JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

A REVIEW ON LEFT VENTRICULAR FAILURE AND AN OVERVEIW OF LEFT VENTRICULAR DYSFUNCTION

Dr. Manchineni Prasada Rao [1*], Dr. V Rajini [2], Dr. Y. Narasimha Rao [3], Shaik Karishma [4]

[1] Head of the institute, M. Pharm, Ph.D.

[2] Head of the Department, Pharmacy Practice

[3] Head of the Department, Pharmacology

[4] Pharm D Student, Department of Pharmacy Practice

M.A.M College of Pharmacy, Kesanupalli, Narasaraopet (522601), Palnadu district, Andhra Pradesh

Corresponding Author : Dr. Manchineni Prasada Rao

M.A.M College of pharmacy, Kesanupalli, Narsaraopet-522601, Palnadu District, Andhra Pradesh.

ABSTRACT

Left heart failure occurs when there is dysfunction of the left ventricle, resulting insufficient delivery of blood to vital organs. Left ventricular failure can be further subdivided into heart failure with preserved ejection fraction (HFpEF) with ejection fraction over 50 percent, heart failure with reduced ejection fraction (HFrEF) with ejection fraction less than 40 percent, or heart failure with mid-range ejection fraction, with ejection fraction between 41 and 49 percent. This activity describes the evaluation and management of left heart failure and highlights the role of the interprofessional team in improving care for affected patients.

Key Words: *HEpEF, HFrEF, ejection fraction, left heart failure.*

INTRODUCTION

The heart is comprised of the pericardium, myocardium, and endocardium. Pathology in any of those structures can lead to heart failure. Left ventricular failure occurs when there is dysfunction of the left ventricle causing insufficient delivery of blood to vital body organs. Left ventricular failure can further subdivide into heart failure with preserved ejection fraction (HFpEF with EF over 50%), heart failure with reduced ejection fraction (HFrEF with EF less than 40%), or heart failure with mid-range ejection fraction (EF between 41 and 49 percent.

EPIDEMIOLOGY

Heart failure is more prevalent and has a higher incidence in the elderly population. Approximately 5.7 million people in the United States have diagnosed heart failure. The incidence is around 10 per 1000 in people over

Approximately 50% of all patients with heart failure are considered to have HFrEF, this diagnosis is becoming more prevalent with time. HFpEF, as opposed to HFrEF, is more common in women (79% versus 49%) and also tends to affect an older population.

ETIOLOGY

The most common etiologies of left heart failure are coronary artery disease and hypertension. The latter can cause left heart failure through left ventricular hypertrophy (leading to HFpEF) and also serves as a risk factor for coronary artery disease (which can lead to HFrEF). Diabetes, smoking, obesity, male gender, and a sedentary lifestyle are also considered risk factors.Many of these causes are preventable, and as such risk factor control remains of extreme importance in preventing heart failure.

PATHOPHYSIOLOGY

Multiple mechanisms can lead to left heart failure. Chronic or poorly controlled hypertension causes increased afterload and therefore increased cardiac workload, which can lead to hypertrophy of the left ventricle. Initially, this hypertrophy serves as a compensatory mechanism and can help maintain cardiac output, but long-term can inhibit relaxation of the myocardium leading to impaired cardiac filling and decrease left ventricular output. Coronary arterial disease causes direct ischemic damage to the myocardium, leading to remodeling and scar formation, which decreases contractility and cardiac output. Arrhythmias can cause remodeling, but in general, decrease cardiac output by impaired ventricular filling and decreased ventricular relaxation. Cardiomyopathies encompass a diverse pathologic spectrum and have variable mechanisms causing cardiac dysfunction.

DIAGNOSIS

The diagnosis of heart failure is clinical. However, several tests are available for further evaluation. Laboratory tests: brain natriuretic peptide (BNP) or NT-pro BNP may be the most helpful as it can differentiate acute heart failure from other causes of shortness of breath. However, this test lacks specificity, and a high level of this hormone is not diagnostic of acute heart failure. Other laboratory tests include troponin T (to detect myocardial infarction, although the levels may be high due to heart failure itself), complete blood count, basic metabolic panel (low sodium, in particular, indicates advanced disease) and liver function tests (to detect liver injury due to volume overload).

Electrocardiography can show nonspecific findings, like ischemic changes, left ventricular hypertrophy, or arrhythmias. Echocardiography can help distinguish HFrEF from HFpEF by determining the ejection fraction, and diastolic left ventricular function can evaluate associated regional wall motion abnormalities that may be suggestive of an ischemic component, as well as valvular and pericardial pathologies.Coronary angiography is indicated in patients with anginal symptoms and may also be indicated in patients with worsening heart failure symptoms.

TREATMENT AND MANAGEMENT

Patients should receive education on the importance of lifestyle modification for improving the outcome of their disease. This includes reasonable salt consumption and avoidance of alcohol, nicotine, and recreational drugs.

Treating the underlying cause is of extreme importance as some heart failure conditions may be reversible when the precipitating factors are addressed, like cardiomyopathies induced by alcohol, tachycardia or ischemia. Tight control of blood pressure will also help prevent further deterioration. Besides loop diuretics for volume overload, the pharmacologic treatment differs between HFrEF and HFpEF, for HFrEF, the mainstay of treatment is the combination of an angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) with a beta blocker (carvedilol, metoprolol or bisoprolol). If the patient remains symptomatic on a maximal dose of ACE inhibitor or ARB, an angiotensin receptor-neprilysin inhibitor may be substituted. Other medications include hydralazine, nitrates, mineralocorticoid receptor antagonists such as spironolactone, ivabradine, and digoxin (as a last resort). Nitrates in combination with hydralazine may be especially efficacious in African American patients. Digoxin, ivabradine and the diuretics have not been shown to offer any mortality benefit.

For HFpEF, the treatment focuses on the underlying cause or contributing factors: control of blood pressure, revascularization if ischemic cardiomyopathy, and management of arrhythmias. A mineralocorticoid receptor antagonist may be beneficial in these patients.

Severely symptomatic patients with an ejection fraction less than 35% should obtain a referral for implantable cardioverter-defibrillator (ICD) or cardiac resynchronization therapy (CRT) (depending on QRS width and the type of intraventricular conduction delay) after medical optimization. In advanced cases, mechanical circulatory assist devices such as an LVAD, continuous infusion of inotropic medications such as dobutamine or milrinone (which is still possible in the ambulatory setting), and cardiac transplantation are options.

DIFFERENTIAL DIAGNOSIS

When approaching a patient with shortness of breath on exertion, a broad differential diagnosis exists. A patient with established left heart failure might have a concomitant disease that might contribute to the patient's presentation, and additional workup for other conditions is warranted in case of atypical presentations. Those conditions include other cardiovascular causes (like primary pulmonary hypertension), pulmonary causes (like chronic obstructive pulmonary disease and interstitial lung disease) and extra-cardiopulmonary causes (like anemia). Isolated lower limb edema is less likely heart failure, and other causes must be ruled out first, like venous insufficiency, cirrhosis, nephrotic syndrome, lymphedema and thrombosis of the veins.

CONCLUSION

Compliance with lifestyle modification and pharmacologic treatment and control of precipitating factors (like hypertension and arrhythmias) are crucial to prevent hospitalizations for heart failure exacerbation and improve the quality of life. Many of those precipitating factors are potentially under patient control. That is why patient

education is an integral part of a multidisciplinary approach to decrease mortality and morbidity due to heart failure.

Conflict of Interest: Nill

Acknowledgements: The authors thank to curious personalities who answered the call for proposals and provide information on the innovative initiatives.

REFERENCE

1.He J, Ogden LG, Bazzano LA, Vupputuri S, Loria C, Whelton PK. Risk factors for congestive heart failure in US men and women: NHANES I epidemiologic follow-up study. Arch Intern Med. 2001 Apr 09;161(7):996-1002.

2.Ho KK, Pinsky JL, Kannel WB, Levy D. The epidemiology of heart failure: the Framingham Study. J Am Coll Cardiol. 1993 Oct;22(4A):6A-13A.

3.Dunlay SM, Weston SA, Jacobsen SJ, Roger VL. Risk factors for heart failure: a population-based case-control study. Am J Med. 2009 Nov;122(11):1023-8.

4.Vasan RS, Levy D. The role of hypertension in the pathogenesis of heart failure. A clinical mechanistic overview. Arch Intern Med. 1996 Sep 09;156(16):1789-96.

5.Writing Group Members. Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, Das SR, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Isasi CR, Jiménez MC, Judd SE, Kissela BM, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Magid DJ, McGuire DK, Mohler ER, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Rosamond W, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Woo D, Yeh RW, Turner MB., American Heart Association Statistics Committee. Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2016 Update: A Report From the American Heart Association. Circulation. 2016 Jan 26;133(4): e38-360.

6.Lloyd-Jones DM, Larson MG, Leip EP, Beiser A, D'Agostino RB, Kannel WB, Murabito JM, Vasan RS, Benjamin EJ, Levy D., Framingham Heart Study. Lifetime risk for developing congestive heart failure: the Framingham Heart Study. Circulation. 2002 Dec 10;106(24):3068-72.

7.Owan TE, Hodge DO, Herges RM, Jacobsen SJ, Roger VL, Redfield MM. Trends in prevalence and outcome of heart failure with preserved ejection fraction. N Engl J Med. 2006 Jul 20;355(3):251-9.