

Lung Cancer

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.

THE ONE-LINE TRUTH

Lung cancer kills more people than any other cancer — and most cases start with cigarette smoke damaging the DNA in lung cells until one cell breaks free and multiplies without stopping.

The chain — how it unfolds

1. Years of cigarette smoke (or radon, asbestos, diesel exhaust) land on the airway lining cells, and toxic chemicals — benzopyrene, nitrosamines, formaldehyde — slice into DNA thousands of times. — why: 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. — why: 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). — why: 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. — why: 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. — why: 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). — why: 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. — why: 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). — why: Lung cancer is the #1 cancer killer (~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.

Spot it — what you see, 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.

Red flags — act now

- 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

Treat it — in this order

1. 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%.
2. 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.
3. 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.
4. 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.
5. 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.

Your top priorities at the bedside

1. Monitor oxygen saturation continuously and keep SpO₂ above 90 percent with supplemental oxygen as needed — why: 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 — why: 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 — why: Upright posture uses gravity to expand lower lung lobes and prevents atelectasis (collapsed air sacs), while deep breathing opens airways that tumors partially block