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Uric acid levels as a predictor of cognitive function in the elderly

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ABSTRACT

This study aimed to examine the association between serum uric acid (SUA) levels and cognitive function among elderly individuals. An observational case-control study was conducted involving 37 elderly participants aged 60–75 years from As-Sakinah Elderly Integrated Health Post in Grendeng Sub-District, North Purwokerto, Indonesia. SUA levels were measured using a point-of-care testing (POCT) device, while cognitive function was assessed using the Montreal Cognitive Assessment–Indonesian version (MoCA-Ina). The mean SUA level was 5.52 ± 1.57 mg/dL, and the mean MoCA-Ina score was 19.05 ± 5.07, indicating that most participants demonstrated cognitive impairment. Pearson correlation revealed a non-significant negative association between SUA and cognitive scores ($r = -0.225$, $p = 0.180$). However, simple linear regression showed a significant inverse relationship ($B = -0.214$, $p < 0.001$), higher SUA levels were associated with lower cognitive performance. These findings suggest that while SUA may contribute to cognitive decline, it is not a standalone biomarker. Further longitudinal studies with larger samples are warranted to clarify its predictive value.

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INTRODUCTION

Cognitive function often declines with age, leading to impairments in memory, attention, reasoning, and orientation. Among older adults, this decline can range from normal age-related changes to more serious conditions such as mild cognitive impairment (MCI) and dementia, both of which are associated with reduced independence and increased risks of hospitalization and mortality (Hajek et al., 2025; Yuan et al., 2021). Therefore, early detection and monitoring of cognitive status in the elderly are essential for delaying disease progression and improving quality of life.

Various biological and metabolic factors have been investigated in relation to cognitive decline in older populations, one of which is serum uric acid (SUA) level. Uric acid is the final product of purine metabolism in the human body and exhibits both antioxidant and pro-oxidant properties. As a major plasma antioxidant, UA can neutralize free radicals such as peroxynitrite and

hydroxyl radicals, inhibit lipid peroxidation, and protect the integrity of the endothelium and the blood–brain barrier (Mijailovic et al., 2022). However, at elevated levels, UA may act as a pro-oxidant by activating NADPH oxidase, increasing intracellular ROS production, reducing nitric oxide bioavailability, and activating the NLRP3 inflammasome, which triggers chronic inflammation (Du et al., 2024) . These processes contribute to endothelial dysfunction, atherosclerosis, reduced cerebral blood flow, and neuroinflammation, ultimately accelerating cognitive decline in the elderly. This relationship is complex and context-dependent, with epidemiological evidence indicating a U-shaped association between UA levels and cognitive function (Mijailovic et al., 2022).

It may serve as a neuroprotective agent by neutralizing free radicals and reducing oxidative damage in neural tissues (Niu et al., 2020). Geng et al. (2022) reported a positive association between SUA levels and cognitive scores in older American adults. Conversely, hyperuricemia has been linked to vascular dysfunction, inflammation, and metabolic disturbances that may adversely affect cognitive performance (Du et al., 2024). Khaled et al. (2023) found that elevated SUA levels were associated with poorer cognitive function among the elderly, indicating potential vascular or inflammatory mechanisms. Perna et al. (2016) emphasized that such findings remain inconsistent across different population groups. These contradictory results underscore the need for further research, particularly in diverse population settings.

SUA levels in both clinical and research settings can be measured using various methods, including automated laboratory analyzers, enzymatic colorimetric assays, and point-of-care testing (POCT) devices. In the present study, SUA levels were measured using a POCT device, which provides rapid results from a small capillary blood sample obtained via finger prick. This method is particularly practical for field studies involving elderly participants, as it minimizes discomfort and eliminates the need for venipuncture (Paraskos et al., 2016).

Cognitive function in older adults can be evaluated using standardized neuropsychological tools. The Montreal Cognitive Assessment (MoCA) is widely utilized to detect mild cognitive impairment. Its Indonesian version, the MoCA-Ina, has been culturally adapted and validated for use among the elderly population in Indonesia. The MoCA-Ina assesses multiple cognitive domains—including memory, executive function, attention, language, visuospatial skills, abstraction, and orientation—with a maximum score of 30 points. Scores below 26 generally indicate cognitive impairment (Rambe & Fitri, 2017).

This study was conducted to examine the correlation between SUA levels and cognitive function in older adults. The findings are expected to contribute to a better understanding of the role of uric acid as a potential predictor of cognitive decline in the Indonesian elderly population.

RESEARCH METHOD

Study Design, Population, and Sample

This study employed an observational case-control design and was conducted from June to July 2025 at the As-Sakinah Elderly Community Health Post (Posyandu Lansia As-Sakinah) in the Grendeng District, North Purwokerto, Indonesia. The objective was to assess the correlation between levels of SUA and cognitive performance.

The study population comprised elderly individuals who visited the As-Sakinah Elderly Community Health Post during the study period and met the following criteria for inclusion: 1) Aged between 60 and 75 years; 2) Willing to participate and provide informed consent; 3) Able to communicate effectively and follow instructions during the assessment; 3) No diagnosed mental disorders such as schizophrenia or major depression; 4) No previous diagnosis of major neurological impairments (e.g., stroke, Parkinson's disease, Alzheimer's disease) that could interfere with cognitive testing; and 5) No severe visual or hearing impairments that could hinder administration of the MoCA-Ina. The exclusion criteria included: 1) Presence of acute illness during the data collection period; 2) Ongoing treatment with medications known to significantly affect SUA levels

(e.g., high-dose diuretics, allopurinol), unless treatment had been stable for ≥3 months; and 3) Refusal to complete the required examinations. A total of 37 elderly participants were enrolled in the study.

Data Collection Procedures

Measurement of Serum Uric Acid

Serum uric acid (SUA) levels were measured using a point-of-care testing (POCT) device (Easy Touch GCU). Capillary blood samples were obtained via finger-prick using sterile lancets. Participants were advised to fast for at least 8 hours before testing to minimize postprandial variation. The POCT method provides immediate quantitative SUA results in mg/dL, facilitating on-site data collection.

Assessment of Cognitive Performance

Cognitive performance was evaluated with the Indonesian edition of the Montreal Cognitive Assessment (MoCA-Ina), a standardized and validated instrument for identifying mild cognitive impairment among elderly individuals in Indonesia (Rambe & Fitri, 2017). This tool evaluates multiple cognitive domains, such as visuospatial and executive function, naming, memory, attention, language, abstract reasoning, delayed retrieval, and orientation. The total possible score is 30; scores below 26 may indicate potential cognitive impairment (Nasreddine et al., 2005).

Additional Data

Sociodemographic data (age, sex), health-related characteristics (history of hypercholesterolemia, hypertension, diabetes mellitus), and the frequency of exercise were collected through structured interviews.

Data Analysis

The collected data were examined through SPSS Statistics version 26.0. Serum uric acid (SUA) levels and MoCA-Ina scores were condensed into averages and standard deviations, while the categorical variables were displayed as frequencies and percentages. Normality of the data was examined using the Shapiro–Wilk test. Pearson's correlation analysis was employed to investigate the link between SUA levels and cognitive function. Additionally, simple linear regression was carried out to evaluate whether SUA could predict cognitive performance. A p-value below 0.05 was considered indicative of statistical significance in all tests.

RESULTS AND DISCUSSIONS

Participants Characteristics

A total of 37 elderly individuals participated in this study, comprising 7 men (18.9%) and 30 women (81.1%). The predominance of female participants reflects the generally higher life expectancy of women compared to men. This demographic trend has been consistently reported in various epidemiological studies and may influence both the prevalence and risk factors of cognitive decline, particularly due to sex-related differences in hormonal profiles, cardiovascular health, and longevity (Wang et al., 2024).

The majority of participants (48.7%) were aged 60–65 years, followed by those aged 71–75 years (27.0%) and 66–70 years (24.3%). Advancing age is closely linked to deterioration in various cognitive domains, such as memory, executive functioning, processing speed, and attentional capacity (Depestele et al., 2020; Jiang et al., 2025; Zihl & Reppermund, 2023). The inclusion of a substantial proportion of younger elderly individuals is thought to have contributed to the relatively low overall prevalence of cognitive impairment observed in this study.

Regarding metabolic and health-related characteristics, most participants had normal cholesterol levels (n = 26, 70.3%), while 11 participants (29.7%) were identified with hypercholesterolemia. A history of hypertension was reported by 21 participants (56.8%), whereas

16 participants (43.2%) had no such history. Diabetes mellitus was present in 8 participants (21.6%), while 29 (78.4%) had no history of the condition.

In terms of physical activity, the majority of participants (n = 27, 73.0%) reported exercising only occasionally. Meanwhile, 6 participants (16.2%) engaged in frequent physical activity, and 4 participants (10.8%) exercised regularly (Table 1).

Table 1. Demographic characteristics of participants (n = 37)

Variable	Category	n	%
Age (years old)	60-65	18	48.7
	66-70	9	24.3
	70-75	10	27.0
Sex	Male	7	18.9
	Female	30	81.1
Hypercholesterolemia	Yes	11	29.7
	No	26	70.3
Hypertension	Yes	21	56.8
	No	16	43.2
Diabetes mellitus	Yes	8	21.6
	No	29	78.4
Exercise frequency	Regular	4	10.8
	Often	6	16.2
	Occasionally	27	73.0

Hypercholesterolemia was identified in 29.7% of participants. Elevated cholesterol levels are known to contribute to atherosclerosis and cerebrovascular disease, both of which are associated with vascular cognitive impairment (Livingston et al., 2020). In addition to dyslipidemia, hypertension also emerged as a prevalent condition among the participants. More than half among the participants (56.8%) indicated a prior diagnosis of hypertension, a primary risk element for cognitive decline, particularly vascular neurocognitive disorder (Huang & Aronow, 2024). Alongside these conditions, diabetes mellitus was present in 21.6% of participants. This metabolic disorder is strongly correlated with elevated risk of cognitive dysfunction and neurodegenerative disorders through multiple mechanisms, including chronic hyperglycemia, insulin resistance, and microvascular damage in the brain (Khaliq et al., 2025; Szablewski, 2025). The coexistence of diabetes and hyperuricemia may exert additive or synergistic effects on cognitive function due to shared vascular and metabolic pathways.

In terms of lifestyle factors, the majority of participants (73.0%) reported engaging in physical activity only occasionally, while only 10.8% exercised regularly. Physical inactivity has been consistently linked to reduced cognitive performance in elderly individuals, as regular aerobic activity is known to improve cerebral blood flow, stimulate neurogenesis, and reduce vascular risk factors (Brouwer et al., 2021; Tomoto et al., 2023; Vidyanti et al., 2025). These findings underscore the importance of lifestyle modification—particularly increasing physical activity—as a possible approach for promoting cognitive health in this population.

Taken together, the demographic and health profiles of this study population revealed several overlapping determinants of cognitive impairment, including advanced age, a predominance of female participants, cardiometabolic comorbidities, and low levels of physical activity. These factors should be carefully considered as potential confounders or effect modifiers in the observed association between SUA levels and cognitive function.

Serum Uric Acid (SUA) Levels and MoCA-Ina Scores

The mean serum uric acid level was 5.52 ± 1.57 mg/dL (range 2.0–9.1 mg/dL), and the mean MoCA-Ina score was 19.05 ± 5.07 (range 10–29). These results suggest that, on average, participants had uric acid levels within the normal reference range but scored below the threshold for normal cognitive function (≥ 26) (Table 2).

Table 2. Descriptive statistics

Variable	n	Min	Max	Mean	SD
Serum uric acid (mg/dL)	37	2.0	9.1	5.52	1.57
Cognitive Score (MoCA-Ina)	37	10	29	19.05	5.07

The descriptive analysis of this study revealed that the mean serum uric acid (SUA) level among elderly participants was 5.52 ± 1.57 mg/dL, with values ranging from 2.0 to 9.1 mg/dL. This means it falls within the normal physiological range for adults—3.4–7.0 mg/dL in men and 2.4–6.0 mg/dL in women (Ali et al., 2019). However, the broad distribution indicates substantial interindividual variability, with some participants exceeding the threshold for hyperuricemia. This variation may be influenced by several factors, including dietary habits, renal function, comorbidities (e.g., hypertension and diabetes), and lifestyle behaviors (Maiuolo et al., 2016). Regarding cognitive performance, the mean MoCA-Ina score was 19.05 ± 5.07, with individual scores ranging from 10 to 29. Given that a MoCA-Ina score of 26 or above is typically regarded as reflecting normal cognitive performance (Nasreddine et al., 2005), the majority of participants in this sample exhibited signs of cognitive impairment. This finding aligns with epidemiological data reporting elevated rates of cognitive impairment among older individuals, particularly in community-dwelling populations with multiple vascular and metabolic risk factors (Livingston et al., 2020).

The co-occurrence of normal average SUA levels and reduced average cognitive scores raises important considerations. Although elevated uric acid has traditionally been linked to gout and cardiovascular risk, emerging evidence suggests a more nuanced role in neurodegeneration. On one hand, uric acid plays a key antioxidant role by eliminating reactive oxygen and nitrogen species, thereby offering potential neuroprotection (Ashok et al., 2022; Niu et al., 2020). On the other hand, chronic hyperuricemia may promote inflammation, oxidative stress, and endothelial dysfunction—all of which contribute to accelerated cognitive decline (Khaled et al., 2023).

The relatively common incidence of cognitive impairment observed in this study is likely influenced not solely by SUA levels, but also by a constellation of factors, including advanced age, comorbid conditions such as hypertension, hypercholesterolemia, and diabetes, as well as low levels of physical activity—all recognized contributors to cognitive deterioration (Mone et al., 2022; Pillai et al., 2021). These data affirm the importance of considering SUA as part of a broader constellation of interrelated risk factors, rather than as an isolated biomarker of cognitive decline.

Association between Serum Uric Acid (SUA) Levels and MoCA-Ina Scores

The Shapiro–Wilk test indicated that both serum uric acid and MoCA-Ina scores were normally distributed ($p > 0.05$), justifying the use of Pearson's correlation test (Table 3).

Table 3. Saphiro-Wilk normality test

Variable	W statistic	p-value
Serum uric acid	0.99	0.95
Cognitive Score	0.96	0.17

Pearson's correlation analysis showed a negative but non-significant association between SUA levels and MoCA-Ina scores ($r = -0.225$, $p = 0.180$), suggesting that higher uric acid levels tended

to be related with lower cognitive scores, although the relationship was statistically non-significant (Table 4).

Table 4. Pearson's correlation between uric acid and MoCA-Ina scores

Variable 1	Variable 2	r	p-value
Serum uric acid	MoCA-Ina Score	-0.225	0.180

The Pearson correlation analysis conducted in this study showed a weak, negative, and non-statistically significant association between SUA levels and MoCA-Ina scores ($r = –0.225$, $p = 0.180$). These findings indicate a possible tendency for higher uric acid levels to be linked with reduced cognitive function in older adults, although this association was not statistically significant within the study sample.

The direction of the correlation is consistent with several prior studies that have proposed a detrimental effect of elevated uric acid on cognitive health. Chronic hyperuricemia has been associated with increased oxidative stress, inflammation, endothelial dysfunction, and reduced cerebral blood flow—all of which can contribute to neurodegeneration and cognitive decline (Khaled et al., 2023; Perna et al., 2016). Moreover, uric acid may exacerbate microvascular damage in individuals with comorbid conditions such as hypertension or diabetes, both of which were common in the present study population.

However, the lack of statistical significance could be attributed to several factors. First, the sample count ($n = 37$) may not have been sufficient to detect small to moderate associations. Second, the variability in uric acid levels was relatively limited, with the majority of participants falling within the normal reference range. Third, the complexity of cognitive aging may mean that multiple interacting variables—including age, sex, comorbidities, and lifestyle factors—affect cognition more strongly than uric acid alone. Interestingly, some studies have reported a neuroprotective role for uric acid, particularly in the early stages of cognitive decline or in specific target groups, for instance, patients with Parkinson's disease or Alzheimer's disease (Geng et al., 2022). In those contexts, uric acid acts as a natural antioxidant, scavenging reactive oxygen and nitrogen species and reducing stress of oxidative in the brain.

The current findings, while not statistically significant, support the hypothesis that higher uric acid may be linked to poorer cognitive performance, at least in a community-based elderly population. However, the weak strength of the association and the absence of statistical significance indicate that uric acid alone is not a reliable predictor of cognitive function. In contrast to our results, several studies conducted in other countries have reported a positive relationship between serum uric acid and cognitive function in older adults. Latourte et al. (2018) observed that older French adults with higher UA levels had a lower risk of dementia over a 12-year follow-up, supporting the hypothesis that UA may help counteract oxidative stress in the brain. These findings appear to contrast with the present study, which highlights the potential adverse effects of elevated UA, particularly in the context of vascular and inflammatory risk factors prevalent among Indonesian elderly. Such discrepancies may be explained by differences in population characteristics, comorbidity profiles, dietary patterns, and baseline UA levels, as well as methodological variations in cognitive assessment. This emphasizes the importance of interpreting UA's role in cognitive health within the specific socio-demographic and clinical context of each population. Future studies with larger samples and multivariate models must clarify whether uric acid plays a causal or merely associative role in cognitive aging.

A basic linear regression test was carried out to examine the relationship between MoCA-Ina cognitive scores (independent variable) and SUA levels (dependent variable). The results demonstrated a statistically significant inverse association ($B = –0.214$, $p < 0.001$), indicating that for every 1-point decrease in MoCA-Ina score, serum uric acid level increased by 0.214 mg/dL (Table 5). The model explained 34.6% of the variance in uric acid levels ($R^2 = 0.346$) (Table 6).

Table 5. Simple linear regression analysis between MoCA-Ina score and uric acid levels

Variable 1	B	SE	Beta	t	p-value
Constant	9.56	0.98	-	9.72	0.000
MoCA-Ina score	-0.21	0.05	-0.59	-4.30	0.000

Table 6. Model summary of regression analysis

R	R^2	Adjusted R^2	SEE
0.59	0.35	0.33	1.33

The simple linear regression analysis demonstrated a statistically notable negative correlation between MoCA-Ina scores and SUA levels ($B = –0.214$, $p < 0.001$), indicating that for every 1-point decrease in cognitive score, serum uric acid level increased by approximately 0.214 mg/dL. The model explained 34.6% of the variance in uric acid levels ($R^2 = 0.346$), which is considerable for a single-predictor model in a biological and behavioral context.

The negative coefficient aligns with findings from studies suggesting that elevated uric acid is linked to poorer cognitive outcomes, potentially through several pathophysiological mechanisms. These include oxidative stress induction, systemic inflammation, and endothelial dysfunction, all of which may impair cerebral perfusion and contribute to neurodegenerative processes (Khaled et al., 2023). Chronic hyperuricemia has also been associated with microvascular brain injury, which could explain the observed association in this study's elderly (Xu et al., 2023).

From a statistical perspective, the R^2 value of 0.346 suggests that cognitive performance accounts for about one-third of the variability in uric acid levels in this sample, leaving the remaining two-thirds potentially explained by other factors such as diet, renal function, genetic predisposition, comorbidities (hypertension, diabetes, dyslipidemia), and medication use. This reinforces the multifactorial nature of uric acid regulation and cognitive aging.

Despite the findings, some constraints need to be considered. Causal inferences cannot be made due to the cross-sectional design —whether high uric acid contributes to cognitive decline, or whether cognitive impairment leads to metabolic changes influencing uric acid levels, remains unclear. Additionally, the limited number of participants potentially reduces the external validity of the study and raises the chance of overestimating the strength of the association. Despite these limitations, the regression results highlight a potentially important link between serum uric acid and cognitive health in the elderly population. If confirmed in longitudinal studies, uric acid measurement could be incorporated into multimodal risk assessment models for early detection of cognitive decline, particularly when combined with other metabolic and vascular biomarkers.

Overall, findings from the current study reveal that higher SUA levels are linked to lower cognitive function in older individuals as measured by the MoCA-Ina. While regression analysis demonstrated a statistically significant relationship, correlation analysis did not reach significance, likely due to sample size limitations. This research design does not support causal conclusions due to its cross-sectional approach, and the relatively small, community-based sample may not fully represent the broader elderly population. Furthermore, unmeasured confounding factors—such as diet, renal function, and genetic predisposition—may influence SUA levels and cognitive performance. Further investigations should adopt long-term study designs with increased sample sizes, greater demographic diversity, and explore multimarker approaches that integrate uric acid with other metabolic, inflammatory, and neuroimaging measures to improve the prediction and understanding of cognitive decline in aging populations.

CONCLUSION

This research revealed that higher SUA levels were significantly linked to lower cognitive function in older adults, as measured by the MoCA-Ina. While higher SUA levels were associated with lower

cognitive scores in regression analysis, correlation analysis did not reach statistical significance. Upcoming long-term investigations involving larger participant groups and multimarker models should be conducted to clarify causality and improve predictive accuracy.

This study suggests that monitoring serum uric acid could be integrated into routine geriatric check-ups to support early detection of cognitive decline. Maintaining UA within an optimal range may help reduce dementia risk, making it important to pair UA testing with cognitive screening and cardiovascular risk assessment. Public health programs promoting healthy lifestyles and training healthcare providers to recognize abnormal UA levels as a modifiable risk factor could strengthen national healthy aging strategies.

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