Heart Failure

Overcoming the Physiologic Dilemma Through Evidence-Based Practice

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KEYWORDS

• Heart failure • Evidence-based practice • Reduced ejection fraction
• Preserved ejection fraction • Treatment

KEY POINTS

- Heart failure occurs with a decline in myocardial performance leading to pulmonary and systemic congestion.
- The pathophysiologic derangement of systolic and diastolic heart failure and right heart failure differ dramatically, necessitating a focused, evidenced-based treatment regimen.
- Disease classification and treatment guidelines from the American Heart Association and the American College of Cardiology play a key role in the treatment paradigm.
- Nonpharmacologic interventions (eg, fluid and sodium restriction, daily weights, obesity management, hypertension management, exercise, routine vaccines, and tobacco cessation) are the foundation of treatment.
- There is a robust body of evidence to prove that pharmacologic interventions reduce morbidity and mortality of heart failure.

Cardiovascular disease remains the leading global cause of death, with 17.3 million deaths per year. This astounding statistic is expected to increase to 23.6 million by 2030. Within that label the prevalence of heart failure (HF) remains a major public health problem of more than 5.8 million in the United States and more than 23 million worldwide.[1] In 2011, 1 in 9 death certificates in the United States mentioned HF. The number of any-mention deaths attributable to HF was approximately as high in 1995 as it was in 2011, and hospital discharges for HF remained stable from 2000 to 2010.[2] Total costs for HF were estimated to be $30.7 billion in 2012. Of this total, 68% was attributable to direct medical costs. Projections show that by 2030, the total cost of HF will increase almost 127% to $69.7 billion from 2012.[2]

Disclosures: Speaker agreement with Novartis Pharmaceutical.
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Nurs Clin N Am 51 (2016) 13–27
http://dx.doi.org/10.1016/j.cnur.2015.11.004 nursing.theclinics.com
0029-6465/16/$ – see front matter

Treatment paradigms revolve around primary and secondary prevention models. Age, coronary artery disease, valvular disease, and poorly controlled hypertension are identified as major contributing factors in the development of HF. Hypertension remains the leading contributor to HF, with 75% of HF cases having antecedent hypertension.[2] Our health care system is burdened by escalating medical costs, and the need for evidence-based best practices to contain this increasing burden is great. As survival rates among patients with cardiovascular disease and, specifically, HF continue to increase, strategies to improve our utilization of evidence-based therapies and interventions to prevent and manage HF are required.

PATHOPHYSIOLOGY

Hypertension, ischemic coronary disease, idiopathic myopathies, and valvular disorders are among the initial causes leading to HF. Heart failure is characterized by a decline in myocardial performance which leads to a decrease in exercise tolerance and ultimately pulmonary and systemic congestion. Although cardiac remodeling occurs at the organ, cellular, and molecular levels and is compensatory, it becomes a progressive and lethal process. This derangement further decreases myocardial function and increases arrhythmia potential, which are the major causes of morbidity and mortality in patients with HF.[3] Patients with HF experience congestive symptoms and vacillate between states of compensation and decompensation. HF is classified as congestive HF with reduced ejection fraction (HFrEF) and congestive HF with preserved ejection fraction (HFpEF). The cause of each differs substantially.

Heart Failure with Reduced Ejection Fraction

HF with reduced ejection fraction, also known as systolic HF, results from the destructive outcomes of ischemic processes, such as coronary artery disease. Nonischemic cardiomyopathy represents another type of HFrEF with genetic, viral, chemotherapeutic, valvular, and alcoholic causes. In both ischemic and nonischemic cases, myocardial injury and maladaptive myocyte compensation lead to remodeling of the left ventricle and a cascade of neurohormonal responses (eg, sympathetic stimulation, renin-angiotensin-aldosterone, endothelin, epinephrine, growth hormone, cortisol, tumor necrosis factor, prostaglandins, substance P, adrenomedullin, and natriuretic peptides) that further impact the failing left ventricle.[4,5] Sympathetic nervous system activation provides inotropic drive for the failing heart, resulting in increased stroke volume and peripheral vasoconstriction in a compensatory attempt to maintain mean arterial perfusion pressure. The renin-angiotensin aldosterone system (RAAS) is a compensatory mechanism to maintain homeostatic control of mean arterial pressure, tissue perfusion, and extracellular volume. The role of endogenous natriuretic peptides in protecting against sodium and volume overload is well recognized, and this family of peptides is thought to have a range of other beneficial cardiac, vascular, and renal actions.[6] Current research reveals the role tissue neutral endopeptidase (NEP) plays in HF treatment by cleaving and inactivating the natriuretic peptides. NEP inhibition has been shown to increase endogenous atrial natriuretic peptide and B-type natriuretic peptide (BNP) levels in association with beneficial hemodynamic and renal effects in HF.[7] Neurohormonal activation has net effects that include vasoconstriction, volume expansion, tachycardia, and inotropic stimulation.[8] These compensatory mechanisms continue cycling as the pathophysiologic response to the failing pump. The optimal myocardial threshold is surpassed, and systolic dysfunction results in cardiac remodeling that increases preload and afterload, thus, increasing left ventricle