CASE REPORT

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Evaluation of the etiological, clinical, and therapeutic profile of patients with heart failure seen at a university outpatient clinic

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HIGHLIGHTS

What is already known?

- Heart failure has a high mortality and healthcare impact.
- Diverse etiologies of heart failure require multifaceted management.
- Beta-blockers, ACE inhibitors, and diuretics are standard HF treatments

What was shown?

- Dilated cardiomyopathy is the leading cause of heart failure in this study.
- Hypertension and diabetes are common comorbidities.
- Medication combinations improve patient outcomes and reduce hospitalizations.

How can the study aggregate to the literature?

- Provides insights into the Brazilian population's heart failure profile.
- Highlights the importance of integrated therapeutic strategies.
- Contributes to public health policies for heart failure management.

ABSTRACT

Introduction: Heart failure (HF) is a complex and progressive syndrome with high mortality and a significant impact on the healthcare system. Between 2004 and 2014, more than 301,000 deaths were recorded due to HF in Brazilian public hospitals, highlighting the need for precise diagnosis and clinical management. Objective: Analyze the etiological and clinical profiles of patients with heart failure treated at a university outpatient clinic, as well as the therapeutic interventions employed. Method: This retrospective study reviewed the medical records of patients with heart failure treated at a university outpatient clinic from 2021 to 2023. The sample was selected for convenience, and the patients were analyzed based on the etiological profile of heart failure. Results: The sample consisted of 103 patients with a median age of 67 years and a similar sex distribution (51% male). The most common etiology was dilated cardiomyopathy (59%), followed by ischemic (20%), hypertensive (16%), and Chagas disease (5.9%). Main treatments included beta-blockers (85%), angiotensin-converting enzyme inhibitors (ACE inhibitors) or angiotensin II receptor blockers (ARBs) (78%), diuretics (62%), spironolactone (57%), and statins (64%). Common comorbidities included systemic arterial hypertension (84%), type II diabetes mellitus (33%), and coronary artery disease (35%). Conclusion: The study reveals the etiological and therapeutic diversity of HF, highlighting the importance of a multifaceted approach in clinical management. The combination of medications such as beta-blockers, ACE inhibitors/ARBs, diuretics, and statins is essential to reduce hospitalizations and improve the quality of life of patients.

Keywords: Heart Failure; Clinical Epidemiology; Comorbidity.

INTRODUCTION

Heart failure (HF) is characterized as a progressive and complex syndrome, resulting in significant impacts on patients' quality of life, increased burden on healthcare services, and higher early mortality rates. It is estimated that approximately 64 million people worldwide suffer from HF, with a rising prevalence, especially in low- and middle-income countries[1]. The probability that one in five individuals will develop this condition over their lifetime underscores the importance of understanding patients' clinical profiles to guide public policies and improve clinical care[2].

In Brazil, between 2004 and 2014, more than 301,000 deaths attributed to HF were documented in public hospitals[2]. Additionally, HF remains the leading cause of rehospitalization in the country, with a high five-year mortality rate, accounting for approximately 5% of healthcare expenditures[2]. Given this scenario, studying this condition becomes essential for developing more effective prevention and management strategies.

This syndrome arises from multiple causes, often representing the terminal stage of various cardiopathies, making its management challenging[3,4]. In response to reduced cardiac function, the body activates compensatory mechanisms such as the renin-angiotensin-aldosterone system (RAAS) and the sympathetic nervous system, leading to vasoconstriction and sodium and water retention, increasing both preload and afterload. Initially, these compensatory responses help maintain cardiac output, but over time, they contribute to volume overload and ventricular remodeling, worsening myocardial dysfunction[3,4].

These structural changes include cardiomyocyte hypertrophy and fibrosis in HF with preserved ejection fraction (HFpEF), as well as eccentric remodeling in HF with reduced ejection fraction (HFrEF), which leads to marked systolic dysfunction. Inflammation

and endothelial dysfunction are also observed, not only promoting disease progression but also influencing the therapeutic strategies needed to manage HF[3,4].

Although previous studies, such as Nogueira (2010), have explored the clinical and etiological profile of HF in patients from a university hospital, the literature still presents significant gaps regarding the specific characterization of the population in Belo Horizonte and the southeastern region. The variability in risk factors and causes of HF across different regions of Brazil highlights the importance of studies focused on specific areas, such as Belo Horizonte, which have not yet been conducted. Understanding local particularities is essential to support evaluation, research, public health policies, and healthcare, as well as to guide more effective prevention and treatment strategies[5].

HF is primarily categorized based on the left ventricular ejection fraction (LVEF), a crucial parameter obtained through transthoracic echocardiography (TTE)[6]. Patients with normal LVEF (≥ 50%) have HF with preserved ejection fraction (HFpEF); reduced LVEF (< 40%) indicates HF with reduced ejection fraction (HFrEF); and LVEF between 40-49% characterizes HF with mildly reduced ejection fraction (HFmrEF).

HF can also be classified based on functionality according to the New York Heart Association (NYHA), which considers exercise tolerance. Patients in Class I do not experience discomfort during routine daily activities; in Class II, they have mild limitations in usual activities due to symptoms; in Class III, symptoms occur during light activities and improve with rest; and in Class IV, symptoms are disabling, occurring even at rest[6].

Therefore, this study aims to analyze the etiological and clinical profiles of HF patients treated at a university outpatient clinic, as well as the therapeutic interventions employed.

METHOD

Study Design

This is an etiological survey study with a retrospective, observational, and descriptive analysis, conducted through the review of medical records of HF patients treated at a university outpatient clinic in the city of Belo Horizonte.

Sample

The inclusion criteria adopted were: patients over 18 years of age, of both sexes, diagnosed with HF based on the NYHA functional classification or echocardiography. The diagnosis could have been established either during follow-up or before the patient arrived at the service. Patients included had to have been followed for at least six months within the period from January 2021 to July 2023.

Patients with diagnostic uncertainty, incomplete medical records, or more than three missing variables were excluded from the study. In total, 286 records were excluded for not meeting the study's exclusion criteria. After selection, a final sample of 103 records met the requirements for complete analysis.

Study Environment Characterization

This study was conducted at a university outpatient clinic that provides a cardiovascular care pathway specifically for treating cardiac conditions that require continuous monitoring, such as heart failure (HF). This setting offers specialized and ongoing follow-up, emphasizing planned interventions and longitudinal management. In contrast to the tertiary hospital described by Nogueira et al. (2010), which supports the management of acute and severe cases, the outpatient clinic used in this study focuses on chronic disease management and the long-term control of HF patients, prioritizing preventive and multidisciplinary practices. This comparison underscores the distinct demands and clinical profiles managed in each type

of setting, enriching the etiological and therapeutic analysis across studies.

Instruments

The data used in this study were obtained from electronic medical records of patients treated at the university outpatient clinic. Data collection was carried out through a structured review of medical records, using a standardized Excel spreadsheet to organize the information. The researchers responsible for data collection followed a standardized protocol agreed upon by all team members to ensure data uniformity and consistency. The collected data were stored in electronic databases, ensuring security and easy access for future analyses. To maintain patient anonymity, records were identified only by numerical codes, ensuring confidentiality and compliance with ethical research guidelines. These procedures guarantee the integrity and quality of the data, allowing for an accurate analysis of the etiological, clinical, and therapeutic characteristics of HF patients treated at the university outpatient clinic.

Procedures

The data were organized in a standardized spreadsheet in Excel, developed by the researchers, including variables such as age, sex, functional class and/or LVEF from TTE, etiology, physical examination data (systolic blood pressure, diastolic blood pressure, heart rate, third heart sound, pulmonary rales, lower limb edema, jugular venous distension), associated comorbidities, and prescribed medications.

HF etiologies were classified into four major groups: Chagas cardiomyopathy, hypertensive cardiomyopathy, ischemic cardiomyopathy, and dilated and other cardiomyopathies. The final sample consisted of 103 medical records selected by convenience sampling, ensuring the necessary diversity and comprehensiveness for the study.

Statistical Analysis

Statistical analysis was performed using RStudio software (version 1.3.1073, Boston, United States). Categorical variables were presented as absolute and relative frequencies and compared using Fisher's exact test, which is particularly suitable for low expected frequencies. Quantitative variables were analyzed using the median and interquartile range (IQR). The Shapiro-Wilk test was applied to assess data normality. To compare study groups, the Kruskal-Wallis test was used when appropriate. A significance level of 5% was considered for all statistical tests. To evaluate the statistical significance of multiple comparisons, Holm's adjustment was applied, ensuring greater precision in the results.

Ethical Considerations

This study adhered to the ethical guidelines established by Resolution 466/12 of the Brazilian National Health Council. The project was approved by the Research Ethics Committee of the Faculdade Ciências Médicas de Minas Gerais (CEPCM-MG) under CAAE 67119822.9.0000.5134 and was granted an exemption from obtaining the Informed Consent Form (ICF).

RESULTS

The sample consisted of 103 individuals, with a median age of 67 years (interquartile range: 57–75 years) and a similar sex distribution (49% female, 51% male). Transthoracic echocardiography was used in 96% of cases to confirm HF (Chart 1).

The median ejection fraction was 42% (range: 33%–54%). Among the patients, 45 (46%) had reduced ejection fraction (< 40%), 17 (18%) had mildly reduced ejection fraction (40%–49%), and 35 (36%) had preserved ejection fraction (> 50%). In six cases, the ejection fraction was not determined, indicating varying degrees of ventricular function impairment (Chart 1).

Chart 1. General Characteristics of Heart Failure Patients (n = 103)

Sample Characterization	
	Mediana [AIQ]
Age (years)	67 [57, 75]
	n (%)
Sex	
Female	50 (49%)
Male	53 (51%)
Etiology	
Chagas-related	6 (5%)
Dilated and others	61 (59%)
Hypertensive	16 (16%)
Ischemic	20 (20%)
Comorbidities	
HTN	87 (84%)
T2DM	34 (33%)
CAD	36 (35%)
Dyslipidemia	48 (47%)
Atrial fibrillation	14 (14%)
Chronic kidney disease	14 (14%)
Anemia	4 (3.9%)
Valvular disease	22 (21%)
Stratified Ejection Fraction	
<40%	45 (46%)
40-49%	17 (18%)
>50%	35 (36%)
Desconhecido	6 (5%)

HTN – Hypertension; **T2DM** – Type 2 Diabetes Mellitus; **CAD** – Coronary Artery Disease; **IQR** – Interquartile Range.

Source: Developed by the authors.

Most participants were classified as NYHA Class II (11%) and Class III (9%), with only 6% in Class I. In 74 cases, the NYHA classification was unknown. The median systolic blood pressure was 130 mmHg (range: 120–146 mmHg), and the median diastolic pressure was 80 mmHg (range: 75–90 mmHg). The median heart rate was 72 bpm (range: 63–80 bpm), with 19 cases lacking available data. Most participants did not present a third heart sound (99%) or pulmonary rales (100%), but 21% had lower limb edema. Jugular venous distension was negative in all cases (Chart 2).

The most prevalent etiology in the sample was dilated cardiomyopathy, accounting for 59% of cases, followed by ischemic heart disease, which represented 20% of cases. Hypertensive and Chagas-related etiologies were less frequent, observed in 16% and 5.9% of patients, respectively. Among comorbidities related to etiologies, hypertension (HTN) was present in all patients with Chagas and hypertensive cardiomyopathy and 81% and 80% of those with dilated and ischemic cardiomyopathy, respectively. Another chronic disease, type 2 diabetes mellitus (T2DM), was observed in 36% of patients with dilated cardiomyopathy, 19% with hypertensive cardiomyopathy, and 45% with ischemic cardiomyopathy, without statistical significance (p = 0.12). Coronary artery disease (CAD) showed a strong association of 95% with ischemic etiology, 25% with hypertensive etiology, 20% with dilated cardiomyopathy, and 17% with Chagas-related cardiomyopathy (p < 0.001) (Chart 2).

Additionally, dyslipidemia occurred in 33% of Chagas-related cases, 42% of dilated cardiomyopathy cases, and 38% and 70% of hypertensive and ischemic etiologies, respectively. Conversely, chronic kidney disease was associated with dilated and ischemic cardiomyopathy in 17% and 20% of cases, respectively. Furthermore, anemia was observed in 5.1% of patients with dilated HF and 5.0% of those with ischemic HF. Valvular diseases were identified in all etiology groups, with a prevalence of 33% in Chagas-related HF, 24% in dilated HF, 19% in hypertensive HF, and 10% in ischemic HF (Chart 2).

Chart 2. Clinical Characteristics of Patients According to the Main Etiology of Heart Failure

Characteristics	Total, N = 1011	Etiology				
		Chagas related, N = 61	Dilated and others, N = 591	Hypertensive, N = 161	Ischemic, N = 201	P-Value 2
Age	66 (57, 74)	67 (59, 72)	64 (54, 76)	69 (65, 73)	65 (57, 73)	0.9
Sex						0.3
Female	48 (48%)	2 (33%)	25 (42%)	11 (69%)	10 (50%)	
Male	53 (52%)	4 (67%)	34 (58%)	5 (31%)	10 (50%)	
Ejection fraction	42 (33, 54)	47 (42, 51)	43 (32, 56)	44 (35, 62)	39 (34, 48)	0.7
Systolic BP	130 (120, 146)	126 (113, 134)	130 (120, 153)	142 (120, 150)	130 (127, 140)	0.5
Diastolic BP	80 (75, 90)	82 (76, 95)	80 (70, 90)	82 (80, 91)	80 (80, 86)	0.5
Heart rate (bpm)	72 (63, 80)	70 (68, 80)	72 (63, 80)	76 (66, 81)	75 (63, 81)	>0.9
Third heart sound	1 (1.0%)	0 (0%)	0 (0%)	0 (0%)	1 (5.0%)	0.4
Pulmonary rales	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	
Lower limb edema	20 (19%)	0 (0%)	14 (23%)	4 (25%)	2 (10%)	0.9
Jugular venous distension	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	
HTN	86 (85%)	6 (100%)	48 (81%)	16 (100%)	16 (80%)	0.2
T2DM	33 (33%)	0 (0%)	21 (36%)	3 (19%)	9 (45%)	0.12

Characteristics	Total, N = 1011	Etiology				
		Chagas related, N = 61	Dilated and others, N = 591	Hypertensive, N = 161	Ischemic, N = 201	P-Value 2
CAD	36 (36%)	1 (17%)	12 (20%)	4 (25%)	19 (95%)	<0.001
Dyslipidemia	47 (47%)	2 (33%)	25 (42%)	6 (38%)	14 (70%)	0.13
Atrial fibrillation	13 (13%)	1 (17%)	7 (12%)	2 (13%)	3 (15%)	0.9
Chronic kidney disease	14 (14%)	0 (0%)	10 (17%)	0 (0%)	4 (20%)	0.2
Anemia	4 (4.0%)	0 (0%)	3 (5.1%)	0 (0%)	1 (5.0%)	>0.9
Valvular disease	21 (21%)	2 (33%)	14 (24%)	3 (19%)	2 (10%)	0.5

BP – Blood pressure in mmHg (millimeters of mercury); bpm – beats per minute; [1] Median (IQR); n (%), [2] Kruskal-Wallis test; Fisher's exact test.

Source: Developed by the authors.

Among the patients, 85% used beta-blockers, while 78% were treated with angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor blockers (ARBs). Diuretics were used by 62% of patients, and spironolactone by 57%. Statins were prescribed to 64% of patients, while vasodilators and calcium channel blockers had limited prescription, each used by 15% of patients. Anticoagulants or antiplatelet agents were used by 52% of patients. Finally, only 8% of patients were receiving treatment with the Sacubitril-Valsartan combination (Chart 3).

Chart 3. Frequency of Heart Failure Treatments Used (n = 103)

Treatments	n (%)
Anticoagulant | Antiplatelet agent	54 (52%)
Beta-blockers	88 (85%)
Calcium channel blockers	16 (15%)
Diuretics	64 (62%)
Spironolactone	59 (57%)
Statins	66 (64%)
ACEIs or ARBs	81 (78%)
Sacubitril + Valsartan	9 (8%)
Vasodilators	16 (15%)

Source: Developed by the authors.

DISCUSSION

Our data revealed that, in our sample, the median age of participants was 67 years, with a slight predominance of males (51%). Heart failure with reduced ejection fraction had a prevalence of 46%. Dilated cardiomyopathy emerged as the most common etiology, affecting 59% of patients, followed by ischemic (20%), hypertensive (16%), and Chagas-related cardiomyopathy (5.9%). Hypertension (HTN) and type 2 diabetes mellitus (T2DM)

were frequently observed comorbidities, particularly among patients with ischemic etiology. Most patients were undergoing treatment with beta-blockers (85%) and ACEIs or ARBs (78%), although only 8% were using the innovative Sacubitril-Valsartan combination.

Clinical profile

The absence of pulmonary rales and jugular venous distension, along with the low prevalence of gallop rhythm, may suggest effective HF management in our sample. The absence of clinical signs such as crackles is associated with better clinical outcomes[8]. Although 19% of patients presented lower limb edema, the lack of investigation into other classic signs limits a comprehensive understanding of congestion. The literature suggests that the combination of natriuretic peptides and echocardiography is crucial for a more accurate prognostic assessment, particularly in patients with HFpEF. The low prevalence of these signs may be interpreted as an indication of adequate outpatient follow-up[7].

Most HF patients are 65 years or older[8]. However, recent reports indicate an increasing incidence of the condition among younger individuals, attributed to the rise in early-onset risk factors such as obesity. In our study, the median age of patients was 67 years, with an interquartile range of 57 to 75 years, reflecting the expected age range for HF. Although HF is more prevalent in women, particularly after 69 years of age[9], our analysis revealed an almost equal distribution between sexes, with 49% women and 51% men. Physical examination data showed that 19% of patients had lower limb edema and 1% presented gallop rhythm, while pulmonary rales and jugular venous distension were not observed. The mean heart rate was 72 bpm, indicating variability in the clinical presentation of HF patients.

In patients with HFpEF, HF according to the Framingham criteria was present in one-quarter of cases, with 10% classified as NYHA Class IV. Most patients met the criteria for natriuretic peptides and echocardiography in diagnosing HFpEF. The persistence of Framingham criteria during follow-up was associated with an increased risk of mortality and HF hospitalization, highlighting the importance of clinical risk assessment and identifying patients for future research on HFpEF[8].

Etiological profile

The most common HF etiology in our sample was dilated cardiomyopathy, contrasting with Nogueira (2010), who observed a higher prevalence of Chagas-related etiology. This difference may be attributed to the time and location of the studies[10]. The original study was conducted in Goiás in 2010, during intensified efforts by the Ministry of Health to control Chagas disease.

Ischemic etiology, previously considered highly prevalent[10], accounted for only 20% of the analyzed cases. This may be explained by the expansion of percutaneous coronary intervention (PCI), which has improved the treatment of acute myocardial infarction (AMI), leading to better myocardial preservation and a reduction in HF cases due to ischemia[11]. The etiologies reflect different HF causes, including dilated cardiomyopathy, CAD, HTN, and

Chagas disease. The relationship between hypertensive HF (16%) and dilated HF (58%) is close. HTN, which affects 30% of the Brazilian population[12], can lead to left ventricular concentric hypertrophy and, over time, progress to dilated HF[13].

Among HF patients, 33% had T2DM, with variations across etiologies: 0% in Chagas-related cases, 45% in ischemic cases, 19% in hypertensive cases, and 36% in dilated and other etiologies. Studies suggest a causal relationship between genetic predisposition to T2DM, insulin resistance (IR), and HF, exacerbated

by the presence of CAD[14]. The high prevalence of T2DM, particularly in dilated HF, may indicate an interconnection between these conditions, as supported by the literature[14].

A significant association was observed between CAD and HF, with a prevalence of 36%, varying across etiologies: 17% in Chagas-related cases, 20% in dilated cases, 25% in hypertensive cases, and 95% in ischemic cases. Studies indicate that coronary flow alterations in HF result from both direct and indirect mechanical mechanisms, influenced by RAAS and the sympathetic nervous system, leading to coronary circulation dysfunction even in the absence of obstructive CAD[15].

In HFPEF, key mechanisms include myocardial abnormalities, microcirculatory compression, capillary rarefaction, endothelial dysfunction, and fibrosis. In HFrEF, the "microvascular paradigm" is currently the most accepted model, replacing the "myocardial overload" theory. Myocardial ischemia has an unfavorable prognosis, highlighting the need for targeted management strategies for this condition. The high prevalence of CAD in dilated HF in our study suggests the presence of these mechanisms, reinforcing the complexity of the CAD-HF interaction and the importance of tailored management approaches[15].

Dyslipidemia plays a significant role in HF etiology, especially when mediated by CAD. In this study, 47% of patients had dyslipidemia. These findings emphasize the relevance of dyslipidemia as both a direct and indirect risk factor for HF. The pathogenesis of CAD in dyslipidemia is well understood, with advances in genomics identifying monogenic and polygenic variants that influence lipid levels. Elevated LDL-C, triglycerides, and ApoB levels have been associated with a higher HF risk, whereas high HDL-C levels are inversely related to risk[16]. Although the direct relationship between dyslipidemia and HF is less frequent, lipid control is crucial in CAD prevention, reducing HF incidence. The use of statins is recommended for high-risk individuals, emphasizing their importance in preventing and mitigating HF progression[16]. Thus, effective dyslipidemia management not only prevents CAD but also serves as a vital strategy to reduce HF burden.

In our study, comorbidities such as atrial fibrillation (13%), chronic kidney disease (14%), anemia (4%), and valvular diseases (21%) had low prevalence and did not show significant statistical differences among HF etiologies. These data suggest that, although these comorbidities are clinically important, they did not play a relevant role in distinguishing HF etiologies in our sample, possibly reflecting a lower influence of these conditions in our specific study population.

HF is a multifactorial condition with various etiologies, including dilated cardiomyopathy, CAD, HTN, and Chagas disease. Our study revealed that, despite these etiological variations, no statistically significant differences in ejection fraction were observed between groups after Holm's adjustment. This suggests that, regardless of the underlying cause, clinical outcomes in terms of ventricular function may be similar. These findings highlight the complexity of HF and the need for integrated treatment approaches that account for its diverse causes while maintaining the common goal of improving patient outcomes.

Therapeutic profile

HF requires a multifaceted therapeutic approach to improve patients' quality of life and reduce hospitalizations. In our study, we analyzed the therapeutic profile of HF patients treated at a university outpatient clinic, highlighting the frequent use of diuretics (62%), spironolactone (57%), ACEIs or ARBs (78%), and beta-blockers (85%). These findings underscore the importance of these medications in HF management, aligning with best clinical practices.

Diuretics, despite the lack of placebo-controlled trials, are essential in treating both HFpEF and HFrEF, demonstrating efficacy in reducing hospital readmissions[17]. Spironolactone, a mineralocorticoid receptor antagonist, has shown benefits in reducing HF-related readmissions[17].

ACEIs were the first class of drugs to demonstrate a significant reduction in mortality and morbidity in HFrEF patients. In addition to improving symptoms, they are widely recommended for all patients with this condition unless specific contraindications exist[18]. Beta-blockers also play a crucial role in HFrEF management, often used in combination with ACEIs and diuretics. They have proven efficacy in reducing both mortality and hospitalizations while providing additional symptom relief[18].

Regarding digoxin, although this drug has a long history in HF treatment, particularly in patients with HFrEF associated with atrial fibrillation, it was not prescribed in our sample. This absence may be related to current guidelines recommending its use only in cases where symptoms are not controlled by first-line medications such as ACEIs, beta-blockers, and mineralocorticoid receptor antagonists. Digoxin may reduce HF hospitalizations and improve symptoms in some patients, but its use requires caution due to its narrow therapeutic window and risk of toxicity, which may explain its low prescription rate[19, 20, 21].

Statin use was observed in 64% of patients, reflecting its importance in preventing cardiovascular events, particularly in patients with dyslipidemia. Studies indicate that statins reduce the risk of developing HF by preventing myocardial infarction and reducing myocardial ischemia[22]. However, in patients with established HF, statins do not reduce the risk of cardiovascular death, which is primarily caused by pump failure and ventricular arrhythmias[22].

Vasodilators, used by 15% of patients, help reduce peripheral vascular resistance and improve blood flow. However, among randomized trials on HF and vasodilator efficacy (V-HeFT I, V-HeFT II, FIRST, PROFILE), only the V-HeFT I trial demonstrated mortality reduction; thus, their indication in HF management does not offer significant benefits[23].

Sacubitril/valsartan was used by 8% of the patients in our study. This treatment has proven effective in reducing hospitalizations and improving symptoms in HFrEF patients. The PARADIGM-HF trial demonstrated a significant 20% reduction in the risk of cardiovascular death or HF hospitalization with sacubitril/valsartan compared to enalapril, emphasizing the need for a paradigm shift in clinical practice to implement this life-saving therapy[24].

In HFrEF patients with NYHA class II to III symptoms, a combination therapy including a neprilysin inhibitor with an angiotensin receptor blocker (such as sacubitril/valsartan), beta-blockers, mineralocorticoid receptor antagonists, and SGLT2 inhibitors is recommended. The sequential introduction of each drug facilitates the identification of potential adverse effects, although some experts advocate for the simultaneous initiation of multiple drugs. Regardless of systolic function recovery, pharmacological therapy for HFrEF is maintained indefinitely[25].

Our study highlights the complexity and importance of a comprehensive therapeutic approach in HF management. The combination of diuretics, spironolactone, ACEIs/ARBs, beta-blockers, statins, vasodilators, and sacubitril/valsartan reflects best clinical practices, providing significant benefits in reducing hospitalizations and improving symptoms. These therapeutic strategies are crucial for enhancing the quality of life of HF patients and reducing the mortality associated with the condition.

Limitations

As a retrospective and observational study, this research is subject to the inherent limitations of medical record analysis, such as the availability and quality of recorded data. Undoubtedly, the quality of clinical records in medical charts is a critical issue due to the difficulty in accessing information that validates adherence to the study's inclusion criteria. Consequently, a convenience sample was necessary. The lack of essential data, such as NYHA classification and heart rate, is concerning. This gap in medical records may compromise clinical management, patient safety, and the right to a competent and accessible health history. Although poor-quality records are a significant issue in healthcare, studies investigating their causes are still lacking[26]. Additionally, the convenience sampling method may introduce selection biases, potentially limiting the generalizability of the results to other populations. The absence of some clinical data in medical records may also have influenced the outcome analysis.

CONCLUSION

This study analyzed the etiological and clinical profiles of HF patients treated at a university outpatient clinic and outlined the therapeutic interventions employed in the studied context. The predominant etiology was dilated cardiomyopathy, followed by ischemic and hypertensive causes, highlighting the impact of changes in Chagas disease prevalence and the importance of strict control of HTN and ischemia. The population was predominantly elderly, with a significant prevalence of comorbidities and a low occurrence of congestion signs, suggesting an effective outpatient approach to clinical management. This profile reinforces the need for management strategies focused on controlling comorbidities and maintaining clinical stability in high-risk patients.

The therapeutic profile revealed a multifaceted approach, emphasizing the use of diuretics, spironolactone, ACEIs/ARBs, and beta-blockers, which are essential for symptom control and mortality reduction. The use of these medications reflects therapeutic advancements that improve quality of life and reduce hospitalizations, particularly in HFrEF.

A detailed understanding of the clinical and etiological profiles of HF is crucial for guiding public health policies and optimizing care, especially in overburdened healthcare systems, aiming for more efficient interventions that meet the specific needs of this population.

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