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21 Common Diagnoses in ICU

A Case Based Approach

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CHAPTER

2

Intensive Care Management of Acute Heart Failure

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CASE PRESENTATION

A 62-year-old woman with a history of type 2 diabetes mellitus and hypertension presented with one week history of worsening exertional chest pain and dyspnoea. She denied any fever, cough or wheeze. At the time of presentation, she was conscious and rational. Her peripheries were cold, respiratory rate was 40 breaths per minute and her oxygen saturation (SpO_2) was 90% on room air. She had a heart rate of 128 beats per minute and her blood pressure was 98/60. Lungs revealed bi-basal fine crepitations. The 12-lead electrocardiogram (ECG) showed ST depressions and T inversions in anterolateral leads, troponin I level was 0.8 ng/mL (0–0.04 ng/mL), while liver and functions were within normal range. Her chest X-ray (CXR) is shown in Fig. 2.1. Bedside echocardiogram showed hypokinesia involving the anterior and lateral walls and septum, with left ventricular ejection fraction (LVEF) of 30–35%. She was given the loading doses of antiplatelets and statin and was started on an intravenous (IV) frusemide infusion followed by subcutaneous enoxaparin 1 mg/kg twice daily. Her oxygenation progressively deteriorated and ultimately was started on non-invasive ventilation (CPAP) with pressure support (PS) of 10 cm H_2O and positive end expiratory pressure (PEEP) of 8 cm H_2O. She became progressively hypotensive needing close hemodynamic monitoring and support.

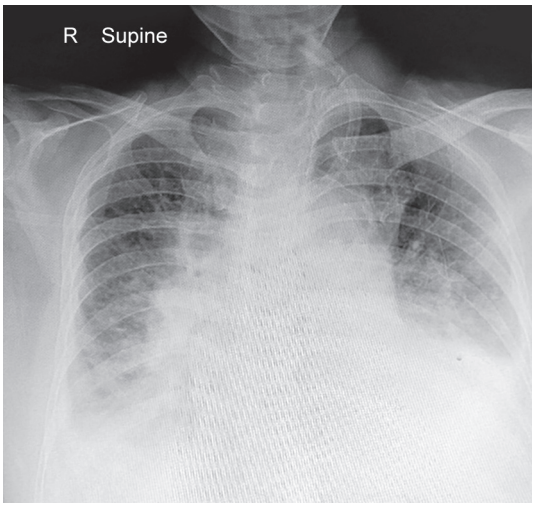

Fig. 2.1: Chest X-ray

How would you Improve this Patient's Haemodynamic Instability?

Vasoactive agents and close hemodynamic monitoring: Her vital parameters were regularly monitored and continued on CPAP. Serial arterial blood gases were carried out to assess oxygenation, acid–base balance and lactate levels. Adequate arterial oxygenation was maintained on the above CPAP settings throughout the stay and she avoided the need for invasive ventilation. Invasive arterial blood pressure monitoring was established with a defined mean arterial pressure (MAP) target of 70–80 mmHg. She was started on

IV noradrenaline infusion titrated according to the target MAP. She was continued on IV frusemide infusion which was titrated and weaned according to the diuresis and lung decongestion. She was started on dual antiplatelet therapy and statin along with enoxaparin. Fluid intake was restricted to 1.5–1.8 litres initially which was gradually increased as she recovered, to 2.4 litres a day. Her electrolyte balance was looked into as well as the blood sugar control was achieved with basal-bolus insulin regime. The course was not complicated with any other organ failures and her inflammatory markers were within normal range. She made an uneventful recovery with gradual weaning from CPAP and noradrenaline. Subsequently she was stabilised on long term heart failure medications along with antiplatelets. She was planned for an interval coronary angiography and was discharged to the cardiology follow up.

PRINCIPLES OF MANAGEMENT

Diagnosis

Heart failure (HF) is defined as inability to deliver oxygen in sufficient amounts to metabolising tissues, resulting from an abnormality of the structure and function of the heart. When there is rapid onset of symptoms and signs of HF, needing urgent interventions, it is called acute heart failure (AHF). The diagnosis of AHF can be challenging, especially in older populations, due to the similarity of AHF symptoms with other respiratory comorbidities like chronic obstructing pulmonary disease (COPD), bronchial asthma (BA) and pneumonia.[1,2] Symptoms and signs of HF result from congestion due to elevated filling pressures, of left (dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, lung crepitations, hypoxia, noticeable S3, loud P2 and murmurs) or right (systemic congestion resulting in oedema in dependent regions, hepatic congestion with tender hepatomegaly, congestion in kidneys, intestine and lower extremities, ascites, high jugular venous pressure) side of the heart or both. Any type of HF can give rise to symptoms and signs attributable to low cardiac output states, including cold extremities, hypotension, narrow pulse pressure, pulses alternans and oliguria. The diagnosis of AHF is further supported by advanced haemodynamic parameters monitored via pulse contour analysis combined with either transpulmonary thermodilution (PiCCO) or bolus indicator dilution (LiDCO) methods.[1,2,3] Pulmonary artery catheterisation (PAC) studies can be very useful in properly selected patients, especially with unclear aetiology.[3]

Aetiologies and Precipitating Factors

The commonest etiologies for AHF include ischaemia, inflammation, arrhythmia and acute valve insufficiency. Decompensation of existing HF can occur without a clear precipitant, and also with any of the causes above plus infection, uncontrolled hypertension and non-compliance (Table 2.1). Among the causes which need urgent attention and treatment include, acute ***C***oronary syndromes (ACS), ***H***ypertensive emergency, unstable ***A***rrhythmias, acute ***M***echanical causes, ***P***ulmonary embolism, ***I***nfection and ***T***amponade (Acronym: ***CHAMPIT***).[1,3,4] Patients with AHF commonly have other co-existing co-morbidities (e.g. Type II Diabetes mellitus, atherosclerosis, renovascular disease, anaemia, obstructive sleep apnoea) which may contribute to the pathology, thus need to be considered as therapeutic targets.

Investigations

ECG is essential to exclude ischaemia and arrhythmias. CXR will give evidence of pulmonary oedema and other comorbidities which will present in a similar way as AHF. Radiological

Table 2.1: Aetiological factors triggering AHF	
Precipitants of rapid decompensation	***Precipitants of slower decompensation***
ACS	Non-compliance with diuretics and fluid restriction
Complications of ACS	Infection
E.g.: Ruptures intraventicular septum/papillary muscle/Chordae of mitral valve	
RV infarction	Anaemia
Tachyarrhythmias or severe bradyarrhythmias	Worsening pre-existing valvular dysfunction
Pulmonary embolism	Severe thyroid dysfunction
Cardiac tamponade	Exacerbation of lung disease: Chronic obstructive pulmonary disease
Aortic dissection	Renal failure
Hypertensive crisis	Hypertension
Peripartum cardiomyopathy	Iatrogenic–NSAIDs, steroids
Cerebrovascular insult (neurogenic pulmonary oedema)	Metabolic/hormonal derangements

evidence of pulmonary venous congestion, interstitial oedema and cardiomegaly suggest AHF. Full blood count, urine full report, blood urea, serum creatinine, serum electrolytes and troponin levels detect precipitants of decompensation.[1,2] The B-type natriuretic peptide (BNP) and N-terminal-proBNP (NT-proBNP) are useful in the diagnosis of AHF. A BNP level less than 100 pg/mL strongly suggests non-heart failure aetiology for dyspnoea, greater than 400 pg/mL strongly suggests AHF, while a BNP level between 100–400 pg/mL is considered indeterminate.[3] Ultrasound imaging of the lungs aids in identifying evidence of pulmonary oedema ("B" profile). Doppler echocardiography is the most useful non-invasive modality of imaging in AHF, providing information on cardiac structure and function. Relative wall motion abnormalities and estimates of haemodynamics help in the management.[1,3] Arterial blood gas analysis is handy in evaluating acid–base and lactate status, to assess the response to treatment.

Clinical Classification of AHF

The clinical classification is based on the presence or absence of congestion ("wet" *vs* "dry") and/or the presence or absence of hypoperfusion ("cold" *vs* "warm").[3,4,5] The combinations of these findings define 4 major clinical categories with possible overlaps between them (Table 2.2). These clinical entities require different treatments.

Initial Management

a. Goals of Management

Immediate aim of management is to improve symptoms by restoring oxygenation and improving organ perfusion (Fig. 2.2), simultaneously avoiding cardiac, renal and other organ damage as well. One should consider life-saving therapies, including mechanical circulatory support (MCS) to stabilise the patient and optimize treatment. Following acute management, treatment should be targeted to prevent early readmission and to improve symptoms and survival.

Table 2.2: Clinical presentations of acute heart failure

	Acute decompensated heart failure	*Acute pulmonary oedema*	*Isolated right ventricular failure*	*Cardiogenic shock*
Main mechanisms	LV dysfunction Sodium and water renal retention	Increased afterload and/or predominant LV diastolic dysfunction Valvular heart disease	RV dysfunction and/or pre-capillary pulmonary hypertension	Severe cardiac dysfunction
Main cause of symptoms	Fluid accumulation, increased intraventricular pressure	Fluid redistribution to the lungs and acute respiratory failure	Increased central venous pressure and often systemic hypoperfusion	Systemic hypoperfusion
Onset	Gradual (days)	Rapid (hours)	Gradual or rapid	Gradual or rapid
Main haemodynamic abnormalities	Increased LVEDP and PCWP[a] Low or normal cardiac output Normal to low SBP	Increased LVEDP and PCWP[a] Normal cardiac output Normal to high SBP	Increased RVEDP Low cardiac output Low SBP	Increased LVEDP and PCWP[a] Low cardiac output Low SBP
Main clinical presentations	Wet and warm or Dry and cold	Wet and warm[b]	Dry and cold or Wet and cold	Wet and cold
Main treatment	Diuretics Inotropic agent/ vasopressors (if peripheral hypoperfusion/ hypotension) Short-term MCS or RRT, if needed	Diuretics Vasodilators[b]	Diuretics for peripheral congestion Inotropic agents/ vasopressors (if peripheral hypoperfusion/ hypotension) Short-term MCS or RRT if needed	Inotropic agents/ vasopressors Short-term MCS RRT

LV = Left ventricular; LVEDP = Left ventricular end-diastolic pressure; MCS = Mechanical circulatory support; PCWP = Pulmonary capillary wedge pressure; RV = Right ventricular; RVEDP = Right ventricular end-diastolic pressure; RRT = Renal replacement therapy; SBP = Systolic blood pressure.

[a]May be normal with low cardiac output.

[b]Wet and cold profile with need of inotropes and/or vasopressors may rarely occur.

The process of management needs an integrative approach where continuous assessment and treatment are provided within a shorter time frame. A suggested framework for the early phase management of AHF and associated cardiogenic shock are demonstrated in Figs 2.3 and 2.4.

b. Oxygen Therapy and Ventilatory Support

Correction of hypoxia is critical as hypoxia (and hypercarbia) observed in patients with pulmonary congestion, has negative effects on coronary and pulmonary perfusion. Administration of oxygen should be reserved for hypoxic patients with SpO_2 <90% or partial

Acute cardiac decompensation	
Therapeutic goals	• Restoration of organ perfusion • Weaning of vasoactive drugs • Avoid complications of disease and treatment • Planning of specific intervention(s) • Establish treatment goals - Recovery, Bridge, Palliation
Respiratory support	Aim: Correction of life-threatening hypoxia • Airway management • Oxygen delivery • CPAP/NIPPV • IPV
Haemodynamic assessment	Aim: Assessment of severity of compromise • Examination • ABG (Lactate) and blood to assess organ injury • Echocardiography • CO monitoring
Haemodynamic stabilization	Aim: Organ and tissue perfusion • Optimization of preload, afterload and CO • Commence vasoactive agents • Correction of ischaemia, arrhythmias and specific triggers
Causative etiology	Aim: Identify cause of decompensation • Diagnostic work up using CHAMP • Aetiological specific interventions • Cardiology specialty input • Consider transfer to specialist centre
Progressive or refractory shock	Aim: Establish escalation strategy • MDT/shock team involvement • Consider short team MCS • Assess eligibility for durable MCS or transplantation • Palliation

Fig. 2.2: Management principles of acute cardiogenic shock

ABG, Arterial blood gas; CHAMP, acute Coronary syndrome, Hypertension emergency, Arrhythmia, acute Mechanical cause, Pulmonary embolism; CO, cardiac output; CPAP, continuous positive airway pressure; MCS, mechanical circulatory support; MDT, multidisciplinary team; NIPPV, non-invasive positive-pressure ventilation; IPPV, invasive positive pressure ventilation

pressure of oxygen (PaO_2) < 8.0 kPa (60 mmHg).[4] The use of non-invasive positive pressure ventilation (NIPPV) in the form of continuous (CPAP) or Bi-level (BiPAP) positive airway pressure, helps in alveolar recruitment, redistribution of extravascular lung water, and in improving compliance and surfactant production, thus reducing the shunt and work of breathing.[5] Use of NIPPV needs to be used in patients with respiratory distress as evidenced by tachypnoea (respiratory rate >25 breaths/min) and desaturation (SpO_2 <90%). It should be instigated immediately to alleviate symptoms, to reduce the rate of invasive ventilation and hospital mortality[4,5] paying careful attention to avoid hypotension. Invasive positive pressure ventilation (IPPV) is indicated, if there is respiratory failure leading to hypoxaemia (PaO_2 <60 mmHg), hypercapnia ($PaCO_2$ >50 mmHg) and acidosis (pH <7.35), which cannot be managed non-invasively. PPV reduces pulmonary wedge pressure, LV afterload, myocardial oxygen demand and work of breathing, thus improves cardiac index and oxygenation.

c. Cardiovascular Management

One main target in managing AHF patients is to restore adequate cardiac output aiming to establish adequate tissue perfusion to prevent end organ damage. Specific precipitants

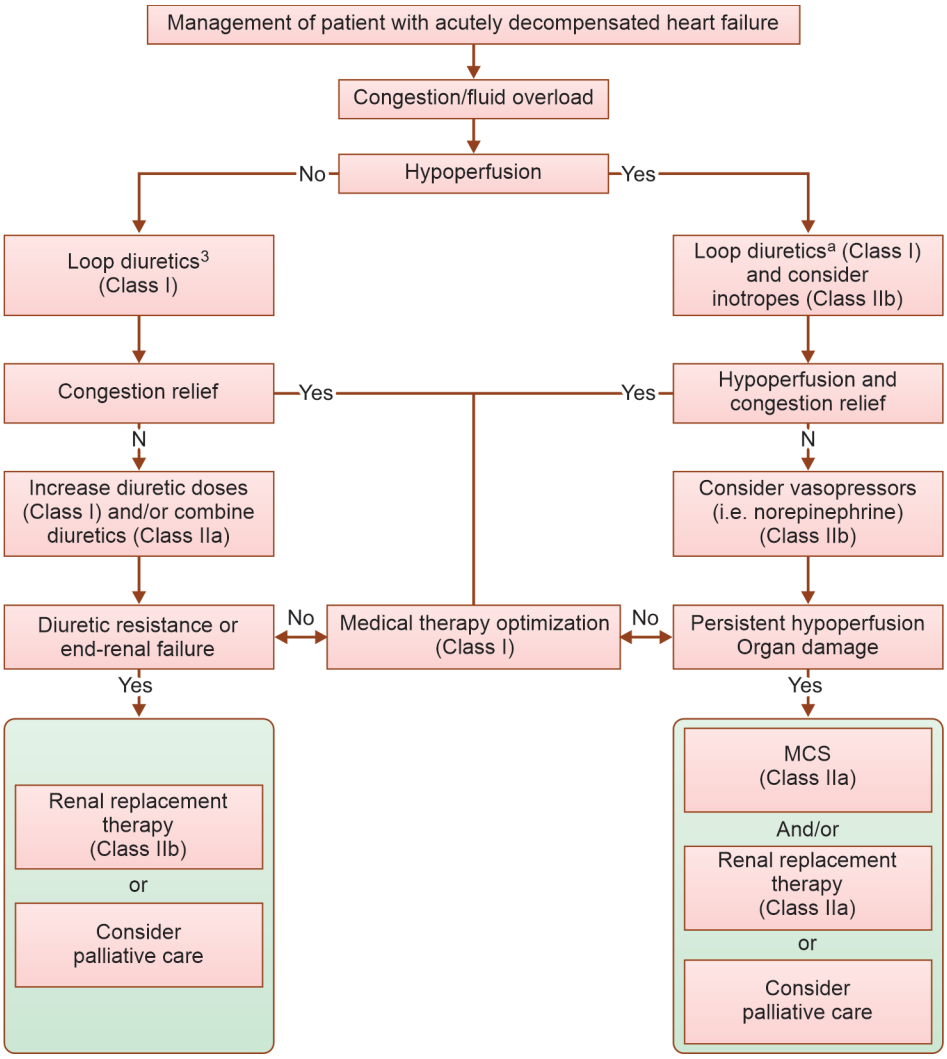

Fig. 2.3: Management of acute decompensated heart failure

MCS = Mechanical circulatory support. [a]Adequate diuretic doses to relieve congestion and close monitoring of diuresis is recommended regardless of perfusion status.

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need to be managed as they are diagnosed, e.g. referring for coronary angiography within the first 120 minutes with an intent to achieve complete revascularisation, in acute coronary syndromes (ACS).[4,5] Diuretics (mainly loop diuretics) play a major role in alleviating symptoms and signs of fluid overload by dual action of diuresis and vasodilation. To start with intravenous diuretics and vasodilators are indicted to relieve dyspnoea, titrated to blood pressure (BP). Intravenous furosemide is used most commonly as the first-line diuretic. One should use the lowest dose possible to provide adequate clinical effect and should be modified according to patients renal function.[4] Attention should be paid to avoid hypokalaemia, renal impairment and hypovolaemia. Intravenous vasodilators (Table 2.3) are also used in AHF for symptomatic relief; however, there is no robust evidence confirming their beneficial effects. Opioid agents have both anxiolytic action and vasodilatory effects in the setting of acute

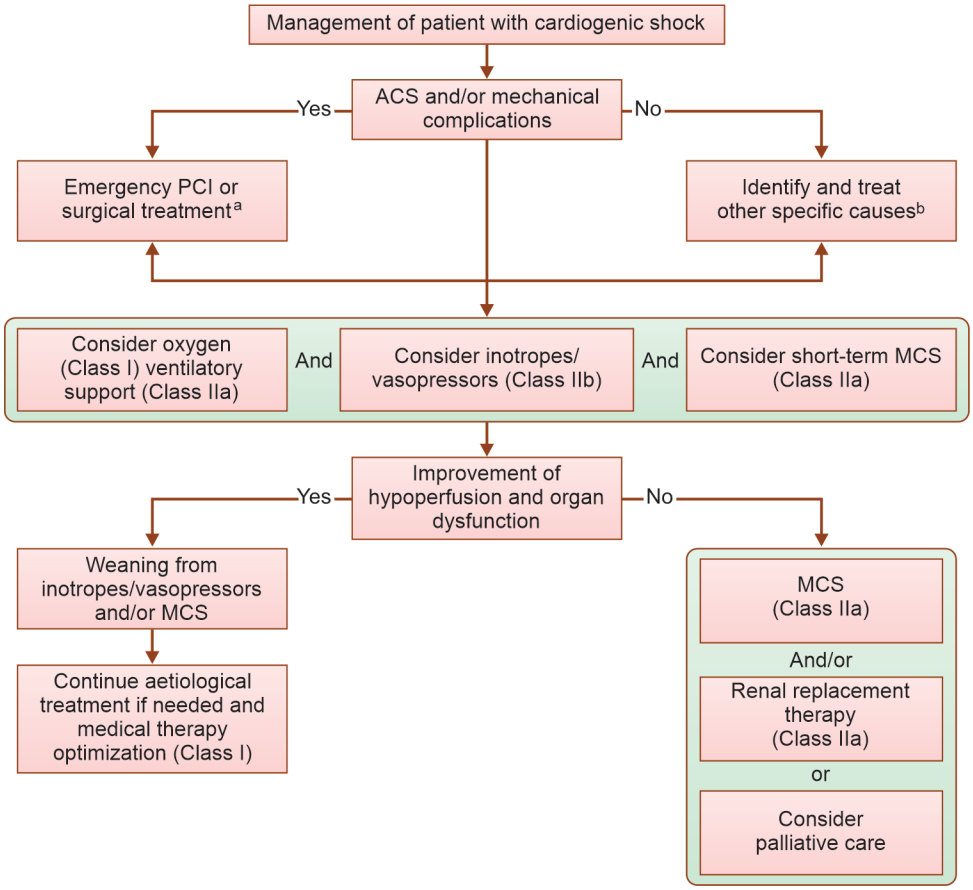

Fig. 2.4: Management of cardiogenic shock

ACS = Acute coronary syndrome; BTT = Bridge to transplantation; MCS = Mechanical circulatory support; PCI = Percutaneous coronary intervention. [a]PCI in ACS, pericardiocentesis in tamponade, mitral valve surgery in papillary muscle rupture. In case of inter-ventricular septum rupture, MCS as BTT should be considered. [b]Other causes include acute valve regurgitation, pulmonary embolism, infection, acute myocarditis, arrhythmia

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Table 2.3: Intravenous vasodilators used to treat acute heart failure

Vasodilator	*Dosing*	*Main side effects*	*Other*
Nitroglycerine	Start with 10–20 µg/min, increase up to 200 µg/min	Hypotension, headache	Tolerance on continuous use
Isosorbide dinitrate	Start with 1 mg/h, increase up to 10 mg/h	Hypotension, headache	Tolerance on continuous use
Nitroprusside	Start with 0.3 µg/kg/min and increase up to 5 µg/kg/min	Hypotension, isocyanate toxicity	Light sensitive

pulmonary oedema. However, they can cause respiratory depression and potentiate the need for invasive ventilation. Their use is not routine and should be considered cautiously.[4] Furthermore, prompt correction of acidosis, optimisation of cardiac preload with balanced crystalloid challenges where clinically indicated and optimising afterload, need to be given

Table 2.4: Summary of the effects of positive inotropes and/or vasopressors used to treat acute heart failure

Medication	*Bolus dose*	*Infusion Rate 0.05–0.5 µg/kg/min unless stated*	*Effect*	*Receptors*				
				Alpha 1	*Beta 1*	*Beta 2*	*Dopamine*	*V1*
Catecholamines								
Epinephrine	Bolus: 1 mg can be given IV during resuscitation, repeated every 3–5 min	0.01–0.1 0.1–0.5	↑ SVR, ↑↑ CO ↑↑ SVR, ↑ CO					
Norepinephrine	No	0.2–1.0	↑↑ SVR, ↑ CO					
Dopamine	No	0.5–2 5–10 >10	↑ CO ↑↑ CO, ↑ SVR ↑↑ SVR, ↑ CO	 + +++	+ +++ ++	 + 	+++ ++ ++	
Dobutamine	No	2–20	↑↑ CO, ↓ SVR, ↓ PVR	+	++++	++		
Phosphodiesterase inhibitors								
Milrinone	25–75 µg/kg over 10–20 min	0.375–0.75	↑ CO, ↓ SVR, ↓ PVR					
Calcium sensitisers								
Levosimendan	12 µg/kg over 10 min (optional)	0.1, which can be decreased to 0.05 or increased to 0.2	↑ CO, ↓ SVR, ↓ PVR					
Vasoconstrictors								
Phenylephrine		0.1–10	↑↑ SVR	++++				
Vasopressin	No	0.02–0.04 U/min	↑↑ SVR ↔ PVR					++++

↑, Increase; ↑↑, Significant increase; ↓, Decrease; ↔, No effect or modest decrease; +, Increase
CO, cardiac output; PVR, peripheral vascular resistance; SVR, systemic vascular resistance; V1, vasopressin one receptor.