

Furosemide Dose Progression after Sodium-Glucose Transporter 2 Inhibitors in Advanced Heart Failure

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Abstract

Background: Studies on Sodium–Glucose cotransporter-2 inhibitors (SGLT2i) in the treatment of heart failure (HF) investigate their potential as a therapeutic alternative for managing congestion and their influence on diuretic efficacy in acute decompensated HF.

Objectives: To analyze the impact of introducing SGLT2i in patients with HF, with emphasis on the hypothesis of a potential reduction in outpatient furosemide use over time, as well as concomitant changes in renal function and clinical signs of congestion.

Methods: Retrospective study of 85 patients with advanced HF under outpatient follow-up between January 2022 and July 2025, including those on furosemide at the time of SGLT2i prescription and excluding those without follow-up visits. Comparative analysis was performed on furosemide doses, signs of congestion, and changes in renal function across four subsequent visits.

Results: The sample was predominantly male (56.5%), with a high prevalence of HF with reduced ejection fraction (82.4%). After the introduction of SGLT2i, results showed a reduction in jugular venous distension and lower-limb edema, accompanied by increased creatinine levels and a rise in mean furosemide dose from 59.66 ± 4.87 mg to 79.05 ± 7.28 mg ($p = 0.003$).

Conclusion: Following the introduction of SGLT2i, an increase in the mean furosemide dose was observed. This finding was associated with elevated creatinine levels, despite modest improvement in clinical signs of congestion, highlighting the dynamic scenario analyzed and the need for prospective studies with follow-up across different stages of HF.

Keywords: Heart Failure; Diuretics; Furosemide; Sodium-Glucose Transporter 2 Inhibitors.

Introduction

Sodium-Glucose cotransporter-2 Inhibitors (SGLT2i) have emerged as a therapeutic class in the treatment of heart failure (HF), initially for glycemic control in patients with type 2 diabetes mellitus. Currently, they demonstrate broad cardiovascular benefits, as evidenced in clinical trials such as DAPA-HF and EMPEROR-Reduced.^{1,2} SGLT2i act on the renal proximal tubule, promoting glucosuria, natriuresis, and diuresis, with the potential to relieve volume overload frequently present in patients with HF.^{3,4} In addition to these hemodynamic effects, SGLT2i are associated with blood pressure reduction, improved

endothelial function, weight loss, and favorable effects on cardiac remodeling.^{5,6}

The natriuretic and hemodynamic effects of SGLT2i are being investigated in the context of congestion relief through studies exploring their potential to enhance diuretic efficacy in patients with acute decompensated HF. Thus, beyond the documented benefits in reducing cardiovascular events and hospitalizations, SGLT2i emerge as possible therapeutic alternatives for the management of congestion. Given these findings, the hypothesis arises that SGLT2 inhibitors could help relieve volume overload and contribute to reducing the dose of diuretics used in patients with HF, thereby favoring a more individualized management approach and identifying patients who would benefit from their early introduction, expanding therapeutic options and improving quality of life.

This study aimed to analyze the impact of introducing SGLT2 inhibitors in the management of HF, with emphasis on the hypothesis of a potential reduction in outpatient furosemide use over time. In addition, it sought to longitudinally monitor clinical changes in congestion signs and laboratory alterations in renal function.

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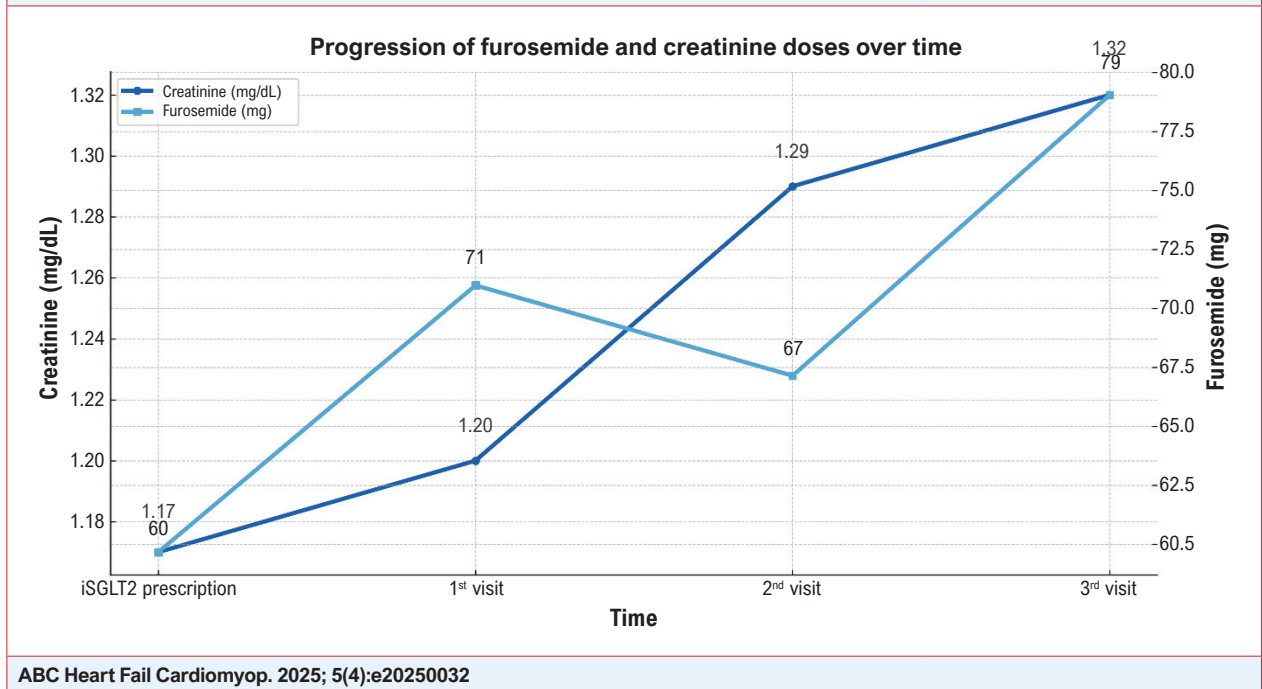
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Central Illustration: Furosemide Dose Progression after Sodium-Glucose Transporter 2 Inhibitors in Advanced Heart Failure



ABC Heart Failure & Cardiomyopathy



Furosemide dose progression after the introduction of sodium-glucose cotransporter-2 inhibitors in advanced heart failure.

Methods

Study design

This is a retrospective study, with data collected from electronic medical records of patients treated at the advanced HF outpatient clinic between January 2022 and July 2025.

Advanced HF was defined in patients followed at the specific outpatient clinic analyzed, who met criteria such as: persistent symptoms (NYHA class III or IV); severe ventricular dysfunction, such as Left Ventricular Ejection Fraction (LVEF) $\leq$ 30% and isolated right HF; episodes of pulmonary or systemic congestion requiring high doses of intravenous diuretics, or episodes of low output requiring inotropes or vasoactive drugs, or malignant arrhythmias requiring emergency care within the past 12 months; severely reduced exercise capacity, with inability to exercise or poor performance on the six-minute walk test.

Inclusion criteria

Patients with a diagnosis of HF who attended outpatient consultations and were on furosemide at the time of initiating SGLT2 inhibitors (dapagliflozin 10 mg/day or empagliflozin 10 mg/day) between January 2022 and July 2025 were included.

Exclusion criteria

Patients who did not have a follow-up visit after initiating SGLT2 inhibitors, those whose follow-up occurred at intervals

shorter than 30 days between visits, or those without sufficient medical record data for study analysis were excluded.

Data collection

Data collection began after approval of the project by the Research Ethics Committee on September 17, 2024, under opinion number 7,081,707. Data were collected sequentially by consultation date and time within the study period, using a specific Google Forms instrument and subsequently stored in an Excel spreadsheet. Information included the prescribed furosemide dose at the time of SGLT2 initiation and during the first three follow-up visits. Clinical data were also collected, such as left ventricular ejection fraction, renal function, epidemiological characteristics, HF etiology, hospitalization history, physical examination signs of congestion (e.g., jugular venous distension, lower limb edema, pulmonary crackles on auscultation), and reported symptoms stratified according to the New York Heart Association (NYHA) functional classification.

Data analysis

The collected data were stored in a Microsoft Excel spreadsheet. Data analysis was performed using SPSS software (IBM SPSS Statistics, version 29.0.2.0). The significance level adopted for all analyses was $p < 0.05$. Continuous variables were expressed as mean $\pm$ standard deviation (SD). The normality distribution of these variables was assessed using the Shapiro–Wilk test, chosen for its sensitivity in small to

moderate samples. Categorical variables were described as absolute and relative frequencies (%).

Comparison of continuous variables

The progression of total furosemide dose and creatinine levels across visits was analyzed using non-parametric tests for paired data, since both variables did not present a normal distribution.

Initially, the Friedman test was used to assess differences across the four evaluated time points (initial prescription, first visit, second visit, and third visit). When a global effect was identified, paired comparisons were performed using the Wilcoxon test, with Bonferroni correction for multiple comparisons.

In addition, specific comparisons between consecutive time points were analyzed using the unadjusted paired Wilcoxon test, with the aim of describing early trends throughout follow-up.

Comparison of categorical variables

Binary variables

The analysis of binary categorical variables, such as the presence of jugular venous distension, lower limb edema, and pulmonary crackles, between two time points was performed using the McNemar test. This test was applied to assess proportional changes in paired data.

Variables with more than two categories

HF functional class, based on NYHA criteria and categorized as I, II, III, and IV, was analyzed between the prescription and first visit using the Marginal Homogeneity test, which is appropriate for paired ordinal categorical variables.

Interval between visits

The mean interval between visits was calculated as mean $\pm$ SD and included to contextualize the clinical and laboratory changes observed throughout the follow-up period.

Results

Medical records of patients treated at a cardiology outpatient clinic were analyzed over a period of one and a half years. Of the 150 records initially evaluated, 65 were excluded due to intervals between visits shorter than 30 days. Thus, 85 records of patients with advanced HF who were using furosemide at the time of SGLT2 inhibitor prescription were included in the analysis.

Most patients were male (56.6%), predominantly White (88.2%), with a mean age of 68.9 ± 1.4 years. Table 1 provides a detailed description of the clinical characteristics, main comorbidities, and chronic medications of these patients at the time of SGLT2 initiation. Notably, the prevalence of HFrEF was 82.4%, with a mean LVEF (Teichholz method) of $32.9 \pm 1.5\%$. It is worth emphasizing that LVEF was obtained using the Teichholz method, as it was the most readily available in the medical records.

In the comparative analysis of furosemide doses across visits, as illustrated in Figure 1, a progressive trend of increasing absolute dose was observed throughout follow-up. At the time of SGLT2 inhibitor initiation, all patients were using furosemide, with a mean dose of 59.66 ± 4.87 mg. In the initial paired analysis using the Wilcoxon test, a statistically significant difference was found between the prescription time point and the first evaluation, when the mean dose reached 70.97 ± 5.70 mg ($p = 0.001$). However, this difference did not remain statistically significant after correction for multiple comparisons using the Bonferroni method. Consistently, in the global analysis across the four evaluated time points, despite a progressive increase in mean dose, reaching 79.05 ± 7.28 mg at the last visit, no statistically significant differences were observed in the adjusted paired comparisons.

Regarding renal function, a progressive increase in serum creatinine levels was observed throughout the follow-up visits, as shown in Figure 2 ($p = 0.002$). This increase was statistically significant between the time of SGLT2 inhibitor prescription and the first follow-up visit ($p = 0.023$), as well as between the first and second visits ($p = 0.007$).

The Friedman test demonstrated a significant difference among the four time points evaluated ($p = 0.002$). In the paired comparisons adjusted by the Bonferroni method, a trend was observed between the first and third visits (adjusted $p = 0.069$), without statistical significance after correction.

Regarding clinical signs, the progression of findings was assessed between the time of SGLT2 inhibitor prescription and the first, second, and third subsequent visits. The mean interval between these visits was 111.96 ± 8.7 days from prescription to the first visit, and 125.58 ± 11.15 days between the first and second visits.

The presence of jugular venous distension was observed in 32.9% of patients at the time of SGLT2 inhibitor prescription, decreasing to 24.7% at the first visit and 27.1% at the second. Lower-limb edema showed a lower incidence throughout follow-up, being present in 35.3% of patients initially, followed by 23.5% and 22.4%. On pulmonary auscultation, crackles were found in 10.9% of patients and remained unchanged during the entire follow-up period.

The NYHA functional classification showed a trend toward improvement: the proportion of patients in functional class I increased from 25.7% to 31.4%, while in class IV it remained at 2.9% between the time of prescription and the first visit.

Discussion

The use of SGLT2 inhibitors in HF has been extensively studied, with consistent benefits in reducing cardiovascular mortality and hospitalizations, as demonstrated in clinical trials such as DAPA-HF, EMPEROR-Reduced, and EMPULSE.⁵⁻⁷ In addition to cardiovascular outcomes, these agents have shown potential nephroprotective effects, as studies reported a slower decline in Glomerular Filtration Rate (GFR) in treated groups compared with placebo.^{6,7} Trials such as EMPAG-HF and EMPA-RESPONSE-AHF also demonstrated that early use of SGLT2 inhibitors

Table 1 – Characteristics of the sample (n=85)

Variables	
Age (years)	68.94 ± 1.4
Male sex - %	56.5% (48)
Race	
White	88.2%
Black	1.2%
Pardo	5.9%
Others	1.2%
Not informed	3.5%
School attainment	
Incomplete primary education	11.8%
Primary education	18.8%
Incomplete higher education	3.5%
Higher education	15.3%
Illiterate	1.2%
Not informed	49.4%
Etiology of HF	
Ischemic	47.1% (40)
Hypertension	4.7% (4)
Valvar	8.2% (7)
Dilated cardiomyopathy	4.7% (4)
Myocarditis	5.9% (5)
Chagas disease	0%
Alcoholic	2.4% (2)
Chemotherapy-induced toxicity	1.2% (1)
Others	5.9% (5)
Idiopathic	20% (17)
Comorbidities	
SAH	80% (68)
DM	48.2% (41)
CAD	44.7% (38)
DSLPL	23.5% (20)
CKD	3.5% (3)
AF	29.4% (25)

COPD	10.6% (9)
Smoking	8.2% (7)
Alcohol consumption	4.7% (4)

NYHA (pre-iSGLT2)

Not informed	12.9% (11)
I	18.8% (16)
II	42.4% (36)
III	23.5% (20)
IV	2.4% (2)

Medications (pre iSGLT2)

ARB	36.5% (31)
INRA	18.8% (16)
ACEI	29.4% (25)
Beta-blocker	30.6% (26)
Spironolactone	71.8% (61)
Finerenone	2.4% (2)
Furosemide (mean dose, mg)	59.66 ± 4.87

Others

LVEF (%Teichholz)	32.9 ± 1.5
Creatinine (mg/dL)	1.17 ± 0.04
SPB (mmHg)	122.7 ± 2.6
DPB (mmHg)	78.29 ± 1.7
HR (bpm)	72.7 ± 1.7

Classification of HF

HFrEF	82.4% (70)
HFmrEF	10.6% (9)
HFpEF	7.1% (6)

HAS: Systemic arterial hypertension; DM: Diabetes mellitus; CAD: Coronary artery disease; DLP: Dyslipidemia; CKD: Chronic kidney disease; AF: Atrial fibrillation; COPD: Chronic obstructive pulmonary disease; NYHA: Functional classification by the New York Heart Association; SGLT2i: Sodium-glucose cotransporter-2 inhibitors; ARB: Angiotensin II receptor blockers; ARNI: Angiotensin receptor-neprilysin inhibitor; ACEI: Angiotensin-converting enzyme inhibitors; LVEF: Left ventricular ejection fraction; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HR: Heart rate; HF: Heart failure; HFrEF: HF with reduced ejection fraction; HFmrEF: HF with mildly reduced ejection fraction; HFpEF: HF with preserved ejection fraction. Source: study data.

may promote a modest increase in diuresis, raising the hypothesis that these drugs could allow for the gradual reduction of traditional diuretic doses, such as furosemide, during follow-up.^{3,8-10}

In the present study, we sought to analyze renal function and diuretic therapy in patients with HF after the introduction of SGLT2 inhibitors, focusing on the hypothesis of an influence on furosemide titration, based on reports in the literature of favorable natriuretic and hemodynamic effects of SGLT2 inhibitors and the hypothesis of a progressive reduction in the dose of this medication.⁵⁻⁷ However, the results showed the need for an increased furosemide dose associated with mild worsening of renal function, as represented in the central figure. This pattern may be related to mild and progressive impairment of renal function, which possibly interfered with the expected natriuretic effect and contributed to the need for maintenance or increase of furosemide dose over time. This finding may be associated with specific characteristics of the studied population, which was typically more severe with advanced HF, or with limitations of outpatient follow-up that may have influenced the observed outcomes.

Moreover, although large clinical trials have consistently demonstrated benefits, none were designed to evaluate the reduction of furosemide dose as a primary endpoint, and the effects on decongestion or diuretic burden in the outpatient setting remain heterogeneous.^{5-7,11} Our results, therefore, illustrate the gap between the efficacy observed in randomized clinical trials and the real-world effectiveness for this specific outcome.

Population factors justifying the increase in furosemide dose

Clinical profile of the sample

Most of the patients evaluated presented characteristics consistent HFrEF. This profile is similar to that observed in major studies focusing on HFrEF, such as EMPEROR-Reduced and DAPA-HF.^{5,6} However, unlike these studies, which included predominantly patients in NYHA functional class II, the present study comprised a significantly higher proportion of individuals in more advanced functional classes (III and IV) at the time of SGLT2i prescription. This more severe clinical profile may have required higher diuretic doses and hindered the reduction of furosemide during follow-up.^{3,10}

Persistent congestion signs

Congestion is the main determinant for the indication and adjustment of diuretic doses in HF. Despite a slight clinical improvement observed during follow-up visits, signs of volume overload, such as edema and jugular venous distension, remained in a significant portion of the sample, indicating residual congestion. This persistence may have limited the possibility of reducing the furosemide dose and contributed to the maintenance or increase of diuretic therapy. Zannad et al.⁷ emphasize that the benefits of decongestive interventions are more pronounced when applied early, before the establishment of significant congestion, which may explain the less favorable clinical response observed.

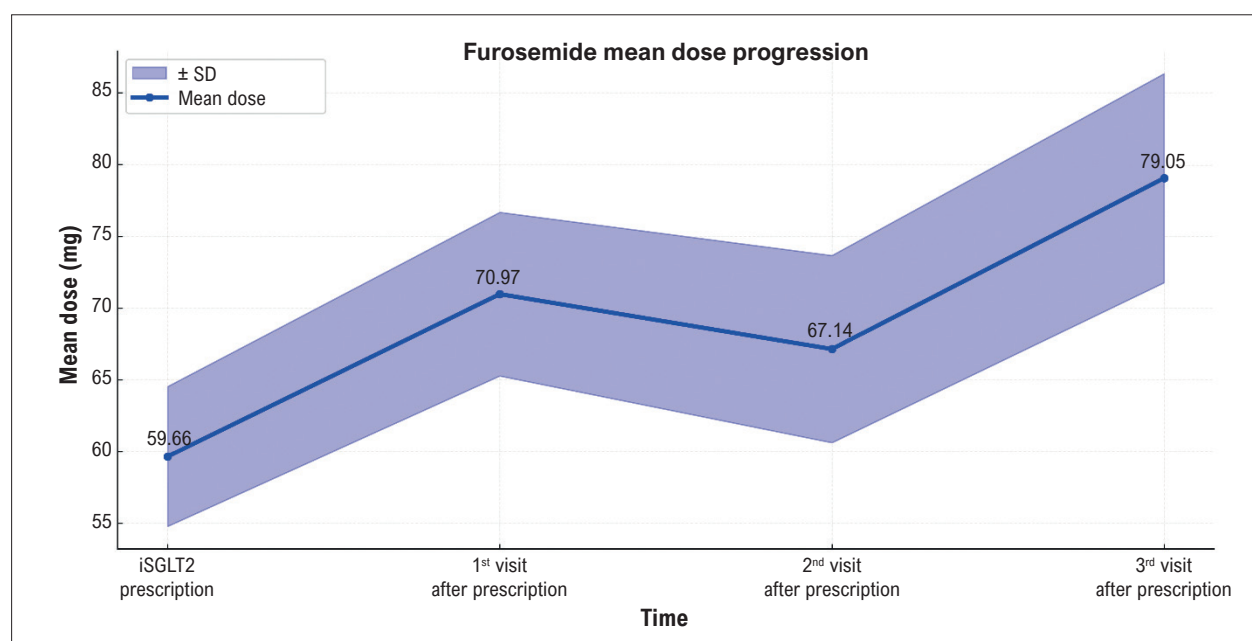


Figure 1 – Furosemide dose progression between the time of SGLT2 inhibitor prescription and subsequent visits; illustrative figure of mean furosemide dose values over time: at the time of SGLT2 prescription (59.66 ± 4.87 mg), at the first follow-up visit (70.97 ± 5.70 mg), at the second visit (67.14 ± 6.52 mg), and at the third visit (79.05 ± 7.28 mg), with $p = 0.003$; SGLT2i: Sodium-glucose cotransporter-2 inhibitors. Source: study data.

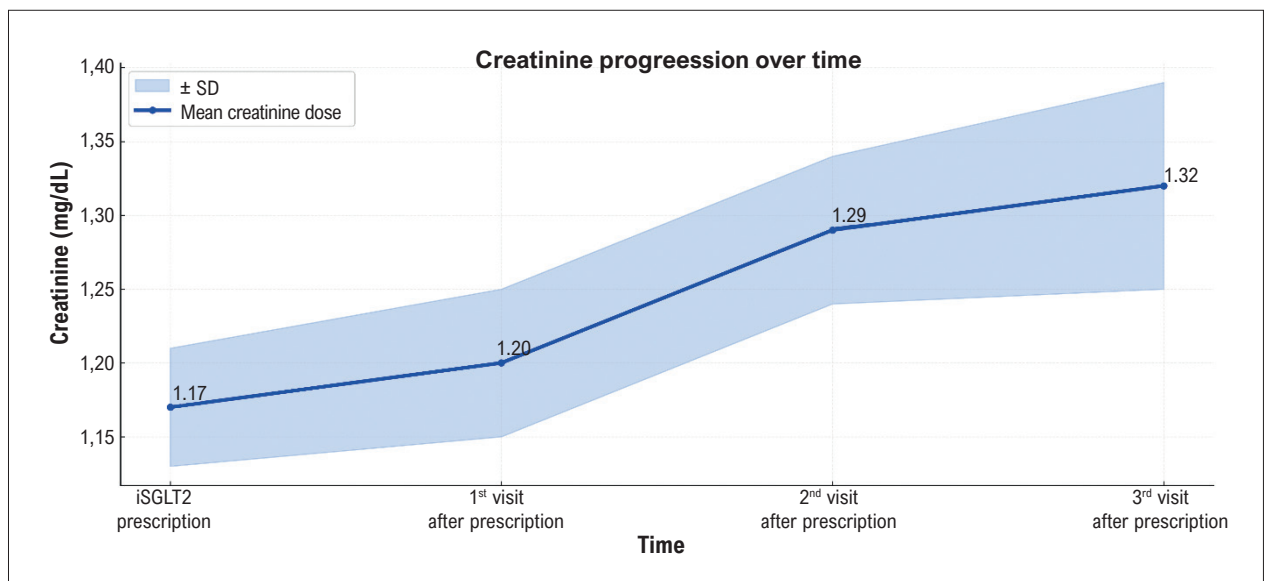


Figure 2 – Evolution of creatinine values between the time of SGLT2 inhibitor prescription and subsequent visits; illustrative figure of mean creatinine values over time: at the time of SGLT2 prescription (1.17 ± 0.04 mg/dL), at the first follow-up visit (1.20 ± 0.05 mg/dL), at the second visit (1.29 ± 0.05 mg/dL), and at the third visit (1.32 ± 0.07 mg/dL), with $p = 0.002$; SGLT2i: Sodium-glucose cotransporter-2 inhibitors; source: study data.

Progression of renal dysfunction

During follow-up, a mild but progressive decline in renal function was observed, expressed by the increase in creatinine levels across visits, which may suggest the presence of cardiorenal syndrome.¹² In the outpatient setting, patients may develop this syndrome mainly due to persistent venous congestion, reduced cardiac output, and chronic neurohormonal activation. Renal venous congestion leads to decreased renal perfusion and activation of compensatory mechanisms that perpetuate sodium and water retention, which may worsen volume overload and renal function.¹³ In this context, the increase in furosemide dosage may have occurred as an attempt to compensate for volume accumulation secondary to renal dysfunction, differing from the results of studies such as DAPA-HF and EMPEROR-Reduced, which demonstrated consistent nephroprotective effects.^{5,6}

It is important to acknowledge that, in the present study, serial assessments of GFR were not performed, which represents a limitation in the interpretation of renal function throughout follow-up. In the EMPAG-HF study, an initial transient decline in GFR was observed, followed by recovery, suggesting that the shorter follow-up period in the present study may not have been sufficient to identify stabilization and the full manifestation of the nephroprotective effect.⁸ Thus, the results may reflect an early phase of hemodynamic adaptation, aggravated by clinical factors such as persistent congestion and greater disease severity, which may have negatively impacted the renal response to treatment.

Mean follow-up time

The interval between visits was relatively short, consistent with quarterly outpatient follow-up. Compared with the

observation period of large clinical trials, this reduced timeframe may have been insufficient to demonstrate sustained clinical stabilization or reversal of transient renal dysfunctions.^{5,6,8,11} Thus, the limited follow-up period, combined with the greater clinical severity of the studied population, may have contributed to the absence of diuretic therapy reduction and to the divergent results in relation to the literature.

An interval shorter than 30 days was considered inadequate, as it does not represent sufficient time for definitive clinical changes in furosemide dose or renal function secondary to the effect of SGLT2 inhibitors. Moreover, in cases of recent decompensation, very early follow-up visits coincide with the most critical phase of the vulnerable period, characterized by marked clinical variability, so that the fluctuations observed tend to reflect transient instability capable of distorting longitudinal assessment. Nevertheless, it is acknowledged that the definition of this cutoff point is arbitrary, since there is no robust evidence establishing the ideal period for such analysis.

Implications

In advanced HF outpatient clinics, SGLT2 inhibitors did not lead to an automatic reduction in furosemide dose in the short term. The decision to adjust diuretic therapy remains individualized, depending on symptoms, clinical signs, and renal function, consistent with real-world practice. This reinforces the observational and hypothesis-generating nature of the present study. The findings, therefore, highlight the need for prospective and controlled studies, with standardized targets for euvolemia and diuretic equivalents, to estimate the specific impact of SGLT2 inhibitors on diuretic burden.

Limitations

The present study has important limitations that must be considered when interpreting the findings. The retrospective design (i), the absence of a control group (ii), and the possibility of selection bias resulting from the exclusion of follow-up visits with intervals shorter than 30 days (iii) limit causal inferences. In addition, the assessment of congestion was extracted from medical record entries (iv), with the possibility of recording bias, heterogeneity in therapeutic management, and lack of standardization of diuretic equivalents (v).^{3,10} These limitations restrict the generalizability of the results and reinforce the exploratory nature of the analysis.

The exclusion of follow-up visits with intervals shorter than 30 days, adopted to avoid transient fluctuations that could distort longitudinal assessment, may have introduced selection bias by removing potentially more unstable patients. Although necessary for temporal consistency of the study, this criterion limits the extrapolation of results to populations monitored more frequently. Inherent to its retrospective design, this study also presents limitations regarding relevant clinical information – such as functional class, blood pressure, objective markers of congestion, NT-proBNP, and detailed parameters of therapeutic optimization – which were not uniformly available across all visits. The absence of these data precluded robust multivariate adjustments or stratified subanalyses without substantial loss of statistical power. Thus, the findings should be interpreted as exploratory, although consistent with the advanced HF phenotype observed in outpatient practice.

Overall, the results suggest that the clinical response to SGLT2 inhibitors may vary according to the profile and severity of patients with HF, as well as the duration of follow-up. Although randomized trials consistently demonstrate benefits in reducing volume overload and in renal protection, the findings of the present study indicate that such effects may not fully manifest in short-term

outpatient settings or in populations with persistent congestion and pre-existing renal dysfunction.^{5-8,11-13} This pattern likely reflects real-world clinical practice, in which diuretic titration is guided by symptoms, clinical signs, and renal function – factors not controlled in our retrospective design. The modest improvement in edema/jugular distension, despite the increased dose, suggests that SGLT2 inhibitors did not eliminate the need for diuretic adjustments in the short term in this predominantly symptomatic HFrEF population.

Author Contributions

Conception and design of the research, Acquisition of data, Analysis and interpretation of the data, Statistical analysis, Obtaining financing, Writing of the manuscript and critical revision of the manuscript for intellectual content: Bolfe I, Ribeiro RKM, Prager C, Bolfe JV, Freitas AKE.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Universidade Positivo under the protocol number 7.081.707. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013.

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