Heart Failure Council of Thailand (HFCT) 2019 Heart Failure Guideline: Acute Heart Failure

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Acute heart failure (AHF) is defined as heart failure with rapid onset or worsening of symptoms or signs. It may present as a first occurrence (de novo) or as an acute decompensation of chronic heart failure. The etiologies and precipitating factors of AHF can be primary cardiac disorders or non-cardiac causes (Table 1). The Thai-ADHERE registry showed that 66.5% of AHF patients had prior HF, and that 39.6% had left ventricular ejection fraction (LVEF) of less than 40% before admission(1). Common etiologies of AHF in this registry were coronary artery disease (44.7%), valvular heart disease (18.7%), cardiomyopathy (13.9%), and hypertensive heart disease (12.2%). New cardiac events, concurrent illness, dietary non-compliance, inadequate diuretics, and medication non-compliance were the precipitating causes that presented in 31.9%, 21.7%, 10.7%, 7.4%, and 4.3% of AHF patients, respectively (Table 2). The etiologies and precipitating factors should be identified and appropriately managed to improve clinical outcomes.

Table 1. Etiologies of AHF

Acute coronary syndrome
Cardiac arrhythmias: tachyarrhythmia, bradyarrhythmia
Acute mechanical complications: free wall rupture, acute valvular insufficiency, prosthetic valve dysfunction
Progression of cardiac lesion, such as valvular lesion
Acute elevated blood pressure: hypertensive emergency
Increased sympathetic activity: stress induced cardiomyopathy
Acute pulmonary embolism

AHF=acute heart failure

Clinical assessment of AHF

Common clinical manifestations of AHF included dyspnea (96.7%), fatigue (36%), peripheral edema (59.5%), and pulmonary rales (84.5%)[1]. Most patients had normal blood pressure (59.5%), while systolic hypertension and hypotension presented in 36.9% and 3.6% of cases, respectively. Systolic blood pressure of less than 90 mmHg was an independent predictor for in-hospital mortality[2]. Variations in clinical manifestations and the clinical classification of AHF patients suggest fluid status and tissue perfusion evaluation as a helpful strategy for guiding treatment (Figure 1). Fluid status can be considered as wet or dry according to the presence or absence of congestion. Tissue perfusion can be characterized as cold or warm according to the presence or absence of signs of peripheral tissue hypoperfusion. The most commonly observed profile "warm and wet" can be classified as "cardiac type" in patients with fluid accumulation, and "vascular type" in patients with elevated blood pressure without fluid accumulation. While the left-sided HF symptoms such as pulmonary congestion are common presentation in majority of

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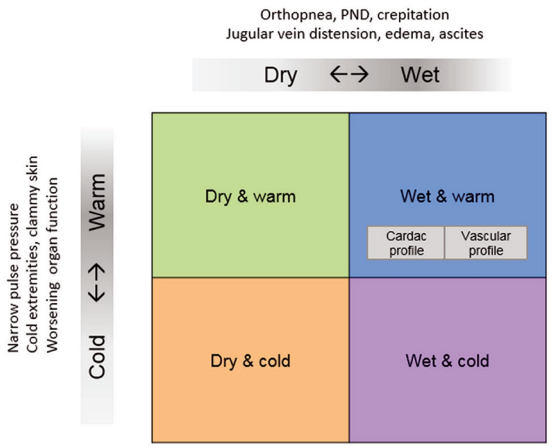

Figure 1. Clinical classification of AHF patients based on congestion and tissue perfusion status.

AHF etiologies, which are the triad of hypotension, clear lung sounds, and elevated jugular vein suggest acute pulmonary embolism.

Table 2. Factors that precipitate AHF

Cardiovascular causes
Acute coronary syndrome or myocardial ischemia
Cardiac arrhythmias: tachyarrhythmia, bradyarrhythmia
Poorly controlled hypertension
Non-cardiac causes
Medication non-compliance
Salt restriction non-compliance
Volume overload
Pulmonary embolism
Infection
Anemia
Exacerbation of lung disease
Drugs: non-steroidal anti-inflammatory drugs, corticosteroids, negative inotropic agents, cardiotoxic agents, TZD, dronedarone
Metabolic derangement: hyperthyroidism, hypothyroidism, adrenal insufficiency
Nutritional deficiency: iron deficiency thiamine deficiency
Toxic substances: alcohol, amphetamine

AHF=acute heart failure; TZD=thiazolidinediones

Initial management of AHF

In parallel to identify the life-threatening causes, the hemodynamic and respiratory status should be evaluated and managed accordingly (Figure 2). The patients with cardiogenic shock and respiratory failure should be managed in intensive care unit (Table 3). Diuretics should be used in patients with cardiac type of wet and warm AHF, while vasodilator should be used in patients with vascular type of wet and warm AHF. Oxygen should not be routinely used in non-hypoxemic AHF patients, because it

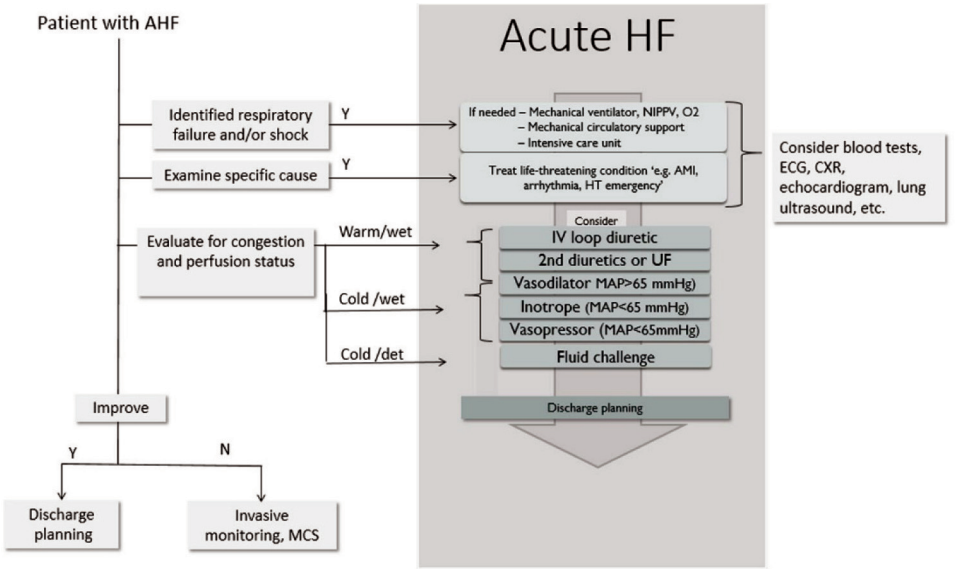

Figure 2. Acute heart failure management summary.

AHF=acute heart failure, AF=atrial fibrillation, AMI=acute myocardial infarction, CAD=coronary artery disease, CCU=coronary care unit, CXR=chest X-ray, ECG=electrocardiogram, HF=heart failure, HT=hypertension, MAP=mean arterial pressure, MCS=mechanical circulatory support, NIPPV=non-invasive positive pressure ventilation, O_2=oxygen, UF=ultrafiltration

Table 3. Recommendations regarding the initial management of AHF

Recommendations	COR	LOE
Management of patients with cardiogenic shock or respiratory failure in the intensive care unit is recommended.	I	C
Identification of etiology and management of precipitating factors in AHF patients are recommended.	I	C
Oxygen therapy is recommended in AHF patients with SpO_2 <90% or PaO_2 <60 mmHg to correct hypoxemia.	I	C
Intubation is recommended in patients with respiratory failure who cannot be managed noninvasively.	I	C
Noninvasive positive pressure ventilation (e.g., CPAP, BiPAP) should be considered in patients with respiratory distress (respiratory rate >25 breaths/minute, SpO_2 <90%) who have SBP >85 mmHg and normal consciousness.	IIa	B
Oxygen therapy is not routinely recommended.	III	C

AHF=acute heart failure; BiPAP=bilevel positive airway pressure; COR=class of recommendation; CPAP=continuous positive airway pressure; LOE=level of evidence; PaO_2=partial pressure of oxygen in arterial blood; SBP=systolic blood pressure; SpO_2=saturation of peripheral oxygen

Table 4. Recommendations regarding diuretic use in patients with AHF

Recommendations	COR	LOE
Intravenous loop diuretic is recommended in patients with signs and symptoms of fluid overload to reduce congestive symptoms.	I	C
In patients not receiving oral diuretics, an initial 20 to 40 mg intravenous dose of furosemide is recommended. In those receiving oral diuretics, an initial equivalent dose of intravenous furosemide is recommended.	I	B
Intravenous loop diuretics can be given either as intermittent boluses or as a continuous infusion, and the dose and duration should be adjusted according to patient symptoms and clinical status.	I	C
Regular daily monitoring of urine output, body weight, renal function, and electrolytes is recommended during the use of intravenous diuretics.	I	C
Intravenous loop diuretic dose should be adjusted according to patient renal function.	IIa	C
In patients with insufficient diuretic response...		
Increase the dose of loop diuretics.	I	B
Re-evaluate patient clinical status for tissue perfusion and volume status.	I	C
Low sodium diet (sodium<2 grams/day) is recommended in patients with recurrent or refractory volume overload despite appropriate diuretic therapy.	I	C
Tolvaptan (V2-receptor antagonist) should be considered in patients with congestion and/or hyponatremia. It should be given for a short duration.	IIa	B
Switch from intermittent bolus to continuous infusion of loop diuretics.	IIa	C
Combination of loop diuretic with either thiazide-type diuretic or spironolactone should be considered in patients with insufficient diuretic response.	IIa	C
Ultrafiltration may be considered in patients with refractory congestion who fail to respond to a diuretic-based strategy.	IIb	B

AHF=acute heart failure; COR=class of recommendation; LOE=level of evidence

causes vasoconstriction and reduction in cardiac output[3]. Non-invasive positive-pressure ventilation has been shown to reduce respiratory distress and intubation rate. However, its effect on mortality is still inconclusive[4].

Diuretics in AHF

Diuretics increase renal salt and water excretion, and they exert some vasodilatory effect. Although the effects of time to diuretics on in-hospital mortality is still inconclusive, the appropriate use of diuretics to improve clinical symptoms and reduce adverse effects is important[5,6]. The Diuretic Optimization Strategies Evaluation (DOSE) study[7] showed similar effects between intermittent bolus and continuous infusion of intravenous furosemide on patient symptoms or change in renal function. High-dose diuretic was found to be associated with greater diuresis, but increased risk of transient worsening renal function. In AHF, the smallest clinically effective dose of diuretic should be used to avoid over-diuresis and renal dysfunction (Table 4). In patients with insufficiency response to diuretics, patient clinical status for tissue perfusion and volume status should be evaluated. Switching from intermittent bolus to continuous infusion of intravenous loop diuretics and/or combination of

Table 5. Recommendations regarding the use of intravenous vasodilators (sodium nitroprusside, nitroglycerine) in patients with AHF

Recommendations	COR	LOE
Invasive BP monitoring is recommended during intravenous vasodilator use.	I	C
In warm and wet (vascular type) heart failure, intravenous vasodilator should be used for relief of congestive symptoms in patients with normal or elevated blood pressure (MAP >65 mmHg).	IIa	B
Patients with signs/symptoms of hypoperfusion and congestion (cold and wet) with MAP >65 mmHg, intravenous vasodilators should be considered with caution.	IIa	C
Intravenous sodium nitroprusside should be avoided in patients with acute myocardial ischemia or renal insufficiency.	IIa	C

AHF=acute heart failure; BP=blood pressure; COR=class of recommendation; LOE=level of evidence; MAP=mean arterial pressure

Table 6. Recommendations regarding the use of inotropic agents (dobutamine, dopamine, milrinone, levosimendan) in patients with AHF

Recommendations	COR	LOE
Use inotropic agents in the following conditions...		
• Cardiogenic shock	I	C
• Signs/symptoms of hypoperfusion and/or end organ damage with hypotension (MAP <65 mmHg) despite adequate filling status.	I	C
• Close monitoring of ECG and BP during intravenous inotrope infusion is recommended.	I	C
• Refractory AHF with inadequate response to intravenous loop diuretics or vasodilators.	IIb	C
• Intravenous infusion of milrinone or levosimendan may be considered to reverse the effects of beta-blocker if beta-blocker is suspected as the cause of hypoperfusion.	IIb	C
Vasopressors (e.g., norepinephrine) may be considered in patients with cardiogenic shock despite treatment with inotropic agents.	IIb	B
Inotropic agents are not routinely recommended due to safety concerns unless the patient has symptomatic hypotension or hypoperfusion.	III	A

AHF=acute heart failure; BP=blood pressure; COR=class of recommendation; ECG=electrocardiogram; LOE=level of evidence; MAP=mean arterial pressure

diuretics such as thiazide-type diuretics should be considered to enhance diuresis[8]. Even though, adding high dose spironolactone to usual care for patients with AHF did not improve net urine output, the study was not performed in patients with diuretic resistance[9]. Tolvaptan (vasopressin-receptor antagonist) has been shown to increase free water excretion (aquaresis) although it did not showed benefits in long term clinical outcomes in hospitalized heart failure patients[10,11]. It should be considered in patients with congestion and/or hyponatremia to enhance fluid loss. Aggressive water (less than 800 ml/day) and sodium restriction (less than 800 mg/day of sodium) had no effect on weight loss or clinical stability[12], however low sodium diet (less than 2 grams/day of sodium) is recommended in patients with recurrent or refractory volume overload despite appropriate diuretic therapy. Ultrafiltration can reduce congestion by removing plasma fluid through the super-permeable membrane. However, there is no evidence favoring ultrafiltration over intravenous diuretic-based therapy. Ultrafiltration should be considered in patients with inadequate response to intravenous diuretic-based therapy[13-15].

Vasoactive agents and inotropic agents

Vasodilators have demonstrated favorable hemodynamic effects in AHF by decreasing preload and afterload. However, there was no association with favorable clinical outcomes observed[16,17]. The favorable hemodynamic effects of vasodilators may be useful in hypertensive patients or vascular type AHF (Table 5).

Inotropic agents should be reserved for patients with hypoperfusion with adequate volume status. Because some inotropic agents, such as dobutamine or levosimendan, have vasodilator effects, close BP monitoring is recommended[18,19] (Table 6). Inotropic agents do not provide morbidity and mortality benefits and should not be routinely used. Furthermore, intermittent or prolong used of inotropic agents may be associated with increased mortality[18].

Table 7. Inotropic agents and vasoactive agents

	Bolus	Infusion rate	Side effects
Inotropic agents and vasopressors			
Dobutamine	No	2 to 20 µg/kg/minute	Tachycardia
Dopamine	No	3 to 5 µg/kg/minute (inotropic agents)	Tachycardia
		>5 µg/kg/minute (vasopressor)	
Levosimendan	12 µg/kg over 10 minutes	0.1 µg/kg/minute	
Milrinone	25 to 75 µg/kg slowly bolus over 20 minutes	0.375 to 0.75 µg/kg/minute	Hypotension, tachycardia
Norepinephrine	No	0.2 to 1.0 µg.kg/minute	Reflex bradycardia, tissue hypoxia from severe vasoconstriction
Epinephrine	1 mg IV push and repeat every 3 to 5 minutes during resuscitation	0.05 to 0.5 µg/kg/minute	Tachycardia
Vasodilators			
Nitroglycerine		Start 10 to 20 µg/minute increased up to 200 µg/minute	Hypotension, headache, tolerance
Nitroprusside		Start 0.3 µg/kg/minute and increase up to 5 µg/kg/minute	Light sensitive, hypotension, isocyanate toxicity

Table 8. Recommendations regarding invasive procedures in patients with AHF

Recommendations	COR	LOE
Pulmonary artery pressure monitoring may be considered in the following...		
• Persistent hypotension and/or worsening renal function with inadequate assessment of left ventricular filling pressure.	IIa	C
• To assess pulmonary artery pressure and pulmonary vascular resistance for heart transplantation or mechanical circulatory support devices.	IIa	C
• Refractory heart failure despite standard treatment with intravenous diuretics, intravenous inotropes, and intravenous vasodilators.	IIb	C
• Invasive hemodynamic monitoring and right heart catheterization are not routinely recommended in AHF.	III	B
Mechanical circulatory support devices should be considered in patients with cardiogenic shock despite adequate medical therapy.	IIa	C

AHF=acute heart failure; COR=class of recommendation; LOE=level of evidence

Vasopressors, such as norepinephrine and dopamine, should be used only in cardiogenic shock to raise blood pressure and preserve perfusion to vital organs[19]. Because inotropic agents and vasopressors increase the risk of cardiac arrhythmia and myocardial ischemia, ECG monitoring is required (Table 7).

In patients with hypoperfusion or worsening organ damage, the assessment of left ventricular filling pressure may be necessary before initiation of inotropic agents or vasopressors. The pulmonary artery pressure monitoring should be considered (Table 8). In addition, it may be considered in patients with refractory heart failure despite standard treatment of intravenous diuretics and vasoactive agents. The routine use of invasive hemodynamic monitoring including pulmonary pressure monitoring is not recommended due to increased adverse events without benefits on mortality and hospitalization[20,21].

Mechanical circulatory support device should be considered in patients with cardiogenic shock despite adequate medical therapy (Table 8).

General management of AHF

During hospitalization with AHF, neurohormonal blockage including renin-angiotensin-aldosterone blockage (RAAS blockage) or beta-blocker should be continued except in the presence of contra-indication. Discontinuation of treatments such as beta-blocker may be associated with adverse outcomes[22]. Medical

Table 9. Recommendations regarding the general management of AHF

Recommendations	COR	LOE
OMT for chronic heart failure is recommended in AHF patients with HFrEF after hemodynamic stabilization and no contraindications.	I	A
Pre-discharge evaluation and optimization of medical therapy are recommended.	I	C
Multidisciplinary HF management should be consulted especially in high-risk AHF patients.	IIa	C
Thromboembolism prophylaxis should be considered in patients not already anticoagulated.	IIa	B

AHF=acute heart failure; COR=class of recommendation; HF=heart failure; HFrEF=heart failure with reduced ejection fraction; LOE=level of evidence; OMT=optimal medical treatment

Table 10. Pre-discharge evaluation topics

1. Identify underlying and precipitating causes
2. Patient clinical status: volume and perfusion
3. Guideline-directed medical therapy
4. Guideline-directed device therapy
5. Patient and caregiver education emphasizing signs and symptoms of worsening HF and management
6. Follow-up schedule (including telephone follow-up) in high-risk HF patients

HF=heart failure

therapy should be optimized before discharge (Table 9). The incidence of venous thromboembolism (VTE) is increased in AHF patients. The benefits of VTE prophylaxis during hospitalization have been shown from HF subgroup in several studies[23]. Thromboembolic prophylaxis should be considered in patients without anticoagulant.

Discharge planning

Appropriate patient and caregiver education, and appropriate AHF management to optimize euvolemic status and tissue perfusion can prevent rehospitalization. Discharge planning should be considered when patient clinical status becomes stable. Pre-discharge evaluation should be performed and focuses on diagnosis, investigation, and management of underlying and precipitating causes, optimal medical treatment (OMT), and device therapy according to patient status (Table 10). Multidisciplinary HF management is recommended, especially in high-risk AHF patients.

Conclusion

AHF is a serious and life-threatening condition. Appropriate and timely assessment and management are essential to improve outcomes.

Conflicts of interest

The authors declare no conflict of interest.

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