Testicular Cancer

A painless lump in a young man's testicle needs an ultrasound of the scrotum right away.

THE STORY IN ONE SENTENCE

A painless lump in a young man's testicle needs an ultrasound of the scrotum right away.

“Testicular cancer shows as a painless, hard lump; ultrasound confirms it, surgery removes the testicle, blood tumor markers guide treatment, and staging scans tell us if it spread—then we give chemo or radiation to kill any leftover cancer cells.”

Normal anatomy — the body's job

The testicles are two oval glands that hang in the scrotum (the skin sack below the penis). Each testicle has tiny coiled tubes inside called seminiferous tubules where sperm are made, and clusters of Leydig cells between the tubes that pump out testosterone into the blood. Keep this picture in your head: a testicle is a sperm factory with a built-in hormone pump. When you understand that normal job, the weird findings from cancer make sense as a break from it.

BODY'S JOB

The testicles do two main jobs. First, they make millions of sperm every day so a man can father children. Second, Leydig cells release testosterone, which travels through the blood to build muscle, deepen the voice, grow body hair, make bones strong, and keep sex drive going. The testicles hang outside the body because sperm need to stay a few degrees cooler than body temperature to survive—that is why the scrotum pulls up when it is cold and hangs loose when it is warm, adjusting the temperature like a thermostat.

1. Germ cells inside the seminiferous tubules divide and mature into sperm over about 74 days.
2. Leydig cells between the tubules release testosterone into tiny blood vessels that carry it to the rest of the body.
3. Sperm travel from the tubules into the epididymis (a coiled storage tube on the back of each testicle) where they finish maturing.

4. During ejaculation, sperm shoot from the epididymis up the vas deferens tube, mix with fluid from the prostate and seminal vesicles, and leave the body as semen.
5. Testosterone in the blood signals bones, muscles, the brain, and other organs to do their male-pattern jobs.

What breaks

Testicular cancer starts when germ cells (the cells that normally become sperm) or other cells in the testicle get DNA damage and start copying themselves out of control. Instead of maturing into sperm and stopping, the broken cells keep dividing and pile up into a solid lump. Most testicular cancers are germ cell tumors—either seminomas (slow-growing, very sensitive to radiation) or non-seminomas (faster-growing, often make hormones like AFP or hCG, and need chemo). The cancer usually stays in one testicle at first, but if left alone, rogue cells can break off and travel through lymph channels to lymph nodes in the back of the belly, then to the lungs and other organs.

> **CLINICAL TERM**
>
> Testicular cancer is most often a germ cell tumor arising from seminiferous tubules, driven by DNA mutations and often linked to cryptorchidism (undescended testicle), family history, contralateral tumor, and age 15–35. The tumor grows as a firm, painless mass, and may produce tumor markers (AFP, hCG, LDH) that guide diagnosis and treatment. If untreated, malignant cells spread via lymphatics to retroperitoneal nodes, then hematogenously to lungs, liver, brain, and bone.

How the body compensates

The body cannot kill testicular cancer on its own, so it tries to wall off the tumor with scar tissue and keep the other testicle working. Vital signs stay normal because the tumor does not bleed fast or dump toxins—so the man can feel fine even with advanced disease. The danger is missing the cancer until it has spread, because early-stage testicular cancer is almost 100% curable, but late-stage needs much harsher chemo and has a higher chance of coming back.

1. The immune system tries to attack the tumor, sending white blood cells to the area, but cancer cells hide from immune detection or grow faster than the immune system can kill them.
2. The body walls off the tumor with fibrous tissue to keep it from invading nearby structures, but this does not stop the cancer—it just slows it down.
3. The other testicle keeps pumping out testosterone and making sperm, so hormone levels and fertility stay normal if only one testicle is affected.

4. If the tumor makes hCG, the body responds as if the man is pregnant, growing breast tissue and sometimes causing nausea or fatigue—these are clues, not separate diseases.
5. Vital signs (heart rate, blood pressure, oxygen) stay stable because the tumor does not bleed into the belly, dump infection into the blood, or shut down organs—so labs and vital signs can look reassuring even when cancer is spreading.
6. The crash point to watch for is: tumor markers (AFP, hCG, LDH) climbing on repeat blood tests, new lung nodules on CT scan, back pain from swollen retroperitoneal lymph nodes pressing on nerves, shortness of breath from lung metastases, or the tumor breaking through the scrotum skin (very late). By the time you see these, the window for easy cure is closing, so we hunt for the lump early with ultrasound and self-exams.

Domino effect

1. A germ cell in the testicle picks up DNA damage and starts dividing out of control. — Risk factors—undescended testicle (cryptorchidism) exposing cells to higher body temperature, family history passing down DNA mutations, or prior cancer in the other testicle—make it more likely that a germ cell will break its stop signals and become a tumor. Age 15–35 is peak time because germ cells are dividing fast during and after puberty, giving more chances for copying errors.
2. The rogue cells pile into a hard, painless lump inside the testicle. — Cancer cells stick together and grow as a solid mass. Because the tumor grows slowly and does not stretch the scrotum's outer layers fast, it usually does not hurt—pain comes from infections or twists that happen suddenly, not from cancer creeping up over weeks.
3. The man (or his doctor) feels the lump during a testicular self-exam or physical. — The testicle normally feels smooth and squishy, so a hard spot or a lump that does not match the other side stands out. This is the first domino that gets noticed—the painless lump is the alarm bell.
4. Some men notice a dull ache or heavy, pulling-down feeling in the scrotum or lower belly. — The growing tumor adds weight and pulls on the spermatic cord (the bundle of blood vessels, nerves, and the vas deferens tube that connects the testicle to the body). The ache is not sharp pain—it is more like wearing a heavy weight in your underwear.
5. If the tumor makes hCG, the man's chest tissue swells and gets tender (gynecomastia). — Non-seminoma tumors, especially choriocarcinoma type, pump out hCG (the hormone pregnant women make). hCG travels in the blood and tells breast tissue to grow, just like it does during pregnancy. The man notices his nipples hurt or his chest looks puffy—this is a red flag that the tumor is making hormones.

6. Ultrasound of the scrotum shows a solid mass inside the testicle, and blood tests check tumor markers (AFP, hCG, LDH). — Ultrasound uses sound waves to see inside the testicle—cancer shows up as a dark or mixed-texture lump that is different from the normal tissue. Blood tests measure AFP (made by yolk sac tumors), hCG (made by choriocarcinoma), and LDH (a general marker of cell turnover that goes up when cancer is growing fast). These numbers help confirm cancer and tell us what type.
7. Surgery removes the whole testicle through a cut in the groin (radical inguinal orchiectomy). — We take out the entire testicle (not just the lump) because cancer cells can hide anywhere in it, and cutting into the scrotum might spill cancer cells into local tissues. Going through the groin lets us clamp the spermatic cord first, stopping blood flow and any cancer cells from escaping during surgery. The testicle goes to pathology, where a specialist slices it thin and looks under a microscope to name the exact cancer type and see if it broke through the outer shell.
8. CT scans of the chest, belly, and pelvis check if cancer spread to lymph nodes or lungs. — Testicular cancer loves to spread first to retroperitoneal lymph nodes (bean-shaped filters along the back wall of the belly), then to the lungs. The CT scan is a detailed X-ray that shows if nodes are swollen or if there are spots in the lungs. This staging tells us if the cancer is still local (Stage I), in nearby nodes (Stage II), or in distant organs (Stage III)—and that picks the treatment plan.
9. If cancer spread or tumor markers stay high after surgery, chemo (like BEP: bleomycin, etoposide, cisplatin) kills leftover cancer cells throughout the body. — Chemo drugs travel in the blood and kill any cancer cells that escaped the testicle. BEP is the standard recipe: bleomycin damages cancer DNA, etoposide stops cells from dividing, and cisplatin crosslinks DNA so cancer cells die. Testicular cancer is exquisitely sensitive to chemo—even advanced disease has a high cure rate. We also recheck tumor markers every few weeks; if they drop to zero and stay there, we know the cancer is gone.
10. If the tumor is pure seminoma and only in nearby lymph nodes, radiation therapy to the belly can kill it instead of chemo. — Seminomas are very sensitive to radiation. A focused beam aimed at the retroperitoneal lymph nodes kills cancer cells there while sparing the rest of the body from chemo side effects. Non-seminomas are not as radiation-sensitive, so they need chemo.
11. Follow-up appointments track tumor markers and scans for years to catch any return early. — Testicular cancer can come back (recur) in the first two years after treatment, so we check blood markers and do scans every few months at first, then space them out. If markers climb or a new lump shows up, we restart treatment right away. The earlier we catch a recurrence, the better the cure rate stays.

Symptoms · what the patient feels, and why

- A hard lump or swelling in one testicle: Cancer cells multiply inside the testicle and form a solid mass that pushes the normal tissue out of the way, making the testicle feel bigger or heavier on one side.
- A heavy or dragging feeling in the scrotum: The growing tumor adds weight to the testicle, pulling down on the sac (scrotum) like carrying a small stone in your pocket.
- Dull ache in the lower belly or groin: The tumor stretches the capsule (outer shell) around the testicle and tugs on the spermatic cord (the bundle of tubes and blood vessels feeding the testicle), sending dull pain signals to nearby nerves.
- The lump does not shine light through (does not transilluminate): Cancer tissue is solid and blocks light completely, unlike a fluid-filled cyst (hydrocele) that glows when you shine a flashlight through the scrotum.
- Breast tenderness or growth (gynecomastia): Some testicular tumors release human chorionic gonadotropin (hCG, a pregnancy hormone) or make extra estrogen (a female hormone), which causes breast tissue to swell and become tender.
- Back pain that will not go away: Cancer that has spread to lymph nodes in the back of the belly (retroperitoneal nodes) presses on nerves along the spine, causing steady aching pain.
- Cough or shortness of breath: Testicular cancer can spread through the blood to the lungs, where tumor nodules take up space and trigger coughing or make it harder to breathe.
- Sudden sharp pain in the testicle: Rapid bleeding into the tumor or twisting of the spermatic cord (torsion) caused by the heavy mass can cut off blood flow and cause sudden, severe pain.

Focused exam

- Hard, painless lump in the testicle: This is the red flag for testicular cancer. A hard bump that does not go away tells you the testicle tissue changed — cancer grows fast and hard, not soft like fluid. Most testicular cancers show up this way in men age 15 to 35.
- Testicle feels heavier or pulls down on one side: A growing tumor adds weight inside the testicle. The body feels the extra mass and the scrotum tries to support it. This heaviness is how testicular cancer often announces itself before a man even finds a lump.
- No pain when you touch the lump: Testicular cancer is sneaky — it grows without hurting. Pain usually comes from infection (swelling and germs) or injury (a hit or twist), not cancer. No pain does not mean no danger in testicular cancer. This silence tricks men into waiting too long.

- Breast tissue gets bigger or tender (gynecomastia): Some testicular cancer cells make a hormone called beta-hCG (a pregnancy-like signal). This hormone tells the body to grow breast tissue. Breast growth in a young man points back to a tumor making hormones in the testicle.
- Belly or lower back pain that does not go away: Testicular cancer spreads first to lymph nodes deep in the belly along the back wall (the retroperitoneal nodes). When these nodes swell with cancer, they press on nerves and muscles, causing pain that does not go away with rest. This pain means the cancer has already spread beyond the testicle.
- Cough or trouble breathing: After belly lymph nodes, testicular cancer spreads to the lungs. Tumors growing in the lungs block air sacs and make it hard to breathe, or irritate the lung tissue and cause coughing. Lung symptoms mean advanced-stage disease that needs chemotherapy, not just surgery.

Diagnostics · every test answers a question

- Scrotal ultrasound with Doppler → Is the lump solid tissue (likely cancer) or fluid (likely a harmless cyst)?
- Alpha-fetoprotein (AFP) blood level → Is there a marker leaking from a germ-cell tumor, and if so, which type?
- Beta-hCG (human chorionic gonadotropin) blood level → Is the tumor making a pregnancy-like hormone that signals cancer?
- Lactate dehydrogenase (LDH) blood level → How much tumor is growing, and how fast are cancer cells dying and spilling their insides into the blood?
- CT scan of the abdomen and pelvis with IV contrast → Has the cancer spread to the retroperitoneal lymph nodes (the nodes deep in the belly along the back wall)?
- CT scan of the chest with IV contrast → Has the cancer spread to the lungs or chest lymph nodes?

Treatment logic

- Radical inguinal orchiectomy (surgical removal of the testicle through a groin incision) → Removes the entire testicle, spermatic cord, and tumor in one piece through the groin (not the scrotum) so cancer cells do not spill into the scrotum's lymph drainage, which goes to different nodes and could spread the cancer. This surgery cures stage I testicular cancer in most men and gives a tissue diagnosis (seminoma vs. non-seminoma) that guides all further treatment.

- Sperm banking (cryopreservation) before surgery or chemotherapy → Saves healthy sperm cells frozen in liquid nitrogen so a man can father biological children later. One testicle removal usually leaves enough sperm production, but chemotherapy (especially cisplatin-based regimens) and radiation both damage the cells in the remaining testicle that make sperm, often causing permanent infertility.
- Active surveillance (for low-risk stage I seminoma or stage I non-seminoma) → Skips immediate chemotherapy or radiation and instead watches closely with blood tumor markers (AFP, beta-hCG, LDH) every 1–2 months and CT scans every 3–6 months for 2–3 years. If cancer comes back, treatment starts then. This approach avoids chemo side effects in the 70–80% of men whose cancer never comes back after surgery alone. It works because testicular cancer is so curable even if it recurs.
- BEP chemotherapy (bleomycin, etoposide, cisplatin) for stage II or III disease → Poisons all fast-dividing cancer cells throughout the whole body by damaging their DNA and stopping them from copying themselves. BEP shrinks tumors in belly lymph nodes and distant sites (like lungs) that surgery cannot reach. It cures more than 90% of men with advanced testicular cancer. Given as 3–4 cycles (each cycle is 3 weeks).
- Retroperitoneal lymph node dissection (RPLND) after chemotherapy → Surgically removes all lymph nodes in the back of the belly (the retroperitoneum) after chemotherapy shrinks tumors. This clears out any residual cancer masses ≥ 1 cm that might still have living tumor cells hiding inside dead tissue (teratoma or viable cancer). RPLND can cure men even when chemo does not kill every cell.
- Radiation therapy (for pure seminoma stage I or IIA) → Aims high-energy X-rays at the retroperitoneal lymph nodes to kill any microscopic cancer cells that spread there after orchiectomy. Seminoma is very sensitive to radiation. A short course (about 3 weeks, 20–30 Gray total dose) cuts recurrence risk from 15–20% to under 5%. Radiation is an alternative to surveillance or one cycle of carboplatin chemo in stage I seminoma.
- Single-dose carboplatin chemotherapy (for stage I seminoma) → Gives one dose of carboplatin (a gentler cousin of cisplatin) after orchiectomy to kill any hidden cancer cells and cut recurrence risk in half (from 15–20% to about 5–10%). It is faster and easier than radiation (one infusion vs. 3 weeks of daily treatments) and avoids radiation's late cancer risk. Carboplatin is only for seminoma, not non-seminoma.

Medications

Cisplatin · Platinum-based chemotherapy (DNA-damaging agent)

Cisplatin is a heavy metal drug that slips into fast-growing cancer cells and twists their DNA strands together so tightly that the cell cannot copy itself and dies. Testicular cancer cells multiply very quickly, so cisplatin hits them hard.

1. Cisplatin enters the bloodstream through an IV and travels to all parts of the body.
2. The drug crosses into cancer cells, especially fast-growing germ cell tumors (the type that starts in the cells that make sperm — seminomas and non-seminomas).
3. Inside the nucleus, cisplatin binds to the DNA strands and creates permanent cross-links (like tangling two ropes together so tightly you cannot pull them apart).
4. The cell's machinery tries to copy the DNA to divide, but the cross-links block it.
5. The cell realizes the damage is too severe to fix and triggers its own death (a process called apoptosis, where the cell shuts itself down on purpose).
6. Over several cycles (usually given in three- or four-day blocks repeated every three weeks), the tumor shrinks as more and more cancer cells die.

SAFETY WATCH

Kidney damage (nephrotoxicity): Cisplatin is filtered by the kidneys and can scar the filtering units (nephrons, the tiny tubes that clean your blood). Doctors give large amounts of IV fluid (saline) before and after each dose to flush the drug through and protect the kidneys. Blood tests check kidney function (creatinine and blood urea nitrogen, which go up when kidneys are struggling) before every cycle. · Hearing loss (ototoxicity): Cisplatin damages the tiny hair cells in the inner ear (the cochlea) that pick up high-pitched sounds. This loss is permanent. You may notice ringing in the ears (tinnitus) or trouble hearing conversations in noisy rooms. Hearing tests (audiograms) are done before treatment starts and repeated if symptoms appear. · Nerve damage (peripheral neuropathy): The drug can injure the long nerves in the hands and feet, causing numbness, tingling, or burning pain in the fingers and toes (like static or pins and needles). This may improve slowly after treatment stops, but it can be permanent in some patients. Dose may be reduced or switched to a different drug if nerve damage gets worse. · Nausea and vomiting: Cisplatin is one of the strongest triggers of nausea. Doctors give anti-nausea medicines before every infusion: 5-HT3 blockers (like ondansetron or granisetron, which block serotonin — the brain messenger that triggers the vomit reflex), NK1 blockers (like aprepitant or rolapitant, which block substance P — another vomit signal), and dexamethasone (a steroid that calms inflammation in the gut and brain). · Low blood counts (myelosuppression): Cisplatin kills rapidly dividing cells in the bone marrow (the spongy tissue inside bones that makes blood cells). This lowers white blood cells (raising your risk of infection), red blood cells (causing anemia — not enough oxygen carriers — which makes you tired and short of breath), and platelets (raising your risk of bleeding and bruising). Blood counts are checked weekly. Growth factors (filgrastim for white cells, erythropoietin or darbepoetin for red cells) may be needed if counts drop too low. · Heart and blood vessel damage (cardiovascular toxicity): Years after treatment, cisplatin increases the risk of high blood pressure, high cholesterol, heart attacks, and strokes. The drug damages the lining of blood vessels (the endothelium) and raises inflammation. Long-term survivors need regular cardiovascular screening (blood pressure, cholesterol panels, EKG). · Infertility: Cisplatin damages the sperm-producing cells (spermatogonia) in the testicles. All patients should be offered sperm banking (freezing sperm samples at a fertility clinic) before starting chemotherapy, because fertility may not return. Testosterone production (the hormone that controls sex drive, muscle, and energy) is usually preserved. · Second cancers: Years later, cisplatin slightly raises the risk of leukemia (cancer of the white blood cells) and solid tumors in other organs (like lung, colon, or bladder). Survivors need lifelong cancer screening according to American Cancer Society guidelines. · Low magnesium (hypomagnesemia): Cisplatin causes the kidneys to waste magnesium. Low magnesium can trigger muscle cramps, weakness, irregular heartbeats, and seizures. Magnesium levels are checked regularly and replaced with oral or IV supplements. · Allergic reactions (hypersensitivity): Rarely, cisplatin causes severe

allergic reactions (rash, hives, swelling, trouble breathing, low blood pressure, or anaphylaxis). This is more common with repeated doses. The infusion is stopped immediately if a reaction occurs, and you are given antihistamines, steroids, and epinephrine if needed.

Etoposide · Topoisomerase II inhibitor (DNA-damaging chemotherapy)

Etoposide blocks an enzyme (topoisomerase II, the molecular scissors that cut and re-glue DNA so it can unwind and copy itself) inside cancer cells. Without this enzyme, the DNA strands break and cannot be repaired, and the cancer cell dies. Etoposide is always given with cisplatin and bleomycin in the BEP regimen (the standard three-drug combination) for testicular cancer.

1. Etoposide is given through an IV (over 30 to 60 minutes to avoid low blood pressure) or as pills and enters the bloodstream.
2. The drug crosses into fast-growing cancer cells.
3. Inside the nucleus, etoposide locks onto topoisomerase II, the enzyme that normally cuts both DNA strands, lets them unwind (so the cell can copy the code), and then glues them back together.
4. Etoposide freezes the enzyme in the 'cut' position, leaving permanent breaks in the DNA (double-strand breaks, the most lethal kind of DNA damage).
5. The cell tries to divide, but the broken DNA triggers alarms (checkpoint proteins like p53) that force the cell to self-destruct (apoptosis).
6. Repeated cycles (usually five days in a row, repeated every three weeks) kill more and more cancer cells, shrinking the tumor.

SAFETY WATCH

Low blood counts (myelosuppression): Etoposide strongly suppresses the bone marrow. White blood cells drop (raising your risk of serious infection), red blood cells fall (causing anemia — tiredness, pale skin, shortness of breath), and platelets drop (raising your risk of bleeding and bruising). Blood counts are checked before each cycle. Treatment may be delayed if counts are too low (absolute neutrophil count below 1,000 or platelets below 75,000). · Infection risk (febrile dangerously low infection-fighting cells (neutropenia)): When white blood cells are very low (neutropenia, usually 7 to 14 days after each dose), even minor infections can become life-threatening. You should report fever over 100.4°F (38°C) immediately. Go to the emergency room — do not wait. IV antibiotics (usually a broad-spectrum drug like cefepime or piperacillin-tazobactam) are started right away, before culture results come back. · Nausea and vomiting: Etoposide causes moderate nausea. Anti-nausea drugs (5-HT3 blockers like ondansetron, NK1 blockers like aprepitant, and dexamethasone) are given before each dose. · Hair loss (alopecia): Etoposide damages the rapidly dividing cells in hair follicles. Most patients lose scalp hair, eyebrows, eyelashes, and body hair. Hair grows back after treatment ends, sometimes with a different texture or color. · Mouth sores (mucositis): The drug can damage the lining of the mouth and throat, causing painful ulcers that make eating and swallowing difficult. Gentle mouth rinses (salt water or baking soda solution, four times a day) and good oral hygiene (soft toothbrush, no alcohol-based mouthwash) help. Severe cases may need prescription mouth rinses with lidocaine (a numbing agent) or magic mouthwash. · Allergic reactions (hypersensitivity): Rarely, etoposide triggers hives, itching, flushing, wheezing, chest tightness, low blood pressure, or anaphylaxis during the infusion. Nurses monitor vital signs closely. If a reaction starts, the IV is slowed or stopped and you are given antihistamines, steroids, and epinephrine if needed. · Second cancers (treatment-related leukemia): Years after treatment, etoposide raises the risk of acute myeloid leukemia (AML) and myelodysplastic syndrome (MDS, a bone marrow disorder that can turn into leukemia). These are caused by damage to the bone marrow stem cells. The risk is about 1 to 2% over 10 to 20 years. Survivors need long-term monitoring (annual blood counts). · Low blood pressure (hypotension): Rapid IV infusion can drop blood pressure suddenly, causing dizziness, fainting, or shock. The drug is given slowly over 30 to 60 minutes, and you lie down during the infusion. · Diarrhea: Etoposide can damage the lining of the intestines, causing watery diarrhea. Drink plenty of fluids and tell your doctor if diarrhea is severe (more than four loose stools a day) — you may need anti-diarrheal medicine like loperamide. · Liver toxicity: Rarely, etoposide raises liver enzymes (ALT, AST, bilirubin). Liver function tests are checked before each cycle.

Bleomycin · Antitumor antibiotic (DNA-damaging chemotherapy)

Bleomycin is a drug (originally made by the bacterium Streptomyces verticillus) that cuts DNA strands inside cancer cells. The breaks pile up faster than the cell can fix them, and the cell dies. Bleomycin is the 'B' in the BEP regimen for testicular cancer.

1. Bleomycin is given through an IV (sometimes as a slow continuous infusion over 24 hours, or as a quick push once or twice a week) and enters the bloodstream.
2. The drug crosses into cancer cells.
3. Inside the nucleus, bleomycin binds to iron (Fe2+, the form of iron inside cells) and oxygen.
4. This combination generates oxygen free radicals (superoxide and hydroxyl radicals — highly reactive molecules that attack nearby structures like a tiny bomb).
5. The free radicals slice through both strands of the DNA helix at the same time, creating double-strand breaks (the hardest kind of damage for the cell to fix).
6. The cell's repair systems (like the proteins ATM and BRCA) are overwhelmed by the number of breaks.
7. The cell stops dividing and dies (apoptosis).

SAFETY WATCH

Lung damage (pulmonary fibrosis and pneumonitis): Bleomycin's most serious side effect is scarring of the lungs. The drug damages the air sacs (alveoli, the tiny balloon-like structures where oxygen enters the blood) and the tissue between them (the interstitium). Inflammation (pneumonitis) comes first, then scar tissue forms (fibrosis), making the lungs stiff and less able to absorb oxygen. Early signs are dry cough, shortness of breath with activity (like climbing stairs), and low oxygen levels (measured by pulse oximeter). Later signs are shortness of breath at rest, rapid breathing, and crackling sounds (crackles or crackles in the lungs (rales)) when the doctor listens to your lungs with a stethoscope. Doctors check pulmonary function tests (PFTs — you blow into a machine that measures how much air you can move and how fast) and oxygen saturation (the percentage of oxygen in your blood, measured by a clip on your finger) before treatment and watch closely during therapy. Bleomycin is stopped immediately if lung symptoms appear or if the total dose reaches 400 units (the risk of lung damage rises sharply above this dose). Scarring can be permanent and may worsen over time, even after the drug is stopped. CRITICAL SAFETY RULE: Patients who have received bleomycin should NEVER be given supplemental oxygen concentrations above 28 to 30% (even during surgery or anesthesia years later, or if you have pneumonia or a heart attack) because high oxygen can trigger sudden, severe worsening of bleomycin lung injury. Always tell every doctor, anesthesiologist, and emergency responder that you have received bleomycin. Wear a medical alert bracelet. · Skin changes: Bleomycin causes darkening (hyperpigmentation, like a deep tan or brown streaks) in skin creases (knuckles, elbows, knees), nail beds, and areas of pressure, friction, or injury (like where an IV was placed or a belt rubs). The skin may thicken, peel, crack, or develop painful sores (especially on the fingertips). Nails may develop dark vertical lines or fall off. These changes usually fade slowly (over months to years) after treatment stops, but some darkening may be permanent. · Raynaud phenomenon: Bleomycin can damage the small blood vessels in the fingers and toes (the capillaries and arterioles). When exposed to cold (like washing your hands in cold water or going outside in winter), the vessels clamp down too tightly, cutting off blood flow. The fingers or toes turn white (because no blood is reaching them), then blue (because oxygen is running out), then red and painful as blood rushes back in (rebound vasodilation). Wearing gloves and warm socks helps. Severe cases may need drugs to relax blood vessels (like calcium channel blockers such as nifedipine). · Fever and chills (pyrexia): Many patients develop flu-like symptoms (fever up to 103°F, shaking chills, muscle aches, headache) a few hours after the first dose. This is an inflammatory reaction, not an infection. It usually improves with later doses as your body gets used to the drug. Acetaminophen or ibuprofen (given before the dose) can help. If fever occurs later in treatment (especially when white blood cell counts are low), it could be a sign of infection — call your doctor immediately. · Allergic reactions (anaphylaxis): Rarely, bleomycin causes severe allergic reactions (sudden rash, hives, itching, swelling of the face or throat,

wheezing, chest tightness, trouble breathing, drop in blood pressure, fainting, or shock). This can happen during the first or second dose. Some doctors give a small test dose (1 to 2 units) first and watch you closely for one hour. If a reaction occurs, the infusion is stopped immediately and you are given antihistamines (like diphenhydramine), steroids (like hydrocortisone), and epinephrine (adrenaline) if needed. · Mouth sores (stomatitis and mucositis): Bleomycin can irritate and damage the lining of the mouth, tongue, and throat, causing painful red ulcers that make eating, drinking, and talking difficult. Good oral care (brushing gently with a soft toothbrush after every meal, rinsing with salt water or baking soda solution four times a day, avoiding alcohol-based mouthwash, spicy or acidic foods, and tobacco) helps prevent and heal sores. Severe cases may need prescription pain rinses (lidocaine gel or magic mouthwash). · Low blood counts (myelosuppression): Bleomycin is the gentlest of the three BEP drugs on the bone marrow, but it can still lower white blood cells, red blood cells, and platelets mildly. Blood counts are checked regularly. · Liver toxicity: Rarely, bleomycin raises liver enzymes (ALT, AST). Liver function tests are checked before each cycle. · Kidney damage: Bleomycin is cleared by the kidneys. High doses or pre-existing kidney disease can lead to kidney damage. Kidney function (creatinine, blood urea nitrogen, estimated glomerular filtration rate) is checked before treatment.

Carboplatin · Platinum-based chemotherapy (DNA-damaging agent)

Carboplatin is a platinum drug (chemically similar to cisplatin but gentler on the kidneys and nerves) that cross-links DNA strands inside cancer cells, stopping them from dividing. In testicular cancer, one dose of carboplatin after surgery is sometimes used instead of radiation or observation in stage I seminoma (cancer that has not spread beyond the testicle) to lower the chance of the cancer coming back.

1. Carboplatin is given as a single IV infusion (the dose is calculated by your kidney function using the Calvert formula: dose in milligrams equals target AUC — area under the curve, a measure of drug exposure — times your GFR plus 25; usually the target AUC is 7).
2. The drug travels through the bloodstream to any remaining cancer cells (microscopic clusters in the retroperitoneal lymph nodes — the lymph nodes at the back of the abdomen — are the most common hiding place).
3. Carboplatin enters the cells and binds to the DNA inside the nucleus, creating cross-links between and within strands (like gluing two pages of a book together so you cannot read or copy the words).
4. The cell tries to divide, but the tangled DNA blocks the copying machinery (DNA polymerase).
5. The cell's damage-detection system (checkpoint proteins like ATM and p53) recognizes the problem and triggers the cell to self-destruct (apoptosis).

6. This one-time treatment reduces the risk of recurrence from about 15% to about 3% in stage I seminoma (based on large randomized trials like MRC TE19 and SWENOTECA).

SAFETY WATCH

Low blood counts (myelosuppression): Carboplatin's main side effect is bone marrow suppression. Platelets drop first (thrombocytopenia, raising your risk of bleeding and bruising), then white blood cells (neutropenia, raising your risk of infection), then red blood cells (anemia, causing tiredness, pale skin, and shortness of breath). The lowest counts (nadir) usually occur 2 to 3 weeks after the dose. Blood counts are checked weekly. Most patients recover fully within 4 to 6 weeks. Severe cases (platelets below 25,000 or neutrophils below 500) may need platelet transfusions or growth factors (filgrastim for white cells). · Nausea and vomiting: Carboplatin causes moderate nausea (less than cisplatin but more than bleomycin). Anti-nausea medicines (5-HT3 blockers like ondansetron or granisetron, NK1 blockers like aprepitant, and dexamethasone) are given before the infusion. Nausea usually peaks 6 to 12 hours after the dose and improves over 1 to 2 days. · Kidney damage (nephrotoxicity): Carboplatin is much easier on the kidneys than cisplatin (it does not require aggressive IV hydration), but the dose is based on kidney function (glomerular filtration rate, GFR — the rate at which your kidneys filter blood, measured by a blood test or 24-hour urine collection). Overdosing can cause kidney injury. Blood tests (creatinine, blood urea nitrogen, and estimated GFR) are checked before treatment. Patients with pre-existing kidney disease need dose adjustment. · Allergic reactions (hypersensitivity): Rarely, carboplatin causes allergic reactions (rash, itching, hives, flushing, wheezing, chest tightness, trouble breathing, low blood pressure, or anaphylaxis). This is more common in patients who have received carboplatin before (the immune system becomes sensitized). Reactions usually occur during or within minutes of the infusion. The infusion is stopped immediately, and you are given antihistamines (like diphenhydramine), steroids (like hydrocortisone or dexamethasone), and epinephrine if needed. If you need carboplatin again, your doctor may give it very slowly with pre-medications (antihistamines and steroids) or switch to a different drug. · Nerve damage (peripheral neuropathy): Carboplatin is much less likely than cisplatin to cause numbness, tingling, or pain in the hands and feet, but it can happen, especially with repeated doses or high cumulative doses. Symptoms usually improve slowly after treatment stops. · Hearing loss (ototoxicity): Carboplatin can damage the hair cells in the inner ear (cochlea), causing hearing loss (especially high-frequency sounds) and ringing in the ears (tinnitus). This is less common than with cisplatin but still possible. A hearing test (audiogram) may be done before treatment if you already have hearing problems. · Infertility: Even a single dose of carboplatin can harm sperm production (spermatogenesis). The cells that divide to make sperm (spermatogonia) are very sensitive to chemotherapy. Sperm banking (freezing sperm samples at a fertility clinic) should be offered before treatment. Sperm counts may recover over time (often 1 to 3 years), but recovery is not guaranteed. Testosterone production (made by different cells, the Leydig cells) is usually preserved, so sex drive, erections, and muscle mass are typically not affected. · Second cancers (treatment-related malignancies): Long-term (10 to 20 years after treatment), carboplatin raises the risk of

leukemia (especially acute myeloid leukemia, AML) and solid tumors (like lung, colon, bladder, or stomach cancer). The risk is lower than with multi-cycle cisplatin regimens (like BEP), but it is still increased. Survivors need lifelong cancer screening according to American Cancer Society and National Comprehensive Cancer Network guidelines. · Fatigue: Carboplatin can cause tiredness and weakness, partly from anemia and partly from the body's inflammatory response to the drug. Fatigue usually improves as blood counts recover. · Liver toxicity: Rarely, carboplatin raises liver enzymes (ALT, AST, alkaline phosphatase, bilirubin). Liver function tests are checked before treatment.

Red flags

- A hard, painless lump in the testicle that does not go away
- Breast swelling or tenderness in a young man
- Back pain that lasts more than a few weeks without injury
- Coughing up blood or trouble breathing
- Sudden severe testicular pain with a known mass

Nursing priorities

1. Arrange urgent scrotal ultrasound with Doppler within 24 hours of presentation — Ultrasound uses sound waves to show if the lump is solid tumor or fluid-filled cyst (hydrocele), and Doppler checks blood flow patterns that differ between cancer and benign (not cancer) lumps
2. Draw blood for tumor markers AFP, hCG, and LDH before any surgery or treatment — Baseline tumor marker levels help classify cancer type and stage, and later levels track if treatment is working or if cancer comes back
3. Provide sperm banking referral and education before orchiectomy or chemotherapy starts — Chemotherapy drugs damage rapidly dividing sperm cells, and removing one testicle plus treatment can cause permanent azoospermia (no sperm in semen), taking away the chance to father biological children
4. Teach patient that the surgeon will remove the testicle through a groin incision, not through the scrotum — Inguinal approach prevents cancer cells from spreading to scrotal lymphatics (drainage vessels) which drain to different lymph nodes than testicular lymphatics, keeping cancer contained for better cure rates
5. Assess for signs of tumor lysis syndrome if starting chemotherapy, including checking potassium, phosphorus, uric acid, and calcium levels — When chemotherapy kills cancer cells rapidly, the cells burst and release potassium and phosphorus into blood while calcium drops, causing dangerous heart arrhythmias (irregular beats) and kidney failure

6. Monitor pulmonary function and oxygen saturation closely during bleomycin administration — Bleomycin causes pulmonary fibrosis (lung scarring) in up to 10 percent of patients, reducing oxygen exchange and causing permanent breathing problems or respiratory failure
7. Schedule and reinforce surveillance imaging and tumor marker follow-up every 1 to 3 months for the first year — Testicular cancer recurrence (cancer coming back) peaks in the first 2 years, and rising AFP or hCG levels detect relapse weeks to months before CT scans show visible tumors, allowing earlier salvage treatment
8. Provide emotional support and mental health referral for young men facing cancer and fertility concerns — Testicular cancer strikes men aged 15 to 35 during peak reproductive and career years, causing psychological distress about masculinity, body image, and future family planning that affects treatment adherence

Sources

- NCCN Clinical Practice Guidelines in Oncology: Testicular Cancer 2024
- American Cancer Society
- American Urological Association
- Journal of Clinical Oncology

Last reviewed: 2026-07-07