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CARDIOVASCULAR AND RENAL BENEFITS OF SGLT2 INHIBITORS IN CHRONIC HEMODIALYSIS: A CASE REPORT

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Abstract: *Sodium–glucose cotransporter-2 (SGLT2) inhibitors provide established cardiovascular and renal protection in patients with heart failure and chronic kidney disease, irrespective of diabetes status, but evidence in individuals receiving maintenance hemodialysis remains limited because this population has been excluded from major randomized trials. We report the case of a 76-year-old non-diabetic patient with heart failure with reduced ejection fraction (HFrEF) and end-stage kidney disease undergoing thrice-weekly hemodialysis in whom empagliflozin was initiated 12 months after dialysis initiation. Over two years of follow-up, treatment was well tolerated and associated with improvements in volume status, blood pressure control, and heart failure–related symptoms and overall quality of life, as reflected by a substantial increase in Kansas City Cardiomyopathy Questionnaire scores. Residual kidney function remained preserved, with sustained urine output and measurable residual glomerular filtration rate. Bioimpedance spectroscopy demonstrated reduced overhydration and improved fluid management, accompanied by lower loop diuretic requirements. No major adverse events were observed, apart from a single low urinary tract infection. This case highlights the potential feasibility and cardiorenal benefits of SGLT2 inhibitor initiation in selected hemodialysis patients with HFrEF and residual diuresis. Further randomized controlled studies are needed to clarify safety, mechanisms, and clinical efficacy in this high-risk population.*

Key words: *SGLT2 inhibitors, hemodialysis, heart failure, residual kidney function.*

1. Introduction

Sodium–glucose cotransporter 2 inhibitors (SGLT2i) have emerged as a cornerstone therapy for heart failure with reduced ejection fraction (HFrEF), demonstrating consistent reductions in heart failure hospitalizations and

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cardiovascular mortality irrespective of diabetes status [1,2]. SGLT2i are also current standard-of-care therapies for chronic kidney disease (CKD) patients, irrespective of diabetes status or CKD stages [3]. Large placebo-controlled randomized clinical trials have demonstrated renal protective effects by reducing CKD progression, cardiovascular and overall mortality [4-6]. Although patients with an eGFR bellow 20 mL/min/1,73 m^2 at baseline, patients on maintenance dialysis or kidney transplant recipients have been excluded from these studies, a continuation of these therapies until the initiation of renal replacement therapies is proposed by current guidelines [3]. Individuals on chronic hemodialysis (HD) bear an exceptionally high burden of cardiovascular disease, with heart failure representing a leading cause of morbidity and mortality. Therapeutic options for HFrEF in this population are limited by hyperkalemia, hypotension and drug intolerance, and evidence supporting guideline-directed medical therapy is often extrapolated from non-dialysis populations.

The objective of this study is to report a clinical case of a patient with HFrEF receiving chronic hemodialysis in whom empagliflozin was started after dialysis initiation, and to provide a focused review of the current literature on the use of SGLT2i in hemodialysis patients, with particular emphasis on cardiovascular effects and residual kidney function preservation.

2. Case report

The patient is a 76-year-old male with long-standing CKD and HFrEF with a left ventricular ejection fraction of 40% prior to hemodialysis initiation with no history of diabetes or impaired fasting glucose. The decline in renal function, assessed by estimated glomerular filtration ratio (eGFR) was gradual, over a period of 20 years and, although no kidney biopsy was performed, the etiology of the CKD was interpreted as both hypertensive nephropathy and chronic tubulointerstitial disease related to hyperuricemia with recurrent gout flares over the past 25 years.

Following progression to end-stage kidney disease (ESKD), the patient had recurrent episodes of decompensated heart failure with exertional dyspnea consistent with New York Heart Association (NYHA) functional class IV in the context of hypervolemia which prompted the decision to initiate thrice weekly maintenance hemodialysis in December 2022 on a radio-cephalic fistula previously created in March 2022. Despite dialysis initiation and guideline-directed heart failure therapy, the patient remained symptomatic with exertional dyspnea consistent with New York Heart Association (NYHA) functional class III. The coronarography could not be performed prior to HD initiation due to azotemia, so the procedure was performed in January 2023 and revealed no significant coronary artery stenosis - the coronary angiogram revealed a left dominant circulation with no lesions at the level of left main, left anterior descending artery or the left circumflex artery and a 50-60% stenosis at the level of the mid segment of the right coronary artery.

Empagliflozin was initiated at a dose of 10 mg per day 12 months after HD initiation, after stabilization of fluid status on chronic hemodialysis, with multiple body composition monitor (BCM)

evaluations using bioimpedance spectroscopy and the refinement of ultrafiltration volume according to the clinical evaluation and BCM reports performed before the HD session. The BCM analysis results depicted in Table 1 were performed 4 months after HD initiation and were compared with the analysis performed 6 months after empagliflozin initiation and after 2 years of follow-up after empagliflozin initiation. A clear improvement in the estimated overhydration, as well as the average overhydration was found, with a relatively constant extracellular to intracellular water
ratio, but with a reduction in relative overhydration, which is the overhydration to extracellular water ratio, from 5% to 4.6% and 2.9%, respectively.

Body composition monitor (BCM) analysis results Table 1

	BCM after HD initiation	**BCM after empagliflozin initiation**	**BCM after 2 years of follow-up**
Average pre-dialysis weight	76.4 kg	74.3 kg	74.1 kg
Average post-dialysis weight	74.3 kg	72.9 kg	72.1 kg
Normohydration weight	75.2 kg	73.3 kg	73.6 kg
Intracellular water (ICW)	23.3 L	25.4 L	22 L
Extracellular water (ECW)	19.7 L	20.4 L	18.5 L
Extracellular/intracellular water ratio (ECW/ICW)	0.84	0.8	0.84
Total body water (TBW)	43 L	45.8 L	40.6 L
Overhydration (OH)	1 L	0.9 L	0.6 L
Average OH	1.2 L	1 L	0.5 L
Relative overhydration (OH/ECW)	5 %	4.6 %	2.9 %
Average pre-dialysis systolic blood pressure	144 mmHg	138 mmHg	138 mmHg
Average pre-dialysis diastolic blood pressure	68 mmHg	69 mmHg	64 mmHg
Lean tissue mass (LTM)	50.7 kg	57.4 kg	47.4 kg
Adipose tissue mass (ATM)	23.2 kg	14.5 kg	24.8 kg
Body mass index (BMI)	26.4 kg/m^2	25.7 kg/m^2	25.6 kg/m^2
Lean tissue index (LTI)	17.6	19.9	16.4
Fat tissue index (FTI)	8	5	8.6
LTM/weight	66.6 %	77.3 %	64 %
ATM/weight	22.4 %	14.4 %	24.6 %

A small reduction in pre-dialysis systolic and diastolic blood pressure was also found, from 144 mmHg and 68 mmHg after hemodialysis initiation to 138 mmHg and 64 mmHg, respectively after 2 years of empagliflozin administration, without changes in the antihypertensive medication. The nutritional status as assessed by BCM analysis showed changes in the lean tissue mass (LTM) and lean tissue index (LTI) and corresponding changes in adipose tissue mass (ATM) and adipose tissue index (ATI), with an initial improvement in LTM and LTI and a rise in LTM to body weight ratio from 66.6% to 77.3% and a drop in ATM to body weight ratio from 22.4% to 14.4% assessed after HD initiation and after empagliflozin

initiation, respectively. These results can be explained by the dietary changes after HD commencement – the patient was on a ketoanalogue-supplemented very low protein diet before HD and had a low appetite due to azotemia and heart failure symptoms, but, after HD initiation, the appetite significantly improved, and the LTM and LTI also improved. The results after 2 years of follow-up after empagliflozin initiation showed a loss in the initial improvement, with a lower LTM to body weight ratio of 64% and a higher ATM to body weight ratio of 24.6%, which is partly explained by the coexistence of terminal chronic kidney disease and heart failure. This result can also derive from the current estimated normalized protein catabolic rate (nPCR) of 0.97 grams of protein per kilogram of body weight per day, which estimates daily dietary protein intake of a stable dialysis patient and, in this case, was lower than the target of 1.1-1.2 grams of protein/kg/day, which prompted nutritional intervention for dietary protein intake increase.

An improvement in diuresis was also observed, with a 24-hour urine volume of 1200 mL at HD initiation, which decreased to 900 mL/day the following year, a rise to 1100 mL/day after empagliflozin initiation and a current diuresis of 1300 mL/day – Figure 1. The loop diuretic dose was halved – 40 mg of furosemide per day orally in the first year of maintenance hemodialysis, 20 mg per day after empagliflozin initiation.

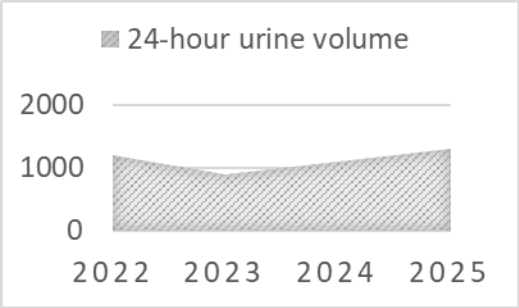

Fig. 1. *Average diuresis at HD initiation and after empagliflozin initiation (per year)*

The improvement in diuresis is consistent with the improvement in the fluid status assessed by BCM, with a reduction in interdialytic weight gain and a reduction in the ultrafiltration volume required to achieve euvolemia.

Besides residual urine output, in order to assess the residual kidney function (RKF), an estimation of the kidney clearance was also performed at 2 years after empagliflozin initiation. A 24-hour urine collection and blood samples were analysed and the clearance of urea and creatinine were calculated, as shown in Table 2.

Estimated residual kidney function (RKF) Table 2

24-hour urine volume	1300 mL
Kt/V	1.5
Creatinine clearance (K_{Cr})	11.4 mL/min
Urea clearance (K_{Urea})	4.4 mL/min
Residual glomerular filtration ratio (GFR)	7.9 mL/min
Body surface area	1.86 m^2
Normalized residual GFR	7.3 mL/min/1.73 m^2

The residual glomerular filtration ratio (GFR) was estimated at 7.9 mL/min and the result was adjusted to the body surface area, with a normalized residual GFR of 7.3 mL/min/1.73 m^2, which, in conjunction with a residual urine volume of 1300 mL per day represent substantial residual kidney function, especially after 3 years of maintenance hemodialysis. The current urinary albumin to creatinine ratio (ACR) is 183 mg/g, with a proteinuria of 475 mg/day (the urinary ACR prior to hemodialysis initiation was 88 mg/g). Although a significant RKF was found, the level of glycosuria was only 2.8 g/day. The 24-hour urinalysis also revealed a natriuresis of 144 mmol/24h and a kaliuresis of 29 mmol/24h and no episodes of kyperkalemia were noted. Hemodialysis efficacy was quantified using the Kt/V (K – dialyzer clearance of urea, t – dialysis time, V – volume of distribution of urea) with an average of 1.5 since hemodialysis initiation.

The patient also demonstrated clinical improvement, with reduction in heart failure symptoms and improved dialysis tolerance. A subjective assessment of heart failure burden was performed using the 12 items Kansas City Cardiomyopathy Questionnaire (KCCQ-12) – Table 3.

Kansas City Cardiomyopathy Questionnaire (KCCQ-12) Table 3

Domain	**Score**		
	At HD initiation	**After HD initiation**	**After empagli-flozin**
Physical limitation	37.5	75	93.75
Symptom frequency	62.5	81.25	87.5
Quality of life	0	37.5	75
Social limitation	0	50	87.5
Overall summary score (OSS)	**25**	**60.9**	**85.94**

At HD initiation, the results showed a high frequency of symptoms, with important physical limitation and severely impaired quality of life and social function, with an overall summary score (OSS) of 25, which is relevant for a poor health status. The score was also performed 6 months after HD initiation and a significant improvement was found, but the quality of life remained the most affected domain. The OSS score improved by 35.9 points, which is consistent with a fair to good health status. At 2 years of follow-up after empagliflozin initiation, a further improvement in all domains was observed, with a further rise in the OSS to 85.94, which is considered a clinically meaningful change from the previous assessment, with major improvement in reported quality of life.

Baseline (at hemodialysis initiation) and follow-up transthoracic echocardiography revealed a stable left ventricular ejection fraction of approximately 40%, consistent with moderate left ventricular systolic dysfunction, the left and right ventricular dimensions were within normal limits, the left ventricular diastolic function assessment was compatible with grade I diastolic dysfunction (impaired relaxation pattern), a mild mitral regurgitation (grade 1) was present, a regional wall-motion analysis demonstrated anteroseptal dyskinesia, the estimated pulmonary artery systolic pressure was within the

normal range and no pericardial effusion was detected.

Over a period of 2 years of follow-up after empagliflozin initiation, no episodes of symptomatic hypotension, ketoacidosis or symptomatic hypoglycemia were observed and interdialytic weight gain remained stable. There was no hospitalization since hemodialysis initiation. Throughout the follow-up period the patient presented an episode of lower tract urinary infection (UTI). Considering the risk associated with the presence of benign prostate hypertrophy, the drug was temporary discontinued. The UTI episode resolved with a seven-day oral antibiotic regime and a urinary antiseptic, with a negative urine culture obtained 10 days after the antibiotic treatment cessation, which prompted reintroduction of empagliflozin. The patient reported an average of 3 gout episodes per year in the years prior to hemodialysis initiation and in the first HD year, only 2 episodes in the following year and no episodes thereafter, with no change in the allopurinol prescribed dose (100 mg per day) and a normal range serum uric acid. A reduction of C-reactive protein was also found, from 10 mg/L before empagliflozin initiation and a current value of 6 mg/L. The calcium and phosphorus levels were within range, with an average calcium-phosphorus product of 36.2 $(mg/dL)^2$ and the serum parathormone level was 196 ng/L at HD initiation and a current level of 76 ng/L on 2400 mg/day of sevelamer. Anemia was well controlled with in-target hemoglobin and ferritin levels, on an average dose of darbepoetin alfa of 7.5 ug per week and iron sucrose of 100 mg per month.

3. Discussion

There is limited data of SGLT2i use in hemodialysis patients and, to my knowledge, this is the first case report of SGLT2i initiation in a non-diabetic patient with HFrEF undergoing maintenance hemodialysis with thrice weekly sessions, for both cardiovascular benefit and preservation of RKF, with 2 years of follow-up. This case illustrates the feasibility and cardio-renal benefits of initiating empagliflozin in a patient receiving chronic hemodialysis with concomitant HFrEF and residual diuresis.

Emerging observational data and early-phase clinical trials suggest that SGLT2i may be safe and potentially beneficial in selected dialysis patients, through mechanisms largely independent of glycemic control. The reason for the paucity of data in the population of HD patients subsides in the renal effects of SGLT2i, which act on specific transporters in the proximal convoluted tubule, leading to increased natriuresis and glycosuria, with subsequent activation of the tubulo-glomerular feed-back by increased delivery to macula densa, which generates vasoconstriction of the afferent arteriole, thus reducing intraglomerular pressure. This effect reduces barotrauma and albuminuria, important factors in CKD progression [7].

In heart failure and in hyperfiltering remnant nephrons characteristic of CKD, SGLT2 expression and transport capacity are upregulated in association with tubular hypertrophy and increased activity of the renin–angiotensin and sympathetic nervous systems. These patho-physiological states are accompanied by stimulation of basolateral Na^+/K^+-ATPase, which facilitates increased apical solute

reabsorption mediated by SGLT2, NHE3, and URAT1, transporters that are functionally integrated through coordinated neurohormonal signaling and shared scaffolding protein complexes [8].

In patients with advanced CKD, the attenuation of glomerular hyperfiltration is reduced in the context of low nephron number, hence a reduction of tubular effect on SGLT2 receptors, but there are several other mechanisms that exert cardiovascular and renal protection by reducing inflammation, oxidative stress and fibrosis [9].

Large-scale clinical trials have demonstrated that SGLT2i reduce cardiovascular death and heart failure-related hospitalizations [10]. Also, a recent meta-analysis demonstrated that initiation of SGLT2i after an acute coronary syndrome is associated with a significant reduction in all-cause and CV mortality [11]. The natriuretic effects of SGLT2i, as well as a reduction in arterial stiffness are mechanisms involved in blood pressure (BP) and volume overload reduction, but other SGLT2i effects are significant for cardiovascular protection [12]. Several studies demonstrated a reduction in proinflammatory pathways with SGLT2i use, and a recent meta-analysis revealed a reduction in serum biomarkers such as C-reactive protein, ferritin, interleukin-6, tumor necrosis factor-α, plasminogen activator inhibitor-1 [13]. Clinical implications of anti-inflammatory effects of SGLT2i have been assessed in several cardiovascular outcome studies, and the results demonstrated an improvement of cardiac function, with the reduction of epicardial adipose tissue, cardiac fibrosis, aortic and vascular stiffness [14-16]. The endothelium is an important modulator of vascular tone, of the hemostasis-thrombosis equilibrium and atherosclerosis and is interlinked with immune cells through pro-inflammatory cytokines and oxygen reactive species [17]. SGLT2 inhibition has been shown to prevent endothelial cell dysfunction and to enhance endothelial-dependent vasodilation in preclinical studies and studies in human endothelial cells in vitro have demonstrated that SGLT2i have direct anti-inflammatory effects [17, 18]. An in vitro study showed that dapagliflozin inhibits pro-inflammatory pathways by reducing the expression of TLR-4 and activation of nuclear factor-κB in response to stimulation by bacterial lipopolysaccharides [19]. A similar effect of SGLT2i has been demonstrated in stretch-induced endothelial cell dysfunction [20]. In a meta-analysis that included six randomized controlled trials, SGLT2 inhibitors significantly improved flow-mediated dilation, although without a significant effect on pulse wave velocity [21]. An experimental study using a co-culture of human cardiac endothelial cells and cardiomyocytes demonstrated that empagliflozin reduced reactive oxygen species accumulation and increased endothelial nitric oxide bioavailability, restoring the impaired cardiomyocyte relaxation and contraction [22]. It has also been shown that SGLT2i exert cardiovascular protection by regulating cytosolic sodium and calcium via inhibition of sodium-hydrogen exchanger-1 (NHE1) and sodium-calcium exchanger (NCX) in endothelial cells, which ameliorates endothelium-derived vasodilation through an increase in nitric oxide bioavailability, inflammation ant platelet aggregability [23].

Another pleiotropic effect of this class of drugs is the effect on the gut microbiome, with beneficial cardiorenal effects [23]. A

comprehensive omics analysis showed that SGLT2i reduced microbiome formation of uremic toxins such as p-cresol sulfate, thereby reducing tubular glucotoxicity and a broad downregulation of sodium, glucose, amino acid and urate uptake [24].

These broad effects of SGLT2i can be taken into consideration for this patient, with a presumed low effect on the proximal tubule of empagliflozin in the case reported, considering the low level of glycosuria found, although with a well-preserved natriuresis and kaliuresis, which can also be attributed to the loop diuretic. The significant preservation of residual kidney function has a major impact on the heart failure symptoms control, although without an improvement in the left ventricle ejection fraction in this case. The preserved diuresis is crucial for the fluid management in this patient, with favorable BCM results, including lower interdialytic weight gain, a lower level of pre-dialysis overhydration, the absence of intradialytic hypotension, with a stable extracellular to intracellular water ratio and lower pre-dialysis systolic and diastolic blood pressure. The preservation of residual glomerular filtration ratio, with adjustment to the body surface area of 7.3 mL/min/1,73 m^2 and the good dialysis adequacy, assessed by a Kt/V of 1.5 are important for a lower uremia burden, which impacts cardiovascular health and nutritional status parameters and may also explain the reduction in gout flares and the normalization of plasma uric acid. Also, a reduction in C-reactive protein values was noted, which may be attributed to the anti-inflammatory properties of SGLT2i.

The effects of SGLT2i on anemia and calcium-phosphorus metabolism were also investigated in several studies. An indirect cardiovascular benefit of SGLT2 inhibitors is the mitigation of anemia and the improvement in iron utilization. It is postulated that SGLT2i decrease the expression of HIF-1α and enhance sirtuin-1 activation, which stimulates hypoxia-inducible factor (HIF)-2α, leading to erythropoietin production in the liver and has antifibrotic effects in the heart [25, 26]. In the proximal tubule, SGLT2 inhibition reduces luminal sodium reabsorption, thereby augmenting the electrochemical gradient driving sodium–phosphate cotransport and increasing phosphate reabsorption, which stimulates fibroblast growth factor-23 (FGF-23) secretion and reduces ionized calcium, leading to secondary hyperparathyroidism and enhanced phosphaturia [27]. At the renal level, parathyroid hormone and FGF-23 exert opposing effects on 1α-hydroxylase activity, resulting in counterregulatory modulation of 1,25-dihydroxyvitamin D synthesis and an uncertain net impact on vitamin D homeostasis and skeletal metabolism [28]. In the case reported, a low level of darbepoetin alfa and iron sucrose was needed to maintain adequate levels of hemoglobin and ferritin, which is consistent with the mechanisms aforementioned, but also with the preservation of RKF. The serum phosphorus and calcium levels are also in-target. Interestingly, the serum parathormone level decreased, which may be explained by the adequacy of hemodialysis and preservation of RKF, but also by the serum phosphorus control through non-calcium binder therapy and the dietary approach to control the phosphorus intake.

The pleiotropic effects of SGLT2i are currently being investigated in several ongoing clinical trials, but there are few completed studies of SGLT2i use in hemodialysis patients in regards to safety and tolerability, as well as cardiorenal and survival benefits – Table 4.

Completed SGLT2i studies in hemodialysis patients Table 4

Author, year (Ref)	Type of study	Intervention	Patients	Control group	Follow-up	Outcome
Heerspink HJL, 2021 [29]	Exploratory analysis of RCT	Dapagliflozin	68	Placebo, 99	Not specified	Lower all-cause mortality after initiation of HD
De la Flor, 2023 [30]	Retrospective	Dapagliflozin or empagliflozin	7	No	12 months	Improvement in RKF
Barreto J, 2023 [31]	Single-center, prospective	Dapagliflozin	7	Age, sex matched with eGFR>60 mL/min, 7	7 days	Difference in peak concentration and area under the curve vs. controls
Wang CA, 2024 [32]	Retrospective	SGLT2i	771	Propensity-score matched, 771	2 years	Reduction in the risk of mortality and MACE
Lin DS, 2025 [33]	Exploratory single-arm safety study	Empagliflozin	17	No	4 weeks	Safety, tolerability

In an exploratory analysis of a landmark randomized controlled trial (RCT) of dapagliflozin use in patients with CKD (the DAPA-CKD trial), a lower all-cause mortality was found in patients on chronic dialysis [29]. De la Flor et al. demonstrated an improvement in residual kidney function, measured by urea clearance, from 4.91 ± 1.14 mL/min to 7.28 ± 1.68 mL/min and also improvements in blood pressure, blood uric acid, hemoglobin A1c, urine albumin/creatinine ratio (UACR), and 24 h proteinuria with dapagliflozin or empagliflozin use in patients on incremental hemodialysis [30]. Barreto J et al. explored the pharmacokinetics of dapagliflozin in patients on regular dialysis and found low dialyzability of the drug, with no serious adverse events [31]. In a retrospective, propensity-score matched clinical trial with a follow-up of 2 years of patients with type 2 diabetes mellitus found that SGLT-2i users were associated with a 48% lower risk of major cardiovascular events (MACE), 51% lower all-cause mortality, with an increased likelihood to become dialysis-free 90 days after the index date and with no significant differences in the incidence of ketoacidosis, urinary tract or genital infection, hypoglycemia, dehydration, bone fractures, below-knee amputations, or 90-day readmissions [32]. An exploratory single-arm safety study assessed the safety and short-term effects of empagliflozin on patients on maintenance hemodialysis with

heart failure demonstrated a favorable safety profile for doses of 5 mg, 10 mg and 25 mg [33].

This report is limited by its single-patient design and relatively short follow-up, precluding causal inference. Other limitations of the study include no active monitoring of ketones, the absence of ketoacidosis being self-reported after a comprehensive education about possible symptoms. There is also no comparison between current renal function and the one prior to SGLT2i initiation assessed using 24 hours urine collections. No assessment of glucose tolerance or insulin resistance were performed, but fasting plasma glucose was within normal range. Lastly, there is no measurement of the hemodialysis arterio-venous fistula blood flow, which can also impact cardiac function through modifications in ventricle filling pressures or in left ventricle ejection fraction. Nevertheless, this case report adds to the growing body of real-world evidence suggesting that SGLT2 inhibitors may represent a valuable adjunctive therapy for selected hemodialysis patients with HFrEF and residual kidney function.

4. Conclusions

In this case report, initiation of empagliflozin after the start of chronic hemodialysis was well tolerated and associated with clinical stabilization in a patient with HFrEF. Together with emerging observational data and ongoing clinical trials, this experience supports the hypothesis that SGLT2 inhibitors may confer cardiovascular benefit in selected hemodialysis patients. Robust randomized controlled trials are required to define safety, efficacy, and optimal patient selection before routine use in this population is recommended.

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