

Clinical Characteristics and Efficacy of Individualized Treatment for IgA Nephropathy Combined with Metabolic Syndrome in Western Guangxi

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Abstract

Objective: To systematically analyze the epidemiological characteristics of metabolic syndrome in patients with IgA nephropathy in western Guangxi, and evaluate the efficacy of an individualized treatment regimen combining metabolic indicators and lifestyle interventions in protecting renal function.

Methods: A retrospective cohort study was conducted. A total of 142 patients with primary IgAN confirmed by renal biopsy at Baise People's Hospital between January 1, 2020 and December 31, 2025 were enrolled. Patients were stratified by MetS status and then divided into a conventional treatment group and an individualized treatment group according to actual treatment received, with 71 cases in each group. The conventional group received standard RAAS inhibitor plus glucocorticoid therapy. The individualized group additionally received targeted agents (SGLT2 inhibitors, atorvastatin calcium, bezafibrate, or amlodipine) based on metabolic abnormalities, plus structured lifestyle intervention. We reported the number and proportion of patients meeting the IDF MetS definition and compared baseline and outcome differences between patients with and without MetS. Primary outcomes included 1-year proteinuria remission rate, annual eGFR decline rate, and cardiovascular event rate. Propensity score analysis and multivariable adjustment, including meta-

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bolic variables, were performed to control confounding due to nonrandom treatment allocation. Multivariate Logistic regression identified independent prognostic factors, and an individualized decision-tree model was constructed. **Results:** Among 142 patients, 68 (47.9%) had MetS and 74 (52.1%) did not; MetS patients had higher baseline waist circumference, fasting glucose/HbA1c, triglycerides, lower HDL-C, heavier proteinuria, and lower eGFR (all $P < 0.05$). Baseline age, gender, eGFR, 24-h proteinuria, blood pressure, and BMI were comparable between groups (all $P > 0.05$). After propensity score matching, baseline metabolic parameters (waist circumference, fasting glucose/HbA1c, triglycerides, HDL-C) and the proportion meeting each MetS component were balanced (all $P > 0.05$). The 1-year complete proteinuria remission rate was 56.3% (40/71) in the individualized group vs. 35.2% (25/71) in the conventional group ($P = 0.012$). Annual eGFR decline was (2.8 ± 1.5) vs. (4.3 ± 2.1) mL/min/1.73 m²/year ($P < 0.001$). Cardiovascular event rates were 8.5% (6/71) vs. 18.3% (13/71) ($P = 0.048$). eGFR slope was estimated via linear mixed models using ≥ 3 serum creatinine measurements per patient (baseline, 6-month, 12-month), with multiple imputation for missing follow-up data. Multivariate Logistic regression showed that baseline proteinuria (OR = 0.42, 95% CI: 0.23 - 0.77), baseline eGFR (OR = 1.03, 95%CI: 1.01 - 1.05), baseline Oxford MEST-C T-stage, and lifestyle score (OR = 1.41, 95% CI: 1.10 - 1.81) were independent predictors of remission (all $P < 0.05$). The decision-tree model achieved 76.4% accuracy and AUC = 0.79 in internal validation. **Conclusion:** MetS comorbidity is common in IgAN patients in western Guangxi, and MetS is associated with worse renal prognosis and accelerates renal progression. An individualized strategy integrating metabolic-targeted therapy and structured lifestyle intervention significantly improves proteinuria remission, slows renal function decline, and reduces cardiovascular risk. This is a single-center retrospective study with internal validation only; claims regarding regional prevalence and strong promotion should be interpreted cautiously. The proposed model provides a practical pathway for precise CKD management in the region, with clinical reference value.

Keywords

IgA Nephropathy, Metabolic Syndrome, Individualized Treatment, Renal Function, Decision-Making Model, Western Guangxi

1. Introduction

IgA nephropathy (IgAN) is the most common primary glomerular disease in China, accounting for 30% - 40% of renal biopsies and a leading cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) in young adults [1]. Although some patients progress slowly, 20% - 40% develop ESRD within 10 - 20 years, imposing a heavy burden [2]. In recent decades, the prevalence of metabolic syndrome (MetS) among CKD patients has risen, emerging as an independent

risk factor for accelerated renal decline [3].

MetS is defined by central obesity, hypertension, hyperglycemia, and dyslipidemia. Underlying mechanisms include insulin resistance, chronic low-grade inflammation, oxidative stress, and lipotoxicity, promoting glomerular hypertension, mesangial proliferation, basement membrane thickening, and interstitial fibrosis [4]. In IgAN, MetS exacerbates proteinuria, poor blood pressure control, and faster eGFR decline, impairing treatment response and prognosis.

Western Guangxi is an ethnic minority region with high rates of high-salt, high-fat, high-carbohydrate diets, physical inactivity, and tobacco/alcohol use. Adult MetS prevalence ranges 18.7% - 22.3%, higher than the national average [5]. However, data on IgAN with MetS in this region remain limited [6]. Current IgAN management follows KDIGO guidelines (RAAS inhibitors + glucocorticoids) without systematic metabolic intervention, leading to suboptimal outcomes in some patients [7].

Precision medicine has advanced nephrology care. SGLT2 inhibitors and statins confer renal protection independent of glycemic or lipid effects, reducing proteinuria and slowing eGFR decline in non-diabetic CKD [8]. Integrating metabolic and lifestyle data into individualized strategies may improve outcomes.

This study analyzed six-year real-world data to characterize IgAN with MetS in western Guangxi, clarify the impact of MetS on renal prognosis, compare treatment efficacy, and identify prognostic factors, aiming to develop a practical individualized decision framework for regional CKD management.

2. Materials and Methods

2.1. Study Design

This was a retrospective cohort study based on a single-center electronic medical record system. Data were collected from patients with primary IgA nephropathy who were hospitalized and confirmed by renal biopsy in the Department of Nephrology of Baise People's Hospital and Youjiang Medical University for Nationalities between January 1, 2020 and December 31, 2025. The study protocol was reviewed and approved by the hospital's Ethics Review Committee (Batch No.: KY2025122505). All patient information was anonymized, and strict medical data confidentiality principles were followed. In view of the non-interventional retrospective nature of the study, informed consent was waived.

2.2. Study Subjects

A total of 142 eligible patients were included. Treatment allocation reflected routine clinical decisions based on metabolic severity, renal function, and comorbidities; propensity score analysis and multivariable adjustment controlled nonrandom confounding. Patients were divided into conventional ($n = 71$) and individualized ($n = 71$) groups.

Inclusion criteria: 1) age 18 - 70 years; 2) primary IgAN by renal biopsy (2017 KDIGO); 3) complete data including demographics, laboratory results, renal pa-

thology (Oxford MEST-C), and ≥ 12 -month follow-up.

Exclusion criteria: 1) secondary IgAN (e.g., Henoch-Schönlein purpura nephritis, cirrhosis-related glomerulopathy); 2) malignancy, NYHA III–IV heart failure, acute infection, or life-threatening systemic disease; 3) $>10\%$ missing key data (eGFR, 24-h proteinuria).

2.3. Diagnostic Criteria

IgAN diagnosis required mesangial proliferation (light microscopy), dominant IgA mesangial deposition (immunofluorescence), and electron-dense deposits (electron microscopy). Pathology severity was assessed by Oxford MEST-C, with focus on T-stage (tubular atrophy/interstitial fibrosis).

Metabolic syndrome was diagnosed according to the 2005 International Diabetes Federation (IDF) criteria: central obesity (waist circumference ≥ 90 cm in men, ≥ 80 cm in women) plus at least two of the following four indicators: 1) Systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg, or use of antihypertensive drugs; 2) Fasting blood glucose ≥ 5.6 mmol/L or confirmed diabetes mellitus; 3) Serum triglycerides ≥ 1.7 mmol/L; 4) High-density lipoprotein cholesterol (HDL-C) < 1.03 mmol/L in men, < 1.29 mmol/L in women.

2.4. Treatment Regimens

The conventional treatment group followed the current clinical pathway, mainly including valsartan (80 - 160 mg/d), an angiotensin receptor blocker (ARB), combined with short-term glucocorticoid therapy (prednisone 0.5 mg/kg/d, tapered and discontinued after 8 weeks), supplemented with symptomatic supportive measures such as blood pressure control and lipid regulation, but without systematic metabolic targeted intervention or standardized behavioral guidance.

The individualized treatment group received comprehensive interventions in addition to the above regimen:

Metabolic targeted medication: Fasting glucose ≥ 7.0 mmol/L or HbA1c $\geq 6.5\%$: dapagliflozin 10 mg/d [8]; triglycerides ≥ 2.26 mmol/L: bezafibrate 400 mg/d, atorvastatin calcium 20 mg/d if needed [9]; BP not controlled ($<130/80$ mmHg): amlodipine 5 - 10 mg/d. We recorded the number of patients receiving each agent: 28 SGLT2 inhibitor alone, 22 bezafibrate alone, 16 amlodipine alone, 5 combined two targeted drugs.

Structured lifestyle intervention: A 6-month intervention plan was jointly formulated by specialist dietitians and nursing teams, including a low-salt diet (sodium intake < 5 g/d), a low-fat diet (fat energy supply ratio $< 25\%$), regular moderate-intensity aerobic exercise (≥ 150 minutes per week, including brisk walking, swimming, cycling, etc.), smoking cessation and alcohol restriction advice, and psychological and emotional counseling. Implementation was supervised through regular outpatient and telephone follow-up [10].

2.5. Outcome Measures

Primary endpoints:

1) **Remission rate of proteinuria:** Defined as 24-hour urinary protein quantification reduced to <0.5 g and a decrease of $\geq 50\%$ from baseline after 1 year of treatment;

2) **Annual decline rate of eGFR:** eGFR calculated via CKD-EPI [11]. At least three serum creatinine measurements (baseline, 6-month, 12-month) per patient; linear mixed models estimated individual slopes with multiple imputation for missing data.

3) **Cardiovascular events:** Including angina pectoris acute attack, myocardial infarction, ischemic or hemorrhagic stroke, hospitalization for heart failure, etc.

Secondary endpoints: Baseline metabolic parameters (waist circumference, fasting glucose/HbA1c, triglycerides, HDL-C) and proportion meeting each MetS component; BP, fasting glucose, lipid targets; Oxford T-stage; adverse events (hypotension, hypoglycemia, elevated liver enzymes, myalgia).

2.6. Lifestyle Assessment

Lifestyle level was assessed using the self-developed *Chronic Kidney Disease Patients' Lifestyle Rating Scale*, which includes four dimensions: dietary structure, frequency and duration of physical activity, smoking and drinking habits, and sleep quality. Each dimension is scored 0 - 2.5 points, with a total score of 0 - 10 points. A higher score indicates a healthier lifestyle. Assessment was completed by trained research nurses at enrollment to ensure consistency and reliability; long-term monitoring can be combined with digital health tools in the future [12] [13].

2.7. Statistical Analysis

SPSS 30.0 and R 4.2.0 were used. Continuous data: mean $\pm$ SD, independent t-test; categorical data: n (%), χ^2 /Fisher's exact test. Spearman correlation; propensity score analysis balanced baseline metabolic and confounding variables; multivariate Logistic regression adjusted for baseline metabolic parameters and Oxford T-stage. Predictors of 1-year remission were identified. A decision-tree model was built (rpart package) with cross-validation. $P < 0.05$ was significant.

3. Results

3.1. Baseline Characteristics

142 patients: 62 male (43.7%), 80 female (56.3%), mean age 42.3 ± 10.5 years. MetS: 68 (47.9%), non-MetS: 74 (52.1%); MetS patients had higher waist circumference (92.5 ± 8.3 vs. 78.2 ± 7.6), fasting glucose (6.8 ± 1.2 vs. 5.1 ± 0.8), triglycerides (2.5 ± 0.9 vs. 1.3 ± 0.5), lower HDL-C (0.98 ± 0.21 vs. 1.35 ± 0.28), heavier 24-h proteinuria (3.5 ± 1.8 vs. 2.2 ± 1.4), lower eGFR (52.1 ± 19.5 vs. 63.8 ± 17.2) (all $P < 0.05$). Baseline age, gender, eGFR, proteinuria, BP, BMI were balanced (all $P > 0.05$). Baseline metabolic parameters and MetS component proportions were balanced (all $P > 0.05$); Oxford T-stage: T0 = 45, T1 = 62, T2 = 35 (Table 1).

Table 1. Baseline characteristics (n = 142).

Indicator	Conventional (n = 71)	Individualized (n = 71)	P
Age (years)	42.1 ± 10.8	42.5 ± 10.2	0.832
Males, n (%)	31 (43.7)	31 (43.7)	1.000
eGFR (mL/min/1.73 m ²)	58.4 ± 18.3	57.9 ± 19.1	0.854
24-h proteinuria (g)	2.8 ± 1.6	2.9 ± 1.7	0.712
SBP (mmHg)	138.5 ± 14.2	139.1 ± 13.8	0.789
DBP (mmHg)	86.3 ± 9.5	87.1 ± 8.9	0.583
BMI (kg/m ²)	25.6 ± 3.4	25.8 ± 3.6	0.701
Waist circumference (cm)	85.2 ± 9.1	84.8 ± 8.7	0.785
Fasting glucose (mmol/L)	5.9 ± 1.1	6.0 ± 1.2	0.621
Triglycerides (mmol/L)	1.8 ± 0.7	1.9 ± 0.8	0.543
HDL-C (mmol/L)	1.15 ± 0.25	1.13 ± 0.27	0.618
Oxford T-stage (T0/T1/T2)	22/31/18	23/31/17	0.957

3.2. Treatment Outcomes

Proteinuria remission: individualized 56.3% vs. conventional 35.2% (P = 0.012). Annual eGFR decline: 2.8 ± 1.5 vs. 4.3 ± 2.1 mL/min/1.73m²/year (P < 0.001). Cardiovascular events: 8.5% vs. 18.3% (P = 0.048). Adverse events: 22.5% vs. 26.8% (P = 0.532) (**Table 2**).

Table 2. Comparison of treatment outcomes between the two groups.

Index	Conventional Group	Individualized Group	P Value
Proteinuria remission (%)	35.2	56.3	0.012
Annual eGFR decline (mL/min/year)	4.3 ± 2.1	2.8 ± 1.5	<0.001
Cardiovascular events (%)	18.3	8.5	0.048
Adverse events (%)	22.5	26.8	0.532

3.3. Multivariate Analysis

Independent predictors of remission: baseline proteinuria (OR = 0.42, 95% CI: 0.23 - 0.77, P = 0.005), baseline eGFR (OR = 1.03, 95% CI: 1.01 - 1.05, P = 0.012), Oxford T-stage (OR = 0.78, 95% CI: 0.65 - 0.94, P = 0.008), lifestyle score (OR = 1.41, 95% CI: 1.10 - 1.81, P = 0.007). Better renal function, metabolic control, milder pathology, and healthier lifestyle predicted remission.

3.4. Construction of Decision-Making Model

Based on the above independent predictors, an individualized treatment decision tree model was constructed in this study. The model uses “baseline proteinuria level” as the root node for the first split: if the baseline proteinuria level ≤ 2 g/d, structured lifestyle intervention combined with basic RAAS inhibitor therapy is

prioritized; if the baseline proteinuria level > 2 g/d and the lifestyle score is <6 points, enhanced RAAS inhibition combined with SGLT2 inhibitor therapy is recommended; if the baseline proteinuria level > 2 g/d and complicated with hypertriglyceridemia, combined atorvastatin calcium for lipid regulation is suggested. The model achieved a prediction accuracy of 76.4% and an area under the ROC curve (AUC) of 0.79 in the internal validation set, showing good discriminative efficacy and clinical applicability.

4. Discussion

This six-year real-world study characterized IgAN with MetS in western Guangxi, confirming MetS as a key risk factor for renal deterioration, and demonstrated superior outcomes with individualized therapy. The regimen increased remission by 21.1%, slowed eGFR decline by 34.9%, and reduced cardiovascular risk by 53.6% without increasing adverse events.

Despite retrospective design, propensity score analysis balanced metabolic and confounding variables, and Oxford T-stage adjusted for pathology confounding, while baseline characteristics were comparable, enhancing real-world generalizability. Multivariate regression and decision-tree modeling identified key prognostic factors, informing future prospective trials.

The core contribution is a dual-support framework: metabolic targeting plus lifestyle intervention. Targeted agents (SGLT2 inhibitors, bezafibrate/atorvastatin, amlodipine) addressed hyperglycemia, hypertriglyceridemia, and resistant hypertension, shifting from generalized to precision care. We clarified bezafibrate's lipid-lowering and renoprotective role in hypertriglyceridemia. SGLT2 inhibitors reduced intraglomerular pressure, improved tubular oxygen balance, and exerted anti-inflammatory/antioxidant effects, slowing CKD progression independent of glycemia [8] [14].

Lifestyle score independently predicted remission (OR = 1.41), highlighting self-management. In western Guangxi, structured interventions improved metabolic control and adherence, supporting long-term prevention [10] [15]. The decision tree integrated lifestyle scores for practical, individualized care.

Limitations: single-center retrospective design, residual confounding despite adjustment; findings are regional and not generalizable nationwide; 1-year follow-up limits assessment of ESRD/survival; lifestyle assessment relied on questionnaires without objective monitoring; model lacks external validation.

5. Conclusion

MetS comorbidity is high in western Guangxi IgAN patients, with worse renal prognosis in MetS patients. Individualized therapy integrating metabolic targeting and structured lifestyle intervention improves proteinuria remission, slows renal decline, and reduces cardiovascular risk. This single-center retrospective study requires cautious interpretation of regional generalization claims, with only internal validation. The decision-tree model shows good predictive performance and clin-

ical utility, offering a replicable pathway for regional CKD management.

6. Limitations of the Study

Single-center, regional sample (predominantly Zhuang ethnicity); limited national generalizability; short follow-up; subjective lifestyle assessment; residual confounding from unmeasured metabolic variables; decision-tree model without external validation.

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Conflicts of Interest

All authors declare no economic or non-economic conflicts of interest.

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