Innovative GI

Understanding & Managing Ulcerative Colitis

A Patient Guide to Ulcerative Colitis Care

What Is Ulcerative Colitis?

Ulcerative colitis (UC) is a chronic inflammatory bowel disease in which the immune system causes ongoing inflammation in the lining of the colon and rectum. Unlike Crohn's disease, which can affect any part of the digestive tract in a patchy pattern, UC inflammation is continuous and almost always starts in the rectum, extending upward through part or all of the colon. Common symptoms include bloody diarrhea, urgency, a persistent sense of needing to have a bowel movement (tenesmus), abdominal cramping, fatigue, and in more active disease, unintentional weight loss. UC typically follows a relapsing-remitting course — periods of active symptoms (flares) alternating with periods of little or no symptoms (remission).

What Causes It?

- **Immune system dysregulation.** The immune system mistakenly targets the lining of the colon, producing ongoing inflammation instead of only responding to genuine threats.
- **Genetics.** Having a first-degree relative with UC increases your risk, and over 200 genetic variations have been linked to the condition — many involved in keeping the gut's lining intact and regulating immune responses.
- **Gut microbiome imbalance.** People with UC tend to have less diverse gut bacteria, with fewer beneficial species; this imbalance is thought to help drive and sustain inflammation, though it's still unclear whether it's a cause, a consequence, or both.
- **Environmental factors** that aren't fully understood, including early antibiotic exposure, diet, and prior gastrointestinal infections. Interestingly, smoking is associated with somewhat lower UC risk (the opposite of its effect in Crohn's disease) — but given everything else smoking causes, this is never a reason to start or continue.
- **What UC is not caused by:** diet and stress don't cause UC, though both can influence how symptoms feel day to day once the disease is present.

How Ulcerative Colitis Is Treated

Treatment has two goals: calming an active flare (called induction) and then keeping the disease quiet long-term (called maintenance) — ideally reaching “mucosal healing,” meaning the colon lining looks normal on colonoscopy, not just that symptoms have improved, since this is linked to better long-term outcomes. A few terms come up below: a study “compared to a dummy pill (placebo)” means some patients got the real treatment and others a fake version, without knowing which, so researchers could isolate the treatment's true effect. A study that “combines” or “pools” results (sometimes called a meta-analysis) means researchers gathered data from several separate studies for a more reliable answer. A “boxed warning” is the strongest safety warning the FDA issues on a medication label. Finally, the “biologic” medications below are named for the specific part of the immune system each one blocks — they aren't different strengths of the same drug, but different approaches to calming inflammation, which is why one may work for you when another hasn't.

5-ASA (Aminosalicylate) Medications

Goal: reduce inflammation directly in the colon lining; typically first-line for mild-to-moderate UC.

- **Oral mesalamine** (e.g., Lialda, Apriso, Pentasa, Asacol HD) or **sulfasalazine**, taken daily.
  Evidence: As a group, 5-ASAs are significantly more effective than a dummy pill for both calming a flare and keeping UC in remission afterward. Sulfasalazine is modestly more effective specifically for long-term maintenance, but causes more side effects (headache, nausea, rash) than newer mesalamine formulations, which is why those are generally preferred first.
- **Topical mesalamine** (enemas or suppositories) for disease limited to the rectum or left side of the colon.
  Evidence: Considered first-line specifically for inflammation confined to the rectum, with higher remission and response rates in trials than oral therapy alone for this pattern of disease.
- **Combination oral plus topical mesalamine** for more extensive disease (beyond the left side of the colon).
  Evidence: Produces a better response than oral therapy by itself in patients with more extensive inflammation, and is recommended over oral therapy alone for this group.

Corticosteroids

Goal: quickly calm inflammation during a flare; a bridge to longer-term treatment, not a long-term solution on their own.

- **Oral prednisone** for moderate-to-severe flares not controlled by 5-ASAs.
  Evidence: Effective and fast-acting for inducing remission, but not effective for keeping UC in remission long-term, and prolonged use carries real risks — bone thinning, elevated blood sugar, mood changes, and higher infection risk. Guidelines are clear that steroids should be tapered off once a flare improves, with a steroid-sparing medication (5-ASA, biologic, or other maintenance therapy) already in place to prevent relapse as the steroid is withdrawn.
- **Budesonide (Uceris)** — a corticosteroid formulated to release mostly in the colon, for mild-to-moderate flares.
  Evidence: Effective for inducing remission with meaningfully fewer of the whole-body steroid side effects above, since less of the medication is absorbed into general circulation. Still intended for short-term flare control, not indefinite use.
- **IV corticosteroids**, reserved for severe flares requiring hospitalization.
  Evidence: Standard of care for severe, hospitalized UC flares; also used short-term for the same reasons and with the same limitations described above.

Biologics & Advanced Therapies

Goal: longer-term disease control for moderate-to-severe UC, or when 5-ASAs and steroids aren't enough — typically also used to help taper off steroids.

• **Anti-TNF biologics** — infliximab (Remicade), adalimumab (Humira), golimumab (Simponi); given by infusion or injection. These block TNF, a signaling protein that drives inflammation throughout the body.

Evidence: The longest track record of this group: in studies that directly compared different biologics against each other, infliximab ranked among the most effective options for patients who haven't tried a biologic before.

• **Anti-integrin biologic** — vedolizumab (Entyvio); given by infusion or injection. This works differently, by blocking inflammatory cells from leaving the bloodstream and entering the gut in the first place, rather than blocking a signaling protein throughout the body.

Evidence: Ranks among the most effective options for patients who haven't tried a biologic before, with one of the more favorable safety profiles in the class, since it acts mainly in the gut rather than throughout the body.

• **Anti-IL-12/23 biologic** — ustekinumab (Stelara); given by infusion then injection. This blocks two different signaling proteins (interleukin-12 and -23) that help drive the immune response involved in UC.

Evidence: Current gastroenterology guidelines specifically favor ustekinumab for patients who've already tried and lost response to an anti-TNF medication, an area where it performs particularly well.

• **Anti-IL-23 biologics** (newer) — risankizumab (Skyrizi), mirikizumab (Omvoh), and guselkumab (Tremfya); given by infusion and/or injection depending on the agent. These block just IL-23, a more targeted version of the approach above.

Evidence: All three are recent FDA approvals (2023–2025) and rank among the higher-efficacy options for patients who haven't tried a biologic before, in studies that directly compared these medications against each other, with mirikizumab additionally standing out for a favorable safety profile.

• **Oral small-molecule therapies** (technically not biologics, since they're chemically made rather than grown from living cells, but often grouped with them) — JAK inhibitors (tofacitinib/Xeljanz, upadacitinib/Rinvoq) and S1P modulators (ozanimod/Zeposia, etrasimod/Velsipity), taken as pills instead of by infusion or injection.

Evidence: Effective options, including for patients who've already tried and failed a biologic. JAK inhibitors specifically carry an FDA boxed warning for increased risk of serious heart events, blood clots, cancer, and death, identified in a large safety trial — they're generally reserved for patients without other good options, used with extra caution in those with heart disease risk factors, and require a specific conversation about this risk before starting. S1P modulators don't carry this same warning but require a baseline heart-rhythm check (an EKG) before the first dose.

Adjunct & Complementary Therapies

Goal: additional, evidence-supported options layered on top of standard medical therapy — not a replacement for it.

• **Curcumin** (the active compound in turmeric), typically 1–3 grams daily alongside your regular UC medication.

Evidence: A placebo-controlled trial found curcumin added to standard 5-ASA therapy significantly reduced relapse over 6 months compared to 5-ASA plus a dummy pill, in patients already in remission. Well tolerated with no serious side effects across trials.

• **CurQD (curcumin-QingDai combination)** — a specific herbal combination, coated so it releases further along in the gut rather than dissolving in the stomach; studied for active flares, not just maintenance.

Evidence: A placebo-controlled trial in patients with active UC found 43% reached the study's main goal on CurQD versus 8% on placebo at 8 weeks, with clinical remission in 50% versus 8%. Promising, dedicated trial evidence specifically for this combination — though QingDai (a component also known as indigo naturalis) has separately been linked to rare cases of liver injury and a rare lung blood-pressure condition in case reports outside these trials, so this should only be used under your doctor's supervision with appropriate monitoring, not started independently.

• **Probiotics** — specifically VSL#3 (also sold as Vivomixx), a high-dose multi-strain product; other probiotic products haven't shown the same evidence.

Evidence: A placebo-controlled trial, and a later study pooling results across several trials for a more reliable answer, both found VSL#3 effective as an add-on for inducing remission in mild-to-moderate UC. Evidence is specific to this product and dose — it doesn't necessarily generalize to other probiotics.

• **Butyrate** (a short-chain fatty acid your gut bacteria normally produce from fiber), as an oral supplement or enema.

Evidence: Evidence is mixed: enema trials for left-sided UC show inconsistent results — some found no difference from a dummy treatment in overall symptoms and inflammation, though one showed fewer inflamed segments of colon and improved bleeding. More recent, smaller trials of oral butyrate supplementation show improvement in inflammation blood markers and symptom-severity scores compared to placebo. Reasonable as a low-risk adjunct, but not established enough to rely on alone.

• **Fecal microbiota transplant (FMT)** — transferring stool from a screened healthy donor to help restore a healthier gut microbiome; done through research protocols or specialized programs, not something to pursue independently.

Evidence: A study pooling results across several placebo-controlled trials found FMT more effective than a dummy treatment for inducing remission, and more effective than VSL#3 in direct comparison. That said, one of the field's own experts described the ulcerative colitis data as showing a "signal of benefit" that remains "unconvincing" overall — still considered investigational (meaning still being studied, not yet standard practice) for UC rather than routine care.

Dietary Management

Goal: support symptom control and nutrition alongside — not instead of — medical therapy.

• **Mediterranean-style eating pattern** — vegetables, fruit, legumes, whole grains, olive oil, less red meat and ultra-processed food.

Evidence: A randomized trial found this pattern improved measured intestinal inflammation and reshaped gut bacteria in UC patients, likely through reduced saturated fat and processed food intake alongside increased fiber and antioxidants. One of the better-supported dietary approaches for this condition, and reasonable to follow long-term given its broader health benefits.

• **A lower-fiber, lower-residue diet during active flares**, returning to a more fiber-inclusive diet once symptoms improve.

Evidence: Reduces mechanical irritation and stool frequency during active inflammation; a practical, widely used short-term measure rather than a disease-modifying treatment — it's not meant to be permanent, since fiber has independent long-term health benefits once the flare settles.

• **A structured exclusion diet** (removing specific food groups thought to promote inflammation), done under medical or dietitian guidance.

Evidence: A small trial in adolescents and young adults with mild-to-moderate UC found meaningful rates of remission and response using a structured whole-food exclusion diet. Promising but based on a small study population so far — best pursued with professional guidance rather than on your own, and as a complement to, not a substitute for, medical therapy.

Vaccinations & Immune Health

Goal: reduce infection risk during immunosuppressive therapy — ideally by bringing vaccines up to date before starting treatment, since both timing and vaccine type matter.

• **Non-live vaccines** — the annual flu shot (the injected version, not the nasal spray), COVID-19, Tdap, hepatitis A and B, HPV, and meningococcus — on the usual schedule.

Evidence: These vaccines can't cause the infection they protect against, so current guidelines say they shouldn't be delayed for patients on a biologic, immunomodulator, or steroid. Hepatitis B vaccination is particularly important for anyone without existing immunity, since immunosuppression can reactivate a dormant hepatitis B infection.

• **Live vaccines** — MMR (measles, mumps, rubella), chickenpox (varicella), the nasal-spray flu vaccine, and yellow fever — should be avoided once a biologic, thiopurine, methotrexate, or JAK inhibitor has started.

Evidence: These contain a weakened but still-live virus, which carries a small risk of causing the very infection it's meant to prevent if given while the immune system is significantly suppressed. If you've never had chickenpox and haven't started immunosuppressive therapy yet, current guidelines recommend completing that vaccination first.

• **Shingles vaccine** (Shingrix, a 2-dose series) — recommended starting at age 19 for anyone on or about to start immune-modifying therapy, rather than waiting until age 50 as in the general population.

Evidence: Guidelines lowered the recommended starting age specifically because immunosuppressive therapy raises the risk of shingles at any age. Shingrix isn't a live vaccine, so it's safe to receive while on treatment.

• **Pneumococcal vaccine** — recommended for all patients 50 and older, and for younger adults (19–49) who are on immune-modifying therapy.

Evidence: People on immunosuppressive therapy face a higher risk of serious pneumococcal infection, which is why current guidelines extend this recommendation to younger patients on treatment rather than waiting until age 50 as in the general population.

When to Contact Our Office

- More than 6 bloody bowel movements a day, fever, a rapid heart rate, or severe abdominal pain or swelling — these can signal a severe flare requiring urgent evaluation
- New symptoms of infection, unusual fatigue, or yellowing of the skin or eyes while on a biologic, immunosuppressant, or CurQD
- Before starting, stopping, or adjusting any medication or supplement in this guide
- Any new or worsening symptoms despite following your current treatment plan

This guide supports your personalized plan and isn't a substitute for individualized medical advice. Check with your provider before starting, stopping, or combining anything mentioned here.