

Acute heart failure

Acute heart failure syndromes (AHFS) may be defined as new-onset, gradual, or rapidly worsening HF signs and symptoms that require urgent therapy. These symptoms reflect congestion behind the failing ventricle and/or hypoperfusion. Patients can be categorized into hemodynamic subsets based on assessment of physical signs and symptoms of congestion and/or hypoperfusion. Patients can be described as “wet” or “dry” depending on volume status, as well as “warm” or “cool” based on adequacy of tissue perfusion.

Patients with AHFS can be further classified as having new-onset or worsening chronic HF. Approximately 80% of patients with AHFS have chronic HF. Patients with advanced HF have low blood pressure (BP), renal impairment, and signs or symptoms refractory to standard medical therapy and represent up to 10% of hospitalized patients with AHFS.

Clinical presentation of acute heart failure

Subset I (Warm and Dry)

- Patients considered well compensated and perfused, without evidence of congestion
- No immediate interventions necessary except optimizing oral medications and monitoring

Subset II (Warm and Wet)

- Patients adequately perfused and display signs and symptoms of congestion
- Main goal is to reduce preload (PCWP) carefully with loop diuretics and vasodilators

Subset III (Cool and Dry)

- Patients are inadequately perfused and not congested
- Hypoperfusion leads to increased mortality, elevating death rates fourfold compared with those who are adequately perfused
- Treatment focuses on increasing CO with positive inotropic agents and/or replacing intravascular fluids
- Fluid replacement must be performed cautiously because patients can rapidly become congested

Subset IV (Cool and Wet)

- Patients are inadequately perfused and congested
- Classified as the most complicated clinical presentation of AHF with the worst prognosis
- Most challenging to treat; therapy targets alleviating signs and symptoms of congestion by increasing CI as well as reducing PCWP while maintaining adequate mean arterial pressure
- Treatment involves a delicate balance between diuretics, vasodilators, and inotropic agents
- Use of vasopressors is sometimes necessary to maintain blood pressure.

Laboratory Assessment

BNP, electrolytes and blood glucose, serum creatinine and blood urea nitrogen to assess renal function. Complete blood cell count is measured to determine if anemia or

infection is present. Creatine kinase and/or troponin concentrations are used to diagnose ischemia, and hepatic transaminases are measured to assess hepatic congestion. Thyroid function tests are measured to assess hyperthyroidism or hypothyroidism as causes of AHF. A urinalysis is attained in patients with an unknown history of renal disease to rule out nephrotic syndrome.

TREATMENT OF ACUTE HEART FAILURE

Desired Therapeutic Outcomes

The goals of therapy for AHF are to (a) correct the underlying precipitating factor(s); (b) relieve the patient's symptoms; (c) improve hemodynamics; (d) optimize a chronic oral medication regimen; and (e) educate the patient, reinforcing adherence to lifestyle modifications and the drug regimen.

The ultimate goal for a patient hospitalized for AHF is the return to a compensated HF state and discharge to the outpatient setting on oral medications. Only through aggressive management to achieve all of these goals will a patient's prognosis be improved and future hospitalizations for acute decompensations be prevented.

Oral agents such as β -blockers, ACE inhibitors or ARBs, and aldosterone antagonists should be initiated as soon as possible during the hospitalization. These chronic oral medications not only improve mortality and prevent readmissions, acutely they also contribute to improvement in hemodynamics. Patient education prior to discharge from the hospital is recommended to assist in minimizing adverse effects and nonadherence.

Pharmacologic Approaches to Treatment

Treatment of AHF targets relief of congestion and optimization of CO utilizing oral or IV diuretics, IV vasodilators, and, when appropriate, inotropes, based on presenting hemodynamics. Current treatment strategies in AHF target improving hemodynamics while preserving organ function.

Diuretics

1. Mechanism and onset

Loop diuretics, including furosemide, bumetanide, and torsemide, are the diuretics of choice in the management of AHF. Furosemide is the most commonly used agent. Diuretics decrease preload by functional venodilation within 5 to 15 minutes of administration and subsequently by an increase in sodium and water excretion. This provides rapid improvement in symptoms of pulmonary congestion.

2. Route

Diuretics is recommended to be administered by intravenous route in AHF due to concern about adequate absorption of oral administration due to bowel oedema Bolus injection should be administered at a rate not exceeding 4 mg per minute to avoid ototoxicity

3. Dose and renal function

Patients who received double their regular oral diuretic dose by intravenous route experienced more weight loss, diuresis and subsequent symptom relief compared to those who receive intravenous doses equivalent to their oral doses. Relatively higher doses of diuretics are needed in patients with renal impairment because they need adequate glomerular filtration to reach their site of action. For example if creatinine clearance is more than 75 ml/min, infusion rate of intravenous furosemide is 10 mg/hour. However, in patients with creatinine clearance less than 25 ml/min, the dosing rate would be 20 mg increased to 40 mg/hour. Lower doses are needed if the patient improves.

6. Diuretic resistance

Occasionally, patients with HF do not respond to a diuretic, defined as failure to achieve a weight reduction of at least 0.5 kg (or negative net fluid balance of at least 500 mL) after several increasing bolus doses can be managed by either increasing the dose of the diuretics, switching to intravenous infusion or addition of oral diuretic with a different mechanism of action such as thiazide (hydrochlorothiazide, bendroflumethiazide) or thiazide like diuretics (metolazone) to counteract diuretic resistance. The addition of these diuretics provide synergistic diuretic effect by preventing sodium uptake from the distal tubule. It should be noted that this co-administration of these diuretics increase the risk of electrolyte abnormalities (hypokalaemia, hyponatremia) and renal dysfunction. Therefore, the renal function and electrolytes should be closely monitored. Combining diuretics should be used with caution due to an increased risk for cardiovascular collapse due to rapid intravascular volume depletion. Strict monitoring of electrolytes, vital signs, and fluid balance is warranted.

7. Monitoring

Careful use of diuretics is recommended to avoid overdiuresis. Monitoring parameters for diuretics includes: ↓HF symptoms, weight (loss or gain), Signs of volume depletion (Weakness Hypotension, dizziness Orthostatic changes in BP, ↓Urine output ↑BUN), Serum potassium and magnesium (avoid hypokalemia and hypomagnesemia), ↑Uric acid, ↑Glucose, Weight loss.

Finally, poor CO may contribute to diuretic resistance. In these patients, it may become necessary to add vasodilators or inotropes to enhance perfusion to the kidneys. Care must be taken because vasodilators can decrease renal blood flow despite increasing CO through dilation of central and peripheral vascular beds.

Vasodilators

IV vasodilators cause a rapid decrease in arterial tone, resulting in a decrease in SVR and a subsequent increase in SV and CO. Additionally, vasodilators reduce ventricular filling pressures (PCWP) within 24 to 48 hours, reduce myocardial oxygen consumption, and decrease ventricular workload. Vasodilators are commonly used in patients presenting with AHF accompanied by moderate to severe congestion. This

class includes nitroglycerin, nitroprusside, and nesiritide. Usual Doses and Monitoring of Commonly Used Hemodynamic Medications: BP, HR, urinary output and kidney function, ECG, extremity perfusion.

IV nitroglycerin is primarily used as a preload reducer for patients exhibiting pulmonary congestion or in combination with inotropes for congested patients with severely reduced CO. Continuous infusions of nitroglycerin should be initiated at a dose of 5 to 10 mcg/min and increased every 5 to 10 minutes until symptomatic or hemodynamic improvement. Effective doses range from 35 to 200 mcg/min.

Inotropic Agents

There are several practical considerations to dobutamine therapy in AHF. First, owing to its vasodilatory potential, monotherapy with dobutamine is reserved for patients with systolic blood pressures greater than 90 mm Hg. However, it is commonly used in combination with vasopressors in patients with lower systolic blood pressures.

In patients on β -blocker therapy, it is recommended that consideration be given to the use of phosphodiesterase inhibitors such as milrinone, which do not depend on β -receptors for effect.

Dopamine is most commonly reserved for patients with low systolic blood pressures and those approaching cardiogenic shock. As with other inotropes, dopamine is associated with a risk for arrhythmias.