Objectives

To know the following: ARDS, Occupational and Environmental lung disease.

Epidemiology
Etiology
Pathogenesis
Clinical presentation
Diagnosis
Treatment
Complication and prognosis

Acute respiratory distress syndrome (ARDS)

- This describes the acute, diffuse pulmonary inflammatory response to either direct (via airway or chest trauma) or indirect (blood-borne) insults that originate from extrapulmonary pathology.

The criteria defining ARDS are:

- Hypoxaemia.
- Chest X-ray showing diffuse bilateral infiltrates.
- Absence of a raised left atrial pressure.
- Impaired lung compliance.

Berlin definition of ARDS

- Onset within 1 week.
  - Respiratory failure not fully explained by cardiac failure or fluid overload.
  - Objective assessment (e.g. by echocardiography).

- Severity according to Impaired oxygenation:
  Mild
  Moderate:
  Severe:
It is characterised by:

- neutrophil sequestration in pulmonary capillaries
- increased capillary permeability
- protein-rich pulmonary oedema with hyaline membrane formation
- damage to type 2 pneumocytes leading to surfactant depletion
- alveolar collapse
- reduction in lung compliance
- If the early phase does not resolve with treatment of the underlying cause.
- Late: fibroproliferative phase ensues and causes progressive pulmonary fibrosis.
- It is frequently associated with other organ dysfunction as part of MOF.

**Conditions predisposing to ARDS**

- **Inhalation (direct)**
  - Aspiration of gastric contents
  - Toxic gases/burn injury
  - Pneumonia
  - Blunt chest trauma
  - Near-drowning

- **Blood-borne (indirect)**
  - Sepsis
  - Necrotic tissue (particularly bowel)
  - Multiple trauma
  - Pancreatitis
  - Cardiopulmonary bypass
  - Severe burns
  - Drugs (heroin, barbiturates, thiazides)
• Major transfusion reaction
• Anaphylaxis (wasp or bee stings, snake venom)
• Fat embolism
• Carcinomatosis
• Obstetric crises (amniotic fluid embolus, eclampsia)

Chest X-ray in acute respiratory distress syndrome (ARDS). Note bilateral lung infiltrates, pneumomediastinum, pneumothoraces with bilateral chest drains, surgical emphysema, and fractures of the ribs, right clavicle and left scapula

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**OCCUPATIONAL LUNG DISEASE**

Exposure to dusts, gases, vapours and fumes at work can lead to the following types of lung disease:

1. **Acute bronchitis and pulmonary oedema** from irritants such as Sulphur dioxide, chlorine, ammonia or oxides of nitrogen

2. **Pulmonary fibrosis** from inhalation of inorganic dust, e.g. coal, silica, asbestos, iron and tin
3. **Occupational asthma** – this is the commonest industrial lung disease in the developed world.

4. **Hypersensitivity pneumonitis**

5. **Bronchial carcinoma** due to asbestos, polycyclic hydrocarbons and radon in mines.

**Occupational ASTHMA**

<table>
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<th>Most frequently reported causative agents</th>
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<td>• Isocyanates</td>
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<td>• Flour and grain dust</td>
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<tr>
<td>• Colophony and fluxes</td>
</tr>
<tr>
<td>• Latex</td>
</tr>
<tr>
<td>• Animals</td>
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<tr>
<td>• Aldehydes</td>
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<td>• Wood dust</td>
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<th>Workers most commonly reported to occupational asthma schemes</th>
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<tr>
<td>• Paint sprayers</td>
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<tr>
<td>• Bakers and pastry-makers</td>
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<tr>
<td>• Nurses</td>
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<td>• Chemical workers</td>
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• **Byssinosis** occurs in workers of cotton and flax mills exposed to cotton brack (dried leaf and plant debris)

• **Reactive airways dysfunction** syndrome or acute irritant-induced asthma refers to the development of a persistent asthma-like syndrome following the inhalation of an airway irritant:

**II-Pneumoconiosis**

**Coal worker’s pneumoconiosis:**

Cause: prolonged inhalation of coal dust causing pulmonary fibrosis through aggregation of alveolar macrophages.

- **Classification:**
  - **A/ Simple CWP:** asymptomatic.
  - **B/ Progressive massive fibrosis:**
III—Lung diseases due to organic dusts:

- A wide range of organic agents may cause respiratory disorders.
- Inhalation of organic dust leading to type III or IV diffuse immune complex reaction within the alveolar & bronchial walls.

**Hypersensitivity pneumonitis (extrinsic allergic alveolitis)**

- farmers (farmer’s lung)
- pigeon fanciers (bird fancier’s lung) with symptoms related to the extent of exposure to the triggering organic dusts.
- Symptoms either acute as influenza-like syndrome or chronic with progressive pulmonary fibrosis.
Hypersensitivity pneumonitis

**Clinical predictors:**

- Exposure to a known offending antigen
  - Positive precipitating antibodies to offending antigen
  - Recurrent episodes of symptoms
  - Inspiratory crackles on examination
  - Symptoms occurring 4–8 hours after exposure
  - Weight loss
Investigations:

CXR-

HRCT-.

Pulmonary function test- restrictive defect,

Serology-.

Management:

1. Avoid exposure to possible triggers.
2. Wearing masks.
3. Glucocorticoids.
4. O2 therapy.

Complications:

- Pulmonary fibrosis,
- Respiratory failure,
- Pulmonary hypertension
- Right side heart failure.

Case-studies

CASE NO 1

A 65-year-old man presents with progressive shortness of breath. On examination he is found to have fine crackles in both lung bases and oxygen saturations of 93% on room air.

Case 2

A diagnosis of idiopathic pulmonary fibrosis is suspected. Which one of the following chest x-ray findings develops first in patients with idiopathic pulmonary fibrosis?

A/ Asymmetrical upper zone 'ground-glass' changes

B/ Small, peripheral opacities in the lower zones

C/ Perihilar horizontal septal lines

D/ Honeycombing

E/ Loss of left heart border

CASE 3
On examination, he is comfortable at rest with oxygen saturations 95% on air. There is no evidence of lymphadenopathy, clubbing or cyanosis. He has fine crackles at both lung bases that do not alter on coughing.

Which of the following investigation findings would support a diagnosis of idiopathic pulmonary fibrosis?

A/ Reticular changes on CT imaging that is worse at the bases
B/ Obstructive picture on spirometry
C/ Extensive ground glass opacities on CT imaging
D/ Increased transfer factor on spirometry
E/ A lymphocytosis on bronchoalveolar lavage

CASE 4

A 68-year-old male patient presents with a 6-month history of shortness of breath that is worse on exertion with reduced exercise tolerance & no associated wheeze, or haemoptysis but does have a dry cough.

He has hypertension and takes amlodipine 5mg once a day. He has never smoked or worked with asbestos.