



Consensus Document

Management protocols for chronic heart failure in India



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ABSTRACT

Heart failure is a common clinical syndrome and a global health priority. The burden of heart failure is increasing at an alarming rate worldwide as well as in India. Heart failure not only increases the risk of mortality, morbidity and worsens the patient's quality of life, but also puts a huge burden on the overall healthcare system. The management of heart failure has evolved over the years with the advent of new drugs and devices. This document has been developed with an objective to provide standard management guidance and simple heart failure algorithms to aid Indian clinicians in their daily practice. It would also inform the clinicians on the latest evidence in heart failure and provide guidance to recognize and diagnose chronic heart failure early and optimize management.

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1. Introduction

Chronic heart failure (HF), a progressive and debilitating disease, is increasing in epidemic proportions and affecting both the developed and the developing world.^{1,2} Heart failure is associated with shorter life expectancy, increased frequency of hospitalization and poor quality of life (QoL), and is a major public

health challenge even in India.^{3–6} However, there is no large study that has explored the burden and impact of HF in India.⁶ The available data is primarily based on extrapolation of Indian data for risk factors of HF, i.e., hypertension, ischemic heart disease (IHD), obesity, diabetes mellitus (DM), and rheumatic heart disease (RHD).⁷

2. Definition

Heart failure is a complex clinical syndrome that underlines the inability of the heart to perform its circulatory function with the desired efficiency due to structural and/or functional (systolic or diastolic) alterations.^{3,8,9}

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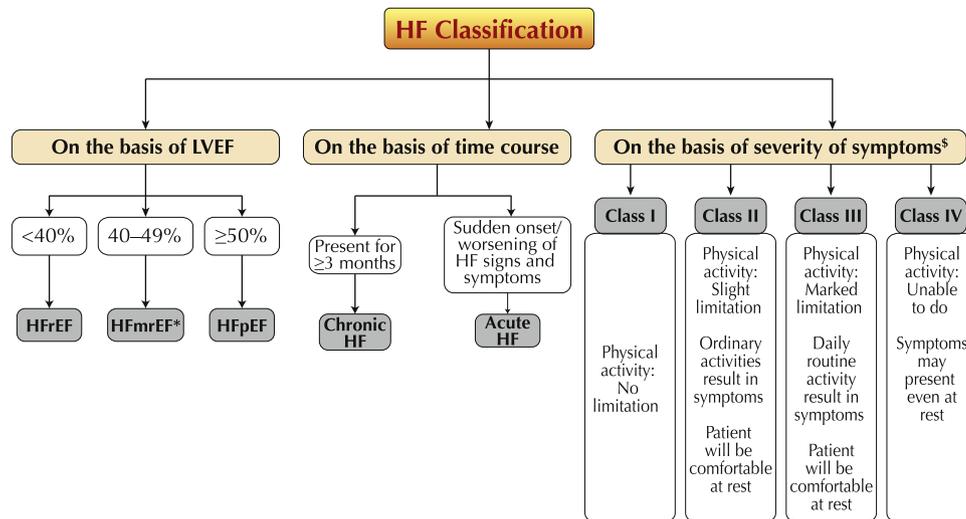


Fig. 1. Different classification systems for HF.

*It is referred to as a grey zone as the protocol and treatment for this group is not clear.

\$ New York Heart Association classification.

HF = heart failure; LVEF = left ventricular ejection fraction; HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mid-range ejection fraction; HFpEF = heart failure with preserved ejection fraction

3. Classification

There is no single agreed classification system for HF. Fig. 1 summarizes the commonly followed classification systems in HF management.^{8,9,10}

Other terms commonly used in HF are as below:^{8,9}

- **Stable HF:** When a HF patient on treatment does not exhibit any major change in the symptoms and signs of HF for at least one month, then the patient's condition is referred to as "stable".
- **Decompensated HF:** When the condition of a "chronic" previously "stable" HF patient deteriorates suddenly or slowly, it is referred to as "decompensated".
- **New-onset/de novo HF:** A patient with new-onset/de novo HF may present with symptoms in an acute or subacute (gradual) fashion.
- **Advanced HF:** It refers to patients with severe cardiac dysfunction, recurrent decompensation and severe symptoms despite optimal standard medical therapy.

4. Epidemiology, etiology and prognosis

4.1. Global data

Heart failure has emerged as a major global health issue, with an estimated worldwide prevalence of >37.7 million.¹¹ The burden

is rapidly increasing and it is projected that by 2030, the number of HF patients would rise by 25%.¹²

The prevalence of HF increases with age.¹³ At 55 years of age, the lifetime risk of HF is 33% and 28.5% for men and women, respectively.¹⁴ The key risk factors and causes of HF are mentioned in Box 1.^{8,9,11} The exponential rise in the incidence of hypertension and DM over the last couple of years has shaped the trajectory of HF development seen today.^{11–13}

Data indicates that the mortality rate is ~50% at 5 years from the initial diagnosis of HF.⁹ HF is a leading cause of hospitalization and represents 1–5% of total hospital admissions.^{3,11,15} About 2–17% of HF patients admitted to hospital die while in the hospital.³ Patients who survive have a high rate of rehospitalization and poor QoL.^{3,5} Despite improvements in medical care over the years, the prognosis of patients with HF remains poor and the survival rate is worse than in those with breast, bowel or prostate cancer.³

Studies estimate that in patients with clinical HF, the prevalence of HF with preserved ejection fraction (HFpEF) is ~50% (range: 22–73%) in the Western population.^{8,9} Key differences in the HF with reduced ejection fraction (HFrEF) and HFpEF patient profiles are mentioned in Table 1.^{8,16,17,18}

4.2. Indian data

The current estimates about incidence of HF in India vary widely from 1.3 to 23 million.¹⁹ The Trivandrum Heart Failure Registry (THFR) reported that HFpEF accounted for 25% of the total

Box 1. Common risk factors and causes of HF

- IHD
- Hypertension
- DM
- AF
- CKD
- VHD

IHD = ischemic heart disease; DM = diabetes mellitus; AF = atrial fibrillation; CKD = chronic kidney disease; VHD = valvular heart disease

Table 1
HFrEF vs. HFpEF—salient differences.

HFrEF	HFpEF
Basic pathophysiology—reduction of LV systolic function	Alteration of LV filling
Men > Women	Women > Men
Younger (compared to HFpEF)	More often older
History of MI and DCM is more common	History of hypertension, AF and VHD is more common
	Impact of co-morbidities may be more profound
LV is dilated with eccentric remodeling	Concentric remodeling (with or without LVH) is present in many
	LV end-diastolic volume is not increased relative to the stroke volume
Vasodilators improves LV systolic performance	Vasodilators have little impact on LV systolic performance
Myocardial stress/injury is more pronounced	Myocardial inflammation and fibrosis are more prominent

HFrEF = heart failure with reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; LV = left ventricular; MI = myocardial infarction; DCM = dilated cardiomyopathy; AF = atrial fibrillation; VHD = valvular heart disease; LVH = left ventricular hypertrophy

HF burden, indicating that in Indian clinical practice, HFrEF is predominantly observed.¹⁹ The available Indian data on HF is presented in Table 2.^{19–24} The overall incidence is likely to increase in the future owing to the following factors:^{6,22,25,26}

- Aging population
- Rising coronary artery disease (CAD) prevalence
- Epidemic rise of key risk factors such as hypertension and DM
- Persistence of diseases such as RHD and untreated congenital heart disease

Table 2
Data on HF in India.

	The Asian Sudden Cardiac Death in Heart Failure (Asian-HF): Indian subset data ^a	Trivandrum Heart Failure Registry (THFR) ^b	Practice Innovation and Clinical Excellence (PINNACLE) India Quality Improvement Program (PIQIP) ^c	INTERNATIONAL Congestive Heart Failure (Inter-CHF): Indian patient data ^d	Medanta Registry ^e
	(n = 1436)	(n = 1205)	(n = 15,870)	(n = 858)	(n = 6141)
Mean age (years)	57.8	61.2	56	56	58.9
Male (%)	75.7	69	77	62	83
HFpEF	–	26%	–	–	0.4%
HFrEF	–	–	–	53%	99.6%
Common etiology	- ischemic (37.3%)	- IHD (72%) - DCM (13%) - RHD (8%)	–	- IHD (46%) - hypertensive heart disease (14%) - VHD (12%) - idiopathic DCM (11%)	- CAD: 76.9% - DM: 18.1% - RHD: 5%
Common co-morbidities	- CAD (51.1%) - hypertension (37.9%) - DM (37.1%)	- hypertension (58%) - DM (55%) - CKD (18%) - COPD (15.4%) - AF/atrial flutter (15%)	- hypertension (37%) - CAD (27.3%) - DM (23%) - MI (17.4%)	- DM (26%) - COPD (16%)	- DM(48.8%) - hypertension (48.3%) - AF (4.8%)
Prognosis	–	Length of hospital stay (median): 6 days In-hospital mortality: 8.5%		1-year mortality: 23.3%	6-month mortality: 13.7%
		30-day mortality rate: 12.5% 90-day mortality rate: 18.1% 1-year mortality rate: 30.8% Rate of re-admission at 1 year: 30.2%		Mean age at time of death: 59 years (vs. 72 years in South America and 69 years in China)	
Patients on guideline-based medical therapy (%)	- ACEI: 45.4% - ARB: 33.8% - BB: 67.3% - Diuretics: 79.6% - MRAs: 61.3%	At discharge: - ACEI: 38.6% - ARB: 10.1% - BB: 58.2% - Diuretics: 81.2% - MRAs: 45.9%	ACEI/ARB: 33.5% BB: 34.9%	- ACEI: 51% - ARB: 17% - BB: 57% - Loop diuretics: 81% - MRAs: 47%	- ACEI/ARB: 67.1% - BB: 81.5% - Diuretics: 78.9% - MRAs: 11.8% - Ivabradine: 17%

HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; IHD = ischemic heart disease; DCM = dilated cardiomyopathy; RHD = rheumatic heart disease; VHD = valvular heart disease; CAD = coronary artery disease; DM = diabetes mellitus; CKD = chronic kidney disease; COPD = chronic obstructive pulmonary disorder; AF = atrial fibrillation; MI = myocardial infarction; ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin II receptor blocker; BB = beta-blocker; MRAs = mineralocorticoid receptor antagonists; LVEF = left ventricular ejection fraction.

^a It only enrolled patients with HFrEF (LVEF ≤ 40%).

^b First organized HF registry in India. It defined HFpEF as LVEF ≥ 40%.

^c Extension of the American College of Cardiology's PINNACLE Registry in the United States. It only enrolled patients with HFrEF (LVEF ≤ 40%).

^d It defined HFrEF as LVEF < 40%.

^e It defined HFrEF as LVEF ≤ 35%.

- Rising burden of lung diseases

4.3. How are Indian HF patients different?^{19–24,27}

- Indian patients present with HF at a younger age than those in the West. E.g., the mean age in the THFR, Medanta Registry and the INTER-CHF (Indian subset) study was 61.2, 58.9 and 56 years, respectively, as compared to 72.4 years in the ADHERE Registry of the USA.
- The male to female ratio is also different in India (70:30 as per the THFR and 83:17 in the Medanta Registry) compared to USA and Africa (almost 50:50). This may partly reflect that, in India, more males seek healthcare than the females unlike the West.
- The etiology of HF in India is also different from that in the West. Though IHD is the most common etiology both in the USA and India (as per the THFR and Medanta Registry), RHD is also a major contributor (accounts for 8% of the burden in the THFR and 5% in the Medanta Registry) in India but not in the USA.
- The prevalence of risk factors also differs between India and the West. E.g., DM is much more prevalent among Indians than those in the West as per the THFR data.
- The prognosis of HF in Indian patients appears to be worse than those in the West. The in-hospital mortality observed in the THFR (8.4%) was almost double compared to that reported in the ADHERE registry (4%) of the USA. The INTER-CHF study also showed that the 1-year mortality of HF is high in India i.e., 37%. As HF presents about a decade earlier in Indians, it generally strikes patients in the prime of their lives. Majority of the Indian patients are from low socio-economic strata, who have to spend out-of-pocket for their treatment, unlike in the West.

5. Diagnosis

Early diagnosis of HF is important to initiate appropriate treatment to reduce mortality, hospitalizations and healthcare costs.²⁸ However, the diagnosis can be challenging at times as not all HF patients exhibit typical symptoms and neither do all patients who have seemingly typical symptoms have HF.³ Hence, clinical evaluation and diagnostic tools are the two key aspects for the accurate diagnosis of HF (Box 2).^{8,9}

5.1. Clinical evaluation

Clinical evaluation involves a detailed history (individual and family) and thorough physical examination.^{8,9} Observation of various signs and symptoms elucidated during the clinical evaluation aid in the process of diagnosis.^{8,9} Some of the important points to consider are mentioned in Box 3.^{8,9,28,29}

It may be specifically difficult to recognize and interpret the signs and symptoms of HF in patients who are obese or elderly or have co-morbid chronic lung disease.⁸ It is also important to note

that the mere presence of risk factors for HF or their structural consequences (e.g., ventricular hypertrophy or asymptomatic diastolic dysfunction) should not be considered as a basis to label someone as a patient with HF.⁸ Moreover, symptoms and signs of HF (particularly evidence of congestion) should be assessed at each visit as they prove important in monitoring a patient's response to treatment.⁸

History-taking (Box 4) involves eliciting personal history and family history.^{8,9} Detailed history provides clues about the etiology, duration and severity of the disease and prognosis of the patient. It also helps identify the opportunities for interventions.⁹

5.2. Diagnostic tests

Goal-directed use of diagnostic tests aids the clinical evaluation and confirms the diagnosis of HF and presence of co-morbidities.

5.2.1. Essential initial investigations

The essential initial investigations should include the following^{8,9}

- 1) Basic blood estimations
- 2) 12-lead electrocardiography (ECG)
- 3) Chest X-ray
- 4) 2D echocardiography,
- 5) Biomarkers (brain natriuretic peptide [BNP] or N-terminal pro-BNP [NT-pro-BNP])

5.2.1.1. *Basic blood estimations.* The important tests to be considered for the basic blood work are listed in Box 5.^{8,9}

5.2.1.2. *Electrocardiography.* As part of the initial evaluation, a 12-lead ECG (Box 6) is recommended in **all patients** presenting with signs and symptoms of HF.^{8,9,28,29,30} A normal ECG may exclude HF in nearly 90% of the cases.⁸ An abnormal ECG increases the probability of HF diagnosis; however, it has low specificity.⁸ It is also important to note that although unusual, ECG can be normal in patients with severe HF.

Specific points to be noted in an ECG include the following: heart rate, PR interval, QRS duration, QT interval, pathological Q waves, evidence of left atrial (LA) overload, left ventricular (LV) hypertrophy, bundle branch block.^{29,30}

5.2.1.3. *Chest X-Ray.* A chest X-ray (Box 7) is recommended in patients with suspected, acute decompensated, or new-onset HF.^{9,28,29,31} It is a useful tool to identify pulmonary venous congestion or edema in HF.^{8,29} It also plays a key role in identifying an alternative explanation for the pulmonary symptoms and signs experienced by a patient, e.g., pulmonary malignancy and interstitial pulmonary disease.^{8,9,31}

5.2.1.4. *Echocardiography.* The most useful and widely used test to establish the diagnosis of HF is echocardiography.⁹ A detailed

Box 2. Essential components to confirm a diagnosis of HF

- 1) Symptoms and signs of pulmonary and/or systemic venous congestion
- 2) Structural abnormality of atria and/or ventricles or heart valves
- 3) Evidence of impaired ventricular filling at rest or effort
- 4) Exclusion of other diagnoses of overlapping symptoms
- 5) Objective documentation of reduced exercise capacity
- 6) Elevated natriuretic peptide

Box 3. Key points in the clinical evaluation of HF**Key symptoms**

- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Reduced exercise tolerance
- Fatigue and more time to recover post-exercise
- Ankle swelling
- Weight gain
- Nocturia
- Nocturnal cough
- Puffy face in the morning
- Bendopnea (shortness of breath when leaning/bending forward)

Key signs

- Elevated jugular venous pressure
- Hepatojugular reflux
- Third heart sound (gallop rhythm)
- Clinically evident cardiomegaly
- Tachycardia
- Tachypnea
- Hepatomegaly
- Pleural effusion
- Pedal edema
- Rales
- Mitral regurgitation
- Cardiac murmur
- Fourth heart sound

Points to consider during physical examination

- Orthostatic changes in BP and heart rate
- Strength and regularity of pulse rate
- BP (supine and upright), especially response of BP to Valsalva maneuver
- Peripheral edema
- Hepatomegaly and/or ascites
- Jugular venous pressure (at rest and following abdominal compression)
- Presence of extra sounds and murmurs—presence of S₃ sound indicates an adverse prognosis in HFrEF. S₄ sound may be present in HFpEF. Presence of murmurs may suggest VHD.
- Size and location of point of maximal impulse
- Presence of right ventricular heave
- Pulmonary status: respiratory rate, rales, pleural effusion (more prominent on the right compared to the left); rales may be absent in advanced HF despite major congestion
- BMI and evidence of weight loss
- Temperature of lower extremities

BP = blood pressure; HFrEF = heart failure with reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; VHD = valvular heart disease; HF = heart failure; BMI = body mass index

echocardiographic examination would provide information about the structure and function of the heart hence, *2D echocardiography* is the **cornerstone of HF diagnosis** (Box 8).^{8,9,32,33} Echocardiography is also an important test to differentiate between HFrEF and HFpEF.^{32,33}

Transthoracic echocardiography (TTE) should be the preferred choice to assess both LV and right ventricular systolic and diastolic function.⁸ *Transesophageal echocardiography* (TOE) has limited use in the routine diagnosis of HF; however, it could be an important modality in certain clinical scenarios (e.g., patients with congenital heart disease, valve disease, suspected aortic dissection or suspected endocarditis, and to rule out intracavitary thrombi in patients with AF who need cardioversion).⁸ TOE can also be used when the severity of mitral or aortic valve disease does not match the patient's symptoms using TTE alone.⁸ *Stress echocardiography*

(exercise or pharmacological) can help assess inducible ischemia, exercise capacity and/or myocardium viability.⁸ It can also help in the assessment of valve morphology for possible intervention.³²

If echocardiography does not provide definite clues, invasive testing may be required for the diagnosis of HF.

5.2.1.5. Biomarkers. Of the array of biomarkers available for the diagnosis of HF, BNP and NT-pro-BNP are the ones that are extensively used clinically.^{8,34,35} In healthy adults, BNP secretion is very low.³⁶ In response to abnormal myocardial stretch (as in HF), the ventricular myocyte secretes a large amount of prohormone BNP 1–108.³⁶ This is quickly cleaved into a biologically active (but less stable) BNP 1–32 and an inert (but more stable) NT-pro-BNP 1–76.³⁶ It needs to be underscored that the negative predictive value of BNP/NT-pro-BNP is very high (0.94–0.98), while the positive

Box 4. Points to consider during history-taking

- Duration and severity of disease
- Aggravating factors

Key triggers of HF are as follows:

- Drugs like NSAIDs, analgesics and steroids
- Infection
- Ischemia
- Anemia
- Chest infection
- Recent onset of fever
- Influenza or pneumococcal infections

- Assess salt and fluid intake
- Effect on exercise capacity, physical activity and sexual activity
- Presence of the following:
 - peripheral edema or ascites
 - chest pain
 - anorexia
 - early satiety
 - weight loss/gain
 - sleep problems and presence of disordered breathing at night
 - signs of palpitations, (pre)syncope and ICD shocks
 - symptoms of TIA or thromboembolism
 - drugs that may exacerbate HF

- Severity of dyspnea and fatigue
- Recent or history of frequent hospitalization for HF
- Compliance with current medication (including drugs for HF)
- Vaccination (influenza and pneumococcal vaccines)
- Comprehensive family history

HF = heart failure; NSAIDs = non-steroidal anti-inflammatory drugs; ICD = implantable cardioverter defibrillator; TIA = transient ischemic attack

predictive value is low (0.64–0.67), making them a good tool to **rule out** HF but a poor tool to help establish diagnosis.^{35,37}

As per the European Society of Cardiology guidelines, a cut-off value of BNP >35 pg/mL and NT-pro-BNP of >125 pg/mL is abnormal in the non-acute setting and is indicative of HF.⁸ However, for clinical purposes in India, this writing committee suggests that a **BNP value of >500 pg/mL or a NT-pro-BNP value of 1000 pg/mL strongly suggests the possibility of HF.**

The utility of BNP and NT-pro-BNP in HF is outlined in [Box 9](#).^{8,34–38}

Age, gender or co-morbid conditions can impact BNP and NT-pro-BNP levels and, hence, these factors should be considered during interpretation.^{34,35} Conditions due to which levels of natriuretic peptides can be spuriously high or low are mentioned in [Table 3](#).^{34,35}

Though the European Society of Cardiology guidelines on HF recommend BNP testing ahead of echocardiography in their diagnosis algorithm, this writing committee suggests using **echocardiography ahead of the BNP/NT-pro-BNP test.** Today, ultrasound machines are available even in remote and small

Box 5. Important investigations in blood work

- Complete blood count
- Serum electrolytes (sodium, potassium, etc.)
- Renal function test
- Liver function test
- Blood glucose (fasting plasma glucose and postprandial plasma glucose)
- Glycosylated hemoglobin (HbA1c)
- Lipid profile
- Thyroid function test
- Iron profile—serum iron, total iron-binding capacity, ferritin and folate

Box 6. Role of ECG in HF diagnosis

- Helps to understand the etiology of the disease, e.g., presence of ST-segment deviation, T-wave inversion or pathologic Q-waves might indicate the presence of CAD
- Provides information about the prognosis of the disease
- Helps understand the indications for therapy, e.g., anticoagulation for AF, pacing for bradycardia etc.
- Indicates presence of ventricular tachyarrhythmia

CAD = coronary artery disease; AF = atrial fibrillation

Box 7. Essential observations—chest X-ray.

- Enhanced cardiothoracic ratio
- Presence of pulmonary venous congestion or pulmonary edema
- Presence of pleural effusion
- Lung abnormalities
- LA and pulmonary artery enlargement
- Kerley B lines

LA = left atrial

Box 8. Essential echocardiographic features to look for in suspected HF

- Enlargement of heart chambers
- LVEF
- Grade of diastolic dysfunction
- Presence and severity of mitral regurgitation and tricuspid regurgitation
- Functional mitral regurgitation
- Estimation of pulmonary artery pressure
- LA volume index
- Global longitudinal strain, if possible
- IVC diameter and collapsibility
- Pericardial effusion and pleural effusion
- Other structural abnormalities such as aneurysm, scar, thrombus, etc.

LVEF = left ventricular ejection fraction; LA = left atrial; IVC = inferior vena cava

centers of India. Besides, training physicians and generating expertise in getting an echocardiogram done is far more simple, cost-effective and practical in India than opting for a biomarker test as it would be substantially costlier and would need the availability of a sophisticated biochemistry laboratory.

Suppressor of tumorigenicity 2 (ST2) and galectin 3 are the new biomarkers that have shown promise in the diagnosis of HF.³⁵ While an elevated ST2 levels at discharge tends to give prognostic information over and above BNP levels in patients with HF, galectin 3 was found to be a better predictor of 60-day HF mortality.³⁵ The

Box 9. Role of BNP and NT-pro-BNP in HF

- To rule out HF
- To assess short - term prognosis (admission BNP)
- To estimate long - term prognosis (discharge BNP)
- For guided (tailored) HF therapy*

*When pharmacotherapy for HF involves an ARNI, NT-pro-BNP and not BNP, should be the preferred biomarker; because ARNI acts by increasing BNP levels. Lower NT-pro-BNP levels indicates better prognosis.

HF = heart failure; BNP = brain natriuretic peptide; ARNI = angiotensin receptor neprilysin inhibitor; NT-pro-BNP = N-terminal pro-BNP

Table 3
Conditions that can show higher or lower natriuretic peptide levels than expected.

Higher levels than expected	Lower levels than expected
Increasing age ^a	Obesity
ACS ^a	Flash pulmonary edema
Renal insufficiency	Pericarditis/tamponade
RV dysfunction ^a	Genetic polymorphism
AF	End-stage cardiomyopathy
PH ^a	
Pulmonary embolism ^a	
Anemia ^a	
Sepsis	
Mitral regurgitation ^a	

^aDelineates likely elevation from ventricular stretch.

ACS = acute coronary syndrome; RV = right ventricular; AF = atrial fibrillation; PH = pulmonary hypertension

high cost and availability issues of these markers have resulted in them getting a backseat in the Indian scenario.

5.2. Other diagnostic tests

The other diagnostic tests that can aid diagnosis of HF are mentioned in [Box 10](#).^{8,9,39–44}

5.3. Key points to consider for the diagnosis of HFpEF

HFpEF is a heterogeneous clinical syndrome of effort intolerance with preserved ejection fraction (EF).⁸ The diagnosis of chronic HFpEF, especially in the typical elderly patient with comorbidities and no obvious signs of central fluid overload, is cumbersome and a validated gold standard is missing.^{8,45,46} Proposed criteria for the diagnosis of HFpEF are mentioned in [Box 11](#).^{8,45}

Diagnostic accuracy increases as more criteria are met. If all criteria are not being fulfilled, a greater reliance should be placed on natriuretic peptides.

Mentioned below are the important points with regards to HFpEF that needs to be kept in mind.^{4,6}

1. There is a difference between diastolic dysfunction, diastolic heart failure, and HFpEF; the latter two are clinical syndromes. Demonstration of diastolic dysfunction is not a prerequisite for diagnosing HFpEF.
2. Diagnosing HFpEF may require invasive hemodynamic testing in some patients
3. A normal BNP does not exclude the diagnosis of HFpEF
4. Elevated pulmonary artery systolic pressure on echocardiography in the presence of a normal EF and no lung disease should prompt consideration of HFpEF
5. There is no standardized protocol for dynamic testing and, hence, it is not recommended in routine clinical practice
6. All patients with HFpEF should be systematically evaluated for the presence of CAD

A practical algorithm for the diagnosis of HF is outlined in ([Fig. 2](#)).

6. Management

Successful management of HF involves patient education, non-pharmacological management, pharmacological management and, in certain scenarios, implantation of devices and/or the option of revascularization.^{8,9}

6.1. Patient education^{8,9}

A one-on-one discussion with the patient and caregiver ([Box 12](#)) should be initiated as soon as the diagnosis of HF is confirmed. The strategies and tools used for patient education should be aimed towards improving patient knowledge about HF, removing misconceptions, motivating self-care, and providing tips

Box 10. Other diagnostic tests for HF

- o **Cardiac Magnetic Resonance (CMR):** It is an important imaging technique for patients with nondiagnostic echocardiographic studies (particularly for imaging of the right heart) and in patients with complex congenital heart diseases and in suspected infiltrative myocardial disease.
- o **Cardiac Computed Tomography (Cardiac CT):** It is a useful test to exclude a diagnosis of CAD, in the absence of relative contraindications, as it allows visualization of the coronary anatomy in HF patients with low intermediate pre-test probability of CAD or those with equivocal non-invasive stress tests.
- o **Single-photon Emission Computed Tomography (SPECT):** It is useful in assessing ischemia and myocardial viability.
- o **Positron Emission Tomography (PET):** It is a useful tool to assess ischemia and viability. It is also useful for diagnosis of diseases such as nonspecific aortoarteritis, IgG gammopathy and other inflammatory disorders such as sarcoidosis.
- o **Coronary Angiography:** As CAD is a common etiology of HF in India, coronary angiography is indicated in most patients, especially in patients with DM. In undiagnosed HF (especially in those aged >40 years) where the etiology is unclear, it should be mandatory to do a coronary angiography to rule out CAD (in view of the high incidence of CAD, especially in diabetics). Hence, in Indian HF patients, even in the absence of angina, it is important to rule out CAD using coronary angiography. Even though the incidence of conditions such as pulmonary AV fistula is rare, it is important to rule out these conditions using coronary angiography or CT.
- o **Late Gadolinium Enhancement (LGE):** It may be important for diagnosis, prognosis and therapy design. The presence of LGE on CMR is an indicator of myocardial fibrosis and it also helps in the detection of scars.
- o **Right Heart Catheterization:** Invasive hemodynamic monitoring with a pulmonary artery catheter should be performed to guide therapy in patients who have respiratory distress or clinical evidence of impaired perfusion and in whom the adequacy or excess of intracardiac filling pressures cannot be determined from clinical assessment. It is useful for the assessment of pulmonary vascular assistance (in specific situations).
- o **Endomyocardial Biopsy:** This investigation is not a part of routine evaluation for HF but should be considered in patients with rapidly progressive HF or worsening ventricular dysfunction that persists despite standard treatment. It should be used in patients when a specific diagnosis is suspected (that can be confirmed only by biopsy) that would influence treatment.

CAD = coronary artery disease; HF = heart failure; IgG = immunoglobulin G; DM = diabetes mellitus; AV = arteriovenous; CT = computed tomography

Box 11. Criteria for the diagnosis of HFpEF

- Symptoms and signs of HF
- Normal or near normal LV systolic function
- LA volume ≥ 34 mL/m²
- Elevated natriuretic peptides, i.e., BNP >35 pg/mL and/or NT-pro-BNP >125 pg/mL
- Corroborative evidence such as elevated pulmonary systolic pressure, reduced 6-minute walk-time and increase in mitral E/e' on modest exercise

HF = heart failure; LV = left ventricular; LA = left atrial; BNP = brain natriuretic peptide; NT-pro-BNP = N-terminal pro-BNP; E/e' = ratio between early mitral inflow velocity and mitral annular early diastolic velocity

for disease management. The perils of poor adherence should be communicated and regular follow-up and discussion with the consulting physician should be encouraged.

6.2. Non-pharmacological management^{8,9}

Non-pharmacological management of HF (Box 13) is as important as the drugs and devices used to manage HF.

6.3. Vaccination^{8,9,47,48}

Though generally neglected, vaccination is an important aspect in the management of HF.

- Pneumococcal vaccination: First dose after confirmation of HF diagnosis and a second dose after 5 years.

- Influenza vaccination: To be given every year before the onset of winter (September/October).

6.4. Pharmacological management

Pharmacological treatment is the cornerstone of HF management.⁴⁹ Even though there is not much difference between patients with HFrEF and HFpEF as far as prognosis is concerned, they differ in terms of response to therapies.^{8,9} Notably, it is only in

patients with HFrEF that treatments have been proven to reduce both morbidity and mortality.^{8,9}

6.4.1. Patients with HFrEF

Neurohormonal activation plays a major role in the propagation of HF.⁵⁰ Its modulation is important not only in relieving symptoms but also in improving long-term prognosis.^{8,9,50} Various randomized controlled trials have demonstrated the mortality benefits (Fig. 3) of neurohormonal modulators such as angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), mineralocorticoid receptor antagonists (MRAs), beta-blockers and the recently approved angiotensin receptor neprilysin inhibitors (ARNIs) in patients with HFrEF.^{8,9,50,51} Diuretics are mainly used to manage the signs and symptoms of congestion and may improve clinical outcomes.^{8,9}

6.4.1.1. ACE inhibitors^{8,9,12,34,52}

- Should be used in all patients, symptomatic and asymptomatic, unless contraindicated.
- Should be up-titrated gradually to the maximally tolerated dose.
- Shown to reduce mortality and morbidity in HFrEF.
- Watch out for hyperkalemia, hypotension, renal dysfunction and angioneurotic edema.
- *Monitor*: renal function and serum potassium levels

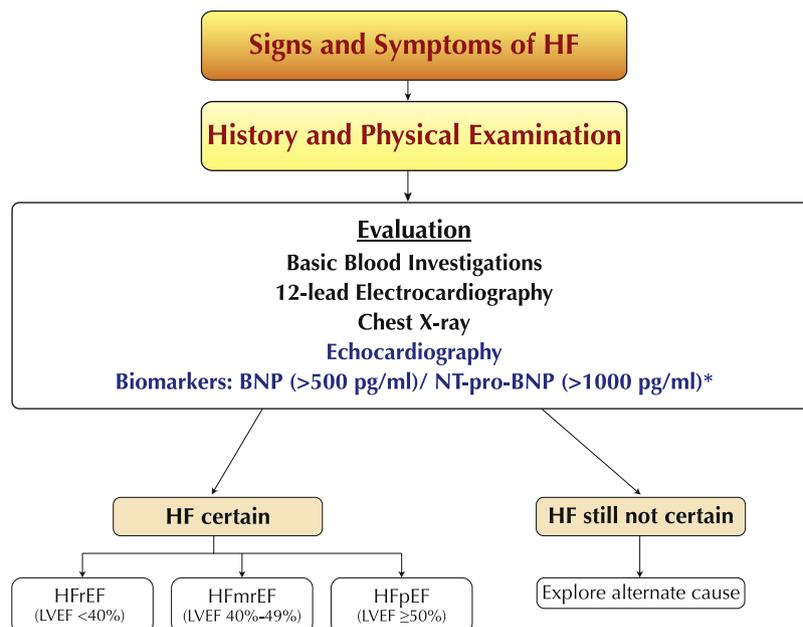


Fig. 2. Algorithm for diagnosis of patients with chronic HF.

*As suggested by this committee

HF = heart failure; BNP = brain natriuretic peptide; NT-pro-BNP = N-terminal pro-BNP; HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mid range ejection fraction; HFpEF = heart failure with preserved ejection fraction; LVEF = left ventricular ejection fraction

Box 12. Points to communicate to an HF patient

- What happens in HF?
- Causes and triggers of HF
- Commonly seen symptoms of HF
- Treatment for HF and importance of adherence; expected key side-effects with the treatment
- Lifestyle modifications
- Early recognition of signs/symptoms of worsening HF
- Monitoring of disease on regular basis (pulse, BP and weight)
- Importance of regular medical appointments

HF = heart failure; BP = blood pressure

Box 13. Non-pharmacological management of HF*Fluid and diet*

- Advise the patient about fluid intake (based on the patient's body weight), physical activity, exposure to heat, etc. Fluid intake should be reduced, especially in advanced HF patients.
- Alcohol should be totally avoided in patients with alcoholic cardiomyopathy. In other patients, alcohol moderation needs to be discussed.
- Limit salt intake, especially in patients with advanced HF. Advise on the use of salt substitutes with caution as they may contain potassium. If salt substitutes are used in large quantities with RAAS blockers, it may lead to hyperkalemia.
- Advise patients to maintain normal weight.

Exercise

- A stable HF patient should be encouraged to exercise, but the functional capacity of the patient should be taken into consideration.

Other lifestyle changes

- Smoking should be strongly discouraged.
- Pneumococcal and influenza immunization should be encouraged.
- Stress management should be advised.

HF = heart failure; RAAS = renin angiotensin aldosterone system

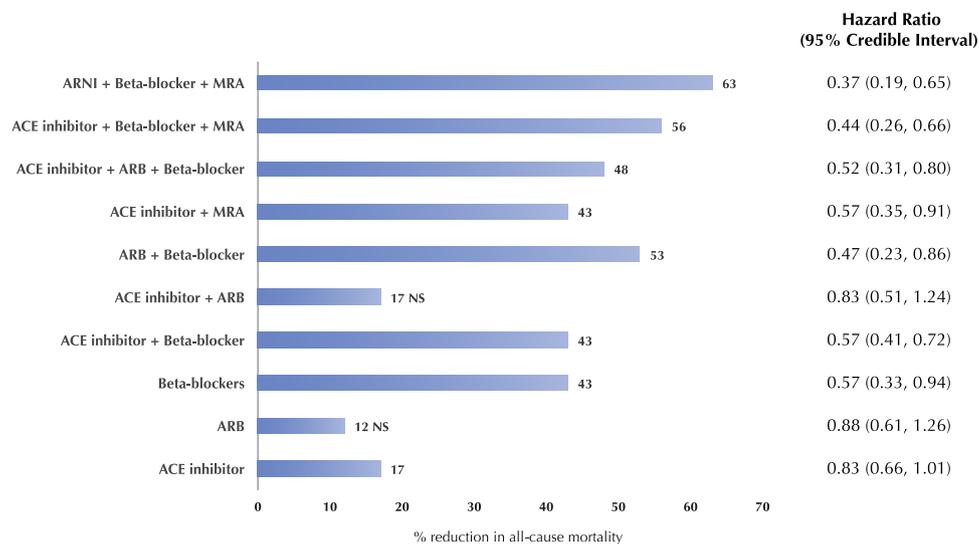


Fig. 3. Reduction in all-cause mortality, with; different classes of drugs vs. placebo.

NS = Non-significant

ARNI = angiotensin II receptor blocker neprilysin inhibitor; MRA = mineralocorticoid receptor antagonist; ACE = angiotensin converting enzyme; ARB = angiotensin II receptor blocker

- Assess around 1–2 weeks after initiation and periodically thereafter.
- Monitoring is especially important in patients with pre-existing hypotension, hyponatremia, DM or azotemia, and in patients taking potassium supplements.

6.4.1.2. ARBs^{8,9,12,34,53}.

- o Recommended as an alternative to ACE inhibitors in patients who cannot tolerate an ACE inhibitor.
- o Do not combine with an ACE inhibitor, except in symptomatic patients receiving a beta-blocker but who cannot tolerate MRA. Strictly monitor patients who receive the combination of ACE inhibitor and ARB.
- o Watch out for hyperkalemia, hypotension, angioneurotic edema and renal dysfunction.
- o *Monitor:* Blood pressure (BP) (including postural BP changes), renal function, and potassium levels
 - Check within 1–2 weeks after initiation and when the administered dose is changed.
 - Close surveillance is recommended in patients with systolic BP <80 mmHg, low serum sodium, DM, and impaired renal function.

6.4.1.3. ARNI^{8,34,38,54,55}.

- o New therapeutic class of drug that acts via inhibition of both renin angiotensin aldosterone system (RAAS) and the neutral endopeptidase system.
- o The first and the only drug approved to be used in this class is sacubitril-valsartan. Sacubitril is a neprilysin inhibitor (NI) while valsartan is an ARB.
- o Sacubitril-valsartan has been shown to be superior to ACE inhibitors (enalapril) in terms of reduction in mortality and morbidity.
- o Sacubitril-valsartan can be used instead of an ACE inhibitor/ARB, if cost is not an issue
- o Should not be combined with an ACE inhibitor/ARB. Due to the potential risk of angioedema, a gap of at least 36 h should be kept between an ACE inhibitor and an ARNI.
- o Watch out for hypotension, hyperkalemia, and angioneurotic edema.
- o Risk of hypotension is greater in patients with activated RAAS; hence, it is important to correct volume or salt depletion prior to the administration of this drug or it can be started at a lower dose.
- o For patients on ARNI, NT-pro-BNP, but not BNP, is a suitable biomarker to monitor overall disease status.

Table 4
Recommended doses of different drugs.

	Initiation dose	Maximum dose
ACE inhibitors		
Enalapril	2.5 mg b.i.d.	10–20 mg b.i.d.
Lisinopril	2.5–5 mg o.d.	20.0–40 mg o.d.
Perindopril	2 mg o.d.	8–16 mg o.d.
Ramipril	1.25–2.5 mg o.d.	10 mg o.d.
ARBs		
Candesartan	4–8 mg o.d.	32 mg o.d.
Losartan	25–50 mg o.d.	50–150 mg o.d.
Valsartan	20–40 mg b.i.d.	160 mg b.i.d.
ARNI		
Sacubitril-Valsartan	100 mg b.i.d. Patients not on a RAAS blocker/Patients who were previously on low-dose RAAS blocker/Patients with severe renal impairment (eGFR <30 mL/min/1.73 m ²)/Patients with moderate hepatic impairment (Child-Pugh B classification): 50 mg b.i.d. Dose can be doubled after 2–4 weeks, as tolerated	200 mg b.i.d.
Beta-blockers		
Bisoprolol	1.25 mg o.d.	10 mg o.d.
Carvedilol	3.125 mg b.i.d.	25.0–50 mg b.i.d.
Metoprolol	12.5–25 mg o.d.	200 mg o.d.
succinate (CR/XL)		
Nebivolol	1.25 mg o.d.	10 mg o.d.
MRAs		
Spironolactone	12.5–25 mg o.d.	50–200 mg
Eplerenone	12.5–25 mg o.d.	50 mg
Diuretics		
Furosemide	20–40 mg o.d. or b.i.d.	400 mg
Torsemide	5–20 mg o.d.	200 mg o.d.
Chlorthalidone	12.5–25 mg o.d.	100 mg
Hydrochlorothiazide	12.5–25 mg o.d. or b.i.d.	100 mg
Indapamide	2.5 mg o.d.	5 mg
Metolazone	2.5 mg o.d.	20 mg
Funny channel inhibitors		
Ivabradine	5 mg b.i.d.	7.5 mg b.i.d. Increase the dose if the heart rate continues to be >70 bpm.

ACE = angiotensin converting enzyme; ARB = angiotensin II receptor blocker; ARNI = angiotensin receptor neprilysin inhibitor; RAAS = renin angiotensin aldosterone system; eGFR = estimated glomerular filtration rate; MRA = mineralocorticoid receptor antagonist

○ **Monitor:**

- BP: If hypotension occurs, consider dose adjustment of diuretics, concomitant antihypertensive drugs, and treatment of other causes of hypotension (e.g., hypovolemia). If hypotension persists, reduce the dosage or temporarily discontinue sacubitril-valsartan.
- Serum creatinine: Down-titrate or interrupt therapy in patients who develop a clinically significant decrease in renal function.
- Serum potassium: Monitor periodically and treat appropriately, especially in patients with risk factors for hyperkalemia (severe renal impairment, DM, hypoaldosteronism, or a high-potassium diet).
- Angioedema: If angioedema occurs, discontinue the drug immediately, provide appropriate therapy, and monitor for airway compromise. Sacubitril-valsartan must not be re-administered in such scenarios.

6.4.1.4. **Beta-blockers**^{8,9,12}.

- Shown to reduce mortality and morbidity in HFrEF.
- Should be started when euvolemic. Can be used both in sinus rhythm and atrial fibrillation (AF).
- Dose should be up-titrated gradually. Closely monitor the vital signs and symptoms during up-titration.
- Watch out for bradycardia, hypotension, and wheezing.
- Abrupt withdrawal should be avoided as it can lead to clinical deterioration.

6.4.1.5. **MRAs**^{8,9,56}.

- Recommended in all symptomatic patients.
- Both spironolactone and eplerenone are well established in HF, though androgenic side-effects are lesser with eplerenone.
- Watch out for hyperkalemia, especially in DM.
- **Monitor:**
 - Serum potassium and renal function: Check within 2–3 days of initiation, especially in patients with DM or renal impairment. The values should be rechecked at day 7 post-initiation and at least once a month for the first 3 months and every 3 months thereafter
 - A fresh monitoring cycle should be initiated when an ACE inhibitor/ARB/ARNI has been added to the treatment or if the dose of the existing ACE inhibitor/ARB/ARNI is increased.

6.4.1.6. **Diuretics**^{8,9}.

- Should be used in all patients who have evidence of fluid retention and in patients with a prior history of fluid retention
- Torsemide/Frusemide:
 - Start the drug early, oral or IV, depending on the degree of congestion.
 - Continuous infusion may be used in case of resistant HF.
 - As congestion improves gradually, titrate towards the smallest dose needed.
- Metolazone/Hydrochlorothiazide:
 - Add in case of resistant HF.
 - Watch out for hyponatremia, hypokalemia and hypotension.
- **Monitor:** Serum potassium and other electrolytes on a periodic basis.

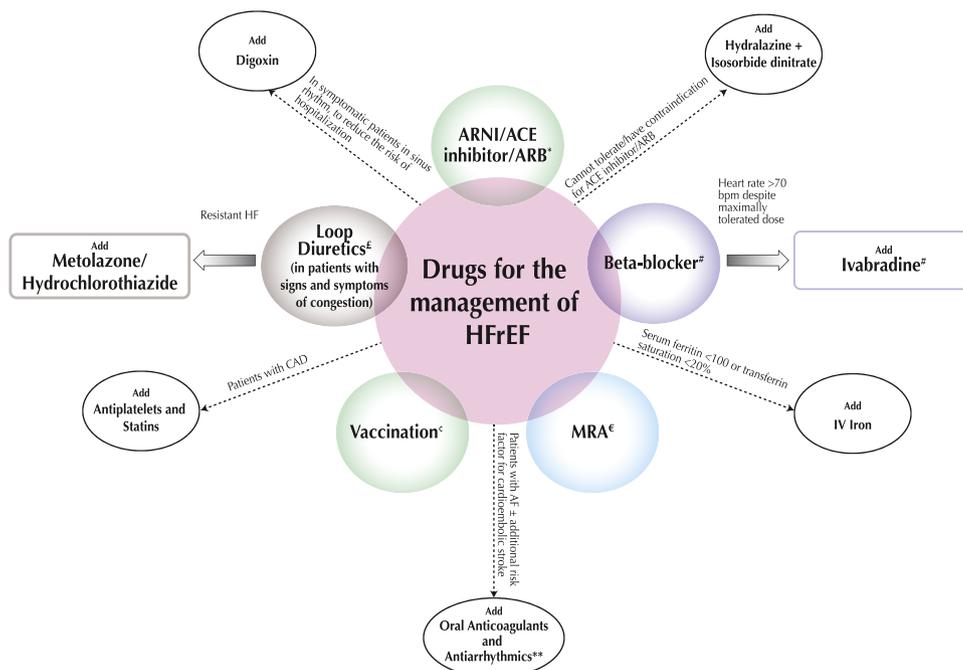


Fig. 4. Core therapy plus other drugs for the management of HFrEF.

Note:

*If affordability is not an issue, then an ARNI can be used instead of an ACE inhibitor. In patients who cannot tolerate ACE inhibitors, ARBs can be considered.

Beta-blocker should be titrated to its maximum tolerated dose to obtain heart rate <70 bpm. Ivabradine is recommended in patients with sinus rhythm and heart rate >70 bpm despite being treated with a maximally tolerated dose of a beta-blocker. Ivabradine can also be used in patients who have contraindication/s for beta-blocker/s.

€ Spironolactone and eplerenone are equal in terms of efficacy, but spironolactone has lesser androgenic side-effects.

£ First dose of pneumococcal vaccination should be given after confirmation of HF diagnosis and second dose after 5 years. Influenza vaccination should be given every year before the onset of winter (September/October).

£ Loop diuretics (Torsemide or Frusemide) should be used in patients with volume overload.

**Among antiarrhythmics, only amiodarone and dofetilide have a neutral effect on mortality in HF patients.

HFrEF = heart failure with reduced ejection fraction; ARNI = angiotensin receptor neprilysin inhibitor; ACE = angiotensin converting enzyme; ARB = angiotensin II receptor blocker; MRA = mineralocorticoid receptor antagonist; IV = intravenous; AF = atrial fibrillation; CAD = coronary artery disease; HF = heart failure

6.4.1.7. Ivabradine^{8,34,57}.

- Recommended to be used in patients with sinus rhythm, who have a heart rate >70 beats per minute (bpm) despite being treated with a maximally tolerated dose of a beta-blocker.
- Usually combined with a beta-blocker; however, if beta-blockers are contraindicated, it can be used alone.
- Watch out for bradycardia and luminous phosphene.

Table 4 lists the initiation and maximum recommended doses of different drugs approved for use in HFrEF.^{8,9,34,54,57}

6.4.1.8. Other drugs^{8,9,58}

6.4.1.8.1. Digoxin.

- May be considered in symptomatic patients in sinus rhythm to reduce the risk of hospitalizations.
- In patients with co-morbid AF, digoxin can be used to reduce ventricular rate when other options cannot be used.
- Use of digoxin has gradually diminished as it has a narrow therapeutic window.
- Should be used with caution in the elderly and in patients with chronic kidney disease (CKD).

6.4.1.8.2. Combination of hydralazine and isosorbide dinitrate.

- Can be used in symptomatic patients who cannot tolerate/have a contraindication for an ACE inhibitor/ARB/ARNI.
- Does not adversely affect electrolytes.
- Watch out for hypotension.

6.4.1.8.3. Intravenous iron.

- Iron deficiency is common in HF patients with and without anemia.
- If serum ferritin level is <100 or transferrin saturation is <20%, use intravenous (IV) ferric carboxymaltose.
- The recommended dose of IV ferric carboxymaltose is 500–1000 mg in 50 mL saline given over 10–15 min.
- It is recommended to test plasma iron level after 2–3 months. If the level is still low, then the dose may be repeated.

6.4.1.8.4. Statins.

- Not shown to be effective in HF.
- Should continue use in patients with clinically proven CAD to reduce future coronary events.
- Should be withdrawn in patients with dilated cardiomyopathy (DCM).

6.4.1.8.5. Oral anticoagulants and antiplatelets.

- Patients with chronic HF with permanent/persistent/paroxysmal AF, intracardiac thrombus and an additional risk factor for cardioembolic stroke (history of hypertension, DM, previous stroke or transient ischemic attack, or ≥75 years of age) should receive chronic anticoagulant therapy.
- Non-vitamin K antagonist oral anticoagulants (NOACs) are preferable if there is no metallic prosthetic heart valve and if the cost of therapy is not a major issue.
- Chronic anticoagulation is also reasonable for patients with chronic HF who have AF but do not have an additional risk factor for cardioembolic stroke.
- Antiplatelets should be used in patients with CAD.

6.4.1.8.6. Antiarrhythmics.

- Amiodarone and dofetilide are the only antiarrhythmic agents that have neutral effects on mortality in patients with HF.

The recommendations for the management of patients with HFrEF are represented in Fig. 4.

6.4.1.9. Drugs of uncertain benefit in patients with HFrEF^{8,9}. Use of drugs that have uncertain benefits in HF should be discouraged as they affect/prevent the use of proven drugs and add to the cost of the overall therapy for HF. These drugs include the following:

- 1) Trimetazidine
- 2) L-carnitine
- 3) Co-enzyme Q
- 4) n-3 polyunsaturated fatty acids

Table 5

List of drugs with definite benefits, uncertain benefits and with potential to cause harm in HF patients.

Beneficial	Uncertain	Harmful
Core drugs <ul style="list-style-type: none"> ○ ACE inhibitors ○ ARNI ○ Beta-blockers ○ MRAs ○ Diuretics ○ Ivabradine 	<ul style="list-style-type: none"> ○ Trimetazidine ○ L-carnitine ○ Co-enzyme Q ○ n-3 polyunsaturated fatty acids 	<ul style="list-style-type: none"> ○ Thiazolidinediones (glitazones) ○ NSAIDs or COX-2 inhibitors ○ Diltiazem/verapamil
Other drugs <ul style="list-style-type: none"> ○ Digoxin ○ Combination of hydralazine and isosorbide dinitrate ○ IV iron ○ Statins ○ Oral anticoagulants/antiplatelets ○ Antiarrhythmics 		

ACE = angiotensin converting enzyme; ARNI = angiotensin receptor neprilysin inhibitor; MRA = mineralocorticoid receptor antagonist; ACE = angiotensin converting enzyme; IV = intravenous; NSAIDs = non-steroidal anti-inflammatory drugs; COX-2 = cyclooxygenase 2

6.4.1.10. Drugs contraindicated in patients with HF_{rEF}^{8,9,59}

The following drug classes increase the risk of worsening of HF and HF-related hospitalization and, hence, should be avoided in patients with HF_{rEF}:

- 1) Thiazolidinediones (glitazones)
- 2) Non-steroidal anti-inflammatory drugs (NSAIDs) or COX-2 inhibitors
- 3) Diltiazem/verapamil (dihydropyridine calcium channel blockers such as amlodipine and felodipine should be used only if there is a compelling indication—management of coexistent hypertension)

Table 5 outlines the utility of different drugs in HF management.

6.4.2. Patients with HF_{pEF}^{8,45,46,60,61}

- Patients with HF_{pEF} are often elderly, female and highly symptomatic, and often have a poor QoL.
- It is recommended to screen HF_{pEF} patients for both cardiovascular (CV) and non-CV co-morbidities.
- Treatment strategies should focus on appropriate management of co-morbidities such as hypertension, DM, etc., alleviation of symptoms and improvement of overall well-being.
- Diuretics are recommended to alleviate symptoms and signs in congested patients.
- There is lack of evidence of any benefit with ACE inhibitors and ARBs. There is some evidence that beta-blockers and MRAs may have a beneficial effect on survival in this group of patients.
- For patients in sinus rhythm, there is some evidence that nebivolol, spironolactone and candesartan might reduce HF hospitalization.

- Combined endurance/resistance training appears safe in these patients and has been shown to improve exercise capacity, physical functioning score and diastolic function.

6.4.3. Patients with HF_{mrEF}⁸

- There is no specific management protocol for HF_{mrEF}. Hence, it is logical to treat these patients based on their etiology and presentation.
- Very often, these patients behave like those with HF_{rEF}.

Fig. 5 summarizes the management of HF patients, as was described above.

6.5. Risk scores for assessing prognosis in HF^{8,62,63}

Risk scores are used for predicting mortality and hospitalization and, hence, can guide the interventions used for HF management. Thus, low-risk patients may be able to receive lower intensity monitoring and vice versa.

Of all the different risk scores, the panel recommends the use of **The Meta-Analysis Global Group in Chronic Heart Failure (MAGGIC) Risk Calculator**, which calculates 3-year mortality risk. The variables included in the risk score are as follows: age, gender, body mass index (BMI), New York Heart Association (NYHA) class, systolic BP, smoking, DM, left ventricular ejection fraction (LVEF), left bundle branch block (LBBB), use of RAAS blockers, beta-blocker use, chronic obstructive pulmonary disorder (COPD), and HF diagnosed >18 months ago.

Another simple risk score that can be considered is the **SHIFT Prognostic Model**, which is based on simple clinical

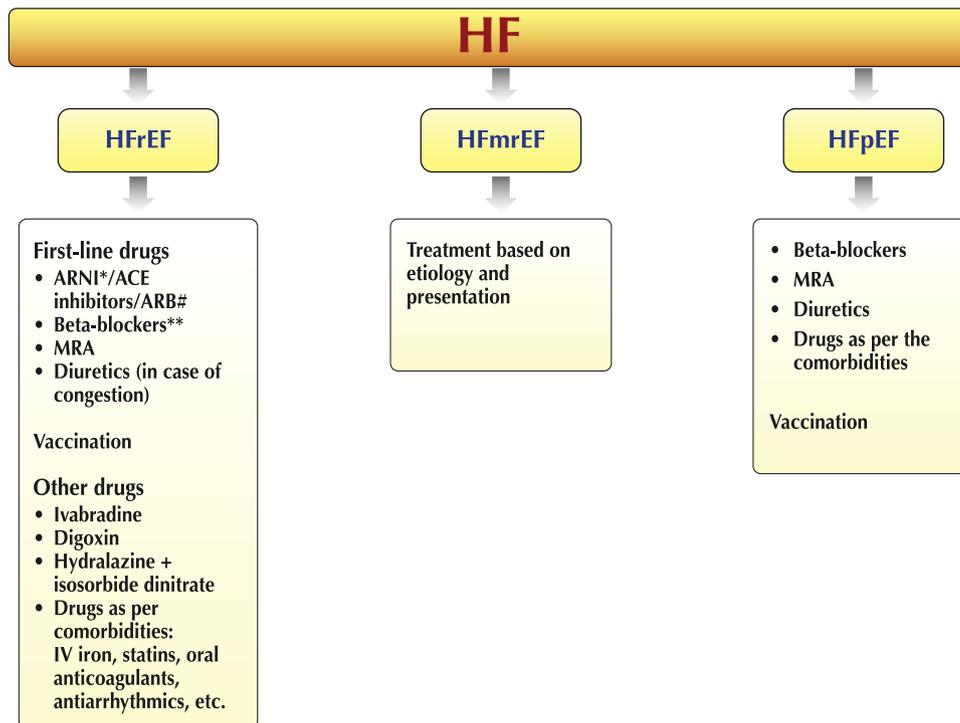


Fig. 5. Management of patients with HF.

*If affordability is not an issue, then ARNI can be used over RAAS inhibitors.

ARB should be used when ACE inhibitor is not tolerated

** Beta-blocker should be titrated to its maximum tolerated dose to obtain heart rate <70 bpm. If the beta-blocker is not tolerated or contraindicated, then add or replace it with ivabradine.

ARNI = angiotensin II receptor blocker neprilysin inhibitor; MRA = mineralocorticoid receptor antagonist; ACE = angiotensin converting enzyme, ARB = angiotensin II receptor blocker

characteristics (increased resting heart rate, low EF, raised creatinine, NYHA Class III/IV, duration of HF, history of LBBB, low systolic BP, age, AF, and use of ivabradine) and predicts CV death and/or hospitalization for the next 2 years.

Both MAGGIC and SHIFT scores are readily available online and can be used to predict prognosis in a HF patient.

6.6. Advanced treatment

6.6.1. Device therapy^{8,9}

Many patients with HFrEF would need device therapy for managing HF, either due to the severity of their condition or due to the presence of co-morbid conditions (Fig. 6).

6.6.2. Implantable cardioverter Defibrillator^{8,9,12}

The risk for ventricular arrhythmias and sudden cardiac death (SCD) is high in patients with HFrEF. Implantable cardioverter defibrillators (ICD) prevent bradycardia and correct potentially lethal ventricular arrhythmias. Box 14 lists the indications and contraindications for ICD use in patients with HF.

Benefits with ICD are not certain in patients who are at a high risk of non-sudden death, as predicted by advanced frailty, frequent hospitalizations, or the presence of co-morbidities such as severe renal impairment or systemic malignancy. In patients at risk of SCD for a limited period, a wearable ICD can be considered. It can also be used as a bridge to an implanted device.

6.6.3. Cardiac resynchronization therapy^{8,9}

Half of HFrEF patients develop conduction-related issues, which can result in electrical dys-synchrony and compromised ventricular efficiency. Treatment with cardiac resynchronization therapy (CRT) restores electrical and mechanical co-ordination, reduces hospitalization and mortality, and improves exercise capacity, LV structure and function, and QoL. There are two kinds of CRT devices:

- 1) CRT pacemaker (CRT-P), also known as biventricular pacing, is a special kind of pacemaker.
- 2) CRT defibrillator (CRT-D) is the same device as a CRT-P but it also includes a built-in ICD.

Indications and contraindications for the use of CRT are listed in Box 15.

If symptomatic relief is the main objective of CRT, then either of the two types of CRT, i.e., CRT-P or CRT-D, can be chosen. If improvement in prognosis is the main reason, then CRT-D should be preferred in patients with NYHA Class II and CRT-P should be preferred in patients in NYHA Class III–IV. If a patient is scheduled to receive an ICD and is in sinus rhythm with a QRS duration ≥ 130 ms, CRT-D should be considered if the QRS interval is between 130 and 149 ms and is recommended if QRS is ≥ 150 ms.

6.7. Revascularization^{8,9,64}

As IHD is a common underlying risk factor for HF, diagnosis of IHD is critical for the proper management of HF. It has been observed that CAD is usually missed in patients with DM; hence, proper diagnostic tests should be considered in patients with DM.

All HF patients with significant CAD and reversible ischemia (detected by PET scan, or dobutamine stress thallium) should be considered for revascularization. However, even in patients with significant CAD (despite optimal medical therapy) and with unknown reversible ischemia, revascularization was found to be beneficial and should be considered. The decision between percutaneous coronary intervention (PCI) and coronary artery bypass graft (CABG) should be based on the following: 1) co-morbidities, 2) coronary anatomy 3) expected completeness of revascularization, and 4) patient's clinical status. Correct revascularization strategies play an important role in prolonging life, reducing hospitalization, and improving the QoL.

6.8. Follow-up

A regular follow-up can help review the prognosis, and assess the patient's adherence to non-pharmacological and pharmacological therapy.⁸ It also helps decide the future course of the treatment, i.e., need for titration of current medications or addition of new drugs or need for devices or revascularization. Box 16 provides some key questions that one should ask the patient during follow-up visits.

The preferred targets that one should aim to achieve while managing Indian HF patients are given in Table 6.

6.9. Management of patients with advanced HF^{8,9,65,66}

Advanced HF, also known as end-stage HF or refractory HF, includes HF patients who continue to progress and develop

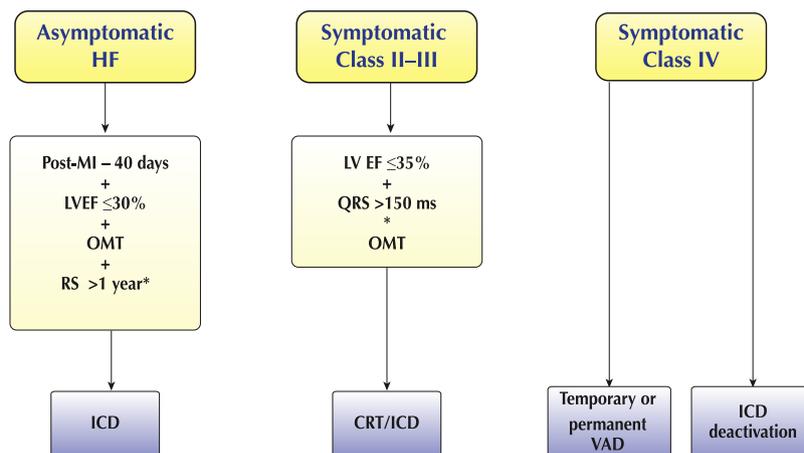


Fig. 6. Device therapy in HF.

*RS = Residual Survival ≥ 1 year

HF = heart failure; MI = myocardial infarction, LVEF = left ventricular ejection fraction, OMT = optimal medical therapy; ICD = implantable cardioverter defibrillators; CRT = cardiac resynchronization therapy; VAD = ventricular assist device

Box 14. Indications and contraindications for use of ICD in HF*Indications*

1) For primary prevention of SCD in order to reduce mortality in the following:

- Patients with IHD at least 40 days post-MI or non-ischemic DCM, with NYHA Class II or III and LVEF $\leq 35\%$ on chronic guideline-directed medical therapy and who have reasonable expectation of meaningful survival for >1 year
- Patients at least 40 days post-MI with NYHA class I HF and LVEF $\leq 30\%$ on chronic guideline-directed medical therapy and who have reasonable expectation of meaningful survival for >1 year.

2) For secondary prevention in patients who have recovered from ventricular arrhythmia causing hemodynamic instability and who are expected to survive for >1 year with good functional status.

Contraindications

1. Within 40 days of MI.
2. In NYHA Class IV patients with severe symptoms refractory to pharmacological therapy.

SCD = sudden cardiac death; IHD = ischemic heart disease; MI = myocardial infarction; DCM = dilated cardiomyopathy; NYHA = New York Heart Association; LVEF = left ventricular ejection fraction; HF = heart failure

Box 15. Indications and contraindications for the use of CRT*Indications*

1) Symptomatic patients with HF in sinus rhythm with a QRS duration ≥ 150 msec and with LVEF $\leq 35\%$ despite optimal medical therapy.

- With LBBB QRS morphology (strongly recommended)
- Without LBBB QRS morphology (should be used)

2) Symptomatic patients with HF in sinus rhythm with QRS duration of 130–149 msec and with LVEF $\leq 35\%$ despite optimal medical therapy.

- With LBBB QRS morphology (should be used)
- Without LBBB QRS morphology (may be used)

3) Patients with HFrEF regardless of NYHA Class who have an indication for ventricular pacing and high-degree AV block (includes patients with AF).

4) Patients with LVEF $\leq 35\%$ in NYHA Class III–IV despite optimal medical therapy, if they are in AF and have a QRS duration ≥ 130 msec, provided a strategy to ensure biventricular capture is in place or the patient is expected to return to sinus rhythm.

5) Treatment can be upgraded to CRT in patients with HFrEF who have received a conventional pacemaker or an ICD and subsequently develop worsening HF despite optimal medical therapy and who have a high proportion of RV pacing (this does not apply to patients with stable HF).

Contraindications

1. Patients with a QRS duration <130 msec.
2. Patients whose co-morbidities and/or frailty limit survival with good functional capacity to <1 year.

HF = heart failure; LVEF = left ventricular ejection fraction; LBBB = left bundle branch block; HFrEF = heart failure with reduced ejection fraction; NYHA = New York Heart Association; AV = atrioventricular; AF = atrial fibrillation; CRT = cardiac resynchronization therapy; ICD = implantable cardioverter defibrillators; RV = right ventricular

Box 16. Key questions to ask the HF patient during follow-up

1. Do you remember the signs and symptoms indicating worsening of HF and what to do if you experience these signs or symptoms?
2. Has your QoL improved after taking the medications?/Do you still experience any problem while performing daily activities?
3. Were you able to take all doses of all the medications, every day?
4. How well were you able to incorporate the instructions related to lifestyle modification in your daily life?
5. Do you have any questions or concerns?

QoL: quality of life

Table 6
Targets in Indian HF patients.

Parameter	Target to achieve
Heart rate	<70 bpm
BP	<130/80 mmHg
HbA1c	<7%
Hb	>12 gm
BMI	<25
6-min walking distance	>400 m

BP = blood pressure; HbA1c = glycated hemoglobin; Hb = hemoglobin; BMI = body mass index

persistent severe symptoms despite receiving optimal medical therapy. The most important first step for the management of these patients is to thoroughly evaluate and ascertain that the diagnosis of advanced HF is correct. It should be confirmed that there are no treatable etiologies or alternative explanations (including non-adherence to medications, sodium restriction, and/or daily weight monitoring) for the advanced symptoms. Key clinical events or findings that can help in the identification of patients with advanced HF are listed in [Box 17](#).

These patients need advanced and special treatment strategies, such as mechanical circulatory support (MCS), cardiac transplantation, or end-of-life care such as a hospice.

Heart transplantation improves survival, exercise capacity and QoL, when used in appropriate candidates. However, it is associated with challenges such as shortage of donor hearts and complications of immunosuppressive therapy in the long-term. Recent data suggests that 2- to 3-year survival rate in carefully selected patients with latest continuous flow devices is comparable to heart transplantation. Hence, these devices can be used as an alternative to heart transplantation, either in case of long-term wait for transplant or in patients who are ineligible for transplantation. However, it should be noted that long-term data is not available with left ventricular assist devices (LVADs). Key clinical points for managing a patient with advanced HF are listed in [Box 18](#).

7. Managing co-morbidities

HF is a multimorbid condition.⁶⁷ Co-morbid conditions may interfere with the optimal treatment of HF and, alternatively, pharmacological treatment for HF may impact co-morbidities.^{8,9} Moreover, the presence of co-morbidities in HF is associated with a poor prognosis.^{8,9} The key points that need to be borne in mind are enumerated below.

7.1. Diabetes^{8,67,68}

There is a bi-directional relationship between HF and DM, and half of chronic HF patients have DM.

- Metformin is an effective antihyperglycemic agent and can be safely used in stable HF patients with DM, unless contraindicated.
- Sodium-glucose co-transporter 2 (SGLT-2) inhibitors (empagliflozin and canagliflozin) have been shown to reduce CV mortality or HF hospitalization in DM patients at high CV risk. They are the only oral antihyperglycemic drugs that have shown benefit in terms of CV event reduction.
- Of all approved glucagon-like peptide 1 receptor agonists (GLP-1 RA), only liraglutide has shown reduction in mortality and a trend towards reduction in HF hospitalization in DM patients at high CV risk.
- Dipeptidylpeptidase-4 (DPP4) inhibitors or gliptins have shown variable effect on the risk of HF hospitalization (sitagliptin—no increased risk; vildagliptin—no increased risk [only gliptin with a dedicated trial in DM patients with HF Class I–III]; saxagliptin and alogliptin—increase risk; linagliptin—not evaluated). It is recommended to consider the risk–benefit ratio when using these drugs in HF patients with DM. In fact, a recent network meta-analysis suggests that vildagliptin may be the least harmful DPP-4 inhibitor with regards to risk of HF.
- Sulfonylurea derivatives should be used with caution as they are associated with increased risk of worsening HF.
- Thiazolidinediones can cause fluid retention. They are contraindicated in NYHA Class III–IV HF patients.
- Insulin leads to powerful sodium retention, and when combined with its effect of reduction in glycosuria, it may exacerbate fluid retention. Hence, insulin can worsen HF, especially when combined with thiazolidinediones.

[Box 19](#) categorizes the available antidiabetic drugs on the basis of their efficacy and safety in HF patients with DM.

7.2. Hypertension^{8,34,69}

It is estimated that ~1 in 3 or 4 adults who have BP > 160 mmHg develop HF.

- If BP is not controlled with an ACE inhibitor/ARB, a beta-blocker, an MRA and a diuretic, then hydralazine and amlodipine/felodipine can be added.

Box 17. Clinical events for the identification of advanced HF

- Two or more hospitalizations or visits to the emergency department for HF in the past year
- Progressive deterioration in renal function
- Weight loss that can't be attributed to other cause/s
- Intolerance to ACE inhibitors because of hypotension and/or worsening renal function
- Intolerance to beta-blockers because of worsening HF or hypotension
- Frequent systolic BP <90 mmHg
- Persistent dyspnea; activities such as dressing or bathing require rest
- 6-min walk distance <300 m
- Recent need to escalate diuretics to maintain volume status, often reaching daily furosemide equivalent dose >160 mg/d and/or use of supplemental metolazone therapy
- Progressive decline in serum sodium, usually to <133 mEq/L
- Frequent ICD shocks

HF = heart failure; ACE = angiotensin converting enzyme; BP = blood pressure; ICD = implantable cardioverter defibrillator

Box 18. Key clinical pointers in the management of patients with advanced HF

- 1) Restrict fluid to 1.5–2 L/day, especially in patients with hyponatremia.
 - 2) Provide IV inotropic support in the following ways:
 - a As ‘temporary use’ in patients with cardiogenic shock until definitive therapy or resolution of the acute precipitating problem is provided.
 - b As ‘bridge therapy’ in patients who are refractory to guideline-directed medical therapy and device therapy and who are eligible for and awaiting MCS or cardiac transplantation.
 - c Short-term use may be reasonable in hospitalized patients who have documented severe systolic dysfunction and who present with low BP and significantly depressed cardiac output.
 - d Long-term use may be considered as palliative therapy in select advanced HF patients already receiving optimal medical therapy and device therapy, who are not eligible for either MCS or cardiac transplantation.
 - 3) Nondurable MCS, including the use of percutaneous and extracorporeal VADs, is reasonable as a ‘bridge to recovery’ or ‘bridge to decision’ for carefully selected patients with HFrEF with acute, profound hemodynamic compromise.
 - 4) A LVAD should be considered in the following scenarios:
 - a Patients who have advanced HFrEF despite optimal medical and device therapy and who are eligible for heart transplantation.
 - b Patients who have end-stage HFrEF despite optimal medical and device therapy and who are not eligible for heart transplantation.
 - c Patients with >2 months of severe symptoms despite optimal medical and device therapy and more than one of the following: LVEF <25% and, if measured, peak VO_2 <12 mL/kg/min; ≥ 3 HF hospitalizations in previous 12 months without an obvious precipitating cause; dependence on IV inotropic therapy; progressive end-organ dysfunction (worsening renal and/or hepatic function) due to reduced perfusion and not due to inadequate ventricular filling pressure (PCWP ≥ 20 mmHg and systolic BP ≤ 80 –90 mmHg or CI ≤ 2 L/min/m²); absence of severe RV dysfunction together with severe tricuspid regurgitation.
 - 5) Patients to be considered for heart transplantation: end-stage HF with severe symptoms, a poor prognosis, and no remaining alternative treatment options; motivated, well informed, and emotionally stable; capable of complying with the intensive treatment required post-operatively.
 - 6) Patients with contraindication for heart transplantation: cancer (decision should be made in consultation with an oncologist); active infection; pharmacologically irreversible pulmonary hypertension (LVAD should be considered and then the candidate should be reevaluated); irreversible renal impairment; severe PAD or cerebrovascular disease; serious co-morbidities with poor prognosis; systemic disease with multi-organ involvement; current alcohol or drug abuse; BMI >35 kg/m² before transplant (recommended to achieve BMI <35 kg/m²); patient who cannot achieve compliant care in outpatient setting.
 - 7) Ultrafiltration should be used in patients with refractory congestion, who do not respond to diuretics. It can also be considered in elderly HF patients as these patients usually have co-morbid kidney disease and volume overload.
- IV = intravenous; MCS = mechanical circulatory support; BP = blood pressure; HF = heart failure; VAD = ventricular assist device; HFrEF = heart failure with reduced ejection fraction; LVAD = left ventricular assist device; LVEF = left ventricular ejection fraction; LVAD = left ventricular assist device; PCWP = pulmonary capillary wedge pressure; CI = cardiac index; RV = right ventricular; PAD = peripheral arterial disease; BMI = body mass index

- In HFpEF, ACE inhibitors, ARBs, and MRAs are preferred. CCBs, beta-blockers and alpha-blockers have limited data in this patient group.
- HFrEF patients with hypertension and HFpEF patients with persistent hypertension after management of volume overload should be treated to achieve systolic BP <130 mmHg.

Box 19. Recommendations for antidiabetic drugs in HF patients with DM*Preferred*

- Metformin (unless contraindicated)
- SGLT-2 inhibitors—empagliflozin, canagliflozin or dapagliflozin

Safe

- Liraglutide
- Vildagliptin/sitagliptin
- Basal insulin

Potentially harmful

- Sulfonylurea derivatives
- Insulin

Harmful

- Thiazolidinediones

SGLT-2 = Sodium-glucose co-transporter 2

- Diltiazem and verapamil should not be used in HFrEF but might be safe in HFpEF.
- Moxonidine should be avoided in HFrEF.

Recommendations regarding the use of antihypertensive agents in HF with hypertension are summarized in [Box 20](#).

7.3. Coronary artery disease^{8,70}

Among the various predisposing conditions, CAD (along with hypertension) exhibits the strongest association for increase in the risk of developing HF.

- Beta-blockers and ivabradine are effective agents for the management of angina in HF patients.
- Trimetazidine, in addition to beta-blockers, has been shown to be beneficial in HFrEF patients with angina.
- Amlodipine, nicorandil and nitrates have been shown to be safe in HFrEF.
- Diltiazem and verapamil are unsafe in HFrEF but can be considered for HFpEF.

Recommendations regarding the use of drugs in HF patients with CAD are summarized in [Box 21](#).

7.4. Atrial fibrillation⁸

AF, the most common arrhythmia in HF, not only impairs cardiac function but also increases the risk of thromboembolic complications (particularly stroke) and contributes to worsening of HF.

For rate control:

- Beta-blockers, digoxin and their combination may be used to control ventricular rate.
- Persistently high ventricular rates may indicate thyrotoxicosis or excessive sympathetic activity due to congestion, which might respond to diuresis.
- Nondihydropyridine CCBs should be avoided.
- A consultation from an electrophysiologist should be considered to arrive at a best strategy.

For rhythm control:

- This strategy is probably a better option for patients with a reversible secondary cause of AF (e.g., hyperthyroidism) or an obvious precipitant (e.g., recent pneumonia) and in patients with troublesome symptoms due to AF after optimization of rate control and HF therapy.
- Class I antiarrhythmic agents and dronedarone increase morbidity and mortality and should be avoided.
- Amiodarone can help some chronic AF patients to revert to sinus rhythm, may reduce symptomatic paroxysms of AF, and can help maintain patients with HF in sinus rhythm after spontaneous or electrical cardioversion. Its continued use should be reviewed regularly.

Thromboembolism prophylaxis:

- Patients with CHA₂DS₂-VASc score ≥ 2 should receive an oral anticoagulant.
- NOACs should be preferred for patients with non-valvular AF.
- For patients who have mechanical heart valves or at least moderate mitral stenosis, only oral vitamin K antagonists should be used for the prevention of thromboembolic stroke.

7.5. Electrolyte dysfunction^{8,71–73}

Electrolyte dysfunction, especially hypokalemia, hyperkalemia and abnormal serum sodium levels, is common in patients with HF. It is not only the disease that contributes to serum potassium level abnormalities but also the drugs that are used for the management of HF (loop diuretics and thiazide diuretics: reduce serum potassium; ACE inhibitors, ARBs and MRAs: increase serum potassium).

[Box 22](#) lists the key points to consider when managing HF patients with electrolyte dysfunction.

7.6. Other common co-morbidities

Other common co-morbidities in HF patients include iron deficiency (serum ferritin $<100 \mu\text{g/L}$ or ferritin between 100 and $299 \mu\text{g/L}$ and transferrin saturation $<20\%$) and anemia, CKD (eGFR

Box 20. Recommendations for antihypertensive agents in HF patients with hypertension

Beneficial

- ACE inhibitors/ARBs
- Beta-blockers
- MRAs
- Diuretics
- Hydralazine
- Spironolactone

Safe

- Dihydropyridine CCBs

Potentially harmful

- Alpha-blockers
- Moxonidine
- Non-dihydropyridine CCBs (might be safe in HFpEF)

ACE = angiotensin converting enzyme; ARB = angiotensin II receptor blocker; MRA = mineralocorticoid receptor antagonist; CCB = calcium channel blocker; HFpEF = heart failure with preserved ejection fraction

Box 21. Recommendations for the use of drugs in HF patients with CAD*Preferred*

- Beta-blockers
- Ivabradine
- Trimetazidine

Safe

- Amlodipine
- Nicorandil
- Nitrates

Potentially harmful (in HFrEF)

- Diltiazem
- Verapamil

HFrEF = heart failure with reduced ejection fraction

Box 22. Key considerations when managing HF patients with electrolyte imbalance.*Hypokalemia*

- Treat using foods with high potassium content or potassium supplements.
- Potassium-sparing diuretics (amiloride and triamterene) can be used as adjunct diuretics in case of resistant edema.

Hyperkalemia

- More common in patients with co-morbid DM and/or renal impairment.
- Management of acute hyperkalemia:
 - Short-term cessation of potassium-retaining agents and RAAS inhibitors may be needed. However, as RAAS inhibitors are important to improve prognosis in chronic HF, they should be reintroduced as soon as possible with continuous monitoring of serum potassium levels.
 - Use of aerosolized beta₂-agonists and IV insulin can be considered.
 - IV calcium can be used in patients with electrocardiographic changes and/or potassium levels >7.0 mmol/L
- Loop diuretics are effective in the management of intermediate hyperkalemia.
- Management of chronic hyperkalemia
 - Dietary potassium restriction and use of diuretics can be considered.
 - All dietary and herbal supplements and salt substitutes should be reviewed.
 - If needed, the dosage of drugs that impair potassium excretion can be lowered or they can be administered every other day or discontinued.
- SPS can manage hyperkalemia, but its chronic use can have detrimental hemodynamic effects.
- Dialysis can be considered in patients with renal impairment or in those not responding to other treatments.
- In patients with CKD and metabolic acidosis, use of sodium bicarbonate can be considered.

Abnormal serum sodium levels

- Most common electrolyte abnormality encountered in hospitalized HF.
- Mostly hypotonic hyponatremia (plasma osmolality <280 mOsm/kg H₂O).
- Treatment depends on: (i) acute hyponatremia or chronic; (ii) severity of hyponatremia; and (iii) neurological status.
- Major treatment
 - Fluid restriction <800–1000 mL/day is necessary, in order to achieve a negative water balance.
 - Use of loop diuretics is recommended as it induces water diuresis (salt-free water excretion) in patients who are fluid-overloaded.
 - Hypertonic saline (3% NaCl) is the mainstay in acute symptomatic hyponatremia (concerns: volume expansion and neurological side-effects associated with rapid correction).
 - Hypertonic saline + loop diuretic therapy may also be useful in selected patients.
 - Vasopressin receptor antagonists or vaptans are indicated in euvolemic or hypervolemic hyponatremia and not indicated in hypovolemic hyponatremia. They are also not useful if serum creatinine >2.5 mg%. Vaptan therapy is recommended if the Na levels are below 125 mmol/L (tolvaptan—start at 15 mg on day 1, and titrate to 30 and 60 mg at 24-h intervals. Concerns: rapid correction, liver toxicity).
 - Ultrafiltration has also been tried in patients with persistent hyponatremia.
 - In case of diuretic-induced hyponatremia, diuretics should be discontinued at the earliest and careful correction of volume deficits may be followed by a rapid water diuresis. Besides, 6- to 8-hourly monitoring of Na levels is recommended.

DM = diabetes mellitus; RAAS = renin angiotensin aldosterone system; HF = heart failure; IV = intravenous; SPS = sodium polystyrene sulfonate; CKD = chronic kidney disease; NaCl = sodium chloride

Box 23. Key considerations in HF patients with iron deficiency/anemia

- Patients should be screened for potentially treatable/reversible causes such as gastrointestinal sources of bleeding.
- Treatment with IV ferric carboxymaltose is effective in HFrEF patients with iron deficiency.
- In case of anemia, evaluate and rectify the cause, e.g., occult blood loss, iron deficiency, B₁₂/folate deficiency, blood dyscrasias, etc.
- ESA have been shown to increase hemoglobin levels. Darbepoetin-alfa, however, is not recommended in HFrEF as it may increase thromboembolic events.

IV = intravenous; HFrEF = heart failure with reduced ejection fraction; ESA = erythropoiesis-stimulating agents

<60 mL/min/1.73 m² and/or presence of albuminuria, i.e., high 30–300 or very high >300 mg albumin/1 g of urine creatinine) and COPD.^{8,9} Key considerations regarding the management of these patients are outlined in [Box 23–25](#).^{8,67,74}

7.7. Special population: Elderly patients with HF^{8,9,75,76}

- These patients might differ in terms of causes of HF, comorbidities, pathophysiology of their symptoms, and prognostic determinants.
- They are under-represented in randomized controlled clinical trials.
- Special issues in pharmacotherapy that should be considered are mentioned below:
 - Changes in drug-handling capacity—lean body mass/total fat/water, etc.
 - More side-effects—renal dysfunction, orthostatic hypotension, and bradycardia
 - Co-polypharmacy may worsen HF or increase the risk of drug–drug interactions
 - Social issues, caregiver-related, financial and cognitive impairment—access and adherence to HF therapy
- The benefits of recommended HF medications, such as neurohormonal antagonists, may be similar in the elderly and in younger patients.
- Diuretics should be used judiciously. Monitor electrolytes and orthostatic BP regularly.
- Utility of advanced therapy, such as ICD, CRT and revascularization procedures is doubtful, especially in the elderly with expected shorter life expectancy.
- For elderly ineligible for transplant, LVADs as destination therapy can be a potential option.

Box 24. Key considerations in HF patients with CKD

- Renal function can worsen during initiation and up-titration of RAAS inhibitors. These drugs can cause a small decrease in GFR but this should not lead to discontinuation of therapy. However, if there is a large increase in serum creatinine, the patient should be evaluated thoroughly and assessed for possible renal artery stenosis, excessive hyper- or hypovolemia, concomitant medication and hyperkalemia.
- Men with HF and renal impairment should be screened for prostatic obstruction as it is common in older men and can interfere with renal function.
- Diuretics (especially thiazides) may not be of much benefit in patients with very low GFR. However, if used, appropriate doses should be considered. Loop diuretics and metolazone can be considered.
- Drugs that are excreted via the kidneys, e.g., digoxin, insulin and low molecular weight-heparin should be monitored. If needed, the dose of these drugs should be adjusted.
- Alpha-adrenoceptor blockers may not be safe in HFrEF and, hence, should be avoided.

RAAS = renin angiotensin aldosterone system; GFR = glomerular filtration rate; HF = heart failure; HFrEF = heart failure with reduced ejection fraction

Box 25. Key considerations in HF patients with lung disease

- Beta-blockers have shown to reduce mortality even in COPD. They are relatively contraindicated in asthma, and, if needed, should be used under close medical supervision.
- Cardioselective beta-blockers such as bisoprolol or nebivolol should be preferred in HF patients with lung disease. Carvedilol also appears to be safe.
- When used, beta-blockers should be used with caution; started with a low dose and monitored closely for signs of airway obstruction, i.e., wheezing, shortness of breath with lengthening of the expiration.
- In HF patients with COPD, adjunct use of ivabradine may be considered.
- ACE inhibitors and ARBs can reduce lung injury in COPD and prevent smooth muscle atrophy and improve respiratory muscle strength in HFrEF. ACE inhibitors are particularly beneficial in HFrEF with COPD.
- Inhaled corticosteroids should be preferred over oral corticosteroids as they do not cause sodium and water retention.

COPD = chronic obstructive pulmonary disease; HF = heart failure; ACE = angiotensin converting enzyme; ARB = angiotensin II receptor blocker; HFrEF = heart failure with reduced ejection fraction

NEED FOR THIS DOCUMENT AND WHAT DOES THIS DOCUMENT ADD?

With the burgeoning burden of HF^{6,11} and with an ever increasing clinical practice, clinicians need simple protocols and algorithms as a guide to optimize HF management. Guidelines being large documents are not user friendly, which limits their use. Besides, there are many guidelines already available to aid practice of the clinicians in the developed world but these guidelines may not necessarily fit the needs of clinicians in developing countries.^{77, 78} This document is an attempt to aid the Indian clinicians in managing HF more effectively. The authors of this document deliberately avoided large amounts of text and focused on providing vital information in a user-friendly format. Easy-to-follow algorithms and tables are the highlight of this document. These algorithms can be easily incorporated in a clinician's daily practice and can aid optimization of HF care in India.

Conflict of interest

The authors have none to declare.

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