Heart Failure : Current Concepts

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The clinical syndrome of heart failure is the final pathway for myriad diseases that affect the heart. Incidence of heart failure today is approaching 10 per 1000 population among persons older than 65 years of age. Heart failure is the reason for at least 20% of all hospital admissions among persons older than 65 years. Symptomatic heart failure has a one-year mortality of approximately 45%. Although there has been a substantial reduction in mortality of patients with systolic heart failure, overall death rates in large epidemiologic surveys have remained the same [1].

Why have the newer and successful therapies failed to result in a meaningful reduction in mortality due to heart failure? It is important to recognize that heart failure is a clinical syndrome arising from diverse causes. Not all patients with the condition have poorly contracting ventricles and a low ejection fraction. Many have uncorrected valvular disease, such as aortic stenosis or mitral regurgitation, or abnormal filling, resulting in diastolic heart failure. A large majority of patients with heart failure are elderly, and 75% of patients have a history of hypertension. Many patients have at least one serious coexisting condition, in addition to advanced age.

Heart failure is largely preventable, primarily through the control of blood pressure and other vascular risk factors. The new approach to the classification of heart failure [2] emphasizes its evolution and progression and defines four stages :

Stage A : Patients with risk factors for the development of heart failure.

Stage B : Patients with structural abnormality of the heart.

Stage C : Patients with structural abnormality of the heart and current or previous symptoms of heart failure.

Stage D : Symptoms of endstage heart failure that are refractory to standard treatment.

Pathophysiology

Systolic heart failure

The hemodynamic model of heart failure emphasizes the effect of an altered load on the failing ventricle and ushers in the era of vasodilators and inotropic agents. The neurohumoral model recognizes the importance of activation of the renin-angiotensin aldosterone axis and the sympathetic nervous system in the progression of cardiac dysfunction.

Left ventricular remodelling is the process by which mechanical, neurohormonal and possibly genetic factors induce dilatation and make it more spherical. The eventual change in the shape of the ventricle becomes deleterious to the overall function of the heart as a pump [3]. As the left ventricle assumes a more globular shape, the distortion of the mitral apparatus and dilatation of the annulus leads to mitral regurgitation which results in an increasing volume overload on the overburdened left ventricle that further contributes to remodelling, the progression of disease and symptoms.

Altered conduction properties of myocardial conduction system lead to supraventricular and ventricular arrhythmias that herald the onset of either systolic or diastolic heart failure. The rate of sudden cardiac death among persons with heart failure is six to nine times that seen in the normal population.

Abnormal myocardial conduction can also lead to delays in ventricular conduction and bundle branch block. Left bundle-branch block (LBBB) is a significant predictor of sudden death and also causes abnormal ventricular activation and contraction, ventricular dyssynchrony leading to a reduced ejection fraction, decreased cardiac output and abnormal diastolic function.

Diastolic heart failure

Approximately 20 to 50% of patients with heart failure have normal left ventricular systolic function with

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abnormal diastolic relaxation. They are typically elderly, female, obese and hypertensive. Cardiac output is limited by the abnormal filling characteristics of the ventricles [4]. Mortality among these patients may be as high as that among patients with systolic heart failure.

Clinical Assessment

Breathlessness, fatigue, weight gain, peripheral edema, raised jugular venous pressure, hepatjugular reflux and presence of a gallop rhythm, are key findings on physical examination that may indicate a need for additional diuretic therapy and may be prognostically important.

Treatment

Stage A : Effective control of risk factors (eg. hypertension, coronary artery disease and diabetes mellitus) has a favourable effect on the incidence of later cardiovascular events, reducing the incidence of heart failure by 30 to 50%. Angiotensin Converting Enzyme (ACE) inhibitor treatment of asymptomatic high-risk patients with diabetes or vascular disease and no history of heart failure has yielded significant reduction in the rate of death, myocardial infarction and stroke [5].

Stage B, C and D : The goals are to minimize risk factors, slow the progression of disease, alleviate symptoms and improve survival. Moderate sodium restriction, weight monitoring and adherence to medication schedules, aid in avoiding fluid retention. Nonsteroidal-antiinflammatory drugs (NSAIDs) should be avoided as they have been associated with worsening of heart failure. For selected patients, a regularly scheduled exercise programme may have beneficial effects on symptoms.

ACE-inhibitors improve survival, the rate of hospitalization, symptoms, cardiac performance, neurohormonal levels and reverse remodelling. Side effects are fairly predictable and reversible and can usually be successfully managed. Angiotensin receptor antagonists are an alternative in patients who cannot tolerate ACE-inhibitors because of severe cough or angioedema, but should not be used as first line therapy for heart failure of any stage.

The primary action of beta-blockers is to counteract the harmful effects of the sympathetic nervous system that are activated during heart failure. This leads to improvement in survival, morbidity, ejection fraction, remodelling, quality of life, the rate of hospitalization and the incidence of sudden death. Beta-blockers should be used in all patients in stable condition without substantial fluid retention and without recent exacerbations of heart failure requiring inotropic therapy.

In patients with reactive airway disease, diabetes and bradyarrhythmias or heart block, they should be used with extreme caution.

Two beta blockers are specifically approved for the treatment of heart failure : carvedilol and long-acting metoprolol. Carvedilol is a nonselective-adrenergic antagonist with α blocking effects also; metoprolol is a selective β_1 adrenergic antagonist with no α blocking effects. A large trial comparing these drugs has just been published, which has shown significant superiority of carvedilol over metoprolol.

There is evidence to support the use of spironolactone, an aldosterone antagonist, in patients with advanced symptoms of heart failure - specifically, NYHA class III or IV symptoms. In patients with advanced heart failure, circulating levels of aldosterone become elevated in response to stimulation by angiotensin II which leads to the retention of salt, myocardial hypertrophy and potassium excretion; spironolactone counteracts these responses. Diuretics are a mainstay for controlling symptoms of congestion. Thiazide or loop diuretics and combination therapy may be used to promote effective diuresis in advanced cases.

Randomized, placebo-controlled study of digoxin for symptomatic patients with a low ejection fraction has shown no difference in mortality between patients receiving digoxin and patients receiving placebo, but there was decrease in the digoxin group in the rate of worsening heart failure and hospitalization. Vasodilator therapy with hydralazine and nitrates has a role in patients intolerant to standard therapy.

Cardiac resynchronization therapy

This is an innovative, pacemaker-based approach to the treatment of patients with heart failure who have a wide QRS complex on 12-lead electrocardiography. The purpose of resynchronization is to provide electromechanical coordination and improved ventricular synchrony in symptomatic patients who have severe systolic dysfunction and clinically significant intraventricular conduction defects, particularly LBBB. A percutaneous, three-lead, biventricular pacemaker system is used; one lead is placed in the right atrium, one is placed in the right ventricle, and a third is passed through the right atrium, through the coronary sinus and into a cardiac vein on the lateral wall of the left ventricle. Beneficial effects include reverse remodelling, resulting in decreased heart size and ventricular volumes, improved ejection fraction and decreased mitral regurgitation. Clinical improvement in exercise tolerance, quality of life and the rate of hospitalization have been documented. To date, however, resynchronization therapy has not been shown to enhance survival.

Increasing use of implantable cardioverter-defibrillators has unequivocally reduced mortality in a subgroup of patients with heart failure. However, its role in all patients of heart failure is not established.

Revascularization and surgical therapy

Patients with heart failure of any stage who are at risk of coronary artery disease should be screened for myocardial ischemia. Revascularization, through either catheter-based or surgical approach, often improves ischemic symptoms, improves cardiac performance and reduces the risk of sudden death.

Similarly, the role of mechanical devices that serve to support patients who are awaiting heart transplantation or are on definitive therapy for endstage heart failure continues to evolve, and such devices offer great hope to many patients who are not eligible for cardiac transplantation.

Conclusion

Heart failure has emerged as an important health problem all over the world, especially in India, with better life expectancy, increased incidence of coronary artery disease and improved survival after acute myocardial infarction. Many common clinical problems encountered in patients with heart failure remain unresolved. There may be important adverse interactions between drugs used for polytherapy (e.g. aspirin and ACE inhibitors)

that need to be resolved. The optimal care for patients with heart failure and preserved systolic function (diastolic heart failure) awaits further research. Newer modes of therapy such as gene therapy, angiogenesis, myoblast and stem-cell transplantation are promising but await validation [6].

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