PULMONARY OEDEMA

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EN to SN Conversion Course
Can be due to:

- Hydrostatic (Pressure Related)
- Increased Permiablity
- A combination of both
Types of Oedema

- HYDROSTATIC – known as Cardiogenic
  Occurs in the presence of elevated left ventricular filling pressure

- PERMIABILTIY – Non Cardiogenic
  Oedema occurs in the absence of elevated left ventricular pressures
Pulmonary Circulation
Alveolar Unit
Common Causes of P.O.

- Congestive Heart Failure
- Mitral Stenosis (leading to left heart failure)
- Cor Pulmonale (Pulmonary Heart Disease)
- Myocardial Infarction
Common Causes of Non Cardiogenic P.O.

- **A.R.D.S.** — The presence of proteins in the interstitial spaces reverse the oncotic pressure gradient and draws water from the capillaries

- Smoke inhalation burns
In both types of Oedema:

- Excess Extra vascular water fills the interstitium.

- Interstitial lymphatics located around the bronchi and the branches of the pulmonary arteries are unable to drain excess water.
The alveolar spaces become flooded

The alveoli become unable to participate in gas exchange due to ventilation/perfusion (V/Q) mismatch.

This gives a picture of acute pulmonary oedema.
Formation of Tissue Fluid

Venous End

Arterial End

B.P. 12mmHg

B.P. 32mmHg

Osmotic Pressure 25mmHg

Osmotic Pressure 25mmHg
Capillary Fluid Exchange

B.P. 32mm Hg

B.P. 25mm Hg

B.P. 12mm Hg
Capillary Fluid Exchange

\[ NDF = (P_C - P_T) - \sigma (\pi_C - \pi_T) \]

When \( NDF > 0 \) → Filtration
When \( NDF < 0 \) → Reabsorption

Hydrostatic (\( P \)) and oncotic (\( \pi \)) pressures within the capillary and tissue interstitium (\( T \)) determine the net driving force (NDF) for fluid movement into the capillary (reabsorption) or out of the capillary (filtration). The oncotic pressure difference is multiplied by the reflection coefficient (\( \sigma \)) that represents the permeability of the capillary barrier to the proteins responsible for generating the oncotic pressure.
Signs & Symptoms

- The onset is usually sudden – require immediate medical & nursing attention.

- The sudden V/Q mismatch causes intense dyspnoea.

- Patient becomes very anxious and frightened.
Respirations are noisy due to presence of secretions in the larynx and trachea (rattling sound).

Skin becomes moist, cold and clammy.

Cyanosis develops rapidly (late stage of respiratory failure).
A cough develops with copious, frothy and blood-stained sputum.

On auscultation, crepitations are heard throughout the chest.

A chest X-ray usually shows the classical bat like picture of the lungs.
Acute P.O.
Treatment

- To calm the patient and relieve anxiety, morphine is used. This will also dilate the peripheral circulation therefore reducing left ventricular pressure during diastole.

- The patient must never be left alone during the attack.
Vital signs recording.

Diuretics are very effective to get rid of excess fluid.

Careful monitoring of fluid balance and electrolyte levels.

Bronchodilators are useful to ease bronchospasm and facilitate bronchial toilet.
Treatment (Cont…..)

- Nursing the patient upright in bed increases the vital capacity of the lungs.

- High concentration of Oxygen to relieve cyanosis.

- Intubation and mechanical ventilation may be required if the condition worsens.
Treatment (Cont.....)

- If ventilated P.E.E.P. sometimes helps to prevent further water leak into the alveoli.

- Identify and treat the cause.

- Haemodynamic (B.P. and P.A.W.P.) monitoring and ABGs are useful guides during the management of artificial ventilation.
REMEMBER

PULMONARY OEDEMA

is a

CONDITION

not a DISEASE