

Advances in Therapeutic Agents for Diabetic Nephropathy

Sha Meng ^{1,†}, Ximei Cai ^{1,2,†} and Fang Wang ^{1,3,*}

¹ Department of Operations Management, Comprehensive Management Office of Wenjiang Campus, West China School of Medicine and West China Hospital, Sichuan University, Chengdu 610041, China

² Health Management Center of West China Tianfu Hospital, Sichuan University, Chengdu 610041, China

³ Innovation Center of Nursing Research, Nursing Key Laboratory of Sichuan Province, West China Hospital, Sichuan University, Chengdu 610041, China

* Corresponding author: Fang Wang

† These authors have made equal contributions.

Received: December 24, 2025; Revised: December 31, 2025; Accepted: January 7, 2026; Published: January 13, 2026

Abstract: Diabetic nephropathy, the prevalent microvascular complication of diabetes, presents a significant challenge to global healthcare systems. The pathogenesis is primarily driven by chronic hyperglycemia-induced renal damage, manifested as increased glomerular filtration rate, inflammation, and oxidative stress. Current clinical management aims to delay disease progression, reduce complications, and improve patients' quality of life, utilizing agents such as renin-angiotensin-aldosterone system inhibitors, sodium-glucose cotransporter 2 inhibitors, and glucagon-like peptide-1 receptor agonists. Emerging therapies, including stem cell and gene therapies, are under active investigation to provide more effective treatment avenues. Furthermore, comprehensive strategies incorporating lifestyle medicine and nutritional supplementation show promise in diabetic nephropathy management and warrant further exploration. This review summarizes current clinical strategies and pharmacological agents for diabetic nephropathy and explores potential novel therapeutic strategies, aiming to provide a scientific reference for future research and clinical practice.

Keywords: Diabetic nephropathy; clinical strategy; pharmacological agent

1. Introduction

Diabetes Mellitus (DM) is one of the leading chronic non-communicable diseases globally, affecting approximately 8–10% of the population worldwide. Among them, China accounts for over a quarter of the global diabetic population, making it the country with the largest number of diabetes patients. The incidence and mortality rates have remained persistently high [1]. Diabetic nephropathy is one of the most common microvascular complications in diabetic patients, with approximately 40% of individuals with diabetes ultimately developing DN. DN is a leading cause of end-stage renal disease and cardiovascular complications, contributing significantly to the mortality of diabetic patients. Currently, the rapid global increase in the diabetic population has made DN management a significant burden and major challenge for healthcare systems [2,3].

Persistent hyperglycemia causes long-term kidney damage, which is the main cause of DN. Increased glomerular filtration rate, thickening of the glomerular basement membrane, oxidative stress and inflammation, extracellular matrix buildup in the glomeruli and renal tubulointerstitial space, and raised intraglomerular pressure are all signs of this [4]. The Mogensen staging system usually divides DN into five stages. Patients typically don't start to feel physically uncomfortable obviously until the disease reaches stage IV. By stage V, the end-stage renal disease phase, the kidneys can no longer function properly. Dialysis or kidney transplantation are currently the only clinical treatment options available [5].

DN is a chronic, progressive illness that is currently incurable. Delaying disease progression, minimizing complications, and enhancing patients' quality of life are the main tenets of clinical management. Commonly used

therapeutic agents include renin-angiotensin-aldosterone system (RAAS) blockers, sodium-glucose cotransporter 2 (SGLT2) inhibitors, glucagon-like peptide-1 (GLP-1) agonists, and dipeptidyl peptidase-4 (DPP-4) inhibitors [6]. Despite extensive clinical drug management and new drug development research for DN, current efforts remain inadequate and fall short of therapeutic expectations. In an effort to serve as a scientific resource for relevant medication research and clinical practice, this review examines potential treatment approaches and summarizes both conventional and cutting-edge therapeutic agents for DN.

2. Clinical Staging and Diagnosis of DN

2.1. *Mogensen Staging and Diagnosis Based on Urinary Albumin Excretion*

A series of clinical indicators and markers of renal injury serve as the foundation for the clinical staging and diagnosis of DN. The Mogensen classification system is the main tool used in clinical staging. Stage I is the hyperfiltration phase, characterized by elevated glomerular filtration rate without significant clinical symptoms; Stage II is the silent phase, defined by aberrant glomerular structure but no obvious proteinuria. Stage III is early nephropathy, where patients exhibit microalbuminuria (urinary albumin excretion rate 30–300 mg/day); Stage IV is clinical nephropathy, characterized by overt proteinuria (urinary albumin excretion rate >300 mg/day); Stage V is end-stage renal disease, where patients exhibit severe renal impairment and usually necessitates dialysis or kidney transplantation for clinical management [5]. Clinical diagnosis of DN requires assessment of patient history and risk factors, including diabetes duration, glycemic control, hypertension, and family history. To assess the severity of proteinuria, it also requires testing for urinary protein using urine analysis, the urine albumin-to-creatinine ratio (UACR), or 24-h urine protein quantification. Renal function needs to be assessed concurrently, mainly using serum creatinine levels and estimated glomerular filtration rate (eGFR). Renal morphology and structure can also be evaluated using imaging modalities like CT, MRI, and ultrasound. Corresponding tests are also required because diabetic retinopathy and elevated cardiovascular risk are frequently linked to the advancement of renal disease. To confirm DN and rule out other kidney diseases, a renal biopsy may be required in specific situations [7]. In general, a thorough evaluation process that incorporates the patient's clinical presentation, laboratory test results, and imaging findings is required for the diagnosis and staging of DN.

2.2. *Investigation of Novel Diagnostic Biomarkers*

Beyond traditional methods, early diagnosis has attracted a lot of interest from researchers due to its potential to improve prognosis and slow the progression of DN. Research suggests that molecules like neutrophil gelatinase-associated lipocalin (NGAL), liver fatty acid-binding protein (L-FABP), and kidney injury molecule-1 (KIM-1) may be useful biomarkers [8,9]. Furthermore, related biomarkers like C-reactive protein (CRP), tumor necrosis factor- α (TNF- α), and oxidative stress markers are currently being studied due to the important role that inflammation and oxidative stress play in the pathogenesis of DN [10]. Additionally, DN susceptibility and progression are linked to particular genetic polymorphisms and epigenetic changes. In order to investigate new biomarkers, technologies such as proteomics, metabolomics, and genomics have been used more frequently in recent years. New approaches and techniques for the early diagnosis of DN have been made possible by discoveries and developments in biomarker research. Future research will concentrate on examining the combined use of these biomarkers and confirming their clinical utility.

3. Treatment Strategies and Therapeutic Agents for DN

DN is traditionally treated with blood pressure and glucose control, blood lipid regulation, sodium restriction, anti-inflammatory and antioxidant therapy, anticoagulant therapy, anti-fibrotic therapy, and renal protective therapy. Clinically, various drugs call for different safety measures and monitoring techniques.

3.1. *Agents for Glycemic, Blood Pressure, and Lipid Control*

It is critical for DN patients to control their blood glucose, blood pressure, and cholesterol levels. These measures not only slow the progression of DN, but they also postpone or avoid the need for dialysis and kidney transplantation, reduce complications, and improve patient outcomes. Commonly used hypoglycemic agents include sulfonylureas (e.g., glibenclamide), non-sulfonylurea insulin secretagogues (e.g., nateglinide), alpha-glucosidase inhibitors (e.g., acarbose), thiazolidinediones (e.g., rosiglitazone), and biguanides (e.g., metformin) [11]. Insulin therapy is required to manage blood glucose levels in patients with type 1 diabetes and types 2 diabetes whose blood glucose cannot be adequately controlled. Antihypertensive drugs commonly used in DN include angiotensin-converting enzyme inhibitors (ACEIs, e.g., benazepril) and angiotensin receptor blockers (ARBs, e.g., losartan). Both

classes of drugs can lower blood pressure and reduce proteinuria, thereby providing renal protection [12]. Additionally, lipid-lowering medications such as atorvastatin can reduce cholesterol levels, lowering the risk of cardiovascular disease [13]. Ultimately, clinical drug selection should be individualized, taking into account the patient's renal function, personal preferences, potential side effects, and drug-drug interactions.

3.2. Sodium-Glucose Cotransporter-2 (SGLT2) Inhibitors

SGLT2 inhibitors are a new family of antidiabetic agents that lower blood glucose levels and lessen renal load by blocking the SGLT2 transporter in the kidneys' proximal tubules [14]. Studies indicate that SGLT2 inhibitors also reduce urinary protein excretion in DN patients, delay renal function decline, and exert cardiovascular protective effects by lowering the incidence of cardiovascular events [15]. Currently, commonly used SGLT2 inhibitors in clinical practice include dapagliflozin, empagliflozin, canagliflozin, and etagliflozin. Renal function, blood pressure, diabetic ketoacidosis, urinary tract and genital infections, electrolyte imbalances, and drug interactions must all be regularly monitored during clinical administration. Medication adjustments should be made based on the specific condition and complications of DN patients.

3.3. GLP-1 Receptor Agonists and DPP-4 Inhibitors

GLP-1 receptor agonists and DPP-4 inhibitors are more recent kinds of antihyperglycemic medications that assist regulate blood glucose by promoting insulin production and preventing glucagon release. They offer extra-glycemic advantages such as increased renal blood flow, decreased renal inflammation, and cardiovascular protection in addition to enhancing islet cell activity [16]. Commonly used GLP-1 receptor agonists in clinical practice include liraglutide, dulaglutide, and exenatide; while frequently employed DPP-4 inhibitors encompass sitagliptin and saxagliptin. Notably, GLP-1 receptor agonists are in significant demand among both diabetic patients and obese populations due to their sustained efficacy and weight-reducing benefits. Consequently, they have become a key focus of current research and development efforts for numerous pharmaceutical companies [17].

3.4. Therapeutic Agents for Alleviating Oxidative Stress and Inflammation

Cellular damage, inflammatory responses, and vascular dysfunction are key pathological features in DN, driven by oxidative stress and inflammation. In clinical practice, N-acetylcysteine is a commonly used antioxidant. It mitigates the progression of DN by reducing the overall oxidative stress burden in patients [18]. Patients with DN may also use nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen, naproxen, and celecoxib to alleviate inflammation and pain symptoms. However, NSAIDs may affect renal blood flow, exacerbate renal impairment, and increase the risk of cardiovascular events. Therefore, blood pressure and renal function must be closely monitored during use to adjust medication dosages as needed [19]. To summarize, when DN patients require the use of antioxidants and anti-inflammatory drugs in clinical settings, these medications must be administered under the direction and supervision of physicians and pharmacists to avoid potential side effects and drug interactions. Individual circumstances and disease progression should be considered when making dosage adjustments.

3.5. Anticoagulant Agents

Patients with DN often exhibit a heightened cardiovascular risk and a hypercoagulable state, leading to an elevated thrombotic risk that may necessitate anticoagulant therapy. In specific clinical scenarios, these agents can improve renal hemodynamics and mitigate ischemia-reperfusion injury. Treatment options for such patients include vitamin K antagonists (e.g., warfarin), direct thrombin inhibitors (e.g., dabigatran), factor Xa inhibitors (e.g., rivaroxaban), and low molecular weight heparins (e.g., enoxaparin). The choice among them should be individualized based on the clinical context and the specific type of thromboembolism being prevented [20]. To avoid impairing drug clearance and increasing bleeding risk, appropriate anticoagulant medications for DN patients must be chosen while taking renal function into account. In addition, potential interactions between anticoagulants and other blood coagulation-affecting medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs) and antiplatelet agents, must be considered [21]. In conclusion, in order to guarantee both safety and effectiveness, DN patients must take anticoagulants under close supervision by doctors and clinical pharmacists, with ongoing drug monitoring during treatment.

3.6. Antifibrotic Therapeutic Agents

The progression of DN is closely linked to renal fibrosis. Antifibrotic agents act by mitigating the excessive accumulation of extracellular matrix during renal tissue injury and repair, thereby slowing the decline of renal

function [22]. Consequently, drugs such as pirfenidone are increasingly employed to attenuate renal fibrosis, mitigate kidney injury, and improve functional outcomes. Beyond their primary antifibrotic effect, these agents often possess concomitant anti-inflammatory and antioxidant properties, which help alleviate the underlying inflammation and oxidative stress in DN. This multifaceted action contributes to reduced fibrosis and delays the progression to end-stage renal disease [23]. The clinical use of antifibrotic drugs requires regular monitoring of renal function parameters, including serum creatinine, blood urea nitrogen, and estimated glomerular filtration rate. Additionally, potential side effects such as hepatotoxicity and gastrointestinal adverse events, as well as drug interactions with concurrent hypoglycemic therapies, must be carefully managed to optimize treatment efficacy and patient safety.

3.7. Renoprotective Agents

Aldosterone receptor antagonists can preserve renal function, slow further deterioration, and effectively reduce renal fibrosis and proteinuria. In addition, these drugs have cardiovascular protective effects, which help to reduce the risk of cardiovascular events in DN patients [24]. Commonly used aldosterone receptor antagonists include eplerenone and spironolactone. Due to spironolactone's potential to cause hyperkalemia and other sex hormone-related side effects, eplerenone has become the more widely used renal protective agent [25]. When using these agents in clinical settings, it is critical to monitor serum potassium levels, sex hormone-related side effects (such as menstrual cycle changes), and renal function. Furthermore, interactions with other medications that may affect serum potassium levels should be carefully considered in order to make timely adjustments to the treatment regimen.

3.8. Traditional Chinese Medicine Therapy

Traditional Chinese Medicine (TCM) emphasizes holistic regulation and treatment based on pattern differentiation, demonstrating favorable effects in managing DN. Common therapeutic modalities include acupuncture, herbal prescriptions, emotional regulation, and dietary therapy. Acupuncture frequently targets specific acupoints such as Shenshu (BL23), Zusanli (ST36), and Sanyinjiao (SP6) to restore balance [26]. The management of DN with Traditional Chinese Medicine (TCM) is grounded in the principles of syndrome differentiation. Shengmai Decoction and Yuquan Pills are Chinese herbal formulas that can help DN patients with Qi-Yin deficiency. Xuefu Zhuyu Decoction and Taohong Siwu Decoction are effective treatments for DN with blood stasis obstructing the collaterals. Bazheng Powder and Sanren Decoction are suitable formulas for DN with internal damp heat accumulation. Zhenwu Decoction and Wuling Powder are effective formulations for DN with yang deficiency and water retention [27,28]. Consequently, TCM therapy for DN requires an individualized approach, demanding careful customization and continuous monitoring of the patient's condition to ensure treatment efficacy.

3.9. Novel Therapeutic Strategies for DN

Novel therapeutic approaches and agents are being actively studied to address unmet clinical needs in DN and overcome the limitations of conventional therapies. Among these, stem cell therapy can regenerate damaged renal cells to restore basic kidney function. Gene therapy offers curative potential by repairing or replacing genetic mutations causing DN. The bioartificial pancreas, which integrates an insulin pump with continuous glucose monitoring, enables precise glycemic control. Additionally, developing novel immunomodulatory therapies targeting the immune responses involved in DN progression may help mitigate inflammation and renal damage [29,30]. Exploring the biological mechanisms underlying DN development will help reduce the toxic side effects of existing therapies, prevent and intervene in potential complications, and provide more personalized treatment options to meet the unresolved clinical needs of DN patients.

4. Discussion and Conclusions

DN, as a common microvascular complication of diabetes, poses ongoing challenges in treatment and management within the medical field. Although current treatment approaches can somewhat slow the progression of DN, they are unable to stop the decline in renal function. Conventional therapies, like aldosterone receptor antagonists, RAAS blockers, and anti-oxidative stress drugs, have proven effective in regulating blood sugar levels and safeguarding renal function. Nevertheless, these treatments frequently have adverse effects and drug interactions, which limits their widespread application.

In recent years, novel drugs and therapeutic approaches have emerged, offering renewed hope for DN management. SGLT2 inhibitors and GLP-1 receptor agonists not only effectively control blood glucose but also

demonstrate renal and cardiovascular protective effects [31]. However, the long-term efficacy and safety of these drugs require further clinical validation. Moreover, although cutting-edge therapies like stem cell and gene therapy are promising for treating DN, they are not yet ready for widespread clinical use and remain primarily in the research domain [29,30]. Moreover, comprehensive disease management strategies, including lifestyle medicine and nutritional supplementation, are playing an increasingly vital role in managing DN. By improving patients' lifestyle habits and dietary patterns, these approaches help control blood glucose, blood pressure, and blood lipids, thereby reducing the risk of DN progression [32]. However, the effectiveness of these approaches is highly dependent on strong patient compliance and ongoing support from the healthcare team.

In conclusion, the management of DN remains a complex clinical challenge. Future efforts should not only focus on elucidating the underlying pathophysiology of DN but also on refining existing treatments and developing personalized therapeutic strategies. Concurrently, improving patient education and self-management, coupled with fostering multidisciplinary collaboration, is crucial for enhancing clinical outcomes and quality of life. Advances in medical science hold the promise of yielding more effective solutions to alleviate the global burden of DN.

Funding

This work was supported by the Science and Technology Project (Social Science Foundation) of Sichuan Province (No. SCJJ25RKX136).

Author Contributions

Writing—original draft, S.M., X.C. and F.W.; writing—review and editing, S.M., X.C. and F.W. All authors have read and agreed to the published version of the manuscript.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflicts of Interest

The authors declare no conflict of interest.

Reference

1. Deng W, Zhao L, Chen C, *et al.* National Burden and Risk Factors of Diabetes Mellitus in China from 1990 to 2021: Results from the Global Burden of Disease Study 2021. *Journal of Diabetes* 2024; **16**(10): e70012. <https://doi.org/10.1111/1753-0407.70012>.
2. Samsu N. Diabetic Nephropathy: Challenges in Pathogenesis, Diagnosis, and Treatment. *BioMed Research International* 2021; **2021**: 1497449. <https://doi.org/10.1155/2021/1497449>.
3. Fenta ET, Eshetu HB, Kebede N, *et al.* Prevalence and Predictors of Chronic Kidney Disease among Type 2 Diabetic Patients Worldwide, Systematic Review and Meta-Analysis. *Diabetology & Metabolic Syndrome* 2023; **15**(1): 245. <https://doi.org/10.1186/s13098-023-01202-x>.
4. Gajjala PR, Sanati M, Jankowski J. Cellular and Molecular Mechanisms of Chronic Kidney Disease with Diabetes Mellitus and Cardiovascular Diseases as Its Comorbidities. *Frontiers in immunology* 2015; **6**: 340. <https://doi.org/10.3389/fimmu.2015.00340>.
5. Mogensen CE, Christensen CK, Vittinghus E. The Stages in Diabetic Renal Disease: With Emphasis on the Stage of Incipient Diabetic Nephropathy. *Diabetes* 1983; **32**: 64–78. <https://doi.org/10.2337/diab.32.2.s64>.
6. Rao V, Rao LV, Tan SH, *et al.* Diabetic Nephropathy: An Update on Pathogenesis and Drug Development. *Diabetes & Metabolic Syndrome* 2019; **13**(1): 754–762. <https://doi.org/10.1016/j.dsx.2018.11.054>.
7. Thipsawat S. Early Detection of Diabetic Nephropathy in Patient with Type 2 Diabetes Mellitus: A Review of the Literature. *Diabetes and Vascular Disease Research* 2021; **18**(6). <https://doi.org/10.1177/14791641211058856>.
8. Satirapoj B. Tubulointerstitial Biomarkers for Diabetic Nephropathy. *Journal of Diabetes Research* 2018; **2018**: 2852398. <https://doi.org/10.1155/2018/2852398>.
9. Liu H, Feng J, Tang L. Early Renal Structural Changes and Potential Biomarkers in Diabetic Nephropathy. *Frontiers in Physiology* 2022; **13**: 1020443. <https://doi.org/10.3389/fphys.2022.1020443>.
10. Ye Z, Xian W, Ling GU, *et al.* Efficacy of Danggui Buxue Decoction on Diabetic Nephropathy-Induced Renal Fibrosis in Rats and Possible Mechanism. *Journal of Traditional Chinese Medicine* 2023; **43**(3): 507–513. <https://doi.org/10.19852/j.cnki.jtcm.20230214.004>.

11. Zhang R, Wang Q, Li Y, *et al.* A New Perspective on Proteinuria and Drug Therapy for Diabetic Kidney Disease. *Frontiers in Pharmacology* 2024; **15**: 1349022. <https://doi.org/10.3389/fphar.2024.1349022>.
12. Zain M, Awan FR. Renin Angiotensin Aldosterone System (RAAS): Its Biology and Drug Targets for Treating Diabetic Nephropathy. *Pakistan Journal of Pharmaceutical Sciences* 2014; **27(5)**: 1379–1391.
13. Kilpatrick ES, Kallner A, Atkin SL, *et al.* The Variability of Measured and Calculated Low-Density Lipoprotein (LDL) Cholesterol in Statin-Treated Diabetes Patients. *Annals of Clinical Biochemistry* 2024; **62(3)**: 184–190. <https://doi.org/10.1177/00045632241305936>.
14. Gao FM, Ali AS, Bellomo R, *et al.* A Systematic Review and Meta-Analysis on the Safety and Efficacy of Sodium-Glucose Cotransporter 2 Inhibitor Use in Hospitalized Patients. *Diabetes Care* 2024; **47(12)**: 2275–2290. <https://doi.org/10.2337/dc24-0946>.
15. Sheu JY, Chang LY, Chen JY, *et al.* The Outcomes of SGLT-2 Inhibitor Utilization in Diabetic Kidney Transplant Recipients. *Nature Communications* 2024; **15(1)**: 10043. <https://doi.org/10.1038/s41467-024-54171-8>.
16. Sidra FNU, Agarwal S, Lockhart Pastor P, *et al.* Glucagon-Like Peptide-1 Receptor Agonists Versus Dipeptidyl-Peptidase 4 Inhibitors in Advanced Chronic Kidney Disease and End Stage Kidney Disease: Real World Effectiveness and Persistence of Therapy. *Journal of Diabetes and its Complications* 2024; **39(1)**: 108925. <https://doi.org/10.1016/j.jdiacomp.2024.108925>.
17. Ning X, Munir KM, Davis SN. Drugs Stimulating Insulin Secretion in Early Clinical Development for the Treatment of Type 1 Diabetes: What's New? *Expert Opinion on Investigational Drugs* 2024; **33(12)**: 1199–1208. <https://doi.org/10.1080/13543784.2024.2439501>.
18. Weng SW, Kuo HM, Chuang JH, *et al.* Study of Insulin Resistance in Cybrid Cells Harboring Diabetes-Susceptible and Diabetes-Protective Mitochondrial Haplogroups. *Mitochondrion* 2013; **13(6)**: 888–897. <https://doi.org/10.1016/j.mito.2013.08.001>.
19. Solomon DH, Paynter NP, Guan H, *et al.* External Validation of a Risk Score for Major Toxicity Among Nonsteroidal Anti-Inflammatory Drug Users: Real-World Application. *ACR Open Rheumatology* 2020; **2(5)**: 269–275. <https://doi.org/10.1002/acr2.11134>.
20. Zahoor MM, Mazhar S, Azhar A, *et al.* Factor Xa Inhibitors Versus Warfarin in Patients with Non-Valvular Atrial Fibrillation and Diabetes Mellitus: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Annals of Medicine and Surgery* 2024; **86(2)**: 986–993. <https://doi.org/10.1097/MS9.0000000000001621>.
21. Chen YF, Li SJ, Chen QT. Analysis of Coagulation Function Changes and Influencing Factors of Hypercoagulable State in Type 2 Diabetic Nephropathy. *Journal of Medicine & Frontier* 2024; **14(02)**: 122–124.
22. Karihaloo A. Anti-Fibrosis Therapy and Diabetic Nephropathy. *Current Diabetes Reports* 2012; **12(4)**: 414–422. <https://doi.org/10.1007/s11892-012-0290-7>.
23. Avila G, Osornio-Garduño DS, Ríos-Pérez EB, *et al.* Functional and Structural Impact of Pirfenidone on the Alterations of Cardiac Disease and Diabetes Mellitus. *Cell Calcium* 2014; **56(5)**: 428–435. <https://doi.org/10.1016/j.ceca.2014.07.008>.
24. Zavatta G, Casadio E, Rinaldi E, *et al.* Aldosterone and Type 2 Diabetes Mellitus. *Hormone Molecular Biology and Clinical Investigation* 2016; **6(1)**: 53–59. <https://doi.org/10.1515/hmbci-2015-0065>.
25. Veneti S, Tziomalos K. The Role of Finerenone in the Management of Diabetic Nephropathy. *Diabetes Therapy* 2021; **12(7)**: 1791–1797. <https://doi.org/10.1007/s13300-021-01085-z>.
26. Li XC, Li XJ. Research Progress of Acupuncture and Moxibustion in Treating Diabetic Nephropathy. *Inner Mongolia Journal of Traditional Chinese Medicine* 2023; **42(11)**: 153–155. <https://doi.org/10.16040/j.cnki.cn15-1101.2023.11.004>.
27. Wang SH, Fang ZH, Li YF, *et al.* Research Progress on Internal Treatment of Diabetic Nephropathy with Traditional Chinese Medicine. *Journal of Henan University (Medical Science)* 2024; **43(05)**: 313–317+340. <https://doi.org/10.15991/j.cnki.41-1361/r.2024.05.011>.
28. Sun S, Zheng YL, Cheng H, *et al.* Research Progress on Traditional Chinese Medicine in Treating Diabetic Nephropathy. *Integrated Traditional Chinese and Western Medicine Chronic Disease Journal* 2025; **2(02)**: 89–96.
29. Hatipoglu BA, Blanchette J. Islet Cell Therapy and Stem Cell Therapy for Type 1 Diabetes: There Will Always Be a Hope. *Endocrinology and Metabolism Clinics of North America* 2023; **52(1)**: 187–193. <https://doi.org/10.1016/j.ecl.2022.07.001>.
30. Kioulaphides S, García AJ. Encapsulation and Immune Protection for Type 1 Diabetes Cell Therapy. *Advanced Drug Delivery Reviews* 2024; **207**: 115205. <https://doi.org/10.1016/j.addr.2024.115205>.
31. Zhu JJ, Wilding JPH, Gu XS. Combining GLP-1 Receptor Agonists and SGLT-2 Inhibitors for Cardiovascular Disease Prevention in Type 2 Diabetes: A Systematic Review with Multiple Network Meta-Regressions. *World Journal of Diabetes* 2024; **15(10)**: 2135–2146. <https://doi.org/10.4239/wjd.v15.i10.2135>.
32. Nieto-Martinez R, Neira C, de Oliveira D, *et al.* Lifestyle Medicine in Diabetes Care: The Lifedoc Health Model. *American Journal of Lifestyle Medicine* 2022; **17(3)**: 336–354. <https://doi.org/10.1177/15598276221103470>.