

## Presentation of acute pulmonary oedema

### Definition

- Acute pulmonary oedema: Accumulation of fluid in the lung parenchyma leading to impaired gas exchange between the air in the alveoli and pulmonary capillaries.
- Cardiac failure: clinical syndrome where the heart is unable to function adequately as a pump to meet the demands of the body; characterised by:
  - Typical symptoms, including dyspnoea, orthopnoea, ankle swelling
  - Typical signs, including bibasal crepitations, raised jugular venous pressure (JVP)
  - Objective evidence of a structural or functional abnormality including cardiomegaly, third heart sound, abnormality on echocardiogram (echo)
- Left ventricular failure (LVF): failure of the LV causing congestion of the pulmonary veins
- Right ventricular failure (RVF): failure of the RV causing congestion of the systemic veins
- Congestive cardiac failure (CCF): LVF and RVF co-exist, the latter usually secondary to the former
- Cor pulmonale: RVF secondary to chronic lung pathology eg chronic obstructive pulmonary disease (COPD)
- Low output cardiac failure: cardiac failure secondary to inadequate pumping and supply
- High output cardiac failure: cardiac failure secondary to excessive demand
- Acute pulmonary oedema: rapid accumulation of fluid in the alveoli and parenchyma of the lung
  - Cardiogenic pulmonary oedema: caused by elevated pulmonary capillary pressure due to decompensated LVF
  - Non-cardiogenic: caused by injury to the lung parenchyma or vasculature

### Causes of acute pulmonary oedema

- Cardiac
  - Acute coronary syndrome (ACS)
  - Cardiac arrhythmia e.g. atrial fibrillation (AF)
  - Valvular heart disease
  - Hypertension
  - Cardiomyopathy
  - Cardiac tamponade
- Non-cardiac
  - Non-compliance with medication
  - Negatively inotropic medication
  - Fluid overload
  - High output cardiac failure
    - Anaemia
    - Thyrotoxicosis
    - Sepsis
  - Acute respiratory distress syndrome (ARDS)
  - Renal artery stenosis (RAS)

### Pathophysiology of acute pulmonary oedema

- Blood pressure (BP) = cardiac output (CO) x systemic vascular resistance (SVR)
- CO = heart rate (HR) x stroke volume (SV)
- SV depends on preload, myocardial contractility and afterload
- As the heart begins to fail, compensatory mechanisms maintain CO & BP
- Increased sympathetic tone increases SVR and stimulates renin secretion
- The renin-angiotensin-aldosterone system (RAAS) increases salt and fluid retention, which initially increases preload, end diastolic volume (EDV), SV and therefore CO via the Frank-Starling mechanism, but over time leads to cardiac dilatation and a reduction in contractility and CO, together with congestion of the pulmonary and systemic veins with associated tissue oedema
- As alveolar oedema increases, diffusion of oxygen into the pulmonary capillaries is impaired, which manifests as dyspnoea; venous return to the already congested heart and lungs increases when the patient lies flat, which manifests as orthopnoea and paroxysmal nocturnal dyspnoea (PND)
- Acute pulmonary oedema can be precipitated by sudden increases in preload (volume overload or fluid retention), decreases in contractility (ischaemia, infarction, arrhythmia, valvular failure, cardiomyopathy, drugs), increases in afterload (systemic or pulmonary hypertension) or direct damage to the lungs themselves

### History in acute pulmonary oedema

- Fatigue
- Worsening dyspnoea progressing from an exercise tolerance of dyspnoea on exertion to at rest
- Orthopnoea
- PND
- Cough productive of pink, frothy sputum
- Ankle swelling

### Examination in acute pulmonary oedema

- LVF
  - Respiratory distress
  - Tachypnoea
  - Bibasal crepitations
  - Cardiac wheeze
  - Tachycardia
  - Displaced apex beat
  - Third heart sound
- RVF
  - RV heave
  - Raised JVP
  - Hepatomegaly
  - Peripheral oedema
- Patients with acute pulmonary oedema are likely to have features of LVF and RVF simultaneously

### Initial investigation of acute pulmonary oedema

- Arterial blood gas (ABG)
- Full blood count
- Urea & electrolytes
- Magnesium
- Calcium
- Thyroid function tests
- 12 lead ECG. Unlikely to be normal; may show:
  - Ischaemia
  - Infarction
  - Left ventricular hypertrophy (LVH)
  - Arrhythmia
- Chest radiograph (CXR) – ABCDE mnemonic
  - **A**lveolar oedema
  - **B**ats wing hilar shadowing and Kerley B lines
  - **C**ardiomegaly
  - **D**iversion to the upper lobes (distension of upper pulmonary veins)
  - **E**ffusions: blunting of the costophrenic angles

### Further investigation of acute pulmonary oedema:

- Echocardiogram (echo)

### Initial management of acute pulmonary oedema

- Assess the patient from an ABCDE perspective
- Sit patient upright
- Maintain a patent airway: use manoeuvres, adjuncts, supraglottic or definitive airways as indicated and suction any sputum or secretions
- Deliver high flow oxygen 15L/min via reservoir mask and titrate to achieve oxygen saturations ( $S_pO_2$ ) 94-98% or 88-92% if known to have COPD
- Attach monitoring
  - Pulse oximetry
  - Non-invasive blood pressure
  - Three-lead cardiac monitoring
- Request 12 lead ECG and portable CXR
- Obtain intravenous (IV) access and take bloods
- Perform ABG sampling
- Give furosemide 40 mg IV [to diurese]
- Give morphine 2.5-10 mg IV [to dilate venous system, decreasing preload and also improve breathing symptomatically]
- Commence glyceryl trinitrate (GTN) 1mg/ml IV infusion at 2 ml/hour and titrate upwards maintaining SBP >90 mmHg; if there is any delay in gaining IV access, 2 puffs of GTN can initially be given sublingually
- If severe, or if inadequate response to medical therapy, consider continuous positive airway pressure (CPAP) starting with a positive end expiratory pressure (PEEP) of 5 cmH<sub>2</sub>O and titrating up to 10 cmH<sub>2</sub>O
- CPAP improves oxygenation via the following mechanisms
  - Delivers high flow oxygen
  - Improves functional residual capacity (FRC)
  - Recruits alveoli
  - Splints airways

- Reduces the work of breathing
- Drives pulmonary oedema back into the circulation
- If evidence of cardiogenic shock is present, avoid/discontinue nitrates and give cautious fluid challenges of 250 ml; if this fails to correct hypotension, patients will require inotropic support

#### Further management of acute pulmonary oedema

- Any patient requiring ongoing CPAP or inotropic support will need referral to high dependency unit (HDU) for:
  - Arterial line: facilitates continuous BP monitoring and frequent ABG sampling
  - Central line: facilitates central venous pressure (CVP) and central venous oxygen saturation ( $S_{cvO_2}$ ) monitoring as well as delivery of inotropic agents
  - Cardiac monitoring
  - Urine output monitoring

#### Common questions concerning acute pulmonary oedema

- Define and classify acute pulmonary oedema
  - Rapid accumulation of fluid in the alveoli and parenchyma of the lung
    - Cardiogenic pulmonary oedema: caused by elevated pulmonary capillary pressure due to decompensated LVF
    - Non-cardiogenic: caused by injury to the lung parenchyma or vasculature
- List the cardiac precipitants of acute pulmonary oedema
  - Acute coronary syndrome (ACS)
  - Cardiac arrhythmia e.g. atrial fibrillation (AF)
  - Valvular heart disease
  - Hypertension
  - Cardiomyopathy
  - Cardiac tamponade
- List the non-cardiac precipitants of acute pulmonary oedema
  - Non-compliance with medication
  - Negatively inotropic medication
  - Fluid overload
  - High output cardiac failure
    - Anaemia
    - Thyrotoxicosis
    - Sepsis
  - Acute respiratory distress syndrome (ARDS)
- By what mechanism do the symptoms dyspnoea, orthopnoea and PND occur?
  - As alveolar oedema increases, diffusion of oxygen into the pulmonary capillaries is impaired, which manifests as dyspnoea; venous return to the already congested heart and lungs increases when the patient lies flat, which manifests as orthopnoea and paroxysmal nocturnal dyspnoea (PND)
- What other symptoms would you enquire about in the history?
  - Fatigue
  - Cough productive of pink, frothy sputum
  - Ankle swelling
- What signs on examination would be suggestive of LVF?
  - Respiratory distress
  - Tachypnoea
  - Bibasal crepitations
  - Cardiac wheeze

- Tachycardia
- Displaced apex beat
- Third heart sound
- What signs on examination would be suggestive of RVF?
  - RV heave
  - Raised JVP
  - Hepatomegaly
  - Peripheral oedema
- What signs would you look for on CXR?
  - Alveolar oedema
  - Bats wing hilar shadowing and Kerley B lines
  - Cardiomegaly
  - Diversion to the upper lobes (distension of upper pulmonary veins)
  - Effusions: blunting of the costophrenic angles
- What postural adjustment may help a patient with acute pulmonary oedema
  - Sit the patient upright
- Give pharmacological therapies excluding oxygen that you would institute
  - Furosemide 40 mg IV
  - Morphine 2.5-10 mg IV
  - GTN IV infusion 1 mg/ml starting at 2 ml/hour and titrating upwards maintaining SBP >90 mmHg
- If patients exhibit an inadequate response to the above therapies, what procedure can be initiated?
  - Commence CPAP starting with a PEEP of 5 cmH<sub>2</sub>O and titrating up to 10 cmH<sub>2</sub>O
- By what mechanisms does CPAP improve oxygenation?
  - Delivers high flow oxygen
  - Improves functional residual capacity (FRC)
  - Recruits alveoli
  - Splints airways
  - Reduces the work of breathing
  - Drives pulmonary oedema back into the circulation