Innovative GI

Understanding & Managing Crohn's Disease

A Patient Guide to Crohn's Disease Care

What Is Crohn's Disease?

Crohn's disease is a chronic inflammatory bowel disease in which the immune system causes ongoing inflammation anywhere along the digestive tract, from the mouth to the anus — though it most commonly involves the end of the small intestine (terminal ileum) and the colon. It differs from ulcerative colitis in two important ways: the inflammation is often patchy, with normal-looking sections of bowel between affected areas ("skip lesions"), and it goes through the full thickness of the bowel wall (transmural) rather than staying confined to the inner lining. This full-thickness involvement is why Crohn's can lead to complications not typically seen in ulcerative colitis — strictures (narrowed segments of bowel), fistulas (abnormal tunnels connecting the gut to other organs or the skin), and abscesses. Common symptoms include abdominal pain and cramping (often lower right-sided), diarrhea (which may or may not contain blood, unlike UC), fatigue, unintentional weight loss, and sometimes fever or perianal symptoms (fissures, drainage, or swelling). Like UC, Crohn's typically follows a relapsing-remitting course.

What Causes It?

- **Immune system dysregulation.** The immune system mounts an ongoing inflammatory response against the gut, likely triggered by an abnormal reaction to the bacteria that normally live there rather than to genuine invaders.
- **Genetics.** Crohn's has a stronger hereditary component than UC — having a first-degree relative with Crohn's meaningfully raises your risk. A gene called NOD2 is the single best-established genetic risk factor, accounting for roughly a fifth of the inherited risk; it normally helps immune cells sense bacteria and regulate inflammation appropriately.
- **Gut microbiome imbalance.** As in UC, people with Crohn's tend to have less diverse gut bacteria, which may help drive and sustain inflammation.
- **Smoking** is one of the strongest known risk factors for developing Crohn's, and for the disease behaving more aggressively once diagnosed — more flares, more need for steroids, and a higher chance of eventually needing surgery. This is the opposite of UC, where smoking is associated with somewhat lower risk. Quitting smoking is one of the single most impactful things a Crohn's patient can do for their disease course.
- **Other environmental factors** that aren't fully understood, including early antibiotic exposure, diet, and prior gastrointestinal infections.
- **What Crohn's is not caused by:** diet and stress don't cause Crohn's, though both can influence how symptoms feel day to day once the disease is present.

How Crohn's Disease Is Treated

Treatment has two goals: calming an active flare (called induction) and then keeping the disease quiet long-term (called maintenance), with an eye toward preventing the strictures, fistulas, and need for surgery that untreated inflammation can eventually cause. 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 "boxed warning" is the strongest safety warning the FDA issues on a medication label. And the "biologic" medications below are named for the specific part of the immune system each one blocks — different approaches to calming inflammation, not different strengths of the same drug.

> *A note on 5-ASA medications (like mesalamine): these are a mainstay of treatment in ulcerative colitis, but the evidence for them in Crohn's disease is much weaker — trials have not shown a clear benefit over a dummy pill for calming a flare or preventing relapse, and most current guidelines recommend against routine use. The one setting with somewhat better support is maintaining remission after bowel surgery, at higher doses. Given this, we generally don't reach for 5-ASAs as primary Crohn's therapy the way we do in UC.*

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.
  Evidence: Effective and fast-acting for inducing remission, but not effective for keeping Crohn's 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 already in place to prevent relapse as the steroid is withdrawn.
- **Budesonide (Entocort EC)** — a corticosteroid formulated to release mainly in the last part of the small intestine and first part of the colon, for mild-to-moderate flares limited to that area.
  Evidence: Effective for inducing remission in ileal or right-sided colonic disease 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, and not useful for disease elsewhere in the colon given where it releases.
- **IV corticosteroids**, reserved for severe flares requiring hospitalization.
  Evidence: Standard of care for severe, hospitalized Crohn's flares; used short-term for the same reasons and with the same limitations described above.

Immunomodulators (Thiopurines & Methotrexate)

Goal: broadly dial down immune activity to maintain remission, taper off steroids, and — especially when combined with a biologic — help that biologic work better and last longer.

- **Azathioprine or 6-mercaptopurine (6-MP)** — oral, once-daily thiopurine medications.
  Evidence: On their own, not clearly better than a dummy pill at inducing remission in an active flare, so they're used mainly for maintenance and as a partner to biologics rather than as flare treatment by themselves.
- **Methotrexate** — given by weekly injection or pill, an alternative for patients who can't tolerate thiopurines.
  Evidence: A head-to-head trial found azathioprine and methotrexate similarly effective for maintaining steroid-free remission, so the choice often comes down to tolerability and preference.
- **Combination therapy** — adding a thiopurine (usually azathioprine) to a biologic like infliximab, rather than using either alone.
  Evidence: The landmark SONIC trial found combination therapy significantly more effective than infliximab alone for steroid-free remission (60% versus 48%). The immunomodulator appears to work partly by improving how the body handles the biologic itself — keeping drug levels more stable and reducing the chance the body develops antibodies against it over time.

Biologics & Advanced Therapies

Goal: longer-term disease control for moderate-to-severe Crohn's, or when steroids alone aren't enough — typically also used to help taper off steroids.

• **Anti-TNF biologics** — infliximab (Remicade), adalimumab (Humira), and certolizumab pegol (Cimzia, a Crohn's-specific option not used in UC); 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. Infliximab in particular also has dedicated trial evidence for fistulizing Crohn's specifically (discussed further below), beyond its general anti-inflammatory effect.

• **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, rather than blocking a signaling protein throughout the body.

Evidence: An effective option 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 Crohn's.

Evidence: A well-established option, including 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, approved 2022), mirikizumab (Omvoh, approved 2025), and guselkumab (Tremfya, approved 2025); 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, dedicated Crohn's approvals with supporting trial data; in one head-to-head trial, guselkumab outperformed ustekinumab on several measures of healing seen on colonoscopy, though real-world experience with these newer agents is naturally more limited than with the longer-established options above.

• **Oral small-molecule therapies** (technically not biologics, since they're chemically made rather than grown from living cells, but often grouped with them) — the JAK inhibitor upadacitinib (Rinvoq), taken as a pill.

Evidence: Effective, including for patients who've already tried and failed a biologic. This class carries an FDA boxed warning for increased risk of serious heart events, blood clots, cancer, and death, identified in a large safety trial of a related JAK inhibitor — it's generally reserved for patients without other good options, used with extra caution in those with heart disease risk factors, and requires a specific conversation about this risk before starting.

Nutritional Therapy

Goal: calm active inflammation through diet composition alone — a uniquely well-supported, medication-free option in Crohn's that doesn't have a direct equivalent in UC.

• **Exclusive enteral nutrition (EEN)** — replacing all regular food with a complete liquid nutrition formula for several weeks, typically to induce remission.

Evidence: One of the best-studied non-drug treatments in all of Crohn's disease, particularly in children and adolescents, where it's recommended as a first-line option for mild-to-moderate disease with effectiveness comparable to steroids — without the steroid side effects. Its main practical limitation is that consuming only formula for weeks is difficult to sustain, which is why it's used less in adults.

• **Crohn's Disease Exclusion Diet (CDED)** — a structured whole-food diet that removes specific components thought to disrupt the gut lining and microbiome, combined with partial (not exclusive) enteral nutrition.

Evidence: Randomized trials in both children and adults found CDED plus partial enteral nutrition induced remission about as effectively as exclusive enteral nutrition, with meaningfully better tolerance and adherence since regular food is still allowed. A well-supported, more sustainable alternative to EEN, ideally followed with dietitian guidance.

Managing Fistulizing & Perianal Disease

Goal: close fistulas and control perianal complications — a combined medical and surgical effort in most cases.

• **Infliximab**, specifically for fistulizing disease.

Evidence: A placebo-controlled trial found that among patients whose fistulas closed with infliximab, continuing infliximab on a regular schedule kept them closed significantly longer than switching to a dummy infusion — this is dedicated, fistula-specific trial evidence, not just an extension of infliximab's general anti-inflammatory effect.

• **Seton placement** (a surgical drain left in place through a fistula tract) **combined with infliximab**, rather than either alone.

Evidence: A study comparing the combination to infliximab by itself found better initial response (100% versus 83%), a lower recurrence rate (44% versus 79%), and a longer time before recurrence when surgery and medication were used together — the current standard approach for most perianal fistulas rather than relying on medication alone.

Surgical Management

Goal: relieve blockages or remove severely damaged bowel when medication isn't enough — an important difference from UC, where removing the colon is curative. In Crohn's, surgery treats the affected segment but doesn't cure the underlying disease, since inflammation can recur elsewhere.

• **Strictureplasty** — widening a narrowed segment of bowel surgically without removing it, generally preferred in adults when possible since it preserves bowel length.

Evidence: Long-term studies show a relatively low recurrence rate at the specific site treated (under 4% in one large series), though full disease recurrence elsewhere in the bowel remains common over time, consistent with Crohn's being a whole-gut disease rather than one confined to a single damaged segment.

• **Bowel resection** — surgically removing a severely damaged or obstructed segment.

Evidence: Effective for relieving obstruction, but endoscopic evidence of disease recurrence near the surgical connection is seen in roughly half of patients within a year without further treatment, which is why starting or continuing a biologic after surgery is now standard practice for higher-risk patients.

• **Biologic therapy after surgery**, to prevent the disease from returning at the surgical site.

Evidence: Multiple studies support starting or continuing a biologic (most commonly an anti-TNF agent) after resection in patients at higher risk of recurrence, rather than waiting for symptoms or endoscopic signs of disease to reappear first.

Adjunct & Complementary Therapies

Goal: additional options layered on top of standard medical therapy — worth knowing the evidence here is meaningfully thinner than for the treatments above, and thinner than for the same supplements in ulcerative colitis.

• **Curcumin** (the active compound in turmeric).

Evidence: Curcumin has much stronger, more consistent trial evidence in ulcerative colitis than in Crohn's specifically. A small placebo-controlled pilot trial in Crohn's patients already on azathioprine found curcumin well tolerated, but the evidence for added effectiveness in Crohn's remains limited and inconsistent — reasonable to consider as a low-risk add-on, but we wouldn't describe it as established therapy the way we would in UC.

• **Probiotics.**

Evidence: Unlike ulcerative colitis, where one specific high-dose product (VSL#3) has dedicated placebo-controlled trial support, no probiotic has shown clear, reproducible benefit specifically in Crohn's disease. We don't discourage a reasonable trial, but wouldn't rely on any product as a treatment for active Crohn's.

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

- Severe abdominal pain, vomiting, or visible abdominal swelling — possible signs of a bowel blockage, requiring urgent evaluation
- High fever, new or worsening drainage from a fistula, or a new lump or swelling near the anus — possible signs of an abscess
- Signs of significant bleeding, or symptoms of anemia such as unusual fatigue or lightheadedness
- New symptoms of infection, or yellowing of the skin or eyes, while on an immunomodulator or biologic
- Before starting, stopping, or adjusting any medication or supplement in this guide

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.

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