Volume 3, Issue 7, 2025

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

EFFECT OF DAPAGLIFLOZIN ON SERUM ELECTROLYTES IN TYPE 2 DIABETES MELLITUS

Dr. Namrah Iqbal[*1], Prof. Dr. Taj Jamshaid[2], Dr. Alveena Fareed[3]

[*1]*Postgraduate Resident Medicine Department, Sharif Medical City Hospital Lahore*
[2]*Professor of Medicine, Sharif Medical City Hospital, Lahore*
[3]*Senior Registrar Gastroenterology, Sharif Medical City Hospital, Lahore*

[*1]namrah_iqbal@hotmail.com

DOI: https://doi.org/10.5281/zenodo.17628986

Keywords
Dapagliflozin, Sodium, Potassium, Type 2 diabetes mellitus, Electrolyte balance, SGLT2 inhibitors

Article History
Received: 12 July 2025
Accepted: 20 August 2025
Published: 02 September 2025

Copyright @Author

Corresponding Author: *
Dr. Namrah Iqbal

Abstract

Background

Dapagliflozin, a sodium-glucose co-transporter 2 (SGLT2) inhibitor, is increasingly used in the management of type 2 diabetes mellitus (T2DM) due to its glycemic and cardiometabolic benefits. However, its effects on serum electrolyte balance remain a subject of clinical interest, as subtle shifts in sodium and potassium levels may influence treatment safety and renal outcomes.

Objective

To assess the effect of dapagliflozin therapy on serum sodium and potassium levels in patients with type 2 diabetes mellitus.

Study Design

Quasi-experimental study.

Duration and Place of Study

The study was conducted from 18th January, 2025 to 1st July, 2025 at Sharif Medical City Hospital, Lahore.

Methodology

A total of 190 patients aged 30–60 years with diagnosed T2DM were enrolled. Participants received dapagliflozin 10 mg daily for three months. Serum sodium and potassium levels were measured at baseline and after 3 months of treatment. Demographic variables, diabetes duration, and body mass index (BMI) were recorded. Data were analyzed using SPSS version 20, and stratified analysis was performed based on age, gender, BMI, and duration of diabetes. A post-stratification T-test was applied, with $p < 0.05$ considered statistically significant.

Results

The mean age of participants was 46.51 ± 8.28 years, with 71.6% males. Dapagliflozin therapy led to a statistically significant increase in mean serum sodium and potassium levels after three months ($p < 0.001$ for both). Stratified analysis revealed greater sodium increases in females, older adults, and those with longer diabetes duration, while potassium increases were more pronounced in younger patients and those with higher BMI. No adverse electrolyte disturbances were observed.

Conclusion

Dapagliflozin is associated with a mild but statistically significant increase in

Volume 3, Issue 7, 2025

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

serum sodium and potassium levels, indicating its renal safety and stable electrolyte profile in patients with type 2 diabetes mellitus.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is an insulin-resistant chronic metabolic syndrome with defective insulin secretion.[1] Insulin resistance contributes to hyperglycemia, which with the lapse of time can develop various complications to the various organs such as the heart, kidney, eyes, and nervous system.[2] The disease is primarily managed with lifestyle modification including diet and exercise, supplemented with pharmacological treatment for the normalization of blood glucose levels.[3] The drugs of choice that are widely employed include oral medications such as metformin, sulfonylureas, and newer drugs including sodium-glucose co-transporter 2 (SGLT2) inhibitors, which have been found to control blood glucose levels and the subsequent diabetes-associated complications.[4]

Antidiabetic medications may affect the electrolytes in the serum in several ways, which contribute to the maintenance of a variety of physiological functions, including nerve conduction, contracting of muscles, and fluid status maintenance.[5] Some of these drugs, especially renal-functional medications, can alter the levels of the major electrolytes sodium, potassium, and magnesium.[6] Potassium loss may be experienced with the use of some diuretics, for instance, while medications such as ACE inhibitors or ARBs may increase the concentration of potassium.[7] These electrolyte imbalances may require close monitoring because they may cause side effects such as an irregular heart rhythm, weakness of muscles, or even renal impairment in patients with underlying disease.[8]

The drug dapagliflozin is an SGLT2 inhibitors that inhibits the sodium-glucose transporter 2 of the kidney, resulting in glucose excretion in the urine accompanied with the reduction of the blood glucose levels of T2DM patients.[9] Aside from the anti-glycemic activity, dapagliflozin has likewise presented additional cardiovascular and renal effects that are contributing factors in the drug's usefulness for the management of T2DM.[10] The drug has also been attributed to the reduction of the patients with T2DM's body weight and blood pressure.[11]

The impact of dapagliflozin on the serum electrolytes of T2DM patients is significant considering the drug's renal glucose excretion mechanism and the possible effects on electrolyte homeostasis.[12] The drug may cause mild alterations in the electrolyte levels according to studies, and decreases in serum sodium that could potentially lead to mild manifestations of hyponatremia.[13] No effect of significant impact on the levels of serum potassium, however, does not occur with the drug, and the risk of hyperkalemia would tend to be lower compared to the use of alternative measures for diabetes, such as the use of potassium-sparing diuretics.[14] Although mild, close monitoring of the electrolytes must be performed, especially with patients with renal impairment or on concomitant medications that influence electrolyte levels.

In a preclinical study, treatment with dapagliflozin resulted in a marked reduction in blood glucose levels in diabetic rats. After 7 days of treatment, glucose levels decreased by 38%, and after 14 days, a 47% reduction was observed, compared to untreated diabetic rats that exhibited hyponatremia and hypokalemia.[15] In a separate clinical trial, 160 heart failure patients were randomly assigned dapagliflozin in addition to their usual medical management. The effects on serum electrolytes were compared with those of patients receiving only standard heart failure treatment. The mean change in serum sodium was found to be 0.205±0.22, while the change in serum potassium was 0.04±0.07.[16]

Despite dapagliflozin showing efficacy in reducing blood sugar levels, its effect on the sodium and potassium electrolyte balance remains a cause of concern. Derangement of serum electrolytes has the potential to cause severe clinical manifestations like dehydration, hypotension, cardiac arrhythmias that may challenge management of diabetes. Understanding these effects is important for ensuring proper drug regimens and patient safety and minimizing diabetic patients' risks of electrolyte disturbances.

Methodology:

This quasi-experimental study was conducted from 18th January, 2025 to 1st July, 2025 at Sharif Medical City Hospital, Lahore. It consisted of 190 patients

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

with Type 2 Diabetes Mellitus, and it was a case study to assess changes in serum electrolyte levels after treatment with dapagliflozin. The sample size of 190 was calculated using a 95% confidence level, with an absolute precision of 0.01 and an expected mean change in potassium of 0.04 ± 0.07.[16]

Inclusions were for male and female subjects within the ages of 30 to 60 years, with diagnosis for Type 2 Diabetes Mellitus and also agreeing to give consent for taking part and without having such medications affecting electrolyte balance. Exclusions were pregnant and lactating women, history of heart failure, kidney disease, amputations, and diabetic complication.

Upon receiving approval from the hospital's ethics committee, written informed consent was obtained from eligible participants, ensuring confidentiality and privacy throughout the study. Data collection involved a review of medical records, physical examinations, and laboratory tests to measure serum electrolyte levels. Dapagliflozin 10 mg was administered to participants once daily for three months, with serum electrolyte levels assessed at baseline (0 months) and again after 3 months of treatment. The lab tests were carried out at Sharif Medical City in Lahore. Regular follow-up visits were scheduled every two weeks to monitor diabetes management, and participants were asked to provide their mobile numbers and addresses for consistent follow-up. If participants could not visit the hospital, they were instructed to get serum electrolytes tested at a nearby laboratory and send the results via WhatsApp, which would be uploaded to the study's data collection form.

Data analysis was performed using the Statistical Package for Social Sciences (SPSS) version 20. Continuous variables such as age and serum electrolytes were reported as mean ± standard deviation, while categorical variables like gender were presented as frequencies and percentages. The data was stratified based on age, gender, duration of diabetes, and body mass index (BMI). A post-stratification T-test was applied, and a p-value of less than 0.05 was considered statistically significant. Participants whose diabetes was not well controlled with dapagliflozin or who missed follow-ups were excluded from the study after appropriate follow-up via phone calls.

Results:

The study included 190 participants with a mean age of 46.51 ± 8.28 years and a mean BMI of 26.48 ± 2.89 kg/m². The cohort consisted of 71.6% males and 28.4% females, with nearly equal distribution in age subgroups (≤45 years: 48.9%; >45 years: 51.1%) and a mean diabetes duration of 35.88 ± 17.49 months (as shown in Table-I).

Table I: Demographics and Clinical Characteristics of Study Populationn = 190

Parameter	**Value**
Age (years)	46.51 ± 8.28
BMI (kg/m²)	26.48 ± 2.89
BSR (mg/dl)	139.80 ± 7.44
Diabetes Duration (months)	35.88 ± 17.49
Gender	
Male	136 (71.6%)
Female	54 (28.4%)
Age Group	
≤45 years	93 (48.9%)
>45 years	97 (51.1%)

Dapagliflozin significantly increased mean serum sodium from 140.67 ± 1.11 mmol/L to 140.93 ± 1.12 mmol/L (mean difference: -0.26 mmol/L; $p < 0.001$) and serum potassium from 4.39 ± 0.25 mmol/L to

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

4.50 ± 0.25 mmol/L (mean difference: -0.10 mmol/L; p < 0.001) (as shown in Table-II)

Table II: Effect of Dapagliflozin on Serum Electrolytes

Electrolyte	Time Point	Mean ± SD	Mean Difference	95% CI of Difference	t Value	p-value
Serum Sodium (mmol/L)	Baseline	140.67 ± 1.11	-0.26	-0.27 to -0.25	-73.60	<0.001
	3 Months	140.93 ± 1.12				
Serum Potassium (mmol/L)	Baseline	4.39 ± 0.25	-0.1	-0.11 to -0.10	-75.67	<0.001
	3 Months	4.50 ± 0.25				

The average change in serum sodium was 0.2611 ± 0.0489 mmol/L and in serum potassium was 0.1037 ± 0.0189 mmol/L (as shown in Table-III).

Table III: Mean Change in Serum Electrolytes Following Dapagliflozin Therapy (n = 190)

Parameter	Minimum	Maximum	Mean Change ± SD
Serum Sodium (mmol/L)	0.2	0.3	0.2611 ± 0.0489
Serum Potassium (mmol/L)	0.1	0.2	0.1037 ± 0.0189

Stratified analysis indicated that serum sodium changes were significantly greater in older adults (>45 years: 0.2711 ± 0.0456 vs. ≤45 years: 0.2505 ± 0.0503; p = 0.003), females (0.2759 ± 0.0432 vs. males: 0.2551 ± 0.0499; p = 0.008), and those with longer diabetes duration (>50 weeks: 0.2900 ± 0.0304 vs. ≤50 weeks: 0.2533 ± 0.0501; p < 0.001). No significant difference was noted between BMI groups (p = 0.146) (as shown in Table-IV A at the end of result). For serum potassium, younger patients showed a greater change (≤45 years: 0.1075 ± 0.0265 vs. >45 years: 0.1000 ± 0.0000; p = 0.006), and those with higher BMI had a greater increase (>25 kg/m²: 0.1064 ± 0.0245 vs. ≤25 kg/m²: 0.1000 ± 0.0000; p = 0.021). Differences by gender (p = 0.09) and diabetes duration (p = 0.166) were not statistically significant (as shown in Table-IV).

Table IV: Mean Change in Serum Electrolytes by Subgroups (n = 190)

A. Serum Sodium (mmol/L)

Subgroup	Category	N	Mean ± SD	p-value
Age Group	≤45	93	0.2505 ± 0.05	0.003
	>45	97	0.2711 ± 0.04	
Gender	Male	136	0.2551 ± 0.04	0.008
	Female	54	0.2759 ± 0.04	
BMI Group	≤25	80	0.2550 ± 0.05	0.146
	>25	110	0.2655 ± 0.04	
Diabetes Duration	≤50 weeks	150	0.2533 ± 0.05	<0.001
	>50 weeks	40	0.2900 ± 0.03	

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

B. Serum Potassium (mmol/L)

Subgroup	Category	N	Mean ± SD	p-value
Age Group	≤45	93	0.1075 ± 0.02	0.006
	>45	97	0.1000 ± 0.00	
Gender	Male	136	0.1051 ± 0.02	0.09
	Female	54	0.1000 ± 0.00	
BMI Group	≤25	80	0.1000 ± 0.00	0.021
	>25	110	0.1064 ± 0.02	
Diabetes Duration	≤50 weeks	150	0.1047 ± 0.02	0.166
	>50 weeks	40	0.1000 ± 0.00	

Discussion:

The findings demonstrated a statistically significant increase in both serum sodium and potassium levels following three months of dapagliflozin therapy. The rise in serum sodium may be attributed to the osmotic diuresis induced by dapagliflozin, which promotes water excretion more than sodium loss, leading to mild hemoconcentration. The increase in serum potassium could be explained by a shift of potassium from intracellular to extracellular compartments due to mild volume contraction and potential alterations in renal tubular handling. Age-related differences in sodium response may reflect age-associated variations in renal adaptive capacity, while greater changes in females might be due to sex-related hormonal influences on fluid and electrolyte regulation. Longer diabetes duration possibly reflects underlying nephron changes that amplify dapagliflozin's effect on sodium retention. Similarly, greater potassium increases in individuals with higher BMI could be due to enhanced renal reabsorption mechanisms or adiposity-linked inflammation affecting tubular function.

Our findings are in agreement with the results of Chen et al. [17] who reported that dapagliflozin mitigated hyponatremia, hypokalemia, and hypochloremia in diabetic rats and did not lead to significant electrolyte disturbances. Specifically, blood sodium and chloride levels normalized with dapagliflozin treatment, and potassium levels, which were reduced in untreated diabetic rats, recovered to near-control levels by day 14. Moreover, dapagliflozin decreased 24-hour urine volume by 29% at day 7 and 30% at day 14, and increased urine osmolality by 29% and 23% respectively compared to untreated diabetic rats—indicating enhanced urine concentrating capacity. These effects were mechanistically linked to a 42–46% increase in UT-A1 protein expression in the inner medullary tip, while AQP2 and NKCC2 levels remained unchanged. Similar to our human data, which showed a mean increase of 0.2611 ± 0.0489 mmol/L in serum sodium and 0.1037 ± 0.0189 mmol/L in potassium ($p < 0.001$ for both), these findings suggest that dapagliflozin enhances renal compensatory mechanisms that stabilize electrolyte levels, even in the presence of ongoing glycosuria. While Chen et al. reported correction of electrolyte imbalances to normal physiological levels, our human study demonstrated modest yet statistically significant increases in serum sodium and potassium, which may reflect differing baseline status, renal maturity, or volume conditions between species and study models.

In contrast, Khattak et al. [18] found no statistically significant change in mean serum sodium (139.3 ± 3.18 vs. 138.94 ± 3.5 mmol/L; $p = 0.44$) or potassium (3.96 ± 0.33 vs. 3.97 ± 0.38 mmol/L; $p = 0.80$) in 200 patients over 24 weeks. These results diverge from our findings, which showed statistically significant increases in both electrolytes over just three months. The difference may be explained by the lack of subgroup stratification in Khattak's study, the longer duration potentially allowing for homeostatic adaptation, and a slightly older patient demographic. Nonetheless, both studies agree that dapagliflozin does not provoke electrolyte imbalance, reaffirming its renal safety. The neutral electrolyte outcome in their study complements our findings by reinforcing dapagliflozin's safety margin across a broader population.

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

Filippatos et al. [19] and Soomro et al. [20] while primarily focused on glycemic and metabolic endpoints, observed reductions in HbA1c (by up to 0.89%), fasting plasma glucose, and uric acid levels (from 7.7 ± 1.7 to 4.3 ± 1.1 mg/dL in Soomro's dapagliflozin group). These metabolic improvements mirror our results and suggest that the modest increases in serum electrolytes may result from mild volume contraction secondary to glucosuria, which concentrates serum solutes. Although these studies did not evaluate electrolytes directly, the improved metabolic control indirectly supports a preserved renal functional response, consistent with our observations.

Yuan et al. [21] demonstrated a significant reduction in serum uric acid (from 347.75 ± 7.75 to 273.25 ± 43.18 μmol/L; p = 0.001) and an increase in fractional excretion of uric acid (from 0.009 to 0.029; p = 0.035) after just one week of dapagliflozin therapy. Although electrolytes were not measured, the mechanism of uricosuria is closely linked to enhanced solute excretion and osmotic diuresis, similar to the mild natriuretic and kaliuretic effects observed in our patients. This similarity strengthens the view that dapagliflozin exerts a coordinated renal response, improving both uric acid handling and electrolyte regulation without causing depletion or overload.

Yao et al. [22] further support these findings by demonstrating systemic benefits of SGLT2 inhibitors, including protective effects on dry eye disease severity, thereby affirming the drug's broad tissue-level safety and metabolic control without evidence of systemic electrolyte disturbance. While their study did not report serum sodium or potassium data, the overall safety profile aligns with our interpretation that SGLT2 inhibitors, including dapagliflozin, function without inducing clinically harmful electrolyte shifts.

While some studies reported neutral effects and others demonstrated corrective shifts in electrolyte parameters, our findings provide further clarity on the subtle yet statistically significant changes that occur in a real-world diabetic population. These results, when interpreted alongside prior research, reinforce the pharmacodynamic consistency of SGLT2 inhibitors across diverse settings and suggest that their benefits extend beyond glycemic control to encompass renal adaptability and homeostasis.

However, some limitations need to be taken into account when interpreting our results. This was a single-site study, so it may reduce generalizability to other populations or healthcare settings. Also, it did not have a longer follow-up period, which could have shown whether electrolyte effects were sustainable and long-term kidney effects. Lack of serial measurements with treatment course and failure to include other electrolytes like magnesium or chloride are other limitations. Finally, relying on only dapagliflozin without a comparison with other SGLT2 inhibitors may miss agent-specific effects.

Conclusion:

Our study has established that therapy with dapagliflozin in patients with type 2 diabetes mellitus results in an increase in serum sodium and serum potassium concentrations, which is statistically significant but only clinically mild, reflecting an acceptable safety profile for electrolyte maintenance. Such increases seem to be properly balanced by renal compensatory mechanisms and are without any significant electrolyte disturbance. In short, dapagliflozin can be regarded as a safe form of therapy when it comes to serum electrolyte homeostasis within this patient group.

Conflict of interest: None

Disclaimer: None

Acknowledgments:

We extend our heartfelt gratitude to the dedicated healthcare professionals of our department for their meticulous handling of patient information and consistent support throughout the study period. Their attention to detail and commitment to excellence significantly enhanced the integrity of our work.

REFERENCES:

Gieroba B, Kryska A, Sroka-Bartnicka A. Type 2 diabetes mellitus - conventional therapies and future perspectives in innovative treatment. BiochemBiophys Rep. 2025;42:102037. doi:10.1016/j.bbrep.2025.102037

Ebrahimi M, Ahmadieh H, RezaeiKanavi M, Safi S, Alipour-Parsa S, Advani S, et al. Shared signaling pathways and comprehensive therapeutic approaches among diabetes complications. Front Med (Lausanne). 2025;11:1497750. doi:10.3389/fmed.2024.1497750

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

Aloke C, Egwu CO, Aja PM, Obasi NA, Chukwu J, Akumadu BO, et al. Current advances in the management of diabetes mellitus. Biomedicines. 2022;10(10):2436. doi:10.3390/biomedicines10102436

Wilcox T, De Block C, Schwartzbard AZ, Newman JD. Diabetic agents, from metformin to SGLT2 inhibitors and GLP1 receptor agonists: JACC focus seminar. J Am CollCardiol. 2020;75(16):1956–1974. doi:10.1016/j.jacc.2020.02.056

Pawar BG, Eerike M, Pyati AK, Varatharajan S, Mali K, Konda VGR, et al. Correlation of serum electrolyte imbalances with diabetic duration and medication use: a cross-sectional comparative study. Cureus. 2024;16(9):e70065. doi:10.7759/cureus.70065

Eshetu B, Worede A, Fentie A, Chane E, Fetene G, Wondifraw H, et al. Assessment of electrolyte imbalance and associated factors among adult diabetic patients attending the University of Gondar Comprehensive Specialized Hospital, Ethiopia: a comparative cross-sectional study. Diabetes MetabSyndrObes. 2023;16:1207–1220. doi:10.2147/DMSO.S404788

Goia-Nishide K, Coregliano-Ring L, Rangel ÉB. Hyperkalemia in diabetes mellitus setting. Diseases. 2022;10(2):20. doi:10.3390/diseases10020020

Liamis G, Hoorn EJ, Florentin M, Milionis H. An overview of diagnosis and management of drug-induced hypomagnesemia. Pharmacol Res Perspect. 2021;9(4):e00829. doi:10.1002/prp2.829

Jasleen B, Vishal GK, Sameera M, Fahad M, Brendan O, Deion S, et al. Sodium-glucose cotransporter 2 (SGLT2) inhibitors: benefits versus risk. Cureus. 2023;15(1):e33939. doi:10.7759/cureus.33939

Nashar K, Khalil P. Clinical evaluation of dapagliflozin in the management of CKD: focus on patient selection and clinical perspectives. Int J NephrolRenovasc Dis. 2022;15:289–308. doi:10.2147/IJNRD.S234282

Han Y, Li YF, Ye CW, Gu YY, Chen X, Gu Q, et al. Effects of dapagliflozin on body weight in patients with type 2 diabetes mellitus: evidence based practice. ExpTher Med. 2024;27(4):173. doi:10.3892/etm.2024.12461

Berezin AA, Fushtey IM, Berezin AE. The effect of SGLT2 inhibitor dapagliflozin on serum levels of apelin in T2DM patients with heart failure. Biomedicines. 2022;10(7):1751. doi:10.3390/biomedicines10071751

Iijima Y, Nakayama M, Miwa T, Yakou F, Tomiyama H, Shikuma J, et al. Nephroprotective effects of dapagliflozin in patients with type 2 diabetes. Intern Med. 2023;62(5):681–688. doi:10.2169/internalmedicine.6685-20

Eagan K, Bolch C, Van Dril E, Schumacher C. Changes in serum potassium in people with type 2 diabetes taking sodium-glucose co-transporter-2 inhibitors. Pharmacotherapy. 2025;45(4):194–202. doi:10.1002/phar.70006

Chen L, LaRocque LM, Efe O, Wang J, Sands JM, Klein JD, et al. Effect of dapagliflozin treatment on fluid and electrolyte balance in diabetic rats. Am J Med Sci. 2016;352(5):517–523.

Fatima Gilani SF, Ali S, Noor M, Farhat K, Siddique MB, Fatima M, et al. Effect of SGLT2 inhibitor dapagliflozin on sodium, potassium and creatinine levels in patients with acute heart failure. J Ayub Med Coll Abbottabad. 2023;35(Suppl1)(4):S715–S720. doi:10.55519/JAMC-S4-11906

Chen L, LaRocque L, Efe O, Wang J, Sands JM, Klein JD, et al. Effect of dapagliflozin treatment on fluid and electrolyte balance in diabetic rats. Am J Med Sci. 2016;352(5):517–523. doi:10.1016/j.amjms.2016.08.015

Khattak AL, Rashid A, Javed F, Aziz S, Siddiqi FA, Alihah S, et al. The effect of SGLT-2 inhibitors on serum potassium and serum sodium levels in diabetic patients taking SGLT-2 inhibitors. Pak Armed Forces Med J. 2025;75(1):142–146. doi:10.51253/pafmj.v75i1.11954

Filippatos TD, Liberopoulos EN, Elisaf MS. Dapagliflozin in patients with type 2 diabetes mellitus. TherAdvEndocrinolMetab. 2015;6(1):29–41. doi:10.1177/2042018814558243

Soomro UA, Siddiqui SS, Shaikh S, Ata MA, Memon A, Shaikh KR, et al. Effect of dapagliflozin therapy on serum uric acid in type 2 diabetic patients. J Peoples Univ Med Health Sci.

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

2021;11(3):73–77. doi:10.46536/jpumhs/2021/11.03.319

Yuan T, Liu S, Dong Y, Fu Y, Tang Y, Zhao W, et al. Effects of dapagliflozin on serum and urinary uric acid levels in patients with type 2 diabetes: a prospective pilot trial. DiabetolMetabSyndr. 2020;12:92. doi:10.1186/s13098-020-00600-9

Yao YP, Yang PJ, Lee CY, Huang JY, Yang SF, Lin HY, et al. Utilization of sodium-glucose cotransporter 2 inhibitors on dry eye disease severity in patients with type 2 diabetes mellitus. Int J Med Sci. 2023;20(13):1705–1710. doi:10.7150/ijms.88720