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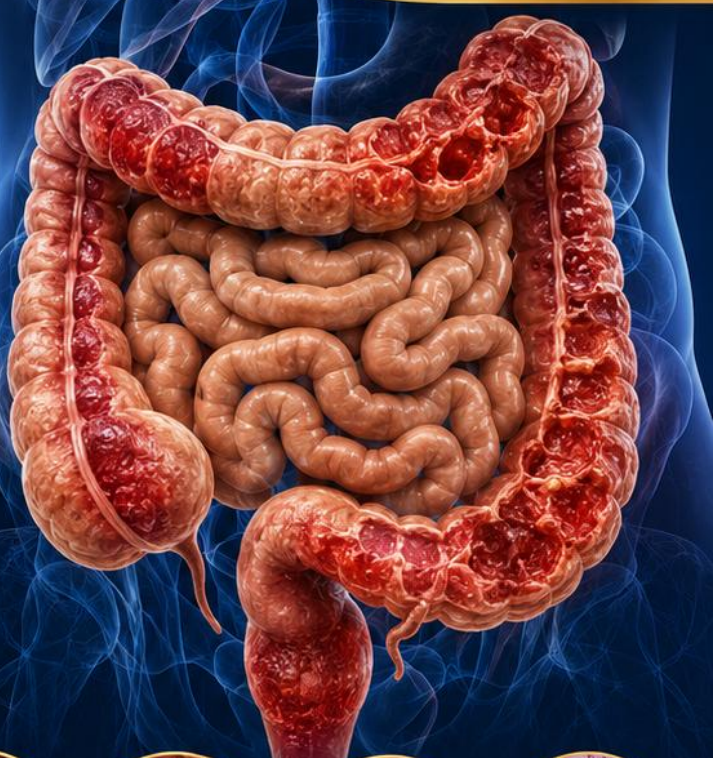
CHAPTER 5

INFLAMMATORY BOWEL DISEASE



ULCERATIVE COLITIS | CROHN'S DISEASE | PERIANAL DISEASE | ENDOSCOPIC & HISTOLOGIC INFLAMMATION

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GE and Hepatology Consultant

Dedication

In loving memory of my mother,

who recently returned to the mercy of Allah.

Her love, prayers, patience, and sacrifices were among the greatest blessings of my life. Whatever good I achieve carries something of her guidance and support.

May Allah forgive her, have mercy upon her, illuminate her grave, and grant her the highest place in **Jannat al-Firdaws**.

This work is dedicated to her memory.

أسأل الله أن يرحم أمي رحمة واسعه وأن يدخلها الفردوس الأعلى من الجنة، ورجاءً أسألكم لها خالص الدعاء..

Chapter 5: Inflammatory Bowel Disease

Ulcerative Colitis, Crohn Disease, Complications, Surgery and Postoperative Care

50 difficult original consultant-level mechanism-based MCQs

Style: **advanced fellowship level**, source-structured from uploaded references only, with **randomized correct-answer positions** and **bold colored key words**.

1. A 24-year-old man has ileal **Crohn disease**, aphthous ulcers, granulomas, and penetrating complications. A translational biopsy study shows abundant IL-12, IL-23, IFN-gamma, IL-17, TNF-alpha and activated macrophage/dendritic-cell signaling. Which mechanism most coherently explains this phenotype?

A. Th1/Th17 polarization driven by antigen-presenting cells responding to commensal microbial antigens

B. Pure epithelial chloride-channel hypersecretion without mucosal immune activation

C. CD1d-restricted NKT-cell IL-13 production causing superficial continuous colitis

D. Classical complement activation by circulating cryoglobulins with small-vessel deposition

E. IgE-mediated mast-cell degranulation against dietary carbohydrate antigens

Answer: A - Th1/Th17 polarization driven by antigen-presenting cells responding to commensal microbial antigens

Mechanism pearl: Crohn disease is best framed as dysregulated mucosal immunity to commensal microbiota in a susceptible host, with **Th1/Th17**, **IL-12/23**, **IL-17**, **IFN-gamma**, and **TNF-alpha** biology. Granulomas and penetrating disease fit this pathway.

2. A 31-year-old woman has continuous colitis from rectum to splenic flexure. Research sampling shows epithelial barrier disruption with increased IL-13, TNF-alpha and IL-6. Which cellular mechanism best distinguishes the classic immune model of **ulcerative colitis** from Crohn disease?

A. Loss of inhibitory nitrergic neurons causing mucosal ulceration

B. B-cell clones producing IgM rheumatoid factor against anti-colon IgG

C. CD1d-restricted NKT-cell cytokine activity producing IL-13-mediated epithelial injury

D. Direct cytopathic invasion of colonocytes by commensal anaerobes

E. Dominant eosinophilic IL-5 signaling identical to eosinophilic esophagitis

Answer: C - CD1d-restricted NKT-cell cytokine activity producing IL-13-mediated epithelial injury

Mechanism pearl: UC is often modeled as a **Th2-like/NKT** process, with **IL-13** disrupting the epithelial barrier. Both UC and Crohn may still share innate cytokines such as **TNF-alpha** and **IL-6**.

3. A patient with quiescent IBD reports bloating and irregular stools. CRP is normal, fecal calprotectin is low, and colonoscopy 3 months earlier showed mucosal healing. Which interpretation is most mechanistically sound before escalating biologic therapy?

A. Fecal calprotectin is a hepatocyte-derived protein and therefore cannot assess intestinal inflammation

B. Low calprotectin confirms cytomegalovirus colitis

C. Symptoms alone are a sufficient treat-to-target marker in IBD

D. Normal CRP and calprotectin prove irreversible fibrostenotic Crohn disease requiring immediate surgery

E. Low fecal calprotectin argues against active neutrophil-mediated intestinal inflammation and favors functional overlap or another non-inflammatory cause

Answer: E - Low fecal calprotectin argues against active neutrophil-mediated intestinal inflammation and favors functional overlap or another non-inflammatory cause

Mechanism pearl: Fecal calprotectin is a neutrophil cytosolic biomarker. It helps separate active inflammatory diarrhea from IBS overlap, bile-acid diarrhea, SIBO, carbohydrate malabsorption, or pancreatic insufficiency in IBD patients.

4. A 28-year-old woman with indeterminate colitis has p-ANCA positive and ASCA negative serology. The surgeon asks whether these markers should determine the operation. Which answer best reflects biomarker biology?

A. ASCA and p-ANCA directly measure transmural fibrosis and should replace histology

B. Serologic patterns are supportive but insufficient to override phenotype, endoscopy, imaging, and pathology

C. p-ANCA has perfect sensitivity for UC and eliminates the possibility of Crohn disease

D. ASCA negativity excludes postoperative Crohn disease of the pouch

E. Positive p-ANCA proves pouchitis before pouch formation

Answer: B - Serologic patterns are supportive but insufficient to override phenotype, endoscopy, imaging, and pathology

Mechanism pearl: p-ANCA and **ASCA** are supportive biomarkers, not decisive surgical tests. They may enrich probability but do not replace clinical phenotype, distribution, histology, imaging, and postoperative evolution.

5. A Crohn patient in clinical remission has persistently elevated fecal calprotectin and ileal ulcers on colonoscopy. Another patient has symptoms but normal biomarkers and healed mucosa. Which concept best explains why management should differ?

A. Fecal calprotectin is useful only after colectomy

B. Biologic therapy should be escalated whenever IBS-type symptoms occur

C. Endoscopic findings are irrelevant if abdominal pain is absent

D. Objective inflammation predicts complications and should guide escalation more than symptoms alone

E. Symptom control alone is the only validated therapeutic target

Answer: D - Objective inflammation predicts complications and should guide escalation more than symptoms alone

Mechanism pearl: Modern IBD care uses **objective inflammation** - endoscopy, CRP, and fecal calprotectin - because symptoms can reflect IBS overlap while silent inflammation can drive strictures, fistulas, dysplasia risk, and surgery.

6. A man with moderate-severe Crohn disease receives adequate induction infliximab. He remains symptomatic, colonoscopy confirms active ulcers, trough level is therapeutic, and no anti-drug antibodies are detected. What is the most mechanism-based next principle?

A. Swap to a different inflammatory mechanism because this suggests mechanistic primary nonresponse

B. Switch within anti-TNF class because adequate drug level proves immunogenicity

C. Increase infliximab indefinitely because every failure is pharmacokinetic

D. Add cholestyramine because therapeutic trough proves bile-acid diarrhea

E. Stop all immunotherapy because objective inflammation is absent

Answer: A - Swap to a different inflammatory mechanism because this suggests mechanistic primary nonresponse

Mechanism pearl: Active inflammation despite **adequate anti-TNF trough** and absent antibodies suggests **mechanistic failure**. Cycling to another anti-TNF is less rational than switching to another pathway such as **anti-integrin**, **IL-12/23**, **IL-23**, or a small molecule.

7. A woman with fistulizing Crohn disease initially responds to infliximab but relapses after 11 months. Trough level is undetectable and anti-drug antibodies are high. Which mechanism best explains the relapse?

A. TNF-alpha receptor mutation induced by vedolizumab

B. JAK-STAT inhibition producing colonic eosinophilia

C. Immunogenic clearance of the monoclonal antibody causing loss of effective TNF-alpha neutralization

D. S1P receptor trapping of infliximab inside lymph nodes

E. Cytokine escape with preserved infliximab exposure

Answer: C - Immunogenic clearance of the monoclonal antibody causing loss of effective TNF-alpha neutralization

Mechanism pearl: Low/absent trough plus high **anti-drug antibodies** indicates **pharmacokinetic failure** due to immunogenic clearance. Combination with thiopurine can reduce immunogenicity in selected anti-TNF strategies.

8. A hospitalized patient with acute severe UC has albumin 21 g/L, CRP 118 mg/L, frequent bloody stools, and severe mucosal ulceration. After standard infliximab induction, response is poor and trough level is very low without antibodies. Which mechanism is most likely?

A. Direct infliximab neutralization by IgA in stool only

B. Thiopurine hypermethylation producing high 6-MMP

C. Excess S1P receptor signaling blocking lymphocyte egress

D. Bile-acid malabsorption causing false low infliximab levels

E. High inflammatory burden, protein-losing mucosa, and hypoalbuminemia increasing biologic clearance

Answer: E - High inflammatory burden, protein-losing mucosa, and hypoalbuminemia increasing biologic clearance

Mechanism pearl: Severe colitis can create **rapid monoclonal antibody clearance** through high inflammatory burden and mucosal protein loss. **Hypoalbuminemia** is a pharmacokinetic warning sign.

9. A 62-year-old man with Crohn colitis and NYHA III systolic heart failure needs steroid-sparing therapy. Which selection is most mechanistically appropriate?

A. Adalimumab because anti-TNF therapy improves cardiac contractility

B. Ustekinumab or vedolizumab rather than anti-TNF therapy

C. Natalizumab without JC-virus testing

D. Golimumab because subcutaneous anti-TNF avoids heart failure risk

E. Infliximab plus high-dose prednisone as preferred long-term maintenance

Answer: B - Ustekinumab or vedolizumab rather than anti-TNF therapy

Mechanism pearl: In IBD with significant **heart failure**, uploaded sources favor **ustekinumab** or **vedolizumab** and avoidance of **anti-TNF** therapy because anti-TNF agents can worsen heart failure.

10. A patient with UC is planned for anti-TNF therapy. HBsAg is negative, anti-HBc is positive, anti-HBs is positive, and TB screening is pending. Which mechanism-based safety principle is correct?

A. Positive anti-HBc excludes any risk of HBV reactivation under immunosuppression

B. Anti-TNF therapy only suppresses neutrophils, so viral and granulomatous infections are irrelevant

C. Vaccination history becomes irrelevant once a monoclonal antibody is selected

D. Screening for TB and HBV is required because cytokine blockade can impair immune containment and permit reactivation

E. Vedolizumab uniquely requires no infection assessment of any kind

Answer: D - Screening for TB and HBV is required because cytokine blockade can impair immune containment and permit reactivation

Mechanism pearl: Before **biologics** and **small molecules**, sources emphasize TB and HBV screening. Blocking **TNF-alpha** can impair granuloma maintenance and antiviral immune surveillance.

11. A woman with moderate UC has recurrent systemic infections and wants an organ-selective biologic. Which mechanism best supports vedolizumab?

A. Alpha-4 beta-7 blockade preventing lymphocyte binding to intestinal MAdCAM-1 with relative gut selectivity

B. Blockade of IL-12/23 p40, reducing Th1 and Th17 differentiation in all tissues

C. JAK1/3 inhibition preventing STAT signaling in circulating lymphocytes

D. S1P1 modulation trapping lymphocytes in lymph nodes

E. Nonselective alpha-4 integrin blockade, including CNS alpha-4 beta-1 trafficking

Answer: A - Alpha-4 beta-7 blockade preventing lymphocyte binding to intestinal MAdCAM-1 with relative gut selectivity

Mechanism pearl: Vedolizumab targets **alpha-4 beta-7** interaction with **MAdCAM-1**, limiting gut lymphocyte trafficking. This explains lower systemic immunosuppression compared with broader trafficking blockade.

12. A Crohn patient asks why natalizumab requires JC-virus antibody monitoring whereas vedolizumab is preferred when available. Which mechanistic explanation is best?

A. Natalizumab blocks IL-23 p19, which is required for JC-virus latency

B. Vedolizumab directly depletes B cells and therefore prevents PML

C. Natalizumab blocks alpha-4 integrins including alpha-4 beta-1 in the CNS, increasing PML risk in JC-virus-positive patients

D. Natalizumab is a JAK inhibitor causing herpes zoster rather than PML

E. Vedolizumab crosses the blood-brain barrier but is rapidly degraded

Answer: C - Natalizumab blocks alpha-4 integrins including alpha-4 beta-1 in the CNS, increasing PML risk in JC-virus-positive patients

Mechanism pearl: Natalizumab blocks broader alpha-4 integrins, including CNS trafficking pathways. **Vedolizumab** is gut-selective through alpha-4 beta-7, so **PML** has not been the same concern.

13. A biologic-naive patient with moderate-severe Crohn disease and psoriasis is offered ustekinumab. Which mechanism best explains why it can treat both diseases?

A. H+/K+-ATPase inhibition reduces epithelial antigen presentation

B. Alpha-4 beta-7 blockade prevents leukocyte entry into skin and gut equally

C. Calcineurin inhibition blocks IL-2 transcription in all T cells

D. S1P1 receptor modulation blocks lymphocyte egress and causes bradycardia

E. p40 blockade inhibits both IL-12 and IL-23 signaling, modulating Th1/Th17 pathways

Answer: E - p40 blockade inhibits both IL-12 and IL-23 signaling, modulating Th1/Th17 pathways

Mechanism pearl: Ustekinumab targets **p40**, shared by **IL-12** and **IL-23**. This suppresses Th1/Th17-related inflammation relevant to Crohn disease and psoriasis.

14. A patient with anti-TNF-exposed Crohn disease is considered for risankizumab rather than ustekinumab. What is the most precise target difference?

A. Risankizumab blocks JAK1 while ustekinumab blocks JAK3

B. Risankizumab selectively blocks IL-23 p19, whereas ustekinumab blocks the shared IL-12/23 p40 subunit

C. Risankizumab blocks TNF-alpha while ustekinumab blocks IL-6 receptor

D. Risankizumab prevents lymphocyte egress through S1P1 modulation

E. Risankizumab blocks alpha-4 beta-7 while ustekinumab blocks alpha-4 beta-1

Answer: B - Risankizumab selectively blocks IL-23 p19, whereas ustekinumab blocks the shared IL-12/23 p40 subunit

Mechanism pearl: Risankizumab is an **IL-23 p19** inhibitor. **Ustekinumab** blocks **p40**, shared by IL-12 and IL-23. This is a comparative cytokine-target question, not a brand-name recall question.

15. A 58-year-old man with UC, prior myocardial infarction, dyslipidemia, and previous shingles asks about tofacitinib. Which mechanism-to-risk pairing is most accurate?

A. p40 blockade with obligatory severe neutropenia and no infection risk

B. Metalloprotease inhibition causing bile-acid malabsorption

C. Alpha-4 beta-7 blockade with PML risk requiring JC-virus monitoring

D. JAK inhibition reducing STAT-mediated cytokine signaling, with herpes zoster and thrombotic/cardiovascular safety concerns

E. TNF-alpha neutralization with rapid lymphocyte egress from lymph nodes

Answer: D - JAK inhibition reducing STAT-mediated cytokine signaling, with herpes zoster and thrombotic/cardiovascular safety concerns

Mechanism pearl: Tofacitinib inhibits **JAK1/3** and interrupts **STAT** signaling. Uploaded sources emphasize **herpes zoster**, lipid changes, and black-box cardiovascular/thrombotic concerns in risk-enriched patients.

16. A patient with severe UC has albumin 19 g/L and high inflammatory burden. The team worries that monoclonal antibody exposure may be poor. Which therapeutic class is least dependent on monoclonal antibody pharmacokinetics?

A. Oral small molecules such as JAK inhibitors when otherwise appropriate

B. Ustekinumab because p40 binding prevents fecal protein loss

C. Infliximab dose escalation only

D. Adalimumab because subcutaneous monoclonals cannot be cleared rapidly

E. Vedolizumab because all gut-selective antibodies ignore albumin

Answer: A - Oral small molecules such as JAK inhibitors when otherwise appropriate

Mechanism pearl: Source tables note **hypoalbuminemia** as a reason to consider **small molecules**. They are not monoclonal antibodies and are less exposed to protein-losing mucosal clearance mechanisms, though safety constraints still matter.

17. A woman with UC starts ozanimod and develops transient bradycardia during initiation. Which mechanism explains both therapeutic effect and this adverse event?

A. Alpha-4 beta-7 blockade preventing MAdCAM-1 binding

B. TNF-alpha neutralization impairing granuloma maintenance

C. S1P receptor modulation preventing lymphocyte egress from lymphoid tissue, with S1P1 cardiac effects

D. JAK1 inhibition reducing IL-6 signaling in T cells

E. IL-12/23 p40 blockade impairing Th1/Th17 cells

Answer: C - S1P receptor modulation preventing lymphocyte egress from lymphoid tissue, with S1P1 cardiac effects

Mechanism pearl: Ozanimod modulates **S1P receptors**, trapping lymphocytes in lymphoid tissue. **S1P1** effects explain dose titration and bradycardia screening logic.

18. A young man is considered for azathioprine. TPMT activity is absent. Which mechanism-based consequence is the main danger?

A. Failure to generate any immunosuppressive metabolite causing immediate flare only

B. Selective IL-23 p19 blockade with psoriasis improvement

C. S1P1 modulation causing first-dose bradycardia

D. Alpha-4 beta-1 blockade causing PML

E. Rapid conversion to excess active thioguanine nucleotides causing severe myelotoxicity

Answer: E - Rapid conversion to excess active thioguanine nucleotides causing severe myelotoxicity

Mechanism pearl: Low/absent **TPMT** or high-risk thiopurine metabolism predisposes to excess active metabolites and **severe leukopenia/myelotoxicity**. TPMT/NUDT15 logic is pharmacogenetic safety, not efficacy recall.

19. A UC patient on azathioprine develops leukopenia with high 6-thioguanine nucleotide levels. Another patient develops transaminitis with preferential 6-methylmercaptopurine production. Which principle best explains the different toxicities?

A. Both are caused by anti-drug antibodies against azathioprine

B. Different thiopurine metabolic shunting patterns produce different active or hepatotoxic metabolite profiles

C. All thiopurine toxicity proves CMV colitis

D. 6-TGN is a fecal neutrophil protein

E. Thiopurines are not metabolized; toxicity reflects direct TNF-alpha neutralization

Answer: B - Different thiopurine metabolic shunting patterns produce different active or hepatotoxic metabolite profiles

Mechanism pearl: Thiopurine metabolite testing separates active **6-TGN**-related efficacy/myelotoxicity from **6-MMP**-skewed hepatotoxicity. This is precision pharmacology, not simple dose recall.

20. A patient with moderate-severe UC has steroid dependence and is offered thiopurine monotherapy for induction. Which statement best reflects the mechanism and clinical positioning?

A. Thiopurines are rapid topical anti-inflammatory drugs equivalent to rectal mesalamine

B. Thiopurines are preferred over all biologics for acute severe UC rescue

C. Thiopurines prevent lymphocyte egress like S1P modulators

D. Thiopurines are slow lymphocyte antiproliferative agents and are not appropriate rapid induction therapy for active moderate-severe UC

E. Thiopurines directly neutralize TNF-alpha within hours

Answer: D - Thiopurines are slow lymphocyte antiproliferative agents and are not appropriate rapid induction therapy for active moderate-severe UC

Mechanism pearl: Azathioprine/6-MP are slow immunomodulators. They may support maintenance or combination anti-TNF strategies, but are poor choices for rapid induction or acute severe colitis rescue.

21. A man with isolated ileal Crohn disease is prescribed high-dose mesalamine because it is safe. Six months later he has a fibrostenotic stricture. Which reasoning best critiques the original strategy?

A. 5-ASA has limited evidence for altering Crohn disease course, especially complicated ileal disease

B. Mesalamine induces rapid mucosal healing in penetrating Crohn disease

C. 5-ASA works by alpha-4 beta-7 blockade and therefore should prevent strictures

D. Mesalamine is the strongest therapy for maintaining Crohn remission

E. Mesalamine prevents anti-TNF antibody formation

Answer: A - 5-ASA has limited evidence for altering Crohn disease course, especially complicated ileal disease

Mechanism pearl: Uploaded sources describe **5-ASA** as much better established in UC than Crohn disease. In complicated or high-risk Crohn disease, safety should not be mistaken for disease-modifying efficacy.

22. A 26-year-old woman is admitted with acute severe UC: 9 bloody stools/day, fever, tachycardia, anemia, high CRP. She is not peritonitic. Which initial package is most mechanism-based?

A. Thiopurine monotherapy because it has rapid onset

B. High-dose oral loperamide, full colonoscopy, and outpatient prednisone

C. Stool testing for C. difficile, early flexible sigmoidoscopy with CMV biopsies, IV corticosteroids, VTE prophylaxis, and surgical awareness

D. Broad-spectrum antibiotics for all patients regardless of infection, no endoscopy

E. Immediate IPAA because acute severe UC is best treated by restorative surgery in one stage

Answer: C - Stool testing for C. difficile, early flexible sigmoidoscopy with CMV biopsies, IV corticosteroids, VTE prophylaxis, and surgical awareness

Mechanism pearl: Acute severe UC is an inflammation-and-complication emergency. Sources emphasize excluding **C. difficile**, assessing severity with **flexible sigmoidoscopy** and **CMV biopsies**, IV steroids, VTE prophylaxis, and early surgical involvement.

23. A hospitalized ASUC patient has no meaningful improvement after 4 days of IV methylprednisolone, CDI is excluded, CMV is not seen, and there is no perforation. Which next step is most defensible?

A. Start azathioprine because it induces remission within 48 hours

B. Continue the same steroid dose for 3 more weeks because delayed response is expected

C. Perform segmental colectomy of the most inflamed left colon

D. Use loperamide to reduce stool number and reassess after diet

E. Use rescue infliximab or cyclosporine while maintaining close surgical backup

Answer: E - Use rescue infliximab or cyclosporine while maintaining close surgical backup

Mechanism pearl: Steroid-refractory **ASUC** requires **rescue therapy** with infliximab or cyclosporine, or colectomy depending on urgency. Delay increases risk of toxic megacolon, perforation, and sepsis.

24. Cyclosporine is chosen as rescue therapy for steroid-refractory ASUC in a selected patient. Which intracellular mechanism explains its immunosuppressive action?

A. Neutralization of soluble and membrane TNF-alpha

B. Inhibition of calcineurin, reducing NFAT-driven IL-2 transcription and T-cell activation

C. Blockade of alpha-4 beta-7 binding to MAdCAM-1

D. S1P1 receptor down-modulation trapping lymphocytes in nodes

E. Blockade of IL-23 p19

Answer: B - Inhibition of calcineurin, reducing NFAT-driven IL-2 transcription and T-cell activation

Mechanism pearl: Cyclosporine is a **calcineurin inhibitor**. It suppresses T-cell activation by reducing **IL-2 transcription**, mechanistically distinct from infliximab rescue.

25. A patient with UC develops fever, tachycardia, abdominal distention, transverse colon diameter 6.3 cm, and systemic toxicity. Which pathophysiologic concern drives urgent management?

A. Isolated bile-acid diarrhea requiring cholestyramine

B. Functional diarrhea with normal inflammatory risk

C. Pouchitis because UC always has an ileal pouch

D. Toxic megacolon with risk of perforation, stool spillage, sepsis, and death

E. Benign gas trapping that should be treated with colonoscopic decompression under full bowel prep

Answer: D - Toxic megacolon with risk of perforation, stool spillage, sepsis, and death

Mechanism pearl: Toxic megacolon is severe colitis plus colonic dilation and systemic toxicity. It can progress to **perforation** and sepsis; antidiarrheals and unsafe colonoscopy are dangerous.

26. A patient with UC undergoes total proctocolectomy and later develops progressive cholestatic enzymes with MRCP showing multifocal strictures. Why did colectomy not prevent this complication?

A. PSC is an extraintestinal hepatobiliary manifestation that may persist or appear despite removal of colonic mucosa

B. PSC after colectomy proves the original diagnosis was Crohn disease

C. PSC is caused by pouchitis antibiotics

D. Colectomy cures all immune phenomena linked to UC

E. IPAA produces biliary obstruction by mechanical compression

Answer: A - PSC is an extraintestinal hepatobiliary manifestation that may persist or appear despite removal of colonic mucosa

Mechanism pearl: Surgery can cure colonic **UC**, but not all extraintestinal biology. **PSC** may precede or follow IBD and can persist after colectomy; cholangiocarcinoma risk also remains.

27. A malnourished, septic patient with fulminant UC and peritonitis requires emergency surgery. Which operation best matches the physiologic principle?

A. Single-stage IPAA with pouch creation during septic shock

B. Cecoanal anastomosis because rectal disease is limited

C. Subtotal/abdominal colectomy with end ileostomy and rectal stump management, deferring restorative surgery

D. Strictureplasty of the colon

E. Segmental left colectomy to preserve uninvolved colon

Answer: C - Subtotal/abdominal colectomy with end ileostomy and rectal stump management, deferring restorative surgery

Mechanism pearl: Emergent fulminant UC is managed by damage-control logic: remove the diseased colon and avoid pelvic pouch construction in a physiologically unstable patient. **IPAA** can be staged after recovery.

28. A woman planning IPAA asks about stapled versus hand-sewn anastomosis. Which trade-off is most accurate?

A. Stapled IPAA removes all rectal mucosa but increases incontinence

B. Hand-sewn mucosectomy leaves a rectal cuff but improves continence

C. Both techniques eliminate the need for pouchoscopy forever

D. Hand-sewn IPAA is contraindicated in all patients with UC

E. Stapled IPAA preserves better continence but leaves residual rectal cuff at risk for cuffitis and dysplasia surveillance

Answer: E - Stapled IPAA preserves better continence but leaves residual rectal cuff at risk for cuffitis and dysplasia surveillance

Mechanism pearl: Stapled IPAA has better functional continence but leaves a **rectal cuff**. **Hand-sewn mucosectomy** removes more mucosa but may increase gas/stool incontinence risk.

29. A patient with stapled IPAA presents with urgency and bright bleeding. Pouch body is normal, but the residual rectal cuff is inflamed. Which diagnosis and therapy are most logical?

A. Irritable pouch syndrome treated with seton placement

B. Cuffitis from residual rectal mucosa; topical mesalamine or topical steroid therapy

C. Pouchitis requiring lifelong anti-TNF without endoscopic confirmation

D. Anastomotic leak requiring azathioprine

E. Crohn disease of the pouch requiring immediate pouch excision

Answer: B - Cuffitis from residual rectal mucosa; topical mesalamine or topical steroid therapy

Mechanism pearl: Cuffitis occurs in residual rectal mucosa after stapled IPAA and often presents with **bleeding** and urgency. Pouchitis more often causes frequency, urgency, diarrhea, pain, and is usually antibiotic responsive.

30. A man with IPAA for UC develops increased stool frequency, urgency and cramps 2 years after surgery. Symptoms alone are nonspecific. Which evaluation is most complete before labeling idiopathic pouchitis?

A. CT chest because pouchitis is primarily pulmonary

B. Immediate pouch excision without stool tests

C. Serum p-ANCA only, because it replaces pouchoscopy

D. Pouchoscopy assessing neoterminal ileum, pouch body, rectal cuff/transitional zone, plus exclusion of C. difficile and other infections

E. Treat every pouch symptom as Crohn disease without endoscopy

Answer: D - Pouchoscopy assessing neoterminal ileum, pouch body, rectal cuff/transitional zone, plus exclusion of C. difficile and other infections

Mechanism pearl: Pouch symptoms correlate imperfectly with inflammation. **Pouchoscopy** should examine the neoterminal ileum, pouch, cuff, and transitional zone while excluding **C. difficile**, CMV, NSAID injury, ischemia, and Crohn disease of the pouch.

31. Eight days after IPAA, a patient on perioperative high-dose steroids develops fever and pelvic pain. CT shows a peripouch abscess. Which mechanism-based management is best?

A. CT-guided drainage if feasible plus antibiotics, because pelvic sepsis threatens pouch function and survival

B. Immediate biologic escalation without source control

C. Observation only because all postoperative collections are sterile

D. High-dose loperamide to reduce pouch output

E. Pouchoscopy with aggressive dilation as first step

Answer: A - CT-guided drainage if feasible plus antibiotics, because pelvic sepsis threatens pouch function and survival

Mechanism pearl: Pelvic sepsis after IPAA is linked to steroid exposure, leaks, poor function, and pouch loss. Abscess biology requires **source control** - percutaneous or surgical drainage when feasible.

32. A Crohn patient has an enteroenteric fistula found on MRE but is asymptomatic, nutritionally stable, and has no abscess or recurrent infection. Which surgical principle is best?

A. All fistulas should be closed endoscopically before treating inflammation

B. Fistulas are superficial mucosal complications like UC and do not need assessment

C. Presence of fistula alone does not mandate surgery; operate for infection, malabsorption, intractable diarrhea, persistent symptoms, or quality-of-life impact

D. Every fistula mandates immediate resection of all connected organs

E. Fistulas are treated by segmental colectomy in UC only

Answer: C - Presence of fistula alone does not mandate surgery; operate for infection, malabsorption, intractable diarrhea, persistent symptoms, or quality-of-life impact

Mechanism pearl: Crohn fistulas reflect **transmural inflammation**, but surgery is complication-driven. Noninflamed organs connected by fistula may be repaired rather than widely resected.

33. A Crohn patient has fever and RLQ pain. CT shows a 5-cm intra-abdominal abscess adjacent to an inflamed ileal segment. What is the best initial strategy before elective resection or biologic escalation?

A. Immediate anti-TNF therapy without drainage to close the fistula

B. Colonic strictureplasty

C. Antibiotics alone because abscess size is irrelevant

D. High-dose oral steroids to reduce abscess wall inflammation

E. Antibiotics plus percutaneous drainage if technically feasible

Answer: E - Antibiotics plus percutaneous drainage if technically feasible

Mechanism pearl: For Crohn abscesses, uploaded sources use a pragmatic cutoff: <3 cm may respond to antibiotics; **>3 cm** generally needs **antibiotics plus percutaneous drainage** before definitive surgery or biologics.

34. A man with perianal Crohn disease has severe pain, fever and MRI showing a complex fistula with abscess. What is the correct sequence?

A. Immediate anti-TNF alone because biologics sterilize abscess cavities

B. Drain sepsis with EUA/abscess drainage and seton when needed, then use medical therapy such as anti-TNF after infection control

C. Topical mesalamine only because perianal disease is mucosal

D. Fistulotomy through sphincter muscle in all complex fistulas

E. IPAA creation to cure perianal Crohn disease

Answer: B - Drain sepsis with EUA/abscess drainage and seton when needed, then use medical therapy such as anti-TNF after infection control

Mechanism pearl: Complex perianal Crohn requires **sepsis control** first. **Setons** maintain drainage; anti-TNF therapy is more rational after abscess drainage, not as a substitute for source control.

35. A Crohn patient has obstructive symptoms. MRE shows a 3-cm ileal stricture, no fistula, no abscess, no dysplasia, and minimal active inflammation. Which intervention matches mechanism?

A. No therapy because strictures cannot obstruct

B. Vedolizumab because it mechanically dilates scar tissue

C. High-dose systemic steroids indefinitely because all strictures are inflammatory

D. Endoscopic balloon dilation because a short fibrostenotic stricture without penetrating or malignant features is suitable

E. Segmental colectomy because all ileal strictures are malignant

Answer: D - Endoscopic balloon dilation because a short fibrostenotic stricture without penetrating or malignant features is suitable

Mechanism pearl: Inflammatory strictures may respond to medical therapy; **fibrostenotic strictures** need dilation or surgery. Endoscopic dilation is considered when short (<5 cm) and without abscess, fistula, dysplasia, or cancer.

36. A patient with longstanding jejunoileal Crohn disease has multiple non-adjacent fibrotic strictures and only 140 cm of small bowel remaining. Which surgical approach best preserves future absorptive capacity?

A. Small bowel strictureplasty, combined with limited resection only where necessary

B. Wide resection of every stricture regardless of bowel length

C. Colonic mucosectomy

D. Permanent avoidance of surgery despite high-grade obstruction

E. Total colectomy with IPAA

Answer: A - Small bowel strictureplasty, combined with limited resection only where necessary

Mechanism pearl: Strictureplasty preserves small bowel length in Crohn disease, especially with multiple strictures or short-bowel risk. It is a bowel-preservation operation, not a curative anti-inflammatory therapy.

37. A Crohn patient has a new tight colonic stricture. Biopsies are nondiagnostic because the scope cannot traverse the lesion. Why is colonic strictureplasty generally avoided?

A. Colonic strictures are always inflammatory and never malignant

B. Colonic strictureplasty causes bile-acid diarrhea by design

C. Concern for dysplasia or cancer makes resection/evaluation preferred over strictureplasty

D. Anti-TNF antibodies make all strictures disappear

E. Strictureplasty is contraindicated only in ileal disease

Answer: C - Concern for dysplasia or cancer makes resection/evaluation preferred over strictureplasty

Mechanism pearl: Colonic strictures in IBD require malignancy concern. Unlike small bowel Crohn strictures, colonic strictures are generally not treated with strictureplasty because occult dysplasia/cancer must be addressed.

38. A 19-year-old presents to surgery with suspected appendicitis. At laparotomy, the terminal ileum is inflamed and thickened, with features suggestive of Crohn disease. Which principle best reflects modern surgical reasoning?

A. The appendix must never be removed when terminal ileitis is present

B. Crohn disease is cured by appendectomy alone in most cases

C. IPAA is the operation of choice for ileocecal Crohn disease

D. All terminal ileitis should be ignored even if cecum and ileocecal region are diseased

E. Ileocecal Crohn may masquerade as appendicitis and many such patients ultimately require ileocolic resection; decision depends on cecal involvement, obstruction, perforation and operative risk

Answer: E - Ileocecal Crohn may masquerade as appendicitis and many such patients ultimately require ileocolic resection; decision depends on cecal involvement, obstruction, perforation and operative risk

Mechanism pearl: Ileocecal Crohn can mimic appendicitis. Older teaching favored appendectomy alone when cecum is normal, but sources note many patients found to have Crohn at laparotomy require early ileocolic resection.

39. A Crohn patient with diarrhea >10/day, no vomiting, CT ileal thickening, mesenteric stranding, mild proximal dilation and high CRP is labeled 'partial SBO'. Which interpretation best avoids unnecessary surgery?

A. Anti-inflammatory therapy is contraindicated when any dilation is present

B. This may represent active inflammatory ileitis with chronic partial narrowing; medical therapy is appropriate if no high-grade obstruction or strangulation

C. The finding proves colonic cancer

D. Diarrhea excludes Crohn inflammation and proves adhesive obstruction

E. All CT reports of partial SBO in Crohn require immediate resection

Answer: B - This may represent active inflammatory ileitis with chronic partial narrowing; medical therapy is appropriate if no high-grade obstruction or strangulation

Mechanism pearl: Crohn can cause imaging language of 'partial SBO' during active ileitis. The clinical pattern - diarrhea, no vomiting, inflammatory markers - may favor **active inflammation** over acute mechanical obstruction.

40. A 27-year-old smoker with penetrating ileocolonic Crohn disease undergoes his second ileocolic resection. What postoperative strategy best targets recurrence biology?

A. Delay therapy until obstructive symptoms recur

B. Avoid smoking cessation because smoking protects Crohn disease

C. Use mesalamine only because surgery cures Crohn disease

D. Start anti-TNF prophylaxis within about 4 weeks and perform colonoscopy at 6-12 months

E. No surveillance if symptoms resolve after surgery

Answer: D - Start anti-TNF prophylaxis within about 4 weeks and perform colonoscopy at 6-12 months

Mechanism pearl: High-risk postoperative Crohn features include **smoking**, penetrating disease, young age, prior resections, extensive/upper GI disease. Sources recommend risk-based prophylaxis and **6-12 month colonoscopy**.

41. An asymptomatic patient 8 months after ileocolic resection has neo-terminal ileal colonoscopy showing >5 aphthous ulcers with normal intervening mucosa. Which interpretation is best?

A. This is endoscopic recurrence by Rutgeerts logic and should prompt therapy initiation or intensification

B. Symptoms are absent, so this is complete remission and no action is needed

C. This finding is compatible only with pouchitis

D. Aphthae after resection always represent ischemia, not Crohn disease

E. The Rutgeerts score applies only after IPAA for UC

Answer: A - This is endoscopic recurrence by Rutgeerts logic and should prompt therapy initiation or intensification

Mechanism pearl: Rutgeerts score detects postoperative Crohn recurrence before symptoms. Scores 0-1 suggest remission; **i2 or higher** indicates recurrence and guides escalation.

42. A patient with Crohn disease asks why stopping cigarettes is emphasized more strongly after surgery than in many other GI disorders. Which mechanism-based answer is best?

A. Smoking reduces risk of small bowel recurrence after ileocolic resection

B. Smoking protects Crohn disease but worsens UC only

C. Smoking is a patient-related risk factor for postoperative Crohn recurrence and worse disease course

D. Smoking directly blocks IL-23 and prevents strictures

E. Smoking eliminates anti-drug antibody formation

Answer: C - Smoking is a patient-related risk factor for postoperative Crohn recurrence and worse disease course

Mechanism pearl: Active cigarette smoking is a major modifiable risk factor for Crohn disease recurrence, including postoperative recurrence. This is a high-yield patient-level recurrence variable.

43. A man with pancolitis for 12 years has persistent histologic inflammation despite mild symptoms. He asks why surveillance intensity and treatment targets focus on microscopic activity. Which mechanism best explains cancer risk?

A. Cancer risk disappears when symptoms are absent

B. Anti-TNF therapy directly mutates p53 in colonic epithelium

C. Only pseudopolyps, not histologic inflammation, cause cancer

D. CRC risk in UC is unrelated to inflammation duration or extent

E. Inflammation-driven epithelial injury and repair promote dysplasia and colorectal neoplasia

Answer: E - Inflammation-driven epithelial injury and repair promote dysplasia and colorectal neoplasia

Mechanism pearl: Chronic **histologic inflammation** is a neoplasia risk signal in colitis. The pathway is inflammation -> epithelial injury/repair -> dysplasia, explaining surveillance and treat-to-target logic.

44. A patient with PSC-IBD has quiescent bowel symptoms but extensive colitis. Which surveillance principle is mechanistically justified?

A. PSC proves the diagnosis is Crohn disease, not UC

B. PSC-associated colitis carries high neoplasia concern even when bowel symptoms are mild, so careful colonoscopic surveillance is needed

C. PSC removes the need for colonoscopy after colectomy in all cases

D. Cholangiocarcinoma risk is eliminated by mesalamine

E. Colitis surveillance should be based only on abdominal pain

Answer: B - PSC-associated colitis carries high neoplasia concern even when bowel symptoms are mild, so careful colonoscopic surveillance is needed

Mechanism pearl: PSC-IBD can have mild intestinal symptoms yet high colorectal neoplasia concern. Hepatobiliary and colonic risks may persist independently of symptom burden.

45. A UC patient has painful tender erythematous nodules over the shins during a colitis flare. Another patient has a rapidly progressive ulcer with violaceous undermined border despite quiescent colitis. Which distinction is most important?

A. Pyoderma gangrenosum is a fungal infection caused by anti-TNF therapy

B. Both always require colectomy regardless of bowel activity

C. Erythema nodosum proves small-bowel lymphoma

D. Erythema nodosum often parallels bowel activity; pyoderma gangrenosum may be independent and requires directed therapy

E. Skin disease in IBD never reflects systemic immune activity

Answer: D - Erythema nodosum often parallels bowel activity; pyoderma gangrenosum may be independent and requires directed therapy

Mechanism pearl: IBD **extraintestinal manifestations** differ biologically. **Erythema nodosum** often improves with bowel control, whereas **pyoderma gangrenosum** may be independent and needs urgent specialist therapy.

46. A patient with IBD develops painful red eye, photophobia and blurred vision. Which reasoning is most appropriate?

A. Urgent ophthalmologic evaluation is needed because uveitis can threaten vision and may not parallel bowel activity

B. Anti-TNF therapy is contraindicated in every ocular manifestation

C. Treat as benign episcleritis only if bowel symptoms are active

D. Colectomy immediately cures all ocular immune disease

E. Eye symptoms are caused by bile-acid malabsorption

Answer: A - Urgent ophthalmologic evaluation is needed because uveitis can threaten vision and may not parallel bowel activity

Mechanism pearl: Uveitis/iritis can threaten vision and requires urgent assessment. Extraintestinal manifestations are not all bowel-activity dependent.

47. A Crohn patient with ileal resection, fat malabsorption, intact colon, recurrent calcium oxalate renal stones and high urinary oxalate asks why this occurs. Which mechanism is best?

A. Anti-TNF antibodies crystallize in urine

B. PSC causes renal oxalate secretion

C. Fatty acids bind luminal calcium, leaving more free oxalate for colonic absorption

D. Loss of colon prevents oxalate absorption

E. Bile acid sequestrants increase urinary oxalate directly

Answer: C - Fatty acids bind luminal calcium, leaving more free oxalate for colonic absorption

Mechanism pearl: Enteric hyperoxaluria requires malabsorption and often an intact colon. Unabsorbed fatty acids bind calcium, freeing **oxalate** for colonic absorption and renal stone formation.

48. A patient with long-standing ileal Crohn disease and ileal resection develops recurrent cholesterol gallstones. Which mechanism best explains this association?

A. Anti-TNF therapy converts bilirubin to cholesterol

B. PSC always produces cholesterol stones

C. Crohn disease directly increases gallbladder alpha-4 beta-7 expression

D. Colonic inflammation increases pancreatic bicarbonate secretion

E. Ileal disease impairs bile acid reabsorption, reducing the bile acid pool and favoring cholesterol supersaturation

Answer: E - Ileal disease impairs bile acid reabsorption, reducing the bile acid pool and favoring cholesterol supersaturation

Mechanism pearl: Ileal Crohn or resection can reduce **bile acid reabsorption**, deplete bile-acid pool, impair cholesterol solubilization, and increase **gallstone** risk.

49. A hospitalized patient with severe UC has active bleeding but no contraindication to anticoagulant prophylaxis. The team hesitates because of hematochezia. Which reasoning is best?

A. VTE prophylaxis should wait until colectomy is completed

B. IBD creates a postoperative/inpatient thrombotic risk, so VTE prophylaxis is still important unless contraindicated

C. UC bleeding excludes venous thrombosis risk

D. Elevated CRP protects against thrombosis

E. Only Crohn disease, not UC, causes thrombosis

Answer: B - IBD creates a postoperative/inpatient thrombotic risk, so VTE prophylaxis is still important unless contraindicated

Mechanism pearl: IBD hospitalization and surgery increase **thromboembolic risk**. Sources emphasize a low threshold for VTE risk assessment and prophylaxis, especially in severe colitis and perioperative care.

50. A patient with axial spondyloarthritis and Crohn disease asks why etanercept is not chosen even though it is an anti-TNF used in rheumatology. Which mechanism-based answer is most accurate?

A. All anti-TNF agents are equally effective in IBD and axial disease

B. Sulfasalazine treats axial disease but not peripheral arthritis

C. Vedolizumab is the best therapy for axial spondyloarthritis because it enters joints selectively

D. Etanercept neutralizes TNF in a way that is ineffective for IBD, whereas monoclonal anti-TNF agents such as infliximab/adalimumab are effective for intestinal disease

E. Secukinumab is preferred because IL-17 blockade reliably improves Crohn disease

Answer: D - Etanercept neutralizes TNF in a way that is ineffective for IBD, whereas monoclonal anti-TNF agents such as infliximab/adalimumab are effective for intestinal disease

Mechanism pearl: IBD-associated spondyloarthritis requires therapy that covers both gut and joints. Monoclonal **anti-TNF** agents treat IBD; **etanercept** is ineffective for IBD despite rheumatologic TNF targeting.