

# Leukemia and Lymphoma

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## THE STORY IN ONE SENTENCE

Leukemia is when one white blood cell in the bone marrow loses its growth controls and multiplies out of control, crowding out normal blood cells.

“Learn the leukemia and lymphoma patterns first — each type grows differently and needs different treatment — then treat the exact disease you have.”

## Normal anatomy — the body's job

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Bone marrow (the spongy factory inside your bones) makes red cells, white cells, and platelets in perfect balance every day. Lymph nodes (tiny bean-shaped filters scattered throughout your body) catch germs and help white cells do their job. Lymphocytes (one kind of white cell) grow up in the marrow, travel through blood and lymph fluid, patrol for invaders, and die on schedule when they are old. Keep that picture in your head, because every weird finding only makes sense when you see it as a change from normal.

## BODY'S JOB

The marrow's job is to keep making the right number of each cell type — enough red cells to carry oxygen, enough white cells to fight infection, and enough platelets to stop bleeding. Lymph nodes filter fluid from your tissues and give white cells a place to meet germs and multiply when needed. Lymphocytes are supposed to grow, work, and die on a clock so they never pile up. If you do not know this normal job first, the weird findings seem random and confusing.

1. Stem cells in the bone marrow split and grow into three lines: red cells, white cells (including lymphocytes, neutrophils, monocytes), and platelets.
2. Each line matures step by step — baby cells (blasts) grow into working adult cells — then leave the marrow and enter the blood.

3. Red cells carry oxygen for about 120 days, then die. Neutrophils (the main infection-fighting white cell) live only hours to days. Platelets last about 10 days. Lymphocytes can live months to years, patrolling and remembering past infections.
4. Lymphocytes move between blood, lymph fluid, and lymph nodes — they scan for invaders, multiply when they find one, then most die when the job is done, keeping the system in balance.
5. The marrow watches how many cells are out in the blood and makes more or fewer to keep the count steady, like a thermostat keeping a room at the right temperature.

## What breaks

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One cell in the bone marrow or a lymph node picks up a DNA mistake (mutation) that breaks its growth controls. Instead of growing up, working, and dying on schedule, it keeps copying itself forever, like a factory machine stuck in the 'on' position. That one broken cell becomes two, then four, then millions, crowding out normal cells and stealing their space and food.

### CLINICAL TERM

A somatic mutation (a DNA mistake that happens after birth, not inherited) in a blood stem cell or lymphocyte disables the genes that normally stop cell division or trigger cell death (apoptosis). The cell becomes a clone — all its copies carry the same mutation — and it outcompetes normal marrow cells (causing leukemia) or piles up in lymph nodes and other organs (causing lymphoma). The type depends on which cell line breaks: lymphoid (B-cells, T-cells, or natural killer cells) or myeloid (the line that makes neutrophils, monocytes, red cells, or platelets). The speed depends on how immature the cells are: acute leukemias (mostly immature blasts, fast) or chronic leukemias and indolent lymphomas (more mature cells, slow). Each combination creates a different disease pattern and needs different treatment.

## How the body compensates

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The body tries to keep oxygen delivery, infection defense, and clotting going even as the marrow fails. The heart pumps faster to move what red cells are left. The spleen and liver may try to make extra blood cells (extramedullary hematopoiesis). Fever and inflammation ramp up to fight infections. For a while, vital signs look okay — but underneath, the reserve is vanishing. This is the dangerous middle: the patient seems stable, but one more infection, one more bleed, or one oncologic emergency (tumor lysis, leukostasis, SVC syndrome) tips them

over the edge. The job is to see the crash coming before it happens, because once it tips, the window to recover closes fast.

1. {'label': 'Cardiovascular compensation for anemia', 'plain': 'As red cells drop, the heart pumps faster (tachycardia) and harder (increased stroke volume) to deliver enough oxygen with fewer carriers. You might see a flow murmur (a soft whoosh from blood moving fast) and feel a bounding pulse. This works for a while, but the heart cannot keep that pace forever — eventually it tires (high-output heart failure).'}
2. {'label': 'Extramedullary hematopoiesis', 'plain': 'When the marrow fails, the spleen and liver try to help by making blood cells the way they did before you were born. You see an enlarged spleen (splenomegaly) (big spleen) and an enlarged liver (hepatomegaly) (big liver). This helps a little but never makes enough normal cells, and the big organs can rupture or press on other structures.'}
3. {'label': 'Immune escalation in dangerously low infection-fighting cells (neutropenia)', 'plain': 'With too few neutrophils, the body cranks up fever and inflammation to try to wall off infections. You see high fevers, but often no pus or obvious infection site (because neutrophils make pus). This is neutropenic fever — a medical emergency even when vitals look stable, because the patient has almost no defense and can crash into septic shock in hours.'}
4. {'label': 'Clotting attempts in a low platelet count (thrombocytopenia)', 'plain': 'The body tries to clot with fewer platelets by tightening blood vessels (vasoconstriction) and using what platelets are left more aggressively. You see pinpoint bleeding spots (petechiae) (tiny red dots under the skin from broken capillaries), purple bleeding blotches (purpura) (larger purple bruises), and mucosal bleeding (gums, nose, GI tract, vagina). Once platelets drop very low (below 10,000), the risk of spontaneous brain or gut bleeding jumps.'}
5. {'label': 'Vital signs stay stable — until they don't', 'plain': 'Blood pressure, heart rate, and oxygen saturation can look normal even while marrow failure worsens, because the compensations above are buying time. This is the dangerous middle, because it lets the team miss where the patient really is on the curve. The patient may feel tired, bruise easily, or have a low-grade fever, but still walk and talk — then suddenly tip into septic shock, bleeding crisis, or respiratory failure.'}
6. {'label': 'Watch the trajectory, not just the snapshot', 'plain': 'Look at whether counts are dropping (worse), flat (holding), or rising (better). Look at how fast symptoms are piling up. A patient with stable vitals but falling neutrophils and rising fevers is not really stable — they are on a downhill slope. Catch the crash point before it happens, because once sepsis, bleeding, or an oncologic emergency hits, the window to turn things around narrows fast.'}

## Domino effect

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1. Marrow crowding causes low counts of all blood cells (pancytopenia) — The broken cell clones multiply wildly and fill the bone marrow, stealing space and nutrients from normal stem cells. The marrow cannot make enough red cells (anemia), white cells (leukopenia, especially dangerously low infection-fighting cells (neutropenia)), or platelets (thrombocytopenia). This triad — low counts across the board — is called low counts of all blood cells (pancytopenia), and it explains the next three dominoes.
2. Anemia causes fatigue, pale skin, shortness of breath, and fast heartbeat — With too few red cells, your blood cannot carry enough oxygen to muscles and organs. Muscles tire quickly (fatigue, weakness). Skin and gums look pale. Your heart pumps faster (tachycardia) and you breathe faster (dyspnea on exertion) to try to deliver what oxygen you have. If anemia is severe, you may feel dizzy, get chest pain (angina), or even faint.
3. Dangerously low infection-fighting cells (Neutropenia) causes life-threatening infections — Neutrophils (a type of white cell) are your front-line defense against bacteria and fungi. With too few neutrophils (neutropenia, especially absolute neutrophil count below 500), even normal skin and gut bacteria can invade and cause sepsis (blood infection). You get high fevers but often no pus or obvious infection site. Neutropenic fever is a medical emergency — untreated, it can become septic shock and multi-organ failure in hours.
4. A low platelet count (Thrombocytopenia) causes bleeding into skin, gums, gut, and brain — Platelets plug holes in blood vessels. With too few platelets (thrombocytopenia, especially below 20,000), you bleed easily: pinpoint bleeding spots (petechiae) (tiny red dots under the skin), purple bleeding blotches (purpura) (larger purple bruises), nosebleeds, bleeding gums, heavy periods, blood in stool or urine. Below 10,000, spontaneous bleeding into the brain or gut becomes a real risk and can be fatal.
5. Lymph node masses cause pain, airway squeeze, and SVC syndrome — In lymphoma (or leukemia with big nodes), clumps of cancer cells pile up in lymph nodes in the neck, chest, belly, or groin. The swollen nodes press on nearby structures: throat (causing trouble swallowing), airway (causing cough, wheezing, a high-pitched noise on breathing in (stridor)), or the superior vena cava (the big vein bringing blood back to the heart), blocking blood return and causing face and neck swelling, bulging veins, and shortness of breath (SVC syndrome, an oncologic emergency).
6. Hyperleukocytosis makes blood thick and clogs tiny vessels — In some acute leukemias, the white cell count climbs above 100,000 (hyperleukocytosis). The blood becomes thick and sludgy, like honey instead of water. It jams up in the tiniest vessels in the lungs (causing shortness of breath, low oxygen) and brain (causing confusion, vision loss, stroke). This is called leukostasis, another oncologic emergency — leukapheresis (filtering the blood) or urgent chemotherapy is needed to bring the count down fast.

7. Tumor lysis syndrome poisons the kidneys and heart — When chemotherapy kills billions of cancer cells at once (especially in fast-growing leukemias and lymphomas like ALL, Burkitt lymphoma, high-grade NHL), the cells burst open and dump their insides into the blood: potassium (causing dangerous heart rhythms), phosphate (which binds calcium, dropping it dangerously low and causing muscle spasms, seizures), and uric acid (which crystallizes in the kidneys, causing acute kidney injury). This cascade is called tumor lysis syndrome. It can kill within hours if not caught early. Prevention includes IV fluids, allopurinol or rasburicase (to lower uric acid), and close monitoring of electrolytes and kidney function.
8. None of this is random — every symptom flows backward from the one broken cell — Tired, pale, infected, bruised, swollen nodes, thick blood, electrolyte chaos — every single finding traces back to that first mutation that broke the growth controls. The body is not randomly falling apart; it is trying to compensate (pump faster, fight harder, clot with less) while the cancer spreads. When you see a weird lab or symptom, ask yourself: 'Which domino is this?' and trace it back upstream. That is how you learn to recognize the pattern early, before the patient crashes.

## Symptoms · what the patient feels, and why

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- Swollen lymph nodes that don't hurt: Cancer cells crowd into the lymph nodes (the body's filter stations) and make them puff up, but because the swelling happens slowly without infection, there's no pain signal.
- Fever that comes and goes without any infection: Cancer cells release chemicals called cytokines (messenger proteins) that reset the brain's temperature thermostat higher, triggering fever even though there's no germ causing it.
- Soaking night sweats that drench your clothes and sheets: The same cytokines that cause fever also tell the hypothalamus (the brain's temperature control center) to dump heat by sweating heavily, often at night when the body is at rest.
- Losing more than 10 percent of your body weight without trying: Cancer cells burn a huge amount of energy and also release chemicals that kill your appetite and speed up how fast your body breaks down fat and muscle for fuel.
- Feeling exhausted all the time: Cancer crowds out the bone marrow (the spongy inside of bones where blood is made), so you make fewer red blood cells to carry oxygen, and the cancer itself steals nutrients and energy your body needs.
- Easy bruising or tiny red or purple dots on the skin: Cancer takes over space in the bone marrow so it can't make enough platelets (the tiny cells that plug holes in blood vessels), which means even small bumps cause bruises and capillaries (the tiniest blood vessels) leak under the skin.

- Getting infections over and over: The bone marrow fills with cancer cells instead of making healthy white blood cells (the body's infection fighters), so germs can take hold more easily and harder to clear.
- Belly pain or feeling full fast when you eat: The spleen or liver (organs that filter blood and sit in your belly) can swell up with cancer cells, pressing on your stomach and nearby organs so there's less room for food.
- Bone pain, especially in the back, ribs, or legs: In leukemia, cancer cells pack the bone marrow so tightly they stretch the bone's outer cover (the periosteum, which has pain nerves), and in some lymphomas cancer can invade bone directly.
- Shortness of breath or cough: Lymph nodes in the chest can swell and press on the windpipe or lungs, or cancer cells can build up in the blood and clog the tiny vessels in the lungs, making it hard to move air in and out.

## Focused exam

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- Pale skin and nail beds: Anemia happens because leukemia cells crowd out the normal red-cell-making spots in the bone marrow, so your body cannot build enough red cells to carry oxygen.
- Fever without a clear infection site: Dangerously low infection-fighting cells (Neutropenia) (too few white cells that fight germs) happens because leukemia or lymphoma cells take over the marrow and push out the normal germ-fighting white cells, so your body cannot defend against infections.
- Bruising or tiny red dots (petechiae) on skin: A low platelet count (Thrombocytopenia) (too few platelets) happens because cancer cells crowd out platelet-making spots in the marrow, so blood cannot clot the way it should and you bleed or bruise easily.
- Swollen lymph nodes in neck, armpit, or groin: In lymphoma, the broken cell multiplies inside lymph nodes instead of bone marrow, making lumps you can feel and measure under the skin to track how big the tumor is.
- Shortness of breath that got worse in the last few hours: Hyperleukocytosis (too many leukemia cells make the blood thick like honey) or a chest tumor can block oxygen from reaching your lungs or brain — this is a medical emergency.
- Belly pain with a puffy belly: Leukemia or lymphoma cells can pile up in the spleen or liver and make them swell, or dying cancer cells can dump waste that poisons the kidneys — a careful belly exam and ultrasound can find this.
- Confused or acting sleepy: Hyperleukocytosis can plug tiny blood vessels in the brain like sand clogging a pipe, or dying cancer cells can dump poison (like too much potassium or uric acid) that harms brain cells — this is a medical emergency.

## Diagnostics · every test answers a question

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- Complete blood count (CBC) with differential → Are red cells, white cells, and platelets all normal, or is the bone marrow failing?
- Peripheral blood smear → Are there abnormal young white cells (blasts) in the blood that should only be in the marrow?
- Bone marrow biopsy with flow cytometry → What exact type of blood cancer is this, and which markers are on the broken cells?
- Cytogenetics and molecular testing (FISH, PCR for gene mutations) → Which genetic breaks or mutations are driving this cancer, and do they predict how it will respond to treatment?
- Chest X-ray or CT chest → Is there a tumor mass in the chest pressing on the windpipe, heart, or big blood vessels?
- Metabolic panel (potassium, phosphate, uric acid, creatinine, calcium, LDH) → Are dying cancer cells dumping waste into the blood (tumor lysis syndrome), and are the kidneys keeping up?
- PET-CT scan (for lymphoma staging) → Where has the lymphoma spread in the body, and how active is it?
- Lumbar puncture (spinal tap) with cerebrospinal fluid analysis → Has leukemia or lymphoma spread into the fluid around the brain and spinal cord?

## Treatment logic

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- IV fluids (150–200 mL/kg/day), allopurinol (or rasburicase in high-risk patients), and continuous heart monitoring → Prevents tumor lysis syndrome by flushing out potassium, phosphate, and uric acid that dying cancer cells dump into the blood when chemotherapy starts. Keeps kidneys working and prevents dangerous heart rhythms (arrhythmias from high potassium).
- Leukapheresis (machine that spins extra white cells out of the blood) if symptomatic hyperleukocytosis is present → Thins the blood so it flows through tiny lung and brain blood vessels again, stopping the blockage that causes shortness of breath, confusion, or stroke.

- Disease-specific chemotherapy regimen matched to cancer type and genetic markers: APL gets all-trans retinoic acid (ATRA) and arsenic trioxide; AML gets cytarabine + anthracycline (7+3 regimen); ALL gets multi-agent induction (vincristine, daunorubicin, asparaginase, steroid); diffuse large B-cell lymphoma (DLBCL) gets rituximab, cyclophosphamide, doxorubicin, vincristine, prednisone (R-CHOP); Hodgkin lymphoma gets doxorubicin, bleomycin, vinblastine, dacarbazine (ABVD); chronic lymphocytic leukemia (CLL) gets BTK inhibitors (ibrutinib) or BCL-2 inhibitors (venetoclax) → Kills the one broken cell that lost growth control, stopping the wild multiplication and letting normal bone marrow cells grow back. Each regimen is matched to the surface markers and gene mutations found in the biopsy.
- Blood product transfusions (packed red cells for anemia, platelets for bleeding) and growth factors (G-CSF for severe dangerously low infection-fighting cells (neutropenia)) → Props up red cells so oxygen reaches your body and you stop feeling tired and dizzy. Raises platelet counts so bleeding stops. Boosts white cells (neutrophils) so infection risk drops while chemotherapy is killing marrow cells.
- Infection prevention (prophylaxis): antibacterial (levofloxacin or cefepime if feverish (febrile) dangerously low infection-fighting cells (neutropenia)), antiviral (acyclovir for herpes simplex virus), antifungal (fluconazole or posaconazole), and Pneumocystis jirovecii pneumonia (PCP) prevention (trimethoprim-sulfamethoxazole) → Prevents bacteria, viruses, and fungi from invading and causing life-threatening infections while neutrophil counts are dangerously low ( $< 500/\mu\text{L}$ ) from both the cancer and chemotherapy.
- Intrathecal chemotherapy (methotrexate or cytarabine injected into spinal fluid) for CNS prevention (prophylaxis) or treatment → Delivers chemotherapy directly into the fluid around the brain and spinal cord, killing leukemia or lymphoma cells hiding there where IV chemotherapy cannot reach well.
- Hematopoietic stem cell transplant (bone marrow transplant) in selected patients with high-risk disease, relapsed disease, or in first remission for certain types (e.g., AML with poor-risk genetics) → Replaces the entire broken bone marrow with healthy donor stem cells (allogeneic transplant) or the patient's own stored cells (autologous transplant), giving a fresh start and a much lower chance of the cancer coming back. The donor immune cells can also attack leftover cancer cells (graft-versus-leukemia effect).

## Medications

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### Allopurinol · Xanthine oxidase inhibitor

When cancer cells die quickly during treatment, they spill their insides into the blood — especially a chemical called uric acid. Too much uric acid can form sharp crystals in the

kidneys and shut them down. Allopurinol blocks the enzyme (xanthine oxidase) that makes uric acid, so levels stay safer.

1. Cancer treatment kills many tumor cells all at once (tumor lysis).
2. Dead cells release purines, which the liver normally turns into uric acid.
3. Allopurinol blocks xanthine oxidase, the enzyme that does this job.
4. Less uric acid is made, so kidneys are safer.

#### **SAFETY WATCH**

Rare but serious skin rash (Stevens-Johnson syndrome) — stop the drug and call the doctor if any new rash appears. · Can rarely cause liver enzyme changes — blood tests are checked. · Needs dose adjustment if kidneys are already weak.

### **Rasburicase · Recombinant urate oxidase enzyme**

Rasburicase is a lab-made copy of an enzyme (urate oxidase) that breaks down uric acid into a harmless compound that the kidneys can easily clear. Humans do not naturally make this enzyme, so giving it can rapidly lower dangerous uric acid levels during tumor lysis.

1. Cancer treatment causes massive tumor cell death.
2. Cells release purines, which turn into uric acid.
3. Rasburicase converts uric acid into allantoin, which dissolves easily and leaves through urine.
4. Uric acid levels drop quickly, protecting the kidneys.

#### **SAFETY WATCH**

NEVER give to anyone with G6PD deficiency — can cause severe anemia and red blood cell destruction (hemolysis) (red blood cells burst). · Can cause allergic reactions (rash, trouble breathing) — watch closely during the first dose. · Blood samples for uric acid must be kept on ice or the drug will keep working in the tube and give a falsely low reading.

### **Hydroxyurea · Ribonucleotide reductase inhibitor**

When white blood cell counts skyrocket (hyperleukocytosis), blood gets thick like syrup and can clog small vessels in the lungs and brain. Hydroxyurea blocks an enzyme (ribonucleotide reductase) that cancer cells need to copy their DNA, so they cannot multiply. This quickly lowers the cell count and thins the blood.

1. Leukemia cells multiply out of control, flooding the blood.
2. Hydroxyurea blocks ribonucleotide reductase, the enzyme that makes DNA building blocks.
3. Cancer cells cannot finish copying their DNA, so they stop dividing and die.

4. White blood cell count drops, blood flows normally again.

**SAFETY WATCH**

Lowers all blood counts (white cells, red cells, platelets) — regular blood tests are needed.  
· Can cause mouth sores and skin changes. · Rarely causes leg ulcers with long-term use. · Must avoid pregnancy — can harm a developing baby.

**All-trans retinoic acid (ATRA) · Differentiation agent (vitamin A derivative)**

In acute promyelocytic leukemia (APL), immature blood cells are stuck at an early stage and cannot grow up into working white cells. ATRA is a form of vitamin A that nudges these stuck cells to mature (differentiate) into normal-functioning cells that then naturally die off, clearing the leukemia without the usual heavy chemotherapy toxicity.

1. APL cells have a broken gene fusion (PML-RARA) that locks them in baby form.
2. ATRA binds to the RAR-alpha receptor inside the cell nucleus.
3. This unlocks the growth block, and cells mature into normal neutrophils.
4. Mature cells live out their natural short lifespan and die, so the leukemia clears.

**SAFETY WATCH**

Differentiation syndrome (formerly called retinoic acid syndrome) — fever, fluid in the lungs, trouble breathing, weight gain. Treated with steroids; can be life-threatening if missed. · Can raise liver enzymes and triglycerides — blood tests are monitored. · Severe birth defects — absolutely cannot be used during pregnancy. · Headache and dry skin are common but manageable.

**Arsenic trioxide · Differentiation and apoptosis inducer**

Arsenic trioxide works two ways in APL: it helps stuck leukemia cells mature (like ATRA does), and it also triggers the cancer cells to self-destruct (apoptosis). Together with ATRA, it has turned APL from one of the deadliest leukemias into one of the most curable, often without traditional chemotherapy.

1. Arsenic binds to the abnormal PML-RARA protein in APL cells.
2. This breaks apart the protein, releasing the growth block.
3. Cells mature into normal neutrophils.
4. Arsenic also damages mitochondria in cancer cells, triggering them to die.
5. The leukemia clears.

**SAFETY WATCH**

Can lengthen the heart's electrical cycle (QT prolongation), which may cause dangerous heart rhythms — ECG monitoring is required. · Can cause differentiation syndrome (same as ATRA). · Can raise liver enzymes. · Low potassium and magnesium must be corrected before and during treatment. · Long-term use (maintenance) can rarely cause nerve damage or skin changes.

**Cytarabine (Ara-C) plus Daunorubicin (7+3 induction) · Antimetabolite (cytarabine) plus anthracycline (daunorubicin)**

This is the backbone of treatment for most acute myeloid leukemia (AML, not APL). Cytarabine is a fake DNA building block — when cancer cells try to use it to copy their DNA, the process jams and the cell dies. Daunorubicin slips between DNA strands and breaks them, also killing fast-dividing cells. Together (7 days of cytarabine, 3 days of daunorubicin), they wipe out the leukemia in the bone marrow so healthy blood cells can regrow.

1. Leukemia cells divide rapidly in the bone marrow.
2. Cytarabine enters cells and is mistaken for a normal DNA piece (cytidine).
3. When the cell tries to copy DNA, cytarabine jams the machinery (DNA polymerase), and the cell dies.
4. Daunorubicin slides into DNA strands and creates breaks (via topoisomerase II inhibition).
5. Cancer cells cannot repair the damage and die.
6. Normal stem cells survive better because they divide more slowly and can repair some damage.

**SAFETY WATCH**

Severe bone marrow suppression — all blood counts drop for weeks, high infection and bleeding risk. · Nausea, vomiting, diarrhea, mouth sores. · Daunorubicin can damage the heart muscle (cardiomyopathy) — lifetime dose is limited. · Cytarabine at high doses can cause brain and eye toxicity (cerebellar syndrome, conjunctivitis). · Hair loss is common. · Red urine from daunorubicin is harmless (the drug is red) but can be alarming.

**Vincristine, Prednisone, Asparaginase, Anthracycline (multi-agent ALL regimen) · Vinca alkaloid (vincristine), corticosteroid (prednisone), enzyme (asparaginase), anthracycline (daunorubicin or doxorubicin)**

Acute lymphoblastic leukemia (ALL) requires hitting cancer cells from several angles at once. Vincristine stops cells from dividing by freezing their internal scaffolding (microtubules). Prednisone (a steroid) directly kills lymphoid cells by triggering apoptosis (cell suicide). Asparaginase starves leukemia cells by destroying asparagine, an amino acid they cannot make

themselves but need to survive. An anthracycline breaks DNA strands. Together, these drugs overwhelm the cancer's defenses.

1. ALL cells divide rapidly and depend on external asparagine to grow.
2. Vincristine binds to tubulin and stops microtubules from forming, so cells cannot divide (freeze in metaphase).
3. Prednisone enters lymphoid cells and activates genes that trigger apoptosis.
4. Asparaginase (from bacteria) breaks down asparagine in the blood; ALL cells starve because they cannot make their own.
5. Anthracycline damages DNA directly.
6. Cancer cells die from multiple attacks at once.

#### SAFETY WATCH

Vincristine: nerve damage (peripheral neuropathy) — tingling, pain, constipation, rarely foot drop. · Prednisone: high blood sugar, mood swings, increased appetite, bone thinning with long use, higher infection risk. · Asparaginase: allergic reactions (sometimes severe), pancreatitis (inflamed pancreas causing severe belly pain), blood clotting problems (both clots and bleeding), liver inflammation. · Anthracycline: heart damage (dose-limited). · Bone marrow suppression common to all — infection and bleeding risk. · Hair loss with anthracyclines.

### **Rituximab plus CHOP (R-CHOP for DLBCL) · Monoclonal antibody (rituximab) plus cyclophosphamide, doxorubicin, vincristine, prednisone**

Diffuse large B-cell lymphoma (DLBCL) is a fast-growing lymphoma of mature B cells. Rituximab is a lab-made antibody that locks onto CD20, a protein on the surface of B cells (including lymphoma cells), and tags them for destruction by the immune system. CHOP is a four-drug chemotherapy cocktail that kills fast-dividing cells. Together, R-CHOP is the standard cure-intent treatment.

1. Lymphoma cells express CD20 protein on their surface.
2. Rituximab binds to CD20 and marks the cell for attack.
3. Immune cells (natural killer cells, macrophages) recognize the tag and destroy the lymphoma cell.
4. Rituximab also directly triggers some cells to die (apoptosis).
5. CHOP drugs (cyclophosphamide damages DNA, doxorubicin breaks DNA, vincristine stops cell division, prednisone kills lymphoid cells) attack from other angles.
6. Lymphoma shrinks or disappears.

**SAFETY WATCH**

Rituximab: infusion reactions (fever, chills, low blood pressure, trouble breathing) are common with the first dose — pre-medications and slow infusion help. · Rare reactivation of hepatitis B in carriers — screen before treatment. · Rare progressive multifocal leukoencephalopathy (PML), a brain infection, in severely immunosuppressed patients. · CHOP: heart damage from doxorubicin, nerve damage from vincristine, bone marrow suppression, nausea, hair loss, risk of second cancers years later. · Prednisone side effects (high blood sugar, mood changes, infection risk).

**ABVD (Doxorubicin, Bleomycin, Vinblastine, Dacarbazine for Hodgkin lymphoma) · Anthracycline, glycopeptide antibiotic, vinca alkaloid, alkylating agent**

Hodgkin lymphoma has Reed-Sternberg cells (the cancer) surrounded by a lot of normal immune cells. ABVD is a four-drug mix that attacks cancer cells in different ways: doxorubicin breaks DNA, bleomycin cuts DNA strands with oxygen radicals, vinblastine stops cell division, and dacarbazine adds DNA damage. Together, they cure most Hodgkin lymphoma cases.

1. Reed-Sternberg cells and their variants divide in lymph nodes.
2. Doxorubicin (an anthracycline) intercalates into DNA and creates breaks via topoisomerase II.
3. Bleomycin generates oxygen free radicals that slice DNA strands.
4. Vinblastine binds tubulin and stops mitotic spindle formation, freezing cell division.
5. Dacarbazine adds alkyl groups to DNA, jamming replication.
6. Cancer cells die; normal tissue (which divides slower) has more time to repair.

**SAFETY WATCH**

Bleomycin: lung toxicity (pulmonary fibrosis) — monitor with breathing tests; risk increases with cumulative dose, older age, smoking, or supplemental oxygen. · Doxorubicin: heart damage (cardiomyopathy) — dose-limited; heart function monitored. · Vinblastine: nerve damage (less severe than vincristine but still possible) — constipation, tingling. · Dacarbazine: nausea and vomiting (often severe), flu-like symptoms. · Bone marrow suppression (less severe than with some other regimens but still present). · Hair loss. · Infertility risk — discuss fertility preservation before starting.

**Ibrutinib (BTK inhibitor for CLL) · Bruton tyrosine kinase (BTK) inhibitor**

Chronic lymphocytic leukemia (CLL) cells survive by getting constant grow signals through a pathway involving BTK, a protein inside the cell. Ibrutinib blocks BTK, cutting off survival

signals. Without these signals, CLL cells die off naturally. It is taken as a daily pill and has largely replaced chemotherapy for many CLL patients.

1. CLL cells receive survival signals through the B-cell receptor (BCR) on their surface.
2. The BCR signal travels through a chain of proteins inside the cell, including BTK.
3. Ibrutinib irreversibly binds to BTK, blocking the signal.
4. Without the signal, CLL cells stop dividing and undergo apoptosis (natural cell death).
5. CLL burden drops over time.

#### **SAFETY WATCH**

Atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat) and other heart rhythm problems — more common in older patients or those with heart disease. · Bleeding risk (easy bruising, nosebleeds, or more serious bleeding) because BTK is also in platelets — avoid other blood thinners if possible, stop before surgery. · Diarrhea, muscle aches, rash. · Rare serious infections (including fungal and viral reactivations). · High blood pressure. · Second cancers (skin cancers especially) — regular skin checks recommended.

### **Venetoclax (BCL-2 inhibitor for CLL and AML) · B-cell lymphoma-2 (BCL-2) inhibitor**

Cancer cells, including CLL and some AML cells, survive by overproducing BCL-2, a protein that blocks apoptosis (the cell's self-destruct program). Venetoclax binds to BCL-2 and shuts it down, re-activating apoptosis. The cancer cells then die naturally. It is often combined with other drugs for faster, deeper responses.

1. CLL or AML cells overproduce BCL-2, which sits on the mitochondria and blocks death signals.
2. Venetoclax binds to BCL-2, kicking out the proteins that trigger apoptosis (BAX and BAK).
3. BAX and BAK punch holes in the mitochondria, releasing signals that activate caspases (enzymes that dismantle the cell).
4. The cancer cell undergoes apoptosis and dies.
5. Disease burden drops.

**SAFETY WATCH**

Tumor lysis syndrome (especially in CLL with high disease burden) — start at a very low dose and ramp up slowly over weeks, with close lab monitoring and hydration. · Low white blood cells and infections (especially in AML) — preventive (prophylactic) antibiotics often given. · Diarrhea, nausea, fatigue. · Low platelet count (thrombocytopenia) — bleeding risk. · Drug interactions — venetoclax levels rise dangerously with certain antifungals (posaconazole, voriconazole) and other CYP3A inhibitors.

**Filgrastim (G-CSF) · Granulocyte colony-stimulating factor**

Chemotherapy kills fast-dividing cells, including the bone marrow cells that make infection-fighting white blood cells (neutrophils). When neutrophil counts drop dangerously low (neutropenia), infection risk skyrockets. Filgrastim is a lab-made copy of G-CSF, a natural signal that tells the bone marrow to crank out more neutrophils faster, shortening the vulnerable window.

1. Chemotherapy wipes out neutrophil precursors in the marrow.
2. Filgrastim binds to G-CSF receptors on surviving stem cells and precursors.
3. This signal tells them to divide faster and mature into neutrophils more quickly.
4. Neutrophil counts rise sooner, reducing infection risk.
5. The patient can often receive the next chemotherapy cycle on time.

**SAFETY WATCH**

Bone pain (especially in the lower back, hips, and sternum) — common but manageable with acetaminophen or ibuprofen. · Rare spleen rupture (if spleen is enlarged, abdominal pain is a warning). · Rarely, can worsen sickle cell disease or cause capillary leak syndrome. · Very rare risk of pushing myeloid leukemia cells to grow (usually not a concern in leukemia treatment because the goal is to kill those cells anyway).

**Leukapheresis · Not a medication — a procedure (mechanical blood filtering)**

When white blood cell counts go dangerously high (hyperleukocytosis, usually  $>100,000/\mu\text{L}$ ), blood becomes thick and can clog small blood vessels in the lungs and brain, causing strokes or respiratory failure. Leukapheresis is an emergency procedure where blood is pulled out of one vein, run through a machine that separates and removes excess white cells, and returned through another vein. It buys time while chemotherapy starts working.

1. Blood is drawn continuously through a large IV or central line.
2. The machine spins the blood to separate cells by weight.
3. Excess white blood cells are skimmed off and discarded.

4. The remaining blood (red cells, platelets, plasma) is returned to the patient.
5. White count drops immediately, blood flows better.
6. Chemotherapy or hydroxyurea is started to keep counts down long-term.

**SAFETY WATCH**

Requires large-bore IV access or central line — risk of bleeding or infection at the site. · Can cause low calcium (citrate used as anticoagulant binds calcium) — tingling around the mouth, muscle cramps; treated with calcium supplements during the procedure. · Blood pressure can drop during the procedure. · Rare risk of bleeding (if platelets are very low) or clotting. · Not all centers have leukapheresis capability — may need transfer.

## Red flags

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- Fever, drenching night sweats, and losing more than 10 percent of your body weight in six months (called B symptoms — they mean the cancer is more aggressive)
- Trouble breathing, chest pain, or swelling in the face and neck (the cancer may be pressing on the big veins or windpipe in the chest)
- Sudden severe confusion, headache, or weakness on one side of the body (cancer may have spread to the brain or spinal cord)
- Bleeding that won't stop or bruises appearing all over without any injury (platelet count is dangerously low)
- Severe belly pain with a hard, swollen abdomen (the spleen or liver may be hugely enlarged or rupturing)

## Nursing priorities

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1. Check oxygen saturation and listen to lung sounds every 2 to 4 hours and watch for new shortness of breath or a high-pitched noise on breathing in (stridor) (a high-pitched breathing sound) — A big tumor mass in the chest can press on the airway or the white cell count can go so high that cells get stuck in the tiny lung blood vessels causing breathing failure
2. Monitor continuous telemetry (heart rhythm) and check potassium, calcium, phosphorus, and uric acid levels every 6 to 12 hours when chemotherapy starts — When cancer cells die fast they spill potassium and phosphorus into the blood causing deadly heart rhythms and the breakdown makes uric acid crystals that shut down the kidneys in tumor lysis syndrome

3. Give IV fluids at 150 to 200 milliliters per hour to keep urine output above 100 milliliters per hour and administer allopurinol or rasburicase as ordered before chemotherapy — Fast fluids wash uric acid and other waste out through the kidneys before crystals form and allopurinol or rasburicase (medicines that lower uric acid) prevent kidney failure from tumor lysis
4. Check platelet count daily and watch for new bruising, blood in urine or stool, or bleeding from gums and IV sites — Leukemia crowds out the bone marrow so platelets drop below 10,000 causing brain bleeds and other dangerous bleeding and some leukemias trigger DIC which uses up clotting factors
5. Take temperature every 4 hours and call the provider immediately if temperature goes above 100.4 degrees Fahrenheit even once — Low white blood cell counts from disease or chemotherapy mean the body cannot fight bacteria so fever is an emergency needing blood cultures and antibiotics within one hour to prevent septic shock
6. Use strict hand hygiene, avoid fresh flowers and raw foods in the room, and limit visitors to healthy people only — The immune system is broken so normal germs from hands or food or sick visitors can cause life-threatening infections that healthy people would fight off easily
7. Transfuse packed red blood cells when hemoglobin drops below 7 to 8 grams per deciliter or the patient has chest pain, dizziness, or severe fatigue — Leukemia stops red blood cell production so oxygen delivery to the heart and brain drops causing heart attacks or falls and transfusions keep organs alive until chemotherapy works
8. Check mental status every shift and ask about numbness, tingling, or new weakness in arms or legs — Leukemia cells can invade the brain and spinal fluid causing seizures, strokes, or paralysis that need emergency treatment with chemotherapy injected into the spine

## Sources

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- NCCN Clinical Practice Guidelines in Oncology: Acute Lymphoblastic Leukemia 2024
- NCCN Clinical Practice Guidelines in Oncology: Non-Hodgkin Lymphomas 2024
- American Society of Hematology
- National Cancer Institute

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