Acute Pulmonary Oedema

Pulmonary oedema occurs when fluid leaks from the pulmonary capillary network into the lung interstitium and alveoli, and the filtration of fluid exceeds the ability of the lymphatics to clear the fluid.

There are two main types of pulmonary oedema[1]:

- **Cardiogenic (or hydrostatic) pulmonary oedema** caused by an elevated pulmonary capillary pressure from left-sided heart failure.
- **Non-cardiogenic pulmonary oedema**:
  - There is usually minimal elevation of pulmonary capillary pressure (except in volume overload due to oliguric renal failure).
  - The oedema may be caused by altered alveolar-capillary membrane permeability - eg, acute respiratory distress syndrome (ARDS), or lymphatic insufficiency - eg, following lung transplant or lymphangitic carcinomatosis.
  - Oedema is uncommon in diminished plasma oncotic pressure in hypoalbuminaemic states such as severe liver disease, nephrotic syndrome and protein-losing enteropathy.
  - The mechanism for non-cardiogenic oedema is unknown in some conditions - eg, narcotic overdose, high-altitude or neurogenic pulmonary oedema.

Aetiology

- Raised pulmonary capillary pressure:
  - Heart:
    - Coronary heart disease: acute myocardial infarction, acute coronary syndromes.
    - Mechanical complications of acute coronary syndrome (eg, rupture of interventricular septum, mitral valve chordal rupture, right ventricular infarction).
    - Valvular - acute aortic regurgitation or mitral regurgitation, severe aortic stenosis, endocarditis.
    - Hypertensive crisis.
    - Acute pulmonary embolism.
    - Acute arrhythmia: rapid arrhythmia or severe bradycardia/conduction disturbance.
    - Acute myocarditis.
    - Left atrial myxoma.
    - Cardiac tamponade.
    - Aortic dissection.
    - Cardiomyopathy - eg, peripartum cardiomyopathy.
    - Drugs: myocardial depression (eg, alcohol), fluid retention (eg, non-steroidal anti-inflammatory drugs (NSAIDs)).
    - Surgery and peri-operative problems.
  - Renal:
    - Acute kidney injury, chronic kidney disease.
    - Renal artery stenosis.
    - Iatrogenic fluid overload.
    - High-output heart failure - eg, sepsis, thyrotoxic crisis, anaemia, shunts.
  - Increased pulmonary capillary permeability:
    - Acute respiratory distress syndrome (ARDS).
    - High altitude.
    - Inhaled or aspirated toxic substances.
    - Radiation.
    - Liver failure.
    - Fat embolism or amniotic fluid embolism.
    - Lymphatic obstruction - eg, mediastinal carcinomatosis, silicosis.
    - Acute or chronic upper airway obstruction.
    - Neurogenic (associated with changes in capillary hydrostatic pressure and changes in pulmonary capillary permeability): develops within a few hours after a neurological insult - eg, status epilepticus, head injury or cerebrovascular insult.

Presentation

Acute pulmonary oedema is a very frightening experience for the patient and represents a genuine medical emergency. This does not preclude a systematic assessment with a rapid, focused history and examination.
Signs:
- The patient is usually severely breathless, sweaty, nauseated and anxious.
- Initially they may have a dry or productive cough (sometimes with pink, frothy sputum).
- Patients may also develop paroxysmal nocturnal dyspnœa or orthopnoea.

History:
- Check for a past history of relevant conditions - eg, coronary heart disease, valvular heart disease, diabetes.
- Review current medication.
- Ask about smoking and alcohol use.

Signs:
- The patient is in respiratory distress, pale, sweaty, tachypnoeic and tachycardic.
- They may be cyanosed, have evidence of congested neck veins and a raised JVP.
- Basal/widespread rales or fine crackles are usually heard when listening to the chest.
- Oxygen saturation is usually <90% on room air.
- Assess for a gallop rhythm (3rd heart sound) and murmurs suggestive of valve stenosis or regurgitation.
- Hypotension - the triad of hypotension (systolic blood pressure <90 mm Hg), oliguria, and low cardiac output is known as cardiogenic shock.
- In hypertensive heart failure, a high blood pressure, tachycardia and vasoconstriction present with signs of pulmonary oedema without extensive systemic congestion.
- Where pulmonary oedema occurs in association with right heart failure, hepatomegaly and peripheral oedema are usual.

Investigations

These will not be available in the pre-hospital setting. For severe acute heart failure, treatment should be started immediately and the condition stabilised before results of investigations are available.

Blood tests:
- Renal function, electrolytes, glucose, cardiac enzymes, LFTs, clotting tests (INR).
- Arterial blood gases and pH.
- Brain natriuretic peptides - helpful in distinguishing acute pulmonary oedema from other causes of dyspnoea.

ECG: look for evidence of arrhythmia, myocardial infarction or other cardiac disease - eg, left ventricular hypertrophy.

CXR: to exclude other causes of breathlessness and confirm pulmonary oedema.

Echocardiogram: transthoracic echocardiography is usually adequate. Transoesophageal echocardiography is not needed in routine diagnostic assessment unless transthoracic echocardiography is inadequate (eg, because of obesity, chronic lung disease, ventilated patients) and an alternative modality such as cardiac magnetic resonance (CMR) imaging is not available.

A urinary catheter enables accurate measurement of urinary output, which helps rapidly to assess diuretic response and fluid balance.

More invasive procedures are required for intensive support, including arterial and central venous pressure lines and pulmonary artery catheters.

For patients with suspected heart failure, the National Institute for Health and Care Excellence (NICE) recommends [2]:

In people presenting with new suspected acute heart failure, use a single measurement of serum natriuretic peptides (B-type natriuretic peptide [BNP] or N-terminal pro-B-type natriuretic peptide [NT-proBNP]) and use the following thresholds to rule out the diagnosis of heart failure: BNP less than 100 ng/L; NT-proBNP less than 300 ng/L.

In people presenting with new suspected acute heart failure with raised natriuretic peptide levels, perform transthoracic Doppler 2D echocardiography to establish the presence or absence of cardiac abnormalities.

Management [3]

See also separate Heart Failure Management article.

Because of the fundamental differences between cardiogenic and non-cardiogenic pulmonary oedema, each requires different management and has a different prognosis [1]. This section is mainly directed at the management of acute cardiogenic pulmonary oedema.

Treatment should be directed at reversing the specific cause - although this may not be possible. See ‘Aetiology’, above, for links to relevant articles.

Management is otherwise supportive and directed at improving oxygenation, perfusion and haemodynamics, and preventing further cardiac and/or renal damage.

Pre-hospital treatment

- Resuscitation - as necessary: sit the patient up; give oxygen (if available) by face mask: 100% if no pre-existing lung disease (but even in patients with chronic obstructive pulmonary disease (COPD), give high oxygen flow initially but monitor with blood gases to ensure hypercapnia is avoided). Aim for oxygen saturations ≥95% (>90% in those with COPD).
- Judge clinical severity: some patients do not require hospital admission - eg, those with mild and stable pulmonary oedema with a known cause that is treatable without admission. However, most patients require urgent admission to hospital.
- Insert an intravenous cannula and give:
Nitrates: first-line vasodilators where systolic blood pressure is >90 mm Hg and there is no serious obstructive valvular disease. Give sublingual or buccal nitrate - eg, glyceryl trinitrate (GTN) spray two puffs sublingual, or 1-3 mg buccal isosorbide dinitrate.

Furosemide: 20-40 mg intravenously (slowly) produces transient venodilation and subsequent diuresis. This may need to be repeated based on response of clinical symptoms - diuretic effectiveness is greatly reduced in the presence of hypotension and when patients have been taking oral diuretics for a long time.

Opiates: the use of opiates is controversial and opiates should not be given to patients with acute decompensated heart failure (see below). However, analgesia and sedation may be appropriate where the patient is in pain or distressed - eg, diamorphine 2.5-5 mg intravenously slowly (or morphine 5-10 mg intravenously slowly).

Acute heart failure with pulmonary congestion/oedema without shock

Initial treatment

- An intravenous loop diuretic is recommended to improve breathlessness and relieve congestion. Symptoms, urine output, renal function and electrolytes should be monitored regularly during use of the intravenous diuretic.
- High-flow oxygen is recommended in patients with a capillary oxygen saturation <90% or PaO$_2$ <60 mm Hg (8.0 kPa) to correct hypoxaemia. Oxygen should not be used routinely in non-hypoxaemic patients, as it causes vasoconstriction and a reduction in cardiac output.
- Thrombo-embolism prophylaxis (eg, with low molecular weight heparin (LMWH)) is recommended in patients not already anticoagulated and with no contra-indication to anticoagulation, to reduce the risk of deep venous thrombosis and pulmonary embolism.
- Opiates - eg, morphine:
  - May be useful in some patients with acute pulmonary oedema, as they reduce anxiety and relieve distress associated with dyspnoea.
  - However, opiates depress respiratory drive, potentially increasing the need for invasive ventilation. Alertness and ventilatory effort should be monitored frequently after administration.
  - Studies have suggested a strong association between the use of opiates and increased mortality and morbidity (eg, intensive care unit admissions or intubation rates) and opiates should not be used routinely in the treatment of acute pulmonary oedema[4, 5].
- Opiates should always be avoided in acute decompensated heart failure[6].

Vasodilators:

- An intravenous infusion of a nitrate should be considered in patients with pulmonary congestion/oedema and a systolic blood pressure >110 mm Hg, who do not have severe mitral or aortic stenosis, to reduce pulmonary capillary wedge pressure and systemic vascular resistance.
- Nitrates may also relieve dyspnoea and congestion. Symptoms and blood pressure should be monitored frequently during administration of intravenous nitrates.

Inotropic agents:

- Are NOT recommended unless the patient is hypotensive (systolic blood pressure <85 mm Hg), hypoperfused, or shocked because of safety concerns (atrial and ventricular arrhythmias, myocardial ischaemia, and death).
- Inotropes cause sinus tachycardia and may induce myocardial ischaemia and arrhythmias. There is concern that inotropes may increase mortality.
- Levosimendan (or a phosphodiesterase III inhibitor such as milrinone) may be used to counteract the effect of a beta-blocker.
- Phosphodiesterase inhibitors (milrinone and enoximone) may be preferred to dobutamine in patients on beta-blocker therapy or with inadequate response to dobutamine. They are peripheral vasodilators and should only be used if systolic blood pressure is adequate. They may increase mortality in those with coronary artery disease.
- Levosimendan is a calcium sensitisier that appears effective in patients with decompensated chronic heart failure already on beta-blockers.

Patients with hypotension, hypoperfusion or shock

- Electrical cardioversion is recommended if an atrial or ventricular arrhythmia is thought to be contributing to haemodynamic compromise.
- An intravenous infusion of an inotrope (eg, dobutamine) should be considered in patients with hypotension (systolic blood pressure <85 mm Hg). The ECG should be monitored continuously because inotropic agents can cause arrhythmias and myocardial ischaemia.
- Short-term mechanical circulatory support should be considered in patients remaining severely hypoperfused despite inotropic therapy and with a potentially reversible cause (eg, viral myocarditis) or a potentially surgically correctable cause (eg, acute interventricular septal rupture).
- A vasoressor (eg, dopamine or noradrenaline (norepinephrine)) may be considered in patients who have cardiogenic shock, despite treatment with an inotrope. Intra-arterial blood pressure measurement should be considered.

Monitoring

- Examination:
  - Systolic blood pressure, heart rhythm and rate, saturation of peripheral oxygen (SpO$_2$) using a pulse oximeter, and urine output should be monitored on a regular and frequent basis until the patient is stabilised.
• It is important to examine the patient repeatedly, including assessment for any new heart murmurs - eg, a patient who has severe pulmonary oedema following a myocardial infarction may go on to develop a ventricular septal defect or mitral regurgitation (confirm with an echocardiogram).

• **Blood tests:**
  - Renal function, electrolytes, serial ECGs and cardiac enzymes should also be closely monitored.

• **Intra-arterial line:**
  - Insertion of an intra-arterial line should only be considered in patients with persistent heart failure and a low systolic blood pressure despite treatment.

• **Pulmonary artery catheterisation:**
  - Right heart catheterisation does not have a general role in the management of acute heart failure, but may help in the treatment of a minority of selected patients with acute heart failure.

**After stabilisation**

• Angiotensin-converting enzyme (ACE) inhibitor, or angiotensin-II receptor antagonist (AIIRA):
  - In patients with reduced EF not already receiving an ACE inhibitor (or AIIRA).
  - This treatment should be started as soon as possible, blood pressure and renal function permitting.

• Beta-blocker:
  - In a person presenting with acute heart failure who is already taking beta-blockers, continue the beta-blocker treatment unless they have a heart rate less than 50 beats per minute, second or third degree atrioventricular block, or shock.
  - Start or restart beta-blocker treatment during hospital admission in people with acute heart failure due to left ventricular systolic dysfunction, once their condition has been stabilised - for example, when intravenous diuretics are no longer needed.

• Mineralocorticoid (aldosterone) receptor antagonist (MRA):
  - In patients with reduced EF not already receiving an MRA.
  - This treatment should be started as soon as possible, renal function and potassium permitting.
  - As the dose of MRA used to treat heart failure has a minimal effect on blood pressure, even relatively hypotensive patients may be started on this therapy during admission.

• Digoxin:
  - In patients with reduced EF, digoxin may be used to control the ventricular rate in atrial fibrillation, especially if it has not been possible to up-titrate the dose of beta-blocker.
  - Digoxin may also provide symptom benefit and reduce the risk of hospital admission for heart failure in patients with severe systolic heart failure.

• It is common to restrict sodium intake to <2 g/day and fluid intake to <1.5-2.0 L/day, especially during the initial management of an acute episode of heart failure associated with volume overload, although there is no firm evidence to support this practice.

**Non-invasive ventilation**

• Continuous positive airway pressure (CPAP) and non-invasive positive pressure ventilation (NIPPV) relieve dyspnoea and improve certain physiological measures (eg, oxygen saturation) in patients with acute pulmonary oedema.

• Non-invasive ventilation should be considered in dyspnoeic patients with pulmonary oedema and a respiratory rate >20 breaths/minute to improve breathlessness and reduce hypercapnia and acidosis.

• Non-invasive ventilation can reduce blood pressure and should not generally be used in patients with a systolic blood pressure of <85 mm Hg (and blood pressure should be monitored regularly when this treatment is used).

• A large RCT showed that neither type of non-invasive ventilation reduced mortality or the rate of endotracheal intubation when compared with standard therapy, including nitrates and opiates.

**Endotracheal intubation and invasive ventilation**

• The primary indication for endotracheal intubation and invasive ventilation is respiratory failure leading to hypoxaemia, hypercapnia, and acidosis.

• Physical exhaustion, diminished consciousness, and inability to maintain or protect the airway are other reasons to consider intubation and ventilation.

**Mechanical circulatory support**

• The conventional indications for an intra-aortic balloon pump (IABP) are to support the circulation before surgical correction of specific acute mechanical problems, during severe acute myocarditis and in selected patients with acute myocardial ischaemia or infarction before, during and after percutaneous or surgical revascularisation.

• There is no good evidence that an IABP is of benefit in other causes of cardiogenic shock.
More recently, balloon pumps (and other types of short-term, temporary circulatory support) have been used to bridge patients until implantation of a ventricular assist device or heart transplantation.

Ventricular assist devices and other forms of mechanical circulatory support (MCS) may be used as a 'bridge to decision' or longer-term in selected patients.

Ultrafiltration

Venovenous isolated ultrafiltration is sometimes used to remove fluid in patients with heart failure, although it is usually reserved for those unresponsive or resistant to diuretics.

Other treatments

- **Arrhythmias**: see separate Atrial Fibrillation article. **Pacing** is recommended in patients haemodynamically compromised by severe bradycardia or heart block.
- **Renal dysfunction** may limit the use of ACE inhibitors and AIIRAs; progressive acute kidney injury and volume overload may require renal replacement therapy.
- **Surgical options** include coronary revascularisation, correction of anatomical lesions, valve replacement or reconstruction, mechanical assist devices for temporary circulatory support and heart transplantation.

For ongoing management, see separate Heart Failure Management article. Review the patient's current medication and care needs.

Prognosis

The prognosis for patients with acute pulmonary oedema depends on the underlying cause, the patient’s age and comorbidities, and the speed of diagnosis and initiation of effective treatment.

Further reading & references

2. Diagnosing and managing acute heart failure in adults; NICE Clinical Guidelines (Oct 2014)

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