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Managing Heart Failure and Enhancing Quality of Life for Patients with Preserved and Reduced Ejection Fraction

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Abstract

Heart failure (HF) is a chronic condition characterized by the heart's inability to pump blood effectively. Hypertension is a frequent contributing factor. The reduced cardiac output leads to fluid accumulation, causing respiratory distress and chest pain. Ejection fraction (EF), a measure of the left ventricle's pumping capacity, is crucial in HF diagnosis. HF with reduced EF (HFrEF) is defined by an EF below 40%, often caused by coronary artery disease—approximately half of all HF patients present with HFrEF. The remaining cases encompass HF with mid-range EF (40% - 50%) or HF with preserved EF (HFpEF, EF ≥ 50%).

Keywords

Heart Failure, Pathophysiology, Etiology, Reduced Ejection Fraction, Preserved Ejection Fraction, Revascularization, CABG, Ventricular Remodeling, Myocardial Viability

1. Introduction

Over time, heart failure can damage the kidneys and other organs and lead to anemia, fluid retention, and fatigue, among other symptoms [1]. Medications and procedures help heart failure sufferers manage their symptoms and lead healthier lives [2]. HFpEF is a growing problem in cardiac health, associated with getting older, being overweight, having high blood pressure, or even metabolic disorders [3]. Unfortunately, no available treatment is effective against this cardiac ailment [3]. It can be marked by an excessive amount of left ventricular pressure building up due to diastolic dysfunction, and a rise in pressure can be observed both at rest and during exercise, which can ultimately result in pulmonary hypertension [3].

Although the ejection fraction appears normal, people with HFpEF still experience mild deficiencies in systolic functioning and cannot take advantage of their systolic reserve capacity under stress. This results in a lesser degree of ejection fraction increase when compared to healthy subjects [3]. In this case, heart failure is due to left ventricular hypertrophy, or an enlargement of the left ventricle, which is often a result of the extra strain put on the heart by high blood pressure [4]. Heart failure with reduced ejection fraction is a complex and progressive medical condition associated with shortness of breath and impaired physical function. With its high mortality rate and risk of readmission, it has become one of the greatest public health challenges [5] [6].

A person with high blood pressure will develop heart volume overload, leading to left ventricular hypertrophy. Therefore, a person with high blood pressure and HFpEF will have signs and symptoms of both conditions. As a result of the heterogeneous pathophysiology that exists within the broad spectrum of HFpEF, there is no effective treatment for heart failure with reduced ejection fraction, which is an increasingly common form of cardiac disease associated with aging, obesity, and hypertension. It is likely to have a poor short-term and long-term prognosis [7]. The underlying pathophysiological mechanisms that contribute to the development of heart failure with reduced ejection fraction are complex and multifactorial and may include alterations in cardiac structure and function and deficits in the vasculature and activity of the sympathetic nervous system [8].

One of the main challenges in treating heart failure with reduced ejection fraction is the highly individualized nature of the disease, which can be influenced by age, comorbidities, and patient-specific factors [8]. As a result, no single treatment approach is effective in all cases of heart failure with a reduced ejection fraction. However, interventions such as exercise regimens and lifestyle modifications can help reduce the symptoms of heart failure with a reduced ejection fraction [9]. Additionally, certain medications, such as angiotensin-converting enzyme inhibitors and beta-blockers, can help slow the progression of the disease and improve the quality of life of patients suffering from heart failure with reduced ejection fraction [10]. Statins have been shown to reduce left ventricular hypertrophy and fibrosis under laboratory conditions. A small study indicated that statins could decrease the risk of death among patients with HFpEF [10].

It is now understood that the greatest predictor of long-term survival after a heart attack is the capacity of the left ventricle. This function has conventionally been described in terms of ejection fraction, but it remains unclear if this metric holds much value in a post-heart attack context [11]. Hence, the relevance of discussing this topic is proven. A low ejection fraction may be caused by a lack of contractility from severe cardiac damage or ongoing ischemia and may also arise from the dilation of the left ventricle due to expansion of the infarct. Therefore, the end-systolic end-systolic volume (ESV) or the end-diastolic end-diastolic volume (EDV) could provide better prognostic indicators than an ejection fraction alone [11].

2. Pathophysiology of Heart Failure

Heart failure (HF) can be broadly categorized into heart failure with preserved ejection fraction (HFpEF) and heart failure with reduced ejection fraction (HFrEF). HFpEF is characterized by normal ejection fraction but diastolic dysfunction, whereas HFrEF involves reduced ejection fraction due to systolic dysfunction. Table 1 summarizes the key differences between HFpEF and HFrEF:

Table 1. Key differences between HFpEF and HFrEF.

Aspect	HFpEF	HFrEF
Pathophysiology	Normal EF, diastolic dysfunction	Reduced EF, systolic dysfunction
Common Causes	Hypertension, aging, obesity	Coronary artery disease, MI
Symptoms	Shortness of breath, fatigue	Shortness of breath, fatigue
Treatment	Diuretics, lifestyle changes	ACE inhibitors, beta-blockers
Prognosis	Variable, often chronic	Higher mortality, progressive

The amount of blood the heart pumps in a certain period is called the cardiac output. This is calculated as the product of heart rate (HR) and stroke volume (SR) and is usually between 4 and 8 L/min. Factors such as ventricular contraction strength, wall integrity, and valvular competence influence cardiac output [12].

Cardiac failure can be the result of systolic or diastolic dysfunction or both [13]. The etiological factors behind heart failure can be attributed to myocardial cell damage and decreased functional cells [12]. The three main culprits of this condition are ischemic heart disease, hypertension, and diabetes, doubling the risk of developing heart failure compared to those with normal blood pressure [10] [12]. In addition, cardiomyopathy, certain types of viral myocarditis, Chagas disease (a viral infection), excessive alcohol consumption, exposure to toxic drugs, valvular disease, and prolonged arrhythmias all cause heart failure. However, these are much less common than the primary three factors [12].

Physiological processes in heart failure with preserved ejection fraction (HFpEF) can be explained as a condition that occurs more and more in people as they age or for those who are obese, those who have diabetes, and those who have high blood pressure. One of the other main characteristics of heart failure with a preserved ejection fraction is reduced ability or physical activity [14]. In any such case, the left ventricular filling pressure is affected, resulting in pressure overload [15]. This extra pressure can have other consequences, such as secondary pulmonary hypertension [15]. Furthermore, this has become the most common type of heart failure, and its rate of occurrence compared to heart failure with a reduced ejection fraction is still increasing [16].

Heart failure with reduced ejection fraction is when the left ventricle of the heart cannot pump adequate blood to the rest of the body [16]. This results in symptoms such as shortness of breath and fatigue caused by weakened and ineffective heart pumping or decreased heart pumping [9]. According to epidemiological research,

approximately half of heart failure (HF) patients have a normal or preserved ejection fraction [9]. Therefore, patients with heart patients fail are classified into two groups:

1) Those with reduced or decreased ejection fraction, known as systolic failure (Mann, 2008).

2) Those with a preserved ejection fraction are generally called diastolic failure (Mann, 2008).

According to the ejection fractions, heart failure can be classified as HFpEF and heart failure with reduced ejection fraction [15]. Despite having a normal range of ejection fractions, those with HFpEF have a worse prediction [10]. Epidemiology The epidemiology and etiology of HFpEF and heart failure with reduced ejection fraction differ; patients with HFpEF tend to be older and more female, with comparatively fewer incidents of ischemic events but higher risk factors such as obesity, hypertension, hypertension, and diabetes mellitus. There are comparable rates of mortality and hospitalization between the two types of heart failure. Treatment with neuroendocrine antagonists has been shown to provide prognostic benefits in heart failure with a reduced ejection fraction but not in patients with HFpEF, patients with HFpEF, probably due to an incomplete understanding of the pathophysiology of the condition [10].

3. Signs and Symptoms

As the heart weakens, patients experience shortness of breath due to lung buildup and swelling of their extremities and stomach area due to their blood not circulating correctly [17]. Other symptoms, such as nausea, lack of appetite, exercise intolerance, heart palpitations, rapid or irregular heartbeat, chest discomfort, weakness, and dizziness, also become apparent [17]. Patients may also experience clammy or sweaty skin. Often, cardiac disorders may have signs such as swelling or edema, abnormal heart rhythm, abnormal lung sounds, and symptoms such as dyspnea, fatigue, and inability to perform physical functions [15]. Signs and symptoms associated with reduced ejection fraction are almost the same as in any heart disease; sometimes, it may be asymptomatic. Similarly, heart failure with preserved ejection fraction presents similar signs and symptoms [17].

The heart attempts to combat these symptoms by increasing cardiac output using the Frank-Starling mechanism, increasing ventricular volume and wall thickness through remodeling, and increasing blood pressure by working with the nervous system. These adaptations can be useful initially, but eventually, they lead to a never-ending cycle of deterioration of heart health [17].

According to the American Heart Association, there are two common types of heart failure, each requiring a different course of treatment [18]. The first type, systolic heart failure, is caused by an enlarged and weakened heart muscle. The second type, diastolic heart failure, is caused by stiffened, stiffened, and thickened heart muscle. Both types of heart failure can be fatal if left untreated [18]. Diastolic heart failure (DHF) and systolic heart failure (SHF) are the two distinct forms of

a chronic condition known as heart disease [18].

4. Risk Factors

The elderly population is more prone to develop cardiovascular when the disease is present compared to the younger generation [18]. Risk factors associated with any chronic heart disease, including the reduced ejection fraction and the preserved ejection fraction, in addition to aging, are hypertension, obesity, diabetes, high cholesterol, and a sedentary lifestyle [15]. Hypertension is a major factor causing diastolic heart failure or HFpEF. However, many patients with systolic heart failure or heart failure with a reduced ejection fraction may have a history of hypertension. The most common etiology of heart failure with a reduced ejection fraction is ischemic heart disease, which is found to be a contributing factor in more than 60% of diagnoses. Patients with heart problems caused by a lack of blood flow to the left ventricle have a much higher mortality rate than those with non-ischemic non-ischemic conditions [19].

Patients with diastolic heart failure may have coronary artery disease [18] [19]. As a preventive measure, the same risk factors should be modified in both cases [18]. Additionally, demographic elements such as age, sex, patient history, social history, and co-occurring illnesses that can predict BP sensitivity in humans are comparable to those of HFpEF. Likewise, sodium intake and other nutritional characteristics may influence the risk of heart failure in obese individuals and those over a certain age [20]. Above all, there are social or behavioral factors that can have an adverse effect on heart health, such as smoking and drinking too much alcohol [21]. These activities can badly affect various organs and hinder their ability to function properly [22]. Additional social or behavioral factors can be modifiable risk factors [21]. See **Figure 1** for an illustration of risk factors in heart failure.

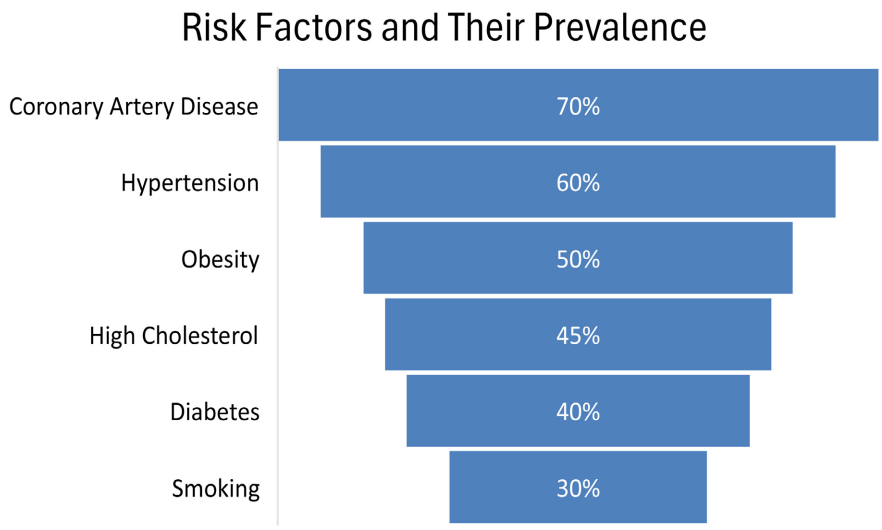

Figure 1. This graph visually represents the percentage of HF patients affected by each risk factor, making it easier to understand the relative prevalence of these conditions.

5. Clinical Events in Patients with CHD and Reduced EF

The degree of left ventricular dysfunction is the most significant predictor of mortality after acute myocardial infarction, but it is not clear whether this assessment should be based on ejection fraction or end-systolic or end-diastolic volume [11]. Since chronic heart disease (CHD) is the leading cause of heart failure with reduced ejection fraction, a condition in which the left ventricle cannot pump enough blood to meet the body's body needs, surgeons sometimes need to up blocked arteries in people with CHD [7]. Also, it is not clear whether the only lifestyle changes of a patient with coronary heart disease with asymptomatic reduced ejection fraction can delay surgical procedures or not [23].

It is believed that a high-fat, high-carbohydrate diet can cause diabetes, obesity, and cholesterol, causing coronary heart disease [24]. High-high-carbohydrate intake can increase triglycerides, total cholesterol, and low-density lipoprotein (LDL), which is the bad cholesterol, and the decrease in high-density lipoprotein (HDL). This good cholesterol can also cause hyperinsulinemia [24]. The increased triacylglycerol to high-density lipoprotein ratio is a crucial predictor of a heart attack [24].

Several studies have found that the ketogenic diet is beneficial for obesity and diseases associated with obesity. Therefore, a ketogenic diet may help obese patients as a natural therapy [24]. In a ketogenic diet, carbohydrates and the healthy fat diet will be increased; hence, certain other controversial studies showed that the high keto diet resulted in fat accumulation and led to a significant factor in the high cholesterol that causes heart disease [24]. The ketogenic diet alone does not help with chronic heart disease.

6. Pharmacological Management

6.1. Drug Therapy

Several different types of drugs are often used to treat heart failure. They are classified according to how they improve health and manage symptoms. Cardiac glycosides, beta-adrenergic blocking agents, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers (ARB) (ARB) are the four main drug classes used to treat heart failure [25]. Examples of cardiac glycosides include digoxin and Digitoxin. Nonpharmacological therapy can improve patients' quality of life with heart failure, provided that the ejection fraction remains above 20% [25].

HFpEF manifests differently in all patients and is associated with a wide range of underlying causes, as well as extracardiac extracardiac manifestations and cardiac abnormalities. Regardless of the individualized nature of HFpEF, the standard course of treatment has been based on an approach that works best for chronic heart failure with reduced ejection fraction [26]. When considering iron deficiency intravenously (IV) rather than orally, iron can improve quality of life and reduce symptoms in patients with heart failure with reduced ejection fraction, measured by transferrin saturation levels [10].

In treating patients with suspected HFpEF, it is crucial to identify the causes

that may be treated, such as contributing factors that contribute to risk, comorbid illnesses, and specific cardiac conditions. Unfortunately, there are no therapies that improve survival in these cases; treatment focuses on relieving symptoms and improving quality of life, controlling fluid retention, and modulating risk factors and any related diseases. To this end, renin-angiotensin-aldosterone system inhibitors, diuretics, calcium channel blockers (CBB), beta-blockers, diet and exercise advice remain essential, although they have not been shown to reduce mortality through large randomized controlled trials [10].

In HFpEF, diuretics are often proven effective as the kidneys are affected due to chronic systemic inflammation, which impairs their ability to excrete sodium and sodium, further leading to gradual volume expansion during the transition from compensated to decompensated HFpEF, and diuretics help reduce the fill pressure of the left ventricular filling pressure [26]. If a patient does not follow the prescribed diet, take their medication, and make the necessary lifestyle changes for heart failure, it can lead to further complications, complications, including a worsening of the condition or issues with treatment, which shows the importance of patient compliance [27].

6.2. Phenotype-Specific Treatment

Remodeling refers to compensatory changes in the heart after a heart attack. It is known as maladaptive remodeling because some of the changes that occur do not have a good effect on the heart. This remodeling is often seen in patients with myocardial infarction or heart attack [26]. The heart shrinks and becomes abnormally shaped. This is not the same as a normal, healthy heart, which is stronger and more resilient than before it was injured [26].

The ventricular surgical reconstruction technique is designed to reverse the maladaptive morphological changes of post-infarction ventricular remodeling by restoring left ventricular volume and a more normal elliptical shape to the left ventricle, thus reducing myocardial wall stress and improving ventricular function. The surgical ventricular reconstruction technique has been added to coronary artery bypass graft (CABG) surgery, especially for patients at risk for recurrent myocardial infarction (heart attack). Adding surgical ventricular remodeling to CABG surgery reduces left ventricular end-diastolic volume compared to CABG alone [19].

However, this structural alteration is not related to a significant improvement in symptoms or exercise tolerance or a reduction in the death or hospitalization rate due to cardiac problems. The study concluded that adding ventricular surgical reconstruction to CABG is not beneficial in reducing morbidity or mortality [19] [26].

The etiological factor of HFpEF differs from that of heart failure, with a reduced ejection fraction, which is linked to the death of the heart cells due to a lack of blood flow, infection, or toxicity [26]. The uneven remodeling of the left ventricle under these two conditions reflects its separate origins. Biomarkers provide

additional evidence for this difference; they indicate lower myocardial damage or strain levels in HFpEF compared to heart failure with reduced ejection fraction [26].

6.3. Direct Cardiac Shockwave Therapy

The buildup of plaque on the inside of the arteries that causes blockage of blood flow to the heart is atherosclerosis [19]. Doctors have found a way to regenerate these affected sections through shockwave therapy. This well-established regenerative tool uses controlled shockwaves and waves to deliver unique chemical signals to stimulate the body's natural healing processes. Shockwave therapy creates new blood vessels in the ischemic myocardium [16] [28].

One such chemical signal is vascular endothelial growth factor, a signaling protein that directs the body to create new blood vessels and prevent others from breaking down. These new blood vessels form due to angiogenesis (forming new blood vessels controlled by chemical signals in the body). Through induction of angiogenesis and reduction of fibrotic scar tissue, shockwave therapy has helped regenerate the ischemic myocardium [16] [28].

Although conventional medical treatment and cardiovascular revascularization with CABG are considered the go-to for addressing chronic cardiovascular problems, problems, there are times when this procedure may not be helpful due to its high costs or health limitations [28]. The novel regenerative technique known as cardiac shockwave therapy may provide a solution at these moments.

Many studies have observed the beneficial effect of shock wave therapy on cardiovascular disease. However, quality research related to the benefits of shockwave therapy in coronary heart disease is limited and is mainly observational and retrospective studies. However, these provide some indication of the benefits of shockwave therapy, which are valid through a study of the relatively small number of patients with heart attack patients in the emergency room. A 2016 meta-analysis of randomized controlled trials demonstrated a significant reduction in the mortality of heart attack patients who received shockwave therapy [16].

Reducing mortality is the primary goal of heart attack treatment, as it benefits the patient and the nation. In light of this, researchers believe that shockwave therapy may be a suitable alternative for patients who cannot undergo coronary artery bypass graft surgery [16] [28]. A study on the use of shock wave therapy for patients with ischemic heart disease concluded that it was a safe and effective treatment, especially for those suffering from angina pectoris, chest pain or discomfort, which is often considered a sign of increasing the risk of myocardial infarction or perhaps a symptom for coronary artery disease [28].

Cardiac shockwave therapy can create a unique type of air current that can provide shear stress to the microenvironment of the heart muscle. This could potentially lead to changes in cell membranes, hyperpolarization, and hyperpolarization, an increase in the activity of the RAS protein (the protein associated with growth and development), as well as an increased presence of endothelial nitric

oxide synthases and vascular endothelial growth factors in areas with reduced blood flow, and, thereby, lack of oxygen [28]. Also, shockwave treatment increases stromal cell-derived factor 1 production, leading to higher amounts of cells with CXC chemokine receptor type 4 (membrane proteins that bind and respond to cytokines) in the heart [28].

Although shockwave therapy is innovative, it is essential to remember that it is not new and has been performed on other parts of the body, in addition to the heart and blood vessels. Some doctors are already performing shockwave therapy for knee osteoarthritis and treating various other disorders. For example, the Mayo Clinic uses shockwave shock wave therapy to treat tennis elbow. The National Institute of Health has approved its application for treating erectile dysfunction and chronic lower back pain [26].

HFpEF accounts for fifty percent of all heart failure cases [26]. It also presents a diverse syndrome initiated by various comorbidities and inflammatory mediators with extracardiac manifestations, cardiovascular abnormalities, and other diseases. Consequently, drug trials have generally been inconclusive regarding their primary objectives. Only exercise training and weight loss appear to improve exercise intolerance and quality of life [20].

Additional approaches to address the underlying pathophysiology of HFpEF, beyond the optimization of medications, nutritional interventions, and the use of pacemakers and implants to prolong survival and alleviate disease progression, may include reducing chronic inflammation, modulating autonomic nervous system activity, correcting cardiac volume or loading conditions, and enhancing adaptive cardiac responses to mechanical stimuli such as exercise training or increased sympathetic nervous system activity [20].

New therapies are directed at preventing or attenuating vascular remodeling and/or endothelial dysfunction to reduce CVD risk in this group. It is also important to address the progression of structural changes in patients with milder symptoms, who are more likely to live longer than patients with more classic manifestations of HFpEF [16] [26].

6.4. Coronary Artery Bypass Graft Surgery—Revascularization

Coronary artery bypass graft surgery (CABG) is used to treat coronary artery disease and dramatically increases survival chances for patients. Jones *et al.* (2009)'s studies have concluded that myocardial revascularization, achieved through CABG, can improve patients' quality of life and survival with chronic heart disease [29].

Before any cardiac surgery, measuring the ejection fraction is an important part of assessing the patient's risk for the procedure [7]. Coronary artery disease occurs when the arteries that supply blood to the heart become clogged or narrowed by substances called plaque, restricting blood flow. The condition is serious and can cause chest pain, heart attacks, and even death in severe cases. The procedure involves taking a healthy blood vessel from another body part, usually the leg, chest,

or arm, and bypassing a blocked or narrowed artery [9].

Veins are most commonly used because they are naturally flexible and durable. A vein is taken from elsewhere in the patient's body and connected to the artery on either side of the blockage, allowing blood to flow freely around it. In other words, coronary artery bypass surgery creates an alternate route for blood to flow around the blockage and reach the heart muscle [29]. Having a history of heart failure is considered a major risk factor for both short-term and long-term complications after coronary artery bypass graft surgery, even if the person had a normal ejection fraction before the procedure. Individuals with an ejection fraction lower than normal more than doubled the chances of mortality after CABG [7] [19].

Scientific research has indicated that healthy heart muscle in the left ventricle is a good predictor of whether people experiencing coronary heart failure will benefit from CABG [5]. There are controversies about the same. Some research suggests that those who undergo bypass surgery can expect an excellent quality of life and a 10-year survival rate [22].

Although a ten-year survival rate after bypass grafting is expected, expected, but patients who have alcohol use disorder and chain smokers must stop those unhealthy habits to prevent further negative impacts that could adversely affect their life expectancy or worsen their condition [22]. It has not been scientifically proven that age can affect a person's lifespan, but research suggests that age is not necessarily associated with reduced quality of life if the individual is in good health and not engaging in unhealthy habits [22].

This is especially true if the person has no preexisting conditions that could influence their well-being [22]. Bypass surgery prevents further blockages from occurring, and, in most cases, the arteries surrounding the grafts heal over time so that the grafts become permanent. CABG can be life-saving and has a positive effect on patients with heart failure and a reduced ejection fraction in all common categories of death, including sudden, pump failure, and myocardial infarction-related death [19]. However, it has certain risks and complications. One of the most common complications is graft failure, where the grafts do not heal correctly and must be removed, requiring the procedure to be repeated [29]. From the risks and complications associated with CABG, there are limitations too, which will limit the patients with heart failure and reduced or preserved ejection fraction to undergo surgery [28].

6.5. CABG with or without Surgical Ventricular Reconstruction

Surgical ventricular reconstruction can effectively reverse the maladaptive morphologic changes of post-infarction ventricular remodeling. By restoring the volume and creating a more normal elliptical shape of the LV, ventricular function can be improved. However, this operation was ineffective in reducing the risk of death or hospitalization due to worsening heart function after myocardial infarction [29].

The study concluded that adding surgical ventricular remodeling by CABG does not reduce morbidity or mortality. However, surgical ventricular reconstruction remains an important consideration for improving symptoms in heart patients and future research on cardiac procedure protocols. Patients with a dilated left ventricle or size irregularities in the ventricle prior to bypass surgery cannot restore their condition after surgery after surgery as the procedure does not influence it [5].

Ventricular reconstruction is also helpful in heart transplantation. In the wake of the death of heart failure due to a failing myocardium, ventricular reconstruction is a viable option to improve cardiac output, reduce the progression of heart failure, and improve quality of life [5]. There is potential to restore the atrial contribution to cardiac output by creating a hole between the left and right atria of the heart (upper chambers of the heart). Chambers of the heart). The ventricular reconstruction procedure has a high perioperative mortality rate, with an immediate death rate of 2.8% [5].

6.6. Effects of Myocardial Viability and Left Ventricular Remodeling after CABG

Every year, millions of people suffer from heart problems. The only solution is sometimes the coronary artery bypass graft [7]. Since chronic heart disease (CHD) is the leading cause of heart failure with reduced ejection fraction, a condition in which the left ventricle cannot pump enough blood to meet body needs, surgeons sometimes need to unblock arteries in people with CHD [5].

Researchers examined whether various factors, including left ventricular end-systolic volume index (LVESVI) and myocardial viability, affected patients' risk of death perioperatively and long-term survival after coronary artery bypass grafting (CABG). In a study, researchers followed 118 patients with coronary heart disease for approximately 30 months after CABG surgery. The main findings indicated that patients with low myocardial viability (LMV) had higher perioperative mortality compared to those with high myocardial viability (HMV). Although perioperative death was more likely in patients with LMV, it did not affect long-term survival.

The findings for patients with LMV were statistically significant compared to those with HMV. Researchers noted that the results could not be generalized to the entire CABG population. The study was presented at the European Society of Cardiology (ESC) Congress. The study included 118 patients with coronary heart disease in a single center who underwent CABG surgery. The researchers analyzed 121 CABG procedures involving a median of three grafts, two arteries, and two different vessels; two grafts were used as a bridge and one as a cross-clamp. The patients were divided into HMV and LMV groups, with characteristics including age, gender, sex, multivessel disease, diabetes mellitus, renal failure, and hypertension. For the HMV group, there was a significant difference between the LMV and HMV groups in the total number of grafts [5].

The difference between SHF and DHF remodeling can be summarized as follows: SHF involves an enlarged heart cavity with increased end-diastolic and end-systolic volume changes, reduced or unchanged wall thickness, increased wall stress, and decreased ejection fraction [18]. The mass is higher, but the mass or cavity ratio stays the same or decreases. Ventricular changes in ventricular shape and geometry occur primarily in the transverse axis of the heart. Some patients may also have electrical and mechanical delays in normal sequential AV contraction or desynchrony. In DHF, the size of the cavity may remain unchanged or even decrease with normal or end-diastolic reduced end-diastolic and end-systolic volumes. The thickness and mass can increase, leading to a high mass-to-cavity ratio. Diastolic The stress of the diastolic stress increases, and systolic wall stress remains normal, with a normal or increased ejection fraction. Ventricular shape and geometry changes are uncommon in DHF, but mechanical desynchrony can still occur without electrical desynchrony [18].

Compared to healthy hearts, echocardiographic studies have shown that SHF has impaired contractile function, which reduces the ejection fraction, and increased wall stress is another mechanism for lowering the lower ejection fraction. Meanwhile, in DHF, abnormal left ventricular relaxation and increased passive stiffness are observed on observation in echo-Doppler studies, which shifts the pressure-volume relationship upward and to the left, resulting in increased diastolic pressure [18].

7. Non-Pharmacological Management

Nonpharmacological therapy has long been recognized as an important part of treatment for patients with heart failure, particularly if the ejection fraction is relatively low. This therapy involves many interventions, including lifestyle changes, diet modifications, cardiac rehabilitation programs, and stress reduction techniques [9].

The nonpharmacological approach is used as a complement to medications intended to treat heart failure. One of the primary goals of nonpharmacological therapy is to improve the quality of life for patients with heart failure by helping them manage their condition more effectively. It involves addressing symptoms such as fatigue, shortness of breath, and chest pain and improving patients' overall physical and emotional well-being. In addition to medications, it is essential to provide nonpharmacological interventions for heart failure patients. Such measures can help slow the progression of the disease and delay the need for pharmaceutical treatment since classically recognized modifiable risk factors for ischemic heart disease and other atherosclerotic conditions are smoking, unbalanced diet, physical inactivity, unhealthy plasma lipid levels, high blood pressure, and being overweight or obese [21]. There is also evidence to suggest that nonpharmacological therapy can positively affect survival time [21].

For those with mild to moderate heart failure, treatment without medication is often enough to treat their symptoms and decrease impairment. However, more

severe heart failure typically requires a medical intervention, and successful Management of these issues can be achieved by logical behavior changes along with these interventions. This way, it can increase the quality of the outcome [21].

Nonpharmacological therapy is also vital in treating heart failure in children and adolescents, although pharmacological therapy is the mainstay of treatment in this population. Clinical trials have been conducted with random participants to examine the effectiveness of lifestyle interventions as a nonpharmacological and nonpharmacological approach on diverse groups, such as healthy individuals and patients from general practice. It was determined that these interventions resulted in a moderate but worthy decrease in blood pressure, total cholesterol, body weight, and coronary risk score. Furthermore, observed results have shown a considerable decrease in the number of fatal and nonfatal myocardial infarctions and unexplained deaths among those exposed to the intervention compared to those operating under regular circumstances [21].

Another effective nonpharmacological intervention for the symptoms and prevention of heart failure is cardiac rehabilitation. Cardiac rehabilitation is a program designed to promote exercise, maximize cardiac function, and improve quality of life. Cardiac rehabilitation can be performed in an outpatient setting but is more commonly performed in an inpatient rehabilitation facility hospital. However, the ejection fraction must remain above 20% for nonpharmacological therapy to be effective. This is because many treatment methods, such as lifestyle changes and cardiac rehabilitation programs, are ineffective if the heart cannot pump sufficiently [30].

The general goal of nonpharmacological and nonpharmacological therapy is to improve the quality of life of patients with heart failure, as it provides a valuable and effective way to do this. If they have a relatively high ejection fraction, patients can work with their healthcare providers to develop a customized treatment plan that best meets their needs. In most cases, this will include treatment with pharmacological therapy. This type of therapy aims to improve symptoms associated with heart failure and slow the progression of the disease. It is suggested that behavioral changes in the daily routine, such as stopping cessation and limiting alcohol intake, participating in healthy eating habits, and exercising regularly, should be made to improve the outcomes of pharmaceutical therapies [21] [22] [31].

A small study of 13 participants showed that a DASH diet limited in salt improved diastolic function, arterial stiffness, stiffness, and ventricular–arterial coupling after 3 weeks [10] [20]. DASH improved both the relaxation and stiffness measures of ventricular diastolic function. The energy available for diastolic filling appeared to be lower, yet stroke volume stayed constant, suggesting increased diastolic filling efficiency [20].

Studies on animal models indicate that limiting dietary sodium consumption can provide relief by improving cardiac muscle and vascular stiffness and functioning. It was hypothesized that adhering to the Dietary Approaches to Stop

Hypertension Diet in combination with Sodium Restrictive Diet (DASH/SRD) would enhance left ventricular diastolic function, arterial elastance, and the connection between the heart and artery in hypertensive HFPEF [20].

An obese group of HFpEF patients found success with a 20-week caloric restriction diet, and their symptoms, maximum oxygen consumption, and well-being also improved [10]. In particular, the quality-of-life improvements of this diet were greater than those achieved through exercise training alone. HFpEF causes persistent systemic inflammation, affecting the heart and other bodily systems, such as the lungs, skeletal muscles, and kidneys. Often, HFpEF patients cannot exercise due to a spike in LV filling pressures. However, for some individuals, the tolerance to the effort is limited by pulmonary hypertension or inadequate use of perfusion and oxygen use of peripheral muscles. The kidneys are also affected, which limits the ability to excrete sodium [26].

When combining diet and exercise, each appeared to be the effect of another build [10]. Other healthy food options include eating a fair amount of vegetables, fruits, legumes, whole grains, and nuts, which are staples of a vegetarian diet and have been associated with a reduced risk of cardiovascular disease [31]. Adherence to the prescribed treatment course, including the other dietary and lifestyle, is essential to achieve a 100 percent success rate when treating the disease [27]. Even any deviation from the nonpharmacological plan, the nonpharmacological plan increases the risk of complications and further deterioration [27].

8. The Present Clinical Treatment and Future Emerging Therapies

New clinical treatments, including pharmacological therapy, device therapy, and surgical interventions, are emerging, and this involves an evolution toward more precise, safe, and effective treatment methods [32]. In terms of pharmacological treatments, this can include the use of traditional drugs such as ACE inhibitors, beta-blockers, and mineralocorticoid receptor antagonists, which are foundational in managing HF with reduced ejection fraction (HFrEF), and newer therapies like sodium-glucose cotransporter-2 (SGLT2) inhibitors and soluble guanylate cyclase stimulators, which have shown promise in improving outcomes for HF patients [33]. Device therapies, such as cardiac resynchronization therapy (CRT) and implantable cardioverter-defibrillators (ICDs), improve survival and quality of life for HF patients [32] [34].

Other emerging interdisciplinary treatments that hold potential for advancing HF management include stem cell therapy, which aims to stimulate myocardial healing and regeneration [35], and the use of exosomes for targeted drug delivery, the use of biomaterials and nanotechnology in developing innovative treatment strategies, such as cardiac patches and targeted nanoparticle delivery systems [32]. These emerging therapies represent a multidisciplinary approach to HF treatment, integrating advances in molecular biology, materials science, and nanotechnology to address the complex pathophysiology of HF and improve patient outcomes

[32]-[35].

SGLT2 inhibitors are a relatively new class of drugs used to treat heart failure. They effectively reduce hospitalization and cardiovascular death in patients with HFpEF and work by blocking glucose reabsorption in the kidneys, increasing glucose excretion in the urine. This also promotes sodium and water excretion, reducing fluid buildup in the body and easing strain on the heart [36] [37].

9. Conclusions

In conclusion, HFpEF with reduced ejection fraction is the most common syndrome in heart failure. It accounts for 50% of all heart failure cases. Patients with HFpEF can be asymptomatic or experience shortness of breath, fatigue, and or edema. They may also be diagnosed with hypertension, diabetes, or atrial fibrillation. As the trigger for heart failure, these conditions play a crucial role in the development of HFpEF. Unfortunately, current treatment strategies for HFpEF are not disease-specific. They, therefore, do not focus on the mechanism of action but rather on symptoms through diuretics and ACE inhibitors to address fluid retention and reduce blood pressure. As a result, patients with HFpEF often must take multiple medications to control them effectively. Symptoms. The growing understanding of the phenotypic diversity in HFpEF suggests that personalized therapeutic strategies may be valuable in treating this syndrome. Personalized therapeutic strategies encompass nonpharmacological therapy that can help manage and improve quality of life.

Lifestyle changes such as diet control and exercise can reduce weight, while stress management can help reduce or eliminate stress. Lifestyle changes effectively reduce weight and improve quality of life, along with pharmacological therapy to improve cardiac function, symptoms, and exercise tolerance in patients with heart failure with preserved ejection fraction. Unlike patients with systolic dysfunction, patients with HFpEF can live relatively everyday lives for many years with proper treatment. However, confusion and controversies continue regarding the definitions, pathophysiology, prognosis, and Management of diastolic and systolic heart failure.

In general, HFpEF and heart failure with a reduced ejection fraction share almost similar clinical characteristics, but that does not mean that the same thing causes them or that they should be treated in the same way. Patient compliance with pharmacological or nonpharmacological Management treatment is crucial for a successful outcome.

Manifestations like signs, symptoms, exercise intolerance, hemodynamics, and outcomes can be similar or identical, yet this alone cannot justify combined treatment options. The actual point of separating diseases should be tried to determine what causes them and what could potentially fix them.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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