

IBD • Inflammatory Bowel Disease

The immune system mistakenly attacks the gut lining over and over, causing long-lasting swelling and damage.

THE STORY IN ONE SENTENCE

The immune system mistakenly attacks the gut lining over and over, causing long-lasting swelling and damage.

“IBD is chronic gut swelling from the immune system attacking itself. Crohn disease can strike anywhere from mouth to anus and punches through all gut layers. Ulcerative colitis only hits the colon and rectum and stays in the inner lining. Where and how deep the swelling goes decides treatment.”

Normal anatomy — the body's job

The gut is a muscular tube lined by a single-cell-thick barrier that absorbs nutrients, keeps bacteria and food particles contained, and blocks germs from entering the bloodstream. The immune system in the gut wall normally tolerates friendly bacteria and food proteins while attacking true pathogens.

BODY'S JOB

The gut lining acts like a selective screen: it lets water, vitamins, and broken-down nutrients pass into the blood to feed the body, but keeps bacteria, toxins, and undigested food locked inside the tube. Immune cells stationed in the gut wall — especially T cells and dendritic cells (cells that train the immune response) — constantly sample what is in the gut and decide whether to tolerate it or attack. This learned tolerance keeps you from reacting to food and harmless gut bacteria every single day.

1. Food enters the mouth and travels down the esophagus into the stomach, where acid and enzymes start breaking it into smaller pieces.
2. The stomach empties into the small intestine (duodenum, jejunum, ileum), where most nutrients are absorbed through tiny finger-like projections called villi that line the inner wall.

3. What is left moves into the colon (large intestine), which absorbs water and forms stool, then stores it in the rectum until you are ready to pass it.
4. The gut lining is a single layer of tightly joined cells that form a barrier; beneath it sits a layer of immune cells that patrol for invaders.
5. Trillions of bacteria live in the gut — mostly helpful ones that make vitamins, break down fiber, and train the immune system to stay calm.
6. Mucus coats the lining to keep bacteria at a safe distance, and immune cells release signals that tell T cells not to attack the friendly bacteria or food proteins.

What breaks

The immune system loses its ability to tolerate harmless gut bacteria and food, so T cells flood the gut wall and cause chronic swelling and tissue damage.

CLINICAL TERM

IBD arises from a combination of genetic susceptibility (over 200 risk genes identified, including NOD2, IL23R, and ATG16L1), environmental triggers (infection, antibiotics, diet, smoking), and gut microbiome changes (dysbiosis) that together break immune tolerance. CD4+ T helper cells — especially Th1, Th17, and sometimes Th2 subsets — become overactive and release pro-inflammatory cytokines such as tumor tissue death (necrosis) factor-alpha (TNF- α), interleukin-17 (IL-17), interleukin-12 (IL-12), and interleukin-23 (IL-23). These cytokines recruit neutrophils (white cells that release tissue-damaging enzymes) and macrophages (cells that eat debris and release more inflammatory signals) into the gut wall, forming ulcers, abscesses, and granulomas (clumps of immune cells). In Crohn disease, inflammation is transmural (all the way through the gut wall) and can occur anywhere from mouth to anus, leading to fistulas, strictures, and skip lesions (patches of disease with normal gut in between). In ulcerative colitis, inflammation is limited to the mucosa and submucosa (inner lining) of the colon and rectum, spreads continuously from the rectum upward, and causes friable mucosa (tissue that bleeds easily) and pseudopolyps (islands of surviving tissue that look like polyps).

How the body compensates

The body tries to contain the damage by walling off inflamed areas with scar tissue, speeding up bowel movements to flush out blood and debris, and shunting blood away from the gut to protect vital organs. These fixes can hide how sick the gut really is — until sudden

complications like a torn colon, massive bleeding, or toxic megacolon (a hugely swollen colon that stops moving) force an emergency.

1. The colon speeds up contractions and pours out mucus and fluid to flush away blood, pus, and damaged tissue — that is why diarrhea and urgency happen.
2. The body forms granulation tissue (new, fragile tissue) to try to heal ulcers, but ongoing inflammation prevents true healing, so the tissue stays raw and bleeds easily.
3. Scar tissue (fibrosis) builds up in areas of repeated injury, narrowing the gut tube into strictures that slow or block food and stool from passing.
4. The immune system walls off deep ulcers by forming abscesses (pockets of pus) and fistulas (tunnels) that connect the gut to skin, bladder, or other loops of bowel to drain the infection.
5. Chronic blood loss from ulcers slowly drains iron stores, causing anemia (low red blood cells) that makes you tired and pale, but the body boosts heart rate to keep oxygen delivery steady — so you may not feel the full impact until the anemia is severe.
6. The liver and spleen may enlarge slightly as they filter the extra immune signals and damaged red blood cells, but vital signs can stay near normal for months or years.
7. Watch for the crash: toxic megacolon (colon swells, stops moving, and risks tearing), perforation (a hole punches through the gut wall, spilling stool into the belly and causing sepsis), massive bleeding (hemorrhage) (uncontrolled bleeding), or colon cancer (chronic inflammation raises cancer risk 2–3 times normal after 8–10 years of disease).
8. Trend over time matters more than a single snapshot — worsening anemia, rising inflammatory markers (C-reactive protein, fecal calprotectin), weight loss, and new fevers mean the disease is winning despite compensation.

Domino effect

1. Genetic susceptibility plus an environmental trigger (infection, antibiotics, stress, diet) breaks immune tolerance in the gut. — People inherit variants in over 200 genes — such as NOD2 (which senses bacteria), IL23R and IL12B (which control T cell signals), and ATG16L1 (which helps cells clear bacteria) — that make the gut barrier leaky and the immune system jumpy. When a trigger arrives, the immune system misfires.
2. T cells (CD4+ helper cells) flood the gut wall and release inflammatory cytokines: TNF- α , IL-12, IL-17, and IL-23. — Dendritic cells in the gut wall mistakenly tag harmless bacteria as dangerous and activate T helper 1 (Th1) and T helper 17 (Th17) cells, which pump out signals that call more white blood cells to the scene and tell them to attack.

3. Neutrophils and macrophages invade the gut lining, releasing enzymes and free radicals that chew up tissue, forming ulcers and bleeding. — Neutrophils are the body's first responders to infection — they release bleach-like chemicals to kill germs, but here they damage healthy gut cells by mistake. Macrophages eat debris and release even more TNF- α , keeping the fire burning.
4. In ulcerative colitis: continuous inflammation in the colon's inner lining causes bloody diarrhea, urgency, a constant urge to pass stool (tenesmus) (feeling that more stool is left inside), and cramping. — Ulcers bleed easily because the surface layer of cells is destroyed, and the colon contracts rapidly to try to expel blood and mucus, giving no time to absorb water — so stool stays liquid and bloody.
5. In Crohn disease: patchy, transmural inflammation anywhere from mouth to anus causes belly pain, non-bloody diarrhea, strictures, fistulas, abscesses, and perianal sores. — Inflammation bores through all layers of the gut wall, creating deep ulcers that can tunnel into nearby organs (fistulas) or trap pus (abscesses). Repeated healing lays down thick scar tissue that narrows the tube (strictures), causing cramping and partial blockages.
6. Chronic gut inflammation spills inflammatory cytokines into the bloodstream, triggering extra-intestinal manifestations: arthritis (joint pain and swelling), uveitis (eye redness and pain), redness (erythema) nodosum (painful red bumps on shins), and pyoderma gangrenosum (deep skin ulcers). — The same overactive immune cells and cytokines circulate everywhere, so joints, eyes, skin, and liver become collateral damage. These flares often mirror gut disease activity.
7. The body tries to contain the damage with scar tissue and abscesses, but ongoing inflammation prevents healing — leading to strictures, fistulas, malabsorption (poor nutrient uptake), anemia, weight loss, and growth delay in children. — Scar tissue is stiff and narrows the gut, blocking food. Fistulas leak gut contents into places they do not belong. Inflamed gut cannot absorb iron, B12, fats, or calories well, so the body starves even if you eat.
8. Without treatment, complications arise: toxic megacolon (the colon balloons and stops moving, risking perforation), perforation (a hole in the gut spills stool into the belly, causing peritonitis and sepsis), massive bleeding, or colorectal cancer (chronic inflammation damages DNA in colon cells over years). — The gut wall weakens from repeated ulcers — one deep ulcer can punch through, or the colon can swell so much that its muscle layer stops working. Chronic inflammation also mutates colon cells, raising cancer risk starting around 8–10 years of disease, especially if the whole colon is involved.

Symptoms · what the patient feels, and why

- **Diarrhea (loose, watery poops) that keeps coming back or won't go away:** The immune system attacks the lining of the intestine by mistake, damaging the cells that normally absorb water and nutrients. The inflamed tissue leaks fluid and can't grab water back from the stool, so the poop stays loose and comes more often.
- **Blood or mucus in the stool:** The inflamed, damaged lining of the colon (the large intestine) bleeds easily, like a scraped knee. Mucus is the slippery coating the gut makes to protect itself; when it's irritated, it makes extra mucus that mixes with the poop.
- **Cramping belly pain, often worse before or during a bowel movement:** The inflamed intestine is swollen and sensitive. The muscles in the intestine wall squeeze to move stool along, and when they squeeze an inflamed area, it hurts—like pressing on a bruise.
- **Urgent need to poop, sometimes waking you up at night:** The inflamed rectum (the last part of the intestine before the anus) is extra sensitive and can't stretch or hold stool well. Even a small amount of stool triggers a strong signal to the brain: 'Go now.'
- **Weight loss without trying:** The damaged intestine can't absorb nutrients and calories properly. Plus, inflammation speeds up the body's metabolism (the rate it burns energy), and pain or nausea can make eating less appealing—so you lose weight even if you're eating normally.
- **Fever and feeling very tired:** The immune system is in overdrive, releasing messenger proteins called cytokines (especially TNF-alpha and interleukins) that raise body temperature and make you feel drained, like when you have the flu. Chronic blood loss and poor nutrient absorption also leave you low on iron and energy.
- **Sores or pain around the anus (bottom):** In Crohn's disease (one type of IBD), inflammation can tunnel through the intestine wall and form fistulas (abnormal tunnels) or abscesses (pockets of pus) near the anus. Frequent, loose stools also irritate the skin around the anus.
- **Joint pain or swelling, especially in large joints like knees and ankles:** The same immune proteins (cytokines) that attack the intestine can also trigger inflammation in the joints. This is called an extraintestinal manifestation—a problem outside the gut caused by the same overactive immune response.
- **Red, painful eyes or blurry vision:** The immune system sometimes attacks the eyes, causing inflammation in the uvea (the middle layer of the eye) or the sclera (the white part). This is another extraintestinal manifestation of IBD.
- **Skin rashes, such as painful red bumps (erythema nodosum) or deep ulcers (pyoderma gangrenosum):** Circulating immune proteins and inflammatory signals from the gut can reach the skin and trigger abnormal immune reactions there. The skin becomes inflamed and develops tender nodules or sores.

Focused exam

- **Blood and mucus in the stool:** In ulcerative colitis, the immune system attacks the inner lining of the colon. That attack breaks tiny blood vessels and strips away the protective layer of cells. Blood in the poop tells you the gut wall is raw and bleeding. Mucus is the colon's attempt to protect itself, but it cannot keep up.
- **Needing to rush to the bathroom many times per day (often 6 or more):** When the colon wall swells from inflammation, it shrinks the space inside. The colon cannot hold as much, so it feels full even when it is not. That urgency is the inflamed gut screaming for relief. Frequent, small bowel movements are the signature of an angry colon.
- **Tender belly, especially in the lower right side (Crohn disease) or lower left side (ulcerative colitis):** Crohn disease swells deep into all layers of the gut wall and can hit anywhere from mouth to anus, but often attacks the end of the small intestine (terminal ileum) in the lower right belly. Ulcerative colitis only swells the surface layer of the colon, usually starting from the rectum and moving left. When you press the belly, you are pressing on swollen, inflamed tissue. Tenderness tells you where the disease is hiding.
- **Skin tags, sores, or tunnels (fistulas) near the anus:** Crohn disease swells so deep it punches tunnels through the gut wall. These tunnels (fistulas) can burrow to the skin, bladder, or other loops of bowel. Sores and tags near the anus are proof the inflammation is deep and has created abnormal connections. This almost never happens in ulcerative colitis.
- **Swollen, painful joints (arthritis), red eyes (uveitis or episcleritis), or red bumps on the shins (erythema nodosum):** IBD is not just a gut disease. The same immune attack that targets the gut can also attack joints, eyes, and skin. These extra findings tell you the immune system is out of control in multiple places. About one in four people with IBD has at least one of these problems outside the gut.
- **Weight loss and looking thin or wasted:** When the gut wall is raw and swollen, it cannot absorb food well. The body starves even when the person eats. Muscle melts away because the gut is too sick to do its job. Weight loss also happens because people avoid eating to escape the pain and diarrhea that food triggers.
- **Fever (temperature over 100.4°F or 38°C):** Fever means either massive inflammation or infection. In IBD, fever can signal that the colon is starting to tear (perforation), a pocket of pus has formed (abscess), or the inflammation is out of control. This is a danger sign that demands fast action.

Diagnostics · every test answers a question

- **Complete blood count (CBC) → Is the person losing blood or fighting infection?**

- C-reactive protein (CRP) and red blood cell (erythrocyte) sedimentation rate (ESR) → How much inflammation is there right now?
- Fecal calprotectin → Is the inflammation coming from the gut, or somewhere else?
- Stool culture and testing for *Clostridioides difficile*, parasites, and other infections → Is this an infection pretending to be IBD, or is IBD letting an infection in?
- Colonoscopy with biopsy → Where is the disease, how deep does it go, and what does it look like under the microscope?
- MR enterography or CT enterography → Is there disease in the small intestine where the camera cannot reach, and are there complications like abscesses or fistulas?
- Serology: anti-*Saccharomyces cerevisiae* antibodies (ASCA) and perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA) → Can blood antibodies help tell Crohn disease apart from ulcerative colitis?
- Albumin and pre-albumin levels → Is the person malnourished because the gut cannot absorb protein?

Treatment logic

- 5-aminosalicylic acid (5-ASA) drugs like mesalamine or sulfasalazine for mild-to-moderate ulcerative colitis → 5-ASA drugs block the chemical messengers (prostaglandins and leukotrienes) that call immune cells to the colon lining. This calms the attack on gut tissue, stops ulcers from forming, and lets the lining heal. They work well for surface inflammation in ulcerative colitis.
- Corticosteroids like prednisone (oral) or methylprednisolone (IV) during acute flares → Steroids shut down the entire immune attack fast by blocking genes that make inflammatory chemicals and by killing activated T cells (the immune soldiers attacking the gut). Bleeding stops, diarrhea slows, and pain eases within days. This buys time to start slower, safer drugs.
- Immunomodulators like azathioprine, 6-mercaptopurine (6-MP), or methotrexate for steroid-dependent or moderate-to-severe disease → These drugs block DNA synthesis in rapidly dividing cells, especially T cells and other immune cells. Fewer immune soldiers means less attack on the gut lining. Over 8 to 12 weeks, inflammation drops and you can taper off steroids safely. They help keep the disease quiet (maintain remission) for months to years.

- Biologic drugs: anti-TNF agents (infliximab, adalimumab, golimumab, certolizumab), anti-integrin (vedolizumab, natalizumab), anti-IL-12/23 (ustekinumab), or JAK inhibitors (tofacitinib, upadacitinib) → Each biologic blocks a specific molecule that calls immune cells to the gut or keeps them activated. Anti-TNF drugs block tumor tissue death (necrosis) factor-alpha (TNF- α), a powerful alarm signal that recruits and activates T cells. Anti-integrins block adhesion molecules so immune cells cannot stick to and enter the gut wall. Anti-IL-12/23 drugs block interleukins that help T cells mature and attack. JAK inhibitors block signals inside immune cells. All of these stop the immune attack at a precise step, heal ulcers, and keep the disease quiet longer than older drugs.
- Antibiotics like metronidazole or ciprofloxacin for perianal Crohn disease, abscesses, or bacterial overgrowth → Antibiotics kill bacteria in infected tunnels (fistulas), drain pockets of pus (abscesses), and reduce bacterial load in the gut. In Crohn, they help heal perianal disease and can calm inflammation in some cases by changing the gut microbiome.
- Surgery: total colectomy (remove the entire colon) for ulcerative colitis; bowel resection or stricturoplasty (widen narrow spots) for Crohn disease → In ulcerative colitis, removing the colon cures the disease completely because ulcerative colitis only attacks the colon and rectum. The surgeon creates a pouch from small intestine (ileal pouch-anal anastomosis) or a permanent ostomy bag. In Crohn disease, surgery removes severely damaged or blocked sections, drains abscesses, or closes fistulas, relieving symptoms and complications.
- Colon cancer surveillance: colonoscopy every 1 to 2 years starting after 8 to 10 years of disease → Chronic inflammation in the colon raises the risk of colon cancer by 2 to 3 times normal. Regular camera exams catch pre-cancerous changes (dysplasia) or early cancer when it is still curable. Removing polyps and biopsying flat abnormal areas saves lives.

Medications

Mesalamine (5-ASA) · Aminosalicylate anti-inflammatory

Mesalamine is an anti-inflammatory drug that works right where the gut lining is inflamed. It quiets down inflammation in the intestinal wall by blocking chemicals that fuel the swelling and damage. Think of it like spraying a cooling mist directly on a hot, irritated surface.

1. You swallow the pill or use it as a rectal suppository or enema
2. The drug travels to the inflamed parts of the intestine
3. Mesalamine blocks enzymes called cyclooxygenase and lipoxygenase in the gut wall—these enzymes make prostaglandins and leukotrienes, which are chemicals that trigger inflammation
4. With fewer inflammatory chemicals, the swelling, redness, and damage to the intestinal lining calm down
5. The gut lining can start to heal and bleeding may stop

SAFETY WATCH

Rarely can cause kidney problems—your doctor will check blood and urine tests periodically · Can rarely trigger a flare that looks like worsening colitis (paradoxical worsening); stop the drug and call your doctor if symptoms get worse · Some people get headache, nausea, or abdominal pain · Allergic reactions are rare but possible (rash, fever); people allergic to aspirin or sulfa drugs should use mesalamine cautiously and tell their doctor

Prednisone or budesonide (corticosteroids) · Corticosteroid (systemic or gut-targeted anti-inflammatory)

Corticosteroids are powerful drugs that slam the brakes on inflammation throughout the body (prednisone) or mainly in the gut (budesonide). They mimic cortisol, the body's own stress hormone, and tell immune cells to stop making inflammatory signals. This quickly quiets a flare but does NOT cure the disease or keep it away long-term.

1. You swallow the pill (or sometimes receive IV steroids in the hospital for severe flares)
2. The steroid enters your cells and binds to receptors inside
3. It travels into the cell's nucleus and changes gene activity—it turns off genes that make cytokines (inflammatory messenger proteins like TNF-alpha, IL-1, IL-6) and turns on genes that reduce inflammation
4. Immune cells (like T cells and macrophages) slow down, stop multiplying as fast, and release fewer attack chemicals
5. Swelling, pain, and damage in the gut lining decrease within days
6. Symptoms like diarrhea, bleeding, and cramping often improve quickly

SAFETY WATCH

Short-term (weeks): mood swings, trouble sleeping, increased appetite and weight gain, high blood sugar, acne, puffy face · Long-term (months or more): weakened bones (osteoporosis), high blood pressure, cataracts, increased infection risk, thinning skin, muscle weakness, stunted growth in children · NEVER stop steroids suddenly after using them for more than a couple of weeks—your adrenal glands (which make natural cortisol) need time to wake back up; your doctor will taper the dose slowly · Watch for signs of infection (fever, new pain) because steroids hide the immune response · Blood sugar and blood pressure should be monitored during use

Azathioprine or 6-mercaptopurine (6-MP) · Thiopurine immunomodulator

Azathioprine and 6-MP are drugs that slow down the immune system to prevent it from attacking the gut. They work by blocking the building blocks that immune cells need to

multiply. With fewer overactive immune cells, inflammation stays lower and flares happen less often.

1. You swallow the pill daily
2. Your liver converts azathioprine into 6-MP, and then into active chemicals called thioguanine nucleotides
3. These chemicals get incorporated into the DNA of rapidly dividing cells, especially T lymphocytes (immune cells that drive IBD inflammation)
4. The immune cells can no longer copy their DNA properly, so they stop multiplying as fast
5. With fewer active T cells, the attack on the gut lining slows down
6. Over weeks to months, inflammation decreases and the gut has time to heal; flares become less frequent

SAFETY WATCH

Can lower white blood cell count (making you more prone to infections), red blood cells (anemia), and platelets (raising bleeding risk)—your doctor will check blood counts every few weeks at first, then regularly · Can cause nausea or stomach upset (taking it with food and splitting the dose may help) · Rarely causes liver inflammation—liver blood tests are monitored · Slightly increases the risk of certain cancers (lymphoma, skin cancer) with long-term use—use sunscreen and get regular skin checks · Before starting, your doctor may test your TPMT or NUDT15 genes—some people break down the drug too slowly and are at higher risk of dangerous side effects; dose is adjusted or a different drug is chosen · Avoid live vaccines while on this drug; talk to your doctor about infection prevention

Methotrexate · Folate antagonist immunomodulator

Methotrexate slows down the immune system by blocking an enzyme that immune cells need to make DNA and reproduce. It is like cutting off the supply line that lets inflammatory cells multiply. With fewer active immune cells, the gut stays calmer and flares are less common.

1. You take methotrexate once a week, usually by mouth or as a shot under the skin
2. The drug enters cells and blocks an enzyme called dihydrofolate reductase, which is needed to recycle folate (a B vitamin)
3. Without enough active folate, cells cannot make the building blocks (purines and pyrimidines) needed to copy DNA
4. Immune cells that are multiplying quickly (like the T cells attacking the gut) slow down and stop dividing
5. Inflammation in the intestinal wall decreases over several weeks to months
6. The gut lining heals and symptoms improve; flares become less frequent

SAFETY WATCH

Can cause nausea, mouth sores, and fatigue—taking folic acid (a supplement) every day EXCEPT the day you take methotrexate reduces these side effects; your doctor will prescribe it · Can damage the liver—liver blood tests are checked regularly; avoid alcohol · Can lower blood cell counts—regular blood tests monitor white cells, red cells, and platelets · Can rarely cause lung inflammation (interstitial pneumonitis)—call your doctor if you develop a new dry cough or shortness of breath · Is TOXIC to a developing baby—women and men must use reliable birth control while on methotrexate and for months after stopping; do NOT get pregnant or father a child while taking this drug · Increases infection risk—watch for fever, sore throat, or unusual infections

Infliximab, adalimumab, golimumab, certolizumab (anti-TNF biologics) · Anti-TNF-alpha monoclonal antibody biologic

Anti-TNF biologics are lab-made antibodies that grab and block TNF-alpha, a powerful inflammatory protein that drives the gut damage in IBD. TNF-alpha is like a alarm signal that tells immune cells to attack; these drugs stick to TNF-alpha and silence it, so the inflammation stops spreading and the gut can heal.

1. You receive the drug by IV infusion (infliximab, some others) or injection under the skin (adalimumab, golimumab, certolizumab)
2. The biologic antibody travels through your bloodstream to areas of inflammation
3. It binds tightly to TNF-alpha molecules floating in the blood and stuck on the surface of immune cells
4. By blocking TNF-alpha, the drug stops it from attaching to TNF receptors on other cells
5. Without the TNF signal, immune cells stop multiplying, stop releasing more inflammatory chemicals, and start to die off (apoptosis)
6. Inflammation in the gut wall drops, ulcers begin to heal, and symptoms like diarrhea, bleeding, and pain improve—often within weeks

SAFETY WATCH

Increased risk of serious infections (tuberculosis, pneumonia, sepsis)—you will be tested for latent TB before starting and monitored for any signs of infection · Infusion or injection site reactions (redness, itching, pain at the site; rarely severe allergic reaction during infusion) · Slightly higher risk of certain cancers (lymphoma, skin cancer)—get regular skin checks and report any unusual lumps or symptoms · Can worsen heart failure—tell your doctor if you have any heart problems · Rarely can trigger lupus-like syndrome or nerve problems (numbness, tingling, vision changes)—report new symptoms immediately · Live vaccines should be avoided; make sure you are up to date on vaccines BEFORE starting therapy · Some patients develop antibodies against the drug over time, which can reduce its effectiveness or cause reactions—your doctor may check drug levels and antibody levels

Vedolizumab · Anti-integrin biologic (gut-selective)

Vedolizumab is a biologic antibody that blocks a specific traffic signal (integrin $\alpha4\beta7$) that immune cells use to enter the gut. It is like closing the gate so inflammatory T cells cannot get from the bloodstream into the intestinal wall. With fewer immune cells reaching the gut, inflammation stays lower and the lining can heal.

1. You receive vedolizumab by IV infusion in a clinic or hospital
2. The antibody circulates in your blood and binds to integrin $\alpha4\beta7$ on the surface of T cells
3. These T cells can no longer stick to MAdCAM-1, a protein on the inside lining of gut blood vessels that acts like a docking station
4. Unable to dock and enter the gut wall, the T cells stay in the bloodstream and eventually die or move elsewhere
5. With fewer T cells infiltrating the intestine, inflammation decreases
6. Ulcers heal, symptoms like diarrhea and bleeding improve, and the gut lining repairs itself over weeks to months

SAFETY WATCH

Infusion reactions are possible but usually mild (headache, nausea, itching); serious allergic reactions are rare · Increased risk of infections, though lower than anti-TNF drugs; still watch for signs like fever, persistent cough, or unusual pain · Rarely, progressive multifocal leukoencephalopathy (PML), a serious brain infection, has been reported—report any new confusion, weakness, vision changes, or trouble thinking immediately · Liver enzyme elevations can occur—your doctor will monitor blood tests · Live vaccines should be avoided; update vaccines before starting if possible

Ustekinumab · Anti-IL-12/IL-23 biologic

Ustekinumab is a biologic antibody that blocks two inflammatory messenger proteins: IL-12 and IL-23. These proteins push certain T cells (Th1 and Th17 cells) to multiply and release chemicals that damage the gut. By blocking IL-12 and IL-23, ustekinumab turns down the volume on inflammation and lets the gut heal.

1. You receive an initial IV infusion, then maintenance injections under the skin every 8 weeks
2. The antibody binds to IL-12 and IL-23 circulating in the blood and in the gut wall
3. By blocking these cytokines, it prevents them from signaling T cells to become Th1 and Th17 cells (the types that drive IBD inflammation)
4. Without the IL-12/23 signal, fewer inflammatory T cells are made and activated
5. Existing T cells release less TNF-alpha, interferon-gamma, and IL-17 (all inflammatory chemicals)
6. Inflammation in the gut drops, ulcers heal, and symptoms improve over weeks to months

SAFETY WATCH

Increased risk of infections, including tuberculosis—TB testing is required before starting · Injection site reactions (redness, swelling, itching) are common but usually mild · Headache, fatigue, and upper respiratory infections may occur · Rarely, serious allergic reactions (anaphylaxis) during or after infusion or injection · Slightly increased cancer risk (skin cancers, possibly others) with long-term immune suppression—get regular skin checks · Live vaccines should be avoided; update vaccines before therapy if possible · Report any new lumps, persistent cough, night sweats, unexplained weight loss, or signs of infection immediately

Tofacitinib, upadacitinib (JAK inhibitors) · Janus kinase (JAK) inhibitor (small-molecule oral immunosuppressant)

JAK inhibitors are pills that block enzymes inside immune cells called Janus kinases. These enzymes relay signals from inflammatory proteins outside the cell to the cell's nucleus, telling it to make more inflammation. By blocking JAKs, these drugs turn down multiple inflammatory signals at once, calming the immune attack on the gut.

1. You swallow the pill once or twice a day
2. The drug enters cells and blocks JAK enzymes (JAK1, JAK2, JAK3, or TYK2, depending on the drug)
3. JAKs normally pass signals from cytokine receptors (like those for IL-6, IL-12, IL-23, interferon) into the nucleus
4. Without working JAK enzymes, the inflammatory cytokine signals cannot get through

5. The cell does not turn on genes for making more inflammatory proteins or multiplying
6. Inflammation in the gut wall decreases, ulcers heal, and symptoms improve within weeks

SAFETY WATCH

Increased risk of serious infections (shingles, tuberculosis, pneumonia, urinary tract infections)—TB testing and shingles vaccine (if eligible) before starting are recommended · Increased risk of blood clots (deep vein clot formation (thrombosis), pulmonary embolism (a clot lodging in a lung artery), especially in older adults or those with heart risk factors)—report leg pain, swelling, chest pain, or shortness of breath immediately · Can raise cholesterol levels—your doctor will monitor lipids and may start a statin · Can lower blood cell counts—regular blood tests check white cells, red cells, and platelets · Slightly increased cancer risk (lymphoma, lung cancer in smokers, skin cancer)—do not smoke, get skin checks, and report unusual symptoms · Liver enzyme elevations—monitored with blood tests · May worsen diverticulitis or cause new tears in the intestine—report severe abdominal pain, fever, or change in bowel habits immediately · Avoid live vaccines; update vaccines (especially shingles and pneumonia) before starting if possible

Red flags

- Severe belly pain that doesn't ease, or a belly that becomes hard and very tender (could mean a perforation—a hole in the intestine—or severe blockage)
- Heavy rectal bleeding that soaks through toilet paper quickly or makes you dizzy (sign of major blood loss)
- High fever over 101.5°F (38.6°C) with shaking chills, especially with worsening belly pain (could mean a serious infection or abscess)
- Sudden inability to pass stool or gas, with severe cramping and vomiting (signs of a bowel obstruction)
- Signs of severe dehydration: very little urine, dark urine, extreme thirst, confusion, or a racing heart (from fluid loss through diarrhea)

Nursing priorities

1. Monitor vital signs every 4 hours and report temperature over 100.4°F, heart rate over 90, systolic blood pressure under 90, or signs of dehydration like dry mouth and decreased urine output. — A fast heart rate (Tachycardia), fever, and low blood pressure (hypotension) signal severe inflammation, sepsis, or hypovolemia (low blood volume) from diarrhea and bleeding, which can lead to shock and organ failure without prompt fluid resuscitation and antibiotics.

2. Assess stool frequency, volume, consistency, and presence of blood or mucus every shift, and document using the Bristol Stool Scale and color description. — Objective tracking separates mild flares treatable with oral mesalamine from severe colitis needing IV corticosteroids or biologic rescue therapy, and monitors response to treatment over days.
3. Examine the abdomen for distension, firmness, bowel sounds, and tenderness with each assessment, and immediately notify the provider if rigidity or rebound pain develops. — Toxic megacolon causes the colon to dilate beyond 6 centimeters with risk of perforation; absent bowel sounds and peritoneal signs mean emergency surgery may be needed to prevent death from peritonitis (infection in the belly cavity).
4. Check hemoglobin and hematocrit levels, inspect skin and conjunctiva for pale skin (pallor), and watch for fatigue, dizziness, or shortness of breath that signal anemia. — Chronic gut bleeding depletes iron stores and red blood cells, causing tissue low oxygen (hypoxia) (low oxygen delivery); hemoglobin under 7 grams per deciliter may require blood transfusion to prevent cardiac stress and organ damage.
5. Maintain IV access and administer fluids with electrolytes as ordered, monitor intake and output hourly, and replace potassium and magnesium when lab values are low. — Severe diarrhea causes loss of sodium, potassium, and magnesium through stool; low potassium (hypokalemia) (low potassium) can trigger dangerous heart rhythms, and dehydration reduces blood flow to the kidneys causing acute injury.
6. Provide perianal skin care after each bowel movement with barrier cream, teach sitz baths for comfort, and inspect for new fissures, abscesses, or fistula drainage. — Acidic diarrhea erodes skin integrity, creating entry for bacteria; perianal abscesses can seed bacteremia (bacteria in blood), and untreated fistulas (abnormal tunnels) lead to sepsis or require surgical drainage.
7. Administer ordered corticosteroids, immunosuppressants, or biologic infusions on time, pre-medicate for infusion reactions, and monitor for infection signs since these drugs suppress immune defense. — Delayed or missed doses of biologic agents like infliximab allow antibody formation that reduces drug effectiveness; immunosuppression increases risk of opportunistic infections like tuberculosis reactivation or fungal pneumonia that can be fatal.
8. Screen for anxiety and depression using validated tools, provide emotional support, and connect the patient with IBD support groups or counseling resources. — Chronic unpredictable symptoms, body-image concerns from ostomy or perianal disease, and medication side effects cause rates of depression up to 25 percent in IBD patients, worsening treatment adherence and quality of life.

Sources

- ACG Clinical Guideline: Management of Crohn's Disease in Adults (2018)

- ACG Clinical Guideline: Ulcerative Colitis in Adults (2019)
- Crohn's & Colitis Foundation
- CDC – Inflammatory Bowel Disease

Last reviewed: 2026-07-07