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A Single-Center Retrospective Cohort Study on the Correlation between the Oxford Classification of IgA Nephropathy Based on Multimodal Pathological Features and Clinical Prognosis

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ABSTRACT

Objective: To explore the intrinsic relationship between the Oxford Classification (MEST-C) of IgA Nephropathy (IgAN) and patients' baseline clinical indicators, combined with multimodal pathological findings. Methods: Clinical and pathological data from 40 patients with primary IgAN diagnosed by renal biopsy at our hospital between April 2021 and December 2024 were retrospectively collected. Pathological presentations, including the Oxford Classification, immunofluorescence deposition patterns, and electron microscopy features, were analyzed and correlated with clinical indicators such as 24-hour urine protein (24 hUP), estimated glomerular filtration rate (eGFR), and hypertension. Results: Among the 40 patients, the M1 (95.0%) and S1 (77.5%) scores of the Oxford Classification were the most prevalent. Correlation analysis between pathology and clinical indicators showed that in the patient group with global glomerulosclerosis ≥ 25%, the proportions of 24 hUP ≥ 1 g, eGFR < 60 ml/min/1.73m², and concomitant hypertension were 85.0%, 75.0%, and 85.0%, respectively, all higher than those in the group with global glomerulosclerosis < 25% (55.0%, 35.0%, 45.0%). The incidence of adverse clinical indicators was also higher in groups with tubular atrophy/interstitial fibrosis (T lesions) ≥ 25% and the presence of crescents (C lesions). As the Lee grade increased, the proportions of T1-T2 and C1-C2 lesions showed a significant increasing trend, reaching 100%

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for both lesion types in Grade V patients. Conclusion: The T and C lesions in the Oxford Classification of IgAN are closely associated with adverse clinical indicators, including higher levels of proteinuria and lower eGFR. The multimodal pathological evaluation model, particularly the standardized application of the Oxford Classification, holds significant clinical value for accurately assessing the severity of IgAN.

1. INTRODUCTION

IgA Nephropathy (IgAN) is the most common primary glomerular disease in China and a leading cause of end-stage renal disease (ESRD) [1]. Its clinical manifestations and renal pathological changes are highly heterogeneous. Accurately assessing pathological changes to predict clinical outcomes is a current research focus. The Oxford Classification (MEST-C), an internationally recognized pathological assessment system for IgAN, includes five core indicators: mesangial hypercellularity (M), endocapillary hypercellularity (E), segmental glomerulosclerosis or adhesion (S), tubular atrophy/interstitial fibrosis (T), and crescentic lesions (C), providing a standardized framework for prognostic judgment [2].

In recent years, with deepening understanding of IgAN, the prognostic value of the Oxford Classification has been continuously validated and refined across different populations and clinical scenarios. Multiple studies emphasize that T and C lesions are strong predictors of renal function progression [3, 4]. However, analyses systematically combining the Oxford Classification with multimodal pathological features such as immunofluorescence and electron microscopy, and correlating them with patients' baseline clinical status, especially in local populations, remain scarce. This single-center retrospective cohort study aims to investigate the correlation between the Oxford Classification, various pathological indicators, and key clinical prognostic indicators based on real-world clinical-pathological data, to provide localized evidence for clinical practice.

2. SUBJECTS AND METHODS

2.1. Study Subjects

A retrospective cohort study design was employed, consecutively enrolling patients diagnosed with primary IgAN by renal biopsy in the Department of Nephrology, Baise People's Hospital, between January 2021 and December 2024.

2.1.1. Inclusion Criteria

(1) Age ≥ 14 years; (2) Pathologically confirmed primary IgAN by renal biopsy; (3) Complete and traceable clinical and pathological data.

2.1.2. Exclusion Criteria

(1) Secondary IgA deposition diseases, such as Henoch-Schönlein purpura nephritis, cirrhotic nephropathy, etc.; (2) Coexisting other primary glomerular diseases; (3) Severe missing clinical or pathological data preventing data analysis.

Finally, 40 patients meeting the criteria were included in this study.

2.2. Research Methods

Baseline data at the time of renal biopsy were collected through the hospital information system, including gender, age, clinical diagnosis, 24-hour urine protein (24 hUP), estimated glomerular filtration rate (eGFR, calculated using the CKD-EPI equation), and blood pressure. Hypertension was defined as systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg, or current use of antihypertensive medication.

2.3. Pathological Evaluation

2.3.1. Oxford Classification (MEST-C)

All renal biopsy tissues were independently assessed by two renal pathologists blinded to the clinical data, according to the 2016 Oxford Classification criteria. The definitions for M, E, S, T, and C indicators strictly followed the international consensus [2].

2.3.2. Evaluation of Global Glomerulosclerosis

Global glomerulosclerosis refers to the diffuse sclerosis of the entire capillary loops of glomeruli, resulting in a complete loss of glomerular function. The calculation method involves counting the total number of evaluable glomeruli in renal biopsy tissue sections, calculating the proportion of glomeruli with global sclerosis, and classifying the cases into two groups: ≥25% global sclerosis and <25% global sclerosis. The relationship between this indicator and the segmental glomerulosclerosis (S) score in the Oxford classification: The S score focuses on segmental sclerosis lesions in part of the glomerulus, while global glomerulosclerosis focuses on diffuse sclerosis of the entire glomerulus. The two indicators reflect different scopes and degrees of glomerular injury respectively and can complement each other in evaluating the overall glomerular injury status.

2.3.3. Immunofluorescence Examination

The deposition intensity of immune markers including IgA, IgG, IgM, C3, and C1q was detected, and results were recorded using a semi-quantitative grading scale: negative, +, 2+, 3+.

2.3.4. Electron Microscopy

The deposition sites of electron-dense deposits were observed.

2.3.5. Lee Grade

The Lee grading system was used for the overall assessment of glomerular lesion severity [5].

2.4. Statistical Analysis

Data processing was performed using SPSS software (version 30.0). Measurement data are presented as mean ± standard deviation, and count data as number (percentage). Intergroup comparisons were conducted using Fisher's exact test, with a P-value < 0.05 considered statistically significant.

3. RESULTS

3.1. Patient Baseline Characteristics

This study included 40 patients, comprising 22 males (55.0%) and 18 females (45.0%). The age range was 14 - 75 years, with a mean age of 36.8 ± 11.5 years. Clinical diagnoses included nephrotic syndrome in 15 cases (37.5%), nephritic syndrome in 12 cases (30.0%), and renal insufficiency in 8 cases (20.0%). Detailed baseline characteristics are shown in Table 1.

Table 1. Baseline characteristics of IgAN patients (n = 40).

No.	Pathology No.	Gender	Age	Clinical Diagnosis	Lee Grade	MEST-C Score	24 h Urine Protein (g)	eGFR (ml/min/1.73m^2)	Blood Pressure (mmHg)
1	KB2103752	F	33	Nephrotic Syndrome	IV	M1E0S1T1C1	-	-	-
2	KB2104365	F	45	Renal Insufficiency	IV	M1E0S1T1C1	-	-	-

Continued

3	KB2105234	M	37	Nephritic Syndrome	III	M1E0S1T0C0	-	-	-
4	KB2106673	F	23	Nephrotic Syndrome	V	M1E0S1T1C1	-	-	-
5	KB2107240	M	28	Renal Insufficiency	V	M1E0S1T2C0	-	-	-
6	KB2108257	F	37	Renal Insufficiency	III	M1E0S0T0C0	-	-	-
7	KB2108444	M	40	Nephrotic Syndrome	IV	M1E1S1T1C0	-	-	-
8	KB2109692	M	33	Nephrotic Syndrome	V	M1E1S1T2C1	-	-	-
9	KB2109748	M	51	Nephritic Syndrome	III	M1E0S1T0C0	-	-	-
10	KB2110143	M	39	Proteinuria Etiology	V	M1E1S0T2C0	-	-	-
11	KB2111917	M	29	Nephritic Syndrome	II	M1E0S0T0C0	-	-	-
12	KB2111981	F	27	Edema Etiology	II	M0E0S0T0C0	-	-	-
13	KB2112381	M	44	Proteinuria Etiology	II	M1E0S0T0C0	-	-	-
14	KB2113930	F	33	Nephritic Syndrome	III	M1E0S1T0C1	-	-	-
15	KB2114700	M	35	Nephrotic Syndrome	IV	M1E1S1T1C2	-	-	-
16	KB2200719	M	23	Nephritic Syndrome	IV	M1E1S1T1C1	-	-	-
17	KB2200928	F	64	Nephrotic Syndrome	III	M1E0S1T0C2	-	-	-
18	KB2200985	M	29	Proteinuria Etiology Pending	II	M0E0S0T0C0	-	-	-
19	KB2201146	F	38	-	IV	M1E0S1T1C0	-	-	-
20	KB2201937	M	30	CKD Stage 4	V	M1E0S1T1C1	-	-	-
21	KB2202466	F	48	Nephritic Syndrome	III	M1E0S1T0C0	-	-	-
22	KB2202467	M	15	-	IV	M1E1S1T1C0	-	-	-
23	KB2202883	M	28	Nephritic Syndrome	V	M1E0S1T2C0	-	-	-
24	KB2203159	M	55	Nephrotic Syndrome	III	M1E1S0T0C0	-	-	-
25	KB2203364	F	44	-	IV	M1E0S1T1C0	-	-	-
26	KB2203763	F	55	Proteinuria Etiology	III	M0E0S0T0C0	-	-	-
27	KB2205888	F	34	-	III	M1E1S1T0C0	-	-	-
28	KB2207393	F	25	Nephrotic Syndrome	V	M1E1S1T2C1	-	-	-
29	KB2208458	F	50	-	IV	M1E1S1T1C1	-	-	-
30	KB2209236	F	28	Chronic Nephritic Syndrome	III	M1E0S1T0C0	-	-	-
31	KB2209378	F	43	Nephritic Syndrome	IV	M1E0S1T1C1	-	-	-
32	KB2209902	F	49	-	III	M1E1S1T0C1	-	-	-
33	KB2210725	M	36	-	III	M1E0S1T0C1	-	-	-
34	KB2210908	F	36	Nephritic Syndrome	IV	M1E1S1T1C0	-	-	-
35	KB2210909	M	37	Nephrotic Syndrome	IV	M0E0S1T1C0	-	-	-
36	KB2211663	F	32	-	III	M1E0S0T0C0	-	-	-
37	KB2211761	F	45	-	II	M1E0S1T0C0	-	-	-

Continued

38	KB2212199	F	50	-	III	M1E1S0T0C1	-	-	-
39	KB2213949	F	46	Nephritic Syndrome	V	M1E1S1T2C0	-	-	-
40	KB2302308	F	14	-	III	M1E0S0T0C2	-	-	-

3.2. Distribution of Oxford Classification and Other Pathological Features

Results of the Oxford Classification showed M1 in 38 cases (95.0%) and S1 in 31 cases (77.5%). For the T score, T0 accounted for 42.5% (17/40), T1 for 37.5% (15/40), and T2 for 20.0% (8/40), with T1 + T2 totaling comprising 57.5%. For the C score, C0 accounted for 40.0% (16/40), C1 for 32.5% (13/40), and C2 for 27.5% (11/40), with C1 + C2 comprising 60.0%. E1 was found in 12 cases (30.0%). Specific distributions are shown in Table 2 and Figure 1.

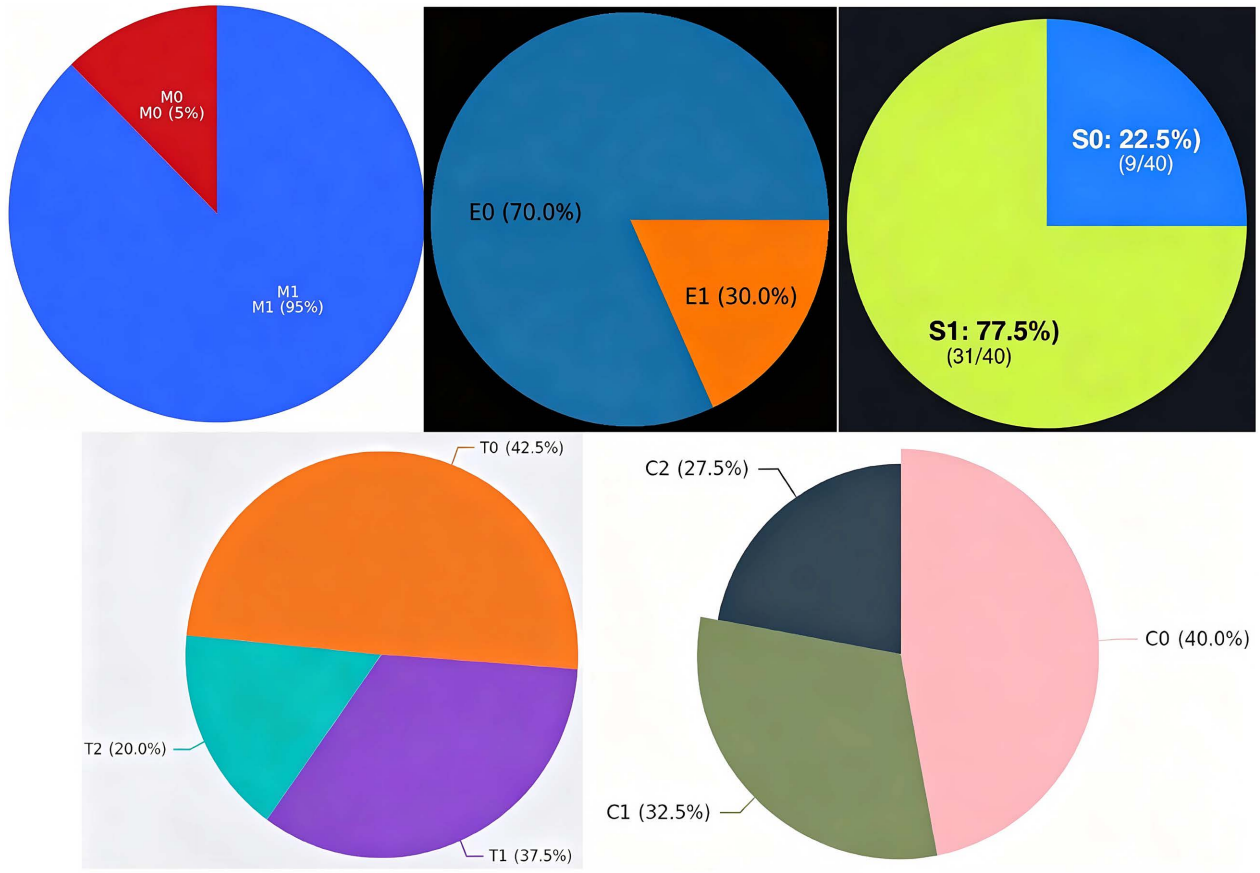

Figure 1. Distribution of oxford classification indicators in IgA nephropathy (M: mesangial hypercellularity; E: endothelial hypercellularity; S: segmental sclerosis; T: tubular atrophy/interstitial fibrosis; C: crescent formation).

Table 2. Distribution of oxford classification parameters in IgAN (n = 40).

Parameter	Score	No. of Cases	Percentage (%)
M Score	M0	2	5.0
	M1	38	95.0

Continued

E Score	E0	28	70.0
	E1	12	30.0
S Score	S0	9	22.5
	S1	31	77.5
T Score	T0	17	42.5
	T1	15	37.5
	T2	8	20.0
C Score	C0	16	40.0
	C1	13	32.5
	C2	11	27.5

Immunofluorescence results showed that IgA deposition was predominantly 2+ and 3+, totaling 36 cases (90.0%), consistent with the typical pathological features of IgAN. C3 positivity (+ to 3+) was observed in 31 cases (77.5%), while C1q positivity was rare, found in only 3 cases (7.5%). Electron microscopy revealed that electron-dense deposits were located primarily in the mesangial region in 35 cases (87.5%), with no deposits found in the subendothelial, subepithelial, or intramembranous areas. Details are shown in Table 3, Table 4, and Figure 2.

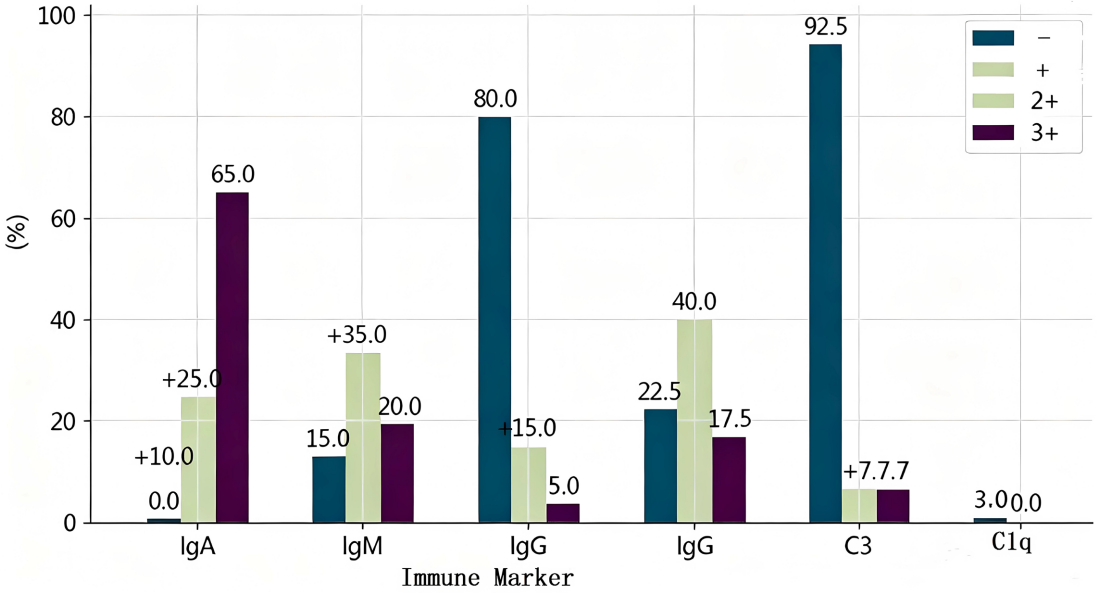

Figure 2. Distribution of immunofluorescence deposition intensity in IgA nephropathy.

Table 3. Immunofluorescence deposition characteristics in IgAN (n = 40).

Immune Marker	Negative (n/%)	Positive+ (n/%)	2+ (n/%)	3+ (n/%)
IgA	0 (0.0)	4 (10.0)	10 (25.0)	26 (65.0)
IgM	6 (15.0)	14 (35.0)	12 (30.0)	8 (20.0)
IgG	32 (80.0)	6 (15.0)	2 (5.0)	0 (0.0)

Continued

C3	9 (22.5)	8 (20.0)	16 (40.0)	7 (17.5)
C1q	37 (92.5)	3 (7.5)	0 (0.0)	0 (0.0)

Table 4. Sites of electron-dense deposit deposition in IgAN by electron microscopy (n = 40).

Deposition Site	No. of Cases	Percentage (%)
Mesangial	35	87.5
Subendothelial	0	0.0
Subepithelial	0	0.0
Intramembranous	0	0.0
No Deposits	5	12.5

3.3. Correlation Analysis between Pathological Changes and Clinical Indicators

In the patient group with global glomerulosclerosis ≥ 25% (n = 20), the proportions of 24 hUP ≥ 1 g, eGFR < 60 ml/min/1.73m^2, and concomitant hypertension were 85.0%, 75.0%, and 85.0%, respectively, all higher than those in the group with global glomerulosclerosis < 25% (n = 20: 55.0%, 35.0%, 45.0%). The incidence of adverse clinical indicators was also generally higher in the group with tubular atrophy/interstitial fibrosis (T lesions) ≥25% (n = 18) and the group with crescents (C lesions present, n = 24) compared to groups with milder lesions. Although some intergroup comparisons did not reach statistical significance, possibly due to sample size limitations, overall clear correlative trends were observed. Despite the limitation of small sample size, some inter-group comparisons (e.g., comparison of 24 hUP ≥ 1 g between the group with tubular atrophy/interstitial fibrosis (T lesion) ≥ 25% and the group with T lesion < 25%, and comparison of eGFR < 60 between the group with crescent formation (C lesion) and the group without C lesion) did not reach statistical significance. However, a clear associative trend was observed overall. Details are shown in Table 5.

Table 5. Correlation analysis between pathological changes and clinical indicators in IgAN.

Pathological Feature	Group	No. of Cases	24 hUP ≥ 1 g (n/%)	eGFR < 60 (n/%)	Hypertension (n/%)
Global Glomerulosclerosis	<25%	20	11 (55.0)	7 (35.0)	9 (45.0)
	≥25%	20	17 (85.0)	15 (75.0)	17 (85.0)
Tubular Atrophy/ Interstitial Fibrosis	<25%	22	13 (59.1)	9 (40.9)	10 (45.5)
	≥25%	18	15 (83.3)	13 (72.2)	15 (83.3)
Crescentic Lesions	Absent (C0)	16	9 (56.2)	8 (50.0)	8 (50.0)
	Present (C1/C2)	24	20 (83.3)	14 (58.3)	19 (79.2)

3.4. Correspondence between Lee Grade and Oxford Classification

Analysis showed that as the Lee grade increased from II to V, the proportions of S1, T1 + T2, and C1 + C2 lesions exhibited a marked increasing trend. Among Grade V patients (n = 8), the proportions of both T1 + T2 and C1 + C2 lesions reached 100.0% (8/8), significantly higher than the 0.0% (0/6) and 33.3% (2/6) observed in Grade II patients, respectively. This suggests that higher Lee grades are associated with more prominent chronic tubulointerstitial damage (T lesions) and acute active lesions (C lesions). Details are

shown in Table 6 and Figure 3.

Table 6. Correspondence between Lee Grade and Oxford classification in IgAN.

Lee Grade	No. of Cases	M1 (n/%)	S1 (n/%)	T1 + T2 (n/%)	C1 + C2 (n/%)
II	6	5 (83.3)	2 (33.3)	0 (0.0)	2 (33.3)
III	15	14 (93.3)	12 (80.0)	4 (26.7)	9 (60.0)
IV	11	11 (100.0)	10 (90.9)	9 (81.8)	7 (63.6)
V	8	7 (87.5)	7 (87.5)	8 (100.0)	8 (100.0)

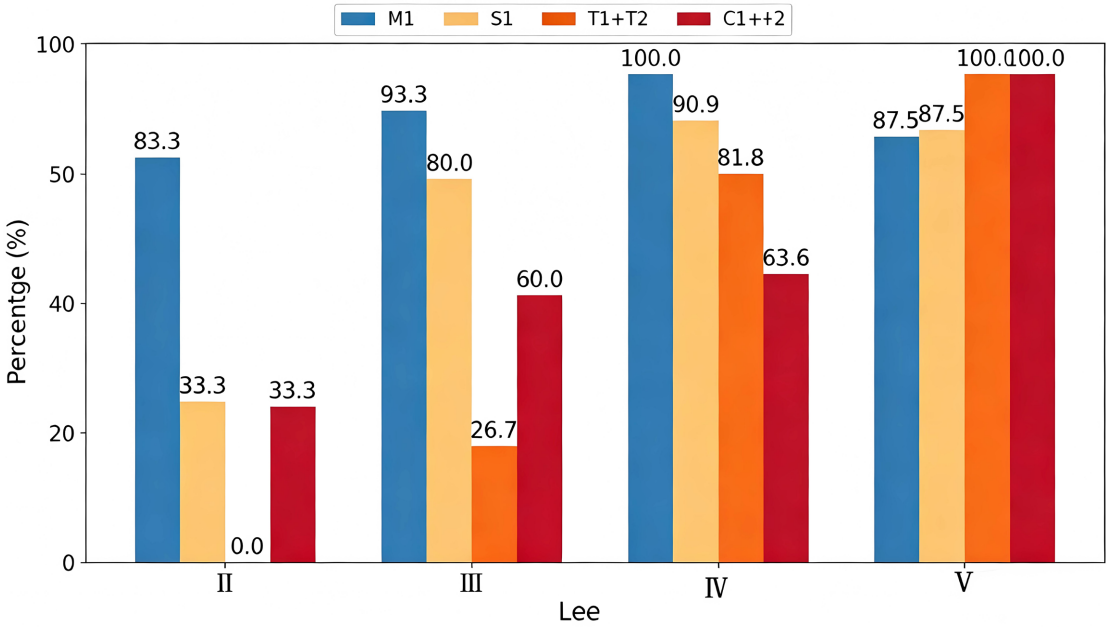

Figure 3. Comparison of oxford classification indicators among different Lee Grade Groups.

4. DISCUSSION

Based on real-world clinical data from 40 IgAN patients at Baise People's Hospital, this study systematically analyzed the correlation between the Oxford Classification and other pathological features with baseline clinical indicators. The core finding is that T lesions, representing chronic irreversible damage, and C lesions, representing acute active injury, are closely associated with higher levels of proteinuria, poorer renal function (eGFR < 60 ml/min/1.73m^2), and a higher prevalence of hypertension. This finding is highly consistent with recent international research consensus. For instance, a 2023 meta-analysis focusing on the Chinese population confirmed that T1/T2 lesions are independent risk factors affecting renal survival in IgAN patients [4]. A 2022 multicenter study also indicated that the presence of crescents, particularly cellular crescents, is an important reference indicator for selecting intensive immunosuppressive therapy [6].

The high prevalence of M1 and S1 lesions in this cohort reflects the relatively common pathological features of mesangial proliferation and segmental sclerosis in newly diagnosed IgAN patients requiring renal biopsy in clinical practice. It is noteworthy that although the correlation of M and E lesions with clinical indicators was less pronounced than that of T and C lesions in this cohort, this does not negate their clinical value. Existing studies have shown that mesangial hypercellularity (M lesion) can serve as a potential indicator for predicting treatment response. For patients with significant M1 lesions, active immunosuppressive therapy is more likely to achieve proteinuria remission [7]. Endothelial hypercellularity (E lesion) usually

indicates an acute inflammatory response in the kidney; timely intervention can reduce inflammatory damage and delay disease progression, and its value in assessing disease activity has been supported by relevant studies [7].

Regarding immunofluorescence and electron microscopy characteristics, this cohort presented a typical multimodal pathological profile of IgAN: predominant mesangial IgA deposition, frequent C3 co-deposition, rare "full-house" or C1q deposition, and deposits confined to the mesangium under electron microscopy. These features provide important basis for the differential diagnosis of IgAN from other glomerular diseases such as lupus nephritis [8].

A key implication of this study is that even in the absence of severe global glomerulosclerosis, the presence of significant T or C lesions is associated with an adverse trend in clinical indicators. This suggests that clinical pathological assessment should comprehensively evaluate damage in the glomeruli, tubulointerstitium, and vessels, rather than focusing solely on the glomeruli themselves. Tubulointerstitial fibrosis, as a common pathological pathway for the progression of various chronic kidney diseases, shows severity closely related to renal function prognosis [3].

5. CONCLUSION

Based on real-world single-center clinical data, this study confirms that the T and C lesions in the Oxford classification of IgA nephropathy are significantly associated with adverse baseline clinical indicators such as higher proteinuria levels, lower eGFR, and higher prevalence of hypertension. This finding has clear clinical practical significance: Patients with pathological findings of T1-2 or C1-2 lesions should be classified as high-risk groups, with closer clinical follow-up to monitor changes in renal function and proteinuria. Meanwhile, for patients with significant C lesions (especially C2 grade), initiation of intensive immunosuppressive therapy may be considered to control acute inflammatory responses and delay disease progression. For patients with prominent T lesions, emphasis should be placed on renal function protection, and active intervention should be implemented for risk factors such as hypertension and proteinuria to prevent further aggravation of tubulointerstitial injury. Standardized application of the Oxford classification, combined with multimodal pathological evaluation information including immunofluorescence and electron microscopy, holds important clinical value for accurately judging the severity of IgA nephropathy, conducting risk stratification, and guiding initial treatment decisions.

STUDY LIMITATIONS

This was a single-center retrospective study with a relatively small sample size (n = 40), which may lead to insufficient statistical power; some intergroup comparisons did not reach statistical significance, and selection bias exists. The lack of long-term follow-up data prevents assessment of the long-term association between the Oxford Classification and disease progression endpoints such as doubling of serum creatinine or ESRD, limiting the analysis to cross-sectional correlations. Some clinical data (e.g., 24hUP, eGFR) were missing in individual cases. Novel biomarkers such as Gd-IgA1 were not included for multidimensional analysis.

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CONFLICTS OF INTEREST

All authors of this study declare no conflicts of interest, financial or otherwise.

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ABSTRACT HIGHLIGHT

This single-center retrospective cohort study investigated the correlation between the Oxford classification (MEST-C) of IgA nephropathy (IgAN) and baseline clinical indicators, integrating multimodal pathological evaluations (immunofluorescence and electron microscopy). A total of 40 patients with primary IgAN diagnosed via renal biopsy (January 2021-December 2024) were enrolled.

KEY FINDINGS

1) The Oxford classification in this cohort was predominantly M1 (95.0%) and S1 (77.5%), with 57.5% of patients presenting T1-2 lesions and 60.0% presenting C1-2 lesions.

2) Patients with glomerular global sclerosis ≥ 25%, tubular atrophy/interstitial fibrosis (T lesion) ≥ 25%, or crescent formation (C lesion) showed significantly higher rates of adverse clinical indicators: 24-hour urinary protein ≥ 1 g (83.3% - 85.0%), estimated glomerular filtration rate < 60 ml/min (58.3% - 75.0%), and hypertension (79.2% - 85.0%)—all markedly higher than those in groups with milder lesions.

3) With increasing Lee grades (from II to V), the proportions of T1-2 and C1-2 lesions increased progressively, reaching 100% in grade V patients.

CLINICAL IMPLICATION

T lesions and C lesions in the Oxford classification are strong correlates of poor baseline clinical status in IgAN. Standardized Oxford classification combined with multimodal pathological assessment is critical for accurate severity stratification and clinical decision-making in IgAN management.