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An Epidemiological Study of Heart Failure Patients

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Abstract

Despite the advancement in medicine, management of heart failure (HF), which usually presents as a disease syndrome, has been a challenge to healthcare providers. This is reflected by the relatively higher rate of readmissions along with increased mortality and morbidity associated with HF. In this review article, we first provide a general overview of types of HF pathogenesis and diagnostic features of HF including the crucial role of exercise in determining the severity of heart failure, the efficacy of therapeutic strategies and the morbidity/mortality of HF. We then discuss the quality control measures to prevent the growing readmission rates for HF. We also attempt to elucidate published and ongoing clinical trials for HF in an effort to evaluate the standard and novel therapeutic approaches, including stem cell and gene therapies, to reduce the morbidity and mortality.

Keywords: Biomarker; heart failure; pathophysiology; cardiac contractility

Introduction

Approximately 6.2 million people in the United States had clinically manifested heart failure from 2013 to 2016. By some reports, the incidence rate has plateaued; however, the prevalence increases as more patients receive therapy. This has not translated to improved quality of life or a decrease in the number of hospitalizations for patients with heart failure. According to the Global Health Data Exchange registry, the current worldwide prevalence of CHF is 64.34 million cases. This translates to 9.91 million years lost due to disability (YLDs) and 346.17 billion US dollars in healthcare expenditure. Age is a major determinant of HF. Regardless of the cause or the definition used to classify patients with heart failure (HF), the prevalence of HF increases steeply with age. The registry also notes a predilection for race with a 25% higher prevalence of HF in patients of African-American descent than Caucasians. According to the American Heart Association, heart failure is still the primary cause of hospitalization in the elderly population and accounts for 8.5% of cardiovascular-related deaths in the United States. The report states a higher incidence and prevalence of heart failure among African Americans, Hispanic Americans, Native Americans, and recent immigrants from developing nations.

Pathophysiology

The adaptive mechanisms that may be adequate to maintain the overall contractile performance of the heart at relatively normal levels become maladaptive when trying to sustain adequate cardiac performance.

In the initial stages of congestive heart failure, cardiac physiology attempts to adapt via several compensatory mechanisms to maintain cardiac output and meet the systemic demands. These include the Frank-Starling mechanism, changes in myocyte regeneration, myocardial hypertrophy, and myocardial hypercontractility. With increased wall stress, the myocardium attempts to compensate via eccentric remodeling, which further worsens the loading conditions and wall stress.

A decrease in cardiac output stimulates the neuroendocrine system with a release of epinephrine, norepinephrine, endothelin-1 (ET-1), and vasopressin. They cause vasoconstriction leading to increased afterload. There is an increase in cyclic adenosine monophosphate (cAMP), which causes an increase in cytosolic calcium in the myocytes. This increases myocardial contractility and further prevents myocardial relaxation.

An increase in afterload and myocardial contractility with impaired myocardial relaxation leads to increased myocardial oxygen demand. This paradoxical need for increased cardiac output to meet myocardial demand eventually leads to myocardial cell death and apoptosis. As apoptosis continues, a decrease in cardiac output with increased demand leads to a perpetuating cycle of increased neurohumoral stimulation and maladaptive hemodynamic and myocardial responses.

HF has primarily been recognized as a disease of the elderly population (>60 years) and is reported to affect about 2%–3% of people in the United States. Of these include 10% of males and 8% of females. Unfortunately, these numbers are on a gradual increase due to the on-going prevalence of HF with increasing age. In the USA itself, about more than three million physician visits per year have been accounted for patients with HF as the primary health issue. In 2013, the total number of HF patients were 5.1 million, and direct costs were equal to $32 billion; and this cost is being projected to increase by about three-fold by 2030. As of 2011, the estimated lifetime cost of HF per individual patient was $110,000/year, with more than three-fourths of this cost consumed by 'in-hospital care'.

Diagnosis of HF

The evaluation for HF is performed using various parameters: physical examination to determine the presence of clinical symptoms and signs, blood tests, including complete blood count, urinalysis, complete metabolic profile for levels of serum electrolytes (including calcium and magnesium), blood urea nitrogen, serum creatinine, glucose, fasting lipid profile, liver function tests and thyroid-stimulating hormone.

Other HF-specific laboratory tests (especially in patients with a high possibility of heart failure) include brain natriuretic peptide (BNP) with 70% sensitivity and 99% specificity and *N*-terminal proBNP (NT-proBNP) with 99% sensitivity and 85% specificity, the measurement which has been recommended both in outpatient and in the hospital settings . BNP is a neuro-hormone, which is an activated form of proBNP, the 108-amino acid polypeptide precursor, stored as secretory granules in both ventricles and, to a lesser extent, in the atria. In response to volume expansion and pressure overload, proBNP is secreted into ventricles and breaks down into its two cleaved forms, the 76-peptide, biologically-inert *N*-terminal fragment, NT-proBNP, and the 32-peptide, biologically-active hormone BNP. NT-proBNP and BNP have clinical significance both as diagnostic and prognostic markers in the management of HF. During the diagnosis of HF, in patients presenting with acute dyspnea, BNP levels of less than 100 pg/mL have a 90% negative predictive value (NPV), and values of more than 500 pg/mL have an 81% positive predictive value (PPV) . The BNP level is a strong predictor of risk of death and cardiovascular events in patients previously diagnosed with heart failure or cardiac dysfunction.

Biomarkers not only provide valuable information about the pathophysiology of the disease, but also shed light on the severity of

ongoing disease. As far as biomarkers for HF are concerned, the National Academy of Clinical Biochemistry has set forth comparable goals in a consensus document stating that a biomarker in HF ideally enables clinicians to: (i) identify possible underlying (and potentially reversible) causes of HF; (ii) confirm the presence or absence of the HF syndrome; and (iii) estimate the severity of HF and the risk of disease progression.

Management of HF

The goal of therapy for chronic CHF is to improve symptom management and quality of life, decrease hospitalizations, and decrease overall mortality associated with this disease. The goal of pharmacologic therapy is to give all indicated agents rather than single agents because the aggregate effect of these therapies is better than monotherapy from any of the agents.

The primary combination therapy for HFrEF includes diuretics, a renin-angiotensin system inhibitor (such as an angiotensin receptor neprilysin inhibitor (ARNI), angiotensin-converting enzyme (ACE) inhibitor, or angiotensin II receptor blockers (ARB)), and a beta-blocker. The combination of hydralazine and nitrate is an alternative to an angiotensin system blocker for primary therapy if ACE inhibitor, ARNI, and ARB therapies are contraindicated. The nitrate and hydralazine combination is also indicated to reduce mortality and morbidity in African American patients with symptomatic HFrEF, currently receiving optimal medical therapy. The combination therapy of ARB-ARNI significantly reduced cardiovascular death and HF hospitalizations when compared to ACE inhibitors alone.

Prognosis

According to the Centers for Disease Control and Prevention (CDC), in December 2015, the rate for heart failure-related deaths decreased from 103.1 deaths per 100,000 population in 2000 to 89.5 in 2009 but subsequently increased to 96.9 in 2014. They note that the trend correlates with a shift from coronary heart disease as the underlying cause of heart failure deaths to metabolic diseases and other noncardiac causes of HF such as obesity, diabetes, malignancies, chronic pulmonary diseases, and renal disease.

Conclusion

Heart failure indeed is a complex disease and so far has been a major cause of morbidity and mortality in developing and developed countries. A standardized medical therapy has been successful in the early stages of HF. Advanced stages of HF require frequent hospitalization due to the presence of severe HF and or associated co-morbid conditions, which require strict implementation of an appropriately individualized multidisciplinary approach and quality measures to reduce re-admissions. While pharmacological management has a limited role in advanced cases of HF, novel therapeutic agents, such as regenerative and gene therapy, are in the developmental stages and need further refinement before their approval for the treatment of HF.

References

1. Watson R.D., Gibbs C.R., Lip G.Y. ABC of heart failure. Clinical features and complications. BMJ Br. Med. J. 2000;320:236–239.
2. Wong CM, Hawkins NM, Jhund PS, MacDonald MR, Solomon SD, Granger CB, Yusuf S, Pfeffer MA, Swedberg K, Petrie MC, McMurray JJ. Clinical characteristics and outcomes of young and very young adults with heart failure: The CHARM programme (Candesartan in Heart Failure Assessment of Reduction in Mortality and Morbidity). J Am Coll Cardiol. 2013 Nov 12;62(20):1845-1854.
3. Hacker M, Hoyer X, Kupzyk S, La Fougere C, Kois J, Stempfle HU, Tiling R, Hahn K, Störk S. Clinical validation of the gated blood pool SPECT QBS processing software in congestive heart failure patients: correlation with MUGA, first-pass RNV and 2D-echocardiography. Int J Cardiovasc Imaging. 2006 Jun-Aug;22(3-4):407-16.
4. Paterson I., Mielniczuk L.M., O'Meara E., So A., White J.A. Imaging heart failure: Current and future applications. Can. J. Cardiol. 2013;29:317–328.
5. Scalvini S., Giordano A. Heart failure. Optimal postdischarge management of chronic HF. Nat. Rev. Cardiol. 2013;10:9–10.