DOI: 10.21088/ijnns.0975.0223.17325.2

International Journal of Neurology and Neurosurgery
Volume 17, Number 3 September-December 2025

Original Article

Assessment of Neurocognitive Functions in Post-Acute Stroke Patients Undergoing Endovascular Intervention or Decompression

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How to cite this article:

Rinku Kumar Sharma, Raman Kumar Shankar, DP Sharma. Assessment of Neurocognitive Functions in Post-Acute Stroke Patients Undergoing Endovascular Intervention or Decompression. Int. J Neuro Neurosurgery. 2025; 17(3): 212-216.

ABSTRACT

Background: Stroke is a leading cause of adult disability and mortality worldwide. Beyond motor and sensory deficits, cognitive dysfunction after stroke substantially affects independence and rehabilitation. Timely detection of post-stroke cognitive impairment (PSCI) allows early intervention. The present study assessed neurocognitive functions in post-acute stroke patients who underwent endovascular or decompressive surgical intervention and compared them with healthy controls using the Addenbrooke's Cognitive Examination III (ACE-III).

Methods: This hospital-based observational case–control study included 40 consecutive patients (18–80 years) with first-ever acute stroke (< 7 days) admitted to the Neurosciences Department, Santokba Durlabhji Memorial Hospital, Jaipur, and 40 age and sex-matched healthy controls. Cases underwent endovascular thrombectomy, aneurysm coiling, clipping, or decompressive craniectomy. Cognitive performance was evaluated at 3 and 6 months using the Hindi-adapted ACE-III assessing five domains attention/orientation, memory, fluency, language, and visuospatial abilities. Statistical analysis used SPSS v22; $p < 0.05$ was considered significant.

Results: Mean patient age was 58.6 ± 11.2 years; 60%were male. Ischemic stroke comprised 72.5%of cases and haemorrhagic 27.5%. Hypertension (42.5%) and diabetes (27.5%) were the most frequent comorbidities. At 3 months, 72.5%of patients demonstrated cognitive impairment versus 62.5%at 6 months. Total ACE-III scores improved from 60.9 ± 10.9 to 74.5 ± 10.7 ($p < 0.001$). Memory and language domains were most affected initially. Cognitive outcomes did not differ significantly by stroke subtype.

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➢ **Received:** 16-10-2025 ➢ **Accepted:** 20-12-2025

Conclusion: Cognitive deficits are highly prevalent in post-acute stroke patients even after successful intervention. Modest recovery occurs by 6 months but multi-domain impairment persists. Routine ACE-III screening and targeted cognitive rehabilitation should be integral to post-stroke care.

KEYWORDS:

• Stroke • Neurocognitive Impairment • Addenbrooke's Cognitive Examination Iii • Endovascular Intervention • Decompressive Craniectomy • Cognitive Rehabilitation • Post-Stroke Dementia.

INTRODUCTION

Stroke represents a major global health burden, ranking as the second leading cause of death and the third leading cause of disability-adjusted life years worldwide.[1] Defined by the World Health Organization as rapidly developing clinical signs of focal or global cerebral dysfunction of vascular origin persisting for at least 24 hours, stroke encompasses ischemic and haemorrhagic subtypes.[2] With the increasing longevity of populations, the prevalence of stroke survivors experiencing long-term neurological sequelae continues to rise.[3]

While motor recovery has been emphasized traditionally, cognitive impairment after stroke is increasingly recognized as a key determinant of functional outcome. Post-stroke cognitive impairment (PSCI) is estimated to affect 30–50% of survivors.[4,5] Cognitive deficits may involve memory, attention, executive function, language, and visuospatial processing.[6] These impairments compromise the ability to perform daily activities, limit participation in rehabilitation, and increase dependence and caregiver burden.[7] Moreover, PSCI is a predictor of recurrent stroke, institutionalization, and mortality.[8]

The mechanisms underlying cognitive dysfunction after stroke are multifactorial, involving direct ischemic or haemorrhagic injury to cortical and subcortical regions, secondary neurodegeneration, inflammatory responses, and pre-existing small-vessel disease.[9] The severity and pattern of impairment depend on lesion location, size, and vascular territory. Socio-demographic and vascular risk factors age, education, hypertension, diabetes, dyslipidemia, and lifestyle further influence cognitive outcome.[10]

Standardized cognitive assessment tools are essential for early identification. The Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA) are widely used but may lack sensitivity for subtle domain-specific deficits.[11] The Addenbrooke's Cognitive Examination-III (ACE-III) provides comprehensive evaluation across five domains with a total score of 100 and high diagnostic accuracy for mild cognitive impairment and dementia.[12] Its Hindi version enables culturally relevant application in Indian patients.[13]

Endovascular interventions and decompressive craniectomy have significantly improved survival in acute stroke. However, data on neurocognitive outcomes following these procedures remain scarce, particularly in Indian settings. Understanding cognitive trajectories can inform post-operative rehabilitation strategies.

This study therefore aimed to (1) evaluate neurocognitive functions in post-acute stroke patients undergoing endovascular or decompressive procedures using the Hindi ACE-III; (2) compare cognitive performance with matched healthy controls; and (3) identify the domains most affected and their temporal progression.

MATERIALS AND METHODS

Study Design and Setting

This prospective observational case–control study was conducted in the Department of Neurosciences, Santokba Durlabhji Memorial Hospital (SDMH), Jaipur, between October 2022 and February 2024. Institutional Ethics Committee approval and written informed consent were obtained.

Participants

Cases: Patients aged 18–80 years with first-ever acute stroke (< 7 days from on set) confirmed by CT or MRI and who underwent endovascular thrombectomy, aneurysm coiling/clipping, or decompressive craniectomy.

Controls: Age-and sex-matched healthy attendants or relatives without neurological or psychiatric illness.

Exclusion criteria: Severe aphasia, unconsciousness, recurrent or chronic stroke, Parkinson's disease, epilepsy, blindness/ deafness, or psychiatric illness in self or first-degree relatives.

Sample Size

Assuming a PSCI prevalence of 74%,[14] 80% power, and 95% confidence, the minimum sample required was 36 per group (EpiInfo v7); 40 patients and 40 controls were included.

Data Collection

Demographic variables (age, sex, education, occupation, residence), vascular risk factors (hypertension, diabetes, dyslipidemia, family history), and clinical features were recorded. All cases underwent standard laboratory evaluation and neuroimaging to confirm stroke subtype and site.

Cognitive Assessment

Neurocognitive evaluation used the Hindi version of the **Addenbrooke's Cognitive Examination-III (ACE-III)** administered at 3 and 6 months post-stroke by trained clinicians. The ACE-III comprises five domains with the following maximum scores:

Domain	Max Score
Attention and Orientation	18
Memory	26
Fluency	14
Language	26
Visuospatial	16
Total	**100**

A total score ≥ 88 was considered normal; 83–87 mild impairment; ≤ 82 significant impairment.[12]

Statistical Analysis

Data were analyzed using SPSS v22 (IBM, USA). Continuous variables were expressed as mean ± SD; categorical data as percentages. Between-group differences were tested using Student's t-test or Chi-square/Fisher's exact test. Within-subject changes between 3 and 6 months were compared with paired t-tests. Significance was set at $p < 0.05$.

RESULTS

Demographic and Socioclinical Profile

Table 1: Sociodemographic Characteristics of Study Participants

Variable	Patients (n = 40)	Controls (n = 40)	p-value
Mean age (yrs)	58.6 ± 11.2	56.9 ± 10.7	0.41 (NS)
Male : Female	24 : 16	23 : 17	0.82 (NS)
Education ≤ Middle school	57.5%	50%	0.74 (NS)
Urban residence	80%	80%	—
Occupation (Business/Farmer/Housewife/Other)	30/22.5/20/27.5%	—	—

Mean age of stroke patients was 58.6 ± 11.2 years (range 32–78). Males constituted 60% (n = 24). The control group had comparable age and sex distribution ($p > 0.05$). Most participants (80%) were urban residents, and 57.5% of cases had education up to middle school. Occupationally, businessmen formed 30%, farmers 22.5%, homemakers 20%, and others 27.5%.

Table 2: Clinical Profile of Stroke Patients

Parameter	Frequency	Percentage (%)
Hypertension	17	42.5
Diabetes mellitus	11	27.5

Hypertension was present in 42.5%, diabetes in 27.5%, and positive family history in 5%. Ischemic stroke accounted for 72.5% and haemorrhagic 27.5%.

Cognitive Impairment Prevalence

At 3 months, 29 patients (72.5%) exhibited cognitive impairment; this decreased to 25 (62.5%) at 6 months. Improvement was statistically significant ($p < 0.05$).

ACE-III Scores

At 3 months, mean total ACE-III score in patients was 60.9 ± 10.9 versus 91.3 ± 8.1 in controls ($p < 0.001$). By 6 months, patient scores improved to 74.5 ± 10.7 ($p < 0.001$ vs 3 months) but remained lower than controls (90.2 ± 8.1).

Memory (14.7 ± 3.9 vs 23.7 ± 2.1) and language (17.7 ± 2.9 vs 25.2 ± 1.1) were most affected at 3 months. At 6 months, memory and language improved (17.9 ± 3.9 and 20.7 ± 3.0) but deficits persisted.

Stroke Subtype and Cognition

Among 29 ischemic strokes, 26 (89.6%) had PSCI at 3 months and 23 (79.3%) at 6 months. Among 11 haemorrhagic strokes, 3 (27%) and 2 (18%) showed PSCI at respective follow-ups ($p > 0.05$).

DISCUSSION

This study provides evidence that cognitive dysfunction remains a frequent and disabling sequel of acute stroke even after advanced endovascular or decompressive interventions. Approximately three-quarters of patients exhibited PSCI at 3 months, with partial recovery at 6 months, mirroring global data reporting 30–70% prevalence.[4,5,15]

Demographic Influences

Older age correlated with higher stroke incidence and poorer cognitive scores, consistent with previous Indian and international findings.[10,16] Male predominance may reflect greater vascular risk exposure. Lower educational attainment was associated with more severe impairment, supporting the cognitive-reserve hypothesis.[17]

Cognitive Pattern

Memory and language were consistently the most affected domains, similar to observations by Tham *et al.*[13] and Chaurasia *et al.*[14] Deficits in these domains suggest involvement of temporal and frontal cortical networks. Improvement in fluency and attention between 3 and 6 months may indicate neuroplastic adaptation facilitated by rehabilitation and compensatory learning.

Stroke Subtype Differences

Although ischemic strokes demonstrated higher cognitive impairment than haemorrhagic, statistical significance was not achieved, possibly due to small sample size. Prior studies show cortical ischemic lesions, particularly in the left hemisphere, correlate strongly with PSCI.[15]

Comparison with Other Studies

Fiedorová *et al.* reported total ACE-R scores of 86.2 in stroke patients versus 91.2 in controls ($p < 0.01$),[6] comparable to our 60.9 vs 91.3 gap considering cultural adaptation. Pendlebury and Rothwell's meta-analysis found 44% cognitive impairment at 3 months and 34% at 1 year[5]; our higher prevalence likely reflects hospital-based recruitment with more severe strokes.

Clinical Implications

Cognitive dysfunction independently predicts poor rehabilitation engagement, increased dependency, and reduced quality of life.[7,8] Routine ACE-III assessment during follow-up allows domain-specific rehabilitation targeting memory, language, and visuospatial skills. Incorporating neuropsychological and occupational therapy early can mitigate chronic disability.

STRENGTHS AND LIMITATIONS

Strengths include prospective design, standardized ACE-III application, and inclusion of both ischemic and haemorrhagic strokes treated with modern interventions. Limitations are single-center scope, modest sample size, lack of lesion mapping, and follow-up limited to 6 months. Longer studies with imaging correlation and neurorehabilitation outcomes are warranted.

CONCLUSION

Post-stroke cognitive impairment remains common despite successful endovascular or surgical management. Significant though incomplete recovery occurs within 6 months, with persistent deficits particularly in memory and language. Incorporating structured cognitive screening such as the ACE-III into stroke care pathways and initiating early cognitive rehabilitation are essential for optimizing outcomes and quality of life among stroke survivors.

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