Asthma

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Asthma

# Among many developed nations, the most commonly occurring disease in children is asthma (Cardon & Palmer, 2003). It is also the most serious disease in adults and about one in thirteen people in America suffer from asthma (CDC, 2019). According to Center for Disease Control and Prevention (CDC) (2018), asthma rates have been significantly rising since 1980s among individuals of all ages, sexes and ethnic groups. However, it was found to be more common in children and adult females compared to males (CDC, 2019). According to World Health Organization (WHO), it is estimated that asthma along with chronic obstructive pulmonary disease (COPD) will be leading cause of death by the year 2020 (Masoli, Fabian, Holt, Beasley, & Program, 2004).

Many medical, social and behavioral factors have been connected to development of asthma. A family history of asthma, tobacco smoke exposure, an allergy-linked condition and mother’s exposure to smoke during pregnancy increase the chances of developing the disease. This document will look at the pathophysiological mechanisms of chronic and acute asthma, the changes in arterial blood gas patterns during an exacerbation and the impact of unhealthy behaviour like smoking on pathophysiology of acute and chronic asthma.

# Chronic and acute asthma exacerbation

Asthma is an obstructive diseases caused by inflammation of airways including trachea, bronchi and bronchioles (Chung & Adcock, 2001). There is no general definition for exacerbation. The episodes of progressive symptoms of asthma are termed as asthma exacerbation. Acute asthmatic exacerbation refers to severe and sudden worsening of related symptoms whereas chronic asthma exacerbation means persistence of these symptoms over a period longer than three months (Chung & Adcock, 2001).

A variety of mediators have been found to have pathological effects on asthma (Barnes & Drazen, 2002). Leukotrienes, prostaglandins and histamines contract the smooth muscles of air passageway, cause leakage from micro vessels, increase mucous secretion and cause inflammation by attracting inflammatory cells (Busse, Banks-Schlegel, & Wenzel, 2000).

# Pathophysiological mechanisms of chronic asthma and acute asthma

Asthma ca be divided into two categories; atopic and non-atopic. Atopic is most common, extrinsic type of asthma caused by inflammation in response to IgE production which is triggered by the environment. Non-atopic, on the other hand is less common, intrinsic asthma caused by inflammation in response to local\self IgE.

## Pathophysiology

Comparing the normal bronchiole to asthmatic bronchiole reveals thickened basement membranes, increased mucous production and goblet cells, increased eosinophils in mucous and tissue. It is also characterized by increased mast cells causing higher histamine release. Increase in other cell types including neutrophils and T-helper cells. Hypertrophy, increase in size due to constriction, is also observed in smooth muscle cells.

Main cells/causative agents: IgE antibodies, eosinophils, dendritic cells and T-helper cells. IgE antibodies bind with receptors on mast cells forming mast cells-IgE complex, this complex recognize allergens and begin release of histamine. Two types: T-helper 1 normally found in lungs, in asthma T-helper2 are up regulated in asthmatics, more T-helper2 than 1. 1 normally promote inflammation by increasing cell mediated immunity, however T-helper 2 cells increase inflammation by increasing humoral immunity, promoting antibody production.

Because of these changes asthmatic cells show three characteristics 1. Airflow obstruction, 2. Bronchiole hyper responsiveness because of histamine release, 3. Inflammation due to increase in neutrophils and other immune cells.

## Mechanism

After an asthmatic person inhales an allergen, the allergen triggers a reaction. The allergen maybe engulfed by dendritic cells (DC) and activate the dendritic cells which is recognized by columnar epithelial cells which. The columnar epithelial cells release thymic stromal lymphocytes which will condition activated DC to produce chemokines to attract specifically T-helper 2 cells. Activated DC will activate t-helper cells to differentiate into T-helper 2 and will also secrete chemokines to attract the T-helper 2 to the bronchioles or the lungs. The activated T-helper 2 cells promote humoral immunity by stimulating plasma cells which will promote IgE production. IgE bind to mast cells to create IgE-mast cell complex. T-helper 2 also stimulate eosinophil production from bone marrows. Eosinophils amounts increase in the lungs through chemotactic activity. Inhaled allergen bind to IgE-mast cell complex and release histamine, prostaglandins and leukotrienes, which will stimulate smooth muscles of airways to pose constriction (bronchoconstriction). Every time the same type of allergen is inhaled, it triggers the whole process, hence leading to bronchoconstriction (Huether and McCance, 2015).

# Impact of smoking behavior on pathophysiology

The Institute of Medicine found relationship between tobacco use and exacerbations of asthma. Tobacco smoke is considered active/passive universal irritant. Irritants are non-allergic chemical substances causing inflammation or bronchospasms. Tobacco smoke causes irritation and infection to upper airway and lower respiratory tract, respectively. Cigarette, fireplaces, campfires and industrial pollutants are various sources of smoke leading to asthma symptoms.

Smoking causes corticosteroid resistance with higher production of neutrophils or lesser eosinophils. Smoke leads to shortness of breath, coughing, wheezing or a combination of all repeating at regular intervals which is called asthma exacerbations (Cardon & Palmer, 2003). Some other symptoms include swelling, itching or increased pulse rate.

## Treatment:

Budesonide inhalation is helpful in newly diagnosed patients reduce the exacerbation by fifty percent. Inhaled corticosteroids (ICS) do the same job in moderate doses. But in asthmatic patients with severe exacerbations, when ICS show no required results, budesonide is administered in high doses. In patients with a history of excessive exacerbations, a combination of budesonide and formoterol is used. Long-Acting Beta-agonists (LABA) and ICS together have proven to be beneficial in patients with severe disease symptoms. Other than the treatment strategies, some preventive measures have proven beneficial in reducing exacerbations. These preventive measures include

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