17/03/2020 Tuesday (Pathophysiology)

Myocardial Infraction (MI):- The term myo cardial infection indicates the development of an area of myocardial necrosis resulting from reduction in coronary blood flow to the segment of myocardium it supplies.

Accute MI, also known as Heart attack.

Causes:- MI cause by coronary artery thrombosis.

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- MI begins with in 20 to 30 minutes of the coronary artery occulsion.
- Lack of oxygen supply to the working myocardial.

Symptoms:-

Chest pain, which may radiate to the arm or jaw, Sweating
Nosia
Chest tightness
breathlessness

Diagnosis:- Electro Cardiogram (ECG) blood test for serum cardiac marker.

Treatment:- Aspirin, morphin, nitroglycerin heparin.

Pathophysiology:-

Atherosclerosis → obstruction

Arterial Spasm → Sudden reversible obstruction

Atherosclerosis + Plaque split + Thrombus → occulsion

→ Ischemia

↓

Hypoxia

↓

Reduced O_2 demand - Anging

↓

Thrombolysis → Unstable angina

↓

Permanent thrombus

↓

Necrosis → Myocardial Infraction.

Arteriosclerosis:- Hardening of arteries. Arteriosclerosis is generally term for thickning & loss of elasticity of arterial walls

Causes:- It is caused by:-

(i) High blood pressure.
(ii) Smoking.
(iii) High Cholestrol.

Symptoms:- Usually does not have any symptom s.-

Treatment:- The Goals of treatment:- lower the risk blood clot forming.

Atherosclerosis:- Is the condition in which an artery wall thickens as a result of a build up of fatty material such as cholestrol.
It is a type of blood vessel disorder.
Refered to as "Hardening of arteries"

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Etiology:- Large scale study of investigation on living population have proove a no. of riskfactors which are associated with increase risk of developing clinical atherosclerosis.

Measure ① Major Risk factor:- Hypertension, Diabetics smoking, age, sex, genetic factor familiar factor, dyslipidemia,

Emerging Risk factor:-
- Environmental factors
- Obesity
- Role of alcohol Infaction
- Physical inactivity.

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20/March/2020 10:45-11:45 FRIDAY SAHIL SHARMA

UNIT-II (6) Respiratory system Date / /

Asthma:- Asthma is a chronic air way disease. The air ways are tubes that carry air in and out of the lungs.

Asthma is a chronic inflamatory lung disease, which is characterised by air ways obstruction, infection and Hyper responsiness of tracheo bronchial smooth muscles to a variety of triggers which leads to increase mucus secretion, mucosal oedema & mucosal plugging.

In asthma, the inside wall of air ways are inflammed. The inflamation makes the air ways very sensitive and when they react strongly with allergic things, it leads to narrowing of air ways.

Etiology:-

(i) Ingestion aspirin.
(ii) Air pollution (iii) pulmonary infection (iv) Smoking.

Types of Asthma:- It is classified into major categories extrinsic ~~or~~ & intrinsic asthma

Extrinsic Asthma:- In which the asthmatic episode is induce by exposure to an extrinsic antigen.

Instrinsic Asthma:- This is most frequently triggered by respiratory tract infection.

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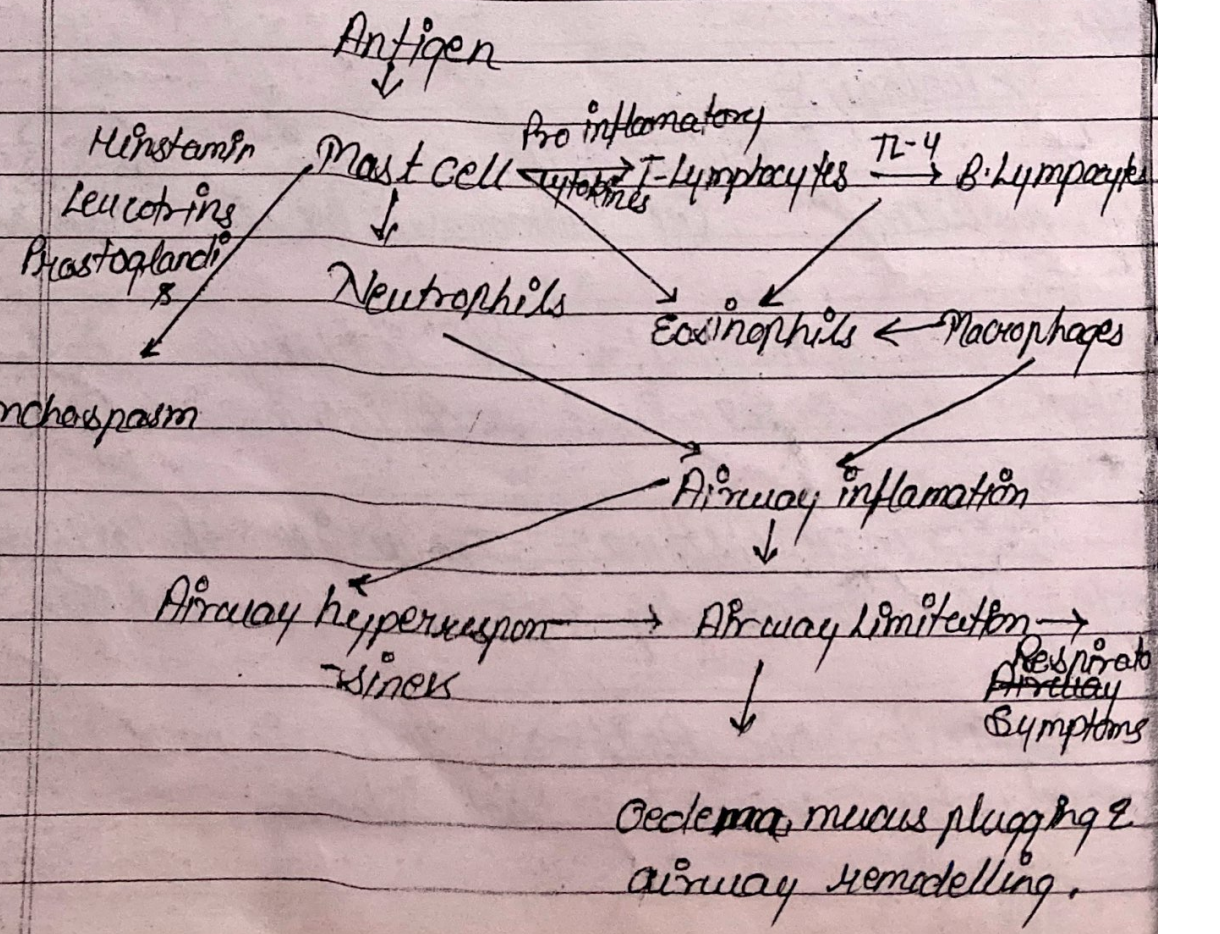
Pathophysiology :- Inhealed antigen activate mast cells in the airway.

They inturn induce the production of mediators of inflamation (histamine, leukotrins, prostaglandis) and cytokines includes, IL4 & IL5

Th5 travels to the bone marrow and cause terminal differentiation of eosinophils.

Circulating eosinophils enters the area of allergic inflamation and begins migrating to the lungs by rolling.

On activation the eosinophils release inflamatory mediators such as leukotrines & granularproteins to injured air way tissues.



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odema → excess of fluid

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Symptoms:- chest tightness, cough, troubles in breathing, wheezing.

Diagnosis:- Chest X-Ray,
Spirometry, histamine test
Sputum investigation

Treatment:- Environmental Control
Pharmacotherapy Education.

Pharmacotherapy:- β-agonist (Salbutamol)
(II) Anticholinergic - e.g Ipratropium bromide
(III) Anti inflamatory agent - eg:- corticosteroid
(IV) Mast cell stabilizers - eg:- ketotifen
(V) Leukotrins modifier - eg:- Montelucast & Zafirlucast

Chronic obstructive Airway disease:- (COAD) OR Chronic obstructive pulmonary disease (COPD):-
COPD is chronic, partial or complete obstruction to the air flow at any level from trachea to the smallest air way resulting in functional disability of lungs.
Chronic airflow obstruction due to chronic bronchitis and pulmonary emphysema.

Chronic bronchitis:- Chronic bronchitis is defined persistant cough and sputum production for atleast three months of the year for two

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or more consecutive year.
The cough is caused by over secretion of mucus.

Etiology:- The two most imp. Etiologic factor responsible of for majority of cases-
(i) Cigarette smoking
(ii) Atmospheric pollution.
(iv) Other Contributory factors are- •occupation •familiar •infection & •genitic factors.

Pathogenesis:-

Irritant
↓
Induce Hyper secretion of bronchial mucus gland
↓ Cause
Hypertrophy of mucus gland
↓
Formation of mucin-secreating goblak cells in the surface of epithelium of bronchi

They also cause inflamation with infiltration of T-cells, macrophages & neutrophills.

Clinical features:→ Some imp. features of bronchitis are-
(1) Persistant Cough with expectoration of long duration

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(II) Patient are called blue bloaters cyanosis and oedema
(III) Features of Right heart failure are common.
(IV) Chest X-Ray show enlarge heart with prominent vessels. (specific)

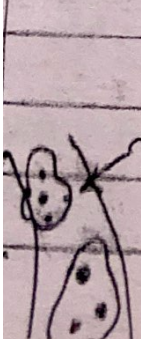
Emphysema :- Is characterised by permanent enlargement of air space due to the destruction of their walls.

Emphysema is defined morphologically, while chronic bronchitis is defined as clinically.

Etiology :- Tobacco, smoke, air pollutants occupational exposures infection -, genetic influence.

Clinical features :- Case of predominant Emphysema develop clinical features after about ½ of the pulmonary parenchema is damage.
The following features is generally characterised -
Weight loss, Cough, dyspnea.
Patients are called pink puffers.
Chest X-Rays shows small heart.

Pathogenesis :-



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TNF - Tumor Necrosis Factor

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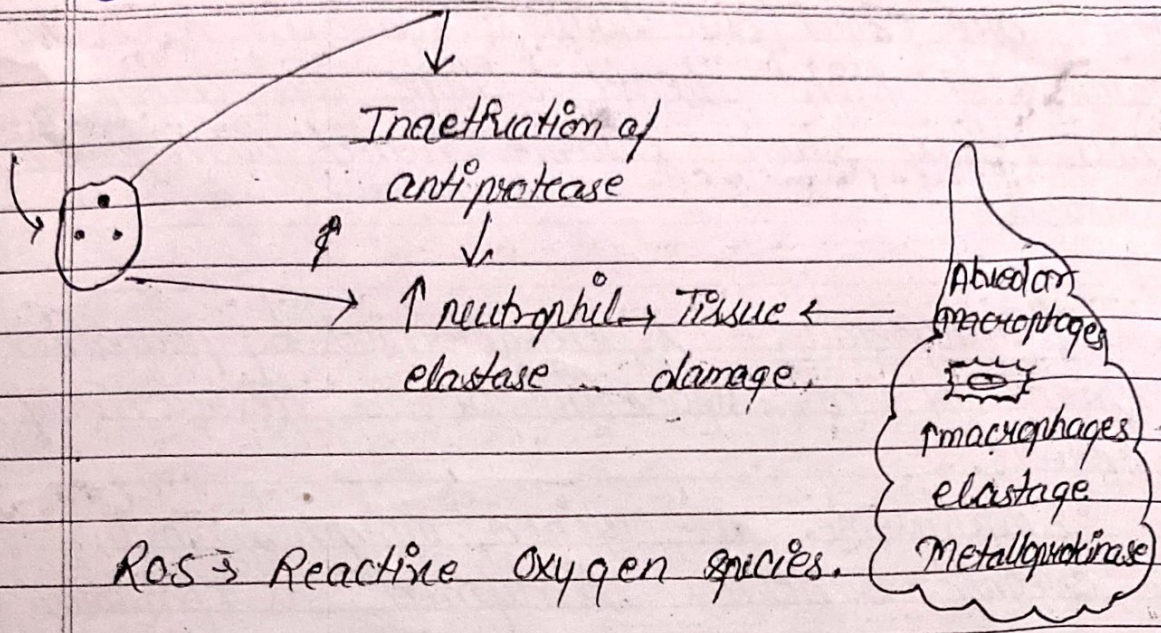

ROS → Reactive Oxygen species.

Nicotine or ROS activate IL-8 and TNF (Tumor Necrosis factor) These Interns attract & activate neutrophils. Accumulated neutrophils activated & release their granules, Rich in a variety of cellular protease (Neutrophil elastase), ~~measure~~ resulting in tissue damage.

Smoking also enhance elastase activity in macrophages elastage is not inhibited by α_1 antitrypsin & can proteolytically digest these antiprotease. Thus metalloproteinase derived from macrophages & neutrophils have a role in tissue disruption.

Diagnosis of COPD :→ Spirometry, chest X-Ray & pulmonary function test

Treatment :→ Quiet smoking, pharmacological treatment. (ex Ipratropium bromide, Salbutamol, aminophyllin, beclomethasone, prednisonolne, bromhexine)

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(7) 26/03/2022 Friday

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Pathophysiology

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Acute Renal failure :-

Acute failure Disease is syndrom characterised by rapid onset renal disfun-ction chefly oligue urea production of abnormaly small amount of urine or an urea failure of kidney to produce urine and sudden increase in metabolic waste product urea in the blood which constiquent development of uraemia (raised level in the blood of urea).

*Etiology :- The causes of acute Renal failure are classified as -

① Pre-Renal :- oligue urea result from reduced GFR.

② Renal :- Damage to the kidney itself due to the acute tubular necrosis (ATN), Glomerular nephritis.

③ Post renal :- obstruction to the carriflow of urine.

*Sign & symptoms :- Increase in level of nitrogeneous base in blood initially produce symptoms like fever.

② More severe and produce the symptoms of altered mental status.

* Pathophysiology :- The primary event in the development of acute Renal failure is constriction of apparent artids and dialation of efferent. This changes in artisols reduce the glomerular rate and therefore formation of urine decrease by the Kidney. The destruction of there function precipitate acute Renal failure.

Chronic Renal failure :-

It is characterised by progressive and irreversible deterodation of renal function due to slow destruction of renal parenchyma Eventually terminating in death when sufficient no. of

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nephron have been damage.

* Sign & symptom :- ① Polyurea ② Fluid Accumulation produce symptoms of edema.

* Pathophysiology: ① Chronic Heart failure ② Hypertension ③ Hypermagnesimia reverse in Bladder, muscle, weakness and coma.

④ Ammonia formation from urea in GPT is responsible for unpleasent taste, Nosea and vomiting.

⑤ Calcium deposition in skin contributive to severe itching in eyes.

* The main causes of chronic Renal failure is Chronic glomerular.

Damage to tubular interstial tissue results in ulteration in reabsorption and secretion of Important constituents leading to excretion of large volume of dilute urin.

Treatment: ① Dsolysis

② Kidney transport.

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