

Lung Cancer

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THE STORY IN ONE SENTENCE

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“Lung cancer has two main types — small-cell (spreads fast, hits smokers hard) and non-small-cell (slower, bigger tumors) — and today we can test each tumor's genes to pick drugs that match the exact mutation driving it.”

Normal anatomy — the body's job

Your lungs are built like an upside-down tree: the windpipe (trachea) splits into two main tubes (bronchi), which branch into smaller and smaller airways (bronchioles) that end in clusters of tiny air sacs called alveoli — thin-walled bubbles wrapped in blood vessels where oxygen jumps into your blood and carbon dioxide jumps out.

BODY'S JOB

The lining cells (epithelium) along those airways do three jobs: (1) make a thin layer of mucus to trap dust and germs, (2) wave tiny hairs (cilia) to sweep that mucus up and out, and (3) divide just enough to replace worn-out neighbors, then stop. This controlled turnover keeps your airways sealed, clean, and ready to move 10,000 liters of air every day.

1. Air flows in through your nose or mouth, down the trachea, and splits at the carina (the fork where the windpipe divides) into the right and left main bronchi.
2. Each bronchus branches like a tree — first into lobar bronchi (three branches on the right, two on the left), then into smaller segmental bronchi, and finally into bronchioles thinner than a pencil lead.

3. At the tips of the smallest bronchioles sit grape-like clusters of alveoli, each wrapped in a net of capillaries so thin that oxygen and carbon dioxide pass through in a fraction of a second.
4. The lining cells along every airway sit on a basement membrane (a thin mat of protein) and are held in place by cell-to-cell anchors (tight junctions and desmosomes) that keep the barrier sealed against germs and irritants.
5. When a lining cell gets old or damaged, a stem cell nearby divides once, one daughter stays a stem cell, and the other matures to replace the lost cell — then both stop dividing. That tight control prevents overgrowth.

What breaks

Cigarette smoke (and other poisons like radon gas, asbestos fibers, or diesel exhaust) lands on the airway lining thousands of times a day, cracking the DNA inside cells. Most cells fix the break or die. But if mutations pile up in the genes that control cell division (oncogenes like KRAS, EGFR) or the genes that force damaged cells to self-destruct (tumor suppressors like TP53, RB1), one cell loses its stop signal and starts copying itself endlessly — that is cancer.

CLINICAL TERM

Carcinogenesis (the birth of cancer) in the lung follows a multi-hit model: repeated exposure to carcinogens (tobacco smoke is responsible for 85% of cases; radon, asbestos, arsenic, and air pollution account for most of the rest) causes DNA adducts (chemical damage to the genome), overwhelming repair pathways. When mutations accumulate in critical genes — activating oncogenes (EGFR, KRAS, ALK, ROS1, BRAF, MET, RET, NTRK) or inactivating tumor suppressors (TP53, RB1, STK11, CDKN2A) — a single clone escapes apoptosis (programmed cell death), evades immune surveillance, and proliferates without restraint, forming a primary tumor that invades local structures and metastasizes via lymphatics and hematogenous spread to brain, bone, liver, and adrenal glands.

How the body compensates

The body has no way to 'fix' a clone of cells that will not stop dividing, so instead it tries to keep you alive by working around the damage — coughing to clear blocked airways, speeding up breathing to pull in more oxygen, shifting blood flow away from the tumor, and ramping up your heart rate. These workarounds can keep your vital signs looking stable even as the cancer

grows, which is why a patient can feel 'okay' until suddenly they crash with a collapsed lung, massive bleeding, or brain swelling.

1. {'what': 'Cough reflex kicks into overdrive', 'why': 'The tumor irritates airway nerve endings (vagal afferents) and blocks mucus flow, so your brainstem (the medulla) fires the cough center over and over to try to clear the obstruction — that is why the cough is dry at first, then brings up blood-streaked coughed-up mucus (sputum) (hemoptysis) when the tumor erodes into a blood vessel.', 'limit': 'Coughing cannot shrink a tumor; it only exhausts your chest muscles and spreads tumor cells into other airways if you aspirate blood.'}
2. {'what': 'Respiratory rate climbs and you switch to mouth breathing', 'why': "As tumor bulk or fluid (malignant pleural a fluid collection (effusion)) reduces the lung's working surface area, oxygen saturation starts to dip; chemoreceptors in the carotid body and aortic arch sense low O₂ or high CO₂ and tell the brainstem to speed up breathing (tachypnea) — you will jump from 12 breaths/min to 24–30 without noticing, and you will feel 'air hunger' (dyspnea) even at rest.", 'limit': 'Faster breathing only helps if you have enough healthy lung left; once the tumor or fluid takes over 50% of lung volume, fast breathing (tachypnea) cannot keep oxygen up and CO₂ starts to rise (hypercapnia), leading to confusion and eventually respiratory failure.'}
3. {'what': 'Heart rate rises (tachycardia) and blood pressure may climb', 'why': "Low oxygen triggers the sympathetic nervous system (your body's gas pedal) to release epinephrine and norepinephrine, which speed your heart and squeeze blood vessels, pushing more blood per minute through the shrinking pool of working alveoli — this is why resting pulse jumps from 70 to 100–120.", 'limit': "The heart cannot keep this pace forever; chronic a fast heart rate (tachycardia) leads to heart failure, and if a big clot (pulmonary a traveling clot lodging in a vessel (embolism)) or tumor mass suddenly blocks a main vessel, the right ventricle (the heart's right pump) stalls (acute cor pulmonale) and blood pressure crashes."}
4. {'what': 'Weight loss (cachexia) as the body burns muscle and fat', 'why': 'The tumor steals glucose, amino acids, and fatty acids from your bloodstream to fuel its wild growth, and it secretes cytokines (TNF- α , IL-6) that crank up your metabolic rate (like running a fever without the heat) and break down muscle (proteolysis) — so you lose 10–20 pounds in weeks even if you eat the same amount.', 'limit': "Cachexia is not simply 'not eating enough'; giving extra calories by mouth or IV does not reverse it because the tumor's metabolic hijacking continues. Cachexia is an independent predictor of shorter survival and poor treatment tolerance."}

5. {'what': 'Collateral circulation tries to route blood around blocked vessels', 'why': 'If the tumor compresses the superior vena cava (SVC, the big vein that drains your head, neck, and arms), venous pressure upstream shoots up; smaller veins in the chest wall dilate to form detours (collaterals) so blood can still reach the heart — you see engorged veins across the chest and neck (SVC syndrome).', 'limit': 'Collaterals are slow and leaky; face and neck swell (edema), the airway can swell shut (stridor), and if pressure spikes in brain veins you get headache, blurred vision, and altered mental status — this is an oncologic emergency requiring urgent radiation or stent.'}
6. {'what': 'Paraneoplastic syndromes — the tumor makes hormones or antibodies by mistake', 'why': 'Some lung cancers (especially small-cell) secrete ACTH (causing Cushing syndrome with high cortisol, moon face, and high blood sugar), ADH (antidiuretic hormone, causing SIADH with dangerously low sodium), or PTHrP (parathyroid-hormone-related peptide, driving calcium sky-high and causing confusion, kidney stones, and heart arrhythmias). Others trigger autoimmune attacks: antibodies meant for tumor antigens cross-react with cerebellum (paraneoplastic cerebellar degeneration), neuromuscular junction (Lambert-Eaton syndrome, causing muscle weakness that improves with use), or peripheral nerves (sensory neuropathy).', 'limit': 'These syndromes can appear months before the tumor is visible on imaging, and they do not respond to the usual hormone pills or IV fluids — you must treat the cancer itself to shut off the ectopic signal.'}

Domino effect

1. Years of cigarette smoke (or radon, asbestos, diesel exhaust) land on the airway lining cells, and toxic chemicals — benzo(a)pyrene, nitrosamines, formaldehyde — slice into DNA thousands of times. — Each puff delivers >7,000 chemicals, at least 70 of which are proven carcinogens; they form DNA adducts (chemical scars on the genome) that, if not repaired before the cell divides, become permanent mutations passed to daughter cells.
2. Most damaged cells either fix the break (using DNA repair enzymes) or die (apoptosis). But if mutations hit the genes that control the cell cycle (KRAS, EGFR, ALK) or block self-destruct (TP53, RB1), one cell escapes and starts dividing endlessly — that single rogue cell is the seed of cancer. — It takes roughly 5–10 critical mutations (the 'multi-hit' model) for a normal cell to become fully malignant: one hit turns on a growth signal, another blocks the stop signal, a third hides the cell from the immune system, and so on — so cancer is not bad luck; it is the endpoint of repeated damage over 20–30 years.

3. The clone grows into a visible tumor (a lump in the airway or lung tissue), crowding and crushing normal structures — airways narrow or collapse (causing wheezing and shortness of breath), blood vessels erode (causing coughing up blood (hemoptysis), coughing up blood), and the tumor eats up body fuel (causing unintended weight loss and fatigue). — Tumors double in size every 100–200 days for non-small-cell, every 30 days for small-cell; once the mass is 1 cm (the size of a pea), it contains ~1 billion cells and starts to show on CT — but it has been growing for 5–8 years before you notice symptoms.
4. Cancer cells break off and travel through lymph channels (to hilar and mediastinal lymph nodes) and through blood vessels (hematogenous spread) to distant organs — brain (20–40% of patients develop brain metastases), bone (spine, ribs, pelvis, causing pain and fractures), liver, and adrenal glands — setting up new tumors in each site. — Metastasis is not random: cancer cells that survive the bloodstream journey 'home' to organs with slow flow and lots of nutrients (the 'seed and soil' hypothesis) — brain mets cause headache, seizures, or stroke-like symptoms; bone mets release calcium into blood (hypercalcemia) causing confusion and kidney stones; liver mets elevate liver enzymes and cause yellowing of the skin and eyes (jaundice).
5. Some tumors secrete hormones or proteins never meant to come from the lung — ACTH (causing Cushing syndrome), ADH (causing SIADH with low sodium and confusion), PTHrP (causing high calcium (hypercalcemia)), or autoantibodies (causing Lambert-Eaton syndrome with muscle weakness, or cerebellar degeneration with loss of coordination (ataxia)) — so the patient presents with bizarre symptoms that seem unrelated to the lungs. — These paraneoplastic syndromes happen because cancer cells revert to a primitive gene program and accidentally turn on genes that were only active in the embryo or in neuroendocrine cells; treating the cancer shuts off the ectopic signal, but until then standard hormone therapy (e.g. giving insulin for high blood sugar) does not work.
6. Local invasion can compress or invade the superior vena cava (SVC syndrome: face/neck swelling, dilated chest-wall veins, a high-pitched noise on breathing in (stridor)), the recurrent laryngeal nerve (hoarse voice), the esophagus (trouble swallowing), the phrenic nerve (paralyzed diaphragm, severe shortness of breath), or the chest wall (severe, unrelenting chest pain). — The mediastinum (the middle compartment of the chest) is packed tight: tumor growing in the right upper lobe or mediastinal lymph nodes has nowhere to go but into adjacent structures — SVC syndrome and airway compression are oncologic emergencies requiring same-day radiation or stent to prevent respiratory arrest.

7. Fluid accumulates in the pleural space (malignant pleural a fluid collection (effusion)) or around the heart (malignant pericardial a fluid collection), collapsing the lung or compressing the heart; air can leak into the pleural space (pneumothorax) if the tumor eats through an airway; and massive coughing up blood (hemoptysis) (coughing up ≥ 100 mL of blood at once) can drown the patient if the tumor erodes into a major pulmonary artery. — Malignant effusions form when tumor cells seed the pleural lining and leak fluid that cannot drain (because lymphatics are blocked); pericardial a fluid collection (effusion) can cause cardiac tamponade (the heart squeezed by fluid around it) (the heart cannot fill between beats, and blood pressure drops dangerously); pneumothorax and massive coughing up blood (hemoptysis) are late-stage emergencies with minutes to intervene.
8. Without treatment, median survival from diagnosis of metastatic non-small-cell lung cancer was 4–6 months (historically); small-cell lung cancer spreads so fast that untreated extensive-stage disease has a median survival of 2–3 months. Today, targeted therapy (for EGFR, ALK, ROS1, BRAF, KRAS G12C, MET, RET, NTRK mutations) and immunotherapy (pembrolizumab, nivolumab, atezolizumab) have pushed median survival in selected groups to 2–5 years, and some patients with driver mutations achieve long-term disease control (>5 years). — Lung cancer is the #1 cancer killer ($\sim 130,000$ U.S. deaths/year, >1.8 million worldwide) not because it is the most common cancer, but because it is usually caught late (stage III or IV) when cure is rare — yet the explosion of molecular testing and immunotherapy since 2015 has transformed outcomes for patients whose tumors harbor actionable mutations or high PD-L1, proving that lung cancer is not one disease but a collection of molecularly distinct diseases, each requiring a different playbook.

Symptoms · what the patient feels, and why

- A cough that won't go away or gets worse over weeks: The tumor irritates the airways or blocks them partly, triggering the cough reflex—your body's way of trying to clear the passage.
- Coughing up blood or rust-colored spit: The tumor erodes into tiny blood vessels in the airway wall, so blood leaks into the mucus you cough up.
- Chest pain that feels deep and aches, especially when you breathe in or cough: The tumor presses on the pleura (the thin wrap around the lung that has pain nerves) or invades the chest wall, stretching those sensitive layers.
- Shortness of breath or wheezing: The tumor narrows a major airway, or fluid builds up around the lung (pleural a fluid collection (effusion)), so less air gets in and out with each breath.
- Hoarse voice that doesn't get better: The tumor presses on the recurrent laryngeal nerve (the nerve that controls your voice box), paralyzing one vocal cord so it can't vibrate normally.

- Losing weight without trying, and feeling very tired: Cancer cells release cytokines (chemical signals that ramp up inflammation) which speed up your metabolism and break down muscle and fat, draining your energy stores.
- Bone pain, especially in the back or hips: Lung cancer can spread (metastasize) to bones; tumor cells eat into the bone tissue and release chemicals that fire up pain nerves.
- New headaches, confusion, or trouble with balance: The cancer has spread to the brain, where growing tumors press on brain tissue and raise pressure inside the skull.
- Swelling in the face, neck, or arms with bulging veins: The tumor squeezes the superior vena cava (the large vein draining blood from your upper body back to the heart), backing up blood and fluid above the blockage.

Focused exam

- Cough that has lasted more than 3 weeks: A tumor growing inside the airway rubs and irritates it constantly, like sandpaper scraping the inside of a straw. In a smoker, a new cough or one that suddenly sounds different is the first warning bell.
- Coughing up blood (hemoptysis): The tumor eats through the wall of tiny blood vessels inside the lung. When blood shows up in spit, it means the cancer has dug deep into living tissue, not just sitting on the surface. Even a small streak matters.
- Shortness of breath and chest pain that gets worse when you breathe in deeply: The tumor takes up space or fluid leaks around the lung (pleural a fluid collection (effusion)), squeezing the lung like a balloon in a box. The lung cannot stretch and fill with air the way it should.
- Weight loss and feeling exhausted without trying to lose weight: Cancer cells burn fuel fast and steal calories and nutrients from the rest of the body. The immune system is fighting around the clock, and the body is losing the battle, like a car idling in the red zone all day.
- Voice sounds raspy (hoarse) or trouble swallowing: The tumor can press on the recurrent laryngeal nerve (the wire that moves the vocal cords) or push on the esophagus (the swallowing tube). When the nerve is pinched, the voice box cannot close properly.
- Swelling of the face, neck, or arms: A lung tumor can squeeze or block the superior vena cava (the big vein bringing blood back from the head and arms to the heart). Blood backs up like a traffic jam, and fluid leaks into the face and arms. This is called superior vena cava (SVC) syndrome.

Diagnostics · every test answers a question

- Low-dose chest CT scan → Is there a mass or nodule in the lung, and where exactly is it sitting?

- Chest X-ray (posteroanterior and lateral) → Is there a visible shadow, mass, or fluid around the lung?
- Tissue biopsy with histology and molecular testing (EGFR, ALK, ROS1, BRAF, PD-L1, KRAS) → What type of cancer cells are these, and which broken genes or proteins do they have?
- PET-CT scan (positron emission tomography with CT) → Has the cancer spread beyond the lung to lymph nodes, liver, bones, or brain?
- Brain MRI with contrast → Has the cancer spread to the brain?
- Complete blood count (CBC) → Is the bone marrow keeping up with making enough red cells, white cells, and platelets?
- Comprehensive metabolic panel (CMP) including calcium → Are the kidneys and liver working, and is calcium dangerously high?
- Pulmonary function tests (spirometry) → How much lung capacity is left, and can the person survive surgery if needed?

Treatment logic

- Low-dose CT screening every year for people aged 50–80 with at least 20 pack-years of smoking history (current smokers or quit within the past 15 years) → Catches tumors early when they are small (stage I or II) and before symptoms show up, so surgery can remove them completely. Early detection cuts lung cancer deaths by about 20%.
- Surgery (lobectomy or wedge resection) to remove the tumor and nearby lymph nodes → Cuts out the tumor entirely along with a margin of healthy tissue and lymph nodes, stopping the cancer from spreading further. This is the only cure for early-stage (stage I–II) lung cancer.
- Targeted therapy matched to the tumor's specific mutation: osimertinib for EGFR mutations, alectinib or brigatinib for ALK rearrangements, sotorasib or adagrasib for KRAS G12C, dabrafenib plus trametinib for BRAF V600E → Blocks the exact broken signal inside cancer cells that tells them to keep copying themselves, like cutting a specific wire in a faulty alarm. Tumors shrink fast and people live months to years longer. Works only if the tumor has that exact mutation.
- Immunotherapy (pembrolizumab, nivolumab, atezolizumab, or durvalumab) alone or combined with platinum-based chemotherapy (carboplatin or cisplatin plus pemetrexed or etoposide) → Chemotherapy poisons all fast-growing cells, including cancer. Immunotherapy lifts the brakes off T cells (the immune system's attack soldiers) so they can recognize and kill cancer cells. Together they shrink tumors and help people live longer, especially when PD-L1 is high or there is no targetable mutation.

- Radiation therapy (stereotactic body radiation or conventional fractionated radiation) → Uses high-energy beams to damage the DNA inside cancer cells so they die and stop dividing. Used to shrink tumors that cannot be cut out, treat tumors that spread to the brain or bones, or kill leftover microscopic cancer after surgery.
- Smoking cessation (stop all tobacco products, avoid secondhand smoke, use nicotine replacement or varenicline if needed) → Stops new toxic chemicals (tar, benzene, formaldehyde) from entering the lungs and breaking more genes. Slows cancer growth, helps chemotherapy and radiation work better, speeds healing after surgery, and cuts the risk of a second lung cancer.
- Supportive care: supplemental oxygen for low oxygen levels, opioids (morphine) for pain and shortness of breath, cough suppressants, thoracentesis (draining fluid around the lung), nutrition support, palliative care team involvement → Keeps the body comfortable and functioning (breathing easier, pain controlled, hunger and nausea managed, fluid balance restored) so the person can tolerate cancer treatment and maintain quality of life.

Medications

Osimertinib · EGFR tyrosine kinase inhibitor (targeted cancer pill)

Blocks the mutated EGFR protein—a broken switch on lung cancer cells that tells them to grow nonstop—so the cells can no longer multiply out of control.

1. In about 15% of lung cancers (more in people who never smoked), a mutation breaks the EGFR switch so it stays stuck in the 'on' position.
2. The locked-on switch floods the cell with grow-and-divide signals.
3. Osimertinib fits into the pocket of the broken EGFR protein and blocks it, like jamming a key into a lock so the real key can't turn.
4. Without the constant grow signal, the cancer cells stop dividing and many die off.
5. This drug works only in tumors that actually have the EGFR mutation; a biopsy test finds the mutation before you start.

SAFETY WATCH

Diarrhea and skin rash (dry, acne-like bumps) are common; keep skin moisturized and tell your team if diarrhea is severe. · Rare but serious lung inflammation (pneumonitis)—call right away if you have new or worsening shortness of breath or cough. · Heart rhythm changes (QT prolongation)—your doctor will check an ECG before and during treatment. · Nail and skin problems; protect your skin from the sun.

Alectinib · ALK tyrosine kinase inhibitor (targeted cancer pill)

Blocks the abnormal ALK protein—a scrambled gene product that drives relentless growth in some lung cancers—so those cancer cells lose their main fuel and shrink.

1. In about 5% of non-small cell lung cancers (especially in younger people and never-smokers), part of the ALK gene fuses with another gene to create a mutant protein that never shuts off.
2. That nonstop signal tells the cell to divide over and over.
3. Alectinib plugs the active site of the ALK protein, silencing the grow command.
4. Cancer cells that depend on ALK stop multiplying; many die.
5. A biopsy test (ALK fluorescence or next-generation sequencing) confirms the rearrangement before starting the drug.

SAFETY WATCH

Muscle and joint pain, leg swelling. · Liver enzyme elevation—regular blood tests monitor liver function. · Vision changes (floaters, flashes)—report new eye symptoms promptly. · Slow heart rate (bradycardia); fatigue and dizziness can be clues. · Take with food to reduce stomach upset.

Pembrolizumab · PD-1 immune checkpoint inhibitor (cancer immunotherapy infusion)

Releases the brake on your own immune T cells so they can recognize and attack lung cancer cells that were hiding by flipping a 'don't eat me' switch.

1. Normally, T cells patrol for abnormal cells, but cancer cells often display a protein called PD-L1 that binds to PD-1 on the T cell and tells it 'I'm harmless, stand down.'
2. That fake-peace signal stops the T cell from killing the cancer.
3. Pembrolizumab is an antibody that covers the PD-1 receptor so PD-L1 can't dock there.
4. With the brake released, the T cells wake up, recognize the cancer as foreign, and destroy it.
5. This works best in tumors with high PD-L1 expression ($\geq 50\%$ of cells); a test on your tumor sample measures PD-L1 before treatment.

SAFETY WATCH

Immune-related side effects: the awakened immune system can mistakenly attack your own organs—lung (pneumonitis), colon (colitis with severe diarrhea), liver (hepatitis), thyroid, pituitary, kidneys, or skin. · Fatigue and rash are common. · Report any new symptoms immediately—fever, persistent cough, severe diarrhea, yellowing of the skin and eyes (jaundice), vision changes, or extreme weakness—because early steroid treatment can reverse most immune attacks. · Infusion reactions (chills, fever during the IV) are possible but uncommon.

Carboplatin · Platinum-based chemotherapy (IV infusion)

Crosslinks the DNA strands inside rapidly dividing cells—both cancer and some normal cells—so they can't copy their genetic instructions and die when they try to split.

1. Carboplatin enters cells and binds to DNA, creating kinks and cross-links between the two DNA strands.
2. When the cell tries to divide, it can't unzip and copy the tangled DNA.
3. The cell's quality-control system detects the damage and triggers cell death (apoptosis).
4. Because cancer cells divide much faster than most normal cells, they take the biggest hit—but fast-growing normal tissues (bone marrow, gut lining, hair follicles) also get damaged.

SAFETY WATCH

Low blood counts (myelosuppression): fewer white cells (infection risk), red cells (anemia, fatigue), and platelets (bleeding/bruising). Blood tests before each cycle track your counts. · Nausea and vomiting—anti-nausea medicines are given before and after infusion. · Kidney damage—drink plenty of fluids; your team monitors creatinine levels. · Numbness and tingling in hands and feet (peripheral neuropathy), though less than cisplatin. · Hearing loss and ringing in the ears (tinnitus) can occur. · Allergic reactions during infusion (rash, wheezing, low blood pressure)—staff watch closely during the drip.

Pemetrexed · Antifolate chemotherapy (IV infusion)

Blocks three enzymes that cells need to build the DNA and RNA letters (nucleotides), so fast-dividing cancer cells can't copy their genetic code and die.

1. Folate (vitamin B9) is essential for making purines and pyrimidines, the letters of the DNA alphabet.
2. Pemetrexed mimics folate and jams three key enzymes: thymidylate synthase, dihydrofolate reductase, and glycinamide ribonucleotide formyltransferase.
3. Without those enzymes, the cell runs out of DNA building blocks.

4. When the cell tries to divide, it can't finish copying its DNA and triggers death signals.
5. Cancer cells divide rapidly and burn through nucleotides fast, so they feel the shortage first.

SAFETY WATCH

Low blood counts (especially white cells and platelets)—watch for fever, unusual bruising, or bleeding. · Fatigue and loss of appetite. · Mouth sores and diarrhea. · You **MUST** take folic acid daily and get a vitamin B12 shot every nine weeks during treatment—these vitamins protect normal cells without feeding the cancer. · Skin rash—steroid premedication (dexamethasone) the day before, day of, and day after infusion reduces this. · Rare severe allergic reactions.

Etoposide · Topoisomerase II inhibitor (chemotherapy, IV or pill)

Freezes topoisomerase II—the enzyme that untangles twisted DNA strands—in a broken state, so the DNA snaps and the cell dies; used mainly in small cell lung cancer combined with a platinum drug.

1. When DNA unzips to copy itself, it gets tangled like a twisted phone cord.
2. Topoisomerase II acts like molecular scissors: it snips both DNA strands, lets them unwind, then glues them back together.
3. Etoposide locks the enzyme onto the DNA at the cut stage, leaving permanent breaks.
4. The cell's repair machinery can't fix the shattered DNA, so it activates cell death.
5. Small cell lung cancer divides so rapidly that it relies heavily on topoisomerase II, making it very sensitive to this drug.

SAFETY WATCH

Severe drop in white blood cells and platelets—fever, infection, and bleeding are real risks; blood counts are checked before each cycle. · Nausea, vomiting, loss of appetite. · Hair loss (temporary; grows back after treatment). · Mouth sores. · Low blood pressure during IV infusion—given slowly and monitored. · Very rare: secondary leukemia years later (the drug can damage bone-marrow DNA).

Atezolizumab · PD-L1 immune checkpoint inhibitor (cancer immunotherapy infusion)

Blocks PD-L1 on cancer cells and immune cells so they can't turn off T cells, freeing your immune system to recognize and destroy the tumor.

1. Many lung cancers and immune cells in the tumor display PD-L1, which acts like a 'safe' badge when it touches PD-1 on patrolling T cells.

2. That handshake sends a 'stand down' signal to the T cell, hiding the cancer.
3. Atezolizumab is an antibody that covers PD-L1 so it can't reach PD-1.
4. With the off-switch blocked, T cells stay active and attack the cancer.
5. Often combined with chemotherapy in extensive-stage small cell lung cancer or non-small cell lung cancer without a targetable mutation.

SAFETY WATCH

Immune-related side effects: the unleashed immune system can attack normal organs—lungs (cough, shortness of breath), colon (diarrhea, belly pain), liver (elevated enzymes, yellowing of the skin and eyes (jaundice)), thyroid or pituitary glands (fatigue, headache, hormone imbalances), skin (rash), kidneys. · Report any new or worsening symptoms immediately; most immune reactions reverse with early steroid treatment. · Infusion reactions (fever, chills, rash during the drip) are uncommon. · Fatigue is frequent.

Durvalumab · PD-L1 immune checkpoint inhibitor (cancer immunotherapy infusion)

Binds to PD-L1 on cancer and immune cells, preventing it from silencing T cells, so the immune system continues to hunt and kill lingering cancer cells after chemoradiation.

1. After chemoradiation for stage III non-small cell lung cancer, microscopic cancer cells often remain.
2. These cells display PD-L1 to evade immune surveillance.
3. Durvalumab covers PD-L1 so T cells don't receive the 'ignore me' signal.
4. Active T cells recognize leftover cancer cells as foreign and destroy them.
5. Given for up to a year after chemoradiation, this consolidation immunotherapy significantly lowers the chance of the cancer coming back.

SAFETY WATCH

Immune-related inflammation: pneumonitis (new cough or shortness of breath), colitis (severe diarrhea), hepatitis (liver enzyme spikes, yellow skin), endocrine problems (thyroid, adrenal, pituitary), nephritis (kidney inflammation), rash. · Any new symptom should prompt immediate contact with your oncology team. · Steroids can reverse most immune side effects if caught early. · Infusion reactions are rare. · Fatigue and cough are common.

Nivolumab · PD-1 immune checkpoint inhibitor (cancer immunotherapy infusion)

Blocks the PD-1 receptor on T cells so cancer cells can't use their PD-L1 disguise to escape immune attack, reawakening the body's ability to fight the tumor.

1. T cells patrol for cancer, but tumor cells often show PD-L1, which locks onto PD-1 and sends a 'do not attack' message.
2. Nivolumab is an antibody that covers PD-1, blocking PD-L1 from docking.
3. The T cell stays activated and recognizes the cancer cell as a threat.
4. The T cell releases toxic proteins that punch holes in the cancer cell and trigger its death.
5. Used in advanced non-small cell lung cancer, especially after chemotherapy or in combination regimens.

SAFETY WATCH

Immune-related toxicity: lung inflammation (pneumonitis), diarrhea and colitis, liver inflammation (hepatitis), skin rash, thyroid or pituitary problems, kidney inflammation. · Fatigue. · Report fever, persistent cough, severe diarrhea, yellowing of the skin and eyes (jaundice), or unusual weakness right away. · Early treatment with steroids usually reverses immune side effects. · Infusion reactions (rare).

Crizotinib · ALK/ROS1/MET tyrosine kinase inhibitor (targeted cancer pill)

Jams the abnormal ALK or ROS1 protein—mutant switches that drive uncontrolled growth in certain lung cancers—so cancer cells lose their main growth engine and shrink.

1. In about 5% of non-small cell lung cancers, a gene rearrangement fuses ALK or ROS1 with another gene, creating a permanently 'on' growth switch.
2. That nonstop signal floods the cell with commands to multiply.
3. Crizotinib fits into the active pocket of the mutant protein and blocks it.
4. Without the growth signal, cancer cells stop dividing and many die.
5. A molecular test (fluorescence in situ hybridization or next-generation sequencing) on a tumor biopsy identifies the rearrangement.

SAFETY WATCH

Vision changes (flashes, floaters, blurry vision)—common and usually mild. · Nausea, vomiting, diarrhea, constipation. · Swelling in legs and around eyes. · Liver enzyme elevation—regular blood tests monitor liver function. · Slow heart rate (bradycardia). · Lung inflammation (rare but serious): report new or worsening cough or shortness of breath immediately.

Dabrafenib plus Trametinib · BRAF inhibitor + MEK inhibitor (targeted cancer pill combination)

Dabrafenib blocks the mutant BRAF protein and trametinib blocks MEK, the next protein in the same growth pathway, shutting down a hyperactive signaling chain that drives cancer cell division.

1. In 1-2% of non-small cell lung cancers, a BRAF V600E mutation locks the BRAF protein in the 'on' position.
2. Active BRAF turns on MEK, which then activates ERK, flooding the cell with grow-and-divide signals.
3. Dabrafenib jams the mutant BRAF; trametinib blocks MEK as backup insurance.
4. Hitting two steps of the same pathway prevents the cancer from bypassing the blockade.
5. With the growth engine stalled, cancer cells stop multiplying and die.

SAFETY WATCH

Fever (sometimes high)—report any fever over 100.4°F; may need to pause the drugs. · Skin rash, thickening of skin on palms and soles. · New skin cancers (squamous cell carcinoma, melanoma)—regular skin checks are essential. · Heart problems: reduced pumping strength (monitor with echocardiograms), irregular heartbeat. · Eye problems: blurred vision, seeing colors differently; tell your doctor promptly. · Bleeding, blood clots. · High blood sugar. · Diarrhea, nausea.

Sotorasib · KRAS G12C inhibitor (targeted cancer pill)

Locks the mutant KRAS G12C protein in its 'off' state, preventing it from sending grow signals that drive cancer cell division in tumors with this specific mutation.

1. KRAS is a molecular switch that flips between 'off' (GDP-bound) and 'on' (GTP-bound) to relay growth signals.
2. The G12C mutation makes KRAS favor the 'on' state, constantly telling the cell to divide.
3. Sotorasib wedges into a pocket on the mutant KRAS protein when it is in the 'off' position and traps it there.
4. With KRAS locked off, downstream growth pathways (MAPK, PI3K) shut down.
5. Cancer cells dependent on this mutant switch stop growing and many die.

SAFETY WATCH

Diarrhea (very common)—anti-diarrheal medicine and hydration are important. · Liver enzyme elevation—regular blood tests; may need dose adjustment. · Nausea, fatigue, muscle and joint pain. · Rare serious liver injury—report yellowing of skin or eyes, dark urine, severe fatigue. · Take on an empty stomach (at least one hour before or two hours after a meal) for best absorption.

Red flags

- Coughing up blood
- Severe chest pain or trouble breathing at rest
- Sudden confusion, seizure, or one-sided weakness (signs of brain spread)
- Swelling of the face and neck with visible bulging veins (superior vena cava syndrome)
- New bone pain that won't go away, especially in the spine

Nursing priorities

1. Monitor oxygen saturation continuously and keep SpO₂ above 90 percent with supplemental oxygen as needed — Lung tumors block airways and destroy gas-exchange surfaces called alveoli, reducing oxygen delivery to blood and risking low oxygen in the blood (hypoxemia) (low blood oxygen) that damages the brain and heart within minutes
2. Assess respiratory rate, work of breathing, and lung sounds every four hours, watching for a high-pitched noise on breathing in (stridor) or new wheezing — Tumors can obstruct large bronchi (breathing tubes) suddenly, causing respiratory distress, or pleural effusions (fluid around lungs) can accumulate fast and collapse lung tissue
3. Position patient sitting upright at 45 to 90 degrees and encourage slow deep breathing or incentive spirometry every hour while awake — Upright posture uses gravity to expand lower lung lobes and prevents atelectasis (collapsed air sacs), while deep breathing opens airways that tumors partially block
4. Check for superior vena cava syndrome signs: facial swelling, neck vein distention, arm swelling, or new confusion — Central lung tumors can compress the superior vena cava (large vein returning blood to heart), causing blood backup that increases brain pressure and can obstruct the airway within hours
5. Administer scheduled pain medication around the clock and assess pain every two hours using a zero-to-ten scale — Uncontrolled pain from chest wall invasion or bone metastases (cancer in bones) causes shallow breathing that leads to pneumonia and prevents coughing to clear secretions

6. Monitor daily weights and calorie intake, offering high-protein snacks every two hours if patient is losing weight — Lung cancer releases cytokines (inflammatory chemicals) that cause cachexia (muscle wasting), and malnourished patients heal poorly from surgery and tolerate chemotherapy worse
7. Teach and document smoking cessation at every visit, offering nicotine replacement patches or gum and referral to quit programs — Continued smoking doubles pneumonia risk after surgery, cuts radiation effectiveness in half, and accelerates tumor growth even during active treatment
8. Screen for depression and anxiety using a two-question screen and notify the provider if patient expresses hopelessness or suicide thoughts — Lung cancer carries high mortality rates and stigma about smoking, causing major depression in forty percent of patients, which worsens treatment adherence and immune function

Sources

- NCCN Clinical Practice Guidelines in Oncology: Non-Small Cell Lung Cancer 2024
- American Cancer Society Lung Cancer Guidelines
- CHEST Guideline and Expert Panel Report on Lung Cancer Screening 2021
- ASCO Clinical Practice Guideline on Systemic Therapy for Stage IV Non-Small Cell Lung Cancer

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