

COPD Exacerbation

COPD flare-up (exacerbation) — an infection or other trigger suddenly worsens the already-narrowed, damaged airways in chronic obstructive pulmonary disease, trapping air and raising CO₂.

THE ONE-LINE TRUTH

The patient's airways are already narrow from long-term damage, and now something (usually an infection) has made them even tighter and filled with mucus.

The chain — how it unfolds

1. A trigger (infection, pollution, or pulmonary embolism (a clot lodging in a lung artery)) hits already-damaged airways. — why: The most common trigger is a viral or bacterial respiratory infection that inflames the airway lining, but any irritant or added lung stress can tip a fragile system into crisis.
2. Airways swell, fill with thick mucus, and squeeze tight (bronchospasm). — why: Inflammation brings more fluid and immune cells into the airway walls (making them thicker), mucus glands pour out thick secretions (clogging the passage), and smooth muscle contracts (narrowing the tube even more).
3. Air gets trapped in the lungs because breathing out through narrow airways is too slow. — why: It takes longer to push air out through a narrow, floppy tube, so the patient does not finish exhaling before the next breath in—leftover air stacks up breath after breath (dynamic hyperinflation).
4. The chest barrel-shapes and the diaphragm flattens. — why: Trapped air over-inflates the lungs like a balloon that never fully deflates, pushing the ribs out and the diaphragm down into a flat, weak position instead of its normal dome.
5. The diaphragm and other breathing muscles tire out from working at a bad angle and against high resistance. — why: A flattened diaphragm cannot contract efficiently, and pushing air through swollen, mucus-filled airways is like breathing through a narrow straw—both exhaust the muscles quickly.

6. The patient shifts to rapid, shallow breathing and uses neck and shoulder muscles to help.
— why: Shallow breaths take less work per breath than deep ones when the diaphragm is weak, and recruiting accessory muscles buys a little more time—but shallow breathing moves air mostly in the dead space (mouth, throat, large airways) and very little reaches the alveoli for gas exchange.
7. Oxygen drops (hypoxemia) and carbon dioxide rises (hypercapnia). — why: Shallow breathing and mucus-plugged airways mean fresh air does not reach the alveoli to load oxygen, and stale air full of CO₂ cannot be pushed out, so both gases go the wrong way in the blood.
8. Blood pH falls (worsening too much acid in the blood (acidosis)) because CO₂ is an acid when dissolved in blood. — why: The kidneys have been holding extra bicarbonate for years to buffer chronic high CO₂, but in an acute flare-up (exacerbation) CO₂ climbs faster than the kidneys can keep up, so pH drops—acidic blood impairs heart rhythm, brain function, and muscle strength.

Spot it — what you see, and why

- Breathing gets harder or faster than usual: The airways are squeezed tighter and filled with more mucus than normal, so air can't flow in and out easily. Your body tries to breathe faster to get enough oxygen.
- More coughing than usual: The airways are irritated and filled with extra mucus. Your lungs try to clear it out by coughing.
- Coughed-up mucus (Sputum) (the mucus you cough up) changes color to yellow, green, or brown, or you cough up more of it: When bacteria grow in the mucus, white blood cells rush in to fight them. Those cells and dead bacteria turn the mucus yellow or green.
- Chest feels tight: The muscles around your airways squeeze down, and the airways themselves swell. This makes your chest feel like a band is wrapped around it.
- Wheezing (a whistling sound when you breathe): Air is being pushed through narrowed, swollen airways. The narrow space makes the air vibrate and whistle.
- Using neck and shoulder muscles to breathe, or belly moving in and out a lot: Your main breathing muscle (the diaphragm) can't keep up alone, so extra muscles in your neck, shoulders, and belly join in to help move air.

Red flags — act now

- New confusion or can't stay awake
- Lips or fingernails turn blue or gray
- Chest pain

- Breathing is so hard you can't speak a full sentence
- Using neck and shoulder muscles to breathe, or sitting and leaning forward to breathe

Treat it — in this order

1. Nebulized short-acting beta-agonist (albuterol) plus short-acting anticholinergic (ipratropium) → Relaxes the smooth muscle squeezing the airways so air can move in and out again. Opens the tubes within minutes and gets oxygen up and carbon dioxide down faster. This is the fastest way to relieve airway obstruction.
2. Systemic corticosteroids (prednisone 40 mg by mouth daily for 5 days, or equivalent IV dose) → Calms down the inflammation and swelling inside the airway walls and reduces mucus production. Keeps the airways more open for days so the patient can breathe easier and recovers faster. Cuts hospital stay by about 1 day and lowers the chance of treatment failure.
3. Antibiotics (azithromycin, doxycycline, or amoxicillin-clavulanate) if at least 2 of these are present: worse trouble breathing (dyspnea), more coughed-up mucus (sputum), or pus-filled (purulent) (thick yellow or green) coughed-up mucus → Kills the bacteria causing the infection so the inflammation and mucus production stop getting worse. Shortens recovery time and lowers the chance the patient will come back to the hospital.
4. Controlled oxygen therapy to target SpO₂ 88 to 92 percent (NOT higher) → Raises oxygen in the blood just enough so organs get what they need and the body works less hard. Keeps a little bit of low oxygen so the brain's breathing center stays awake and reminds the patient to keep breathing.
5. Noninvasive positive-pressure ventilation (BiPAP or CPAP) if pH is below 7.35, PaCO₂ is above 45 mmHg, and respiratory rate is above 25 despite initial treatment → Pushes air into the lungs so the patient does not have to work so hard and carbon dioxide escapes more easily. Lowers the need for intubation (breathing tube) by about half and cuts mortality. Buys time while medications start working.

Your top priorities at the bedside

1. Position the patient sitting upright at 60 to 90 degrees, leaning slightly forward with arms resting on the overbed table. — why: This position (called tripod position) lets the diaphragm (main breathing muscle) drop down fully and uses gravity to help air get into the lower lung areas, which decreases the work of breathing.

2. Start controlled oxygen therapy with a nasal cannula or Venturi mask, targeting oxygen saturation (SpO₂) of 88 to 92 percent, and monitor closely for decreased respiratory rate or increased sleepiness. — why: COPD patients can lose their normal drive to breathe if given too much oxygen because their bodies rely on low oxygen (hypoxia) as the trigger to breathe; too much oxygen can cause hypercapnia (carbon dioxide buildup) and respiratory failure.
3. Administer nebulized albuterol (a short-acting beta-agonist that opens airways) and ipratropium (a short-acting anticholinergic that also opens airways) together, usually every 4 to 6 hours or more often if ordered. — why: These bronchodilators (medicines that relax and widen the airway muscles) work by different mechanisms and together they open the narrowed airways faster and more completely, making it easier to move air in and out.