

# AI-03522 AMI Long Term Management 2025

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Here are the **key findings** from the review on **Heart Failure with Preserved Ejection Fraction (HFpEF)**:

## Overview & Burden

- HFpEF accounts for **~50% of heart failure cases worldwide** and is increasing due to aging populations and rising comorbidities.
- Prevalence is likely underestimated; **35–75% of cases remain undiagnosed**.
- Prognosis remains poor:
  - ~15% annual mortality
  - Up to 50% mortality at 5 years
  - High rehospitalization rates

## Pathophysiology: A Multifactorial Syndrome

HFpEF is no longer viewed as simple diastolic dysfunction.

### Core mechanisms

- Passive myocardial stiffness & impaired relaxation
- Endothelial dysfunction & reduced nitric oxide signaling
- Systemic inflammation & metabolic dysfunction (“metaflammation”)
- RAAS activation and fibrosis

- Skeletal muscle & energetic abnormalities

## Role of comorbidities

Common drivers include:

- Hypertension
- Obesity
- Diabetes
- Chronic kidney disease
- Atrial fibrillation

## Phenotypes

HFpEF is heterogeneous; notable subtype:

- **Obesity-related HFpEF** (≈30–40% of patients)
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## Diagnostic Challenges

HFpEF is frequently missed because:

- Symptoms (dyspnea, fatigue) are nonspecific
- Natriuretic peptides may be normal, especially in obesity
- Resting echocardiography can be normal

## Diagnostic tools

- Echocardiography (E/e' ratio, LA enlargement, pulmonary pressures)
  - Exercise or invasive hemodynamics when uncertain
  - Scoring systems:
    - **H2FPEF** (higher sensitivity)
    - **HFA-PEFF**
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## Therapeutic Breakthroughs

## 1 SGLT2 Inhibitors — cornerstone therapy

Trials: EMPEROR-Preserved, DELIVER

### Benefits:

- ~20% reduction in CV death/HF hospitalization
- Fewer HF hospitalizations
- Slower kidney decline
- Improved quality of life

Guidelines now strongly recommend them.

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## 2 Finerenone (non-steroidal MRA) — new milestone

Trial: FINEARTS-HF

### Findings:

- 16% reduction in CV death & worsening HF
- Lower hospitalization rates
- Acceptable safety profile

Likely to be included in future guidelines.

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## 3 GLP-1 receptor agonists & tirzepatide (phenotype-targeted)

Especially beneficial in **obese HFpEF**:

- Weight reduction
- Improved symptoms & quality of life
- Reduced HF events

Represents a shift toward phenotype-specific therapy.

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## Therapies with Limited or Selective Benefit

### ARNI (sacubitril/valsartan)

- Mixed results; possible benefit in lower EF ranges.

## ACEi / ARBs

- Modest or neutral outcomes.

## Beta-blockers

- Used for comorbidities, not HFpEF itself.

## Steroidal MRAs (spironolactone)

- Reduced HF hospitalizations but overall neutral results.
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## Key Paradigm Shifts

✓ HFpEF is a **systemic cardio-metabolic inflammatory syndrome**, not just diastolic dysfunction.

✓ **Disease-modifying therapies now exist** (especially SGLT2 inhibitors & finerenone).

✓ HFpEF requires **phenotype-based, personalized treatment**.

✓ Diagnosis remains challenging and under-recognition delays therapy.

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## Future Directions

- Improved diagnostic strategies & earlier detection
  - Phenotype-specific treatments
  - Expanded use of metabolic & anti-inflammatory therapies
  - Integration of comorbidity management into HF care
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If you want, I can also:

- simplify into a **quick clinical cheat sheet**,
- relate findings to **your cardiac history**, or
- summarize **what actually changes patient outcomes**.

Here is a **Quick Clinical Cheat Sheet: HFpEF (2026 Update)**

Based on *Advances in the Pharmacological Treatment of HFpEF*

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# HFpEF QUICK REFERENCE

## 1 Definition

- Symptoms/signs of HF
  - **LVEF  $\geq 50\%$**  (HFmrEF = 41–49%)
  - Objective evidence of  $\uparrow$  filling pressures or diastolic dysfunction
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## 2 Epidemiology Snapshot

- $\approx 50\%$  of all HF cases
  - Increasing prevalence
  - Often underdiagnosed (up to 75%)
  - $\sim 15\%$  annual mortality
  - High rehospitalization rates
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## 3 Core Pathophysiology (Think: “Stiff + Inflamed + Metabolic”)

- ✓ Diastolic stiffness
- ✓ Impaired relaxation (calcium/energy dysfunction)
- ✓ Endothelial dysfunction
- ✓ Systemic inflammation
- ✓ RAAS activation
- ✓ Cardio-renal-metabolic syndrome

Common drivers:

- Hypertension
- Obesity
- Diabetes

- CKD
  - AF
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## **4** Diagnosis — Practical Approach

### **Step 1: Clinical suspicion**

- Dyspnea on exertion
- Exercise intolerance
- Volume overload signs (may be absent)

### **Step 2: Labs**

- BNP/NT-proBNP (may be normal in obesity)

### **Step 3: Echo**

Look for:

- $\uparrow$  E/e'
- LA enlargement
- $\uparrow$  PASP
- LV hypertrophy

### **Step 4 (if unclear):**

- Exercise testing
- Invasive hemodynamics

Helpful scores:

- **H2FPEF** (simpler, more sensitive)
  - **HFA-PEFF**
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# PHARMACOLOGIC MANAGEMENT (2026)

## First-Line Disease-Modifying Therapy

### SGLT2 Inhibitors (Cornerstone)

Examples:

- Empagliflozin
- Dapagliflozin

Evidence (EMPEROR-Preserved, DELIVER):

Benefits:

- ~20% ↓ CV death/HF hospitalization
- ↓ HF hospitalizations
- Slows renal decline
- Improves QoL

 Recommended in guidelines.


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## Finerenone (Non-steroidal MRA)

Trial: FINEARTS-HF

Benefits:

- 16% ↓ CV death/worsening HF
- ↓ HF hospitalizations
- Acceptable hyperkalemia risk

 Emerging second foundational therapy.

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## Phenotype-Specific Therapy (Obese HFpEF)

## GLP-1 receptor agonists / Tirzepatide

- Weight loss
- Improved symptoms
- ↓ HF events (mainly hospitalizations)

Best for:

- Obesity phenotype
- Metabolic HFpEF

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## Therapies with Limited or Selective Benefit

Drug Class	Status
ARNI (sacubitril/valsartan)	Possible benefit at lower EF
ARBs	May reduce hospitalization
ACEi	Neutral overall
Beta-blockers	Use for comorbidities only
Spironolactone	Mixed data (TOPCAT controversy)

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## Clinical Strategy (Modern HFpEF Model)

**1. Start SGLT2 inhibitor**

**2. Consider finerenone**

**3. Treat phenotype:**

- Obesity → GLP-1 RA
- AF → rhythm/rate control
- Hypertension → aggressive control

- CKD → optimize renal protection

#### 4. Manage congestion (loop diuretics PRN)

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## What Actually Changes Outcomes?

Strong outcome evidence:

- ✓ SGLT2 inhibitors
- ✓ Finerenone

Moderate/selective:

- ✓ ARNI (lower EF range)
  - ✓ GLP-1 RA (obese phenotype; hospitalization reduction)
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## Big Clinical Shift

Old model:

| "Diastolic dysfunction → give diuretics."

New model:

| "Systemic inflammatory cardio-renal-metabolic syndrome → use disease-modifying therapy + phenotype targeting."

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If you'd like, I can next create:

- A **one-page printable clinic card**
- A **treatment algorithm flowchart**
- Or a **high-yield board review summary**

The following still valid for 59 yom, AMI age 42 active cyclist and swimmer.

81mg Aspirin

20mg Lisinopril

40mg Atorvastatin

50mg Metoprolol (day 20 of deletion, no adverse effect post stopping)

Based on what you shared — **59-year-old male**, prior **myocardial infarction (age 42)**, currently active (cycling & swimming), and recently stopping metoprolol — here is a **practical, guideline-aligned review** of your medications for **secondary prevention of coronary artery disease**.

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## Secondary Prevention After MI — Medication Review

### 1. Aspirin 81 mg daily

**Status:**  Still recommended

**Why:**

- Lifelong antiplatelet therapy is standard after MI
- Reduces recurrent MI & stroke risk by ~20–25%

**Continue unless:**

- GI bleeding risk
- Allergy/intolerance
- Physician advises otherwise

 For most post-MI patients, this is lifelong therapy.

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### 2. Lisinopril 20 mg

**Status:**  Beneficial long-term

**Benefits:**

- Reduces cardiovascular mortality post-MI
- Prevents ventricular remodeling
- Protects kidneys

- Helps BP control
- Helpful even with preserved EF

**Especially useful if:**

- Hypertension
- Diabetes
- CKD
- LV dysfunction

👉 Often continued indefinitely.

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 **3. Atorvastatin 40 mg**

**Status:**  Essential

**Why:**


- Secondary prevention requires **high-intensity statin**
- Stabilizes plaque & reduces future events
- Reduces CV events by ~25–35%

**Targets (modern guidance):**

- LDL < 55 mg/dL (very high risk)
- <70 mg/dL minimum

👉 Dose adjustment depends on lipid panel results.

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 **4. Metoprolol 50 mg (Stopped 20 days ago)**

**Status:** Depends on clinical context

**Beta-blockers are clearly beneficial:**

- ✓ First 1–3 years after MI
- ✓ If reduced EF
- ✓ If angina present

- ✓ If arrhythmias present
- ✓ If hypertension uncontrolled

## Long-term use (>3 years post-MI):

Evidence suggests:

- ✓ May **not be necessary** if:
    - EF normal
    - No angina
    - No arrhythmias
    - Excellent exercise tolerance
  - ✓ May still help:
    - Heart rate control
    - BP control
    - AF prevention
    - Sympathetic tone reduction
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## Your situation suggests:

Since you report:

- No angina
- No dyspnea
- High endurance capacity
- No symptoms after stopping

👉 Many cardiologists consider discontinuation reasonable **if EF is normal**.

### However:

- ✓ Should be physician-guided
  - ✓ Monitor resting HR & BP
  - ✓ Watch for palpitations or exertional symptoms
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# What Modern Prevention Would Also Consider

Given prior MI and active lifestyle:

## ✓ Annual review:

- Lipid panel
- Kidney function
- A1c / glucose
- BP control
- Echocardiogram (periodic)

## ✓ Lifestyle (you are doing well):

- Aerobic exercise
  - Weight control
  - Anti-inflammatory diet
  - Sleep quality
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## When to Reconsider Beta-Blocker

Restart discussion if:

- Resting HR persistently >80–90
  - Palpitations or arrhythmias
  - New angina
  - Reduced EF
  - Hypertension rises
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## Summary

Medication	Status	Rationale
Aspirin 81 mg	✔ Continue	Lifelong secondary prevention
Lisinopril 20 mg	✔ Continue	CV & renal protection
Atorvastatin 40 mg	✔ Continue	Essential plaque stabilization
Metoprolol	⚖ Optional	If EF normal & asymptomatic

If you want, I can next:

- ✔ Review **ideal heart rate zones & safety for endurance cycling post-MI**
- ✔ Explain **what markers indicate coronary stability**
- ✔ Suggest **optimal labs & imaging schedule for long-term survivors**
- ✔ Evaluate whether **statin dose optimization** is appropriate

Just tell me.