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Critical Care of Acute Heart Failure

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Abstract

Acute heart failure is a life-threatening medical condition. Improving acute heart failure care is important. Early diagnosis and evaluating the etiology are important in acute heart failure. Patients with suspected acute heart failure should have a diagnostic workup, and appropriate pharmacological and nonpharmacological management should be started promptly and in parallel. Diagnosis of acute heart failure should be based on history and symptoms. The physical examination typically presents with some combination of increased congestion and decreased peripheral perfusion, further confirmed by electrocardiogram, chest radiograph, biomarkers, and echocardiogram. The first step in the management of a patient is to address life-threatening issues. Patients with respiratory failure or cardiogenic shock should be treated soon. The next step is the identification of precipitants that needs urgent management. Evidence-based medication to reduce morbidity and mortality for patients with heart failure includes an angiotensin converting enzyme inhibitor, angiotensin receptor blocker, or angiotensin receptor-neprilysin inhibitor; a beta blocker; and a mineralocorticoid receptor antagonist. During an acute heart failure episode, management of these agents depends upon whether the patient has contraindications to therapy such as hemodynamic instability or acute kidney injury. Once the patient is stable, therapies are carefully initiated, reinitiated, or titrated with appropriate follow-up.

Keywords: acute heart failure, critical care

1. Introduction

Acute heart failure is a relevant public health problem causing a majority of hospital admissions in patients aged of 65 years or more. Despite major achievements in the treatment of chronic heart failure over the last decades, which led to marked improvement in long-term survival, outcomes of acute heart failure remain poor with 90-day rehospitalization and 1-year

mortality rates reaching 10–30%. There is an unmet need for improving acute heart failure care, including critical care to patient's outcome.

2. Critical care of acute heart failure

2.1. Introduction

Acute heart failure (AHF) is a disease with high rehospitalization and mortality rates. Improving acute heart failure critical care is important.

2.2. Definition of acute heart failure

Acute heart failure (AHF), also known as acute decompensated heart failure or cardiac failure, refers to rapid onset or worsening of symptoms and/or signs of heart failure (HF). It is a life-threatening medical problem requiring evaluation and management soon.

AHF may present as a first occurrence or as a consequence of acute decompensation of chronic HF. AHF may be caused by primary cardiac dysfunction or precipitated by extrinsic factors, often in patients with chronic HF.

2.3. Management of acute heart failure

AHF is a life-threatening medical problem; thus, rapid transfer to the nearest hospital is essential, which has a cardiology department and coronary care/intensive care unit (CCU/ICU). Early diagnosis and evaluating the etiology are important in AHF. Therefore, all patients with suspected AHF should have a diagnostic workup and appropriate pharmacological and nonpharmacological management should be started promptly and in parallel.

2.4. Causes/precipitant factors

The most frequent primary causes of AHF include ischemic, inflammatory, arrhythmia, and acute valve insufficiency. Decompensation of chronic HF can occur without known precipitant factors, but more often with one or more factors, such as infection, uncontrolled hypertension, rhythm disturbances, or nonadherence with drugs/diet (**Table 1**) [1]. Some of them leading to decompensation, which need to be treated/corrected urgently, include acute coronary syndromes (ACS), hypertensive emergency, severe arrhythmias, acute mechanical cause underlying AHF, or acute pulmonary embolism [1].

2.5. Diagnosis

Initial diagnosis of AHF should be based on a thorough history assessing symptoms, prior cardiovascular history, and potential cardiac and noncardiac precipitants. The physical examination of a patient with AHF typically presents with some combination of increased congestion and, less frequently, decreased peripheral perfusion, further confirmed by appropriate additional investigations such as image and laboratory assessment (with specific biomarkers).

Acute coronary syndrome
Arrhythmia (tachyarrhythmia and bradyarrhythmia)
Excessive rise in blood pressure
Nonadherence with diet intake or medications
Toxic substances (alcohol, recreational drugs)
Drugs (e.g., NSAID, corticosteroid, cardiotoxic chemotherapeutics)
Exacerbation of chronic obstruction pulmonary disease
Pulmonary embolism
Surgery
Stress
Metabolic/hormonal derangements
Cerebrovascular insult
Acute mechanical cause

Table 1. Factors triggering acute heart failure.

2.5.1. *The information from physical examination*

2.5.1.1. *Vital sign*

Poorly controlled hypertension is one of the predictor factors of AHF and should be avoided. Hypotension is a dangerous sign of poor prognosis for patients with AHF. Unstable heart rate and rhythm are not only a cause of the AHF episode, but also increase the risk of unstable hemodynamic. Respiratory rate is often not as carefully assessed clinically as other vital signs, but may represent inadequate resolution of the initial episode of lung edema or a new event, such as a pulmonary embolus. Fever reflects the possibility of underlying infectious process, which can instigate AHF exacerbations. Oxygen saturation is also an important vital sign for HF patients and should be measured.

2.5.1.2. *Body weight*

Body weight provides an important information of severity of fluid overload and response to therapy. It is easy to measure and should be obtained as early as possible. Urine output and daily input and output measures are provided in more detail, but many clinical practices appear to be more difficult.

2.5.1.3. *Jugular venous pressure*

Jugular venous pressure (JVP) is a useful physical examination finding to monitor fluid status and response to therapy in the AHF patient. Elevation of the JVP suggests that there is persistent volume overload and need more diuresis. JVP will be influenced by significant tricuspid regurgitation, while the elicitation of hepatojugular reflux can augment the interpretation.

2.5.1.4. Heart sound

Cardiac auscultation is very basic and an important skill to follow the presence and severity of extra heart sounds (S3 and S4) and murmurs. The appearance of an S3 is associated with elevated ventricular pressure and poor left ventricular function. The reduction in the intensity of mitral and tricuspid regurgitation murmurs are the sign of reduction of ventricular filling pressure and volume.

2.5.1.5. Breath sound

Breath sound examination can provide the signs of lung congestion or pleural effusion. It can also evaluate the possibility of an underlying pneumonia.

2.5.1.6. Extremities

Extremities edema reflects the status of fluid overload, and extremities temperature reflects the distal perfusion. Both of them should be closely monitored to understand the severity of low cardiac output/fluid overload and the response to management.

2.5.2. What biochemistry studies should be ordered to help establish the diagnosis?

B-type natriuretic peptide (BNP) and N-terminal-proBNP (NT-proBNP)—plasma concentrations of BNP and NT-proBNP—are increased in the presence of elevated ventricular pressure and volume, and have been used to assist in the diagnosis of AHF. There is no absolute “diagnostic level” of these biomarkers for AHF; most use different ranges. For example, a BNP less than or equal to 100 pg/mL is strongly suggestive of nonheart failure etiology for the dyspnea, BNP greater than 400 pg/mL is strongly supportive of AHF and BNP 100–400 pg/mL is indeterminate. High BNP levels may be the result of factors other than HF (e.g., age, renal dysfunction, myocardial infarction, acute pulmonary embolism, and high output states such as cirrhosis), and some factors may cause lower BNP levels (e.g., obesity, within 1 hour of flash pulmonary edema, acute mitral regurgitation, and mitral stenosis) [2–6].

Renal function markers: blood urea nitrogen (BUN) and creatinine are markers of renal function. Chronic renal insufficiency is a frequent comorbidity in patients with heart failure, and AHF episodes are often accompanied by acute on chronic renal failure.

Troponin: increased troponins reflect myocardial damage. ACS may precipitate AHF, but much more frequently, myocardium damage from AHF itself leading to mild-to-moderate troponin elevations. Distinguishing the “troponinemia” of heart failure from that of ACS or myocardial infarction can often be challenging and requires synthesis of symptoms and other clinical information [7].

2.5.3. What imaging studies should be ordered to help establish the diagnosis?

Electrocardiogram (ECG): the ECG can provide information of myocardial ischemia, chamber dilatation or hypertrophy, arrhythmias, and electrolyte disorders as contributing factors to AHF.

Chest radiography: the chest X-ray (CXR) can reflect pulmonary congestion or pleural effusions. Changes in cardiac silhouette may reflect cardiomegaly or pericardial effusion.

Echocardiogram: echocardiography is probably the most useful noninvasive imaging study for HF. It provides information on structure, and function of all cardiac chambers and valves. Potential wall motion abnormalities and estimates of hemodynamics were also able to be detected by echocardiography.

Pulmonary artery catheterization: the pulmonary artery (PA) catheter is a very useful diagnostic tool in properly selected patients. In patients with evidence of shock with unclear etiology, measurement of pulmonary capillary wedge pressures (PCWP), cardiac output, and vascular resistances can provide key information to guide appropriate selection of therapy. A PA catheter should not be routinely used in most patients with AHF due to potential complication (e.g., infection, vascular injury, etc.).

2.6. Clinical classification

Clinical classification can be based on physical examination to detect the presence of congestion (“wet” vs. “dry” if present vs. absent) and/or peripheral hypoperfusion (“cold” vs. “warm” if present vs. absent) [8, 9]. The combination of these options identifies four groups: warm and wet (well perfused and congested), cold and wet (hypoperfused and congested), cold and dry (hypoperfused without congestion), and warm and dry (compensated, well perfused without congestion). This classification may be helpful to guide therapy in the initial phase and carries prognostic information. The “cold and wet” groups have most poor prognosis and need urgent management [1, 8, 9] (**Figure 1**).

2.7. Monitor

2.7.1. Invasive monitor

The insertion of an arterial line in patients with AHF and cardiogenic shock was recommended. The arterial line allows for repetitive sampling of arterial blood gases, providing important information on oxygenation (PaO_2), ventilation (PaCO_2), acid-base balance, electrolytes, and lactate levels. The continuous measurement of arterial pressure allows for the appropriate titration of vasoactive medication.

The central venous catheter enables the monitoring of central venous pressure and allows the safe and continuous administration of vasoactive drugs and inotropes in patients with AHF who require intensive treatment. Central venous oxygen saturation (ScvO_2) can also be monitored with the central venous catheter. In combination with increased lactate levels and signs of organ dysfunction, $\text{ScvO}_2 < 60\%$ indicates severe hypoperfusion and mandates further diagnostics and urgent treatment.

A pulmonary artery catheter (PAC) may be considered in patients with hypotension and hypoperfusion to evaluate the volume status and cardiac output. However, the use of a PAC did not improve the survival and was associated with more adverse events. Based on the imbalance between potential benefits and known risks, PAC should not routinely be used to

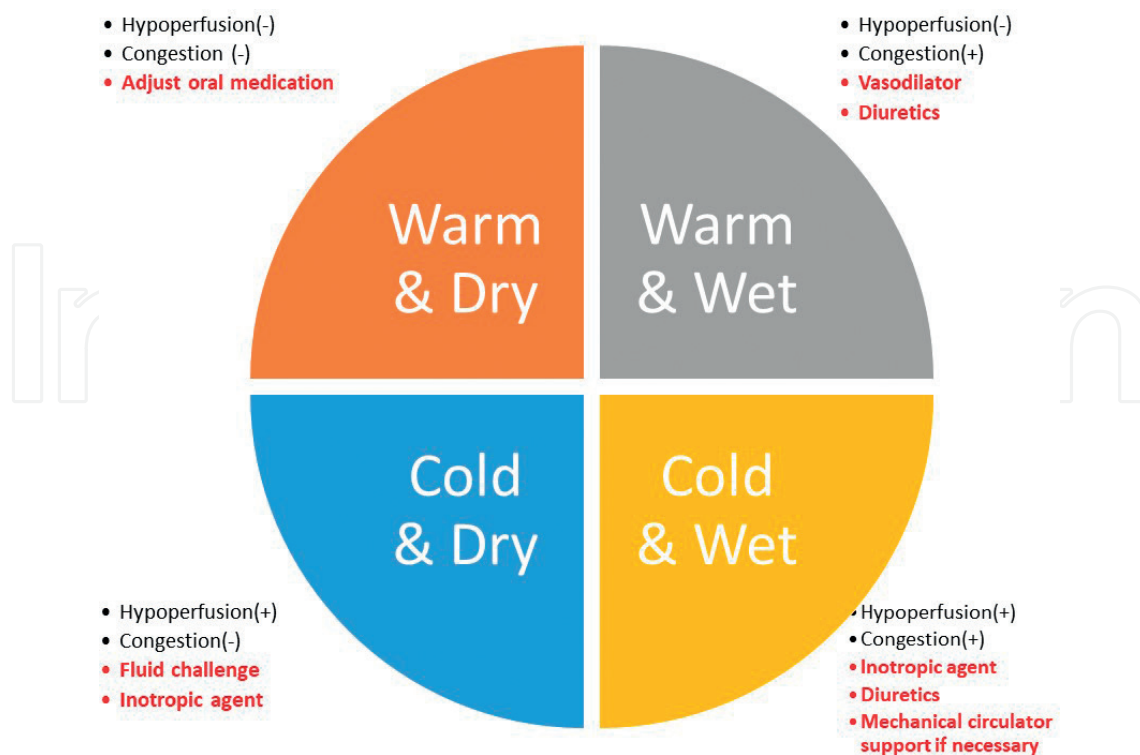


Figure 1. Classification and management of AHF.

monitor patients with AHF, but it can still be justified to use PAC in selected populations by experienced physicians. PAC is most appropriately used in critical patients who need rapid evaluation of vasoactive medications or fluid balance.

2.7.2. Noninvasive hemodynamic monitor

Noninvasive techniques are undergoing considerable development to avoid complications of invasive monitor. However, none can be currently recommended for routine clinical use. Invasive techniques such as the PAC and transpulmonary thermodilution remain the reference standard.

2.7.2.1. Contour of the pulse wave

Several algorithms have been proposed to determine cardiac output based on determination of systolic area by analysis of the contour of the pulse wave. These signals are often obtained from an arterial line.

2.7.2.2. Digital photoplethysmography

Digital photoplethysmography is a technique for continuous measurement of blood volume changes. Severe skin vasoconstriction, which is common in cardiogenic shock, impairs signal quality of blood pressure and is an important limitation of the technique. These monitors are not useful when arterial impedance is variable, such as with vasoconstrictor administration,

unless given continuously and steady state has been reached. Digital photoplethysmography techniques do not appear to be sufficiently effective in assessing cardiac output in resuscitation patients with microcirculatory disorders, peripheral vasoconstriction, or high blood pressure lability.

2.7.2.3. Thoracic bioimpedance

Transthoracic electric bioimpedance is a noninvasive method for cardiac output measurement. Several hemodynamic parameters can be measured and calculated using the technique including flow (e.g., stroke volume/stroke index), resistance (e.g., systemic vascular resistance/index), contractility (e.g., cardiac power index, systolic time ratio, pre-ejection period, left ventricular ejection time, velocity index, acceleration index), and fluid (e.g., thoracic fluid content). Thoracic bioimpedance data may be useful in fluid management in patients with AHF, and the differentiation of cardiogenic from pulmonary causes of acute dyspnea. Bioimpedance might be useful for trend analysis, but the data should be interpreted cautiously, as the method is associated with limitations that may affect its accuracy (e.g., arrhythmias, mechanical ventilation, body motion, and factors that affect conductivity between the electrodes and the skin-like temperature and humidity).

2.8. Management

2.8.1. First step

The first step in management of the patient with AHF is to address life-threatening issues. Patients with respiratory distress/failure or cardiogenic shock should be treated soon and be triaged to a location where immediate respiratory and cardiovascular support can be provided.

2.8.1.1. Respiratory failure

Respiratory failure is the most frequent life-threatening condition for patients with AHF. Immediate management includes oxygen supply and removal of overload fluid.

Oxygen supply and ventilator support: immediate administration of supplemental oxygen is the most readily available means to improve oxygenation. If remain inadequate, rapid use of noninvasive ventilatory support continuous positive airway pressure or nasal intermittent positive pressure ventilation has been shown to be very effective in rapidly improving symptoms, hemodynamics, and metabolic abnormalities associated with AHF [10–12]. If noninvasive measures are insufficient, rapid intubation with mechanical positive pressure ventilation should be done.

Diuretic: most patients also have significant volume overload leading to the respiratory insufficiency, and diuretics remain the most commonly administered agent for AHF. Rapid administration of intravenous loop diuretics is recommended for volume overload and can relieve symptoms quickly as well as decrease the underlying volume overload [13–15].

Ultrafiltration: potential benefits of ultrafiltration over intravenous diuretics include more effective removal of sodium, minimal effects on serum electrolytes, decreased neurohormonal

activation, and adjustable and potentially very rapid fluid removal rates. The cost, need for vascular access, need for nursing training, and support are all potential barriers to ultrafiltration in clinical practice. Identifying the most appropriate patients for ultrafiltration therapy is an area of controversy and active clinical research.

2.8.1.2. Cardiogenic shock

Cardiogenic shock is defined as a hypotension (SBP < 90 mmHg) with signs of hypoperfusion (cold sweated extremities, oliguria, mental confusion, dizziness, narrow pulse pressure, etc.). Emergency management is necessary to improve organ perfusion by increasing cardiac output and blood pressure. After fluid challenge, pharmacologic management consists of positive inotropic agent and a vasopressor as needed. Positive inotropes agents and vasopressors used to treat acute heart failure are discussed below and listed in **Table 2**. However, rather than combining several inotropes, device therapy has to be considered when there is an inadequate response.

Positive inotropes: Positive inotropes increase heart contractility and cardiac output by increasing cAMP and intracellular calcium. Dobutamine is a predominant beta-1 adrenergic receptor agonist, which produces positive inotropic and chronotropic effects. The mechanism of action is the binding of the beta-1 receptor, leading to enhanced myocardial contractility. There is also a modest alpha and beta-2 effect, which causes mild peripheral vasodilation; in the context of increasing cardiac output, this can cause a variable effect on mean arterial pressure. The major side effects of dobutamine are atrial and ventricular tachyarrhythmias. Registry data of AHF patients suggest worse outcomes with dobutamine, and hence, its use is limited to patients with poor response to diuretics and vasodilators and patients in overt cardiogenic shock [16, 17]. Dopamine has variable effects on different receptors at different doses; conventionally at low doses (0–2 mcg/kg/min), there is preferential dopamine receptor activation leading to enhanced renal artery vasodilation and enhanced renal perfusion; at 2–10 mcg/kg/min, there is enhanced norepinephrine release, leading to enhanced myocardial contractility and mild peripheral vasoconstriction; at doses above 10 mcg/kg/min, there is preferential alpha adrenergic receptor activation causing peripheral vasoconstriction and an increase in mean arterial pressure [18]. Phosphodiesterase type III inhibitors, such as milrinone and enoximone, are a positive inotropic agent as well as a vasodilator. Its mechanism of action is the inhibition of the breakdown of cyclic adenosine monophosphate (cAMP) in cardiac myocytes, leading to the increase of cAMP-mediated Ca⁺⁺ in the myocyte and hence enhanced myocyte contractility. Similarly, in the vascular smooth muscle, its action is that of increasing cAMP-mediated contractile protein phosphorylation, leading to vascular relaxation. The hemodynamic changes seen with milrinone include an increased cardiac output, decreased systemic vascular resistance, reduced PCWP, and typically a mild decrease in mean arterial pressure. Milrinone did not significantly decrease hospitalization length of stay, and did lead to significantly more hypotension and atrial arrhythmias. Increased myocardial ischemia and increased mortality in patients with ischemic heart disease may also occur. Although bolus loading was suggested, some clinicians no longer use a bolus loading dose to avoid significant hypotension. Patients should be carefully monitored in intensive care unit when used with milrinone [19–21]. Levosimendan is an ATP-dependent potassium channel activator with myocardial calcium sensitizing and possible PDE III inhibitor effects, and acts

Inotropes/vasopressors	Dose	Recommended
Dobutamine (beta-1 adrenergic receptor agonist)	2–20 µg/kg/min	Also a vasodilator, may increase the risk of arrhythmia and hypotension
Dopamine (variable effects on different receptors at different dose)	3–5 µg/kg/min; inotropic (beta+) >5 µg/kg/min: (beta+), vasopressor (alpha+)	May increase the risk of arrhythmia
Milrinone (phosphodiesterase type III inhibitors)	25–75 µg/kg over 10–20 min (optional) 0.375–0.75 µg/kg/min	Also a vasodilator not recommended in acutely worsened ischemic heart failure. Dose adjustment is required in the presence of renal insufficiency, hypotension, or arrhythmias.
Enoximone (phosphodiesterase type III inhibitors)	0.5–1.0 mg/kg over 5–10 min (optional) 5–20 µg/kg/min	Also a vasodilator similar as Milrinone
Levosimendan (ATP-dependent potassium channel activator with myocardial calcium sensitizing effects and possible PDE III inhibitor effects)	12 µg/kg over 10 min (optional) 0.1 µg/kg/min, which can be decreased to 0.05 or increased to 0.2 µg/kg/min	Also a vasodilator increases the risk of hypotension
Norepinephrine (alpha agonist)	0.2–1.0 µg/kg/min	Increase systemic vascular resistance and afterload
Epinephrine (alpha and beta agonist)	0.05–0.5 µg/kg/min	Increase the risk of arrhythmia

Table 2. Positive inotropes and/or vasopressors for acute heart failure.

as a vasodilator and inotrope. Hypotension and arrhythmias may occur, and patients should be carefully monitored. Using a bolus loading dose or not depends on the blood pressure [22–24]. Monitoring of response to these therapies depends upon the hemodynamic, peripheral perfusion, and other target organ functions.

Vasopressors: Drugs with peripheral arterial vasoconstrictor action such as norepinephrine or dopamine in higher doses (5 mg/kg/min) are given to patients with marked hypotension. These agents are given to raise blood pressure and redistribute blood to the vital organs. However, LV afterload increases under vasopressors use. A subgroup analysis suggested that norepinephrine would have fewer side effects and lower mortality [25]. Epinephrine (adrenaline) should be restricted to patients with persistent hypotension despite adequate cardiac filling pressures and the use of other vasoactive agents, as well as for resuscitation protocols [26].

Mechanical circulatory support: In cases of severe AHF, which is refractory to medical therapy, temporary circulatory support (TCS) can be utilized to improve end organ perfusion. TCS ranges from percutaneously inserted devices, such as intra-aortic balloon pump (IABP) to ventricular assist devices (VAD). IABP can be used at many centers for severe cardiac compromise and provide benefits in decreasing afterload and increasing coronary perfusion and cardiac output. The contraindication is moderate-to-severe aortic insufficiency and aortic aneurysms/dissections, and is limited by vascular access issues and complications. IABP could only partially increase cardiac output, and there is no benefit for right heart failure. In cases of complete hemodynamic collapse or severe right ventricular failure, other mechanical support devices may be used at specialized centers, including VADs and extracorporeal membrane

oxygenation (ECMO). ECMO can be placed to completely bypass the cardiopulmonary circulation. Additional surgically placed TCS includes semidurable continuous-flow ventricular assist devices. TCS can serve as a “bridge to recovery” or as a “bridge to decision” in patients who may need implantation of permanent LV assist devices or cardiac transplantation.

2.8.2. Second step

The next step to identification of precipitants/causes leading to decompensation that needs urgent management, including ACS, hypertensive emergency, arrhythmia, acute mechanical cause underlying AHF, and pulmonary embolism [1].

Acute coronary syndrome: coexistence of ACS and AHF identifies a very-high-risk group where an immediate invasive strategy with intent to perform revascularization is recommended [27, 28].

Hypertensive emergency: aggressive blood pressure reduction (in the range of 25% during the first few hours and cautiously thereafter) should be considered and initiated as soon as possible with i.v. vasodilators in combination with loop diuretics, which is recommended [29, 30].

Arrhythmias: severe rhythm disturbances in patients with AHF and unstable conditions should be corrected urgently with medical therapy, electrical cardioversion, or temporary pacing [31, 32].

Acute mechanical cause underlying AHF: this may present as a complication of ACS (free wall rupture, ventricular septal defect, or acute mitral regurgitation), chest trauma or cardiac intervention, or as acute valve incompetence. Echocardiography is essential for diagnosis, and treatment typically requires emergency surgical repair. Surgery might not be performed soon in some special consideration. Patients suffer from postmyocardial infarction ventricular septal defect, and acute mitral regurgitation with acceptable hemodynamic might use IABP to unload ventricular pressure and perform surgery several days later till cardiac muscle inflammation improved.

Acute pulmonary embolism: patients presenting with acute pulmonary embolism should be managed according to the guidelines. Immediate reperfusion either with thrombolysis, catheter-based approach, or surgical embolectomy is recommended if hemodynamically unstable [33].

2.8.3. Other medications for AHF

2.8.3.1. Vasodilator therapy

Use of vasodilator therapy in patients with AHF is based upon hemodynamic response, since evidence on efficacy and safety of vasodilatory therapy in AHF is limited. The routine use of vasodilators does not improve outcomes, and should be avoided [30, 34, 35].

Nitrates: nitrates can be very effective as vasodilators, producing rapid decreases in pulmonary congestion, left ventricular end diastolic pressure, LV wall stress, and myocardial oxygen consumption. Rapid administration of intravenous nitrates in patients with severe pulmonary edema decreased the need for mechanical ventilation and myocardial infarction compared to a high-dose furosemide strategy in a randomized study. It has coronary vasodilatory effects as well, making it a good option for patient with ongoing ischemia. Initial

intravenous dose is typically 20 mcg/min, with a doubling of the dose every 5–15 min. Other options for administration include sublingual tablets and sprays as well as topical pastes. Major side effects include hypotension and headache.

Nesiritide: nesiritide is a recombinant B-type natriuretic peptide. It has balanced venous and arteriolar actions, and modestly enhances diuresis through direct renal effects. The dose starts at 0.01 mcg/kg/min. Though there may be a role for nesiritide in some special populations (i.e., diuretic-resistant patients), however, there was a higher rate of hypotension and there was no significant change in the rate of death, rehospitalization, or renal function. Routine use of nesiritide in acute decompensate heart failure was not recommended.

2.8.3.2. Digoxin

Digoxin is mostly indicated in patients with AHF and rapid ventricular rate. However, in patients with renal failure or other factors affecting digoxin metabolism (including other drugs and elderly), the maintenance dose should be adjusted and avoid overdose.

2.8.4. Management of evidence-based oral therapies

Evidence-based medication to reduce morbidity and mortality for patients with chronic HF includes an angiotensin-converting enzyme (ACE) inhibitor, angiotensin receptor blocker (ARB), or angiotensin receptor-neprilysin inhibitor (ARNI); a beta blocker; and a mineralocorticoid receptor antagonist (MRA). During an acute HF episode, management of these agents depends upon whether the patient was already receiving these medications and whether the patient has contraindications to therapy such as hemodynamic instability or acute kidney injury. Once the patient is stable, evidence-based therapies are carefully initiated, reinitiated, or titrated with arrangements for appropriate outpatient follow-up. In stable patients, ACE inhibitor, ARB, or ARNI and beta blocker therapy should be initiated prior to hospital discharge, and then MRA should be added as soon as possible. Ivabradine reduces the risk of hospitalization in patients with HF and can increase stroke volume and maintain cardiac output when heart rate slows down. Ivabradine may be considered to be used with dobutamine at the acute phase [36, 37].

2.8.5. Common pitfalls and side effects of management

Management of patients with AHF is complex and highly specific to the individual. However, there are some common pitfalls:

Hypotension: most therapies for AHF can cause hypotension, and the development of hypotension related to poor clinical outcomes. Blood pressure, central venous pressure, body weight, urine output, and renal function should carefully be monitored to avoid hypotension. If hypotension happens, rapidly evaluate the perfusion and delete vasodilator (be especially aware of nitrates), and position the patient as necessary to improve perfusion. In many patients, raising the legs will provide sufficient augmentation of venous return to improve symptoms.

Worsening renal function: since worsening renal function can result from inadequate diuresis or be the result of excessive diuresis with volume depletion. In these situations, measurements of baseline BUN and creatinine as comparators and central venous pressure are very

helpful to have treatment decision. Transient decreases in renal function may not portend the poor prognosis, as previously believed, and may just reflect the response to therapy.

Electrolyte imbalance: electrolyte imbalance is often noted under diuretic use and might lead to life-threatening arrhythmias. Close monitoring and positive correction are necessary.

3. Conclusion

- The first step in management of the patient with AHF is to address life-threatening issues. Patients with respiratory distress/failure or hemodynamic compromise should be treated soon.
- Respiratory failure should be treated first; initial therapy includes supplemental oxygen and assisted ventilation. Patients with respiratory failure due to AHF who fail to improve with non invasive ventilation require endotracheal intubation for conventional mechanical ventilation.
- Most patients will also have significant volume overload leading to the respiratory insufficiency; diuretics remain the most commonly administered agent for AHF. Rapid administration of intravenous loop diuretics is recommended for volume overload. Ultrafiltration should be performed if failed to improve under diuretics use.
- Severe low cardiac output requires aggressive management of their shock to mitigate or prevent the related end organ damage. Positive inotropes agents and vasopressors are used to treat low cardiac output and hypotension. In cases of severe AHF, which is refractory to medical therapy, temporary circulatory support (TCS) can be utilized to improve end-organ perfusion.
- The next step to identification of precipitants/causes leading to decompensation that needs urgent management, including acute coronary syndrome, hypertensive emergency, arrhythmia, and acute mechanical and pulmonary embolism.
- Vasodilators may be required to correct elevated filling pressures. The routine use of vasodilators does not improve outcomes, and should be avoided.
- Evidence-based medication to reduce morbidity and mortality for patients with chronic HF includes an angiotensin converting enzyme (ACE) inhibitor, angiotensin receptor blocker (ARB), or angiotensin receptor-neprilysin inhibitor (ARNI); a beta blocker; and a mineralocorticoid receptor antagonist (MRA). During an acute HF episode, management of these agents depends upon whether the patient was already receiving these medications and whether the patient has contraindications to therapy such as hemodynamic instability or acute kidney injury. Once the patient is stable, evidence-based therapies are carefully initiated.

Conflict of interest

None.

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