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Original Research Article

Correlation of Blood Sodium and Potassium Levels with The Extent of Stroke

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Abstract:

Background: Stroke is a leading cause of disability, often resulting in motor and neurological impairments. Electrolyte disturbances, particularly in sodium, potassium, and calcium, may influence stroke severity and outcomes. This study aimed to evaluate the association between serum electrolyte levels and functional outcomes in ischemic stroke patients.

Methods: A prospective study was conducted over one year at a tertiary care hospital including 168 adult ischemic stroke patients. Stroke severity and motor function were assessed using NIHSS and MAS scores. Serum sodium, potassium, and calcium levels were measured at admission. The primary outcome was death or major disability at 3 months (mRS 3–6).

Results: Patients with death or major disability were older (74.2 vs. 66.5 years) and had higher NIHSS scores (median 6 vs. 3) and lower MAS scores (median 15 vs. 20). Abnormal calcium levels were significantly associated with adverse outcomes ($p = 0.01$), while sodium and potassium showed no significant correlation ($p = 0.12$ and 0.43).

Conclusion: Calcium disturbances are linked to worse functional outcomes in ischemic stroke. Monitoring and correcting calcium levels may help improve prognosis.

Keywords: Ischemic Stroke, Serum Electrolytes, Calcium, Potassium, Sodium, Stroke Severity, mRS.

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Introduction

Stroke is a clinical condition characterized by sudden onset of focal or global neurological deficits lasting more than 24 hours or leading to death, caused by vascular abnormalities in the brain [1]. It arises from either blockage or rupture of cerebral blood vessels, resulting in ischemic or hemorrhagic stroke, respectively [2]. Stroke is one of the leading causes of adult disability worldwide [3], with motor impairments being the most common consequence. Approximately 80% of stroke survivors experience motor deficits, which manifest as reduced muscle strength, loss of motor control, or limitations in voluntary movement [4]. The Motor Assessment Scale (MAS) is a reliable and valid instrument widely used to evaluate motor function in stroke patients. It is simple, rapid, and effective in assessing motor outcomes [5].

In addition to motor dysfunction, hospitalized stroke patients frequently develop electrolyte imbalances. A study by Kembuan and Sekeon (2014) reported that 45.9% of acute stroke patients experienced disturbances in electrolytes such as sodium, potassium, and calcium [6]. These imbalances are associated with increased mortality compared to patients with normal electrolyte levels [7]. In ischemic stroke, electrolyte disturbances can result from cellular changes caused by hypoxia in affected neurons [8]. Electrolytes, particularly sodium, potassium, and calcium, are essential for muscle contraction and proper neuromuscular function [9].

Given the critical role of electrolytes in muscle function, this study aims to investigate the relationship between serum sodium, potassium, and calcium levels in patients with ischemic stroke. Understanding this association could help predict motor recovery and inform clinical management of stroke patients.

Materials and Methods

Study Participants: This prospective study was conducted over one year at a tertiary care hospital. A total of 168 adult patients with confirmed ischemic stroke admitted within seven days of symptom onset were enrolled. Patients with cancer

or missing blood samples were excluded. Written informed consent was obtained from all participants or their immediate family members, and the study was approved by the hospital ethics committee.

Data Collection: Baseline demographic information, medical history, and clinical characteristics were recorded at admission. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS), and motor function was evaluated with the Motor Assessment Scale (MAS). Blood pressure and other routine clinical measurements were recorded according to standard hospital protocols.

Serum Electrolyte Measurements: Fasting blood samples were collected within 24 hours of admission. Serum sodium, potassium, and calcium levels were measured using automated laboratory analyzers. Laboratory personnel were blinded to patients' clinical outcomes. Electrolyte levels were classified as normal or abnormal according to standard reference ranges.

Outcome Assessment: The primary outcome was death or major disability at 3 months after ischemic stroke, defined by a modified Rankin Scale (mRS) score of 3–6. Participants were followed up in person at 3 months, and both mRS and MAS scores were recorded to assess functional and motor outcomes.

Statistical Analysis: Descriptive statistics were used to summarize baseline characteristics, serum electrolyte levels, and outcome measures. Comparisons between groups with and without death or major disability were performed, and a p-value <0.05 was considered statistically significant.

Results

The baseline characteristics of the 168 study participants are summarized in Table 1. Participants who experienced death or major disability at 3 months, defined as mRS scores of 3-6, were significantly older (74.2 ± 10.9 years) compared to those without the primary outcome (66.5 ± 11.8 years, $p < 0.001$). The group with adverse outcomes also had higher baseline NIHSS scores (median 6 vs. 3, $p < 0.001$) and lower median MAS scores (15 vs. 20, $p = 0.02$), indicating more severe neurological and motor impairments. Other characteristics, including sex distribution, history of hypertension, and diabetes, showed no statistically significant differences between the two groups.

Table 1: Characteristics of Study Participants According to Death or Major Disability at 3 Months (mRS 3–6)

Characteristics	Without Death/Major Disability (n=98)	With Death/Major Disability (n=70)	P value
Age, years (mean ± SD)	66.5 ± 11.8	74.2 ± 10.9	<0.001
Male sex, n (%)	55 (56.1)	38 (54.3)	0.78
History of hypertension, n (%)	62 (63.3)	48 (68.6)	0.42
History of diabetes, n (%)	28 (28.6)	25 (35.7)	0.34
Baseline NIHSS score, median (IQR)	3 (2–5)	6 (4–9)	<0.001
MAS score, median (range)	20 (5–48)	15 (4–42)	0.02*

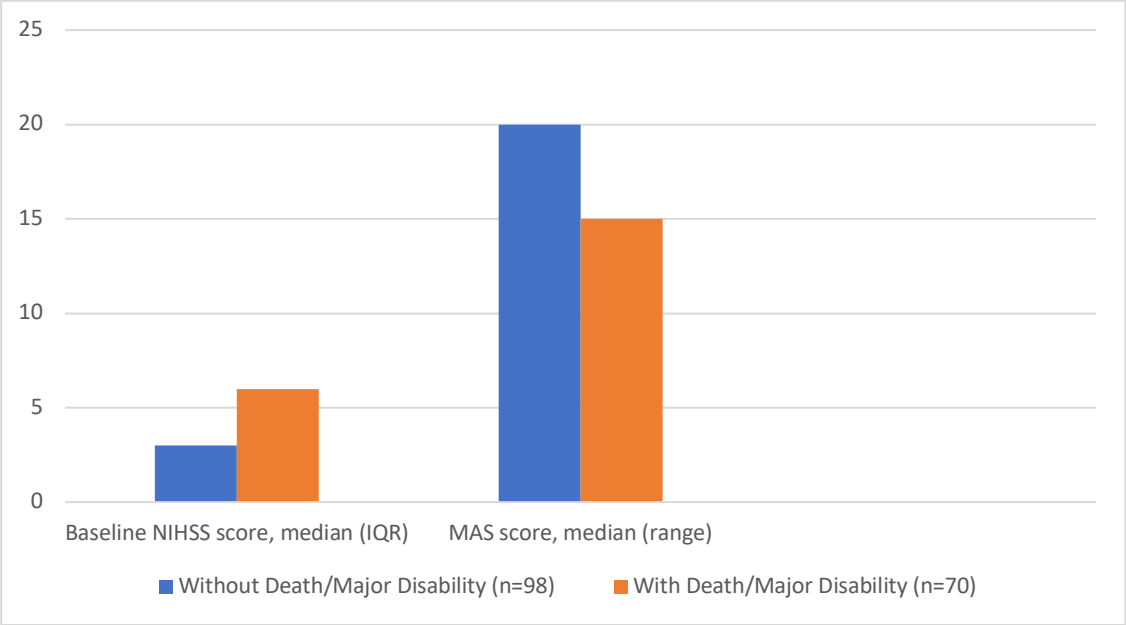

Figure 1: NIHSS and MAS Scores in Patients with and Without Death or Major Disability at 3 Months.

The relationship between serum electrolyte levels and stroke severity is presented in Table 2. Among the participants, abnormal calcium levels were significantly associated with higher rates of death or major disability at 3 months (54.9% vs. 45.1%, p = 0.01). In contrast, serum sodium and potassium levels did not show a significant association with adverse outcomes (p = 0.12 and p = 0.43, respectively). These findings suggest that calcium disturbances may be linked to worse functional outcomes, whereas sodium and potassium levels were not significantly correlated with stroke severity in this cohort.

Table 2: Serum Electrolyte Levels According to Stroke Severity (mRS 3–6)

Electrolyte	Normal Level n (%)	Abnormal Level n (%)	Primary Outcome (mRS 3–6), n (%)	P value
Sodium	140 (83.3)	28 (16.7)	20/28 (71.4%)	0.12
Potassium	138 (82.1)	30 (17.9)	22/30 (73.3%)	0.43
Calcium	86 (51.2)	82 (48.8)	45/82 (54.9%)	0.01*

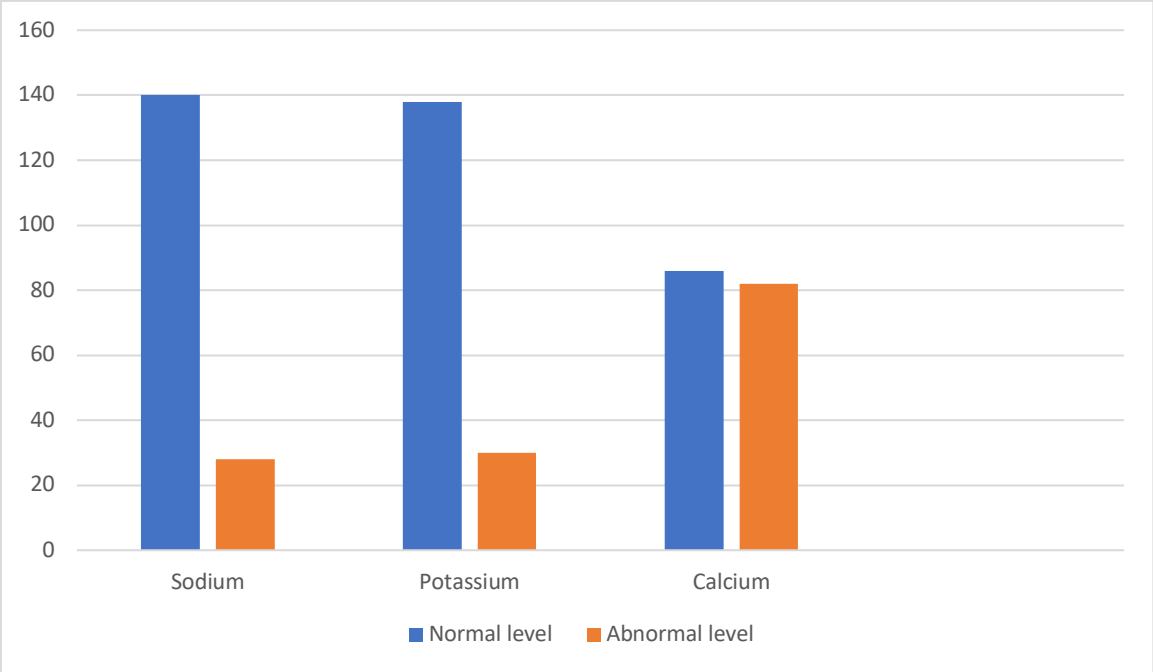

Figure 2: Proportion of Patients with Death or Major Disability (mRS 3–6) According to Normal and Abnormal Serum Sodium, Potassium, and Calcium Levels

Discussion

In this prospective study of 168 patients with ischemic stroke, we observed that patients with normal or higher levels of serum potassium, calcium, and magnesium at baseline tended to have better outcomes at 3 months, with lower rates of death or major disability as measured by mRS 3–6. These associations were most pronounced for calcium, which showed a statistically significant correlation with motor and functional outcomes, while potassium and magnesium showed trends toward protective effects. These findings suggest that these electrolytes may play an important role in stroke prognosis and could serve as accessible biomarkers for identifying patients at risk of poor outcomes [10-13].

Potassium, calcium, and magnesium are critical macrominerals involved in vascular function, thrombosis, inflammation, oxidative stress, and neuronal protection. Animal studies have demonstrated that higher potassium levels may reduce cerebral injury and apoptosis, while calcium and magnesium exert neuroprotective effects during ischemic events [14,15]. Epidemiological data also suggest that low potassium, calcium, or magnesium levels may increase the risk of ischemic stroke or worsen outcomes, supporting our observations [16,17].

Previous clinical studies examining these electrolytes and stroke prognosis have produced inconsistent results, often limited by small sample sizes [18-20]. Some studies reported that hypokalemia is associated with poorer functional outcomes [18], while elevated calcium has been linked to higher mortality in stroke patients [19]. Others found that low magnesium increases the risk of hemorrhagic complications after thrombolysis or thrombectomy [19,20], whereas some studies did not find significant associations for calcium or

magnesium [21-23]. Our study, despite a smaller sample than large cohorts, provides additional evidence that higher baseline electrolyte levels, particularly calcium, are associated with favorable short-term outcomes in ischemic stroke.

Several biological mechanisms may explain these protective effects. Potassium, calcium, and magnesium help regulate vascular tone, reduce blood pressure, and modulate smooth muscle contraction [24]. They also reduce vascular inflammation by inhibiting nuclear factor κB activation [25,26]. High potassium may suppress smooth muscle proliferation and platelet aggregation, decreasing the risk of atherosclerosis and thrombosis [27]. Magnesium supplementation can mitigate oxidative stress, inflammation, and endothelial dysfunction [24]. Elevated calcium may protect ischemic brain tissue by modulating excitotoxic pathways, potentially improving functional recovery [28]. Further studies are needed to clarify the detailed mechanisms behind these associations.

Our findings have important clinical implications. Patients with low baseline potassium, calcium, or magnesium levels may require closer monitoring and targeted interventions to improve outcomes. Evidence from animal studies suggests that supplementation of these electrolytes can reduce infarct size and improve recovery, highlighting a potential avenue for therapeutic intervention [24,29].

This study has some limitations. First, dietary intake of potassium, calcium, and magnesium was not assessed, which may introduce confounding. Second, serum levels were measured only once at admission, so the effects of dynamic changes over time were not captured. Finally, as all participants were from a single hospital, the generalizability of our results to other populations is limited. Future prospective studies with repeated measurements and diverse populations are warranted to confirm these findings.

Conclusion

This study demonstrates that statin therapy is both effective and safe in dyslipidemic patients with coexisting cardiovascular disease and non-alcoholic fatty liver disease. Statin use was associated with significant reductions in serum liver enzymes (ALT and AST) over the follow-up period, indicating an improvement in hepatic biochemical status without evidence of clinically significant hepatotoxicity. In addition, statins effectively improved lipid parameters, reinforcing their central role in cardiovascular risk reduction. These findings support the continued and judicious use of statins in dyslipidemic patients with NAFLD and highlight their potential dual benefit in addressing both metabolic liver dysfunction and cardiovascular disease. Further large-scale and long-term studies are warranted to confirm these findings and explore their impact on long-term hepatic outcomes.

References

1. Kao WT, Parnes LS, Chole RA. Otoconia and otolithic membrane fragments within the posterior semicircular canal in BPPV. Laryngoscope. 2017;127(3):709–14.
2. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Culebras A, Elkind MSV, et al. An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013; 44:2064–89.
3. Siddeswari R, Suryanarayana B, Sudarsi B, Manohar S, Rao N, Abhilash T. Comparative study of risk factors and lipid profile patterns in ischemic and hemorrhagic stroke. J Med Allied Sci. 2016;6(1):8.
4. Langhorne P, Coupar F, Pollock A. Motor recovery after stroke: a systematic review. Lancet Neurol. 2009;8(8):741–54.
5. Brauer SG, Bew PG, Kuys SS, Lynch MR, Morrison G. Prediction of discharge destination after stroke using the Motor Assessment Scale on admission: a prospective, multisite study. Arch Phys Med Rehabil. 2008;89(6):1061–5.
6. Kembuan AHNM, Sekeon SAS. Electrolyte disturbances among acute stroke patients in Manado. Glob J Med Public Health. 2014;3(1):1–6.
7. Kalyan M, Nahdi WK, Kanitkar SA, Moharkar A, Saha R. Electrolyte imbalance in acute stroke. NIJRM. 2017;8(4):2230–9969.
8. Atri A. Ischemic stroke: pathophysiology and principles of localization. Neurology. 2009;13(1):1–15.
9. Aksoy D, Ayan M, Alatli T, Sahin F, Ozdemir MB, Cevik B, et al. Clinical and demographic characteristics of acute stroke patients admitted to the emergency department of a tertiary referral center. J Appl Environ Microbiol. 2014;13(1):135–8.
10. Stone MS, Martyn L, Weaver CM. Potassium intake, bioavailability, hypertension, and glucose control. Nutrients. 2016; 8:444.
11. Peterlik M, Kallay E, Cross HS. Calcium nutrition and extracellular calcium sensing: relevance for the pathogenesis of osteoporosis, cancer, and cardiovascular diseases. Nutrients. 2013; 5:302–27.
12. Grober U, Schmidt J, Kisters K. Magnesium in prevention and therapy. Nutrients. 2015; 7:8199–8226.
13. Mohammadifard N, Gotay C, Humphries KH, Ignaszewski A, Esmaillzadeh A, Sarrafzadegan N. Electrolyte minerals intake and

cardiovascular health. Crit Rev Food Sci Nutr. 2019; 59:2375–85.
14. Dibaba DT, Xun P, Fly AD, Bidulescu A, Tsinovoi CL, Judd SE, et al. Calcium intake and serum calcium level in relation to the risk of ischemic stroke: findings from the REGARDS study. J Stroke Cerebrovasc Dis. 2019; 28:312–23.
15. Ohira T, Peacock JM, Iso H, Chambless LE, Rosamond WD, Folsom AR. Serum and dietary magnesium and risk of ischemic stroke: the Atherosclerosis Risk in Communities study. Am J Epidemiol. 2009; 169:1437–44.
16. Li N, Qin S, Xie L, Qin T, Yang Y, Fang W, et al. Elevated serum potassium concentration alleviates cerebral ischemia–reperfusion injury via mitochondrial preservation. Cell Physiol Biochem. 2018; 48:1664–74.
17. Misonou H, Mohapatra DP, Menegola M, Trimmer JS. Calcium- and metabolic state-dependent modulation of the voltage-dependent Kv2.1 channel regulates neuronal excitability in response to ischemia. J Neurosci. 2005; 25:11184–93.
18. Gao F, Wang CT, Chen C, Guo X, Yang LH, Ma XC, Han JF. Effect of hypokalemia on functional outcome at 3 months post-stroke among first-ever acute ischemic stroke patients. Med Sci Monit. 2017; 23:2825–2832.
19. Chung JW, Ryu WS, Kim BJ, Yoon BW. Elevated calcium after acute ischemic stroke: association with a poor short-term outcome and long-term mortality. J Stroke. 2015; 17:54–59.
20. Cheng Z, Huang X, Muse FM, Xia L, Zhan Z, Lin X, Cao Y, Han Z. Low serum magnesium levels are associated with hemorrhagic transformation after thrombolysis in acute ischemic stroke. Front Neurol. 2020; 11:962.
21. Qiu H, Shen R, Chen L, Pandey S, Sun J, Deng H. Low serum magnesium levels are associated with hemorrhagic transformation after mechanical thrombectomy in patients with acute ischemic stroke. Front Neurol. 2022; 13:831232.
22. Qi YL, Wu Q, Li XQ, Zhou ZH, Xia C, Wang XH, Chen HS. The association of admission ionized calcium with outcomes of thrombolysed patients with anterior circulation ischemic stroke. Brain Behav. 2023;13:e2844.
23. Siegler JE, Boehme AK, Albright KC, Bdeir S, Kar AK, Myers L, Beasley TM, Martin-Schild S. Acute decrease in serum magnesium level after ischemic stroke may not predict decrease in neurologic function. J Stroke Cerebrovasc Dis. 2013;22:e516–e521.
24. Mohammadifard N, Gotay C, Humphries KH, Ignaszewski A, Esmaillzadeh A, Sarrafzadegan N. Electrolyte minerals intake and cardiovascular health. Crit Rev Food Sci Nutr. 2019; 59:2375–2385.
25. Sung IS, Park SY, Jeong KY, Kim HM. Investigation of the preventive effect of calcium on inflammation-mediated choroidal neovascularization. Life Sci. 2019; 233:116727.
26. Shahi A, Aslani S, Ataollahi M, Mahmoudi M. The role of magnesium in different inflammatory diseases. Inflammo-pharmacology. 2019; 27: 649–661.
27. Young DB, Lin H, McCabe RD. Potassium's cardiovascular protective mechanisms. Am J Phys. 1995;268(4 Pt 2):R825–R837.
28. Kunutsor SK, Laukkanen JA. Circulating active serum calcium reduces the risk of hypertension. Eur J Prev Cardiol. 2017; 24:239–243.
29. Tan XF, Qin T, Li N, Yang YG, Zheng JH, Xie L, Chen MH. High-potassium preconditioning enhances tolerance to focal cerebral ischemia-reperfusion injury through anti-apoptotic effects in male rats. J Neurosci Res. 2019;97:1253–1265.