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Research Progress on the Pathogenesis and Chinese and Western Medicine Treatment of Diabetic Kidney Disease

Xiaoduo Ai[1], Genping Lei[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) is a common microvascular complication of diabetes and an important cause of chronic kidney disease progressing to kidney failure. Its onset and progression are not attributable to hyperglycemia alone, but rather to the combined effects of multiple factors, including disorders of glucose and lipid metabolism, abnormal glomerular hemodynamics, oxidative stress, inflammatory responses, impaired autophagy, programmed cell death, and renal fibrosis. Modern medical treatment has evolved from simply controlling blood glucose and blood pressure to comprehensive interventions aimed at reducing proteinuria and providing cardiorenal protection. Sodium-glucose cotransporter 2 (SGLT2) inhibitors, renin-angiotensin-aldosterone system (RAAS) inhibitors, glucagon-like peptide-1 (GLP-1) receptor agonists, and nonsteroidal mineralocorticoid receptor antagonists play important roles in slowing disease progression. In traditional Chinese medicine (TCM), DKD is considered a disorder characterized by deficiency in the root and excess in the branch, with qi-yin deficiency and spleen-kidney deficiency as the root and dampness, blood stasis, phlegm, turbidity, and toxin as the manifestations of excess. Chinese herbal formulas, proprietary Chinese medicines, and herb pairs may ameliorate renal injury through multitarget regulation. This review summarizes the major pathogenic mechanisms of DKD and advances in Western and Chinese medical treatment, with the aim of providing a reference for clinical prevention and management.***

Keywords: Diabetic kidney disease, Pathogenesis, Western medical treatment, Traditional Chinese medicine treatment**.**

1. Introduction

Diabetic kidney disease (DKD) is one of the most common and serious microvascular complications of diabetes, and approximately 20%-40% of patients with diabetes develop varying degrees of renal impairment [1]. DKD is mainly characterized by increased urinary albumin excretion and a progressive decline in glomerular filtration rate. As the disease advances, patients may develop overt proteinuria, edema, and renal dysfunction, eventually progressing to end-stage renal disease [2]. Its onset and progression are influenced by multiple factors. Persistent hyperglycemia and abnormal glomerular hemodynamics constitute important pathophysiological foundations, while oxidative stress, inflammatory responses, impaired autophagy, and renal fibrosis also participate in disease progression [3]. A long duration of diabetes, poor glycemic control, hypertension, dyslipidemia, and obesity may further increase the risk of DKD [4]. With the growing prevalence of diabetes, the disease burden associated with DKD continues to increase. This article systematically reviews the pathogenesis of DKD and advances in Chinese and Western medical treatment, with the aim of providing a reference for clinical prevention and management.

2. Pathological Basis of Diabetic Kidney Disease

DKD may involve the glomeruli, tubulointerstitium, and renal microvasculature. Early pathological changes mainly include glomerular hypertrophy, thickening of the basement membrane, and mesangial matrix expansion, whereas progressive disease may be accompanied by podocyte detachment, foot process effacement, glomerulosclerosis, and nodular lesions [5]. Persistent hyperglycemia, proteinuria, inflammation, and hypoxia may also damage renal tubular epithelial cells, promote fibroblast activation and extracellular matrix deposition, and ultimately lead to renal interstitial fibrosis [6].In addition, hyaline degeneration and luminal narrowing of the renal microvasculature may further reduce tissue perfusion, create an ischemic and hypoxic microenvironment, and interact with tubular injury to aggravate disease progression [7].Therefore, DKD is not merely a glomerular disease, but rather a chronic injury involving the entire nephron, including the glomeruli, tubulointerstitium, and microvasculature. The cumulative burden of these pathological changes ultimately determines the rate of renal function decline [8].

3. Pathogenesis of Diabetic Kidney Disease

3.1 Disorders of Glucose and Lipid Metabolism and Hemodynamic Changes

Persistent hyperglycemia can activate the polyol, protein kinase C, and hexosamine pathways and promote the formation of advanced glycation end products (AGEs) [9]. Binding of AGEs to the receptor for advanced glycation end products (RAGE) can induce oxidative stress and inflammatory responses, resulting in injury to podocytes, mesangial cells, and renal tubular epithelial cells [10]. Lipotoxicity caused by dyslipidemia and insulin resistance may further aggravate renal damage [11]. In the early stages of diabetes, increased sodium-glucose reabsorption in the proximal tubule suppresses tubuloglomerular feedback and causes dilation of the afferent arteriole. At the same time, activation of the RAAS causes relative constriction of the efferent arteriole, leading to glomerular hyperfiltration and intraglomerular hypertension. Persistent mechanical stress subsequently damages the filtration barrier and promotes the

development of proteinuria.

3.2 Oxidative Stress, Mitochondrial Dysfunction, and Inflammation

Hyperglycemia can activate NADPH oxidase and disrupt the mitochondrial electron transport chain, resulting in increased production of reactive oxygen species (ROS) [12]. Excessive ROS can damage lipids, proteins, and DNpA and induce a decrease in mitochondrial membrane potential, reduced ATP production, and impaired mitophagy, thereby forming a vicious cycle of "oxidative stress-mitochondrial injury-aggravated oxidative stress." Meanwhile, hyperglycemia, AGEs, and ROS can activate NF-kappaB and the NLRP3 inflammasome, promoting the release of inflammatory mediators such as TNF-alpha, IL-1beta, IL-6, and IL-18. Inflammation and oxidative stress reinforce each other, continuously amplifying renal injury [13].

3.3 Impaired Autophagy, Programmed Cell Death, and Renal Fibrosis

Autophagy removes damaged organelles and abnormal proteins and is an important mechanism for maintaining homeostasis in podocytes and renal tubular cells. In diabetes, impaired autophagic flux leads to the accumulation of damaged mitochondria and abnormal proteins, while enhanced endoplasmic reticulum stress can further induce apoptosis [14]. In recent years, pyroptosis and ferroptosis have also been implicated in DKD progression. The NLRP3/caspase-1/GSDMD pathway is closely associated with pyroptosis, whereas dysfunction of the SLC7A11/GPX4 antioxidant system promotes iron-dependent lipid peroxidation [15]. Persistent hyperglycemia, inflammation, and oxidative stress ultimately activate profibrotic signaling pathways such as TGF-beta/Smad, resulting in the deposition of collagen and fibronectin and promoting glomerulosclerosis and renal interstitial fibrosis [16]. These mechanisms do not act independently but show extensive cross-talk: oxidative stress can activate inflammasomes, inflammation can further aggravate mitochondrial injury, and impaired autophagy reduces the clearance of damaged organelles. Together, these abnormalities promote cell death and fibrosis and form a continuously progressive pathological network.

4. Advances in Western Medical Treatment of Diabetic Kidney Disease

4.1 Glucose-Lowering and Cardiorenal Protective Therapy

Appropriate glycemic control is the foundation of DKD treatment. Dipeptidyl peptidase-4 (DPP-4) inhibitors improve glucose metabolism by increasing endogenous GLP-1 levels and may also exert certain effects on inflammation and urinary albumin excretion [17]. although their principal role remains glycemic management. SGLT2 inhibitors reduce glucose and sodium reabsorption in the proximal tubule, restore tubuloglomerular feedback, and decrease glomerular hyperfiltration and intraglomerular pressure. They may also reduce inflammation, oxidative stress, and metabolic burden and have therefore become important agents for renal protection in DKD [18]. In addition to improving glycemic control, GLP-1 receptor agonists can reduce body weight and cardiovascular risk. Studies of agents such as semaglutide have shown improvements in important renal outcomes, providing new treatment options for patients with type 2 diabetes and chronic kidney disease [19].

4.2 RAAS Inhibitors and Nonsteroidal Mineralocorticoid Receptor Antagonists

Angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) inhibit the RAAS, dilate the efferent arteriole, and reduce intraglomerular pressure, thereby decreasing proteinuria and slowing the decline in renal function [20]. They are important foundational therapies for patients with concomitant hypertension and albuminuria, and serum creatinine and potassium should be monitored during treatment. Finerenone, a nonsteroidal mineralocorticoid receptor antagonist, can further inhibit inflammation and fibrosis and, when added to standard RAAS blockade, reduce the risk of renal and cardiovascular events [21]; however, hyperkalemia should be closely monitored. In patients with inadequate blood pressure control or concomitant cardiovascular disease, calcium channel blockers or beta-blockers may be added according to the clinical situation [22]. Overall, Western medical treatment emphasizes individualized management according to eGFR, the degree of albuminuria, blood pressure, serum potassium, and cardiovascular risk. Rational combinations of drugs with different mechanisms may further reduce residual renal risk.

5. Advances in Traditional Chinese Medicine Treatment of Diabetic Kidney Disease

Traditional Chinese medicine does not contain a disease entity exactly corresponding to "diabetic kidney disease." According to its clinical manifestations, including prolonged xiaoke, proteinuria, edema, and progressive renal dysfunction, DKD may be categorized under traditional disease concepts such as xiaoke (wasting-thirst), niaozhuo (turbid urine), shuizhong (edema), xulao (consumptive disease), and guange [23]. The kidney is considered the primary organ involved, with a close relationship to the spleen. Prolonged xiaoke consumes qi and yin; impaired spleen transportation and transformation and failure of kidney storage lead to loss of essence and nutrients through the urine. Qi deficiency may impair the movement of blood and give rise to blood stasis, whereas spleen deficiency promotes the accumulation of dampness and turbidity. With prolonged disease entering the collaterals, dampness, blood stasis, turbidity, and toxin may become intertwined. Therefore, DKD is generally characterized by deficiency in the root and excess in the branch, with qi-yin deficiency and spleen-kidney deficiency as the root and dampness-turbidity, blood stasis, and phlegm-toxin as the branch manifestations. Treatment commonly includes replenishing qi and nourishing yin, strengthening the spleen and tonifying the kidney, activating blood and unblocking the collaterals, clearing heat and draining dampness, and eliminating turbidity and toxins [24].

5.1 Chinese Herbal Formula Treatment

Chinese herbal formulas can be flexibly modified according to the stage and syndrome pattern of DKD. Modified Shenqi

Dihuang Decoction is based on the principles of replenishing qi and nourishing yin, tonifying the kidney and securing essence, and activating blood and unblocking the collaterals. Clinical studies have shown that its addition to Western medical treatment may improve TCM symptoms, urinary albumin-to-creatinine ratio (UACR), and certain indices of glucose metabolism and renal function in patients with early DKD [25]. Baoshen Formula, which contains Astragalus, raw and prepared Rehmannia root, and Salvia miltiorrhiza, among other herbs, is mainly used for patients with qi-yin deficiency complicated by blood stasis obstructing the kidney collaterals and may reduce urinary protein and improve renal function [26]. Tangshenkang Formula focuses on strengthening the spleen, tonifying the kidney, replenishing qi, and nourishing yin; related studies suggest that it may delay renal injury by increasing Klotho protein levels [27]. For patients with spleen-kidney yang deficiency and retention of water-dampness, modified Zhenwu Decoction may warm yang and strengthen the spleen, activate blood, and promote diuresis, thereby improving edema, urinary protein, and serum creatinine levels [28]. Chinese herbal formulas are characterized by treatment of both the root and manifestations and by multitarget regulation. Modern studies have implicated effects on oxidative stress, inflammation, autophagy, podocyte injury, and fibrosis. However, substantial heterogeneity remains in formula composition, dosage, treatment duration, and clinical outcome measures, and high-quality clinical trials are still required.

5.2 Proprietary Chinese Medicine Treatment

Proprietary Chinese medicines have relatively standardized dosage forms and compositions and are therefore convenient for long-term combination therapy. Bailing Capsules are traditionally used to tonify the lung and kidney and replenish essence and qi; when combined with dapagliflozin, they have been reported to improve proteinuria, inflammatory markers, and renal function in patients with DKD [29]. Huangkui Capsules, whose main ingredient is Abelmoschus manihot flower, may exert renoprotective effects through anti-inflammatory and antioxidant actions and by reducing renal tubular injury [30]. Rehmannia Leaf Total Glycoside Capsules are traditionally used to nourish yin, clear heat, promote diuresis, and reduce edema and may improve certain indices of glucose metabolism, inflammation, and renal function [31]. Keluoxin Capsules, containing Astragalus, Rehmannia, Scrophularia, Salvia miltiorrhiza, and other herbs, are used to replenish qi, nourish yin, activate blood, and unblock the collaterals and may delay DKD progression by reducing oxidative stress, inflammation, and microvascular injury [32].

5.3 Chinese Herbal Pair Treatment

Herb pairs represent a basic form of combination therapy in Chinese herbal prescriptions. The Huangqin-Huanglian (Scutellaria-Coptis) pair is used to clear heat and dry dampness and may exert therapeutic effects by improving glucose and lipid metabolism and reducing inflammation and fibrosis [33]. The Yinchen-Yuzhu (Artemisia capillaris-Polygonatum odoratum) pair combines the actions of nourishing yin, clearing heat, draining dampness, and dispersing accumulations and may intervene in DKD through multiple mechanisms involving metabolism, inflammation, and oxidative stress [34]. The Cangzhu-Huangbai (Atractylodes-Phellodendron) pair is characterized by strengthening the spleen, dispelling dampness, and clearing damp-heat and is considered suitable for patients with damp-heat accumulation [35]. The Yizhiren-Wuyao (Alpinia oxyphylla-Lindera) pair is traditionally used to warm the kidney, dispel cold, and secure essence; experimental studies suggest that it may protect renal tissue by regulating autophagy [36]. Compared with herbal formulas and proprietary Chinese medicines, research on herb pairs is still largely concentrated on experimental studies and network pharmacology, and stronger clinical evidence is needed.

6. Conclusions and Perspectives

DKD is a chronic kidney disease driven by the combined effects of metabolic abnormalities, glomerular hyperfiltration, oxidative stress, inflammation, impaired autophagy, programmed cell death, and fibrosis. Modern medicine has established a comprehensive cardiorenal protective strategy based on glycemic and blood pressure management and the combined use of SGLT2 inhibitors, RAAS inhibitors, GLP-1 receptor agonists, and finerenone. TCM approaches DKD from pathogenic patterns such as spleen-kidney deficiency, qi-yin deficiency, and the accumulation of dampness, blood stasis, turbidity, and toxin, and applies herbal formulas, proprietary Chinese medicines, and herb pairs for multitarget intervention. These approaches have shown potential in reducing proteinuria, alleviating inflammation, and preserving renal function. However, current TCM research remains limited by relatively small sample sizes, insufficient quality control, and a lack of long-term hard renal endpoints. Future studies should strengthen multicenter randomized controlled trials and integrate technologies such as single-cell sequencing, multi-omics, and organoids to further elucidate the active material basis and key molecular targets of TCM, thereby providing more robust evidence for precise integrated Chinese and Western medical treatment of DKD.

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