Management of heart failure: An update
Talukder AKMNA

Introduction
Heart Failure is a common problem worldwide including in our country particularly in the elderly age group. In our country there is no definite data regarding incidence of heart failure, though the magnitude of the problem is supposed to be definitely significant. In the Western world the annual incidence is 2-4% between 35 and 64 years and 10% of patients over 65 years of age. In USA, 5 million people have heart failure. In recent decades management has improved as well as the prognosis, though in many ways cost of management in hospitals is very high. In USA and Europe there are updated guidelines from "National Advisory Body" on regular basis published at regular intervals regarding management of heart failure. In our country that is yet to come. Doctors should themselves acquire the updated knowledge regarding the management of HF.

Definition
Heart failure is a condition when the heart cannot maintain an adequate cardiac output to meet the demand of the body or can do so at the expense of an elevated filling pressure.

Causes of HF
Almost all forms of heart diseases can cause HF. Extra cardiac factors also contribute due to an increased work load.

Non cardiac causes

- Anaemia
- Thyrotoxicosis
- Beri-beri
- Padget's Disease
- A-V malformation
- Volume overload

Cardiac Causes

- IHD & MI
- Hypertension
- Cardiomyopathy
- DCM
- HCM + HOCM
- Restrictive cardiomyopathy
- Myocarditis
- Valvular H. Diseases - All forms
- Cardiac arrhythmia - Atrial Fibrillation and Atrial Flutter - Atrial Tachycardia

Precipitating Factors
Infection, anaemia, physical and emotional stress, excess fluid and salt intake, some drugs and other physical illness can further precipitate HF.

Forms of HF
Acute vs. Chronic Heart Failure:
Acute HF develops suddenly which may be due to severe IHD, acute MI, Hypertension, Heart block, Tachyarrhythmia, Myocarditis, Endocarditis, Pulmonary embolism, volume overload, valvular heart disease. When there is gradual impairment of cardiac function almost due to any cause, a chronic H.F. supervene. A number of compensatory mechanisms take place. Minor additional factor may precipitate overt or Acute H.F.

Left vs. Right HF and Biventricular HF:
LV, LA, Mitral and Aortic valves comprises the left side of the heart. In LVF, LV output goes down and left atrial, Pulmonary venous pressure goes up causing accumulation of fluid before left side of heart causing Pulmonary congestion and edema. Acute M.I., IHD, Hypertension, Mitral & Aortic Valvular Diseases, cardiomyopathy are the common causes and they presents with dyspnoea, orthopnea, cough, frothy sputum.

Dr. A.K.M. Nurul Alam Talukder, MCPS (Medicine), D.Card (D.U.), Asst. Professor, Department of Medicine, Mowlana Bhashani Medical College & Hospital, Uttara, Dhaka

The ORION Medical Journal 2006 May; 24:355-
which may be blood-tinged, or frank pulmonary edema. A gradual onset causes a reflux increase in pulmonary arterial pressure which can protect pulmonary edema.

In right sided heart failure, right ventricular output goes down causing accumulation of fluid before right side of heart causing edema. Increased JVP, Congestive hepatomegally, Ascites, Pleural effusion etc. Lung disease, pulmonary embolism, pulmonary and tricuspid valvular disease, and pulmonary hypertension are the common causes of isolated right heart failure. However, right sided HF are commonly due secondary to left sided heart failure, so in fact, biventricular failure supervene. A combined pulmonary congestion and accumulation of fluid in periphery are the manifestations of biventricular heart failure.

Systolic vs. Diastolic Failure:
In systolic failure the ventricle is unable to pump out blood due to poor contractility and is usually dilated, commonly due to CAD, DCM, and Pulmonary embolism. It is classical or familiar form of HF. In diastolic failure there is diminished LV filling due to poor ventricular relaxation and compliance due to stiffness. It is equally important like systolic failure. LVH (Hypertensive), HCM, HOCM, IHD, Amyloidosis, constrictive pericarditis, endomyocardial fibrosis are the common causes of diastolic dysfunction. In fact in many instances it is systolic and diastolic disturbances combined, playing the role.

Forward vs. Backward Failure:
Hypothetically in forward failure there is decreased cardiac output causing a decreased renal blood flow, activating RAA system resulting in fluid accumulation. In backward failure theory, the ventricle fails to discharge the blood from it and increases the back pressure causing accumulation of fluid in interstitial places. In fact both mechanism plays along with more complex pathophysiological factors.

High vs. Low Output Failure:
In Hyperthyroidism, Anaemia, Pregnancy, A-V malformation, Beriberi, Padget's disease, there are high cardiac output causing increased workload on heart causing heart failure. In others the cardiac output is low.

Pathophysiological Consideration
Cardiac output is the function of preload, afterload and myocardial contractility. In majority of patient with HF, the primary abnormality is impairment of ventricular function and in others there is increase outflow resistance, i.e. in hypertension aortic and pulmonary valvular diseases, which ultimately causes ventricular dilatation and myocardial functional impairment. In the beginning, sympathetic activation provides ionotropic support and maintains cardiac output. In myocardial failure there is decreased stroke volume causing increased blood accumulation in ventricle after systole and increases venous pressure (increases the preload) which helps to maintain normal cardiac output. But afterwards both the mechanism causes increase HR and increased filling pressure which has deleterious effect. Diminished renal perfusion activates RAA axis and there is increased ADH secretion, causing fluid retention, accumulation in interstitial space and ultimately increases peripheral resistance contributing to deleterious effect on heart and HF and a vicious cycle starts.

However, stretched atrial myocytes secretes ANP (Atrial natriuretic peptide) and stretches left ventricular myocytes secretes BNP (ß-type natriuretic peptide), both has beneficial effects as they cause natriuresis, vasodilatation, smooth muscle relaxation causing decreased afterload making a counter-balance.

Endothelium dependent vasodilatation is impaired (decreased endothelium secretion) and NO activity (vasodilator) is blunted causes deleterious effect on heart also. Also, changes occur in cardiac contractile gene expression involving sarcolemmal Ca-channel contributing to poor myocardial contractility. Currently more focus is being paid to ANP & BNP (or NT-Pro BNP- a byproduct of BNP) as impending biochemical marker of HF, and
also trials are going on for therapeutic benefit of these in HF and benefit of endothelin antagonist.

Clinical Manifestation
Symptoms - Depends on underlying causes & type of HF.
1. Dyspnoea: Initially on effort and then occurs with less effort or even at rest. Orthopnoea and PND may also be present. Patient may also present with cough and cardiac asthma or frank acute pulmonary edema. Cheney-stokes type of breathing may occur particularly HF with HTN or CAD.
2. Swelling of body: Common manifestation often with leg swelling and abdominal distention with low Urinary output.
3. Fatigue & weakness: Common symptom
4. Abdominal symptoms: Anorexia, nausea, abdominal pain and fullness are frequently complained.
5. Cerebral symptoms: Headache, insomnia, anxiety, confusion, lack of concentration, and memory disturbance may be present.

Physical signs

<table>
<thead>
<tr>
<th>Evidence of</th>
<th>Cyanosis</th>
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<tbody>
<tr>
<td>Dyspnoea</td>
<td>Pulse</td>
</tr>
<tr>
<td>Orthopnoea</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>Cold extremity</td>
<td>Pulsus alternance</td>
</tr>
<tr>
<td>Weight loss-cardiac cachexia</td>
<td>Irregular pulse</td>
</tr>
<tr>
<td>Edema</td>
<td>BP-low</td>
</tr>
<tr>
<td></td>
<td>JVP-raised</td>
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</tbody>
</table>

• CVS: Cardiomegally, evidence of valvular heart disease, 3rd and 4th heart sounds, and summation gallop may be present.
• Chest: Creps and dullness over base, evidence of frank pulmonary edema or pleural effusion may be present.
• Abdomen: Ascites, hepatomegally, splenomegally, jaundice, may be present.

Assessment of severity of HF
NYHA guidelines regarding classification of severity of HF can be used to assess severity.

Investigation
• ECG: for evidence of ischaemia, MI, hypertension, arrhythmia.
• X-Ray chest: cardiac size, pulmonary congestion, edema, pleural effusion.
• Haematological and biochemical: CBC, electrolyte, urea, creatinine, sugar, LFT, cardiac enzymes in Acute exacerbation to exclude acute MI.
• ANP, BNP, NT: Pro BNP done to screen LV dysfunction and treatment monitoring. Thyroid function test in appropriate test can be done.
• Urine: routine examination
• Echocardiography: 2D & Doppler echo. done to establish systolic & diastolic dysfunction of LV and RV and severity of HF. Echo may reveal aetiology of HF - valvular disease, IHD, cardiomyopathy, intra-cardiac thrombosis.
• Cardiac Catheterization: It is indicated in acutely decompensated LHF, severe acute HF, not responding to treatment, HF with unknown aetiology, Patient with angina pectoris, patient with MR and AV disease.
• Others: Cardiopulmonary exercise testing, 24 hours holter monitoring, resting and stress radionuclide angiography are done in special circumstances.

Management
A full clinical assessment about severity, type of HF, its aetiology, precipitating factors, and concomitant illness should be done and accordingly management is planned. Aim of treatment is prevention, maintenance, improved quality of life and survival.

General Management
Physical activity - In acute HF or acute on chronic HF rest in bed, if needed propped up position advocated. DVT is avoided by leg exercise where appropriate, elastic support stocking, and S/C heparin. Prolonged bed-rest is avoided and as condition improves, low level endurance exercise is encouraged avoiding strenuous activity.

Dietary Modification - Large meal avoided, weight reducing diet advocated where appropriate. In DM, renal failure, and lipid disorder, modification of diet is advocated accordingly. Generally, salt and water intake is restricted. Smoking should be stopped.
Immunization: Influenza and Pneumococcal vaccine should be given.

Drug Counselling: Patient should avoid:
- NSAID and Coxib
- Class 1 antiarrhythmic drug
- Calcium channel blocker
- Tricyclic anti-depressant
- Lithium salt
- Cortico steroids.

Counseling regarding diuretic self-dose modification according to weight gain and fluid retention should be done.

Pharmacological Therapy
- Oxygen: High concentration oxygen inhalation is given through variable performance mask if patient is breathless particularly in acute HF or HF with acute exacerbation.
- Diuretic: Diuretics give symptomatic relief of generalized edema and pulmonary edema. It reduces the preload also. Different types of diuretics are available. Dose, route and type of diuretics are chosen according to severity of HF keeping in mind the side-effects.

<table>
<thead>
<tr>
<th>Loop diuretics</th>
<th>Thiazides</th>
<th>K+ sparing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>Bendroflazide</td>
<td>Spironolactone</td>
</tr>
<tr>
<td>Bumetamide</td>
<td>Chlorthalidone</td>
<td>Amiloride</td>
</tr>
<tr>
<td>Torasemide</td>
<td>Metolazone</td>
<td>Triamterene</td>
</tr>
<tr>
<td>Indapamid</td>
<td></td>
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</tbody>
</table>

I/V loop diuretics are potent and give quick relief of breathlessness of pulmonary edema. It causes hypokalaemia. Thiazides are mild diuretics. Potassium sparing diuretics are also mild. All diuretics can be used in HF alone or in combination with others. Spironolactone among them, the only one diuretic gives 30% mortality benefit (RALES study) in moderate to severe HF but causes hyperkalaemia. Loop diuretics (e.g. Furosemide) can be combined with spironolactone to get synergistic effect with balancing side-effects of potassium disturbance which can be beneficial in severe HF.

a. ACEI: Benefits of ACEI are well established in HF with significant mortality benefit (CONSENSUS & SOLVD Study) as well as in M.I. with asymptomatic HF (SAVE study). It is recommended as 1st line therapy in HF.

They can cause 1st doze hypotension, dry cough, and allergic angioedema. They are started with low dose and increased thereafter step-wise.

<table>
<thead>
<tr>
<th>ACEI</th>
<th>Initial dose</th>
<th>Maintenance dose</th>
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<tbody>
<tr>
<td>Captopril</td>
<td>6.25mg BD</td>
<td>25-50mg TID</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5mg OD</td>
<td>10mg BD</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5mg OD</td>
<td>5-20mg OD</td>
</tr>
<tr>
<td>Ramipril</td>
<td>1.25-2.5mg OD</td>
<td>2.5-5mg BD</td>
</tr>
</tbody>
</table>

b. ARB: Are also used in HF, particularly if patient cannot tolerate ACEI with mortality benefit though not superior to ACEI. ARB can be combined with ACEI. in managing HF. Losartan Potassium, Valsartan, Irbesartan, Candesartan, are the examples of ARB. They are also started with low doses and are increased step-wise.

- Beta-Adrenoceptor Antagonist: Betablockers are useful in chronic stable heart-failure. MERIT, CIBIS2 study has shown improved symptomatic class, exercise tolerance, LV function & mortality benefit using Metoprolol, Bisoprolol respectively. US Carvedilol study using Carvedilol- a non-selective vasodilator beta-blocker with additional anti-oxidant properties has also demonstrated a significant improvement in mortality. All these drugs can be given with ACEI. Diuretics but should be started with low dose titrating slowly over a month. Transient worsening of HF may occur but afterwards it improves the symptoms of HF. However, it should be used cautiously to manage such cases.

<table>
<thead>
<tr>
<th>BB agent</th>
<th>1st Dose</th>
<th>Increment (mg/day)</th>
<th>Target dose</th>
<th>Titration period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bisoprolol</td>
<td>1.25</td>
<td>2.5, 3.75, 5, 10</td>
<td>10</td>
<td>Week-Month</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>12.5/25</td>
<td>25, 50, 100, 200</td>
<td>200</td>
<td>Week-Month</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3.125</td>
<td>6.25, 12.5, 25, 50</td>
<td>50</td>
<td>Week-Month</td>
</tr>
</tbody>
</table>
Review Article

- **Cardiac Glycosides**: Digoxin gives symptomatic benefit when used in HF particularly if associated with Atrial Fibrillation. However it does not give any mortality benefit. The dose is 0.125-0.25 mg daily. In the elderly and in Renal Failure the does is further reduced. It can cause digitalis toxicity particularly with high dose, dehydration, and hypokalaemia. Serum digoxin level should be monitored to maintain optimum dose. Drug holiday is maintained to avoid tachyphylaxis.

- **Nitrate**: It is used if patient is having angina and symptoms of dyspnoea. Its overall benefit in management of HF by evidence is lacking.

- **Ionotropic Agents**: Intravenous continuous infusion of Dopamine (2-10 ,ug/kg/min) and Dobutamine (2.5-10 ,ug/kg/min) are used in acute LVF, after cardiac surgery, AMI with Shock, Pulmonary Edema, and in patient with refractory HF as a bridge to transplantation. Their use is carefully monitored. Similarly Milrinone, Amrinone can be used simultaneously like Dopamine/ Dobutamine for short period only for symptomatic benefit as probably it eventually increases the overall mortality.

- **Vasodilators**: Sodium nitroprusside and hydralazine can be rarely used if vasoconstriction persists despite ACEI therapy in HF.

- **Antiarrhythmic Agents**: Beta-blockers used in HF are in fact beneficial for different types of Arrhythmia including Ventricular Tachyarrhythmia. Amiodarone is the only drug other than beta-blockers without negative ionotrophic effect which can be used in both supra-ventricular as well as ventricular arrhythmia. DC Shock to restore sinus rhythm and digitalis are the options for managing AF.

- **Anticoagulants**: Patient with HF are at increased risk of pulmonary and systemic embolism including stroke and is treated with I/V heparin followed by oral warferrin. Presence of clot in the LV is also treated in the same line. Patients with AF are also treated with warferrin. Hospitalized bed-ridden patients without such complications should receive S/C prophylactic heparin therapy. Alternatively, low-molecular weight heparin can be used instead of heparin.

**Surgery**

- **Revascularization**: The role of revascularization is unclear because of increased mortality. In selective patients particularly with hibernating, stunned myocardium, revascularization can be beneficial.

- **Pacemaker**: Bi-ventricular pacing can be considered in patient with decreased ejection fraction and ventricular dyssynchrony, SA node disease, and in Heart Block patient.

- **ICD**: Implantable cardioverter defibrillators are considered with bi-ventricular pacing in those who remain symptomatic with severe HF.

- **Heart Replacement Therapy**: Heart transplantation is the treatment of choice in young patients with intractable HF. Availability is limited. One year survival is over 90% and five years survival is over 75%. Ventricular assist device and artificial heart can be used in bridging to transplantation in acute severe myocarditis and in patients with permanent haemodynamic support. These are done in specialized centers with advanced facilities.

**Conclusion**

Judicial approach to the management of HF will eventually reduce the overall mortality and improvement of symptoms of HF. In our country, because of financial constraints and lack of cardiac transplant availability all support for HF cannot be provided for the mass population.

**Reference**

Review Article

Journal of Medicine 343: 246-253


5. Taskforce of the working group on HF of the European Society of Cardiology (1997) Treatment of HF. European Heart Journal 18: 736-753


