Lecture one
lung pathology
4th year MBBS

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Expectation at end of lecture

- Brief review of anatomy and physiology related to lungs
- Atelectasis
- Pulmonary edema
  - Cardogenic pulmonary edema
  - Non cardiogenic pulmonary edema
- Important questions
• REVIEW OF ANATOMY AND PHYSIOLOGY RELATED TO LUNGS
Lungs

• The major function of the lung is to excrete carbon dioxide from blood and replenish oxygen.
  – Trachea
  – Bronchi & Bronchiole
  – Acinus
  – Alveoli
Respiratory system
review of anatomy
Alveoli and capillaries junction
Alveoli and capillaries junction with surfactants

Structure of an Alveolus

- Oxygen rich blood
- Oxygen poor blood
- Capillary
- Fluid with surfactant
- Type II cell
- Type I cell
- Alveolar macrophage
- Respiratory membrane
- $O_2$, $CO_2$
Alveoli and capillaries junction

• Muco cilliary apparatus
  – Cilia beat in rythmic fashion from alveoli to proximal trachea
  – Remove mucous and debris containing bacteria
  – Mucous is then swallowed or coughed out of body
Bronchial muscles

- Smooth muscle contract in response to emotion and parasympathetic activity ....bronchoconstriction

- Smooth muscles relax in response to sympathetic activity
DIAPHRAGM

• Muscular layer separating lung from abdomen
• Major Muscle of inspiration
• Nerve supplies C3, C4 and C 5
DIAPHRAGM

breathing in

increase in volume means lower pressure

ribs move up and out
diaphragm flattens
volume of chest increases

breathing out

decrease in volume means higher pressure

ribs fall
diaphragm moves up
volume of chest decreases

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Common developmental defects in lung

- Agenesis or hypoplasia of both lungs, one lung, or single lobes
  - Tracheal and bronchial anomalies (atresia, stenosis, tracheoesophageal fistula)
- Vascular anomalies
- Congenital lobar overinflation (emphysema)
- Foregut cysts
- Congenital pulmonary airway malformation
Now pathology starts

ATELECTASIS
ATELECTASIS

• an area of collapsed or nonexpanded lung.
• predispose for infection due to decreased mucociliary clearance.
ATELECTASIS

1. Obstruction/resorption atelectasis

– collapse of lung due to resorption of air distal to an obstruction;

– aspiration of a foreign body, chronic obstructive pulmonary disease (COPD), and postoperative atelectasis.
Atelectasis

2. Compression atelectasis
   – atelectasis due to fluid, air, blood, or tumor in the pleural space.

3. Contraction (scar) atelectasis
   – is due to fibrosis and scarring of the lung.

4. Patchy atelectasis is due to a lack of surfactant, as occurs in hyaline membrane disease of newborn or acute (adult) respiratory distress syndrome (ARDS).
• Pulmonary edema
Pulmonary edema

- It is collection of fluid in lung tissue.
Pulmonary edema

• It is collection of fluid in lung tissue.

❖ Causes
  ❖ cardiogenic pulmonary edema
  ❖ Non cardiogenic pulmonary edema
Causes OF pulmonary edema
Pulmonary Edema types cardiogenic

- Increased hydrostatic pressure
  - RAISED PAP AND LVEDP > 18
  - (increased pulmonary venous pressure)
- Left-sided heart failure (common)
- Volume overload
- Pulmonary vein obstruction
Pulmonary edema types

- **EDEMA DUE TO MICROVASCULAR INJURY** (ALVEOLAR INJURY)
- **Infections**: pneumonia, septicemia  
  Inhaled gases: oxygen, smoke
- **Liquid aspiration**: gastric contents, near-drowning
- **Drugs and chemicals**: chemotherapeutic agents (bleomycin), other medications (amphotericin B), heroin, kerosene, paraquat  
  Shock, trauma  
  Radiation  
  Transfusion related
Pulmonary edema types undetermined origin

• EDEMA OF UNDETERMINED ORIGIN
• High altitude
• Neurogenic (central nervous system trauma)
• Cardiogenic pulmonary edema
Cardiogenic pulmonary edema

- Due to left ventricular failure due to IHD and fluid overload
- Pulmonary artery pressure are high > 18mmHg
- Initially basal edema then goes upward..
- Heart failure cell (macrophages filled with hemosiderin)
- Fibrosis and thickening of alveolar walls and septa in long standing cases...
- Brown induction
- Engorged capillaries
Carcinogenic pulmonary edema
• NON CARDIOGENIC PULMONARY EDEMA
  – ARDS and ALI

abrupt onset of significant hypoxemia and diffuse pulmonary infiltrates in the absence of cardiac failure.
NON CARDIOGENIC PULMONARY EDEMA

- Accumulation of fluid inside lung parenchyma due to damage to alveolar walls.
- Fluid from alveolar enter into peri-alveolar tissue and from blood into alveoli.
Acute Respiratory Distress Syndrome

• **INFECTION**
  – Sepsis
  – Pneumonia ...Viral, Mycoplasma, and Pneumocystis pneumonia; miliary tuberculosis
  Gastric aspiration

• **PHYSICAL/INJURY**
  – Mechanical trauma, including head injuries
  – Pulmonary contusions Near-drowning
  – Fractures with fat embolism
  – Burns Ionizing radiation
Acute Respiratory Distress Syndrome

• INHALED IRRITANTS
  – Oxygen toxicity  Smoke  Irritant gases and chemicals

• CHEMICAL INJURY
  – Heroin or methadone overdose  Acetylsalicylic acid  Barbiturate overdose  Paraquat

• HEMATOLOGIC CONDITIONS
  – Multiple transfusions
  – Disseminated intravascular coagulation

• PANCREATITIS  UREMIA  CARDIOPULMONARY BYPASS  HYPERSENSITIVITY REACTIONS
Acute Respiratory Distress Syndrome

• Pathogenesis

  – Damage occurs to alveolar capillary membrane
  – Increased vascular permeability and alveolar flooding, loss of diffusion capacity, and widespread surfactant abnormalities caused by damage to type II pneumocytes.
  – Endothelial injury also triggers the formation of microthrombi that add the insult of ischemic injury
  – Hyaline membranes result from inspissation of protein rich edema fluid that entraps debris of dead alveolar epithelial cells
FIGURE 15-4 The normal alveolus (left side) compared with the injured alveolus in the early phase of acute lung injury and acute respiratory distress syndrome. Pro-inflammatory cytokines such as interleukin 8 (IL-8), interleukin 1 (IL-1), and tumor necrosis factor (TNF) (released by macrophages), cause neutrophils to adhere to pulmonary capillaries and extravasate into the alveolar space, where they undergo activation. Activated neutrophils
Acute respiratory distress syndrome mediator

- **Proinflammatory**
  - Interleukin -1 & 8
  - Platelet activating factor PAF
  - *nuclear factor κB* (NF-κB),
  - TNF
  - Tissue factor
  - Protein C decreased
  - Thrombin
  - Neutrophils
    - Neutrophilic accumulation in lung
    - Increased oxidative damage

- **Anti-inflammatory**
  - Interleukin 10, antioxidants and antiproteases
Acute respiratory distress system

Morphology.

• **Proliferative stage /Acute stage**
  – the lungs are heavy, firm, red, and boggy.
  – They exhibit congestion, interstitial and intra-alveolar edema, inflammation, fibrin deposition, and diffuse alveolar damage.
  – The alveolar walls become lined with waxy hyaline membranes.
Acute Respiratory Distress Syndrome
hyaline membrane
Acute respiratory distress syndrome morphology and staging

• **In the organizing stage,**
  – type II pneumocytes undergo proliferation, and there is a granulation tissue response in the alveolar walls and in the alveolar spaces.

• **Fibrotic stage**
  – fibrotic thickening of the alveolar septa ensues, caused by proliferation of interstitial cells and deposition of collagen
Acute respiratory distress syndrome
Clinical presentation

• Dyspnea shortness of breath, hypoxia and acute respiratory failure and right heart failure

• **Outcome** most resolve by the end of 3\textsuperscript{rd} week while few undergo fibrotic stage and long term dependency on ventilator.

• high mortality unless supportive care with lung protective strategies.
• What is the function of nose?
• What are the functions of mucocilliary apparatus?
• What are the constituent of alveolar capillary membrane?
• What is ALI and ARDS?
• What are the causes of cardiogenic pulmonary edema?
• What are the causes of non cardiogenic pulmonary edema?
Reference

• Robin basic pathology 9\textsuperscript{th} edition
• ARDS group network website
• Kaplan pathology
GET UP AS LECTURE HAS ENDED