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Review Article

Review of best treatment option in heart failure 2023

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ABSTRACT

Congestive heart failure is a clinical condition in which the heart is unable to pump enough blood to meet the metabolic needs of the body because of pathological changes in the myocardium. The three main causes of CHF are coronary artery disease, diabetes mellitus, and hypertension. US hospitalizations for heart failure decreased up until 2012; however, from 2013 to 2017, an increase in HF hospitalizations was observed. In 2017, there were 1.2 million HF hospitalizations in the United States among 924 000 patients with HF. This represents a 26% increase in HF hospitalizations and number of patients hospitalized with HF. For this reason, as a team, we have decided to conduct a review of the best treatment options for heart failure based on multiple articles.

Keywords: SLGT2, ARNIs, Heart failure, Progression, Treatment

INTRODUCTION

Heart failure is one the most common complications of the most prevalent pathologies in the population around the world, like hypertension and diabetes mellitus. In the view of above is important detect and treat the first stages of Heart Failure with best treatment options available. In the few past years, new drugs and recommendations had change the prognostic of the patients.[1]

BACKGROUND

Congestive heart failure (CHF) is a clinical condition in which the heart is unable to pump enough blood to meet the metabolic needs of the body because of pathological changes in the myocardium. The three main causes of CHF are coronary artery disease, diabetes mellitus, and hypertension.[3,4]

These conditions cause ventricular dysfunction with low cardiac output, which results in blood congestion and poor systemic perfusion. CHF is classified as either left heart failure (LHF) or right heart failure (RHF), while a combination of both is called biventricular or global CHF.[4] In addition, is classified by the cause of the heart failure in systolic dysfunction or reduce ejection fraction, and in diastolic dysfunction or preserved ejection fraction. Classification of HF by Left Ventricular Ejection Fraction (LVEF) is considered important in the classification of patients with HF because of differing prognosis and response to treatments and because most clinical trials select patients based on ejection fraction (EF).

Table 1: Etiology.

Etiology of heart failure		
Characteristics	Systolic dysfunction	Diastolic dysfunction
General causes	Coronary artery disease, arterial hypertension, Diabetes mellitus, valvular heart disease, renal disease, infiltrative diseases.	
Specific causes	Dilated cardiomyopathy (Chagas disease, chronic alcohol use, idiopathic), Cardiac arrhythmias, Myocarditis	Restrictive cardiomyopathy, Hypertrophic cardiomyopathy, Pericardial tamponade, Constrictive pericarditis
Risk factors	Obesity, Smoking, COPD, Use of heavy drugs (recreational and prescribed), Alcohol abuse	

Table 2: LVEF.[1]

Type of HF According to LVEF	Criteria
HFrEF (HF with reduced EF)	LVEF ≤40%
HFimpEF (HF with improved EF)	Previous LVEF ≤40% and a follow-up measurement of LVEF >40%[1]
HFmrEF (HF with mildly reduced EF)	LVEF 41%-49%. Evidence of spontaneous or provokable increased LV filling pressures (eg, elevated natriuretic peptide, noninvasive and invasive hemodynamic measurement)
HFpEF (HF with preserved EF)	LVEF ≥50%. Evidence of spontaneous or provokable increased LV filling pressures (eg, elevated natriuretic peptide, noninvasive and invasive hemodynamic measurement)

The ACC/AHA stages of HF is another classification that emphasize the development and progression of disease, and advanced stages and progression are associated with reduced survival. Therapeutic interventions in each stage aim to modify risk factors (stage A), treat risk and structural heart disease to prevent HF (stage B), and reduce symptoms, morbidity, and mortality (stages C and D).[1]

Table 3: AHA Stages.[5]

ACC/AHA classification		
Stages	Objective assessment	Corresponding NYHA functional class
Stage A	High risk of developing heart failure (e.g., preexisting arterial hypertension, coronary artery disease, diabetes mellitus) No structural cardiac changes or symptoms	No
Stage B	Structural damage to the heart (e.g., infarct scars, dilatation, hypertrophy) No signs or symptoms of heart failure	NYHA I
Stage C	Structural damage to the heart Signs or symptoms of heart failure	NYHA I, II, III, IV
Stage D	Terminal stage heart failure	NYHA IV

Epidemiology

US hospitalizations for heart failure (HF) decreased up until 2012; however, from 2013 to 2017, an increase in HF hospitalizations was observed. In 2017, there were 1.2 million HF hospitalizations in the United States among 924 000 patients with HF. This represents a 26% increase in HF hospitalizations and number of patients hospitalized with HF.[1,6] Heart failure is a prevalent condition globally. It affects millions of people worldwide, with estimates varying by region. In the United States, for example, it was estimated that around 6.2 million adults had heart failure in 2019.

Treatment

The initial treatment of patient with risk of develop or actually presenting HF is lifestyle modification this to reduce the progression. Like daily exercise; contraindicated in acute decompensated heart failure, cessation of smoking, alcohol and recreational drug use, weight loss; while there is a paucity of large-scale trials of intentional weight loss in heart failure, there is some evidence that weight loss may improve heart failure, quality of life, and exercise capacity.[7] Other important pillar of the treatment of a patient with HF is education to make understand the physiopathology basis of the disease improve the survival and quality of life of the patient. With

this the patient will understand and follow the next recommendation that are important part of the treatment.

Salt restriction: ≤1.5 g/day in stages A and B, ≤3 g/day in stages C and D. Avoidance of potassium-rich foods while taking aldosterone antagonists, like potatoes, tomatoes, beans, yogurt, salmon, tuna, bananas, peaches, prunes. Fluid restriction of 1.5-2 L/day in stage D patients who have edema and/or hyponatremia. Self-monitoring and symptom recognition of an acute decompensation. Daily weight check; patients with a weight gain of >4-5 lbs (>2 kg) within 3 days should consult a doctor. The majority of the patients with heart failure have one or multiple comorbidities, they should be treated to improve and stop the progression of the disease. The most important and common are hypertension, dyslipidemia, diabetes, iron deficiency[8], obstructive sleep apnea, atrial fibrillation, and coronary heart disease. Treatment is based on the stage of heart failure. Additional therapies are added to the baseline medications as symptoms worsen. From stage B onward, device therapy can be considered alongside medical therapy. Start all new medications at the lowest recommended dose and slowly titrate up to the target dose where applicable.

Table 4: Treatment.[1,9,10]

Initial treatment of heart failure	
Stage A	
Treatment of cardiovascular associated risk factors	
Stage B	
Medications	**Indications**
Angiotensin-converting enzyme inhibitor (ACEI)	Every patient with Heart failure with reduced ejection fraction
Angiotensin receptor blockers (ARB)	Patients who cannot tolerate ACEIs
Beta blockers	Improve adverse cardiac remodeling and outcomes in patients with asymptomatic reduced LVEF after myocardial infarction.
Stage C	
Angiotensin receptor-neprilysin inhibitors (ARNIs)	HFrEF and persistent or worsening symptoms despite adequate treatment regimen with first-line drugs. Increased reduction in mortality compared to ACEI or ARB therapy.
Aldosterone antagonists	All HFrEF patients with NYHA class II–IV symptoms and an LVEF of < 35%
Loop diuretics and thiazide diuretics	All patients with fluid retention, begin treatment with loop diuretics to treat volume overload. Thiazides may be added for a synergistic effect.
SGLT2 inhibitors	HFrEF with NYHA class II–IV symptoms. Administered in conjunction with first-line drugs. Reduction in cardiovascular mortality and hospitalization rates in patients with and without type 2 diabetes.
Isosorbide dinitrate (ISDN) and hydralazine	Patients who cannot tolerate ACEIs or ARBs
Stage D	
Most patients require invasive interventions or a change in focus to palliative care. Consider continuous intravenous inotropic support.	

Complications

Heart failure is a complex condition associated with various complications that significantly affect patients' health. Common complications of heart failure include pulmonary edema, a life-threatening condition characterized by fluid accumulation in the lungs, leading to severe breathing difficulties. Arrhythmias, irregular heartbeats that can be fatal, are also prevalent. Renal dysfunction often occurs due to decreased blood flow to the kidneys, leading to decreased urine output and fluid retention. Additionally, liver dysfunction, cognitive impairment, and decreased exercise tolerance are common complications. Chronic heart failure can result in long-term effects, including progressive debilitation, repeated hospitalizations, and reduced life expectancy. Managing these complications requires comprehensive medical care to prevent further deterioration and improve patients' quality of life.[11] Heart failure often results in cardiorenal syndrome, a condition where heart and kidney dysfunction coexist and significantly impact each other's function. In cardiorenal syndrome, the kidney's ability to effectively regulate fluid balance and excrete waste products is compromised due to the heart's reduced ability to pump blood effectively. Reduced cardiac output can lead to decreased blood flow to the kidneys, resulting in impaired renal function. This interdependence between heart and kidney function contributes to a vicious cycle, where

kidney dysfunction further exacerbates heart failure, and heart failure, in turn, adversely affects kidney function. This syndrome complicates the management of heart failure, as treatment strategies often need to address both cardiac and renal issues simultaneously to effectively manage the patient's condition.[12,13]

DISCUSSION

The background reveals that the treatment of heart failure is a nuanced approach, relying on a diverse array of medication classes, each possessing unique advantages and specific applications depending on individual patient characteristics. Heart failure and diabetes mellitus are disease related due to that the diabetes actually in is one of the most prevalent disease in the western population, and is one the principal etiology of heart failure. One of the main mechanism how diabetes cause heart failure is the oxidative stress is involved in the pathophysiology and development of diabetic cardiomyopathy. It has been proposed that downregulation of peroxisome proliferator-activated receptor α (PPARα) induces dysregulation of nitrogen oxides (NOX) proteins, which are predominant isoforms expressed in cardiac tissue and contribute to the development of myocardial hypertrophy. Continue state of hyperglycemia in DM leads to increased ROS production by enhancing mitochondrial oxygen consumption and damaging mitochondrial function. Increased generation of ROS, reduced activity of endogenous antioxidants or both can induce oxidative stress, which is a potent culprit in diabetes mellitus due to inducing β-cell dysfunction and insulin resistance.[14] In the view of mention above, many clinical trials have showed that anti-diabetic drugs have a main roll in prevention, stop progression and treatment on patient with diabetes mellitus and heart failure. There are great variety of anti-diabetic drug, and the majority have shown improvement in mortality and complications in patients with diabetes and heart failure versus placebo. But the group of anti-diabetic drug that have the greatest benefits are Sodium-Glucose Cotransporter-2 (SGLT-2) Inhibitors and Glucagon-Likepeptide-1 (GLP-1) Receptor Agonists.

Natriuresis induced by SGLT-2 inhibitors leads to an increase in the activation of the renin–angiotensin–aldosterone system (RAAS) and a reduction in plasma volume, systemic blood pressure and vascular stiffness via a decrease in the sympathetic system hyperactivation, resulting in increased vasodilatation and improved vascular function. This has also been demonstrated by dapagliflozin's effectiveness on vascular endothelial function and glycemic control via the use of dapagliflozin on early-stage DM type 2 patients, possibly via antifibrotic mechanisms. Natriuresis and osmotic diuresis also increase glomerular afferent arteriolar vasoconstriction, and as a result, intraglomerular hyperfiltration is reduced, leading to decreased proteinuria and increased glycosuria. In glycosuria, the uric acid in plasma is reduced, as are blood glucose levels and potential hyperglycemia-associated toxicity, especially in hyperglycemic patients.[14]

The medical treatment of HF aims to achieve several objectives: reduce overall and/or cardiovascular mortality, decrease hospitalizations, improve functional capacity and quality of life, and prevent or delay the deterioration of cardiac function, meaning to modify the temporal progression of HF. Controlling cardiovascular risk factors (such as hypertension, diabetes mellitus, obesity) and early treatment of comorbidities that facilitate HF progression or trigger it (e.g., ischemic heart disease) are critical to delay HF progression, reduce hospitalizations, and increase survival.[15] Various clinical trials have demonstrated that treatment with angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor antagonists (ARBs), beta-blockers, or mineralocorticoid receptor antagonists significantly reduces morbidity and mortality in patients with HF with reduced ejection fraction (HFrEF). In recent years, other therapeutic targets involved in HF pathogenesis have been identified, and treatments have been developed, among which sacubitril-valsartan (SAC/VAL) should be highlighted. SAC/VAL is a combination of valsartan and sacubitril (a prodrug that inhibits neprilysin, an enzyme responsible for degrading various endogenous vasoactive peptides). SAC/VAL has shown benefits in terms of morbidity and mortality in the Paradigm-HF trial in patients with HFrEF (EF ≤35%). Another therapeutic innovation is the sodium-glucose cotransporter 2 inhibitors (SGLT2is), which in individuals with type 2 diabetes showed a reduction in HF hospitalizations. Recently, research on SGLT2is (dapagliflozin, empaglifozin, and sotagliflozin) in HF patients, regardless of their metabolic profile, has demonstrated their benefit and utility.[15]

In Paraglide-HF a multicentre, double-blind, randomized, active-controlled trial evaluated the effect of sacubitril/valsartan versus valsartan on changes in NT-proBNP, the safety and tolerability in patients with heart failure with a EF above 40% who had been stabilized after a worsening heart failure event. The trial found that S/V leads to greater reduction in plasma NT-proBNP levels compared to valsartan alone.[16] In the DAPA-HF trial, dapagliflozin showed a reduction in events related to HF worsening or cardiovascular death. In the EMPEROR-Reduced trial, empaglifozin showed a reduction in events related to HF hospitalization or cardiovascular death. Current evidence supports the use of SGLT2 inhibitors (dapagliflozin, empaglifozin) in patients with HFrEF <40%.[15] In light of these mentioned strategies, there is an increasing emphasis on a therapeutic approach in the initial stage with the four pillars that have shown reductions in mortality and hospitalizations in HFrEF patients: ACEIs or ARNIs (sacubitril/valsartan), beta-blockers, mineralocorticoid receptor antagonists, and SGLT2 inhibitors, as long as there are no contraindications or intolerance. In cases of congestion, diuretics will be added.[15] Regarding advances in the treatment of HF with preserved ejection fraction (HFpEF), the EMPEROR-Preserved study, in which empaglifozin significantly reduced the primary endpoint (cardiovascular death or HF hospitalization) in patients with HF and EF > 40%,

regardless of diabetic status.[15] It seems logical to implement the drugs that improve survival as early as possible and move away from a stepwise decision-making approach, which may be a barrier to patients benefiting from these treatments.[15] Preserved-HF was a study to evaluate whether dapagliflozin improves symptoms, physical limitations, and exercise capacity in patients with HFpEF. 324 patients with an LVEF ≥45% were included, who were randomized 1:1 to receive Dapagliflozin and placebo. The primary outcome was improvement in the score of the Kansas City Cardiomyopathy Questionnaire Clinical Summary (KCCQ-CS), which quantifies heart failure symptoms, physical function, and quality of life, with the higher the score representing the better health condition. In the group under treatment with Dapagliflozin, an improvement in KCCQ-CS of 5.8 points was observed at 12 weeks.[17]

Emperor: Preserved was a randomized clinical trial that included 5988 patients with LVEF >40%, randomized to receive 1:1 Empaglifozin or placebo. The primary outcome was cardiovascular death or hospitalization for HF, it occurred in 13.8% of patients treated with empaglifozin and 17.1% of patients in the placebo group.[17] The seniors trial investigated the use of nebivolol in elderly patients (≥70 years) with heart failure, the primary outcome being all-cause mortality and hospitalization for cardiovascular causes. A third of the 2128 participants had LVEF > 35%. Follow-up was carried out for 21 months and the primary outcome was observed in 31.1% and 35.3% of patients receiving nebivolol or placebo, respectively. The study concluded that nebivolol was well tolerated and effective in reducing morbidity and mortality in elderly patients.[17] One study evaluated the efficacy of carvedilol versus placebo in patients with HFpFE, analyzing a composite outcome of death and hospitalization for cardiovascular causes. After 3.2 years of follow-up, the primary outcome occurred in 24.2% and 27.2% of patients in the carvedilol and placebo groups, respectively. In the standard dose group (>7.5 mg/day), the primary composite endpoint was significantly reduced compared to placebo, while in the low dose group (≤7.5 mg/day) the same endpoint assessment was comparable to placebo. The study failed to show prognostic benefits after treatment with carvedilol. However, the administration of carvedilol at standard doses was associated with a decrease in the rates of death or hospitalization from cardiovascular causes, so further studies can be performed.[17] The charm-preserved trial studied the efficacy of candesartan, analyzing a composite primary outcome of cardiovascular death and hospitalization for heart failure. 3023 participants with HFpEF were included and randomly assigned to receive candesartan (32 mg once daily) or placebo. Follow-up was carried out for 36.6 months, and the primary outcome was observed in 22% of the patients treated with candesartan and 24% of the placebo group. Although there was no clear benefit, there was a modest reduction in the rate of hospitalization for heart failure, which prompted the ACC/AHA to make a IIb recommendation for the use of angiotensin receptor blockers for the treatment of HFpEF.[17] Topcat, a randomized control trial, evaluated treatment of HFpEF with mineralocorticoid receptor antagonists. 3445 patients, ≥50 years old, with LVEF ≥45% and hospitalization for heart failure within 12 months or elevated BNP levels within 60 days after randomization were included. 1767 patients from the United States, Canada, Brazil, Argentina (America Group) and 1678 participants from Russia/Georgia were included. Two groups were evaluated, one treated with spironolactone (15-45 mg once a day) and another placebo group, for 3.3 years. The primary outcome was a combination of cardiovascular death, aborted cardiac arrest, and hospitalization for heart failure. One-third of the patients in the spironolactone group discontinued treatment due to elevations in serum creatinine and potassium, but remained in the study. The overall incidence of the primary outcome was not reduced with treatment, with events occurring in 18.6% of those receiving spironolactone and 20.4% of the placebo group. Furthermore, there was a lower incidence of hospitalization for heart failure in the sporonolactone group (12%) compared to the placebo group (14.2%). The study was limited by the enrollment of two different populations, the American group and the Russian/Georgian group, since there are variations between these. It was concluded that treatment with spironolactone can improve prognosis by reducing the rates of cardiovascular death and hospitalization for heart failure, although there is a greater risk of hyperkalemia and renal failure.[17]

Paragon-HF performed a prospective comparison between angiotensin-nerlipressin inhibitors compared with ARB in patients with NYHA class II heart failure, with LVEF ≥45%, elevated BNP levels, and structural heart disease. We included 4822 participants with HFpEF who were randomized to receive sacubitril/valsartan (97/103 mg twice daily) or Valsartan (160 mg twice daily). The primary outcome was the reduction in the incidence of hospitalization or death due to heart failure. They were evaluated for 35 months and there were 894 primary events in 526 patients receiving sacubitril/valsartan treatment and 1009 events in 557 patients receiving valsartan. A possible benefit was observed for women and patients with lower EF (45-57%).[17] Diuretics are used to treat symptoms and signs of congestion. Loop diuretics are the first line treatment in cases of acute heart failure. Thiazide diuretics can be added in patients resistant to treatment with loop diuretics. In this patient it´s important to monitor serum electrolytes, mainly potassium.[18] There are four pillars of disease modifying treatment: Angiotensin converting enzyme inhibitors (ACEi)/Angiotensin II receptor blockers (ARB)/Angiotensin Receptor Antagonism with Neprilysin Inhibition (ARNI); mineralocorticoid receptor antagonism; Antagonism of the sympathetic system with selected beta-blockers; Sodium Glucose Cotransporter 2 Inhibitors (SGLT2i).[18] ACE inhibitors are recommended as first line treatment, which have demonstrated reductions in mortality of 23% and of worsening symptoms by 35%. Angiotensin II receptor blockers have been used as second

line treatment by the NICE guidelines because of the cost-effectiveness, but combined ARB + ARNI (Sacubitril/ Valsartan) has recently demonstrated superiority to ACEi and ARBs as monotherapy. Sacubitril/Valsartan has a 16% relative risk reduction in mortality compared with enalapril, and it has also demonstrated rapid symptomatic and quality of life benefits, for this reason guidelines recommend switching from ACEi/ARB to ARNI.[18]

Mineralocorticoid receptor antagonism, such as Spironolactone and Eplerenone, block the effects of aldosterone. They can be useful to prevent hypokalemia, but are contraindicated in patients with hyperkalemia and its essential to monitorize electrolites. Eplerenone has shown to reduce mortality in patients with heart failure after myocardial infraction.[18] Beta blockers have been shown to reduce mortality by 35%. These can worsen acute heart failure, so it is necessary to give appropriate clinical advice when starting treatment. The dose should be escalated to the maximum tolerated, with a goal of a heart rate of 60-70 bpm. Ivabradine can be added in those patients who do not tolerate BBs and continue with >70 bpm, since it can reduce heart rate and thus improve symptoms and mortality.[18] Both dapaglifozin and empaflifozin (SGLT2i) have been shown to reduce worsening heart failure and cardiobascular death by 25% in patients with reduced ejection fraction heart failure, both in patients with and without diabetes. The ACC/AHA and ESC guidelines support the use of SGLT2i in an early stage of heart failure, and its use has also been approved in the UK by the NICE guidelines.[18]

A study was carried out, the DELIVER trial, in which the use of Dapaglifozin was evaluated, compared with placebo, in patients with heart failure with mildly reduced or preserved ejection fraction, in which it sought to demonstrate a reduction in the risk of worsening of heart failure or cardiovascular death. 6263 patients were included in the study, who were randomly chosen to receive Dapaglifozin or placebo. It was found that in the group of patients treated with dapaglifozin there was a worsening of heart failure and cardiovascular death in 512 (16.4%) compared to patients in the placebo group where this occurred in 610 (19.5%). These data support the use of SGLT2 inhibitors as essential therapy in patients with heart failure.[19] The most common comorbidity in all groups was hypertension (HTA), similar to other studies with prevalence rates of up to 90% in patients with HFpEF.[20]

The latest European HF guidelines from 2021 recommend, as first-line treatment in symptomatic patients with HFrEF, the use of sacubitril/valsartan or angiotensin-converting enzyme inhibitors, beta-blockers, aldosterone antagonists, and SGLT-2 inhibitors, and as second-line treatment, the use of vericiguat. In fact, the European HF guidelines suggest that vericiguat treatment should be considered in symptomatic patients who experience HF deterioration despite first-line treatments to reduce the risk of cardiovascular death or HF hospitalization.[20] The European Medicines Agency has approved vericiguat for the treatment of chronic symptomatic HF in adult patients with HFrEF who have stabilized after a recent episode of decompensation requiring intravenous diuretic treatment. However, it is important to note that despite first-line treatments, the residual risk of events remains high. Therefore, the use of new treatments, such as vericiguat, which act on complementary pathophysiological pathways to help reduce the disease burden of HF, is still necessary.[20] Vericiguat is an oral drug that directly stimulates GCs, increasing the availability of intracellular cGMP. This has beneficial effects by restoring the relative deficiency in the nitric oxide-GCs-cGMP signaling pathway through direct and independent stimulation of GCs, synergistically with nitric oxide. The increase in cGMP results in a reduction in left ventricular remodeling, improved myocardial and vascular function, and a decrease in fibrosis and inflammation.[20] Regarding the use of vericiguat, the recommended initial dose is 2.5 mg of vericiguat once daily, with a doubling of the dose approximately every 2 weeks until reaching the target maintenance dose of 10 mg once daily, based on patient tolerance. The treatment is associated with a slight reduction in systolic blood pressure compared to placebo (1-2 mmHg), and electrolyte monitoring is not necessary.[20] Vericiguat has an excellent safety profile, and the fact that renal function and plasma ions do not need to be monitored makes it a straightforward drug to use and titrate in daily clinical practice, including telemedicine consultations, which are crucial in managing patients with HF. Finally, it has been estimated that the use of vericiguat in clinical practice would be cost-effective because it significantly reduces HF hospitalizations (both first episodes and recurrences), which are the main driver of healthcare costs associated with HF.[20]

CONCLUSION

To conclude, as we have seen in this article, the treatment of heart failure has evolved, and there are increasingly better treatment options to improve the quality of life for patients and prevent severe complications. We believe that further research into therapeutic options should continue, with a focus on making these treatments widely accessible to the entire population and minimizing adverse effects.

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