



RESEARCH ARTICLE

ASSOCIATION OF MICROALBUMINURIA WITH STROKE SEVERITY AND INFARCT SIZE IN ACUTE ISCHEMIC STROKE: A PROSPECTIVE CASE-CONTROL STUDY

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ABSTRACT

Background: Microalbuminuria (MAU), a marker of systemic endothelial dysfunction, has been increasingly recognized for its association with cardiovascular and cerebrovascular diseases. This study aimed to evaluate the relationship between microalbuminuria and the severity of acute ischemic stroke, assessed using the National Institutes of Health Stroke Scale (NIHSS), and to determine whether microalbuminuria can serve as a predictor of infarct size and neurological impairment. **Methods:** A total of 60 patients diagnosed with acute ischemic stroke were enrolled. Spot urine samples were analyzed for microalbuminuria, and stroke severity was assessed using the NIHSS. Infarct size was categorized as either small (SI) or large (LI) based on radiological findings. Statistical analysis was performed to evaluate the correlation between microalbuminuria and stroke severity. **Results:** Spot microalbuminuria was detected in 70.0% of cases compared to 10.0% of controls, with a statistically significant association ($p = 0.000$). A strong correlation was observed between microalbuminuria and stroke severity based on NIHSS scores at admission and one month later ($p = 0.000$), with higher microalbuminuria prevalence in moderate to severe and severe stroke categories. Stroke size also correlated with microalbuminuria, with larger infarcts more frequently associated with its presence. **Conclusion:** The presence of microalbuminuria is significantly associated with both infarct size and stroke severity in acute ischemic stroke patients. These findings suggest that microalbuminuria could serve as a simple, cost-effective prognostic biomarker in the clinical assessment of stroke severity. Incorporating microalbuminuria screening in acute stroke management may enhance early risk stratification and guide therapeutic decisions.

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INTRODUCTION

Acute ischemic stroke (AIS) stands as a predominant cause of mortality and long-term disability globally, resulting from the sudden occlusion of cerebral arteries leading to brain tissue infarction.¹ The severity of AIS varies among individuals, influenced by a myriad of factors including age, comorbid conditions, and the extent of cerebral involvement. Early identification of prognostic markers is crucial for stratifying patients, guiding therapeutic interventions, and improving clinical outcomes. Microalbuminuria (MAU), characterized by the excretion of 30–300 mg of albumin in urine per day, has emerged as a potential biomarker reflecting systemic endothelial dysfunction and microvascular damage.² Traditionally associated with diabetic nephropathy and cardiovascular diseases, recent studies have highlighted its prevalence in AIS patients and its correlation with stroke severity and prognosis. For instance, a study by Li *et al.* reported that MAU was present in approximately 36.88% of AIS patients and was independently associated with higher National Institutes of Health Stroke Scale (NIHSS) scores at admission and poorer outcomes at three months, as measured by the modified Rankin Scale (mRS).³

The pathophysiological basis linking MAU to AIS severity lies in the shared mechanisms of endothelial dysfunction and systemic inflammation. Elevated urinary albumin levels may indicate widespread vascular injury, which not only predisposes individuals to stroke but also exacerbates its severity. Furthermore, MAU has been associated with early neurological deterioration (END) in AIS patients, suggesting its role as a predictor of acute worsening in neurological status.⁴ Given the non-invasive nature and cost-effectiveness of MAU assessment, its incorporation into routine clinical evaluation could enhance risk stratification and management of AIS patients. However, despite accumulating evidence, the utility of MAU as a prognostic marker in AIS remains underexplored in diverse populations.⁵ This study aims to investigate the correlation between MAU and AIS severity, thereby elucidating its potential role in prognostication and guiding clinical decision-making.

METHODOLOGY

This prospective analytical study was conducted at Acharya Shri Chander Medical College, Jammu and Kashmir, over a six-month period from June 2022 to November 2022. The

study involved collaboration between the Department of General Medicine and the Department of Biochemistry. A total of 60 patients diagnosed clinically and radiologically with acute ischemic stroke (AIS) involving the middle cerebral artery (MCA) and anterior cerebral artery (ACA) territories were included. The diagnosis of AIS was confirmed through non-contrast computed tomography (CT) of the brain. An age- and sex-matched control group comprising 60 healthy individuals was also included for comparison of microalbuminuria prevalence. Patients aged 18 years and above, presenting within 24 hours of stroke onset, and willing to provide informed written consent were enrolled in the study. Exclusion criteria included patients with neoplasms, females during menstruation or pregnancy, and those found to have macroalbuminuria (urinary albumin excretion >300 mg/day) at the time of admission. After obtaining written informed consent, clinical and demographic data were collected using a pre-designed structured proforma. The evaluation of all enrolled patients included measurement of serum electrolytes, fasting blood sugar (FBS), HbA1c, serum lipid profile, high-sensitivity C-reactive protein (hs-CRP), electrocardiogram (ECG), and renal artery Doppler ultrasonography. All patients underwent non-contrast CT scan of the brain to confirm the diagnosis and localization of the infarct. Urine samples were obtained within 24 hours of admission to assess for microalbuminuria, defined as urinary albumin excretion between 30–300 mg/day. A repeat urine sample was collected one month later to assess the persistence or resolution of microalbuminuria. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) both at admission and after one month. NIHSS is a validated tool for objectively quantifying the impairment caused by a stroke, where higher scores indicate greater stroke severity. The primary objective of the study was to examine the correlation between microalbuminuria and the severity of acute ischemic stroke, and to determine whether microalbuminuria could serve as a potential biomarker for poor neurological outcomes. The study also aimed to compare the incidence of microalbuminuria in stroke patients with that in the general control population. Data were entered and analyzed using standard statistical software. Descriptive statistics were used to summarize clinical and demographic characteristics. Comparative analyses were performed using the Chi-square test for categorical variables and the Student's t-test or Mann-Whitney U test for continuous variables, as appropriate. A p-value < 0.05 was considered statistically significant. Ethical clearance was obtained from the institutional ethics committee, and all participants provided informed consent prior to participation. No conflict of interest was declared by the investigators.

RESULTS

The study included a total of 120 participants, comprising 60 cases and 60 age- and sex-matched controls. The age distribution showed that the highest proportion of participants belonged to the 61–70 years age group (30.0% in both cases and controls), followed by the 41–50 years group. The lowest representation was observed in the 71–80 years group, with only 5.0% in both groups. Overall, the distribution across age groups was comparable between cases and controls. In terms of gender, males constituted a higher proportion in both groups: 61.7% among cases and 55.0% among controls. Females made up 38.3% of the cases and 45.0% of the controls, indicating a male predominance in both groups.

Table. Age and Gender Distribution of Cases and Controls

Age Group (Years)	Cases (n, %)	Controls (n, %)	Total (n, %)
31–40	10 (16.7%)	11 (18.3%)	21 (17.5%)
41–50	18 (30.0%)	15 (25.0%)	33 (27.5%)
51–60	11 (18.3%)	13 (21.7%)	24 (20.0%)
61–70	18 (30.0%)	18 (30.0%)	36 (30.0%)
71–80	3 (5.0%)	3 (5.0%)	6 (5.0%)
Total	60 (100%)	60 (100%)	120 (100%)
Gender	Cases(n, %)	Controls (n, %)	
Male	37 (61.7%)	33 (55.0%)	
Female	23 (38.3%)	27 (45.0%)	
Total	60 (100%)	60 (100%)	

Table 2. Distribution of Spot Microalbuminuria among Cases and Controls

Group	Positive (n, %)	Negative (n, %)	Total (n)	Chi-Square Test (p-value)
Cases	42 (70.0%)	18 (30.0%)	60	0.000*
Controls	6 (10.0%)	54 (90.0%)	60	
Total	48	72	120	

*p < 0.05; statistically significant.

Table 3. Correlation between Spot Microalbuminuria and NIHSS Stroke Scale

NIHSS Stroke Scale	Spot Microalbuminuria Positive (n, %)	Spot Microalbuminuria Negative (n, %)	p-value
No stroke	0 (0.0%)	5 (8.3%)	0.000*
Minor stroke	0 (0.0%)	8 (13.3%)	
Moderate stroke	0 (0.0%)	5 (8.3%)	
Moderate to severe stroke	20 (33.3%)	0 (0.0%)	
Severe stroke	22 (36.7%)	0 (0.0%)	
Total	42 (70.0%)	18 (30.0%)	

* p < 0.05, statistically significant

The presence of spot microalbuminuria was significantly higher among cases compared to controls. Among the 60 patients with acute ischemic stroke (cases), 42 (70.0%) tested positive for microalbuminuria, whereas only 6 (10.0%) of the 60 controls had a positive result. Conversely, microalbuminuria was absent in 18 (30.0%) cases and 54 (90.0%) controls. The difference between the two groups was statistically significant, with a Chi-square test yielding a p-value of 0.000, indicating a strong association between acute ischemic stroke and microalbuminuria. In the present study, smoking and alcohol habits were assessed among both cases and controls. Among the acute ischemic stroke cases, 10 (16.7%) were smokers and 9 (15.0%) reported a history of alcohol consumption, while the majority were non-smokers (83.3%) and non-alcoholic (85.0%). Among the controls, 13 (21.7%) were smokers and 13 (21.7%) reported alcohol use, while 47 (78.3%) were non-smokers and non-alcoholic. Statistical analysis revealed that there was no significant association between smoking habit (p = 0.487) or alcohol habit (p = 0.345) and the occurrence of acute ischemic stroke in this study population.

The correlation between spot microalbuminuria and stroke severity assessed by the NIHSS stroke scale revealed a statistically significant association (p = 0.000). Notably, none of the patients with mild or no stroke showed microalbuminuria. In contrast, all cases of moderate to severe and severe stroke were associated with positive microalbuminuria, with 33.3% and 36.7% of total cases respectively. This indicates that the presence of microalbuminuria strongly correlates with increased stroke severity, highlighting its potential role as a prognostic

Table 4. The distribution of spot microalbuminuria across different age groups

Age Group (Years)	31–40	41–50	51–60	61–70	71–80	Chi-Square Test
Positive Cases (n)	11	20	13	4	0	p = 0.000*
% within Positive Cases	22.9%	41.7%	27.1%	8.3%	0.0%	

Table 5. Distribution of spot microalbuminuria based on gender

Spot Microalbuminuria	Gender		p-value
	Male	Female	
Positive Cases (n)	28	20	
% within Positive Cases	58.3%	41.7%	
Chi-Square Test		0.546	
		Not Significant	

Table 6. Distribution of One-Month NIHSS Scale Among the Study Group

One-Month NIHSS Scale	Frequency (n)	Valid Percent (%)
No Stroke	11	18.3%
Minor Stroke	12	20.0%
Moderate Stroke	18	30.0%
Moderate to Severe Stroke	4	6.7%
Severe Stroke	15	25.0%
Total	60	100.0%

Table 7. Correlation Between NIHSS Stroke Scale at 1 Month and Spot Microalbuminuria

NIHSS Scale (1 Month)	Spot Microalbuminuria Negative (n)	Spot Microalbuminuria Positive (n)	Total (n)	% within Microalbuminuria (-)	% within Microalbuminuria (+)	% of Total
No Stroke	9	2	11	50.0%	4.8%	18.3%
Minor Stroke	8	4	12	44.4%	9.5%	20.0%
Moderate Stroke	1	17	18	5.6%	40.5%	30.0%
Moderate to Severe Stroke	0	4	4	0.0%	9.5%	6.7%
Severe Stroke	0	15	15	0.0%	35.7%	25.0%
Total	18	42	60	100.0%	100.0%	100.0%

*P-value = 0.000 (Chi-square test, statistically significant)

Table 8. Distribution of Spot Microalbuminuria Based on Stroke Size

Stroke Size	Spot Microalbuminuria Negative (n)	Spot Microalbuminuria Positive (n)	Total (n)	% within Spot Microalbuminuria (-)	% within Spot Microalbuminuria (+)	% of Total
SI (Small Infarct)	18	2	20	100.0%	4.8%	33.3%
LI (Large Infarct)	0	40	40	0.0%	95.2%	66.7%
Total	18	42	60	100.0%	100.0%	100.0%

P-value = 0.000 (Chi-square test, statistically significant)

biomarker in acute ischemic stroke patients. The distribution of spot microalbuminuria across different age groups among patients with acute ischemic stroke revealed a statistically significant association (Chi-square test, **p** = 0.000). The highest proportion of positive cases was observed in the 41–50 year age group (41.7%), followed by the 51–60 year group (27.1%) and the 31–40 year group (22.9%). A smaller proportion (8.3%) was noted in the 61–70 year group, while no positive cases were recorded in the 71–80 year group. These findings suggest that spot microalbuminuria is more prevalent in middle-aged individuals with acute ischemic stroke, with a notable decline in older age groups. The distribution of spot microalbuminuria based on gender showed that out of the total positive cases, 28 (58.3%) were males and 20 (41.7%) were females. Statistical analysis using the Chi-square test revealed a p-value of 0.546, indicating that the difference in the occurrence of spot microalbuminuria between males and females was not statistically significant. This suggests that gender did not have a significant influence on the presence of microalbuminuria among the studied population. The table 6 presents the distribution of neurological impairment among patients one month after an acute ischemic stroke, as assessed by the NIHSS (National Institutes of Health Stroke Scale).

Among the 60 patients studied, 30.0% (n=18) had a moderate stroke, while 25.0% (n=15) continued to have severe stroke symptoms after one month. A total of 18.3% (n=11) showed no residual neurological deficits, and 20.0% (n=12) were classified as having a minor stroke. Only 6.7% (n=4) had moderate to severe stroke symptoms. These findings indicate that a substantial proportion of patients retained moderate to severe disability one month post-stroke. The correlation between NIHSS stroke scale at one month and spot microalbuminuria status revealed a statistically significant association (**p** = 0.000). Among patients with no stroke, 50.0% were negative for microalbuminuria and only 4.8% were positive. Conversely, in patients with moderate to severe and severe strokes, a large proportion were positive for microalbuminuria (40.5% and 35.7% respectively). This indicates that the presence of microalbuminuria was significantly associated with greater stroke severity at one month, suggesting its potential role as a prognostic marker. The distribution of spot microalbuminuria in relation to stroke size was analyzed in the study, and the results demonstrate a strong association between the presence of microalbuminuria and larger infarcts (LI). Out of the 60 patients included in the study, 18 had small infarcts (SI) and 42 had large infarcts (LI).

Among the 18 patients with small infarcts, only 2 (4.8%) tested positive for spot microalbuminuria, and the remaining 16 (100%) were negative for microalbuminuria. In contrast, all 42 patients with large infarcts tested positive for microalbuminuria, with no patients in this group testing negative. This difference is statistically significant, with a *p*-value of 0.000, highlighting the strong correlation between larger infarcts and the presence of microalbuminuria.

DISCUSSION

The present study investigated the association between microalbuminuria (MAU) and the severity of acute ischemic stroke, as determined by the National Institutes of Health Stroke Scale (NIHSS). Our findings demonstrate a statistically significant and clinically relevant correlation between microalbuminuria and stroke severity. Among the 60 patients included in our cohort, 42 (70%) tested positive for microalbuminuria. Notably, all 42 patients who presented with large infarcts were found to be MAU-positive, with no negative cases in this subgroup. The strength of this association, confirmed by a *p*-value of 0.000, reinforces the potential role of microalbuminuria as an indicator of larger infarct burden and more severe neurological impairment in acute ischemic stroke. The observed prevalence of microalbuminuria in our study is consistent with earlier findings reported in the literature. Kachroo *et al.* identified MAU in 47% of ischemic stroke patients, while Beamer *et al.* found a prevalence of 29%.^{6,7} Similarly, Fei L *et al.* reported microalbuminuria in 36.88% of patients with acute cerebral infarction.⁸ These results are further supported by a range of studies reporting prevalence rates between 12% and 60% among stroke patients, suggesting that MAU is a frequently encountered phenomenon, even among non-diabetic individuals.⁹⁻¹¹ This underscores the hypothesis that microalbuminuria may be more closely linked with vascular injury than with glycemic status alone. In addition to its prevalence, our study also highlighted a direct relationship between the presence of microalbuminuria and the degree of neurological deficit, as measured by the NIHSS. Patients with higher NIHSS scores were significantly more likely to exhibit MAU. This observation aligns closely with the findings of Kachroo *et al.*, who reported a strong positive correlation ($r = 0.904$, $p < 0.0001$) between NIHSS scores and microalbuminuria.⁶ Beamer *et al.* also emphasized the prognostic value of MAU, identifying it as an independent predictor of unfavorable outcomes in patients with acute ischemic stroke.⁷ Further evidence was provided by Muralidhara *et al.*, who demonstrated a graded increase in urine albumin-creatinine ratio with rising NIHSS scores, particularly in non-diabetic individuals.¹² These findings collectively suggest that MAU not only reflects the presence of cerebrovascular disease but may also serve as a surrogate marker for its clinical severity. Beyond its association with acute clinical outcomes, microalbuminuria has also been shown to be a predictor of long-term stroke risk. In a comprehensive meta-analysis, Kelly and Rothwell concluded that proteinuria—of which microalbuminuria is an early form—is an independent predictor of stroke, even after adjusting for traditional vascular risk factors such as hypertension, diabetes, and smoking.¹³ This observation strengthens the argument that microalbuminuria is more than a passive marker of comorbidity; rather, it may play an active role in the pathogenesis of cerebrovascular disease. Several

mechanisms have been proposed to explain the association between microalbuminuria and stroke severity. One of the most widely accepted explanations involves endothelial dysfunction. Microalbuminuria is considered an early and sensitive marker of systemic endothelial injury, which is a pivotal factor in the development of atherosclerosis and thromboembolic events (Festa *et al.*).¹⁴ The compromised integrity of the endothelium increases vascular permeability, allowing albumin to leak into the urine and reflecting systemic vascular fragility that likely extends to cerebral vessels. Another possible mechanism involves the close physiological link between the cerebral and renal microvasculature. According to the “strain vessel” hypothesis, small resistance vessels in the brain and kidneys are similarly vulnerable to hypertensive and hemodynamic stress due to their exposure to high pulsatile pressures (Ito S *et al.*).¹⁵

These vessels tend to suffer parallel damage under pathological conditions, which may explain why microalbuminuria often coexists with cerebral infarcts. Additionally, acute ischemic stroke may trigger dysregulation of the renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system, leading to glomerular hyperfiltration and subsequent albumin leakage. Systemic inflammation also offers a plausible biological link between microalbuminuria and stroke. Elevated levels of inflammatory biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor- α (TNF- α) have been associated with both MAU and cerebrovascular events (Mahmud N *et al.*, 1995).¹⁶ These cytokines promote endothelial dysfunction, increase vascular permeability, and contribute to plaque instability and thrombogenesis. Therefore, inflammation may serve as a shared pathogenic pathway in both renal and cerebral vascular injury. Taken together, these mechanisms suggest that microalbuminuria is not merely a byproduct of existing disease but may actively participate in the pathophysiology of ischemic stroke. Clinically, the utility of microalbuminuria lies in its ease of detection with low cost, and non-invasive nature, making it a promising biomarker for risk stratification in patients presenting with acute stroke. Early identification of MAU-positive patients could prompt closer monitoring, more aggressive risk factor modification, and possibly improved outcomes through timely intervention. However, certain limitations of the present study must be acknowledged. The cross-sectional design restricts the ability to infer causality or long-term prognostic implications. The relatively small sample size and single-center setting may also limit the generalizability of our findings. Furthermore, while we accounted for common vascular risk factors, residual confounding cannot be entirely ruled out. Future studies should consider larger, multicenter, longitudinal designs to validate these findings and further explore the causal pathways linking microalbuminuria to stroke severity and prognosis.

CONCLUSION

The findings of our study underscore a robust and statistically significant association between microalbuminuria and the severity of acute ischemic stroke. The high prevalence of microalbuminuria among patients with larger infarcts and elevated NIHSS scores suggests its potential role as a surrogate marker for extensive vascular injury and adverse neurological outcomes. These observations are well-aligned with existing literature and lend further support to the concept that

microalbuminuria reflects systemic endothelial dysfunction, microvascular compromise, and underlying inflammatory activity—factors integrally involved in the pathogenesis and progression of ischemic stroke. Given its ease of detection, cost-effectiveness, and prognostic value, routine assessment of microalbuminuria may offer an important adjunct in the early risk stratification and clinical management of patients with acute ischemic stroke. Future prospective studies with larger, diverse populations are warranted to validate these findings and explore whether interventions targeting microalbuminuria can favorably influence stroke outcomes.

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