

Speaker Declarations

Newton Wong

This presenter has the following declarations of relationship with industry

- NONE

19 Nov 2018

Colorectal disease in the immunocompromised patient

Newton ACS Wong

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Which “immunocompromised patient” ?

- HIV/AIDS patients
- Immunosuppressive drugs and transplant patients.
 - Solid organ
 - Bone marrow
- [Not primary immunodeficiencies]

1. Antibody deficiencies

X-linked Agammaglobulinemia(XLA)

Common variable immunodeficiency(CVID)

Hyper IgM syndrome

Transient hypogammaglobulinemia of infancy(selected cases)

IgG subclass deficiency± Selected IgA deficiency (selected cases)

Impaired specific antibody production with normal plasma immunoglobulin level

2. Combined immunodeficiencies

All type of severe combined immunodeficiencies(SCID)

3. Other well-defined immunodeficiency syndromes

Wiskott –Aldrich syndrome

DNA repair defects; Ataxia-telangiectasia, Nijmegen breakage syndrome

Di George Anomaly

Primary CD4 deficiency

ICF syndrome

4. Diseases of immune dysregulation

X-linked lymphoproliferative syndrome (XLP)

Talk plan

- Diseases common to several immunosuppressed groups
 - Infection
 - Neoplasia
 - Drugs
- Diseases specific to certain immunosuppressed groups

Infection

- Bacteria
 - Infectious colitides
 - ?more common in immunocompromised individuals)



Colorectal disease in liver allograft recipients – a clinicopathological study with follow-up

Newton A.C.S Wong^a, Andrew J. Bathgate^b and Christopher O.C. Bellamy^a

Objective To determine the spectrum and outcome of colorectal diseases occurring in adult liver allograft recipients.

Design A retrospective cohort analysis of clinical, microbiological and histopathological data regarding colorectal disease.

Patients Forty three out of 302 adult primary liver allograft recipients were transplanted and followed up (at median 42 months) at a tertiary referral centre/teaching hospital.

Results Out of 302 patients, 43 (14%) were investigated (by endoscopy and/or laparotomy) for symptoms of colorectal disease after orthotopic liver transplantation. The symptoms were: diarrhoea ($n = 31$); per-rectal bleeding ($n = 5$); and symptoms relating to pre-transplant

activity of pre-transplant ulcerative colitis was unchanged or increased after orthotopic liver transplantation. Two further patients developed new-onset ulcerative colitis after orthotopic liver transplantation.

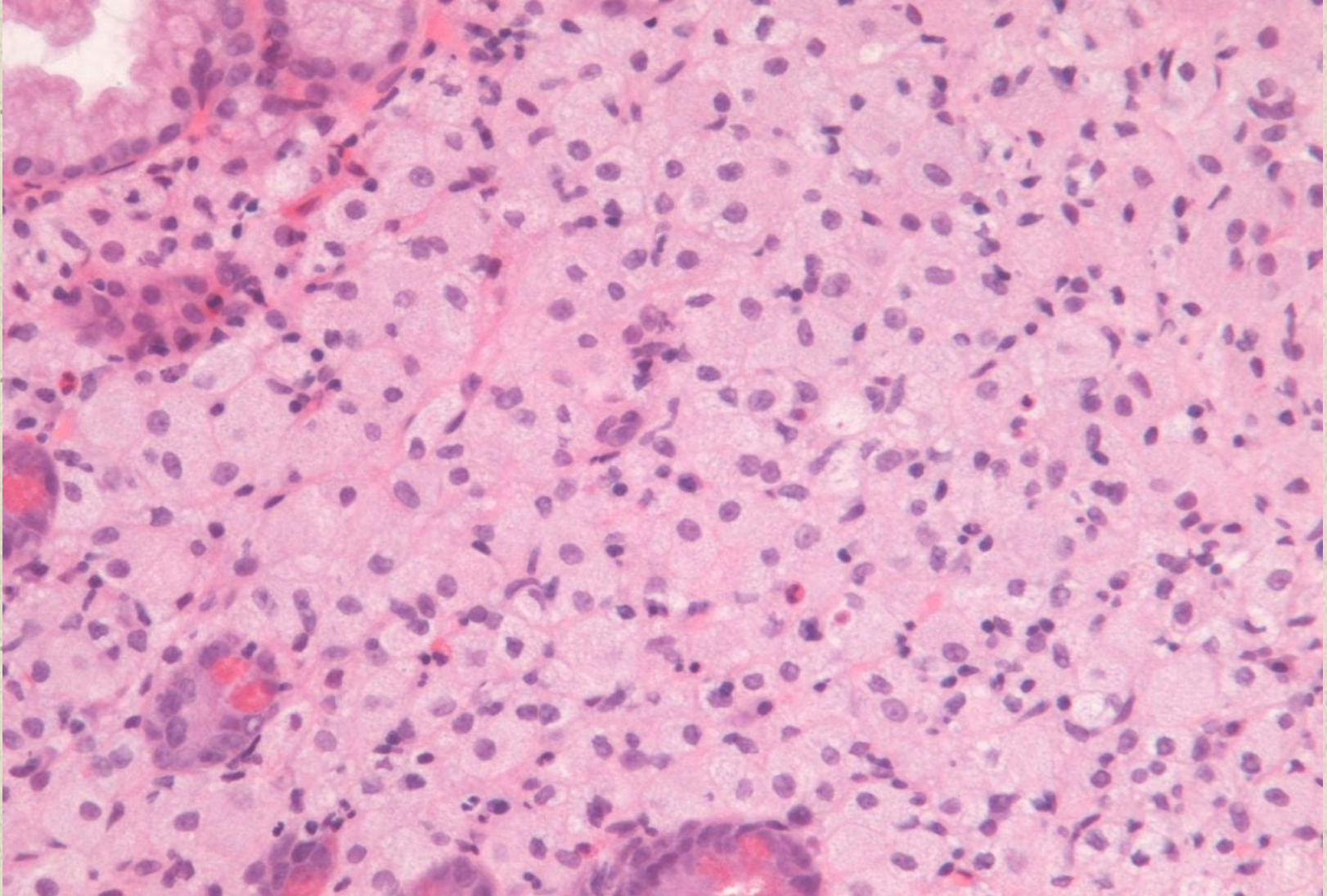
Conclusions Ulcerative colitis, *C. difficile*, cytomegalovirus infection and medications are the commonest colorectal causes of morbidity after orthotopic liver transplantation. Adult liver allograft recipients are, however, unlikely to show certain large bowel diseases encountered in other immunosuppressed groups. Amongst non-ulcerative colitis patients, those presenting with diarrhoea show a good outcome with appropriate management, whereas those with per-rectal bleeding have a more guarded prognosis. *Eur J Gastroenterol Hepatol* 14:231–236
© 2002 Lippincott Williams & Wilkins

Table 2 Causes of diarrhoea amongst 31 liver allograft recipients without pre-transplant ulcerative colitis

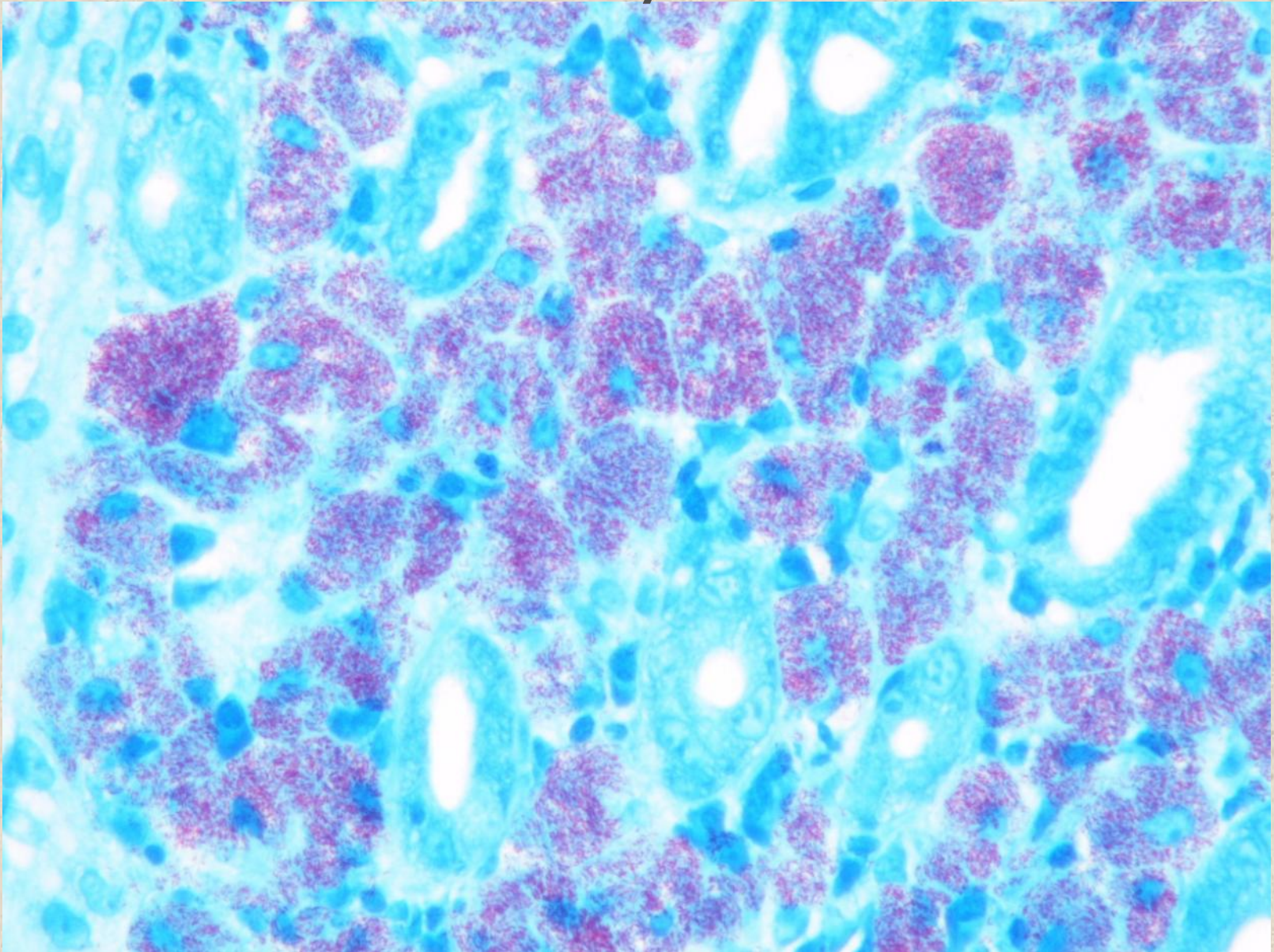
Cause of diarrhoea	Number of patients (%) (%); total $n = 31$	Median (range) onset after orthotopic liver transplantation, in months
<i>Clostridium difficile</i>	10 (32%)	1 (1–11)
Medication*	6 (20%)	6 (< 1–38)
Cytomegalovirus	3 (10%)	1 (1–6)
Colorectal adenocarcinoma	1 (3%)	25
Pancreatic insufficiency	1 (3%)	9
<i>De novo</i> ulcerative colitis	2 (6%)	9.5 (5–14)
Uncertain diagnoses	8 (26%)	2.5 (1–11)

* Four antibiotics, one iron replacement therapy, one mycophenolate mofetil.

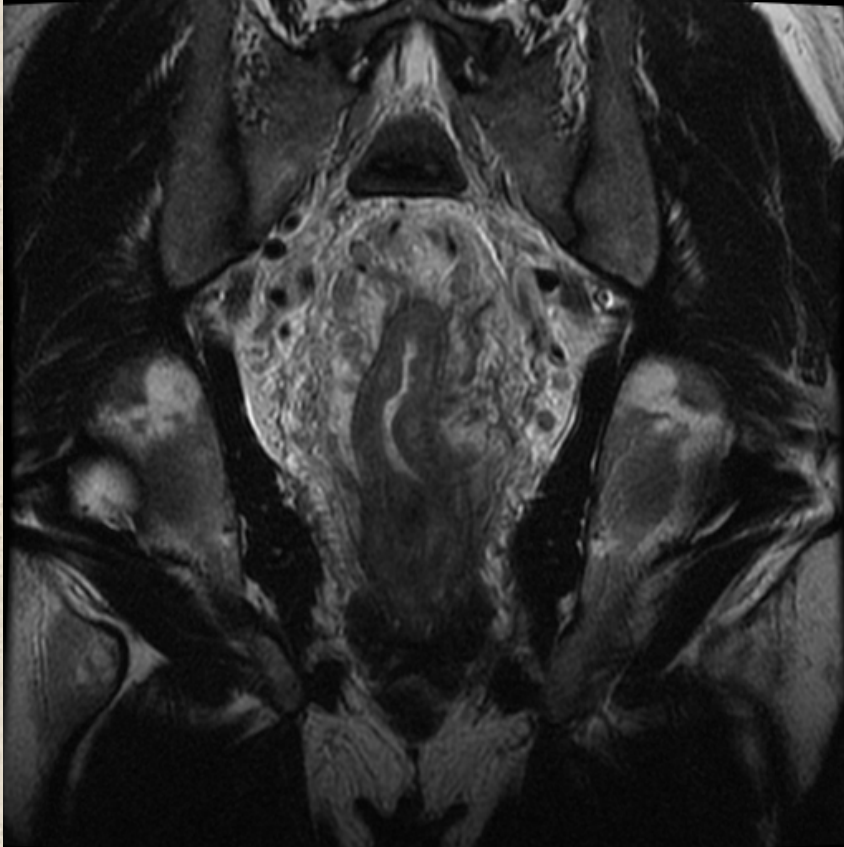
36M HIV patient



ZN+, thus: *Mycobacteria*

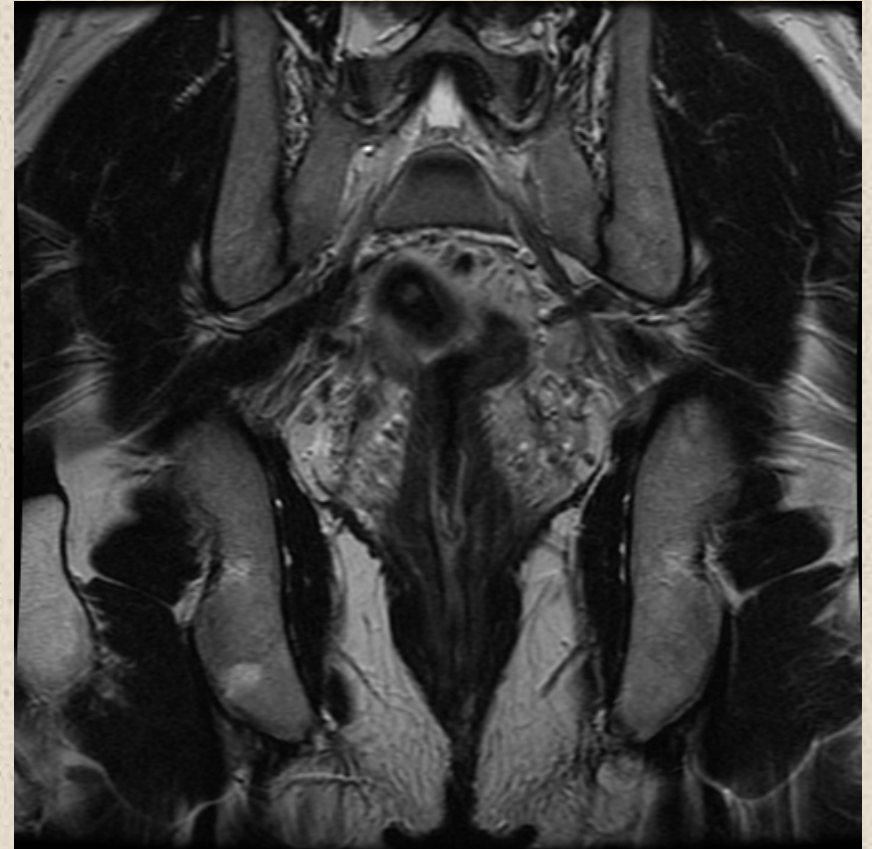
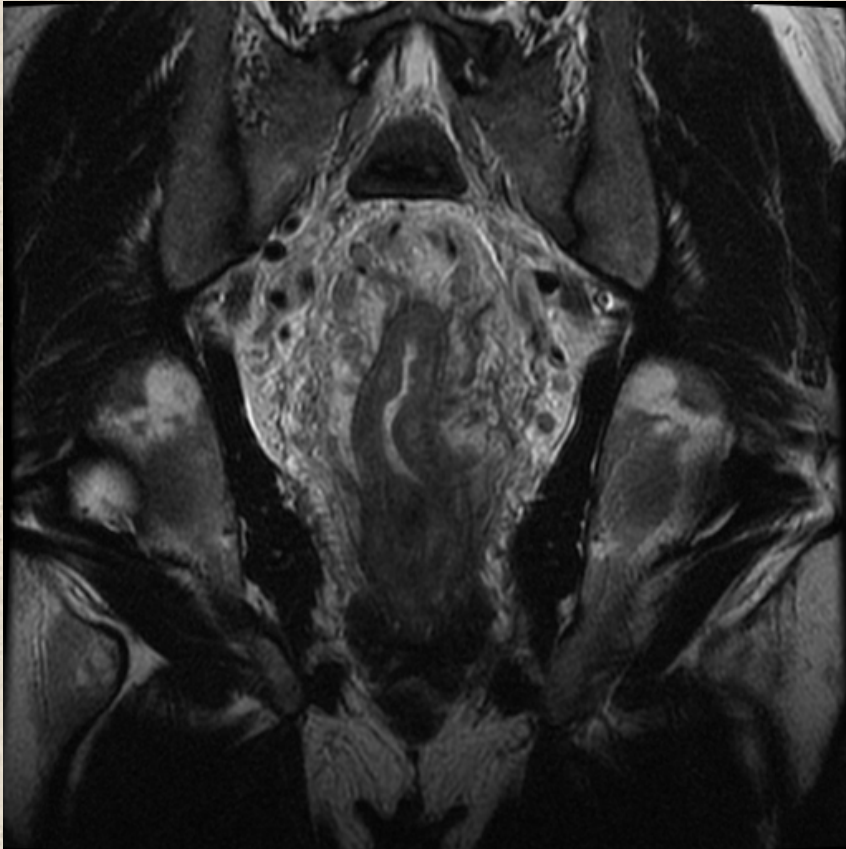


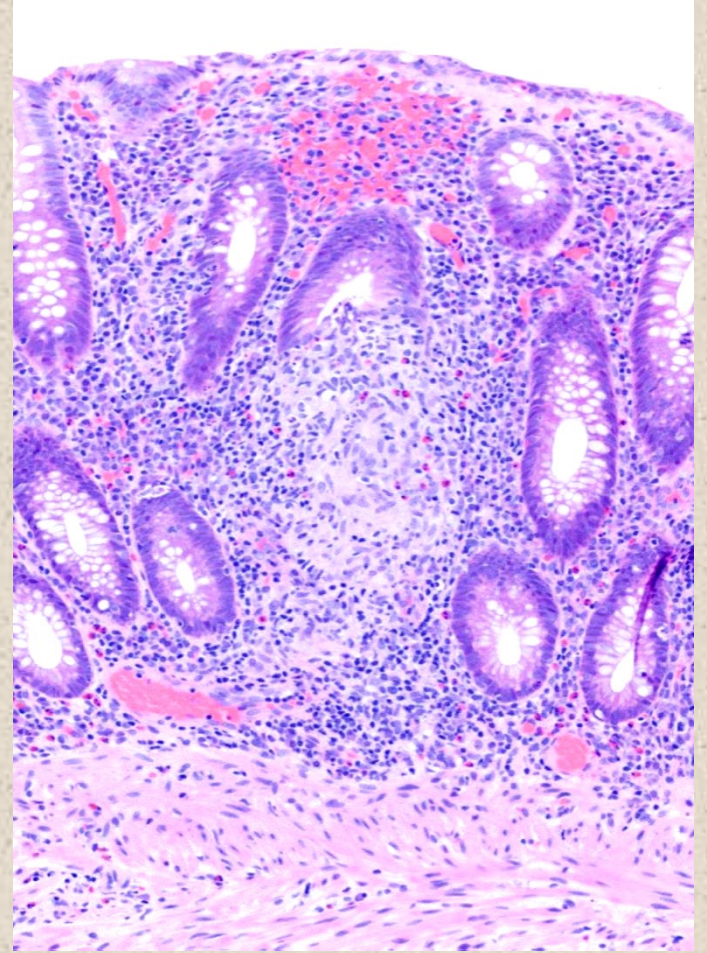
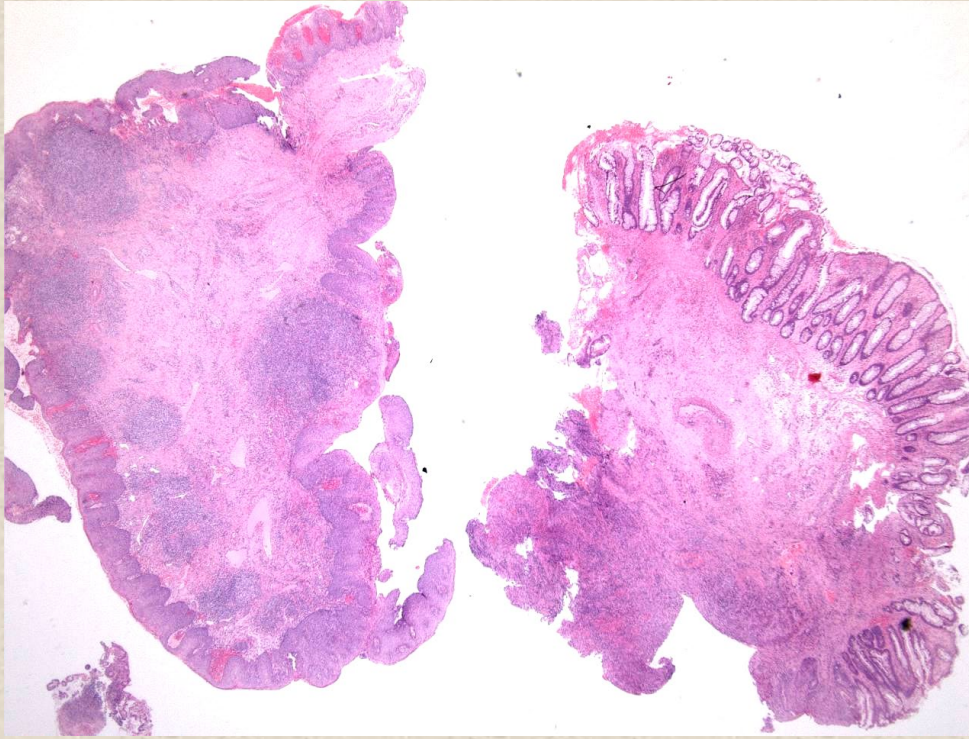
2. 23M. MSM. 10 cms long stricture in rectum and anus, clinically & radiologically malignant. The surgeon was sharpening his scalpel in the MDTM helped by the usual confidently expressed diagnosis by the radiologist. A total of 20 separate biopsies, in two settings, showed fibrinopurulent exudate, granulation tissue and inflamed fibromuscular connective tissue with no mucosa, no granulomas and no tumour.

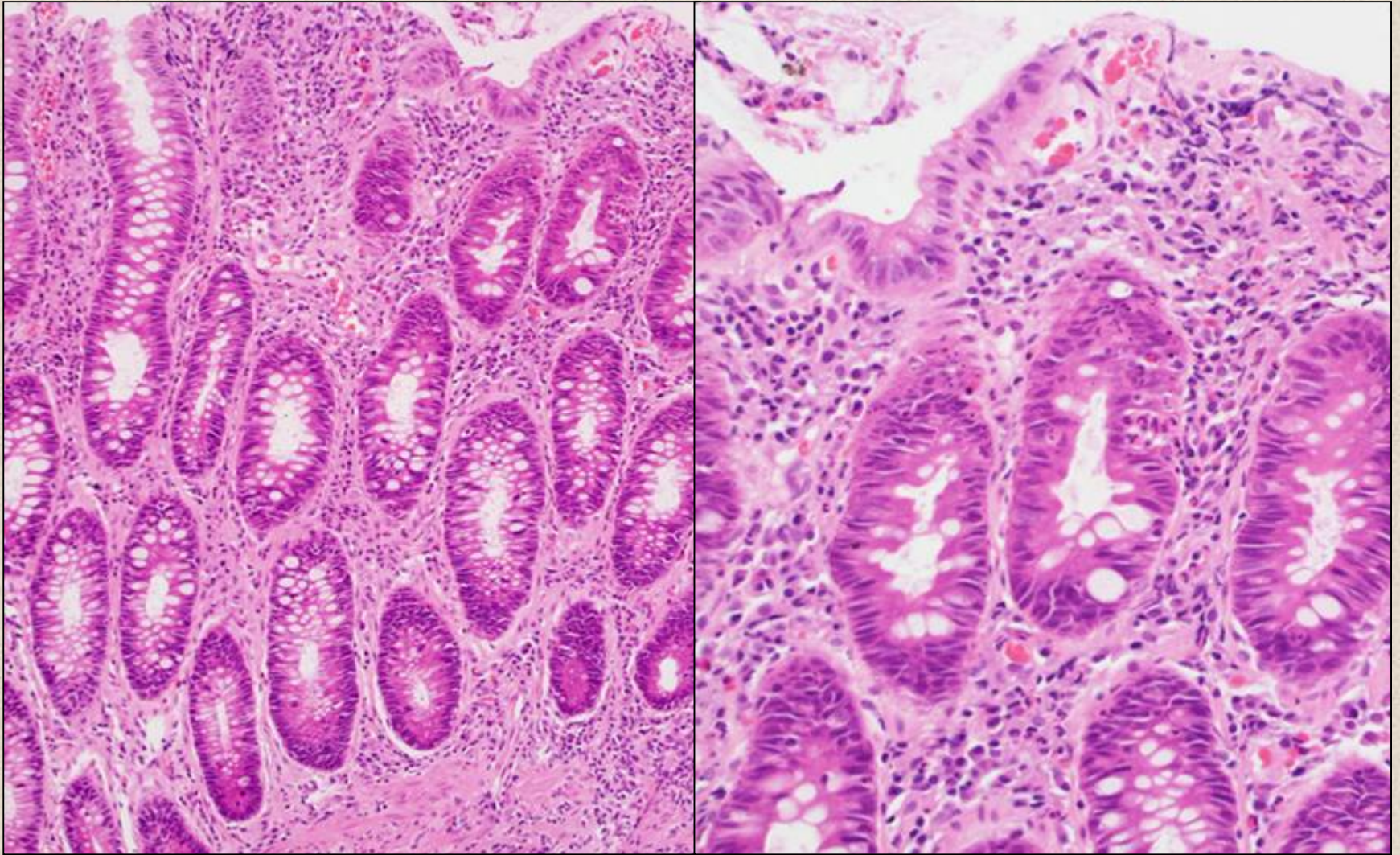


Coronal and sagittal MRI images of the pelvis

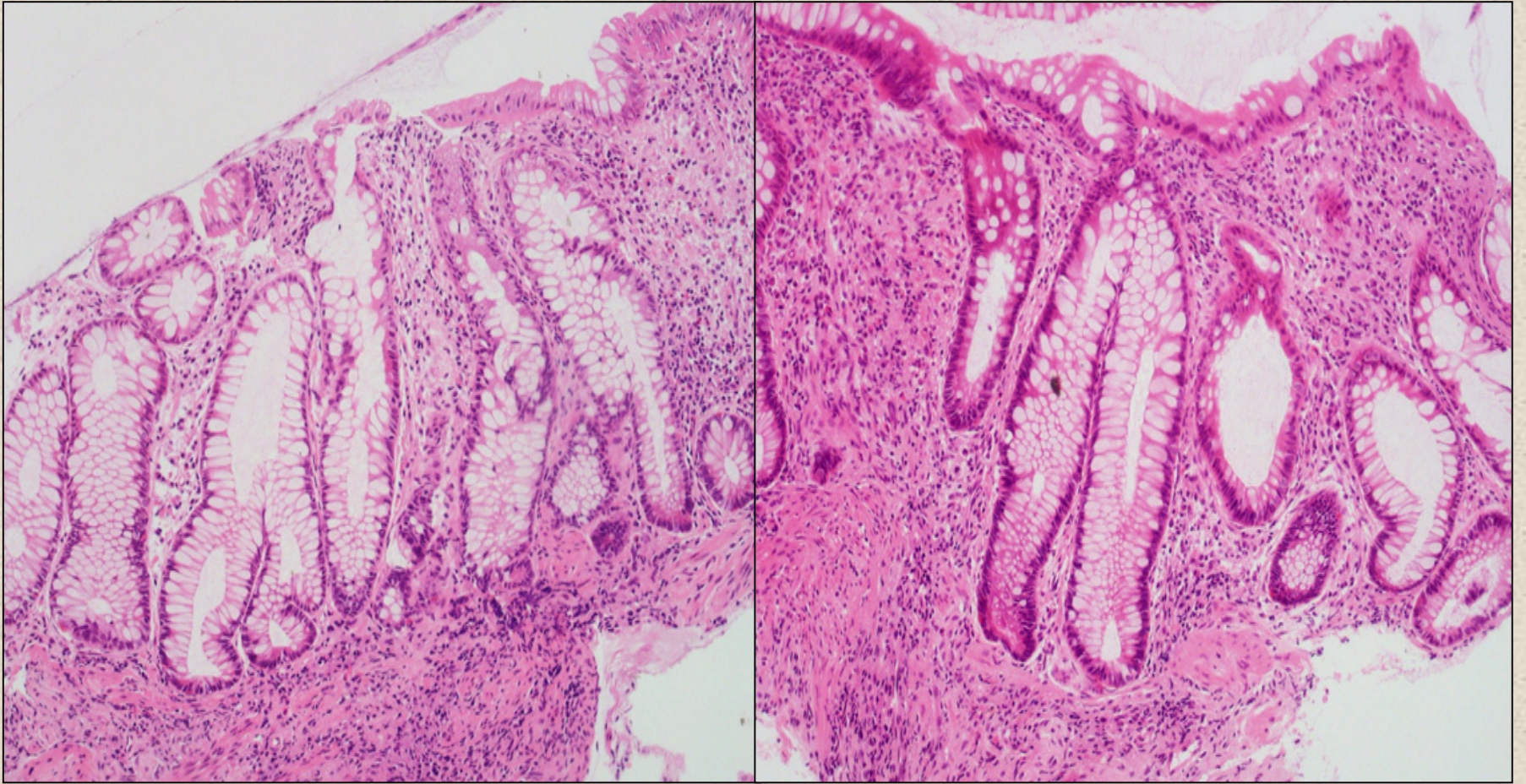
2. This is before and after treatment, with eight weeks separating them. Note the massively thickened rectum before treatment (left) and the normal calibre rectum after treatment (right). What's the diagnosis and what was the treatment?







32 year old male presented bloody diarrhoea
? IBD



Same patient 8 weeks later

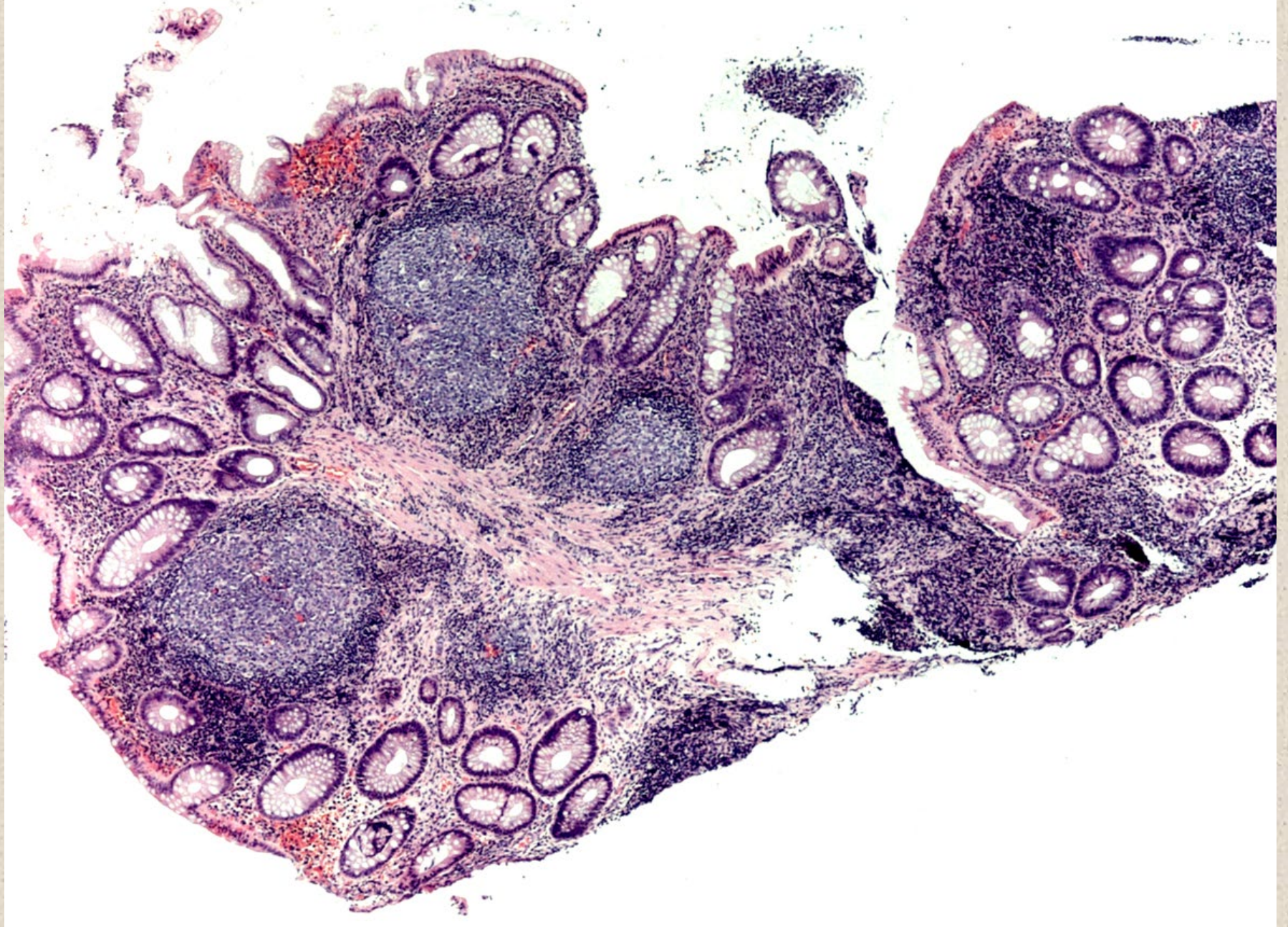
Table 2 | Summary of histological features of rectal biopsies and diagnostic timeline from 12 HIV positive men subsequently diagnosed with LGV proctitis

Histological features									Diagnosis		
Case no.	Time between biopsy & LGV diagnosis (months)	Mucosal ulcers	Cryptitis	Crypt abscess	Crypt distortion	Granuloma	Plasma cell infiltrate	Giant cells present	Initial histological diagnoses/suggestions	Mode of initial LGV diagnosis	LGV DNA results from biopsy (date of biopsy)
1	23		■	■	■ mild			■	Uncertain aetiology Possible IBD	Rectal swab LGV Positive	LGV Positive (September 2004) Negative (December 2002 & June 2003)
2	22		■	■		■			?IBD ?infective	Rectal swab LGV Positive	LGV Positive (May 03)
3	23		■		■ mild				?Due to prolapse ?IBD	Rectal swab LGV Positive	Negative
4	12	■	■				■		Uncertain aetiology	Clinical & Serology WIF titre = 1:4000	Negative (September 2004) Inhibitory (December 2003)
5	12	■				■	■		?Infective ?Crohn's	Rectal swab LGV Positive	Positive
6	3	■		■		■			Exclude LGV	Clinical Rectal swab CT detected, not sent for LGV testing	LGV Positive
7	9		■	■	■ minimal			■	Favours infective aetiology	Rectal swab LGV Positive	Negative (September 2004 and January 2005)
8	2	■	■	■	■ mild				?IBD. Exclude CT/LGV	Clinical & Serology CFT titre = 1:512 WIF titre = 1:4000	LGV Positive (March 2005) Negative (June 2005)
9	4	■	■	■		■	■		?Crohn's	Clinical & Serology CFT titre = 1:1280	LGV Positive (November 2005) August 2005 not tested
10	0	■					■		Diagnosed Crohn's 1999, thought to be recurrence	Rectal swab LGV Positive	Negative (November 2005)
11	0	■	■		■ mild	■			?IBD. Exclude LGV Concurrent Anal SCC present	Rectal swab LGV Positive	Negative (November 2005) December 2005 not tested
12	6		■				■		?Early ulcerative colitis	Rectal swab LGV Positive	Negative

Soni S, Srirajaskanthan R, Lucas SB, Alexander S, Wong T, White JA.
Lymphogranuloma venereum proctitis masquerading as inflammatory bowel disease
in 12 homosexual men. Aliment Pharmacol Ther. 2010 Jul;32(1):59-65.

Chlamydia

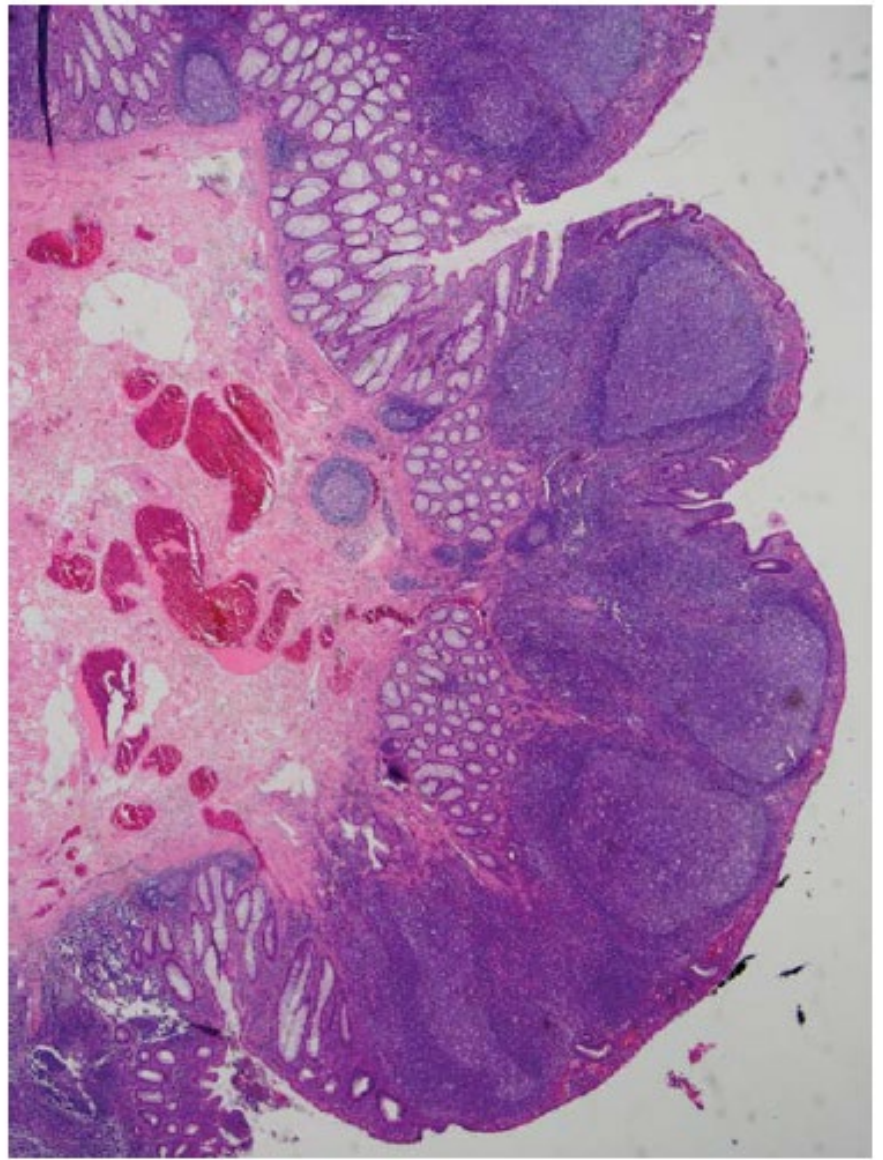
- Proctocolitis ('follicular proctitis')
... can mimic inflammatory
bowel disease



37 year old male - Posterior rectal wall sessile polyp



Obstructing rectal mass seen on endoscopy.



24-year-old Black female.

The Rectal Tonsil— A Reaction to Chlamydial Infection?

Am J Surg Pathol • Volume 33, Number 3, March 2009

the use of cervical or urethral swabs, or urine samples, for routine testing.

Molecular genotyping to evaluate for LGV is not routinely performed. LGV is endemic in Africa, Asia, and South America, but is generally rare elsewhere. It may merit special consideration when there is rectal infection with a concern for a clinically serious disease process (in our case, a mass-like lesion with Crohn-like symptoms), which is an indication to forward specimens to the CDC for specialized testing.

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REFERENCES

1. Boisvert J-F, Koutsky LA, Suchland RJ, et al. Clinical features of *Chlamydia trachomatis* rectal infection by serovar among homosexually active men. *Sex Transm Dis*. 1999;26:392-398.

Chlamydia

- Proctocolitis ('follicular proctitis')
- ??? Hyperplastic MALT tissue in rectum ('rectal tonsil') – or is it EBV-related?

Infection

- Bacteria
 - Infectious colitides
 - Clostridium difficile
 - Mycobacteria
 - Chlamydia

Viruses



CMV

- HIV/AIDS, transplant and chemotherapy patients
- Cause of isolated apoptosis

- 77 year old male
- Presented with PR bleeding
- Endoscopy showed a caecal focus of inflammation (?Crohn's) and a sigmoid colonic sessile lesion (?CRC)

ID No. : ■

Name :

Sex : Age :

D. O. Birth :

09/11/2009

11:56:16

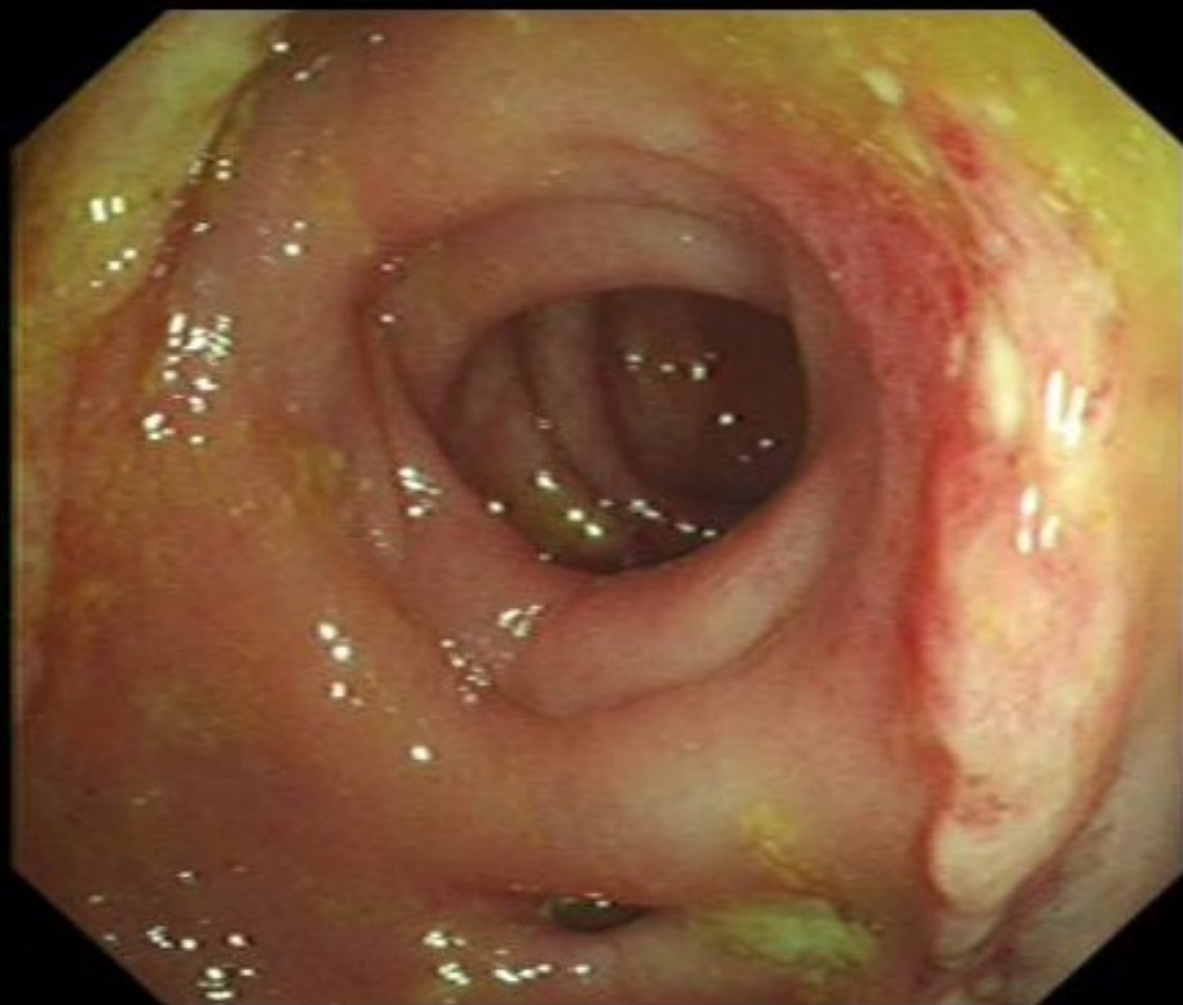
SCV: 99

CVP: D1/1

Ct: N Eh: A0

Ce: 3 Z: 1.0

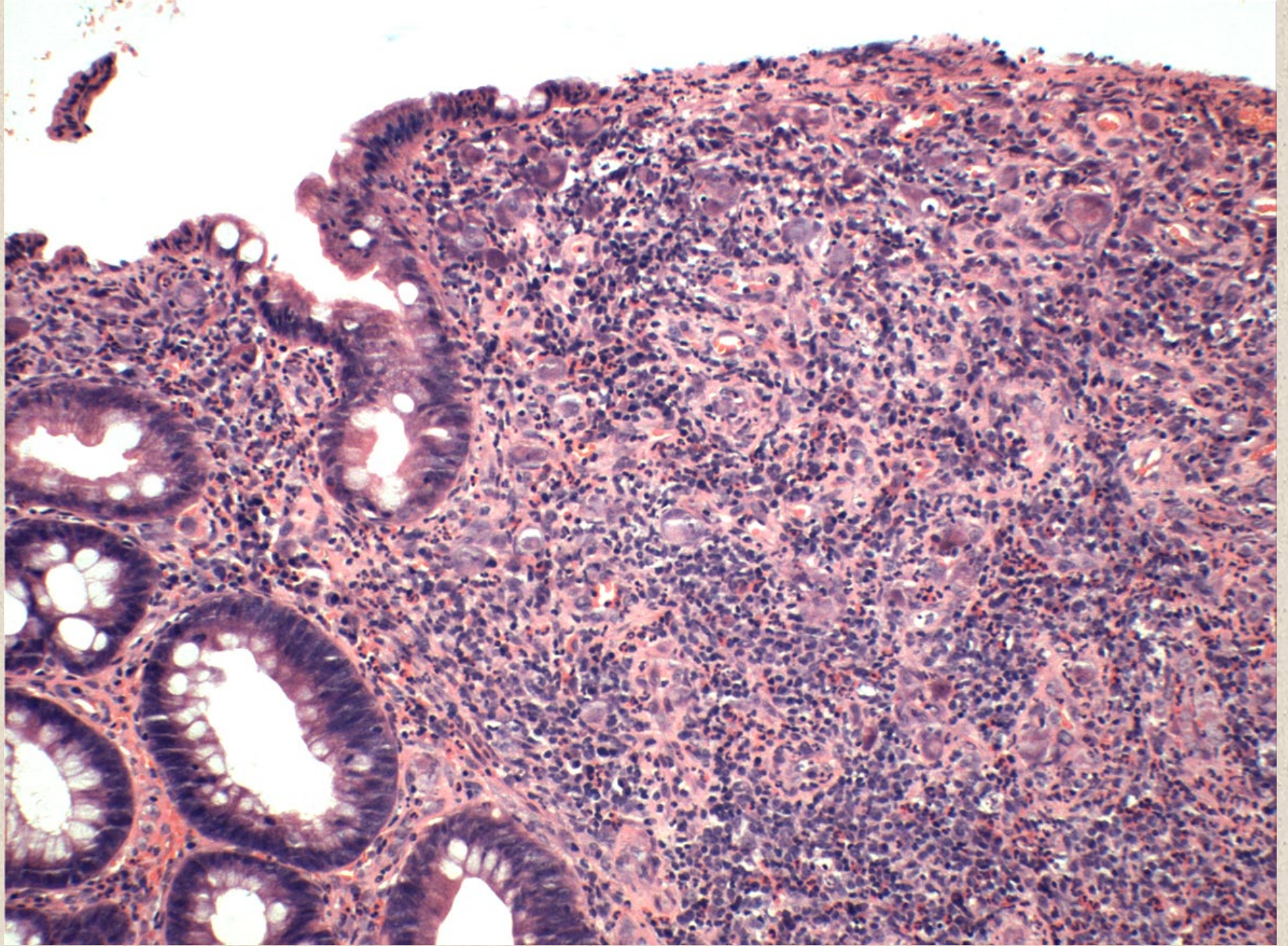
IHb=55



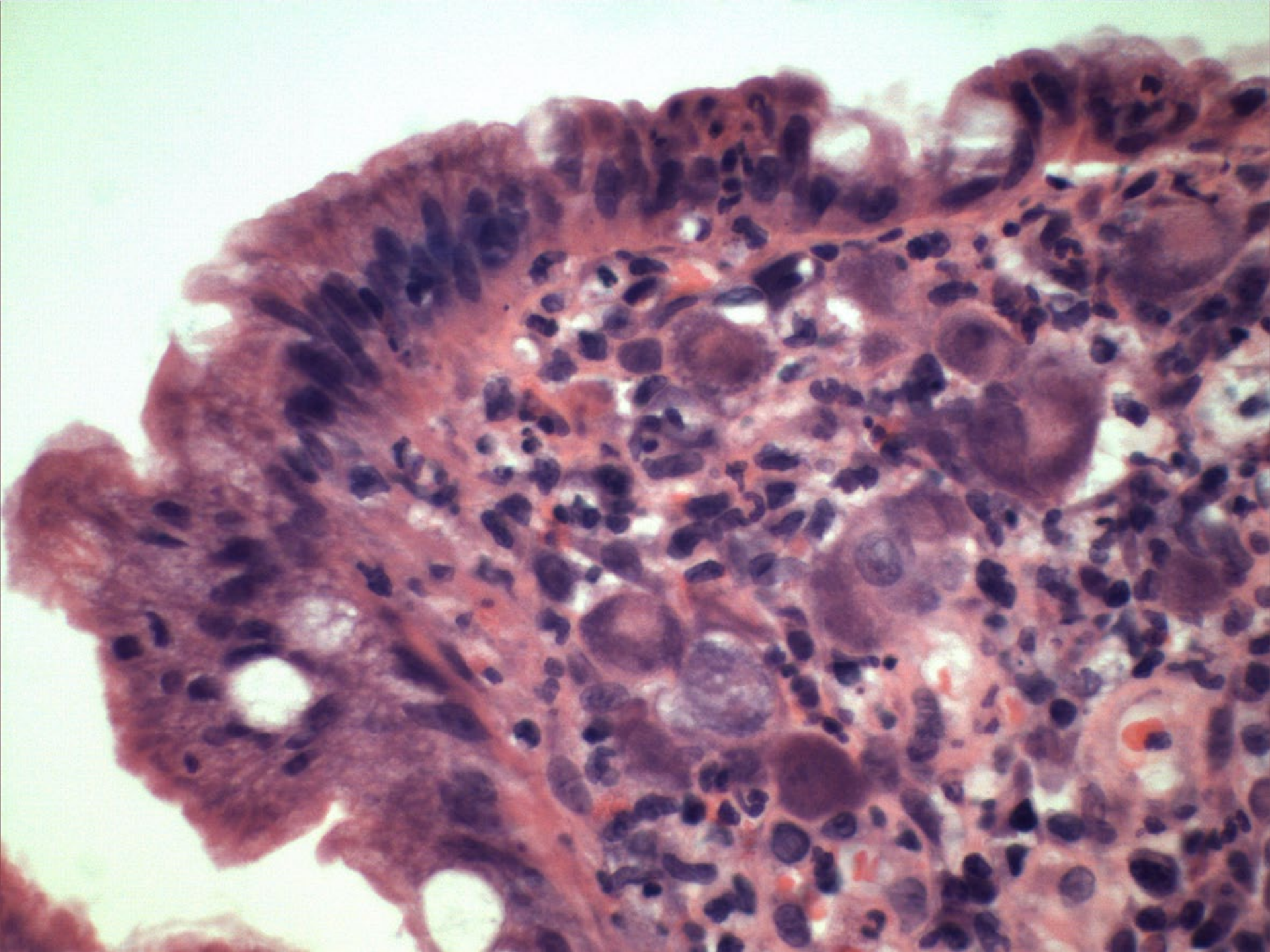
Physician :

Comment :

Sigmoid colonic tumour

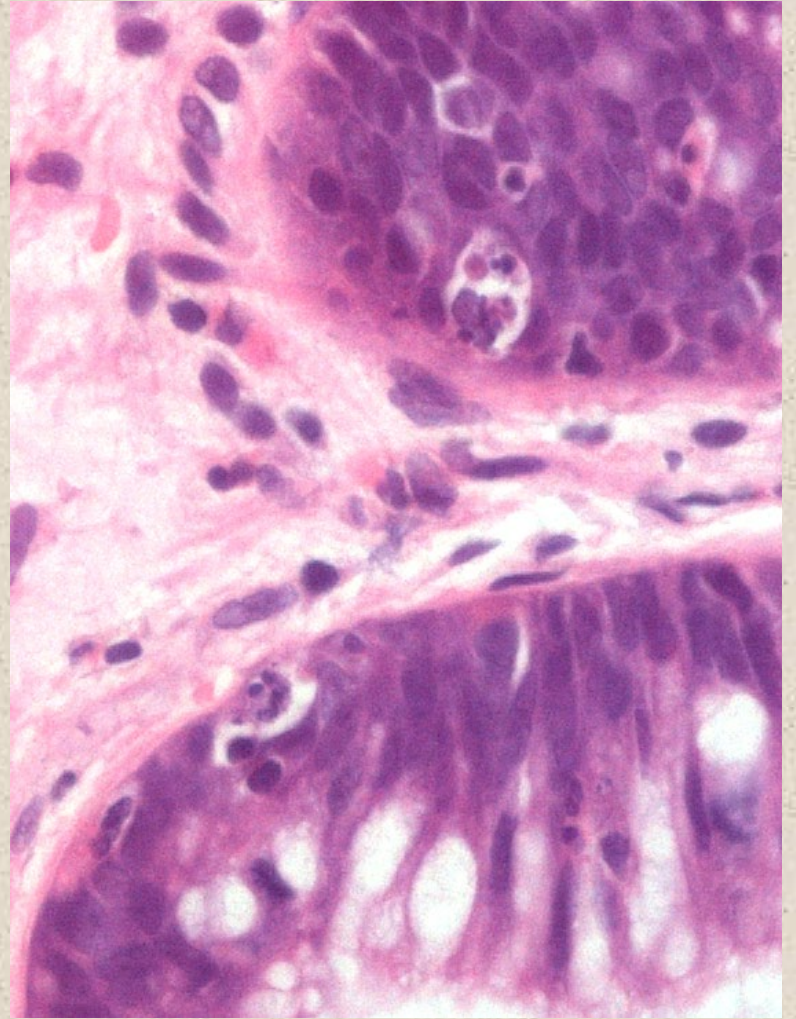
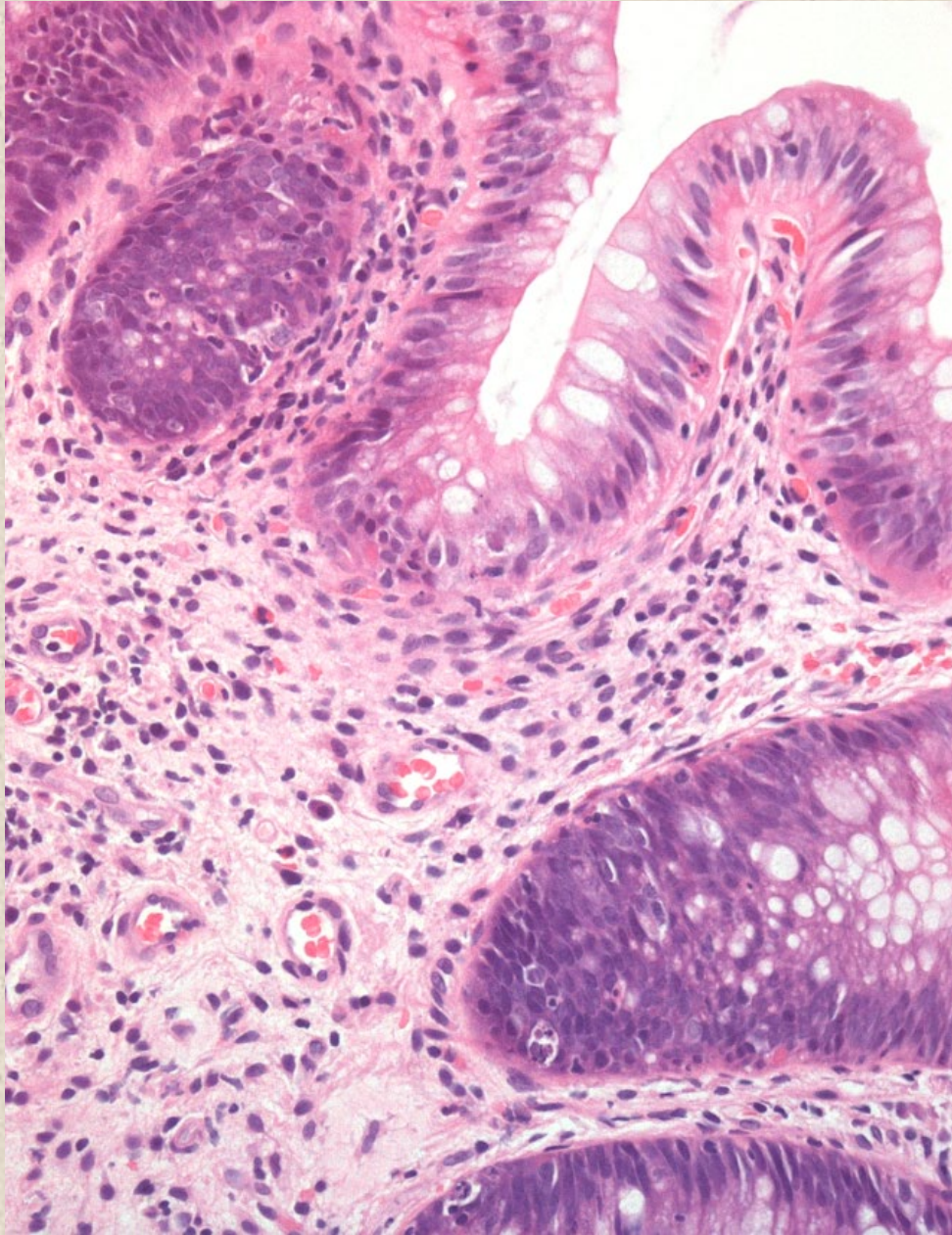


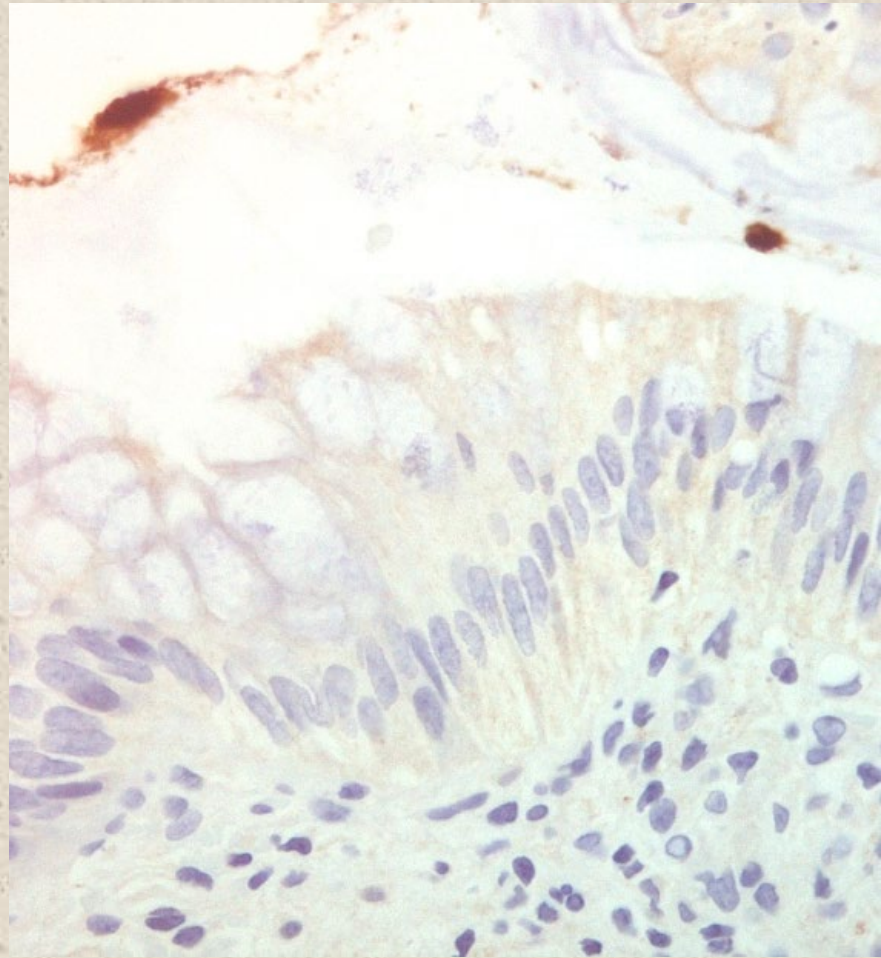
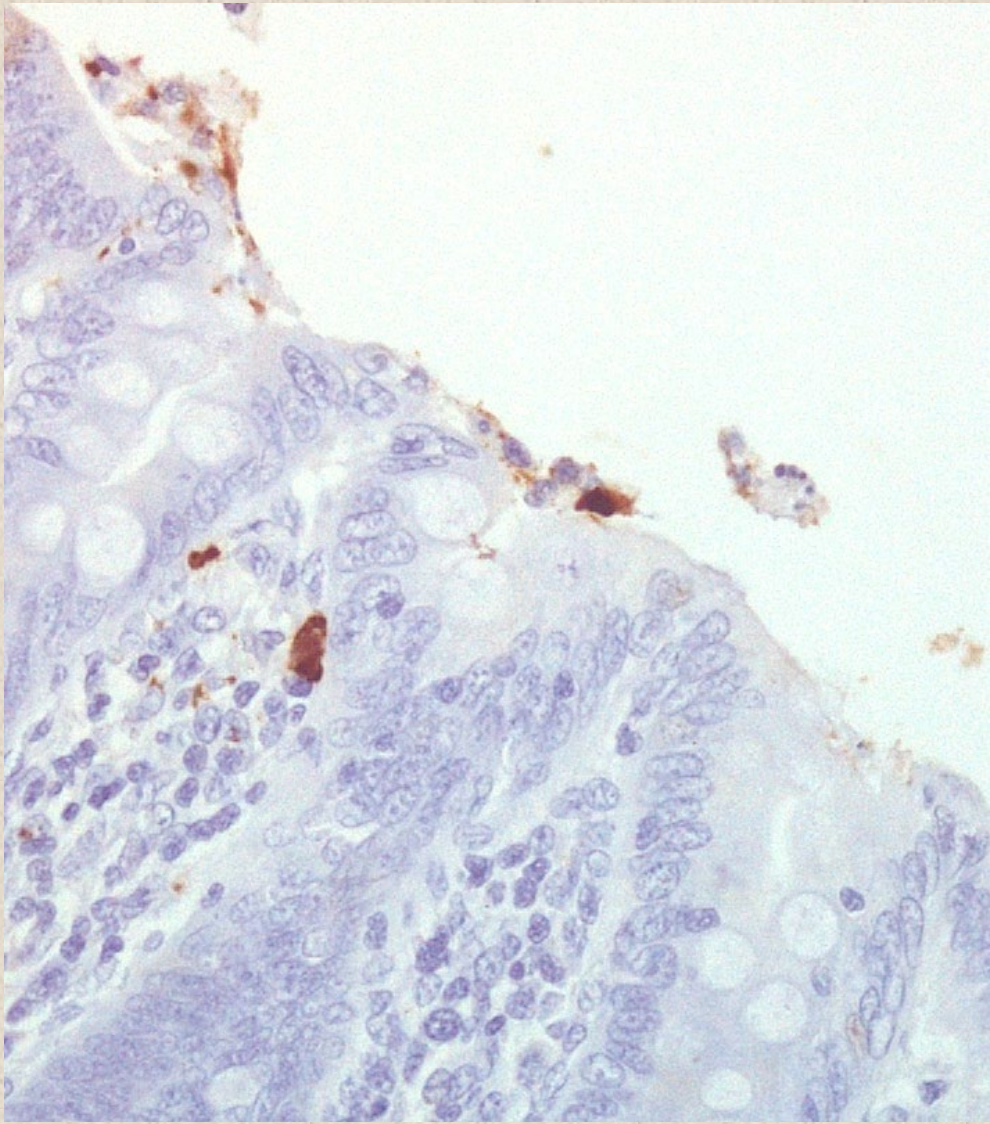
Biopsies of sigmoid colonic tumour



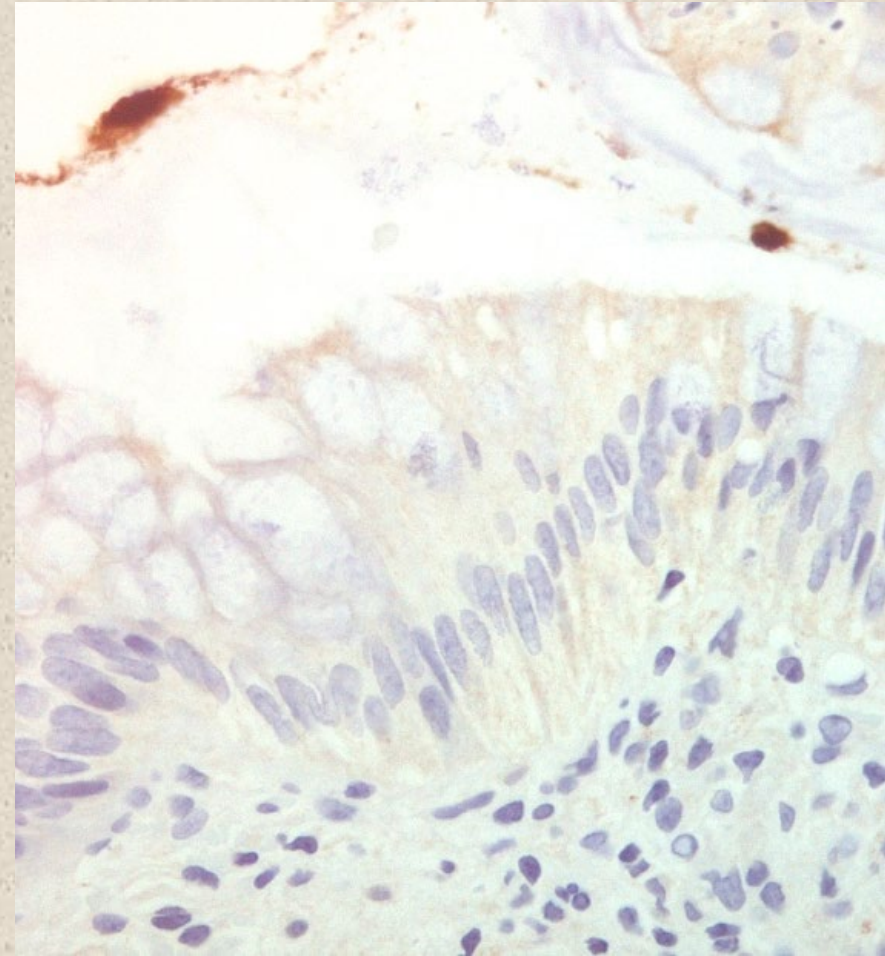
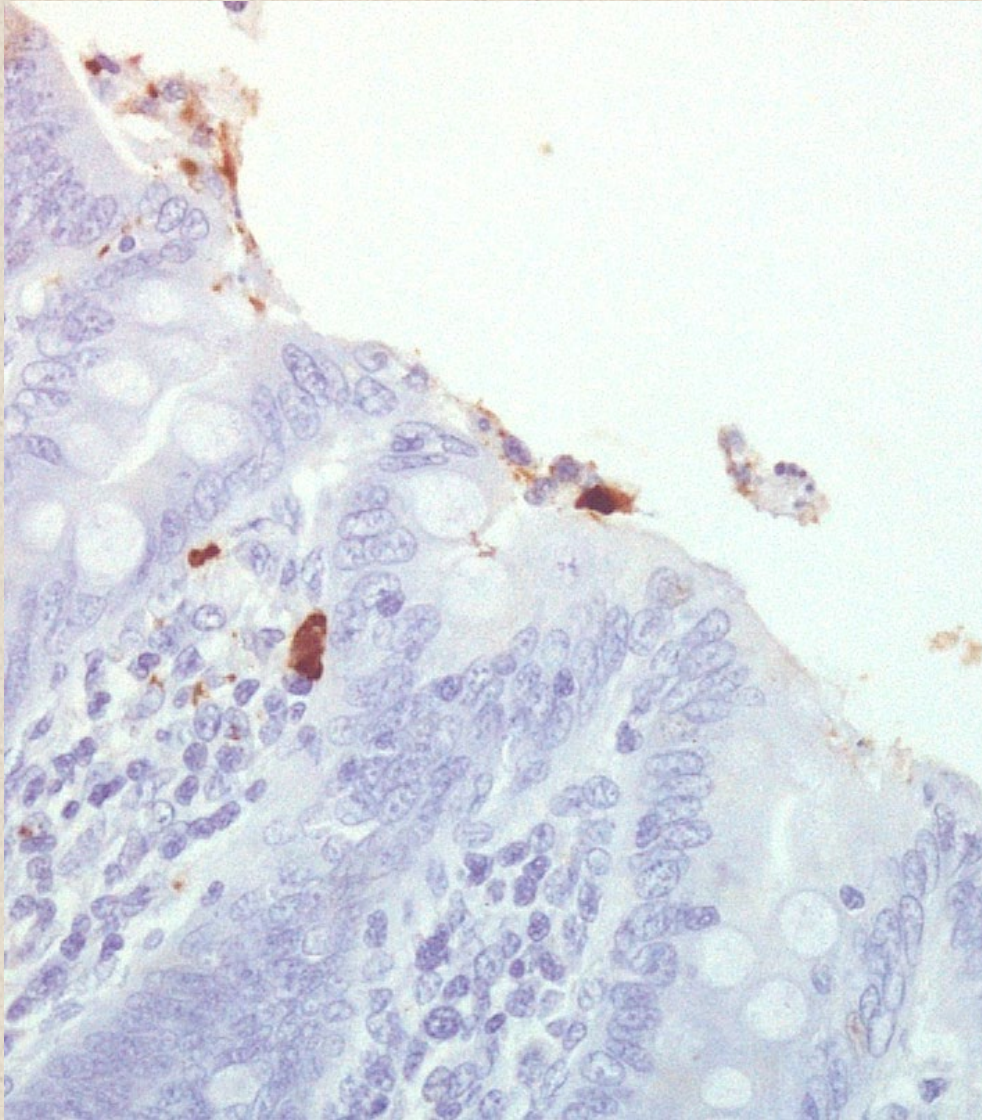
CMV

- HIV/AIDS, transplant and chemotherapy patients
- Cause of isolated apoptosis
- Can present as a focal lesion/polyp in the large bowel





Adenovirus



Viruses

- EBV – PTLD and smooth muscle tumours
- HHV8 – Kaposi sarcoma and primary effusion lymphoma
- HPV – anal squamous neoplasia

Fungi and parasites



Fungal

Candida spp

Cryptococcus neoformans

Histoplasma capsulatum

Pneumocystis jirovecii (*carinii*)

Parasitic

Cryptosporidium parvum

Cyclospora cayetanensis

Entamoeba histolytica

Giardia lamblia (*intestinalis*)

Isospora belli

Leishmania donovani

Microsporidia (*Enterocytozoon bieneusi*, *Encephalitozoon*
[*Septata*] *intestinalis*)

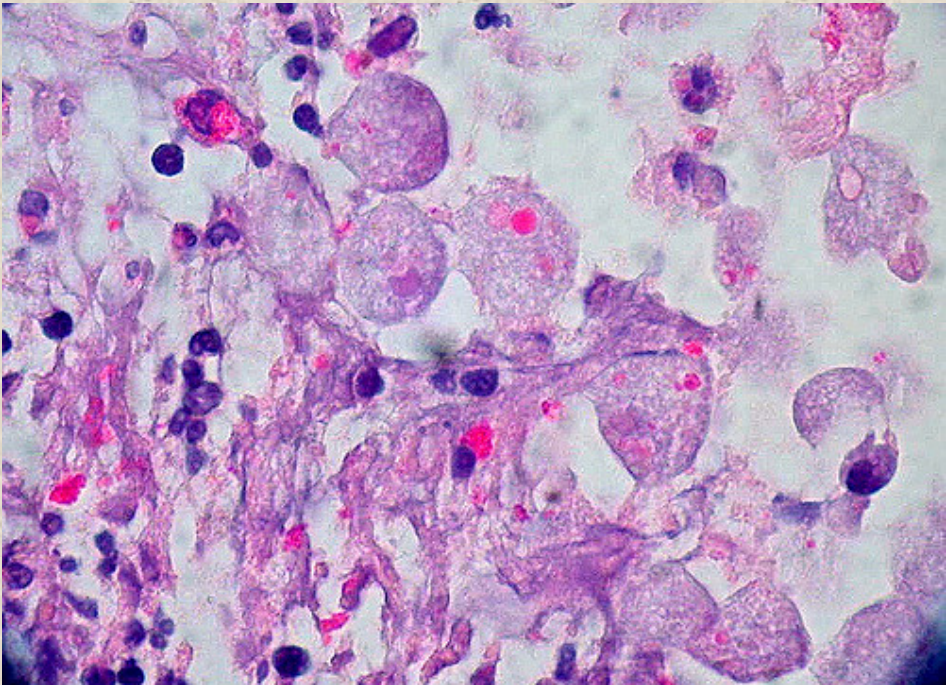
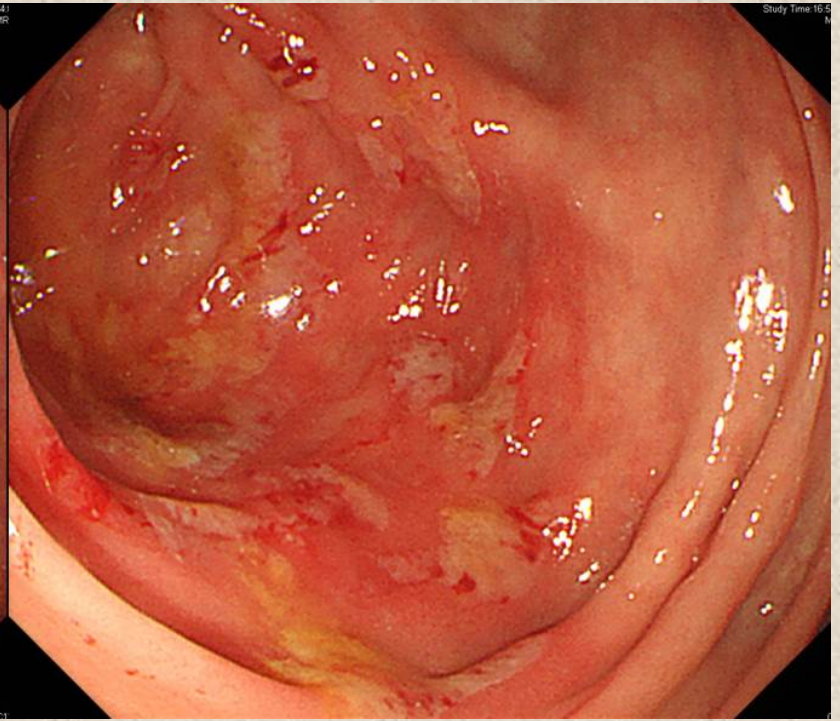
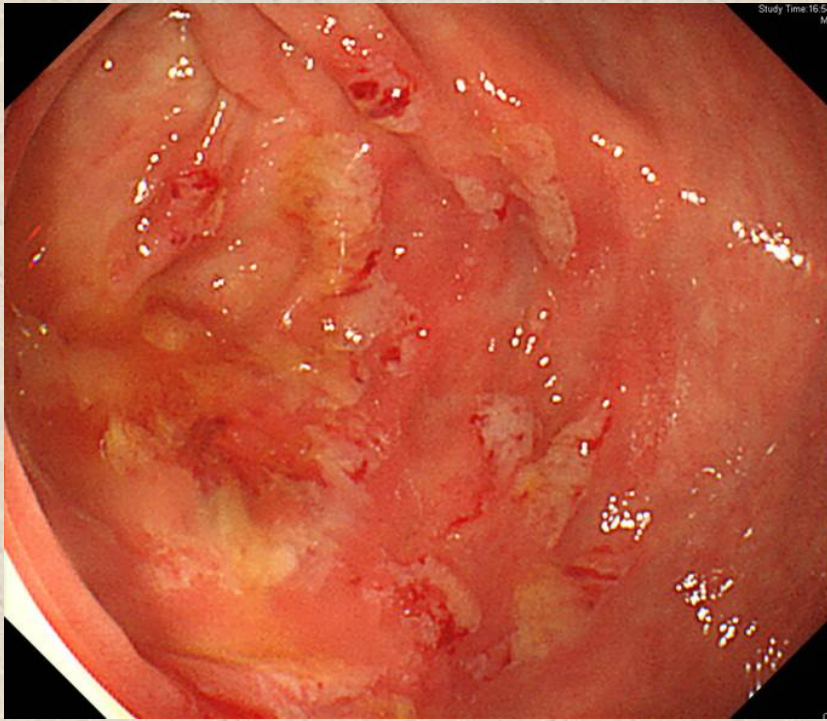
Strongyloides stercoralis

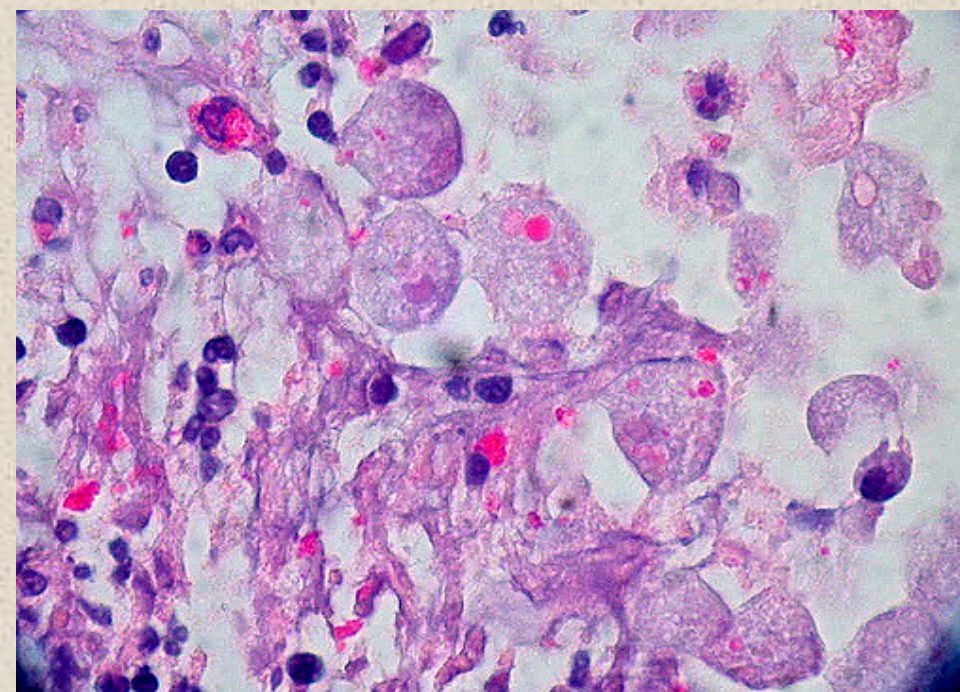
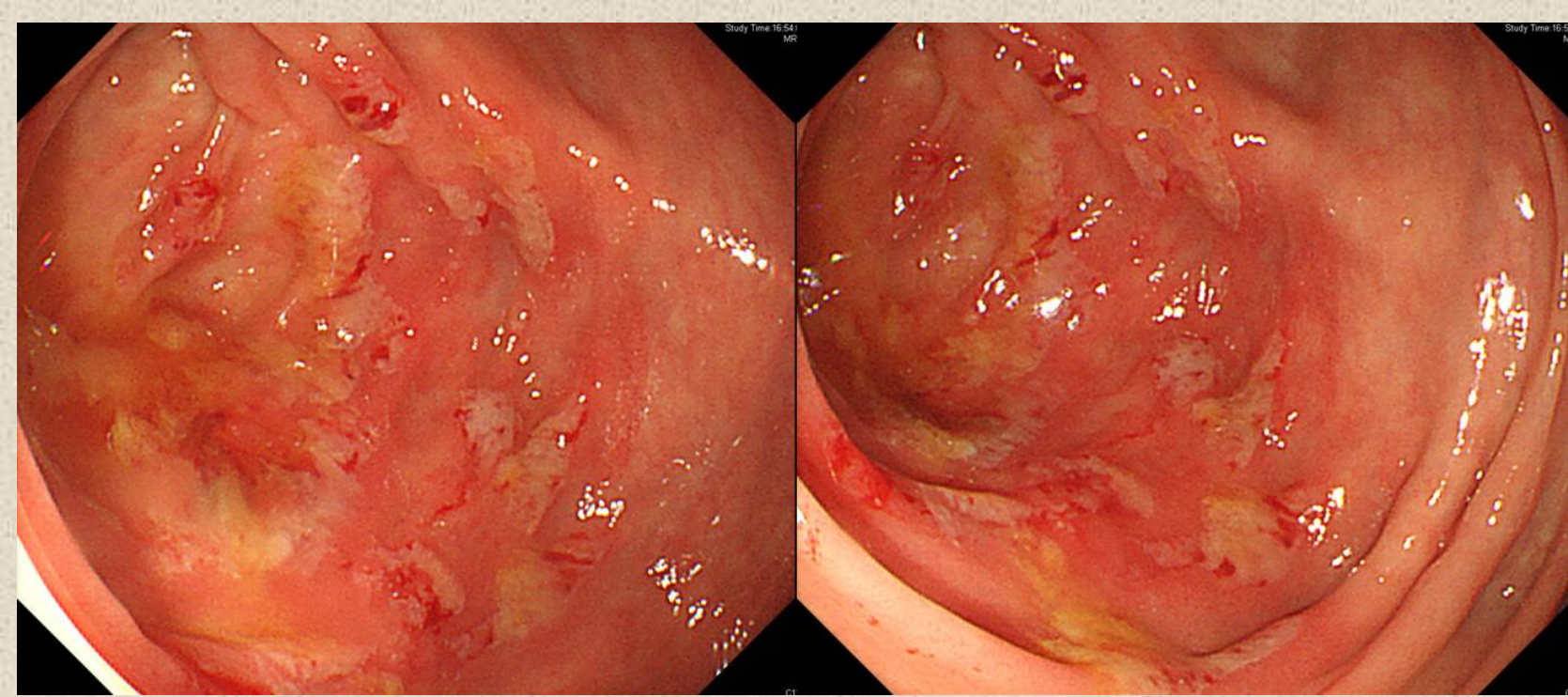
Toxoplasma gondii

Human Immunodeficiency Virus–Related Gastrointestinal Pathology

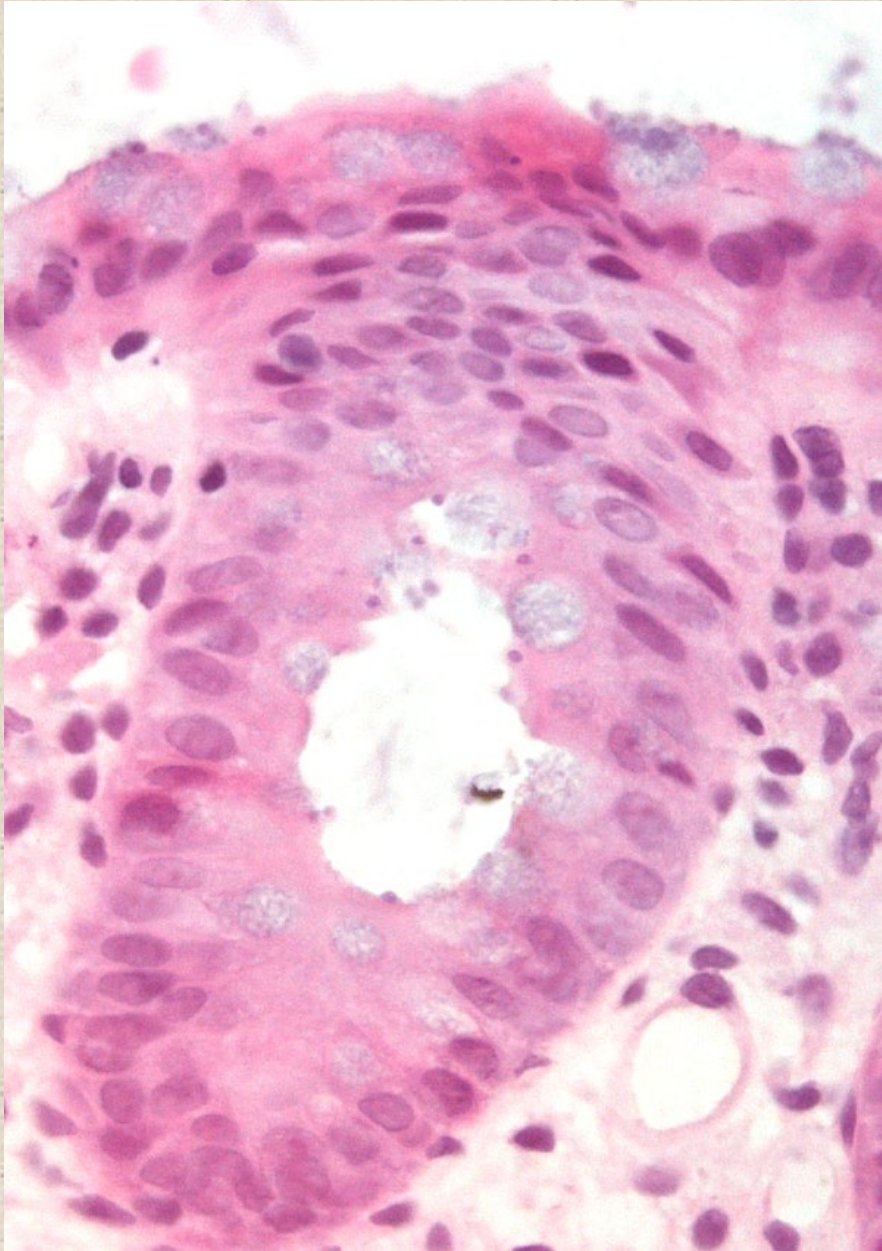
Tomas Slavik, MBChB, FCPATH(SA), MMed

(*Arch Pathol Lab Med.* 2012;136:305–315; doi: 10.5858/arpa.2011-0332-RA)

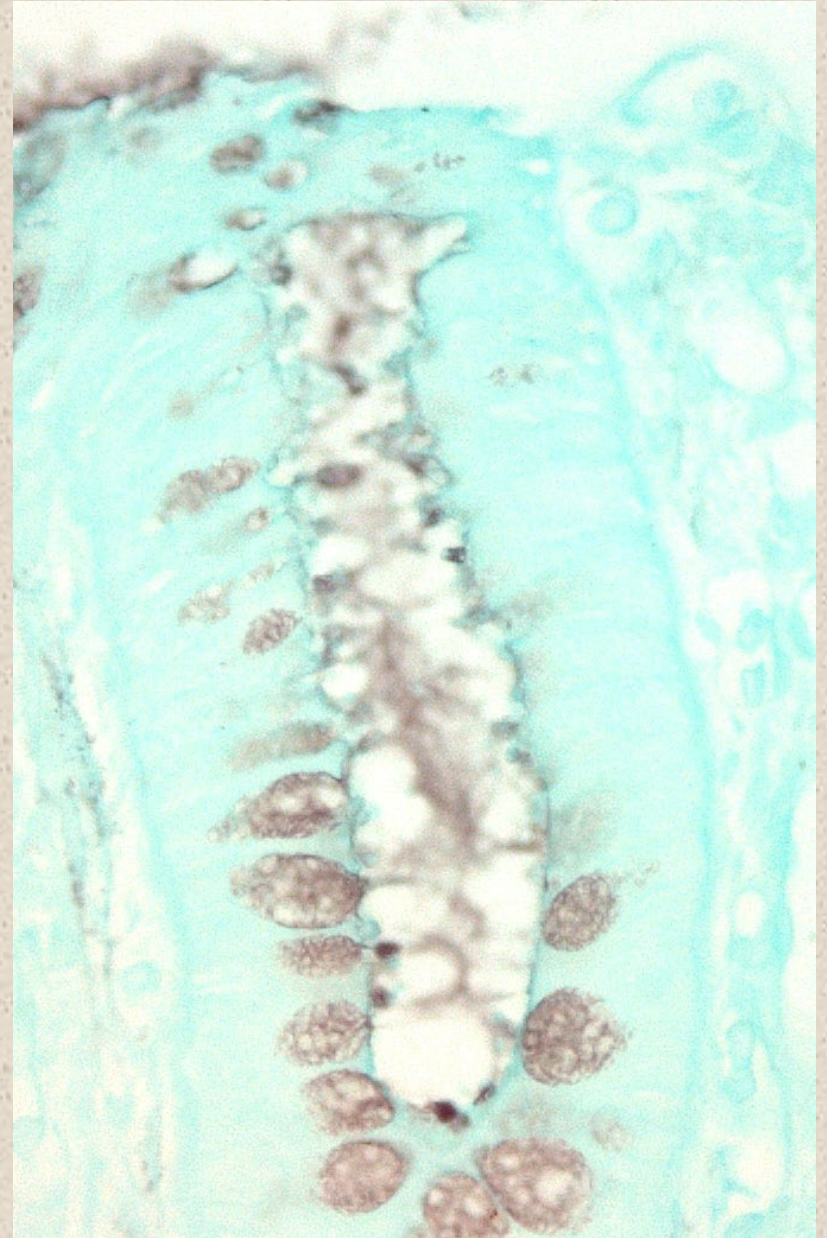
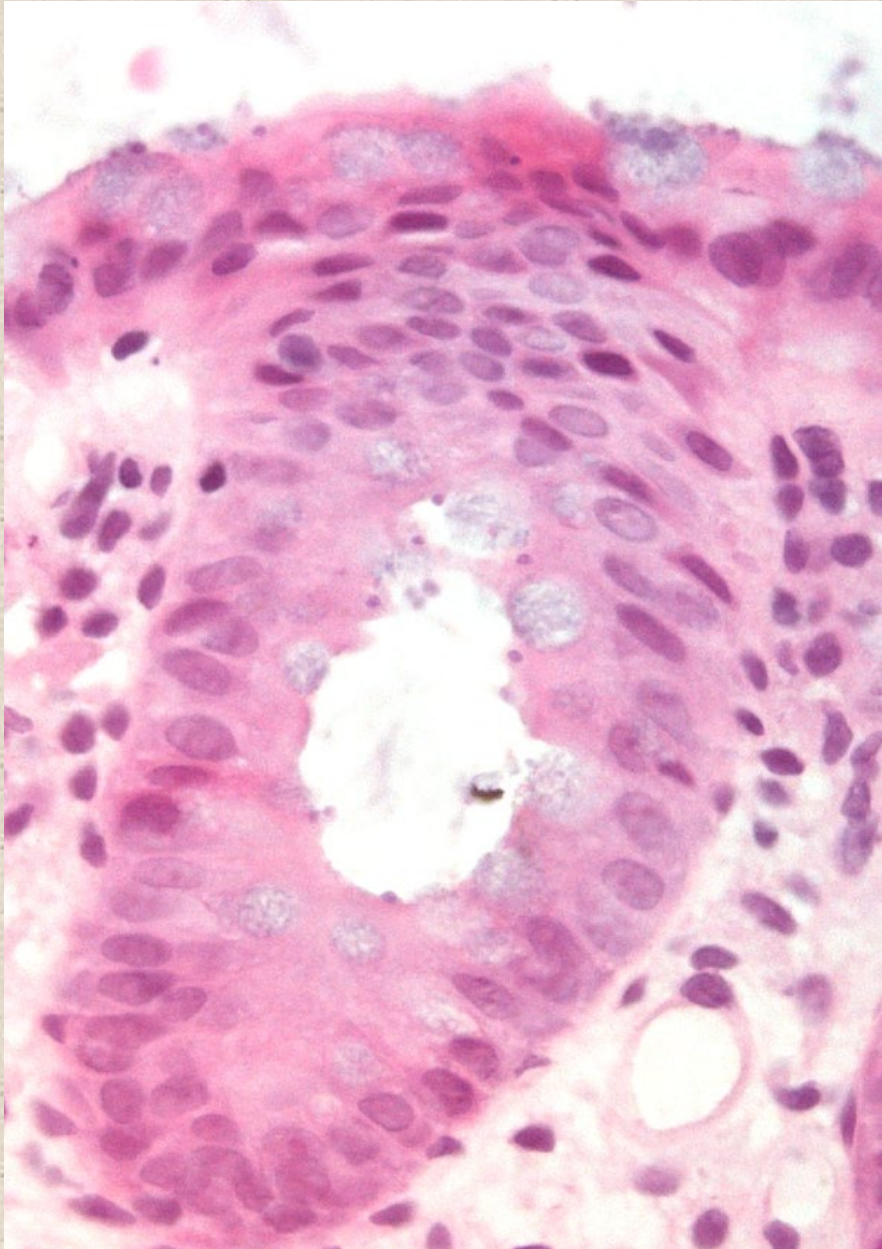


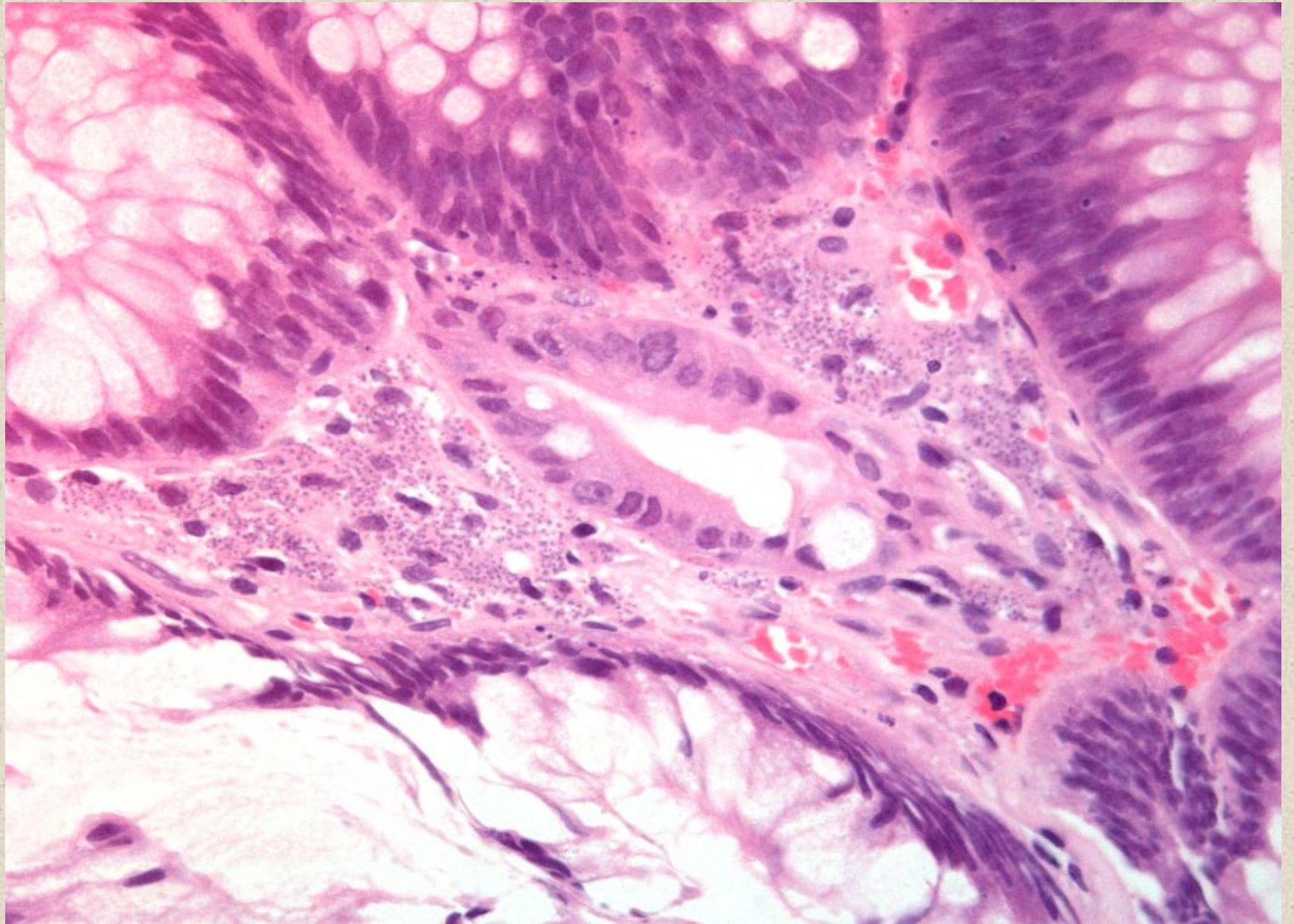


Amoebic
colitis

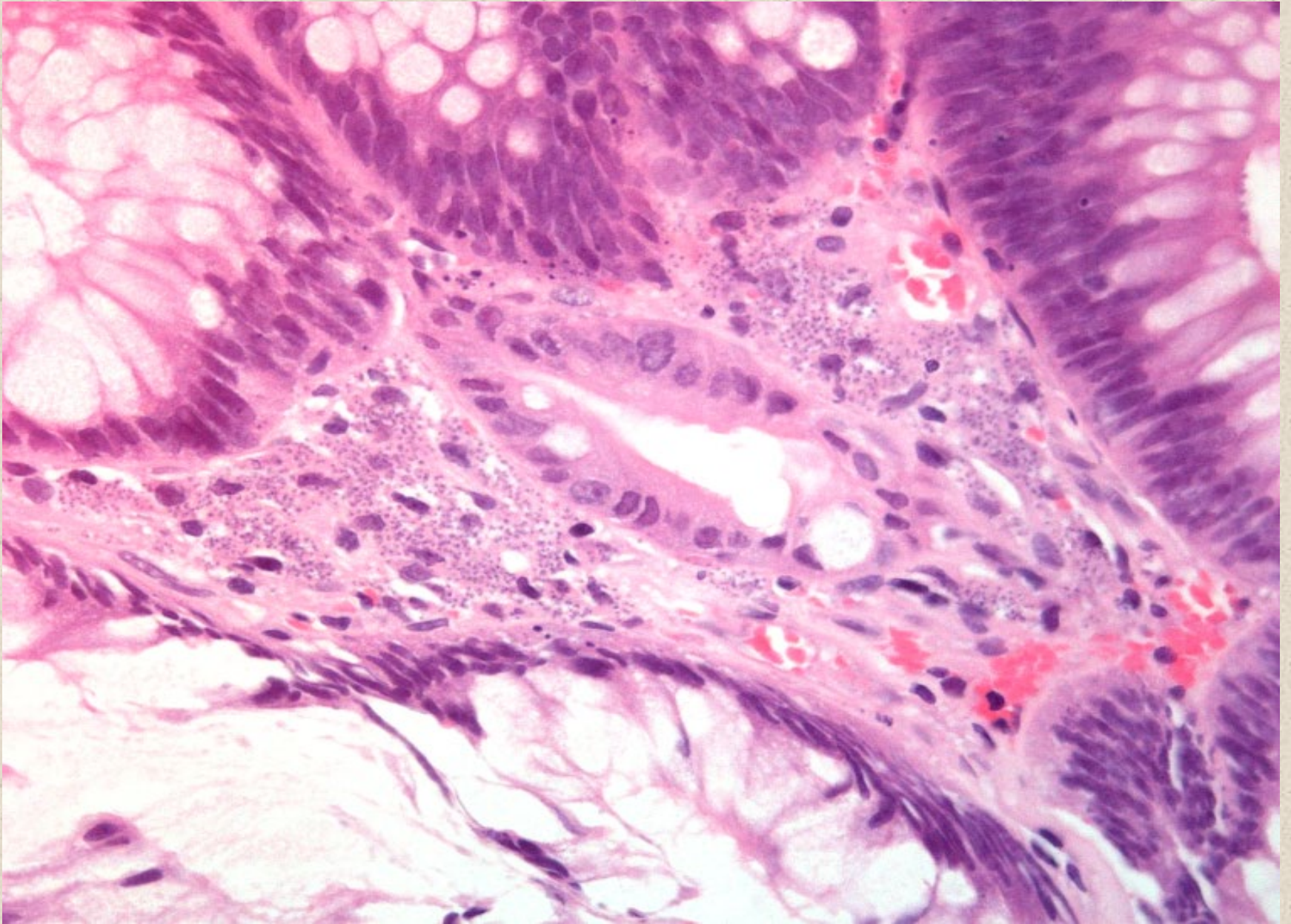


Cryptosporidia





Leishmania

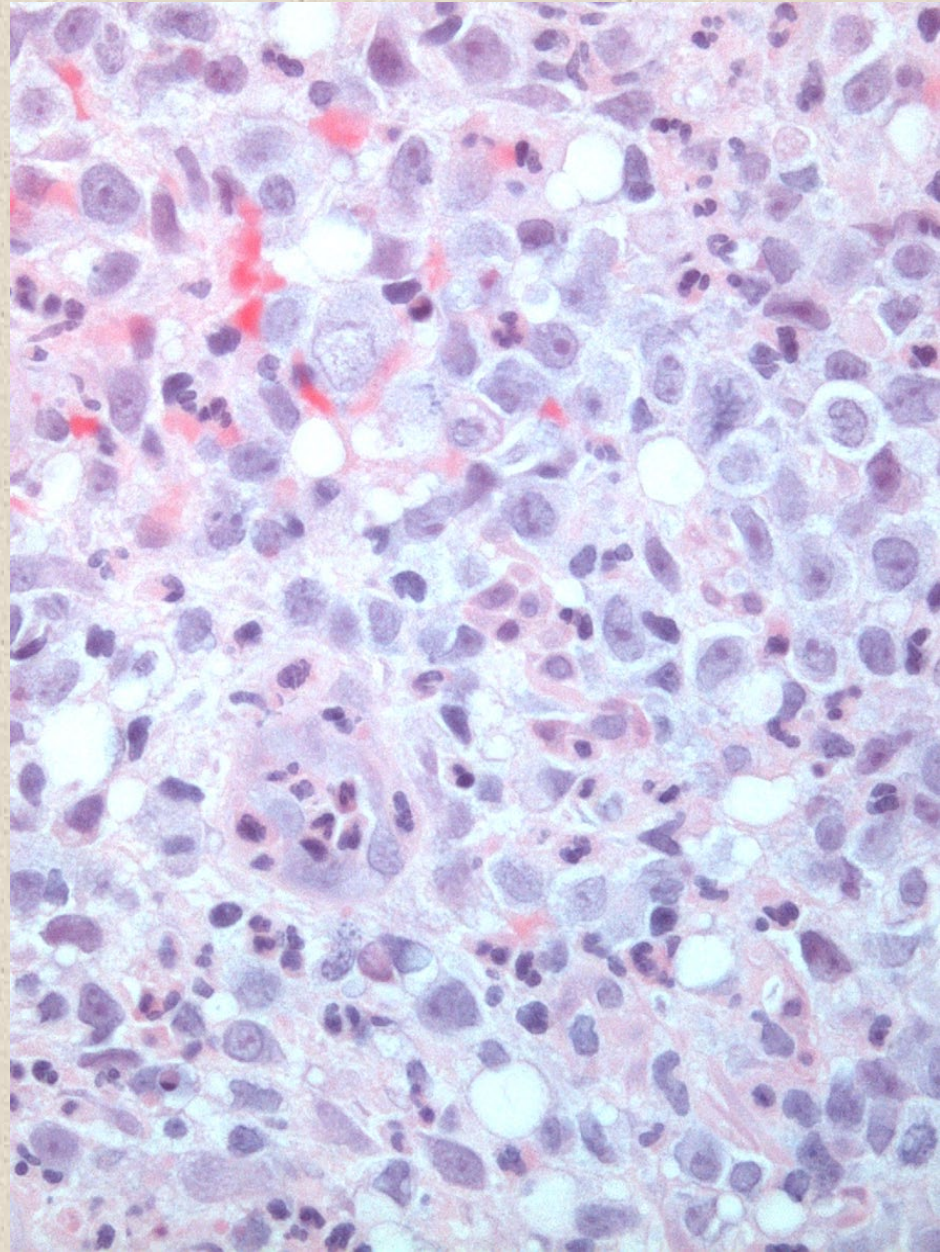
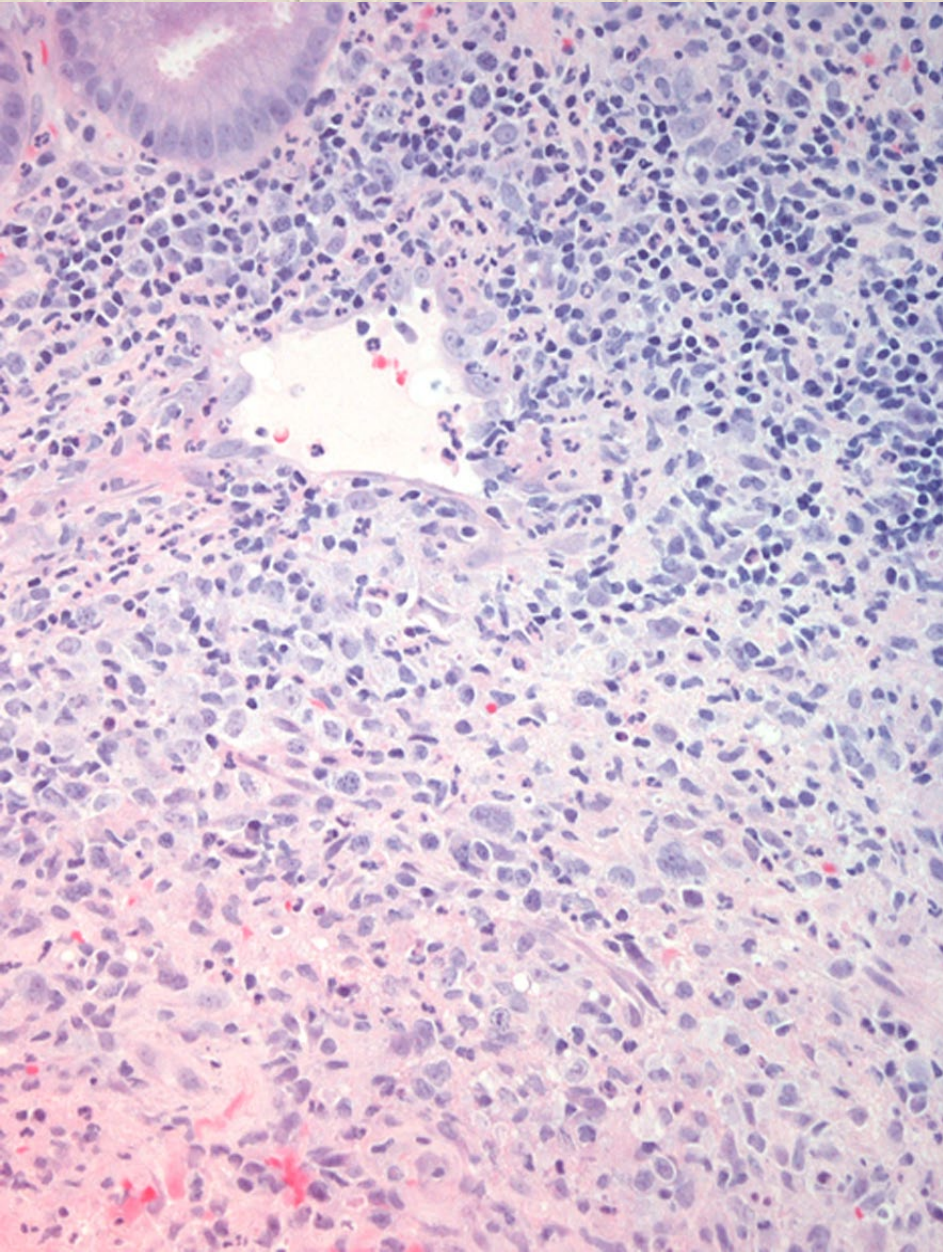


Neoplasia

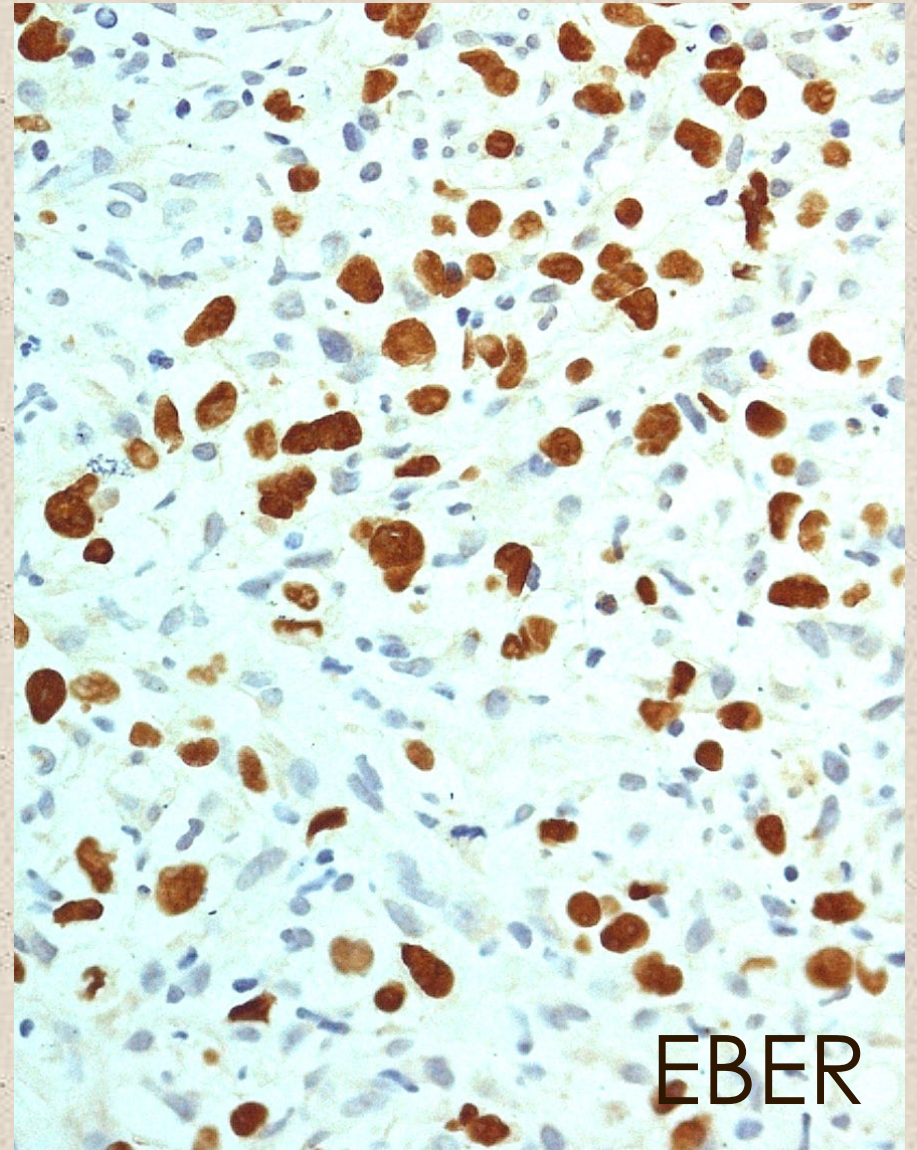
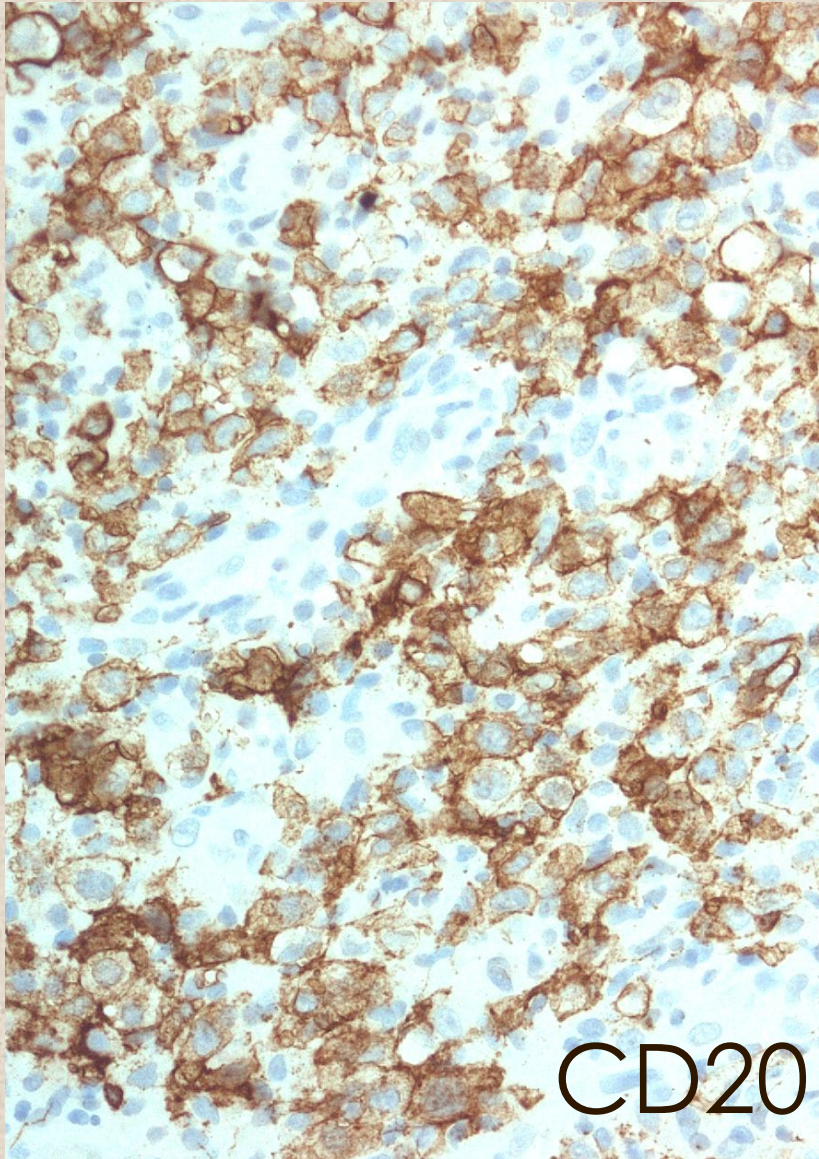
- Lymphoid
- Epithelial
- Mesenchymal

Epstein Barr virus &
Post-transplant
lymphoproliferative disease
(PTLD)

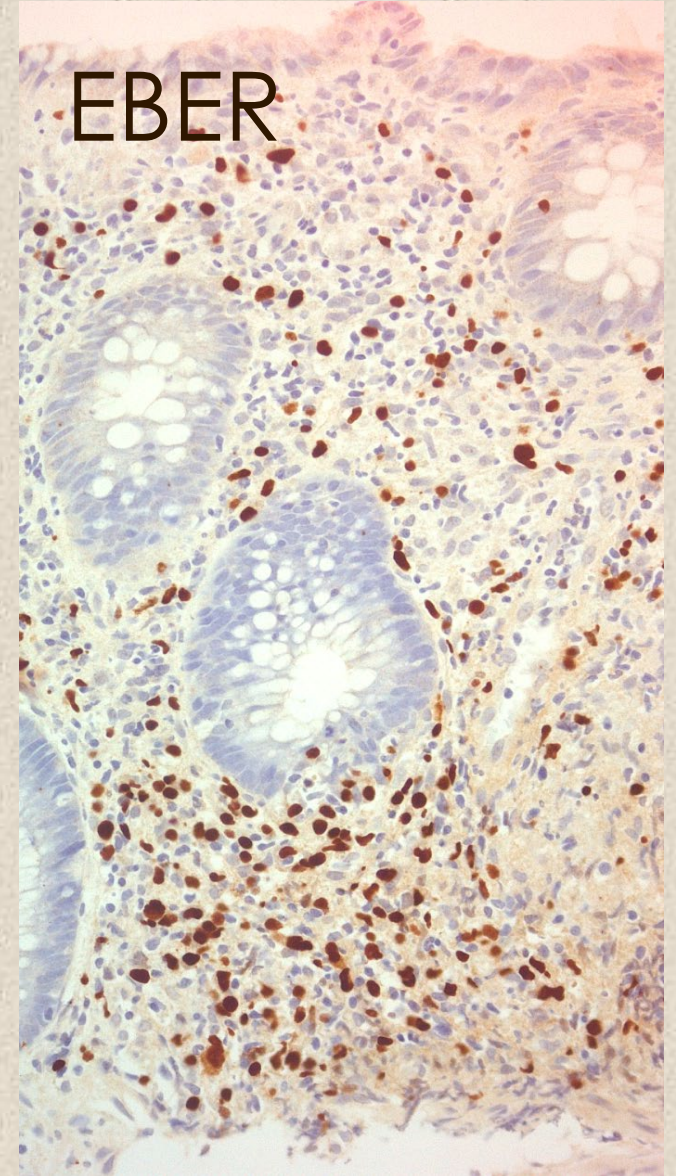
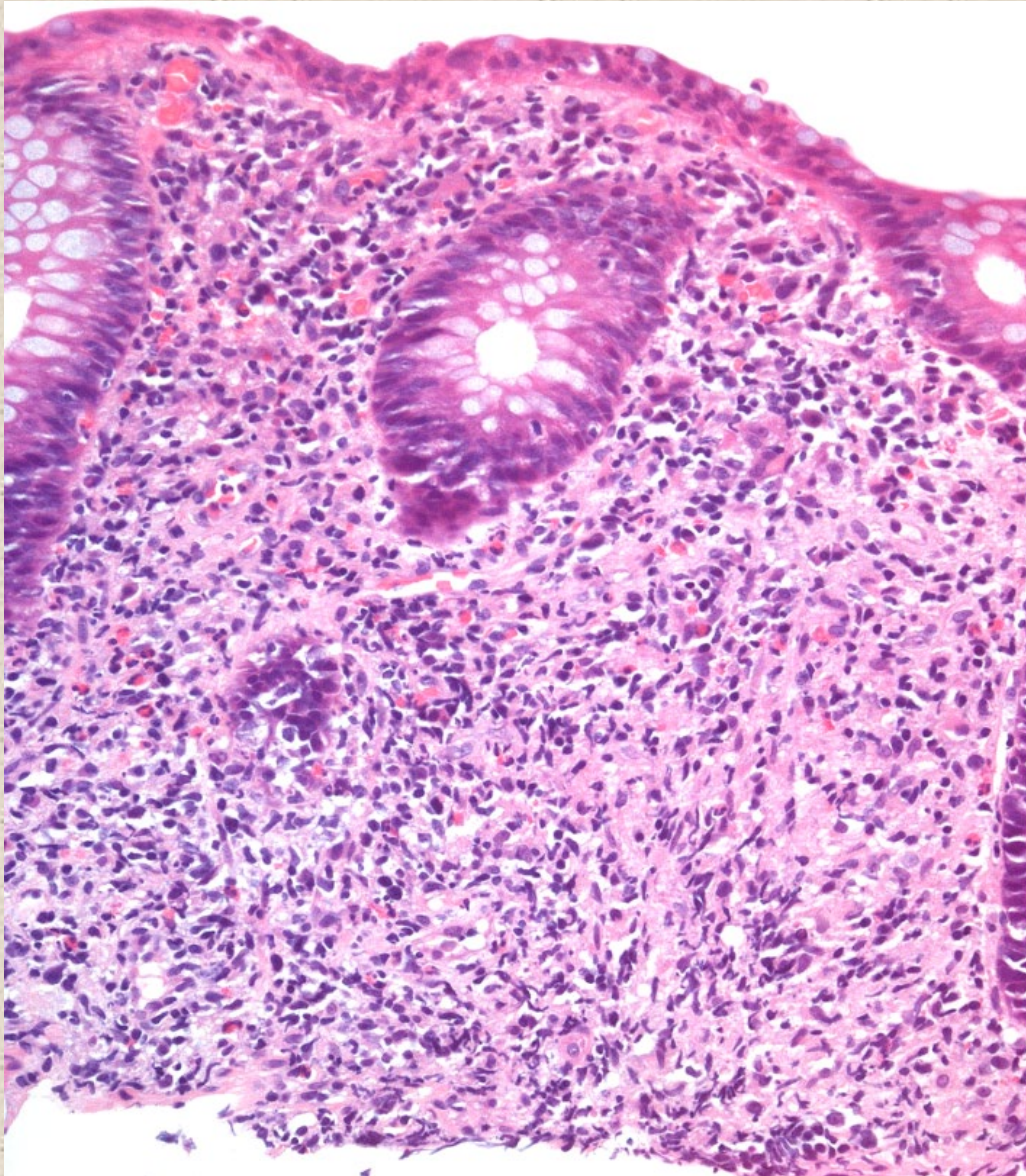
Monomorphic PTLD



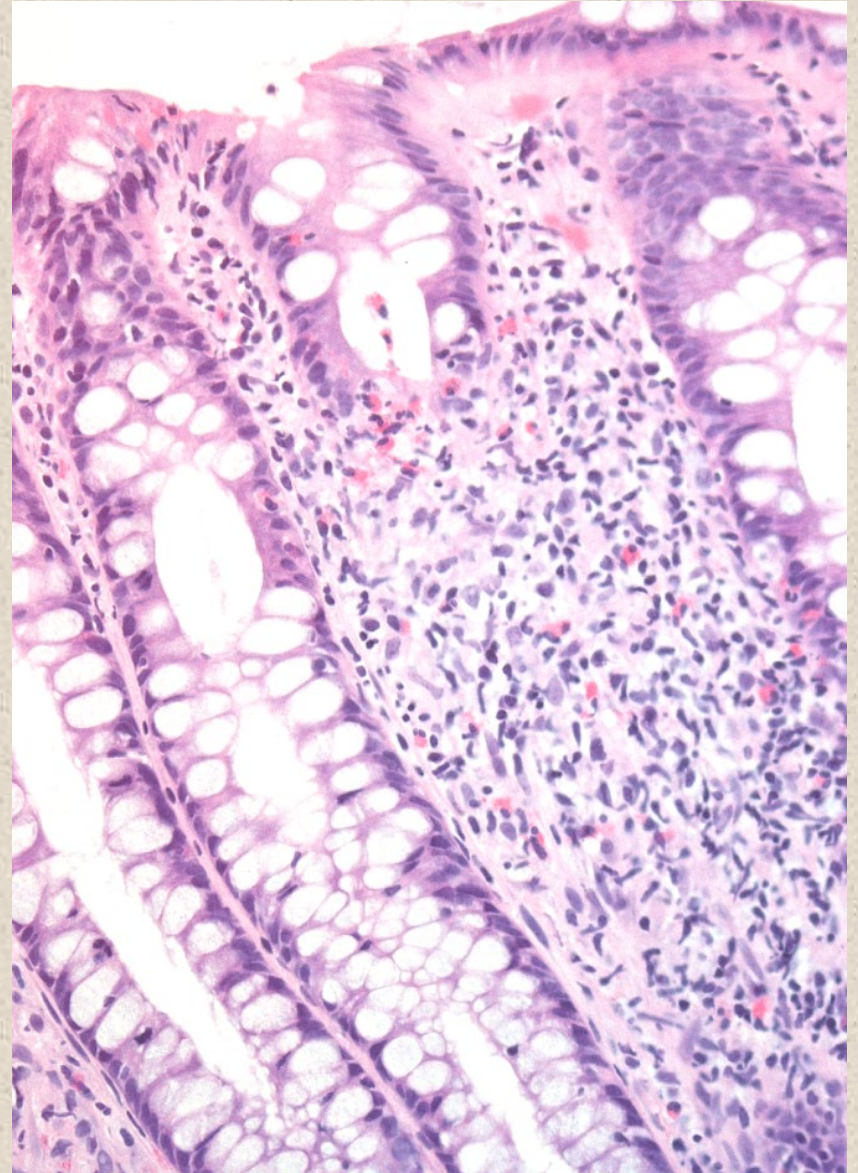
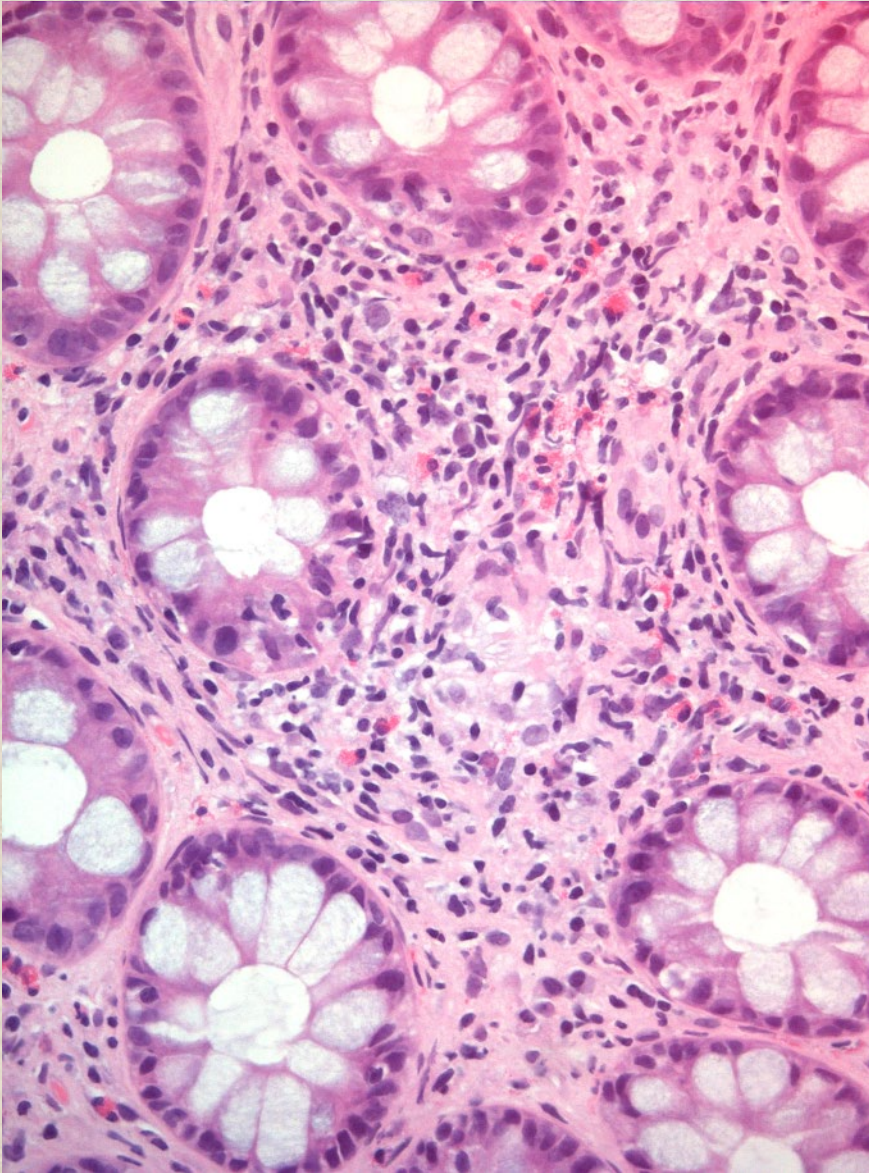
Monomorphic PTLD



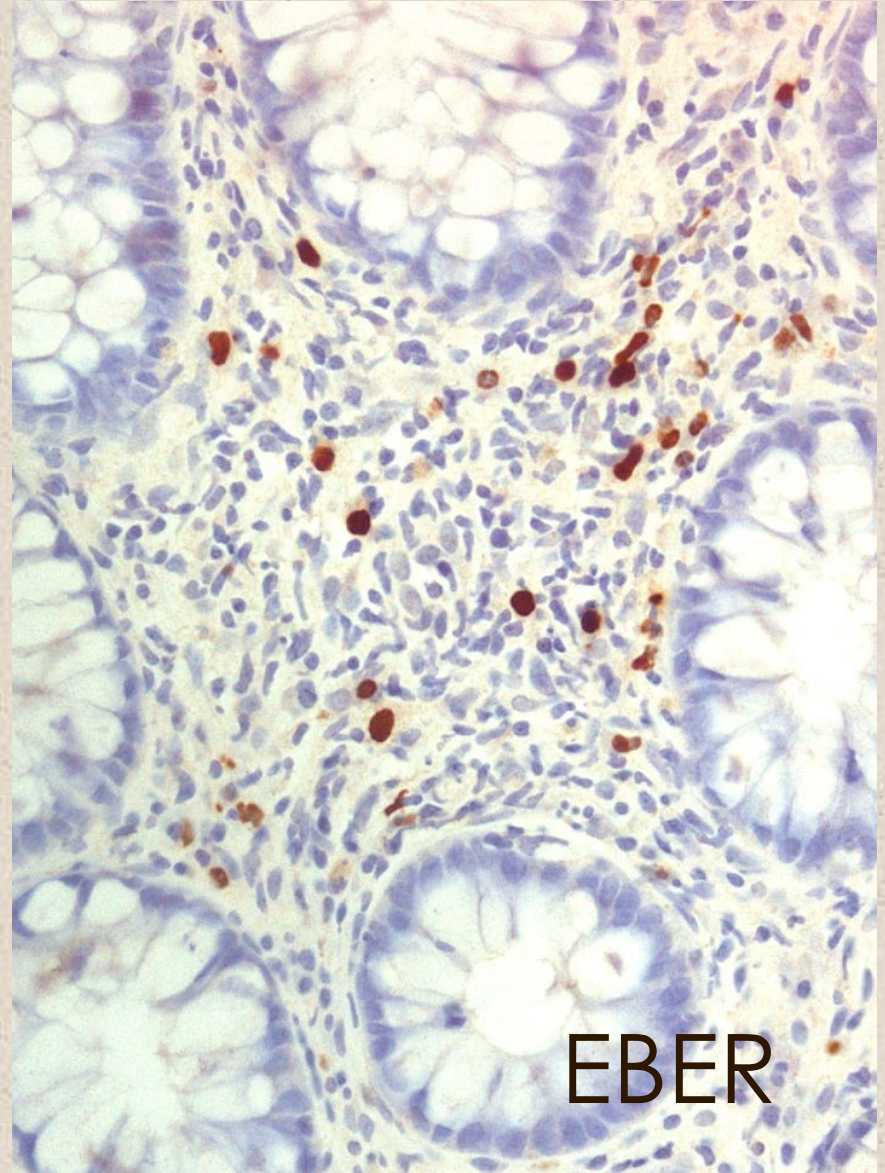
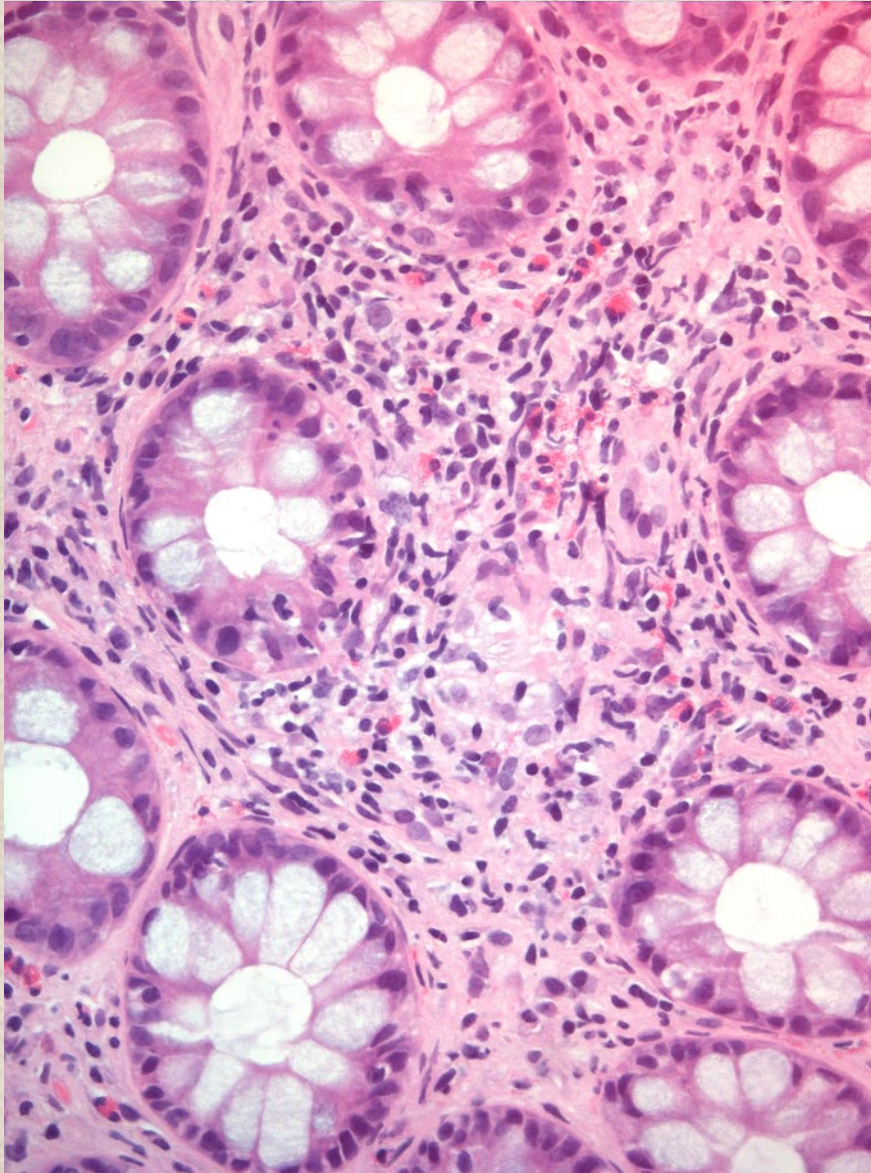
Polymorphic PTLD



Polymorphic PTLD

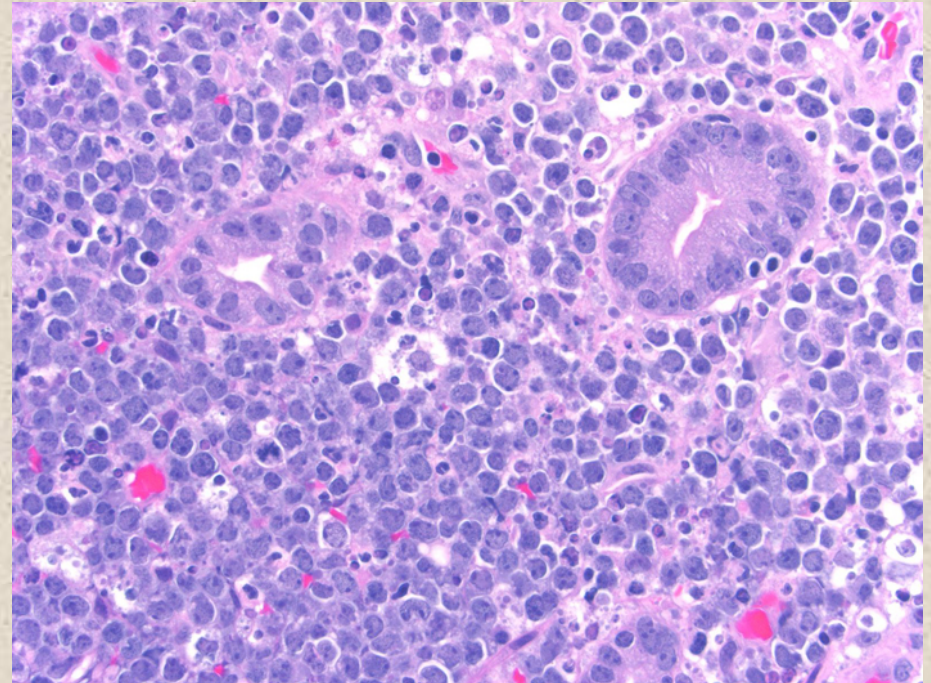
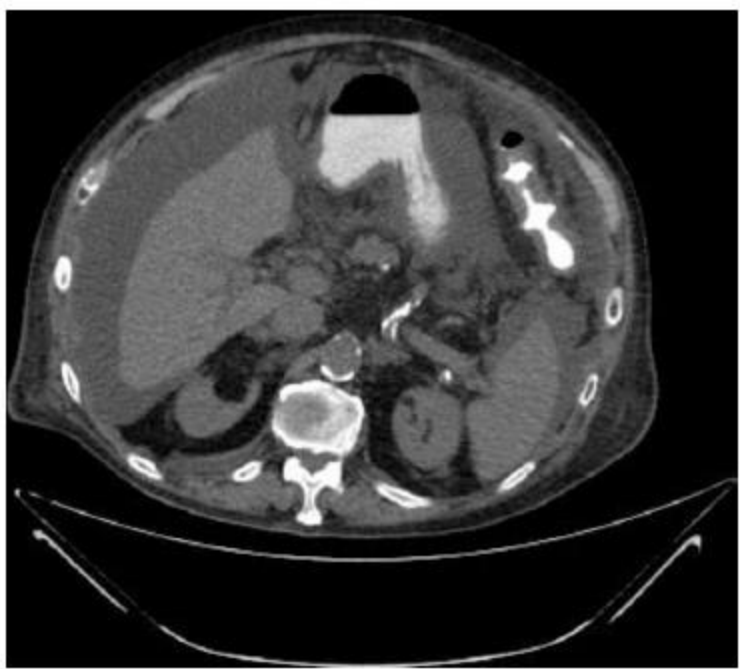


Polymorphic PTLD



- Lymphoid
 - Polymorphic PTLD can mimic Crohn's disease
 - Solid organ >>> bone marrow transplant patients

- Lymphoid
 - HIV and lymphomas (e.g. primary effusion lymphoma, Burkitt lymphoma)



Neoplasia

- Epithelial
 - Anal squamous dysplasia (AIN/SIL) and squamous cell carcinoma

- Mesenchymal
 - Kaposi sarcoma (can be CD117 but ?DOG1 negative)

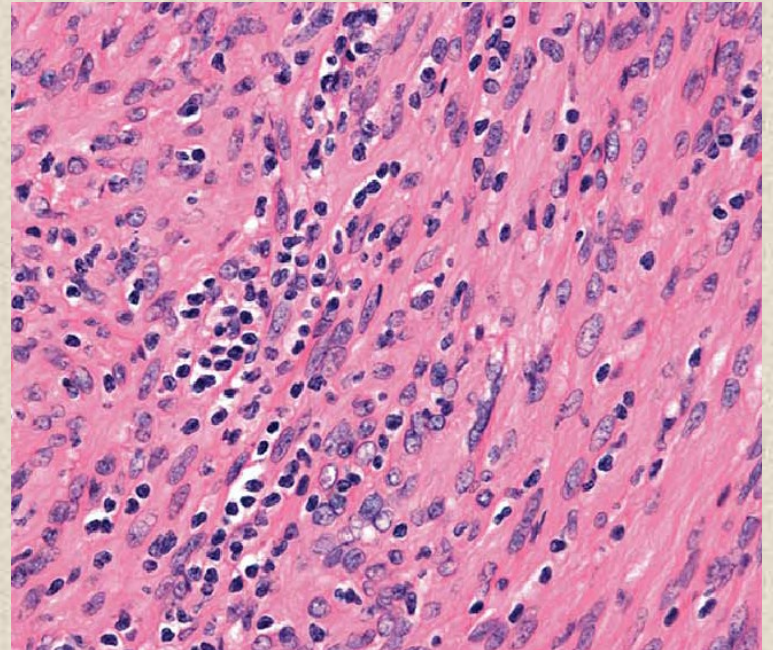
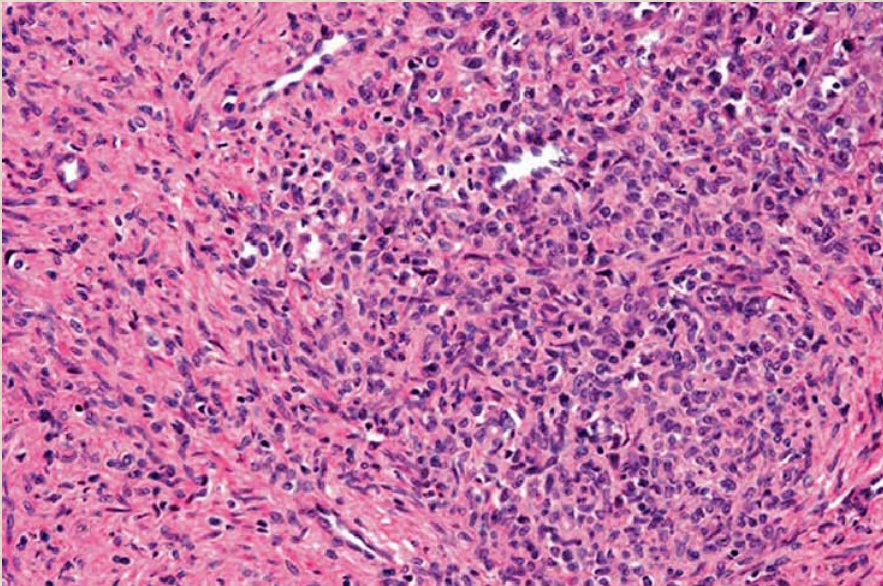
Is *DOG1* Immunoreactivity Specific to Gastrointestinal Stromal Tumor?

William Swalchick, MA, Rania Shamekh, MD, and Marilyn M. Bui, MD, PhD

Table 4. — IHC Negative for *DOG1* in Neoplastic Non-GISTs

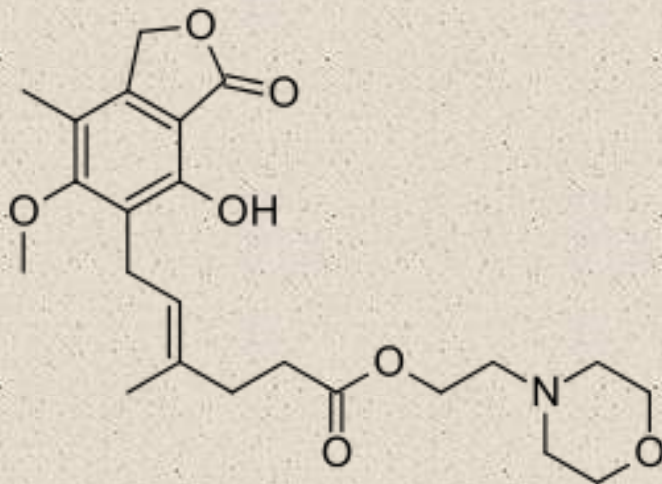
Wong ¹⁰	K9	Dedifferentiated liposarcoma	27
Lopes ¹¹	K9/DOG1.1	Desmoid tumor	59
Miettinen ¹²	K9	Desmoplastic small round cell tumor	16
Hemminger ¹⁴	K9	Endometrial stromal sarcoma	28
		Extraskeletal myxoid chondrosarcoma	16
		Gastrointestinal ganglioneuroma	3
		Inflammatory fibroid polyp	67
		Inflammatory myofibroblastic tumor	23
		Kaposi sarcoma	24

- In HIV/AIDS patients, EBV driven smooth muscle neoplasms:
 - less pleomorphism
 - low mitotic count



Drugs

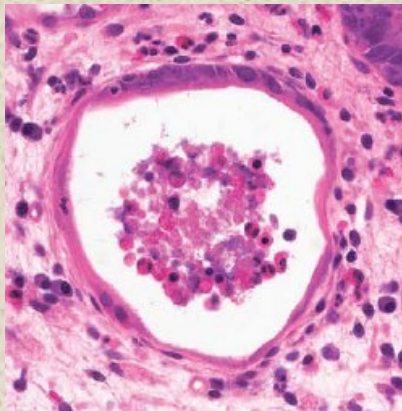
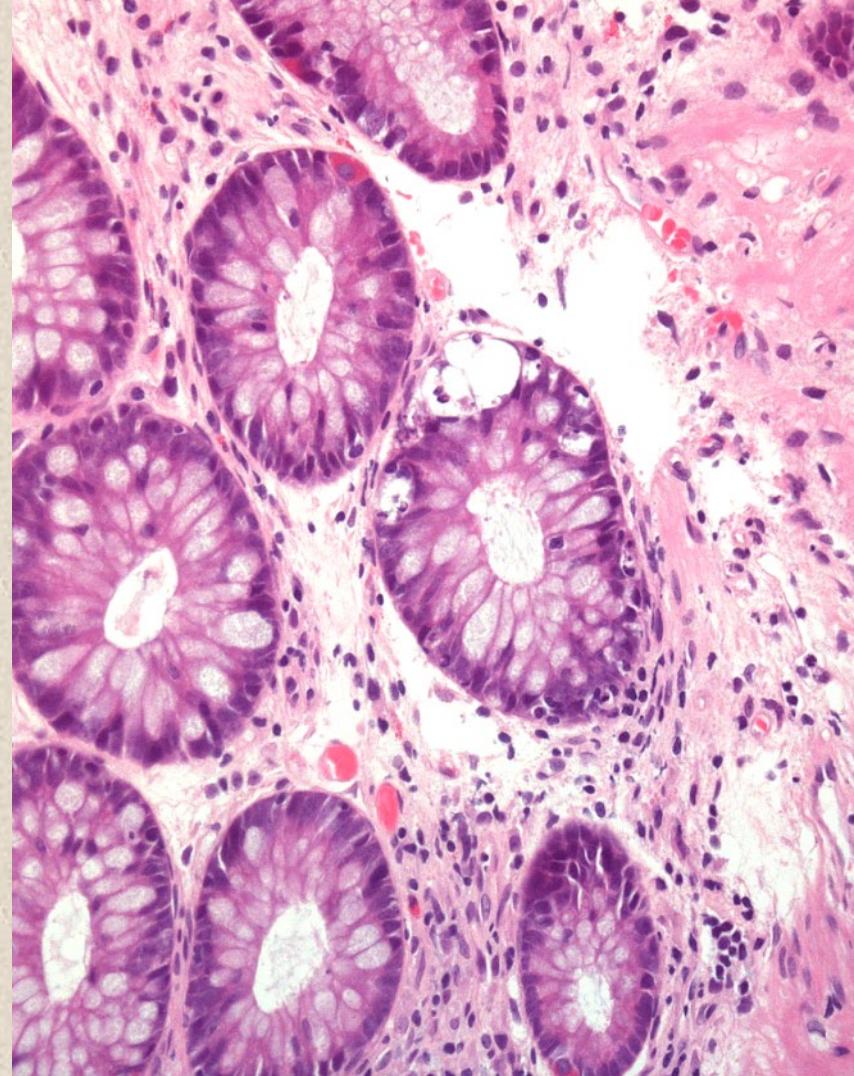
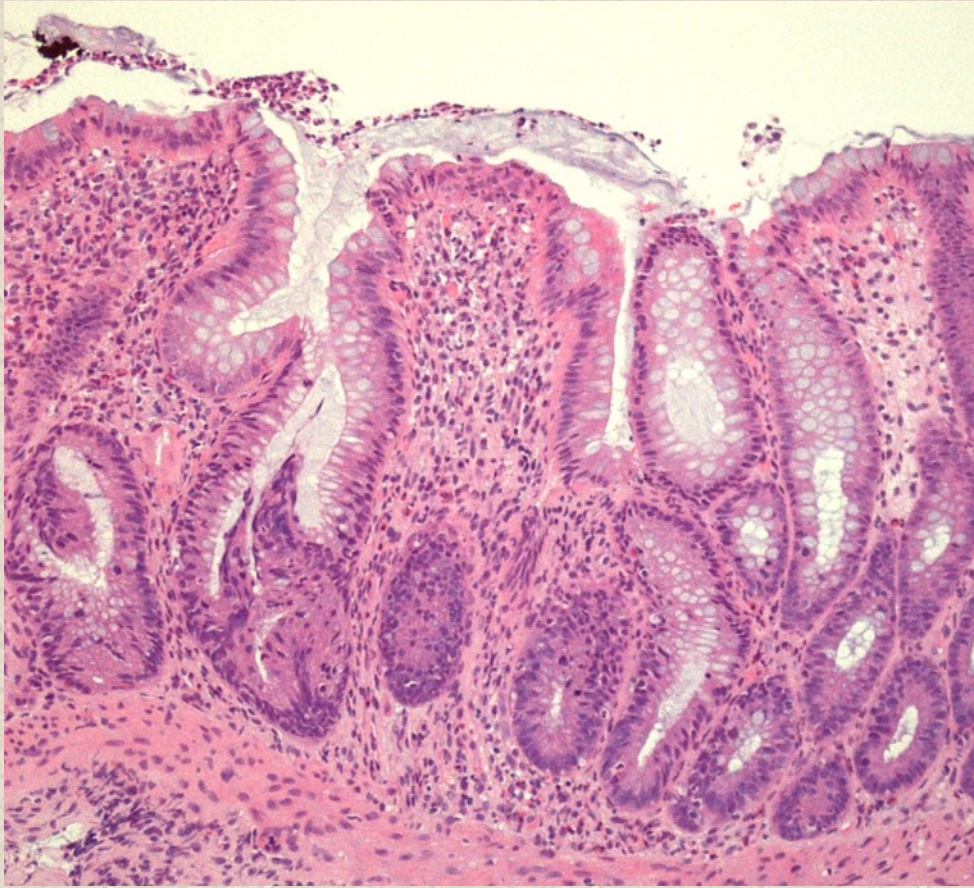
- Diarrhoea and:
 - Antiretrovirals
 - Cyclosporine
 - Tacrolimus
- Mycophenolate mofetil (MMF)



MMF

- Oesophagus to Colorectum
- Histological patterns
 - GvHD like
 - IBD like
 - Ischaemic like
 - Combinations
 - (Dilated damaged crypts)

MMF



Talk plan

- Diseases common to several immunosuppressed groups
 - Infection
 - Neoplasia
 - Drugs
- Diseases specific to certain immunosuppressed groups

Gastrointestinal GvHD

- BMT rather than solid organ transplant patients
- Hallmark histological feature is apoptosis
 - Proliferative compartment
 - ‘Exploding crypt cell’
 - Apoptotic microabscess (cluster of 5+ apoptotic bodies)



Gastrointestinal GvHD

- Other causes of apoptosis
 - Conditioning drugs (< Day 21)
 - Mycophenolate mofetil (MMF)
 - Viruses
- ?Paucity of inflammation
 - Acute inflammation can be seen
 - Granulomas?

Granulomas in GI GvHD?

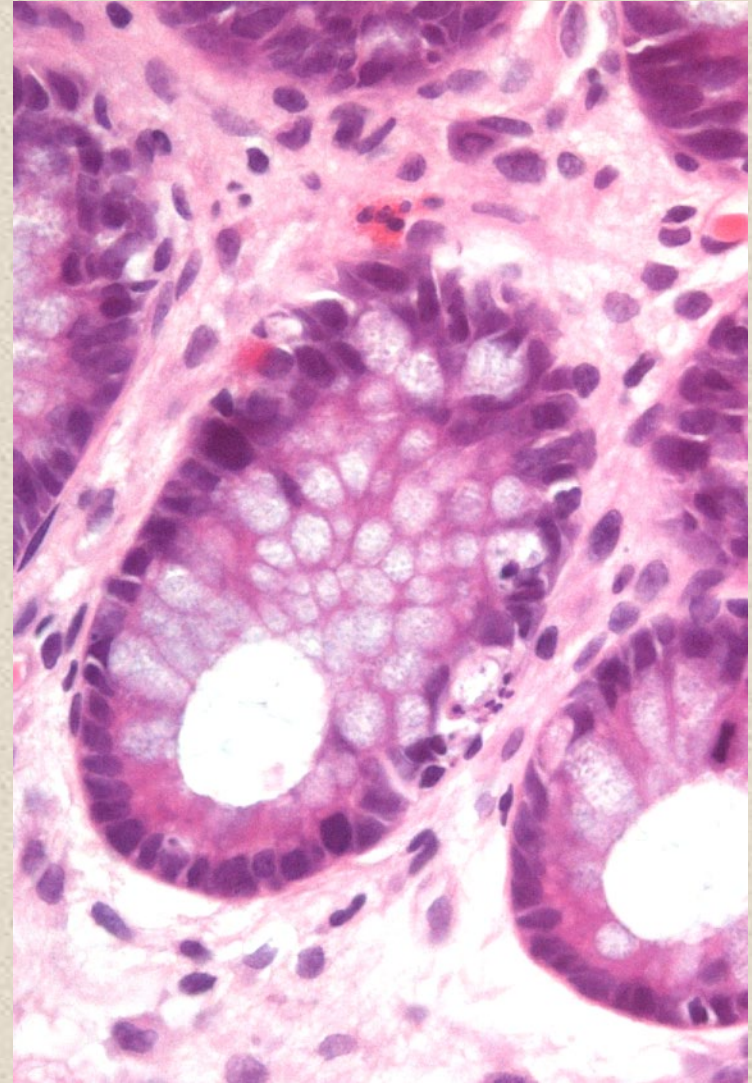
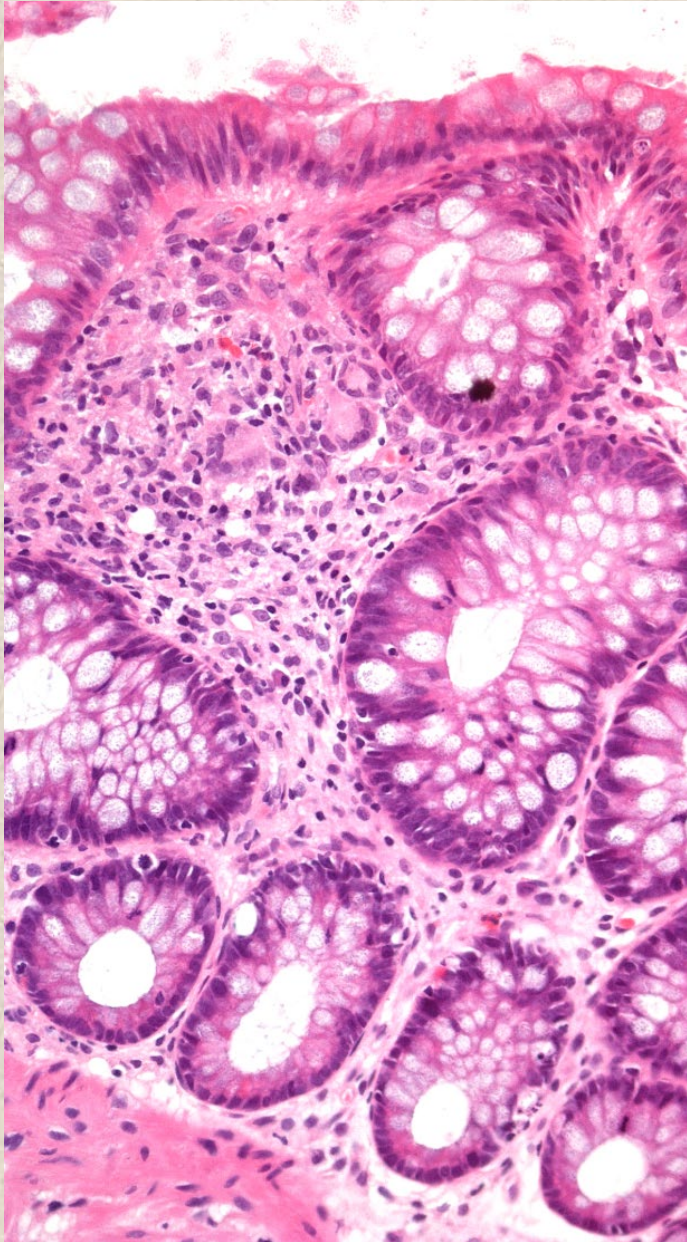


TABLE 1.

Definition and Histologic Grading System for Gut

Grade	Histological Features ^a
I	Crypt abscesses with necrotic or atypical epithelial cells, in vicinity of inflammatory infiltrate composed of lymphocytes, plasma cells, immunoblasts, and occasionally eosinophils (see Fig. 3)
II	Individual crypt loss (see Fig. 4)
III	Loss of two or more contiguous crypts (see Fig. 5)
IV	Total denudation of epithelium (usually followed rapidly by bacterial or fungal superinfection)

Lerner, K.G., Kao, G.F., Storb, R., Buckner, C.D., Clift, R.A., and Thomas, E.D.: Histopathology of graft-vs.-host reaction (GvHR) in human recipients of marrow from HL-A-matched sibling donors.

Transplant Proc 6: 367-371, 1974.

Is the Presence of 6 or Fewer Crypt Apoptotic Bodies Sufficient for Diagnosis of Graft Versus Host Disease? A Decade of Experience at a Single Institution

Jingmei Lin, MD, PhD, Rong Fan, MD, PhD, Zijin Zhao, MD, Oscar W. Cummings, MD, and Shaoxiong Chen, MD, PhD

Abstract: Histopathology assessment is crucial for the diagnosis of graft versus host disease (GVHD), as the presence of crypt apoptosis is the cardinal criterion required. However, crypt apoptosis is not limited to GVHD; it also occurs in other conditions such as infection, drug reaction, or inflammatory reactions unrelated to GVHD. To better determine whether the presence of 6 or fewer apoptotic bodies is sufficient for the diagnosis of GVHD, we retrospectively reviewed 78 colon biopsies from 66 patients who received either hematopoietic stem cell (HSCT) or cord blood cell transplantation and whose colon biopsies exhibited apoptotic bodies. Among them, 41 cases contained 6 or fewer apoptotic bodies in the colon biopsy. These biopsies were compared with 141 colon biopsy controls that showed no significant pathologic changes as well as 16 colon biopsies with cytomegalovirus colitis from patients without a history of bone marrow transplantation. Among the 41 cases reviewed, 7 patients had coexisting GVHD in other organs (skin or liver). However, gastrointestinal symptoms of at least 4 HSCT patients whose colon biopsies contained 6 or fewer

Key Words: graft versus host disease, colon, cytomegalovirus colitis, apoptosis, transplantation

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Transplantation, either hematopoietic stem cell (HSCT) or cord blood cell, is a potentially curative therapy in the management of immunohematopoietic disorders. However, the efficacy of this treatment is greatly impaired by graft versus host disease (GVHD), a donor-derived immune response against recipient antigens, resulting in significant morbidity and mortality.¹ The gastrointestinal (GI) tract is one of the main target organs involved by GVHD. Unfortunately, GI GVHD may present with a variety of nonspecific symptoms; diagnosis based on clinical presentation alone is not reliable or specific.² Gross endoscopic discordance with histologic findings exists^{3,4}; therefore histopathology assessment is crucial to make a diagnosis of GVHD.

Control grp: Non-BMT patients investigated for GI symptoms or being followed up for colorectal polyps

Strikingly, the presence of a single or rare crypt apoptosis was seen in 21.3% (30 of 141) of the normal controls with a mean of 0.2 per 10 contiguous crypts and a maximum count of up to 5 per 10 contiguous crypts.

TABLE 5. Suggested Modified Histopathologic Criteria for Grading GVHD in Colon Biopsies

Indeterminate for GVHD

Rare crypt apoptosis (≤ 6 apoptotic bodies per 10 contiguous crypts)

Grade 1 GVHD

Increased crypt apoptosis without crypt/glands loss (≥ 7 apoptotic bodies per 10 contiguous crypts)

Grade 2 GVHD

Loss of individual crypt/gland with crypt apoptosis

Grade 3 GVHD

Loss of 2 or more contiguous crypts/glands with crypt apoptosis

Grade 4 GVHD

Extensive crypt loss with mucosal denudation or ulceration with crypt apoptosis

When assessing transplant patient GI biopsies

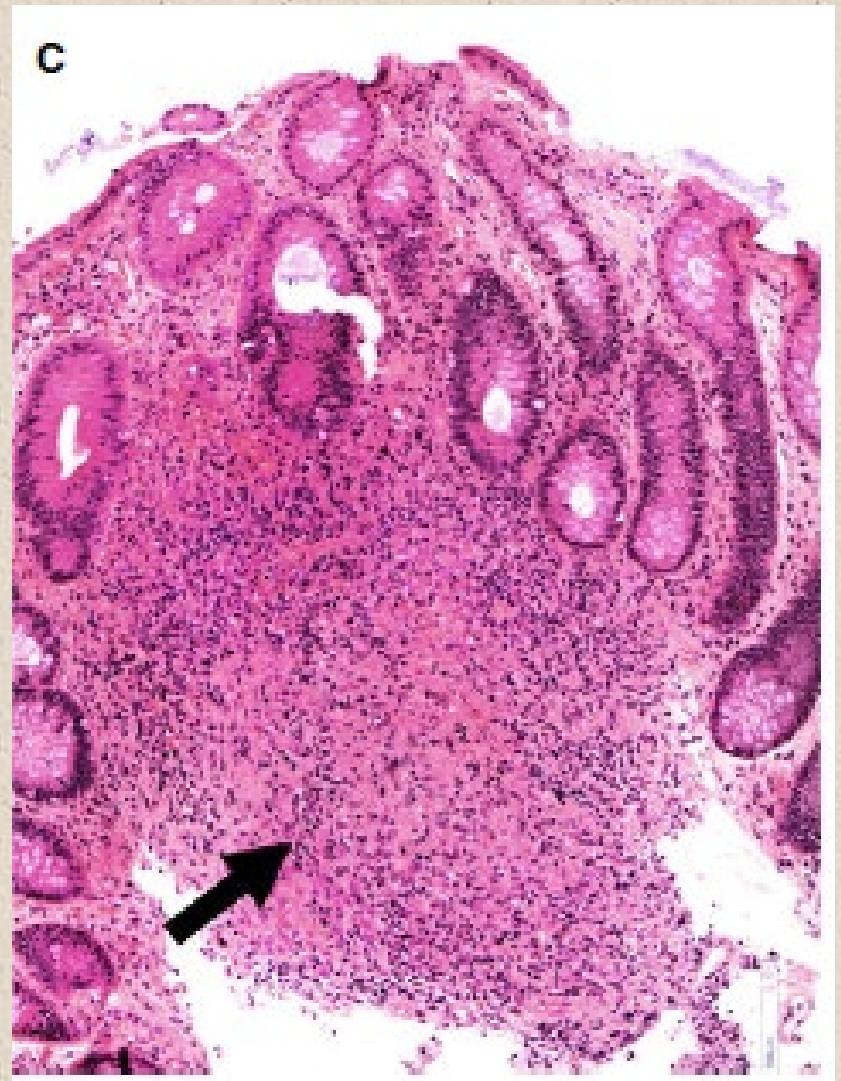
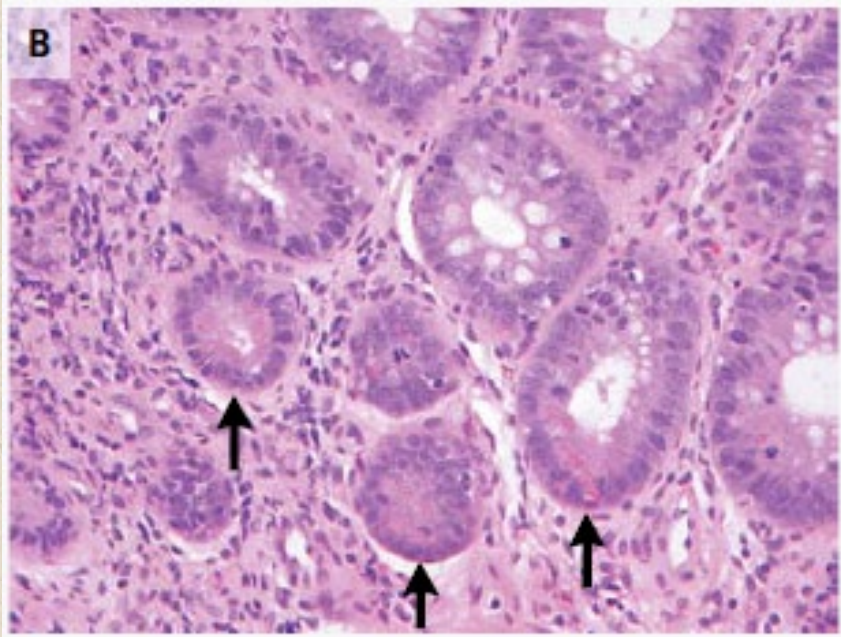
- Time from transplant?
- Transplant type?
- Underlying disease?
- GvHD elsewhere?
- MMF therapy?
- CMV, Adenovirus and EBV titres?

ORIGINAL ARTICLE

Cord Colitis Syndrome in Cord-Blood Stem-Cell Transplantation

Alex F. Herrera, M.D., Gabriela Soriano, M.D., Andrew M. Bellizzi, M.D., Jason L. Hornick, M.D., Ph.D., Vincent T. Ho, M.D., Karen K. Ballen, M.D., Lindsey R. Baden, M.D., Corey S. Cutler, M.D., M.P.H., Joseph H. Antin, M.D., Robert J. Soiffer, M.D., and Francisco M. Marty, M.D.

N Engl J Med 2011;365:815-24.



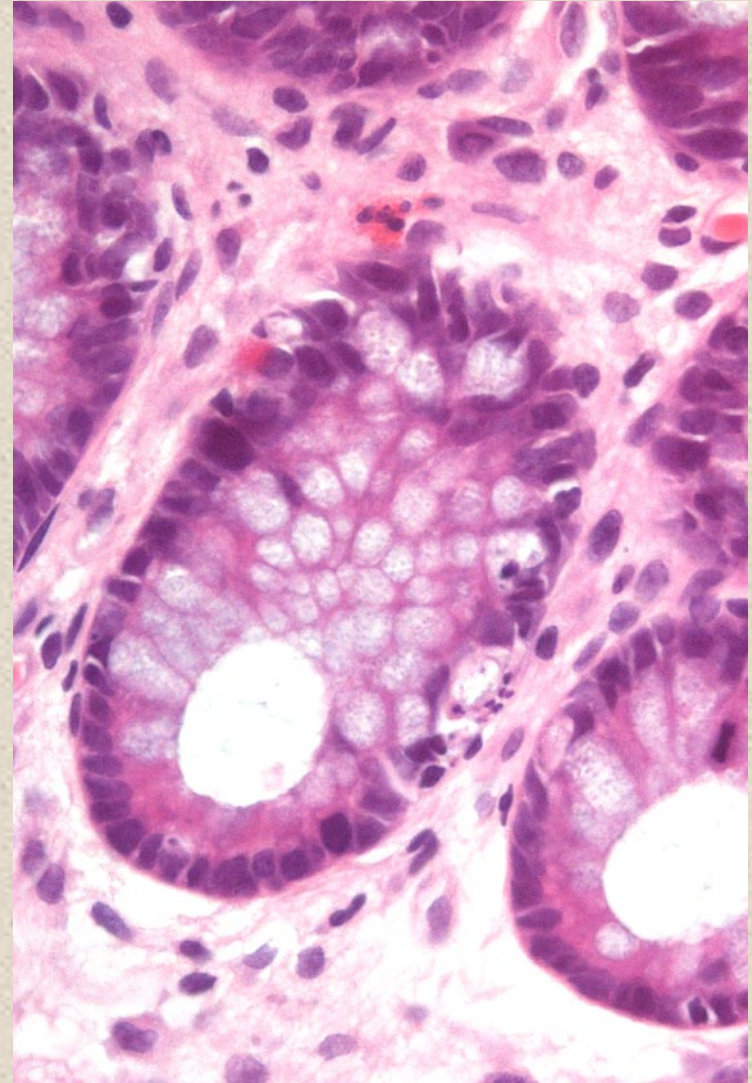
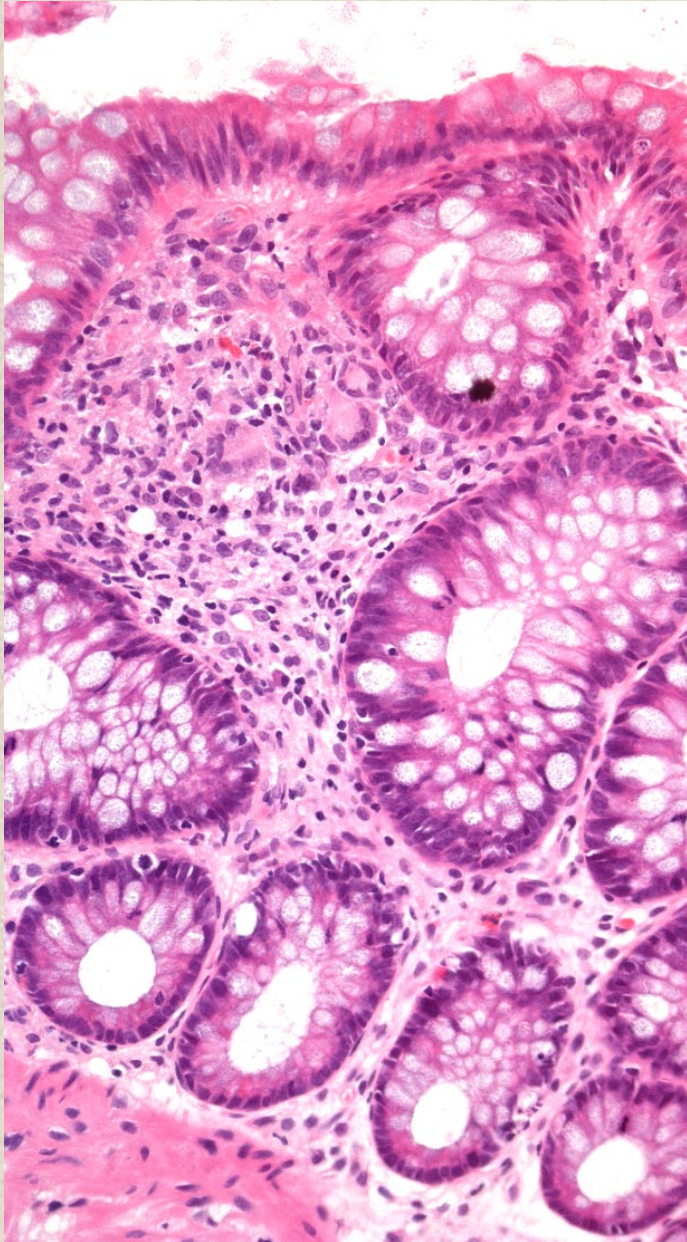
ORIGINAL ARTICLE

Sequence-Based Discovery of *Bradyrhizobium enterica* in Cord Colitis Syndrome

Ami S. Bhatt, M.D., Ph.D., Samuel S. Freeman, B.S.E., Alex F. Herrera, M.D.,
Chandra Sekhar Pedamallu, Ph.D., Dirk Gevers, Ph.D., Fujiko Duke, B.S.,
Joonil Jung, Ph.D., Monia Michaud, M.Sc., Bruce J. Walker, B.S., Sarah Young, Ph.D.,
Ashlee M. Earl, Ph.D., Aleksander D. Kostic, Ph.D., Akinyemi I. Ojesina, M.D., Ph.D.,
Robert Hasserjian, M.D., Karen K. Ballen, M.D., Yi-Bin Chen, M.D.,
Gabriela Hobbs, M.D., Joseph H. Antin, M.D., Robert J. Soiffer, M.D.,
Lindsey R. Baden, M.D., Wendy S. Garrett, M.D., Ph.D., Jason L. Hornick, M.D., Ph.D.,
Francisco M. Marty, M.D., and Matthew Meyerson, M.D., Ph.D.

N Engl J Med 2013;369:517-28.

Granulomas in GI GvHD?



ORIGINAL ARTICLE

Evaluating the association between histological manifestations of cord colitis syndrome with GVHD

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Cord colitis syndrome (CCS) is a recently proposed clinical entity characterized by a persistent diarrheal illness after cord blood transplantation (CBT), which is not caused by GVHD or CMV colitis. CCS is histologically characterized by chronic active colitis with granulomatous inflammation and Paneth cell metaplasia suggesting chronicity. However, the specificity of these pathological features to CCS remains to be validated. We conducted a retrospective study of 49 patients who had diarrhea and underwent diagnostic colonoscopy with biopsy following allogeneic hematopoietic SCT. None of the patients met the clinical criteria for CCS. Chronic active colitis with granulomatous inflammation and Paneth cell metaplasia was present in 12/33 (36%) patients with biopsy-proven GVHD, 4/6 (67%) patients with CMV colitis and 2/15 (13%) patients with nonspecific colitis. In patients with GVHD and/or CMV colitis, these pathological features were present in 4/8 (50%) patients after CBT and in 11/26 (42%) patients undergoing BMT or PBSCT. These results demonstrate that chronic active colitis with granuloma and Paneth cell metaplasia is not only a specific feature of CCS but also is present in GVHD and CMV colitis, irrespective of stem cell source.

Late-Onset Colitis after Cord Blood Transplantation Is Consistent with Graft-Versus-Host Disease: Results of a Blinded Histopathological Review



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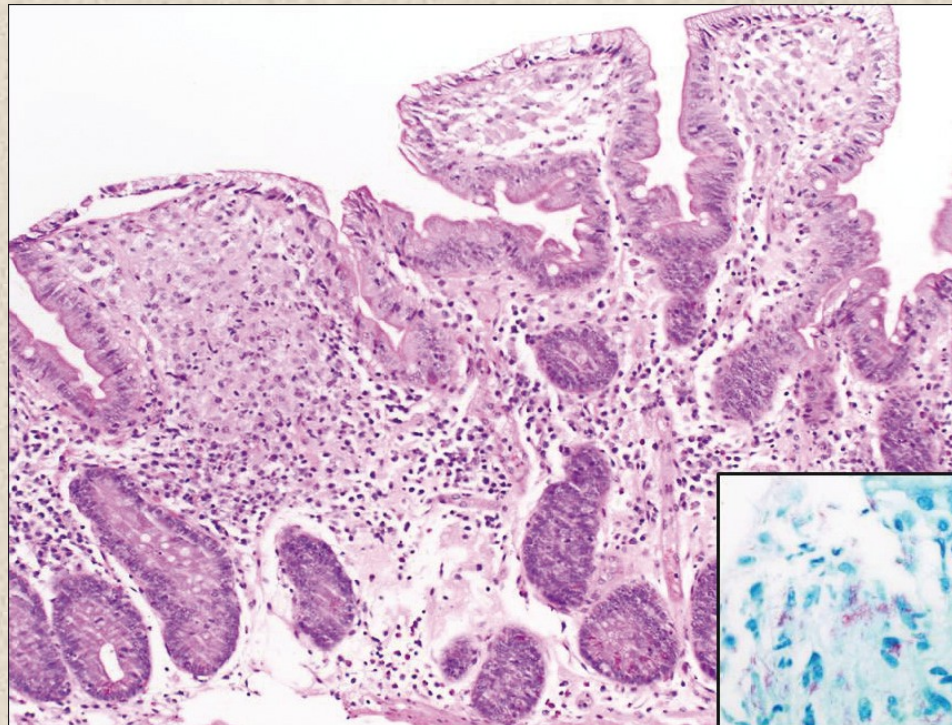
ABSTRACT

Cord colitis syndrome after umbilical cord blood transplantation (UCBT) involves late-onset diarrhea, absence of infection or GVHD, chronic active colitis, and granulomatous inflammation that responds to antibiotics. We tested the hypothesis that Seattle recipients of UCBT had late-occurring colitis distinct from GVHD and colitis in other allograft recipients. We conducted a blinded histological review of 153 colon biopsy specimens from 45 UCBT recipients and 45 matched allografted controls obtained between day +70 and day +365 post-transplantation. Diarrhea was the primary indication for biopsy in 10 UCBT recipients and 11 controls. No histological differences were seen between UCBT recipients and controls with diarrhea or between the entire cohort of UCBT recipients and their controls. Distorted mucosal architecture and apoptotic crypt cells typical of GVHD were common in both groups; Paneth cell metaplasia and granulomas were rare findings. Chronic active colitis was present in 58% of the UCBT recipients and in 62% of controls. No UCBT recipient with diarrhea was treated with antibiotics, and all recipients responded to systemic corticosteroids. Colitis occurring after day +70 in allografted controls was related to acute GVHD, independent of the source of donor cells. We could not identify a histologically distinct cord colitis syndrome in either the UCBT or the non-cord blood allograft recipients.

- Is cord colitis syndrome a real entity?
 - Watch this space but ...

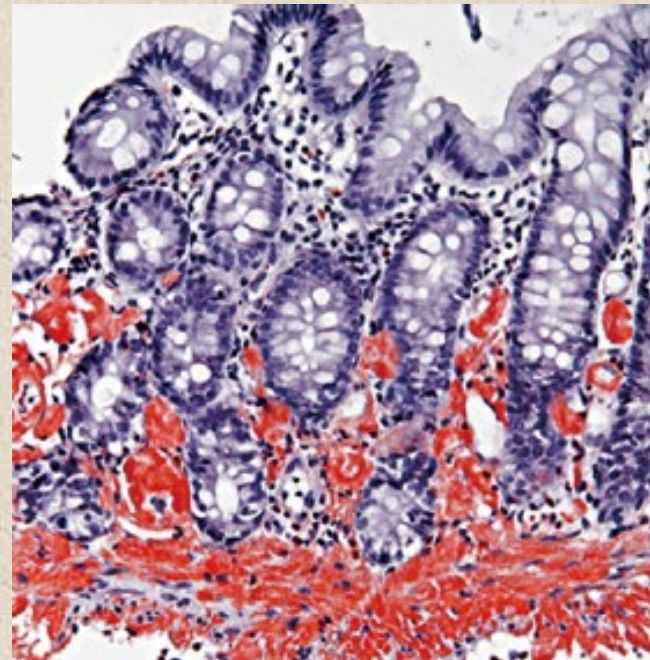
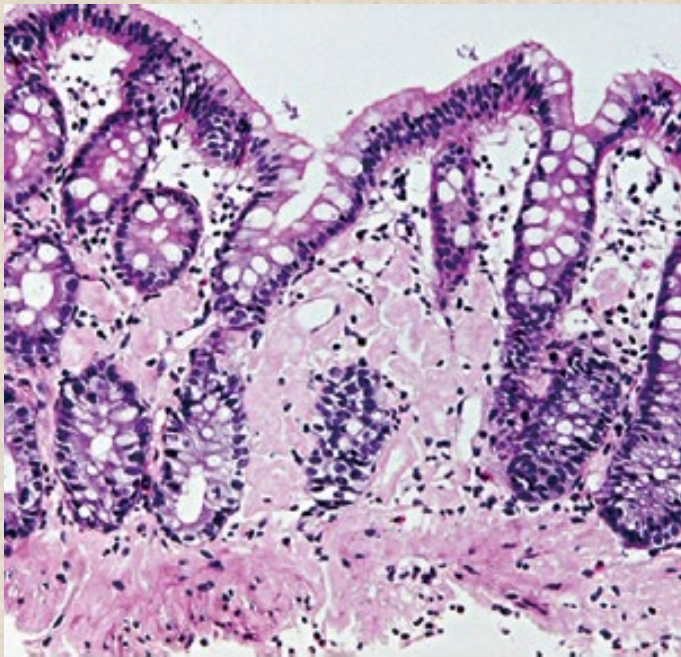
Underlying condition requiring transplant

- BMT transplant
 - Lymphoma/leukaemia



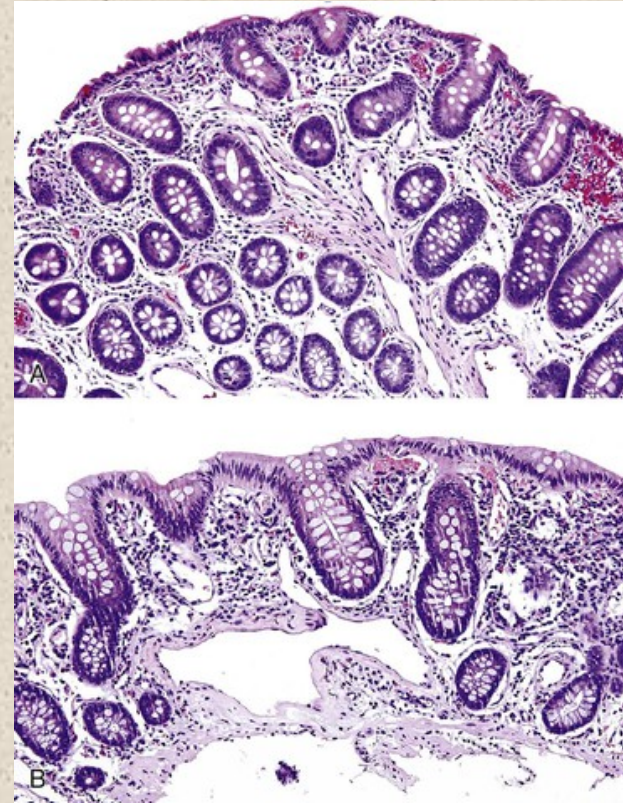
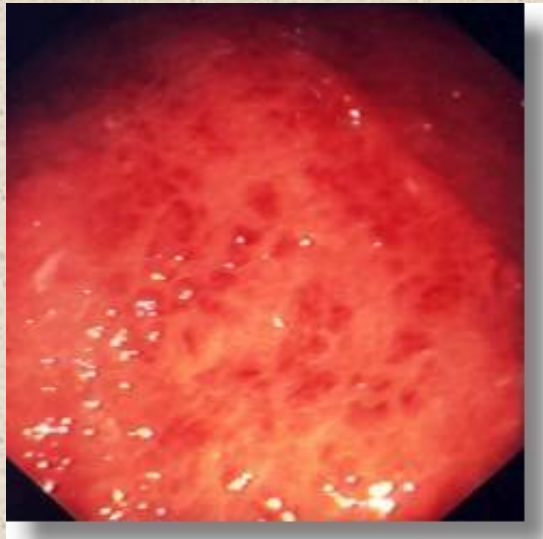
Underlying condition requiring transplant

- Renal transplant
 - Amyloid



Underlying condition requiring transplant

- Liver transplant
 - Portal colopathy



- Primary sclerosing cholangitis and IBD

Transplantation and IBD

activity of pre-transplant ulcerative colitis was unchanged or increased after orthotopic liver transplantation. Two further patients developed new-onset ulcerative colitis after orthotopic liver transplantation.

- Does transplantation improve or worsen IBD?
- Immunosuppression helps but restoration of normal liver function has opposite effect.

Other specific associations

- HIV/AIDS patients and colopathy
 - GvHD-like
 - IBD-like?
 - real entity?



23M AML patient



- Pneumatosis coli:



- Colitis but no ...



Leukaemia and neutropaenic colitis (aka typhlitis)



Typhlon (Grk) = caecum



Summary

- Rare pathology, rare presentation of common pathology, and/or multiple pathology
- Clinical data are crucial – especially when considering GvHD

When assessing transplant patient GI biopsies

- Time from transplant?
- Transplant type?
- Underlying disease?
- GvHD elsewhere?
- MMF therapy?
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