Medicine – Dr. Kawa – Lecture 1 – Asthma

Obstructive & Restrictive Pulmonary Diseases

Obstructive Pulmonary Disease

Indicate obstruction to flow of air *through the airways.*

As asthma , COPD (chronic bronchitis & emphysema) , Bronchiactasis , cystic fibrosis…

TLC (total lung capacity) increase

RV (residual volume) increase

FEV1 / VC less than 70%

Restrictive Pulmonary Disease

Indicate limitation to full expantion of the lungs because of diseases in the lung paranchyma , chest wall or diaphragm.

(Lung volumes are decrease but flow rates are normal).

As ***Interstitial lung disease****(cryptogenic fibsosing alveolitis , sarcoidosis, asbestosis, silicosis , coal worker pneumoconiosis…)*

TLC (total lung capacity) decrease

RV (residual volume) decrease

FEV1 / VC = or more than 70%

Pulmonary function test

Lung volumes

TLC (total lung capacity): is the volume of gas contained in the lungs after a maximal inspiration.

RV (residual volume): is the volume of gas remaining in the lungs at the end of a maximal expiration.

VC (vital capacity): is the volume of gas that exhaled from the lungs during expiration.

TLC = VC + RV

Lung volumes are ***increase*** *in obstructive lung diseases.*

Lung volumes are ***decrease*** *in restrictive lung diseases.*

Gas flow rate

FEV1(forced expiratory volume in the first second):

is the volume of gas exhaled during the first second of expiration.

FVC (forced vital capacity):

is the total volume of gas that exhaled from the lungs during expiration.

Flow rate ***decrease*** *in obstruction to air flow(obstructive lung disease)*

FEV1 / VC less than 70% (in Obstructive lung diseases).

FEV1 / VC = or more than 70%(in Restrictive lung diseases).

Obstructive Pulmonary Disease

ASTHMA

What is Asthma?

Chronic disease of the airways that may cause

Wheezing

Breathlessness

Chest tightness

Nighttime or early morning coughing

Episodes are usually associated with widespread, but variable, airflow obstruction within the lung that is often reversible either spontaneously or with treatment.

Bronchial asthma

Definition

It's paroxysmal reversible airway obstruction

characterized by airway inflammation & increased airway responsiveness to stimuli resulting in symptoms of ***wheeze*** *,* ***cough*** *,* ***dyspnoea*** *&* ***chest tightness*** *.*

Functionally characterized by airway obstruction which is variable over short periods of time or is reversible with treatment (the airway obstruction may be relieved spontaneously or with therapy) .

Increased responsiveness of the airways & reversable airflow obstruction are not unique to asthma .

Many patients with COPD exhibit nonspecific hyperresponsiveness, although obstruction is not completely reversible(partially reversible) .

Asthmatic Bronchitis : *some current or past cigarette smokers with chronic bronchitis & airflow obstruction exhibit episodic wheezing & SOB that closely mimic asthma.(a subcategory of chronic bronchitis that has features in common with asthma) .*

> *In persons older than 40 years who were newly diagnosed as having asthma , approximately one half had a history of cigarette smoking & had been previously diagnosed as having chronic bronchitis & emphysema ,these patients would have been more accurately diagnosed as having* ***Asthmatic Bronchitis.***

In persons older than 40 years with features of asthma who have never smoked cigarettes called ***adult – onset asthma****.*

Prevalence of asthma sssss:

Asthma is very commen ,about 4-5%of population have bronchial asthma .

The prevalence of asthma increased in the last century .

About 300 million people world-wide suffer from asthma ,

and an additional 100 million may be diagnosed with asthma by 2025 .

Asthma occur in all ages but predominantly in early life

(more common in children) .

In childhood , asthma is more common in boys (male :female 2:1), but following puberty females are more frequently affected (sex ratio equilized by the age of 30) .

Asthma prevalence is higher among

children than adults

boys than girls

women than men

Aetiology of asthma :

*The aetiology of asthma is complex and multiple **environmental** & **genetic** factors are implicated .*

*There are **genetic markers** on multiple chromosomes that relate to bronchial hyperresponsiveness & atopy.*

Risk Factors for Developing Asthma

- **Genetic characteristics**
- **Occupational exposures**
- **Environmental exposures**

Genetic characteristics

Occupational exposures

Environmental exposures

Factors may predispose to asthma :

*- Childhood **infection** eg.: respiratory syncytial virus .*

In patients with chronic asthma Mycoplasma & Chlamydia species have been identified in lung specimens , suggesting a possible role of infection in the pathogenesis .

*-**Allergen exposure** eg.: house dust mite .*

***-Indoor pollution**, warm, humid, centrally heated homes.*

*-Dietary **deficiency of antioxidants** .*

*-Exposure to **pets** in early life .*

***-Obesity** (gastroesophegeal reflux) .*

Factors may protect against asthma :

*-**Living on farm** (childhood exposure to antigen –rich environment is associated with a redused incidence of asthma & allergy by changes in the maturation process of the immune system.).*

*-predominance of **lactobacilli** in gut flora .*

-***Milk & antioxidants*** *such as vit. E.*

**According to the aetiology , we can divide asthma in to :*

Allergic asthma *: Atopy is the single largest risk factor for the development of asthma ,personal or family history of allergy ,increase IgE level ,positive skin reaction to intradermal injection of extracts antigens .*

Idiosyncratic asthma *: Non atopic ,no personal or family history of allergy .*

**Onset in early life : Strong allergic components .*

** Onset in late life : non allergic or have a mixed aetiology .*

Pathophysiology of asthma :

Asthma is multifactorial in origin arising from interaction of both genetic & environmental factors .

Airway inflammation occurs when genetically susceptible individuals are exposed to environmental factors .

**Cardinal pathophisiological features of asthma:*

1-Airwflow limitation:

usually reverse spontaneously or with treatment.

2-Airway hyper-responsiveness*:*

Exaggerated bronchoconstriction to a wide range of stimuli eg.: exercise, cold air…..

3-Airway inflammation *:*

Antigen-antibody reaction occur & leads to an inflammatory reaction in which several different cells are involved namely mast cells, macrophages & oesinophils which produce mediators such as histamine, prostaglandin & leukotriens .

These mediators (histamine, prostaglandin & leukotriens)

interact in a complex way resulting in ***bronchial hyperresponsiveness*** *which cause the following pathological changes in asthma :*

-Bronchial muscle spasm, smooth muscle hypertrophy & hyperplasia .

-Mucosal swelling (oedema).

-Hyperplasia of mucous glands with mucous plugging (viscid secretion).

-Thickened basement membrane .

-Epithelial damage .

-vasodilatation .

All these lead to airway obstruction, that is why asthma is not only bronchconstriction ,but also associated with inflammatory reaction.

Pathology of Asthma

With increasing severity & chronicity of asthma

*remodelling of the airways occur leading to **fibrosis of the airway wall,** fixed narrowing of the airways & a reduced response to bronchodilator medications .*

Pathogenesis of allergic asthma:

*Inhaled antigen is processed by **mucosal dendritic cells** & presented to **Tho-T cells**. This results in the generation of either **Th1** or **Th2** – T cells. With Th2 predominating in asthma .*

*Th2-T cells produces Interleukins **IL-4, IL-6** & **IL-13** which stimulate **B-cells** to produce **IgE** , which binds to **mast cells**.(IgE also bind to basophils & other cells.).*

*Inhaled antigen binds to IgE , stimulating the **mast cell** to degranulate , which in turn leads to the release of mediators of the immediate response & the late resonse (mediators like Histamine & Leukotrienes).*

***Histamine & Leukotrienes** produce bronchospasm & airway oedema.*

*Released chemotactic factors, along with factors from Th2 T-cells (**IL-3 , IL-5 & GM-CSF** " Granulocyte-Macrophage –colony – stimulating factor") facilitate **eosinophil** traffic from the bone marrow to the airway walls.*

These late responses lead to : excessive mucous production , airway wall inflammation & hyperresponsiveness.

[There are some notes in the 'Notes' sections of some slides in the PowerPoint. Return to the slideshow if interested.]