Summer 2020

Heart Failure with Reduced Ejection Fraction

Jennifer Klubnik
Otterbein University, klubnik1@otterbein.edu

Heart Failure with Reduced Ejection Fraction

Jennifer Klubnik, BSN, RN
Otterbein University, Westerville, Ohio

Introduction

Heart failure with reduced ejection fraction (HFrEF) is:
A chronic disease
On the rise among the population (will ↑ by 46% in the next 10 years)(Bowers, 2019)
Costs upwards of $70 billion annually (Murphy et al., 2019)
HFrEF leads to high morbidity and mortality as well as repeat hospitalizations for exacerbation of symptoms (Rhiantong et al., 2019). HFrEF is the focus of this poster, however, HF with preserved EF (HFpEF) will be mentioned to compare and contrast the severity of HFrEF. Differences in HFrEF and HFpEF lie in the pathophysiology, treatment, and outcomes. HFrEF is a disease that is complicated with treatment focused on keeping symptoms mitigated. However, there is constant developing research in understanding the pathophysiology and treatments for this and other types of HF affecting people around the world (Ge et al., 2019).

Stages of HF

HF is staged on a four-level progression of symptom and disease state. This A-D range developed by The American College of Cardiology and the American Heart Association defines the different stages of HF. The stage at which a person is in their HF disease progression dictates the type of intervention (Capriotti & Micari, 2019) This progression scale is often used in conjunction with the New York Heart Association classification of HF.

Table 1. Functional Classification Systems for Heart Failure

New York Heart Association classification

I: Asymptomatic
II: Minor symptoms, symptoms with modest exertion
III: Moderate symptoms, symptoms with minor exertion
IV: Symptoms at rest

American College of Cardiology/American Heart Association classification

A: At risk of heart failure but without structural disease
B: Structural heart failure but without symptoms
C: Structural heart failure with current or prior symptoms
D: Symptoms at rest

(Chavey et al., 2017, Table 1)

Clinical Manifestations

- Dyspnea
- Persistent cough
- Frothy or pink sputum
- Edema
- Jugular vein distention
- Low urine output
- Pulmonary edema
- ↑ or ↓ BP
- Potential S3 gallop on auscultation of heart

(McCance & Huether, 2018)

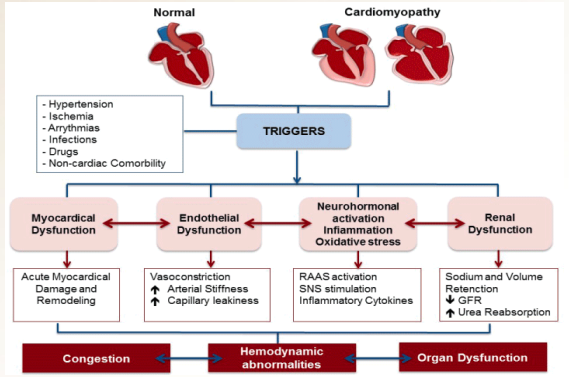
Pathophysiology

Cardiac cell dysfunction
↓
Renin-angiotensin aldosterone system (RAAS) ↑ preload
↓
Ventricular remodeling due to over activation of RAAS
(Wan et al., 2019)

Remodeling & Cellular Changes

- Change in the formation of the heart
- Response to an insult to the myocardium
- Hemodynamic overload
- Poor prognosis if maladaptive *adaptive remodeling is in response to normal changes*
- Cardiac muscle cells decrease in amount and enlarge in size→maintain stroke volume→ventricular wall thickens
- Activation of RAAS quickens the remodeling process
- Increase of proinflammatory cytokines

(Colucci, 2018)



(Solimando, 2017)

Disease Focused Diagnostics

Noninvasive

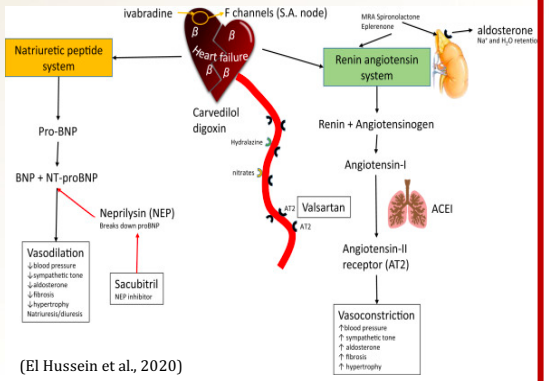
- Thorough history and physical → valuable information based on discussion of history, symptoms, comorbidities, and physical assessment
- Serum blood levels → elevated BNP correlates with HF
- 12-lead ECG → determine if there is ventricular hypertrophy
- Chest x-ray (PA/LAT) → visualize heart and lungs
- Transthoracic echocardiogram → heart size and functionality/determines EF

Invasive

- Coronary angiogram → excludes coronary disease as a cause/assess severity of pulmonary HTN
- Hemodynamic monitoring *heart pressures and pulmonary capillary wedge (PCW) pressures → if elevated, proceed to drug studies
- Drug studies utilizing pulmonary v. systemic vasodilators → assess which medication is effective in improving cardiac output ***last resort testing method***

(Wan et al., 2019)

Mechanism of pharmacotherapies



(El Hussein et al., 2020)

Implications for the Advanced Practice Nurse

- Assess and intervene on modifiable variables r/t medication regimens starting with inpatient initiation of pharmacological interventions →cost, knowledge deficit/depression/mental health (Albert & Kozinn, 2018)
- Lead HF clinics utilizing a multidisciplinary approach →patients will reach optimization in pharmacological therapies and remain adherent
- Evaluate need for implantable cardiac defibrillator (ICD) placement →placement post-cardiac injury with a rEF reduces mortality (Chan et al., 2020)
- Telehealth visits →assess symptoms to avoid rapid decompensation
- Evaluate need for invasive/noninvasive monitoring devices → assess impeding decompensation (Murphy et al., 2019)
- LVAD-education on expectations/risks and complications/effects on QoL/palliative care (Kaiser, 2019)

LAST TREATMENT OPTION FOR HFrEF/NOT ELIGIBLE FOR TRANSPLANT

Treatment

Pharmacotherapies

Angiotensin-converting enzyme (ACE) inhibitor
- Lisinopril/Enalapril

Angiotensin II receptor blocker (ARB) *if ACE is ineffective*
- Valsartan/Candesartan

Angiotensin receptor neprilysin inhibitor (ARNI) *must stop ACE before initiating*
- Sacubitril/Valsartan (Entresto™)

β-Blocker *appropriate if no hypotension and HR>50*
- Carvedilol/Metoprolol

Mineralocorticoid receptor antagonist (MRA) *used in conjunction with Entresto™*
- Spironolactone/Eplerenone

Loop diuretic *assists with excess fluid removal/will cause a decrease in K+ levels*
- Furosemide

Hyperpolarization-activated cyclic nucleotide-gated channel blocker *appropriate if NSR, tolerating all other medications at max level*
- Ivabradine

Cardiac glycoside
- Digitalis

Direct-acting vasodilator *stable/African-American*
- Hydralazine/Isosorbide

Evidence-based pharmacologic treatment for HFrEF displaying a decrease in hospitalizations and improvement in QoL (Albert & Kozinn, 2018, p. 18).

Left Ventricular Assistive Device (LVAD)

- End-stage HF
- Two indications: bridge to transplant (BTT) or destination therapy (DT)
- Takes over the function of the heart muscle and maintains cardiac output
- Risk of complications: infection, blood clots, GI bleed, dysrhythmias, stroke

(Capriotti & Micari, 2019)

Remodeling and Treatment?

HF treatment aims to slow the process of remodeling and reduce the activation of RAAS. The goal is to cease the advancement of the disease (Bowers, 2019). HFrEF and HFpEF may exist concurrently, however, treatment methods vary (Murphy et al., 2019)

Conclusion

HFrEF is a chronic disease in which treatment is constantly evolving. The pathophysiology of the disease and ways to effectively manage HFrEF are everchanging. Hence, research continues on the molecular changes that occur within the heart as well as appropriate therapies for disease management. The APN is in a front-line role to educate patients and initiate therapies to prevent readmissions. APN led HF clinics are beneficial in ensuring faster follow-up after discharge. Knowledge sharing between the APN and patient can increase the patient with HFrEF adherence and quality of life.

References

OTTERBEIN UNIVERSITY