CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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CHRONIC OBSTURCTIVE PULMONARY DISEASES

- DEFINATION:- Chronic obstructive pulmonary disease is one of the most common lung disease it makes it difficult to breathe the airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particle or gases
- It is mainly causes by cigarette ,smoking, although COPD affect the lungs.

Classification of COPD

- They are classify in two type

i. Chronic Bronchitis:-It is defined clinically as chronic productive cough for 3months in each of 2 years in a patients.

- It involves a long term cough with mucus.

ii. Emphysema:- It defined as the presence of permanent enlargement of airspace distal to the terminal bronchiole, accompanied by destruction of their walls without obvious fibrosis.

- It involves destruction of the lungs over time.

Epidemiology of COPD

- According to WHO estimates, 65 million people have moderate to severe chronic obstructive pulmonary disease (COPD). More than 3 million people died of COPD in 2005.
- It is known that almost 90% of COPD deaths occur in low- and middle-income countries.

Etiology/Causes/Risk Factor/Incidence

- COPD is cause by smoking.
- The more person smoke ,the more likely that person will develop COPD.
- However, some people smoke for years & never get COPD
- In rare case, nonsmoker who lack the protein called α-1 antitrypsin can develop emphysema.

Risk factor of COPD

- Genetic factor
- Sex
- Airway hyper reactivity, immunoglobin E & asthma

❑ **Exposures**

- Smoking
- Occupation
- Enviroment pollution
- Diet
- Childhood illness

Other Risk factor

- Exposure to certain gases
- Frequent use of cooking fire without proper ventilation.

Pathophysiology

- The different pathogenic mechanism discussed above produce the pathological changes, which turn to give rise to following physiology abnormalities in COPD like :-

A. Mucus hyper secretion & ciliary dysfunction
B. Airflow limitation & hyperinflation
C. Gas exchange abnormalities
D. Pulmonary hypertension
E. Systemic effect

A. **Mucous hpersecretion & cilliary dysfunction:-**

- These are the first physiological abnormalities of COPD, due to stimulated secretion from enlarged mucous gland & squmous- metaplasia of epithelial cell.
- Mucus hypersecretion and decreased cilliary function occur early in the course of COPD. The result is mucus plugging, primarily in the small peripheral airways. In the presence of increased mucus production and reduced clearance of airway secretions, airways that are typically sterile may become colonized with bacteria. This constant presence of bacteria contributes further to the chronic inflammation associated with COPD.

B. Airflow limitation & hyperinflation:-

- The major site of the air flow limitation is in the smaller conducting airway less than 2mm in diameter & is mainly due to airway remodeling .
- Other factor are inflammatory cells, mucous & plasma exudates in bronchi, smooth muscles contraction & dynamic hyperinflation during exercise.
- The latter is one of the major contributors to exercise limitation in these patient.
- Airflow limitation in COPD is best measured by spirometry which is key to diagnosis of the diseases.
- Airflow limitation is also worsened by a loss of the normal elastic recoil of the lung during exhalation.
- As a result, the patient is required to use abdominal and chest wall muscles to force air out of the lung, resulting in further collapse of airways and air trapping, which leads to thoracic hyperinflation.
- Hyperinflation of the lungs due to excess air trapping inhibits normal diaphragm contraction and reduces the efficiency of this primary muscle of ventilation, especially during exercise.

C. Gas exchange abnormalities:-

- These occur in advanced diseases & are characterized by arterial hypoxemia with or without hypercapnia.
- An abnormal distribution of ventilation perfusion ratio is the main mechanism of abnormal gas exchange in COPD.
- As COPD advances, gas exchange worsens, resulting in significant hypoxemia, which may require chronic supplemental oxygen.
- Some patients with severe disease develop hypercapnia, which affects the primary drive to breathe.
- In these patients, respiratory drive becomes less responsive to changes in arterial pH and $PaCO_2$ and hypoxic drive begins to play a larger role.
- Patients with chronic carbon dioxide retention tolerate the increased carbon dioxide concentrations and do not exhibit the signs of acute hypercapnia, including somnolence and altered mental status. Supplemental oxygen in these patients can result in an acute worsening of hypercapnia and acidosis, so it should be administered cautiously.
- However, concerns about oxygen supplementation causing narcosis should not prohibit its administration.

D. Pulmonary hypertension :-

- This occur late in course of COPD normally after the development of severe gas exchange abnormalities.
- Factor contributing to pulmonary hypertension in COPD include vasoconstriction, Endothelial dysfunction, destruction of pulmonary capillary etc .
- The combination of event may eventually lead to right ventricle hypertrophy & dysfunction.

E. Systemic effect :-COPD is associated with extra pulmonary effect including systemic inflammation & skeletal muscle wasting.

- The systemic effect contribute to limit the exercise capacity of these patient.

- **Pathophysiology of Exacerbations:**
- COPD is characterized by periodic episodes of worsening lung function and symptoms called *exacerbations*.
- Exacerbations have a significant impact on the morbidity and mortality of COPD.
- The inflammatory mediators that contribute to COPD play a role in exacerbations, although it appears that eosinophils are more prominent during exacerbations.
- Exacerbations of COPD typically have an infectious etiology, either viral or bacterial. Some estimates suggest that at least 50% of episodes are viral.
- Some patients' airways are chronically colonized with bacteria, which reduces the utility of sputum cultures in identifying a cause.
- *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Moraxella catarrhalis*, common colonizers of the upper airway, cause most of the bacterial respiratory infections.

- Mucus hypersecretion predisposes patients to repeated infections.
- Decreased removal of bronchial secretions physically impairs the defenses of the lungs against infection, and the mucus provides a good growth medium for bacteria.
- The bacteria, together with the host's immune responses, contribute to further lung tissue damage. During an exacerbation, there are increased symptoms, increased mucus production, and worsening of gas exchange and airflow obstruction.

Symptoms

TABLE 35.3 Components of Medical History for Patients with Chronic Obstructive Pulmonary Disease (COPD)

Evaluate the presence of symptoms:

- **Cough**
- **Sputum production**
- **Dyspnea**

Collect past medical history and review of systems:

- **History of asthma, allergies, or childhood respiratory infections**
- **Episodes of wheezing, chest pain, or morning headache**
- **Past history of COPD exacerbations**
- **Comorbidities related to cigarette smoking**
- **Unexplained weight loss**
- **History of depression or anxiety**
- **Family history of COPD**

Collect risk exposure history:

- **Cigarette smoking (past or present)**
- **Passive cigarette smoke exposure**
- **Occupational or environmental exposures**

Complication

- Irregular heat beat
- Need for breathing machine
- Right sided heart failure
- Pneumonia
- Severe weight loss
- Osteoporosis

Thank you