Pulmonary Edema

Cardiogenic pulmonary edema

- Left heart failure results in increased LV end-diastolic pressure, causing increased pulmonary capillary hydrostatic pressure. Fluid collects in extravascular pulmonary tissues faster than the lymphatics clear it.

Causes of cardiogenic pulmonary edema

Often an acute complication of MI and IHD, or an exacerbation of pre-existing cardiac disease (eg hypertension, aortic/mitral valve disease). Other causes are:

- Arrhythmias.
- Failure of prosthetic heart valve.
- Ventricular septal defect.
- Cardiomyopathy.
- Negatively inotropic drugs (eg β-blockers).
- Acute myocarditis.
- Left atrial myxoma (may cause syncope, fever, high ESR))—very rare.
- Pericardial disease.

History

- Frequently dramatic.
- Dyspnoea and distress may prevent a full history from being taken.
- Find out the length of the history and whether there is any chest pain.
- Check current drug therapy/allergies, and establish what emergency prehospital treatment has been administered.

Examination

- Usually reveals a tachypnoeic, tachycardic, and anxious patient.
- If pulmonary oedema is severe, the patient may be cyanosed, coughing up frothy pink sputum and unable to talk. Check pulse and BP; auscultate the heart for murmurs and third/fourth heart sounds of gallop rhythm.
- Look for raised JVP (also a feature of PE and cardiac tamponade). Listen to the lung fields—fine inspiratory crepitations (crackles) may be limited to the bases or be widespread.

Examination

- Wheeze may be more prominent than crepitations.
- Cardiogenic pulmonary oedema is associated with evidence of d cardiac output (sweaty, peripherally cool, and pale).
- Consider other diagnoses (eg sepsis) in patients with warm, flushed extremities.

Investigations

Commence treatment before completing investigations:

- Attach a cardiac monitor and check SpO2 with a pulse oximeter.
- Obtain an ECG. Check for arrhythmias, LAD, LVH, LBBB, and recent or evolving MI.
- Send blood for U&E, glucose, FBC, troponin, and B-type natriuretic peptide (BNP).
- If severely ill or SpO2 <90% on air, obtain an ABG.

Investigations

Obtain a CXR and look for features of cardiogenic pulmonary oedema:

- Upper lobe diversion (distension of upper pulmonary veins).
- Cardiomegaly (LV and/or left atrial dilatation).
- Kerley A, B, or C septal lines (see Fig. 3.24).
- Fluid in interlobar fifissures.
- Peribronchial/perivascular cuffifing and micronodules.
- Pleural effusions.
- Bat's wing hilar shadows.

• Request old hospital notes/ECGs. In newly diagnosed heart failure, an urgent transthoracic echo will identify the presence or absence of cardiac abnormalities.

Treatment

- Check that the airway is clear.
- Raise the trolley to sit the patient up (support with pillows, if needed).
- Provide high-flow O2 , as required, by a tight-fitting face mask.
- Give furosemide IV 40mg. Note that larger doses may be needed in patients already taking oral furosemide.
- Nitrates and opioids are no longer recommended routinely.Reserve the use of IV nitrates for specific circumstances (eg concomitant myocardial ischaemia, severe hypertension, or regurgitant aortic or mitral valve disease), starting IVI slowly (eg GTN IVI, starting at 10mcg/min), i every few minutes according to clinical response; monitor BP closely—take special care to avoid hypotension.

Treatment

If the patient has chest pain, consider giving very small titrated increments of IV opioid (with antiemetic). Do not give opioids to patients who are drowsy, confused, or exhausted, as this may precipitate respiratory arrest.

- Consider NIV (continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP)) if the patient is very breathless with acidaemia and there is no immediate improvement.
- Monitor urine output (inserting a urinary catheter if necessary).
- Treat the underlying cause and associated problems (arrhythmias, MI, cardiogenic shock, acute prosthetic valve failure).

Prosthetic valve failure

- Always consider valve failure in patients with prosthetic valves—a large variety are in common use. All are associated with some risks (eg embolism, failure, obstruction, infection, haemorrhage from associated anticoagulation), which vary according to the design.

- Acute failure of a prosthetic aortic or mitral valve results in dramatic acute-onset pulmonary oedema with loud murmurs.

Prosthetic valve failure

- The patient may deteriorate rapidly and not respond to standard drug treatment. Resuscitate as described earlier.

- A CXR will show a prosthetic heart valve ± pulmonary oedema. Call urgently for expert help (ICU team, cardiologist, and cardiothoracic surgeon). Emergency transthoracic or transoesophageal echocardiography will confifirm the diagnosis.

- Immediate valve replacement is required.

Non-cardiogenic pulmonary edema

Pulmonary oedema may occur in the absence of high pulmonary venous pressure.

The following mechanisms may be responsible:

- high capillary permeability.
- low plasma oncotic pressure.
- high lymphatic pressure.

- Changes in capillary permeability, secondary to a variety of triggers, is the mechanism most frequently implicated in non-cardiogenic pulmonary oedema, when it occurs as adult respiratory distress syndrome (ARDS).

- Since the mechanisms producing cardiogenic and non-cardiogenic pulmonary oedema differ, so does the approach to treatment.

Causes of non-cardiogenic pulmonary edema

- ARDS (sequel to sepsis, trauma, pancreatitis, COVID-19).
- Intracranial (especially subarachnoid) haemorrhage.
- IV flfluid overload.
- Hypoalbuminaemia (liver failure, nephrotic syndrome).
- Drugs/poisons/chemical inhalation.
- Lymphangitis carcinomatosis.
- Smoke inhalation.
- Near drowning incidents.
- High altitude mountain sickness.

Management

- Distinguishing non-cardiogenic from cardiogenic pulmonary oedema is usually apparent from the history.

- Evaluate the patient and resuscitate according to ABCs.

Management

- Direct treatment towards the underlying cause and according to the physiological disturbance. Use NIV early and consider urinary, intra-arterial, and central venous lines.

- Involve ICU early and provide appropriate IV fluids and inotropes—deterioration may require intubation, whilst being mindful of the risk of hypotension afterwards.