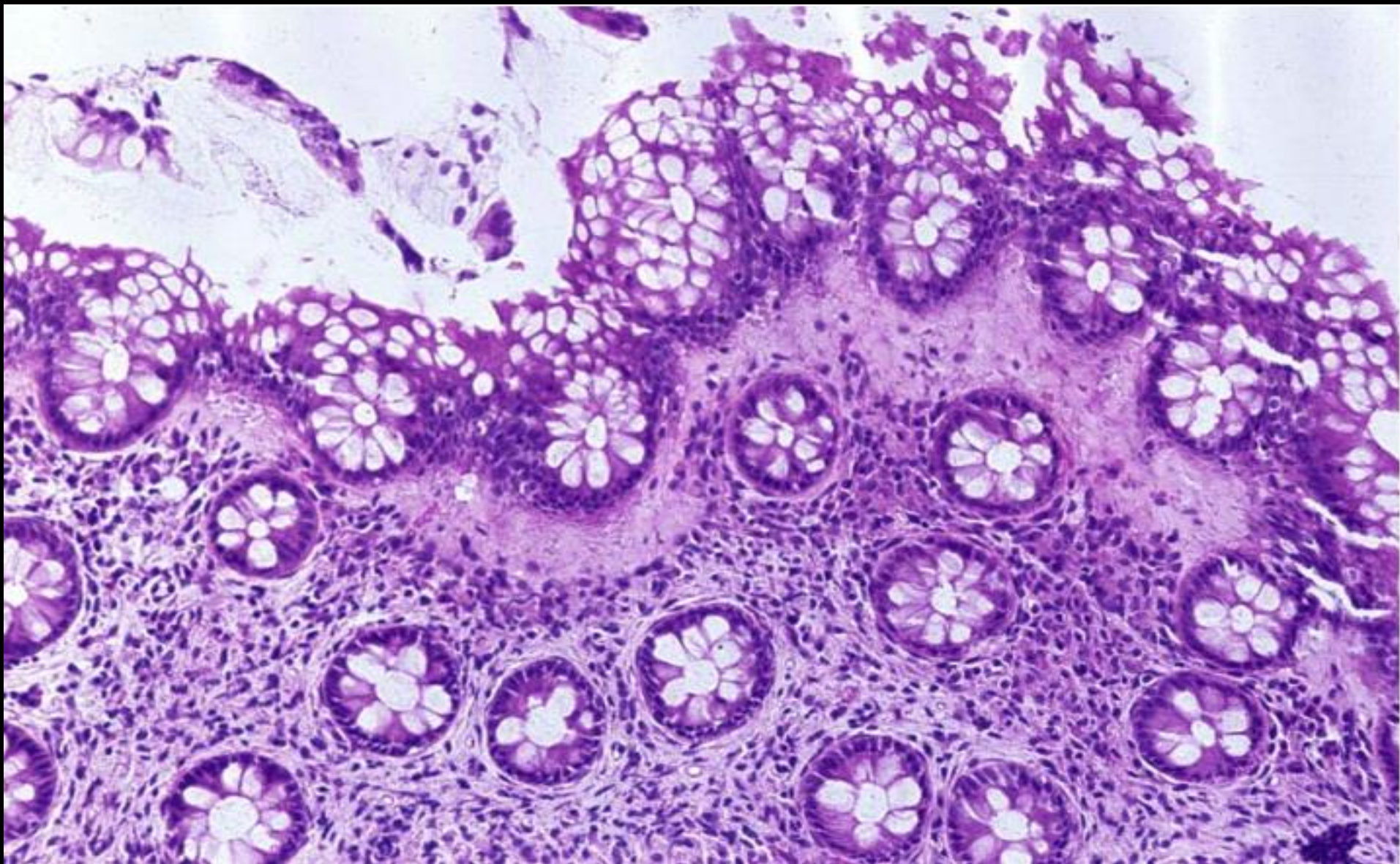


Collagenous Colitis

Pitfalls in Diagnosis

- Rectum can be spared
- Subepithelial collagen can be patchy
 - Need multiple biopsies
- Do not focus exclusively on collagen band, inflammation necessary
- Tangential sections





Collagenous Colitis

Pathogenesis

- Type of chronic idiopathic IBD
- ? Infectious agent or luminal toxin
 - Remission on diverting fecal stream - (Swedish study of 9 patients, with rechallenge)
 - Improvement on bismuth
 - ↑ IELS suggests polarization to luminal agent
 - Luminal agent → cross reactivity to epith Ag
- ? Drug – NSAIDs, lansoprazole
- ? Autoimmune (17–40% other autoimmune)

Collagenous Colitis

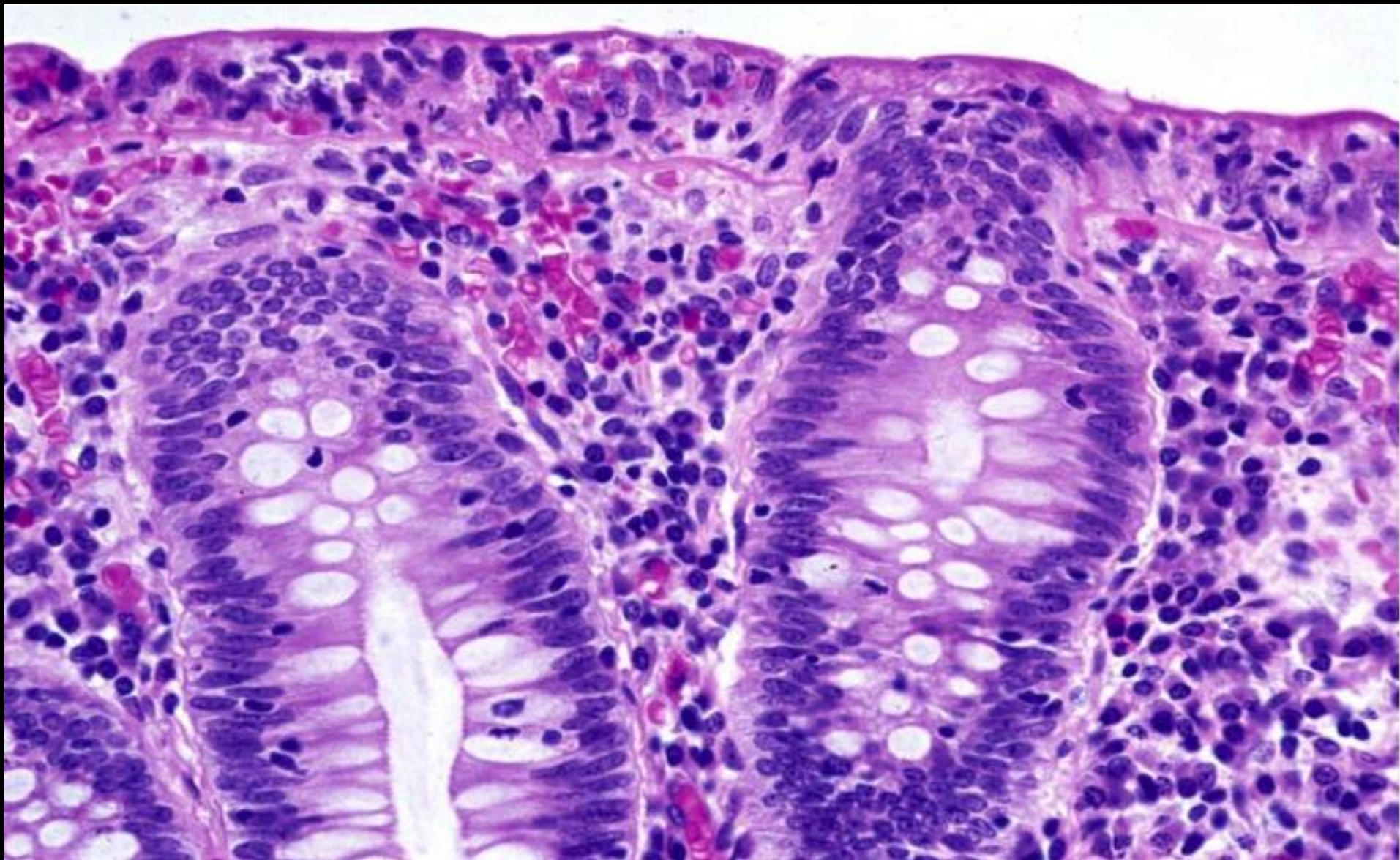
Differential Diagnosis

➤ Lymphocytic colitis

- Similar in ↑IELS, increased CI, little crypt distortion or neutrophils
- Lacks SCL, eosinophils

➤ Ulcerative colitis & Crohn's disease

- Similar increased CI in lamina propria
- Differs in prominent crypt distortion & epithelial neutrophils



Carpenter et al.

Dig Dis Sci 37: 1903-9, 1992

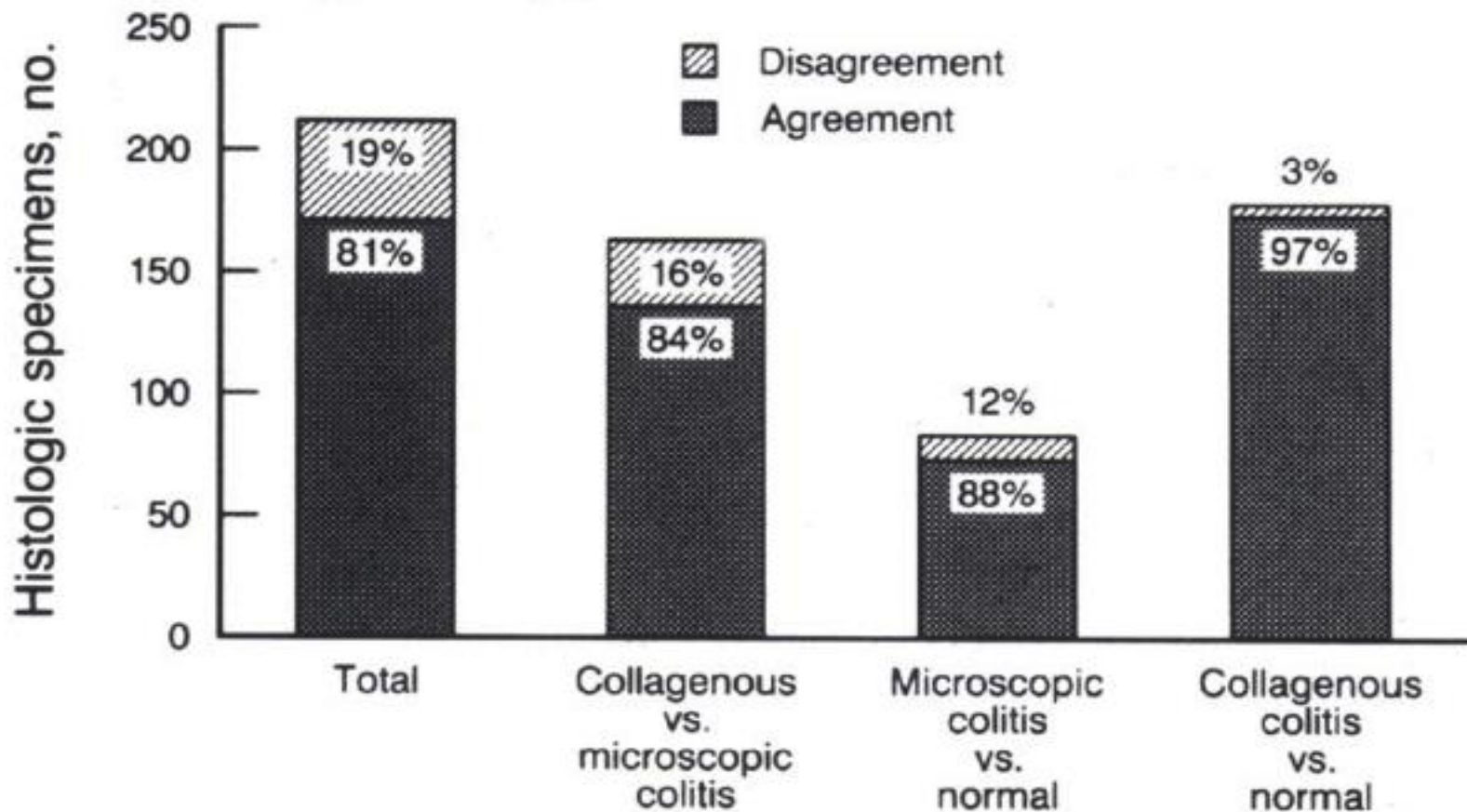
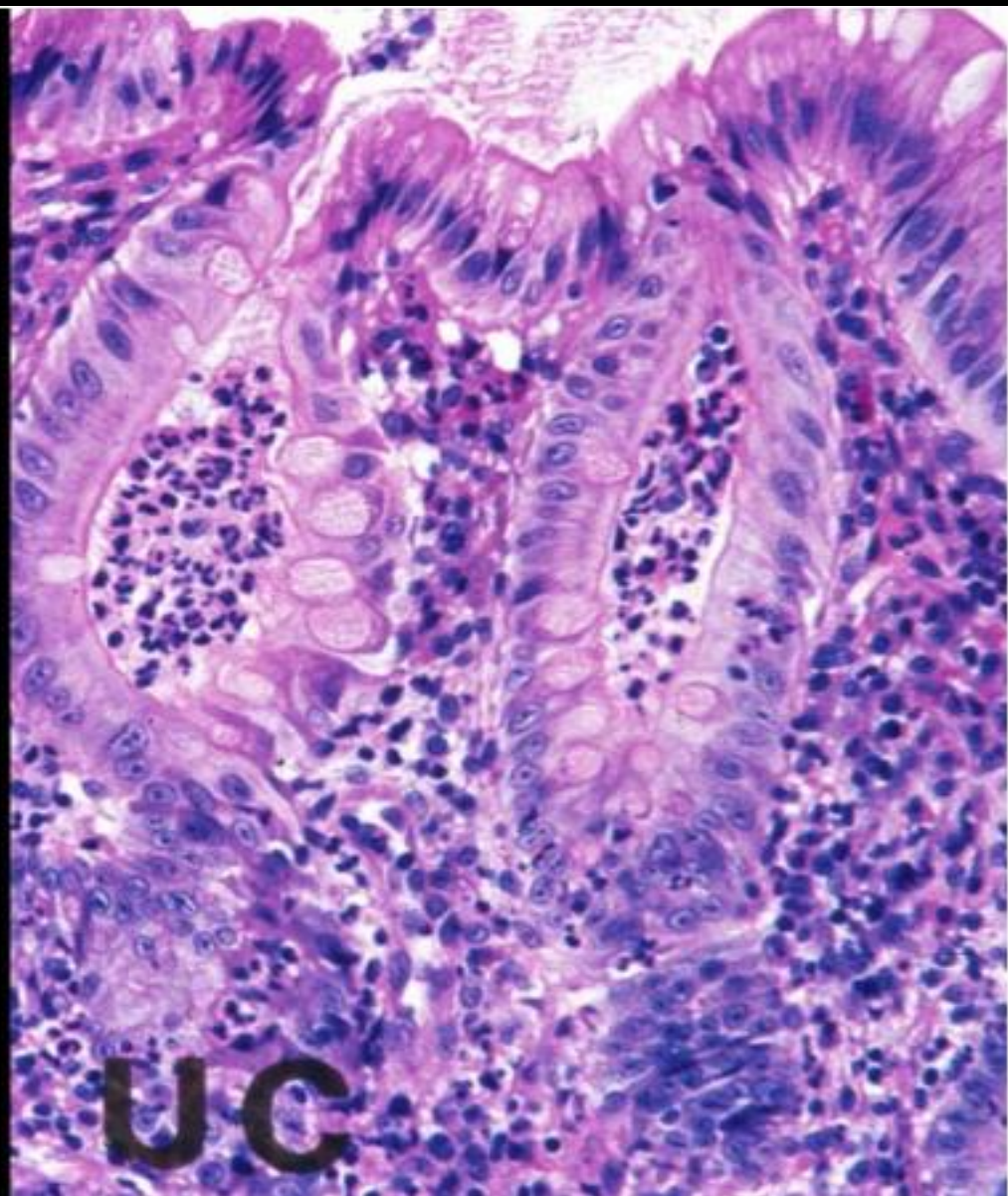
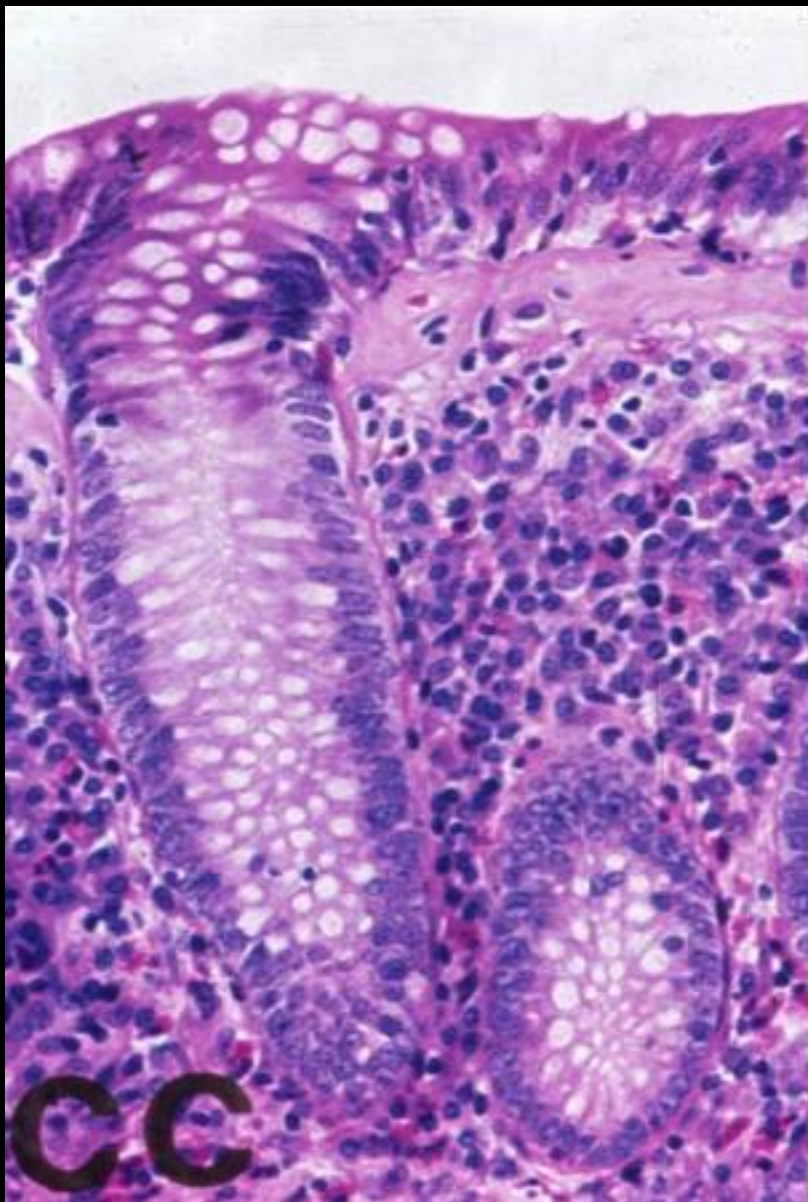


Fig 1. Concordance of the two pathologists in the interpretation of the histologic findings. The frequencies of agreement and disagreement are shown by percentages within each bar for the total experience and for each of the histologic distinctions.



Collagenous Colitis

Therapy

- **Previously**
 - Diet, ASA, Steroids
- **Currently**
 - Bismuth
 - Budesonide

ORIGIN OF THE TERM “MICROSCOPIC COLITIS”

➤ **“A mild increase in the number of inflammatory cells on colonic or rectal biopsy was observed [without] crypt abscesses, pus on a rectal mucosal smear, abnormal sigmoidoscopic appearance, or abnormal barium enema...this mild inflammatory change...was designated ‘microscopic colitis’.”**

Read, et al. Gastroenterology 78:264, 1980

Collagenous Colitis

- Often diffuse, Rt > Lt
- Normal endoscopy
- Intraepithelial lymphocytosis (mild)
- Increase lymph, plasma cells, Eos (mild)
- Thick irregular subepithelial collagen layer
- Surface degeneration/stripping
- Crypt hyperplasia
- +/- chronicity features

IBD Mimickers

1. WITH SPECIFIC ANATOMICAL DISTRIBUTION
2. INFLAMMATION WITH NO SIGNIFICANT ARCHITECTURAL DISORDER
 - a) Mostly active process
 - b) Mostly interstitial process
3. MULTIFOCAL ULCERATIONS
4. CHRONIC INTRAEPITHELIAL PROCESS

1. **Diverticular disease associated colitis**
2. **Inflammation with no significant architectural disorder**
 - a) **Infectious-type colitis**
 - b) **Diversion colitis**
3. **NSAID damage**
4. **Collagenous colitis**

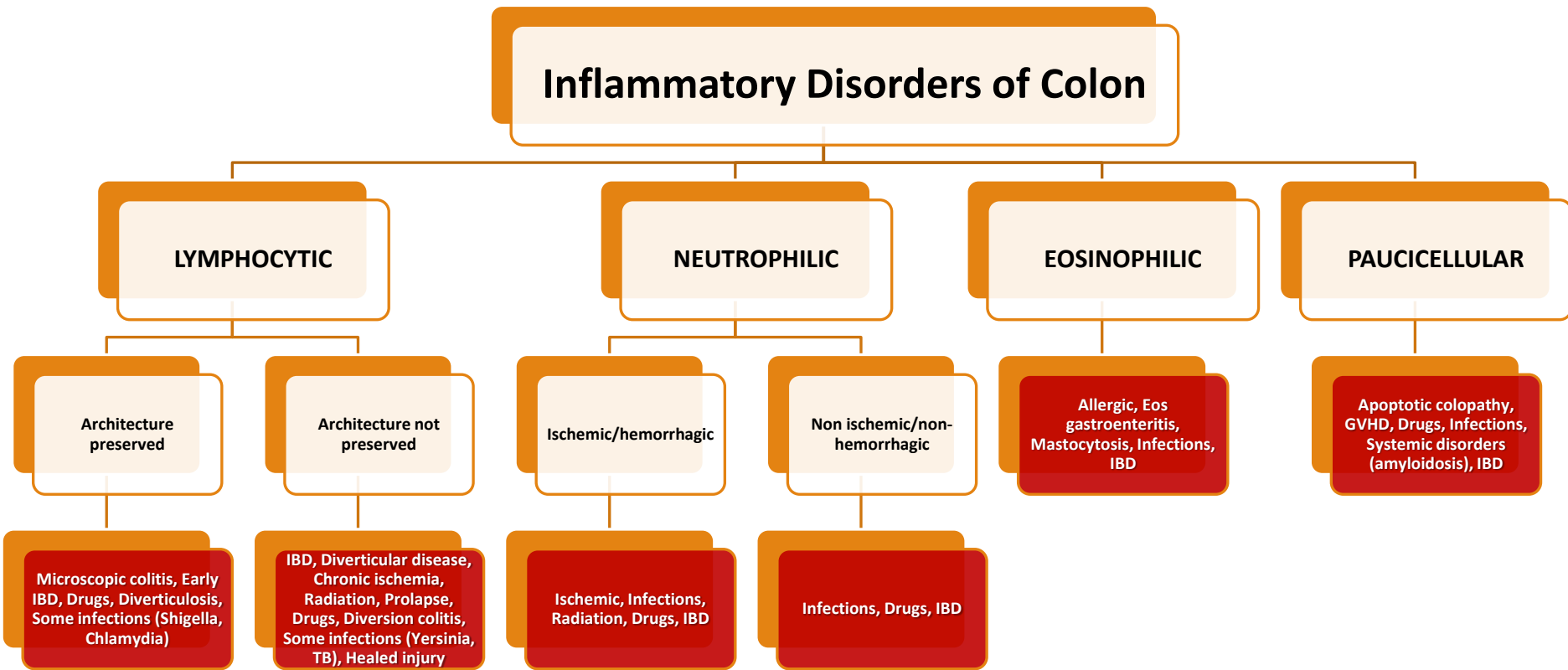
Remember

- **Not all crypt distortion is IBD**
- **Not all IBD reveals crypt distortion**
- **Some infectious colitis show “chronic” features**
- **Thus, there are NO specific features of IBD**

UC vs. Crohn's – Post-treatment Biopsies

Feature	UC	Crohn's
Diffuse disease	+/-	+/-
Rectal involvement	+/-	+/-
Segmental involvement	+/-	+/-
Ileal involvement	Mild, distal 1-5 cm	Mild-severe >5 cm
UGI involvement	Rare	+/-
Granulomas	Mucin related	Non-mucin related
Anal disease	-	+/-

Pattern Analysis of Inflammatory Colonic Bx



Messages to take home

- Evidence of chronicity is the most reliable finding for IBD pathologic diagnosis
 - Architectural changes are paramount
 - Cellularity (intraepithelial and lamina propria)
- Minimal clinical information for correlation
 - Duration of symptoms
 - Endoscopic appearance and topography
- Distribution of damage