**Hayfever**
Is a local inflammatory response to allergens occurring in the upper respiratory tract.
Etiology: The trigger is most often encountered in the air passing through the upper respiratory tract (or it can be absorbed into the blood through the digestive tract). A weakened immune system due to dysbiosis makes it over active or hypersensitive reacting to otherwise unthreatening substances ie: pollens, cut grass, weeds, tree mould spores, dust, feathers, animal hair and dander.
Signs and Symptoms: seasonal illness marked by sneezing, sniffling, runny nose and itchy skin, itchy or watery eyes.
Pathophysiology: IgE is formed to the allergen. When the antibody binds to the allergen, mast cells attach to the IgE burst open releasing histamine causing mucous secretion, swelling and itching in the upper respiratory tract.
Recommendations: Text p. 246
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**Pneumonia**
Infection of the lung alveoli characterized by consolidation due to the outpouring of fluids into the alveolar spaces. The fluid accumulates, preventing the exchange of gases.
Etiology: bacterial infection (typically happens in immunosupressed people)
Signs and Symptoms: sudden onset of fever, shaking, chills and a sharp pleural pain on inspiration, breathing is shallow and rapid with cough with blood stained mucus.
Pathophysiology: Bacteria causes congestion of the alveoli, followed by a phase called red hepatisation which lasts up to four days.
Recommendations: Text p.249-250
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**Upper Respiratory Tract Infection**Any infection that affects the nasal passages, pharynx and bronchi commonly referred to as the common cold, viral sore throat, influenza, infantile croup and infantile acute bronchiolitis.
Etiology: viral, sometimes with secondary bacterial infections, adenovirus, echoviruses, rhinoviruses, parainfluenza, respiratory syncytial and coxsackie virus, bordetella pertussis, gram negative coccobacillus.
Signs and Symptoms: runny nose, congestion, postnasal drainage, cough, malaise and in some cases exacerbations of asthma.
Pathophysiology: distributed by sneezing, direct contact with nasal secretions. The contagious period is before the onset of symptoms (12-72 hours intubation).
Recommendations: Text p. 251-252
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**Asthma**Overresponsiveness to various stimuli results in episodic narrowing and inflammation of the airways.
Etiology: tobacco smoke and viral triggers are common triggers, air pollutants, allergens, dust, cold air, exercise, fumes or medicines, inflammation from dysbiosis & food sensitivies, research suggests that children exposed to anti pyretic medications in utero and or during their first two years of life have increased risk of allergies, asthma and eczema.
Signs and Symptoms: attacks characterized by tightness in the chest, difficulty breathing and especially exhaling, wheezing and coughing. As the tightness subsides, thick sputum is coughed up.
Pathophysiology: widespread bronchial obstruction due to bronchospasm (contraction in the bronchi), edema, swelling and plugging up of the passages with mucus. IgE mediated inflammation. Mucous plugs, fluid, thickening of the walls and the presence of many immune cells impeded normal respiration. Forced expiratory volume is reduced resulting in hyperinflation because it is difficult to exhale. Acidosis is created by retention of carbon dioxide. Sometimes the mucosal lining degenerates and sloughs off adding to the plugging. Mucous glands increase in number.
*Extrinsic*: Attacks triggered by external things (air pollutants, allergens, dust, dust mites, dander, animal fur, dust, mold, pollens etc.)
*Intrinsic*: Attacks as a result of things happening internally (upper respiratory infection, emotion, cold air, stress, chemicals and exercise but not by an allergen encountered by the lungs).
\* both as a result of dysbiosis and resulting weakened immune system and hypersensitivity
Recommendations: Text p. 254-256
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**Chronic Bronchitis and Emphysema**
Debilitating, progressive and potentially fatal lung diseases that have in common increased resistance to air movement , prolongation of the expiratory phase of respiration and the loss of the normal elasticity of the lung including chronic bronchitis and emphysema.
Etiology: chronic irritation of the lungs by dust, pollution or cigarettes, alpha-one antitrypsin deficiency
Signs and Symptoms:
Pathophysiology: chronic inflammation leads to damage to bronchiolar and alveolar walls with subsequent fibrosis, loss of elasticity and reduction of smooth muscle. Once the damage has occurred, the weakened tissue is forced out into expanded emphysematous spaces by the atmospheric pressure of incoming air.
Recommendations: Text p. 258-259
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