

!! JAY AMBE !!

17. COPD

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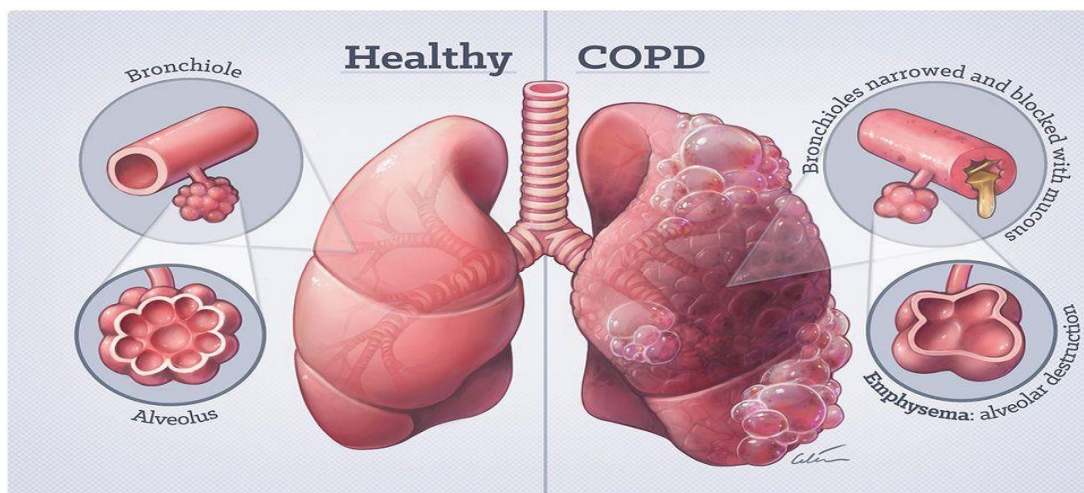
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DEFINATION OF COPD

COPD (chronic obstructive pulmonary disease) is a group of lung diseases that make it hard to breathe and get worse over time.

- ❖ Normally, the airways and air sacs in the lungs are elastic or stretchy.
- ❖ When we breathe in, the airways bring air to the air sacs.
- ❖ The air sacs fill up with air, like a small balloon.
- ❖ When we breathe out, the air sacs deflate, and the air goes out.
- ❖ In COPD, less air flows in and out of airways because of one or more problems:
 - ✓ The airways and air sacs in lungs become less elastic
 - ✓ The walls between many of the air sacs are destroyed
 - ✓ The walls of the airways become thick and inflamed
 - ✓ The airways make more mucus than usual and can become clogged

TYPES OF COPD

1. **Emphysema** affects the air sacs in lungs, as well as the walls between them. They become damaged and are less elastic.
2. **Chronic bronchitis**, in which the lining of airways is constantly irritated and inflamed. This causes the lining to swell and make mucus.

SYMPTOMS

Signs and symptoms of COPD may include:

- ✓ Shortness of breath, especially during physical activities
- ✓ Wheezing
- ✓ Chest tightness
- ✓ A chronic cough that may produce clear, white, yellow or greenish MUCUS
- ✓ Frequent respiratory infections
- ✓ Lack of energy
- ✓ Unintended weight loss (in later stages)
- ✓ Swelling in ankles, feet or legs

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ETIOLOGY:-

- ✓ The cause of COPD is usually long-term exposure to irritants that damage lungs and airways.
- ✓ The cigarette smoke is the main cause. Pipe, cigar, and other types of tobacco smoke can also cause COPD.
- ✓ Exposure to other inhaled irritants can contribute to COPD.
These include second hand smoke, air pollution, and chemical fumes or dusts from the environment or workplace.
- ✓ Rarely, a genetic condition called alpha-1 antitrypsin deficiency can play a role in causing COPD.

| Etiology | Mechanism(s) |
|---------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Cigarette smoke | Presence of smoke particles in the lungs leads to an inflammatory response with increased macrophage and neutrophil infiltration into the lungs. These immune cells release cytokines, chemokines and elastases , which damages the lung parenchyma over time. |
| Occupational exposures | Etiology unclear, however, is hypothesized to be a similar inflammatory response that damages the alveoli. |
| Alpha-1 antitrypsin deficiency | Alpha-1 antitrypsin is a serine protease inhibitor (SERPIN) secreted by the liver into the blood which inhibits the enzyme neutrophil elastase from damaging the lung tissue. Deficiency of this alpha-1 antitrypsin leads to unopposed elastolysis (destruction of the elastin fibers in alveolar walls) and development of early emphysema. This is the protease-antiprotease hypothesis of emphysema development. |
| Chronic IV drug use | IV drug users of cocaine, methadone and heroin are at higher risk for developing COPD; this is attributed to the vascular damage induced by the insoluble filler (cornstarch, cellulose, talc, fiber etc) found in IV drugs. |

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PATHOPHYSIOLOGY

COPD can be caused by environmental exposure, particularly cigarette smoking, in generally genetically susceptible persons.

Emphysema:

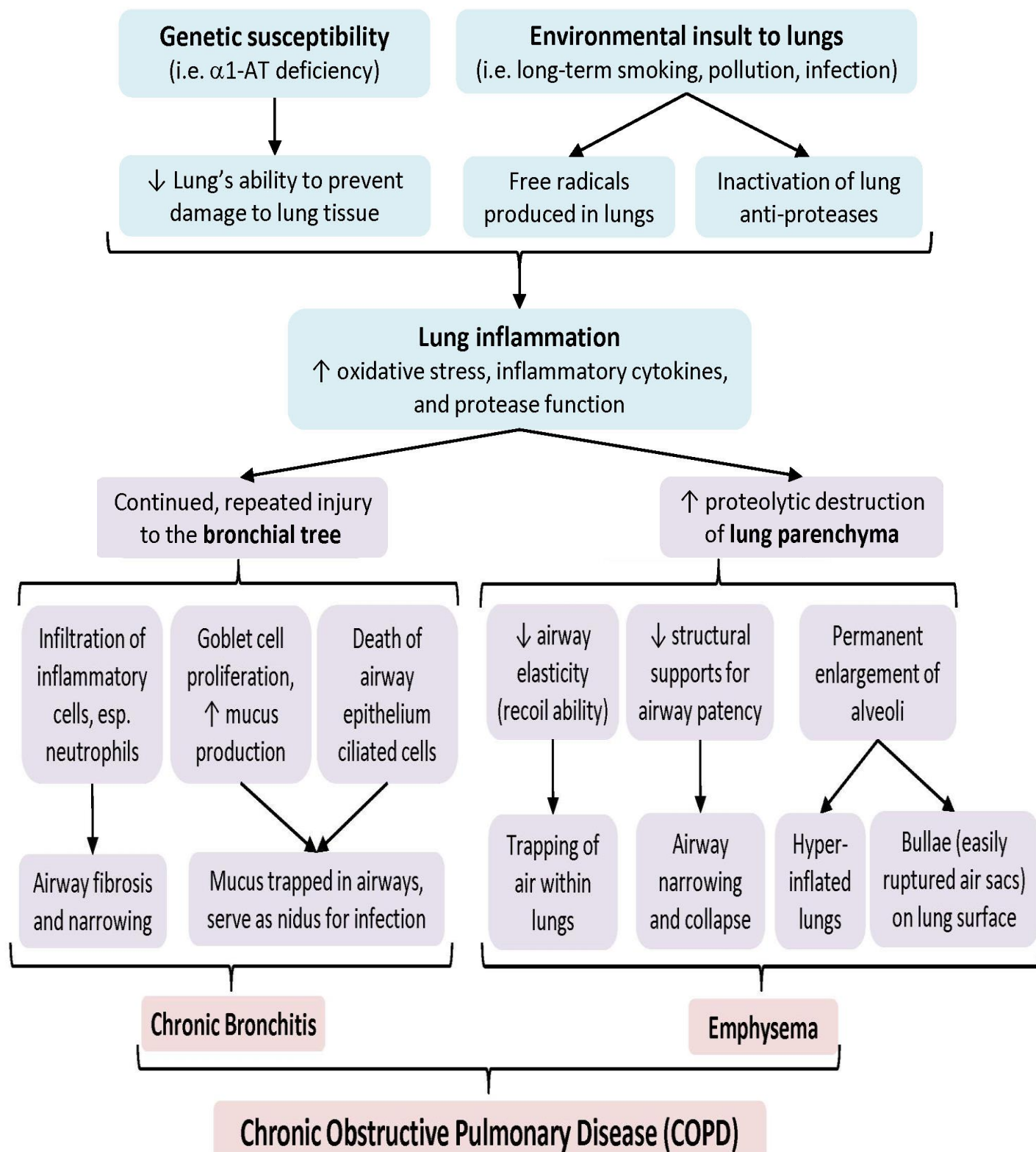
- ❖ This is due to damage of air sacs (alveoli) that destroys the walls inside them and causes them to merge into one big air sac.
- ❖ It can't absorb oxygen properly, so less oxygen goes in blood.
- ❖ Damaged alveoli can make lungs stretch out and lose their elasticity.
- ❖ Air gets trapped in lungs and person can not breathe it out, so you feel short of breath.
- ❖ In emphysema:
 - ✓ Smoking causes inflammation in airways.
 - ✓ Neutrophils and other immune cells are recruited to the small airways.
 - ✓ Releasing protease and oxidative species
 - ✓ Neutrophil elastase breaks down elastin fibers that normally contributes to the elastic recoil during expiration.
 - ✓ Alpha 1 antitrypsin is a protease inhibitor that keeps elastase in check.
 - ✓ Alpha 1 antitrypsin is the best known genetic predisposition to emphysema, especially in smokers with his genetic disorder.
 - ✓ Impaired gas exchange and air trapping are also the features of emphysema

Chronic bronchitis:

- ❖ If the person have coughing, shortness of breath, and mucus that lasts at least 3 months for 2 years in a row, then the person have chronic bronchitis.
- ✓ Hair-like fibers called cilia line of bronchial tubes and help move mucus out.
- ✓ In chronic bronchitis, cilia is damaged due to inflammation.
- ✓ This makes it harder to get clear the mucus, which makes cough more, which creates more mucus.
- ❖ **In chronic bronchitis**
 - ✓ Inflammation from smoke exposure also causes fibrosis of the bronchiolar walls, mucus hypersecretion, airways edema, and bronchoconstriction.
 - ✓ These features make up the small airways disease component of COPD known as chronic bronchitis

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PATHOPHYSIOLOGY - 2



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COMPLICATIONS

COPD can cause many complications, including:

- ❖ **Respiratory infections.** People with COPD are more likely to catch colds, the flu and pneumonia.
- ❖ **Heart problems.** For reasons that aren't fully understood, COPD can increase risk of heart disease, including heart attack
- ❖ **Lung cancer.** People with COPD have a higher risk of developing lung cancer.
- ❖ **High blood pressure in lung arteries.** COPD may cause high blood pressure in the arteries that bring blood to lungs (pulmonary hypertension).
- ❖ **Depression.** Difficulty breathing can keep person from doing activities. And dealing with serious illness can contribute to the development of depression.

DIFFERENCE BETWEEN ASTHMA AND COPD

| ASTHMA AND COPD COMPARED | |
|-----------------------------------------------------------------------|----------------------------------------------------|
| ASTHMA | COPD |
| High proportion of non-smokers | High proportion of smokers/ex-smokers |
| Symptom onset before age 40 is common | Symptoms develop after age 40 |
| Breathing difficulty is intermittent ("attacks") | Breathing difficulty is persistent and progressive |
| Night-time symptoms and/or attacks are common | Night-time attacks are uncommon |
| Patients may be asymptomatic or with minimal symptoms between attacks | Variability of symptoms is rare |
| Symptoms may be affected by exercise | Symptoms worsen with exercise |