

CHRONIC HEART FAILURE: PATHOPHYSIOLOGY, CLINICAL MANIFESTATIONS AND CONTEMPORARY MANAGEMENT

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Abstract

Chronic Heart Failure (CHF) is a complex clinical syndrome resulting from structural or functional impairment of ventricular filling or ejection of blood. It represents a leading cause of hospitalization and mortality worldwide. CHF is characterized by neurohormonal activation, reduced cardiac output, and systemic congestion. This paper examines the epidemiology, pathophysiology, classification, clinical presentation, diagnostic strategies, complications, and modern treatment approaches of chronic heart failure following the IMRAD structure.

Keywords: Chronic Heart Failure, CHF, Left Ventricular Dysfunction, Ejection Fraction, Cardiomyopathy, Neurohormonal Activation, ACE Inhibitors, Beta-blockers, Cardiac Remodeling, Congestive Heart Failure

Introduction

Chronic Heart Failure affects more than 64 million people globally. It is a progressive condition in which the heart is unable to pump sufficient blood to meet the metabolic demands of the body.

The most common causes include:

- Coronary artery disease
- Hypertension
- Cardiomyopathy
- Valvular heart disease
- Myocardial infarction

CHF is not a single disease but a clinical syndrome involving complex hemodynamic and neurohormonal mechanisms. It significantly reduces quality of life and is associated with high morbidity and mortality.

The aim of this study is to analyze the mechanisms of disease progression, clinical features, diagnostic criteria, and current evidence-based management strategies for chronic heart failure.

Methods

This study is based on analysis of international cardiology guidelines (ESC, AHA/ACC), randomized controlled trials, and meta-analyses published between 2015–2024.

Inclusion criteria:

- Adults ≥ 18 years
- Diagnosed chronic heart failure
- Studies with ≥ 500 participants

Exclusion criteria:

- Acute heart failure
- Congenital heart disease
- Pediatric cardiology cases

Key parameters analyzed:

- Left Ventricular Ejection Fraction (LVEF)
- Hospitalization rates
- Mortality outcomes
- Response to pharmacological therapy

Results

1. Classification of Heart Failure

CHF is classified based on Left Ventricular Ejection Fraction (LVEF):

- HFrEF: LVEF $< 40\%$ (Reduced EF)
- HFmrEF: LVEF 41–49%
- HFpEF: LVEF $\geq 50\%$ (Preserved EF)

HFrEF accounts for approximately 50% of cases.

2. Pathophysiology

CHF progression involves multiple mechanisms:

Step 1: Myocardial Injury

Damage from ischemia or hypertension reduces contractility.

Step 2: Neurohormonal Activation

Activation of:

- Renin-Angiotensin-Aldosterone System (RAAS)
- Sympathetic Nervous System

Step 3: Cardiac Remodeling

Left ventricular dilation and hypertrophy develop.

Figure 1. Neurohormonal Activation in Heart Failure (Description)

The diagram illustrates activation of the RAAS system leading to vasoconstriction, sodium retention, increased afterload, and worsening ventricular dysfunction.

3. Clinical Manifestations

Left-sided Heart Failure Symptoms

- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Pulmonary edema

Right-sided Heart Failure Symptoms

- Peripheral edema
- Hepatomegaly
- Ascites
- Jugular venous distension

Fatigue and exercise intolerance are common in both types.

Figure 2. Fluid Accumulation in Congestive Heart Failure (Description)

The illustration demonstrates pulmonary congestion in left-sided failure and peripheral edema in right-sided failure due to increased venous pressure.

4. Diagnostic Evaluation

Key diagnostic tools include:

- Echocardiography (LVEF measurement)
- BNP or NT-proBNP levels
- Chest X-ray
- Electrocardiogram

- Cardiac MRI (advanced cases)
Elevated BNP levels correlate with severity of heart failure.

5. Complications

- Arrhythmias
- Sudden cardiac death
- Renal dysfunction (cardiorenal syndrome)
- Thromboembolism

6. Treatment Strategies

Lifestyle Management

- Sodium restriction
- Fluid management
- Weight monitoring
- Physical rehabilitation

Pharmacological Therapy

1. ACE Inhibitors / ARBs
 - Reduce mortality
 - Decrease remodeling
2. Beta-blockers
 - Improve survival
 - Reduce sympathetic activation
3. Mineralocorticoid receptor antagonists
 - Reduce hospitalization
4. SGLT2 inhibitors
 - Reduce cardiovascular mortality
5. Diuretics
 - Symptom relief

Figure 3. Impact of Guideline-Directed Medical Therapy (Description)

A comparative chart shows reduction in mortality and hospitalization rates in patients receiving combined therapy (ACE inhibitor + beta-blocker + MRA + SGLT2 inhibitor).

7. Advanced Therapies

- Implantable cardioverter-defibrillator (ICD)
- Cardiac resynchronization therapy (CRT)
- Left ventricular assist devices (LVAD)
- Heart transplantation

Discussion

Chronic heart failure is driven by a vicious cycle of myocardial injury and neurohormonal activation. Without intervention, cardiac remodeling worsens ventricular dysfunction.

Modern guideline-directed medical therapy (GDMT) has significantly improved survival over the past two decades. Early initiation of combination therapy is associated with better outcomes compared to stepwise escalation.

Despite therapeutic advances, HFpEF remains challenging to treat due to its complex pathophysiology.

Prevention strategies should focus on:

- Blood pressure control
- Ischemic heart disease prevention
- Early detection of asymptomatic ventricular dysfunction

Conclusion

Chronic Heart Failure is a progressive internal disease with significant global impact. Early diagnosis, comprehensive pharmacological therapy, and device-based interventions significantly improve prognosis and quality of life. Continued research is necessary to optimize treatment for HFpEF and reduce global mortality rates.

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