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LINK MEDICAL INTERFACE

Determinants of Post-Stroke Cognitive Impairment in a Hospital-Based Sialkot Cohort

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ABSTRACT

Background: Post-stroke cognitive impairment (PSCI) is one of the most disabling sequelae of stroke, contributing to functional dependence, poor rehabilitation outcomes, and reduced quality of life. Despite a rising burden of stroke in Pakistan, there remains limited evidence on the prevalence and determinants of PSCI in secondary-care hospital settings. Objective: To assess the frequency and predictors of post-stroke cognitive impairment among survivors in Sialkot hospitals and to identify the independent socio-demographic and clinical factors influencing cognitive outcomes. Methods: An analytical cross-sectional study was conducted among 100 stroke survivors and 100 healthy controls recruited from four major hospitals in Sialkot between January and June 2024. Cognitive function was assessed using the Montreal Cognitive Assessment (MoCA), stroke severity by the National Institutes of Health Stroke Scale (NIHSS), and functional status by the Barthel Index. Data were analyzed using SPSS v25, applying t-tests, ANOVA, correlation, and multiple linear regression to determine independent predictors of MoCA scores. Results: Cognitive impairment (MoCA <26) was observed in 78% of stroke survivors versus 12% of controls ($p<0.001$). Lower MoCA scores correlated significantly with older age ($r=-0.47$), higher NIHSS ($r=-0.59$), and lower Barthel Index ($r=+0.63$). Education, stroke severity, and functional independence independently predicted cognitive performance (adjusted $R^2=0.61$). Conclusion: Post-stroke cognitive impairment is highly prevalent among stroke survivors in Sialkot and is primarily influenced by age, education, and neurological severity. Routine cognitive screening and integrated rehabilitation strategies are recommended to enhance recovery and independence.

Keywords
Post-Stroke Cognitive Impairment, Stroke Severity, Montreal Cognitive Assessment, Functional Independence, Predictors, Pakistan.

INTRODUCTION

Post-stroke cognitive impairment (PSCI) is one of the most consequential sequelae of cerebrovascular disease, affecting attention, memory, language, and executive function, and driving long-term disability, caregiver burden, and health-system costs (1). International estimates suggest that a substantial proportion of survivors develop PSCI across the first post-stroke months, but the magnitude and pattern vary with case-mix, timing of assessment, and tools used (2). Mechanistically, both ischemic and hemorrhagic strokes can damage strategic gray and white matter networks through infarction, microvascular injury, and secondary neuroinflammation, with vascular risk factors compounding small-vessel disease and cognitive decline trajectories (3,4). These concerns are acute in South Asia and Pakistan, where stroke incidence is high, onset occurs at younger ages than in high-income countries, and organized stroke services—including thrombolysis pathways and structured cognitive rehabilitation—remain unevenly accessible (5,6). Public awareness of stroke symptoms and risk modification is also limited, contributing to delayed presentation and missed opportunities for secondary prevention (7), while the population burden of hypertension—the single largest modifiable stroke risk—has been substantial for decades (8).

In low-resource hospital settings, valid, brief cognitive screening is essential for triage and rehabilitation planning. The Montreal Cognitive Assessment (MoCA) is more sensitive than the Mini-Mental State Examination for detecting mild cognitive deficits in stroke cohorts, capturing executive and visuospatial dysfunction often missed by global screens (9). Observational studies consistently implicate older age, greater acute stroke severity, and lower educational attainment as correlates of poorer post-stroke cognition, whereas stroke type and hemisphere show inconsistent associations once overall severity is accounted for (2,10,11). Functional status and cognition are interdependent; lower scores on activities-of-daily-living indices commonly accompany worse cognitive performance, complicating recovery and community reintegration (12). Yet, despite Pakistan's rising stroke burden and service gaps, there are few contemporary data quantifying PSCI severity and identifying independent predictors among survivors receiving care in secondary/tertiary hospitals in smaller cities such as Sialkot, where demographic composition, education levels, and care pathways may differ from national referral centers (5,6).

Within this context, we designed an analytical cross-sectional study of adult stroke survivors (≥3 months post-event) managed in four Sialkot hospitals to quantify cognitive impairment using MoCA and determine independent predictors among socio-demographic, clinical, and stroke-specific variables. Using a PICO framing: the Population comprises brain-stroke survivors in Sialkot hospitals; the Intervention/Exposure is the set of patient characteristics and stroke features (age, education, NIHSS, Barthel Index, stroke type, hemisphere, disease status); the Comparator is between-group contrasts across these exposures; and the Outcomes are MoCA total score (continuous) and PSCI severity categories (mild,

moderate, severe). We hypothesized a priori that higher NIHSS and older age would be associated with lower MoCA scores, while higher educational attainment would be associated with better MoCA performance; we further anticipated no independent association for stroke type or hemisphere after adjustment (2,10,11). The primary objective is to quantify PSCI burden and identify independent predictors to inform screening priorities and rehabilitation targeting in a resource-limited, hospital-based Sialkot cohort.

MATERIAL AND METHODS

This analytical cross-sectional study was designed to determine the prevalence and predictors of post-stroke cognitive impairment (PSCI) among adult stroke survivors in a hospital-based cohort from Sialkot, Pakistan. The rationale for selecting this design was to allow assessment of multiple socio-demographic, clinical, and stroke-related factors at a single point in time to identify independent determinants of cognitive function measured by standardized instruments. The study was conducted across four major hospitals in Sialkot—Civil Hospital, Saeed Medical Complex, Idrees Hospital, and Kiran International Hospital—between January and June 2024, representing the primary neurological and rehabilitation care centers serving the district's urban and peri-urban population.

Participants were recruited consecutively from outpatient and inpatient follow-up clinics if they were at least three months post-confirmed stroke diagnosis to ensure stabilization of acute neurological symptoms. Diagnosis of ischemic or hemorrhagic stroke was verified through neuroimaging, including computed tomography (CT) or magnetic resonance imaging (MRI). Eligible participants were adults aged 30 years and above who were clinically stable and able to provide informed consent directly or via a caregiver. Individuals with prior psychiatric or neurological illnesses such as schizophrenia, major depressive disorder, or aphasia before stroke onset were excluded to minimize confounding from pre-existing cognitive deficits. A control group of 100 healthy individuals without a history of stroke or chronic psychiatric disorders was recruited from the same community for comparative analyses. Written informed consent was obtained from all participants or their legal guardians, with additional verbal reconfirmation before each interview.

Data were collected by trained medical professionals using structured questionnaires and validated instruments administered in the hospital setting under supervision. Each participant underwent a standardized interview and assessment protocol comprising three major domains: (1) socio-demographic information, (2) clinical history, and (3) cognitive and functional evaluation. Demographic variables included age, gender, marital status, and education level. Clinical variables encompassed comorbidities (hypertension, diabetes mellitus, and dyslipidemia), type of stroke (ischemic or hemorrhagic), hemisphere involvement (left or right), and stroke severity. Hypertension was operationally defined as systolic blood pressure ≥140 mmHg or diastolic ≥90 mmHg or current use of antihypertensive medication; diabetes was defined as fasting plasma glucose ≥126 mg/dL or use of hypoglycemic drugs. Dyslipidemia was characterized as total cholesterol ≥4.5 mmol/L or LDL ≥2.5 mmol/L, or use of lipid-lowering agents.

Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS), a 15-item clinician-rated instrument scored from 0 to 42, where higher values indicate greater neurological deficit. Scores of 1–4, 5–15, 16–20, and 21–42 corresponded to minor, moderate, moderate-to-severe, and severe stroke categories, respectively (13). Cognitive performance was evaluated using the Montreal Cognitive Assessment (MoCA), which measures multiple domains—visuospatial/executive function, naming, attention, language, abstraction, delayed recall, and orientation—on a 30-point scale. A score ≥26 indicated normal cognition, 25–18 mild impairment, 17–10 moderate impairment, and <10 severe impairment (14). Functional independence was assessed using the Barthel Index (BI), evaluating activities of daily living such as mobility, feeding, grooming, dressing, and continence, with scores <60 classified as dependent, 61–90 moderately dependent, 91–99 mildly dependent, and 100 independent (15).

To minimize observer bias, all assessors were trained in standardized administration of NIHSS, MoCA, and BI, and inter-rater reliability was verified prior to data collection. The sequence of tool administration was fixed across participants to reduce measurement variability, with rest intervals provided to prevent fatigue. To address confounding, multivariable analyses were planned a priori to adjust for age, education, and stroke severity as potential covariates, and sensitivity checks were performed to assess robustness across subgroups stratified by stroke type and hemisphere. Selection bias was minimized by including consecutive eligible cases across multiple hospitals, thereby enhancing representativeness within the local stroke population.

A sample size of 100 stroke survivors and 100 controls was deemed adequate to detect medium effect sizes (Cohen's $d \approx 0.6$) in MoCA score differences between groups at $\alpha=0.05$ and power=0.90, based on prior regional studies showing comparable cognitive score distributions. Missing data were handled by pairwise deletion after verifying that less than 5% of total observations were incomplete, with no significant deviation in demographic or clinical variables between complete and incomplete cases.

Data were entered and analyzed using IBM SPSS Statistics version 25.0. Descriptive statistics summarized categorical variables as frequencies and percentages and continuous variables as means and standard deviations. Between-group differences were analyzed using independent-samples t-tests or chi-square tests, as appropriate. One-way ANOVA with Tukey's post hoc comparisons was applied to explore differences in MoCA across educational and marital subgroups. Pearson's correlation coefficient quantified linear associations among MoCA, NIHSS, age, and BI scores. Multiple linear regression was conducted to identify independent predictors of MoCA, with inclusion of all variables showing $p<0.10$ in bivariate analyses. Collinearity diagnostics were examined (tolerance >0.2, VIF <5). Statistical significance was set at $p<0.05$ (two-tailed).

Ethical approval for this study was obtained from the institutional ethics review committee of Link Medical Interface, Lahore, with reference number LMJ/ERB/2024/041. All participants provided informed consent, and confidentiality was maintained through coded identifiers and restricted data access. Hardcopy data sheets were stored in locked cabinets, and electronic datasets were password-protected. Reproducibility and data integrity were ensured through double-entry verification, regular cross-checks between source documents and databases, and blinded statistical validation of a random subset of cases. The full analytic protocol, dataset structure, and coding templates were archived for potential replication and secondary analyses under institutional governance.

RESULTS

A total of 200 participants were included—100 post-stroke patients and 100 healthy controls. The mean age of stroke survivors was significantly higher than controls, and a higher proportion were male and hypertensive. Cognitive impairment (MoCA <26) was observed in 78% of stroke survivors compared to 12% of controls ($p<0.001$), with moderate-to-severe impairment predominating among ischemic cases.

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Table 1. Demographic and Clinical Characteristics of Participants (n = 200)

Variable	Stroke Group (n=100)	Control Group (n=100)	Mean Difference / χ^2	p-value
Age (years, mean ± SD)	59.8 ± 9.2	52.1 ± 8.4	t = 5.75	<0.001
Male (%)	68 (68%)	56 (56%)	χ^2 = 3.04	0.082
Education (years, mean ± SD)	10.4 ± 4.1	12.7 ± 3.8	t = -4.02	<0.001
Hypertension (%)	77 (77%)	39 (39%)	χ^2 = 25.7	<0.001
Diabetes mellitus (%)	48 (48%)	27 (27%)	χ^2 = 8.39	0.004
Dyslipidemia (%)	36 (36%)	23 (23%)	χ^2 = 4.11	0.043
Smoking (%)	29 (29%)	19 (19%)	χ^2 = 2.29	0.130
Stroke type (Ischemic/Hemorrhagic)	74 / 26	—	—	—
Hemisphere (Left/Right)	58 / 42	—	—	—

Interpretation: Stroke survivors were significantly older and had higher rates of hypertension, diabetes, and dyslipidemia compared to controls, suggesting a clustering of vascular risk factors.

Table 2. Cognitive and Functional Scores among Stroke and Control Groups

Variable	Stroke Group (n=100)	Control Group (n=100)	Mean Difference (95% CI)	p-value	Cohen's d
MoCA Total Score	18.4 ± 5.3	27.8 ± 1.9	-9.4 (-10.5, -8.3)	<0.001	2.20
Barthel Index	77.5 ± 15.8	96.4 ± 5.2	-18.9 (-21.7, -16.1)	<0.001	1.58
NIHSS Score	9.8 ± 4.1	—	—	—	—
MoCA <26 (Impaired, %)	78 (78%)	12 (12%)	OR = 26.0 (12.2–55.5)	<0.001	—

Interpretation: Stroke survivors had markedly lower MoCA and Barthel scores, reflecting substantial cognitive and functional deficits compared to controls. Effect sizes were large for both variables, indicating strong group differences.

Table 3. Comparison of MoCA Scores by Stroke Type, Hemisphere, and Education (Stroke Group Only)

Variable	n	Mean MoCA ± SD	F / t / χ^2	p-value	95% CI / η^2
Stroke Type			F(1,98)=0.74	0.392	η^2=0.008
• Ischemic	74	18.2 ± 5.1			
• Hemorrhagic	26	19.0 ± 5.8			
Hemisphere			t=0.59	0.556	95% CI: -1.8, 3.3
• Left	58	18.1 ± 5.4			
• Right	42	18.8 ± 5.2			
Education (Years)			F(2,97)=8.21	<0.001	η^2=0.145
• ≤8 years	38	15.9 ± 4.8			
• 9–12 years	34	18.8 ± 5.0			
• >12 years	28	21.2 ± 4.6			

Interpretation: Educational attainment showed a strong, graded association with cognitive performance, whereas stroke type and hemisphere had no significant influence on MoCA scores.

Table 4. Correlation Between Cognitive, Functional, and Clinical Variables in Stroke Survivors

Variables	r	95% CI	p-value	Interpretation
MoCA vs Age	-0.47	(-0.61, -0.29)	<0.001	Older patients performed worse cognitively
MoCA vs NIHSS	-0.59	(-0.70, -0.45)	<0.001	Higher stroke severity predicted poorer cognition
MoCA vs Barthel Index	+0.63	(0.49, 0.73)	<0.001	Greater functional independence correlated with better cognition
NIHSS vs Barthel Index	-0.68	(-0.77, -0.56)	<0.001	Severe strokes reduced functional ability

Interpretation: Significant negative correlations were found between MoCA and both age and stroke severity, while positive associations were observed between cognition and functional independence.

Table 5. Multiple Linear Regression Predicting MoCA Scores Among Stroke Survivors (n=100)

Predictor Variable	β (Unstandardized)	SE	95% CI	t	p-value
Age (years)	-0.21	0.06	-0.33, -0.09	-3.45	0.001
Education (years)	+0.39	0.09	0.21, 0.56	4.39	<0.001
NIHSS Score	-0.52	0.10	-0.72, -0.32	-5.20	<0.001
Barthel Index	+0.07	0.03	0.01, 0.13	2.34	0.022
Hypertension (yes=1)	-1.48	0.83	-3.12, 0.16	-1.78	0.078
Adjusted R^2 = 0.61	F(5,94)=31.2	p<0.001	—	—	—

Interpretation: Education, stroke severity, and functional independence were the strongest independent predictors of cognitive outcomes, collectively explaining 61% of MoCA score variance. Age retained a modest but significant negative effect after adjustment.

The present study included 200 participants, equally divided into 100 stroke survivors and 100 healthy controls. Stroke patients were significantly older (mean ± SD 59.8 ± 9.2 years) than controls (52.1 ± 8.4 years, $p < 0.001$), and although males predominated in both groups, gender difference did not reach statistical significance (68% vs 56%, $p = 0.082$). Years of formal education were lower among stroke survivors (10.4 ± 4.1) compared

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with controls (12.7 ± 3.8, $p < 0.001$). Vascular risk factors were markedly more prevalent in the stroke cohort: hypertension in 77% vs 39% ($p < 0.001$), diabetes mellitus in 48% vs 27% ($p = 0.004$), and dyslipidemia in 36% vs 23% ($p = 0.043$). These results highlight a strong clustering of vascular comorbidities among stroke patients. Cognitive and functional assessments revealed striking deficits among stroke survivors. Mean MoCA scores were 18.4 ± 5.3 in the stroke group versus 27.8 ± 1.9 in controls (mean difference = −9.4, 95% CI −10.5 to −8.3; $p < 0.001$), corresponding to a large effect size (Cohen's $d = 2.20$). Functional independence measured by the Barthel Index was also significantly reduced (77.5 ± 15.8 vs 96.4 ± 5.2; $p < 0.001$, $d = 1.58$). Overall, 78% of stroke survivors demonstrated cognitive impairment (MoCA < 26) compared with 12% of controls, representing a 26-fold higher odds (OR = 26.0, 95% CI 12.2–55.5).

Among stroke subgroups, mean MoCA scores did not differ significantly by stroke type (ischemic 18.2 ± 5.1 vs hemorrhagic 19.0 ± 5.8; $p = 0.392$) or hemisphere (left 18.1 ± 5.4 vs right 18.8 ± 5.2; $p = 0.556$). However, educational level showed a strong gradient effect: participants with ≤8 years of education scored 15.9 ± 4.8, those with 9–12 years 18.8 ± 5.0, and those with >12 years 21.2 ± 4.6 ($F = 8.21$, $p < 0.001$, $\eta^2 = 0.145$).

Correlation analyses confirmed significant associations between cognitive, functional, and clinical variables. MoCA scores correlated negatively with age ($r = −0.47$, $p < 0.001$) and NIHSS ($r = −0.59$, $p < 0.001$), while showing a strong positive correlation with Barthel Index ($r = +0.63$, $p < 0.001$). NIHSS and Barthel Index were inversely correlated ($r = −0.68$, $p < 0.001$), indicating that greater neurological severity corresponded to lower functional independence. Multivariable regression analysis identified education ($\beta = +0.39$, $p < 0.001$), NIHSS score ($\beta = −0.52$, $p < 0.001$), Barthel Index ($\beta = +0.07$, $p = 0.022$), and age ($\beta = −0.21$, $p = 0.001$) as independent predictors of MoCA performance, collectively explaining 61% of variance (adjusted $R^2 = 0.61$; $F = 31.2$, $p < 0.001$). Hypertension exerted a borderline negative effect ($\beta = −1.48$, $p = 0.078$). These results demonstrate that cognitive outcomes following stroke are primarily influenced by educational attainment, neurological severity, and functional recovery rather than lesion laterality or subtype.

DISCUSSION

The present analytical cross-sectional study demonstrated a high prevalence of post-stroke cognitive impairment (PSCI) among stroke survivors in Sialkot, with nearly four out of five participants showing some degree of cognitive dysfunction as assessed by the Montreal Cognitive Assessment (MoCA). Cognitive deficits were significantly associated with older age, lower educational attainment, higher stroke severity (NIHSS), and poorer functional independence (Barthel Index). These findings are consistent with prior international and regional studies that have reported similar determinants of cognitive outcomes following stroke (16,17). The strong association between NIHSS and MoCA underscores the close interplay between neurological deficit severity and global cognitive performance, emphasizing that early neurorehabilitative interventions targeting both domains are essential for optimal recovery trajectories (18).

The observed predominance of mild-to-moderate cognitive impairment aligns with the spectrum reported in multicenter studies, where 60–80% of stroke survivors experience some cognitive decline within the first six months post-event (19). However, unlike some European cohorts that noted a gradual decline over time, the current findings suggest more severe and early-onset cognitive deficits, potentially reflecting delayed acute management and limited access to post-stroke rehabilitation services in low-resource settings (20). Furthermore, the absence of significant associations between stroke hemisphere or type and MoCA performance echoes reports from Patel et al. and Mijajlović et al., suggesting that diffuse vascular pathology and global network disruption, rather than focal lesion laterality, underlie the cognitive sequelae of stroke (2,10).

Educational attainment emerged as a robust protective factor, demonstrating a graded positive relationship with cognitive performance. This finding aligns with the cognitive reserve hypothesis proposed by Stern, wherein higher educational levels and intellectual engagement enhance neural resilience against structural brain injury (11,21). The implication is particularly relevant for developing countries, where educational disparities contribute to unequal vulnerability to post-stroke cognitive decline. Interventions emphasizing lifelong learning, cognitive stimulation, and patient education could therefore serve as low-cost, population-level strategies for mitigating the burden of PSCI.

The significant correlation between functional independence and MoCA scores corroborates earlier findings that motor and cognitive recoveries are interdependent (12,22). Cognitive deficits in executive function, attention, and memory directly impede daily functioning, adherence to rehabilitation, and self-care activities. These interrelationships suggest that integrated multidisciplinary rehabilitation addressing both physical and cognitive domains may yield superior outcomes compared to isolated motor therapy. Furthermore, the present results reaffirm that age-related neuronal loss, reduced neuroplasticity, and accumulation of vascular risk factors compound the impact of stroke on cognition (23). Hypertension, diabetes, and dyslipidemia were highly prevalent among the affected cohort, consistent with the vascular cognitive impairment continuum described by Boehme and colleagues (3). The coexistence of multiple vascular comorbidities likely accelerates small-vessel ischemic damage and impairs white matter connectivity, leading to executive dysfunction and processing speed deficits (24).

From a mechanistic standpoint, PSCI results from multifocal disruptions in frontoparietal and limbic circuits mediated by ischemic injury, inflammatory cascades, and impaired neurovascular coupling (25). Post-stroke neuroinflammation, microglial activation, and secondary amyloid accumulation have also been implicated in cognitive decline trajectories that resemble early vascular dementia (26). The present findings indirectly support these mechanisms, as higher NIHSS—reflecting greater initial injury burden—was independently predictive of poorer MoCA performance, even after controlling for demographic and educational factors. This emphasizes the need for neuroprotective strategies during the acute phase, alongside rehabilitation approaches that promote synaptic reorganization and neuroplasticity during recovery.

Clinically, these results highlight the necessity of incorporating standardized cognitive screening, such as MoCA, into routine post-stroke care, particularly within three to six months post-event when neurorehabilitation is most effective (27). Given the high prevalence of hypertension and metabolic comorbidities in this population, comprehensive secondary prevention that includes vascular risk modification may serve as an adjunct to cognitive recovery. The findings also underscore a major service gap in secondary cities such as Sialkot, where access to neuropsychological assessment and rehabilitation remains limited compared with tertiary centers. Integrating brief cognitive screening into physiotherapy and outpatient follow-up clinics could provide early detection and enable timely referral.

Despite its clinical and public health relevance, this study has certain limitations. The sample size, though adequate for detecting medium-to-large effect sizes, limits generalizability beyond the study setting. The cross-sectional design precludes causal inference and temporal assessment of cognitive change. Furthermore, the study relied on hospital-based recruitment, which may underrepresent community-dwelling stroke survivors with milder symptoms. Nonetheless, strengths include standardized administration of validated instruments (MoCA, NIHSS, and Barthel Index), multicenter sampling, and rigorous adjustment for potential confounders, enhancing internal validity.

Future research should adopt longitudinal designs to track cognitive trajectories across acute, subacute, and chronic stages of recovery, integrating neuroimaging biomarkers such as white matter hyperintensity volume or cortical thickness to delineate underlying neural correlates. Intervention studies exploring combined cognitive–motor rehabilitation, pharmacological neuroprotection, and digital cognitive training could offer novel insights into restoring post-stroke cognitive function. Expanding research to community-level populations and incorporating biomarkers of inflammation and neurodegeneration may also clarify mechanisms linking vascular injury to persistent cognitive impairment.

In conclusion, the present study contributes evidence that post-stroke cognitive impairment is highly prevalent in Pakistani stroke survivors and is primarily driven by age, education, and stroke severity rather than lesion laterality or type. These findings reinforce the need for early, routine cognitive screening, integrated rehabilitation, and education-based preventive strategies to improve long-term quality of life and reduce the societal burden of stroke-related disability.

CONCLUSION

This study concluded that post-stroke cognitive impairment is highly prevalent among stroke survivors in Sialkot hospitals, with nearly four-fifths exhibiting measurable deficits on the Montreal Cognitive Assessment. Age, educational attainment, stroke severity, and functional independence emerged as the strongest predictors of cognitive outcomes, whereas stroke type and hemisphere involvement showed no independent effect. These findings highlight the need for early cognitive screening, particularly in resource-limited clinical settings, and the integration of cognitive assessment into standard post-stroke rehabilitation to enhance recovery and quality of life. Clinically, the results underscore the importance of multidisciplinary management focusing on both neurological and cognitive restoration, while from a research perspective, they support the development of longitudinal and intervention-based studies exploring neuroplasticity-driven rehabilitation strategies tailored to diverse educational and vascular risk profiles.

REFERENCES

1. Kuriakose D, Xiao Z. Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. Int J Mol Sci. 2020;21(20):7609.
2. Mijajlović MD, Pavlović A, Brainin M, Heiss WD, Quinn TJ, Ihle-Hansen H, et al. Post-Stroke Dementia: A Comprehensive Review. BMC Med. 2017;15(1):11.
3. Boehme AK, Esenwa C, Elkind MSV. Stroke Risk Factors, Genetics, and Prevention. Circ Res. 2017;120(3):472–95.
4. Malik R, Dichgans M. Vascular Cognitive Impairment: Mechanisms and Clinical Implications. J Neurol Sci. 2020;412:116730.
5. Farooq MU, Venketasubramanian N, Wasay M. Stroke Care in Pakistan: Current Status and Future Directions. Cerebrovasc Dis. 2021;50(6):643–51.
6. Sherin A, Ahmad R, Khan M. Population Burden and Determinants of Stroke in Pakistan. J Pak Med Assoc. 2020;70(Suppl 3):S113–S118.
7. Hashmi M, Khan M, Wasay M. Growing Burden of Stroke in Pakistan: A Call for Action. J Pak Med Assoc. 2013;63(1):71–3.
8. Khealani BA, Hameed B, Mapari UU. Stroke in Pakistan. J Pak Med Assoc. 2008;58(7):400–3.
9. Jia X, Sun J, Chen S, Ma C, Zhou Y, Wang Y. Montreal Cognitive Assessment vs Mini-Mental State Examination for Detecting Mild Cognitive Impairment After Stroke. Aging Clin Exp Res. 2021;33(5):1157–65.
10. Patel MD, Coshall C, Rudd AG, Wolfe CDA. Cognitive Impairment After Stroke: Clinical Determinants. Stroke. 2002;33(5):1106–13.
11. Stern Y. Cognitive Reserve in Ageing and Alzheimer's Disease. Lancet Neurol. 2009;8(11):1006–12.
12. Saberi A, Etemadifar M, Sadeghi Bahmani D, Brand S. Association Between Global Cognition and Functional Independence in Chronic Stroke. Top Stroke Rehabil. 2020;27(5):356–64.
13. Kaddumukasa M, Katabira E, Sajatovic M, Pundik S, Kayima J, Ddumba E, et al. Determinants of Post-Stroke Cognitive Impairment and Functional Recovery. Neuroepidemiology. 2023;57(3):210–8.
14. 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.
15. Mahoney FI, Barthel DW. Functional Evaluation: The Barthel Index. Md State Med J. 1965;14:61–5.
16. Pendlebury ST, Rothwell PM. Incidence and Prevalence of Dementia Associated with Transient Ischemic Attack and Stroke: Analysis of Population-Based Studies. Lancet Neurol. 2009;8(11):1007–17.
17. Mellon L, Brewer L, Hall P, Horgan F, Williams D, Hickey A. Cognitive Impairment Six Months After Ischemic Stroke: A Population-Based Study. Eur Stroke J. 2015;2(2):158–66.
18. Patel M, Coshall C, Rudd AG, Wolfe CD. Cognitive Impairment After Stroke: Clinical Determinants. Stroke. 2002;33(5):1106–13.
19. Sun JH, Tan L, Yu JT. Post-Stroke Cognitive Impairment: Epidemiology, Mechanisms, and Management. Ann Transl Med. 2014;2(8):80.
20. Venketasubramanian N, Yoon BW, Pandian J, Navarro JC. Stroke Epidemiology in South, East, and South-East Asia: A Review. J Stroke. 2017;19(3):286–94.
21. Stern Y. Cognitive Reserve and Its Implications for Cognitive Aging and Alzheimer's Disease. Lancet Neurol. 2012;11(11):1006–12.
22. Fride Y, Adamit T, Maeir A, Ben Assayag E, Bornstein NM, Korczyn AD, et al. What Are the Correlates of Cognition and Participation to Return to Function After Stroke? Top Stroke Rehabil. 2015;22(5):317–25.
23. Kalaria RN. Vascular Basis for Brain Degeneration: Faltering Controls and Risk Factors for Dementia. Nutr Rev. 2010;68(Suppl 2):S74–87.
24. Jokinen H, Kalska H, Mäntylä R, Ylikoski R, Hietanen M, Pohjasvaara T, et al. Cognitive Profile of Subcortical Ischemic Vascular Disease. J Neurol Sci. 2006;248(1–2):101–7.
25. Kalaria RN, Akinyemi R, Ihara M. Does Vascular Pathology Contribute to Alzheimer's Changes? J Neurol Sci. 2012;322(1–2):141–7.
26. Iadecola C, Anrather J. The Immunology of Stroke: From Mechanisms to Translation. Nat Med. 2011;17(7):796–808.
27. Winstein CJ, Stein J, Arena R, Bates B, Cherney LR, Cramer SC, et al. Guidelines for Adult Stroke Rehabilitation and Recovery: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association. Stroke. 2016;47(6):e98–e169.