Students Name

Professor's Name

Course Code

Date Due

Asthma Disorder

Asthma is a chronic disorder affecting respiratory airways in the lungs. Usually, asthmatic patients suffer from inflammation of the lung airways cells (Busse & Herman 17). Asthma identification is commonly referred to as pathophysiology analysis (Bosco et al. 37). Airways inflammations occur when two or more mediators react with each other. Siddiqui et al. (31) asserts that lung inflammation causes bronchial hyperresponsiveness that leads to wheezing, dyspnea, chest tightening, and coughing. Weiss et al. (282) reports that lung inflammation stimulates hyperresponsiveness of the mucus glands and airways muscles. Health physicians use macrophages, lymphocytes, neutrophilis, and mast cells in pathophysiology analysis to identify lung inflammation (Martinez 41).

Hyperresponsiveness causes peeling of the mucus edema, epithelial cell wall, and hypersecretion of mucus (Bosco et al. 20). Pathophysiology researchers assert that edema mucus triggers plasma extravasation, airways venous dilation, and cell inflammation infiltration. Increased Inflammation causes contraction, hypertrophy, and hyperplasia of the smooth muscles of the airways (Weiss et al. 284). Airways inflammation, bronchoconstriction, or both characterize acute asthma in case lung inflammation persists. Persistent inflammation increases rate of gravid ventilation leading to high respiratory alkalinity (Hwang et al. 725).

Increased airways constriction reduces airflow and alveolus ventilation. The constrictions also increase carbon dioxide retention in the blood and hypoxemia of the arteries that further increase respiratory alkalinity (Busse & Herman 45). Further inflammation leads to imbalanced perfusion and

ventilation that leads to reduced or partial arterial oxygen pressure (pO2) in the blood (Bosco et al. 78). Respiratory scientists argue that if asthma is not treated at this stage, the airways constriction, partial arterial oxygen imbalance, and carbon dioxide gas exchanges is fatal (Hwang et al. 723).

Siddiqui et al. (30) defines Asthma etiology as a scientific classification of risk factors that cause Asthma both in children and adult. Etiology risk factors are classified into non-modifiable (intrinsic) and modifiable (extrinsic) and (Szefler 604). Modifiable asthmatic risk factors are caused by inhalation of various antigens and are responsible for childhood asthma. Antigens and antibodies cause hypersensitivity reactions in the mast cells of the respiratory duct. Examples of extrinsic factors include genetic asthma history, obesity, tobacco smoke, illnesses, external pollutants, and male sex (Bosco et al. 85).

Non-modifiable (intrinsic) etiological asthma factors cause adulthood asthma. Busse and Herman (43) report that intrinsic etiological factors lead to imbalanced autonomic nervous system (ANS) due to mismatch between alpha-adrenergic and ANS beta. Etiological experts assert that non-modifiable factors exhibit no hypersensitivity to the external antigens (Szefler 599). Airways hyper-reaction, lifestyle behaviors, work experiences, and female sex are the common modifiable risk factors (Weiss 280).

Modifiable and non-modifiable factors provides clinical manifestations framework that categorizes childhood and adulthood asthma conditions (Martinez 31). Research states that smoking or inhaling tobacco is the primary causes of childhood asthma. Health experts, therefore, advice mothers to avoid tobacco to prevent infecting their children with asthma (Busse & Herman 44).

On the other hand, adolescence and adulthood asthma is caused by risky lifestyle factors like professional smoking, exposures, and obesity (Hwang et al. 723). Adults can prevent this by eating balanced diet, avoiding smoking, and engaging in healthy occupational practices (Busse & Herman 41). There are various methods physicians use to diagnose Asthma in health centers. In Sputum

analysis method, asthmatic patient's sputum is characterized by Cursch-mann mucous, presence of eosiophilis debris, and purulent appearance (Martinez 32).

Hematologial asthmatic analysis establishes the presence of eosiophilis content for both modifiable and non-modifiable asthma conditions (Weiss 283). On the other hand, Pulmonary Function Testing analyses Forced Vital Capacity (FVC) status (Busse & Herman 45). Most asthmatic patients suffer from reduction of lung volume, total lung capacity, and lung conformity. Asthma can also diagnosed through chest x-ray test analysis. The infected people have their x-ray images with inflated lung airways (Hwang et al. 728). Finally, asthma can be tested through carbon dioxide blood test. Asthmatic victims exhibit (pCO2) content of more than 40 mm Hg. The condition is attributed to airways constriction due to muscles and epithelial walls inflammation (Bosco et al. 123).

Asthma may complicate into adverse health complications if not treated in early stages. Research shows that asthma complication in children and adult are almost similar (Weiss et al. 281). Common health complications are work or schools absenteeism, home confinement, permanent airways damage, and breathing difficulties (Siddiqui et al.133). In most cases, asthma complicates to pneumonia, finally death, wheezing, respiratory failure, lung, and asthmatics (Szefler 599)

Works Cited

Bosco, Anthony, et al. “Decreased activation of inflammatory networks during acute asthma exacerbations is associated with chronic airflow obstruction.” Mucosal immunology (2010). Print.

Busse, William W., and Herman Mitchell. “Addressing issues of asthma in inner-city children.” Journal of allergy and clinical immunology 119.1 (2007): 43-49. Print.

Hwang, Sohyun, et al. “A protein interaction network associated with asthma.” Journal of theoretical biology 252.4 (2008): 722-731. Print.

Martinez, Fernando D. “Asthma treatment and asthma prevention: a tale of 2 parallel pathways.” Journal of allergy and clinical immunology 119.1 (2007): 30-33. Print.

Siddiqui, Salman, Sherif Gonem, and Andrew J. Wardlaw. “Advances in the management of severe asthma.” Seminars in respiratory and critical care medicine. Vol. 33. No. 06. Thieme Medical Publishers, 2012. Print.

Szefler, Stanley J. “Asthma progression: Can we and should we measure it?.” Journal of Allergy and Clinical Immunology 121.3 (2008): 598-600. Print.