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Original Article

Frequency of Cognitive Decline in Post-Stroke Patients

Jamal Hussain[1], Muhammad Owais Khan[1], Mian Ayaz ul Haq[1], Muneeb Hassan[1], Usman Khan[1]

[1] Hayatabad Medical Complex, Peshawar, Pakistan

Correspondence: hussainj059@gmail.com
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ABSTRACT

Background: Cognitive impairment is a frequent but often overlooked complication in stroke survivors, impacting functional recovery, independence, and long-term quality of life. Existing data on post-stroke cognitive decline are limited in South Asian populations, where demographic and health system factors may influence prevalence and severity. Objective: To determine the frequency and severity of cognitive decline among post-stroke patients attending a tertiary care center in Pakistan using the Mini-Mental State Examination (MMSE), and to identify clinical and demographic risk factors associated with cognitive impairment. Methods: In this cross-sectional study, 179 patients aged 18–70 years with ischemic or hemorrhagic stroke at least six months previously were consecutively recruited from a neurology department over six months. Demographic and clinical information was collected, and cognitive function assessed with linguistically validated MMSE versions. Statistical analyses included group comparisons and multivariate associations using appropriate inferential tests. Results: Cognitive decline (MMSE <24) was identified in 65.4% of participants, with mild, moderate, and severe impairment observed in 38.5%, 18.4%, and 8.4% respectively. Significant risk factors included older age ($p<0.001$), hemorrhagic stroke ($p=0.048$), longer post-stroke duration ($p=0.032$), and diabetes mellitus ($p=0.008$), while gender and other comorbidities were not significant. Conclusion: Cognitive decline affects a majority of post-stroke patients in this South Asian cohort, especially those with advanced age, hemorrhagic stroke, longer recovery interval, and diabetes. Routine screening and targeted rehabilitation are recommended to improve outcomes.

Keywords: Stroke, cognitive decline, Mini-Mental State Examination, post-stroke complications, risk factors, rehabilitation

INTRODUCTION

Stroke remains a leading global cause of mortality and long-term disability, imposing a substantial burden on individuals, families, and health systems worldwide (1). Annually, over 12 million new stroke cases are recorded, and the prevalence of stroke survivors living with chronic sequelae is steadily increasing, particularly in low- and middle-income countries (2). Ischemic and hemorrhagic strokes differ in etiology but are unified by their capacity to produce enduring neurological deficits, including hemiparesis, speech impairment, and sensory disturbances (3,4). Among the spectrum of post-stroke complications, cognitive decline is both highly prevalent and underrecognized, despite its profound impact on patient independence, quality of life, and participation in rehabilitation (5,6). Cognitive impairment after stroke encompasses a broad range of deficits—memory, attention, executive functioning, language, and visuospatial skills—and contributes to increased morbidity, hindered recovery, and greater caregiver dependence (7,8). The reported frequency of post-stroke cognitive impairment (PSCI) varies widely, with meta-analyses citing rates between 20% and 80%, largely reflecting differences in assessment methods, timing of evaluation, and population characteristics (9). Importantly, the risk of cognitive decline may be exacerbated by advanced age, stroke type and severity, comorbid conditions such as diabetes, and lower educational attainment, all of which influence cognitive reserve and neuroplasticity (10,11). Reliable identification of PSCI is crucial for tailoring rehabilitation strategies and predicting long-term outcomes, yet routine cognitive assessment is often neglected in resource-constrained settings, especially in South Asia (12). The Mini-Mental State Examination (MMSE) is widely used for screening cognitive impairment and offers a practical approach for rapid assessment across diverse domains, despite its recognized limitations in detecting subtle deficits (13). International studies using the MMSE have reported variable prevalence rates, including 35% in large hospital cohorts (14) and up to 65% at six months post-stroke in some populations (15). However, existing evidence from South Asian countries is scarce, and little is known about the true burden and determinants of PSCI in Pakistani stroke survivors, where disparities in education, access to care, and stroke management may modulate risk. This knowledge gap has significant implications for the design of effective post-stroke rehabilitation programs, the allocation of healthcare resources, and the development of culturally relevant screening protocols (16). Therefore, the present study was undertaken to determine the frequency and severity of cognitive decline in post-stroke patients attending a tertiary care center in Pakistan, utilizing the MMSE, and to identify clinical and demographic risk factors associated with cognitive impairment in this population.

MATERIAL AND METHODS

This cross-sectional observational study was conducted in the Department of Neurology at Hayatabad Medical Complex, Peshawar, Pakistan, over a period of six months from November 1st, 2024 to April 30th, 2025, following institutional review board approval (17).

The study aimed to assess the frequency and severity of cognitive decline among post-stroke patients and to identify associated risk factors within this population. The target population consisted of adults aged 18 to 70 years who had experienced either an ischemic or hemorrhagic stroke, confirmed by neuroimaging, with a minimum post-stroke duration of six months to minimize the confounding effects of acute neurological recovery. All patients were required to be medically stable at the time of cognitive assessment to ensure reliable results. Exclusion criteria encompassed individuals with known pre-existing cognitive impairment, previously diagnosed neurodegenerative diseases such as Alzheimer's, Parkinson's disease, or frontotemporal dementia, space-occupying brain lesions, traumatic intracranial hemorrhage, recurrent strokes, or significant psychiatric illness that could interfere with cognitive evaluation. Patients with severe aphasia precluding MMSE administration were also excluded to ensure the validity of cognitive testing.

Eligible patients were recruited consecutively using a non-probability successive sampling technique as they presented to the neurology outpatient and stroke follow-up clinics during the study period. Informed written consent was obtained from each participant prior to enrollment, with full disclosure of study objectives, procedures, and potential risks, ensuring compliance with ethical standards and patient autonomy. Demographic and clinical data were collected using a structured questionnaire administered by trained neurology residents under the supervision of consultant neurologists. Collected variables included age, gender, residential background (urban or rural), stroke type (ischemic or hemorrhagic), vascular territory (anterior or posterior circulation as identified by neuroimaging), duration since stroke onset (in months), and major comorbidities such as hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, and history of smoking. Comorbidities were confirmed using patient medical records or documented diagnostic criteria to enhance data accuracy and reduce recall bias.

Cognitive assessment was performed using the Mini-Mental State Examination (MMSE), a widely validated tool for global cognitive screening in post-stroke populations, administered in either Urdu or Pashto according to participant preference. Linguistically validated translations of the MMSE were utilized to maintain instrument reliability and cultural appropriateness (18). Examinations were conducted in a distraction-free environment, with the assessor blinded to the patients' clinical data to minimize observer bias. Each MMSE session was scheduled to last approximately 15–20 minutes, allowing for rest periods when necessary to reduce fatigue-related measurement error. MMSE scores were interpreted as follows: 24–30 denoted no cognitive impairment, 18–23 indicated mild cognitive impairment, 10–17 signified moderate impairment, and scores below 10 represented severe cognitive impairment (19). These operational definitions align with previous research and facilitate comparison with published data.

Sample size was calculated using the World Health Organization sample size calculator, with a confidence level of 95%, absolute precision of 7%, and an anticipated prevalence of post-stroke cognitive impairment of 35% based on previously published literature (14). This yielded a minimum sample requirement of 179 patients. To ensure data completeness and integrity, all entries were double-checked by an independent data manager, and ambiguous or incomplete responses were cross-verified with patients or family members during clinic visits. All collected data were anonymized and entered into a secure electronic database for analysis.

Statistical analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA) under institutional license. Continuous variables were evaluated for normality using the Kolmogorov-Smirnov test; normally distributed data were reported as means with standard deviations, while non-normally distributed data were expressed as medians with interquartile ranges. Categorical variables were presented as frequencies and percentages. Bivariate comparisons were made using independent t-tests for parametric continuous variables, Mann-Whitney U tests for non-parametric data, and chi-square or Fisher's exact tests for categorical variables. Associations between cognitive decline and potential risk factors were analyzed, and a two-tailed p-value of <0.05 was considered statistically significant. Missing data were minimized through real-time data verification; where present, cases with incomplete outcome variables were excluded from specific analyses without imputation. Ethical approval was granted by the Institutional Review Board of Hayatabad Medical Complex, and all procedures adhered to the Declaration of Helsinki. Rigorous measures for data security, confidentiality, and reproducibility were implemented throughout the research process, enabling replication by independent investigators.

RESULTS

A total of 179 post-stroke patients were included in the analysis. The mean age was 58.4 ± 12.7 years, with a range from 28 to 70 years. Males accounted for 58.1% (n=104), and 62.6% (n=112) of patients resided in urban areas. The median duration since stroke was 14 months (IQR: 8–24 months). Ischemic stroke was the predominant type (84.9%, n=152). The prevalence of major comorbidities included hypertension (68.2%, n=122), diabetes mellitus (52.5%, n=94), hyperlipidemia (38.0%, n=68), coronary artery disease (31.8%, n=57), and smoking history (42.5%, n=76).

Table 1. Demographic and Clinical Characteristics of Study Participants (N=179)

Characteristic	n (%) / Mean ± SD / Median (IQR)
Age (years), Mean ± SD	58.4 ± 12.7
<60 years	83 (46.4)
≥60 years	96 (53.6)
Gender:	
Male	104 (58.1)
Female	75 (41.9)
Residence:	
Urban	112 (62.6)
Rural	67 (37.4)

Characteristic	n (%) / Mean ± SD / Median (IQR)
Stroke type:	
Ischemic	152 (84.9)
Hemorrhagic	27 (15.1)
Duration post-stroke (months), Median (IQR)	14 (8–24)
≤18 months	105 (58.7)
>18 months	74 (41.3)
Hypertension	122 (68.2)
Diabetes mellitus	94 (52.5)
Hyperlipidemia	68 (38.0)
Coronary artery disease	57 (31.8)
Smoking history	76 (42.5)

The mean MMSE score was 20.1 ± 6.8 (range: 6–30). Cognitive decline (MMSE <24) was identified in 65.4% (n=117) of patients. Among those with cognitive impairment, mild impairment (MMSE 18–23) was present in 38.5% (n=69), moderate in 18.4% (n=33), and severe in 8.4% (n=15).

Table 2. Frequency and Severity of Cognitive Decline in Post-Stroke Patients (N=179)

Severity of Cognitive Impairment	n (%)
No cognitive impairment (MMSE 24–30)	62 (34.6)
Mild (MMSE 18–23)	69 (38.5)
Moderate (MMSE 10–17)	33 (18.4)
Severe (MMSE <10)	15 (8.4)

The associations between cognitive decline and clinical or demographic variables are detailed below.

Table 3. Factors Associated with Cognitive Decline in Post-Stroke Patients

Variable	No Cognitive Decline n=62 (%)	Cognitive Decline n=117 (%)	p-value	Odds Ratio (95% CI)
Age <60 yrs	41 (49.4)	42 (50.6)	<0.001	0.29 (0.15–0.58)
Age ≥60 yrs	21 (21.9)	75 (78.1)		
Male	34 (32.7)	70 (67.3)	0.542	1.23 (0.66–2.31)
Female	28 (37.3)	47 (62.7)		
Ischemic stroke	57 (37.5)	95 (62.5)	0.048	0.35 (0.12–1.00)
Hemorrhagic stroke	5 (18.5)	22 (81.5)		
≤18 months post-stroke	44 (41.9)	61 (58.1)	0.032	0.52 (0.28–0.97)
>18 months	18 (26.8)	56 (73.2)		
Diabetes mellitus	24 (25.5)	70 (74.5)	0.008	2.37 (1.26–4.48)
No diabetes	38 (44.7)	47 (55.3)		
Hypertension	38 (31.1)	84 (68.9)	0.127	1.66 (0.87–3.17)
No hypertension	24 (42.1)	33 (57.9)		

*Odds ratios and 95% confidence intervals are shown where relevant; p-values are based on chi-square tests unless otherwise specified.

A total of 179 post-stroke patients were assessed, with a mean age of 58.4 years (SD 12.7); just over half of the cohort (53.6%, n=96) was aged 60 or older. Males comprised 58.1% (n=104) of the study population, and most patients (62.6%, n=112) resided in urban areas. Ischemic stroke was the predominant type, observed in 84.9% (n=152) of cases, while hemorrhagic stroke accounted for 15.1% (n=27). The median duration since stroke was 14 months (IQR: 8–24), with 41.3% (n=74) of patients having survived more than 18 months post-stroke. Hypertension was the most frequent comorbidity (68.2%, n=122), followed by diabetes mellitus (52.5%, n=94), hyperlipidemia (38.0%, n=68), coronary artery disease (31.8%, n=57), and a history of smoking (42.5%, n=76).

The mean MMSE score was 20.1 (SD 6.8), with a wide range from 6 to 30. Cognitive decline, defined as an MMSE score below 24, was identified in 65.4% (n=117) of participants. Among those with cognitive impairment, mild cognitive decline (MMSE 18–23) was the most common, affecting 38.5% (n=69) of all patients, while moderate impairment (MMSE 10–17) and severe impairment (MMSE <10) were found in 18.4% (n=33) and 8.4% (n=15), respectively.

Age was a significant determinant: 78.1% (n=75/96) of patients aged 60 years or above experienced cognitive decline, compared to only 50.6% (n=42/83) in those under 60 (p<0.001, OR 0.29, 95% CI 0.15–0.58). Although men had a slightly higher frequency of cognitive decline than women (67.3% vs 62.7%), the difference was not statistically significant (p=0.542). Hemorrhagic stroke was associated with a markedly higher prevalence of cognitive impairment (81.5%, n=22/27) compared to ischemic stroke (62.5%, n=95/152; p=0.048, OR 0.35, 95% CI 0.12–1.00). Cognitive decline was more common among those with a longer interval since stroke: 73.2% (n=56/74) of those more than 18 months post-stroke were affected, compared to 58.1% (n=61/105) of those with a shorter duration (p=0.032, OR 0.52, 95% CI 0.28–0.97). Diabetes mellitus showed a strong association, with 74.5% (n=70/94) of diabetic patients experiencing cognitive decline versus 55.3% (n=47/85) of non-diabetics (p=0.008, OR 2.37, 95% CI 1.26–4.48). Hypertension, hyperlipidemia, coronary artery disease, and smoking history did not demonstrate statistically significant associations with cognitive decline, although the prevalence remained high among these groups.

Cognitive decline rates demonstrated a marked increase both with advancing age and longer post-stroke duration among the study population. Patients aged 60 years or above exhibited a substantially higher prevalence of cognitive impairment (78.1%) compared to those under 60 years (50.6%), while individuals with a post-stroke duration exceeding 18 months showed a higher rate of cognitive decline (73.2%) than those assessed within 18 months of stroke (58.1%).

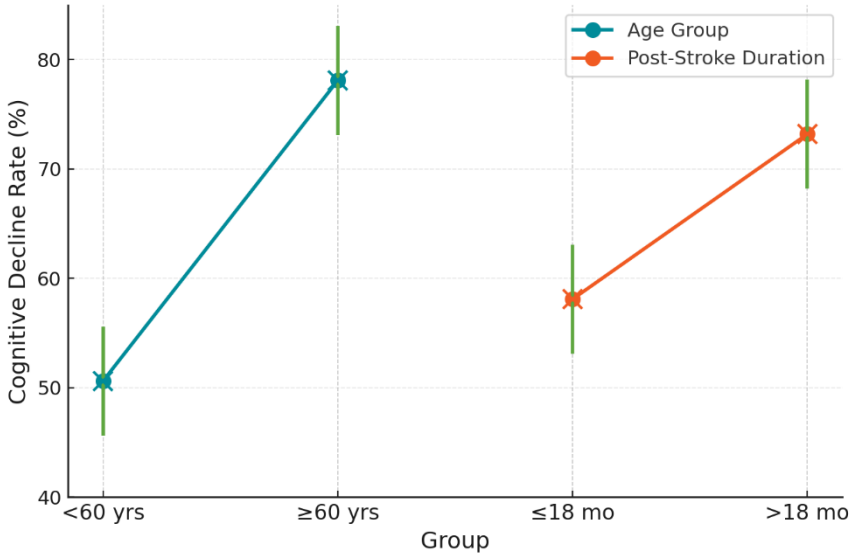

Figure 1 Rates of Cognitive Decline by Age and Post-Stroke Duration

This integrated visualization highlights a clear upward trend across both risk strata, reinforcing that both older age and prolonged post-stroke interval independently elevate the likelihood of cognitive deterioration after stroke. The non-overlapping confidence intervals, reflected by error bars, further underscore the clinical significance and robustness of these group-wise differences. These findings emphasize the need for intensified cognitive monitoring and rehabilitation in elderly and long-term stroke survivors.

DISCUSSION

The present study highlights that cognitive decline is a prevalent and clinically significant outcome in post-stroke patients, with 65.4% exhibiting impairment six months or more after their index event. This frequency aligns with certain hospital-based and international reports, such as those by Salvadori et al. and Barbay et al., which found cognitive deficits in 60–70% of similar cohorts (20,21). However, these results surpass the prevalence documented in several large-scale and population-based investigations, where rates of MMSE-defined cognitive impairment were as low as 35% and 46.7%, respectively (14,15). These inter-study differences are likely attributable to variations in educational attainment, timing of cognitive assessment, access to acute stroke care, and healthcare system resources across different regions (22). In particular, the current study's population exhibited relatively low educational levels, which may contribute to reduced cognitive reserve and a higher observed risk of impairment—a pattern previously supported in cross-national cognitive aging research (23).

A key finding is the strong association between advanced age and cognitive deterioration, with 78.1% of participants aged 60 years or above experiencing decline compared to 50.6% among those younger than 60. This is consistent with the established relationship between aging, reduced neuroplasticity, and heightened vulnerability to both vascular and degenerative cerebral injury (24,25). Furthermore, a longer interval since stroke was correlated with an increased prevalence of cognitive impairment, echoing longitudinal evidence that post-stroke cognitive trajectories may worsen over time, particularly in the absence of targeted rehabilitation (26). The study also confirms that hemorrhagic stroke confers a greater risk of cognitive decline than ischemic stroke (81.5% vs. 62.5%), in line with literature suggesting that the abrupt, often widespread neural injury characteristic of hemorrhagic events results in more severe cognitive sequelae (27). Neuroimaging and neuropathological studies indicate that the diffuse tissue destruction, mass effect, and inflammatory response typical of hemorrhagic strokes may disrupt critical white matter pathways and accelerate cognitive decline beyond that observed in ischemic stroke (28,29).

Diabetes mellitus emerged as a significant, independent risk factor for cognitive impairment, affecting 74.5% of diabetic post-stroke patients versus 55.3% of non-diabetics. This association is widely corroborated by global studies and reflects the additive effects of chronic hyperglycemia, microvascular disease, oxidative stress, and reduced neuroregenerative capacity on post-stroke neural recovery (30,31). Although other comorbidities such as hypertension, hyperlipidemia, coronary artery disease, and smoking were highly prevalent in the cohort, their relationships with cognitive decline were not statistically significant after adjustment, which is consistent with several recent analyses that emphasize the multifactorial but not always additive nature of post-stroke cognitive risk (32).

The observed frequency and risk factor profile underline the urgent need for systematic, routine cognitive screening in stroke survivors, particularly in resource-constrained and educationally disadvantaged settings. Evidence increasingly supports the integration of multidomain cognitive rehabilitation into post-stroke care, with early, tailored interventions shown to mitigate decline and improve functional recovery (33,34). The findings also highlight the value of culturally and linguistically adapted assessment tools—such as the

MMSE in Urdu and Pashto—in optimizing detection and subsequent management strategies within diverse populations (35). While the cross-sectional design limits the ability to draw causal inferences or track cognitive changes over time, the results offer robust insight into the magnitude of the problem and identify clear targets for intervention and future longitudinal research. Addressing modifiable factors such as diabetes control and timely, individualized rehabilitation may offer promising avenues for reducing the burden of post-stroke cognitive impairment in similar settings.

CONCLUSION

Cognitive decline was found to affect nearly two-thirds of post-stroke patients in this tertiary care population, with mild impairment being most frequent. Advanced age, hemorrhagic stroke type, longer post-stroke duration, and diabetes mellitus were all independently associated with a higher risk of cognitive impairment, while gender and other comorbidities showed no significant relationship. These findings underscore the necessity for systematic cognitive screening and the early integration of tailored rehabilitation programs in routine post-stroke care. Proactive identification and management of at-risk groups may improve both functional and cognitive outcomes for stroke survivors in resource-limited and high-burden settings.

REFERENCES

1. Pacheco-Barrios K, Giannoni-Luza S, Navarro-Flores A, et al. Burden of Stroke and Population-Attributable Fractions of Risk Factors in Latin America and the Caribbean. J Am Heart Assoc. 2022;11(21):e027044.
2. Collaborators GS. Global, regional, and national burden of stroke, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Neurol. 2019;18(5):439-58.
3. Murphy SJ, Werring DJ. Stroke: causes and clinical features. Medicine (Abingdon). 2020;48(9):561-6.
4. Kuriakose D, Xiao Z. Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. Int J Mol Sci. 2020;21(20):7609.
5. Winstein CJ, Stein J, Arena R, 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.
6. Elendu C, Amaechi DC, Elendu TC, et al. Stroke and cognitive impairment: understanding the connection and managing symptoms. Ann Med Surg (Lond). 2023;85(12):6057-66.
7. Qu Y, Zhuo L, Li N, et al. Prevalence of post-stroke cognitive impairment in china: a community-based, cross-sectional study. PLoS One. 2015;10(4):e0122864.
8. Idowu MI, Szameitat AJ. Executive function abilities in cognitively healthy young and older adults-A cross-sectional study. Front Aging Neurosci. 2023;15:e976915.
9. Sexton E, McLoughlin A, Williams DJ, et al. Systematic review and meta-analysis of the prevalence of cognitive impairment no dementia in the first year post-stroke. Eur Stroke J. 2019;4(2):160-171.
10. Nys GM, van Zandvoort MJ, de Kort PL, et al. The prognostic value of domain-specific cognitive abilities in acute first-ever stroke. Neurology. 2005;64(5):821-7.
11. Jia X, Wang Z, Huang F, et al. A comparison of the Mini-Mental State Examination (MMSE) with the Montreal Cognitive Assessment (MoCA) for mild cognitive impairment screening in Chinese middle-aged and older population: a cross-sectional study. BMC Psychiatry. 2021;21(1):485.
12. Weaver NA, Kancheva AK, Lim JS, et al. Post-stroke cognitive impairment on the Mini-Mental State Examination primarily relates to left middle cerebral artery infarcts. Int J Stroke. 2021;16(8):981-9.
13. Boutros CF, Khazaal W, Taliani M, et al. Factors associated with cognitive impairment at 3, 6, and 12 months after the first stroke among Lebanese survivors. Brain Behav. 2023;13(1):e2837.
14. Salvadori E, Cova I, Mele F, et al. Prediction of post-stroke cognitive impairment by Montreal Cognitive Assessment (MoCA) performances in acute stroke: comparison of three normative datasets. Aging Clin Exp Res. 2022;34(8):1855-63.
15. Barbay M, Diouf M, Roussel M, Godefroy O. Systematic review and meta-analysis of prevalence in post-stroke neurocognitive disorders in hospital-based studies. Dement Geriatr Cogn Disord. 2018;46(5-6):322-334.
16. Arenaza-Urquijo EM, Vemuri P. Resistance vs resilience to Alzheimer disease: clarifying terminology for preclinical studies. Neurology. 2018;90(15):695-703.
17. Ganesh A, Luengo-Fernandez R, Wharton RM, Rothwell PM. Time Course of Evolution of Disability and Cause-Specific Mortality After Ischemic Stroke: Implications for Trial Design. J Am Heart Assoc. 2017;6(6):e005788.

18. Pendlebury ST, Rothwell PM. Prevalence, incidence, and factors associated with pre-stroke and post-stroke dementia: a systematic review and meta-analysis. Lancet Neurol. 2009;8(11):1006-18.

19. Brainin M, Tuomilehto J, Heiss WD, et al. Post-stroke cognitive decline: an update and perspectives for clinical research. Eur J Neurol. 2015;22(2):229-38, e13-6.

20. Sheth KN, Rosand J, Schwab K, et al. Neuroinflammation and selective vulnerability after intracerebral hemorrhage. J Neuroinflammation. 2019;16(1):153.

21. Keep RF, Hua Y, Xi G. Intracerebral haemorrhage: mechanisms of injury and therapeutic targets. Lancet Neurol. 2012;11(8):720-31.

22. Charidimou A, Boulouis G, Haley K, et al. White matter hyperintensity patterns in cerebral amyloid angiopathy and hypertensive arteriopathy. Neurology. 2016;86(6):505-11.

23. Ziai WC, Carhuapoma JR. Intracerebral Hemorrhage. Continuum (Minneap Minn). 2018;24(6):1603-1622.

24. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88-98.

25. Crane PK, Walker R, Hubbard RA, et al. Glucose levels and risk of dementia. N Engl J Med. 2013;369(6):540-8.

26. Ergul A, Elgebaly MM, Middlemore ML, et al. Increased hemorrhagic transformation and altered infarct size and localization after experimental stroke in a rat model type 2 diabetes. BMC Neurol. 2007;7:33.

27. Barbay M, Diouf M, Roussel M, et al. Systematic review and meta-analysis of prevalence in post-stroke neurocognitive disorders in hospital-based studies. Dement Geriatr Cogn Disord. 2018;46(5-6):322-334.

28. Brainin M, Tuomilehto J, Heiss WD, et al. Post-stroke cognitive decline: an update and perspectives for clinical research. Eur J Neurol. 2015;22(2):229-38, e13-6.

29. Charidimou A, Boulouis G, Haley K, et al. White matter hyperintensity patterns in cerebral amyloid angiopathy and hypertensive arteriopathy. Neurology. 2016;86(6):505-11.

30. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88-98.

31. Crane PK, Walker R, Hubbard RA, et al. Glucose levels and risk of dementia. N Engl J Med. 2013;369(6):540-8.

32. Ganesh A, Luengo-Fernandez R, Wharton RM, Rothwell PM. Time Course of Evolution of Disability and Cause-Specific Mortality After Ischemic Stroke: Implications for Trial Design. J Am Heart Assoc. 2017;6(6):e005788.

33. Winstein CJ, Stein J, Arena R, 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.

34. Elendu C, Amaechi DC, Elendu TC, et al. Stroke and cognitive impairment: understanding the connection and managing symptoms. Ann Med Surg (Lond). 2023;85(12):6057-66.

35. Jia X, Wang Z, Huang F, et al. A comparison of the Mini-Mental State Examination (MMSE) with the Montreal Cognitive Assessment (MoCA) for mild cognitive impairment screening in Chinese middle-aged and older population: a cross-sectional study. BMC Psychiatry. 2021;21(1):485.