Urinary microalbumin-to-creatinine ratio as a predictor of cerebral vasospasm in patients with aneurysmal subarachnoid hemorrhage

Eurasian Clinical and Analytical Medicine Original Research

Urinary microalbumin/creatinine ratio and vasospasm

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Abstract

Aim: Cerebral vasospasm is a major complication of aneurysmal subarachnoid hemorrhage (SAH), leading to increased morbidity and mortality. Reliable, easily applicable biomarkers for predicting vasospasm remain limited. This study investigated the relationship between urinary microalbumin-to-creatinine ratio and vasospasm in aneurysmal SAH patients.

Materials and Methods: In this prospective observational study, forty patients with aneurysmal subarachnoid hemorrhage (SAH) were included between May 2010 and February 2011. Neurological status was assessed using the Hunt–Hess scale, hemorrhage burden by the Fisher classification, and vasospasm was confirmed angiographically. Twenty-four–hour urinary microalbumin levels and serum creatinine were measured to calculate the microalbumin-to-creatinine ratio. Clinical, radiological, and laboratory parameters were compared between patients with and without vasospasm.

Results: Vasospasm occurred in 20 patients. Age and sex distributions did not differ between groups ($p > 0.05$). Patients with vasospasm had lower Glasgow Coma Scale scores (13.1 ± 1.8 vs. 14.9 ± 0.4, $p < 0.001$). Urinary microalbumin (173.5 ± 208.4 vs. 2.0 ± 2.3 mg/L, $p < 0.001$) and the microalbumin-to-creatinine ratio (310.9 ± 426.3 vs. 2.6 ± 2.6 mg/g, $p < 0.001$) were markedly higher in the vasospasm group. Both Hunt–Hess and Fisher grades were strongly associated with elevated urinary markers ($p < 0.001$), while BUN and serum creatinine showed no differences.

Discussion: The urinary microalbumin-to-creatinine ratio showed a strong association with vasospasm and disease severity in aneurysmal SAH. This easily measurable, non-invasive marker may help identify patients at higher risk for vasospasm, supporting earlier clinical intervention.

Keywords

aneurysmal subarachnoid hemorrhage, vasospasm, urinary microalbumin, microalbumin-to-creatinine ratio

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Introduction

Subarachnoid hemorrhage (SAH) is defined as bleeding into the cerebral and/or spinal subarachnoid space [1]. The most common cause of SAH is the rupture of cerebral aneurysms. Other etiologies include arteriovenous malformations, head trauma, hypertensive hemorrhage, coagulation disorders, sickle cell anemia, pituitary apoplexy, and cocaine use [2,3]. Cerebral vasospasm, characterized by pathological narrowing of cerebral arteries following SAH, remains one of the most critical complications, significantly contributing to morbidity and mortality in affected patients [4].

Although cerebral vasospasm most frequently develops after SAH, it may also occur following intracranial surgery, intracranial infections, or endovascular interventions. The incidence and severity of vasospasm correlate with neurological status. For instance, one study reported vasospasm in 22.2% of patients classified as grade I, compared with 73.3% in grade V [5]. A similar association is evident in mortality rates: 9% in patients without vasospasm, 15.5% in those with localized vasospasm, and 22% in those with diffuse vasospasm [6].

Despite extensive research, the pathophysiology of cerebral vasospasm remains incompletely understood, with recent studies highlighting a multifactorial etiology involving microcirculatory dysfunction, neuroinflammation, and thrombo-inflammation. For example, Stragier et al. discuss how microvascular injury and inflammatory cascades contribute to delayed ischemia [7]. Hoh et al.'s 2023 guideline update also frames delayed cerebral ischemia (DCI) as stemming from combined effects beyond pure vasospasm, including endothelial and metabolic derangements [8].

In this study, we investigated whether the urinary microalbumin-to-creatinine ratio is associated with vasospasm in patients with aneurysmal SAH. By focusing on a laboratory-based marker, we aimed to evaluate whether this parameter could serve as a simple and accessible tool for predicting vasospasm development in clinical practice.

Materials and Methods

Study Design and Setting

This prospective observational study was conducted between May 2010 and February 2011 at the Departments of Neurosurgery and Radiology, Çukurova University Faculty of Medicine. Ethical approval was obtained from the institutional review board, and informed consent was provided by all patients or their legal representatives.

Patient Selection

A total of 40 patients diagnosed with aneurysmal subarachnoid hemorrhage (SAH) were enrolled. Patients were admitted either to the Neurosurgery Intensive Care Unit or the Radiology Angiography Unit. The diagnosis of aneurysmal SAH was confirmed using computed tomography (CT). Only patients with normal renal function were included, defined as serum creatinine ≤1.2 mg/dL and an estimated glomerular filtration rate (eGFR) >60 mL/min/1.73 m². Exclusion criteria were evidence of renal impairment or comorbid conditions that could influence renal function.

Clinical and Radiological Evaluation

Neurological status was assessed using the Hunt–Hess grading system and the Glasgow Coma Scale (GCS). The amount of hemorrhage detected on CT scans was graded according to the Fisher classification. All patients underwent selective cerebral digital subtraction angiography (DSA) via femoral artery catheterization under local anesthesia. Vasospasm was diagnosed radiologically, and patients were divided into two groups:

- Group 1: No vasospasm
- Group 2: Vasospasm present

Laboratory Measurements

For each patient, 24-hour urine samples were collected to measure microalbumin levels, and the microalbumin-to-creatinine ratio was calculated from these 24-hour samples. Simultaneously, venous blood samples were obtained to measure blood urea nitrogen (BUN) and serum creatinine levels. Routine laboratory investigations included complete blood count, liver and renal function tests, coagulation profile, urinalysis, chest radiography, and electrocardiography.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 22.0 (IBM Corp., Armonk, NY, USA) at the Department of Biostatistics, Çukurova University Faculty of Medicine. Data distribution was tested for normality. Continuous variables with normal distribution were analyzed using Student's t-test or one-way ANOVA, while non-normally distributed variables were analyzed with the Mann–Whitney U or Kruskal–Wallis test. Categorical variables were compared using the chi-square test or Fisher's exact test as appropriate. Results were expressed as mean ± standard deviation, median (min–max), and percentages. A p-value of <0.05 was considered statistically significant.

Ethical Approval

This study was approved by the Ethics Committee of the Medical Faculty of Çukurova University (Date: 2010-05-11, No: TF2010LTP19).

Results

A total of 40 patients with aneurysmal subarachnoid hemorrhage were included in the study, of whom 20 developed vasospasm and 20 did not. The mean age of the patients was 50.3 ± 11.2 years, and no significant difference in age was observed between the vasospasm and non-vasospasm groups (48.7 ± 11.2 vs. 51.9 ± 11.3 years, p = 0.383). Similarly, sex distribution did not differ significantly between the groups (p = 0.744), with females representing 62.5% of the total cohort. In contrast, neurological grading showed clear differences: all patients without vasospasm were classified as Hunt–Hess grade I–II, whereas 90% of those with vasospasm were grade III–IV (p < 0.001). The mean Glasgow Coma Scale score was also significantly lower in patients with vasospasm compared with those without (13.1 ± 1.8 vs.

Table 1. Baseline characteristics of patients with aneurysmal subarachnoid hemorrhage according to vasospasm status

Variable	No Vasospasm (n = 20)	Vasospasm (n = 20)	Total (n = 40)	p-value
Sex, n (%)				
· Male	7 (35.0)	8 (40.0)	15 (37.5)	0.744
· Female	13 (65.0)	12 (60.0)	25 (62.5)	
Age (years)	51.9 ± 11.3	48.7 ± 11.2	50.3 ± 11.2	0.383
Hunt–Hess grade, n (%)				
· Grade I–II	20 (100.0)	2 (10.0)	22 (55.0)	<0.001
· Grade III–IV	0 (0.0)	18 (90.0)	18 (45.0)	
Glasgow Coma Scale	14.9 ± 0.4	13.1 ± 1.8	14.0 ± 1.6	<0.001
Fisher grade, n (%)				
· Grade 2	11 (55.0%)	2 (10.0%)	–	<0.001
Variable	**No Vasospasm (n = 20)**	**Vasospasm (n = 20)**	**Total (n = 40)**	**p-value**
· Grade 3–4	9 (45.0%)	18 (90.0%)	–	
BUN (mg/dL)	13.7 ± 5.1	12.9 ± 4.5	13.3 ± 4.8	0.675
Serum creatinine (mg/dL)	0.70 ± 0.26	0.74 ± 0.30	0.72 ± 0.28	0.645
Urinary microalbumin (mg/L)	2.0 ± 2.3	173.5 ± 208.4	87.7 ± 169.4	<0.001
Microalbumin/creatinine ratio	2.6 ± 2.6	310.9 ± 426.3	156.8 ± 336.0	<0.001

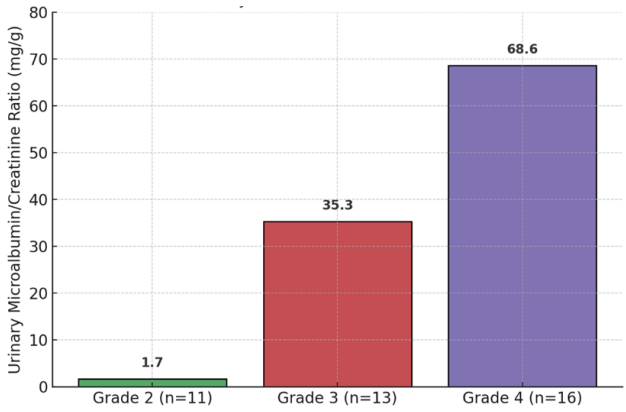

Figure 1. Relationship between Fisher grade and urinary microalbumin/creatinine ratio (mg/g). Error bars represent standard deviation. The difference between groups is statistically significant (p < 0.001)

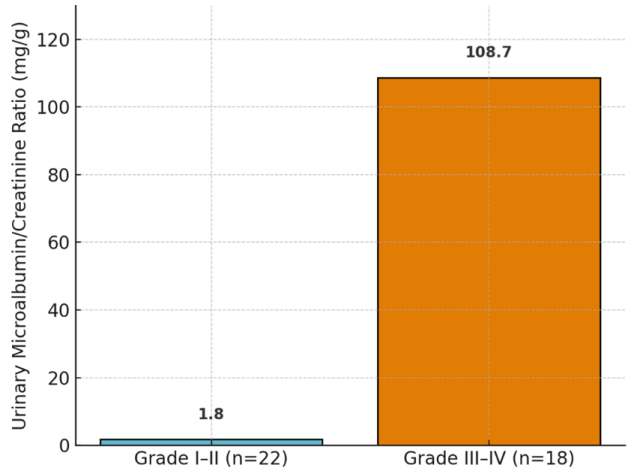

Figure 2. Relationship between Hunt–Hess grade and urinary microalbumin/creatinine ratio (mg/g). Error bars represent standard deviation. The difference between groups is statistically significant (p < 0.001)

Table 2. Comparison of clinical and laboratory parameters between the vasospasm and no vasospasm groups

Parameter	No Vasospasm (n=20)	Vasospasm (n=20)	p value
Day of hemorrhage (median, range)	3 (1–15)	5 (1–14)	0.054
Glasgow Coma Scale (mean ± SD)	14.9 ± 0.4	13.1 ± 1.8	<0.001
Urinary microalbumin (mg/L)	1.96 ± 2.33	173.5 ± 208.4	<0.001
BUN (mg/dL)	13.7 ± 5.1	12.9 ± 4.5	0.675
Serum creatinine (mg/dL)	0.70 ± 0.26	0.74 ± 0.30	0.645
Microalbumin/creatinine ratio (mg/g)	2.6 ± 2.6	310.9 ± 426.3	<0.001

14.9 ± 0.4, p < 0.001). Fisher grade analysis revealed that higher grades were predominantly associated with vasospasm (p < 0.001). Regarding laboratory parameters, no significant differences were found in BUN or serum creatinine levels between groups. However, urinary microalbumin levels (173.5 ± 208.4 vs. 2.0 ± 2.3 mg/L, p < 0.001) and the urinary microalbumin-to-creatinine ratio (310.9 ± 426.3 vs. 2.6 ± 2.6 mg/g, p < 0.001) were markedly elevated in patients with vasospasm (Table 1). When clinical and laboratory parameters were compared between groups, no significant difference was observed regarding the day of hemorrhage (median 5 vs. 3 days, p = 0.054), serum creatinine (0.74 ± 0.30 vs. 0.70 ± 0.26 mg/dL, p = 0.645), or BUN levels (12.9 ± 4.5 vs. 13.7 ± 5.1 mg/dL, p = 0.675). In contrast, patients with vasospasm had significantly worse neurological status, as reflected by lower Glasgow Coma Scale scores (13.1 ± 1.8 vs. 14.9 ± 0.4, p < 0.001). Moreover, urinary microalbumin levels were substantially higher in the vasospasm group compared with those without vasospasm (173.5 ± 208.4 vs. 2.0 ± 2.3 mg/L, p < 0.001). A similar pattern was seen for the urinary microalbumin-to-creatinine ratio, which was markedly elevated in patients with vasospasm (310.9 ± 426.3 vs. 2.6 ± 2.6 mg/g, p < 0.001) (Table 2). Analysis of laboratory parameters according to neurological grading demonstrated a strong association between urinary markers and disease severity. Patients with higher Hunt–Hess grades (III–IV) had significantly elevated urinary microalbumin levels (190.3 ± 213.5 vs. 3.8 ± 6.4 mg/L, p < 0.001) and microalbumin-to-creatinine ratios (341.7 ± 439.4 vs. 5.5 ± 9.6 mg/g, p < 0.001) compared with those with lower grades (I–II). In contrast, BUN and serum creatinine levels did not differ significantly between Hunt–Hess subgroups. A similar pattern was observed with Fisher grades: patients classified as grade 3 or 4 exhibited markedly higher urinary microalbumin (148.7 ± 207.8 and 134.2 ± 193.7 mg/L, respectively) and microalbumin-to-creatinine ratios (287.6 ± 448.5 and 176.5 ± 241.2 mg/g) compared with grade 2 patients (1.7 ± 1.9 mg/L and 2.3 ± 2.0 mg/g, respectively; both p < 0.001). Again, no significant differences were detected for BUN or serum creatinine across Fisher grades (Table 3, Figure 1-2).

Discussion

In this study, we investigated the association between urinary microalbumin-to-creatinine ratio and the occurrence of cerebral vasospasm in patients with aneurysmal subarachnoid hemorrhage (SAH). Our findings demonstrated that urinary microalbumin levels and, more importantly, the microalbumin-to-creatinine ratio were significantly higher in patients who developed vasospasm compared with those who did not. In addition, both Hunt–Hess and Fisher grades were strongly correlated with elevated urinary markers, whereas conventional renal function parameters such as blood urea nitrogen (BUN) and serum creatinine showed no significant relationship with vasospasm. These results suggest that urinary microalbumin and its ratio to serum creatinine may serve as useful, non-invasive biomarkers for predicting vasospasm risk following SAH.

We observed a clear parallel between neurological grading and the

Table 3. Association of laboratory parameters with Hunt–Hess and Fisher grades

Parameter	Hunt–Hess I–II (n = 22)	Hunt–Hess III–IV (n = 18)	p-value	Fisher Grade 2 (n = 11)	Fisher Grade 3 (n = 13)	Fisher Grade 4 (n = 16)	p-value
Urinary Microalbumin (mg/L)	3.8 ± 6.4 (median 1.3)	190.3 ± 213.5 (median 64.6)	<0.001*	1.7 ± 1.9 (med. 1.3)	148.7 ± 207.8 (med. 30.5)	134.2 ± 193.7 (median 54.1)	<0.001*
BUN (mg/dL)	13.7 ± 5.0	12.8 ± 4.5	0.663	14.2 ± 4.9	12.5 ± 5.0	13.6 ± 3.8	0.837
Serum creatinine (mg/dL)	0.70 ± 0.25	0.75 ± 0.31	0.644	0.73 ± 0.28	0.72 ± 0.32	0.71 ± 0.11	0.869
MCR (mg/g)	5.5 ± 9.6 (med. 1.8)	341.7 ± 439.4 (med. 108.7)	<0.001*	2.3 ± 2.0 (med. 1.7)	287.6 ± 448.5 (med. 35.3)	176.5 ± 241.2 (median 68.6)	<0.001*

MCR: Microalbumin/creatinine ratio

presence of vasospasm. Patients with Hunt–Hess grades III–IV had markedly higher rates of vasospasm compared with those with grades I–II. This is consistent with previous reports. In a clinical series, Rumalla et al. showed that vasospasm was present in 22.2% of grade I patients but in as many as 73.3% of grade V patients, indicating that increasing clinical severity strongly predisposes to vasospasm [9]. Yang et al. demonstrated higher mortality and morbidity rates among patients with diffuse vasospasm compared with those without vasospasm [10]. Our findings further support the prognostic value of neurological status at admission.

Beyond subarachnoid hemorrhage, urinary biomarkers such as microalbumin and the microalbumin-to-creatinine ratio have also been explored in ischemic and hemorrhagic stroke cohorts. Studies by Garcia-Garcia et al. and Zhao et al. demonstrated that elevated urinary microalbumin levels correlate with infarct size, blood–brain barrier disruption, and poor neurological recovery [11,12]. These findings suggest that urinary microalbuminuria reflects a broader pathophysiological mechanism involving systemic endothelial dysfunction and inflammatory activation. Our results extend this concept to aneurysmal SAH, highlighting that similar vascular injury pathways may underlie microalbuminuria across different cerebrovascular disorders.

One of the most novel aspects of our study is the evaluation of urinary microalbumin and its ratio to serum creatinine as predictors of vasospasm. We found that patients with vasospasm had markedly elevated microalbumin/creatinine ratios compared with those without vasospasm. Kedziora et al. reported that increased urinary albumin excretion was correlated with poor neurological status and lower Glasgow Coma Scale scores in aneurysmal SAH patients [13]. In another investigation, Garcia et al. compared urinary microalbumin-to-creatinine ratio, estimated glomerular filtration rate (eGFR), and cystatin C in stroke patients and concluded that the microalbumin/creatinine ratio was the most reliable biomarker of cerebrovascular injury [11]. Our study confirms and extends these findings, showing that the urinary microalbumin/creatinine ratio is not only associated with neurological deterioration but also strongly linked to angiographically confirmed vasospasm.

We also demonstrated that Fisher grade was significantly associated with urinary markers: patients with Fisher grades 3 and 4 had markedly higher microalbumin/creatinine ratios than those with Fisher grade 2. This aligns with the work of Aldrich et al., who showed that increasing clot burden on CT, particularly in the Sylvian fissure and basal cisterns, was associated with more severe vasospasm [14]. Wolfert et al. also reported that patients with diffuse SAH had vasospasm in 74% of cases, along with high rates of hydrocephalus [15]. Our findings are consistent with these observations and suggest that urinary markers may reflect the underlying inflammatory and hemodynamic burden associated with increasing hemorrhage grade.

The underlying mechanisms linking urinary microalbumin to vasospasm may involve systemic inflammatory responses. Albuminuria is considered an early marker of endothelial dysfunction and increased vascular permeability. Experimental and clinical studies have shown that acute cerebral injury triggers systemic inflammatory cascades that affect glomerular permeability, leading to transient microalbuminuria [12,16,17]. This is consistent with our observation that the microalbumin/creatinine ratio increased in parallel with clinical and radiological severity of SAH. Ganta et al. further demonstrated that in critically ill trauma patients, an elevated urinary microalbumin/creatinine ratio correlated positively with APACHE II, SAPS II, and MODS scores, and predicted worse prognosis [18]. Recent studies also emphasize the broader inflammatory role of albuminuria beyond renal dysfunction. Ma et al. reported strong correlations between albuminuria and systemic inflammation indices such as the systemic immune-inflammation ratio and neutrophil-to-albumin ratio [19]. Similarly, Kanbay et al. described albuminuria as a marker of endothelial injury and systemic inflammation independent of kidney disease [20]. Taken together, these data suggest that urinary microalbuminuria may be a surrogate of systemic inflammation and vascular injury in SAH-related vasospasm.

Our results highlight the potential clinical utility of monitoring urinary microalbumin/creatinine ratio as a simple, inexpensive, and non-invasive tool for predicting vasospasm risk. Unlike angiography, which is invasive, and transcranial Doppler, which may have operator-dependent variability, urinary markers can be obtained routinely and serially in intensive care settings. If validated in larger cohorts, this parameter could aid in early identification of patients at high risk of vasospasm, thereby facilitating timely interventions and potentially improving outcomes.

Limitations

Some limitations of our study should be acknowledged. The sample size was relatively small (n = 40), which may limit generalizability. We did not evaluate long-term outcomes, such as delayed cerebral ischemia or mortality, in relation to urinary markers. Although patients with known diabetes mellitus or hypertension were not excluded, these comorbidities were not systematically analyzed as potential confounding factors. Other conditions that may influence microalbuminuria, such as infections, were also not evaluated in detail. Future studies with larger, multicenter cohorts and longitudinal follow-up are warranted to confirm our findings and further explore the predictive value of urinary markers.

Conclusion

In conclusion, our study demonstrates that urinary microalbumin levels and the urinary microalbumin-to-creatinine ratio are significantly elevated in patients who develop cerebral vasospasm following aneurysmal subarachnoid hemorrhage. These parameters showed strong correlations with neurological severity (Hunt–Hess grade), radiological hemorrhage burden (Fisher grade), and clinical deterioration, whereas conventional renal markers such as BUN and serum creatinine were not predictive.

The findings suggest that urinary microalbumin and its ratio to serum creatinine may serve as simple, non-invasive biomarkers for early identification of patients at high risk of vasospasm. Incorporating these measurements into routine clinical practice could enable closer monitoring and earlier initiation of therapeutic strategies, potentially improving outcomes in this high-risk population. Future multicenter studies with larger sample sizes and long-term follow-up are warranted to validate these results and further clarify the prognostic value of urinary markers in SAH.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content, including study design, data collection, analysis and interpretation, writing, and some of the main line, or all of the preparation and scientific review of the contents, and approval of the final version of the article.

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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