COPD PATHOPHYSIOLOGY

FLAME LECTURE: 15

MEHAN 10.28.18

LEARNING OBJECTIVES

- To understand the pathophysiology of COPD
- To be able to understand and explain to patients the cause of COPD
- Prerequisites:
- NONE
- See also – for closely related topics
- FLAME lectures on COPD diagnosis, treatment, and management of acute exacerbations

BACKGROUND

- Risk Factors
  - Age >35 with significant smoking history
  - Alpha-1 antitrypsin deficiency
  - History of exposure to noxious stimuli such as air pollution, occupational dusts or chemicals
- Signs & Symptoms
  - Cough, Wheezing, Shortness of Breath at rest or with exertion
- Diagnostic Confirmation
  - Spirometry (discussed in FLAME 16)

PATHOPHYSIOLOGY

- COPD is due to poorly reversible airflow obstruction which occurs 2/2 inflammatory responses from long term exposure to noxious stimuli, most often cigarette smoke
- All smokers have some degree of inflammatory response, however in patients with COPD, the inflammatory response is amplified
- The amplified inflammatory response results in pathological changes that cause:
  - Air trapping
  - Increased airway resistance
  - Increased lung compliance
  - Progressive airflow obstruction

PATHOLOGIC CHANGES

- Pathologic changes seen in COPD predominantly occur in the airways, however are also seen in the bronchioles and lung parenchyma
  - Chronic inflammation
  - Increased number of goblet cells, neutrophils, macrophages, CD8 T-lymphocytes
  - Mucus gland hyperplasia
  - Narrowing and reduction of small airways
  - Airway collapse due to alveolar wall destruction
  - Pulmonary artery vasoconstriction due to poor gas exchange/hypoxia

PATHOPHYSIOLOGY CONTINUED

- Noxious Agents → Increased activation of macrophages which trigger epithelial cells to release chemo-attractant factors that recruit neutrophils and CD 8+ lymphocytes
- Neutrophils release protease, which is an enzyme that breaks down protein, thereby causing alveolar wall destruction
- CD 8+ lymphocytes also cause alveolar wall destruction and activate fibroblasts → abnormal tissue repair/fibrosis
- Oxidative Stress from noxious stimuli → increased inflammatory mediators and mucous hypersecretion as well as inactivation of anti-proteases which exacerbates neutrophil-mediated damage
  - Alpha-1 Antitrypsin is an example of an anti-protease, which is why patients who have a severe deficiency have an increased risk of developing COPD

REFERENCES

1. Chronic obstructive pulmonary disease: Definition, clinical manifestations, diagnosis, and staging
2. Macnee W. Pathology, pathogenesis, and pathophysiology. BMJ 2006; 332(7551):1202-1204