

Neutropenic Fever

Fever in a patient whose neutrophil count is below 500 cells per microliter—no white blood cell army left to fight infection.

THE STORY IN ONE SENTENCE

Fever in a patient whose neutrophil count is below 500 cells per microliter—no white blood cell army left to fight infection.

“Neutropenic fever means fever when you cannot make pus. Start broad antibiotics within one hour or bacteria win.”

Normal anatomy — the body's job

Neutrophils are white blood cells made in the bone marrow—the spongy factory inside your bones. They are your body's first responders against bacteria and fungi. A healthy adult keeps 2,500 to 7,000 neutrophils in every microliter of blood, ready to swarm any infection site within minutes.

BODY'S JOB

When bacteria enter through a cut, your gut lining, or an IV line, neutrophils rush to the spot, surround the germs, swallow them, and release chemicals that kill the invaders. This battle creates pus—a thick fluid packed with dead neutrophils, bacteria, and tissue debris. Pus, redness, warmth, and swelling are all signs that neutrophils are working. Without enough neutrophils, infections spread silently because the alarm system is broken.

1. Bone marrow stem cells divide every day to make new neutrophils.
2. Neutrophils mature in the marrow over about two weeks, then enter the bloodstream.
3. They patrol blood vessels, squeezing through vessel walls into tissues when chemical danger signals appear.
4. At the infection site, neutrophils swallow bacteria, release toxic chemicals, and call more immune cells for backup.
5. Used-up neutrophils die after a few hours and become part of pus, so the marrow must keep making replacements constantly.

What breaks

Chemotherapy drugs kill fast-dividing cells to stop cancer, but they also kill the bone marrow stem cells that make neutrophils. The marrow factory shuts down, neutrophil counts fall below 500 per microliter, and the infection army disappears. Bacteria from your gut, skin, or central line—germs that healthy neutrophils would stop in minutes—now spread unopposed into the bloodstream.

CLINICAL TERM

Chemotherapy-induced myelosuppression destroys hematopoietic stem cells in the bone marrow, dropping the absolute neutrophil count (ANC) below 500 cells/ μ L and eliminating the innate immune response to bacterial and fungal pathogens. Gram-negative bacteria (*Escherichia coli*, *Klebsiella*, *Pseudomonas*) translocate from the gut through damaged mucosa. Gram-positive organisms (*Staphylococcus aureus*, coagulase-negative staph, *Streptococcus*) seed from skin and central venous catheters. Fungal infections (*Candida*, *Aspergillus*) emerge after seven to ten days of profound dangerously low infection-fighting cells (neutropenia) because neutrophils are required to kill fungal hyphae. The broken piece is the absent neutrophil response—fever is often the only clinical sign because there are no neutrophils to produce local inflammation.

How the body compensates

The body tries to keep blood pressure and oxygen delivery steady even as bacteria flood the bloodstream. It releases stress hormones, squeezes blood vessels, and raises heart rate to hold things together. These tricks work for a short window, so vital signs can look deceptively normal while sepsis builds. The danger is missing the crash point—once bacteria release enough toxins, blood vessels leak, the heart weakens, blood pressure collapses into septic shock, and organs fail fast.

1. {'step': "The hypothalamus (the brain's thermostat in the center of the brain) raises body temperature to make fever.", 'why': "Fever is triggered by cytokines (immune alarm chemicals) released when bacteria enter the bloodstream. Heat slows bacterial growth and speeds immune cell activity—it is the body's first defense when neutrophils are gone."}
2. {'step': 'The adrenal glands (small glands sitting on top of each kidney) release epinephrine and norepinephrine (stress hormones that prepare the body for danger).', 'why': 'These hormones make the heart beat faster and squeeze blood vessels tighter to keep blood pressure up and push oxygen to vital organs like the brain and heart, even as infection spreads.'}

3. {'step': 'The heart rate climbs (tachycardia) to pump more blood per minute.', 'why': 'With fewer oxygen-carrying red cells reaching tissues because blood vessels are leaking and blood flow is uneven, the heart compensates by beating faster to move whatever blood is left.'}
4. {'step': 'Blood vessels in the skin, gut, and muscles constrict (narrow) to keep blood flowing to the brain, heart, and kidneys.', 'why': 'The body sacrifices less vital areas to protect organs that will kill you fastest if they fail—this is why hands and feet may feel cool even when core temperature is high.'}
5. {'step': 'The liver and spleen try to filter bacteria from the blood, but without neutrophils to kill the germs, bacteria keep multiplying.', 'why': 'Filtering slows the flood but does not stop it—bacteria double every twenty to thirty minutes in the bloodstream when neutrophils are absent.'}
6. {'step': 'The kidneys hold onto sodium and water to keep blood volume up and prevent blood pressure from dropping.', 'why': 'If blood pressure falls, organs do not get enough oxygen. Holding fluid is a short-term fix, but it fails once blood vessels start leaking from bacterial toxins.'}
7. {'step': 'All these tricks buy time—minutes to hours—but they cost energy and cannot last. The crash point is septic shock: bacteria release endotoxins (poisons from bacterial cell walls) that make blood vessels leak and the heart pump weakly. Blood pressure collapses, organs starve for oxygen, and multi-organ failure follows.', 'why': 'Compensation works only while the body has reserves. Once bacterial toxins overwhelm the system, every organ begins to fail at once because they all depend on steady blood flow and oxygen.'}
8. {'step': 'The goal is to start broad-spectrum antibiotics within one hour of fever to kill bacteria before the crash point arrives.', 'why': 'Every hour of delay increases mortality because bacteria double so fast. Antibiotics stop the toxin flood and give the body time to recover marrow function and rebuild the neutrophil army.'}

Domino effect

1. Chemotherapy kills bone marrow stem cells. — Chemotherapy targets all fast-dividing cells to stop cancer, but it cannot tell cancer cells from the stem cells in bone marrow that make neutrophils, so both are destroyed.
2. The absolute neutrophil count (ANC) drops below 500 cells per microliter within days to weeks. — Neutrophils only live a few hours in the bloodstream, so once the marrow stops making new ones, the circulating count falls fast—below 500 is the danger zone where infection risk skyrockets.

3. Bacteria from the gut (*E. coli*, *Klebsiella*, *Pseudomonas*), skin, or central line (*Staph aureus*, coagulase-negative staph) enter the bloodstream unopposed. — Healthy neutrophils kill bacteria within minutes of contact. With fewer than 500 neutrophils per microliter, there is no army to stop the invaders, so bacteria spread from the gut or catheter site into the blood.
4. Fever appears—often the only sign of infection. — Bacteria in the bloodstream trigger the hypothalamus (the brain's thermostat) to raise body temperature through cytokines (immune alarm chemicals). There is no swelling, redness, or pus because neutrophils are needed to make those signs.
5. The body releases epinephrine, norepinephrine, and cortisol (stress hormones) to hold blood pressure steady. — These hormones squeeze blood vessels, speed the heart, and keep blood flowing to the brain and heart. This buys time, so vital signs can look deceptively normal for a short window even as bacteria multiply.
6. Bacteria double every twenty to thirty minutes in the bloodstream, releasing endotoxins (poisons from bacterial cell walls). — Without neutrophils to kill them, bacteria grow exponentially. Endotoxins trigger a massive inflammatory cascade that damages blood vessel linings and makes vessels leak fluid into tissues.
7. Blood vessels leak, the heart pumps weakly, and blood pressure crashes into septic shock. — Endotoxins activate the endothelium (the inner lining of blood vessels), causing widespread leaking. Fluid shifts out of vessels into tissues, the heart cannot pump enough blood forward, and blood pressure collapses.
8. Organs starve for oxygen—kidneys fail, lungs fill with fluid (ARDS), the brain becomes confused, and the liver cannot clear toxins. — Every organ depends on steady blood flow and oxygen delivery. When blood pressure drops and vessels leak, oxygen does not reach tissues, so cells switch to anaerobic metabolism (making energy without oxygen), which produces lactic acid and fails quickly. Multi-organ failure follows.

Symptoms · what the patient feels, and why

- Fever (temperature at or above 100.4°F / 38°C once, or 100.4°F for one hour or longer): When white blood cells drop too low (neutropenia), the body cannot fight off germs that sneak in from the skin, gut, or lungs. Even a small infection that would normally be stopped right away can now spread quickly. The immune system raises the body's heat to try to slow the germs down — this is the fever. Because there are so few fighter cells, this fever is a serious alarm: something is wrong and help is needed fast.

- Chills and sweating: The hypothalamus (the brain's thermostat, sitting deep in the middle of the brain) resets the body's target temperature higher to fight infection. Muscles shake (chills) to make heat and raise the temperature up to the new set point. Once the body gets there, sweating cools it back down. In neutropenic fever, these swings can be intense because even a tiny infection can trigger a big inflammatory surge.
- Redness, warmth, swelling, or pus around central line sites (where IV catheters enter the skin): A central line is a tube placed into a big vein to give medicines or draw blood. Skin bacteria (like *Staphylococcus*) can crawl down the outside of the catheter and infect the tunnel under the skin or the bloodstream itself. The body tries to send immune cells to the spot, causing redness and warmth, but with so few white cells available, the infection spreads easily instead of being walled off.
- Pain, redness, or sores in the mouth or throat: The lining of the mouth and throat sheds and regrows constantly. Chemotherapy damages the fast-growing cells that rebuild that lining, leaving raw spots (mucositis). Bacteria and fungi that normally live in the mouth can now invade those raw areas. With dangerously low infection-fighting cells (neutropenia), the body has no troops to keep them in check, so sores spread and infections take hold quickly.
- Pain or tenderness around the rectum (perirectal area): The skin around the anus and the lining just inside the rectum are thin and can tear easily, especially if stools are hard or if the area is irritated. Bacteria from stool can enter those tiny breaks. Normally white blood cells rush in and seal the infection off into a small abscess, but in dangerously low infection-fighting cells (neutropenia) there are not enough cells to do that, so the infection can spread into deeper tissues and the bloodstream.
- New cough or shortness of breath: Bacteria or fungi (like *Aspergillus*, a mold that lives in the air and soil) can settle into the lungs. With low white blood cells, the lungs cannot clear out these invaders, and patches of infection (pneumonia) form. Inflamed lung tissue gets stiff and fills with fluid, making it hard to breathe. Because neutropenic patients cannot mount a strong immune response, the usual thick phlegm and infiltrates on X-ray may be missing at first, but the infection is still dangerous.
- Confusion or sudden change in thinking: Infection can release toxins and trigger widespread inflammation (sepsis) that reduces blood flow and oxygen delivery to the brain. Low blood pressure from septic shock means the brain does not get enough fuel. Also, bacteria or fungi can rarely cross into the brain itself (meningitis or encephalitis). Any of these paths can scramble the brain's electrical signals, causing confusion, drowsiness, or disorientation.

- Low blood pressure (hypotension) or fast heart rate (tachycardia): When infection spreads into the bloodstream (sepsis), bacteria release toxins that make blood vessels leak and widen. Blood pressure drops because the pipes are too wide and leaky to keep pressure up. The heart speeds up (tachycardia) to pump harder and try to keep enough blood flowing to the brain and kidneys. In neutropenic fever, this can happen very fast because there are no white cells to contain the infection early.

Focused exam

- Temperature $\geq 38.3^{\circ}\text{C}$ (100.9°F) or $\geq 38.0^{\circ}\text{C}$ (100.4°F) for ≥ 1 hour in a patient with ANC < 500 : This is the definition of neutropenic fever. Neutrophils (the white blood cells that eat bacteria) are nearly gone, so your body cannot make pus, redness, or swelling when germs invade. Fever is often the ONLY warning sign. The thermometer reading IS the alarm bell.
- Central line site (if present): look for warmth, redness, oozing, or tenderness along the tunnel under the skin: Central lines (the plastic tubes that deliver medicine directly into big veins) sit right where Staphylococcus and other skin bacteria live. Even when the line is infected, the site may look almost normal because there are no neutrophils to cause swelling. You must examine it closely every time—central line infection is one of the most common sources in neutropenic fever.
- Mouth and throat: look for white cottage-cheese patches (oral candidiasis), painful sores, or bleeding gums: The mouth is crawling with bacteria and fungi that normally stay put. Without neutrophils to guard the doorway, these germs slide through tiny cracks in the gums or tongue and enter the bloodstream silently. Mouth infection is a hidden but common starting point in neutropenic fever.
- Perirectal area: gently check (do NOT do a rectal exam) for pain, tenderness, swelling, or drainage: The rectum and lower bowel hold huge numbers of gram-negative bacteria (like E. coli and Pseudomonas) that can tunnel into the bowel wall and cause typhlitis (infection and swelling of the cecum, the pouch where the small intestine meets the colon). This can turn deadly fast. Pain or tenderness may be the only early clue because there are no neutrophils to make the area visibly swollen or red.
- Lung exam: listen for crackles, wheezes, or absent breath sounds; check oxygen saturation with a pulse oximeter: Pneumonia (infection in the air sacs of the lungs) and sepsis (when infection spreads through the bloodstream and triggers body-wide inflammation) can both attack the lungs and make them fill with fluid or collapse. Low oxygen ($\text{SpO}_2 < 94\%$ on room air) means the lungs are struggling and septic shock may be close.

- Blood pressure, heart rate, and skin blood flow (perfusion): is the skin warm and pink, or cold, clammy, and pale?: In septic shock, blood vessels leak fluid and lose their ability to squeeze, so blood pressure drops and organs do not get enough blood. The tricky part: blood pressure can stay normal for a while (the heart beats faster to make up for it), then suddenly fall. Cold, clammy skin and a fast pulse mean shock is starting—organs are running out of oxygen and you are running out of time.
- Mental status: is the patient alert, answering questions, and tracking conversation, or confused, slow, or drowsy?: The brain is extremely sensitive to low oxygen. Confusion, sleepiness, or slow responses mean the brain is not getting enough blood or oxygen—a sign that sepsis has spread and the body is losing the fight. This is a red flag that you need immediate, aggressive treatment.

Diagnostics · every test answers a question

- Blood cultures (two sets: one from each lumen of the central line if present, and one from a peripheral vein) → What bacteria or fungus is growing in the bloodstream, and where did it come from?
- Complete blood count (CBC) with differential → Exactly how low is the absolute neutrophil count (ANC), and are platelets or red blood cells also dropping?
- Serum lactate → Are the tissues getting enough oxygen, or is the body switching to emergency backup energy because blood flow is failing?
- Chest X-ray (posteroanterior and lateral) → Is there pneumonia (infection in the lungs), fluid buildup, or a collapsed lung?
- Comprehensive metabolic panel (CMP): creatinine, blood urea nitrogen (BUN), liver enzymes (ALT, AST), bilirubin → Are the kidneys and liver still working, or are they starting to fail from low blood flow or sepsis?
- Urinalysis and urine culture (if urinary symptoms present or no other source found) → Is the bladder or kidney infected?
- Chest CT scan (if chest X-ray is normal but patient has cough, shortness of breath, or low oxygen) → Is there early pneumonia, fungal infection (like *Aspergillus*), or a lung abscess that the X-ray missed?
- Procalcitonin (optional, not required for initial diagnosis but sometimes used to track treatment response) → Is this bacterial infection, and is it getting better or worse?

Treatment logic

- Broad-spectrum antibiotic with anti-pseudomonal coverage (cefepime, piperacillin-tazobactam, or meropenem) within 60 minutes of presentation → Kills the most dangerous bacteria—gram-negative organisms like *Pseudomonas aeruginosa* and *E. coli* from the gut, and gram-positive organisms like *Staphylococcus* from skin and lines—before they multiply unchecked in the bloodstream. Without neutrophils, the body cannot slow the bacteria down, so the antibiotic must do all the work. Every hour of delay increases the risk of septic shock and death.
- Add vancomycin (or linezolid if vancomycin-resistant) if the patient is hemodynamically unstable (low blood pressure, high lactate), central line infection is suspected, MRSA is known or common in your hospital, or there is skin/soft tissue involvement → Covers methicillin-resistant *Staphylococcus aureus* (MRSA) and other resistant gram-positive bacteria that the first antibiotic may not kill. Central lines are coated with skin bacteria, and MRSA is especially dangerous in neutropenic patients because it can cause aggressive bloodstream infection or endocarditis (infection of the heart valves). Adding vancomycin closes the gap when the infection is more severe or when the source is likely the line or skin.
- Blood cultures (before antibiotics), careful physical exam of central line sites, perirectal area, mouth, and lungs; chest X-ray → Identifies the source of infection (line, gut, mouth, lungs) and the exact organism growing in the blood. This guides whether to remove a contaminated central line, switch antibiotics based on culture sensitivities, or add antifungals if cultures stay negative but fever persists. The exam and imaging tell you where to look; the cultures tell you what you are fighting.
- Add empiric antifungal therapy (echinocandin like micafungin or caspofungin, or voriconazole) if fever persists beyond 4–7 days on broad-spectrum antibiotics and dangerously low infection-fighting cells (neutropenia) continues → Kills fungi—especially *Candida* (yeast) and *Aspergillus* (mold)—that grow when neutrophils stay low for a long time. Fungi are slower to cause symptoms than bacteria, so they are not the first suspect, but they become the main suspect if fever does not break after several days of antibiotics. Adding an antifungal stops a second, hidden infection from taking over while the bone marrow is still recovering.
- IV fluids (crystalloid like normal saline or lactated Ringer's, 30 mL/kg bolus) and vasopressors (norepinephrine) if blood pressure drops, lactate rises, or signs of shock appear → Restores blood volume and squeezes blood vessels to maintain blood flow to vital organs (brain, heart, kidneys) when septic shock causes vessels to leak fluid and lose tone. Fluid alone often is not enough—norepinephrine (a medication that mimics adrenaline) tightens blood vessels and keeps pressure up while antibiotics work. This buys time for the immune system (and the antibiotics) to catch up.

- Consider granulocyte colony-stimulating factor (G-CSF, filgrastim) in high-risk patients (ANC <100, prolonged dangerously low infection-fighting cells (neutropenia) expected, severe infection, organ dysfunction) to accelerate neutrophil recovery → G-CSF is a protein that tells the bone marrow to make neutrophils faster. It shortens the window of danger by bringing neutrophils back sooner (usually 1–3 days earlier). This is most helpful in patients with deep dangerously low infection-fighting cells (neutropenia) (ANC <100) or septic shock, where every extra day without neutrophils raises the risk of death. It does NOT work for everyone—some patients' marrows are too damaged by chemotherapy to respond.
- Remove the central line if blood cultures from the line turn positive much earlier than peripheral cultures, the line site looks infected, or the patient stays sick despite appropriate antibiotics (especially if *Staphylococcus aureus*, *Pseudomonas*, or *Candida* is growing) → Takes away the plastic surface where bacteria or fungi have formed a biofilm (a sticky shield that antibiotics cannot penetrate). Some organisms—especially *Staph aureus*, *Pseudomonas*, and *Candida*—cling to the line and keep reseeding the bloodstream even while antibiotics are running. Removing the line removes the source and lets the antibiotics finally clear the infection.

Medications

Cefepime · Antipseudomonal beta-lactam antibiotic (fourth-generation cephalosporin)

Cefepime punches holes in the walls of bacteria, causing them to burst and die. It is especially strong against *Pseudomonas aeruginosa* and gram-negative bacteria that often cause infections when white blood cells are very low.

1. Cefepime enters the bacterial cell wall and finds penicillin-binding proteins (the construction workers that build and repair the wall).
2. It locks onto these proteins and stops them from connecting the wall's building blocks together.
3. Without a strong wall, water rushes into the bacterium and the cell pops like an overfilled water balloon.
4. The infection begins to clear as bacteria die off.

SAFETY WATCH

Can cause allergic reactions, especially if you are allergic to penicillin—tell your doctor about any antibiotic allergies before starting. · May trigger seizures in patients with kidney disease or if the dose is too high—kidney function is checked and the dose adjusted. · Can cause diarrhea, including *C. difficile* infection (a dangerous gut infection) if normal gut bacteria are wiped out. · Must be dosed carefully in patients with kidney problems because it is cleared through the kidneys.

Piperacillin-tazobactam · Antipseudomonal beta-lactam antibiotic (penicillin plus beta-lactamase inhibitor)

Piperacillin punches holes in bacterial walls just like cefepime. Tazobactam is its bodyguard—it blocks the scissors (enzymes called beta-lactamases) that some bacteria use to cut and destroy piperacillin, so the antibiotic can keep working.

1. Piperacillin binds to penicillin-binding proteins in the bacterial wall, stopping wall repair.
2. Tazobactam binds to and disables beta-lactamase enzymes that bacteria use to chew up antibiotics.
3. With the scissors blocked, piperacillin can freely destroy the wall.
4. Bacteria burst and die, and the infection begins to clear.

SAFETY WATCH

Can cause severe allergic reactions, especially in patients allergic to penicillin—always tell your doctor about drug allergies. · May cause low potassium levels (hypokalemia), especially with high doses or long treatment. · Can lead to *C. difficile* diarrhea by wiping out healthy gut bacteria. · Requires dose adjustment in kidney disease because it is cleared by the kidneys.

Meropenem · Antipseudomonal carbapenem antibiotic

Meropenem is one of the strongest bacterial wall-breakers we have. It works against a very wide range of bacteria, including many that resist other antibiotics. It is used when the infection is severe or when resistant bacteria are suspected.

1. Meropenem enters bacteria and binds tightly to penicillin-binding proteins, stopping wall construction.
2. Unlike many other antibiotics, meropenem resists destruction by most bacterial enzymes, so it stays active longer.
3. The bacterial wall weakens and the cell bursts from internal pressure.
4. Even tough bacteria die, and the infection clears.

SAFETY WATCH

Can cause seizures, especially in patients with brain injury, kidney disease, or high doses—neurologic monitoring is important. · May cause allergic reactions; patients with penicillin or cephalosporin allergies have a small risk of cross-reaction. · Can lead to *C. difficile* diarrhea by disrupting normal gut bacteria. · Requires dose adjustment in kidney disease.

Vancomycin · Glycopeptide antibiotic

Vancomycin is added when doctors suspect MRSA (methicillin-resistant *Staphylococcus aureus*), a tough skin bacterium, or when there is an infection in a central line (IV catheter). It works by blocking the final step of bacterial wall construction, causing the wall to fall apart.

1. Vancomycin binds to the building blocks (D-alanyl-D-alanine) of the bacterial wall before they can be linked together.
2. Without those links, the wall stays weak and incomplete.
3. The bacterium cannot withstand its own internal pressure and bursts.
4. MRSA and other resistant gram-positive bacteria are killed.

SAFETY WATCH

Can damage the kidneys (nephrotoxicity), especially when combined with other kidney-toxic drugs or given in high doses—kidney function is monitored closely. · Can cause 'red man syndrome'—flushing, itching, and low blood pressure if infused too quickly; must be given slowly over at least 60 minutes. · May damage hearing (ototoxicity) with prolonged use or high blood levels. · Blood levels are checked regularly to keep the dose safe and effective.

Micafungin (or other echinocandin: caspofungin, anidulafungin) · Echinocandin antifungal

If fever continues for 4 to 7 days despite antibiotics, doctors suspect a fungal infection, usually *Candida*. Micafungin attacks the fungal cell wall by blocking beta-1,3-D-glucan synthase, the enzyme that builds the wall's main scaffolding. Without that scaffold, the fungal cell wall collapses.

1. Micafungin enters the fungus and binds to beta-1,3-D-glucan synthase, the builder enzyme.
2. The enzyme is blocked and cannot make glucan, the tough fiber that holds the fungal wall together.
3. The wall weakens and the fungus dies or stops growing.
4. *Candida* infections in the blood and tissues begin to clear.

SAFETY WATCH

Generally well tolerated with fewer side effects than older antifungals. · Can cause liver enzyme elevation—liver tests are monitored during treatment. · May cause histamine-related reactions (flushing, rash) during infusion, though rare. · Rarely causes low blood counts or kidney problems.

Voriconazole · Triazole antifungal

Voriconazole is used for suspected or proven mold infections, especially *Aspergillus*, which can invade the lungs and sinuses in neutropenic patients. It blocks an enzyme (14-alpha-demethylase) that fungi need to make ergosterol, a key fat in their cell membrane. Without ergosterol, the membrane leaks and the fungus dies.

1. Voriconazole enters the fungus and blocks 14-alpha-demethylase, the enzyme that turns lanosterol into ergosterol.
2. Without ergosterol, the fungal membrane becomes unstable and leaky.
3. Toxic substances enter the fungus and essential contents leak out.
4. The fungus stops growing and dies; mold infections begin to clear.

SAFETY WATCH

Can cause visual disturbances (blurry vision, bright lights, color changes)—these are usually temporary but tell your doctor. · May cause serious liver damage—liver enzymes are checked regularly. · Can cause hallucinations, confusion, or nightmares, especially at high blood levels. · Interacts with many other drugs through the liver enzyme CYP2C19—drug levels are monitored and doses adjusted. · Can make skin very sensitive to sunlight—use sunscreen and protective clothing.

Filgrastim (G-CSF, granulocyte colony-stimulating factor) · Hematopoietic growth factor

Filgrastim is a man-made version of a natural signal (G-CSF) that tells the bone marrow to speed up production of neutrophils (infection-fighting white blood cells). It is used in selected high-risk patients to shorten the time the immune system is dangerously low.

1. Filgrastim binds to G-CSF receptors on the surface of stem cells and immature neutrophils in the bone marrow.
2. The signal activates genes that speed up cell division and maturation.
3. More neutrophils are made and released into the bloodstream faster.
4. The neutrophil count rises, improving the body's ability to fight infection while antibiotics do their work.

SAFETY WATCH

Can cause bone pain (aching in the hips, back, or long bones) because the marrow is working hard—usually managed with acetaminophen or ibuprofen. · Rarely causes the spleen to enlarge or even rupture—tell your doctor if you have sudden left upper belly pain. · Can rarely trigger acute respiratory distress syndrome (ARDS), especially in patients with lung disease. · Should not be given within 24 hours before or after chemotherapy to avoid overstimulating damaged marrow.

Red flags

- Temperature at or above 100.4°F (38°C) in anyone with known low white blood cells (absolute neutrophil count below 500 cells per microliter)
- Signs of septic shock: confusion, very low blood pressure, fast breathing, cold or mottled skin, or little to no urine output
- Severe pain, redness, or drainage around a central line, port, or catheter site
- New trouble breathing, chest pain, or coughing up blood
- Severe belly pain or inability to have a bowel movement, which may signal a perirectal abscess or infection in the gut wall

Nursing priorities

1. Notify the provider immediately and prepare to draw two sets of blood cultures from different sites before any antibiotics are given. — Blood cultures identify the exact bacteria causing infection so antibiotics can be adjusted later. Drawing before antibiotics gives the best chance of growing bacteria in the lab. Two separate draws reduce false positives from skin contamination.
2. Administer broad-spectrum antibiotics within 60 minutes of fever recognition, using cefepime, piperacillin-tazobactam, or meropenem as ordered. — Neutropenic patients have almost no white blood cells to fight infection. Every hour of delay increases risk of septic shock and death. Broad-spectrum antibiotics kill many different bacteria types until culture results identify the specific bug.
3. Take full vital signs including blood pressure, heart rate, respiratory rate, oxygen saturation, and temperature every 1 to 2 hours until stable. — Neutropenic patients can progress to septic shock quickly because they lack the white blood cells that usually contain infection. Low blood pressure, high heart rate, fast breathing, or low oxygen signal deterioration requiring immediate intervention.

4. Examine the central line site, any IV sites, mouth, throat, and skin very carefully for redness, swelling, drainage, or sores. Inspect but never perform a digital rectal exam. — Neutropenic patients may not show typical signs of infection like pus because white blood cells create pus. Careful visual inspection finds hidden infection sources. Digital rectal exams can tear fragile tissue and introduce bacteria into the bloodstream.
5. Place the patient in a private room and enforce strict hand hygiene for all staff, visitors, and the patient. — Neutropenic patients are highly susceptible to any bacteria or virus. Hand hygiene with soap and water or alcohol gel kills most germs before they can enter the patient's body through touch or contact with surfaces.
6. Monitor intake and output hourly and maintain IV access for fluids and antibiotics. — Sepsis causes vasodilation (widening of blood vessels) and capillary leak (fluid escaping into tissues), leading to low blood pressure. IV fluids keep blood pressure adequate for organ blood flow (perfusion) (blood flow to kidneys, brain, and heart).
7. Hold all medications and foods that could injure the mouth or rectal lining, including suppositories, enemas, rectal thermometers, and hard or sharp foods. — Any break in the mucosa (lining of the mouth and intestines) creates an entry point for bacteria. Without neutrophils (infection-fighting white blood cells), even tiny tears can cause life-threatening bloodstream infections.
8. Document the absolute neutrophil count (ANC) and notify the provider if fever continues beyond 3 days or if the patient develops new symptoms like cough, rash, or increased confusion. — Persistent fever despite broad antibiotics suggests resistant bacteria, fungal infection, or a non-infectious cause. Fungal infections like Candida or Aspergillus become more likely after 4 to 7 days of antibiotics, requiring antifungal medication.

Sources

- IDSA Clinical Practice Guideline for the Use of Antimicrobial Agents in Neutropenic Patients with Cancer (2010 update)
- NCCN Guidelines: Prevention and Treatment of Cancer-Related Infections (2023)
- CDC Healthcare Infection Control Practices Advisory Committee (HICPAC)
- American Society of Clinical Oncology (ASCO) guidelines on fever and neutropenia

Last reviewed: 2026-07-07