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A Review on Heart Failure

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[1]Prasanna Mahendra Sapkal, [2]Prasanna Deepak Madane

1.Govt. College of Pharmacy, Karad.
2.Yashvantrao Chavan Institute of Science, Satara., India.

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ABSTRACT

Cardiovascular disease remains one of the most important causes of morbidity and mortality in western society. Heart failure is common clinical disorder that results in pulmonary vascular congestion and reduced cardiac output. The diagnosis of heart failure is often determined by a careful history and physical examination and characteristic chest radiograph finding. The measurement of serum brain natriuretic peptide and echocardiography have subsequently improved accuracy of diagnosis. The pathogenesis of heart failure is complex and there is no single lesion any form of heart disease can lead to heart failure. A fundamental response to myocardial injury or altered loading condition include “remodelling” of heart so that the size, shape and function of the affected chamber are grossly distorted. Early diagnosis and appropriate therapy help in reversing the presence of remodelling and clinical improvement in most of the patient.

INTRODUCTION

Heart failure (HF) is a common cardiovascular condition with increasing incidence and prevalence. Several large clinical trials on use of pharmacological therapy and device have resulted in an increasing use of evidence-based therapy(1). The important risk factor for heart failure includes coronary artery disease, hypertension, diabetes mellitus, cardiotoxic drug, valvular heart disease and obesity(1). Congestive heart failure is a complex clinical syndrome that can result from any functional or structural cardiac disorder that impairs the ventricle's ability to fill or eject blood(2).

Heart failure is common disease, approximately 5 million persons in United State and more than 500,000 new cases reported each year. Heart failure is predominantly a disease of the elderly with prevalence rates ranging from 1% in person younger than 50 years to 10% in persons aged 80 years and older. Approximately 80% of patients hospitalized with HF are older than 65 years(3). There is no definitive diagnostic test for heart failure, it remains a clinical diagnosis that is largely based on careful history and physical examination and supported by ancillary tests such as chest radiograph, electrocardiograph and echocardiography(2). The standard treatment for heart failure will be diuretic and ACE inhibitors, sometimes supplemented by digitalis and other vasodilators. As short-term measure this treatment may be supplemented by drug with positive isotropic capacity(4). For better understanding of its pathophysiology, it has become clear that become pathological changes involve not only the cardiovascular system but also the renal, neuroendocrinological, immunological, musculoskeletal, hematologic and gastrointestinal system as well as nutritional status(5).

The syndrome of heart failure:

Heart failure is constellation of sign and symptom caused by inadequate performance of heart focuses on only one aspect of the pathophysiology involved in syndrome. Currently, a complex blend of structural, functional and biological alteration are evoked to account for the progressive nature of heart failure and to explain the efficacy or failure of therapies used in clinical trials(6). The syndrome may occur as the end-result of damage caused number of disease progress. The presence and severity of heart failure can be assessed by questionnaires, physical and radiographic examination and by measure of ventricular performance and exercise capacity(7).

As heart failure syndrome advance, patient become less physically active and there can be substantial salt and water retention leading to edema. Many patients eventually die from progressive pump dysfunction, which include hypotension, low cardiac output and multi-organ dysfunction(8).

Classification of heart failure:

Heart failure may be either predominantly systolic and diastolic. Patient with systolic heart failure also have some amount of diastolic dysfunction(1).

Diastolic heart failure:

Patient with diastolic heart failure are typically elderly, often female, obese and frequently have hypertension and diabetes(1). It is estimated that 20 to 50 percent of patients with heart failure have preserved systolic function or a normal left ventricular ejection fraction. Although such hearts contract normally, relaxation is abnormal. Cardiac output, especially during exercise, is limited by the abnormal filling characteristics of the ventricles (1,6). The abnormal high filling pressure leads to pulmonary congestion, dyspnoea and oedema(1,9).

Diagnosis of heart failure:

The diagnosis of heart failure is mainly clinical but various investigations help us to understand underlying cause and assessment of severity of heart failure.

The classical clinical symptoms of heart failure are exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnoea, fatigue and the sign are elevated jugular venous pressure, pulmonary rales, third heart sound and peripheral oedema(1). Most specific symptoms are orthopnea and paroxysmal nocturnal dyspnoea(1).

Diagnostic modalities in heart failure:

- Careful history and physical examination
- Laboratory investigation including complete blood count, test for renal and hepatic function, urinalysis, electrocardiogram and chest X-ray
- Two dimensional and Doppler electrocardiogram
- Careful exclusion of coronary artery disease and thyroid disease in all patient , and

- Selective use of other diagnostic tests including serologic studies in selected patient based upon clinical characteristics, risk factor, past medical, family history(1,10).

Table no:1

Modified Framingham criteria for diagnosis of chronic heart failure
Major criteria
Neck-vein distention
Orthopnea or proximal nocturnal dyspnea
Crackles (> 10 cm above base of lung)
Cardiomegaly on chest radiograph
S_3 gallop
Central venous pressure >12 mm Hg
Left ventricular dysfunction on echocardiogram
Weight loss >4.5 kg in response to CHF treatment
Acute pulmonary edema
Minor Criteria
Bilateral ankle edema
Night cough
Dyspnea on exertion
Hepatomegaly
Pleural effusion
Tachycardia(> 120 beats/min)
(*ref. No.*:2)

Remodelling

Great emphasis has been put on fact that a remodelling process, characterised by myocardial restructuring causing progressive dilation and a deterioration of left ventricular mechanical efficacy, is frequently observed in various clinical situation causing left ventricular strain. Remodelling is association with poor prognosis, causing a successive deterioration of congestive heart failure and thereby adding to the impact of the disease that triggered the remodelling process(4). Left ventricular remodelling is process by which mechanical, neurohormonal and possibly genetic factors alter ventricular size, shape and function. Remodelling occurs in several clinical condition, including myocardial infraction, cardiomyopathy, hypertension and valvular heart disease; its hallmark includes hypertrophy, loss of myocytes and increased interstitial fibrosis(6,11). Reverse remodelling, in which the therapy promoted a return to more normal ventricular size and shape(6,12).

Stages of Heart Failure:

There are four stages of heart failure

Patient with stage A:

HF (heart failure) are at high risk of developing heart failure because of the presence of condition strongly association with development of heart failure. These patients have hypertension, CAD, diabetes mellitus, history of cardiotoxic drug therapy, alcohol abuse, a history of rheumatic fever or a family history of cardiomyopathy. These patients have no evidence of structural heart disease(3). ACE- treatment of asymptomatic high risk patients with diabetes or vascular disease and no history of heart failure has yielded significant reductions in rates of death, myocardial infarction and stoke(6,13). The goal of treatment in stage A is to prevent remodelling(6).

Patient with Stage B:

They have structural heart diseases associated with development of heart failure but have never shown symptom or sign of heart failure. These patients have a prior myocardial infraction (MI), left ventricle hypertrophy or fibrosis, left ventricle dilation or hypercontracility or asymptomatic valvular heart disease(3). The goals of therapy for patients with heart failure and low ejection fraction are to improve survival, slow the progressive disease, alleviate symptoms, and minimize

risk factor. Modification of lifestyle can be helpful in controlling the symptom of heart failure(6). A measure treatment ACE inhibitors and beta blocker and valve replacement or repair for patient with hemodynamically significant valvular stenosis or regulation(3).

Patients with stage C have current of prior symptoms of heart failure associate with structural heart disease(3). Patients with stage D have advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy and who require specialization intervention(3). In patients with advanced heart failure, circulating levels of aldosterone becomes elevated in response to stimulation by angiotensin I and there is decreasing in the hepatic clearance of aldosterone due to hepatic congestion. Aldosterone stimulate the retention of salt, myocardial hypertrophy and potassium excretion; spironolactone counteracts these response. The beneficial effect of spironolactone is heart failure may also include a decrease in collagen synthesis that promotes organ fibrosis(6).

Treatment of heart failure

Diuretic

These are the main stay of therapy to relieve congestive symptom(1). Diuretic are first line drug in the treatment of older patient with heart failure and volume overload. Diuretic decrease venous return, reduce ventricular filling pressure, cause loss of fluid from body and decrease symptom of pulmonary and systemic congestion and edema(3). Age-related decrease in renal function and in circulating plasma volume may decrease the efficacy of diuretic in elderly patient with heart failure (3). Three classes of drug are available: loop diuretic, thiazide group and potassium sparing diuretics(1).

- *Loop diuretic*: Agent belonging to this group include furosemide, toresemide and bumetinide. These are more potent diuretic that act on ascending loop of Henle.
  The important adverse effects of these agents on long-term medication are hypokalaemia, hypomagnesemia, hypokelemic, alkalosis and orthostatic hypotension.(1).
- *Thiazide diuretic*: These act by inhibiting the reabsorption of sodium and chloride from distal convoluted tubules. Diuretic with these agents is modest and these are ineffective at glomerular filtration rate below 40 ml/min. The untoward effects are hypokalaemia, hypomagnesemia, hyperglycaemia, hyperuricaemia(1).

Metalozane belonging to the quinalozone sulphonamide group. It acts by inhibition of sodium reabsorption in clinical in cortical collecting duct and proximal convoluted tubules(1).

B- blockers

The beneficial role of β-blockers in the treatment of heart failure is well established. Agents commonly used in clinical practice are sustained release metoprolol, carvedilol, bisoprolol and nebivolol(1). Chronic administration of beta blockers after MI (Myocardial Infraction) reduces mortality, sudden cardiac death and recurrent MI especially in elderly persons. These benefits are more marked in patients with a history of HF(3). Non selective β-blockers, propanolol was found useful in our experience in patients with dilated cardiomyopathy (DCM) with class 2-4 symptoms(1). Beta blockers are effective in antagonizing neurohormonal system that cause myocyte apoptosis, myocyte necrosis, myocyte hypertrophy, fetal gene program activation, extracellular matrix alteration and beta receptor uncoupling. Beta blockers may prevent or reverse increased systemic vascular resistance and increased afterload coursed by excessive sympathetic nervous system activation. Beta blockers also decrease levels of atrial natriuretic peptide, brain natriuretic peptide and tumor necrosis alpha level(3).

Angiotensin Converting Enzyme Inhibitors (ACEI):

The ACEI acts on the both arterial and venous capacitance vessels. They act by inhibiting the production of angiotensin II as a consequence preventing the deleterious effects of angiotensin II through its action predominantly on the type 1 receptors. The levels of bradykinin are raised which result in the production of nitric oxide and other important endogenous vasodilators(1). ACE inhibitors may also improve heart failure associated with normal left ventricular ejection fraction by decreasing afterload, lowering elevated blood pressure, decreasing left ventricular mass and arterial and arteriolar wall thickness and stiffness by improving left ventricular relaxation and by attenuation the coronary vasoconstriction of angiotensin II. Increased activation of the rennin-angiotensin- aldosterone system may stimulate the progressive myocardial fibrosis(3).

Digoxin:

Digoxin acts by the sodium potassium adenosine triphosphatase enzyme and increase the intercellular Na. The drug is begin used for more than a century in the treatment of heart failure(1). Digoxin reduces the rapid ventricular rate associated with supraventricular tachyarrhythmias and may be used to treat older patient with heart failure and supraventricular tachyarrhythmias such as atrial fibrillation. However, digoxin should not be used to treat patient with heart failure in sinus rhythm with diastolic heart failure(3). Digoxin has a narrow therapeutic index, especially in elderly patient. Age-related reduction renal function increases serum digoxin level in serum in older person. The decrease in skeletal muscle mass in elderly patient reduces the volume of distribution of digoxin, increasing serum digoxin levels(3). In addition, hypokalemia, hypomagnesemia, myocardial ischemia, hypoxia, acute and chronic lung disease, acidosis, hypercalcemia, and hypothyroidism may cause digitalis toxicity despite normal serum digoxin levels(3).

Table no: 2

Treatment of older patient with Diastolic heart failure
• Treat with caution use of diuretic and with beta blockers. • Add angiotensin-converting enzyme(ACE) inhibitors if heart failure persists or angiotensin 2 type 1 receptor antagonist if patient cannot tolerate ACE inhibitors because of cough, angioneurotic edema, rash or altered taste sensation. • Add isosorbide dinitrate plus hydrazine if heart failure persists. • Add calcium channel blocker if heart failure persists. • Avoid digoxin if sinus rhythm is present(3).

Prevention of heart failure

Primary prevention

As mentioned earlier, the most important risk factor are hypertension, coronary artery disease, diabetes mellitus and rheumatic heart disease. Physical activity, dietary control and lifestyle

modification can drastically bring down some of these modifiable risk factors and reduce the load of heart failure (1).

Secondary prevention

Screening through population based approach and high risk approach can yield satisfactory results and greater reduction in congestive heart failure. Screening of hypertension, screening of diabetes, cholesterol screening these screening test can be incorporated into the existing cardiovascular and chronic disease control programmes(3). Health education and health promotion strategies are quite important in success of these programmes.

Non pharmacological treatment of heart failure may include *exercise training, diet and nutrition education and counselling*(3). Currently, physicians are merely delaying progressions of heart failure for few months without being given chance to actually prevent its root cause(8).

Device therapy in heart failure:

Cardiac resynchronization therapy:

Cardiac resynchronization therapy (CRT) is an established therapy for patients with left ventricular systolic dysfunction and moderate to serve heart failure symptoms despite optimal medical therapy and evidence of wide QRS >120 ms (1,14).

Implantable Cardiac Defibrillator (ICD):

MADIT-II and SCD-HeFT trials confirmed the effectiveness if ICD for primary prevention in heart failure(1,15). In patients with heart failure and left ventricular *dyssynchrony*, the choice of CRT versus CRT-D is individualized as the randomized clinical trial comparing the above therapies did not demonstrate any satisfactory significant difference between the two treatment arm(1,16).

Ventricular assist device:

Mechanical circulatory assist devices are used in both acute and chronic heart failure. They are used as bridge to recovery, bridge to transplant or as permanent destination therapy(1,17).

Coronary Disease Severity:

Disease severity is usually discussed as a potential confounder of the relation between depression and cardiac outcomes rather as a candidate mechanism. If the sickest patient in a particular population tends to be the who develop depression, the relative severity of their heart disease might explain both their depression and their high risk of adverse cardiac outcome.

There is little evidence to support either possibility, at least in terms of standard physiological indicator of the severity of heart disease, such as the extend coronary atherosclerosis, the size of the infract in post-myocardial infarction patient or the degree to which their left ventricular ejection fraction has been reduced(18).

CONCLUSION

Heart failure is common clinical syndrome resulting from multiple underlying causes. Chronic heart failure is complex cardiac condition that encompasses several etiologies and comorbidities. Early identification of risk factor and initiation of appropriate theory at early stages prevents development of heart failure. Pharmacological therapy of heart failure is still a matter of great concern. Thus here we discuss over review to give you information about heart failure, pathology, and treatment.

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