DOI: 10.53469/jcmp.2026.08(04).14

Research Progress in Integrated Traditional Chinese and Western Medicine for Diabetic Kidney Disease at Stage III–IV

Qianqian Wei[1], Chunying Liu[2,*]

[1]Shaanxi University of Chinese Medicine, Xianyang 712046, Shaanxi, China
[2]Affiliated Hospital of Shaanxi University of Chinese Medicine, Xianyang 712000, Shaanxi, China
**Correspondence Author*

Abstract: ***Diabetic kidney disease (DKD), one of the major microvascular complications of diabetes, can progress to end-stage renal disease and severely threatens patients' quality of life and lifespan. The pathogenesis of DKD is complex and is currently believed to be related to glucose and lipid metabolism disorders, oxidative stress, abnormal renal hemodynamics, inflammatory responses, and other factors. Western medical treatments for stage III–IV DKD mainly include lifestyle modification, hypoglycemic therapy, antihypertensive therapy, lipid-lowering therapy, and renal triple therapy, which are relatively limited and pose great challenges for prevention and treatment. Traditional Chinese medicine (TCM) offers unique insights into the intervention of DKD in terms of etiology, pathogenesis, syndrome differentiation, and TCM treatment.***

Keywords: Stage III–IV diabetic kidney disease, Glucose and lipid metabolism, Integrated traditional Chinese and western medicine, Research progress**.**

1. Introduction

Diabetic kidney disease (DKD) is characterized primarily by glomerular vascular damage, and clinical manifestations commonly include proteinuria and decreased glomerular filtration rate [1]. Due to the insidious onset of early-stage DKD with no obvious symptoms or signs, most patients are diagnosed clinically when the disease has progressed to stages III–IV. Western medical management of DKD mainly consists of lifestyle modification, hypoglycemic, antihypertensive, and lipid-lowering interventions; in the advanced stage with progressive renal function deterioration, blood purification and kidney transplantation may be performed [2], but these approaches are relatively limited. Numerous clinical studies have confirmed that TCM has definite efficacy and advantages in the syndrome differentiation and treatment of DKD [3]. With the advantages of multi-component, multi-pathway action and low toxicity, TCM can reduce lipotoxicity, regulate lipid metabolism disorders, and improve renal function in DKD to a certain extent [4]. Studies have shown that TCM has significant advantages in alleviating clinical symptoms, reducing urinary protein and serum creatinine levels, improving patients' quality of life, and delaying the progression of DKD [5]. This paper reviews the research progress in integrated traditional Chinese and western medicine for stage III–IV DKD based on relevant literature.

2. Western Medical Understanding of Diabetic Kidney Disease

2.1 Epidemiology

Statistics show that the prevalence of DKD among patients with type 2 diabetes in China has reached as high as 40% [6], and DKD is the leading cause of end-stage renal failure worldwide [7]. In recent years, with the significant increase in the number of diabetic patients, China has become one of the countries with the largest diabetic population globally [8].

2.2 Pathogenesis

The main pathological features of DKD include glomerulosclerosis, renal tubular injury, and interstitial fibrosis, which are typical lesions of DKD [9]. However, its pathogenesis is complex and is currently believed to involve glucose and lipid metabolism disorders, oxidative stress, inflammatory responses, abnormal renal hemodynamics, genetic factors, etc. Among them, glucose and lipid metabolism disorders play a crucial role.

Glucose and lipid metabolism are key in the development and progression of DKD. In the pathogenesis of DKD, persistent hyperglycemia is not only a marker of metabolic imbalance but also damages renal cells by generating various toxic intermediate metabolites [10]. Abnormal glucose metabolism also exacerbates the dysmetabolism of advanced glycation end products (AGEs) in the glomerular basement membrane and renal tubulointerstitium, leading to aggravated glomerulosclerosis and proteinuria. After binding to the receptor for advanced glycation end products (RAGE), AGEs activate signaling pathways that induce parenchymal cell injury and mesangial cell apoptosis. Furthermore, AGEs disrupt the negative feedback mechanism of the renin-angiotensin-aldosterone system (RAAS) by stimulating proximal tubular cells to secrete angiotensinogen, causing abnormal activation of the RAAS, resulting in high intraglomerular pressure and inducing more diabetes-related renal lesions. Long-term hyperglycemia can lead to renal microangiopathy and impair normal renal function. In addition, insulin resistance and impaired β-cell function exacerbate glucose metabolism disorders, thereby aggravating renal damage. Studies have reported that long-term hyperglycemia in DKD patients damages renal cell DNA, renal vascular endothelial cells, and podocytes, and promotes the production of cytokines such as transforming growth factor-β (TGF-β), interleukins (IL), and insulin-like

growth factors, leading to glomerulosclerosis [11].

Lipid accumulation and its metabolites induce oxidative stress, fibrosis, inflammation, and apoptosis, which aggravate glomerulosclerosis. Hyperlipidemia can cause arteriosclerosis and damage the kidneys, and has been listed as an independent risk factor for DKD [12]. Normal low-density lipoprotein undergoes oxidation to form oxidized low-density lipoprotein (ox-LDL), which leads to vascular sclerosis, endothelial damage, and inflammatory responses. As a highly metabolically active organ, the kidney requires continuous and sufficient energy supply to maintain normal function, most of which comes from lipid oxidation [13]. In DKD, lipid metabolism disorders are not only related to disrupted energy supply but also cause abnormal renal lipid accumulation (lipotoxicity), an independent pathogenic factor that directly drives cell injury and inflammation [14].

2.3 Western Medical Treatment

2.3.1 Lifestyle Intervention

Lifestyle intervention is necessary for DKD patients. Moderate exercise can effectively enhance cardiopulmonary function, inhibit renal oxidative stress, reduce aldosterone levels, and improve renal vascular atherosclerosis [15]. A meta-analysis showed a positive correlation between smoking and the occurrence of DKD; therefore, smoking cessation has an important impact on the prognosis of DKD [16]. Guidelines recommend a daily total energy intake of 25–30 kcal/kg and sodium intake below 2.3 g for DKD patients [17].

2.3.2 Hypoglycemic Therapy

The 2022 Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guideline for Diabetes in Chronic Kidney Disease recommends a target glycated hemoglobin (HbA1c) range of 6.5%–8.0% for non-dialysis DKD patients [18]. A first-line regimen of metformin and sodium-glucose cotransporter 2 inhibitors (SGLT2i) is recommended, with assessment of the patient's estimated glomerular filtration rate (eGFR) [19]. Insulin, α-glucosidase inhibitors (AGI), sulfonylureas (SU), dipeptidyl peptidase-4 inhibitors (DPP-4i), or thiazolidinediones (TZD) can be selected based on the patient's complications, creatinine and urea nitrogen levels, therapeutic goals, efficacy, and family economic status [20].

2.3.3 Antihypertensive Therapy

Most DKD patients have elevated blood pressure, even refractory hypertension, requiring combination antihypertensive drugs for control. All guidelines recommend angiotensin-converting enzyme inhibitors/angiotensin receptor blockers (ACEI/ARB) as first-line agents in the absence of contraindications, administered at the highest approved tolerable dose. Sacubitril/valsartan sodium tablets exert renal protective, antihypertensive, and urinary protein-lowering effects and reduce cardiac ejection fraction, and are widely used clinically. Serum potassium and creatinine levels should be monitored regularly. When the therapeutic effect is unsatisfactory, other antihypertensive drugs such as mineralocorticoid receptor antagonists (MRA), diuretics, calcium channel blockers (CCB), and β-blockers can be combined.

2.3.4 Lipid-Lowering Therapy

DKD is mostly accompanied by mixed dyslipidemia, mainly characterized by elevated triglycerides and low-density lipoprotein cholesterol (LDL-C). Statins (renal and cardiac protective) are the first choice, with atorvastatin and rosuvastatin commonly used. These drugs are relatively safe for renal function and can reduce cardiovascular and cerebrovascular events (the most dangerous cause of death in DKD patients). Liver function, creatine kinase, and renal function should be monitored regularly during use. When triglycerides are significantly elevated, fibrates (e.g., fenofibrate) can be combined short-term; however, statin-fibrate combination requires caution due to the risk of myopathy and renal injury. Generally, LDL-C is prioritized for target achievement before triglyceride management. High-dose, intensive lipid-lowering regimens are generally avoided, as well as unregulated "lipid-lowering health products," many of which are nephrotoxic.

2.3.5 Renal Triple Therapy

The core of pharmacotherapy for DKD is to delay renal function progression, reduce proteinuria, and lower the risk of adverse cardiovascular events. Three key classes of drugs are commonly used clinically: ACEI/ARB, mineralocorticoid receptor antagonists (MRA), and SGLT2i.

ACEIs (e.g., benazepril, enalapril) and ARBs (e.g., valsartan, irbesartan) are basic antihypertensive and renal protective drugs for DKD with similar mechanisms. They act by inhibiting the RAAS, mainly dilating the glomerular efferent arteriole more than the afferent arteriole, reducing intraglomerular hypertension, hyperperfusion, and hyperfiltration, thereby decreasing urinary protein excretion. They also inhibit renal inflammation and fibrosis, delay glomerulosclerosis and renal interstitial fibrosis, and exert antihypertensive effects to reduce cardiac afterload and lower the risk of cardiovascular events. They are contraindicated in patients with bilateral renal artery stenosis, renal artery stenosis in a solitary kidney, and pregnancy. Serum creatinine and potassium should be closely monitored during administration to guard against hyperkalemia.

Non-steroidal MRAs (e.g., finerenone) are the first choice for DKD treatment, while steroidal MRAs (e.g., spironolactone, eplerenone) are mostly used for heart failure and should be used with caution in DKD patients. Their mechanism involves specifically blocking mineralocorticoid receptors in renal tubules, heart, and blood vessels, reducing renal inflammation, oxidative stress, and fibrosis, decreasing urinary protein excretion, delaying the decline in eGFR, and lowering the risk of uremia and adverse cardiovascular events, with mild diuretic, natriuretic, and potassium-sparing effects. Although finerenone reduces proteinuria, improves renal function, assists in blood pressure control, and alleviates systemic inflammation in DKD, serum potassium should be monitored regularly to prevent hyperkalemia [21]. Finerenone, a non-steroidal MRA (ns-MRA), highly selectively and potently antagonizes mineralocorticoid receptors in renal and

cardiac tissues, directly inhibiting and delaying renal and cardiac inflammation and fibrosis caused by excessive mineralocorticoid receptor activation, exerting dual renal and cardiac protection [22], and has become an important drug for DKD treatment. Finerenone is routinely used in patients with eGFR ≥25 ml/min/1.73m²; when combined with ACEI/ARB, renal protective effects are synergistically enhanced, and concurrent use with potent potassium-sparing diuretics or potassium supplements is avoided.

Representative SGLT2i include dapagliflozin and empagliflozin. Their main mechanism is inhibiting sodium-glucose cotransporter 2 in the proximal renal tubule, reducing renal glucose reabsorption, promoting glucose excretion in urine, and lowering intraglomerular pressure through osmotic diuresis and natriuresis. In addition, they reduce body weight, improve insulin resistance, and exert glucose-independent renal protective effects, reducing proteinuria, delaying eGFR decline, and lowering the risk of uremia, heart failure, and cardiovascular death. SGLT2i are preferentially used in DKD patients with moderate to high progression risk, including those with obesity or overweight, hypertension, dyslipidemia, smoking, family history of premature atherosclerotic cardiovascular disease (ASCVD), and heart failure (regardless of ejection fraction) [23]. They may easily cause genitourinary tract infections (e.g., vulvovaginal candidiasis, urinary tract infection); patients should be instructed to drink plenty of water and maintain personal hygiene during treatment.

The triple renal protective combination follows the principle of "ACEI/ARB + SGLT2i" as the first-line core regimen; if persistent proteinuria or high renal progression risk remains, finerenone can be added to form the "triple renal protective" regimen. During combination therapy, key monitoring includes serum potassium, creatinine, eGFR, blood pressure, and volume status to avoid excessive hypotension and diuresis leading to insufficient renal perfusion, ensuring safe and effective medication.

3. Traditional Chinese Medicine Understanding of Diabetic Kidney Disease

3.1 TCM Disease Names

In traditional Chinese medicine, diabetes is defined as "Xiaoke" (wasting-thirst syndrome). There is no explicit disease name for DKD in TCM. Currently, based on the symptoms of DKD, TCM classifies it as "Shuizhong" (edema), "Xulao" (consumptive disease), "Guange" (block and repulsion syndrome), "Shenxiao" (renal wasting), etc.

3.2 TCM Etiology and Pathogenesis

Modern TCM scholars have different summaries of its etiology and pathogenesis, mostly agreeing on "deficiency in origin and excess in superficiality, mixed deficiency and excess." The root deficiency involves impairment of the lung, stomach, spleen, liver, kidney, and other zang-fu organs, especially the liver, spleen, and kidney, leading to deficiency of qi, blood, yin, and yang over time. The superficial excess refers to excess pathogens such as qi stagnation, blood stasis, phlegm turbidity, water dampness, and turbid toxin, among which dampness and stasis persist throughout the disease course. Studies have shown that as DKD progresses to stages III–IV, spleen-kidney qi deficiency combined with blood stasis is a common syndrome type, which then progresses to yin-yang deficiency combined with phlegm turbidity and blood stasis [24].

3.2.1 Spleen-Kidney Deficiency

The spleen is the acquired base, governing transportation and transformation, and is the source of qi and blood production. The kidney is the congenital base, governing essence storage, qi reception, and water regulation. The spleen and kidney are interconnected, mutually promoting and influencing each other. In literature on treating Xiaoke, besides treating from the kidney, there are also approaches from the spleen. For example, the Song Dynasty General Record of the Holy Benevolence stated when discussing the pathogenesis of Xiaoke with abdominal distension: "The spleen governs water regulation, dredges and regulates water passages, and transports water downward to the bladder. Excessive drinking in Xiaoke impairs the spleen earth, which fails to govern water." Dou Cai's Heart Book of Bian Que also pointed out: "Although Xiaoke has upper, middle, and lower stages, it is always due to consumption of body fluids. The kidney is the source of body fluids, and the spleen is the foundation of body fluids; deficiency of the source and foundation leads to Xiaoke." Yang Shiying's Direct Guidance from the Benevolent Studio considered the pathogenesis of middle Xiaoke as: "Heat accumulates in the middle, and the deficient spleen is affected." Therefore, spleen-kidney deficiency is the pathological basis of DKD onset. Xiang Wenzheng et al. [25] believed that the kidney stores primordial yin and yang and is the congenital base of the human body. Imbalance of kidney yin and yang is the fundamental cause of Xiaoke. Sufficient kidney yin nourishes lung and stomach yin; kidney yin deficiency leads to flaming deficiency fire, depletion of lung metal, impaired water metabolism, and frequent urination. Deficiency fire from kidney yin deficiency scorches the stomach, causing stomach heat and polyphagia. Lung dryness, stomach heat, and kidney deficiency coexist and interact, with kidney deficiency as the main factor. The pathogenesis evolution of Xiaoke is also closely related to the kidney; treatment requires addressing the root, and regulating kidney yin and yang to balance yin and yang is the fundamental principle for Xiaoke.

3.2.2 Damp Turbidity and Blood Stasis

Consumption of body fluids and exuberant internal dryness-heat are the basic pathogenesis of Xiaoke. With prolonged disease course, yang qi is impaired; qi deficiency leads to dysfunction of zang-fu organs and disordered qi movement. Qi deficiency impairs blood circulation, causing blood stasis and collateral obstruction. Blood stasis leads to pain due to obstruction and malnutrition of extremities, resulting in various complications. The theory of blood stasis causing Xiaoke can be traced to Spiritual Pivot · Five Changes, which states: "Seven emotions cause disease; anger leads to qi stagnation, which causes blood stasis, and stasis transforms into heat, leading to Xiaoke." In his work Treatise on Blood Syndromes, the late Qing physician Tang Rongchuan recorded the pathogenesis of blood stasis causing thirst:

"Blood stasis internally causes thirst. The reason is that blood and qi are inseparable; internal blood stasis blocks qi movement, which fails to transport body fluids upward, resulting in thirst." The pathogenesis of blood stasis obstruction was first recorded in Internal Classic: "Anger causes qi counterflow, accumulation in the chest, retention of blood and qi, impaired blood circulation, transformation into heat, heat consuming the skin, hence Pi Xiao." National TCM Master Zou Yanqin [26] held that spleen-kidney deficiency is the basis of disease onset, and damp stasis obstructing collaterals persists throughout the disease course. Liu Hongfang [27] proposed the theory of "essence damage and collateral arthralgia," arguing that DKD involves all five zang-organs with the kidney as the core; kidney deficiency is marked by essence damage, zang-fu dysfunction leads to endogenous "dampness," "turbidity," and "stasis," stagnation due to deficiency, prolonged accumulation, invasion of zang-organs and blood collaterals, and direct obstruction of renal collaterals. Professor Tong Xiaolin [28] believed that DKD is dominated by qi deficiency, manifested mainly by stasis and turbidity. Liu Jiangteng et al. [29] summarized Professor Zhao Jinxi's clinical emphasis on adhering to the pathogenesis and attaching importance to the formation of "micro-abdominal masses," applying the therapeutic method of supplementing qi and activating blood circulation throughout treatment. In summary, damp turbidity and blood stasis are part of the pathogenesis of this disease.

3.3 TCM Treatment

TCM holds that DKD is typically manifested as renal wasting, turbid urine, and edema, with spleen-kidney qi deficiency as the root and blood stasis and turbid toxin obstructing collaterals as the manifestation, forming a complex syndrome system of mixed deficiency and excess. Danxi's Experiential Therapy proposed the pathological evolution rule of "prolonged Xiaoke depletes kidney water and causes internal blood stasis," providing a theoretical basis for the method of tonifying the kidney and activating blood circulation [30]. Metabolomics studies further reveal that DKD patients have tricarboxylic acid cycle disorders and amino acid metabolism disorders, which are highly consistent with the TCM pathogenesis of "spleen failing to transport and transform, kidney failing to store essence" [31]. Based on this, tonifying the kidney and supplementing qi to consolidate the root, and draining turbidity and unblocking collaterals to eliminate pathogens have become the core therapeutic principles of TCM for DKD.

Chen Yanqiang et al. [32] divided DKD into five stages for treatment based on the Western medical Mogensen staging and their own clinical experience: stage I is dominated by yin deficiency and dryness-heat, treated with modified Xiaoke Decoction, Jade Maid Decoction, Liuwei Dihuang Pill, etc.; stage II is dominated by qi-yin deficiency, treated with modified herbs such as Atractylodis Macrocephalae Rhizoma, Dioscoreae Rhizoma, Pseudostellariae Radix, and Astragali Radix; stage III is dominated by spleen-kidney deficiency, treated with modified Four Gentlemen Decoction, Tonify Middle Qi Decoction, etc.; stage IV is dominated by spleen-kidney yang deficiency with water retention, treated with modified Spleen-Strengthening Drink, Jisheng Shenqi Pill, True Warrior Decoction, etc.; stage V is dominated by internal retention of turbid toxin, treated with insect drugs for searching and unblocking collaterals (e.g., Hirudo, Pheretima, Eupolyphaga Steleophaga). Taiping Holy Benevolent Prescriptions of the Song Dynasty stipulated that blood-activating and stasis-resolving methods should be used to treat "renal wasting," such as Shenli Decoction, which contains a large number of qi-regulating, blood-activating, and stasis-resolving herbs including Angelicae Sinensis Radix, Chuanxiong Rhizoma, and Paeoniae Radix Rubra.

Chinese patent medicines are widely used in DKD treatment, combined with Western medicine and Chinese herbal medicines, exerting effects of reducing urinary protein, delaying renal function progression, and improving clinical symptoms: Huangkui Capsule and Keluoxin Capsule effectively reduce urinary protein and improve renal function in DKD patients; Cordyceps preparations such as Bailing Capsule and Bailing Tablet can assist in reducing urinary albumin excretion rate and 24-hour urinary protein quantity and improve renal function [33]; others include Shenyan Kangfu Tablet, Haikun Shenxi Capsule, Shenkang Injection, Niaoduqing Granule, Shenshuaining Capsule, Jinshuibao Capsule, Tripterygium Glycosides Tablet, etc.

3.4 Active Components of Chinese Herbs

Studies have shown that Astragali Radix can protect the kidneys by inhibiting signaling pathways, reducing apoptosis, participating in energy metabolism, and regulating insulin sensitivity in the pathogenesis of DKD [34]. Modern pharmacological research on Atractylodis Macrocephalae Rhizoma [35] shows that its active components including atractylenolide and atractylodes polysaccharide exert anti-inflammatory, antioxidant, anti-platelet aggregation, immunomodulatory, and hypoglycemic effects. Codonopsis Radix can reduce blood viscosity, improve hemodynamics in patients, and prevent microthrombosis [19]. PAN Z S et al. [36] found that pachymic acid can alleviate lipid metabolism disorders in mouse hepatocytes induced by OA-palmitic acid and promote hepatocyte lipolysis by activating the SIRT6 signaling pathway. Experimental studies have shown that total saponins from Dioscoreae Septemlobae Rhizoma have renal protective effects [37]. Wang Shuoshi et al. [38] found that isochlorogenic acid B from the Miao medicine Pyrrosia petiolosa is an important active component for hypoglycemia, blocking AGE-RAGE signaling pathway-mediated pancreatic islet cell apoptosis by regulating key genes JNK, P38, BAX, CASP3, and RAGE to achieve hypoglycemic effects. The chemical components of Chuanxiong Rhizoma are mainly phthalides (e.g., ligustilide), alkaloids (e.g., tetramethylpyrazine), phenolic acids (e.g., ferulic acid, caffeic acid), and polysaccharides [39–40], which exert vasodilatory, anti-platelet aggregation, anti-inflammatory, antioxidant, and analgesic effects [41–42]. Paeoniae Radix Rubra 801 inhibits platelet aggregation and lowers blood lipids [43]. Pheretima mainly exerts anticoagulant and antithrombotic effects [44]. Studies have shown that insect drugs for activating blood circulation and unblocking collaterals such as Eupolyphaga Steleophaga can delay DKD progression by improving hemodynamics [45]. Xu Biqi et al. [46] found that flavonoids from Eucommiae Cortex reduce fasting blood glucose and improve renal function in DN mice, related to ameliorating oxidative stress in renal tissue. Zhang Jie et al. [47] found that

Achyranthis Bidentatae Radix delays and prevents diabetic renal damage and inhibits renal tissue cell apoptosis. Euryales Semen has antioxidant, hypoglycemic, and gastric mucosal protective bioactivities, with polysaccharides, sterols, polyphenols, etc. as the main active compounds [48]. Rosae Laevigatae Fructus has astringent and antidiarrheal, hepatic and renal protective, immunomodulatory, lipid-lowering, antioxidant, antibacterial, and anti-inflammatory bioactivities.

4. Conclusion

DKD is a chronic progressive disease characterized by insidious onset and high missed diagnosis rate, leading most patients to be diagnosed at stages III–IV. Current conventional Western medical treatments are mainly symptomatic, with problems such as unstable efficacy in some patients, numerous toxic and side effects, high cost of some drugs, and poor patient compliance. In contrast, TCM provides an alternative option for clinical diagnosis and treatment of DKD with its advantages of stable action and high safety. Flexible application of TCM as adjuvant therapy on the basis of basic Western medical treatment can reduce renal damage and delay the progression of renal failure, aiming to achieve optimal efficacy, which has become a new trend in current clinical practice.

References

[1] Expert Group of Nephrology Branch of Chinese Medical Association. Chinese guidelines for clinical diagnosis and treatment of diabetic kidney disease[J]. Chinese Journal of Nephrology, 2021, 37(3): 255-304.

[2] Guo Q, Li GL, Liu H, et al. Research progress of adipose-derived mesenchymal stem cells in the treatment of diabetes mellitus and its chronic complications[J]. Chinese Journal of Cell and Stem Cells (Electronic Edition), 2023, 13(1): 56-62.

[3] Wang Y, Zhou JW, Wang Z, et al. Progress in integrated traditional Chinese and western medicine treatment of diabetic kidney disease[J]. Chinese General Practice, 2022, 25(12): 1411-1417.

[4] Liu YT, et al. Research progress on lipid metabolism mechanism of diabetic kidney disease and intervention of traditional Chinese medicine[J]. Journal of Changchun University of Chinese Medicine, 2025, 41(11): 1292-1298.

[5] Zhang L, Miao R, Yu T, et al. Comparative effectiveness of traditional Chinese medicine and angiotensin converting enzyme inhibitors, angiotensin receptor blockers, and sodium glucose cotransporter inhibitors in patients with diabetic kidney disease: A systematic review and network meta-analysis[J]. Pharmacological Research, 2022, 177: 106111.

[6] Microvascular Complications Group of Diabetes Branch of Chinese Medical Association. Clinical guidelines for the prevention and treatment of diabetic kidney disease in China[J]. Chinese Journal of Diabetes Mellitus, 2019, 11(1): 15-28.

[7] Reidy K, Kang HM, Hostetter T, et al. Molecular mechanisms of diabetic kidney disease[J]. Journal of Clinical Investigation, 2014, 124(6): 2333-2340.

[8] China Association of Chinese Medicine; Dongzhimen Hospital of Beijing University of Chinese Medicine; Beijing University of Chinese Medicine. Guidelines for integrated traditional Chinese and western medicine diagnosis and treatment of diabetic kidney disease[J]. Journal of Beijing University of Chinese Medicine, 2024, 47(4): 580-592.

[9] Gupta S, Dominguez M, Golestaneh L. Diabetic kidney disease: An update[J]. Medical Clinics of North America, 2023, 107(4): 689-705.

[10] Zhao MY, Yu M, Cheng YY, et al. Pathogenesis and treatment progress of diabetic kidney disease[J]. Acta Pharmaceutica Sinica, 2026, 61(3): 694-705.

[11] Li HL, Tang CC, Shen YP, et al. Research progress on pathogenesis of diabetic kidney disease and integrated traditional Chinese and western medicine treatment[J]. Clinical Medicine & Engineering, 2023, 30(8): 1175-1176.

[12] Liu YT, Cui CJ, Wang ZK, et al. Research progress on lipid metabolism mechanism of diabetic kidney disease and intervention of traditional Chinese medicine[J]. Journal of Changchun University of Chinese Medicine, 2025, 41(11): 1292-1298.

[13] Lee LE, Doke T, Mukhi D, et al. The key role of altered tubule cell lipid metabolism in kidney disease development[J]. Kidney International, 2024, 106: 24-34.

[14] Mitrofanova A, Merscher S, Fornoni A. Kidney lipid dysmetabolism and lipid droplet accumulation in chronic kidney disease[J]. Nature Reviews Nephrology, 2023, 19: 629-645.

[15] Guo ZC, Lan T, Ma LY, et al. Research progress on mechanism of exercise improving diabetic kidney disease[J]. Chinese Journal of Cell Biology, 2023, 45(10): 1568-1574.

[16] Ding LD, Shao P, Li M, et al. Meta-analysis of correlation between smoking and diabetic kidney disease[J]. Zhejiang Practical Medicine, 2019, 24(1): 75-79.

[17] Hong M, Zhu HQ. Interpretation of guidelines for the prevention and treatment of diabetic kidney disease in China (2021 Edition) [J]. Chinese Journal of Medicine, 2022, 57(2): 133-138.

[18] KDIGO Diabetes Work Group. KDIGO 2022 clinical practice guideline for diabetes management in chronic kidney disease[J]. Kidney International, 2022, 102(5S): S1-S127.

[19] Expert Group on Multidisciplinary Diagnosis, Treatment and Management of Diabetic Kidney Disease. Expert consensus on multidisciplinary diagnosis, treatment and management of diabetic kidney disease[J]. Chinese Journal for Clinicians, 2020, 48(5): 522-527.

[20] KDIGO Diabetes Work Group. KDIGO 2020 clinical practice guideline for diabetes management in chronic kidney disease[J]. Kidney International, 2020, 98(4S): S1-S115.

[21] Zhang QY, Liu JR, Wu X, et al. Clinical efficacy of finerenone combined with sacubitril/valsartan and dapagliflozin in the treatment of early diabetic kidney disease[J]. Shandong Medical Journal, 2025, 65(12): 1-6.

[22] Guo Q, Li GL, Liu H, et al. Research progress of adipose-derived mesenchymal stem cells in the treatment of diabetes mellitus and its chronic complications[J]. Chinese Journal of Cell and Stem Cells (Electronic Edition), 2023, 13(1): 56-62.

[23] ElSayed NA, Aleppo G, Aroda VR, et al. 11. Chronic kidney disease and risk management: standards of care in diabetes-2023[J]. Diabetes Care, 2023, 46(Suppl 1): S197–S215.
[24] Gupta S, Dominguez M, Golestaneh L. Diabetic kidney disease: An update[J]. Medical Clinics of North America, 2023, 107(4): 689-705.
[25] Xiang WZ. Treating Xiaoke disease by regulating kidney yin and kidney yang[J]. Sichuan Journal of Traditional Chinese Medicine, 2010, 28(5): 30-31.
[26] Yan QH, Zou YQ. Professor Zou Yanqin, a national master of traditional Chinese medicine, treats diabetic kidney disease from spleen and kidney[J]. Journal of Nanjing University of Chinese Medicine, 2018, 34(2): 112-115.
[27] Hu JY, Liu HF, Zhang XW. Discussion on the pathogenesis of “essence damage and collateral arthralgia” in diabetic kidney disease[J]. Journal of Beijing University of Chinese Medicine, 2019, 42(1): 8-11.
[28] Tong XL, Zhou Q, Zhao LH, et al. Experience in TCM syndrome differentiation and treatment of diabetic kidney disease[J]. China Journal of Traditional Chinese Medicine and Pharmacy, 2014, 29(1): 11-13.
[29] Liu JT, Ruan ZC, Lin YY, et al. Study on the medication rule of Zhao Jinxi in treating diabetic kidney disease based on multivariate data mining[J]. Chinese Journal of Information on Traditional Chinese Medicine, 2025, 32(11): 71-76.
[30] Guo JE, Zhang JH, Zhang Y, et al. Clinical efficacy of modified Shuilu Erxian Dan combined with Difang Decoction on patients with early and middle stage diabetic kidney disease[J]. Chinese Traditional Patent Medicine, 2024, 46(5): 1514-1519.
[31] Liu N, Liu CX, Huang JM. Research progress in targeted metabolomics of diabetic complications[J]. Drug Evaluation Research, 2022, 45(6): 1166-1172.
[32] Chen YQ, Shi W, Huang YY. Discussion on TCM staging and syndrome differentiation treatment of diabetic kidney disease[J]. New Journal of Traditional Chinese Medicine, 2009, 41(3): 3-4.
[33] Expert Group of Nephrology Committee of Chinese Research Hospital Association; Liu DW; Fan WW. Expert consensus on integrated traditional Chinese and western medicine treatment and follow-up management of primary diabetic kidney disease[J]. Chinese Journal of Research Hospitals, 2024, 11(5): 1-15.
[34] Hong M, Zhu HQ. Interpretation of guidelines for the prevention and treatment of diabetic kidney disease in China (2021 Edition) [J]. Chinese Journal of Medicine, 2022, 57(2): 133-138.
[35] KDIGO Diabetes Work Group. KDIGO 2022 clinical practice guideline for diabetes management in chronic kidney disease[J]. Kidney International, 2022, 102(5S): S1-S127.
[36] KDIGO Diabetes Work Group. KDIGO 2020 clinical practice guideline for diabetes management in chronic kidney disease[J]. Kidney International, 2020, 98(4S): S1-S115.
[37] Expert Group of Nephrology Branch of Chinese Medical Association. Chinese guidelines for clinical diagnosis and treatment of diabetic kidney disease[J]. Chinese Journal of Nephrology, 2021, 37(3): 255-304.
[38] Jia YJ, Xue YM. Interpretation of guidelines for the prevention and treatment of diabetic kidney disease in China (2021 Edition) [J]. Journal of Clinical Internal Medicine, 2022, 39(5): 303-306.
[39] Ali A, Bain S, Hicks D, et al. Correction to: SGLT2 inhibitors: cardiovascular benefits beyond HbA1c—translating evidence into practice[J]. Diabetes Therapy, 2019, 10(5): 1623-1624.
[40] American Diabetes Association Professional Practice Committee; Draznin B, Aroda VR, et al. Chronic kidney disease and risk management: Standards of medical care in diabetes-2022[J]. Diabetes Care, 2022, 45(Suppl 1): S175-S184.
[41] Wu L, Li GJ, Fan T, et al. Research progress of SGLT-2 inhibitors and diabetic kidney disease[J]. Journal of Xiangnan University (Medical Edition), 2020, 22(3): 72-75.
[42] Zhang QY, Liu JR, Wu X, et al. Clinical efficacy of finerenone combined with sacubitril/valsartan and dapagliflozin in the treatment of early diabetic kidney disease[J]. Shandong Medical Journal, 2025, 65(12): 1-6.
[43] Guo Q, Li GL, Liu H, et al. Research progress of adipose-derived mesenchymal stem cells in the treatment of diabetes mellitus and its chronic complications[J]. Chinese Journal of Cell and Stem Cells (Electronic Edition), 2023, 13(1): 56-62.
[44] Wang Y, Zhou JW, Wang Z, et al. Progress in integrated traditional Chinese and western medicine treatment of diabetic kidney disease[J]. Chinese General Practice, 2022, 25(12): 1411-1417.
[45] Liu YT, et al. Research progress on lipid metabolism mechanism of diabetic kidney disease and intervention of traditional Chinese medicine[J]. Journal of Changchun University of Chinese Medicine, 2025, 41(11): 1292-1298.
[46] Zhang L, Miao R, Yu T, et al. Comparative effectiveness of traditional Chinese medicine and angiotensin converting enzyme inhibitors, angiotensin receptor blockers, and sodium glucose cotransporter inhibitors in patients with diabetic kidney disease: A systematic review and network meta-analysis[J]. Pharmacological Research, 2022, 177: 106111.
[47] Diabetes Branch of Chinese Medical Association. Guidelines for the prevention and treatment of type 2 diabetes in China (2020 Edition) [J]. Chinese Journal of Diabetes Mellitus, 2021, 13(4): 279-337.
[48] Microvascular Complications Group of Diabetes Branch of Chinese Medical Association. Guidelines for the prevention and treatment of diabetic kidney disease in China (2021 Edition) [J]. Chinese Journal of Diabetes Mellitus, 2021, 13(8): 757-777.