Heart Failure In Elderly

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INTRODUCTION

Syndrome of heart failure (HF) is one of the most common causes of disability and hospital discharge diagnosis in the elderly population. More than 75% of patients with HF are older than 65 years. The increased numbers of older people with heart HF is due to improved treatment of ischemic, valvular and hypertensive heart disease. Many patients with acute myocardial disease in their early age are surviving more and more, but with residual left ventricular dysfunction. Improved control of blood pressure has led to a 60% decline in stroke mortality rates, but become vulnerable for HF. Surprisingly, treatment of the condition is mostly extrapolative and involves many of the same methods as in younger patients. The reason is most of the trials for HF exclude elderly population. Recently trials like SENIOR with d nevibolol and MUST with ICDs have been carried out in the elderly group. The new guidelines emphasise the importance of combination therapy with ACEI and BB as a cornerstone with or without ARB or Aldosterone as an adjuvant therapy. Patients in NYHA class III-IV symptomatic with persistently low EF, life expectancy above 12 months and QRS duration above 120 milliseconds will have mortality and morbidity benefit with CRT.

DEFINITION AND CLASSIFICATION

Heart failure (HF) is conglomerate of diverse and complex spectrum of pathologies bound by a limited number of clinical characteristics with ever expanding evolution of the nomenclatures. American College of Cardiology/ American Heart Association (ACC/AHA) task force on practice guidelines defined HF as ‘a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood’1. European Society of Cardiology defined HF as a clinical syndrome with i) symptoms typical of heart failure (breathlessness at rest or on exercise, fatigue, tiredness, ankle oedema), ii) signs typical of HF (tachycardia, tachypnoea, pulmonary rales, pleural effusion, raised jugular venous pressure, peripheral oedema, hepatomegaly) and iii) objective evidence of a structural or functional abnormality of the heart at rest (cardiomegaly, third heart sound, cardiac murmurs, abnormality on the echocardiogram, raised natriuretic peptide level). Other overlapping terms are congestive heart failure to emphasise congestive signs and symptoms, acute heart failure syndrome to highlight rapid onset of symptoms and signs secondary to abnormal cardiac function, acute decompensated heart failure to denote patients with known HF experiencing acute or sub-acute worsening of HF state. Table I depicts the classification of HF by symptoms related to structural capacity (NYHA) and or by structural abnormality (ACC/AHA)2.

Pathophysiology conundrum

Unlike in young, older ones experience heart failure more after stresses. Four principal changes are associated with cardiovascular aging that impair the ability to respond to stress3:
1. Reduced responsiveness to ß-adrenergic stimulation.
2. Increased vascular stiffness in the elderly.
3. The heart getting stiff and less compliant with age produces raised diastolic pressure causing left atrial stretching and predisposing to atrial arrhythmias like atrial fibrillation.
Table I

<table>
<thead>
<tr>
<th>NYHA functional classification (severity based on symptoms-fatigue, palpitation or dyspnoea and Physical activity)</th>
<th>ACC/AHA Stages of HF (Stage of HF based on structure and damage of heart muscle)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I No limitation of physical activity. No symptom on ordinary activity</td>
<td>Stage I At high risk of developing HF. No structural or functional abnormality. No signs or symptom</td>
</tr>
<tr>
<td>Class II Slight limitation of activity. Comfortable at rest, but ordinary activity results in symptoms</td>
<td>Stage II Developed structural heart disease strongly associated with development of HF. No signs or symptom</td>
</tr>
<tr>
<td>Class III Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in symptoms</td>
<td>Stage III Symptomatic HF associated with structural heart disease</td>
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<tr>
<td>Class IV unable to carry out and physical activity without discomfort. Symptom at rest. Any physical activity results discomforts</td>
<td>Stage IV Advanced structural heart disease and marked symptom of HF at rest despite maximal medical therapy</td>
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</table>

4. Older mitochondria may be unable to perform adequately under stress.

Other factors influencing the course and progress of the disease are shown in Fig.1. Orthostatic hypotension is present in one-third of patients over the age of 65 years. Decreased glomerular filtration rate, reduced tubular transport, leading to reduced renal function and impaired capability to dilute urine and renal retention correlate with the presence of HF.

There could be either right sided HF or left sided HF or biventricular failure as per which ventricle/s are invoved. The following figure (Fig.2) depicts the pathophysiology.

CLINICAL MANIFESTATIONS
They maintain a precarious balance between the managed symptom state and acute symptom exacerbation. During the former, they are usually without symptoms on treatment. Acute symptom exacerbation, often requiring emergency management, can be precipitated by poor compliance with sodium restriction, infection, or stress.

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Fig.1

Fig.2

Often co-morbid conditions mask the symptoms.
1. Dyspnea on exertion may result from lung disease, lack of exercise, and deconditioning. Restlessness may be the presentation.
2. Nocturia, an early symptom may be caused by prostatic hypertrophy.
3. Oedema may be caused by venous insufficiency.
4. Increasing lethargy and confusion, probably the result of impaired cerebral perfusion & activity intolerance may be common.
5. Anorexia and loss of lean body mass due to GI hypoperfusion may be masked by oedema.

Epidemiological consideration and prognostication
HF afflicts about 5 million people in the United States, and a million admissions each year. Incidence increases with age, and older people >65 years becoming the fastest growing
segment of the population, the personal and financial burden of heart failure increase. Huynh et al followed 282 elderly heart failure patients on various treatment schedules for up to 14 years and identified seven factors affecting survival: This are shown in Table II.

**Table I**

1. Advanced age  
2. History of dementia  
3. Coronary artery disease  
4. Peripheral vascular disease  
5. Hyponatremia  
6. High blood urea  
7. Hypotension

Patients with four or more of the risk factors had a low probability of surviving longer than six months. Patients with two to three factors were likely to live at least a year.

Invasive, uncomfortable or risky procedures are not advised if longevity is jeopardised. Aggressive treatment such as a defibrillator may be considered in low risk patients. Similarly, a person having probable survival less than 6 months, need to be hospitalised.

The prevalence of AF is greater in elderly people, and the risk increases with age and underlying heart disease. The overall prevalence of AF is about 1%, but 70% are at least 65 years old, and about 50% are older than 75 years old. AF occurs in about 10-30% of patients with HF, and patients with HF are at increased risk of developing AF (HR 4.5 for men and 4.9 for women).

**Co-morbid conditions**

HF in elderly people usually does not occur as an isolated condition as in middle-aged patients. It has been estimated than more than two-thirds of patients with HF have two or more non-cardiac comorbidities and more than 25% may have six or more concomitant diseases. and management should be undertaken taking multiple comorbidities into consideration. They could be: 1) Anaemia, 2) Bad nutrition 3) Chronic lung diseases, 4) Depression, 5) Eye and ear dysfunction, 6) Failure of kidney function 7) Gout and arthritis.

**Anaemia** associated with chronic diseases like renal diseases, malignancies or under treatment on aspirin, non-steroidal anti-inflammatory drugs (NSAID), warfarin or inadequate intake of iron, folate or vitamin B₁₂ may confers adverse prognosis.

**Bad or Malnutrition** in older adults whether due to other diseases or HF itself increase frailty, immune deficiency and contribute to poor prognosis.

**Chronic lung diseases** may increase breathlessness and exercise intolerance in elderly patients with HF. Plasma brain natriuretic peptide (BNP) helps to distinguish between primary pulmonary symptoms and HF.

**Depression** specially caused by loss of spouse may lead to non-compliance of drug intake and affects the prognosis adversely with increased hospitalisation rates.

Eye and ear malfunction, associated with altered taste and smell function may interfere with the compliance with treatment advice. Cognitive impairment that may decrease compliance, postural hypotension and urinary incontinence, which may be aggravated by medical treatment of HF. Taken together all these factors may increase the risk of falls, the leading cause of hip fractures in elderly people, a condition associated with increased morbidity and mortality.

**Failure of kidney function** may be worsened by diuretics, angiotensin converting enzyme (ACE) inhibitors and may lead to volume overload in persons prone to HF. Renal failure is a bad prognostic indicator in patients with HF.

**Gout or arthritis** makes elderly people disabled who are often treated with NSAID that may enhance salt and water retention and thus impairs the function of diuretic and ACE inhibitors. They are also notorious for causing gastrointestinal bleedings.

**DIAGNOSTIC TESTS**

Clinical findings are the first and most important diagnostic tool for HF. Presence of symptoms at rest or during exercise plus the objective evidence of cardiac dysfunction like sinus tachycardia, hypotension, cold clammy sweating with pale or cyanotic extremities, 53 or 54 , murmurs, crepitations in bases of lungs, raised JVP, hepatomegaly and oedema etc.

**Electrocardiography** is first line diagnostic test. A normal ECG almost rules out systolic HF. Abnormalities could be: abnormal Q-waves, LBBB or atrial or ventricular arrhythmias. LBBB with severe LV systolic dysfunction
Diagnosis of systolic and/or diastolic dysfunction is clinched by **Echocardiography**. It is a widely available, easy, safe, rapid, non-invasive technique. Simple measure of LVEF is key to the diagnosis of either systolic or diastolic dysfunction. Systolic LV dysfunction is considered when LVEF is < 40% and LV systolic function is “preserved” when the LVEF is > 50%. The grey area between 40-50% needs to be explored further. Other conditions are: valve dysfunction, various chamber enlargement, hypertrophic cardiomyopathy, restrictive cardiomyopathy, Taka- Subo’s disease. Dobutamine Stress Echocardiography (**DSE**) may be useful to diagnose ischaemic origin of HF and myocardial viability.

**Chest X-ray** may show cardiomegaly, a clue to systolic HF and absence of cardiomegaly may indicate diastolic HF.

**Therapeutic trial** may help in the diagnosis of HF.

**Plasma BNP** level < 100 pg/ml makes the diagnosis of HF unlikely, while a value > 400 pg/ml strongly suggest the diagnosis. On the contrary values between 100 and 400 g/ml may be not diagnostic.

Other tests like **radionuclide angiography**, **cardiac magnetic resonance**, **pulmonary function tests**, **ambulatory ECG monitoring** give either re-confirmatory or additional information regarding structural abnormality, size of chambers, pleural effusion or pulmonary collapse or various arrhythmias.

**TREATMENT**

Optimal management includes control of risk factors, patient education, self-management and specific therapy. The goals are multiple: a) relieve symptoms, b) reducing hospitalisations, c)improve the quality of life, and 4) prolonging survival.

General treatment includes 1) management of hypertension, 2) control of diabetes and diabetes-induced complications, 3) dyslipidaemia 4) discontinuation of tobacco and control of alcohol intake (1-2 drinks daily), 5) Symptomatic coronary artery disease requires anti-platelet agents, statin, beta-blockers, nitrates and may need coronary revascularisation. 6) Precipitating conditions, such as anaemia, thyroid diseases, renal failure, infections and drugs (for instance NSAID), pulmonary embolism should be prevented and treated. 7) Low intake of salt (less than 2000 mg daily) and total fluid intake. 8) Measurement of weights should be done regularly, generally in the morning. Patients should maintain their weight within narrow limits (e.g. ±1 Kg in mild-to-moderate HF, ±1/2 Kg in severe HF). 9) Learning to self-adjust the dosage of drugs, like diuretics, anti-coagulants, 10) Regular low-intensity aerobic exercise (e.g. walking, stationary cycling) three to five times per week. Clear instructions about how and when to contact the Emergency Room for aggressive treatment should be provided.

Drug therapy is not significantly different from that recommended in younger patients and management still remains mainly empirical. The SENIORS study was specifically done to study elderly patients with HF with the beta-blocker, nebivolol, in patients ≥ 70 years with HF regardless of ejection fraction. After a mean follow-up of 21 months, decreased all-cause mortality or cardiovascular hospitalisation (HR 0.86, 95% CI 0.74-0.99; p = 0.039) was found in the treatment arm. Previous studies bisoprolol, carvedilol or metoprolol demonstrated a decreased relative risk of death in HF in younger group (excluding patients > 80 years) and focussed on those with low left ventricular ejection fraction (LVEF).

**Diastolic heart failure**

Diastolic HF has exactly similar symptoms and signs of HF, but with a normal or near normal left ventricular systolic function, and evidence of diastolic dysfunction (abnormal left ventricular filling and elevated filling pressures, decreased E/E’ ratio as obtained by tissue doppler). The exercise intolerance caused by impaired left ventricular filling leads to raised left atrial and pulmonary venous pressures and pulmonary congestion. Low cardiac output causes skeletal muscles fatigue. Rarely they are diagnosed while investigating associated disorders. Among patients with HF, 40-60% of cases may have a normal or near normal LVEF. The prevalence of diastolic HF among patients with HF has been estimated 15%, 33% and 50% at ages < 50, 50-70 and > 70 years respectively. Another 15% of elderly patients with HF have only mild systolic dysfunction (LVEF 45-54%). Patients with diastolic HF are more likely to be older, female, and hypertensive and less likely to have a previous myocardial infarction or to be treated with ACEI/ARB. They have lower in-hospital mortality (3% vs. 4%) but same length of stay in hospital.

Differential diagnosis include : i) obesity, ii) lung disease, iii) poorly controlled atrial fibrillation (AF), iv) coronary
ischaemia, v) volume overload (for instance renal failure) and vi) increased afterload (like hypertensive crisis)

Management principles for patients with diastolic HF include: i) the control of systolic and diastolic hypertension, ii) control of ventricular rate in patients with AF, iii) maintenance of sinus rhythm whenever possible, iv) prevention of sinus tachycardia, v) dehydration with diuretics for reduction of pulmonary congestion and peripheral oedema, vi) treatment and prevention of myocardial ischaemia. Regression has better prognosis than systolic HF but a worse outcome and the Cardiovascular Health Study reveal that diastolic HF is a common entity.

Data from the Framingham Heart Study, the V-HeFT trials and the Cardiovascular Health Study reveal that diastolic HF has better prognosis than systolic HF but a worse outcome than matched controls (annual mortality 8–9% vs. 15–19% in systolic HF vs. 1–4% in matched controls). Older age, male gender, NYHA class, lower LVEF, the extent of coronary artery disease, peripheral vascular disease, diabetes, and impaired renal function are independent predictors of mortality. Better outcome of diastolic HF may be not seen after adjustment for age, sex, NYHA class and coronary artery disease.

Atrial fibrillation, pacemaker and internal cardioverter defibrillator in HF in elderly:

Restoration and maintenance of sinus rhythm has importance in patients with HF and needs to be achieved in symptomatic patients on treatments for rate control.

Ongoing studies, and a prospective evaluation of the management of AF in HF (AF-CHF trial) will provide data to develop an evidence-based approach.

AFFIRM and RACE trials offered no survival benefit with rhythm control. Anticoagulation should be considered and continued in this group of high-risk patients not withstanding fears of bleeding.

Beta-blockers are useful for rate control and improve survival. Digoxin is useful for those intolerant to betablockers or CCBs. Beta-blockers appear to effectively prevent occurrence of AF in patients with systolic HF and are also safe in chronic obstructive pulmonary disease.

Complete AV nodal ablation and permanent pacemaker implantation may be required in drug-failed cases. The efficacy has been confirmed in a trial in which 66 patients with clinical HF, AF and a resting rate > 90 beats/min were randomly assigned to pharmacologic AV nodal blockade or AV nodal ablation and implantation of a VVIR pacemaker. After 1-year follow-up patients undergoing AV nodal ablation and VVIR pacemaker implantation, there were significant symptomatic improvement, however, with similar overall quality of life and NYHA functional class in the two groups.

In patients with moderate to severe left ventricular dysfunction along with LBBB with 1st degree AV block, the beneficial effects of DDD pacing are reduced or even reversed by the detrimental effects of right ventricular apical pacing. Here is the indication of biventricular pacing which improves left ventricular function and may be considered as a default option.

REFERENCES

2. Dickstein K, Cohen-Sotoal A, Filipatos G et al. ESC guideline for the diagnosis and management of acute and chronic heart failure 2008, the task force for diagnosis and treatment of acute and chronic heart failure 2008 of the European Society of Cardiology. Developed in collaboration with the HFA and endorsed by the ESMC. EHJ 2008;29:2388-442.


