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Chronic Obstructive Pulmonary Disease (COPD)

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Chronic Obstructive Pulmonary Disease (COPD)

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Why COPD?

As of 2021 the CDC reports more than 16 million Americans are diagnosed with COPD and estimate millions more are undiagnosed (Centers for Disease Control and Prevention, 2021).

In 2019, Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide, causing 3.23 million deaths (World Health Organization 2022).

Nearly 90% of COPD deaths in those under 70 years of age occur in low- and middle-income countries (World Health Organization 2022).

Health care costs due to potentially preventable hospitalizations due to COPD exacerbations resulted in \$7.27 billion spent in the United States in 2017 ("Aggregate costs of potentially preventable adult inpatient stays by condition U.S. 2017," 2020).

Clinical Manifestations

Signs and symptoms of COPD (McCance et al., 2018):

- Shortness of breath, dyspnea on exertion (DOE)
- Wheezing, increased sputum production, cough
- Hypoxemia with exercise or everyday tasks requiring supplemental oxygen
- History of smoking
- Barrel chest
- Prolonged expiration, chronic hypoventilation
- Cyanosis, polycythemia, cor pulmonale

Diagnosis

Based on history of symptoms, physical exam, chest imaging, pulmonary function tests (PFTS) and blood gas (McCance et al., 2018).

PFT reveals airway obstruction (decreased FEV1) (Soriano et al., 2018).

By the time individual seeks medical care due to symptoms, considerable airway damage is already present (McCance et al., 2018).

Underlying Pathophysiology

Chronic Bronchitis

Chronic exposure to irritant engages inflammatory cells such as neutrophils, macrophages, and lymphocytes. These inflammatory cells cause bronchial edema, increase the number and size of mucosal and goblet cells in the airway, hypertrophy with fibrosis, and narrowing of the airway. Ciliary function is impaired causing inability to clear thick mucus. Frequent bacterial infections occur from compromised defense mechanisms in the lung (McCance et al., 2018).

Emphysema

Emphysema is the destruction of alveoli caused by an imbalance of proteases, antiproteases, oxidative stress, and apoptosis of structural lung cells (McCance et al., 2018). Like chronic bronchitis, inflammatory cells (TNF- α , IL-8, and chemokines) are increased fostering the destruction of bronchi and alveoli (Wang et al., 2018). This results in significantly reduced surface area for gas exchange, ventilation-perfusion mismatch, and hypoxemia (McCance et al., 2018).

Asthma

Asthma-Chronic Obstructive Pulmonary Disease Overlap Syndrome (ACOS) is a presentation of clinical features from both diseases. Like COPD, Asthma is an inflammatory disease causing bronchial hyperresponsiveness, bronchoconstriction, and airway obstruction (McCance et al., 2018). Asthmatics may clinically resemble patients with COPD with several similar, but different underlying mechanisms (Hikichi, et al., 2018).

Significance of Pathophysiology

Irreversible, pathologic inflammatory modifications occur in large central airways, small peripheral airways, and in the lung. Bronchial edema and increased goblet cell activity considerably increases mucous production and its tenacity. This is seen in combination with the destruction of alveoli and loss of elasticity in the bronchial walls. Together these phenotypes cause airway obstruction, hypoxemia, and increase susceptibility to infection (McCance et al., 2018).

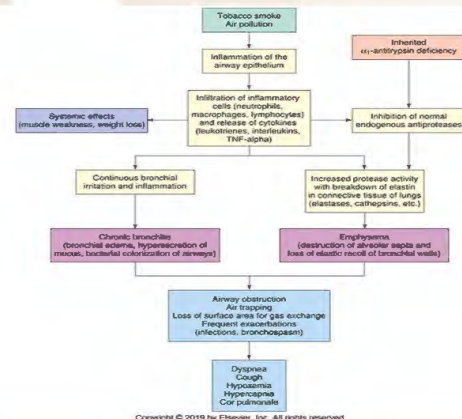
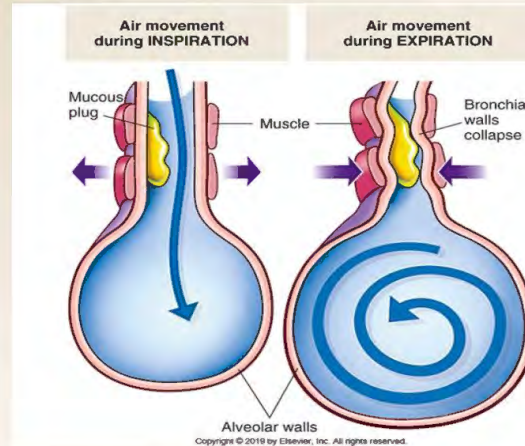


Diagram 1. Pathophysiology behind the process of COPD (McCance et al., 2018).

COPD

Topic: Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable respiratory disease characterized airflow limitation that is not fully reversible and typically progressive. It includes two phenotypes that overlap with each other: chronic bronchitis and emphysema (McCance et al., 2018). Significant exposure to noxious particles or gasses creates chronic inflammation causing airway and alveolar abnormalities (Soriano et al., 2018).



Implications for Nursing Care

- COPD is not reversible. Prevention is best treatment. Treatment is based on symptom management (McCance et al., 2018).
- Smoking cessation is paramount in the prevention of COPD and to slow down the progression of the disease (Soriano et al., 2018).
- Chest physical therapy including deep breathing, incentive spirometry, and postural drainage. Teaching nutritional counseling, respiratory hygiene, recognize early signs of infection, and pursed-lip breathing to relieve dyspnea (McCance et al., 2018).
- COPD commonly presents alongside other comorbidities such as cardiovascular disease, causing increased risk of exacerbation. Identification and treatment of cardiac comorbidity in COPD patients can help positively affect prognosis (André et al., 2019).
- COPD patient will commonly require non-invasive ventilation (NIV) with supplemental oxygen. Standard of care for acute respiratory failure and hypoxemia (Viniol & Vogelmeier, 2018).

Conclusion

COPD is comprised of two phenotypes: emphysema and chronic bronchitis. Frequent exposure to noxious irritants causes inflammation and progressive irreversible damage in the airway and lung. Smoking is a major cause of COPD and cessation is key in preventing progression. Treatment is based upon symptom management to reduce incidence of exacerbation (McCance et al., 2018).

Treatment

Prevention of Exacerbations

Smoking cessation: key to prevent further morbidity and mortality. Cessation can slow decline in forced expiratory volume in 1 second (FEV1) (Hikichi et al., 2018).

Pharmacotherapy: current data suggests bronchodilation with long-acting antimuscarinic antagonists (LAMA) (e.g., tiotropium) monotherapy has a greater effect on exacerbation over long-acting B2-agonist (LABA) (e.g., salmeterol) monotherapy (Viniol & Vogelmeier, 2018).

Inhaled corticosteroids: long-term oral corticosteroids not recommended. Long-term treatment with inhaled corticosteroids (ICS) may be considered for patients with history of exacerbations especially if patient has high blood eosinophil count with asthma and COPD overlap (Viniol & Vogelmeier, 2018).

Combination therapy: significant decrease in incidence of exacerbations by combining LAMA and LABA/ICA compared to using one or the other. ICS should be saved for certain circumstances (Viniol & Vogelmeier, 2018).

Phosphodiesterase-4 inhibitor: GOLD guidelines recommend PDE-4 inhibitors with very severe airflow limitation (Viniol & Vogelmeier, 2018).

Prophylactic antibiotics: macrolide antibiotics indicated for patients with moderate to severe airflow obstruction. Antibiotic resistance should be considered when using therapy (Viniol & Vogelmeier, 2018).

Mucolytics: many inflammatory cells involved in COPD causing stimulation of mucus production from goblet and submucosal cells (Wang et al., 2018). Mucolytics useful in patients with moderate to severe airflow obstruction despite inhaled therapy (McCance et al., 2018).

Monoclonal antibodies: patients with elevated eosinophil level using mepolizumab has been associated with decreased incidence of exacerbation, however, no formal data to currently recommend this treatment (Viniol & Vogelmeier, 2018).

Other Preventative Recommendations

Physical activity and pulmonary rehabilitation: strongest predictor of mortality in COPD patients is activity level. COPD patients with low activity levels have a higher mortality risk (Viniol & Vogelmeier, 2018).

Education and self-management

Vaccination: current guidelines recommend receiving both influenza and pneumococcal vaccinations with evidence showing reduced incidence of exacerbation (Viniol & Vogelmeier, 2018).

Treatment of COPD Exacerbations

Noninvasive ventilation: supplemental oxygenation is first-line intervention in COPD exacerbation. Standard of care in acute respiratory failure (Viniol & Vogelmeier, 2018).

B-agonist and anticholinergics: initial exacerbation treatment includes short acting B2-agonists and short-acting anticholinergics (Viniol & Vogelmeier, 2018).

Corticosteroids: have been shown to improve lung function and oxygenation, shorten recovery time and duration of hospitalizations. Should use no longer than 5-day treatment (Viniol & Vogelmeier, 2018).

Antibiotics: a 5-7 day course of antibiotics can be initiated according to ERS/ATS guidelines if purulent sputum is expectorated. Procalcitonin level may be checked as a general marker for antibiotic treatment (Viniol & Vogelmeier, 2018).

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