DOI: 10.53469/jcmp.2025.07(07).38

Experience of Traditional Chinese and Western Medicine in the Treatment of IgA Nephropathy

Fuyu Chen[1], Xinbo Yang[2,*]

[1]Shaanxi University of Chinese medicine, Xianyang 712046, Shaanxi, China
[2]The Second Affiliated Hospital of Shaanxi University of Chinese medicine, Xianyang 712046, Shaanxi, China
**Correspondence Author*

Abstract: ***IgA nephropathy (hereinafter referred to as IgAN) is the most common primary glomerular disease in the world at present. Its pathogenesis is not clear and its clinical symptoms are diverse. The treatment is mainly glucocorticoids, immunosuppressants, antihypertensive and anticoagulant treatment. There is no IgAN disease in traditional Chinese medicine. According to its clinical manifestations, it can It is classified in the categories of "hematuria" and "edema". Most of the pathogenesis is based on the deficient root and excessive branch, in the process of the disease, there are both stasis blood pattern, wind evil and dampness-heat and other pathogenic factors. It also puts forward the treatment principles such as supporting vital Qiand expelling evils, activating blood to resolve stasis, clearing heat and eliminating dampness, benefiting qi and nourishing yin, paying attention to the overall concept, putting people-oriented, treating both the symptoms and root and branch, and improving the prognosis and long-term survival quality of patients. It has significant advantages in terms of quantity. Therefore, this article aims to briefly discuss the clinical experience of traditional Chinese and Western medicine treatment of IgAN and provide reference for clinical treatment of IgAN.***

Keywords: IgAnephropathy, Glucocorticoids, Immunosuppressant, Traditional Chinese and drugs, Cause of diseased and pathogenesis**.**

1. Introduction

IgAN, a common kidney disease first described and published in 1968 by Begger and Hinglais, is also known as Begger's disease [1]. Its main clinical manifestations include episodic gross hematuria, microscopic hematuria with or without asymptomatic proteinuria, proteinuria, hypertension, and renal dysfunction. The pathogenesis remains unclear, though it may be associated with infections. Abnormal immune function and dysregulated secretion of cytokines and inflammatory mediators might contribute to IgAN development. Immunofluorescence studies primarily show deposition of IgA and C3 in the mesangial zone, often accompanied by mesangial cell proliferation and stromal dilation [2]. The "four sequential hits" [3] is currently widely accepted. The incidence varies regionally, reaching 40%-50% in Asia-Pacific regions, and it frequently progresses to end-stage renal disease (ESRD) [4]. While IgAN can occur at any age, it predominantly affects young adult males aged 20-30 [5]. Treatment mainly focuses on symptomatic relief or measures to reduce non-specific kidney damage [6]. However, recent research indicates that only a small percentage of patients achieve significant recovery. In Asia-Pacific regions, IgAN is one of the primary glomerular diseases leading to uremia, ultimately progressing to ESRD [7]. Given the significant variations in clinical manifestations, pathological changes, and prognoses of IgAN, its treatment approaches also differ substantially. The primary clinical principles focus on blood pressure control and infection management, with therapeutic strategies mainly involving anti-inflammatory agents, immunosuppressants, anticoagulants, and antihypertensives. These measures aim to reduce inflammatory responses, regulate immune dysregulation, and prevent thrombosis. However, their effectiveness in improving patients' long-term quality of life remains limited. Traditional Chinese Medicine (TCM), however, demonstrates distinct advantages in this regard. By adopting a disease mechanism-based approach and implementing staged treatment plans, TCM has achieved remarkable efficacy in symptom relief and delaying disease progression.

2. Western Medicine Treatment

2.1 Renin-angiotensin System Inhibitors (RASIs)

Renin-angiotensin receptor blockers (RASIs) are primarily divided into two categories: Angiotensin-converting enzyme inhibitors (ACEIs) and Angiotensin II receptor blockers (ARBs). These medications act on the Renin-angiotensin-aldosterone system (RASS) by inhibiting processes like angiotensin-induced blood pressure elevation, thereby controlling blood pressure and reducing glomerular mesangial cell damage. Cao Hongna's study on the efficacy of telmisartan in primary IgA nephropathy demonstrated that this medication can improve glomerular filtration function and enhance nutritional status by antagonizing RAS activation, which reduces oxidative stress markers and prevents ischemia-reperfusion injury-induced renal unit or glomerular damage [8]. However, RASIs may increase risks of hyperkalemia, renal impairment, and hypotension [9].

2.2 Glucocorticoids (GC)

Glucocorticoids (GC) exhibit potent anti-inflammatory effects, capable of modulating inflammatory cell activity, enhancing immune regulation, and improving renal hemodynamics. They demonstrate efficacy in reducing proteinuria, preserving renal function, and alleviating or delaying renal tissue damag [10]. Commonly used GCs in clinical practice include prednisone methylprednisolone, and others. The involvement of immune dysregulation and autoimmune responses in the pathogenesis of IgAN suggests that GCs may confer therapeutic benefits for this disease [11].

In a clinical observation study evaluating the use of GC for IgAN treatment [12], showed that compared to conventional therapy, the glucocorticoid-adjunct therapy group demonstrated superior efficacy, lower 24-hour urinary protein

quantification, reduced serum creatinine (Scr), and shorter duration of 24-hour urinary protein <0.3g compared to conventional treatment. A systematic review of GC monotherapy for IgAN indicated that glucocorticoids significantly reduced the risk of developing end-stage renal disease (ESRD) in patients with IgAN. However, it should be noted that only high-dose glucocorticoids effectively lowered this risk, possibly due to insufficient immune suppression effects at low doses. While glucocorticoid therapy for IgAN demonstrates therapeutic benefits, cautious application is still required to minimize adverse events [13]. In a clinical study analyzing glucocorticoid intervention for IgA nephropathy, subgroup analysis of glucocorticoid doses revealed no significant difference in treatment outcomes between different GC doses combined with renin-angiotensin-sparing (RASI) inhibitors when urinary protein quantification <0.75g/d. However, when urinary protein >0.75g/d, the glucocorticoid group exhibited significantly better renal prognosis compared to the monotherapy RSA group. Additionally, conventional-dose glucocorticoids showed markedly higher infection risks than low-dose glucocorticoids, confirming glucocorticoids' clinical efficacy in improving renal prognosis for IgAN patients. When urinary protein>0. 75g/d or relevant pathological criteria are met, using low-dose glucocorticoids can further reduce adverse reactions [14].

These studies demonstrate that glucocorticoids (GCs) show significant efficacy in IgA nephropathy (IgAN) treatment, particularly in reducing urinary protein levels and delaying progression to end-stage renal disease (ESRD) in patients with impaired renal function. Even when administered at low doses, GCs maintain these benefits while minimizing adverse events. However, the lack of standardized dosing guidelines and patient eligibility criteria continues to spark debate. Given that systemic glucocorticoid therapy remains the primary approach, optimizing treatment strategies has become crucial to mitigate adverse effects [15,16]. The Global Kidney Disease Organization (KDIGO) recommends that glucocorticoid therapy should only be considered for high-risk patients (eGFR>30mL·min-11.73m^{-2}) with persistently elevated urinary protein levels (>1g/d) after three months of optimized therapy, emphasizing that individualized risk assessment is essential for clinical decision-making.

2.3 Immunosuppressants

The pathophysiology of IgAN primarily involves deposition of immune complexes in the glomerular basement membrane zone, which may ultimately lead to glomerular lesions and interstitial fibrosis. Although evidence-based support remains limited, numerous clinical studies have demonstrated that immunosuppressants serve as effective therapeutic agents for IgAN. The KDIGO Guidelines specifically recommend [17] therapy for patients with IgAN accompanied by crescent formation and rapid renal function decline. Commonly used immunosuppressive agents in clinical practice include Cyclophosphamide (CTX), Facrolimus (FK506), Cyclosporine A (CsA), Mycophenolate mofetil (MMF), and Rituximab (RTX) among others [18].

Cyclophosphamide (CTX), a cytotoxic immunosuppressant, inhibits immunoglobulin secretion, B cell differentiation, and T cell activity while reducing inflammatory factor release, thereby alleviating systemic inflammation [19]. Clinical observations by Wang Mengping et al. using adequate doses of glucocorticoids (GC) and CTX for IgAN treatment demonstrated the efficacy of CTX [20,21]. However, Su's study revealed significant differences in cyclophosphamide's effectiveness across varying degrees of hematuria. As a traditional immunosuppressant, CTX's therapeutic effects on IgAN have been confirmed by researchers [22].

Mycophenolate Mofeti (MMF), a highly selective immunosuppressant that acts on lymphocytes [23], has shown significant efficacy in treating IgAN when combined with low-dose glucocorticoids (GC). A clinical analysis demonstrated that MMF-GC therapy significantly outperformed GC monotherapy in alleviating IgAN-related symptoms, with marked improvements in renal function and proteinuria [24]. Feng Shengmin's experimental results further confirmed this finding [25,26]. Clinical trials comparing CTX and MMF revealed no substantial difference in therapeutic outcomes between the two agents, though MMF exhibited notably lower incidence of adverse reactions compared to CTX, demonstrating superior safety profile [27]. However, studies have noted significant ethnic variations in MMF efficacy, with favorable results observed exclusively in Asian populations [28].

Ciclosporine A (CsA) and tacrolimus (FK506) are representative calcineurin inhibitors. Numerous studies have demonstrated that the combination of CsA with corticosteroids in the treatment of IgAN outperforms monotherapy with steroids in alleviating proteinuria and improving serum albumin levels [29-32]. Li Zan's serological studies showed that combining FK506 with low-dose glucocorticoids significantly reduced IL-17, IL-23, and VEGF levels in IgAN patients, which play crucial roles in the immune response during disease onset [33]. Wang Jiang's research also reached similar conclusions [34]. Although the combination of tacrolimus with low-dose steroids demonstrates significant clinical efficacy, studies indicate that FK506 monotherapy or FK506 plus steroids in the treatment of IgAN patients with mild-to-moderate renal insufficiency not only fails to provide clear benefits in reducing proteinuria or preserving renal function but may even elevate serum creatinine levels [35]. This is likely attributed to the nephrotoxic effects of FK506 [33].

Rituximab (RTX), a chimeric monoclonal antibody that specifically binds to CD20 and mediates B cell lysis, has seen limited research on its application in IgAN treatment. A long-term follow-up study of PTX therapy for adult-onset minimal change nephropathy and membranous nephropathy conducted abroad demonstrated that PTX effectively alleviated proteinuria with low recurrence rates [36]. Similar findings have been reported domestically, with evidence showing better remission rates for IgAN patients with minimal change nephropathy compared to those with membranous nephropathy [37].

In summary, numerous domestic and international retrospective studies indicate that immunosuppressants can effectively slow renal function deterioration in IgAN patients with concurrent renal insufficiency. However, the incidence of adverse events associated with immunosuppressive therapy

has significantly increased in these patients [38]. Although immunosuppressants demonstrate notable therapeutic efficacy in treating IgAN, a retrospective study highlighted that eGFR and urinary protein levels are independent predictors of treatment outcomes. Notably, only eGFR independently predicts renal prognosis [39]. The evidence supporting the effectiveness of immunosuppressive therapy for IgAN and its long-term prognostic value still require further investigation.

2.4 Other Effective Drugs

2.4.1 Hydroxychloroquine (HCQ)

Hydroxychloroquine (HCQ), a cornerstone drug in rheumatology and immunology, demonstrates significant efficacy in modulating immune responses. It effectively alleviates proteinuria while reducing urinary IL-6 and TNF-α levels—both of which are associated with renal fibrosis [40]. Studies have demonstrated that hydroxychloroquine (HCQ) can alleviate mesangial cell proliferation, inhibit inflammatory cell infiltration, and reduce fibrosis severity, along with significantly decreasing the deposition density of IgA immune complexes, thereby mitigating renal injury in rats [41]. However, the long-term therapeutic benefits and renal protective effects of HCQ remain subjects of ongoing debate.

2.4.2 Treatment based on Gut-kidney axis theory

IgA is extensively present in the intestinal mucosal immune system. Therefore, gut microbiota imbalance partially influences the pathogenesis of IgA nephropathy. Chronic bacterial infections and dysbiosis accelerate excessive IgA production [42]. Consequently, recent studies have focused on regulating gut microbiota to treat IgAN. Drugs developed based on the entero-Kidney axis theory include modified corticosteroid agents, B-cell immunopathology-targeted therapies, complement and lectin pathway-targeting drugs, as well as angiotensin and endothelin-targeting therapies. Budesonide Release Capsules (TRF-Budesonide), a novel intraintestinal targeted release formulation, can exert anti-inflammatory and immunosuppressive effects on the intestinal mucosal immune system locally [43]. Most of these drugs remain in preclinical or early clinical trial stages, and their safety and efficacy for widespread clinical application still require further validation.

3. Traditional Chinese Medicine Research

3.1 Ancient Books

Hematuria, one of the most prominent clinical manifestations of IgAN, has been documented in ancient Chinese medical texts. The classic Jin Gui Yao Lue first coined the term "urinary bleeding," while later editions like Huang Di Nei Jing referred to it as "bloody urine". The Xue Lun Zheng explains that residual heat from the heart channel heat transmitting to the small intestine, whereas liver heat resides in the blood chamber, which is the pathogenesis behind hematuria. Similarly, Zhu Bing Yuan Hou Luns stated: "The heart governs blood and interacts with the small intestine. When heat accumulates in the heart affecting the small intestine, hematuria occurs." Furthermore, Danxi Xinfa differentiated hematuria from strangury syndrome by noting: "Bloody urine with pain is strangury; without pain, it is hematuria. These historical records demonstrate that traditional Chinese medicine (TCM) has long analyzed hematuria symptoms. Through accumulated case studies and evolving TCM systems, a systematic understanding and theoretical framework have developed, significantly influencing modern integrated TCM-Western medicine approaches in managing IgAN.

3.2 Etiology and Pathogenesis

Traditional Chinese Medicine (TCM) posits that IgAN primarily manifests as a syndrome of fundamental deficiency with superficial excess, where the deficient root constitutes the primary pathogenic factor. Professor Wang Xiangsheng proposes a six-channel differentiation approach: Initial heat pathogens first invade the Taiyang channel, causing blood-heat accumulation that leads to hematuria. When unresolved, this pathogen travels along meridians to the Shaoyang channel, establishing the disease's foundation. In excess syndromes, the Taiyang and Shaoyang channels dominate, while spleen-lung qi deficiency in the Taiyin channel obstructs dampness transformation, resulting in internal damp-heat accumulation. Conversely, unresolved Taiyin pathogen penetrates to Shaoyin, damaging kidney yin and causing deficient heat to disturb blood vessels, making the Taiyin and Shaoyin channels predominant in deficiency syndromes [44]. Professor Fu Yi identifies congenital constitution as the root cause, supplemented by factors like excessive sexual activity, chronic fatigue, and qi-yin deficiency. This combination of deficiency and external pathogenic factors triggers disease onset, with congenital constitution being the primary cause. The pathogenesis involves deficient vital energy disturbing the kidneys, manifesting as renal network dysfunction and blood proteinuria [45]. Professor Chen Shunhe emphasizes that IgAN is not caused by deficiency, stasis, or latent pathogens, but rather by "heat" pathogens damaging renal networks, leading to essence leakage through hematuria and proteinuri [46]. Professor Lu Keda attributes significant causative factors to wind pathogen invasion and congenital constitution deficiencies [47]. Professor Zhu Caifeng suggests that IgAN's pathogenesis originates from kidney qi deficiency and impaired renal network nourishment, with rheumatic pathogen, blood stasis, and turbid toxins constituting the exterior manifestations. The failure of the Pumen (gate of vital energy) to maintain its function represents the deficiency aspect, applying the theory that "the Pumen governs all five zang organs" for treatment [48].

3.3 Traditional Chinese Medicine Treatment

3.3.1 Treatment from the theory of "Deficiency and Excess"

The Zhu Bing Yuan Hou Luns states: "Wind-water pathologies arise from spleen-kidney qi deficiency. Kidney fatigue leads to deficiency, which manifests as sweating. When sweating encounters wind, internal pathogens invade the kidneys. Since spleen deficiency fails to control water retention, fluid leakage occurs in the skin, where it interacts with rheumatic pathogens, hence termed wind-water

syndrome." Professor Ren Jixue posits that IgAN's pathogenesis involves kidney deficiency and spleen insufficiency, with latent pathogenic factors in the kidneys being the critical trigger. Treatment strategies focus on kidney-strengthening and spleen-tonifying therapies, eliminating latent pathogens through staged treatment addressing both symptoms and root causes [49]. Professor Fu Yi identifies congenital constitution weakness combined with external pathogen invasion as the fundamental cause of IgAN, characterized by vital energy deficiency and pathogenic factors disturbing the kidneys. Treatment follows the principle of "tonifying deficiency and purging excess," prioritizing pathogen elimination during acute phases caused by damp-heat or blood stasis, while chronic persistent cases manifest qi-yin deficiency syndrome treated with modified Ginseng-Astragalus-Digested Rehmannia Decoction [45]. Analysis of Xiong Guoliang's medication practices reveals a complementary approach: Astragalus, Cornus officinalis, and Rehmannia nourish liver-kidney functions and replenish yin-energy, aiming to strengthen deficiency and support vital energy. Meanwhile, Salvia miltiorrhiza, Cicada slough, and Imperata root activate blood circulation, resolve stasis, clear heat, and eliminate dampness—effectively purging excess—forming a dual-supplement and purgation regimen balancing deficiency and excess [50].

3.3.2 Treatment from the perspective of "blood stasis"

The XuezhengLun states: "Blood stasis transforms into fluid retention, manifesting as edema—a blood disorder accompanied by water retention." Clinical practice has shown that blood stasis persists throughout the entire disease process of IgAN, aligning with research on related syndromes [51]. Therefore, blood-activating and stasis-resolving medications should be administered throughout treatment. Professor Li Ping's experience in treating chronic kidney disease reveals that hematuria in IgAN nephropathy is rooted in yin deficiency with turbid toxins and blood stasis as secondary manifestations., the modified Xiao Ji Yin Zi formula demonstrated remarkable efficacy [52]. Wang Qingqing innovatively incorporated moutan, white reed rhizome, and corydalis charcoal into her modified Xiaochaihu Tang formula for sore throat, effectively stopping bleeding without leaving residual stasis [53]. Professor Xiang Guangsheng's clinical application of a cooling-blood and stasis-resolving formula combined with conventional Western medicine showed significantly better outcomes than monotherapy in alleviating basic symptoms and regulating inflammatory factors in IgAN patients [54].

3.3.3 Treatment from the theory of "dampness-heat"

Zhu Danxi posited that "among the six qi (wind, dampness, heat, phlegm, and fire), dampness-heat causes disease in 80-90% of cases." Studies indicate that dampness-heat syndrome accounts for approximately half of IgAN pathotypes [55], aligning with Professor Zhu's clinical philosophy. Professor Liu Baohou suggests that persistent proteinuria in IgAN is closely linked to spleen deficiency with dampness accumulation. When dampness-heat pathogens obstruct kidney function, they impair the kidneys' regulatory capacity, leading to excretion of vital essence through urine and subsequent proteinuria formation. In clinical practice, he often combines heat-clearing and dampness-eliminating herbs like mulberry leaves, honeysuckle flower, and platycodon root [56]. Professor Qi Airong emphasizes location-specific differentiation when treating water-damp pathogens in IgAN, prescribing appropriate diuretic herbs based on affected areas while incorporating wind-dispelling agents to combat dampness [57]. Professor Chen Shunhe and colleagues propose a "heat" theory for IgAN treatment: during acute phases, modified Xiaoji Yinzi and Yinqiao San are used to clear heat, resolve exterior patterns, and drain dampness; chronic progression stages employ Shengjiang San to purge interior heat, supplemented by yin-nourishing herbs for comprehensive treatment, with variations of Yinqiao San as needed [47].

3.3.4 Treatment from the perspective of "wind evil"

The Suwen: Qi Jue Lun states: "The kidney's Foot-Shaoyin pulse originates below the little finger, runs diagonally along the sole, emerges beneath the sacrum, follows the inner ankle, and enters the heel. . . It then enters the lungs, traverses the throat, and passes between the tongue roots. This meridian governs kidney-related disorders such as dry mouth, sore throat, swollen throat, shortness of breath, belching, and pain." The meridian pathways elucidate the relationship between the throat, lungs, and kidneys. External pathogens invade the lung system through the throat and disrupt kidney meridians via these pathways. Professor Wang Bokui posits that IgAN fundamentally stems from wind-heat disturbing the kidneys. The rapid clinical progression of IgAN aligns with pathogenic characteristics of "wind dominance leading to movement" and "wind's tendency to move and change frequently." Patients with inherent weakness develop diseases when internal and external winds combine. Treatment should emphasize the rational use of heat-clearing and wind-eliminating herbs [58]. Professor Zhang Yu contends that wind pathogens permeate the entire IgAN pathogenesis. Constitutional weakness combined with internalized five pathogens transforms into internal wind over time, followed by external wind invasion triggering disease. As wind pathogens often accompany dampness and stasis, Master Zhang proposed multiple wind-dispelling methods including "eliminating wind, extinguishing wind, and resisting wind" [59]. Professor Tian Yun et al. linked IgAN to mucosal immunity, which resembles wind pathogens. They recommended specialized use of wind-dispelling and exterior-releasing herbs, providing a sustainable clinical pathway [60].

4. Conclusion

In summary, the pathogenesis of IgAN remains unclear. Western medical treatment for IgAN primarily focuses on antihypertensive therapy and symptomatic management such as reducing proteinuria and hematuria. Given the immunopathological changes associated with IgAN, the use of hormones and immunosuppressants has become common. Although these approaches have achieved some therapeutic effects, their long-term prognosis and adverse reaction rates remain unpredictable, necessitating further clinical research. Traditional Chinese medicine (TCM) treatment for IgAN has demonstrated remarkable success in numerous clinical reports, particularly when combined with Western medicine. This integrated approach shows significant efficacy in improving

renal function, reducing proteinuria and hematuria, mitigating side effects of Western treatments, and enhancing patients 'quality of life. Despite these achievements, challenges persist in TCM-Western medicine collaboration, including differences in theoretical frameworks, difficulties in integrating both systems, lack of unified treatment guidelines, complexities in medication selection and efficacy evaluation, and unresolved safety concerns during treatment. The author's discussion on clinical approaches to IgAN aims to provide insights for advancing research and exploration in this field.

References

[1] BERGER J, HINGLAIS N. Intercapillary deposits of IgA-IgG [J]. J Urol Nephrol (Paris), 1968, 74(9): 694-695.

[2] Shi Sheng, Jiang Xiao, Xu Xingxin et al. Clinicopathologic features and risk factors of primary IgA nephropathy with foot process effacement [J]. Journal of Anhui Medical University, 2023, 58(12): 2119-2123.

[3] Pattrapornpisut P, Avila-Casado C, Reich HN. IgA Nephropathy: Core Curriculum 2021. Am J Kidney Dis. 2021 Sep;78(3):429-441.

[4] Zhao Fanbo, Ji Yue, Qi Xin et al. Progress in the Pathogenesis, Diagnosis and Treatment of IgA Nephropathy in TCM and Western Medicine [J]. Acta Chinese Medicine and Pharmacology, 2023, 51 (09): 97-103.

[5] Zhang Pengwei, Cheng Xia, Cheng Lanlan et al. Advances in the study of sodium-glucose cotransporter-2 inhibitors for kidney protection in IgA nephropathy [J]. Clinical Focus, 2022, 37(11): 1053-1056.

[6] Floege J, Rauen T, Tang SCW. Current treatment of IgA nephropathy. Semin Immunopathol. 2021 Oct; 43(5): 717-728.

[7] Gao Xin, Tian Yun. Progress in TCM Syndrome Differentiation and Treatment of IgA Nephropathy [J]. Journal of Liaoning University of Traditional Chinese Medicine, 2018, 20(12):73-75.

[8] Cao Hongna. Effects of Telmisartan on Primary IgA Nephropathy and Its Analysis of Renal Function Improvement [J]. Jiangxi Medical Journal, 2021, 56(08):1214-1217.

[9] RAQUEL, ESTERAS, VANESSA M, et al. Combination use of medicines from two classes of renin -angiotensin system blocking agents: risk of hyperkalemia, hypotension, and impaired renal function [J]. Ther Adv Drug Saf, 2015, 6(4):166-176.

[10] Peng Xiaojie, Zheng Weimin, Fu Rui et al. Systematic Evaluation of Tacrolimus in IgA Nephropathy: Efficacy on Proteinuria and Risk of Infections [J]. Jiangxi Medical Journal, 2019, 54(7):761-765.

[11] Prognostic Analysis of Ig A Nephropathy Patients with High Risk of CKD Progression Treated with Glucocorticoid: A Retrospective Study [J]. Chinese General Practice, 2024, 27(06):692-698.

[12] Dongfang Yuehua. Observation and Evaluation of Glucocorticoid Therapy in IgA Nephropathy [J]. Electronic Journal of Clinical Medical Literature, 2020, 7(36):150.

[13] Liu Kaixiang, Zhan Zhipeng, Xie Xisheng. Advances in the Diagnosis and Treatment of IgA Nephropathy [J]. Chinese Journal of Integrated Traditional and Western Nephrology, 2018, 19(02):173-176.

[14] Zhong Yongzhong, Fei Dan, LinYi et al. Clinical Study of Glucocorticoid Intervention in IgA Nephropathy Based on Real-world Retrospective Analysis [J]. Chinese Journal of Integrated Traditional and Western Nephrology, 2023, 24(03):213-217.

[15] Jin Yue. Advances in Diagnosis and Treatment of IgA Nephropathy [J]. Heilongjiang Medical Journal, 2022, 46(19): 2427-2430.

[16] Huang Jiali, Yu Shuwen, Yu Dongrong et al. Advances in Glucocorticoid Therapy for IgA Nephropathy [J]. Chinese Journal of Integrated Traditional and Western Nephrology, 2018, 19(05):430-432.

[17] Li Zhiheng, Wen Qing, Cai Xueni et al. Research Advances in the Treatment of IgA Nephropathy [J]. Journal of Xinxiang Medical University, 2023, 40(03): 295-300.

[18] Zheng Zhuolong, Lin Gang, Luo Mianna et al. Research Progress on Immunosuppressive Therapy for IgA Nephropathy in Adults [J]. Journal of Medical Information, 2023, 36(14):183-187.

[19] Wang Mengping, Sun Jianchao, Jin Chang et al. Efficacy and Influencing Factors of Adequate Prednisone Combined with Cyclophosphamide Therapy in the Treatment of IgA Nephropathy [J]. Herald of Medicine, 2023, 42(08):1172-1177.

[20] Su Jili, Zhou Xiaoshuang, Fan Xiuxi et al. Efficacy of cyclophosphamide combined with glucocorticoids therapy with different degrees of hematuria IgA nephropathy [J]. Chinese Journal of General Practice, 2023, 21(07):1105-1108.

[21] Ni Zhaohui, Li Shu. Immunosuppressive Therapy for Patients with IgA Nephropathy [J]. Journal of Internal Medicine Concepts & Practice, 2016, 11(03):150-155.

[22] Zhu Mingzhi, Zhong Ruiqiong, Tong Yu. Analysis on effects of different treatment regimens for patients with IgA nephropathy [J]. Journal of Clinical Nephrology, 2019, 19(07):512-515.

[23] Tang Fei, Tao Cai, Wan QIjun. Recent Research Progress on Mycophenolate Mofetil (MMF) Therapy for IgA Nephropathy [J]. Chinese Journal of Integrated Traditional and Western Nephrology, 2019, 20(01): 87-89.

[24] Zhan Ju. Efficacy Analysis of Mycophenolate Mofetil Combined with Low-Dose Glucocorticoids in IgA Nephropathy Patients [J]. Harbin Medical Journal, 2016, 36(04):455-456.

[25] Feng Shengmin. Clinical Efficacy of Mycophenolate Mofetil Combined with Low-Dose Prednisone Acetate in IgA Nephropathy Patients [J]. The Journal of Medical Theory and Practice, 2020, 33(16):2664-2666.

[26] Liu Chunhua, Yang Dingping. Clinical efficacy of mycophenolate mofetil plus low-dose glucocorticoid for IgA nephropathy with crescent formation [J]. Journal of Clinical Nephrology, 2023, 23(05):363-368.

[27] Zhu Mingzhi, Zhong Ruiqiong, Tong Yu. Analysis on effects of different treatment regimens for patients with IgA nephropathy [J]. Journal of Clinical Nephrology, 2019, 19(07):512-515.

[28] LAFAYETTE R A, CANETTA P A, ROVIN B H, et al. A randomized, controlled trial of rituximab in IgA nephropathy with proteinuria and renal dysfunction [J]. J Am Soc Nephrol, 2016, 28(4):1306-1313.
[29] Dong Xiaohong, Chen Wenjun, Miu Jinglong. Therapeutic Efficacy of Cyclosporine A Combined with Prednisone in Progressive IgA Nephropathy [J]. Contemporary Medical Symposium, 2016, 14(24): 52-53.
[30] Suo Bincheng. Clinical Effect Observation of Cyclosporin A Combined with Prednisone in the Treatment of Progressive IgA Nephropathy [J]. Chinese and Foreign Medical Research, 2017, 15(09):6-7.
[31] Tian Yan, Zeng Chunlan. To Compare the Efficacy of Cyclosporine A and Cyclophosphamide Combined with Hormone in Severe IgA Nephropathy [J]. Chinese and Foreign Medical Research, 2021, 19(18):143-145.
[32] Wang Xiaowen, Wang Anwen, Zhang Quanquan et al. Research of Cyclosporine A Combined with Union/Low-dose Prednisone on the Treatment of Progressive IgA Nephropathy [J]. Chinese Journal of Pharmacoepidemiology, 2017, 26(03):168-170+218.
[33] Li Zan, Wang Fengping. Therapeutic effects of tacrolimus combined with low-dose glucocorticoid on IgA nephropathy and the changes of IL-17 and IL-23 levels [J]. Hebei Medical Journal, 2020, 42(11): 1681-1683+1687.
[34] Wang Jiang, Xu Gaosi. Clinical Efficacy Analysis of Tacrolimus in IgA Nephropathy Patients [J]. Jiangxi Medical Journal, 2023, 58(07):815-818+850.
[35] DU B, JIA Y, ZHOU W, et al. Efficacy and safety of mycophenolatemofetil in patients with IgA nephropathy: an update Meta analysis [J]. BMC Nephrol, 2017, 18(1): 245-255.
[36] Bruchfeld A, Benedek S, Hilderma n M, et al. Rituximab for minimal chang e disease in adults: long-term follow-up [J]. Nephrol Dial Transplant, 2014, 29(4): 851-856.
[37] Zhao Ying, Zhang Lijie, Ma Shuang et al. Efficacy and safety analysis of rituximab in the treatment of primary IgA nephropathy [J]. Practical Pharmacy and Clinical Remedies, 2023, 26(06):516-521.
[38] Jia Qin. Efficacy and safety of immunosuppressive therapy forIgA nephropathy with stage 3 or 4 chronic kidneydisease [D]. Air Force Medical University of the Chinese People's Liberation Army, 2022.
[39] Rui Yuanfan, Tang Lin. Effect and prognosis of immunosuppressive therapy of IgA nephropathy: a report of 97cases [J]. Anhui Medical and Pharmaceutical Journal, 2021, 25(07):1302-1307.
[40] Wang Chaochao, Lin Yongqiang, Cai Xiaoqiao et al. Effect of hydroxychloroquine on serum and urinary IL-6 and TNF-a levels in patients with IgA nephropathy [J]. Zhejiang Medical Journal, 2023, 45(05):514-516+522.
[41] Li You, Jiang Hong, Li Wei. Effect of hydroxychloroquine on renal pathology and Toll-like receptor 4 expression in a rat model of IgA nephropathy [J]. Journal of China Medical University, 2020, 49(11): 997-1001.
[42] Gao Hang, Zhao Ya Zhou, Liu Chunying. Treatment of IgA nephropathy based on the gut-kidney axis in traditional Chinese and Western medicine [J]. Modern Chinese Clinical Medicine, 2022, 29(02):72-76.
[43] Subinur Elken, Li Jing. Recent advances in targeted therapy of primary IgA nephropathy [J]. Journal of Clinical Nephrology, 2023, 23(01):74-78.
[44] Liu Na, Wang Xiangsheng, Zaho Xutao. Experience of Professor WANG Xiangsheng in Treating IgA Nephropathy with the Perspective of Six-Channel [J]. Clinical Journal of Traditional Chinese Medicine, 2018, 30(10):1802-1804.
[45] Liu Yongfang, Wang Ying. Chief Physician Fu Yi's Expertise in IgA Nephropathy Treatment [J]. Journal of Medical Information, 2020, 33(21):155-156.
[46] Chen Shunhe, Zheng Xiaojing, Qu Kai. Treatment of IgA Nephropathy from "Heat" [J]. Journal of Chengdu University of Traditional Chinese Medicine, 2021, 44(01): 43-46.
[47] Yang Dan, Fu Qianqian, Lu Keda. Lu Keda's Experience in Treating IgA Nephropathy [J]. Journal of Chengdu University of Traditional Chinese Medicine, 2022, 45(01):76-79.
[48] Yuan Mingyi, Zhu Caifeng, Bao Ziyang. Professor Zhu Caifeng's Application of 'The Anus Serving the Five Zang Organs' Theory in IgA Nephropathy Treatment [J]. Zhejiang Journal of Traditional Chinese Medicine, 2022, 57(02):92-93.
[49] Zou Di, Liu Yanhua, Zhang Shoulin et al. Master Traditional Chinese Medicine Physician Ren Jixue's Clinical Experience in Treating IgA Nephropathy: A National Heritage Perspective [J]. Journal of Li-shizhen Traditional Chinese Medicine, 2022, 33(05):1213-1215.
[50] Wan Peijun, Lu Lingfei, Xiong Guoliang. Analysis of Medication Rule of Xiong Guoliang for the Treatment of IgA Nephropathy [J]. Asia-Pacific Traditional Medicine, 2023, 19(07):157-162.
[51] Ye Lingyan, Wu Yuchi, Lin Qizhan. Study on Correlation Between Traditional Chinese Medicine Syndrome Types and Clinical Indicators in Patients with IgA Nephropathy [J]. Acta Chinese Medicine, 2024, 39(02): 421-427.
[52] Shen Zhengri, Liu Peng, Li Ping. Professor Li Ping's Clinical Experience in Treating Chronic Kidney Disease with Blood-Activating and Stasis-Resolving Therapy [J]. Chinese Journal of Integrated Traditional and Western Nephrology, 2023, 24(12):1044-1045.
[53] Wang Qingqing, Lu Yan, Tang Ying, et al. Treatment Outcome of TCM & WM Therapy on IgA Nephropathy with Chaiqin Decoction [J]. Chinese Journal of Integrated Traditional and Western Nephrology, 2020, 21(04): 309-312.
[54] Xiang Guangsheng, Tang Chensi, Lu Boyu et al. Effects of Liangxue Sanyu decoction combined with conventional western medicine on hemorheology and inflammatory cytokine levels in patients with IgA nephropathy [J]. Shaanxi Journal of Traditional Chinese Medicine, 2024, 45(01):63-66.
[55] Liu Yongfang, Chen Bangming, Wang Jinxaing et al. Distribution Characteristics of TCM Constitution Types and Excess Patterns in 240 Cases of IgA Nephropathy: A Clinical Study [J]. Chinese Journal of Integrated Traditional and Western Nephrology, 2017, 18(03): 238-239.
[56] Mi Panpan, Wang Xinqiang, Sun Nating et al. An analysis of famous TCM doctor Liu Baohou's medication rulesfor treating IgA nephropathy based on

TCM inheritance support system [J]. Clinical Journal of Chinese Medicine, 2021, 13(17):15-18.
[57] Zou Yanhong, Qi Airong. QI Ai-rong's Experience in Treating IgA Nephropathy from the Perspective of "Deficiency, Rheumatism and Stasis" [J]. Jiangxi Journal of Traditional Chinese Medicine, 2023, 54(10):41-43.
[58] Miao Linzhi, Wang Baokui. Discussion on pathogenic effect of pathogenic wind-heat on IgA nephropathy and diabetic nephropathy [J]. Beijing Journal of Traditional Chinese Medicine, 2023, 42(09):1018-1020.
[59] Ma Sijia, Zahng Yu. Zhang Yu′s Thinking and Methods in Treating IgA Nephropathy Proteinuria by Removing Wind Evil Factor [J]. Acta Chinese Medicine, 2022, 37(02):341-344.
[60] Tian Haoyi, Tian Yun. Treatment of IgA nephropathy from the wind pathogen [J]. China Medical Herald, 2022, 19(34):117-119+139.