Emergency Department Stabilization of Heart Failure

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KEYWORDS

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• Ventilation • Vasodilators • Ultrafiltration • Interventions
• Nesiritide • Morphine • Emergency department
• Stabilization

Patients who have heart failure (HF) make up a clinically diverse population. They are a heterogenous group that has multiple complicating comorbities, various etiologies of HF, and differing pathophysiologic triggers resulting in acute decompensation.[1,2] Increased understanding of the diversity of HF patients has led to new insights in the emergent management of these patients. Physicians and researchers are re-evaluating the properties of intravenous diuretics, vasodilators, and inotropes commonly used to alleviate congestion and restore hemodynamic stability. In particular, the shift has been to re-examine how these therapies should be administered, which HF patients should receive them, and the consequences of these therapeutic decisions.

CLASSIFICATION OF HEART FAILURE

HF patient types have not been well described or tailored with specific treatment strategies in prospective randomized studies. The selection of existing treatments tends to be empiric due to the paucity of randomized clinical trial data. In addition, HF trials have largely focused on enrolling subjects based on prespecified ejection fraction criteria.[3] Some of the HF-specific treatments, when used without caution, may result in myocardial injury,[4] impaired renal function,[5] and increased mortality risk,[6] further complicating therapeutic decisions.

The European Society of Cardiology guidelines were the first to classify patients who have HF into distinct clinical conditions.[7] These guidelines classified patients into clinical conditions based on symptoms and hemodynamic parameters. Despite the publication of these guidelines, there are no inclusive, evidence-, or consensus-based treatment algorithms that address the individual treatment needs of each type of HF patient, particularly in the emergency department (ED) setting. Recommendations should focus on therapeutic management, emphasizing the identification and matching of HF patient types to specific treatment strategies. Management algorithms should supplement these recommendations.

Traditional methods of categorizing HF patient types use classification based on hemodynamic characteristics obtained through invasive monitoring at presentation and a clinical symptom profile that suggests HF: peripheral edema, weight gain, fatigue, dyspnea due to pulmonary congestion, and history of HF.[4,8–12] Although most EDs do not obtain hemodynamic parameters such as pulmonary capillary wedge pressure or cardiac output by way of invasive means, they do rely on an easily obtainable parameter—blood pressure.

Patients can subsequently be classified into normotensive, hypertensive, and hypotensive HF. Although the exact pathophysiology, clinical characteristics, and appropriate treatment options of each of these patient types has yet to be clarified,

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recommendations for the initial treatment, based largely on observational data and expert consensus, can be suggested. Signs, symptoms, and hemodynamic characteristics of the normotensive and hypertensive groups are described in the following paragraphs.

Normotensive Heart Failure

These patients may represent nearly half of the HF population.[2] Blood pressure is normal (systolic blood pressure range of 90–140 mm Hg), and there is usually a history of progressive worsening of chronic HF. In this group, symptoms and signs develop over days, and pulmonary and systemic congestion (seen as jugular venous distension and peripheral edema) are present. Ejection fraction is usually reduced. Management is often difficult because many patients are refractory to therapy and continue to have signs of congestion despite the initial improvement in symptoms. In some patients, the clinical or radiographic signs of pulmonary congestion are not evident despite elevated left ventricular filling pressures.[2,4,7,13] These patients have acute decompensation as a result of their cardiac failure.[14]

Hypertensive Acute Heart Failure

Data from the Acute Decompensated Heart Failure National Registry demonstrates that 50% of HF patients have a systolic blood pressure greater than 140 mm Hg on presentation.[2] These patients are more likely to have diastolic dysfunction with preserved left ventricular ejection fraction, are more often women, and are older.[1,4] Symptom onset is generally acute, with severe dyspnea and signs of end-organ hypoperfusion. Acute pulmonary edema is the hallmark of hypertensive HF and is usually auscultated on examination as rales and identified on chest radiography as pulmonary edema. The clinical target is systemic blood pressure control, with a focus on early, aggressive vasodilation, more so than on diuresis. This holds particularly true when pulmonary congestion is related to fluid maldistribution, rather than an increase in total fluid volume.[4,8] These patients have a syndrome that has been referred to as ''acute vascular failure,'' and the initial treatment in this group reflects that etiology.[14]

The novel concept of identifying and varying treatment based on systemic blood pressure addresses the diversity of the presentation of HF that is often seen in the ED. It is important to note that this classification is not entirely inclusive of all the challenges faced when evaluating patients who have HF but encompasses a large proportion of the patients seen.[12]

MECHANISM OF SYMPTOMS IN HEART FAILURE

Acute Decompensated Cardiac Heart Failure

In the euvolemic state, there is a well-defined balance between the actions of the renin-angiotensin-aldosterone (RAA) system and the natriuretic peptides that maintains fluid status. This perfect homeostasis, however, is lost in disease states such as HF, in which the mechanisms of sodium and water retention far outweigh natriuretic effects. In HF states, excess sodium and fluid retention occurs mainly within the extracellular fluid volume space. This retention results in an equal increase of fluid volume in each of the interstitial and plasma spaces. Despite this rise in total body volume and, therefore, plasma volume (PV), the arterial filling pressure remains low, which in turn continues to stimulate retention of sodium and water.[15]

Patients who have acute decompensated HF have decreased cardiac reserve, and the acute process occurs as progression of this state. Worsening cardiac contractility can be a result of ischemia, arrhythmias, inflammatory activation, or progressive deterioration in myocardial dysfunction due to the underlining mechanism causing the HF process. Subjects who have poor cardiac contractility may also develop decompensation as a result of medication noncompliance and may therefore not have further contractility impairment. The results of this event are worsening forward perfusion, increased left ventricular pressure, and alterations in the neurohormonal states that maintain fluid balance.[14] Increased left ventricular filling pressures influence changes in neurohormonal activation, activation of gene expression programs, and induction of myocte apoptosis in HF patients. Through indirect activation of the RAA, adrenergic, and cytokine systems and by way of a direct effect on myocardial stretch, fluid accumulation fosters left ventricular remodeling. Increased intraventricular pressure can cause coronary hypoperfusion, leading to subendocardial ischemia and, thus, worsening cardiac function.[16]

One study looked at PV in acute HF patients compared with normal subjects. The patients who had acute HF had visible evidence of volume overload, such as peripheral edema, jugular venous distention, and ascites; they also had PV measurements that were 34% higher compared with healthy subjects.[17]

Feigenbaum and colleagues[18] looked at PV in HF patients undergoing treatment and found a 23% PV contraction in patients treated with diuretics. They concluded that standard drug therapy may lead to a contracted PV in chronic HF