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Body mass index and cognitive recovery after acute ischemic stroke: A 3-month prospective analysis using mini-mental state examination at Tashkent Medical Academy, Uzbekistan

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Keywords
Ischemic Stroke; Body Mass Index; Cognitive Dysfunction; Obesity; Stroke Rehabilitation; Overweight

Abstract

Background: The association between body mass index (BMI) and cognitive recovery after acute ischemic stroke (AIS) remains controversial, with some studies suggesting a protective effect of overweight status. This study aims to investigate the relationship between BMI and cognitive recovery at three months post-stroke using data from the clinic of Tashkent Medical Academy, Tashkent, Uzbekistan.

Methods: We conducted a prospective cohort study including patients with AIS from the clinic of Tashkent Medical Academy between 2022 and 2024. Patients were categorized into five BMI groups based on World Health Organization (WHO) Asian population criteria. Cognitive recovery was assessed using the Mini-Mental State Examination (MMSE) at three months, with favorable recovery defined as an improvement of at least 3 points. Multivariate logistic regression and linear mixed-effects modeling (LMM) were used to evaluate the association between BMI and cognitive recovery, adjusting for demographic and clinical variables.

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Results: Among 728 patients with AIS included, 48.1% were overweight, 30.0% had normal weight, 13.5% were obese, 4.4% were underweight, and 4.1% were severely obese. Favorable cognitive recovery was most frequent in overweight patients (60.9%) and least common in underweight patients (50.0%) (P < 0.001). Overweight status was independently associated with better cognitive recovery [odds ratio (OR): 1.22, 95% confidence interval (CI): 1.10-1.37], whereas severe obesity showed no statistically significant association with cognitive outcomes (OR: 1.06, 95% CI: 0.71-1.58).

Conclusion: Overweight status may be associated with improved cognitive recovery after AIS, whereas severe obesity and underweight status are linked to worse outcomes. These findings highlight the need for individualized weight management strategies in post-stroke rehabilitation. Further research is needed to explore the underlying mechanisms and potential clinical interventions.

Introduction

Stroke remains one of the leading causes of disability and cognitive impairment worldwide, significantly affecting patients' quality of life (QOL) and long-term functional outcomes.[1] Cognitive recovery following acute ischemic stroke (AIS) is influenced by various factors, including age, vascular comorbidities, stroke severity, and neurorehabilitation strategies.[2] Among these, body mass index (BMI) has emerged as a potentially modifiable factor affecting stroke prognosis, yet its role in post-stroke cognitive recovery remains controversial.[3]

The relationship between BMI and stroke outcomes has been extensively studied, but findings remain inconsistent. Some research suggests that overweight and mildly obese individuals exhibit better survival rates and functional recovery, a phenomenon known as the "obesity paradox".[4] Possible explanations include increased metabolic reserves, protective effects of adipose tissue on neuroinflammation, and improved nutritional status, which may enhance post-stroke recovery.[5] Conversely, underweight patients are often frail and at a higher risk for malnutrition, sarcopenia, and atrial fibrillation (AF), all of which may negatively impact cognitive function.[6] Severe obesity, on the other hand, is associated with metabolic dysregulation, chronic inflammation, and increased cardiovascular burden, potentially leading to worse cognitive trajectories after stroke.[7,8]

Despite these findings, limited studies have directly assessed the association between BMI and cognitive recovery rather than overall functional outcomes. Given the conflicting evidence, further investigation is warranted to determine whether BMI independently influences post-stroke cognitive trajectories.

Therefore, this study aimed to address the existing gap in understanding the relationship between BMI and cognitive recovery after AIS. Previous studies have primarily focused on functional or survival outcomes, while the influence of BMI on post-stroke cognitive trajectories remains insufficiently explored. We hypothesized that overweight patients would demonstrate more favorable cognitive recovery, while underweight and severely obese individuals would experience poorer outcomes.

Materials and Methods

Study design and participants: This prospective cohort study was conducted at the clinic of Tashkent Medical Academy, Tashkent, Uzbekistan, between January 2022 and December 2024. All patients diagnosed with AIS within 14 days of symptom onset were consecutively enrolled, provided they met the predefined inclusion criteria. No selective recruitment was applied. Inclusion criteria were: (1) age between 40 and 80 years, (2) diagnosis of AIS confirmed by neuroimaging [computed tomography (CT) or magnetic resonance imaging (MRI)], (3) onset of stroke symptoms within 14 days prior to enrollment, and (4) availability of baseline cognitive assessment. Exclusion criteria were: (1) history of prior dementia or other severe neurodegenerative disorders, (2) presence of intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), or transient ischemic attack (TIA), (3) severe comorbidities limiting life expectancy to less than six months (e.g., terminal cancer, end-stage renal disease), (4) severe aphasia or cognitive impairment preventing cognitive testing, (5) uncontrolled psychiatric illness, and (6) loss to follow-up due to inability to participate in three-month assessment.

A total of 1262 patients were screened for eligibility. Of these, 956 patients met the inclusion criteria and were initially enrolled. During the 3-month follow-up period, 228 patients were excluded due to death (n = 64), loss to follow-up (n = 138), or incomplete cognitive assessments (n = 26), resulting in a final analytic cohort of 728 patients. Ethical approval for this study was obtained from the Institutional Review Board of the Tashkent Medical Academy (approval code: TMA-IRB/2022-03). All participants or their legally authorized representatives provided written informed consent before enrollment, in accordance with the ethical principles of the Declaration of Helsinki.

Baseline assessments: Demographic characteristics, vascular risk factors, and laboratory results were documented. The baseline vascular risk factors assessed included a history of stroke (confirmed through medical records), hypertension (HTN) (previous diagnosis or use of antihypertensive medication), diabetes mellitus (DM) (previous diagnosis or diabetes medication use), dyslipidemia (diagnosed dyslipidemia or lipid-lowering therapy), AF or atrial flutter [confirmed by at least one electrocardiogram (ECG) or presence of arrhythmia during hospitalization], coronary heart disease (CHD), and current or past smoking habits. The classification of AIS subtypes followed the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) criteria.

Upon hospital admission, nurses recorded patients' weight and height. The BMI was calculated as weight in kilograms divided by the square of height in meters. According to the World Health Organization (WHO) guidelines for the Asian population, BMI categories were defined as follows: underweight (BMI < 18.5 kg/m^2), normal weight (BMI = 18.5-24.9 kg/m^2), overweight (BMI = 25-29.9 kg/m^2), and obese (BMI ≥ 30 kg/m^2).[9]

Outcomes and follow-up: Three months following stroke onset, cognitive recovery was evaluated among AIS survivors using the Mini-Mental State Examination (MMSE). Baseline cognitive assessment was performed within 14 days after stroke onset. Cognitive improvement was defined as an increase of at least 3 points on the MMSE scale compared to baseline.[10] Assessments were conducted by trained evaluators who were blinded to the participants' baseline BMI classification and clinical characteristics, ensuring objectivity in outcome assessment. This procedural blinding was maintained throughout follow-up.

Descriptive statistics were used to summarize demographic and clinical characteristics across BMI groups. Continuous variables were assessed for normality using the Shapiro-Wilk test; those violating normality assumptions (P < 0.05) were analyzed using the Kruskal-Wallis test, while categorical variables were compared using the Pearson chi-square test.

MMSE change was analyzed using two complementary approaches. Additionally, mean changes in MMSE scores between baseline and 3 months were calculated for each BMI category, and differences across groups were compared using one-way analysis of variance (ANOVA). First, the primary outcome – favorable cognitive recovery – was defined as a binary variable, indicating a ≥ 3-point increase in MMSE score at 3 months compared to baseline. This binary outcome was analyzed using multivariable logistic regression, with the normal-weight group (BMI = 18.5-24.9 kg/m^2) serving as the reference category. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were calculated.

Covariates were selected based on preliminary univariate logistic regression analyses, with variables showing a P-value < 0.20 considered for inclusion in the multivariable model. In addition, clinically important covariates – such as HTN, smoking, and heart failure – were retained regardless of their univariate significance to account for potential confounding and theoretical relevance. The final adjusted model included age, sex, baseline MMSE, TOAST stroke subtype, HTN, DM, dyslipidemia, coronary artery disease (CAD), AF/atrial flutter, heart failure, smoking status, and prior stroke. Model diagnostics included assessment of multicollinearity using variance inflation factors (VIFs) (all VIFs < 2), model fit via the Hosmer-Lemeshow test (P = 0.39), and discrimination using the area under the receiver operating characteristic curve (AUC = 0.76).

Second, MMSE score was treated as a continuous outcome to evaluate within-subject cognitive change over time. A linear mixed-effects model (LMM) was used, including fixed effects for time (baseline vs. 3 months), BMI group, and their interaction, with random intercepts for participants to account for individual variability. Effect modification was further assessed by testing interaction terms between BMI and key covariates (age, sex, and diabetes), none of which reached statistical significance (all P-values > 0.10).

Missing data were minimal (< 5% for all variables) and handled through complete case analysis, yielding a final analytic sample of 728 participants. No imputation was performed.

All analyses were conducted using SPSS software (version 25.0, IBM Corporation, Armonk, NY, USA), with two-sided P-values < 0.05 considered statistically significant.

Results

Participant flow and attrition: Among the 728 patients with AIS who completed the 3-month follow-up and met all eligibility criteria, 55% were women and 45% were men. These patients represent the final analytic sample used to evaluate the association between BMI and cognitive recovery. We performed a comparative analysis between the baseline characteristics of patients retained in the final analytic sample (n = 728) and those lost to follow-up (n = 228). The groups did not differ significantly in terms of age (P = 0.31), sex (P = 0.44), or baseline BMI distribution (P = 0.29). However, a slightly higher proportion of severely aphasic patients was noted among those excluded.

Baseline characteristics by BMI category: Based on BMI classification, 32 patients (4.4%) were categorized as underweight, 218 (30.0%) had normal weight, 350 (48.1%) were overweight, 98 (13.5%) were obese, and 30 (4.1%) were classified as severely obese. The baseline characteristics of these BMI groups are presented in table 1. Statistically significant differences were observed across BMI groups. The obese group had higher frequencies of HTN (76.5%), diabetes (32.7%), and dyslipidemia (57.1%). In contrast, the underweight group showed a higher prevalence of AF/atrial flutter (18.8%) and heart failure (6.3%), and a greater proportion of cardioembolic stroke cases (9.4%) compared to other BMI categories.

Univariate logistic regression analysis: To explore factors associated with favorable cognitive recovery, we first conducted univariate logistic regression analyses for all baseline demographic and clinical covariates. As shown in table 2, several variables were significantly associated with favorable cognitive recovery. Younger age, male sex, and higher baseline MMSE scores were linked to better outcomes. In contrast, DM, prior stroke, and cardioembolic or large artery atherosclerosis (LAA) stroke subtypes were associated with lower odds of cognitive improvement. Variables with a P < 0.20 were considered for inclusion in the multivariable logistic regression model. Additionally, clinically important variables such as HTN, smoking status, and heart failure were retained regardless of their univariate significance to account for potential confounding effects.

Multivariable logistic regression analysis: Cognitive recovery, defined as an improvement of at least 3 points on the MMSE scale, was more frequently observed in higher BMI groups: 50.0% (16/32) in underweight, 54.6% (119/218) in normal weight, 60.9% (213/350) in overweight, 58.2% (57/98) in obese, and 60.0% (18/30) in severely obese individuals (P < 0.001).

Table 1. Baseline characteristics of 728 patients with acute ischemic stroke (AIS) according to body mass index (BMI)

Category	Underweight (< 18.5 kg/m²)	Normal (18.5-24.9 kg/m²)	Overweight (25-29.9 kg/m²)	Obese (30-34.9 kg/m²)	Severely obese (≥ 35 kg/m²)	P
Number	32 (4.4)	218 (30.0)	350 (48.1)	98 (13.5)	30 (4.1)	-
Age (year)	75 (66-80)	68 (58-76)	65 (55-73)	64 (55-73)	66.5 (56-74)	< 0.0001
Gender (women)	19 (59.4)	119 (54.6)	175 (50.0)	58 (59.2)	21 (70.0)	< 0.0001
Medical history						
Hypertension	15 (46.9)	122 (56.0)	231 (66.0)	75 (76.5)	23 (76.7)	< 0.0001
Diabetes	5 (15.6)	45 (20.6)	94 (26.9)	32 (32.7)	10 (33.3)	< 0.0001
Dyslipidemia	12 (37.5)	104 (47.7)	185 (52.9)	56 (57.1)	17 (56.7)	< 0.0001
Peripheral vascular disease	1 (3.1)	3 (1.4)	5 (1.4)	1 (1.0)	1 (3.3)	0.3350
Coronary heart disease	4 (12.5)	27 (12.4)	50 (14.3)	18 (18.4)	6 (20.0)	< 0.0001
Atrial fibrillation/flutter	6 (18.8)	25 (11.5)	32 (9.1)	9 (9.2)	3 (10.0)	< 0.0001
Heart failure	2 (6.3)	5 (2.3)	7 (2.0)	2 (2.0)	1 (3.3)	< 0.0001
Current or previous smoking	8 (25.0)	65 (29.8)	140 (40.0)	34 (34.7)	6 (20.0)	< 0.0001
Stroke	10 (31.3)	72 (33.0)	120 (34.3)	34 (34.7)	8 (26.7)	0.1220
Baseline MMSE (score < 24)	4 (12.5)	17 (7.8)	28 (8.0)	9 (9.2)	3 (10.0)	0.2300
LAA	12 (37.5)	95 (43.6)	160 (45.7)	45 (45.9)	14 (46.7)	
CE	3 (9.4)	14 (6.4)	18 (5.1)	5 (5.1)	1 (3.3)	
SAO	5 (15.6)	37 (17.0)	58 (16.6)	14 (14.3)	5 (16.7)	
Other determined	1 (3.1)	5 (2.3)	8 (2.3)	3 (3.1)	1 (3.3)	
Undetermined	11 (34.4)	67 (30.7)	106 (30.3)	31 (31.6)	9 (30.0)	

Data are presented as number and percentage for nominal data or median and interquartile range (IQR) for continuous data
CE: Cardioembolism; LAA: Large artery atherosclerosis; SAO: Small artery occlusion; MMSE: Mini-Mental State Examination

Table 2. Univariable logistic regression of demographic and clinical covariates associated with favorable cognitive recovery at 3 months post-stroke

Covariate	OR	95% CI	P
Age (per year increase)	0.96	0.95-0.97	< 0.001
Female sex	0.81	0.72-0.91	0.001
Baseline MMSE < 24	0.55	0.43-0.69	< 0.001
TOAST: LAA (ref: SAO)	0.62	0.54-0.72	< 0.001
TOAST: CE	0.53	0.38-0.73	< 0.001
TOAST: Undetermined	0.60	0.51-0.71	< 0.001
Hypertension	1.02	0.91-1.14	0.678
Diabetes mellitus	0.74	0.67-0.83	< 0.001
Dyslipidemia	1.03	0.93-1.14	0.575
Coronary artery disease	0.91	0.80-1.03	0.135
Atrial fibrillation/flutter	1.16	0.94-1.42	0.154
Heart failure	0.72	0.49-1.05	0.081
Smoking (current or former)	1.01	0.89-1.14	0.932
Prior stroke	0.78	0.69-0.88	< 0.001

P-values < 0.05 were considered statistically significant
OR: Odds ratio; CI: Confidence interval; MMSE: Mini-Mental State Examination; TOAST: Trial of ORG 10172 in Acute Stroke Treatment; SAO: Small artery occlusion; LAA: Large artery atherosclerosis; CE: Cardioembolism

Following adjustments for potential confounding factors, multivariate logistic regression analysis indicated that being overweight was independently associated with improved cognitive recovery at 3 months (OR = 1.22, 95% CI: 1.10-1.37). Although the OR for severe obesity suggested a potential association with lower cognitive recovery, this finding was not statistically significant (OR = 1.06, 95% CI: 0.71-1.58, P = 0.753), and should be interpreted with caution (Table 3).

Longitudinal cognitive change (LMM analysis): Longitudinal analysis using an LMM further supported these findings. The model included BMI group, time (baseline and three months), and their interaction as fixed effects, with random intercepts for individuals. The detailed parameter estimates from the LMM are presented in table 4. The BMI × time interaction term was statistically significant (P = 0.03). Specifically, overweight patients demonstrated greater cognitive improvement compared with normal-weight individuals, while improvement was less pronounced among underweight and severely obese groups.

The mean MMSE improvement over the three-month follow-up period differed across BMI categories. Patients with overweight exhibited the greatest mean increase in MMSE scores (3.2 ± 2.8), followed by those with obesity (2.9 ± 2.7) and normal weight (2.4 ± 2.6). In contrast, the underweight group demonstrated the least improvement (1.8 ± 2.4), while severely obese individuals showed moderate gains (2.1 ± 2.9). Between-group comparison using one-way ANOVA indicated that these differences were statistically significant (P = 0.041). Consistent with these findings, cognitive decline occurred in 12.5% (4/32) of underweight, 7.8% (17/218) of normal-weight, 6.9% (24/350) of overweight, 7.1% (7/98) of obese, and 10.0% (3/30) of severely obese patients (P < 0.001).

Table 3. Multivariable logistic regression analysis of factors associated with favorable cognitive recovery at 3 months

Category	Favorable cognitive recovery (MMSE score improvement ≥ 3) OR (95% CI)	P
Weight status		
Underweight	1.18 (0.90-1.54)	0.110
Normal (reference)	1	-
Overweight	1.22 (1.10-1.37)	0.001
Obese	1.14 (0.98-1.33)	0.071
Severely obese	1.06 (0.71-1.58)	0.753
Age (per 1-year increase)	0.97 (0.96-0.98)	0.001
Female gender	0.82 (0.74-0.92)	0.001
Hypertension	1.01 (0.92-1.12)	0.951
Diabetes	0.76 (0.69-0.85)	0.001
Dyslipidemia	1.04 (0.94-1.14)	0.605
Coronary heart disease	0.94 (0.82-1.08)	0.373
Atrial fibrillation/flutter	1.18 (0.96-1.47)	0.098
Heart failure	0.75 (0.52-1.08)	0.123
Current or previous smoking	1.00 (0.89-1.12)	0.951
Stroke history	0.79 (0.71-0.87)	0.001
Baseline MMSE score	0.51 (0.42-0.61)	0.001
LAA	0.56 (0.49-0.64)	0.001
CE	0.53 (0.39-0.71)	0.001
SAO (reference)	1	-
Other determined	0.47 (0.31-0.74)	0.001
Undetermined	0.61 (0.52-0.70)	0.001

CE: Cardioembolism; CI: Confidence interval; LAA: Large artery atherosclerosis; MMSE: Mini-Mental State Examination; OR: Odds ratio; SAO: Small artery occlusion

Sensitivity and interaction analyses: Sensitivity analyses were performed to confirm the robustness of the main findings (Table 5).

Table 4. Linear mixed-effects model (LMM) evaluating longitudinal changes in Mini-Mental State Examination (MMSE) scores by body mass index (BMI) category

Variable	β (estimate)	SE	95% CI	P
Time (3 months vs. baseline)	+2.85	0.21	2.44, 3.26	< 0.001
Overweight × time	+0.74	0.28	0.19, 1.29	0.030
Obese × time	+0.62	0.31	0.01, 1.23	0.047
Severely obese × time	+0.41	0.44	-0.45, 1.27	0.338
Underweight × time	-0.58	0.36	-1.29, 0.13	0.108

SE: Standard error; CI: Confidence interval

Repeating the multivariable logistic regression after excluding patients with missing baseline covariates or extreme BMI values (BMI < 17 or ≥ 37 kg/m^2) yielded similar results. Interaction analyses were also conducted to evaluate whether the effect of BMI on cognitive recovery differed by key covariates, including age, sex, and DM. None of the BMI × covariate interaction terms reached statistical significance (all Ps > 0.10).

Discussion

The findings of this study provide important insights into the relationship between BMI and cognitive recovery following AIS. Our results demonstrate a significant association between BMI and post-stroke cognitive outcomes, with overweight individuals being more likely to exhibit favorable cognitive recovery at three months. Conversely, severe obesity was linked to a higher likelihood of cognitive decline, even after adjusting for potential confounders. These findings align with previous research suggesting a complex interplay between adiposity and stroke recovery.[3,11]

Our study corroborates earlier reports on the potential protective effect of overweight status on post-stroke recovery. The observation that overweight patients exhibited a greater likelihood of cognitive improvement (OR = 1.24, 95% CI: 1.12-1.38) suggests that moderate adiposity may be associated with neuroprotective advantage. This observation was supported by both categorical and continuous analyses. Our findings were consistent across binary and continuous modeling of MMSE change. While logistic regression demonstrated a significantly higher likelihood of cognitive improvement in the overweight group, the LMM confirmed greater mean MMSE gains over time in the same group, suggesting both clinically and statistically significant recovery trajectories. This phenomenon, often referred to as the "obesity paradox", has been described in prior observational stroke studies.[3] The proposed mechanisms include enhanced metabolic reserves, anti-inflammatory properties of adipose tissue, and improved nutritional status, which may collectively mitigate neurodegenerative processes.[12]

However, the relationship between BMI and cognitive outcomes appears to be nonlinear, as patients in the underweight and severely obese categories had worse cognitive trajectories. In our sample, underweight patients demonstrated higher rates of AF, heart failure, and cardioembolic strokes compared to other BMI groups. These clinical characteristics may partially explain the less favorable cognitive recovery observed in this subgroup and are consistent with the hypothesis that underlying frailty and cardiovascular comorbidity are associated with poorer outcomes in underweight individuals.[13,14]

Table 5. Sensitivity and interaction analyses of body mass index (BMI)-cognition associations

Analysis type	Description	Main finding	P
Sensitivity analysis 1	Excluding cases with missing data (n = 728 complete cases)	Overweight remained independently associated with favorable recovery (OR = 1.21, 95% CI: 1.09-1.36)	< 0.01
Sensitivity analysis 2	Excluding extreme BMI (< 17 or ≥ 37 kg/m^2)	Results unchanged; overweight OR = 1.20 (95% CI: 1.08-1.35)	< 0.01
Interaction analysis: BMI × age	Evaluating modification by age group (< 65 vs. ≥ 65 years)	No significant interaction	0.42
Interaction analysis: BMI × sex	Evaluating effect modification by sex	No significant interaction	0.27
Interaction analysis: BMI × diabetes	Evaluating effect modification by DM	No significant interaction	0.33

BMI: Body mass index; OR: Odds ratio; CI: Confidence interval; DM: Diabetes mellitus

Although a numerical trend toward poorer cognitive outcomes was observed among individuals with severe obesity, the association was not statistically significant (OR = 1.06, 95% CI: 0.71-1.58). This non-significant finding may be explained by metabolic dysregulation, chronic inflammation, or increased vascular burden,[15] but should be interpreted cautiously due to the limited sample size in this subgroup.

Our study also highlights the association between vascular risk factors and post-stroke cognitive recovery. As expected, HTN, diabetes, and dyslipidemia were more prevalent in higher BMI groups, particularly among obese individuals. While these conditions are known to exacerbate stroke severity and impair neurovascular function, their precise contributions to BMI-related cognitive outcomes warrant further investigation.[16,17] Notably, diabetes was significantly associated with poorer cognitive recovery (OR: 0.76, 95% CI: 0.69-0.85), reinforcing the need for rigorous glycemic control in post-stroke patients.[18]

Our study has several limitations. First, BMI was measured only once at admission during the acute hospitalization phase and may not reflect weight fluctuations occurring during recovery, potentially introducing misclassification bias. Future studies should incorporate longitudinal assessments of body composition to better understand its relationship with cognitive outcomes. Second, we used only the MMSE to assess cognition, which, despite being practical and widely validated, lacks sensitivity for specific cognitive domains such as executive function and attention, and is susceptible to ceiling effects and educational bias. More comprehensive neuropsychological tools are recommended in future research.[19] Third, as our study was observational, causality cannot be definitively established, and residual confounding remains possible. Further randomized controlled trials (RCTs) and longitudinal studies are warranted to elucidate the causal mechanisms underlying BMI-related cognitive outcomes after stroke. Lastly, our study was not powered based on a priori calculations, as it was embedded in a prospective observational registry. However, the final sample size (~700 participants) was adequate to detect the observed association between BMI and cognitive recovery with > 90% power at $\alpha = 0.05$. Subgroup analyses – particularly for the severely obese group (n = 30) – should be interpreted with caution due to limited statistical power. Future studies with larger samples in underrepresented BMI categories are needed to validate these findings.

Conclusion

Our findings indicate that overweight status is significantly associated with better early cognitive recovery following AIS. While underweight and severe obesity groups exhibited trends toward poorer outcomes, these associations were not statistically significant and should be interpreted with caution. These findings suggest that individualized weight management may represent a potential area for further investigation in post-stroke cognitive rehabilitation. Further research is warranted to determine whether interventions targeting weight status are associated with improved cognitive outcomes, and to evaluate their clinical applicability in stroke rehabilitation settings.

Conflict of Interests

The authors declare no conflict of interest in this study.

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References

1. Feigin VL, Stark BA, Johnson CO, Roth GA, Bisignano C, Abady GG, et al. Global, regional, and national burden of stroke and its risk factors, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Neurol 2021; 20(10): 795-820.
2. Cumming TB, Marshall RS, Lazar RM. Stroke, cognitive deficits, and rehabilitation: still an incomplete picture. Int J Stroke 2013; 8(1): 38-45.
3. Doehner W, Schenkel J, Anker SD, Springer J, Audebert HJ. Overweight and obesity are associated with improved survival, functional outcome, and stroke recurrence after acute stroke or transient ischaemic attack: observations from the TEMPiS trial. Eur Heart J 2013; 34(4): 268-77.
4. Vemmos K, Ntaios G, Spengos K, Savvari P, Vemmou A, Pappa T, et al. Association between obesity and mortality after acute first-ever stroke: the obesity-stroke paradox. Stroke 2011; 42(1): 30-6.
5. Katsiki N, Ntaios G, Vemmos K. Stroke, obesity and gender: a review of the literature. Maturitas 2011; 69(3): 239-43.
6. Kang SH, Choi EK, Han KD, Lee SR, Lim WH, Cha MJ, et al. Underweight is a risk factor for atrial fibrillation: A nationwide population-based study. Int J Cardiol 2016; 215: 449-56.
7. Lavie CJ, McAuley PA, Church TS, Milani RV, Blair SN. Obesity and cardiovascular diseases: implications regarding fitness, fatness, and severity in

the obesity paradox. J Am Coll Cardiol 2014; 63(14): 1345-54.
8. Yatsuya H, Folsom AR, Yamagishi K, North KE, Brancati FL, Stevens J. Race- and sex-specific associations of obesity measures with ischemic stroke incidence in the Atherosclerosis Risk in Communities (ARIC) study. Stroke 2010; 41(3): 417-25.
9. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004; 363(9403): 157-63. Erratum in: Lancet 2004; 363(9412): 902.
10. Clark CM, Sheppard L, Fillenbaum GG, Galasko D, Morris JC, Koss E, et al. Variability in annual Mini-Mental State Examination score in patients with probable Alzheimer disease: a clinical perspective of data from the Consortium to Establish a Registry for Alzheimer's Disease. Arch Neurol 1999; 56(7): 857-62.
11. Liu Z, Sanossian N, Starkman S, Avila-Rinek G, Eckstein M, Sharma LK, et al. Adiposity and Outcome After Ischemic Stroke: Obesity Paradox for Mortality and Obesity Parabola for Favorable Functional Outcomes. Stroke 2021; 52(1): 144-51.
12. Quiñones-Ossa GA, Lobo C, Garcia-Ballestas E, Florez WA, Moscote-Salazar LR, Agrawal A. Obesity and Stroke: Does the Paradox Apply for Stroke? Neurointervention 2021; 16(1): 9-19.
13. Zhu W, Wan R, Liu F, Hu J, Huang L, Li J, et al. Relation of Body Mass Index with Adverse Outcomes among Patients with Atrial Fibrillation: A Meta-Analysis and Systematic Review. J Am Heart Assoc 2016; 5(9): e004006.
14. Bucholz EM, Krumholz HA, Krumholz HM. Underweight, Markers of Cachexia, and Mortality in Acute Myocardial Infarction: A Prospective Cohort Study of Elderly Medicare Beneficiaries. PLoS Med 2016; 13(4): e1001998.
15. Kramer CK, Zinman B, Retnakaran R. Are metabolically healthy overweight and obesity benign conditions?: A systematic review and meta-analysis. Ann Intern Med 2013; 159(11): 758-69.
16. Gorelick PB, Scuteri A, Black SE, Decarli C, Greenberg SM, Iadecola C, et al. Vascular contributions to cognitive impairment and dementia: a statement for healthcare professionals from the American heart association/American stroke association. Stroke 2011; 42(9): 2672-713.
17. Iadecola C, Gottesman RF. Neurovascular and Cognitive Dysfunction in Hypertension. Circ Res 2019; 124(7): 1025-44.
18. Biessels GJ, Despa F. Cognitive decline and dementia in diabetes mellitus: mechanisms and clinical implications. Nat Rev Endocrinol 2018; 14(10): 591-604.
19. Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, Whitehead V, Collin I, et al. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. J Am Geriatr Soc 2005; 53(4): 695-9.