ULCERATIVE COLITIS

Determining Ideal Targets for Treatment

Audience: RN/APRN | **Contact hrs:** 1.5 | **Pharmacology hrs:** 0.5

Expected revision: October 5, 2028

Pharmacology hours are included in the total contact hours.

Aiming beyond symptom relief

A patient who no longer sees blood may still have active colonic inflammation. Another patient may have urgency despite substantial mucosal healing. These situations require different responses. Treat-to-target care connects what the patient experiences with objective evidence of inflammation, the burden of treatment, and the life the patient wants to resume. The aim is durable, steroid-free disease control rather than repeated short-lived improvement. (Rubin et al., 2025; Turner et al., 2021).

Educational purpose. Develop a practical approach to selecting, measuring, and acting on treatment targets in ulcerative colitis (UC), with particular attention to medication safety and timely escalation. This article is for RNs and APRNs. Adult management is emphasized; children require pediatric disease-activity, growth, dosing, and monitoring pathways. U.S. prescribing information is identified where used and does not establish worldwide approval. RNs assess, administer authorized treatments, teach, monitor, and escalate concerns; diagnostic and prescribing decisions require appropriate licensure and privileges.

Learning outcomes

After completing this activity, the learner should be able to: **1.** Relate mucosal inflammation and disease extent to presentation and assessment; **2.** Distinguish symptomatic, biomarker, endoscopic, and histologic findings when evaluating targets; **3.** Explain selection and safe administration of conventional and advanced therapies; **4.** Prioritize recognition, monitoring, and escalation in acute severe UC; **5.** Construct a follow-up plan that addresses function, adherence, prevention, and patient preferences; and **6.** Identify when an apparent treatment failure requires investigation rather than an automatic drug change.

Clinical cases in this article are fictional teaching examples. They illustrate reasoning and communication, not standing orders or a substitute for patient-specific assessment.

Biology, risk, and disease extent

UC is a chronic inflammatory bowel disease involving the colon and rectum. In untreated typical disease, inflammation begins in the rectum and extends proximally in a continuous pattern. Injury primarily affects the mucosa, but severe inflammation can compromise the bowel wall and produce life-threatening complications. The characteristic pattern helps distinguish UC from Crohn's disease, without making distribution alone sufficient for diagnosis. Treatment can modify the apparent distribution; an atypical finding warrants interpretation rather than an automatic change of diagnosis. (Kucharzik et al., 2025; Rubin et al., 2025).

The mucosal barrier is an active interface. Goblet cells produce mucus; epithelial cells and their junctions restrict access to underlying tissue; innate and adaptive immune responses normally tolerate much of the intestinal microbial community. In UC, barrier dysfunction, altered microbial ecology, genetic susceptibility, and dysregulated immune signaling reinforce one another. Neutrophils enter damaged crypts and the lumen, helping explain crypt abscesses and elevated fecal calprotectin. Ulceration explains visible blood; inflamed distal mucosa contributes to urgency and tenesmus. Figure 1 links distribution to mucosal injury. (Kucharzik et al., 2025; Rubin et al., 2025).

No single infection, food, emotional state, or immune-cell pathway explains every patient's disease. Family history is relevant, but currently available routine genetic or antibody testing does not reliably select the ideal UC medication. Research on epithelial peroxisome proliferator-activated receptor gamma (PPAR-γ) helps explain part of mesalamine's local anti-inflammatory activity; it does not justify prescribing unrelated PPAR-γ agonists as established UC treatment. (Rousseaux et al., 2005; Rubin et al., 2025).

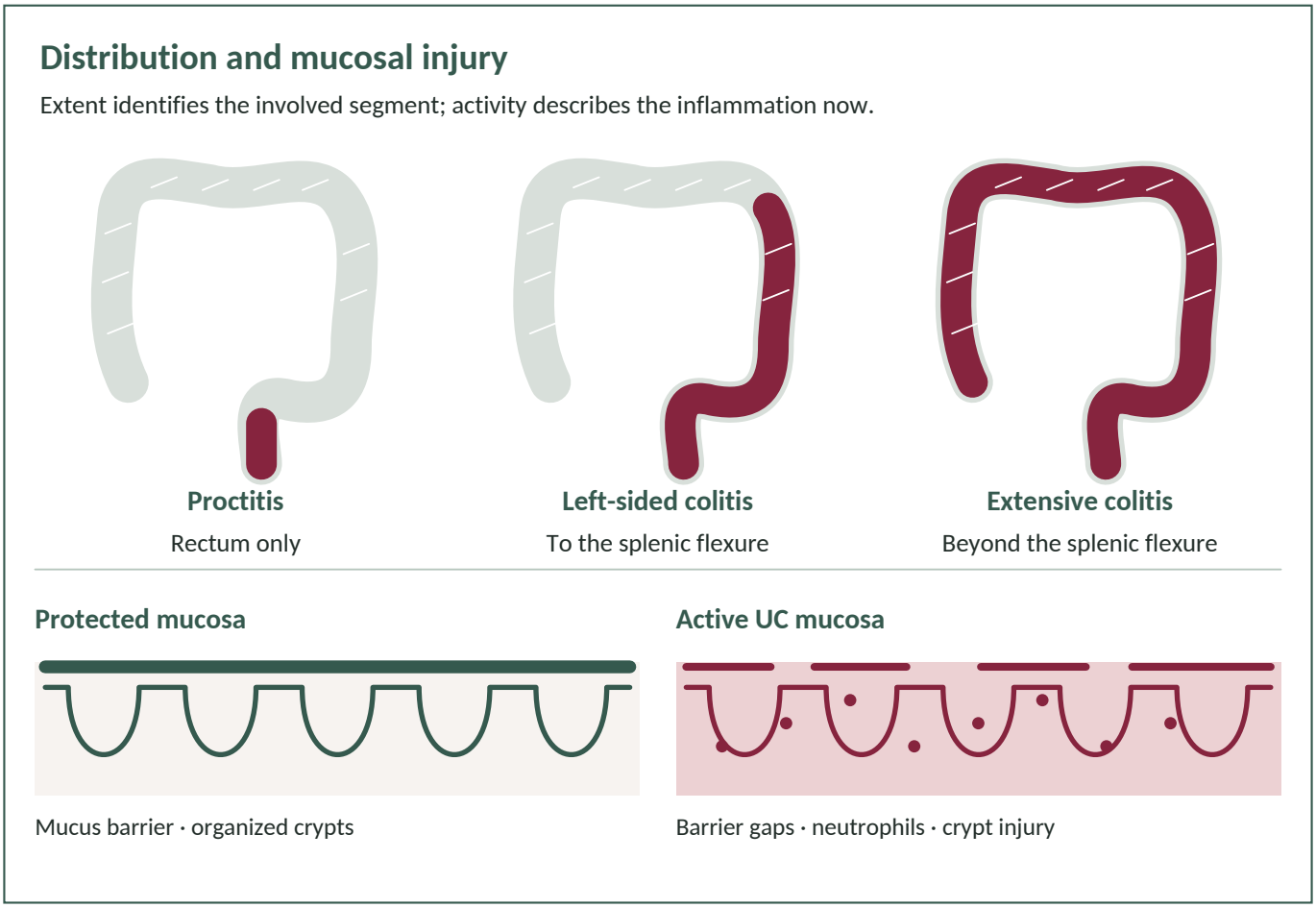


Figure 1. Typical untreated UC distribution and a schematic comparison of protected and inflamed mucosa. The colored segment indicates extent, not activity severity. Original educational diagram; not a diagnostic image. Sources: Rubin et al., 2025; Kucharzik et al., 2025.

Environmental associations require careful counseling. A statistical relationship between smoking exposure and UC is not a reason to start or resume smoking. Similarly, observational associations involving appendectomy, diet, or medication exposure do not establish a preventive prescription for an individual. Ask about tobacco, recent antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), infections, travel, and family history without blaming the patient or presenting one exposure as the proven cause. (Ko et al., 2019; Rubin et al., 2025).

Record extent anatomically: **proctitis** is confined to the rectum; **left-sided colitis** extends proximally but not beyond the splenic flexure; **extensive colitis** extends beyond that landmark. Extent informs topical drug delivery and cancer-risk assessment, whereas activity describes how inflamed the bowel is now. A small affected area can still cause severe symptoms and major disability. Severe refractory proctitis should not be dismissed merely because it is not pancolitis. (Rubin et al., 2025).

Recognize activity and exclude mimics

Ask about stool frequency relative to the patient's usual baseline, visible blood, urgency, nighttime awakening, incontinence, and the ability to defer defecation. Include fatigue, appetite, weight change, fever, abdominal pain, and missed work or school. Proctitis may present mainly with bleeding and tenesmus rather than frequent loose stools; some patients have constipation. A description such as "three stools" loses important information when it omits blood, nocturnal symptoms, or an inability to leave the bathroom. (Rubin et al., 2025; Turner et al., 2021).

The initial examination assesses hydration, perfusion, abdominal tenderness and distension, fever, and the consequences of anemia or malnutrition. Marked distension, worsening tenderness, tachycardia, hypotension, or altered mentation must redirect attention to urgent complications. Severe abdominal pain is not simply a symptom to suppress. Perianal fistulas, prominent small-bowel disease, or other atypical features should prompt specialist evaluation for

Crohn's disease or another diagnosis. (Kucharzik et al., 2025; Rubin et al., 2025).

At diagnosis and during a suspected flare, consider enteric infection, especially *Clostridioides difficile*. Stool studies are selected according to symptoms, epidemiology, travel, and exposures. Infection and UC can coexist. A positive test therefore needs clinical interpretation, and an established UC diagnosis does not make every episode of diarrhea an inflammatory flare. In severe or steroid-refractory colitis, mucosal biopsies may be needed to evaluate cytomegalovirus (CMV); a routine stool panel is not equivalent to that tissue assessment. (Kucharzik et al., 2025; Rubin et al., 2025).

Diagnostic confirmation generally combines the history with ileocolonoscopy and biopsies from affected and apparently unaffected areas. Histology supports chronic inflammatory disease and helps evaluate mimics. During severe acute colitis, limited flexible sigmoidoscopy with cautious technique is usually more appropriate than a routine full colonoscopy with aggressive bowel preparation. Laboratory assessment commonly includes a complete blood count, renal function, electrolytes, liver tests, albumin, and C-reactive protein (CRP), together with stool inflammatory markers when clinically useful. (Kucharzik et al., 2025; Rubin et al., 2025).

Do not equate a normal CRP with absence of colonic inflammation. Some patients produce little systemic CRP despite active mucosal disease. Albumin is influenced by inflammation, intestinal protein loss, nutrition, and other illnesses; it is not a stand-alone nutrition score. Hemoglobin, ferritin, transferrin saturation, symptoms, and inflammatory context help distinguish iron deficiency from other contributors to anemia. The trend and the clinical setting often matter more than a single isolated value. (Hashash et al., 2024; Singh et al., 2023).

Assess disease severity across several dimensions: symptoms, objective inflammation, previous hospitalizations, steroid exposure, treatment failures, and functional impact. Deep ulceration, extensive disease, high inflammatory burden, or recurrent steroid need can indicate a more difficult course. Stool count alone is not an adequate severity classification. Documenting both extent and activity makes referral and handoff more useful than a nonspecific label such as "moderate UC." (Ko et al., 2019; Rubin et al., 2025).

Define targets before choosing a drug

A treatment target is useful only when the team and patient know how it will be measured and what will happen if it is not reached. Induction aims to control active disease; maintenance aims to preserve remission without ongoing corticosteroids. A prescription should therefore be accompanied by an early clinical review, a plan for objective reassessment, and a maintenance strategy. The time required for response varies by therapy and severity; deterioration should never wait for the end of a routine induction window. (Rubin et al., 2025; Singh et al., 2024).

The near-term goals are resolution of rectal bleeding, movement toward the patient's normal bowel pattern, improved urgency, and recovery of everyday function. Long-term goals include sustained steroid-free remission and endoscopic healing, with prevention of hospitalization, surgery when avoidable, and disability. Figure 2 presents these as complementary domains rather than a ladder on which symptoms become unimportant after the first step. (Rubin et al., 2025; Turner et al., 2021).

Be precise about endoscopic terminology. The 2025 American College of Gastroenterology (ACG) guideline uses a Mayo endoscopic subscore of 0 or 1 for *endoscopic improvement*. A score of 0 represents a more stringent absence of visible activity. Calling every score of 1 "complete healing" obscures residual disease. Reports should state the instrument and actual score, along with extent and relevant findings, rather than relying on an unqualified phrase such as "mucosa looks better." (Rubin et al., 2025).

STRIDE-II separates short- and intermediate-term measures from longer-term outcomes and emphasizes quality of life and absence of disability. Histologic healing adds information about depth of remission but is not a universal stand-alone target that automatically requires changing an otherwise effective drug. Shared decisions must weigh the potential benefit of deeper control against infection risk, adverse effects, cost, inconvenience, and the possibility of losing a therapy that is working. (Rubin et al., 2025; Turner et al., 2021).

The patient's goal should be concrete: sleeping through the night, traveling without fear of incontinence, attending class, or tapering prednisone without another relapse. Measure that goal alongside disease activity. A negative stool test does not restore confidence by itself, and good quality of life does not prove absence of

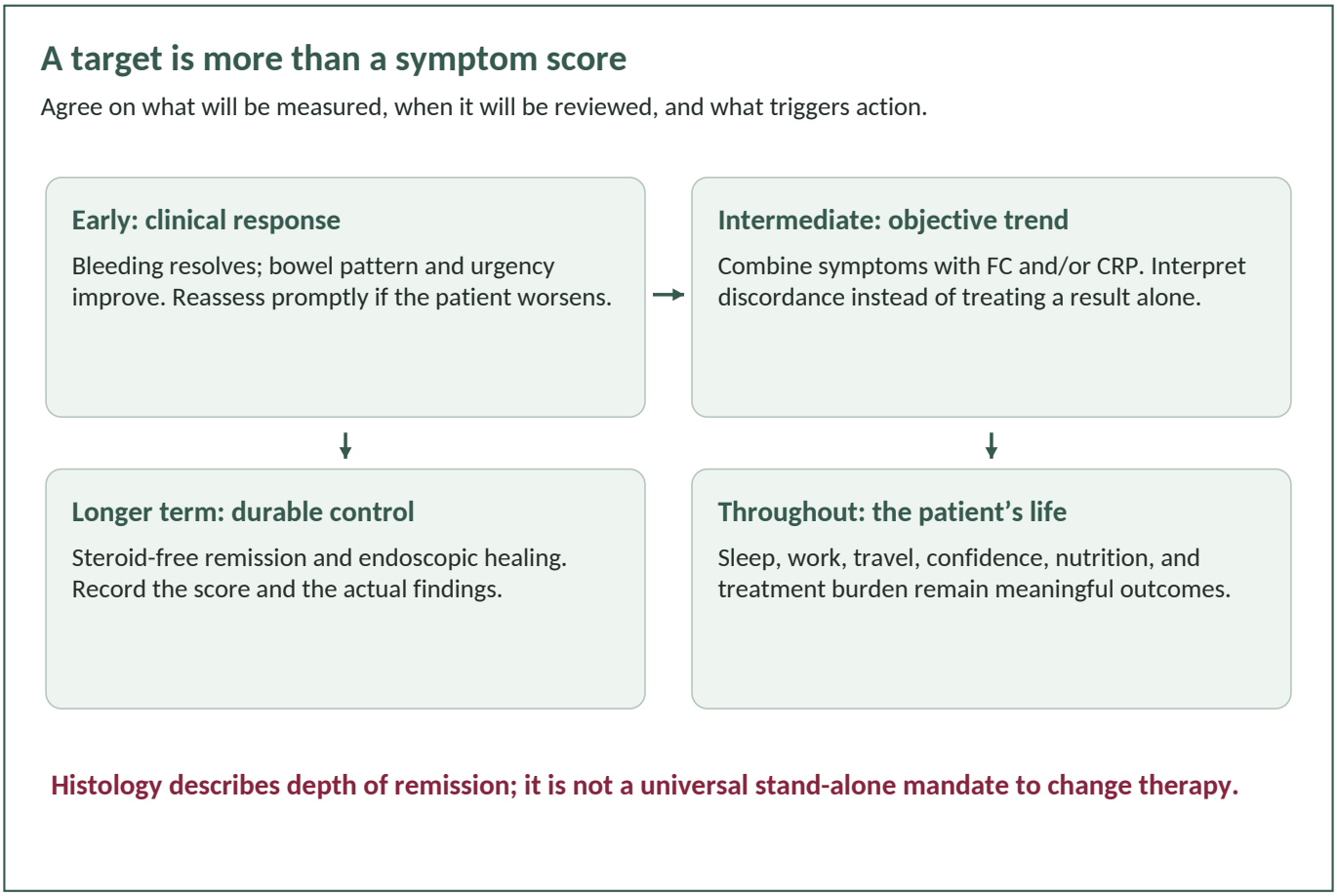


Figure 2. Complementary treatment-target domains. Timing depends on the therapy, severity, and clinical trajectory; the map does not impose a fixed calendar for every patient. Sources: Turner et al., 2021; Rubin et al., 2025.

inflammation. Both deserve attention. Record the agreed target, monitoring method, review interval, responsible clinician, and escalation contact in the same plan. (Turner et al., 2021).

Make biomarkers useful, not absolute

Fecal calprotectin (FC) reflects neutrophil-associated intestinal inflammation and is useful for following UC over time. It is not specific for UC and is not a cancer-screening test. Enteric infection, some medications, assay differences, sampling variation, and the clinical context can affect interpretation. When possible, follow serial results using the same laboratory method and compare them with prior endoscopic findings. A trend from a high baseline is informative, but improvement alone is not the same as reaching the agreed target. (Singh et al., 2023).

The American Gastroenterological Association (AGA) recommends combining symptoms with biomarkers rather than relying on symptoms alone. In a patient in symptomatic remission, **FC below 150 µg/g**, normal fecal lactoferrin, and/or normal CRP can help rule out active inflammation and avoid routine endoscopy solely to assess current activity. This is a decision threshold for a defined scenario, not a universal definition of histologic remission. Normal results do not remove a separate indication for dysplasia surveillance. (Singh et al., 2023).

In symptomatic remission with elevated biomarkers, endoscopic assessment is generally preferred to an automatic empirical treatment change. In patients with moderate-to-severe symptoms but normal biomarkers, endoscopic evaluation also helps resolve the discrepancy. With mild symptoms, the AGA favors endoscopic assessment regardless of whether the biomarker is normal or elevated. Figure 3 summarizes this logic. Acute toxicity, suspected infection, or another urgent indication takes precedence over this outpatient monitoring framework. (Singh et al., 2023).

Moderate-to-severe symptoms accompanied by elevated inflammatory markers can support a treatment decision without routine endoscopy merely to prove inflammation again, provided the diagnosis and clinical context are clear. That does not eliminate endoscopy when needed to assess complications, obtain biopsies, investigate a different diagnosis, or guide a major

Read symptoms and biomarkers together

Outpatient monitoring in established UC—not an emergency triage or cancer-surveillance rule.

CLINICAL STATE	BIOMARKER RESULT	NEXT INTERPRETIVE STEP
Symptomatic remission	FC <150 µg/g	May avoid routine endoscopy solely for current activity.
Symptomatic remission	Elevated biomarkers	Clarify with endoscopic assessment; do not escalate automatically.
Mild symptoms	Normal or elevated	Endoscopic assessment helps guide the treatment decision.
Moderate–severe symptoms	Normal biomarkers	Resolve the discrepancy with endoscopic evaluation.
Moderate–severe symptoms	Elevated biomarkers	May support a treatment decision without a routine activity-only scope.

Toxicity, infection, complications, biopsy needs, or dysplasia surveillance can create separate indications.

Figure 3. Symptom–biomarker interpretation using the AGA framework. FC: fecal calprotectin. The <150 µg/g threshold applies to the specified remission scenario, not every clinical question. Sources: Singh et al., 2023.

change. Avoid translating an algorithm into "scope everyone" or "never scope when FC is high." The question the test must answer should determine the investigation. (Singh et al., 2023).

During stable remission, biomarker monitoring at approximately 6–12-month intervals is a reasonable guideline-supported framework; active disease, a treatment change, or a concerning trend warrants earlier assessment. Arrange who will review the result and how the patient will be contacted. A test ordered but not collected, an abnormal value filed without action, and a message not received are different failures. Nurses can close these gaps by tracking completion and confirming the follow-up plan. (AGA, 2023; Singh et al., 2023).

CASE 1 · Feeling well is not the whole assessment

Maria, 36, reports one or two formed stools daily and no visible blood. Her FC has risen from 70 to 480 µg/g on the same assay. She is afebrile and has no acute abdominal findings. She asks whether the result can be ignored because she feels well. (Singh et al., 2023).

The result is a signal to investigate, not an instruction to double treatment. The team reviews medication access and adherence, recent infection or NSAID exposure, and the reliability of the specimen, then arranges appropriate endoscopic assessment. A repeat test may help clarify an unexpected result, but repeated testing should not become indefinite avoidance of evaluation. (Singh et al., 2023).

The nursing explanation is: "Your symptoms have improved, but this test suggests inflammation may still be present. We need to clarify that before changing a treatment that may be helping." This preserves shared decision-making while avoiding both false reassurance and unverified escalation. (Singh et al., 2023).

Endoscopy, histology, and imaging

Endoscopy directly assesses visible mucosal disease and enables biopsy. The Mayo endoscopic subscore is familiar in routine practice; the Ulcerative Colitis Endoscopic Index of Severity provides a different structured assessment. They are not interchangeable

scales. The report should identify which was used and describe ulceration, bleeding, vascular pattern, and extent. Serial comparison is more useful when terminology, scoring, and documentation are consistent. (Kucharzik et al., 2025; Rubin et al., 2025).

Biopsies can show active microscopic inflammation despite a reassuring endoscopic appearance. Histologic activity is associated with less favorable outcomes, but an association does not prove that escalating every patient solely to normalize a microscopic score improves net outcomes. The Nancy index and Robarts Histopathology Index are structured approaches, not a license to treat a number in isolation. Persistent histologic activity should lead to review of the entire course, adherence, objective trends, cancer risk, and patient preferences. (Rubin et al., 2025; Turner et al., 2021).

Microscopic remission does not require erasing every chronic architectural change. Absence of active neutrophilic inflammation and complete histologic normalization are distinct concepts. Similarly, a pathology report of chronic inactive colitis need not mean ongoing acute injury. Ask the reporting team to clarify terminology when it would change treatment. Communicating the exact histologic finding prevents an unnecessary alarm over a word such as “chronic” or inappropriate reassurance when active inflammation is present. (Kucharzik et al., 2025).

Intestinal ultrasound (IUS) is increasingly used for noninvasive assessment and monitoring where trained operators and reliable services are available. It can assess bowel-wall thickness and vascularity and support early response assessment. It does not provide mucosal biopsies, replace dysplasia-surveillance colonoscopy, or establish histologic remission. Rectal assessment and operator dependence remain practical limitations. CT or MRI has a different role when complications or an alternative diagnosis are suspected; repeated CT is not the default tool for routine remission monitoring. (Kucharzik et al., 2025; Rubin et al., 2025).

When symptoms persist despite objective control, consider residual anorectal dysfunction, disorders of gut–brain interaction, medication effects, infection, diet-related symptoms, and other causes guided by the history. A reassuring inflammatory evaluation should open this assessment rather than end the consultation. Empirically adding immunosuppression to treat every remaining symptom exposes the patient to harm without proving an inflammatory mechanism. Conversely, labeling symptoms “functional” before evaluating new bleeding or nocturnal diarrhea risks missing active disease. (Rubin et al., 2025; Singh et al., 2023).

Optimize mild-to-moderate treatment

For appropriately selected mild-to-moderate UC, mesalamine and other 5-aminosalicylates (5-ASAs) remain central. The route must match the diseased segment. A suppository treats the rectum; an enema can cover more distal colon, with reach and retention varying between patients. Oral therapy provides broader delivery. Oral and rectal mesalamine together can improve control in left-sided or extensive disease. Asking whether a patient can retain an enema is as important as documenting that it was prescribed. (Ko et al., 2019; Rubin et al., 2025).

For mild-to-moderate proctitis, the ACG recommends rectal mesalamine 1 g daily for induction, with an appropriate maintenance plan. Left-sided disease often warrants rectal mesalamine at least 1 g daily together with oral mesalamine at least 2 g daily. Product-specific U.S. labeling may differ from guideline regimens. For example, Lialda delayed-release tablets are labeled for adult induction at 2.4–4.8 g once daily and maintenance at 2.4 g daily. Table 1 separates these examples rather than treating every formulation as identical. (Rubin et al., 2025; Takeda, 2026).

Verify the exact formulation, dose, route, instructions about food, and whether the preparation can be divided or crushed. Lialda tablets are swallowed whole and taken with food. Different release systems should not be casually exchanged milligram-for-milligram without checking the product instructions. Once-daily oral regimens can support adherence when suitable. Before declaring treatment failure, explore affordability, refill gaps, tolerability, administration technique, and whether topical therapy was actually used. (Ko et al., 2019; Takeda, 2026).

Mesalamine can cause renal injury, including interstitial nephritis. Assess renal function before therapy and periodically thereafter; a rising creatinine requires evaluation rather than routine continuation. Review dehydration and concomitant nephrotoxic drugs, including NSAIDs. Acute mesalamine intolerance can resemble a flare, with cramping, abdominal pain, bloody diarrhea, fever, headache, or rash. Suspected intolerance calls for prompt prescriber assessment and discontinuation according to the treatment plan, not uncritical dose escalation. (Takeda, 2026).

Table 1. Match conventional treatment to disease and formulation

Clinical use	Adult regimen example	Administration or safety distinction
Mild–moderate proctitis	Rectal mesalamine 1 g daily (ACG induction recommendation).	Confirm the suppository was used and can be retained; plan maintenance.
Mild–moderate left-sided disease	Rectal mesalamine ≥1 g/day plus oral mesalamine ≥2 g/day (ACG).	Topical delivery complements oral treatment; assess technique and tolerability.
Oral mesalamine: Lialda	U.S. induction: 2.4–4.8 g once daily. Maintenance: 2.4 g once daily.	Take with food; swallow whole. Assess renal function before and during treatment.
Budesonide extended-release: Uceris	U.S. adult induction: 9 mg each morning for up to 8 weeks.	Swallow whole. Check CYP3A4 interactions and grapefruit; not routine maintenance.

Selected adult examples, not a complete prescribing guide. Guideline regimens and U.S. product labels are distinguished. Verify organ function, interactions, and local authorization. Sources: Rubin et al., 2025; Takeda, 2026; Salix, 2024.

Sulfasalazine can be useful in selected patients, including those with associated arthritic symptoms, but the sulfapyridine component contributes to intolerance. Headache, nausea, rash, hepatic injury, and blood dyscrasias require appropriate assessment and laboratory monitoring. Folate supplementation is relevant because sulfasalazine affects folate metabolism. These considerations distinguish it from mesalamine; a history of tolerating one does not establish that the other needs no safety review. (Ko et al., 2019).

If optimized 5-ASA treatment is inadequate, budesonide MMX or systemic corticosteroids may be used for induction according to disease activity and specialist judgment. The U.S. Uceris extended-release tablet regimen is 9 mg orally each morning for up to eight weeks; swallow whole. Controlled ileal-release budesonide is not the same colonic-delivery product. CYP3A4 inhibitors and grapefruit can increase exposure. “Locally acting” does not mean free of infection risk, adrenal effects, or systemic toxicity. (Salix, 2024).

Neither oral nor rectal corticosteroids are maintenance treatment. The taper after systemic steroid use must reflect the clinical course, duration, prior exposure, and adrenal risk; it is not a universal schedule copied from an old handout. Repeated rescue courses, relapse during tapering, or inability to stop prednisone indicate a need to reconsider the maintenance strategy. Monitor glucose, blood pressure, sleep, mood, infection symptoms, and bone-health risk during relevant exposure. (Rubin et al., 2025; Salix, 2024).

CASE 2 · A route problem can look like drug failure

James, 29, has inflammation confined to the rectum. He reports persistent urgency and small amounts of blood despite taking his oral mesalamine. His vital signs are normal, and infection assessment is unrevealing. He never started the prescribed suppository because he thought oral and rectal medication were duplicates. (Ko et al., 2019; Rubin et al., 2025).

The team explains that topical treatment places medication directly at the inflamed site. The nurse reviews administration privately, explores discomfort and practical barriers, and confirms the agreed regimen and review date. Oral adherence alone did not establish that the intended treatment had been optimized. (Ko et al., 2019; Rubin et al., 2025).

Persistent symptoms after an adequate, correctly used regimen still require reassessment and possible escalation. This example is not permission to delay referral when bleeding worsens, systemic symptoms appear, or severe activity is found. (Ko et al., 2019; Rubin et al., 2025).

Escalate toward steroid-free control

Moderate-to-severe activity, high-risk disease, or failure of optimized conventional therapy may warrant early advanced treatment. Current AGA guidance favors an early advanced-therapy approach over prolonged gradual escalation through ineffective 5-ASAs in appropriate patients. A history of repeated prednisone courses should not be normalized because each course temporarily helps. The maintenance plan should aim to remove steroid dependence, not simply issue the next taper. (Singh et al., 2024).

Available mechanisms include tumor necrosis factor (TNF) blockade, inhibition of gut-selective lymphocyte trafficking, interleukin-12/23 or selective interleukin-23 blockade, Janus kinase (JAK) inhibition, and sphingosine-1-phosphate (S1P) receptor modulation. These act at different points in immune activation and cell movement (Figure 4). A therapy's mechanism is not a complete safety profile, and its success in another inflammatory disease does not automatically establish a UC indication or the correct UC dose. (Singh et al., 2024).

The choice considers previous advanced-therapy exposure, likelihood of response, needed speed of control, comorbidities, extraintestinal disease, pregnancy plans, infection history, and the route the patient can sustain. Access, insurance authorization, infusion travel, refrigeration, and injection ability can determine whether a theoretically excellent plan is executable. Discuss these before the first dose rather than discovering an unaffordable maintenance regimen after induction has begun. (Rubin et al., 2025; Singh et al., 2024).

The AGA groups therapies by comparative efficacy in advanced-therapy-naïve and previously exposed populations. Many comparisons are indirect and carry uncertainty. They should inform discussion, not become a universal ranking that disregards a patient's risks or a drug's local label. For example, a high-efficacy oral option may be unsuitable for someone with a substantial thrombotic risk. The 2024 living recommendations remained unchanged after the AGA's March 2026 evidence review. (AGA, 2026; Singh et al., 2024).

Table 2 identifies the major classes and practical safety distinctions. Biological agents can have intravenous induction and subcutaneous maintenance formulations, depending on the exact product. Approved subcutaneous infliximab or vedolizumab maintenance should not be treated as interchangeable with every intravenous induction or intensified regimen. Likewise, a biosimilar is an approved biological alternative, but product, indication, route, strength, and transition instructions still require verification. (Rubin et al., 2025; Singh et al., 2024).

Safer biologics and immunomodulators

Infliximab, adalimumab, and golimumab inhibit TNF. Infliximab is an important option for outpatient disease and hospitalized steroid-refractory acute severe UC. The U.S. standard intravenous adult UC regimen is 5 mg/kg at weeks 0, 2, and 6, then every eight weeks; rescue intensification is a separate specialist decision, not an automatic substitute for this schedule. Pretreatment screening includes tuberculosis and hepatitis B assessment, and active serious infection requires evaluation before treatment. (Janssen Biotech, 2025; Rubin et al., 2025).

During an infusion, recognize fever, urticaria, chest tightness, dyspnea, hypotension, or other features of a reaction. Follow the local infusion-reaction pathway, stop or interrupt the infusion as indicated, assess airway and circulation, and obtain urgent assistance for severe reactions. Do not simply label new breathlessness "anxiety." Anti-TNF selection also requires attention to heart failure, demyelinating disease, prior malignancy, and cumulative immunosuppression. Infliximab doses above 5 mg/kg are contraindicated in moderate or severe heart failure under U.S. labeling. (Janssen Biotech, 2025).

Vedolizumab blocks α4β7-mediated gut lymphocyte trafficking. Ustekinumab targets the p40 subunit shared by interleukin-12 and interleukin-23. Mirikizumab, risankizumab, and guselkumab selectively target interleukin-23 p19. These are distinct mechanisms and products, not different names for the same regimen. Gut selectivity or a favorable comparative infection profile does not mean that fever, serious infection, hypersensitivity, or other warnings can be ignored. (Singh et al., 2024).

Liver monitoring is especially important with the selective interleukin-23 products that carry specific inflammatory-bowel-disease hepatotoxicity precautions. For risankizumab and mirikizumab, U.S. prescribing information calls for liver enzymes and bilirubin before therapy and monitoring during induction according to the product schedule. New jaundice, dark urine, marked fatigue, nausea, or unexplained abdominal symptoms

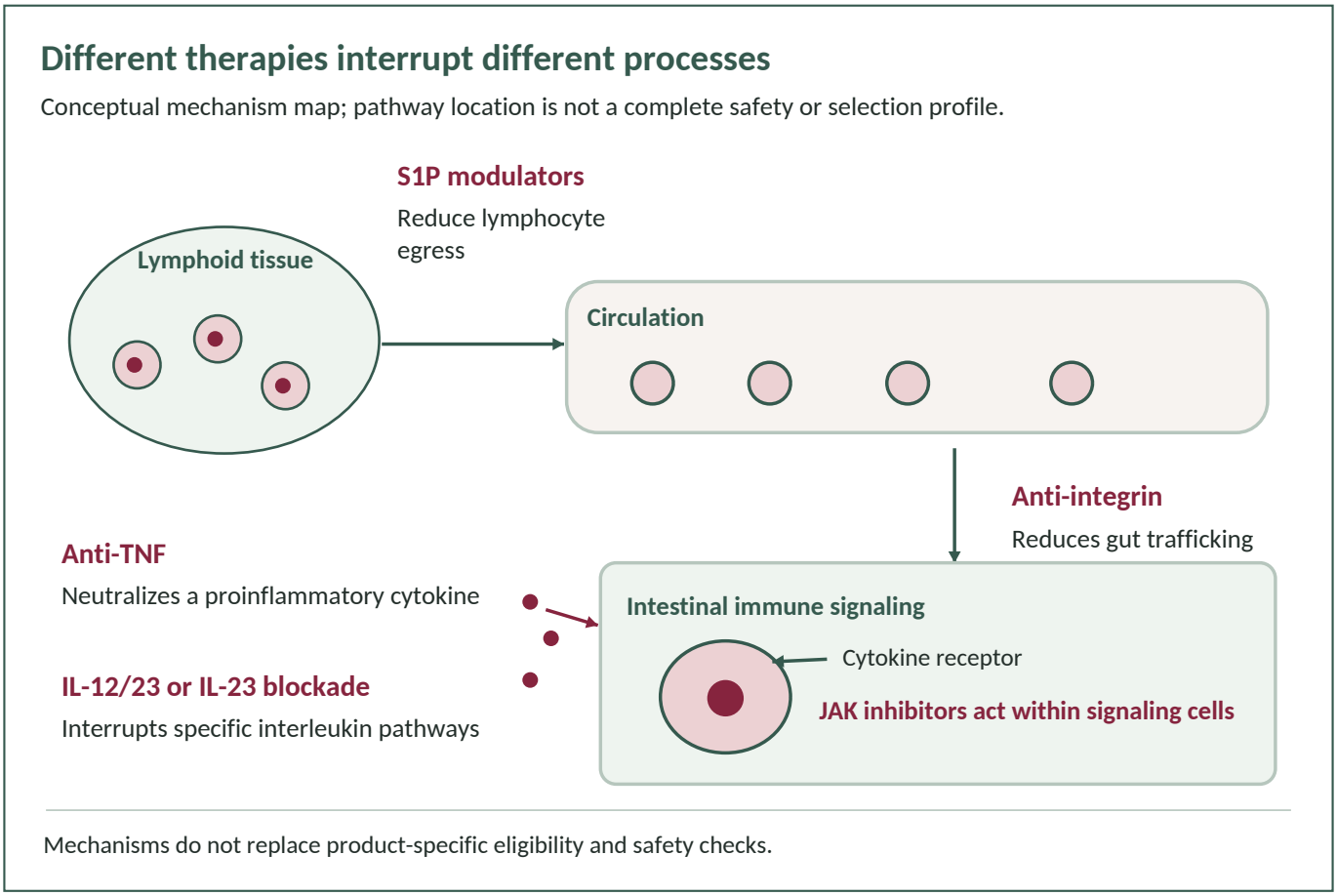


Figure 4. Selected immune processes targeted by advanced therapies. S1P: sphingosine-1-phosphate; TNF: tumor necrosis factor; IL: interleukin; JAK: Janus kinase. Original conceptual schematic, not a complete pathway model. Sources: Singh et al., 2024; Pfizer, 2025; AbbVie, 2026a.

require assessment. Do not substitute a psoriasis dosing or monitoring handout for the UC regimen. (AbbVie, 2026b; Eli Lilly, 2025).

Thiopurines such as azathioprine are too slow and are not recommended as monotherapy to induce remission in active moderate-to-severe UC. They may have a maintenance role in selected patients and can be combined with infliximab to improve treatment durability. Combination benefit must be weighed against infection and malignancy risks. Methotrexate monotherapy is not recommended for UC induction or maintenance. In the United States, azathioprine treatment of UC is guideline-supported off-label practice, not an FDA-approved UC indication. These distinctions matter when reconciling a regimen inherited from another inflammatory diagnosis. (Legacy Pharma USA, 2026; Rubin et al., 2025; Singh et al., 2024).

Before a thiopurine, assess thiopurine methyltransferase (TPMT) status and, where appropriate, NUDT15-related risk, along with baseline blood counts and liver tests. Genotype or enzyme testing does not replace ongoing laboratory monitoring. Unexplained leukopenia, recurrent infection, bruising, jaundice, or severe abdominal pain warrants prompt review. Check interactions, particularly allopurinol or febuxostat; specialist low-dose thiopurine–allopurinol strategies are not routine unsupervised coadministration. (Legacy Pharma USA, 2026; Singh et al., 2024).

For every advanced agent, confirm the actual dose administered, next due date, screening results, vaccine plan, laboratory schedule, and who will respond to abnormal results. A drug listed on a medication record may never have been started because authorization failed. A well-designed handoff distinguishes intended therapy from therapy actually received and makes missed maintenance doses visible before relapse occurs. (Rubin et al., 2025; Singh et al., 2024).

Small molecules: high-value safety distinctions

Tofacitinib and upadacitinib are oral JAK inhibitors. Rapid activity and avoidance of infusions can be attractive, but oral administration does not imply low risk. U.S. boxed warnings address serious infection, mortality, malignancy, major adverse cardiovascular

Table 2. Advanced therapies: mechanism is only the starting point

Class and examples	Therapeutic distinction	Priority safety and delivery checks
Anti-TNF Infliximab, adalimumab, golimumab	Cytokine blockade; infliximab also has an ASUC rescue role.	TB/HBV assessment, serious infection, reactions; consider heart failure and demyelination.
Anti-integrin Vedolizumab	Blocks α4β7-mediated gut trafficking.	Verify induction vs maintenance route and exact product; assess infection and reactions.
IL-12/23 Ustekinumab	Targets the shared p40 subunit.	Confirm induction and maintenance instructions, infection screening, and hypersensitivity precautions.
Selective IL-23 Mirikizumab, risankizumab, guselkumab	Targets p19; not interchangeable with p40 blockade.	Use the UC regimen. Check label-specific liver monitoring, infection, and hypersensitivity precautions.
JAK inhibitors Tofacitinib, upadacitinib	Oral intracellular signaling inhibition; eligibility differs by product.	Boxed warnings; CBC, liver tests, lipids, TB/hepatitis, interactions, pregnancy, and glucose in diabetes.
S1P modulators Ozanimod, etrasimod	Reduce lymphocyte egress; oral schedules differ.	Cardiac/conduction, liver, infection, eye, interaction, vaccine, and fetal-risk assessment.

These are class-level reminders, not complete contraindication lists. Use the current product label and individual benefit–risk assessment. Sources: Singh et al., 2024; Rubin et al., 2025; Janssen Biotech, 2025; Pfizer, 2025, 2026; AbbVie, 2026a, 2026b; Eli Lilly, 2025.

events, and thrombosis. Much of the comparative safety signal arose in an older rheumatoid arthritis population with cardiovascular risk factors; it should inform individualized UC decisions without pretending that the exact event rates transfer unchanged to every person with UC. (AbbVie, 2026a; Pfizer, 2026).

U.S. indications are not identical. Tofacitinib is indicated for adults with moderate-to-severe UC after inadequate response or intolerance to one or more TNF blockers. Current upadacitinib labeling also allows use when TNF blockers are clinically inadvisable, provided the patient has received at least one approved systemic therapy. Confirm the actual product's eligibility language rather than applying an obsolete class-wide rule. Neither is recommended in combination with biological UC therapies or potent immunosuppressants such as azathioprine or cyclosporine. (AbbVie, 2026a; Pfizer, 2026).

For an adult without dose-modifying organ dysfunction or interactions, tofacitinib immediate-release induction is 10 mg twice daily for eight weeks. An inadequate response may justify extension to a maximum of 16 weeks, with discontinuation if adequate benefit is not achieved. Maintenance is generally 5 mg twice daily; higher-dose maintenance is reserved for selected situations and the shortest appropriate duration. Extended-release strengths and conversion instructions differ. Never assume that a maintenance dose is the normal induction dose. (Pfizer, 2026).

Standard adult upadacitinib induction for UC is 45 mg once daily for eight weeks; maintenance is usually 15 mg daily, with 30 mg considered for refractory, severe, or extensive disease. Use the lowest effective maintenance dose. Renal or hepatic impairment and strong CYP3A4 inhibitors can change the regimen; strong inducers may reduce effectiveness. Do not copy the longer Crohn's induction duration into a UC prescription. Extended-release tablets are swallowed whole. (AbbVie, 2026a).

Review tuberculosis, viral hepatitis, vaccination status, blood counts, liver enzymes, lipids, pregnancy potential, cardiovascular risk, prior thrombosis, cancer history, and smoking exposure. Laboratory thresholds and interruption rules are product-specific. The June 2026

U.S. JAK-label updates also highlight hypoglycemia in patients with diabetes: consider more frequent glucose monitoring and teach symptom recognition. Falling glucose during steroid tapering has other possible causes too; medication reconciliation must consider the full regimen. (AbbVie, 2026a; Pfizer, 2026).

Ozanimod and etrasimod modulate S1P receptors, reducing lymphocyte egress from lymphoid tissue. Etrasimod's U.S. regimen is 2 mg once daily, whereas ozanimod uses a product-specific titration and maintenance schedule. Before etrasimod, review an electrocardiogram, blood count, liver tests, immunizations, interacting drugs, and a baseline fundus examination including the macula. Monitor for infection, bradycardia, hypertension, liver injury, and visual change. Ophthalmic screening should not be omitted simply because the patient reports normal vision. (Pfizer, 2025; Singh et al., 2024).

Etrasimod is contraindicated with certain recent major cardiovascular events and important conduction disorders without a functioning pacemaker. Medications that slow heart rate or atrioventricular conduction merit special attention. Fetal-risk counseling and contraception instructions are product-specific, as are vaccine and post-discontinuation precautions. The oral agents should not be presented as freely interchangeable alternatives to a biological infusion. (Pfizer, 2025).

CASE 3 · Convenience does not settle the safety decision

Robert, 67, has active UC after loss of response to an anti-TNF agent. He prefers a tablet. His history includes pulmonary embolism, coronary disease, diabetes treated with insulin, and repeated prednisone exposure. (AbbVie, 2026a; Pfizer, 2026).

The prescriber and patient must compare the potential efficacy of a JAK inhibitor with his thrombotic and cardiovascular risks and suitable alternatives. The nurse should make those risks visible before treatment starts rather than assuming that prior anti-TNF failure makes any oral agent appropriate. (AbbVie, 2026a; Pfizer, 2026).

If a JAK inhibitor is selected after specialist review, the plan must include the correct induction and maintenance doses, laboratory follow-up, infection and clot warning symptoms, and closer glucose attention. A new glucose of 54 mg/dL with sweating requires immediate hypoglycemia management under protocol and prompt regimen review; it is not a routine symptom to wait out. (AbbVie, 2026a; Pfizer, 2026).

Verify response before declaring failure

Review early symptom response and adverse effects, then reassess objective inflammation at a time appropriate to the drug and clinical trajectory. For outpatient treatment, the ACG recommends response assessment within eight weeks, but a worsening patient needs earlier action. Schedule reassessment when treatment begins. Clinical improvement, steroid withdrawal, biomarker change, and endoscopic findings answer different questions; recording each prevents an early symptomatic response from being mistaken for durable remission. (Rubin et al., 2025).

Before changing an advanced therapy, confirm that inflammation is active and that the intended induction, maintenance, and adherence actually occurred. Consider infection, incorrect administration, inadequate exposure, accelerated drug clearance in severe inflammation, immunogenicity, and a noninflammatory cause of symptoms. "The biologic failed" is not a complete assessment when the patient missed two doses or never received the prescribed induction sequence. (Rubin et al., 2025; Singh et al., 2024).

Reactive therapeutic drug monitoring is particularly useful with anti-TNF loss of response. A low trough without important antidrug antibodies may support an exposure-optimization strategy; substantial antibodies or active disease despite an adequate concentration may favor a different approach. Results depend on the assay, timing, phenotype, and clinical goal. There is no single universal trough target for every drug and every situation. Routine proactive testing in all asymptomatic patients and extrapolation to every newer biologic are not equally supported. (Rubin et al., 2025).

Do not withdraw a successful maintenance therapy solely because a patient feels well. De-escalation decisions consider remission duration and depth, prior course, toxicity, combination therapy, and the consequences of relapse. Conversely, indefinite continuation of an ineffective drug while repeatedly prescribing steroids is not a treat-to-target strategy. Explain the reason for either course and ensure that the next monitoring step is explicit. (Singh et al., 2024).

Acute severe UC: protect the rescue window

Acute severe ulcerative colitis (ASUC) is a hospital-level emergency, classically characterized by at least six bloody stools daily plus systemic toxicity such as fever, tachycardia, anemia, or an elevated inflammatory marker. The exact score is less important than recognizing a deteriorating patient promptly. Dehydration, electrolyte disturbance, severe anemia, infection, venous thromboembolism, toxic megacolon, perforation, and hemorrhage require active surveillance. Figure 5 organizes the early hospital priorities. (Rubin et al., 2025).

Initial management includes gastroenterology involvement, early colorectal surgical awareness, stool testing for C. difficile, blood tests, fluid and electrolyte correction, and assessment for colonic dilation or another complication. Flexible sigmoidoscopy should occur within 72 hours, preferably within 24 hours, with biopsies for CMV when appropriate. Avoid routine complete colonoscopy when the acutely inflamed, vulnerable colon makes that approach unsafe. Infection evaluation proceeds alongside a time-sensitive treatment plan. (Rubin et al., 2025).

The ACG recommends intravenous methylprednisolone totaling 60 mg daily or hydrocortisone 100 mg three or four times daily as initial corticosteroid treatment. These are alternative regimens, not instructions to combine both. Routine broad-spectrum antibiotics and total parenteral nutrition solely for bowel rest are not recommended without another indication. Nutrition should be individualized, preferably using the gut when feasible. Antibiotics may be essential when infection, sepsis, or a surgical complication is suspected. (Hashash et al., 2024; Rubin et al., 2025).

Pharmacologic venous-thromboembolism prophylaxis is recommended for hospitalized ASUC unless a patient-specific contraindication exists. Stable rectal bleeding caused by colitis does not automatically justify withholding prophylaxis. Major uncontrolled hemorrhage or another true contraindication requires an individualized plan. Nurses should identify omitted doses, refusal, or automatic hold parameters and clarify the reason rather than silently accepting an unprotected high-risk patient. (Rubin et al., 2025).

Document actual stool frequency and visible blood, vital signs, abdominal examination, intake and output, hydration, pain, mobility, and serial laboratory trends. Avoid NSAIDs, opioids, and anticholinergic or antimotility agents that can worsen colonic dilation or obscure deterioration. A fall in stool output accompanied by greater distension, tachycardia, and tenderness is not necessarily recovery; ileus or toxic megacolon may reduce stool passage while the patient becomes sicker. (Rubin et al., 2025).

Assess response to intravenous steroids by day 3. An inadequate response should trigger rescue therapy with infliximab or cyclosporine and a concurrent surgical decision, not several more days of unchanged observation. Rescue selection considers prior treatment, contraindications, albumin, anticipated maintenance therapy, local expertise, and patient preferences. Routine JAK-inhibitor rescue is not established as the default ACG pathway; specialized use requires evidence-aware expert judgment and should not delay necessary surgery. (Rubin et al., 2025).

Cyclosporine inhibits calcineurin and can serve as a rescue bridge rather than indefinite maintenance. This UC rescue use is guideline-supported and off-label in the United States. It requires close blood-level monitoring, renal function and electrolyte surveillance, blood-pressure checks, and interaction review. Tremor, confusion, seizures, rising creatinine, and hypertension are important toxicity signals. The team should identify the intended maintenance agent before discharge. Infliximab intensification in severe disease is likewise a specialist decision; low albumin can influence exposure, but it does not create one universal accelerated regimen. (Novartis, 2026; Rubin et al., 2025).

Toxic megacolon, perforation, severe refractory hemorrhage, or medically refractory deterioration can require urgent surgery. Do not postpone indicated surgery because the patient recently received infliximab or cyclosporine. Early discussion allows the patient to hear about options before exhaustion and instability make decision-making harder. Surgery is a treatment for disease and complications, not proof that the patient or team has failed. (Rubin et al., 2025).

CASE 4 · Day 3 is a decision point

Leah, 42, was admitted with ten bloody stools daily, pulse 112/min, temperature 38.2°C, and hemoglobin 9.7 g/dL. She has received the prescribed intravenous steroid regimen. On hospital day 3, she still has nine bloody stools, CRP remains elevated, and abdominal discomfort is increasing. (Rubin et al., 2025).

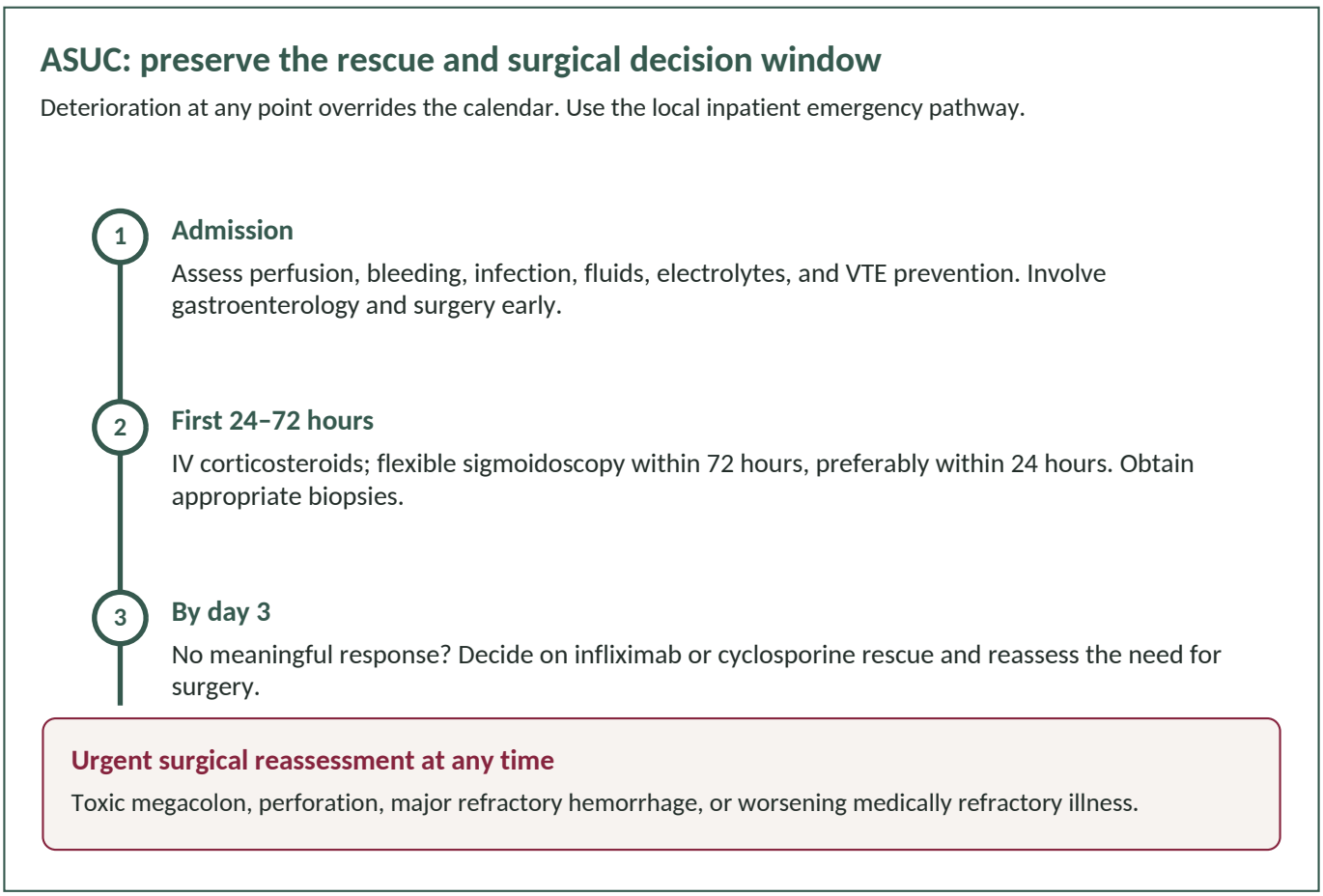


Figure 5. Early hospital priorities in acute severe ulcerative colitis. Inpatient treatment, monitoring, and rescue decisions require authorized orders and specialist assessment. Sources: Rubin et al., 2025.

> The nurse reports the trajectory directly: “There has been no meaningful improvement by day 3. We need the rescue and surgical review plan today.” The team reassesses infection and complications, reviews the endoscopy and biopsy findings, and decides between appropriate rescue therapy and surgery. (Rubin et al., 2025).

> If she subsequently passes fewer stools but develops worsening distension and tachycardia, that change requires urgent reassessment. It must not be documented as improvement merely because the stool count fell. Bowel activity, perfusion, examination, and systemic illness must be interpreted together. (Rubin et al., 2025).

Recovery, surgery, and long-term prevention

A discharge plan should explain the maintenance regimen, any steroid taper, laboratory and stool-test appointments, the first follow-up visit, and what to do if symptoms return. Teach the difference between routine contact, same-day assessment, and emergency care. Heavy bleeding, severe or increasing abdominal pain, distension, fainting, fever with systemic illness, chest pain, dyspnea, or unilateral leg swelling warrant prompt escalation. Include a direct contact pathway; “follow up with GI” is not an executable appointment. (Rubin et al., 2025).

Assess iron deficiency, anemia, weight loss, food restriction, and nutritional risk. Patients may avoid eating to reduce bowel movements and become progressively depleted. A dietitian can help preserve adequate energy, protein, and micronutrients while adapting food choices to tolerance. A generally Mediterranean-style eating pattern can support health, but no single diet has been shown to prevent flares in every adult with IBD. Do not promise that dietary change can replace effective UC medication. (Hashash et al., 2024).

Probiotics, curcumin, and other supplements should be discussed without dismissing the patient’s interest, but they should not substitute for proven therapy. Their preparations and evidence are heterogeneous, and supplement use should be included in medication reconciliation. Fecal microbiota transplantation for UC disease control is not routine care outside an appropriate

research setting; this is a different question from its role in selected recurrent C. difficile infection. Avoid selling a microbiome explanation as a proven individualized cure. (Ko et al., 2019).

Screen for extraintestinal problems, including inflammatory joint symptoms, painful red eyes or visual changes, skin lesions, and hepatobiliary disease. Eye pain or visual loss deserves urgent assessment rather than a routine future appointment. Fatigue may reflect active inflammation, anemia, disrupted sleep, depression, medication effects, or other illness. Normal bowel symptoms do not remove the need to ask about these domains. Primary sclerosing cholangitis (PSC) changes colorectal surveillance needs even when bowel symptoms are mild. (East et al., 2025; Rubin et al., 2025).

Inflammation control and cancer surveillance are complementary. The BSG colorectal surveillance guideline recommends risk assessment approximately eight years after symptom onset in colonic IBD, with PSC-associated colitis entering surveillance from diagnosis. Endoscopically confirmed isolated proctitis generally follows population screening rather than an IBD-specific surveillance program. Subsequent intervals depend on inflammation, disease extent, dysplasia, strictures, family history, and the local guideline. A low FC never cancels surveillance that is otherwise due. (East et al., 2025).

Review vaccinations before and during immunosuppressive treatment using the current local schedule and the specific drug label. Plan indicated nonlive vaccines, including infection-risk-appropriate vaccination, rather than assuming that treatment must wait until every routine vaccine is complete. Live-vaccine decisions require attention to the agent, degree of immunosuppression, and timing. Address smoking cessation, skin protection and relevant skin assessment, cervical screening where applicable, and bone health after substantial corticosteroid exposure. (Pfizer, 2025; Pfizer, 2026; Singh et al., 2024).

Patients considering colectomy need balanced education about staged surgery, an ileostomy, or an ileal pouch–anal anastomosis where suitable. Removing the colon treats colitis in that organ but does not guarantee freedom from pouchitis, cuffitis, extraintestinal disease, altered bowel function, or all future surveillance. Involve a colorectal surgeon and ostomy nurse early. Teach hydration and output monitoring, medication absorption concerns, skin care, and the signs of obstruction or dehydration appropriate to the operation. (East et al., 2025; Rubin et al., 2025).

Shared decisions and special populations

Ask what makes the current treatment difficult. Embarrassment about rectal administration, fear of cancer or infection, needle anxiety, limited transportation, work schedules, and cost can all alter adherence. Use teach-back: ask the patient to explain the difference between induction and maintenance, the next monitoring step, and when to call. Offer accessible written instructions and an interpreter when needed. A patient who cannot obtain a medicine does not have a knowledge deficit that can be solved by repeating the same advice. (Singh et al., 2024; Turner et al., 2021).

Older adults and people with multimorbidity need a deliberate comparison of uncontrolled disease risk with infection, cardiovascular, thrombotic, malignancy, and medication-interaction risks. Frailty and functional ability can matter more than age alone. Diabetes is relevant during both corticosteroid use and JAK-inhibitor treatment. Cognitive, visual, or dexterity limitations may make an injection device difficult to use; assess technique and support instead of assuming a prescription has solved delivery. (AbbVie, 2026a; Pfizer, 2026; Singh et al., 2024).

Pregnancy planning should begin before conception whenever possible. Active disease can threaten maternal and infant outcomes; reflexively stopping an effective maintenance biological therapy is not a safe general rule. The 2025 global consensus supports continuation of appropriate low-risk therapies and monoclonal antibodies through pregnancy and lactation, while small molecules require different avoidance and transition planning. Coordinate gastroenterology, obstetric care, medication-specific counseling, and the infant’s vaccination plan rather than providing one blanket rule for all immunosuppressants. (Mahadevan et al., 2025).

Children and adolescents need pediatric specialist pathways, attention to growth, puberty, nutrition, school participation, and age- or weight-specific medication approvals. Adult dose tables should not be extrapolated to them. In transition to adult care, ensure that disease extent, prior severe episodes, treatment exposure, vaccine history, and the patient’s ability to manage refills and follow-up are transferred, not just the current medication list. (Kucharzik et al., 2025; Turner et al., 2021).

CASE 5 · Healing does not erase the patient's burden

Aisha, 31, has no rectal bleeding, FC 65 µg/g, and a recent examination showing a Mayo endoscopic subscore of 0 with no active histologic inflammation. She still avoids travel because of urgency and reports poor sleep and fatigue. She is considering pregnancy and wonders whether a stronger drug or no drug would be better. (Mahadevan et al., 2025; Singh et al., 2023; Turner et al., 2021).

The team validates her symptoms, evaluates noninflammatory contributors and anemia or other causes of fatigue, and discusses supportive strategies. Escalating immunosuppression solely for urgency is not justified by the current inflammatory findings. Stopping maintenance treatment without a pregnancy plan is also inappropriate. (Mahadevan et al., 2025; Singh et al., 2023; Turner et al., 2021).

Her follow-up includes a functional goal she chooses, review of symptoms and objective monitoring, and preconception counseling. Success is not a contest between the patient's experience and laboratory results; it is a plan that responds to both. (Mahadevan et al., 2025; Singh et al., 2023; Turner et al., 2021).

Evolving strategies and clinical synthesis

Research is expanding precision treatment, barrier biology, histologic endpoints, intestinal ultrasound, and combinations of targeted therapies. Promising mechanisms and early trial results are not equivalent to a licensed indication or a proven improvement in long-term outcomes. Advanced combination treatment and molecular response signatures are not routine substitutes for careful clinical assessment. Distinguish a research proposal from established practice and use current local labels and specialist guidance when introducing a new treatment. (Kucharzik et al., 2025; Rubin et al., 2025; Singh et al., 2024).

The practical target is durable, steroid-free control that protects the bowel and restores the patient's life. Measure symptoms and function; check objective inflammation; distinguish infection, underexposure, toxicity, and noninflammatory symptoms; and choose the next action with the patient. For acute severe disease, the priority changes from routine outpatient monitoring to preserving the rescue window and recognizing surgical complications. Across both settings, nurses make treatment safer by noticing discordant findings, verifying what actually happened, and closing the loop on the next decision. (Rubin et al., 2025; Turner et al., 2021).

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