Heart Failure: What Every PCP Must Know

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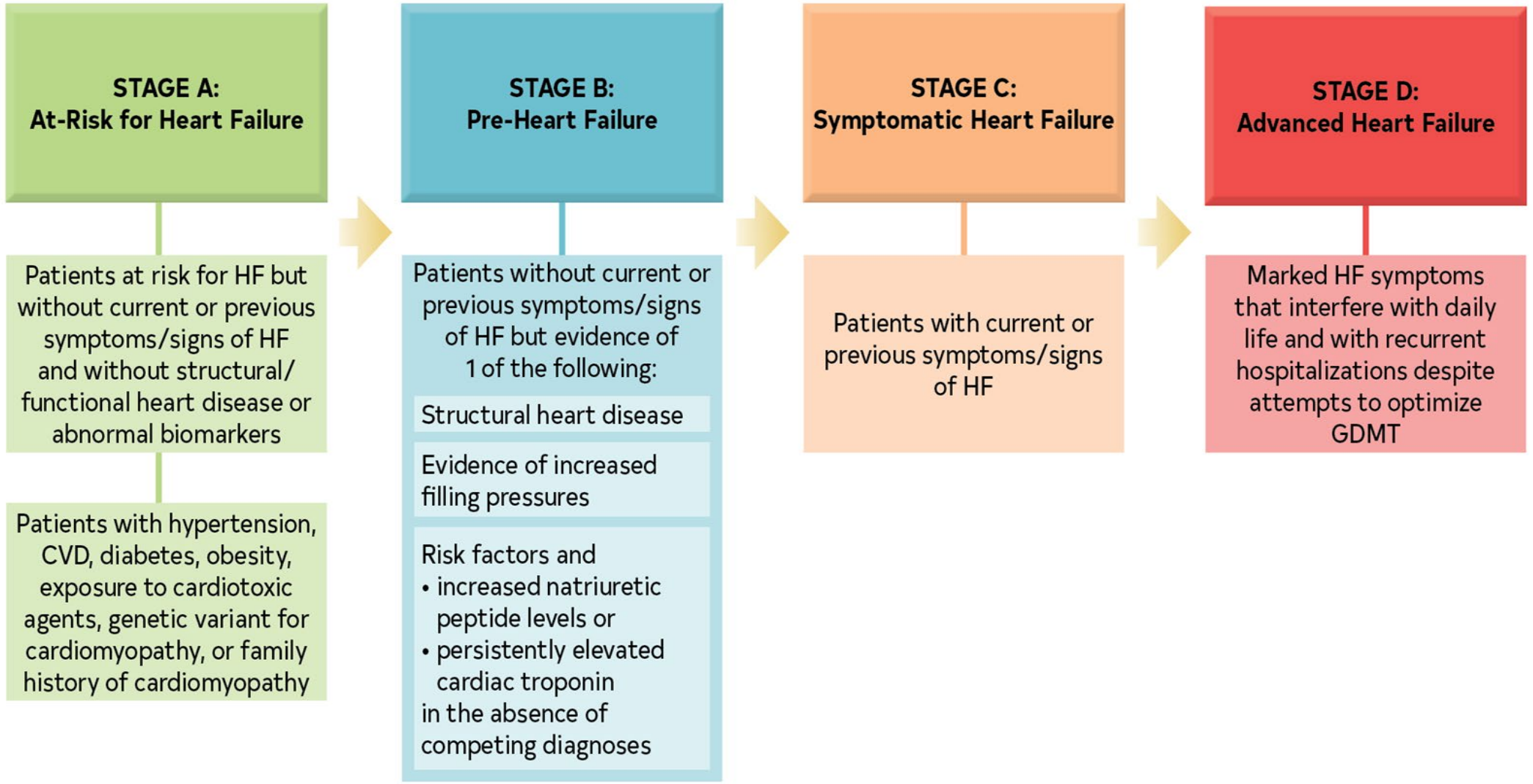
Learning Outcomes

- Recognize presentations of heart failure (HF)
- Explain diagnostic approach & first-line management
- Know when to refer

Why it Matters

- 6 million+ Americans with HF
- Leading cause of hospitalization >65 years
- 50% 5-year mortality
- Prevention, early diagnosis & GDMT improves survival

Heart Failure Stages: Prevention Opportunities



Clinical Vignette 1

- Mr. J, 72M with HTN and CAD
- Presents with fatigue, orthopnea, PND, LE edema
- Exam: BP 110/70, HR 85, rales, JVD elevated
- ❓ What is your initial approach?

Key Symptoms

- Breathlessness
- Orthopnea
- Paroxysmal nocturnal dyspnea

- Reduced exercise tolerance
- Fatigue

- Ankle swelling

Key Signs

- Elevated JVP
- Pulmonary rales
- S3 (HFrEF) or S4 (HFpEF)
- Hepatomegaly, ascites, LE edema

Diagnosis

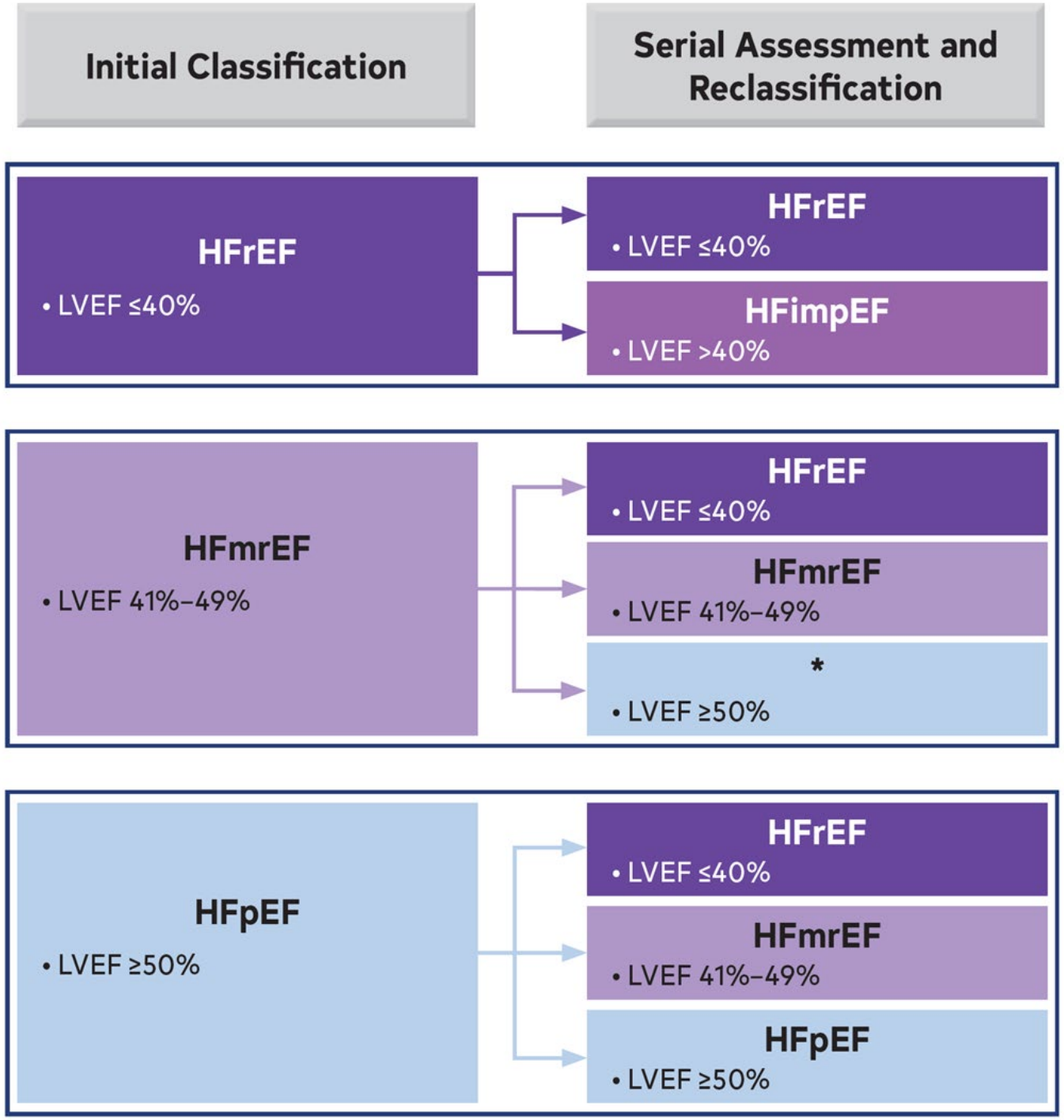
- Labs: BNP/NT-proBNP, CBC, CMP, TSH
- EKG: Arrhythmia, LVH, ischemic changes
- CXR: Cardiomegaly, pulmonary congestion
- Echo: Essential for EF, valvular disease, wall motion

Clinical Vignette 2

- Ms. L, 68F with obesity and DM
- CC: Dyspnea and leg swelling
- BNP 350, EKG NSR, Echo EF 55% with LV
- ❓ What type of HF and initial management?

➡️ HFpEF; manage volume, *control risk factors*, consider SGLT2i

Classification determines treatment strategy



Non-Pharmacologic Management

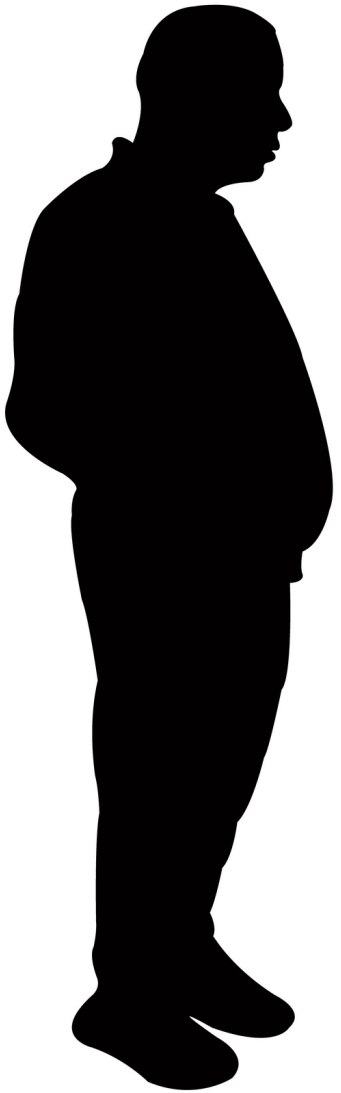
- Low sodium intake ~2g/day
- Daily weights: 3lb/day or 5lb/week gain = concerning
  - Weight monitoring = volume management
- Exercise as tolerated
  - Cardiac rehabilitation programs
  - Focus on functional training

HFrEF Pharmacologic Therapy

HFpEF Pharmacologic Therapy

- No mortality-proven therapy until recently
- SGLT2 inhibitors reduce hospitalizations & improve outcomes
- Volume management with diuretics
- Manage comorbidities: obesity, DM, OSA, CAD, AFib

Clinical Vignette 3



- Mr. P, 58M with EF 25%
- On max tolerated doses of ACEi/ARNi, BB, MRA, SGLT-2
- Still NYHA III symptoms despite diuresis
- ❓ Next step?

➡️ Refer for advanced therapies

When to Refer

- New onset heart failure to determine etiology and develop treatment plan
- Persistent NYHA III-IV symptoms despite therapy
- Consideration for ICD, CRT, advanced therapies

Prevention & Screening

- Early preventive strategy: Control HTN, DM, **obesity**, CAD, OSA
- Encourage general wellness
- Encourage vaccination: flu, COVID, pneumococcus
- Early identification + GDMT improves outcomes

Key Pearls

- Prevention at every stage is key
- BNP <100 pg/mL makes HF unlikely (unless BMI > 30)
- Start GDMT early in HFrEF
- HFpEF = comorbidity management + SGLT2i
- Refer to cardiac rehab
- Daily weights = volume management
- Refer new onset HF to identify cause and guide management

Thank You

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2025 Family Medicine POP: Questions & Answers

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1. When starting GDMT for heart failure, which of the 4 agents do you prioritize starting first?

There is no right or wrong on starting GDMT. The most important thing is to get the meds on board, so confidently start them with something! The ARNi and SGLT-2 seem to have better impact on patients' quality of life, so if they can tolerate I make sure they take these two. All agents have data for mortality benefit but what people want is to feel better. ARNIs and SGLT-2 seem to make patients feel better and reduce their mortality risk.

2. What about checking albumin especially with dm?

Symptoms of chf, 3rd spacing, can be similar with low albumin because of renal loss: There are many reasons to understand albumin levels. Patients are often referred to me because of lower extremity edema and concern for HF. HF is a specific syndrome (DOE, orthopnea, PND and the edema, +/_ elevated BNP). Edema alone does not mean HF. Albumin levels are helpful to understand the mechanism and pathophysiology for edema and to help address the actual cause of the problem. My differential for edema include 1) HF 2) renal disease 3) liver disease 4) Venus insufficiency —> Albumin helps understanding possible nephrotic range proteinuria vs liver failure vs cachexia. And just recently learned that albumin can help as marker for disease progression in my patient with ATTR Amyloid CM!

3. Which beta blockers are preferred for GDMT? And how do you know what dose to use?

GDMT BB include 1) Metoprolol SUCCINATE (not tartrate). 2) Carvedilol. 3) Bisoprolol. Start low and adjust as tolerated. Based on the clinical trial, the recommendation is to get them to the high dose e.g. Metoprolol succinate 100mg and Carvedilol 25mg PO BID (I hardly ever use Bisoprolol). In practice I aim for high dose, but it all depends on the patient's HR tolerance.

4. How frequently should echos be performed in a stable patient?

Routine echocardiogram assessment in stable patients is not indicated nor necessary. Echos should only be performed with changes in their clinical status.

5. Can you address if patients with advanced heart failure benefit from AICD?

Yes! Patients with LV EF < 35% on Max tolerated GDMT for at least 3 months who have not improved should get an AICD to reduce SCD risk. If they have a left bundle branch block with QRS duration of > 150 ms then they should get Bi-Ventricular pacing in addition to their ICD.

6. If patients are having side effects to SGLT2, is there evidence to support even lower doses or a few doses/week like we see with statins?

I don't have data to support lower doses BUT I do it anyway… What I have learned is that clinical trials do not include many of the patients we typically see in clinic. For example, an elderly frail female of small size may not tolerate the full dose, so I start her with half or alternating days… I have seen improvements in their functionality and they feel better even with smaller doses. Please note, this is anecdotal and I have not strategically followed outcomes on my patients. It is all my perception when I meet with them.

7. For HFpEF, can BB be used as well in addition to diuretics and SGLT2?

I prefer to avoid BB if at all possible in HFpEF. Most HFpEF patients are women, obese or overweight with DM. In these groups of patients BB can be detrimental for their metabolism which is often the root cause of their disease. If the medication is needed to treat their atrial fibrillation for example, or if they have hypertension that requires a third agent, then I may be forced to use it, but do my best to try everything else before adding it.

8. How long are you comfortable prescribing a patient with phentermine?

I do not have any patients on phentermine now. The hypertension and tachycardia side effects make it hard to use in most of my patients. Now with modern weight loss meds, phentermine is really reserved for those who have no other options.

9. What max dose do you normally prescribe for phentermine?

I haven't used phentermine for years, but in my previous use I start at 15 mg and watch response. If weight loss seen and normal HR and BP, then increase 30mg progressively with no more than 3 months of treatment. To really help the patients they need a good lifestyle intervention program to teach them to cut calories and have a plan for maintenance. Please note, I don't use phentermine anymore, with access to GLP-1s I much prefer that. With the upcoming changes to Medi-Cal will need to figure out how to access but GLP-1 are so much better for cardiac patients.

10. Are there any guidelines around MEMS devices for heart failure exacerbation detection?

There are. The device can be recommended in patients with recurrent hospitalization on max tolerated GDMT. Personally, I do not use. I consider MEMs for very sick patients, but for the bulk of my patients I find that educating them on signs and symptoms of early congestion: orthopnea, DOE, do not wait for edema! And weights every morning.

11. Can you discuss different NYHA treatment management for different ethnicities if you consider it before prescribing therapies?

This is the reason I spent 2 years studying CVD epidemiology :) Peoples' ethnic backgrounds do have an impact on the particular pathophysiology of disease but we don't have enough data or guidance from a therapeutic standpoint to address all CVD manifestations or for prevention. I trust we will move toward understanding this better in the future. There are some studies. For example, A-HeFT trial published in

2004 demonstrating benefit of Hydralazine + Isosorbide Dinatrate for treatment of HFrEF in Black men. The study showed a 43% all cause mortality reduction! So in HFrEF patients on GDMT who remain symptomatic I seek to add. In many instances when other GDMT is not accessible this is my go to for my Black patients.

12. Is ARNI usually well covered by insurance?

It WAS :(when Entresto had not become generic it was well covered. Now, I am having a very hard time getting it for patients. Entresto became available as Sucubitril/Valsartan and insurances are requesting the generic form, but when patients show up at the pharmacy their copayment is too high and cannot afford it. There was a PAP through Novartis but the program is now closed since the med is generic. Per Novartis representatives, I am being told that the high copayment has to do with the patients' specific insurance prescription plan. I am trying to learn what patients need to do to have more reasonable copayments for use. It will be interesting to see what the new year brings with the changes in coverage we are seeing.

Any medications or treatment methods suggested in this document should not be used by the practitioner without evaluation of their patient's condition(s) and possible contraindication(s) or danger(s) of use of any specific medication.