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KSHF Guidelines for the Management of Acute Heart Failure: Part III. Specific Management of Acute Heart Failure According to the Etiology and Co-morbidity

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ABSTRACT

The prevalence of heart failure (HF) is on the rise due to the aging of society. Furthermore, the continuous progress and widespread adoption of screening and diagnostic strategies have led to an increase in the detection rate of HF, effectively increasing the number of patients requiring monitoring and treatment. Because HF is associated with substantial rates of mortality and morbidity, as well as high socioeconomic burden, there is an increasing need for developing specific guidelines for HF management. The Korean guidelines for the diagnosis and management of chronic heart failure (CHF) were introduced in March 2016. However, CHF and acute heart failure (AHF) represent distinct disease entities. Here, we introduce the Korean guidelines for the management of AHF with reduced or preserved ejection fraction. Part III of this guideline covers management strategies optimized according to the etiology of AHF and the presence of co-morbidities.

Keywords: Heart failure; Guideline; Comorbidity

INTRODUCTION

The number of patients with heart failure (HF) has been on the rise as a consequence of the aging of society and the improvement in screening and diagnostic techniques. Therefore, there is an increasing need for developing guidelines for the diagnosis and treatment of

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HF. Although guidelines for HF management have already been issued by American and European associations, many aspects of such guidelines do not reflect the domestic reality in Korea due to the social and anthropological characteristics of the Korean population. Thus, to help clinicians establish the best treatment plan for Korean patients with HF, it is necessary to develop specific guidelines that reflect the clinical situation in Korea. In 2012, the Korean Society of Heart Failure (KSHF) established the Guideline Writing Committee to develop the treatment guidelines for HF. The Korean guidelines for the diagnosis and management of chronic heart failure (CHF) were introduced in March 2016.[1] However, CHF and acute heart failure (AHF) are distinct disease entities, warranting different approaches for diagnosis and treatment. Here, we introduce the Korean guidelines for the management of AHF with reduced or preserved ejection fraction (HFrEF and HFpEF, respectively). In Part III of this guideline, we discuss management strategies optimized according to the etiology of AHF and the presence of co-morbidities.

This guideline was developed based on previously issued international guidelines and amended to reflect the clinical situation in Korea. A committee of KSHF members decided on the format of the guideline, the selection of topics addressed, and the composition of the Writing Committee. To develop the guidelines, we considered all clinical studies and evidence included in the international guidelines, as well as domestic research conducted in Korean patients with HF. Members of the Writing Committee first drafted thematic manuscripts, which were then assembled and arranged according to the evidence-based scales. To ensure transparency and facilitate future revision of the guidelines, we documented the process followed for issuing a recommendation for each topic based on the gathered evidence. The guideline was drafted, reviewed by the advisory committee, and finalized after receiving endorsement from the Korean Society of Cardiology, Korean Society of Hypertension, Korean Society of Interventional Cardiology, Korean Society of Echocardiography, and Korean Society of Lipid and Atherosclerosis. While working on this guideline, the members of the Writing Committee were not affected by external influences and made every effort to exclude conflicts of interests.

The Writing Committee issued the level of recommendation upon a comprehensive analysis of evidence from studies describing real clinical experience, from surveys, from epidemiologic, observational, and randomized clinical studies, and from meta-analyses. The level of evidence and class of recommendation were defined so as to have a clear formulation, provide straightforward instructions, and be easily adopted in daily clinical practice (**Supplementary Tables 1** and **2**).

This guideline is intended to help improve clinical practice by providing recommendations based on clinical evidence. As such, the guideline does not serve as a basis for clinical judgement. The final decision in the treatment of each patient should be made by the treating physician according to their personal opinion and judgment, while using the guideline to support these decisions.

ISCHEMIC HEART DISEASE/ACUTE CORONARY SYNDROME

1. In patients with ST-segment elevation or left bundle branch block, percutaneous coronary intervention or coronary artery bypass grafting must be performed to reduce cardiomyocyte necrosis and early mortality rates (class of recommendation I, level of evidence A).
2. If percutaneous coronary intervention and coronary artery bypass grafting are not suitable options for controlling ST-segment elevation or new left bundle branch block, intravenous thrombolytic therapy must be performed (class of recommendation I, level of evidence A).
3. In hemodynamically unstable patients with non-ST elevation acute coronary syndrome, emergency reperfusion techniques such as percutaneous coronary intervention or coronary artery bypass grafting should be performed as early as possible (class of recommendation I, level of evidence A).
4. Intravenous opioids can be considered in patients with ischemic chest pain to improve symptoms and dyspnea (class of recommendation IIb, level of evidence B).

To reduce cardiomyocyte necrosis and early mortality, primary percutaneous coronary intervention must be performed promptly patients with acute coronary syndrome and ST-segment elevation or new left bundle branch block.[2-7] If percutaneous coronary intervention or coronary artery bypass grafting cannot be performed, intravenous thrombolytic therapy must be administered.[8] If symptoms develop within 12–24 hours or signs of congestive HF, hemodynamic instability, or persistent ischemia is detected, it is appropriate to conduct primary percutaneous coronary intervention.[8-10] If patients who experienced serious HF-associated symptoms, cardiogenic shock, or hemodynamically unstable ventricular arrhythmia did not receive primary percutaneous coronary intervention, coronary angiography to enable percutaneous coronary intervention must be performed immediately unless it is deemed unlikely to bring any benefits.[11)12]

Despite the recent advances in medical treatment, patients with severe coronary artery disease and decreased left ventricular contractility typically have poor prognosis with drug treatment alone.[13] Elective reperfusion therapy can increase the survival rate of such patients,[14-22] whereas coronary artery bypass grafting can be considered in patients with ischemic heart disease and severe left ventricular dysfunction, where it is expected to increase survival regardless of myocardial variability.[15)20)23-27]

Intravenous opioids can be used to alleviate symptoms and improve dyspnea in patients with ischemic chest pain. Routine injection of opioids is not recommended in AHF patients but can be used in patients with severe dyspnea. As opioids can hinder respiration, consciousness and breathing must be checked frequently.[28)29]

HYPERTENSIVE EMERGENCY

1. A rapid and excessive rise in blood pressure can cause AHF accompanied by pulmonary edema, and thus must be managed immediately with antihypertensive therapy (class of recommendation I, level of evidence C).
2. Within the first few hours of AHF treatment, it is appropriate to use intravenous vasodilators and loop diuretics in combination with active antihypertensive therapy to lower blood pressure within 25% of the initial value (class of recommendation IIa, level of evidence B).

> 3. In AHF, vasodilator injection is appropriate as a primary treatment to alleviate symptoms and decrease congestion (class of recommendation IIa, level of evidence B).

Hypertensive emergency refers to a rapid rise in blood pressure (>180/120 mmHg) and progression of target organ damage.[30] In patients with left ventricular failure accompanied by pulmonary edema, immediate hospital admission and prompt initiation of antihypertensive therapy are required.[31)32] Organ damage can also occur if blood pressure rises quickly without exceeding 180/120 mmHg. However, excessive lowering of blood pressure can cause ischemia in the kidney, brain, and coronary arteries. Therefore, this process must be performed with caution to ensure that the extent of the blood pressure drop does not exceed 25% of the initial blood pressure in the first few hours.[33-35] It is sensible to use aggressive therapy with combined vasodilator injection and loop diuretics. Vasodilator injection can be considered in patients with AHF, to alleviate symptoms and decrease congestion.[34)36-38]

MYOCARDITIS

> 1. In patients with myocarditis, endomyocardial biopsy can help diagnose and predict prognosis (class of recommendation IIa, level of evidence C).
> 2. Cardiac magnetic resonance imaging (MRI) can be considered for diagnosing myocarditis (class of recommendation IIb, level of evidence B).
> 3. In patients with myocarditis, routine immunosuppressive therapy not increase survival (class of recommendation III, level of evidence B).
> 4. Patients with hemodynamically unstable fulminant myocarditis refractory to medication require mechanical circulatory support (class of recommendation I, level of evidence B).

Myocarditis can have many etiologies. While viral infection is the most common etiology, systemic autoimmune diseases such as drug hypersensitivity, lupus erythematosus, or Churg-Strauss syndrome may also cause myocarditis. Endomyocardial biopsy is the standard method for diagnosing myocarditis. The findings of lymphocytic infiltration and heart muscle damage in the biopsy specimens are used to diagnose and classify myocarditis according to Dallas criteria.[39] Moreover, endomyocardial biopsy can accurately differentiate specific myocarditis types such as giant-cell myocarditis or eosinophilic myocarditis, and helps in predicting patient prognosis. According to a small-scale research study on myocarditis, survival rate is lower in patients with inflammatory cell infiltration evident on immunohistochemical staining of endomyocardial biopsy specimens.[40] With the development of cardiac MRI technology, diagnosing myocarditis noninvasively has recently become possible. Cardiac MRI is used to indirectly diagnose myocarditis by detecting early-stage myocardial edema or inflammatory myocardial damage,[41] so patients with suspected myocarditis must also have relevant clinical manifestations. However, this approach is limited because it may yield false negative results if there are no other indicators of myocarditis, such as in patients with giant-cell myocarditis or less-than-severe myocarditis.

Observational studies reported that immunoregulatory treatment (e.g., with immunosuppressants) may help control inflammation in patients with myocarditis. However, a larger randomized study reported that the clinical effects of immunosuppressive or immunoregulatory treatment were ambiguous or even harmful at times; therefore, routine immunosuppressive treatment is not recommended in patients with myocarditis.[42]

Thus, in principle, myocarditis patients should receive the same treatment as that recommended in HF patients. However, if drug therapies cannot maintain adequate blood pressure in patients with hemodynamically unstable fulminant myocarditis patients, a mechanical cardiac support device (MCSD) must be used promptly. In patients who receive an MCSD and later have it removed as their health improves, the long-term survival is very high (>90% for 10 years after the onset of myocarditis).[43)44)]

Cardiac transplantation must be considered in myocarditis patients if HF does not improve with appropriate drug treatment or after insertion of an MCSD. Research revealed that the time from symptom onset to cardiogenic shock was shorter in patients who received an MCSD that was removed after recovery than in those who eventually needed cardiac transplantation.[45)] Therefore, cardiac transplantation can be seen as a treatment option better suited for chronic than for acute myocarditis. Recent studies reported that the survival rate of cardiac transplant recipients is similar to that of other HF patients.[46)47)] However, patients with sarcoidosis or giant-cell myocarditis have a risk of recurrence after cardiac transplant and thus should be monitored carefully.[46)]

VALVULAR HEART DISEASE

1. The prognosis of AHF caused by aortic valve stenosis can be improved more dramatically with percutaneous or surgical therapy than with symptomatic therapy (class of recommendation I, level of evidence A).
2. If surgery is not possible in a patient with AHF caused by aortic valve stenosis, a more substantial improvement in prognosis can be achieved with percutaneous aortic valve replacement than with symptomatic therapy (class of recommendation I, level of evidence A).
3. The prognosis of patients with AHF caused by mitral insufficiency can be improved more extensively by surgical therapy than by symptomatic therapy (class of recommendation I, level of evidence A).

Valvular heart disease can act as both a cause and an aggravating factor of HF. For this reason, HF patients with valvular heart disease are categorized as high risk. After a complete medical examination, the treatment direction must be consulted and planned by a multidisciplinary team involving a valvular heart disease specialist, an HF specialist, a cardiac surgeon, a valve intervention specialist, a cardiovascular imaging specialist, and an anesthesiologist. All patients must receive the best drug treatment for HF, according to the guidelines. Patients with severe aortic valve stenosis may develop hypotension when taking vasodilators, so caution is needed.

Aortic valve stenosis
Aortic valve stenosis is a cause of HF, and its prevalence is increasing due to the aging of society. During the early stages of AHF caused by aortic valve stenosis, diuretic therapy can improve the patient's condition; however, unless systemic vascular circulation improves, poor prognosis is unavoidable. Aortic valve replacement surgery is a classic treatment for aortic valve stenosis. However, as the mean patient age increases together with the range of associated diseases, more and more patients are categorized as having high surgical risk and thus are poor candidates for aortic valve replacement surgery. Percutaneous aortic valve replacement was developed as a suitable intervention strategy in such high-risk patients, but no study to date has compared the outcomes of surgical and percutaneous aortic valve replacement in patients with aortic valve stenosis accompanied by AHF.

Several randomized studies reported no difference between percutaneous aortic valve replacement and surgical therapy in terms of overall or cardiac mortality in patients with aortic valve stenosis.[48-57] Compared to patients who received surgery, those who underwent percutaneous aortic valve replacement had lower incidence of hemorrhage and new-onset atrial fibrillation, but higher incidence of major cerebrovascular accidents and vascular complications. On the other hand, compared to patients who received medical treatment, those who underwent percutaneous aortic valve replacement had lower readmission rates, lower mortality rates, and higher survival rates but were at a high risk of major cerebrovascular accidents. A multidisciplinary approach involving a valve intervention specialist, cardiovascular imaging specialists, anesthesiologist, and cardiac surgeon is needed when determining the percutaneous aortic valve replacement indications of patients with aortic valve stenosis.

Aortic valve insufficiency

Aortic valvuloplasty or valve replacement can be performed in symptomatic patients with severe aortic valve insufficiency or left ventricular systolic dysfunction (left ventricular ejection fraction <50%), regardless of symptoms. However, there is currently no clear recommendation for the treatment of AHF accompanied with aortic insufficiency.

Mitral insufficiency

Mitral insufficiency is the most common valvular heart disease in patients with HFpEF or HFrEF, occurring secondary to morphological changes induced by HF-related hemodynamic changes. Mitral insufficiency accompanied by HF is usually not severe and can be improved if the disease responsible for the insufficiency is treated. However, different therapeutic approaches are needed depending on the type of mitral insufficiency (primary or secondary). Thoracotomy is generally used for treating primary mitral insufficiency, but percutaneous mitral valve intervention can be considered in patients who are not candidates for surgery due to high risk. In patients with mitral insufficiency, the 12-month and 2-year outcomes of percutaneous mitral valve intervention were similar to those of surgery.[58-60] Percutaneous mitral valve intervention was associated with reduced need for transfusion and mechanical ventilation but increased need for reparative mitral valve surgery. However, there no randomized controlled studies have compared percutaneous mitral valve intervention and symptomatic therapy or surgical therapy plus symptomatic therapy. The study results of the Efficacy of Vasopressin Antagonism in Heart Failure Outcome Study with Tolvaptan (EVEREST) and EVEREST II trials revealed no difference between percutaneous mitral valve intervention and surgery regarding mortality or incidence of severe mitral insufficiency at 30 days, 1 year, 2 years, and 4 years, respectively, although these conclusions may not be generalizable to all mitral insufficiency patients.

Tricuspid insufficiency

Secondary tricuspid insufficiency in relation to elevated right ventricular pressure and volume overload is frequently observed as a natural course of HF. Because severe tricuspid insufficiency worsens the symptoms and signs of right HF, diuretics are used to reduce peripheral edema. Passive hepatic congestion is frequently observed in these patients, and a high-dose aldosterone antagonist is used to improve such symptoms.[61)62] When secondary tricuspid insufficiency is accompanied by HF, tricuspid insufficiency is alleviated through active HF treatment. The optimal surgical strategy in patients with secondary tricuspid insufficiency accompanied by HF has not been established, but surgical therapy is advised in patients with tricuspid insufficiency who needed mitral or aortic valve surgery. Currently,

percutaneous intervention is attempted in patients with tricuspid insufficiency, but there are no clear recommendations in patients with AHF accompanied with tricuspid insufficiency.

RIGHT HEART FAILURE

1. Coronary reperfusion must be performed in patients with right ventricular myocardial infarction (MI) accompanied by ST-segment elevation acute MI (class of recommendation I, level of evidence A).
2. Use of thrombolytics is appropriate in patients with acute right HF due to pulmonary embolism (class of recommendation IIa, level of evidence B).
3. MCSD use can be very helpful in patients with right HF and cardiogenic shock refractory to medication (class of recommendation IIa, level of evidence C).

The pathophysiologic mechanisms underlying right ventricular dysfunction can be classified into 3 categories: decreased right ventricular contractility, right ventricular pressure overload, and right ventricular volume overload.[63] If there is decreased right ventricular contractility (like in MI), right ventricular output is reduced, leading to decreased left ventricular preload and, consequently, reduced left ventricular output.[64] The right ventricular pressure and volume overload observed in acute pulmonary artery embolism causes right ventricular enlargement and creates a leftward shift of the interventricular septum, decreased left ventricular preload, and reduced left ventricular output resulting in hypotension.[65] Moreover, due to right ventricular pressure overload, the right ventricular wall tension increases, thereby increasing the oxygen demand of the right ventricular myocardium. Hypotension and increased wall tension of the right ventricle causes reduced left ventricular output, decreasing right coronary artery flow. This forms a vicious cycle, creating an environment that facilitates the development of right ventricular ischemia.[66]

Since the right ventricle has good recovery, its functions can be regained if right HF is treated early. Thus, the treatment for right HF and the treatment for the causes of right HF must go hand in hand. Right heart function is determined by preload (volume status), contractility, and afterload, so controlling these 3 parts can improve the symptoms and signs of HF.

Right ventricular myocardial infarction

Right coronary artery occlusion causes right ventricular ischemia, but the right ventricle has a lower oxygen demand[67] and its blood supply flows during both systole and diastole.[68] A collateral circulation develops from the left coronary artery,[69] which can help avoid right ventricular MI even in patients with long-term ischemia.[70] True right ventricular MI is very rare.[71] Therefore, the treatment guidelines for right ventricular MI are identical to those of left ventricular MI.[72][73]

Pulmonary embolism

The increase in afterload induced by acute pulmonary embolism can cause right HF.[74-76] If the patient's vital signs are stable, anticoagulants such as heparin or a new oral anticoagulant (non-vitamin K oral anticoagulant) can improve symptoms; however, if the patient is hemodynamically unstable (e.g., experiencing hypoxia), thrombolytics must be administered and embolectomy must be performed.[77] The use of thrombolytics was shown to improve prognosis in moderate risk patients with acute pulmonary embolism with stable vital signs but with signs of right HF on echocardiography.[78]

Clinical role of mechanical cardiac support in acute right heart failure

The advantage of mechanical cardiac support is that it decreases the myocardial oxygen demand while maintaining or increasing blood flow, without causing myocardial ischemia.[79] The use of an intra-aortic balloon pump can improve right heart function by decreasing left ventricular filling pressure and right ventricular afterload. Veno-arterial extracorporeal membrane oxygenation bypasses the right heart and pulmonary circulation and can be used as a temporary treatment for right HF until heart function is recovered.[80-82]

CARDIORENAL SYNDROME

Because the heart and kidneys interact hemodynamically and neurohormonally, acute or chronic disease in 1 organ can induce acute or chronic dysfunction of the other organ. This phenomenon is referred to as the cardiorenal syndrome and can be classified into 5 types according to disease progression (acute or chronic) and according to the culprit organ (heart or kidneys): type 1, abrupt worsening of heart function inducing acute kidney injury; type 2, chronic abnormalities in heart function inducing chronic kidney disease; type 3, abrupt worsening of kidney function inducing acute cardiac disorder; type 4, chronic kidney disease contributing to decreased cardiac function; and type 5, acute or chronic systemic condition inducing heart and kidney dysfunction.[83] AHF patients have type 1 cardiorenal syndrome.

An estimated 30–60% of HF patients experience a greater-than-moderate degree of renal dysfunction,[84][85] and it has been reported that 20–30% of patients undergoing AHF treatment experience renal function deterioration.[86-89] Among Korean patients hospitalized with AHF, 14.9% have serum creatinine levels >2.0 mg/dL.[90] Both underlying renal dysfunction and renal function deterioration during treatment have a negative prognostic effect in HF patients.[84-86][90][91]

Some studies reported improved renal function after use of a left ventricular assist device and cardiac resynchronization therapy, implying that cardiorenal syndrome is partially reversible.[92][93] However, decreased renal function can be an indicator of severe HF. In this case, solely improving renal function might not improve prognosis. No medication available to date is known to directly improve the renal function of HF patients. If there are signs of elevated jugular venous pressure or evidence of congestion such as peripheral edema, the use of diuretics must be continued despite elevated blood urea nitrogen or serum creatinine levels. The effects of diuretics on renal function might vary. In some patients, renal function can deteriorate due to decreased renal perfusion caused by decreased ventricular filling pressure.[94] Nevertheless, most patients with HF on the plateau of the Frank Starling curve would not experience renal function deterioration because changes in ventricular filling pressure would not have much effect on cardiac output,[95] or would experience decreased renal venous pressure and intraperitoneal pressure, which would lead to right ventricular enlargement and improved renal function.[96][97] Although diuretic therapy was correlated with renal function deterioration in the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) study[98] and in the EVEREST study,[99] the death rate was lower in the group that received active diuretic therapy.

Although no clinical research on renin-angiotensin-aldosterone antagonists has focused on patients with cardiorenal syndrome, subgroup analyses and cohort studies showed that the long-term use of renin-angiotensin-aldosterone antagonists had positive prognostic effects even in patients with decreased renal function.[100-103] Although partial aggravation of renal function may occur in the early phase of renin-angiotensin-aldosterone antagonist

administration, long-term prognosis was better in groups that received high doses.[104-106] However, if decreased renal function is also present, there is a high possibility that side effects such as hyperkalemia or deteriorated renal function would occur.

The use of vasodilators has no clear advantage in patients with cardiorenal syndrome. In the Acute Decompensated Heart Failure National Registry (ADHERE) study, the patients who took combination therapy with diuretics and nitroglycerin or nesiritide experienced renal function deterioration more frequently than noted among those who took only diuretics.[107] The Acute Study of Clinical Effectiveness of Nesiritide in Decompensated Heart Failure (ASCEND-HF) and Renal Optimization Strategies Evaluation (ROSE) studies showed that nesiritide had no effect on renal function.[108)109] In the Dopamine in Acute Decompensated Heart Failure (DAD-HF) and ROSE studies of dopamine, the use of inotripic agents had no effect on renal function,[109)110] while the effect of inotropic agents on cardiorenal syndrome was unclear.

Ultrafiltration can be used in patients with deteriorated renal function and diuretic resistance. In the Ultrafiltration versus Intravenous Diuretics for Patients Hospitalized for Acute Decompensated (UNLOAD) study[111] and the Relief for Acutely Fluid-Overloaded Patients with Decompensated Congestive Heart Failure (RAPID-CHF) study,[112] ultrafiltration resulted in greater loss of fluids but did not affect serum creatinine levels. In the Cardiorenal Rescue Study in Acute Decompensated Heart Failure (CARESS-HF) study,[113] which targeted HF patients with gradually deteriorating renal function and persistent congestion, ultrafiltration resulted in similar weight loss but increased the serum creatinine levels and the risk of side effects. Thus, although ultrafiltration can help decrease fluid overload in some patients with diuretic resistance, it cannot be considered a primary treatment for AHF or an effective solution for cardiorenal syndrome.

STRESS-INDUCED CARDIOMYOPATHY

1. Transthoracic echocardiography and coronary angiography or coronary computed tomography angiography must be used to diagnose stress-induced cardiomyopathy (class of recommendation I, level of evidence C).
2. In patients with stress-induced cardiomyopathy, it is appropriate to also treat underlying diseases when treating AHF (class of recommendation IIa, level of evidence C).

In 1990, the first case was reported of a patient who had symptoms and signs very similar to acute coronary syndrome but showed no meaningful stenosis or spasm of the coronary artery. Since then, there have been many reports of patients with an abnormal wall motion of the left ventricular apex related to physical and mental stress causing pathognomonic wall motion abnormalities of apical ballooning and left ventricular dysfunction. This was originally called Takotsubo cardiomyopathy because the affected left ventricle resembled the pot used to caught octopuses, but was later renamed stress-induced cardiomyopathy and categorized as a subgroup of acquired cardiomyopathy.[114-120] Stress-induced cardiomyopathy frequently occurs in aged women after mental and physical stress. Stress-induced cardiomyopathy occurs in 1–2% of patients with suspected acute coronary syndrome and is known to accompany AHF in 45% of all cases.[114-120] According to numerous domestic single-center studies, stress-induced cardiomyopathy occurs in 28% of intensive care unit patients.[121)122] Although the causes of stress-induced cardiomyopathy are not yet clear, the presence of a preceding stressful condition is a known characteristic.[123] Triggers generally

include emotional factors (30%), physical factors (40%), and unclassified factors (30%). Recent studies report an increasing prevalence of stress-induced cardiomyopathy due to physical stress.[123-125] Mechanisms proposed to underlie stress-induced cardiomyopathy include coronary artery vasoconstriction, microcirculatory disturbances, left ventricular outflow obstruction, catecholamine-induced myocardial stunning, hormonal interactions, and inflammation.[123]124]

The modified Mayo Clinic diagnostic criteria are used to diagnose stress-induced cardiomyopathy:[126] transient localized wall motion abnormality in the left mid-ventricular to apical area; new changes on electrocardiography, including ST-segment elevation or T-wave inversion, coupled with changes in the levels of cardiac enzymes; no evidence of significant coronary stenosis or acute rupture of the atherosclerotic plaque; exclusion of acute myocarditis and pheochromocytoma. To diagnose stress-induced cardiomyopathy, coronary artery disease must be excluded by confirming that there is no significant coronary stenosis or rupture of the atherosclerotic plaque on coronary angiography. Furthermore, as vasospasm cannot be excluded on angiography alone in the absence of significant coronary stenosis, a provocative vasospasm test could be considered if the patient is stable. Coronary angiography is required for making the definite diagnosis of stress-induced cardiomyopathy, but if invasive coronary angiography cannot be carried out, coronary artery disease can be ruled out through cardiac computed tomography. Performing echocardiography multiple times to monitor localized wall motion abnormalities can help with the diagnosis, and a nuclear scan can also be considered.[126-128]

Since there are currently no specialized treatment guidelines for stress-induced cardiomyopathy, the treatment for HF and its causative diseases is implemented based on general AHF treatment guidelines.[116]119]129-131] In patients with congestion, diuretics can be administrated if blood pressure is stable. Until left ventricular function is recovered, an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker can be administered. If the patient is hemodynamically unstable, an inotrope injection may be needed.[129] The most common complication of stress-induced cardiomyopathy is AHF, as 15–50% of patients show symptoms and signs of HF.[119] Cardiogenic shock (9.3%), pulmonary edema (25%), and life-threatening arrhythmias (4.2%) can occur as comorbid conditions. Mitral insufficiency, pericardial effusion, and dynamic left ventricular outflow tract obstruction can occur as well. Left ventricular apical thrombus formation can cause thromboembolism.[132]

Stress-induced cardiomyopathy tends to improve after a few weeks. Localized wall motion abnormalities improve within 4–8 weeks, so if they are persistently observed on echocardiography, the condition must be re-evaluated for the possibility of a different diagnosis.[116]120] The in-hospital mortality rate of stress-induced cardiomyopathy is 1–2%, but some high-risk groups have a death rate of up to 16%. Thus, if the severity of the underlying disease is high or stress-induced cardiomyopathy negatively affects the progress of HF, the prognosis is poor.[133]134] On long-term follow-up, the recurrence rate of stress-induced cardiomyopathy is 2–10%.[116]120]

TACHYCARDIA-INDUCED CARDIOMYOPATHY

1. It is appropriate to control heart rate or restore sinus rhythm as a treatment for tachycardia-induced cardiomyopathy due to atrial fibrillation (class of recommendation IIa, level of evidence B).
2. Catheter ablation to restore sinus rhythm can be considered for patients with symptomatic tachycardia-induced cardiomyopathy due to atrial fibrillation that is refractory to medication (class of recommendation IIb, level of evidence B).

Tachycardia-induced cardiomyopathy is a type of HF caused by increased heart rate leading to left ventricular dysfunction.[135] Because left ventricular dysfunction is proportional to tachycardia rate and duration, the systolic dysfunction can be partially or completely normalized by controlling the heart rate. Left ventricular dysfunction can occur in supraventricular tachycardia, ventricular tachycardia, or even frequent premature ventricular contractions.[136] If tachycardia continues, it can cause atrio-ventricular dysynchrony, decreased myocardial contractility, elevated left ventricular filling pressure, reduced cardiac output, increased systemic vascular resistance, increased left ventricular wall stress, left ventricular enlargement, mitral insufficiency, and activation of the renin-angiotensin-aldosterone system and sympathetic nervous system,[135][137][138] which induces ventricular remodeling. This not only results in cellular structure changes but also in the depletion of stored energy, increased oxidative stress, and impaired calcium signaling.

Tachycardia-induced cardiomyopathy can be suspected if a patient with idiopathic HF has persistent tachycardia. However, it is difficult to determine whether tachycardia is the cause or effect of HF. Thus, it is difficult to differentiate tachycardia-induced cardiomyopathy from general cardiomyopathy. Tachycardia-induced cardiomyopathy is easier to diagnose if ventricular function is recovered after the restoration of sinus rhythm or control of ventricular rate.

The key principles when treating tachycardia-induced cardiomyopathy are maintaining sinus rhythm and controlling ventricular rate,[139] which helps improve left ventricular function, relieve symptoms, and prevent arrhythmia recurrence.[135] Atrial fibrillation is the most common arrhythmia accompanied by HF, while tachycardia-induced cardiomyopathy can occur if the pulse exceeds 150 beats/min.[61] It is appropriate for atrial fibrillation patients who also experience HF to maintain a ventricular rate of 60–100 beats/min. It was suggested in numerous treatment guidelines that controlling atrial fibrillation is not superior to restoring sinus rhythm,[140][141] but this view cannot necessarily be applied to tachycardia-induced cardiomyopathy caused by atrial fibrillation.[135][137] Studies conducted in patients with left ventricular failure accompanied by atrial fibrillation have found that, compared to heart rate control, the restoration of sinus rhythm provides better outcomes in terms of improving left ventricular function and quality of life.[142][143] Therefore, restoring the sinus rhythm can be considered in patients with tachycardia-induced cardiomyopathy caused by atrial fibrillation. Methods of achieving sinus rhythm restoration include drug treatment, cardioversion, and catheter ablation. Amiodarone is the most effective drug for maintaining sinus rhythm.[144] Dronedarone is dangerous to AHF patients and its use is not recommended.[145] Catheter ablation can be considered as a suitable treatment method for restoring sinus rhythm in patients with HF accompanied by atrial fibrillation, providing increased left ventricular ejection fraction and improved symptoms.[142][146][147]

Tachycardia-induced cardiomyopathy is known to occur in 6–10% of patients with supraventricular tachycardia,[148][149] while left ventricular ejection fraction is known to improve after catheter ablation.[148][150][151] Ventricular tachycardia or excessive premature

ventricular contraction can cause tachycardia-induced cardiomyopathy via ventricular desynchronization.[152] Although there are many definitions of excessive premature ventricular contraction, the occurrence of cardiomyopathy is increased when there are more than 10,000 premature ventricular contractions or more than 24–26% of frequencies over the course of 24 hours.[135][153][154] A beta-blocker is the first-line agent, while amiodarone, sotalol, and dofetilide can also be used.[135][137] Catheter ablation can be considered for treating premature ventricular contraction, with success rates of 70–90%[155] and with improvements in left ventricular ejection fraction and general symptoms.[156] In case reports and small observational studies, tachycardia-induced cardiomyopathy improved after catheter ablation even in patients with ventricular tachycardia.[137]

PERIPARTUM CARDIOMYOPATHY

1. An angiotensin-converting enzyme inhibitor or angiotensin-receptor blocker should not be used in women with peripartum cardiomyopathy prior to childbirth (class of recommendation III, level of evidence C).
2. Limited treatment options are available for peripartum cardiomyopathy; bromocriptine can be considered if needed (class of recommendation IIb, level of evidence C).
3. Subsequent pregnancy is not recommended in women diagnosed with peripartum cardiomyopathy and a left ventricular ejection fraction <25% or non-normalized left ventricular ejection fraction after treatment (class of recommendation III, level of evidence C).

Peripartum cardiomyopathy occurs in pregnant woman between the last month of pregnancy and until up to 6 months postpartum, with no discernable cause. Peripartum cardiomyopathy occurs in one of every 1,300–1,400 pregnant women and, although the occurrence rate is low, the rates of heart transplant and death are high.[157] Risk factors of peripartum cardiomyopathy include advanced maternal age, multifetal gestation, multipara status, pregnancy-induced hypertension, and long-term use of tocolytics.[158][159] While the etiology remains unknown, peripartum cardiomyopathy is considered to arise from immunologic or pregnancy-related hemodynamic causes.[160] Treatment for peripartum cardiomyopathy is quite similar to treatments for other types of HF[61][136]; however, but due to the risk of embryotoxicity, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers cannot be used.[161][162] Instead, hydralazine and nitrates can be used relatively safely, even during pregnancy.[163] One study reported improved ventricular contractility following bromocriptine administration, based on which the recommended dose is 2.5 mg twice daily for 2 weeks.[164]

A pregnant woman with suspected peripartum cardiomyopathy should be referred to a cardiologist and obstetrician. HF drugs must be administered carefully because they can affect the fetus; hence, fetal development must be closely monitored. The optimal delivery method and timing have not been established. If the conditions of the mother and the baby do not worsen, preterm delivery is not needed.[165] However, if the mother is hemodynamically unstable or has severe HF, emergency delivery must be considered regardless of gestational age. A team consisting of a cardiologist, obstetrician, anesthesiologist, intensivist, and neonatologist would need to carefully evaluate the case and discuss the delivery timing. If the mother is hemodynamically unstable or requires mechanical assistance, a cesarean section must be considered. The use of spinal or epidural anesthesia for cesarean section is advised.[165]

The prognosis of peripartum cardiomyopathy usually depends on the recovery of ventricular contractility, which improves within 6 months after the symptom onset in 30–50% of patients and such patients have good long-term prognosis. However, if ventricular contractility does not improve, peripartum cardiomyopathy has a prognosis similar to that of dilated cardiomyopathy.[166] Moreover, women with a history of peripartum cardiomyopathy who experience another pregnancy may die if ventricular function deteriorates; however, if ventricular contractility is normalized, subsequent pregnancy would not be fatal.[167] If ventricular function continues to deteriorate, there is a high risk of venous thrombosis and thus anticoagulant therapy is advised. However, during pregnancy, heparin or low-molecular-weight heparin must be considered due to the embryotoxicity of warfarin.

CONCLUSION

In this part of guideline, the management strategies according to the etiologies or co-morbidities in AHF patients were discussed. Although the general principles of management follow the recommendations of the guidelines for AHF, it is important to identify the etiologies and accompanying disease as there are special considerations for each specific etiology. Various circumstances should be taken into account and appropriate management plan should be established for each patient.

SUPPLEMENTARY MATERIALS

Supplementary Table 1
Criteria used to judge the level of evidence and establish the class of recommendation for AHF

Click here to view

Supplementary Table 2
Formulations typically used with each class of recommendation

Click here to view

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