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Original Research Article

Clinicopathological Features and Risk Factors Analysis of IgA Nephropathy Associated with Acute Kidney Injury: A Single-Center Experience from Eastern India

Pramod Kumar[1], Md Asif Nizami[2], Sarvjeet Kumar[3]

[1]3rd Year DM Resident, Department of Nephrology, Institute of Medical Science and SUM Hospital, Bhubaneswar, Odisha, India

[2]3rd Year DM Resident, Department of Nephrology, Institute of Medical Science and SUM Hospital, Bhubaneswar, Odisha, India

[3]3rd Year DM Resident, Department of Medical Gastroenterology, Institute of Medical Science and SUM Hospital, Bhubaneswar, Odisha, India

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Corresponding Author: Pramod Kumar

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Abstract:

Background: The most common primary glomerulonephritis in the world and a major contributor to chronic kidney disease (CKD) in India is immunoglobulin A nephropathy (IgAN). Although the illness usually progresses slowly to end-stage renal disease (ESRD), a small percentage of patients have acute kidney injury (AKI). Negative long-term renal survival is linked to this condition. There is still a dearth of information about the clinicopathological spectrum of IgAN-associated AKI from Eastern India, especially with regard to the Oxford MEST-C classification.

Objective: To assess the clinicopathological characteristics of individuals with biopsy-proven IgA nephropathy at a tertiary care facility in Odisha and determine independent risk variables linked to AKI.

Methods: From May 2023 to August 2025, this prospective observational study was carried out at the Institute of Medical Sciences and SUM Hospital in Bhubaneswar, Odisha. One hundred patients with primary IgAN confirmed by biopsy were included in the study. According to KDIGO recommendations, patients were divided into AKI and non-AKI groups. The Oxford MEST-C classification was used to grade the histopathological results, laboratory values, and clinical data. To identify AKI factors, logistic regression analysis was utilized.

Results: The cohort had a male-to-female ratio of 2.1:1 and a mean age of 34.5 ± 12.2 years. Of the group, 18% (n=18) had documented AKI. Nephrotic-range proteinuria and macroscopic hematuria were substantially more common in the AKI group ($61.1\% \; vs. \; 15.8\%, p < 0.001$). Acute tubular necrosis (ATN) and cellular/fibrocellular crescents (C1/C2 scores) were substantially more common in the AKI group histopathologically ($72.2\% \; vs. \; 24.4\%, p < 0.01$). The most frequent triggering cause was prior mucosal infections. Macroscopic hematuria (OR 4.2), crescent presence (OR 5.6), and baseline hyperuricemia (OR 2.1) were found to be independent predictors of AKI by multivariate analysis.

Conclusion: Crescentic transformation and hematuria-induced tubular damage are the main causes of AKI in IgA nephropathy, a prominent clinical entity in Eastern India. One reliable indicator of acute dysfunction is the Oxford C-score. Improving renal outcomes and directing immunosuppressive medication need early detection of these risk factors.

Introduction

The most prevalent primary glomerular disease in the world is still immunoglobulin A nephropathy (IgAN), which was initially identified by Berger and Hinglais in 1968 [1]. The dominant or co-dominant deposition of IgA immune complexes, particularly galactose-deficient IgA1 (Gd-IgA1), in the glomerular mesangium is what pathologically defines it. The "Four-Hit Hypothesis" currently explains the pathogenesis: Gd-IgA1 production, autoantibody production against this galactose-deficient hinge region, circulating immune complex formation, and deposition of these complexes in the mesangium, which causes inflammation and fibrosis [2].

IgAN has a strikingly diverse clinical presentation. Although the traditional course of chronic kidney disease (CKD) is characterized by asymptomatic microscopic hematuria and sluggish progression, the disease can also present with severe symptoms. In the context of IgAN, acute kidney injury (AKI) is a

serious consequence that considerably deteriorates the prognosis. Up to 25% of IgAN patients may experience AKI throughout the course of the illness, which is linked to a higher risk of renal function not recovering and a quicker progression to End-Stage Renal Disease (ESRD) [3, 4].

There is significant geographic variation in the epidemiology of IgAN. Compared to fewer than 10% in North America and Europe, IgAN is responsible for 30–40% of all primary glomerular disorders in East Asian and South Asian populations [5]. Additionally, compared to their Western counterparts, patients with the "Asian phenotype" of IgAN typically present at a younger age and have more advanced histological damage at diagnosis. IgAN is a major cause of CKD in India, however there is still little information on acute exacerbations [6]. India's tropical climate, high prevalence of respiratory and gastrointestinal mucosal diseases, and genetic variety may all contribute to particular AKI triggering variables that are not adequately described in Western literature.

Two different pathophysiological processes are typically linked to AKI in IgAN. The first is severe glomerular inflammation, which is a medical emergency needing immediate immunosuppression and frequently appears as crescentic glomerulonephritis (necrotizing lesions). Massive glomerular hematuria causes Acute Tubular Necrosis (ATN), the second non-glomerular mechanism. In these situations, tubular damage is caused by direct cytotoxic effects of heme pigments and intratubular blockage by red blood cell (RBC) casts [7]. It is important to distinguish between these systems since they differ greatly in how they are managed.

Histological lesions in IgAN can be graded consistently using the Oxford classification (MEST-C score). Its usefulness in predicting AKI episodes—especially the relevance of the "C" (crescent) and "E" (endocapillary hypercellularity) scores in the Indian population—needs more validation, even if its value in predicting long-term CKD development is well-established [8].

By examining the clinicopathological features of 100 biopsy-proven IgAN patients at the Institute of Medical Sciences (IMS) and SUM Hospital, Bhubaneswar, this study seeks to close the current knowledge gap. In order to direct early treatment intervention and enhance renal survival in Eastern India, this study aims to uncover strong, region-specific risk factors for AKI by associating clinical presentations with Oxford MEST-C scores.

Methodology

Study Design and Setting: The Department of Nephrology and Pathology at the Institute of Medical Sciences and SUM Hospital in Bhubaneswar, Odisha, served as the site of this prospective observational study. Starting in May 2023 and ending in August 2025, the study lasted 27 months. Before the study started, the Institutional Ethics Committee gave its approval, and each participant gave their informed consent.

Study Population and Eligibility: A total of one hundred patients were enrolled in the trial. Patients who were 18 years of age or older and had a primary diagnosis of IgAN confirmed by renal biopsy—defined as dominant or co-dominant IgA staining in the mesangium on immunofluorescence—were the only ones who could be included. For proper histological scoring, the biopsy material had to have at least eight glomeruli. Individuals with secondary causes of IgA deposition, such as systemic lupus erythematosus, cirrhosis, or IgA vasculitis (Henoch-Schönlein purpura), were excluded. To guarantee a homogeneous group, individuals with co-occurring diabetic nephropathy, other superimposed glomerular diseases, or inadequate clinical data (follow-up less than three months) were also eliminated from the analysis.

Data Collection and Clinical Definitions: At the time of admission, we created a thorough clinical profile for each participant. This procedure started with a thorough interview to record medical history and demographic information, with an emphasis on identifying any recent mucosal infections—such as sore throats or gastrointestinal disturbances—that occurred during the month before presentation. In order to detect macroscopic hematuria, we also particularly asked patients about any instances of noticeable urine staining. We measured blood pressure and evaluated volume status during the physical examination by looking for pedal edema. We defined hypertension as a value greater than 140/90 mmHg or the present use of blood pressure medication.

A series of laboratory tests were carried out after the clinical evaluation. This comprised a lipid profile, a full blood count, and important indicators of renal function as urea, uric acid, and serum creatinine. A 24-hour urine collection was used to gauge the degree of protein loss, and urine sediment was inspected under a microscope to check for casts or red blood cells. The CKD-EPI formula was used to estimate renal function. Patients were categorized as having Acute Kidney Injury (AKI) if their blood creatinine increased by 0.3 mg/dL within 48 hours or if it climbed to 1.5 times their baseline level within the preceding week, according to the standard KDIGO guidelines [9].

Histopathological Evaluation: The main component of this investigation was the histological evaluation, which was carried out on renal biopsy specimens with a minimum of eight glomeruli to guarantee diagnostic precision. To view the

glomerular and tubular architecture, the tissue samples were prepared for light microscopy using a typical battery of stains, including Hematoxylin & Eosin, Periodic Acid-Schiff (PAS), Masson's trichrome, and Silver Methenamine. Studies using immunofluorescence were also conducted to verify that IgA deposits predominated.

Using the most recent Oxford MEST-C classification system, a senior renal pathologist evaluated each slide to determine the disease's severity [8]. Instead of only recording the existence of abnormalities, we measured them: segmental sclerosis (S) and endocapillary hypercellularity (E) were simply marked as present or missing, whereas mesangial hypercellularity (M) was scored according to whether it impacted more or less than half of the glomeruli. The percentage of cortical area damaged was used to grade tubular atrophy (T). Importantly, we graded crescent formation (C) according to the percentage of glomeruli participating. In order to detect non-glomerular causes of kidney failure, we especially searched for indicators of acute tubular injury, such as the flattening of epithelial cells or the presence of red blood cell casts within the tubules, in addition to the MEST-C scores.

Statistical Analysis: We used SPSS software (version 26.0) to handle and analyze the data that was gathered. We started our analytical process by examining the data's distribution. If continuous variables, such as age and creatinine levels, had a normal distribution, we reported them as means with standard deviations; if not, we reported them as medians with interquartile ranges. Percentages were used to describe categorical data, such as gender or the existence of hypertension.

We used the Chi-square test for categorical variables to compare the differences between the AKI and Non-AKI groups. Depending on the data distribution, we used either the independent t-test or the Mann-Whitney U test for continuous variables. We used both univariate and multivariate logistic regression analysis to go beyond basic correlations and determine which characteristics truly predicted the onset of AKI. This made it possible for us to determine independent risk factors and compute Odds Ratios (OR). Any finding that had a p-value of less than 0.05 was deemed statistically significant.

Results

Baseline Characteristics: One hundred patients with biopsy-proven IgAN made up the study cohort. There was a clear male preponderance (68%) and a mean age of (34.5 ± 12.2) years. At the time of presentation or throughout the study period, eighteen individuals (18%) satisfied the criteria for AKI.

Table 1: **Baseline Demographic and Clinical Characteristics**

Parameter	Total (n=100)	AKI Group (n=18)	Non-AKI Group (n=82)	P-value
Age (years)	34.5 ± 12.2	31.2 ± 10.5	35.2 ± 12.5	0.21
Male Gender, n (%)	68 (68%)	14 (77.8%)	54 (65.8%)	0.32
Hypertension, n (%)	42 (42%)	12 (66.7%)	30 (36.6%)	**0.02** *
Gross Hematuria, n (%)	24 (24%)	11 (61.1%)	13 (15.8%)	**< 0.001** *
Edema, n (%)	55 (55%)	15 (83.3%)	40 (48.8%)	**0.01** *
Preceding Infection, n (%)	31 (31%)	10 (55.6%)	21 (25.6%)	**0.01** *

Macroscopic hematuria was much more common in the AKI group (61.1%) than in the non-AKI group. Edema and hypertension, two clinical signs of fluid overload, were also more common in the AKI group.

Laboratory Parameters: In terms of biochemistry, the AKI group showed significant proteinuria and severe renal impairment.

Table 2: **Comparison of baseline laboratory parameters and renal function indices**

Parameter	AKI Group (Mean ± SD)	Non-AKI Group (Mean ± SD)	P-value
Serum Creatinine (mg/dL)	3.8 ± 1.4	1.1 ± 0.4	**< 0.001**
eGFR (mL/min/1.73m²)	28.5 ± 12.1	82.4 ± 24.5	**< 0.001**
Hemoglobin (g/dL)	9.8 ± 1.6	11.2 ± 1.8	**0.003**
Serum Albumin (g/dL)	2.9 ± 0.6	3.6 ± 0.5	**< 0.001**
24h Urinary Protein (g)	3.2 ± 1.8	1.4 ± 0.9	**< 0.001**
Serum Uric Acid (mg/dL)	7.9 ± 1.5	5.8 ± 1.2	**< 0.001**

The AKI group had significantly higher levels of hyperuricemia ($7.9 \pm 1.5\ \mathrm{mg/dL}$), which may indicate tubular stress or decreased clearance.

Precipitating Factors for AKI: We examined the triggering events found in the patient history to gain a better understanding of the clinical setting of AKI episodes.

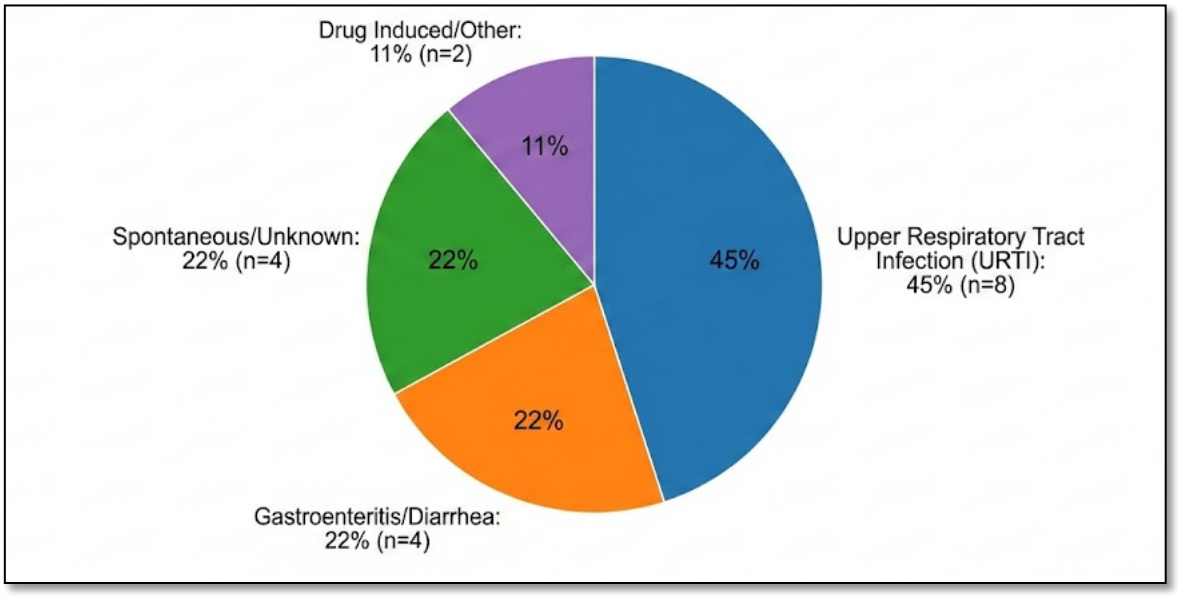

Figure 1: **Distribution of Precipitating Factors for AKI in IgAN Patients (n=18)**

Histopathological Analysis (Oxford MEST-C Classification)

Important distinction between the groups was made possible by histopathology.

- **Crescents (C Score):** The most powerful histological discriminant was the existence of cellular or fibrocellular crescents. Compared to just 10% of the non-AKI group, 72.2% of AKI patients had C1 or C2 lesions.
- **Endocapillary Hypercellularity (E Score):** 20% of non-AKI patients and 55% of AKI patients have it.
- **Tubular Injury:** Acute tubular necrosis (ATN) and intratubular RBC casts were seen in 38.8% of AKI patients, most of whom had extensive hematuria.

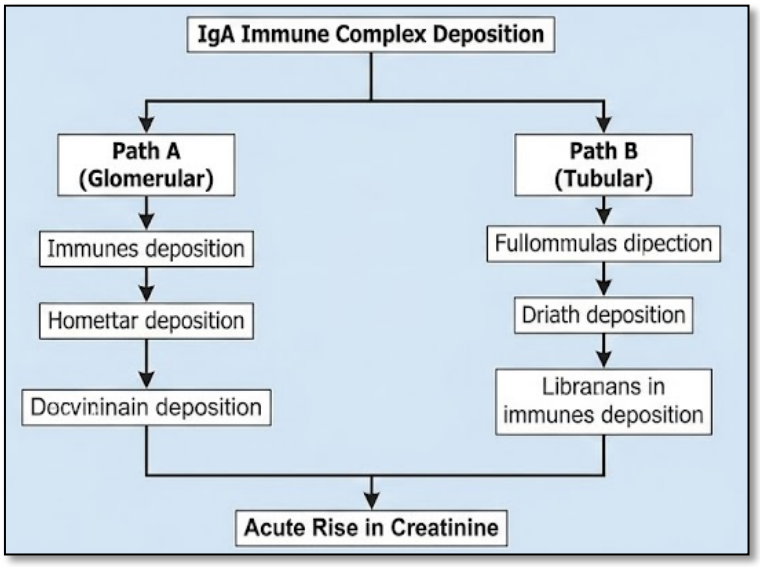

Figure 2: **Schematic Mechanism of AKI in IgA Nephropathy**

Treatment and Outcomes: Depending on the underlying pathophysiology (crescentic vs. ATN), different management approaches and short-term results were observed.

Table 3: Treatment Modalities and Short-term Outcomes in AKI Group (n=18)

Treatment Modality	n (%)	Full Recovery of Renal Function	Partial Recovery	No Recovery (Dialysis Dependent)
IV Methylprednisolone Pulse	10 (55.5%)	4	4	2
Oral Steroids Only	3 (16.6%)	1	2	0
Conservative / Supportive Care	5 (27.7%)	4	1	0
Total	18 (100%)	9 (50%)	7 (38.8%)	2 (11.1%)

Note: Conservative care was primarily utilized for patients with ATN-dominant histology without active crescents. Pulse steroids were reserved for crescentic disease.

Risk Factor Analysis

Table 4: Multivariate Logistic Regression Analysis for Predictors of AKI

Variable	Odds Ratio (OR)	95% CI	P-value
Macroscopic Hematuria	4.21	$1.85 - 9.56$	$\mathbf{0.001}$
Oxford C Score (C1/C2)	5.64	$2.12 - 14.98$	$< \mathbf{0.001}$
Serum Uric Acid > 7.0 mg/dL	2.15	$1.05 - 4.40$	$\mathbf{0.036}$
Massive Proteinuria (> 3g)	1.98	$0.92 - 4.25$	0.081
Hypertension	1.65	$0.75 - 3.60$	0.210

The most powerful independent risk variables for AKI are histological evidence of crescents and clinical presentation of macroscopic hematuria, according to the multivariate analysis.

Discussion

The clinicopathological landscape of IgA nephropathy in Eastern India is thoroughly examined in this prospective study from IMS & SUM Hospital. Our results highlight that AKI, which affects around 18% of patients, is a common and varied consequence of IgAN.

Incidence and Demographic Trends: Our cohort's mean age of 34.5 years is consistent with national patterns shown by Jha et al. and Chacko et al., indicating that IgAN affects a younger population in India as opposed to Western populations, where diagnoses often occur in the fourth or fifth decade [6, 10]. Our study's AKI incidence of 18% is in line with previous data from China and India that indicate a rising frequency of acute presentations, most likely because to increased ambient antigenic exposure rates [11].

Pathogenic Mechanisms: Hematuria vs. Crescents: Two unambiguous routes to AKI are identified by our investigation.

First, AKI linked to hematuria: We discovered a significant association (OR 4.21) between AKI and macroscopic hematuria. These patients often had RBC and ATN casts on histology. This lends credence to the theory of "Warfarin-related nephropathy" or hemoprotein-induced tubular toxicity, in which tubular blockage and oxidative stress are caused by free hemoglobin released from lysed red blood cells [7, 12]. This reversible form of AKI was previously identified by Kveder et al., who noted that if treated with hydration and alkalization, it frequently has a better prognosis than crescentic illness [13].

Second, Crescentic IgAN: AKI was most significantly predicted by the presence of crescents (C1/C2) (OR 5.64). This is consistent with the Oxford Classification validation studies that identified cellular crescents as an indicator of active glomerular damage [8, 14]. Crescents are the main cause of rapid renal deterioration in clinical practice, according to our data and research by Haas et al. and Lv et al., but the original Oxford study was reluctant to add C-scores because it excluded patients with severe renal failure [15, 16].

The Role of Hyperuricemia: The independent correlation between baseline hyperuricemia and AKI (OR 2.15) was an intriguing discovery. Although uric acid has historically been thought of as a secondary effect of decreased GFR, new research by Srivastava et al. indicates that it may cause endothelial dysfunction, activate the renin-angiotensin system, and increase tubulointerstitial inflammation [17]. Urate-lowering medication may be useful in high-risk IgAN patients because pre-existing hyperuricemia may make the kidney more sensitive to acute insults in the context of AKI.

Infection as a Trigger: Mucosal infections (URTI and gastroenteritis) preceded AKI in 67% of cases,

as seen in Figure 1. This supports the "gut-kidney axis" idea, which holds that Gd-IgA1 synthesis is primed by mucosal inflammation [18]. In contrast to respiratory-dominant causes in European research, the significant frequency of gastroenteritis-associated AKI in our group might be unique to Odisha's tropical environment [19].

Treatment Implications: Table 3 shows that pulse steroid-treated patients, especially those with crescentic histology, often had good outcomes. The catastrophic possibility of AKI is highlighted by the fact that around 11% of patients did not regain renal function. The KDIGO 2021 guidelines for intensive immunosuppression in cases that progress quickly are supported by this [20].

Limitations

In order to present a fair scientific viewpoint, it is necessary to acknowledge the various limitations of this study. First and foremost, the results might not accurately reflect the genetic and environmental variety of the entire Indian subcontinent due to its single-center design inside Eastern India. The apparent incidence of AKI may potentially be inflated by referral bias, since tertiary care facilities often handle more serious cases than primary care clinics. Additionally, a higher sample size would have enhanced the statistical strength of the logistic regression analysis, especially when evaluating less frequent factors like individual drug exposures, even though a cohort of 100 biopsies is significant for a single facility.

From a pathophysiological perspective, it was not possible to assess blood levels of anti-Gd-IgA1 autoantibodies or Galactose-deficient IgA1 (Gd-IgA1) due to resource limitations. The depth of our mechanistic understanding is limited by the inability to link these particular molecular biomarkers to the severity of AKI. Lastly, while the study period was long enough to evaluate short-term recovery, it was not long enough to assess the long-term effects of these AKI episodes on the development of End-Stage Renal Disease (ESRD) over a period of five to ten years. Strong correlations between risk factors and outcomes can be seen in this observational study, however causation regarding the effectiveness of the various treatment regimens used cannot be conclusively established.

Conclusion

In conclusion, 18% of patients in Eastern India suffer from acute kidney injury, a common and potentially fatal consequence of IgA nephropathy. This work reveals a unique dual-pathology causing these acute episodes: hematuria-induced acute tubular necrosis, which frequently responds to supportive treatments, and inflammatory crescentic glomerulonephritis, which necessitates strong immunosuppression. Thus, a strong framework for instant risk categorization in this area is established by the Oxford MEST-C score, specifically the presence of cellular crescents (C1/C2), in conjunction with the clinical manifestation of macroscopic hematuria.

Additionally, as mucosal infections were found to be the main precipitating triggers in our sample, clinicians should keep a high index of suspicion for AKI in patients who present with prior infections. The study emphasizes that early kidney biopsy is essential for differentiating between glomerular and tubular injury, which helps make the crucial choice between pulse steroid therapy and conservative management. Half of the AKI cohort had reversible renal impairment, which highlights a critical treatment window that needs to be taken advantage of in order to improve outcomes. To confirm these results and create tailored treatment plans for this aggressive trait, more multicentric research involving genetic study of the Indian population is crucial.

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