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Original Article

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Empagliflozin Decreased Proteinuria in Patients with Glomerulonephritis without Diabetes: A Randomized Clinical Trial

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ABSTRACT

Objective: Empagliflozin (EMPA) is a new class of hypoglycemic drugs acting as a sodium glucose co-transporter 2 inhibitor. In addition to reducing blood sugar, cardiovascular complications, and mortality rate, EMPA also has kidney protective properties (such as reducing proteinuria, progression of albuminuria, and the rate of estimated glomerular filtration rate (eGFR) decline). Therefore, this study aimed at investigating the effect of EMPA on proteinuria in non-diabetes mellitus (non-DM) patients with glomerulonephritis.

Methods: A randomized double-blind clinical trial was conducted on 60 non-DM patients with glomerulonephritis that were assigned into 2 groups of 30. All patients received their usual treatment. Patients in the first group received EMPA 10 mg tablets daily (EMPA group), while patients in the control group received placebo daily (placebo group) for 3 months.

Results: The results of the present study showed that the mean change weight decreased in the EMPA group and increased in the control group (−0.25 kg vs. +0.10 kg; $P < .001$). Although eGFR decreased over time, there was not any significant difference between the EMPA and control groups at either the start or the end of study (−2.72 mL/min/1.73m^2 vs. −1.89 mL/min/1.73m^2, $P = .767$). Proteinuria decreased and increased significantly in EMPA and control groups, respectively (−118.06 mg/24h vs. +233.69 mg/24h, $P = .013$).

Conclusion: According to the results of the present study, it seems that EMPA administration can play an effective role in reducing proteinuria in patients with glomerulonephritis.

Keywords: Empagliflozin, proteinuria, glomerulonephritis

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INTRODUCTION

Glomerulonephritis is a kind of kidney disease, characterized by inflammation of glomeruli or small blood vessels in the kidneys, and may be with or without hematuria and proteinuria.[1] Recently, some researchers have indicated that sodium-glucose cotransporter 2 inhibitor (SGLT2i), as a new class of antidiabetic drugs, may reduce proteinuria in diabetic nephropathy patients by reducing podocyte apoptosis. In fact, SGLT2i can repair the damage of proximal tubular cells, reduce podocytes shedding by inhibiting kidney oxidative stress, and further relieve proteinuria.[1,2]

Some of SGLT2i drugs include canagliflozin, dapagliflozin, and empagliflozin (EMPA), which inhibit the SGLT2i transport pump in the kidney, increase the excretion of glucose in the urine, and reduce the serum glucose level.[3] Although the primary purpose of these drugs was to improve the control of blood sugar indicated by reducing glycosylated hemoglobin A1c (HbA1c) by 0.8-0.5%,[4] some additional benefits comprised weight loss,

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blood pressure reduction, and decreased the length of hospitalization, and lower cardiovascular mortality rate.[5,6]

The mechanisms of kidney protection by SGLT2i are not yet fully understood; however, its beneficial effects on the kidney may be mediated by sodium excretion, which leads to systemic and intrarenal hemodynamic changes. In particular, SGLT2i increases the delivery of distal tubular sodium and chloride to the macula densa, which increases the tubuloglomerular feedback and causes arteriolar vasoconstriction. As a result, SGLT2i causes an acute decrease in intraglomerular pressure and estimated glomerular filtration rate (eGFR). This effect is particularly observed in patients with type II diabetes (T2DM) that do not exhibit hyper-filtration.[7,8]

Empagliflozin is hypothesized to reduce the risk of cardiovascular mortality, improve outcomes in septic patients, and decrease hospitalization for heart failure. Furthermore, findings suggest promising potential for reducing complications and mortality rates in patients with kidney damages as well.[5,9,10] Moreover, EMPA reduces the rate of eGFR decline, decreases the development of albuminuria, and reduces the rate of cardiovascular mortality in patients with albuminuria.[11] Besides, dapagliflozin has some effects on proteinuria in non-diabetic patients with chronic kidney disease. Although the mentioned effect was not significant, it has caused not only body weight loss but also an acute and reversible decrease in eGFR.[12,13]

As stated, it seems that additional clinical studies should be conducted to determine whether SGLT2i such as EMPA differ from other SGLT2is in its effect. Moreover, no studies have specifically evaluated the effect of EMPA on patients with glomerulonephritis. Therefore, the present study aimed at investigating the effect of EMPA on proteinuria in non-DM patients with glomerulonephritis during 2020-2024.

MATERIALS AND METHODS

Study Design

This study was a randomized double-blind clinical trial and was approved by the Ethics Committee of Isfahan University of Medical Sciences (Approval no: IR.MUI.MED.REC.1400.419; Date: 2021-09-01 and the Clinical Trial Code: IRCT20090127001598N6v).

MAIN POINTS

- The obtained results showed that the 3-month administration of EMPA with a low dosage (10 mg) had no adverse effects on the patients' hemodynamic parameters and blood factors.
- The obtained results indicated that EMPA administration slightly and non-significantly reduced eGFR.
- The obtained results revealed that EMPA administration significantly reduced proteinuria. Hence its prescription may be considered safe in this category of patients.

Setting and Participants

The sample size in each group was estimated to be 36 at the confidence level of 95%, the test power of 80%, and considering the results of previous studies[11] with regard to the SD values of eGFR in 2 groups with and without kidney problems treated with EMPA equal to 21 and 7.5, respectively, and the mean difference of 12.

The inclusion criteria in the study comprised patients over 18 years old, with a history of proteinuria and glomerulonephritis, and with eGFR ≥ 30 mL/min/1.73 m^2. The patients were excluded from the study and replaced with another sample in case of allergic reaction to EMPA drug, change of drug or drug dosage affecting proteinuria, decrease of patients' fasting blood sugar (FBS) from the normal range during the study, unwillingness to continue cooperation in the study, or failure to refer for further follow-ups. It should be noted that the diagnosis of glomerulonephritis was confirmed by a pathologist through kidney biopsy.

After obtaining written consent from eligible patients to enter the study, 72 patients with glomerulonephritis that referred to Nephrology Clinics in Isfahan during 2020-2024 were randomly selected. Then, the patients were divided into 2 groups of 36 using random allocation software.

Intervention

All patients continued their usual drugs including angiotensin-II receptor blockers (ARBs), calcineurin inhibitors (CNIs), etc. In addition, patients in the first group received EMPA 10 mg tablets (Gloripa®; Abidi Pharmaceutical Company, Tehran, Iran) daily for 3 months (EMPA group). The second group, as a control group, received placebo tablets from the same Pharmaceutical Company daily for 3 months. Furthermore, FBS and creatinine (Cr) were checked by the patients monthly to control hypoglycemia or the incidence of acute kidney injury (AKI).

Empagliflozin and placebo were provided by Prof. Abidi pharmaceutical company. It is worth mentioning that in order to comply with blinding conditions, matching placebo tablets, identical in color, shape, and prescription protocol to the active EMPA tablets, were prepared and given to the researcher. Therefore, the study employed double-blinding, and the data analyst also did not know the grouping categorization of the 2 groups.

Outcomes

At the beginning of the study, the patients' basic information such as age, gender, duration of glomerulonephritis, systolic blood pressure (SBP), diastolic blood pressure (DBP), and their clinical parameters including blood urea nitrogen (BUN), Cr, albumin (Alb), sodium (Na), potassium (K), FBS, HbA1c, triglyceride (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), total cholesterol (Tch), alanine transaminase (ALT), aspartate transaminase (AST), 24-hour proteinuria, eGFR, 24-hour creatinine, and 24-hour urine volume were recorded.

After completing the treatment period (3 months), the patients' clinical parameters including SBP, DBP, BUN, Cr, Na, K, FBS, HbA1c, Alb, TG, HDL, LDL, Tch, ALT, AST, 24-hour proteinuria, eGFR, 24-hour Cr, and the 24-hour urine volume were recorded.

Data Analysis

At the level of descriptive statistics, indicators such as mean, SD, frequency, and frequency percentage were used. At the level of inferential statistics, chi-squared test was used to compare the frequency distribution of qualitative variables between 2 groups. Moreover, independent samples *t*-tests and univariate analysis (with control of the base value) were used to compare the mean of quantitative variables between 2 groups. Furthermore, paired *t*-tests were applied to compare the mean of the quantitative variables after the intervention, as compared to before the intervention. The significance level was considered less than .05 in all analyses. Data analysis was done using SPSS statics software version 25.

RESULTS

Six patients from the control group (3 patients due to vomiting, 3 patients due to skin rash) and 6 patients from the EMPA group (4 patients due to vomiting and 2 patients due to skin rash) were excluded from the study. Ultimately, 30 patients remained in the EMPA group and 30 in the control group for final analyses (Figure 1).

The EMPA group consisted of 15 (50%) men and 15 (50%) women with a mean age of 44.37 ± 11.21 years, while the control group comprised 20 (66.7%) men and 10 (33.3%) women with a mean age of 43.63 ± 10.69 years (*P*-value = .295 and .796, respectively). Moreover, the mean change weight after the intervention, compared to before the intervention, decreased in the EMPA group and increased in the control group (mean difference: −0.25 ± 1.56 kg vs. +0.10 ± 1.71 kg; *P*-value < .001) (Table 1).

Examination of laboratory factors also found that none of the blood factors including BUN, Cr, Alb, Na, K, FBS, HbA1c, TG, HDL, LDL, Tch, ALT, and AST showed a significant difference between the 2 groups, either before and 3 months after the intervention (*P* > .05). Moreover, these factors did not change significantly after the intervention, as compared to before the intervention (*P* > .05) (Table 2).

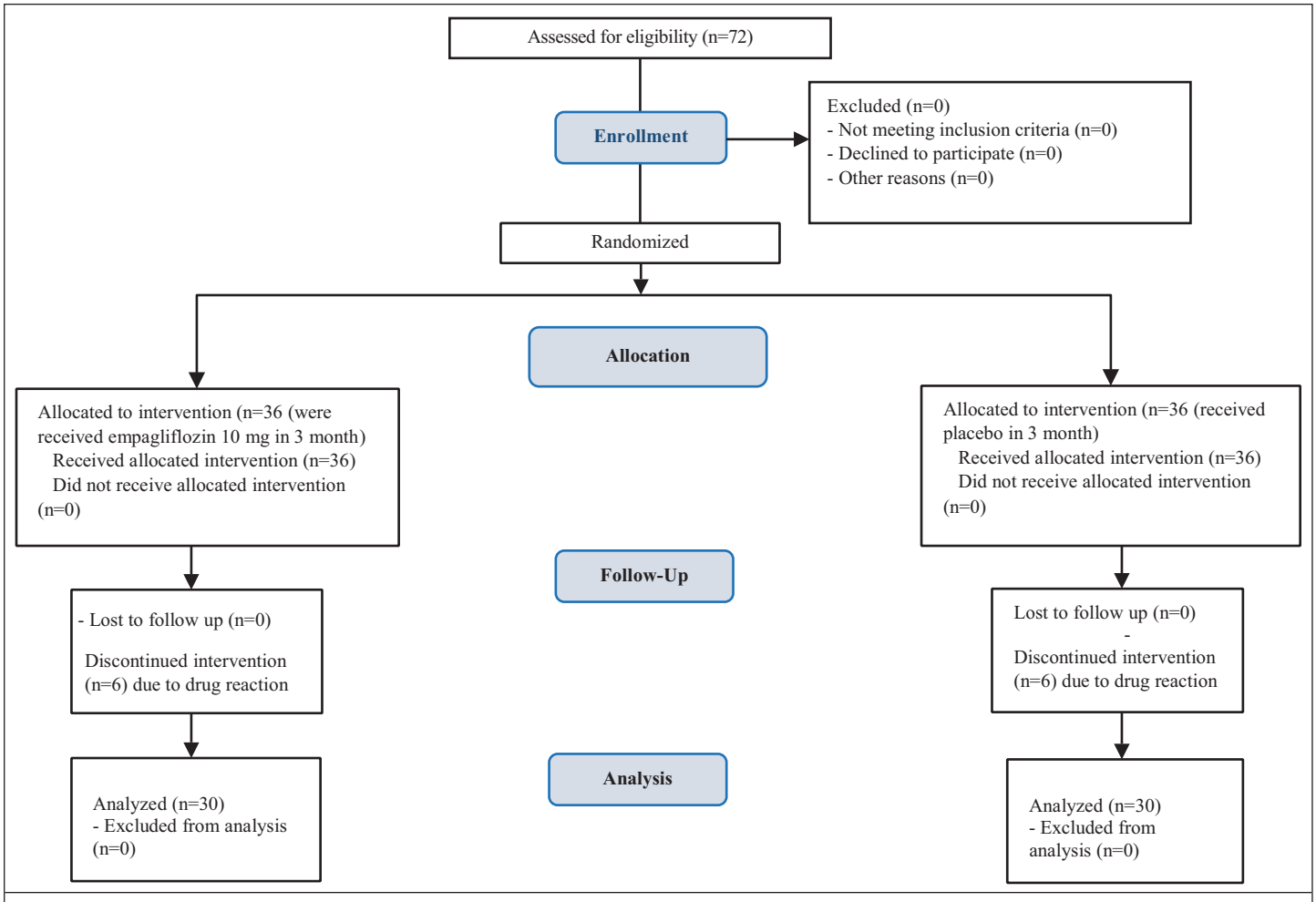

Figure 1. Consort flowchart of patients.

Table 1. Patients' Basic and Clinical Characteristics in the 2 Groups

Characteristics		EMPA Group (n = 30)	Control Group (n = 30)	*P*
Sex	Male	15 (50.0%)	20 (66.7%)	.295*
	Female	15 (50.0%)	10 (33.3%)	
Age±SD (years)		44.37 ± 11.21	43.63 ± 10.69	.796**
Duration of glomerulonephritis±SD (years)		8.05 ± 5.13	8.31 ± 4.80	.844**
Weight changes±SD (kg)		−0.25 ± 1.56	+0.10 ± 1.71	<.001**
Type of pathology	MGN	10 (33.3%)	11 (36.7%)	.143*
	MPGN	1 (3.3%)	0 (0%)	
	IgAN	7 (23.5%)	4 (13.3%)	
	FSGS	10 (33.3%)	11 (36.7%)	
	SLE	1 (3.3%)	3 (10%)	
	MCD	1 (3.3%)	1 (3.3%)	
SBP±SD (mmHg)	Baseline	82.17 ± 10.39	82.03 ± 8.50	.957**
	Third month	81.21 ± 7.63	80.26 ± 6.19	.614**
*P****		.445	.088	
DBP±SD (mmHg)	Baseline	82.17 ± 10.39	82.03 ± 8.50	.957**
	Third month	81.21 ± 7.63	80.26 ± 6.19	.614**
*P****		.610	.327	

DBP, diastolic blood pressure; EMPA, Empagliflozin; FSGS, focal segmental glomerulosclerosis; IgAN, immunoglobulin A nephropathy; MCD, minimal change disease MGN, membranous glomerulonephritis; MPGN, membranoproliferative glomerulonephritis; SBP, systolic blood pressure; SD, standart deviation; SLE, systemic lupus erythematous.
*The significance level obtained from the chi-square test comparing the 2 groups' distribution frequency of variables.
**The significance level obtained from the independent samples t-test comparing the 2 groups' mean of variables.
***The significance level obtained from the paired t-test comparing the mean of the variables after the intervention compared to before the intervention in each of 2 groups.

The eGFR in the EMPA (−2.72 mL/min/1.73m^2) and control (−1.89 mL/min/1.73m^2) groups decreased after the intervention, as compared to before the intervention, although this decrease was not statistically significant (*P* = .767). Proteinuria in the EMPA group was significantly lower after the intervention with a mean of 1326.73 ± 980.39 mg/24 h as compared to before the intervention with a mean of 1444.79 ± 897.18 mg/24h (Mean difference: −118.06 mg/24h; *P* = .008). However, proteinuria in the control group was significantly higher after the intervention with the mean of 1564.86 ± 380.13 mg/24h, as compared to before the intervention with the mean of 1331.17 ± 956.98 mg/24 h (mean difference: +233.69 mg/24 h; *P* = .003). Therefore, the level of proteinuria in the EMPA group was significantly lower than its level in the control group after 3 months (*P* = .022). Creatinine and 24-hour urine volume also increased insignificantly in both groups (*P* > .05), with no significant difference between the 2 groups (*P* > .05) (Table 3 and Figure 2).

DISCUSSION

The results of the present study conducted on 60 patients with glomerulonephritis revealed that none of the patients' laboratory factors and hemodynamic parameters had significant changes by prescribing EMPA for 3 months; however, the patients' weight decreased significantly during this prescription period.

In line with the results of the present study, Neeland et al[14] and Ridderstrale et al[15] have indicated that EMPA, as compared to placebo, significantly reduced body weight, waist circumference, and total and visceral adiposity indices in T2DM patients. Therefore, it seems to have promising potential for improving cardiometabolic risk in T2DM patients. In fact, this drug has diuretic effects, and SGLT-2i reduces blood sugar and body weight by inhibiting the absorption and transfer of glucose in the kidney.[16,17]

Furthermore, EMPA leads to the reduction of arterial stiffness, which is associated with the improvement of blood pressure control. Empagliflozin exerts a consistent weight loss, and which has been demonstrated to reduce blood pressure through various mechanisms including anti-inflammatory effects.[18,19] In the present study, the SBP and DBP slightly reduced, and finally the patients' hemodynamic stability was reported following 3 months of EMPA administration.

Besides, eGFR had a slight and non-significant decrease in both EMPA and control groups after the intervention. Creatinine and 24-hour urine volume also increased slightly

Table 2. Comparison of the Patients' Mean Laboratory Factors before and after the Intervention in the 2 Groups

Variables	Follow-up Time	EMPA Group (n = 30)	Control Group (n = 30)	*P*[1]
BUN (mg/dL)	Baseline	23.91 ± 11.67 20.00 [15.72-31.00]	22.42 ± 9.48 20.00 [16.05-28.00]	.593
	Third month	21.79 ± 10.31 21.00 [14.15-28.25]	21.62 ± 10.67 19.54 [11.97-29.92]	.951
P[2]		.100	.721	
Cr (mg/dL)	Baseline	1.25 ± 0.42 1.07 [0.97-1.55]	1.28 ± 0.42 1.19 [0.95-1.55]	.640
	Third month	1.27 ± 0.45 1.12 [0.95-1.43]	1.30 ± 0.47 1.20 [0.99-1.50]	.786
P[2]		.364	.956	
Na (mEq/L)	Baseline	139.22 ± 2.13 139.20 [137.75-140.77]	139.01 ± 4.42 138.75 [136.00-140.00]	.813
	Third month	140.10 ± 2.95 140.00 [138.00-142.25]	138.96 ± 2.29 138.90 [137.00-140.00]	.099
P[2]		.137	.956	
K (mmol/L)	Baseline	4.36 ± 0.59 4.30 [3.97-4.65]	4.33 ± 0.41 4.25 [3.99-4.62]	.845
	Third month	4.39 ± 0.50 4.30 [4.09-4.52]	4.34 ± 0.46 4.27 [3.97-4.72]	.699
P[2]		.640	.811	
FBS (mg/dL)	Baseline	94.57 ± 11.26 93.50 [85.00-101.75]	95.20 ± 13.23 96.00 [87.50-101.25]	.448
	Third month	93.00 ± 8.57 91.50 [86.75-98.25]	93.53 ± 15.62 89.50 [85.00-99.50]	.774
P[2]		.326	.346	
HbA1C (%)	Baseline	5.38 ± 0.46 5.20 [5.20-5.40]	5.37 ± 0.55 5.40 [5.13-5.65]	.938
	Third month	5.35 ± 0.59 5.15 [5.10-5.40]	5.36 ± 0.93 5.40 [5.10-5.90]	.188
P[2]		.727	.100	
Alb (g/dL)	Baseline	4.13 ± 0.55 4.20[3.80-4.52]	4.29 ± 0.42 4.40 [3.90-4.58]	.234
	Third month	4.12 ± 0.50 4.10 [3.80-4.47]	4.19 ± 0.35 4.30 [3.90-4.45]	.579
P[2]		0.954	0.145	
TG (mg/dL)	Baseline	155.62 ± 83.58 134.00 [93.50-190.00]	152.65 ± 50.59 127.00 [90.50-179.00]	.885
	Third month	149.03 ± 85.63 134.00 [85.00-183.00]	151.43 ± 99.29 140.5 [92.50-172.00]	.862
P[2]		.162	.986	
Tch (mg/dL)	Baseline	170.23 ± 51.99 163.00 [134.00-187.25]	156.86 ± 44.07 148.00 [137.50-173.00]	.292
	Third month	164.07 ± 42.95 163.00 [134.00-188.00]	156.10 ± 28.02 157.00 [140.50-174.00]	.406
P[2]		.244	.772	

(*Continued*)

Table 2. Comparison of the Patients' Mean Laboratory Factors before and after the Intervention in the 2 Groups (*Continued*)

Variables	Follow-up Time	EMPA Group (n = 30)	Control Group (n = 30)	*P*[1]
LDL (mg/dL)	Baseline	90.83 ± 37.84 85.50 [66.00-107.50]	80.74 ± 31.54 73.00 [57.00-101.25]	.287
	Third month	89.03 ± 31.92 89.50 [64.25-106.75]	78.68 ± 22.05 76.00 [65.50-94.50]	.154
P[2]		.696	.343	
HDL (mg/dL)	Baseline	48.80 ± 16.82 43.50 [35.77-63.00]	51.89 ± 15.15 50.00 [41.00-61.75]	.467
	Third month	45.89 ± 14.42 39.00 [35.00-58.00]	48.17 ± 11.68 48.50 [38.75-55.25]	.515
P[2]		.138	.169	
ALT (IU/L)	Baseline	26.40 ± 13.06 22.50 [17.75-34.25]	25.50 ± 13.64 21.00 [18.50-28.50]	.795
	Third month	25.60 ± 12.86 22.00 [14.75-34.00]	22.21 ± 8.72 20.00 [17.00-26.00]	.242
P[2]		.704	.232	
AST (IU/L)	Baseline	22.30 ± 8.96 18.00 [16.75-28.00]	23.30 ± 9.72 20.00 [17.75-26.25]	.680
	Third month	23.90 ± 9.68 22.00 [18.00-27.50]	19.76 ± 6.26 19.00 [15.50-22.00]	.057
P[2]		.281	.080	

Data shown mean ± SD and median [IQR]. Alb, albumin; ALT, alanine transaminase; AST, aspartate transaminase; BUN, blood urea nitrogen; Cr, creatinine; EMPA, Empagliflozin; FBS, fasting blood sugar; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; K, potassium; LDL, low-density lipoprotein; Na, sodium; TG, triglyceride; Tch, total cholesterol.
[1]The significance level obtained from the independent samples t-test comparing the 2 groups' mean of variables in each of the follow-up times.
[2]The significance level obtained from the paired t-test comparing the mean of the variables after the intervention compared to before the intervention in each of the 2 groups.

and non-significantly in both groups. In contrast, the patients' proteinuria decreased significantly following the EMPA administration. The level of proteinuria in the EMPA group was significantly lower than that of the control group after 3 months.

Sodium glucose co-transporter 2 inhibitors may protect the kidney through mechanisms similar to hyperglycemia-dependent pathways, potentially benefiting proteinuria from various causes characterized by hyperfiltration (e.g., obesity-related chronic kidney disease (CKD), focal segmental glomerular (FSGS), immunoglobulin A nephropathy (IgAN)).[20] Clinical trials showed that SGLT2is like empagliflozin significantly reduced albumin–creatinine ratio and preserved eGFR while lowering kidney/cardiovascular mortality.[17,21] Some other studies have reported that SGLT2i either have no adverse effects on kidney function[3] or may exert non-protective effects.[22,23]

Large-scale clinical trials on SGLT2i have revealed that although initial eGFR decreases rapidly, eGFR improves in SGLT2i users after 192 weeks of follow-up.[6,17] Another study also revealed that the cumulative reduction of more than 40% in eGFR <60 mL/min/1.73 m^2 of body surface area, kidney disease progression, or death due to kidney or cardiovascular causes occurred less frequently with dapagliflozin use, as compared to placebo.[24]

In fact, EMPA reduces the rate of eGFR decline, the progress of albuminuria, and the rate of cardiovascular mortality in albuminuric individuals.[17] Considering these results obtained from the EMPAREG trial, the recommended drug regimen was modified in patients with T2D and established cardiovascular disease.[25]

In addition, a significant decrease in urinary albumin-to-creatinine ratio has been observed in SGLT2i-treated patients, as compared to control patients, especially those with CKD after a certain follow-up period.[21,24]

Cherney et al[18] demonstrated that SGLT2i dapagliflozin did not reduce proteinuria in non-DM CKD patients after 6 weeks, despite causing transient eGFR reduction and weight loss. Although the findings of the mentioned study were contrary to those of the current study, in which EMPA had a significant role in reducing proteinuria, it may be stated that the difference of the studied sample can be the source of this variation in findings. Moreover,

Table 3. Comparison of the Patients' Mean Kidney Factors before and after the Intervention in the 2 Groups

Variables	Follow-up Time	EMPA Group (n = 30)	Control Group (n = 30)	P^1
eGFR mL/min/1.73 m^2	Baseline	69.73 ± 22.89	70.45 ± 27.68	.922
	Third month	67.01 ± 21.71	68.56 ± 23.27	.809*
eGFR difference		−2.72 −4.00 [−11.20 ± 4.62]	−1.89 −2.00 [−6.92 ± 4.00]	.767
P^2		0.224	0.417	
Proteinuria mg/24 h	Baseline	1444.79 ± 897.18	1331.17 ± 956.98	.637
	Third month	1326.73 ± 980.39	1564.86 ± 380.13	.022*
Proteinuria difference		−118.06 −248.00 [−524.25 ± 162.25]	233.69 −223.00 [−789.00 ± 135.12]	.013
P^2		.008	.003	
Cr mg/24 h	Baseline	1155.23 ± 411.26	1080.07 ± 290.86	.600
	Third month	1397.23 ± 330.64	1214.14 ± 446.79	.754*
Cr difference		242.00 −67.00 [−285.00 ± 213.00]	134.07 −83.00 [−378.00 ± 209.00]	.261
P^2		.461	.055	
Urine volume mL/24 h	Baseline	1681.00 ± 693.31	1705.86 ± 642.09	.887
	Third month	1711.17 ± 758.17	1755.00 ± 620.39	.861*
Urine volume difference		30.17 100.00 [−275.00 ± 325.00]	49.14 0.00 [−285.00 ± 425.00]	.997
P^2		.786	.730	

Data shown mean ± SD or median [IQR]. Cr, creatinine; eGFR, estimated glomerular filtration rate; EMPA, Empagliflozin; IQR, interquartile range; SD, standard deviation.
[1]The significance level obtained from the independent samples *t*-test comparing the 2 groups' mean of variables in each of the follow-up times.
[2]The significance level obtained from the paired *t*-test comparing the mean of the variables after the intervention compared to before the intervention in each of the 2 groups.
*Univariate analysis test comparing variables after the intervention between 2 groups while controlling the baseline value, sex, age, duration of glomerulonephritis, and type of pathology.

EMPA, as compared to dapagliflozin, may have a different effect on proteinuria, which highlights the necessity of conducting further studies in this respect.

In line with the present study, Hammad et al[26] presented that the administration of 25 mg of EMPA once a day for 3 months as a supplement to the usual treatment protocol (RAAS blockers

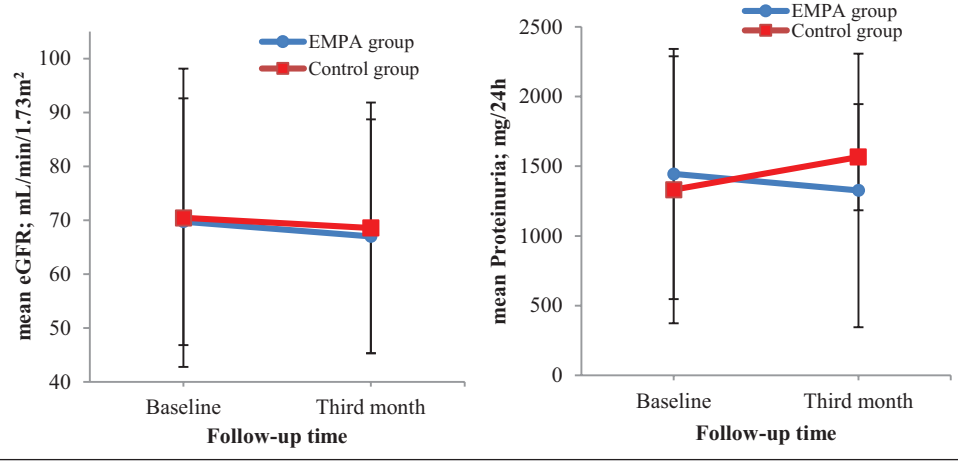

Figure 2. Changes in eGFR and proteinuria before and after the intervention in the 2 groups.

and immuno-suppressants) had a satisfactory effect on the improvement of proteinuria in patients with glomerulonephritis. Empagliflozin, as compared to placebo, has tended to preserve kidney function in patients with glomerulonephritis.[26]

Likewise, the results of another study have addressed the possible role of SGLT2i in reducing proteinuria and delaying the progression of chronic kidney disease in patients with nephrotic-range proteinuria.[27]

Some studies have reported very rare genital infections as well as urinary tract and asymptomatic bacteriuria after treatment with EMPA;[28,29] however, none of these complications were observed in the present study.

In addition to the small size of the sample, the lack of long-term follow-up can be considered as one of the other limitations of this study. Therefore, it is recommended to conduct further clinical trials with a larger sample size and a longer treatment duration evaluating the trend of changes in kidney and inflammatory factors, body weight, visceral fat, and waist size. In addition, the comparative evaluation of the effect of different doses of EMPA or the effect of different SGLT2i drugs on proteinuria as well as hemodynamic and metabolic stability of patients with various diabetic and non-diabetic diseases or type of pathology of glomerulonephritis such as (membranoproliferative glomerulonephritis (MPGN), IgAN, FSGS, systemic lupus erythematous (SLE), etc) can be fascinating.

CONCLUSION

According to the results of the present study, the 3-month administration of EMPA with a low dosage (10 mg) had no adverse effects on the patients' hemodynamic parameters and blood factors. Furthermore, the rate of decline in eGFR, and increase in Cr and urinary output was not significant, while it was associated with a significant decrease in proteinuria in patients with glomerulonephritis. Therefore, in this patient population, prescribing this drug may be considered a safe therapeutic option for reducing selective proteinuria.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author, J.G. The data are not publicly available due to their containing information that could compromise the privacy of research participants.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of Isfahan University of Medical Sciences (Approval no: IR.MUI.MED.REC.1400.419 Date: 01.09.2021).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer reviewed.

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