DOI: 10.53469/jcmp.2025.07(04).48

Research Progress on Micro Inflammatory Status of Chronic Kidney Disease

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Abstract: ***The occurrence of microinflammation is affected by various factors, and its mechanism is relatively complex. Moreover, this state can have adverse effects on patients with chronic kidney disease (CKD). Currently, for the microinflammatory state existing in CKD patients, Western medicine mainly intervenes through methods such as drug therapy, exercise therapy, and optimized dialysis. In terms of traditional Chinese medicine's role in improving the microinflammatory state in the bodies of CKD patients, Reflecting the unique advantages possessed by traditional Chinese medicine.***

Keywords: Chronic kidney disease, Microinflammatory state, Treatment, Literature review**.**

1. Introduction

Chronic kidney disease (CKD) is a disease that seriously endangers human health. Its characteristics such as high prevalence rate, low awareness rate, poor prognosis, and high medical costs make it another important threat besides cardiovascular and cerebrovascular diseases, diabetes mellitus, and malignant tumors [1]. As the renal function of CKD patients declines, they will experience water and sodium retention, toxin accumulation, abnormal hormone secretion, metabolic disorders, and a microinflammatory state. Some studies have shown that with the decline of glomerular filtration rate (GFR), the incidence rate of the microinflammatory state gradually increases. The incidence rate of the microinflammatory state in patients with CKD stage 1 is 8.2%, and the incidence rate in patients with CKD stages 3 to 5 can reach 49% [2].

Currently, the commonly studied markers of the microinflammatory state at home and abroad include high-sensitivity C-reactive protein (hs-CRP), C-reactive protein (CRP), Procalcitonin (PCT), and interleukins (IL). The fibrinogen/albumin ratio, systemic immune - inflammation index, platelet-lymphocyte ratio (PLR), and neutrophil-lymphocyte ratio (NLR) are novel inflammatory markers proposed in the research field related to kidney diseases in recent years [3].Previous studies have found that the NLR and PLR levels are elevated in patients with chronic kidney disease (CKD), and they are positively correlated with CRP, IL-6, and TNF-α. This suggests that NLR and PLR can serve as potential alternative indicators of CRP for evaluating the systemic inflammatory state of CKD patients [4]. They can reflect the renal function status during the disease progression in the body and are associated with poor prognosis [5].

2. Factors and Mechanisms Influencing the Occurrence of Microinflammation

The kidney has a dual role in inducing the production and clearance of inflammatory cells [6]. In the CRIC study involving 3, 430 CKD patients [7], it was found that as the glomerular filtration rate decreased, the levels of pro-inflammatory factors in the bodies of CKD patients gradually increased, indicating a positive correlation between the exacerbation of the microinflammatory state and the decline of renal function. On the one hand, as the renal function of CKD patients declines, uremic toxins gradually accumulate in the body, leading to the activation of the immune pathway. The activated immune system leads to the activation of the pro-inflammatory signaling pathway, causing excessive secretion of pro-inflammatory factors. In turn, the elevated levels of pro-inflammatory factors in the circulation will further damage the kidneys and lead to a decrease in renal clearance [8]. As the renal function declines, the intestines compensate by excreting the body's metabolites through the intestines. In chronic renal failure, the excretion of intestinal toxins can reach 80% [9]. Liang Jie and others found that in CKD patients, the levels of inflammatory factors such as CRP, IL-6, and TNF-α were negatively correlated with the numbers of Bifidobacterium and Lactobacillus acidophilus ($P<0.05$), and positively correlated with the numbers of Enterococcus faecalis and Escherichia coli ($P < 0.05$) [10]. For CKD patients who have received dialysis filtration for a long time, their antioxidant stress capacity decreases. The accumulated immune complexes in the body will activate the mononuclear phagocyte system and other parts of the immune system. At the same time, the incompatibility of the dialysis membrane, the contamination of the dialysate, the presence of the vascular access, and frequent operations all lead to the body being in a microinflammatory state for a long time [11-12]. Studies have found [13-14] that as the glomerular filtration rate decreases, the triglyceride levels in CKD patients increase, and the high-density lipoprotein cholesterol levels decrease. High-density lipoprotein is transformed from an anti-inflammatory factor into a pro-inflammatory factor, and the pro-inflammatory properties of low-density lipoprotein are further enhanced. At the same time, the accumulation of lipids induces the activation of multiple inflammatory pathways. These factors work together to lead to the exacerbation of the microinflammatory state.

At present, the mechanism of the occurrence of the microinflammatory state in patients with chronic kidney disease (CKD) is still unclear. Some studies have found that the NF-κB signaling pathway, NLRP3 inflammasome

signaling pathway, MAPK signaling pathway, and JAK/STAT signaling pathway are all involved in the regulation of the microinflammatory state in kidney diseases [15].

3. Adverse Effects of the Microinflammatory State on Patients with Chronic Kidney Disease

The microinflammatory response leads to and exacerbates renal interstitial fibrosis. With the progression of the course of chronic kidney disease (CKD), the microinflammatory state is significantly associated with the occurrence of complications such as cardiovascular events, renal anemia, lipid metabolism disorders, and malnutrition.

3.1 Microinflammation and Cardiovascular Events

Cardiovascular complications are one of the common complications of chronic kidney disease (CKD), and they are also one of the main causes of death in patients with end-stage renal disease. Foreign studies have found that pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) can damage the endothelial cells in blood vessels. IL-1 in combination with TNF-α can lead to the occurrence of vascular calcification (VC) by affecting the functions of nuclear factor-κB receptor activator ligand and osteoprotegerin in the blood vessel wall. Inflammatory mediators such as C-reactive protein (CRP), IL and TNF-α promote the formation of VC by promoting the transformation of vascular smooth muscle cells into an osteoblast-like cell phenotype and inhibiting the production and utilization of nitric oxide (NO). At the same time, they promote the degradation of elastin and collagen in the blood vessel wall, providing a site for mineral deposition, thus participating in the progression of VC [16, 11].

3.2 Microinflammation and Renal Anemia

Under the microinflammatory state of chronic kidney disease (CKD), inflammatory factors such as interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-α) can inhibit the hematopoietic function of the bone marrow. Some inflammatory factors can also counteract the anti-apoptotic activity of erythropoietin, ultimately leading to a low response of patients to erythropoietin (EPO) therapeutic agents. Sustained inflammatory stimuli will continuously shorten the lifespan of red blood cells. Inflammatory mediators can also promote the liver to increase the synthesis of hepcidin. The increase in hepcidin can lead to the obstruction of iron output from cells and a decrease in the absorption of iron by the intestine, resulting in a reduction of iron ions in the serum and further exacerbating anemia [17-18].

3.3 Microinflammation and Malnutrition

Malnutrition is one of the common complications in patients with chronic kidney disease (CKD). The microinflammatory state in CKD can lead to an increase in resting energy expenditure, thus promoting protein catabolism and reducing anabolism. With the progression of the course of CKD, the oxidative stress response of patients is enhanced. As the number of inflammatory factors continues to increase, the nutritional status gradually deteriorates, ultimately affecting the prognosis and quality of life of CKD patients [19].

3.4 Microinflammation and Thrombosis

The microinflammatory state in chronic kidney disease (CKD) can damage the podocytes in the glomeruli, leading to a gradual increase in permeability and exacerbating glomerulosclerosis [20]. It can also increase the concentration of some coagulation-related proteins (such as fibrinogen) in the plasma, making patients at a higher risk of thrombosis [21].

4. Treatment of the Microinflammatory State

Regarding the treatment of the microinflammatory state, currently, it mainly includes drug therapy, exercise therapy, and optimized dialysis.

4.1 Angiotensin Converting Enzyme Inhibitor (ACEI) / Angiotensin Receptor Blocker (ARB)

Angiotensin II is an inflammatory and profibrotic factor, which is highly expressed in the bodies of patients with chronic kidney disease (CKD). It can promote the secretion of interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) by glomerular mesangial cells, thus activating inflammatory cells and participating in the process of microinflammation occurrence. Ordaz et al. [22] found in their study that treatment with ACEI and ARB can significantly reduce the serum level of IL-6, while there are no obvious changes in the levels of C-reactive protein (CRP) and TNF-α, indicating that ACEI and ARB have a certain effect in inhibiting inflammation.

4.2 Statins

In addition to their obvious lipid-lowering effects, statins have been proven by a large number of experimental and clinical studies to also have significant anti-inflammatory effects. Statins can inhibit the inflammatory response through multiple pathways: a. Preventing the accumulation of the inflammatory mediator NF-κB; b. Inhibiting the inflammasome and the subsequent maturation of IL-1β; c. Shifting the differentiation of macrophages from the M1 subset (pro-inflammatory) to the M2 subset (anti-inflammatory); d. Reducing the activation of macrophages and T cells, thereby reducing the inflammatory response. The study by Wang et al. [23] demonstrated that the level of the serum inflammatory biomarker CRP in CKD patients taking statins decreased.

4.3 Aspirin and Antioxidants

Antioxidant therapy can improve the microinflammatory state of CKD patients, enhance the creatinine clearance rate, and delay kidney damage. Corken et al. [24] found through a controlled study that after treatment with aspirin, the levels of the inflammatory markers IL-1β and TNF-α in CKD individuals decreased significantly, and the average change range was positively correlated with a higher glomerular filtration rate.

4.4 Vitamin D

Studies have found that 25-hydroxyvitamin D can play a certain role in protecting the kidneys in CKD by inhibiting the synthesis and release of inflammatory factors such as IL-6 and IL-8. The study by Zhou et al. [25] showed that the lower the level of serum 1, 25-dihydroxyvitamin D3, the higher the level of CRP, and the more obvious the deterioration of renal function.

4.5 Intestinal Microbial Agents

In CKD patients, intestinal flora imbalance is accompanied by the disruption of the intestinal barrier function, allowing the translocation of intestinal endotoxins and bacterial metabolites into the systemic circulation, thus leading to a microinflammatory state. Zhang Yan et al. found that the Bifidobacterium and Lactobacillus triple viable tablets can reduce the inflammatory factors IL-6 and TNF-α in CKD patients [26].

4.6 Exercise Therapy

Correa et al. [27] found in their study that exercise training can improve the levels of inflammatory factors and muscle strength in CKD patients, thereby improving their clinical status. Exercise can also significantly reduce the end products of oxidative stress in patients undergoing maintenance hemodialysis. Song Yanyun et al. found through research that through dynamic resistance exercise training, especially mainly using dumbbell exercises, the levels of CRP and IL-6 in chronic kidney disease patients can be reduced [28].

4.7 Optimized Dialysis

Maintenance hemodialysis is one of the main treatment measures for patients with end-stage renal failure. Since inflammatory factors have a relatively large molecular weight, the use of dialysis membranes with poor biocompatibility and low flux will instead exacerbate the inflammatory response in hemodialysis patients, while the use of high-flux dialysis membranes can effectively remove uremic endotoxins and inflammatory factors and reduce the inflammatory response [29-30]. Studies have shown that the use of sterile dialysate can reduce the levels of inflammation and oxidative stress in HD patients, while contaminated dialysate can keep patients in an inflammatory state for a long time [31]. A randomized controlled study conducted by Jiao Lijuan et al. [32] found that compared with ordinary dialysis methods, the combined treatment of hemoperfusion and hemodialysis can more effectively remove inflammatory factors, alleviate the microinflammatory response in end-stage patients, and have a relatively small impact on the oxidative stress response.

5. Current Situation of Traditional Chinese Medicine Research

In “Huangdi Nei Jing”, it is stated that: “The kidney is the gateway of the stomach. If the gateway is not functioning properly, water will accumulate accordingly. That is, if the qi transformation function of the kidney fails, the gateway will be blocked, and water will accumulate in the body, resulting in water retention in the body, manifested as edema of the limbs and eyelids, chest tightness, poor appetite, fullness and discomfort in the stomach, etc. The spleen and stomach are the foundation of acquired constitution and the hub for the ascending and descending of qi. When the two complement each other in terms of dryness and dampness, the transportation and transformation of water fluids can proceed normally, and the refined substances of water and grains can be distributed properly. If one is old and physically weak, has an unregulated diet, overindulges in physical labor or sexual desires, or suffers from a long-term illness, etc., it will lead to the deficiency of qi, blood, yin and yang of the internal organs, deficiency of the spleen and kidney. The kidney fails to perform its qi transformation function, and the spleen fails to transport and transform properly. There will be an obstacle in the distribution of body fluids, and water will gather into dampness. When it congeals, it becomes phlegm. And phlegm-dampness will further entrap the spleen. If phlegm-dampness persists for a long time and generates heat, turbid toxins will be produced. When it enters the collaterals, blood stasis will occur. There is a mixture of deficiency and excess, and the course of the disease is lingering and difficult to cure. Pathological products such as “phlegm-dampness, turbid toxins, and blood stasis” are in line with the persistent, low-level, and mild inflammatory response in chronic kidney disease. Therefore, the pathogenesis of the microinflammatory state is deficiency in the root and excess in the surface, with deficiency of the spleen and kidney as the root and “phlegm-dampness, turbid toxins, blood stasis”, etc. as the excess in the surface. Among them, blood stasis is a key factor leading to the occurrence, development, and aggravation of CKD, running through the entire course of the disease [33]. After the generation of blood stasis, it accumulates and does not resolve, remaining in the spleen, kidney, and all parts of the body, causing the dysfunction of the internal organs and the disorder of body fluid metabolism. Therefore, the body shows the syndrome of dampness-induced blood stasis and the intermingling of dampness and blood stasis. The core pathogenesis of chronic inflammation is mostly the accumulation of masses due to blood stasis and phlegm coagulation [34].

Li Liguo and others [35] studied 120 patients with chronic kidney disease at stages 2-4 and found that after 8 weeks of treating chronic kidney disease with Shenyan Kangfu Tablets combined with Alprostadil: the levels of IL-6, IL-18, and CRP were significantly lower than before the treatment. Ai Sinan and others [36] found that when treating CKD with Niaoduqing Granules, it mainly acts on signaling pathways such as Hypoxia-inducible factor-1 (HIF-1), TNF-α, Phosphatidylinositol 3-kinase/Protein kinase B (PI3K/Akt), etc., and improves renal function by improving hypoxia, reducing inflammation, and protecting renal tubular epithelial cells and other approaches. Yang Mei and others [37] found through research that deficiency of the spleen and kidney is the most common deficiency syndrome type in CKD patients with a microinflammatory state, and it is also the internal basis for the onset of the disease. Among the excess syndromes, the syndromes of water-dampness and blood stasis are relatively common. Zhu Yunyun and others [38] found that most patients with microinflammation have a red tongue body. Therefore, they believe that the pathogenesis of the microinflammatory state of chronic kidney disease in traditional Chinese medicine should be deficiency of the

spleen and kidney and internal accumulation of heat toxins, and they propose to treat the microinflammatory state of CKD from the perspective of “fire and heat”. Using drugs for clearing heat and detoxifying often achieves good results. Moreover, dampness and blood stasis have the characteristics of viscosity and stubbornness, which are also similar to the pathological characteristics of the microinflammatory state that are difficult to completely cure and prone to recurrence.

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