2019

Heart Failure with Preserved Ejection Fraction

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Introduction

- Heart failure with preserved ejection fraction (HFpEF), formerly known as diastolic heart failure, is a diagnosis that is becoming more prevalent as the population ages. HFpEF affects 10 in every 1,000 individuals over 65 years of age, with 870,000 new cases being diagnosed every year (Tawil & Gelzinis, 2016).
- HFpEF accounts for 54% of all heart failure diagnoses. The five year mortality for patients with HFpEF is 42.3% (Singh & Mehta, 2018).
- HFpEF is a clinical syndrome that encompasses several cardiovascular disease processes and symptoms that relate to diastolic dysfunction while retaining a left ventricular ejection fraction (LVEF) of > 50% (Harper, Patel, & Lyon (2018).
- HFpEF requires careful consideration of the healthcare provider when exploring clinical decisions relating to the management of the disease and beyond.

Topic Importance

- As the population ages, HFpEF is becoming more prevalent. It is the leading cause of hospitalization in patients 65 and older (Tawil & Gelzinis, 2016).
- The management of HFpEF creates unique challenges for the anesthesia provider and intraoperative clinical management and decision making, and the disease is associated with increased mortality postoperatively (Singh & Mehta, 2018).
- Health care expenses related to heart failure totaled $30 billion in 2013 (Gazewood & Turner, 2017).
- In the next 20 years the prevalence of heart failure is projected to increase by 50% (Farris et al., 2017).

Risk Factors

- Age > 70
- Female sex
- Obesity
- Hypertension
- Tobacco use
- Diabetes mellitus
- Coronary artery disease
- Valvular heart disease
- Atrial fibrillation

(Gazewood & Turner, 2013)

Disease Process

- HFpEF consists of a combination of cardiovascular disease processes that become prevalent in advanced age. As the patient ages, the cardiomyocyte function degrades, and the production and utilization of ATP decreases cause the inability of the ventricles to relax as well as stiffness resulting in issues with ventricular compliance and filling (Singh & Mehta, 2018).
- As the heart works to maintain the same contractility to deliver oxygen to the tissues, the walls may hypertrophy to elicit a greater pressure gradient and overcome the cardiac wall stiffness and impair relaxation (Farris et al., 2017).
- As the heart continues to fail, symptoms of HFpEF may arise, such as fatigue, dyspnea on exertion, and edema (Gazewood & Turner, 2017).
- On the onset of symptoms, the patient may seek treatment. The diagnosis of HFpEF consists of the presence of signs and symptoms of heart failure, and echocardiogram confirming LVEF > 50%, and a beta-natriuretic peptide (BNP) of > 100 pg/mL (Tawil & Gelzinis, 2016).
- Because the pathophysiology of HFpEF is irreversible, there is no treatment available. However, the symptoms of HFpEF can be managed with diet and lifestyle changes, as well as medications such as diuretics to reduce preload, beta blockers to reduce afterload, and ACE inhibitors to prevent further remodeling of the myocardial tissue (Harper et al., 2017).
- HFpEF is a chronic disease that when diagnosed will be present for the remainder of the patient's lifetime.

Signs and Symptoms

- General fatigue and weakness
- Dyspnea, paroxysmal nocturnal dyspnea, or orthopnea
- Edema
- Jugular venous distension
- S3 heart sound
- Displaced apical impulse

(Gazewood & Turner, 2017)

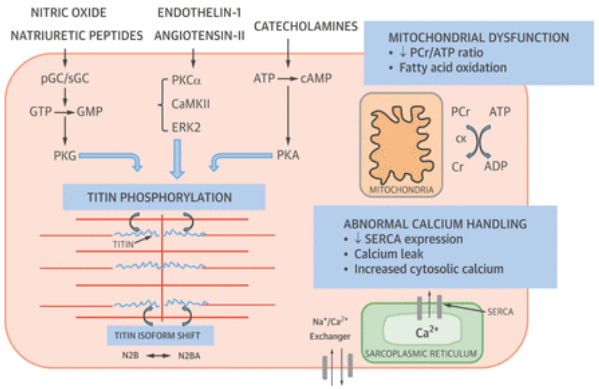

Figure 1. Specific biological phenotypes in HFpEF (Lewis et al., 2017).

Underlying Pathophysiology

- In normal physiology, the heart achieves optimal cardiac output through the augmentation of stroke volume and heart rate. Myocardial contractility, the Frank-Starling law of stretch, and afterload related to ventricular-atrial coupling a determine stroke volume. Diastole affects these components. In beginning diastole, elastic recoil relating to the Frank-Starling law and active myocardial relaxation creates a negative pressure gradient, allowing passive filling of the LV, which accounts for 80% of ventricular filling. Following this passive filling, the LA contraction, or "kick," causes the remainder of ventricular filling. When the ventricle reaches its maximum fill capacity, contraction begins, beginning systole and ejecting blood from the heart. Diastole is an active process, meaning it requires energy in the form of ATP. Diastole is the process of the LV filling from the LA through the mitral valve. It is affected by plasma volume, passive elasticity, and active relaxation of the LV, and the volume and pressure received from the LA (Farris et al., 2017).
- HFpEF has two significant components: alteration of active ventricular relaxation and myocardial stiffness.
- HFpEF impairs the active relaxation of the LV due to reduced cardiac myocyte ATP production related to the dysfunction of the mitochondria. A decrease in nitrous oxide (NO) production, an increase in reactive oxygen species, dysfunction of the endothelium, and ischemia of the microvasculature causes the dysfunction of the mitochondria. Because of the decrease in ATP production, the cardiomyocytes are unable to uptake calcium into the sarcoplasmic reticulum appropriately, which prevents calcium from unbinding to troponin. This calcium mismanagement leaves the myofilaments unable to uncouple, preventing active relaxation (Farris et al., 2017).
- The myocardium becomes stiff in HFpEF due to sarcomere dysfunction and an increase in extracellular matrix proteins. In a non-diseased resting heart, the action of titin protein located in the sarcomeres mediates ventricular stiffness and tension. Titin works as a bidirectional spring and plays a part in early diastolic passive recoil. In HFpEF, the titin protein does not phosphorylate normally and causes an increase in resting myocardial stiffness. Excessive extracellular matrix protein production causes fibrosis and myocardial stiffness in HFpEF by binding to the collagen matrix, cardiomyocytes, and fibroblasts (Farris et al., 2017).
- Because of the impaired relaxation and myocardial stiffness associated with HFpEF, the heart must work harder to achieve the same ejection fraction (EF). In order to maintain the necessary EF, the cardiomyocytes may hypertrophy, causing enlargement of the heart. This enlargement can decrease stroke volume due to reduced available filling space while not affecting contractility. The enlargement of the cardiac muscle maintains the ejection fraction, which is stroke volume divided by end-diastolic volume, or the volume in the heart after ventricular filling has occurred before the start of systole. Because the pathophysiology of HFpEF causes a reduction of both stroke volume and end-diastolic volume, the EF is preserved, remaining > 50% (Farris et al., 2017).

Significance of Pathophysiology

- As the ventricle hypertrophies and becomes stiff, the cardiac output will decrease. This causes blood to back up into the LA and into the pulmonary circulation. The backflow of blood into the pulmonary vasculature can cause pulmonary edema, and acute, life-threatening disorder that must be managed in an acute care setting and may require advanced resuscitation techniques and aggressive medication and airway management. This acute on chronic exacerbation of HFpEF accounts for the majority of hospital admissions in patients 65 years of age or older (Farris et al., 2017).
- In an outpatient setting, the symptoms associated with HFpEF can cause a reduction in the quality of life for the patient without proper management through diet changes, lifestyle changes, and medication (Harper et al., 2018).
- Diuretics are the hallmark of HFpEF management because of their ability to reduce preload. If there is less preload, the heart may not struggle to pump an increased volume of blood forward, reducing the instances of acute on chronic exacerbations (Harper et al., 2018).
- Management can also include medications such as beta blockers, which contribute to a decrease in afterload that can augment the cardiac output, and ACE inhibitors, which can prevent remodeling of the heart muscle (Harper et al., 2017).

Implications for Nursing Care

- The management of HFpEF requires diet and lifestyle changes, as well has a strict medication regimen.
- The nurse has an important role in the education of the HFpEF patient on these expectations for prevention of clinical deterioration and an acute on chronic exacerbation (Cavalcanti & Pereira, 2014).
- Patients that develop an acute on chronic exacerbation of HFpEF may be admitted to inpatient units. In this case, the nurse must be aware that the patient could exhibit exercise intolerance and shortness of breath. This may impede nursing care and require management in an acute care setting (Gazewood & Turner, 2017).
- HFpEF presents challenges to the anesthesia provider, as it causes a reduction in cardiac output which can be amplified by anesthetic agents (Singh & Mehta, 2018).

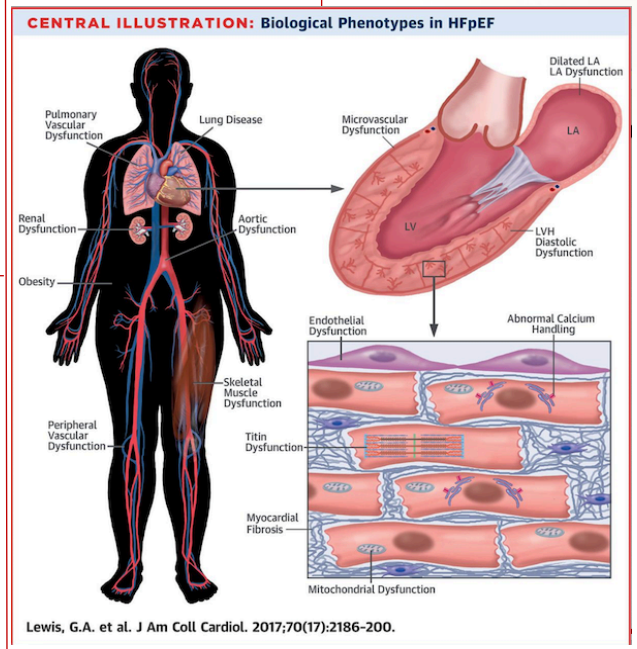

Figure 2. Biological phenotypes in HFpEF (Lewis et al., 2017).

Conclusion

- HFpEF is a complex medical syndrome that affects a significant portion of the population and poses many unique clinical considerations for the healthcare provider.
- It is important for healthcare providers to be aware of the pathophysiology and considerations related to HFpEF in order to make evidence-based decisions involving the care of patients who present with the disease.
- As the population ages, the prevalence of HFpEF is increasing significantly (Tawil & Gelzinis, 2016).
- Although treatment for HFpEF does not exist, symptoms can be managed with medication and lifestyle changes. The nurse is in a unique position to provide education to the HFpEF patient to improve quality of life (Cavalcanti & Pereira, 2014).

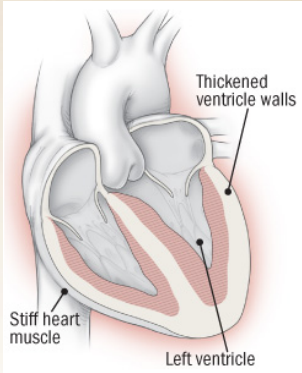

Figure 3. An illustration of heart muscle hypertrophy in HFpEF ("Diastolic Heart Failure," 2010).

References

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