

Review article

Management of heart failure – an update

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Introduction

Heart failure (HF) is a common cardiovascular condition with increasing incidence and prevalence.¹ Several large clinical trials on use of pharmacological therapy and devices have resulted in an increasing use of evidence based therapy of heart failure. Despite these advances the morbidity and mortality of those afflicted with heart failure continues to remain high. Adherence to guidelines results in improved outcomes of heart failure patients. Since the mid-1990s discoveries from basic research and findings from key clinical trials have resulted in considerable change in the scope of therapies available and the continuing advancement of our understanding of the pathophysiological mechanisms of heart failure. In this article, we highlight these new developments.

Definition of heart failure

Heart failure can be defined as an abnormality of cardiac structure or function leading to failure of the heart to deliver oxygen at a rate commensurate with the requirements of the metabolizing tissues, despite normal filling pressures (or only at the expense of increased filling pressures).²

Heart failure may be either predominantly systolic and diastolic.³ The major trials in patients with heart failure and a reduced EF (HF-REF), or 'systolic HF', mainly enrolled patients with an EF $\leq 35\%$. Other, more recent trials enrolled patients with HF who did not have an entirely normal EF (generally considered to be $\geq 50\%$). The term HF with 'preserved' EF (HF-PEF) was created to describe these patients. Patients with an EF in the range 35–50% therefore represent a 'grey area' and most probably have primarily mild systolic dysfunction.⁴ Patients with systolic heart failure also have some amount of diastolic dysfunction. It is estimated that 40 to 50 per cent of patients with heart failure have preserved systolic function or a relatively normal left ventricular ejection fraction.⁵ HF-PEF seems to have a different epidemiological and aetiological profile from HF-REF.^{6,7} Patients with HF-PEF are older and more often female

and obese than those with HF-REF. They are less likely to have coronary heart disease and more likely to have hypertension and atrial fibrillation.⁸

Staging of heart failure

To express the severity of heart failure and to determine its prognosis and management both the ACCF/AHA stages of heart failure and the New York Heart Association (NYHA) functional classification are important.^{9,10} The ACCF/AHA stages of heart failure emphasize the development and progression of disease and can be used to describe individuals and populations, whereas the NYHA classes focus on exercise capacity and the symptomatic status of the disease. (Table-I & II)

Epidemiology of heart failure

Approximately 1–2% of the adult population in developed countries has heart failure, with the prevalence rising to $\geq 10\%$ among persons 70 years of age or older. Heart failure incidence increases with age, rising from approximately 20 per 1,000 individuals 65 to 69 years of age to >80 per 1,000 individuals among those >85 years of age.¹¹

Table-I: New York Heart Association functional classification of heart failure.

Class I	No limitation of physical activity. Ordinary physical activity does not cause undue breathlessness, fatigue, or palpitations.
Class II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in undue breathlessness, fatigue, or palpitations.
Class III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary physical activity results in undue breathlessness, fatigue, or palpitations.
Class IV	Unable to carry on any physical activity without discomfort. Symptoms at rest can be present. If any physical activity is undertaken, discomfort is increased.

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Table II. American College of Cardiology/American Heart Association Stages of Heart Failure

Stage	Definition	Patient description
A	High risk for developing heart failure (HF)	Hypertension, Coronary artery disease, Diabetes mellitus, Family history of cardiomyopathy.
B	Asymptomatic HF	Previous myocardial infarction (MI), LV hypertrophy or systolic dysfunction, asymptomatic valvular disease
C	Symptomatic HF	Known structural heart disease, shortness of breath and fatigue, reduced exercise tolerance.
D	Refractory end-stage HF	Marked symptoms at rest despite maximal medical therapy (eg, those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions

Heart failure in Bangladesh

In Bangladesh, we do not have data regarding the exact prevalence and incidence of heart failure. In one study carried out in a tertiary hospital, about one-seventh of total hospital admitted patient had heart failure. Mean age was 54.1 (±15.3) years. Majority (35.79%) had ischaemic heart disease (IHD) as the principal etiological factor but this frequently coexisted with a history of hypertension (46.8%). Hypertension was considered the primary risk

factor of heart failure in 29.14% of cases. Diabetes Mellitus (DM) co-existed with IHD in 41.4% and it was found more prevalent in Dilated Cardiomyopathy (DCM).¹²

Pathophysiology of heart failure

In patients with LV systolic dysfunction, the maladaptive changes occurring in surviving myocytes and extracellular matrix after myocardial injury (e.g. myocardial infarction)

Table III. Symptoms and signs typical of heart failure.¹⁶

Symptoms		Signs
Typical	More specific	
Breathlessness	Elevated jugular venous pressure	
Orthopnoea	Hepatojugular reflux	
Paroxysmal nocturnal dyspnoea	Third heart sound (gallop rhythm)	
Reduced exercise tolerance	Laterally displaced apical impulse	
Fatigue, tiredness, increased time to recover after exercise	Cardiac murmur	
Ankle swelling		
Less typical	Less specific	
Nocturnal cough	Peripheral oedema (ankle, sacral, scrotal)	
Wheezing	Pulmonary crepitations	
Weight gain (>2 kg/week)	Reduced air entry and dullness to percussion at lung bases	
Weight loss	Tachycardia	
Bloated feeling	Irregular pulse	
Loss of appetite	Tachypnoea (>16 breaths/min)	
Confusion (especially in the elderly)	Ascites, Hepatomegaly	

lead to pathological 'remodelling' of the ventricle with dilatation and impaired contractility, one measure of which is a reduced EF. Two key neurohumoral systems activated in heart failure are the renin–angiotensin–aldosterone system and sympathetic nervous system. In addition to causing further myocardial injury, these systemic responses have detrimental effects on the blood vessels, kidneys, muscles, bone marrow, lungs and liver, and create a pathophysiological 'vicious cycle', accounting for many of the clinical features of the heart failure syndrome, including myocardial electrical instability. Interruption of these two key processes is the basis of much of the effective treatment of heart failure.^{13,14}

Diagnosis of heart failure

The diagnosis of heart failure can be difficult, especially in the early stages. Although symptoms bring patients to medical attention, many of the symptoms of heart failure are non-specific and do not, therefore, help discriminate between heart failure and other problems. Symptoms that are more specific (i.e. orthopnoea and paroxysmal nocturnal dyspnoea) are less common, especially in patients with milder symptoms, and are, therefore, insensitive.¹⁵

The ACC/AHA guidelines emphasize (i) careful history and physical examination, (ii) laboratory investigations including complete blood count, test of renal and hepatic functions, urine analysis, electrocardiogram and chest x-ray, (iii) two dimensional and Doppler echocardiogram (iv) careful exclusion of coronary artery disease and thyroid disease in all patients, and (v) selective use of other diagnostic tests including serologic studies in selected patients based upon the clinical characteristics, risk factors, past medical and family history.⁸

Investigations to diagnose heart failure

Electrocardiogram

Heart failure is very unlikely (likelihood ? 2%) in patients presenting acutely and with a completely normal ECG.¹⁷ The ECG may show evidence of cardiac chamber enlargement or hypertrophy or Q waves (indicating loss of viable myocardium). It will give a possible clue to the aetiology of heart failure. The ECG shows the heart rhythm and electrical conduction, i.e. whether there is sinoatrial disease, atrioventricular (AV) block, or abnormal intraventricular conduction defect. These findings are also important for decisions about treatment (e.g. rate control and anticoagulation for atrial fibrillation, pacing for bradycardia, or Cardiac resynchronization therapy if the patient has Left bundle branch block).

Chest X-ray

A chest X-ray is probably most useful in identifying an alternative pulmonary explanation for a patient's symptoms and signs. It may, however, show pulmonary venous congestion or oedema in a patient with heart failure. It is important to note that significant LV systolic dysfunction may be present without cardiomegaly on the chest X-ray.¹⁶

Echocardiography

The echocardiogram provides immediate information on chamber volumes, ventricular systolic and diastolic function, wall thickness, and valve function.^{18,19} So echocardiography is used as most useful and if possible bed side investigation for diagnosis of heart failure along with its underlying cause and severity and complications. Left ventricular ejection fraction (LVEF) is frequently used for diagnosis of heart failure. LVEF may also be preserved (and stroke volume reduced) in patients with significant mitral regurgitation. Thus LVEF must be interpreted in its clinical context.

Natriuretic peptides

Because the signs and symptoms of heart failure are so non-specific, many patients with suspected heart failure referred for echocardiography are not found to have an important cardiac abnormality. Where the availability of echocardiography is limited, an alternative approach to diagnosis is to measure the blood concentration of a natriuretic peptide, a family of hormones secreted in increased amounts when the heart is diseased or the load on any chamber is increased.²⁰

Other cardiac imaging modalities in the evaluation of patients with suspected or confirmed heart failure

Exercise or pharmacological stress echocardiography is used to identify cardiac function at stress. This is mostly useful for evaluation of cardiac conditions with milder severity to determine whether they can produce symptoms during exercise.

Cardiac magnetic resonance (CMR) and radionuclide ventriculography is a non-invasive technique that provides most of the anatomical and functional information available from echocardiography, including evaluation of ischaemia and viability, as well as additional assessments. CMR is particularly valuable in identifying inflammatory and infiltrative conditions, and in predicting prognosis in patients with these.²¹

The main use of cardiac CT in patients with heart failure is a non-invasive means to visualize the coronary anatomy.²²

Coronary angiography may be required, urgently, in selected patients with acute heart failure (AHF) (shock or acute pulmonary oedema), particularly those with an associated acute coronary syndrome.

Cardiac catheterization and endomyocardial biopsy

In patients with suspected constrictive or restrictive cardiomyopathy, cardiac catheterization used in combination with other noninvasive imaging techniques may help to establish the correct diagnosis. In patients with suspected myocarditis and infiltrative diseases (e.g. amyloidosis) endomyocardial biopsy may be needed to confirm the diagnosis.¹⁶

Exercise testing

Exercise testing allows objective evaluation of exercise capacity and exertional symptoms, such as dyspnoea and fatigue. The 6-min walk test and a variety of treadmill and bicycle protocols are available. A normal exercise capacity in a patient not receiving treatment effectively excludes the diagnosis of symptomatic heart failure, although it must be remembered that there is a poor correlation between exercise capacity and resting haemodynamic measures, including EF.¹⁶

Genetic testing

Currently this is recommended in patients with dilated cardiomyopathy and AV block or a family history of premature unexpected sudden death, as a prophylactic implantable cardioverter defibrillator (ICD) may be indicated.¹⁶

Ambulatory electrocardiographic monitoring

Ambulatory ECG monitoring is valuable in the assessment of patients with symptoms suggestive of an arrhythmia or bradycardia (e.g. palpitations or syncope) and in monitoring ventricular rate control in patients with atrial fibrillation. It is useful for identifying the type, frequency, and duration of atrial and ventricular arrhythmias, silent episodes of ischaemia and bradycardia, and conduction disturbances, which may cause or exacerbate heart failure.¹⁶

Important Comorbidities in heart failure.

Atrial Fibrillation

Patients with heart failure are more likely than the general population to develop atrial fibrillation (AF). There is a direct relationship between the NYHA class and prevalence of AF in patients with heart failure progressing from 4% in those who are NYHA class I to 40% in those who are NYHA class IV. AF is also a strong independent risk factor for subsequent development of heart failure. In addition to those with HF-REF, patients with HF-PEF

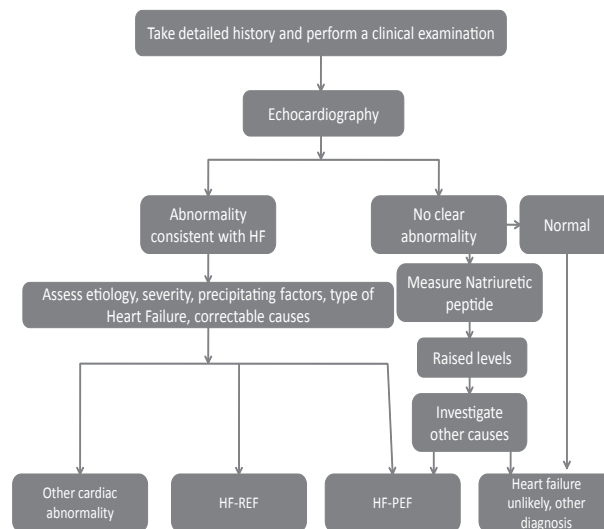


Figure-1 The detailed algorithm for the diagnosis of heart failure. (Serum Natriuretic peptides: High levels-BNP > 400 pg/ml. NT-proBNP > 2000 pg/ml. Raised levels-BNP >100-400 pg/ml. NT-proBNP > 400-2000 pg/ml. Normal levels- BNP < 100 pg/ml. NT-proBNP < 400 pg/ml)

are also at greater risk for AF. Heart failure and AF can interact to promote their perpetuation and worsening through mechanisms such as rate-dependent worsening of cardiac function, fibrosis, and activation of neurohumoral vasoconstrictors. AF can worsen symptoms in patients with heart failure, and, conversely, worsened heart failure can promote a rapid ventricular response in AF.

Angina

Angina and features of heart failure may coexist in patients with IHD. Beta-blockers are effective agents for angina as well as an essential treatment for systolic heart failure. Certain other effective antianginal drugs have been studied in large numbers of patients with systolic heart failure and shown to be safe (e.g., ivabradine, and nitrates).¹⁶

Anemia

Anemia is a common finding in patients with chronic HEART FAILURE. Anaemia is associated with more symptoms, worse functional status, greater risk of heart failure hospitalization, and reduced survival. Anemia is also more common in women and is seen in both patients with HF-REF and HF-PEF.¹²

Cachexia

A generalized process, wasting all body compartments [i.e. lean tissue (skeletal muscle), fat tissue (energy reserves), and bone tissue (osteoporosis)], may occur in 10–15% of patients with heart failure, especially those with HF-REF. This serious complication is associated with worse

symptoms and functional capacity, more frequent hospitalization, and decreased survival.¹⁶

Chronic obstructive pulmonary disease

COPD and asthma may cause diagnostic difficulties; especially in HF-PEF. These conditions are associated with worse functional status and a worse prognosis. Beta-blockers are contraindicated in asthma but not in COPD, although a selective beta-1 adrenoceptor antagonist (i.e. bisoprolol, metoprolol succinate, or nebivolol) is preferred. Oral corticosteroids cause sodium and water retention, potentially leading to worsening of heart Failure, but this is not believed to be a problem with inhaled corticosteroids. COPD is an independent predictor of worse outcomes in heart failure.¹⁶

Depression

Depression is common and is associated with worse clinical status and a poor prognosis in heart failure. It may also contribute to poor adherence and social isolation.¹⁶

Diabetes

Dysglycaemia and diabetes are very common in heart failure, and diabetes is associated with poorer functional status and worse prognosis.¹⁶

Hyperlipidaemia

Elevated low-density lipoprotein cholesterol is uncommon in HF-REF; patients with advanced HF-REF often have low concentrations of low-density lipoprotein, which is associated with a worse prognosis.¹⁶

Hypertension

Hypertension is associated with an increased risk of developing heart failure; antihypertensive therapy markedly reduces the incidence of heart failure (with an exception of alpha-adrenoceptor blockers, which are less effective than other antihypertensives in preventing heart failure). Negatively inotropic CCBs (i.e. diltiazem and verapamil) should not be used to treat hypertension in patients with HF-REF (but are believed to be safe in HF-PEF).^{8,16}

Gout

Hyperuricaemia and gout are common in heart failure and may be caused or aggravated by diuretic treatment. Hyperuricaemia is associated with a worse prognosis in HF-REF.²³

Kidney dysfunction and cardiorenal syndrome

The GFR is reduced in most patients with heart failure, especially if advanced and renal function is a powerful independent predictor of prognosis in heart failure.

Renin–angiotensin–aldosterone blockers (ACE inhibitors, renin inhibitors, ARBs, and MRAs) frequently cause a fall in GFR, although any reduction is usually small and should not lead to treatment discontinuation unless marked.¹⁶

Management of heart failure

Objectives in the management of heart failure

The goals of treatment in patients with established HEART FAILURE are to relieve symptoms and signs (e.g. oedema), prevent hospital admission and improve survival. Reductions in mortality and hospital admission rates both reflect the ability of effective treatments to slow or prevent progressive worsening of heart failure. This is often accompanied by reverse LV remodeling and a reduction in circulating natriuretic peptide concentrations.²⁴

Aggressive management of underlying cause like systemic hypertension, diabetes mellitus, underlying coronary artery disease and surgical correction of valvular heart diseases should be carried out if indicated. Correction of co morbid conditions like anaemia, thyroid disorders and nutritional deficiencies, should be done. Avoidance of alcohol and other toxins is necessary.⁵ Care should be taken to avoid common precipitating factors like infection, arrhythmia, salt and water retaining drugs, cardio depressant drugs. (Figure-2)

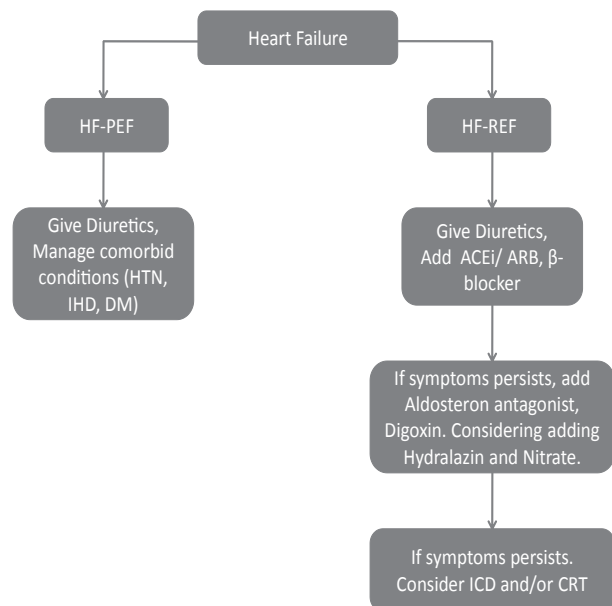


Figure-2 : Algorithm for management of Chronic heart failure.

Non pharmacological treatment of heart failure

Exercise training: Exercise training improves exercise capacity and quality of life in patients with mild to moderate heart failure. Early trials have shown beneficial effects of aerobic exercises. A low level to a moderate intensity (50-80% of maximal capacity) exercise with a warm up period of period of 10 to 15 min, for a duration of 20 to 30 min, 3-5 times a week is recommended in heart failure patients.²³

Diet and nutrition: Heart failure patients are at increased risk of weight loss due to loss of appetite and hypercatabolic status. Adequate calorie intake and nutrients are part of the dietary programmes in these patients. Salt restriction according to symptoms is an essential component. Because sodium intake is typically high (>4 g/d) in the general population, clinicians should consider some degree (e.g. <3 g/d) of sodium restriction in patients with stage C and D heart failure for symptom improvement.⁸ Fluid balance has to be maintained. A supervised nutritional intervention was shown to improve clinical status and quality of life in heart failure patients.²⁴
Education and counselling: The physician and caregivers should educate the patients regarding the nature of the disease and take steps to prevent further progression of disease. Lifestyle modification and strict compliance to medication is important. Psychological support by the treating physicians boosts the confidence of patients.⁵

Pharmacological treatment of heart failure

Diuretics

Diuretics are recommended in patients with HF-REF who have evidence of fluid retention, unless contraindicated, to improve symptoms.⁸ Three classes of drugs are available: loop diuretics, thiazide group and potassium sparing diuretics.

Loop diuretics: Agents belonging to this group include furosemide, torsemide and bumetanide. Recent data suggest that torsemide and bumetanide are more effective than furosemide in the treatment of advanced heart failure.²⁵

Thiazide diuretics: Diuresis with these agents is modest and these are ineffective at glomerular filtration rate below 40 ml/min.²⁵ The two commonly used agents in clinical practice are chlorothiazide and hydrochlorothiazide. The untoward effects are hypokalaemia, hypomagnesemia, hyperglycaemia, and hyperuricaemia.

Aldosterone receptor antagonists

These are recommended in patients with NYHA class II-IV and who have LVEF of 35% or less, unless

contraindicated, to reduce morbidity and mortality. Patients with NYHA class II should have a history of prior cardiovascular hospitalization or elevated plasma natriuretic peptide levels to be considered for aldosterone receptor antagonists. Creatinine should be 2.5 mg/dL or less in men or 2.0 mg/dL or less in women (or estimated glomerular filtration rate >30 mL/min/1.73 m²), and potassium should be less than 5.0 mEq/L. Careful monitoring of potassium, renal function, and diuretic dosing should be performed at initiation and closely followed thereafter to minimize risk of hyperkalemia and renal insufficiency.⁸

β-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 succinate, carvedilol, bisoprolol and nebivolol. Several large randomized placebo-controlled studies in patients with class II-IV heart failure in particular the MERIT-HF, COPERNICUS, CIBIS and COMET trials showed mortality and morbidity benefit with usage of β-blockers.⁵ Current recommendations suggest that the β-blocker therapy should be routinely administered to clinically stable patients with left ventricular systolic dysfunction.⁸

Angiotensin converting enzyme inhibitors (ACEI)

There is a clear-cut and compelling evidence that all patients with heart failure and asymptomatic left ventricular dysfunction should receive ACE inhibitor therapy. Multiple well designed prospective randomized placebo-controlled trials, particularly CONSENSUS I, V-HEFT II and SOLVD showed improvement in symptoms and survival in patients with mild to severe heart failure. These drugs are initiated at low doses and gradually increased to achieve target doses used in clinical trials. Renal function and serum potassium levels are assessed within 1-2 week and periodically thereafter. The adverse effects of these agents are dry cough, hypotension, worsening renal function, hyperkalaemia and angioedema.²⁶⁻²⁸

Angiotensin receptor blockers (ARB)

ARBs are recommended in patients with HF-REF with current or prior symptoms who are ACE inhibitor intolerant, to reduce morbidity and mortality.

Digoxin

Digoxin acts by inhibition of the sodium potassium adenosine triphosphatase enzyme and increases the intracellular Na. This drug is being used for more than a century in the treatment of heart failure. The randomized trials RADIANCE and the DIG trial showed significant

reduction in hospitalizations for worsening heart failure but no reduction in mortality. It is particularly useful in patients of heart failure with atrial fibrillation.²⁹
Other Drug Treatment.⁸

Anticoagulation

Patients with chronic heart failure with permanent/persistent/paroxysmal AF and an additional risk factor for cardioembolic stroke (history of hypertension, diabetes mellitus, previous stroke or transient ischemic attack, or ≥ 75 years of age) should receive chronic anticoagulant therapy.

Statins

Statins are not beneficial as adjunctive therapy when prescribed solely for the diagnosis of heart failure in the absence of other indications for their use.

Drugs of Unproven Value or That May Worsen HF:

Nutritional supplements as treatment for heart failure are not recommended in patients with current or prior symptoms of HF-REF. Hormonal therapies other than to correct deficiencies are not recommended for patients with current or prior symptoms of HF-REF. Drugs known to adversely affect the clinical status of patients with current or prior symptoms of HF-REF are potentially harmful and should be avoided or withdrawn whenever possible (e.g., most antiarrhythmic drugs, most calcium channel blocking drugs (except amlodipine), NSAIDs, or thiazolidinediones). Long-term use of infused positive inotropic drugs is potentially harmful for patients with HF-REF, except as palliation for patients with end-stage disease who cannot be stabilized with standard medical treatment.

Device Therapy

ICD therapy is recommended for primary prevention of SCD to reduce total mortality in selected patients with nonischemic DCM or ischemic heart disease at least 40 days post-MI with LVEF of 35% or less and NYHA class II or III symptoms on optimum treatment, who have reasonable expectation of meaningful survival for more than 1 year.⁸

CRT is indicated for patients who have LVEF of 35% or less, sinus rhythm, left bundle-branch block (LBBB) with a QRS duration of 120 ms or greater, and NYHA class II, III, or ambulatory IV symptoms on medical treatment. CRT is not recommended for patients with NYHA class I or II symptoms and non-LBBB pattern with QRS duration less than 120 ms and whose comorbidities and/or frailty limit survival with good functional capacity to less than 1 year.⁸

Surgery in heart failure:

As coronary artery disease is one of the major causes of heart failure every effort has to be made to undertake complete revascularization after assessment of myocardial viability. In patient with valvular heart diseases repair or replacement as per recommendations and guidelines is helpful in prevention and treatment of heart failure. In patients with secondary or functional mitral regurgitation and ischaemic mitral regurgitation, valve repair surgery preserving the mitral apparatus is advocated. Cardiac transplantation remains the gold standard therapy for end stage heart disease.³⁰

Acute heart failure^{8,16}

Acute heart failure (AHF) is the term used to describe the rapid onset of, or change in, symptoms and signs of heart failure. It is a life threatening condition that requires immediate medical attention and usually leads to urgent admission to hospital. In most cases, AHF arises as a result of deterioration in patients with a previous diagnosis of heart failure (either HF-REF or HF-PEF).

Initial assessment and monitoring of patients

Three parallel assessments must be made during the initial evaluation of the patient. (i) Does the patient have heart failure or is there an alternative cause for their symptoms and signs (e.g. chronic lung disease, anaemia, kidney failure, or pulmonary embolism)? (ii) If the patient does have heart failure, is there a precipitant and does it require immediate treatment or correction (e.g. an arrhythmia or acute coronary syndrome)? (iii) Is the patient's condition immediately life-threatening because of hypoxaemia or hypotension leading to underperfusion of the vital organs (heart, kidneys, and brain)?

Pharmacological therapy

Acute management

Oxygen

Oxygen may be given to treat hypoxaemia ($SpO_2 \leq 90\%$), which is associated with an increased risk of short-term mortality. Oxygen should not be used routinely in non-hypoxaemic patients as it causes vasoconstriction and a reduction in cardiac output.

Diuretics

Most patients with dyspnoea caused by pulmonary oedema obtain rapid symptomatic relief from administration of an i.v. diuretic, as a result of both an immediate venodilator action and subsequent removal of fluid.

Opiates

Opiates such as morphine may be useful in some patients with acute pulmonary oedema as they reduce anxiety and relieve distress associated with dyspnoea. Opiates are also thought to be venodilators, reducing preload, and may also reduce sympathetic drive.

Vasodilators

Vasodilators are probably most useful in patients with hypertension and should be avoided in patients with a systolic blood pressure <110 mmHg.

Nesiritide

Nesiritide—a human BNP that acts mainly as a vasodilator—was recently shown to reduce dyspnoea by a small but statistically significant amount when added to conventional treatment (mainly diuretic).

Inotropes

Use of an inotrope such as dobutamine should usually be reserved for patients with such severe reduction in cardiac output that vital organ perfusion is compromised.

Vasopressors

Drugs with prominent peripheral arterial vasoconstrictor action such as norepinephrine are sometimes given to severely ill patients with marked hypotension. Endotracheal intubation and invasive ventilation

The primary indication for endotracheal intubation and invasive ventilation is respiratory failure leading to hypoxaemia, hypercapnia, and acidosis. Physical exhaustion, diminished consciousness, and inability to maintain or protect the airway are other reasons to consider intubation and ventilation.

Mechanical circulatory support

Intra-aortic balloon pump

The conventional indications for an intra-aortic balloon pump (IABP) are to support the circulation before surgical correction of specific acute mechanical problems (e.g. interventricular septal rupture and acute mitral regurgitation), during severe acute myocarditis and in selected patients with acute myocardial ischaemia or infarction before, during, and after percutaneous or surgical revascularization.

Ventricular assist devices

Ventricular assist devices and other forms of mechanical circulatory support (MCS) may be used as a 'bridge to decision' or longer term in selected patients.

Ultrafiltration

Venovenous isolated ultrafiltration is sometimes used to

remove fluid in patients with heart failure, although is usually reserved for those unresponsive or resistant to diuretics.

Invasive monitoring

Intra-arterial line

Insertion of an intra-arterial line should only be considered in patients with persistent heart failure and a low systolic blood pressure despite treatment.

Pulmonary artery catheterization

Right heart catheterization does not have a general role in the management of Acute heart failure, but may help in the treatment of a minority of selected patients with acute (and chronic) heart failure. Pulmonary artery catheterization should only be considered in patients: (i) who are refractory to pharmacological treatment; (ii) who are persistently hypotensive; (iii) in whom LV filling pressure is uncertain; or (iv) who are being considered for cardiac surgery.

Monitoring after stabilization

Heart rate, rhythm, blood pressure, and oxygen saturation should be monitored continuously for at least the first 24 h of admission, and frequently thereafter. Symptoms relevant to heart failure (e.g. dyspnoea) and related to the adverse effects of treatments used (e.g. dizziness) should be assessed at least daily. Fluid intake and output, weight, and the jugular venous pressure and extent of pulmonary and peripheral oedema (and ascites if present) should be measured daily to evaluate the correction of volume overload. Blood urea nitrogen, creatinine, potassium, and sodium should be monitored daily during i.v. therapy and when renin-angiotensin-aldosterone system antagonists are being initiated or if the dose of any of these drugs is changed.

Readiness for discharge

Before discharge is contemplated, the acute episode of heart failure should have resolved and, in particular, congestion should be absent and a stable oral diuretic regimen established for at least 48 h. Long-term disease-modifying therapy (including a beta-blocker) should be optimized as much as possible and appropriate education provided to the patient and family/caregivers.

Prognosis

Although survival has improved, the absolute mortality rates for heart failure remain approximately 50% within 5 years of diagnosis.³¹ In the ARIC study, the 30-day, 1-year, and 5-year case fatality rates after hospitalization for heart failure were 10.4%, 22%, and 42.3%, respectively.³²

Heart failure in Bangladesh has reached epidemic proportions. Early identification of the risk factors and initiation of appropriate therapy at early stages prevents development of heart failure. Clinical diagnosis and diagnostic imaging, echocardiogram in particular identifies patients with heart failure. The optimum utilization of the available drugs, general measures and surgical procedures appropriate to the condition improves the outcome of these patients.

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