



Andrew Baird

Acute pulmonary oedema

Management in general practice

Background

Acute pulmonary oedema is a life threatening emergency that requires immediate intervention with a management plan and an evidence based treatment protocol.

Objective

This article describes the features, causes, prevalence and prognosis of heart failure and the management of acute pulmonary oedema.

Discussion

Presentations of acute pulmonary oedema and acute heart failure to general practice require a coordinated and urgent response. Initial assessment, management and monitoring should occur concurrently and must be modified in response to clinical changes.

Keywords: heart failure; pulmonary oedema; emergencies



Acute heart failure (AHF) is a clinical syndrome characterised by the rapid onset and progression of breathlessness and exhaustion. There is usually fluid overload.¹ Acute heart failure typically occurs as 'acute decompensated heart failure' (ADHF) either secondary to chronic heart failure (CHF) or de novo. The more severe presentations of acute heart failure are acute pulmonary oedema (APO) and cardiogenic shock. In the EuroHeart Failure Survey II² of patients hospitalised with AHF, 37% had de novo acute heart failure and 16% had APO.

General practitioners are familiar with the clinical presentation of APO – severe dyspnoea, distress, pallor, sweating, tachycardia and poor peripheral perfusion. However, in general practice, the presentation of AHF may be less dramatic than APO and the clinical features may be atypical (eg. delirium or falls in the elderly). Differential diagnoses and potential precipitants for APO are listed in *Table 1*.

The most common cause of heart failure is impaired myocardial function (cardiomyopathy) secondary to one or more of the following¹:

- hypertension (>60% of patients with heart failure)
- ischaemic heart disease (>50% of patients with heart failure)
- idiopathic dilatation (10% of patients with heart failure)
- diabetes
- alcohol excess
- obesity
- drug toxicity.

Other cardiac causes of heart failure include arrhythmias, valve dysfunction and pericardial disease. Noncardiac causes of heart failure are hypovolaemia (dehydration or haemorrhage), pulmonary embolism and high output states (anaemia, septicaemia and thyrotoxicosis).

On echocardiographic criteria, about one-third to one-half of patients with a diagnosis of CHF have normal ventricular systolic function with a left ventricular ejection fraction >40%.¹ There is impaired relaxation of the ventricle in diastole, which is termed 'diastolic heart failure' or synonymously 'heart failure with normal systolic function' (HFNSF). The significance of HFNSF as a distinct clinical entity is uncertain. However, patients with HFNSF do seem particularly dependent on ventricular filling for cardiac output and are very sensitive to overdiuresis.³

**Table 1. Acute pulmonary oedema – differential diagnoses and potential precipitants**

Consider and manage the following as appropriate	
Differential diagnoses of acute dyspnoea	Precipitants of acute pulmonary oedema
Primary respiratory causes <ul style="list-style-type: none"> Exacerbation of chronic obstructive pulmonary disease (COPD) Acute asthma Pneumothorax Pneumonia 	Primary cardiac causes <ul style="list-style-type: none"> Acute coronary syndrome/myocardial infarction Arrhythmia Pericarditis Acute valve dysfunction (aortic stenosis, mitral regurgitation) Endocarditis
Anaphylaxis	Fluid overload
Hyperventilation	Drugs (eg. nonsteroidal anti-inflammatory drugs [NSAIDs], nondihydropyridine calcium channel blockers)
Noncardiogenic acute pulmonary oedema <ul style="list-style-type: none"> Drowning Laryngospasm/upper airway obstruction Toxic inhalation 	Noncompliance with: <ul style="list-style-type: none"> heart failure management medications restrictions on fluid intake or alcohol intake
Pulmonary embolus	Pulmonary embolus
Acute renal failure	Acute renal failure
High output states <ul style="list-style-type: none"> Septicaemia Anaemia Thyrotoxicosis 	High output states <ul style="list-style-type: none"> Septicaemia Anaemia Thyrotoxicosis

Prevalence

The prevalence of CHF increases with age. In Australia, the prevalence in the 55–64 years age group is estimated at 2.5%, in the over 75 years age group it is 8.2%. There are twice as many women as men with CHF.⁴ At the age of 40 years, the lifetime risk of developing heart failure is about 20% in both men and women.¹ There are no data on the incidence of APO, but it is estimated that most patients with CHF will have at least one episode of ADHF or APO.¹

Prognosis

Overall, CHF has a poor prognosis with about 50% mortality 5 years from diagnosis.^{5–7} In-hospital mortality from ADHF ranges from 2.1–21.9%, depending on mortality risk stratification.⁸

Clinical decision tools to predict mortality in ADHF have been developed based on the following recognised indicators of poor prognosis: renal impairment, low systolic blood pressure, tachypnoea, hyponatraemia, anaemia and comorbidities.^{8,9}

Management

The management of ADHF and APO is largely based on clinical experience rather than prospective randomised controlled trials.^{1,5–7,10} Current management described in this article is informed by guidelines from Australia,^{3,6} Europe,⁵ the United States of America⁷ and by a review.¹⁰ The therapeutic interventions recommended by these authorities differ in approach and emphasis but the principles are similar.

Table 2 summarises the assessment and management steps for APO.

There are no guidelines or studies which specifically address the management of APO in the primary care setting. A PubMed search on all fields using the MeSH terms ‘acute heart failure’ and ‘family practice’ produced only one relevant article.¹¹

The goals of APO management are symptom relief, reduction of extracellular fluid excess, improved haemodynamics, improved arterial oxygenation and satisfactory perfusion of the vital organs.¹

General guidelines, approaches and goals must be modified according to clinical setting (eg. rural or metropolitan) and context (eg. APO in an elderly patient with end stage CHF as opposed to a previously healthy patient 50 years of age).

The prerequisites for successful management of APO include appropriate:

- systems (triage, crisis resource management plans, organisation, leadership, teamwork, documentation)
- resources (personnel, equipment and drugs)
- knowledge and skills.

Assessment, management and definitive therapy are concurrent, continuous and iterative. All patients require hospital admission for stabilisation and monitoring unless inappropriate, such as in the palliative care context. Definitive therapy for acute pulmonary oedema is outlined in *Table 3*.

Once the patient is stable, management progresses to postacute care planning.

**Table 2. Assessment and management of acute pulmonary oedema**

Initial	<ul style="list-style-type: none">• Call for help (other GPs, nurses, clinic staff, dial 000)• Commence oxygen• Insert 16 gauge intravenous cannula• Commence definitive treatment while assessing patient
History	<ul style="list-style-type: none">• Focused cardiorespiratory history (Note: nocturnal dyspnoea and orthopnoea are specific but not sensitive for heart failure)• Check past medical history, medications, compliance• Consider third party information
Examination	<ul style="list-style-type: none">• Five vital signs<ul style="list-style-type: none">– temperature– pulse (rate, rhythm)– blood pressure– respiration (rate, pattern)– oxygen saturation• Focused cardiorespiratory examination, particularly:<ul style="list-style-type: none">– colour– diaphoresis– jugular venous pulse– apex beat (shift, loading)– heart sounds (gallop rhythm?)• Murmurs (eg. mitral regurgitation, aortic stenosis)• Peripheral perfusion and oedema• Air entry, crepitations, rhonchi
Monitoring	<ul style="list-style-type: none">• Blood pressure• Continuous ECG (lead II) – if available• Oxygen saturation• Automated external defibrillator on standby• Consider urinary catheter if managing in rural hospital
Investigations (depending on availability)	<ul style="list-style-type: none">• 12 lead electrocardiogram (ECG) as soon as possible (APO is an acute coronary syndrome until proven otherwise)• Chest X-ray (portable, if available)• Point-of-care pathology tests (if available)<ul style="list-style-type: none">– troponin– B-type natriuretic peptide (BNP)*• Other pathology tests: urea and electrolytes, liver function tests, glucose, urinalysis, full blood examination (FBE), arterial blood gases• Echocardiogram at earliest opportunity (depending on access and patient stability)
Reassurance and explanation	<ul style="list-style-type: none">• Patient and relatives

* Useful negative predictive value (if BNP lower than 100 pg/mL, dyspnoea is unlikely to be cardiogenic). This test costs about \$50 and there is a Medicare rebate for the diagnosis of dyspnoea in hospital emergency departments. The role and utility of this test in APO and in primary care settings is not yet well defined

**Table 3. Definitive therapy for acute pulmonary oedema**

Therapy (action)	Dose	Notes
Posture	Patient supported in sitting up position	Supine position if unconscious or in cardiogenic shock
Oxygen (corrects hypoxaemia) (align centre of flowmeter ball to required flow rate)	10–15 L/min via Hudson type mask and reservoir bag ¹² (This is initial treatment even in patients with known COPD who are at risk of hyperoxic hypercapnia as oxygenation is the priority. Monitor conscious state, respiratory rate and oxygenation)	When stable, reduce to 2–6 L/min via nasal prongs or 5–10 L/min via Hudson mask Patients with COPD should ideally receive controlled oxygen therapy via a 28% Venturi mask (flow rate 4 L/min) ¹² Titrate to achieve oxygen saturation of 94–96% (non-COPD) or 88–92% (COPD) ¹²
Glyceryl trinitrate (venodilator, reduces preload)	400 µg sublingual every 5 minutes (up to three doses)	Maintain systolic blood pressure (SBP) above 100 mmHg Contraindicated within 48 hours of PDE5 inhibitor
	Intravenous infusion commencing at 10 µg/min	Double infusion rate every 5 minutes according to clinical response (maintain SBP above 90 mmHg)
Positive airway pressure support (continuous [CPAP] or bi-level [BiPAP] if available) reduces alveolar and pulmonary interstitial oedema; reduces venous return and preload	<ul style="list-style-type: none"> • Continuous positive airway pressure – 10 cmH₂O • BiPAP: inspiratory pressure 15 cmH₂O, expiratory pressure 5 cmH₂O 	Contraindicated if: <ul style="list-style-type: none"> • SBP <90 mmHg • reduced consciousness Clinical improvement may occur within 10 minutes. Duration of use depends on efficacy and tolerability. Some evidence BiPAP may be superior
Frusemide (loop diuretic, reduces fluid overload; possible vasodilator effect)	20–80 mg IV bolus After bolus, consider continuous IV infusion at 5–10 mg/hour (total dose <100 mg in first 6 hours, and <240 mg in the first 24 hours)	Consider repeating a bolus dose after 30–60 minutes if there has been no clinical improvement and no diuresis Risk of hypovolaemia; avoid if no clear fluid overload
Morphine (reduces sympathetic nervous activity; possible venodilator effect)	1–2 mg IV	Does not improve pulmonary oedema or cardiac output Anxiolytic Reduces respiratory work
Other drugs that may be used		
Low molecular weight heparin (venous thromboembolism prophylaxis)	Eg. enoxaparin 40 mg SC daily	Commence if available
Digoxin	500 µg IV (over 5 or more minutes)	Only for digoxin naïve patients with rapid atrial fibrillation
Spironolactone	25–50 mg orally stat	Consider in volume overloaded patient with poor response to IV frusemide ⁵
Other therapies that may be used in intensive care unit		
<ul style="list-style-type: none"> • Adrenaline infusion • Vasopressin antagonists (conivaptan, tolvaptan) • Vasodilators (sodium nitroprusside) • Inotropes (for cardiogenic shock or hypoperfused state with SBP <90 mmHg) <ul style="list-style-type: none"> – beta-adrenergic stimulators (dobutamine, dopamine) – phosphodiesterase inhibitors (milrinone, enoximone) • Ultrafiltration • Mechanical support (eg. intra-aortic balloon pump) for cardiogenic shock 		



Checklist for postacute care

- Structured management plan (Medicare primary care items may apply): patient and GP define problems, goals and actions
- Team based care according to needs and access (Medicare primary care items may apply). This may involve the GP, practice nurse, cardiologist and/or heart failure clinic, pharmacist, Aboriginal health worker, dietician or exercise physiologist
- Education and support for patient and carers (useful resources are available at www.heartfoundation.org.au)
- Home assessment and community support
- Lifestyle
 - smoking cessation
 - diet: follow Heart Foundation guidelines, no added salt
 - fluid restriction: maximum 2 L/day (1.5 L/day if severe CHF)
 - alcohol: no more than one unit per day
 - exercise: individualised program
- Investigations
 - echocardiogram: mandatory for all patients post-AHF
 - full cardiac ‘workup’: ECG, lipid profile, glucose, renal function, liver function, thyroid function, iron studies, FBE
- Monitoring: patient self monitoring (symptoms, weight, BP)
- GP review (frequency determined by severity and stability of CHF):
 - symptoms, weight, BP, cardiorespiratory status
 - risk factor management
 - comorbidities (especially ischaemic heart disease, diabetes, COPD, renal impairment, sleep apnoea, obesity, depression, osteoarthritis)
 - mental state
 - medication review (Medicare primary care items may apply)
 - pathology (urea, creatinine, electrolytes, FBE)
- Medications indicated (*improves prognosis as well as symptoms)
 - angiotensin converting enzyme inhibitor (ACEI)*: all patients with CHF (if not tolerated use angiotensin II receptor blocker)
 - beta blocker*: patients with systolic failure; COPD is not a contraindication (bisoprolol, carvedilol, metoprolol, nebivolol)
 - frusemide: symptoms of fluid overload
 - spironolactone*: add on if symptom control inadequate
 - digoxin: consider for atrial fibrillation, or as add on therapy for sinus rhythm with severe CHF inadequately controlled with the above
- Medications (contraindicated or caution):
 - verapamil and diltiazem
 - NSAIDs or cyclo-oxygenase-2 inhibitors
 - corticosteroids
- Vaccinations: influenza, pneumococcal
- Device therapy for patients with moderate or severe CHF (cardiologist would assess and recommend if appropriate):
 - cardiac resynchronisation therapy (eg. if QRS is greater than 120 ms)
 - implantable cardioverter defibrillator.

Summary of important points

- Acute pulmonary oedema is a life threatening emergency requiring immediate intervention with a crisis resource management plan and an evidence based treatment protocol.
- The principal therapies for APO are oxygen, sitting the patient upright, glyceryl trinitrate, positive airway pressure, frusemide, morphine and inotropes.
- A key component in the management of APO is postacute care which presents an opportunity to optimise wellbeing and prognosis in CHF.

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