Advances in the management of acute decompensated heart failure

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ABSTRACT

Heart failure is a common condition that affects millions of people worldwide and is associated with substantial morbidity and mortality. Acute decompensated heart failure refers to the worsening of symptoms that requires changes in drugs or the start of new treatments such as non-invasive positive pressure ventilation. This review summarizes the current data and provides an evidence based approach to the diagnosis and management of acute decompensated heart failure. The review discusses common nomenclature and classifications of the condition, followed by the diagnostic accuracy of medical history, physical examination, electrocardiography, radiographs of the chest, point-of-care ultrasound, and laboratory testing. Current and emerging medical treatments are also discussed.

Introduction

Heart failure affects millions of people worldwide, leading to substantial morbidity and mortality. Progressive worsening of symptoms, referred to as acute decompensated heart failure (ADHF), is associated with a high rate of unplanned emergency department visits, frequent hospital admissions, and increased risk of mortality.[1] The approach used to diagnose and manage ADHF has evolved over time, and there is a need to understand the current state of the science. In this review, we discuss the most important aspects of ADHF care, focusing on epidemiology, nomenclature, diagnosis, management, risk stratification, and disposition. This review is intended for all clinicians who care for patients with heart failure in the acute care setting.

Sources and selection criteria

We searched PubMed and the Cochrane Database of Systematic Reviews for articles published from inception to 1 April 2025 to identify studies reporting on the diagnosis, management, and disposition of ADHF. We evaluated retrospective and prospective studies, randomized controlled trials, systematic reviews and meta-analyses, and guidelines. When available, systematic reviews and meta-analyses were preferentially selected. These were followed sequentially by randomized controlled trials, prospective studies, retrospective studies, and case series.

Epidemiology

Heart failure is a common condition, affecting over 56 million people worldwide.[2] The risk of heart failure increases with age, with a 20-45% lifetime prevalence in people older than 45 years.[3] Although the overall incidence of heart failure has stabilized or declined in some regions,[2] the absolute number of patients with ADHF remains substantial owing to the aging population and improved survival rates from other cardiovascular conditions.[2 4 5] This trend contributes to a growing prevalence of heart failure across Europe and the United States.

In the US, heart failure is the primary or secondary diagnosis for one to three million hospital admissions each year, accounting for up to 2% of the total healthcare budget.[6] By 2030, more than eight million people (one in 33) will have heart failure.[6]

Approximately 1.4% of all emergency department visits are for heart failure, with a rising incidence over time.[7] Among those presenting to the emergency department, nearly three quarters are admitted to hospital.[7] Most people admitted to hospital with ADHF will have a known history of heart failure, with one study reporting an outpatient heart failure diagnosis in 73.4% and treatment for heart failure in 64.9%.[8] Inpatient mortality ranges from 4% to 12% and can be as high as 25% in patients at high risk.[6 9 10] The one year survival rate among those with heart failure is 86.5%, which has been declining over the past decade.[11 12] Among those requiring hospital admission for ADHF, the mortality rate markedly increases.[5] Moreover, there are substantial public health implications, with overall direct costs related to heart failure exceeding $30 billion (£22.3 billion; €25.5 billion) annually and indirect costs (owing to lost economic productivity) exceeding $14 billion in the US.[13 14] Another study reported the global economic burden exceeded $108 billion annually, with approximately $65 billion in direct costs and $43 billion in indirect costs.[15] A systematic review of 16 international studies reported the lifetime cost for patients with heart failure has been estimated to be at least $126 819 per patient.[16]

Importantly, research has shown inequalities in heart failure, with higher rates among women and black patients.[17-21] Among those with ADHF, black patients are more likely to be admitted to hospital and have higher rates of morbidity and mortality.[8 18 22 23] Heart failure has also shown a disproportionate rise in low and middle income countries, with reduced rates of starting goal directed medical treatment after discharge and higher one year mortality rates.[24 25]

Nomenclature

Heart failure is a complex clinical syndrome characterized by inability of the heart to adequately pump sufficient blood to meet the body's metabolic needs, occurring at rest, with exertion, or because of increased filling pressures. Primary symptoms (shortness of breath, edema, and fatigue) are caused by functional or structural cardiac damage. As symptoms progress, patients may notice reduced exercise tolerance or symptoms of fluid retention (eg, pulmonary congestion, splanchnic congestion, peripheral edema).[26] Heart failure can be classified by several different and inter-related categories, including side of involvement, ejection fraction, and acuity. Table 1 presents a list of definitions for several common categories of heart failure. For the purposes of this review, we focus on ADHF with an emphasis on left sided heart failure.

Traditionally, ADHF was defined by three main components: worsening symptoms, treatment location requiring urgent or inpatient care, and the need for escalated care involving intravenous or invasive treatments.[26] However, a new proposed definition emphasizes that it is not the acuity or the location that defines decompensated heart failure, but rather the need for intensified or escalated treatments.[27] Decompensated heart failure is characterized by the active deterioration of heart failure symptoms despite attempts to optimize heart failure treatment, necessitating intensified or rescue treatments beyond standard heart failure treatment. This definition does not specify a location (such as admission to hospital, urgent care, or emergency department visit). This change aims to align the definition with patient profiles and clinical care, providing better guidance in clinical trial design and regulatory approval processes.[27] In this review, we use the term ADHF because it remains the most common terminology; however, it is important to note that timing is not always sudden onset and can include those with more gradual progression.

Although there are several terminologies for heart failure, patients may present with a variety of signs and symptoms, and the European Society of Cardiology classifies these various presentations into several categories, including ADHF, acute pulmonary edema, right ventricular failure, and cardiogenic shock.[28] These categories are based on phenotypes that incorporate peripheral perfusion (normal perfusion or "warm" *v* hypoperfusion or "cold") and congestion (congestion or "wet" *v* no congestion or "dry"). The combination of these includes "warm and wet" (normal perfusion but congested, 70% of patients), "cold and wet" (hypoperfused and congested, 20%), "cold and dry" (hypoperfused but no congestion, <2%), and "warm and dry" (normal perfusion without congestion, <10%).[29]

Patients with ADHF present because of fluid accumulation associated with left ventricular dysfunction with renal sodium and water retention. As cardiac output drops from myocardial injury or stress, a neurohormonal mediated cascade including activation of the renin-angiotensin-aldosterone and sympathetic nervous systems occurs. The clinical effects of this neurohormonal activation are sodium and water retention and increased systemic vascular resistance. These maintain blood pressure and perfusion, but increase myocardial workload, wall tension, and myocardial oxygen demand. The onset is typically gradual (days) with low or normal cardiac output. Patients may present with warm and wet or cold and dry phenotypes.

Table 1 | Common terminology for heart failure[26 27]

Nomenclature	Definition
Left sided heart failure	Failure of the left ventricle to effectively pump blood, leading to pulmonary congestion (eg, rales, dyspnea, orthopnea) and reduced systemic perfusion
Right sided heart failure	Failure of the right ventricle to effectively pump blood, leading to systemic congestion (eg, peripheral edema, hepatomegaly, jugular venous distension)
Biventricular heart failure	Failure of the left and right ventricles, leading to both pulmonary and systemic congestion
Heart failure with preserved ejection fraction (HFpEF)	Left ventricular heart failure characterized by impaired relaxation and filling, but a normal ejection fraction (≥50%)
Heart failure with mildly reduced ejection fraction (HFmrEF)	Left ventricular heart failure characterized by a mildly reduced ejection fraction (41-49%), considered an intermediate category between HFpEF and HFrEF
Heart failure with reduced ejection fraction (HFrEF)	Left ventricular heart failure characterized by impaired contractility and a reduced ejection fraction (≤40%)
Systolic heart failure	Heart failure characterized by reduced myocardial contractility. This is an older term that has been replaced by HFrEF and HFmrEF
Diastolic heart failure	Heart failure characterized by impaired relaxation and filling. This is an older term that has been replaced by HFpEF
Congestive heart failure	Heart failure accompanied by pulmonary or systemic congestion. This is an older term that is no longer recommended
Acute decompensated heart failure (ADHF)	A sudden worsening of chronic heart failure accompanied by pulmonary or systemic congestion
Decompensated heart failure	Active deterioration of heart failure with symptoms necessitating intensified or rescue treatments beyond standard heart failure treatment
Sympathetic crashing acute pulmonary edema	A hyperacute version of ADHF where sympathetic overactivation leads to hypertension, pulmonary edema, and respiratory distress
Cardiogenic shock	Heart failure resulting in tissue hypoperfusion

Box 1: Differential diagnosis for acute dyspnea

Cardiac causes
- Acute coronary syndrome
- Acute decompensated heart failure
- Arrhythmias
- Constrictive pericarditis
- Myocarditis
- Pericardial effusion/cardiac tamponade
- Takotsubo cardiomyopathy
- Valvular dysfunction (eg, aortic stenosis, mitral regurgitation)

Pulmonary causes
- Acute respiratory distress syndrome
- Asthma
- Chronic obstructive pulmonary disease
- Interstitial lung disease
- Pleural effusion
- Pneumonia
- Pneumothorax
- Pulmonary embolism

Other causes
- Anemia
- Foreign body aspiration
- Hypoalbuminemia
- Metabolic disorders (eg, acidosis, sepsis, thyrotoxicosis)
- Neuromuscular disorders (eg, Guillain-Barré, myasthenia gravis, amyotrophic lateral sclerosis)
- Non-cardiogenic pulmonary edema (eg, high altitude pulmonary edema, toxin induced)
- Panic attack
- Toxic inhalation

Acute pulmonary edema is the second presentation category and is caused by fluid redistribution into the lungs from increased afterload, left ventricular diastolic dysfunction, or valvular heart disease. The onset is typically more rapid (hours) with normal cardiac output and normal to high systolic blood pressure. Acute pulmonary edema can be abrupt, severely symptomatic, and rapidly fatal if it goes untreated. Patients present with a warm and wet phenotype.

Isolated right ventricular failure is the third category and presents because of right ventricular dysfunction or precapillary pulmonary hypertension with increased central venous pressure. The onset may be gradual or rapid. Patients present with cold and dry or cold and wet phenotypes.

The final category is cardiogenic shock caused by severe cardiac dysfunction and systemic hypoperfusion, associated with low cardiac output and blood pressure. The onset can be gradual or rapid, and patients typically present with a cold and wet phenotype, though they may also present as cold and dry.[28] Importantly, the blood pressure should be contextualized by their baseline values because some patients may have proportionally low blood pressure despite not meeting a traditional numerical threshold such as mean arterial pressure <65 mm Hg.

One study evaluated these clinical phenotypes and their association with patient management and outcomes, and found most patients had a warm and wet phenotype (76%), followed by cold and wet (17.1%).[30] Patients presenting with cold phenotypes had higher hospital admission and mortality rates compared with other phenotypes. This is consistent with other studies finding warm and wet to be the most common presenting phenotype, while those with cold phenotypes have the highest mortality rates.[9 30-32]

Diagnosis

History and physical examination

The evaluation should begin with a detailed history and focused physical examination. This should include history of heart failure (including ejection fraction and last echocardiogram, if known), drugs taken (including recent changes or missed doses), dietary changes, weight changes, and relevant associated medical conditions (eg, coronary artery disease, hyperthyroidism). Assessment of acute symptoms should include the time course and severity, comparison with previous episodes of ADHF, and any interventions attempted (eg, increases in diuretic dosing). Dyspnea should be quantified using changes in level of orthopnea and distances walked before experiencing dyspnea. Changes in total body weight and urine output should also be noted. Because heart failure can overlap with other conditions (box 1), it is important to use a combination of historical features, physical examination findings, and testing to determine whether ADHF is the cause of the patient's symptoms. Importantly, existing clinical decision tools for ADHF are intended to assess the risk of adverse outcomes to inform admission or discharge decisions, rather than to diagnose heart failure.[33]

There is no single diagnostic test for ADHF; the diagnosis is based on the combination of all clinical data. Table 2 presents a summary of the positive likelihood ratio and negative likelihood ratio for common history and physical examination findings. The presence of a third heart sound or ventricular filling gallop (S3) helps to rule in ADHF (positive likelihood ratio 4.0), while the presence of fever assists with ruling it out (negative likelihood ratio 0.4).[34] The PREDICA trial identified predictive criteria from the history and physical examination to improve the diagnosis accuracy of patients presenting to the emergency department with dyspnea.[35] The trial showed that an emergency physician's gestalt (their clinical judgment about the probability of ADHF) was a strong predictor of correct diagnosis.

Evaluation for precipitating factors could also help diagnosis. These factors include drug or diet non-adherence (excess salt or fluid intake, unable to fill the drug prescriptions or take as recommended), renal failure (especially missed dialysis), poorly controlled hypertension, iatrogenic (recent addition of negative inotropic drugs, starting salt retaining drugs such as non-steroidal anti-inflammatory drugs, steroids, thiazolidinediones, inappropriate treatment reduction, or new dysrhythmic agents),

Table 2 | Pooled likelihood ratios for history and physical examination findings in acute decompensated heart failure[34]

Component	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Past medical history				
Atrial fibrillation	6 (1935)	51.9 (49.8 to 54.2)	2.1 (1.6 to 2.9)	0.82 (0.71 to 0.93)
Arrhythmia	5 (3469)	40.2 (38.6 to 41.9)	2.7 (2.2 to 3.4)	0.75 (0.68 to 0.83)
Coronary artery disease	14 (4983)	42.9 (41.5 to 44.3)	2.0 (1.7 to 2.4)	0.71 (0.64 to 0.79)
Chronic kidney disease	6 (3009)	42.8 (41.0 to 44.6)	3.4 (2.7 to 4.5)	0.75 (0.71 to 0.80)
Diabetes mellitus	19 (7707)	47.3 (46.2 to 48.4)	1.5 (1.3 to 1.7)	0.89 (0.84 to 0.94)
Heart failure	22 (8493)	46.0 (44.9 to 47.0)	2.7 (2.0 to 3.7)	0.58 (0.49 to 0.68)
Hyperlipidemia	5 (2923)	39.8 (38.1 to 41.6)	1.6 (1.3 to 1.9)	0.85 (0.82 to 0.90)
Hypertension	25 (10 137)	45.6 (44.6 to 46.6)	1.3 (1.3 to 1.4)	0.62 (0.53 to 0.73)
No history of COPD	18 (8053)	42.8 (41.7 to 43.9)	1.2 (1.1 to 1.4)	0.7 (0.6 to 0.8)
Previous myocardial infarction	9 (4208)	40.5 (39.1 to 42.0)	2.1 (1.8 to 2.5)	0.82 (0.76 to 0.89)
Renal failure	5 (2840)	40.9 (39.1 to 42.7)	2.3 (1.3 to 3.9)	0.9 (0.73 to 1.11)
Symptoms				
Absence of productive cough	7 (2414)	43.0 (41.0 to 45.0)	1.13 (1.02 to 1.26)	0.6 (0.5 to 0.8)
Dyspnea at rest	4 (2038)	37.9 (35.9 to 40.0)	1.1 (0.9 to 1.4)	0.88 (0.74 to 1.04)
Orthopnea	15 (5430)	45.5 (44.2 to 46.9)	1.9 (1.4 to 2.5)	0.74 (0.64 to 0.85)
Paroxysmal nocturnal dyspnea;	9 (2216)	44.8 (42.8 to 46.9)	1.6 (1.2 to 2.1)	0.79 (0.71 to 0.88)
Physical examination findings				
Absent fever	7 (3197)	43.6 (41.9 to 45.3)	1.14 (1.02 to 1.27)	0.4 (0.3 to 0.6)
Hepatojugular reflux	4 (1209)	60.4 (57.6 to 63.1)	2.2 (1.3 to 3.7)	0.91 (0.88 to 0.94)
Jugular venous distension	23 (8012)	47.8 (46.7 to 48.9)	2.8 (1.7 to 4.5)	0.76 (0.69 to 0.84)
Leg edema	26 (9626)	47.2 (46.2 to 48.2)	1.9 (1.6 to 2.3)	0.68 (0.61 to 0.75)
Murmur	8 (4004)	45.3 (43.8 to 46.8)	1.9 (0.9 to 3.8)	0.93 (0.79 to 1.08)
Rales	22 (8775)	48.2 (47.1 to 49.2)	1.8 (1.5 to 2.1)	0.60 (0.51 to 0.69)
S3	14 (5900)	45.2 (44.0 to 46.5)	4.0 (2.7 to 5.9)	0.91 (0.88 to 0.95)
Wheezing	13 (6970)	44.2 (43.0 to 45.3)	0.6 (0.5 to 0.8)	1.19 (1.10 to 1.30)

ADHF=acute decompensated heart failure; CI=confidence interval; COPD=chronic obstructive pulmonary disease; S3=presence of a third heart sound or ventricular filling gallop.

and substance abuse (cocaine, methamphetamines, ethanol).

Electrocardiography

Although electrocardiogram findings are not effective in confirming or excluding ADHF, all patients with ADHF should undergo electrocardiography to help identify alternative causes (eg, ST elevation myocardial infarction, pericarditis) and evaluate for dysrhythmias requiring targeted interventions for heart rate control. Table 3 includes the likelihood ratios for electrocardiogram findings in ADHF.

Chest radiography

Chest radiographs are commonly ordered in ADHF to identify the presence and degree of pulmonary edema, and to assess for alternative causes (eg, pleural effusion, pneumonia, pneumothorax).[7] The progression of heart failure on chest radiography has been proposed to follow three stages.[36] Stage 1 consists of redistribution of pulmonary vessels, increased cardiothoracic ratio, and a broad vascular pedicle. Stage 2 involves interstitial edema, which includes Kerley B-lines, peribronchial cuffing, hazy contour of vessels, and subpleural edema. Stage 3 involves alveolar edema, which can present with consolidations, a butterfly appearance, cotton wool appearance, and pleural effusions (fig 1). Although these findings are modestly specific for ADHF, their absence does not exclude the diagnosis.[37] Approximately 20% of patients attending the emergency department subsequently diagnosed with ADHF have chest radiographs without evidence of congestion.[37] Table 4 includes the likelihood ratios for chest radiography findings in ADHF. Interstitial edema (positive likelihood ratio 6.4) and Kerley B-lines (positive likelihood ratio 6.5) increase the probability of heart failure.[34] The absence of any of these findings does not significantly decrease the probability of ADHF.

Table 3 | Pooled likelihood ratios for electrocardiogram findings in acute decompensated heart failure[34]

Finding	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Atrial fibrillation	6 (2242)	55.8 (53.7 to 57.8)	2.2 (1.4 to 3.5)	0.88 (0.85 to 0.91)
Ischemic changes	2 (1138)	42.6 (39.8 to 45.5)	2.9 (1.2 to 7.1)	0.78 (0.73 to 0.84)
Normal sinus rhythm	3 (1207)	39.6 (36.9 to 42.4)	0.7 (0.5 to 0.9)	2.88 (1.26 to 6.57)
ST depression	2 (1024)	60.8 (57.8 to 63.8)	2.0 (1.0 to 3.8)	0.97 (0.95 to 1.00)
ST elevation	1 (219)	61.2 (54.6 to 67.4)	0.6 (0.2 to 1.7)	1.03 (0.96 to 1.11)
T wave inversion	1 (709)	69.4 (65.9 to 72.7)	2.4 (1.2 to 4.8)	0.94 (0.90 to 0.98)

ADHF=acute decompensated heart failure; CI=confidence interval.

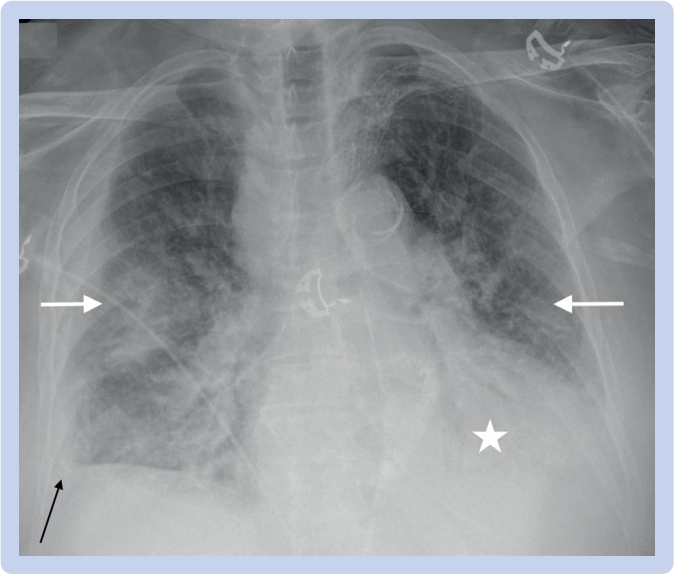

Fig 1 | Chest radiograph with bilateral interstitial infiltrates (white arrows), bilateral pleural effusions (black arrow), and cardiomegaly (star)

Point-of-care ultrasound

Point-of-care ultrasound (POCUS) is a valuable tool allowing for rapid bedside diagnosis. This test can help to reveal emergent causes of dyspnea, such as cardiac tamponade or pulmonary embolism (eg, evidence of acute right heart strain), and can estimate left ventricular function and volume status.[38]

Lung POCUS involves assessment for pulmonary edema, which is defined as the presence of at least three B-lines (hyperechoic imaging artefacts extending two thirds of the length of the ultrasound screen) involving at least two areas bilaterally (fig 2, video 1).[39] A systematic review and meta-analysis showed lung POCUS is more sensitive (91.8% *v* 76.5%) and more specific (92.3% *v* 87.0%) than chest radiography for detecting pulmonary edema in ADHF.[40] Another systematic review of six studies comparing POCUS and chest radiography showed better diagnostic test accuracy of POCUS compared with chest radiography. Chest radiography had a positive likelihood ratio of 7.36 and a negative likelihood ratio of 0.30, while POCUS had a positive likelihood ratio of 8.63 and a negative likelihood ratio of 0.14.[41] Because bilateral B-lines can be found in conditions not caused by pulmonary edema (eg, pulmonary fibrosis, pulmonary contusion, bilateral pneumonia), rapid assessment for raised central venous pressure may follow. An inferior vena cava diameter >2 cm or collapsibility index of <30% is indicative of raised central venous pressure.[42]

Cardiac POCUS can be used in a complementary manner to determine the ejection fraction and diastolic dysfunction, as well as alternative causes such as pericardial effusion or right ventricular dysfunction from pulmonary embolism.[43-45] With training, emergency physicians have reasonable agreement with cardiology interpretations by classifying a visual POCUS estimation of left ventricular ejection fraction into broad categories of normal, moderately reduced, and severely reduced (video 2).[46] More recent data have also shown the role of artificial intelligence to overcome the user dependent nature of POCUS, resulting in high image quality and diagnostic accuracy among more inexperienced users.[47-49] In this capacity, artificial intelligence can assist with image acquisition, image interpretation, and automated measurements. However, while POCUS is valuable in the acute setting, many patients may benefit from subsequent comprehensive transthoracic echocardiography in a delayed fashion. Table 5 includes the likelihood ratios for POCUS findings in ADHF.

Laboratory testing

Laboratory testing can be beneficial to identify potential causes and complications of ADHF. Common testing includes a complete blood count, electrolytes, creatinine, liver function testing, troponin, and B-type natriuretic peptide (BNP) or N-terminal proB-type natriuretic peptide (NT-proBNP). The complete blood count can evaluate for anemia as a potential mimic of ADHF. Electrolytes are valuable because many diuretics can cause electrolyte imbalances. Creatinine and liver function testing can identify renal impairment and hepatic congestion, respectively, which can influence the differential diagnosis and inform prognosis. Troponin can be useful for determining whether acute coronary syndrome is present in patients with suggestive symptoms, as well as for prognosis. Notably, troponin can also be raised owing to demand ischemia and should not automatically lead to invasive coronary angiography in the absence

Table 4 | Pooled likelihood ratios for chest radiograph findings in acute decompensated heart failure[34]

Finding	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Alveolar edema	3 (2001)	48.3 (46.2 to 50.5)	5.3 (3.3 to 8.5)	0.95 (0.94 to 0.97)
Cephalization	5 (1338)	54.0 (51.3 to 56.6)	5.6 (2.9 to 10.4)	0.53 (0.39 to 0.072)
Enlarged cardiac silhouette	12 (3515)	51.7 (49.4 to 52.7)	2.3 (1.6 to 3.4)	0.43 (0.36 to 0.51)
Kerley B-lines	2 (814)	46.8 (43.4 to 50.2)	6.5 (2.6 to 16.2)	0.88 (0.69 to 1.33)
Interstitial edema	3 (2001)	48.3 (46.2 to 50.5)	6.4 (3.4 to 12.2)	0.73 (0.68 to 0.78)
Pleural effusion	5 (1326)	55.1 (52.4 to 57.8)	2.4 (1.6 to 3.6)	0.89 (0.80 to 0.99)
Pulmonary edema	15 (4393)	46.6 (45.1 to 48.1)	4.8 (3.6 to 6.4)	0.48 (0.39 to 0.58)

ADHF=acute decompensated heart failure; CI=confidence interval.

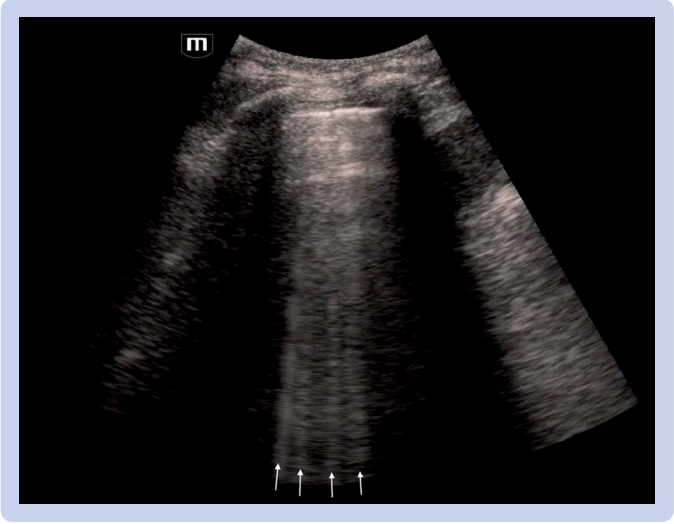

Fig 2 | B-lines (white arrows) on point-of-care cardiac ultrasound suggestive of pulmonary edema

of other findings concerning for acute coronary syndrome.[50]

A raised BNP can suggest volume overload in patients with an unclear clinical picture. However, despite the established use of natriuretic peptide testing, interpretation of results can be challenging in certain patients. Levels can be affected by age, sex, body mass, sacubitril or valsartan treatment, and may have delayed increases in patients with flash pulmonary edema.[51] Dyspnea and modest BNP increases may be seen in pulmonary hypertension, pulmonary embolism, pneumonia, sepsis, and renal failure.[52] As many as 25% of patients fall into the diagnostic "gray zone" (100-500 pg/mL for BNP), confounding test interpretation.[53] Heart failure can largely be excluded in patients with acute dyspnea and NT-proBNP <300 pg/mL or BNP <100 pg/mL. NT-proBNP has age specific cutoffs to further increase accuracy, with cutoff levels of 450, 900, and 1800 pg/mL in patients aged <50, 50-75, and >75 years, respectively.[53] BNP or NT-proBNP testing is best used when diagnostic uncertainty occurs and as an addition to the clinical assessment, rather than in isolation. Results may also be useful for comparison with inpatient testing in a serial fashion.[54 55] Table 6 and table 7 present interval likelihood ratios for BNP and NT-proBNP from a systematic review with individual patient level meta-analysis.[34]

Management

The initial management of ADHF should include hemodynamic stabilization and symptom relief.[32 56] Delays in diagnosis and treatment can worsen morbidity and mortality, with data suggesting an adjusted odds of death increasing by 6.8% for each six hour delay in treatment.[57] Figure 3 summarizes the management of ADHF.

Non-invasive ventilation

Evaluation of oxygenation and respiratory status should be the immediate first step in the emergency department. Non-invasive positive pressure ventilation (NIPPV, including continuous positive airway pressure and bilevel positive airway pressure) should be started rapidly in those presenting with acute respiratory distress to improve oxygenation and reduce work of breathing.[58] Successful NIPPV requires hemodynamic stability, facial anatomy allowing a facemask seal, monitoring, and patient cooperation. NIPPV has been shown to reduce hospital mortality (risk ratio 0.65, 95% confidence interval 0.51 to 0.82; number needed to treat 17) and rates of endotracheal intubation (risk ratio 0.49, 95% confidence interval 0.38 to 0.62; number needed to treat 13), with no difference in adverse events.[59] Randomized trial data have not shown a difference in mortality, endotracheal intubation, myocardial infarction, or length of hospital stay between continuous positive airway pressure and bilevel positive airway pressure.[60] In patients who cannot tolerate NIPPV, high flow nasal cannula may be considered.[61] A recent meta-analysis reported that high flow nasal cannula reduced rates of intubation compared with conventional oxygen treatment (risk ratio 0.31, 95% confidence interval 0.16 to 0.59).[62]

Nitroglycerin

For patients with adequate blood pressure, intravenous vasodilators should be used to reduce afterload and optimize preload, thereby improving symptoms and reducing congestion. These agents are particularly useful in patients with severe hypertension or acute pulmonary edema.[63-65] Nitroglycerin is the drug of choice in patients with ADHF and hypertension (defined as a systolic blood pressure >160 mm Hg).[65-67] An initial dose of 400 µg sublingually (tablets or spray) can be given while obtaining intravenous access. Once intravenous access is established, a nitroglycerin infusion should be started. Studies have shown that an initial high

Table 5 | Pooled likelihood ratios for POCUS in acute decompensated heart failure[34]

Finding	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Positive B-lines	8 (1914)	48.2 (46.0 to 50.5)	7.4 (4.2 to 12.8)	0.16 (0.05 to 0.51)
Pleural effusion	2 (155)	40.7 (33.2 to 48.5)	2.0 (1.4 to 2.8)	0.49 (0.22 to 1.10)
Restrictive mitral pattern	1 (125)	43.2 (34.9 to 52.0)	8.3 (4.0 to 16.9)	0.21 (0.12 to 0.36)
Reduced ejection fraction	3 (325)	41.2 (36.0 to 46.7)	4.1 (2.4 to 7.2)	0.24 (0.17 to 0.35)
Increased left ventricular end diastolic dimension	1 (84)	58.3 (47.7 to 68.3)	2.5 (1.5 to 4.2)	0.30 (0.16 to 0.54)

ADHF=acute decompensated heart failure; CI=confidence interval.

Table 6 | Interval likelihood ratios for BNP in acute decompensated heart failure (n=2202)[34]

BNP value (pg/mL)	No of patients (%)	Interval likelihood ratio (95% CI)
0-100	617 (28)	0.14 (0.12 to 0.18)
100-200	308 (14)	0.29 (0.23 to 0.38)
200-300	188 (9)	0.89 (0.67 to 1.17)
300-400	148 (7)	1.34 (0.98 to 1.83)
400-500	148 (7)	2.05 (1.47 to 2.84)
500-600	115 (5)	3.50 (2.30 to 5.35)
600-800	218 (10)	4.13 (3.01 to 5.68)
800-1000	130 (6)	5.00 (3.21 to 7.89)
1000-1500	160 (7)	7.12 (4.53 to 11.18)
1500-2500	105 (5)	8.33 (4.60 to 15.12)
2500-5001	65 (3)	8.91 (4.09 to 19.43)

BNP=B-type natriuretic peptide; CI=confidence interval.

dose bolus of 1000-2000 µg is well tolerated and can lead to improved patient symptoms and oxygen saturation, and reduced rates of intensive care unit admission.[65-68] A starting intravenous infusion dose of 0.5-0.7 µg/kg/min is common and titrated every few minutes up to 200 µg/min based on blood pressure and symptoms. Patients should be closely monitored to prevent hypotension. Flow limiting, preload dependent states such as aortic stenosis, right ventricular infarction, and hypertrophic cardiomyopathy, and patients with volume depletion are at increased risk of vasodilator associated hypotension.[69]

Other vasodilators

If additional arterial vasodilation is needed despite high dose nitroglycerin and NIPPV, intravenous angiotensin converting enzyme inhibitors or dihydropyridine calcium channel blockers may be considered. One retrospective study showed that intravenous enalaprilat 1.25 mg reduced systolic blood pressure by 30 mm Hg within three hours, with less than 2% of patients experiencing hypotension.[70] A separate retrospective study of intravenous nicardipine reported that all patients had a 30 mm Hg reduction in systolic blood pressure in a median of 18 minutes, with only one patient (2.6%) experiencing hypotension.[71] However, these agents should remain second line only after sufficiently titrated doses of nitroglycerin and NIPPV have been administered.[72]

Table 7 | Interval likelihood ratios for NT-proBNP in acute decompensated heart failure (n=2013)[34]

NT-proBNP value (pg/mL)	No of patients (%)	Interval likelihood ratio (95% CI)
0-100	150 (7.5)	0.09 (0.05 to 0.17)
100-300	205 (10.2)	0.23 (0.16 to 0.33)
300-600	212 (10.5)	0.28 (0.20 to 0.39)
600-900	151 (7.5)	0.63 (0.46 to 0.87)
900-1500	249 (12.4)	0.84 (0.67 to 1.06)
1500-3000	273 (13.6)	1.49 (1.19 to 1.86)
3000-5000	225 (11.2)	2.36 (1.81 to 3.08)
5000-10 000	239 (11.9)	2.48 (1.91 to 3.21)
10 000-15 000	112 (5.6)	2.84 (1.90 to 4.23)
15 000-30 000	111 (5.5)	2.93 (1.95 to 4.39)
30 000-200 000	86 (4.3)	3.30 (2.05 to 5.31)

CI=confidence interval; NT-proBNP=N-terminal proB-type natriuretic peptide.

Nitroprusside dilates venous and arterial vessels, but is less preferred than the other agents because of increased risks of hypotension.[73 74] Owing to their mechanisms, intravenous vasodilators may be more effective than diuretics for patients with acute pulmonary edema caused by increased afterload and fluid redistribution to the lungs, even when there is minimal total body fluid accumulation.[32 64 65 75]

Two recent trials evaluating early intensive and sustained vasodilation showed no difference between intravenous vasodilators and high dose diuretics compared with usual care. The GALACTIC trial included 788 patients randomized to early, intensive and sustained vasodilation compared with usual care. The vasodilation strategy included sublingual and transdermal nitrates, low dose oral hydralazine for 48 hours, and rapid up-titration of angiotensin converting enzyme inhibitors, angiotensin receptor blockers, or sacubitril valsartan. There was no difference in their primary endpoint of composite all cause mortality or readmission within 180 days.[76] The ELISABETH trial was a stepped wedge, cluster randomized trial conducted in 15 emergency departments in France that included 503 patients randomized to a care bundle of intravenous nitrate boluses, management of precipitating factors, and intravenous diuretics compared with usual care. There was no difference in the number of days alive and out of the hospital within 30 days, or secondary outcomes of 30 day all cause mortality, cardiovascular mortality, readmission, length of hospital stay, or renal function deterioration.[77]

Diuretics

Among patients with fluid overload, diuretics increase the excretion of water and salt. Intravenous loop diuretics, such as furosemide, are usually administered. In the DOSE trial, there was no significant difference in patient oriented outcomes when furosemide was administered at a lower dose (a dose equivalent to the patient's oral dose) or a higher dose.[78] The high dose strategy was associated with greater diuresis and more favorable outcomes in some secondary measures, but was associated with a transient worsening of renal function. Therefore, if a patient is already on a diuretic regimen, the intravenous equivalent of double their home dose is a reasonable first approach, with adjustment based upon clinical response (table 8). If the patient presents with new onset heart failure or is not on maintenance diuretic treatment, intravenous furosemide 40 mg is an acceptable starting dose.[63]

The timing is controversial because some studies suggested rapid administration of diuresis within the first 60-90 minutes was associated with improved oxygenation and reduced mortality,[80-82] while others reported no difference in mortality.[83 84] A recent systematic review and meta-analysis reported no significant reduction in mortality in hospital, but did identify a 30 day mortality reduction among those receiving early intravenous diuresis (odds

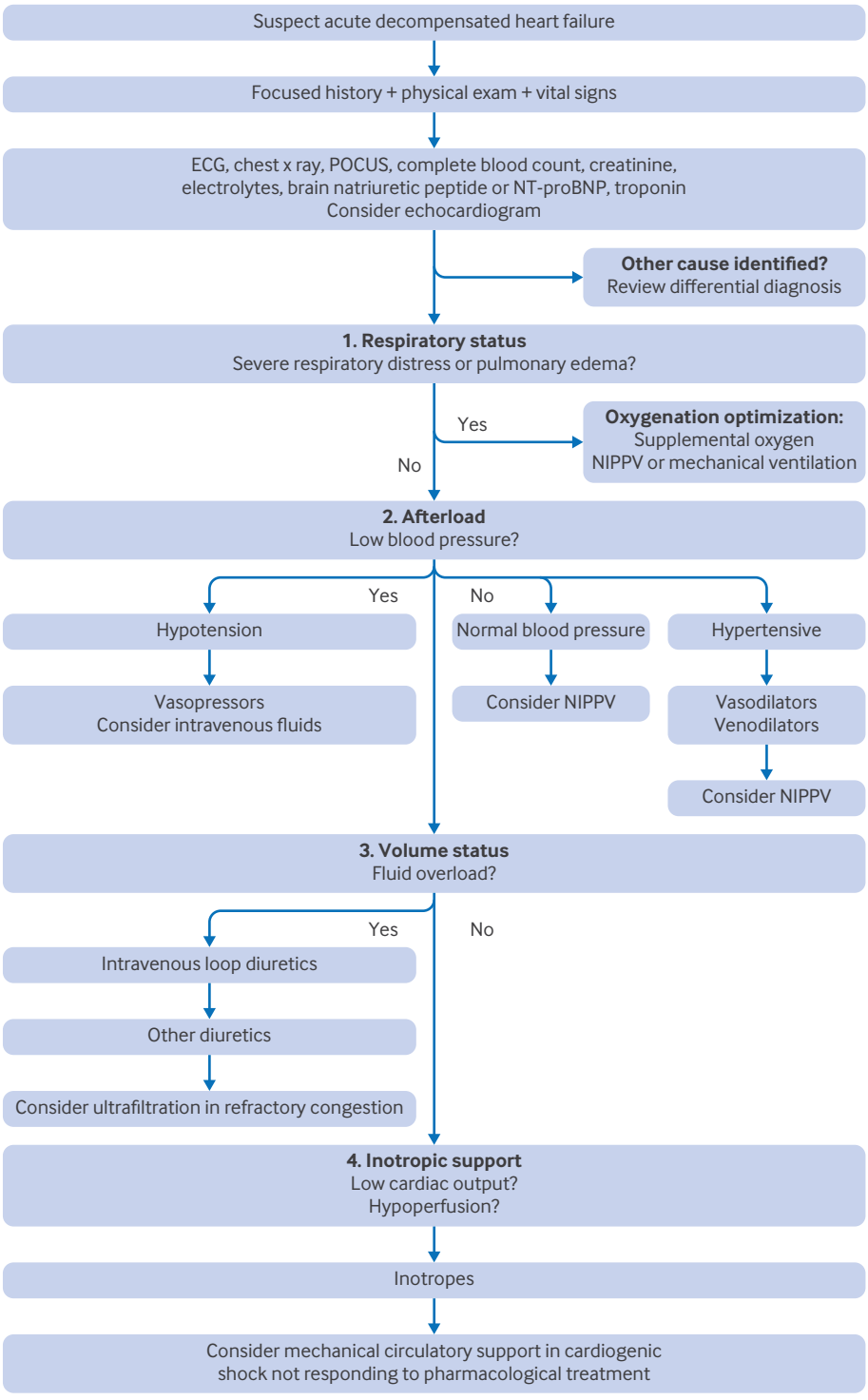

Fig 3 | Management of acute decompensated heart failure. ECG=electrocardiogram; NT-proBNP=N-terminal proB-type natriuretic peptide; NIPPV=non-invasive positive pressure ventilation; POCUS=point-of-care ultrasound

ratio 0.77, 95% confidence interval 0.64 to 0.93).[85] Diuretic response should be evaluated by monitoring urine output or urine sodium with a goal of 100-150 mL/h during the first six hours, or urine sodium content 50-70 mEq/L at two hours.[86 87] If response is inadequate, doses can be doubled, and if still insufficient, additional diuretics acting at different sites of the renal system (eg, thiazides, metolazone, acetazolamide) can be considered with careful monitoring of electrolytes and renal function.[63 88-90] The PUSH-AHF trial found urine sodium guided treatment was associated with improved natriuresis and diuresis, with no difference in adverse events.[91] Studies have also shown a beneficial effect of automated and nursing driven protocols with improved net fluid output and weight loss.[92 93]

Table 8 | Loop diuretic dosing conversion chart[79]

Loop diuretic	Oral (mg)	Intravenous (mg)	Loop diuretic furosemide equivalent
Furosemide	40	20	—
Bumetanide	1	1	Bumetanide 1 mg=furosemide 40 mg by mouth=furosemide 20 mg intravenously
Torsemide	20	20	Torsemide 20 mg=furosemide 40 mg by mouth=furosemide 20 mg intravenously

A recent randomized controlled trial suggested adding acetazolamide 500 mg daily to loop diuretics might increase urine output and expedite systemic decongestion through sequential nephron blockade.[94] The ADVOR trial was a multicenter, randomized, placebo controlled trial of 519 patients with acute heart failure and volume overload, with NT-proBNP levels >1000 pg/mL or BNP >250 pg/mL. Patients received intravenous acetazolamide (500 mg daily) or placebo alongside standard loop diuretics. Successful decongestion within three days was achieved in 42.2% of the acetazolamide group compared with 30.5% of the placebo group (risk ratio 1.46, 95% confidence interval 1.17 to 1.82). Readmission or all cause death occurred in 29.7% of the acetazolamide group and 27.8% of the placebo group (hazard ratio 1.07, 95% confidence interval 0.78 to 1.48). The hospital stay was one day shorter for patients receiving acetazolamide (8.8 days (95% confidence interval 8.0 to 9.5) *v* 9.9 days (9.1 to 10.8)). There were no differences in other outcomes or adverse events.

The CLOROTIC trial enrolled 230 patients with ADHF randomized to oral hydrochlorothiazide (25-100 mg daily) or placebo in addition to intravenous furosemide.[95] Hydrochlorothiazide led to greater weight loss (2.3 *v* 1.5 kg; P=0.002), but there was no difference in dyspnea symptoms. More patients on hydrochlorothiazide had increased serum creatinine (46.5% *v* 17.2%; P<0.001). Heart failure readmission, all cause death rates, and length of stay were similar.

Mineralocorticoid receptor antagonists like spironolactone or eplerenone are commonly used for chronic management because of their role in fluid retention and cardiac remodeling. Mineralocorticoid receptor antagonists can also be added in the acute setting as long as potassium and renal function are not a concern, although the evidence of effectiveness is mixed.[96]

Importantly, a subset of patients with ADHF may be isovolemic with the primary driver of symptoms being sympathetic overactivation resulting in acute hypertension and pulmonary edema. This group, referred to as sympathetic crashing acute pulmonary edema (SCAPE), benefits primarily from vasodilation and NIPPV and may not require routine diuresis.[97]

Ultrafiltration

In the setting of diuretic refractory fluid overload, ultrafiltration or renal replacement therapy may enable more rapid weight loss and reduce readmission for heart failure.[98] The UNLOAD trial compared veno-venous ultrafiltration versus standard intravenous diuresis among 200 patients admitted to hospital for ADHF with evidence of fluid overload and found the ultrafiltration group had more net fluid loss (4.6 *v* 3.3 L; P=0.001) by 48 hours and fewer readmissions for ADHF at 90 days (18% *v* 32%; P=0.037).[99] In contrast, CARRESS-HF randomized 188 patients with ADHF complicated by cardiorenal syndrome and persistent congestion to stepped pharmacological treatment versus ultrafiltration and reported no difference in net fluid loss at 96 hours.[100] Ultrafiltration also carries an increased risk of adverse events, including raised creatinine, bleeding complications, and intravenous catheter related complications.[100] A meta-analysis also found that ultrafiltration was associated with an increased risk of hypotension (odds ratio 2.39, 95% confidence interval 1.20 to 4.76).[98]

Vasopressors and inotropes

A subset of patients will have ADHF with cardiogenic shock, resulting in poor forward flow and hypotension. Cardiogenic shock is defined clinically as a life threatening hypotension with rapidly escalating inotropic or pressor support, and critical organ hypoperfusion (often confirmed by worsening acidosis and lactate levels).[26] Other criteria can include hypotension (systolic blood pressure <90 mm Hg or mean arterial pressure <60 mm Hg), low cardiac output (cardiac index <2.2 L/min/m^2), or pulmonary capillary wedge pressure >15 mm Hg.[26]

These patients often require a combination of vasopressors and inotropic agents.[75] Norepinephrine can be used as a first line agent based on its ability to provide vasoconstriction and inotropic benefits. Epinephrine can also be considered, though

Table 9 | Ottawa Heart Failure Risk Score

Input	Points
History	
History of stroke or transient ischemic attack	1
History of intubation for respiratory distress	2
Examination	
Heart rate on emergency department arrival ≥110	2
Room air oxygen saturation <90% on arrival	1
Heart rate ≥110 during 3 minute walk test after emergency department treatment (or too ill to perform)	1
Investigations	
Electrocardiogram with acute ischemic changes	2
Urea ≥12 mmol/L (33 mg/dL)	1
Serum CO_2 ≥25 mmol/L (mEq/L)	2
Troponin I or T raised to myocardial infarction level	2
NT-ProBNP ≥5000 ng/L	1

NT-ProBNP=N-terminal proB-type natriuretic peptide.
Risk of adverse event within 14 days: 0 points: 2.8% (low); 1-2 points: 5.1-9.8% (medium); 3-4 points: 15.9-26.1% (high); 5-9 points: 39.8-89% (very high).

Box 2: Emergency Heart Failure Mortality Risk Grade Criteria

- Age
- Systolic blood pressure
- Heart rate
- O_2 saturation
- Creatinine
- Potassium
- Transport by emergency medical services
- Troponin positive
- Active cancer
- On outpatient metolazone

one randomized trial of norepinephrine versus epinephrine in cardiogenic shock after myocardial infarction reported higher rates of refractory shock with epinephrine.[101] Inotropes may improve hemodynamics, reduce congestion, and increase cardiac output, thereby improving peripheral perfusion. Among patients with low cardiac output and peripheral hypoperfusion, requiring inotropic support, data suggest that milrinone or dobutamine are reasonable, with milrinone showing a slightly lower mortality rate in overall ADHF compared with dobutamine; however, no difference was seen in the subgroup with ADHF and cardiogenic shock.[102] Close communication with a heart failure specialist is recommended in patients with ADHF requiring vasopressors or inotropes.

Implementation

Importantly, implementation to practice remains a persistent challenge. Among patients admitted to hospital, use of guideline directed medical treatment remains underused during the hospital stay and upon discharge.[103] This can be influenced by time constraints, diagnostic uncertainty, resource limitations, or knowledge translation. To address these gaps, some experts have proposed a framework across the care continuum, including patients, clinicians, and public health initiatives.[104]

Box 3: Multiple Estimation of Risk Based on Spanish Emergency Department Score

- Barthel index at emergency department presentation
- Systolic blood pressure
- Age
- NT-proBNP
- Potassium
- NYHA class IV on presentation
- Positive troponin level
- Respiratory rate
- Low output symptoms
- O_2 saturation
- Episode associated with acute coronary syndrome
- Hypertrophy on electrocardiogram
- Creatinine

NT-ProBNP=N-terminal proB-type natriuretic peptide; NYHA=New York Heart Association

Disposition

Although most patients with ADHF are admitted to hospital, several risk stratification tools exist to identify patients at lower risk who may be appropriate for discharge.[7 105] The Ottawa Heart Failure Risk Score was developed to evaluate the risk of 14 day and 30 day adverse events among patients with ADHF who are older than 50 years and attending the emergency department (table 9).[33 106] The Emergency Heart Failure Mortality Risk Grade calculates a seven day mortality risk based on 10 risk factors using an online risk calculator (https://coachcalculator.ices.on.ca/#/; box 2).[107-109] The Multiple Estimation of Risk Based on Spanish Emergency Department Score predicts 30 day mortality in ADHF using an online calculator based on 13 criteria (https://meessi-ahf-risk.score-calculator-ica-semes.portalsemes.org/; box 3).[110 111] The HEARTRISK6 scale was published in 2024 and includes six criteria to predict 30 day adverse events or 14 day return visits (fig 4).[112] The STRATIFY risk stratification tool was developed, externally validated, and owing to its complexity, has been embedded into the electronic health record, requiring no calculation by clinicians.[113-116] It is designed to predict patients with ADHF at low risk for 30 day complications, and therefore potentially eligible for discharge consideration. Importantly, these tools should only serve as adjuncts to clinical decision making and should not replace it.

The COACH trial was a cross sectional, stepped wedge, cluster randomized trial with 5452 patients enrolled at 10 hospitals in Ontario, Canada, and used the Emergency Heart Failure Mortality Risk Grade 30 Day Mortality-ST Depression score.[117] Patients at low risk were recommended for early discharge (up to three days) with standardized outpatient care, while patients at intermediate and high risk were admitted. Despite similar early discharge rates (57% *v* 58%), the trial showed a 12% reduction in all cause death or admission to hospital for cardiovascular conditions within 30 days in the intervention group compared with the control group (adjusted hazard ratio 0.88, 95% confidence interval 0.78 to 0.99), indicating a positive effect of care after discharge. In the COACH trial, follow-up was with an internist or cardiologist for patients at low risk who were discharged early (median 6 days, interquartile range 3-12) compared with usual care (median 12 days, interquartile range 5-29).

The STRONG-HF trial emphasized the importance of early assessment and up-titration of heart failure treatments in patients discharged after being admitted to hospital for ADHF.[118] The trial included 1078 patients randomized to usual care versus high intensity care. High intensity care included early and rapid intensification of oral heart failure drugs such as angiotensin converting enzyme inhibitors (or angiotensin receptor blockers with or without neprilysin inhibitor), β blockers, and mineralocorticoid receptor antagonists. The primary outcome of heart failure readmission or all cause death at 180 days occurred in 15.2% of patients in

		Points
Initial assessment		
(a) History of valvular heart disease		1
(b) Heart rate		
(i) ≥100 bpm to <120 bpm		2
(ii) ≥120 bpm		3
(c) Treated with non-invasive ventilation		2
Investigations		
(a) Creatinine		
(i) ≥150 µmol/L to <300 µmol/L (≥1.7 mg/dL to <3.4 mg/dL)		2
(ii) ≥300 µmol/L (≥3.4 mg/dL)		3
(b) Troponin		
(i) ≥3x or 4x upper reference limit		1
(ii) ≥5x upper reference limit		2
Fails reassessment after ED treatment (2-6 hours)		
(a) Resting vital signs abnormal (O_2 saturation <90% on room air or usual O_2 heart rate ≥110 bpm, or respiratory rate ≥28) or (b) Unable to start or complete 3 min walk test		1
Risk of short term serious outcomes		
Total score	**Absolute risk (%)**	**Category**
0	6.4	Low
1	8.5	
2	11.3	Medium
3	14.9	
4	19.4	
5	24.8	
6	31.2	High
7	38.3	
8	46.0	
9	53.9	
≥10	61.6	

Fig 4 | HEARTRISK6 scale. bpm=beats per minute; ED=emergency department

the high intensity care group versus 23.3% in the usual care group. Readmissions were reduced in the high intensity group and there was no significant difference in all cause mortality by day 180 or rates of serious adverse events in both groups.

On discharge, it is important to ensure patients have close primary care or cardiology follow-up for re-evaluation and medical optimization. This should include dietary and exercise counseling, drug adjustment (eg, diuretics, antihypertensive agents), and referral for invasive procedures (eg, implantable cardiac defibrillators, cardiac resynchronization treatment) if appropriate. Although beyond the scope of this paper, an in-depth review of modern management of chronic heart failure in the outpatient setting is available from Heidenreich and Sandhu.[119] Before discharge, all patients should have an understanding of the findings, follow-up plan, and indications to return.

Emerging treatments

Sodium-glucose cotransporter 2 inhibitors

Sodium-glucose cotransporter 2 inhibitors (eg, canagliflozin, dapagliflozin, empagliflozin, ertugliflozin) are glucose lowering agents that block the sodium-glucose cotransporter 2 protein located in the proximal convoluted tubule of the nephron for adults with diabetes mellitus. Recent research has proposed a potential role for management in ADHF. EMPULSE was a randomized trial of empagliflozin in ADHF.[120] The primary endpoint was clinical benefit using a composite measure of death, number of heart failure events, and symptom score change at 90 days. Heart failure events included hospital admissions

or urgent, unplanned outpatient visits that required intensification of treatment. Patients were randomized within three days of hospital admission and treated for 90 days. The primary endpoint was achieved more in patients treated with empagliflozin than placebo (stratified win ratio 1.36, P=0.005). Efficacy was independent of ejection fraction and diabetes status. Adverse events rates were similar between groups. A subsequent meta-analysis of randomized controlled trials found early initiation of sodium-glucose cotransporter 2 inhibitors in ADHF was associated with reduced rates of hospital admissions (risk ratio 0.79, 95% confidence interval 0.72 to 0.87) and acute kidney injury (0.76, 0.59 to 0.99).[121]

Soluble guanylate cyclase stimulators

Vericiguat is a new oral soluble guanylate cyclase stimulator, which enhances the cyclic guanosine monophosphate pathway by directly stimulating soluble guanylate cyclase to reduce oxidative stress and improve endothelial dysfunction.[122] A recent meta-analysis of four randomized controlled trials found that the addition of vericiguat 10 mg to those recently admitted to hospital for ADHF had a reduced risk of heart failure related hospital admission (risk ratio 0.92, 95% confidence interval 0.84 to 1.00), but no difference in cardiovascular or all cause mortality.[123]

Cardiac myosin activators

Cardiac myosin activators (eg, omecamtiv mecarbil) are a newer class of myotropes that improve myocardial function by directly augmenting cardiac sarcomere function. A recent randomized controlled trial of cardiac myosin activators in symptomatic chronic heart failure with an ejection fraction less than or equal to 35% reported a reduction in the composite outcome of cardiovascular death, hospital admission for heart failure, or urgent outpatient visit for heart failure at first event (hazard ratio 0.92, 95% confidence interval 0.86 to 0.99).[124]

Guidelines

Several clinical practice guidelines and consensus documents exist for the management of ADHF. Overall, these recommend the use of biomarkers like BNP or NT-proBNP; chest radiographs and echocardiography to assess heart size, pulmonary congestion, and rule out other causes; use of risk scores to estimate mortality risk; maintenance or optimization of guideline directed medical treatment during hospital admission; use of intravenous diuretics for fluid overload; venodilators or vasodilators for afterload; and inotropic support or temporary mechanical circulatory support to maintain systemic perfusion and end organ function among those with cardiogenic shock. A summary of the guidelines is presented in supplementary table 1.[26 28 125 126]

Conclusion

ADHF affects millions of people worldwide and is associated with high morbidity and mortality rates.

QUESTIONS FOR FUTURE RESEARCH

- What is the optimal combination of history, physical examination, and testing to accurately diagnose acute decompensated heart failure (ADHF)?
- Which populations with ADHF will benefit most from angiotensin converting enzyme inhibitors or vasodilators in addition to high dose nitroglycerin and non-invasive positive pressure ventilation?
- What is the optimal combination of elements to identify those with ADHF at low risk to enable safe discharge while reducing hospital admission rates?
- What new pharmacological agents offer advantages in real world settings?

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS MANUSCRIPT

We discussed this review with patients with lived heart failure experiences. They emphasized the importance of clear communication and explaining information in patient centric terminology and language. One patient explained there was a primary focus on their symptoms early on, while no one explicitly told them they had heart failure until they followed up as an outpatient. Patients also emphasized the importance of explaining the consequences of heart failure, risk factors for decompensation, and the role of diet, exercise, and drugs. One patient highlighted the challenges with finding the right combination of drugs and that this led to frequent exacerbations and emergency department visits. There was also fear about the ability to return to work, take care of their family and loved ones, and being able to coordinate care after discharge. Patients stressed the impact on quality of life, including both the physical and psychological impact of heart failure. Finally, they emphasized the role of clear instructions after discharge from the hospital. Based upon this, we expanded our disposition section and added language emphasizing the importance of communication and coordination of care.

The management of ADHF requires a comprehensive approach that includes accurate diagnosis, timely intervention, and effective treatment strategies. Key diagnostic tools include history and physical examination, electrocardiography, chest radiography, POCUS, and laboratory testing. Management strategies focus on hemodynamic stabilization, symptom relief, and addressing underlying causes. NIPPV, intravenous vasodilators, and diuretics are essential components of treatment. Among patients with hypotension or those with cardiogenic shock, vasopressors, inotropic agents, and mechanical circulatory support might be necessary. Evidence based clinical practice guidelines should be followed to optimize patient outcomes in ADHF.

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Web appendix: Supplementary table 1
Video 1: B-lines on point-of-care thoracic ultrasound suggestive of pulmonary edema
bmj-2025-084242-vid1The BMJ Video Player
Video 2: Decreased ejection fraction on point-of-care cardiac ultrasound
bmj-2025-084242-vid2The BMJ Video Player