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Clinical Profile and Prognostic Outcomes of Adult-Onset Nephrotic Syndrome With Acute Kidney Injury: A Prospective Cohort Study at Bach Mai Hospital

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ABSTRACT

Nephrotic syndrome (NS) complicated by acute kidney injury (AKI) is associated with substantial morbidity and an increased risk of long-term renal dysfunction, yet prospective data on histopathological patterns, treatment response, and renal outcomes in this population remain limited. In this prospective observational cohort study, we enrolled 122 adults aged 18–60 years with newly diagnosed NS and concurrent AKI at the Kidney and Urology Center of Bach Mai Hospital, Hanoi, Vietnam, between 2023 and 2024, and followed them for 12 months. Clinical and biochemical parameters, renal biopsy findings, and treatment responses were evaluated, and AKI was classified according to AKIN criteria. Membranous glomerulonephritis (MGN, 36.9%) and focal segmental glomerulosclerosis (FSGS, 35.2%) were the most common histological subtypes, followed by minimal change disease (MCD, 23.0%). Pre-renal AKI accounted for 86.1% of cases, and AKIN Stage I was the most frequent stage at presentation (51.6%). Over 12 months, significant improvements were observed in serum creatinine, uric acid, lipid profile, proteinuria, and hematuria, with serum creatinine decreasing from 1.68 to 1.26 mg/dL and proteinuria from 5.16 to 1.7 g/24 h ($p < 0.001$ for all). Renal recovery was most frequent in patients with AKIN Stage I (95.2%) and least frequent in those with Stage III disease (30.0%). Treatment response also varied markedly by histological subtype: complete remission occurred in all patients with MCD, in 55.8% of those with FSGS, and in none of those with MGN. These findings indicate that histopathological subtype and AKIN stage are important predictors of renal recovery and therapeutic response in adults with NS-associated AKI, and support the clinical value of early recognition of pre-renal causes together with biopsy-guided management to improve outcomes.

Categories: Nephrology
Keywords: acute kidney injury, nephrotic syndrome, renal recovery, AKIN staging, histopathology

1. Introduction

Nephrotic syndrome (NS) represents a prevalent glomerular disorder in the adult population, characterized by the classical triad of heavy proteinuria, hypoalbuminemia, and peripheral edema, frequently accompanied by dyslipidemia [1,2]. Although many patients follow a relapsing–remitting clinical course, NS is far from benign. Among its complications, acute kidney injury (AKI) has emerged as a particularly critical concern, due to its potential to abruptly impair renal function, disrupt homeostatic mechanisms, and substantially increase morbidity and mortality [3,4].

In recent years, the incidence of AKI in patients with NS has shown a marked rise, which may reflect both improved diagnostic vigilance and greater exposure to risk factors such as aging, polypharmacy, and widespread use of nephrotoxic agents[5]. AKI in this context arises through diverse mechanisms—including intravascular volume depletion secondary to hypoalbuminemia, excessive diuretic administration, infections, and nephrotoxic exposure (notably to non-steroidal anti-inflammatory drugs)—as well as, though less commonly, intrinsic renal pathology such as acute tubular necrosis or rapidly progressive glomerulonephritis. Importantly, both the presentation and prognosis of AKI vary substantially, depending on the underlying histopathological subtype of NS and the severity of the acute renal insult [6,7].

Despite advances in the management of glomerular diseases, AKI continues to play a pivotal role in determining both short- and long-term renal outcomes. Severe or persistent episodes may culminate in incomplete recovery, progressive renal dysfunction, or eventual transition to chronic kidney disease (CKD) and

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end-stage renal disease (ESRD) [8]. These risks are further exacerbated by the heterogeneity of NS subtypes—where diseases such as focal segmental glomerulosclerosis (FSGS) and membranous glomerulonephritis (MGN) are frequently associated with poorer renal outcomes and suboptimal responses to immunosuppressive therapy, in contrast to the generally favorable prognosis observed in minimal change disease (MCD) [9].

Although prior research has addressed certain epidemiologic and clinical aspects of AKI in NS, there remains a notable scarcity of prospective studies that comprehensively characterize the clinical presentation, histopathological diversity, and longitudinal outcomes of adults with NS complicated by AKI, particularly under contemporary therapeutic protocols. Moreover, the prognostic implications of AKI severity—classified by Acute Kidney Injury Network (AKIN) staging—and the interplay between histological diagnosis and renal recovery have yet to be clearly delineated in adult cohorts [10,11].

To address these gaps, the present study was designed as a prospective, observational cohort aimed at systematically evaluating the clinical features, histological spectrum, treatment responses, and renal outcomes of adults with newly diagnosed NS complicated by AKI. Through longitudinal follow-up over a 12-month period, this study sought to elucidate the relationship between biochemical trends, AKI staging, and glomerular pathology, with the ultimate goal of improving prognostic stratification and guiding individualized treatment strategies in this complex patient population.

Research Objectives

To evaluate the clinical characteristics, histopathological patterns, and prognostic outcomes of adult-onset nephrotic syndrome with acute kidney injury, with particular emphasis on the predictive roles of AKIN stage and renal biopsy findings

2. Methodology

2.1. Study Design and Setting

This study was designed as a prospective, observational cohort and was conducted at a tertiary care hospital over a 12-month period. The research protocol received ethical approval from the institutional review board, and all procedures adhered to the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

2.2. Study Population

This study enrolled adult patients aged between 18 and 60 years who were newly diagnosed with nephrotic syndrome (NS) accompanied by concurrent acute kidney injury (AKI) at the Kidney and Urology Center of Bach Mai Hospital between 2023 and 2024. The diagnosis of NS was established based on both clinical and laboratory criteria, including generalized edema, proteinuria exceeding 3.5 g/day, and hypoalbuminemia (serum albumin <30 g/L). AKI was identified and staged according to the Acute Kidney Injury Network (AKIN) criteria, which classify AKI severity based on changes in serum creatinine and urine output [10,11].

Exclusion criteria comprised a prior diagnosis of diabetes mellitus, chronic kidney disease (CKD), systemic diseases known to cause secondary proteinuria (such as systemic lupus erythematosus or amyloidosis), acute nephritic syndrome, and rapidly progressive glomerulonephritis. These criteria were applied to ensure a homogeneous study population of patients with primary NS complicated by superimposed AKI.

2.3. Baseline Assessment

At enrollment, comprehensive baseline data were collected, including demographic characteristics (age, sex), clinical features (blood pressure), and detailed medication history, with particular attention to recent use of diuretics and non-steroidal anti-inflammatory drugs (NSAIDs). Baseline laboratory tests included serum creatinine, uric acid, lipid profile (total cholesterol, triglycerides, LDL, HDL), 24-hour urinary protein excretion, hematuria (RBCs/high-power field), serum albumin, total protein, and complete blood count. Complement components C3 and C4 were measured to assess possible immune-mediated disease. Autoimmune work-up included testing for antinuclear antibody (ANA), anti-double-stranded DNA (anti-dsDNA), anti-Smith (anti-Sm), and antineutrophil cytoplasmic antibodies (proteinase 3-ANCA, myeloperoxidase-ANCA) using immunofluorescence assay (IFA) and enzyme-linked immunosorbent assay (ELISA), in order to rule out secondary causes of NS.

All patients underwent percutaneous, ultrasound-guided renal biopsy. Histopathological evaluation was conducted by experienced renal pathologists and classified into subtypes including membranous glomerulonephritis (MGN), focal segmental glomerulosclerosis (FSGS), minimal change disease (MCD), and other less common glomerular diseases.

2.4. Follow-Up and Outcomes

Participants were followed longitudinally for a minimum of 12 months, with scheduled follow-up assessments at baseline, 3 months, 6 months, and 12 months. At each visit, clinical evaluation and laboratory investigations were repeated. Monitored variables included serum creatinine, estimated glomerular filtration rate (eGFR), uric acid, lipid profile, proteinuria, and other relevant biochemical parameters.

The primary outcome was renal recovery, defined as return to baseline or near-baseline renal function. Secondary outcomes included progression to CKD, response to treatment (categorized as complete remission, partial remission, or no response), and the requirement for renal replacement therapy during the follow-up period.

Treatment decisions were individualized based on histopathological diagnosis and clinical condition. Regimens included corticosteroids, immunosuppressive agents (e.g., calcineurin inhibitors, cyclophosphamide), and supportive measures such as diuretics, statins, or anticoagulation where indicated. All therapeutic interventions were documented in detail throughout the follow-up period.

2.5. Statistical Analysis

All statistical analyses were performed using SPSS version 21.0 (IBM Corp., Armonk, NY, USA). Categorical variables were expressed as counts and percentages, while continuous variables were summarized using means with standard deviations or medians with interquartile ranges, as appropriate. Changes in continuous variables across time points were analyzed using paired t-tests or non-parametric equivalents (e.g., Wilcoxon signed-rank test), based on data distribution. The Kruskal-Wallis test was used to compare biochemical changes across multiple time points. Associations between histopathological subtypes, AKIN stages, and treatment outcomes were assessed using logistic regression analysis. A two-tailed p-value <0.05 was considered statistically significant.

2.6. Ethical Considerations

The study protocol was reviewed and approved by the institutional ethics committee. All participants provided written informed consent prior to inclusion in the study. The research was conducted in compliance with the ethical principles of the Declaration of Helsinki and its subsequent amendments.

3. RESULTS

Table 1 presented the baseline characteristics of the study participants. The mean age was 40.7 ± 12.9 years, with 40.2% of patients under 30 years old. Males accounted for 63% of the cohort. Average blood pressure was within normal limits. Anemia was rare (1.6%), and complement levels (C3 and C4) were mostly normal, indicating minimal immunologic abnormality at presentation. Membranous nephropathy (MGN) was the most common diagnosis (36.9%), followed by focal segmental glomerulosclerosis (FSGS, 35.2%) and minimal change disease (MCD, 23%).

Regarding AKI type, pre-renal AKI was predominant (86.1%), while intrinsic and post-renal AKI were reported in 10.7% and 3.3% of patients, respectively. For AKIN staging, more than half of the patients were categorized as Stage I (51.6%), followed by Stage II (32.0%) and Stage III (16.4%). At presentation, 18.9% of patients had recent exposure to non-steroidal anti-inflammatory drugs (NSAIDs), and 58.2% had used diuretics. The mean baseline creatinine was 1.68 ± 0.52 mg/dL, baseline proteinuria was 2.85 ± 0.38 g/day, and serum albumin was 5.16 ± 2.51 g/dL. These findings reflected a population with moderate renal impairment and significant protein loss at diagnosis, frequently associated with pharmacological triggers. (Table 2).

Table 3 summarized the longitudinal evolution of key laboratory markers from baseline to 12 months. Several parameters demonstrated statistically significant changes over time, indicating favorable therapeutic responses. Most notably, serum creatinine decreased significantly from 1.68 ± 0.52 mg/dL at baseline to 1.26 ± 0.31 mg/dL at 12 months ($p < 0.001$), reflecting improved renal function. Similarly, uric acid levels dropped from 7.91 ± 1.71 to 4.42 ± 1.44 mg/dL ($p < 0.001$). Among lipid profiles, cholesterol fell substantially from 266.42 ± 24.02 to 163.79 ± 42.80 mg/dL ($p = 0.02$), while triglycerides showed a sharp decline from 255.09 ± 39.39 to 149.89 ± 43.87 mg/dL ($p < 0.001$). Moreover, markers of nephrotic syndrome activity also improved: proteinuria reduced dramatically from 5.16 ± 2.51 to 1.7 ± 0.88 g/24h ($p < 0.001$), and hematuria decreased from 15.4 ± 33.21 to 2.11 ± 5.26 RBCs/high-power field ($p < 0.001$). In contrast, other variables such as total protein, albumin, LDL, HDL, hemoglobin, WBC, and platelet counts showed no statistically significant changes throughout follow-up ($p > 0.05$). These findings underscored the effectiveness of treatment in improving renal and metabolic outcomes, particularly in reducing disease activity marker.

Figure 1 illustrated the relationship between baseline AKIN stage and renal recovery outcomes at 12 months. Patients initially classified as AKIN Stage I showed the most favorable prognosis, with 95.2% achieving full recovery and only 4.8% progressing to chronic kidney disease (CKD). In contrast, recovery rates declined substantially in more severe stages: 64.1% of Stage II patients recovered, while 35.9% developed CKD. Among those with AKIN Stage III, only 30.0% achieved recovery, and the majority (70.0%) progressed to CKD. These findings underscored the prognostic value of initial AKI severity, with higher AKIN stages being strongly associated with poorer long-term renal outcomes.

Figure 2 illustrated the distribution of treatment responses according to histological subtypes among the study participants. Patients with minimal change disease (MCD) demonstrated the most favorable outcomes, with 100% achieving complete remission (CR). In contrast, those with membranous glomerulonephritis (MGN) had no complete responders, and all patients exhibited partial remission (PR). Among focal segmental glomerulosclerosis (FSGS) cases, only 55.8% achieved complete response, while 23.3% showed partial remission and 20.9% had no response (NR). The “Other” histological group exhibited the poorest response, with only 66.7% achieving partial remission and 33.3% showing no response. These findings underscored a significant variation in treatment efficacy across different histological classes. (Figure 2).

4. DISCUSSION

This 12-month prospective cohort study systematically investigated the clinical characteristics, histopathological profiles, and treatment outcomes in adults with nephrotic syndrome (NS) complicated by acute kidney injury (AKI). The results indicated that membranous glomerulonephritis (MGN, 36.9%) and focal segmental glomerulosclerosis (FSGS, 35.2%) constituted the most prevalent histopathological subtypes, followed by minimal change disease (MCD, 23%) and other glomerulopathies (4.9%). The predominance of MGN and FSGS aligned with existing epidemiological data in adult populations, while the considerable proportion of MCD underscored its continued relevance among this demographic [12].

Histopathological subtype was significantly associated with differential treatment response. All patients diagnosed with MCD achieved complete remission, consistent with the high steroid sensitivity reported by Waldman et al.[13]. In contrast, only 55.8% of FSGS

patients attained complete remission, with the remainder demonstrating partial or no response—reflecting the known clinical heterogeneity and steroid resistance described by D'Agati et al.[14]. Patients with MGN achieved partial remission exclusively, a finding that mirrored those of Ponticelli et al. and was characteristic of the disease's typically indolent course and suboptimal response to conventional immunosuppression [15]. These findings were further corroborated by Yadav et al. [16]., who observed 100% complete remission among MCD patients, 52.2% remission in FSGS, and partial remission in all MGN cases, reinforcing the prognostic utility of histopathological classification [16,17].

Pre-renal AKI emerged as the dominant etiology, accounting for 86.1% of cases in this cohort. Intrinsic and post-renal causes were infrequent. These observations were consistent with those of Yadav et al.[16], who reported a similar pre-renal predominance (95%), emphasizing the pathophysiological role of hemodynamic disturbances such as hypovolemia, overuse of diuretics, and NSAID exposure in this context. More than half of the patients presented with AKIN Stage I, while only 16.4% had Stage III disease at baseline. This distribution suggested timely recognition and intervention in most cases, although a significant proportion still developed severe AKI [18]. These patterns were in concordance with earlier studies by Praga et al.[19] and Kellum et al.[20,21], which also reported the predominance of reversible, pre-renal injury in NS-related AKI.

Substantial improvements in renal and metabolic parameters were observed over the 12-month follow-up period. Serum creatinine declined from 1.68 to 1.26 mg/dL, uric acid from 7.91 to 4.42 mg/dL, cholesterol from 266.4 to 163.8 mg/dL, triglycerides from 255.1 to 149.9 mg/dL, proteinuria from 5.16 to 1.70 g/24h, and hematuria from 15.4 to 2.1 RBCs/hpf. All changes were statistically significant ($p < 0.05$). These findings closely resembled those of Yadav et al.[16], who documented similar biochemical improvements within a three-month treatment window. Clinically, such reductions in proteinuria and serum creatinine indicated disease remission, while improvements in lipid profile and uric acid levels may have translated to decreased cardiovascular and renal risk [22]. These observations were in agreement with Cattran et al.[23], who underscored the prognostic relevance of sustained reductions in proteinuria [24,25].

Renal recovery was closely associated with the severity of AKI at presentation. Patients with AKIN Stage I had the highest rate of recovery (95.2%), followed by those in Stage II (64.1%) and Stage III (30.0%). Notably, progression to chronic kidney disease (CKD) occurred most frequently among Stage III patients (70.0%). This trend mirrored that of Yadav et al.[16], who reported recovery rates of 96.9% and 82.1% in AKIN Stages I and II, respectively. Furthermore, our results aligned with the findings of Coca et al.[26], who demonstrated a direct correlation between AKI severity and adverse long-term renal outcomes. These findings validated the use of AKIN staging not only as a severity index but also as a robust predictor of prognosis [27–29].

The high prevalence of pre-renal AKI and the prognostic significance of AKIN staging emphasized the need for early recognition and prevention of reversible factors, including careful management of diuretics and avoidance of nephrotoxins [30,31]. Additionally, the variation in treatment response across histological subtypes highlighted the value of kidney biopsy in directing therapeutic decisions and follow-up strategies. Patients with FSGS or MGN—particularly those with severe AKI—required closer monitoring and possibly alternative immunosuppressive regimens if standard treatments failed. Conversely, while MCD patients generally responded well to therapy, they remained at risk for relapse, especially in the adult population, and thus warranted ongoing surveillance.

Limitations

This study's strengths included its prospective design, well-defined follow-up intervals, and comprehensive evaluation of histological and biochemical parameters. However, limitations were also acknowledged. As a single-center study with a moderate sample size and a 12-month observation period, its findings may have limited generalizability and were insufficient to assess long-term outcomes such as relapse or late CKD progression. Similar limitations were reported, who advocated for future multicenter investigations with extended follow-up to validate and expand upon these findings.

5. CONCLUSIONS

In conclusion, this study contributed to the growing evidence base on NS-associated AKI by highlighting the predominance of reversible, pre-renal mechanisms and underscoring the prognostic roles of histopathological subtype and AKIN stage. These factors strongly influenced therapeutic response and long-term renal outcomes. Future research should focus on multicenter studies with longer follow-up durations, incorporate biomarker-based risk stratification, and explore novel therapeutic approaches to improve outcomes, particularly in patients with steroid-resistant FSGS or progressive AKI.

Additional Information

Author Contributions: All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Disclosures

Human subjects: The study protocol was reviewed and approved by the Institutional Review Board (IRB) under approval number 2023/PHAD/DER-05-03. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki and the guidelines of the International Conference on Harmonization for Good Clinical Practice (ICH-GCP). Written informed consent was obtained from all participants prior to their enrollment.

Animal subjects: All authors have confirmed that this study did not involve animal subjects or tissue.

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following:

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Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

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