INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

OPEN ACCESS

Case Study

Evaluation Of Renal and Cardiovascular Outcomes with Dapagliflozin Plus Telmisartan in Patients with Diabetic Nephropathy

Ambala Syama*, Asma Bano, Banda Vaishnavi, Nallavelli Lalith Adithya, Shanigaram Deekshith Reddy

Bharat School of Pharmacy.

ARTICLE INFO

Published: 12 Aug 2026
Keywords:
Diabetic Nephropathy, Dapagliflozin, Telmisartan, Serum Creatinine, UACR, eGFR, Blood Pressure
DOI:
10.5281/zenodo.21901590

ABSTRACT

Diabetic nephropathy is a major complication of type 2 diabetes mellitus and a leading cause of chronic kidney disease. This study evaluated the renal and cardiovascular outcomes of dapagliflozin plus telmisartan in patients with diabetic nephropathy. Renal parameters (serum creatinine, eGFR, UACR, and BUN) and cardiovascular parameters (blood pressure and HbA1c) were assessed at baseline and follow-up. Combination therapy significantly improved renal function, reduced albuminuria, improved glycaemic control, and lowered blood pressure ($p < 0.05$). These findings suggest that dapagliflozin plus telmisartan is an effective therapeutic strategy for improving renal and cardiovascular outcomes in patients with diabetic nephropathy

INTRODUCTION

DIABETIC NEPHROPATHY:

Diabetic nephropathy (DN) is a characteristic set of structural and functional kidney abnormalities in patients with diabetes.

Structural abnormalities – Hypertrophy of the kidney.
Increase in glomerular basement membrane thickness.
Nodular and diffuse glomerulosclerosis.
Tubular atrophy.
Interstitial fibrosis.

Functional abnormalities – Early increase in glomerular filtration rate with intraglomerular hypertension.
Subsequent proteinuria.
Systemic hypertension.
Eventual loss of renal function.[1]

STAGES:

***Corresponding Author:** Ambala Syama
Address: *Bharat School of Pharmacy.*
Email ✉: ambalasyama@gmail.com
Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Mogensen et al. has suggested that Diabetic nephropathy can be divided into 5 stages:

STAGES	DESCRIPTION	GFR
Early hypertrophy stage	Kidney damage present (microscopic changes or increased filtration), but overall function is normal.	90 or above(normal)
Silent stage	Early damage, possibly with slightly increased protein (microalbuminuria) in urine, but function is still good.	60 – 89(mildly decreased)
Incipient DN	Moderate to severe loss of function; protein levels in urine increase, indicating ongoing damage.	30 – 59(moderate)
Overt DN	Severe decline in kidney function; symptoms like swelling, fatigue, and changes in urination may appear.	15 – 29(severe)
End-stage renal disease	The kidneys have failed or are very close to failing, requiring dialysis or a kidney transplant.	Below 15(kidney failure)

It is often difficult to document these various stages in a diabetic patient in clinical practice because of confounding factors such as blood pressure medications, as they modify natural course of DN.[1,2]

EPIDEMOLOGY:

About 30-
50% of persons with type 2 diabetes have albumi nuria, and 20% have reduced kidney function (eG FR < 60 mL/min/1.73 m2). Each year, 2-3% of patients with T1DM and 8% of patients wit h T2DM have albuminuria, and 2-
4% of patients with any kind of diabetes have a lo wer eGFR.[3]
Epidemiological data show that about 14% of adults in the United States have CKD, and nearly one-third of these individuals also have diabetes. Overall, 30–40% of people with diabetes develop diabetic nephropathy. With the global prevalence of diabetes expected to exceed 783 million by 2045, diabetic kidney disease will remain a major public health challenge and an important cause of morbidity and mortality.[4]

Diabetic kidney disease(DKD) is one of the most common and serious long-term consequences of diabetes mellitus. About 50% of people with type 2 diabetes (T2DM) and one-third of people with type 1 diabetes (T1DM) have it. DKD is a major contributor to chronic kidney disease (CKD) and has a significant effect on illness, medical costs, and mortality. About 30-50% of persons with type 2 diabetes have albuminuria and 20% have reduced kidney function (eEach

ETIOLOGY:

- Long-standing hyperglycaemia
- Thickening of the glomerular basement membrane
- Macrophage infiltration
- Endothelial cell injury
- Podocyte damage
- Polyol pathway:
- Genetic susceptibility [4]

PATHOGENESIS:

The pathogenesis of DN entails a multifaceted interplay of factors, encompassing alterations in

glomerular function, hormonal influences, and protein glycosylation. Elevated blood glucose levels induce the glycosylation of glomerular proteins, fostering mesangial cell proliferation, matrix expansion, and vascular endothelial impairment. Disease progression is marked by mesangial expansion, thickening of the glomerular basement membrane, and the emergence of characteristic lesions such as Kimmelstiel-Wilson nodules. Furthermore, renal vasodilation, heightened GFR, and elevated blood pressure are hallmark features of DN. While precise etiology remains incompletely understood, the pathogenesis is implicated in hyperglycaemia-induced renal injury, advanced glycation products, and cytokine activation. DN represents a primary cause of chronic kidney disease (CKD) in Western societies and a significant complication of diabetes, impacting up to 50% of individuals with longstanding diabetes.[5]

SYMPTOMS:

For the early diabetic nephropathy condition, there are no symptoms. When the kidney functioning gets worsen then the symptoms are:

- Swelling of the hands, feet, and face
- Trouble in sleeping or concentrating
- Poor appetite
- Nausea
- Weakness
- Dry skin and Itching (end-stage kidney disease)
- Drowsiness (end-stage kidney disease)
- Abnormalities in the heart's regular rhythm (because of increased potassium in the blood)
- Muscle twitching.[6]

RISK FACTORS:

- High blood sugar (hyperglycaemia).
- High blood pressure (hypertension).
- Smoking.
- High blood cholesterol.
- Obesity.
- Family history of diabetes and kidney disease. [7]

COMPLICATIONS:

- Fluid buildup in the body which leads to the swelling of the arms and legs, high blood pressure or fluid in the lungs (Pulmonary oedema).
- Rise or increase of the potassium mineral levels in blood (Hyperkalaemia).
- Heart and blood vessel disease (Cardiovascular disease) which can lead to stroke.
- Decrease in RBC to carry oxygen (Anaemia).
- Pregnancy complications that may cause risk for the mother and foetus.
- Permanent damage to the kidneys (end stage kidney disease), for this the treatment is either dialysis or kidney transplant.[8]

PREVENTION:

- Regular managing of the diabetes, by taking the appointments.
- The whole body checkup should be done every 6 months or yearly.
- Treating the diabetes by keeping the target range of blood sugar levels as much as possible, this helps in preventing or slowing the diabetic nephropathy.
- Managing the high blood pressure or other medical conditions, as it raises the risk of kidney disease.
- If the OTC medications like pain relievers that include aspirin and NSAIDs, such as Naproxen sodium (Aleve) and Ibuprofen (Advil, Mortin IB, etc) are taken, then can lead to the kidney damage.
- Maintaining the healthy body weight by consulting the qualified physician.

- Quit Cigarette smoking and alcohol intake, these can cause kidney damage or worsen the condition of the kidneys.

DIAGNOSIS:

1. **Physical examination:** Clinical signs and symptoms, general examination like vitals, pedal oedema or fluid retention, any skin infections or changes.
2. **Laboratory investigations:** Renal function tests, Complete blood picture.
3. **Urine Albumin-Creatinine Ratio (UACR):** Checks for albumin in urine, an early sign of damage.
4. **Estimated Glomerular Filtration Ratio (eGFR):** Calculates kidney filtering ability using serum creatinine.
5. **Serum Creatinine:** Measures waste product filtered by kidney.
6. **Blood Urea Nitrogen (BUN):** Measures the urea nitrogen in blood.
7. **Electrolytes:** Sodium, chloride, potassium, bicarbonates.
8. **Renal Biopsy:** A tissue sample examined for detailed damage, used when alternative diagnosis is suspected.
9. **Imaging (Ultrasound, CT, MRI):** Shows the size, shape, blockage or blood flow issues in the kidney.[9]

TREATMENT:

NON-PHARMACOLOGICAL TREATMENT:

Dietary modification – Low sodium intake, controlled protein diet, and low glycaemic index foods help reduce albuminuria and slow disease progression.

Regular physical activity – Aerobic and resistance exercises improve insulin sensitivity and may delay progression of diabetic nephropathy.

Weight management – Lifestyle-based weight reduction improves metabolic control and reduces renal hyperfiltration.

Lifestyle blood pressure control – Salt restriction, exercise, and stress reduction help prevent worsening of renal damage.

Smoking cessation – Avoidance of tobacco slows progression of kidney disease and reduces cardiovascular risk.

Patient education and self-management – Diabetes education and lifestyle counselling improve adherence and long-term renal outcomes.

PHARMACOLOGICAL TREATMENT:

Drug/Class	Examples	Mechanism of Action	Clinical Effect/Benefit
Antidiabetic agents	Insulin, Metformin, Sulfonylureas	Control blood glucose	Reduces hyperfiltration, delays nephropathy
ACE inhibitors	Enalapril, Ramipril	Inhibit RAAS → reduce intraglomerular pressure	Reduce proteinuria, slow CKD progression
ARBs	Telmisartan, Losartan	Block angiotensin II receptor → vasodilation	Alternative to ACEi; renoprotective
SGLT2 inhibitors	Dapagliflozin, Empagliflozin	Inhibit renal glucose reabsorption	Reduce albuminuria, protect kidneys & heart
Mineralocorticoid receptor antagonist	Finerenone	Anti-inflammatory, anti-fibrotic	Reduce albuminuria, slow CKD progression
Other antihypertensives	CCBs, Beta-blockers	Lower BP	Support renal and cardiovascular protection

Drug/Class	Examples	Mechanism of Action	Clinical Effect/Benefit
Lipid-lowering agents	Statins	HMG-CoA reductase inhibition	Reduce cardiovascular morbidity
Complication management [10]	Erythropoietin, Sodium bicarbonate	Treat anemia, acidosis	Support renal function and quality of life

ABOUT DRUG:

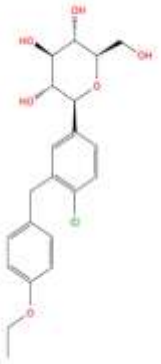
DAPAGLIFLOZIN + TELMISARTAN

INTRODUCTION:

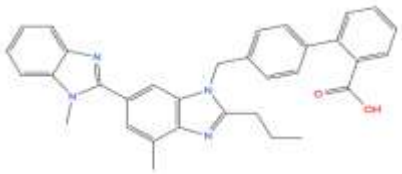
Dapagliflozin is an SGLT2 inhibitor that lowers blood sugar by encouraging the excretion of glucose in the urine. It also has other advantages like helping people lose weight and protecting their hearts. Angiotensin II receptor blockers (ARBs) like telmisartan help protect the kidneys and heart while also successfully regulating blood pressure.

For the treatment of patients with type 2 diabetes mellitus (T2DM) and concurrent hypertension, the fixed-dose combination of dapagliflozin and telmisartan is a sensible therapeutic approach. The high cardiovascular and renal risk burden linked to type 2 diabetes is addressed by this combination which permits efficient control of hyperglycaemia and hypertension while providing organ protection.

MOLECULAR STRUCTURE:



Dapagliflozin



Telmisartan

MOLECULAR FORMULAE:

Dapagliflozin: $C_{21}H_{25}ClO_6$

Telmisartan: $C_{33}H_{30}N_4O_2$

BRAND NAME:

- DAPA-T
- DAPTEL
- DAPTEL-T

CATEGORY:

- Dapagliflozin – Sodium Glucose Cotransporter 2 (SGLT2) inhibitor.
- Telmisartan – Angiotensin II Receptor Blockers (ARBs)

DOSAGE:

- Dosage of Dapagliflozin + Telmisartan (Combination Therapy)
- Dapagliflozin: 10 mg once daily (oral)
- Telmisartan: 40 mg or 80 mg once daily (oral), depending on blood pressure control and patient response

➢ Common fixed-dose combinations available:

- Dapagliflozin 10 mg + Telmisartan 40 mg

- Dapagliflozin 10 mg + Telmisartan 80 mg

MECHANISM OF ACTION:

Dapagliflozin inhibits the SGLT2 transporter in a selective and reversible manner. SGLT2 proteins are expressed in the kidney's proximal convoluted tubule (PCT), where they are in charge of reabsorbing sodium and glucose from the glomerular filtrate. Everyday, the body filters and reabsorbs about 180 gm of glucose primarily through SGLT2 and partially through SGLT1. Therefore, in healthy people, no glucose is eliminated through urine. SGLT2 is upregulated during hyperglycaemia such as in type 2 diabetes which promotes additional glucose reabsorption and contributes to hyperglycaemia. Eventually the capacity is exceeded resulting in the development of glycosuria. SGLT2 inhibitors prevent the reabsorption of glucose of about 80 gm daily causing glycosuria which directly lowers glucose levels without the need for insulin.[11]

Telmisartan is related to the renin angiotensin-aldosterone system (RAAS) which is crucial for controlling blood pressure and fluid balance in the body. The RAAS pathway starts when the kidneys release renin which is an enzyme released in reaction to low blood pressure, low blood sodium or activation of the sympathetic nervous system. Angiotensinogen is a protein which is produced by the liver gets changed into angiotensin-I by renin. Angiotensin-converting enzyme (ACE) then transforms angiotensin-I into angiotensin-II mostly in the lungs. Angiotensin II is a strong vasoconstrictor which causes blood vessels to narrow and raises blood pressure. Additionally, it causes the adrenal glands to release aldosterone which causes the kidneys to retain water and salt raising blood pressure even more. Telmisartan selectively blocks the angiotensin II type 1 (AT1) receptors to produce its antihypertensive effects. Telmisartan blocks the vasoconstrictive effects of angiotensin II by attaching itself to these receptors. This causes blood vessels to dilate and relax which lowers blood pressure.

PHARMACOKINETICS:

Dapagliflozin is a highly selective, competitive, reversible and oral SGLT2 inhibitor. Dapagliflozin was found to provide close-to-maximal SGLT2 inhibition in healthy subjects at doses of about 20-50mg for at least 24 hours indicating that once daily dosages are appropriate. In oral administration, dapagliflozin is quickly absorbed and typically reaches peak plasma concentration in 2 hours. [12]

Telmisartan's maximum plasma concentrations increased proportionally with dose and the median time to maximum plasma concentrations after oral dosing was between 0.5 and 2 hours. [13]

INDICATIONS:

- Dapagliflozin indicates for 3 major conditions-
  Type 2 diabetes mellitus
  Heart failure
  Chronic kidney disease.
- Telmisartan lowers the blood pressure, preventing cardiovascular events like myocardial infarction and stroke, reduction of cardiovascular risk in individuals who are intolerant to ACE inhibitors.

ADVERSE EFFECTS:

The adverse effects of dapagliflozin includes-

- Urinary tract infections
- Genital infections
- Frequent urination
- Electrolyte imbalance
- Hypoglycaemia [14]

The adverse effects of telmisartan includes-

- Dizziness
- Headache
- Back pain

- Sinus congestion with rare serious issues including hyperkalaemia, renal impairment, severe hypotension, angioedema (throat/face swelling) and a rare sprue-like enteropathy (severe diarrhoea).

CONTRAINDICATIONS:

- Dapagliflozin is contraindicated in anaphylactic reactions or angioedema, patient on dialysis [15]

Telmisartan is contraindicated in pregnant and breastfeeding women, history of hypersensitivity to specific drug, severe liver or biliary issues.

CASE DISCUSSION:

The laboratory parameters of all the patients are mentioned in Table – 1, 2, 3, 4 and 5.

CASE STUDY – 1:

Age : 55 years
Sex : Male
Diagnosis : Type 2 Diabetes mellitus with Diabetic Nephropathy
Treatment:
Dapagliflozin – 10mg OD
Telmisartan – 40mg OD
Follow up: 6 months

Table 1: Laboratory parameters of Case study-1

PARAMETERS	BEFORE TREATMENT	AFTER TREATMENT
Serum Creatinine	2.19 mg/dl	1.0 mg/dl
UACR	429 mg/g	56 mg/g
eGFR	30 mL/min/1.73 m²	83 mL/min/1.73 m²
BUN	60 mg/dl	22 mg/dl
HbA1C	9.5 %	6.5 %
Blood Pressure	165/93 mmHg	125/81 mmHg

CASE STUDY – 2:

Age : 75 years
Sex : Male
Diagnosis : Type 2 Diabetes mellitus with Diabetic Nephropathy
Treatment:
Dapagliflozin – 10mg OD
Telmisartan – 40mg OD
Follow up: 6 months

Table 2: Laboratory parameters of Case study-2

PARAMETERS	BEFORE TREATMENT	AFTER TREATMENT
Serum Creatinine	1.87 mg/dl	1.19 mg/dl
UACR	286 mg/g	81 mg/g
eGFR	47 mL/min/1.73 m²	92 mL/min/1.73 m²
BUN	39 mg/dl	24 mg/dl
HbA1C	8.1 %	6.9 %
Blood Pressure	164/96 mmHg	128/72 mmHg

CASE STUDY – 3:

Age : 46 years
Sex : Female
Diagnosis : Type 2 Diabetes mellitus with Diabetic Nephropathy
Treatment:
Dapagliflozin – 10mg OD
Telmisartan – 40mg OD
Follow up: 6 months

Table 3: Laboratory parameters of Case study-3

PARAMETERS	BEFORE TREATMENT	AFTER TREATMENT
Serum Creatinine	2.06 mg/dl	1.01 mg/dl
UACR	303 mg/g	58 mg/g
eGFR	54 mL/min/1.73 m²	79 mL/min/1.73 m²
BUN	67 mg/dl	24 mg/dl
HbA1C	8.2 %	7.0 %
Blood Pressure	168/104 mmHg	125/81 mmHg

CASE STUDY – 4:

Age : 55 years
Sex : Female
Diagnosis : Type 2 Diabetes mellitus with Diabetic Nephropathy
Treatment:
Dapagliflozin – 10mg OD
Telmisartan – 40mg OD
Follow up: 6 months

Table 4: Laboratory parameters of Case study-4

PARAMETERS	BEFORE TREATMENT	AFTER TREATMENT
Serum Creatinine	1.65 mg/dl	1.0 mg/dl
UACR	378 mg/g	39 mg/g
eGFR	29 mL/min/1.73 m²	74 mL/min/1.73 m²
BUN	44 mg/dl	16 mg/dl
HbA1C	9.2 %	7.0 %
Blood Pressure	147/95 mmHg	120/72 mmHg

CASE STUDY – 5:

Age : 55 years
Sex : Male
Diagnosis : Type 2 Diabetes mellitus with Diabetic Nephropathy
Treatment:
Dapagliflozin – 10mg OD
Telmisartan – 40mg OD
Follow up: 6 months

Table 5: Laboratory parameters of Case study-5

PARAMETERS	BEFORE TREATMENT	AFTER TREATMENT
Serum Creatinine	2.13 mg/dl	1.09 mg/dl
UACR	340 mg/g	41 mg/g
eGFR	36 mL/min/1.73 m²	72 mL/min/1.73 m²
BUN	47 mg/dl	20 mg/dl
HbA1C	8.2 %	6.4 %
Blood Pressure	148/103 mmHg	125/72 mmHg

CONCLUSION

The five representative cases shows consistent improvement in renal function, albuminuria, glycaemic control, and blood pressure following 6 months of treatment with dapagliflozin plus telmisartan. Across the cases, the reduction in serum creatinine, UACR, BUN, and HbA1c, and increase in the eGFR, were observed. Although the individual responses vary, the overall conclusion shows that the combination therapy has more Renal and Cardiovascular outcomes in patients with Diabetic Nephropathy.

Case 1

After 6 months of treatment with dapagliflozin 10 mg once daily and telmisartan 40 mg once daily, the patient demonstrated marked improvement in renal function, as evidenced by a reduction in serum creatinine (2.19 to 1.00 mg/dL), increase in eGFR (30 to 83 mL/min/1.73 m²), and decrease in UACR (429 to 56 mg/g). Glycaemic control improved with HbA1c decreasing from 9.5% to 6.5%, while blood pressure decreased from 165/93 mmHg to 125/81 mmHg. Overall, combination therapy was associated with significant improvement in renal and metabolic parameters and effective blood pressure control during the 6-month follow-up.

Case 2

Following 6 months of dapagliflozin plus telmisartan therapy, the patient exhibited clinically meaningful improvements in renal function and albuminuria. Serum creatinine decreased from 1.87 to 1.19 mg/dL, eGFR increased from 47 to 92 mL/min/1.73 m², and UACR declined from 286 to 81 mg/g. HbA1c improved from 8.1% to 6.9%, and blood pressure was reduced from 164/96 mmHg to 128/72 mmHg. These findings suggest that combination therapy was associated with improved kidney function, better glycaemic control, and effective blood pressure management.

Case 3

The patient with earlier-stage diabetic nephropathy showed favourable clinical outcomes after 6 months of treatment. Improvements were observed in serum creatinine (2.06 to 1.01 mg/dL), eGFR (54 to 79 mL/min/1.73 m²), UACR (303 to 58 mg/g), and BUN (67 to 24 mg/dL). HbA1c decreased from 8.2% to 7.0%, and blood pressure improved from 168/104 mmHg to 126/74 mmHg. These findings suggest that early initiation of dapagliflozin plus telmisartan may be associated with improved renal function and metabolic control in patients with diabetic nephropathy.

Case 4

The patient had reduced renal function at baseline (eGFR 29 mL/min/1.73 m²). After 6 months of therapy, serum creatinine decreased from 1.65 to 1.00 mg/dL, eGFR increased to 74 mL/min/1.73 m², and UACR declined from 378 to 39 mg/g. Improvements were also observed in BUN, HbA1c, and blood pressure. Within the limits of this individual case, treatment with dapagliflozin plus telmisartan was associated with marked improvement in renal and metabolic parameters over the follow-up period.

Case 5

The 73-year-old patient experienced clinically relevant improvements following 6 months of combination therapy. Serum creatinine decreased from 2.13 to 1.09 mg/dL, eGFR improved from 36 to 72 mL/min/1.73 m², and UACR was reduced from 340 to 41 mg/g. HbA1c improved from 8.2% to 6.4%, and blood pressure decreased from 148/103 mmHg to 125/72 mmHg. These observations suggest that dapagliflozin plus telmisartan was associated with improved renal function, glycaemic control, and blood pressure management in this older patient during the 6-month follow-up.

ACKNOWLEDGMENTS

The authors would like to acknowledge the facilities provided by the Bharat Institutions-pharmacy in executing this article.

REFERENCES

1. Ayodele OE, Alebiosu CO, Salako BL. Diabetic nephropathy--a review of the natural history, burden, risk factors and treatment. Journal of the National Medical association. 2004 Nov;96(11):1445.
2. Weir MR, Agarwal R, Rossing P, Usman MS, Khan MS, Butler J, et al. *Chronic Kidney Disease and Type 2 Diabetes*. Arlington, VA: American Diabetes Association; 2021. Table 1, Stages of Diabetic Nephropathy. doi:10.2337/db20211-13.
3. Hoogeveen EK. The epidemiology of diabetic kidney disease. Kidney and Dialysis. 2022 Aug 1;2(3):433-42.
4. Varghese RT, Jialal I. Diabetic Nephropathy.[Updated 2020 Mar 9]. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing. 2020.
5. Kulkarni A, Thool AR, Daigavane S. Understanding the clinical relationship between diabetic retinopathy, nephropathy, and neuropathy: a comprehensive review. Cureus. 2024 Mar 21;16(3).
6. National Kidney Foundation. Diabetes – a major risk factor for kidney disease [Internet]. New York: National Kidney Foundation; [cited 2026 Jan 21]. Available from: https://www.kidney.org/kidney-topics/diabetes-major-risk-factor-kidney-disease
7. Diabetic kidney disease. National Institute of Diabetes and Digestive and Kidney Diseases. https://www.niddk.nih.gov/health-information/diabetes/overview/preventing-problems/diabetic-kidney-disease. Accessed May 24, 2023.
8. Rasooly RS, Akolkar B, Spain LM, Guill MH, Del Vecchio CT, Carroll LE. The National Institute of Diabetes and Digestive and Kidney Diseases Central Repositories: a valuable resource for nephrology research. Clinical Journal of the American Society of Nephrology. 2015 Apr 1;10(4):710-5.
9. American Diabetes Association. Chronic kidney disease and risk management: Standards of care in diabetes—2024 [Internet]. Diabetes Care. 2024;49(Suppl 1):S246–S259 [cited 2026 Jan 21]. Available from: https://diabetesjournals.org/care/article/49/Supplement_1/S246/163914/11-Chronic-Kidney-Disease-and-Risk-Management (diabetesjournals.org in Bing).
10. Fan R, Kong J, Zhang J, Zhu L. Exercise as a therapeutic approach to alleviate diabetic kidney disease: mechanisms, clinical evidence and potential exercise prescriptions. Frontiers in Medicine. 2024 Oct 25;11:1471642.
11. Nicholson, M. K., Ghazal Asswad, R., & Wilding, J. P. (2021). Dapagliflozin for the treatment of type 2 diabetes mellitus – an update. *Expert Opinion on Pharmacotherapy*, *22*(17), 2303–2310. https://doi.org/10.1080/14656566.2021.1953471
12. Balakumar P, Sundram K, Dhanaraj SA. Dapagliflozin: glucuretic action and beyond. Pharmacological Research. 2014 Apr 1; 82:34-9.
13. Stangier J, Su CA, Roth W. Pharmacokinetics of orally and intravenously administered telmisartan in healthy young and elderly volunteers and in hypertensive patients.

Journal of international medical research. 2000 Aug;28(4):149-67.

14. Anitha AP, Balasubramanian S, Ramalingam AG, Samuel Kennady SR, Ganamurali N, Dhanasekaran D, Sabarathinam S. An exploration of the experience of dapagliflozin in clinical practice. Future Science OA. 2022 Sep 1;8(8): FSO816.
15. Palandurkar G, Kumar S, Palandurkar IV GS. Current status of dapagliflozin in congestive heart failure. Cureus. 2022 Sep 21;14(9).

HOW TO CITE: Ambala Syama, Asma Bano, Banda Vaishnavi, Nallavelli Lalith Adithya, Shanigaram Deekshith Reddy, Evaluation Of Renal and Cardiovascular Outcomes with Dapagliflozin Plus Telmisartan in Patients with Diabetic Nephropathy, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 8, 2040-2050, https://doi.org/10.5281/zenodo.21901590