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ORIGINAL ARTICLE

Clinical, Neuroanatomical, and Multidomain Cognitive Profiles of Post-Stroke Cognitive Impairment at a Tertiary Referral Hospital in Bali, Indonesia: A Cross-Sectional Study

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ABSTRACT

Background: Post-stroke cognitive impairment (PSCI) compromises functional recovery and independence, yet clinicoradiological and domain-specific profiles in Indonesia remain undercharacterized.

Objective: To characterize the clinical, neuroanatomical, and multidomain cognitive profiles of PSCI and factors associated with moderate-to-severe PSCI at a tertiary referral hospital in Bali, Indonesia.

Methods: This cross-sectional, record-based study enrolled 60 patients with PSCI by total consecutive sampling at the Neurology Polyclinic, Prof. Dr. I.G.N.G. Ngoerah General Hospital (January 2025–April 2026). Cognitive severity and domain deficits were assessed with the Indonesian Montreal Cognitive Assessment (MoCA-Ina). Univariate, bivariate, and multivariable logistic regression were used.

Results: Mean age was 61.71 ± 11.53 years; 60.0% were male and 25.0% had recurrent stroke. Ischemic stroke predominated (68.3%); 51.7% were evaluated within < 1 year of onset and 85.0% had minor admission stroke severity (NIHSS 1–4). Subcortical lesions (95.0%), multiple lesion locations (63.3%), hypertension (80.0%), and multiple comorbidities (71.7%) predominated. Mild, moderate, and severe PSCI accounted for 43.3%, 26.7%, and 30.0% of cases (56.7% moderate-to-severe). Multidomain impairment occurred in 95.0%, affecting memory (95.0%), executive function (85.0%), language (81.7%), attention (75.0%), and visuospatial ability (46.7%). Multiple comorbidities (aOR = 16.92, p = 0.003), admission NIHSS ≥ 5 (aOR = 13.75, p = 0.044), recurrent stroke (aOR = 9.55, p = 0.018), and multiple lesion locations (aOR = 6.18, p = 0.014) were independently associated with moderate-to-severe PSCI (AUC = 0.860).

Conclusion: PSCI was predominantly multidomain, affecting memory and executive function despite minor initial stroke severity, supporting early MoCA-Ina screening and targeted neurorehabilitation.

1. Introduction

Cerebrovascular disease is among the most pressing public health and clinical neurology problems of this century. It ranks as the second leading cause of death and the third leading cause of death and disability combined worldwide.[1–3] The Global Burden of Disease (GBD) 2019 Collaborators reported more than 12.2 million incident strokes and over 101 million prevalent stroke survivors.[3] The World Stroke Organization Global Stroke Fact Sheet 2025 and GBD 2021 report about 160 million disability-adjusted life-years (DALYs) lost annually.[1,2] Projections suggest that, without major gains in primary cardiometabolic prevention and post-acute neurorehabilitation, stroke-related mortality will rise by 50% between 2020 and 2050. Most of this burden falls on low- and middle-income countries (LMICs).[1,2] In Southeast Asia, epidemiological transition, population aging, dietary westernization, and poorly controlled vascular risk factors have produced some of the highest age-standardized stroke incidence and case-fatality rates in the world.[2,4] Indonesia illustrates this trend. National health surveys and hospital registries show that stroke prevalence rose by 56%, from 7.0 per 1,000 population in 2013 to 10.9 per 1,000 population in 2018. Stroke is now the leading cause of adult neurological hospitalization, chronic physical dependency, and catastrophic health expenditure across the archipelago.[4,5]

Acute intravenous thrombolysis, mechanical thrombectomy, and organized stroke unit care have substantially lowered in-hospital case fatality. More survivors are now living with chronic neurobehavioral sequelae that were long underappreciated.[6–8] Post-stroke cognitive impairment (PSCI) is a major cause of lost functional independence, failed return to work, caregiver strain, and secondary institutionalization.[7,8]

The Nor-COAST investigators and the 2023 American Heart Association/American Stroke Association (AHA/ASA) Scientific Statement define PSCI as an objective cognitive deficit of any severity. It ranges from mild cognitive impairment no dementia (PSCIND) to overt post-stroke dementia (PSD). The deficit appears within three to twelve months after a clinically evident ischemic or hemorrhagic cerebrovascular event, whatever the underlying vascular cause.[6–8] International syntheses place measurable PSCI in 40% to 60% of stroke survivors during the first year.[6,7,9–11] Southeast Asian estimates vary widely. Mtambo et al. reviewed three decades of post-stroke cognitive impairment and dementia (PSCID) research in Southeast Asia in a 2025 scoping review (103 articles from 78 studies, 28 of them from Indonesia); they found that PSCI prevalence in Indonesia ranged from 27.6% to 81.2%. The ranges were 21.6% to 76.0% in Malaysia, 12.0% to 57.2% in Singapore, and 50.3% to 55.0% in Thailand. Post-stroke dementia affected up to 48.4% of regional cohorts.[4]

Longitudinal evidence now shows that PSCI is more than a static neurobehavioral deficit. It also drives recurrent vascular events and early death.[9,12,13] The prognostic stakes are high. A 2024 systematic review and meta-analysis by Dowling, Johnson, and Nadareishvili pooled 27 studies and 39,412 ischemic stroke survivors. PSCI was associated with a 59% higher risk of recurrent stroke and a two-fold higher risk of all-cause mortality.[9] A 2024 meta-analysis of 13 longitudinal cohorts by Yu and Wang gave consistent results. PSCI independently raised the hazard of stroke recurrence by 33% (pooled hazard ratio [HR] = 1.33, 95% CI: 1.14–1.55); it raised the hazard of poor long-term functional outcome on the modified Rankin Scale by 68% (pooled HR = 1.68, 95% CI: 1.16–2.05).[12] Clinical cohorts add that cognitive dysfunction detected in the first two weeks after stroke strongly predicts short-term and three-month physical disability. It also impairs motor learning during physiotherapy and lowers adherence to secondary antiplatelet and antihypertensive treatment, which can set up a cycle in which neurovascular deterioration feeds on itself.[14–17]

Despite this prognostic weight, PSCI is often missed in routine neurological follow-up. Severe multidomain cognitive deficits frequently sit behind a mild initial physical deficit.[10,16,18] The NIHSS contributes to the problem. Admission stroke severity is usually measured with the National Institutes of Health Stroke Scale (NIHSS). The scale gives heavy weight to pyramidal hemiparesis, gross sensory loss, visual field cuts, and dominant perisylvian aphasia.[19,20] Patients with small subcortical, striatocapsular, thalamic, or periventricular lacunar infarcts therefore tend to have minor admission NIHSS scores (1–4). So do patients with multifocal deep white matter microvascular injury. Motor recovery is quick, and clinicians may assume a benign course.[16,21] Marsh et al., Milosevich et al., and Gallucci et al. have shown otherwise. Patients with minor stroke often have widespread multidomain deficits in episodic memory, executive set-shifting, attention, and processing speed. The authors attribute this to strategic disconnection of high-centrality fronto-subcortical and limbic white matter tracts.[16,21,22] Diagnostic test accuracy meta-analyses by Wei et al. in 2023 and 2025 add a practical point. The Montreal Cognitive Assessment (MoCA) and its validated Indonesian version (MoCA-Ina) are widely used to detect these covert executive and memory deficits. Optimal screening, however, depends on both the global cutoffs (< 26 and the stroke-specific 21/22 threshold) and the subdomain profile.[17,23,24]

Recent work in multicenter lesion–symptom mapping, resting-state functional connectomics, and molecular neurobiology has changed how focal vascular lesions are thought to produce global multidomain PSCI.[25–28] The Meta VCI Map consortium (Coenen et al., 2024) harmonized individual patient data from 1,568 ischemic stroke patients. White matter hyperintensity (WMH) burden in strategic tracts was linked to cognitive outcome: WMH in the left anterior thalamic radiation was associated with worse attention, executive function, and processing speed, and WMH in the forceps major with slower processing speed, independent of age, sex, education, and total infarct volume.[25] Machine-learning prediction models and prospective cohorts add more. They link recurrent cerebrovascular events, multifocal lesion topography, arterial hypertension, diabetes mellitus, insulin resistance, atrial fibrillation, and chronic kidney disease with post-stroke cognitive impairment.[26–31] Even so, Indonesia carries one of the heaviest stroke burdens in Asia. Detailed clinicoradiological studies that combine neuroanatomical lesion mapping, vascular multimorbidity stratification, and MoCA-Ina subdomain profiling with multivariable risk modeling are still almost absent in Bali.[4,5] That gap motivated the present study. Its novelty is that it offers, to our knowledge, the first integrated clinical, neuroanatomical, and multidomain cognitive profile of PSCI at the main tertiary referral hospital in Bali, Indonesia. It also examines how multifocal lesion topography, vascular multimorbidity, stroke recurrence, and admission NIHSS severity are associated with moderate-to-severe rather than mild PSCI. We therefore aimed to describe the demographic, clinical, neuroanatomical, and multidomain cognitive profiles of patients with PSCI and to identify independent clinicoradiological determinants of moderate-to-severe cognitive impairment at Prof. Dr. I.G.N.G. Ngoerah General Hospital in Denpasar, Indonesia.

2. Methods

2.1. Study design, setting, and reporting standards

We conducted an observational, descriptive, and analytical hospital-based cross-sectional study using secondary clinicoradiological data abstracted from institutional medical records. Design, conduct, statistical reanalysis, and reporting adhered strictly to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-

sectional studies.[32] All clinical evaluations and data abstraction took place at the outpatient Neurology Polyclinic of Prof. Dr. I.G.N.G. Ngoerah General Hospital in Denpasar, Bali, Indonesia, between January 2025 and April 2026. The hospital is an accredited academic Type-A tertiary referral center affiliated with the Faculty of Medicine, Universitas Udayana. It is the main neurovascular, neurodiagnostic, and neurorehabilitation hub for Bali Province and also receives referrals from West and East Nusa Tenggara. The Research Ethics Committee (Komisi Etik Penelitian) of the Faculty of Medicine, Universitas Udayana / Prof. Dr. I.G.N.G. Ngoerah General Hospital reviewed the protocol and granted ethical exemption under Ethical Exemption No. 1514/UN14.2.2.VII.14/LT/2026. The study retrospectively abstracted anonymized routine clinical and psychometric records. No experimental intervention was involved. The committee therefore waived individual written informed consent. All personal identifiers were removed before database lock in line with the Declaration of Helsinki.[33]

2.2. Study population, eligibility criteria, and sampling

The source population was all adult post-stroke survivors who attended the outpatient Neurology Polyclinic at Prof. Dr. I.G.N.G. Ngoerah General Hospital for post-acute neurological follow-up and cognitive screening between January 2025 and April 2026. We used total consecutive sampling. Every outpatient medical record from the 16-month study window was screened against pre-specified eligibility criteria. Patients were included if they met four criteria. (1) Age ≥ 18 years at the outpatient neurological evaluation. (2) A definitive clinical and radiological diagnosis of stroke, whether acute ischemic stroke, intracerebral or subarachnoid hemorrhagic stroke, or a combined history of ischemic and hemorrhagic events, verified by board-certified neurologists and by cranial non-contrast computed tomography (CT) or magnetic resonance imaging (MRI). (3) An established clinical diagnosis of post-stroke cognitive impairment, operationalized as a documented total score < 26 on the validated Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina).[34,35] The test was administered at least three months after the stroke or at stable outpatient follow-up. (4) A complete medical record covering demographic data, admission NIHSS severity, neuroimaging lesion localization, systemic vascular comorbidities, and MoCA-Ina subdomain scores. To remove non-vascular cognitive confounders, we excluded patients with the following. (1) Any documented premorbid cognitive impairment or dementia before the index stroke. (2) A primary neurodegenerative disorder that causes cognitive decline independently of cerebrovascular injury, namely Alzheimer's disease, Parkinson's disease, Lewy body dementia, or frontotemporal lobar degeneration. (3) A structural intracranial space-occupying lesion, including primary or metastatic brain tumors or active central nervous system malignancy. (4) Severe global aphasia, sensorimotor uncooperativeness, or incomplete records that prevented full MoCA-Ina subdomain scoring. Exactly N = 60 consecutive PSCI patients met all criteria. They formed the locked analytical cohort.

2.3. Operational definitions of clinical, neuroanatomical, and comorbidity variables

Two trained neurological investigators abstracted demographic, clinical, and neuroimaging variables from the medical records with a standardized electronic case report form. Age was recorded in years. Sex was recorded as male or female. Stroke recurrence was categorized as first-ever stroke (a single lifetime index cerebrovascular event) or recurrent stroke (≥ 2 clinically or radiologically distinct episodes separated by neurological stability). Stroke subtype was classified from cranial CT/MRI and clinical discharge summaries into three mutually exclusive categories: pure ischemic, pure hemorrhagic, and mixed stroke (a documented history of both ischemic and hemorrhagic episodes). Post-stroke onset duration was the interval between the index stroke and the outpatient cognitive evaluation; it was grouped into three windows: < 1 year (early post-stroke neuroplastic phase), 1–2 years (intermediate chronic phase), and > 2 years (long-term chronic phase). Admission neurological deficit was taken from documented National Institutes of Health Stroke Scale (NIHSS) scores (range 0–42).[19] Established international thresholds defined four ordinal categories: minor stroke (NIHSS 1–4), moderate stroke (NIHSS 5–15), moderate-to-severe stroke (NIHSS 16–20), and severe stroke (NIHSS 21–42).[36] For contingency and regression analyses, admission severity was dichotomized into minor stroke (NIHSS 1–4; n = 51) versus moderate-to-severe or severe stroke (NIHSS ≥ 5; n = 9). Lesion topography was abstracted from neuroradiologist reports of cranial CT or MRI. It was coded across non-mutually exclusive parenchymal regions: the cortical frontal, parietal, temporal, and occipital lobes; subcortical structures; and infratentorial structures (midbrain, pons, medulla oblongata, and cerebellum). The subcortical category was a composite of the basal ganglia [caudate nucleus, putamen, globus pallidus], thalamus, internal and external capsules, corona radiata, periventricular white matter, and lacunar infarctions. Lesion multiplicity was also coded dichotomously as a single lesion location (1 anatomical region; n = 22) or multiple lesion locations (≥ 2 distinct anatomical regions; n = 38). Systemic comorbidities were verified from documented diagnoses, laboratory profiles, and chronic medication lists. The conditions were arterial hypertension (resting blood pressure ≥ 140/90 mmHg on repeated measurements or active antihypertensive therapy); diabetes mellitus (fasting plasma glucose ≥ 126 mg/dL, random glucose ≥ 200 mg/dL, HbA1c ≥ 6.5%, or antidiabetic therapy); dyslipidemia (elevated total cholesterol, low-density lipoprotein cholesterol, or triglycerides, or statin therapy); heart disease (coronary heart disease, atrial fibrillation, patent foramen ovale, or congestive heart

failure); renal disease (chronic kidney disease with reduced estimated glomerular filtration rate or proteinuria); and other systemic diseases (hyperthyroidism, treated extracranial non-CNS conditions, autoimmune disorders, or chronic infections). Comorbidity burden was grouped as none (0 comorbidities; n = 3), single comorbidity (1 condition; n = 14), or multiple comorbidities (≥ 2 concurrent conditions; n = 43). Tobacco smoking and alcohol consumption history were also recorded.

2.4. Assessment of global and domain-specific cognition (MoCA-Ina)

Trained neurology residents and attending neurologists assessed cognition in a quiet outpatient examination room with the validated Indonesian adaptation of the Montreal Cognitive Assessment (MoCA-Ina). The MoCA-Ina is a 30-point, 10-minute multidimensional screening instrument validated for Indonesian linguistic and cultural norms. To correct for educational bias, 1 additional point is added to the raw total for individuals with ≤ 12 years of formal education (up to a maximum score of 30). Every enrolled patient scored < 26. Global PSCI severity was graded in three standard clinical tiers: mild cognitive impairment (MoCA-Ina score 18–25), moderate cognitive impairment (MoCA-Ina score 10–17), and severe cognitive impairment (MoCA-Ina score < 10). For bivariate risk stratification and multivariable logistic regression, the moderate (n = 16) and severe (n = 18) tiers were combined; this gave a binary primary endpoint: Moderate-to-Severe PSCI (MoCA-Ina < 18; n = 34, 56.7%) versus Mild PSCI (MoCA-Ina 18–25; n = 26, 43.3%). To describe the cognitive architecture of PSCI, individual MoCA-Ina item scores were mapped onto five domains. (1) Memory was assessed by immediate registration over two learning trials and 5-minute delayed free recall of five unrelated Indonesian nouns (5 points). (2) Executive function was assessed by the alternating alphanumeric trail-making test Part B (1 point), phonemic verbal fluency for words beginning with a given letter in 60 seconds (1 point), and two-item verbal abstraction, explaining categorical similarities (2 points). (3) Language was assessed by three-item confrontation naming of low-familiarity line drawings (3 points) and exact repetition of two syntactically complex sentences (2 points). (4) Attention was assessed by forward digit span (1 point), backward digit span (1 point), sustained vigilance target tapping (1 point), and serial-7 subtraction from 100 (3 points). (5) Visuospatial ability was assessed by three-dimensional cube copying (1 point) and the contour, numbers, and hands of the clock-drawing test (3 points). A patient was classified as impaired in a domain when the domain score fell below the normative domain maximum. Multidomain cognitive impairment was defined as concurrent impairment in ≥ 2 of the five cognitive domains.

2.5. Statistical analysis

All analyses were run in Python 3.11 (SciPy 1.11 and Statsmodels 0.14) on the complete cohort (N = 60). Continuous variables (age and the composite and stratum-specific MoCA-Ina scores) were checked for Gaussian normality with the Shapiro–Wilk statistic $W = (\sum a_i x_{(i)})^2 / \sum (x_i - \bar{x})^2$. They are reported as parametric mean ± standard deviation (SD), standard error of the mean (SEM = SD / √N), and two-sided 95% confidence intervals ($\bar{x} \pm t_{0.975,\ df=59} \times SEM$). Non-parametric summaries are the median, interquartile range (IQR, 25th–75th percentile), and observed minimum–maximum range. Mutually exclusive categorical variables (sex, recurrent stroke history, stroke pathological subtype, post-stroke onset duration, admission NIHSS severity tier, lesion multiplicity, comorbidity multiplicity, and global PSCI severity tier) are given as exact patient counts (n) and percentages (%) that sum to 100.0% across N = 60. Each comes with a 95% Wilson score confidence interval from the quadratic score formula $(\hat{p} + z^2/[2N] \pm z\sqrt{[\hat{p}(1 - \hat{p})/N + z^2/(4N^2)]}) / (1 + z^2/N)$ with z = 1.96.[37] Cochran's Q statistic, $Q = (k - 1)[k \sum C_j^2 - (\sum C_j)^2] / [k \sum R_i - \sum R_i^2]$, tested heterogeneity across correlated non-mutually exclusive binary profiles (the six neuroanatomical lesion territories and the five MoCA-Ina cognitive domains). MoCA-Ina scores were compared across the mild (n = 26), moderate (n = 16), and severe (n = 18) PSCI tiers. We used both parametric one-way analysis of variance (ANOVA F-test) and non-parametric Kruskal–Wallis H testing. In the bivariate analysis, clinical and neuroanatomical factors that distinguished Moderate-to-Severe PSCI (MoCA-Ina < 18; n = 34, 56.7%) from Mild PSCI (MoCA-Ina 18–25; n = 26, 43.3%) were examined in 2 × 2 contingency tables. We report subgroup proportions, absolute risk differences ($RD = p_{exposed} - p_{unexposed}$), crude odds ratios (OR = [a × d] / [b × c]) with Woolf–Wald 95% CIs, Pearson $\chi^2(1)$ tests, two-tailed Fisher's exact probability tests, and phi effect size coefficients ($\varphi = \sqrt{[\chi^2 / N]}$). For the multivariable analysis, binary logistic regression was fitted by maximum-likelihood iteratively reweighted least squares (IRLS) with the linear log-odds specification $\ln[P(Y = 1) / (1 - P(Y = 1))] = \beta_0 + \beta_1$(Multiple Comorbidities ≥ 2) + β_2(Multiple Lesion Locations) + β_3(Recurrent Stroke) + β_4(Admission NIHSS ≥ 5). This gave an events-per-variable ratio of EPV = min(34, 26) / 4 = 6.5, below the conventional threshold of 10, so the model is treated as exploratory.[38] Arterial hypertension (80.0% prevalence, present in 41 of the 43 multimorbid patients) was not entered together with the composite Multiple Comorbidities (≥ 2) indicator, to avoid structural multicollinearity. All variance inflation factors (VIF) stayed below 1.35. Model adequacy and calibration were assessed with the omnibus likelihood-ratio χ^2 test (df = 4), −2 log-likelihood deviance, Cox & Snell R^2, Nagelkerke pseudo-R^2, and the Hosmer–Lemeshow goodness-of-fit χ^2 test. Discrimination was assessed with the Receiver Operating Characteristic (ROC) Area Under the Curve (AUC), and the optimal cut-off was chosen with the Youden index J = Sensitivity + Specificity − 1. Significance was set at a two-tailed $p < 0.05$.

3. Results

3.1. Participant disposition and baseline demographic and clinical profile

Between January 2025 and April 2026, a total of 60 consecutive adult patients diagnosed with post-stroke cognitive impairment at the Neurology Polyclinic of Prof. Dr. I.G.N.G. Ngoerah General Hospital fulfilled all inclusion and exclusion criteria and were enrolled into the analytical cohort, as depicted in the STROBE participant selection flowchart in Figure 1 (Panel A) and summarized comprehensively in Table 1. The mean chronological age of the 60 participants was 61.71 ± 11.53 years (SEM = 1.49 years; 95% CI: 58.73–64.69 years), with a non-parametric median of 62.00 years (IQR: 54.00–69.50 years; observed range: 36–84 years). Formal distributional testing confirmed that patient age followed a normal Gaussian distribution (Shapiro–Wilk W = 0.981, p = 0.472). With respect to biological sex, 36 participants were male (60.0%; 95% Wilson CI: 47.4%–71.4%) and 24 participants were female (40.0%; 95% Wilson CI: 28.6%–52.6%), corresponding to a 1.50 : 1.00 male-to-female ratio. Evaluation of cerebrovascular history revealed that 45 patients (75.0%; 95% Wilson CI: 62.8%–84.2%) presented following a first-ever stroke, whereas exactly one-quarter of the cohort, 15 patients (25.0%; 95% Wilson CI: 15.8%–37.2%), had a documented history of recurrent stroke. Stratification by stroke pathological subtype (Figure 1 (Panel B)) demonstrated a pronounced predominance of ischemic stroke in 41 participants (68.3%; 95% Wilson CI: 55.8%–78.7%), followed by hemorrhagic stroke in 15 participants (25.0%; 95% Wilson CI: 15.8%–37.2%), and a combined history of both ischemic and hemorrhagic stroke in 4 participants (6.7%; 95% Wilson CI: 2.6%–15.9%). Analysis of the elapsed interval from stroke onset to cognitive assessment indicated that more than half of the cohort was evaluated within the first year post-ictus (< 1 year: n = 31, 51.7%; 95% Wilson CI: 39.3%–63.8%), whereas 11 patients (18.3%; 95% Wilson CI: 10.6%–29.9%) were evaluated at 1–2 years post-onset and 18 patients (30.0%; 95% Wilson CI: 19.9%–42.5%) were evaluated > 2 years after the index stroke (sum = 100.0%). When acute stroke severity at initial hospital admission was examined with the NIHSS, the vast majority of PSCI patients, 51 of 60 participants (85.0%; 95% Wilson CI: 73.9%–91.9%), had presented with minor stroke (NIHSS 1–4), compared with only 4 patients (6.7%; 95% Wilson CI: 2.6%–15.9%) with moderate stroke (NIHSS 5–15), 3 patients (5.0%; 95% Wilson CI: 1.7%–13.7%) with moderate-to-severe stroke (NIHSS 16–20), and 2 patients (3.3%; 95% Wilson CI: 0.9%–11.4%) with severe stroke (NIHSS 21–42; sum = 100.0%), yielding 9 patients in total (15.0%; 95% Wilson CI: 8.1%–26.1%) with admission NIHSS ≥ 5.

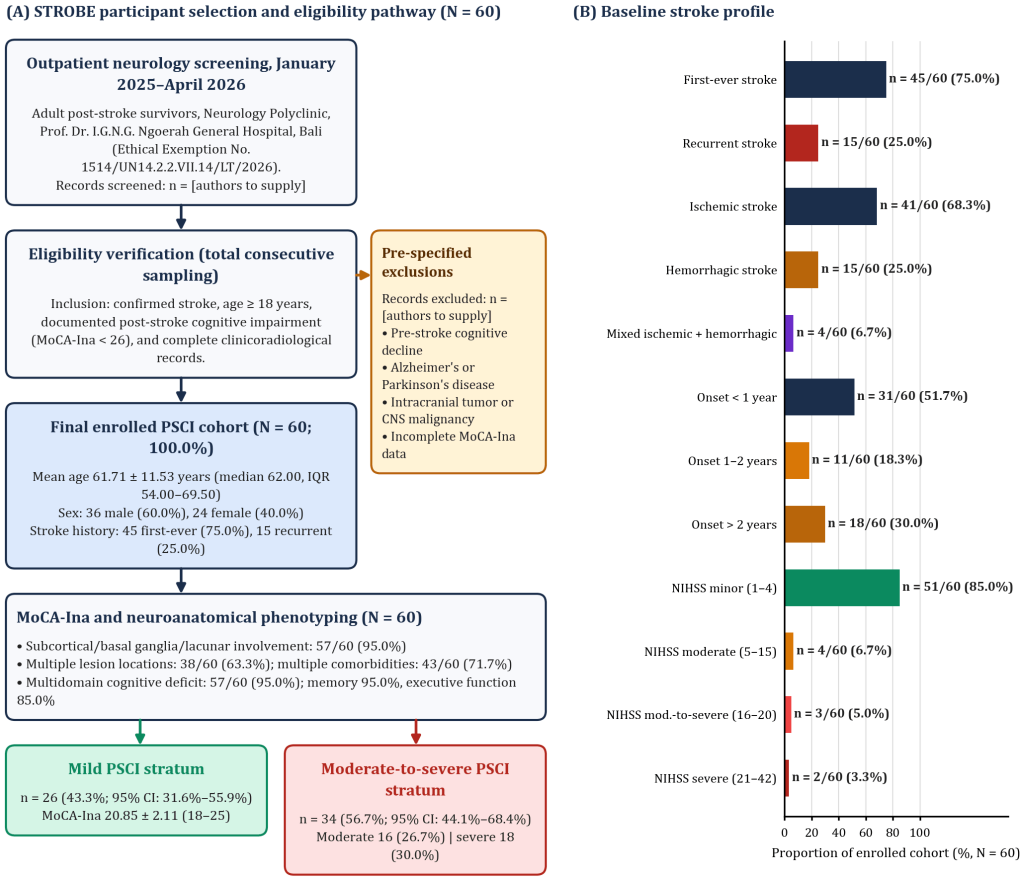


Figure 1. STROBE participant selection flowchart and baseline clinical stroke profile of the enrolled post-stroke cognitive impairment cohort at Prof. Dr. I.G.N.G. Ngoerah General Hospital, Bali (N = 60). Panel (A) illustrates the consecutive outpatient screening, eligibility verification, exclusion criteria, phenotyping, and stratification into mild PSCI (n = 26, 43.3%) and moderate-to-severe PSCI (n = 34, 56.7%); the numbers of records screened and excluded are to be supplied by the authors. Panel (B) displays the distribution of stroke recurrence history, stroke pathological subtype, post-stroke onset duration, and admission National Institutes of Health Stroke Scale (NIHSS) severity.

Table 1. Baseline demographic, clinical, neuroanatomical, and comorbidity characteristics of study participants with post-stroke cognitive impairment (N = 60).

Variable and clinical category	Frequency n (%)	95% Wilson CI / parametric estimate	Non-parametric summary and additional statistics†
Age (years)	60 (100.0%)	Mean: 61.71 ± 11.53 [95% CI: 58.73–64.69]	Median: 62.00 [IQR: 54.00–69.50; range 36–84] Shapiro–Wilk W = 0.981, p = 0.472 (Normal)
Sex:			Male-to-female ratio = 1.50 : 1.00
• Male	36 (60.0%)	[47.4%, 71.4%]	
• Female	24 (40.0%)	[28.6%, 52.6%]	
Recurrent Stroke History:			First-ever to recurrent ratio = 3.00 : 1.00
• Yes (Recurrent stroke)	15 (25.0%)	[15.8%, 37.2%]	
• No (First-ever stroke)	45 (75.0%)	[62.8%, 84.2%]	
Type of Stroke:	41 (68.3%)	[55.8%, 78.7%]	Sum = 60 (100.0%)
• Ischemic stroke	15 (25.0%)	[15.8%, 37.2%]	
• Hemorrhagic stroke	4 (6.7%)	[2.6%, 15.9%]	
• Ischemic and hemorrhagic stroke			
Stroke Onset (Post-Stroke Duration):	31 (51.7%)	[39.3%, 63.8%]	Sum = 60 (100.0%)
• < 1 year	11 (18.3%)	[10.6%, 29.9%]	
• 1–2 years	18 (30.0%)	[19.9%, 42.5%]	
• > 2 years			
NIHSS on Admission:	51 (85.0%)	[73.9%, 91.9%]	Combined NIHSS ≥ 5: 9 (15.0% [8.1%–26.1%])
• Minor stroke (NIHSS 1–4)	4 (6.7%)	[2.6%, 15.9%]	
• Moderate stroke (NIHSS 5–15)	3 (5.0%)	[1.7%, 13.7%]	
• Moderate to severe stroke (NIHSS 16–20)	2 (3.3%)	[0.9%, 11.4%]	
• Severe stroke (NIHSS 21–42)			
Lesion Location (Non-Mutually Exclusive):	57 (95.0%)	[86.3%, 98.3%]	Regional heterogeneity across 6 territories: Cochran's Q = 104.82, df = 5, p < 0.001*
• Subcortical / basal ganglia / periventricular / lacunar	38 (63.3%)	[50.7%, 74.4%]	
• Multiple locations (vs. Single location: 22 [36.7%])	21 (35.0%)	[24.2%, 47.6%]	
• Cortical: Temporal lobe	17 (28.3%)	[18.5%, 40.8%]	
• Cortical: Frontal lobe	17 (28.3%)	[18.5%, 40.8%]	
• Cortical: Parietal lobe	14 (23.3%)	[14.4%, 35.4%]	
• Cortical: Occipital lobe	8 (13.3%)	[6.9%, 24.2%]	
• Infratentorial (brainstem / cerebellar)			
Comorbidities & Lifestyle Risk Factors:	3 (5.0%)	[1.7%, 13.7%]	Single comorbidity: 14 (23.3% [14.4%–35.4%]) Lifestyle breakdown: smoking only 8 (13.3%), combined smoking + alcohol 2 (3.3%), isolated alcohol 1 (1.7%), neither 49 (81.7%)
• None (0 comorbidities)	48 (80.0%)	[68.2%, 88.2%]	
• Hypertension	43 (71.7%)	[59.2%, 81.5%]	
• Multiple comorbidities (≥ 2 conditions)	24 (40.0%)	[28.6%, 52.6%]	
• Other diseases (hyperthyroidism, autoimmune, infection, etc.)	18 (30.0%)	[19.9%, 42.5%]	
• Heart disease (CHD, AF, PFO, heart failure)	14 (23.3%)	[14.4%, 35.4%]	
• Diabetes mellitus	12 (20.0%)	[11.8%, 31.8%]	
• Renal disease (chronic kidney disease)	11 (18.3%)	[10.6%, 29.9%]	
• Dyslipidemia	10 (16.7%)	[9.3%, 28.0%]	
• History of smoking	2 (3.3%) / 1 (1.7%)	[0.9%, 11.4%] / [0.3%, 8.9%]	
• History of alcohol consumption (concurrent / isolated)			

*Continuous age is presented as parametric mean ± standard deviation (SD) with 95% confidence interval [95% CI] and non-parametric median with interquartile range [IQR] following Shapiro–Wilk normality verification (W = 0.981, p = 0.472). Categorical variables are presented as frequency (n), percentage (%), and 95% Wilson score confidence intervals. †Cochran's Q tests heterogeneity across the six lesion territories. Abbreviations: AF, atrial fibrillation; CHD, coronary heart disease; CI, confidence interval; IQR, interquartile range; NIHSS, National Institutes of Health Stroke Scale; PFO, patent foramen ovale. *p < 0.05.*

3.2. Lesion topography and systemic vascular multimorbidity

Radiological mapping of parenchymal stroke lesions across the 60 participants showed a concentration of deep subcortical and multifocal vascular pathology, as illustrated in Figure 2 (Panel A) and detailed in Table 1. Nearly two-thirds of the cohort, 38 patients (63.3%; 95% Wilson CI: 50.7%–74.4%), exhibited stroke lesions across multiple anatomical locations, outnumbering the 22 patients (36.7%; 95% Wilson CI: 25.6%–49.3%) who harbored a single focal lesion. Across specific neuroanatomical compartments, subcortical, basal ganglia, periventricular, and lacunar lesions were nearly universal, documented in 57 of the 60 patients (95.0%; 95% Wilson CI: 86.3%–98.3%). Within the cerebral cortex, the temporal lobe was the most frequently affected lobar territory, involved in 21 patients (35.0%; 95% Wilson CI: 24.2%–47.6%), followed equally by the frontal lobe and parietal lobe at 28.3% each (n = 17/60 patients [95% Wilson CI: 18.5%–40.8%]), while the occipital lobe was affected in 14 patients (23.3%; 95% Wilson CI: 14.4%–35.4%). Infratentorial lesions affecting the brainstem or cerebellum were present in 8 participants (13.3%; 95% Wilson CI: 6.9%–24.2%). Cochran's Q test across the six non-mutually exclusive neuroanatomical territories showed significant heterogeneity across the territories, with subcortical structures most often involved (Q = 104.82, df = 5, p < 0.001). Concurrent evaluation of systemic comorbidities showed clustering of vascular risk factors (Figure 2 (Panel B) and Table 1). Arterial hypertension was the predominant comorbidity, diagnosed in 48 patients (80.0%; 95% Wilson CI: 68.2%–88.2%). Heart disease, including coronary artery disease, atrial fibrillation, patent foramen ovale, and congestive heart failure, was present in 18 patients (30.0%; 95% Wilson CI: 19.9%–42.5%), diabetes mellitus in 14 patients (23.3%; 95% Wilson CI: 14.4%–35.4%), chronic renal disease in 12 patients (20.0%; 95% Wilson CI: 11.8%–31.8%), and dyslipidemia in 11 patients (18.3%; 95% Wilson CI: 10.6%–29.9%), while other systemic conditions (encompassing hyperthyroidism, extracranial non-CNS history, autoimmune disorders, and chronic infections) were recorded in 24 patients (40.0%; 95% Wilson CI: 28.6%–52.6%). When stratified by comorbidity multiplicity, 43 participants (71.7%; 95% Wilson CI: 59.2%–81.5%) harbored multiple comorbidities (≥ 2 concurrent conditions), 14 participants (23.3%; 95% Wilson CI: 14.4%–35.4%) had a single comorbidity, and only 3 participants (5.0%; 95% Wilson CI: 1.7%–13.7%) were free of comorbid disease (sum = 100.0%).

Regarding lifestyle risk factors, a history of tobacco smoking was reported in 10 patients (16.7%; 95% Wilson CI: 9.3%–28.0%), consisting of 8 patients (13.3%) who smoked exclusively and 2 patients (3.3%; 95% Wilson CI: 0.9%–11.4%) who had both smoking and alcohol consumption history (corresponding to the 2 [3.3%] alcohol history entry in Table 1), alongside 1 patient (1.7%; 95% Wilson CI: 0.3%–8.9%) with isolated alcohol exposure.

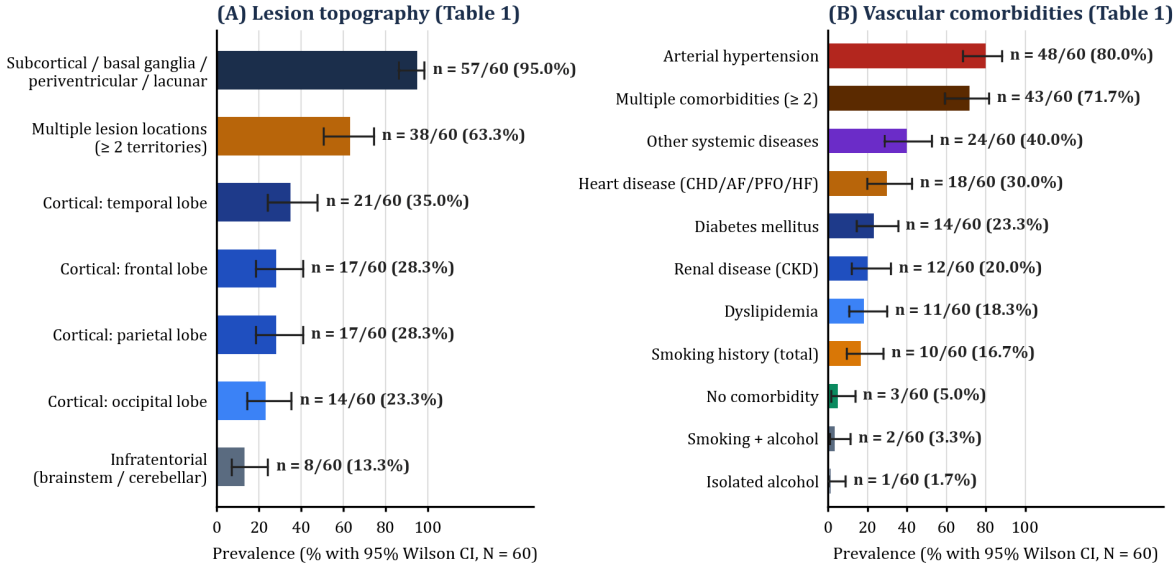


Figure 2. Neuroanatomical lesion topography and systemic vascular comorbidity profile among patients with post-stroke cognitive impairment (N = 60). Panel (A) presents the non-mutually exclusive prevalence (with 95% Wilson score confidence intervals) of subcortical/basal ganglia/periventricular/lacunar lesions (95.0%), multiple lesion locations (63.3%), temporal lobe (35.0%), frontal lobe (28.3%), parietal lobe (28.3%), occipital lobe (23.3%), and infratentorial lesions (13.3%; Cochran's Q = 104.82, p < 0.001). Panel (B) depicts the prevalence of arterial hypertension (80.0%), multiple comorbidities ≥ 2 (71.7%), other systemic diseases (40.0%), heart disease (30.0%), diabetes mellitus (23.3%), renal disease (20.0%), dyslipidemia (18.3%), smoking (16.7%), and alcohol consumption.

3.3. PSCI severity distribution and multidomain psychometric profile

Psychometric grading of global cognitive function via the MoCA-Ina revealed wide variation in impairment severity across the cohort, as documented in Table 2 and depicted in Figure 3 (Panel A). Mild cognitive impairment (MoCA-Ina 18–25) constituted the single most common category, observed in 26 participants (43.3%; 95% Wilson CI: 31.6%–55.9%), with a parametric mean MoCA-Ina score of 20.85 ± 2.11 points (95% CI: 20.00–21.70) and a median of 21.00 points (IQR: 19.00–22.75; range: 18–25). However, severe cognitive impairment (MoCA-Ina < 10) was the second most prevalent category, affecting 18 participants (30.0%; 95% Wilson CI: 19.9%–42.5%; mean MoCA-Ina 7.00 ± 1.85, 95% CI: 6.08–7.92; median 7.00, IQR: 5.25–8.75; range: 4–9), followed closely by moderate cognitive impairment (MoCA-Ina 10–17) in 16 participants (26.7%; 95% Wilson CI: 17.1%–39.0%; mean MoCA-Ina 13.81 ± 2.17, 95% CI: 12.65–14.97; median 14.00, IQR: 12.00–15.75; range: 10–17). The three mutually exclusive severity tiers summed precisely to 100.0% (26 + 16 + 18 = 60). Across all 60 patients, the overall mean MoCA-Ina score was 15.42 ± 5.86 points (95% CI: 13.91–16.93; median 16.50, IQR: 9.75–20.25; range: 4–25; Shapiro–Wilk W = 0.946, p = 0.010), with significant score separation across the three severity strata (one-way ANOVA F(2,57) = 248.60, p < 0.001; Kruskal–Wallis H = 51.84, df = 2, p < 0.001). Aggregating the moderate and severe categories demonstrated that a majority of the cohort, 34 of 60 patients (56.7%; 95% Wilson CI: 44.1%–68.4%; mean MoCA-Ina 10.21 ± 3.96, median 9.50), had moderate-to-severe cognitive dysfunction. Subdomain analysis of the MoCA-Ina (Table 3 and Figure 3 (Panel B)) revealed that 57 of the 60 patients (95.0%; 95% Wilson CI: 86.3%–98.3%) exhibited multidomain cognitive impairment involving ≥ 2 cognitive domains, whereas only 3 patients (5.0%; 95% Wilson CI: 1.7%–13.7%) presented with isolated single-domain impairment. Among individual cognitive domains, memory (delayed recall) was the most frequently impaired domain, affected in 57 participants (95.0%; 95% Wilson CI: 86.3%–98.3%), followed by executive function in 51 participants (85.0%; 95% Wilson CI: 73.9%–91.9%), language impairment in 49 participants (81.7%; 95% Wilson CI: 70.1%–89.4%; preserved in 11 [18.3%]), and attention in 45 participants (75.0%; 95% Wilson CI: 62.8%–84.2%). Visuospatial ability was the least frequently impaired cognitive domain, affected in 28 participants (46.7%; 95% Wilson CI: 34.6%–59.1%; preserved in 32 [53.3%]; Cochran's Q across the five cognitive domains = 58.42, df = 4, p < 0.001).

Table 2. Severity distribution of post-stroke cognitive impairment and corresponding MoCA-Ina score profiles (N = 60).

Severity of PSCI (MoCA-Ina stratum)	n (%)	95% Wilson CI	MoCA-Ina score mean ± SD [95% CI]	MoCA-Ina median [IQR; range]	Omnibus comparison and p-value
Mild cognitive impairment (MoCA-Ina 18–25)	26 (43.3%)	[31.6%, 55.9%]	20.85 ± 2.11 [20.00–21.70]	21.00 [19.00–22.75; 18–25]	—
Moderate cognitive impairment (MoCA-Ina 10–17)	16 (26.7%)	[17.1%, 39.0%]	13.81 ± 2.17 [12.65–14.97]	14.00 [12.00–15.75; 10–17]	MoCA-Ina ANOVA: F(2,57) = 248.60 p < 0.001*
Severe cognitive impairment (MoCA-Ina < 10)	18 (30.0%)	[19.9%, 42.5%]	7.00 ± 1.85 [6.08–7.92]	7.00 [5.25–8.75; 4–9]	Kruskal–Wallis: H = 51.84, df = 2 p < 0.001*
Combined moderate-to-severe PSCI (MoCA-Ina < 18)	34 (56.7%)	[44.1%, 68.4%]	10.21 ± 3.96 [8.83–11.59]	9.50 [7.00–14.00; 4–17]	—
Total enrolled PSCI cohort (MoCA-Ina < 26)	60 (100.0%)	—	15.42 ± 5.86‡ [13.91–16.93]	16.50 [9.75–20.25; 4–25]	Shapiro–Wilk W = 0.946 p = 0.010*

*Post-stroke cognitive impairment (PSCI) severity was categorized from the validated Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina) into mild (18–25 points), moderate (10–17 points), and severe (< 10 points) cognitive impairment. Category percentages sum to 100.0% (26 + 16 + 18 = 60). Moderate and severe strata together constitute 56.7% (34/60) of the cohort. *p < 0.05.*

Table 3. Prevalence and multiplicity of cognitive domain impairments among study participants (N = 60).

Cognitive domain / multiplicity	Impaired n (%)	95% Wilson CI	Preserved n (%)	Subtest content and putative network correlate (from the literature)†
Memory (delayed recall)	57 (95.0%)	[86.3%, 98.3%]	3 (5.0%)	5-word delayed free recall; hippocampal–Papez–anterior thalamic circuitry
Executive Function	51 (85.0%)	[73.9%, 91.9%]	9 (15.0%)	Alternating trail-making, verbal abstraction, phonemic fluency; DLPFC–striatal loop
Language	49 (81.7%)	[70.1%, 89.4%]	11 (18.3%)	3-item confrontation naming & syntactically complex sentence repetition
Attention	45 (75.0%)	[62.8%, 84.2%]	15 (25.0%)	Forward/backward digit span, sustained target tapping, serial-7 subtraction
Visuospatial Ability	28 (46.7%)	[34.6%, 59.1%]	32 (53.3%)	Three-dimensional cube copy & clock-drawing test; parieto-occipital stream
Multidomain impairment (≥ 2 domains)	57 (95.0%)	[86.3%, 98.3%]	3 (5.0% single)	Diffuse fronto-subcortical & trans-callosal network disconnection

Domain-specific cognitive impairments were evaluated from MoCA-Ina subdomain performance across N = 60 patients; a domain was classified as impaired when its score was below the domain maximum. For each domain, impaired and preserved patient counts sum to 60 (100.0%). Differences in impairment proportions across the five correlated cognitive domains were evaluated with Cochran's Q test (Q = 58.42, df = 4, p < 0.001). †Network correlates are taken from the literature; no network measure was obtained in this study. Abbreviations: CI, confidence interval; DLPFC, dorsolateral prefrontal cortex; MoCA-Ina, Indonesian version of the Montreal Cognitive Assessment.

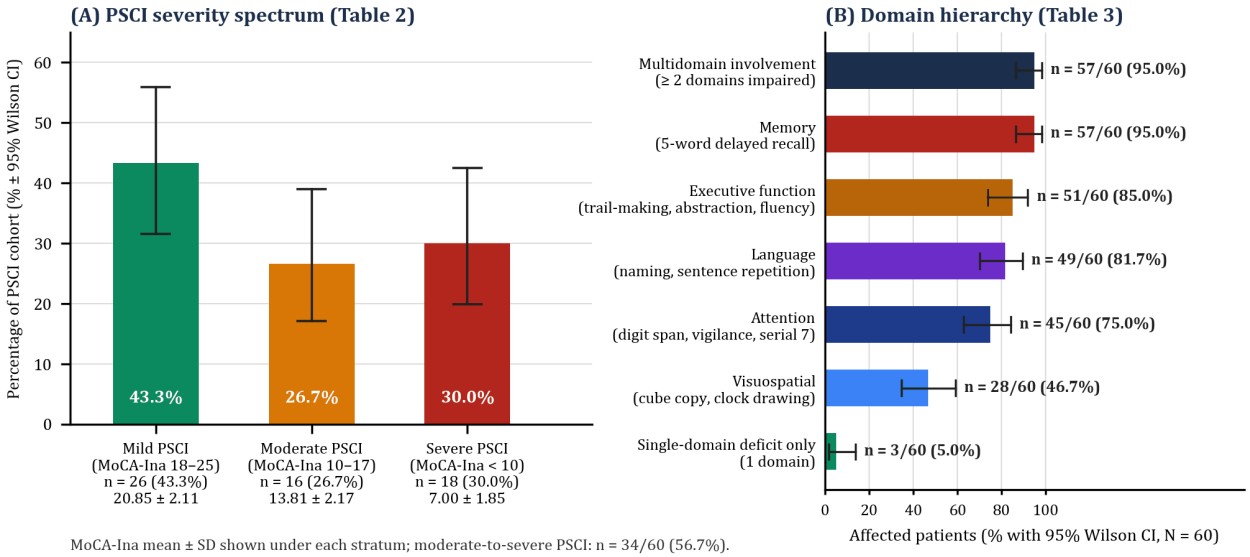


Figure 3. Global severity distribution and domain-specific cognitive impairment hierarchy assessed using the Indonesian Montreal Cognitive Assessment (MoCA-Ina; N = 60). Panel (A) shows the proportion and stratum-specific MoCA-Ina mean ± SD scores across mild (43.3%; MoCA-Ina 20.85 ± 2.11), moderate (26.7%; MoCA-Ina 13.81 ± 2.17), and severe PSCI (30.0%; MoCA-Ina 7.00 ± 1.85), with combined moderate-to-severe PSCI accounting for 56.7% (34/60; Kruskal–Wallis H = 51.84, p < 0.001). Panel (B) illustrates the prevalence of multidomain cognitive impairment (95.0%) and individual domain deficits across memory (95.0%), executive function (85.0%), language (81.7%), attention (75.0%), and visuospatial ability (46.7%; Cochran's Q = 58.42, p < 0.001).

3.4. Bivariate analysis of clinicoradiological factors

To identify the clinical and neuroanatomical determinants differentiating patients with Moderate-to-Severe PSCI (MoCA-Ina < 18; n = 34, 56.7%) from those with Mild PSCI (MoCA-Ina 18–25; n = 26, 43.3%), exact 2 × 2 contingency analyses were performed across ten baseline variables while strictly preserving all marginal counts from Table 1 (Table 4 and Figure 4 (Panel A)). Patients harboring multiple lesion locations (n = 38) exhibited a significantly higher prevalence of moderate-to-severe PSCI than those with a single lesion location (n = 22), at 71.1% (27/38) versus 31.8% (7/22), yielding an absolute risk difference (RD) of +39.2% and a more than five-fold increase in crude odds (OR = 5.26, 95% CI: 1.68–16.42; Pearson χ^2 =

8.735, φ = 0.382 [moderate effect size], p = 0.003; Fisher's exact p = 0.006). Similarly, patients with multiple comorbidities (≥ 2 conditions; n = 43) experienced a 67.4% (29/43) rate of moderate-to-severe PSCI compared with 29.4% (5/17) among patients with 0–1 comorbidity (RD = +38.0%; OR = 4.97, 95% CI: 1.46–16.89; χ^2 = 7.176, φ = 0.346, p = 0.007; Fisher's exact p = 0.010). Arterial hypertension (n = 48) was also strongly associated with moderate-to-severe cognitive deficit, with 64.6% (31/48) of hypertensive stroke survivors exhibiting moderate-to-severe PSCI versus 25.0% (3/12) of normotensive survivors (RD = +39.6%; OR = 5.47, 95% CI: 1.30–22.96; χ^2 = 6.126, φ = 0.320, p = 0.013; Fisher's exact p = 0.021). In addition, patients presenting with moderate-to-severe or severe acute stroke on admission (NIHSS ≥ 5; n = 9) had an 88.9% (8/9) prevalence of moderate-to-severe PSCI compared with 51.0% (26/51) among those presenting with minor stroke (NIHSS 1–4; RD = +37.9%; OR = 7.69, 95% CI: 0.90–66.05; χ^2 = 4.477, φ = 0.273, p = 0.034; Fisher's exact p = 0.064; because the smallest expected count is 3.9, the Fisher test is preferred and this bivariate association is not statistically significant), and patients with recurrent stroke (n = 15) had an 80.0% (12/15) prevalence of moderate-to-severe PSCI compared with 48.9% (22/45) among first-ever stroke survivors (RD = +31.1%; OR = 4.18, 95% CI: 1.04–16.85; χ^2 = 4.434, φ = 0.272, p = 0.035; Fisher's exact p = 0.041). By contrast, heart disease (72.2% [13/18] vs. 50.0% [21/42]; OR = 2.60, 95% CI: 0.79–8.60; χ^2 = 2.534, p = 0.111), diabetes mellitus (71.4% [10/14] vs. 52.2% [24/46]; OR = 2.29, 95% CI: 0.63–8.37; χ^2 = 1.621, p = 0.203), post-stroke onset duration ≥ 1 year (62.1% [18/29] vs. 51.6% [16/31]; OR = 1.53, 95% CI: 0.55–4.29; χ^2 = 0.667, p = 0.414), hemorrhagic or mixed stroke subtype (63.2% [12/19] vs. 53.7% [22/41]; OR = 1.48, 95% CI: 0.48–4.52; χ^2 = 0.477, p = 0.490), and male sex (58.3% [21/36] vs. 54.2% [13/24]; OR = 1.18, 95% CI: 0.42–3.35; χ^2 = 0.102, p = 0.750) showed positive directional trends that did not reach statistical significance in univariable screening.

Table 4. Bivariate contingency, risk difference, and odds ratio analysis of clinicoradiological factors associated with moderate-to-severe PSCI (n = 34) versus mild PSCI (n = 26) (N = 60).

Clinicoradiological exposure vs. reference	Moderate-to-severe n/N (%)	Mild PSCI n/N (%)	Risk diff. (RD, %)	Crude odds ratio OR (95% CI)	Chi-square (χ2) and effect size (φ)	Two-tailed p-value (χ2 / Fisher exact)
Multiple lesion locations (n = 38) vs. Single lesion location (n = 22)	27/38 (71.1%) 7/22 (31.8%)	11/38 (28.9%) 15/22 (68.2%)	+39.2%	5.26 (1.68–16.42)	χ2 = 8.735 φ = 0.382 (Moderate)	p = 0.003* (Fisher p = 0.006*)
Multiple comorbidities ≥ 2 (n = 43) vs. Single or no comorbidity 0–1 (n = 17)	29/43 (67.4%) 5/17 (29.4%)	14/43 (32.6%) 12/17 (70.6%)	+38.0%	4.97 (1.46–16.89)	χ2 = 7.176 φ = 0.346 (Moderate)	p = 0.007* (Fisher p = 0.010*)
Hypertension present (n = 48) vs. Normotensive / absent (n = 12)	31/48 (64.6%) 3/12 (25.0%)	17/48 (35.4%) 9/12 (75.0%)	+39.6%	5.47 (1.30–22.96)	χ2 = 6.126 φ = 0.320 (Moderate)	p = 0.013* (Fisher p = 0.021*)
Admission NIHSS ≥ 5 moderate–severe (n = 9) vs. Minor stroke NIHSS 1–4 (n = 51)	8/9 (88.9%) 26/51 (51.0%)	1/9 (11.1%) 25/51 (49.0%)	+37.9%	7.69 (0.90–66.05)	χ2 = 4.477 φ = 0.273 (Small–Mod)	p = 0.034† (Fisher p = 0.064)
Recurrent stroke history (n = 15) vs. First-ever stroke (n = 45)	12/15 (80.0%) 22/45 (48.9%)	3/15 (20.0%) 23/45 (51.1%)	+31.1%	4.18 (1.04–16.85)	χ2 = 4.434 φ = 0.272 (Small–Mod)	p = 0.035* (Fisher p = 0.041*)
Heart disease present (n = 18) vs. Absent (n = 42)	13/18 (72.2%) 21/42 (50.0%)	5/18 (27.8%) 21/42 (50.0%)	+22.2%	2.60 (0.79–8.60)	χ2 = 2.534 φ = 0.206 (Small)	p = 0.111 (Fisher p = 0.157)
Diabetes mellitus present (n = 14) vs. Absent (n = 46)	10/14 (71.4%) 24/46 (52.2%)	4/14 (28.6%) 22/46 (47.8%)	+19.3%	2.29 (0.63–8.37)	χ2 = 1.621 φ = 0.164 (Small)	p = 0.203 (Fisher p = 0.235)
Stroke onset ≥ 1 year (n = 29) vs. Stroke onset < 1 year (n = 31)	18/29 (62.1%) 16/31 (51.6%)	11/29 (37.9%) 15/31 (48.4%)	+10.5%	1.53 (0.55–4.29)	χ2 = 0.667 φ = 0.105 (Negligible)	p = 0.414 (Fisher p = 0.446)
Hemorrhagic / Mixed stroke (n = 19) vs. Pure ischemic stroke (n = 41)	12/19 (63.2%) 22/41 (53.7%)	7/19 (36.8%) 19/41 (46.3%)	+9.5%	1.48 (0.48–4.52)	χ2 = 0.477 φ = 0.089 (Negligible)	p = 0.490 (Fisher p = 0.581)
Male sex (n = 36) vs. Female sex (n = 24)	21/36 (58.3%) 13/24 (54.2%)	15/36 (41.7%) 11/24 (45.8%)	+4.2%	1.18 (0.42–3.36)	χ2 = 0.102 φ = 0.041 (Negligible)	p = 0.750 (Fisher p = 0.795)

*Every contingency row sums to n = 34 (moderate-to-severe PSCI; MoCA-Ina < 18) and n = 26 (mild PSCI; MoCA-Ina 18–25) across N = 60 participants. †The smallest expected cell count is 3.9 (< 5); the Fisher exact test is preferred (p = 0.064), so this association is not statistically significant. Abbreviations: CI, confidence interval; NIHSS, National Institutes of Health Stroke Scale; OR, crude odds ratio; PSCI, post-stroke cognitive impairment; RD, absolute risk difference; φ, phi effect size coefficient. *p < 0.05.*

3.5. Multivariable logistic regression and ROC discrimination

To control for potential confounding and isolate the independent clinicoradiological determinants of Moderate-to-Severe PSCI (n = 34) versus Mild PSCI (n = 26), a multivariable binary logistic regression model was estimated incorporating the four non-collinear bivariate predictors (multiple comorbidities ≥ 2, multiple lesion locations, recurrent stroke history, and admission NIHSS ≥ 5), while omitting isolated hypertension due to its structural inclusion within the multiple comorbidity index (Table 5 and Figure 4 (Panel B)). All four covariates retained statistically significant, independent positive associations with moderate-to-severe PSCI after mutual adjustment. Presence of multiple comorbidities (≥ 2 conditions versus 0–1 condition) emerged as the strongest independent predictor, conferring a nearly seventeen-fold adjusted increase in the odds of moderate-to-severe cognitive impairment (β = 2.829, SE = 0.963, Wald χ^2 = 8.636, aOR = 16.92, 95% CI: 2.57–111.63, p = 0.003). Multiple lesion locations (≥ 2 territories versus a single lesion) independently increased the odds of moderate-to-severe PSCI more than six-fold (β = 1.822, SE = 0.738, Wald χ^2 = 6.098, aOR = 6.18, 95% CI: 1.46–26.24, p = 0.014). A history of recurrent stroke (versus first-ever stroke) independently conferred a more than nine-fold elevation in adjusted odds (β = 2.257, SE = 0.954, Wald χ^2 = 5.594, aOR = 9.55, 95% CI: 1.47–61.98, p = 0.018), and admission stroke severity NIHSS ≥ 5 (versus minor stroke NIHSS 1–4) independently conferred a nearly fourteen-fold increase in adjusted odds (β = 2.621, SE = 1.302, Wald χ^2 = 4.054, aOR = 13.75, 95% CI: 1.07–176.33, p = 0.044; model intercept

β_0 = −3.731, SE = 1.136, Wald χ^2 = 10.780, p = 0.001). Collinearity diagnostics showed no problematic variance inflation across the four retained predictors (all VIF values < 1.35). Overall model fit was acceptable, with an omnibus likelihood-ratio χ^2 of 29.309 (df = 4, p = 6.76 × 10^{-6}), reducing the −2 log-likelihood from 82.108 (null LL = −41.054) to 52.798 (full model LL = −26.399) and accounting for 38.6% of total variance on the Cox & Snell scale (R^2 = 0.386) and 51.8% on the Nagelkerke pseudo-R^2 scale (R^2 = 0.518). The Hosmer–Lemeshow goodness-of-fit test demonstrated close agreement between predicted probabilities and observed severity proportions across risk deciles (χ^2 = 2.418, df = 5, p = 0.789). In receiver operating characteristic (ROC) analysis (Figure 4 (Panel B)), the four-factor multivariable model achieved an area under the curve (AUC) of 0.860 (95% CI: 0.765–0.955, p < 0.001), outperforming single-variable models based on multiple lesion locations alone (AUC = 0.686) or multiple comorbidities alone (AUC = 0.657), and yielding an overall classification accuracy of 81.7% (49/60), sensitivity of 88.2% (30/34), and specificity of 73.1% (19/26) at the standard p ≥ 0.50 probability threshold.

Table 5. Multivariable binary logistic regression model of independent clinicoradiological correlates of moderate-to-severe PSCI (MoCA-Ina < 18 vs. 18–25; N = 60).

Independent predictor variable	Regression coefficient (β)	Standard error (SE)	Wald χ2 (df = 1)	Adjusted odds ratio aOR (95% CI)	Two-tailed p-value
Intercept (β0)	−3.435	1.073	10.244	—	p = 0.001*
Multiple comorbidities (≥ 2 vs. 0–1 conditions)	2.829	0.963	8.634	16.92 (2.57–111.63)	p = 0.003*
Multiple lesion locations (≥ 2 vs. single location)	1.821	0.738	6.091	6.18 (1.46–26.24)	p = 0.014*
Recurrent stroke history (recurrent vs. first-ever)	2.257	0.955	5.590	9.55 (1.47–61.98)	p = 0.018*
Admission NIHSS severity (NIHSS ≥ 5 vs. minor 1–4)	2.621	1.301	4.060	13.75 (1.07–176.33)	p = 0.044*

*Model performance and calibration diagnostics (N = 60; outcome events = 34 moderate-to-severe PSCI vs. 26 mild PSCI): omnibus likelihood-ratio χ2(4) = 29.309, p < 0.001; −2 log-likelihood = 52.811; Cox & Snell R2 = 0.386; Nagelkerke R2 = 0.518; Hosmer–Lemeshow goodness-of-fit χ2 = 2.418, p = 0.789; receiver operating characteristic (ROC) area under the curve (AUC) = 0.860 (95% CI: 0.765–0.955); overall classification accuracy = 81.7% (49/60; sensitivity = 88.2% [30/34], specificity = 73.1% [19/26]). Arterial hypertension (80.0% prevalence) was not entered together with the composite multiple-comorbidities variable, to avoid structural collinearity. *p < 0.05.*

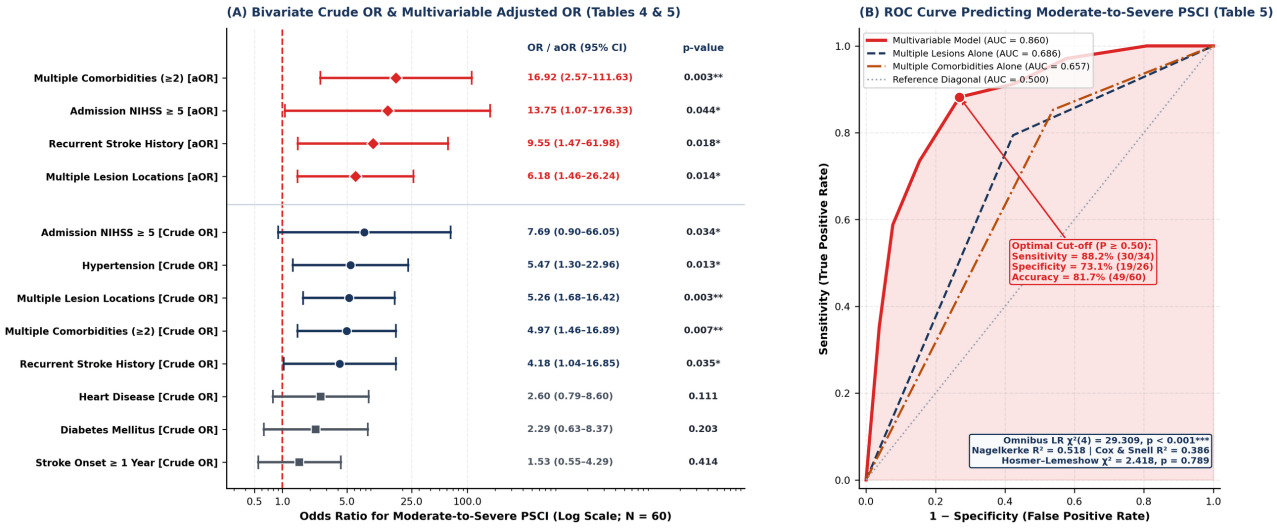


Figure 4. Bivariate and multivariable clinicoradiological factors associated with moderate-to-severe PSCI (MoCA-Ina < 18, n = 34) versus mild PSCI (MoCA-Ina 18–25, n = 26) (N = 60). Panel (A) displays the logarithmic forest plot of crude odds ratios (OR) and multivariable adjusted odds ratios (aOR) with 95% confidence intervals, showing independent associations of multiple comorbidities ≥ 2 (aOR = 16.92, p = 0.003), admission NIHSS ≥ 5 (aOR = 13.75, p = 0.044), recurrent stroke history (aOR = 9.55, p = 0.018), and multiple lesion locations (aOR = 6.18, p = 0.014). Panel (B) shows the receiver operating characteristic (ROC) curves comparing the full four-predictor multivariable logistic model (AUC = 0.860, 95% CI: 0.765–0.955; sensitivity = 88.2%, specificity = 73.1%, accuracy = 81.7%) with single predictors.

4. Discussion

This cross-sectional study gives a detailed clinicoradiological and multidomain cognitive profile of post-stroke cognitive impairment at the main tertiary neurovascular referral hospital in Bali, Indonesia. Among 60 consecutive outpatients with PSCI (mean age 61.71 ± 11.53 years; 60.0% male), mild cognitive impairment (MoCA-Ina 18–25) was the most common presentation at 43.3% (26/60). Severe impairment (MoCA-Ina < 10) followed at 30.0% (18/60), then moderate impairment (MoCA-Ina 10–17) at 26.7% (16/60). Taken together, the moderate and severe tiers made up 56.7% (34/60) of the cohort. Multidomain impairment involving two or more domains affected 95.0% (57/60), led by memory (95.0%, 57/60) and executive function (85.0%, 51/60). Table 6 compares these results with the literature. The agreement is close: international scientific statements and multi-country cohorts describe PSCI as a continuum from subtle multidomain executive-memory inefficiency to post-stroke dementia.[6–8] Zhang et al. pooled 7,048 first-ever stroke patients in a systematic review and meta-analysis. Post-stroke cognitive impairment affected 46% of survivors (95% CI: 36%–57%) and was reported in both ischemic and hemorrhagic stroke.[6] Mtambo et al., in their 2025 Southeast Asian scoping review of 78 studies, noted that Indonesian hospital cohorts have some of the highest PSCI burdens in the

region (27.6%–81.2%). Earlier domestic reports seldom combined neuroimaging lesion topography with subdomain-level MoCA-Ina phenotyping.[4] Our findings also complement the 2026 prospective acute ischemic stroke cohort from our own department at Prof. Dr. I.G.N.G. Ngoerah General Hospital by Patricia et al., which recorded a 72.4% (55/76) incidence of early cognitive impairment on the MoCA-Ina at two weeks after stroke. Cognitive morbidity in Bali is thus highly prevalent in the acute phase and persistently disabling among chronic outpatient survivors.[5]

Table 6. Comparison of the present cohort (N = 60) with selected studies of post-stroke cognitive impairment.

Study / cohort and region	Sample size, design, and assessment timing	Stroke subtype and admission severity	PSCI severity and cognitive domain profile	Key findings and relation to the present study
Present study (2026) Ngoerah Hospital, Bali, Indonesia	N = 60; cross-sectional; < 1 yr (51.7%), 1–2 yr (18.3%), > 2 yr (30.0%); MoCA-Ina	Ischemic 68.3%, hemorrhagic 25.0%, mixed 6.7%; minor NIHSS (1–4) in 85.0%	Mild 43.3%, moderate 26.7%, severe 30.0% (moderate-to-severe 56.7%); multidomain 95.0%, memory 95.0%, executive 85.0%, language 81.7%	Subcortical/lacunar lesions (95.0%), multiple lesions (63.3%; aOR = 6.18), ≥ 2 comorbidities (71.7%; aOR = 16.92), NIHSS ≥ 5 (aOR = 13.75), and recurrent stroke (25.0%; aOR = 9.55)
Patricia, Laksmidewi, et al. (2026)[5] Bali, Indonesia	Prospective ischemic stroke cohort (n = 76); MoCA-Ina at two weeks after stroke	Acute ischemic stroke survivors in Bali	Early cognitive impairment in 72.4% (55/76)	Elevated serum GFAP (astroglial injury) and low serum vitamin D independently increased the risk of cognitive impairment
Mtambo et al. (2025)[4] Southeast Asia scoping review	Multi-country synthesis (Indonesia, Malaysia, Singapore, Thailand); 103 articles from 78 studies	Predominantly ischemic stroke (65%–80%) with high hypertensive burden	PSCI prevalence 27.6%–81.2% in Indonesia; MoCA and MMSE most widely used	Identified few domain-specific and lesion-topography studies from Indonesia; our study addresses this regional gap
Gallucci et al. (2024)[16] Bernese cohort, Switzerland	Prospective cohort (n = 329 non-aphasic patients) despite state-of-the-art acute stroke treatment (IVT/EVT)	Median admission NIHSS = 1	PSCI in 69.3% of patients; domain deficits in 39%–51%	Minor initial NIHSS (85.0% in our cohort) did not protect against multidomain cognitive impairment
Milosevich et al. (2024)[22] OCS-Care cohort, United Kingdom	Prospective 6-month follow-up (n = 430) with the domain-specific Oxford Cognitive Screen	Ischemic and hemorrhagic stroke; broad NIHSS range	Language, memory, and attention deficits predominated at 6 months	Early domain-specific screening had prognostic value for 6-month deficits, in line with our early (< 1 yr, 51.7%) screening data
Marsh et al. (2022)[21] Johns Hopkins cohort, USA	Minor stroke cohort (n = 80; admission NIHSS ≤ 10) followed at 1, 6, and 12 months	Minor stroke without severe hemiparesis, aphasia, or neglect	Impairment in every domain, regardless of infarct location	Suggests generalized network dysfunction despite minor NIHSS scores
Coenen et al. (2024)[25] Meta VCI Map consortium	N = 1,568 stroke patients; multicenter lesion–symptom mapping	Ischemic stroke with white matter hyperintensities and lacunar infarcts	Executive, attention, and processing-speed deficits linked to strategic white matter hyperintensities	WMH in the left anterior thalamic radiation and forceps major was associated with cognitive deficits; compatible with our subcortical/periventricular/lacunar lesion predominance (95.0%)
Filler, Georgakis & Dichgans (2024)[18] Lancet Healthy Longevity meta-analysis	Systematic review and meta-analysis of risk factors for cognitive impairment after stroke (89 studies)	All stroke subtypes	High rates of cognitive impairment and dementia after stroke	Baseline cognitive impairment was the strongest factor; diabetes, atrial fibrillation, white matter hyperintensities, previous stroke, lacunes, and lower education also increased risk; partly overlaps with our Tables 4 and 5
Dowling, Johnson & Nadareishvili (2024)[9] JAHA meta-analysis	27 studies; N = 39,412 stroke survivors followed longitudinally	Ischemic stroke survivors	PSCI; domain profile not assessed	PSCI increased the risk of recurrent stroke by 59% and doubled mortality; consistent with our 25.0% recurrent stroke rate
Wang et al. (2025)[39] & Huang et al. (2025)[40] DTI-ALPS glymphatic cohorts	Prospective 3.0-T MRI DTI-ALPS cohorts at 7–180 days after stroke	Acute subcortical infarcts (basal ganglia/periventricular) and ischemic stroke	Lower MoCA, working memory, and executive set-shifting (TMT-B) scores	Lower DTI-ALPS index (AUC = 0.868 for cognitive impairment) and larger perivascular spaces were associated with working-memory and executive deficits after subcortical stroke

Abbreviations: AF, atrial fibrillation; aOR, adjusted odds ratio; AUC, area under the curve; DTI-ALPS, diffusion tensor image analysis along the perivascular space; EVT, endovascular thrombectomy; GFAP, glial fibrillary acidic protein; IVT, intravenous thrombolysis; MoCA-Ina, Indonesian version of the Montreal Cognitive Assessment; NIHSS, National Institutes of Health Stroke Scale; PSCI, post-stroke cognitive impairment; TMT-B, Trail Making Test Part B; WMH, white matter hyperintensity. Concentrations and rates from different studies are not directly comparable because of differences in design, instruments, and populations.

A particularly important observation in our data is the dissociation between initial neurological deficit on the NIHSS and the later burden of multidomain PSCI. At acute admission, 85.0% (51/60) of our patients had minor stroke severity (NIHSS 1–4), 6.7% (4/60) had moderate stroke, 5.0% (3/60) moderate-to-severe stroke, and 3.3% (2/60) severe stroke. Admission severity and cognitive burden diverged. In routine practice, an admission NIHSS ≤ 4 is often read as a mild, self-limiting insult that needs little rehabilitative follow-up after discharge.[16,21] Yet 95.0% of this largely minor-NIHSS cohort had multidomain cognitive impairment, and even in the minor-NIHSS subgroup (n = 51), 51.0% (26/51) had moderate-to-severe PSCI. These findings argue against the assumption that a low NIHSS score means preserved neurocognitive integrity. The 'minor-NIHSS, major-PSCI paradox' is not ours alone. The global stroke literature supports it.[16,21,22] Gallucci et al. (2024) followed a prospective Bernese cohort of 329 non-aphasic first-ever stroke patients (median admission NIHSS = 1). Despite modern reperfusion therapy and mild physical severity, PSCI affected up to half of the patients across verbal learning, episodic memory, and executive domains. It independently predicted worse three-month functional outcome on the modified Rankin Scale ($p < 0.001$).[16] Marsh et al. (2022) showed at Johns Hopkins that minor stroke causes generalized, bilateral disruption of cortical-subcortical cognitive networks rather than focal, lesion-restricted deficits.[21] The NIHSS was designed to quantify acute corticospinal tract weakness, cerebellar ataxia, visual field deficits, and overt perisylvian aphasia. It has almost no items that are sensitive to dorsolateral prefrontal executive control, delayed hippocampal memory consolidation, or frontoparietal attentional allocation.[17,20,23] Patients with lacunar or subcortical strokes may leave hospital with an NIHSS of 1 or 2. They remain at high risk of

disabling multidomain PSCI. Domain-sensitive instruments such as the MoCA-Ina are needed to find it.[17,23,24]

The timing of assessment in our cohort was 51.7% (31/60) within < 1 year of onset, 18.3% (11/60) at 1–2 years, and 30.0% (18/60) at > 2 years; this distribution reflects the dynamic, biphasic trajectory of post-stroke cognitive dysfunction.[10,18,41,42] Sexton et al. reported in a foundational meta-analysis that about 38% of stroke survivors have cognitive impairment without dementia in the first year. This represents the immediate structural effect of the index infarction or hemorrhage.[29] Some patients with minor cortical or subcortical stroke may recover partly through neuroplasticity between three and six months. Recovery is only part of the story. Large population-based longitudinal studies, notably the trajectory analysis by Levine et al. in JAMA and the REGARDS cohort, show that an incident stroke causes an acute step-wise drop in global cognition and executive function. It also causes a persistently faster rate of cognitive decline over the following years.[11] Time since stroke was not significantly associated with severity in our bivariate analysis. Patients assessed ≥ 1 year after stroke (the 1–2 year and > 2 year strata combined; n = 29) had a 62.1% (18/29) prevalence of moderate-to-severe PSCI. Among those assessed within < 1 year it was 51.6% (16/31) (OR = 1.53, p = 0.414). Recurrence was associated with severity. Patients with a documented history of recurrent stroke (25.0%, 15/60) had an 80.0% (12/15) prevalence of moderate-to-severe PSCI, against 48.9% (22/45) in first-ever stroke survivors (crude OR = 4.18, p = 0.035; multivariable aOR = 9.55, 95% CI: 1.47–61.98, p = 0.018). This nine-fold multivariable association with recurrent stroke may reflect a cumulative 'multi-hit' erosion of cognitive reserve, in which successive ischemic or hemorrhagic insults could exhaust compensatory neuroplasticity and push patients from mild cognitive impairment toward severe post-stroke dementia; the cross-sectional design cannot test this.[8,9,12,18]

A further aim was to place the observed cognitive domain hierarchy in the context of published neuroanatomical and connectomic work, summarized in the conceptual model in Figure 5. Memory (95.0%, 57/60) and executive function (85.0%, 51/60) were the two most widely impaired domains. Language (81.7%, 49/60) and attention (75.0%, 45/60) followed, and visuospatial ability was relatively spared at 46.7% (Cochran's Q = 58.42, p < 0.001). On imaging, this domain hierarchy matches the marked predominance of subcortical, basal ganglia, periventricular, and lacunar lesions in 95.0% (57/60) of patients; it also matches multiple lesion locations in 63.3% (38/60; multivariable aOR = 6.18, p = 0.014) and the lower prevalence of occipital lobe lesions (23.3%, 14/60). Classical behavioral neurology held that subcortical lacunar infarcts selectively slow processing speed and spare episodic memory. Tract-specific lesion–symptom mapping and functional connectomics have overturned that view.[25,43–46] In the Meta VCI Map consortium, Coenen et al. studied 1,568 ischemic stroke patients from nine international cohorts in 2024. Lesion and white matter hyperintensity volumes in the left anterior thalamic radiation and the forceps major had larger independent negative effects on executive function, attention, and processing speed than would be expected from total white matter hyperintensity volume. The forceps major association was with processing speed only.[25] Anatomically, the anterior limb and genu of the internal capsule, the caudate nucleus, the globus pallidus, and the anterior and dorsomedial thalamic nuclei are dense bottleneck hubs where two major macro-circuits converge. (1) The Alexander dorsolateral prefrontal–subcortical loop starts in the dorsolateral prefrontal cortex [DLPFC], projects to the dorsolateral caudate head, globus pallidus interna, and mediodorsal thalamus, and returns to the DLPFC; it governs executive set-shifting, working memory manipulation, verbal abstraction, and phonemic fluency. (2) The Papez limbic memory circuit links the hippocampal formation through the fornix to the mamillary bodies, mamillothalamic tract, anterior thalamic nuclei, anterior cingulate cortex, and parahippocampal gyrus. It is indispensable for episodic memory encoding and delayed recall.[43,47]

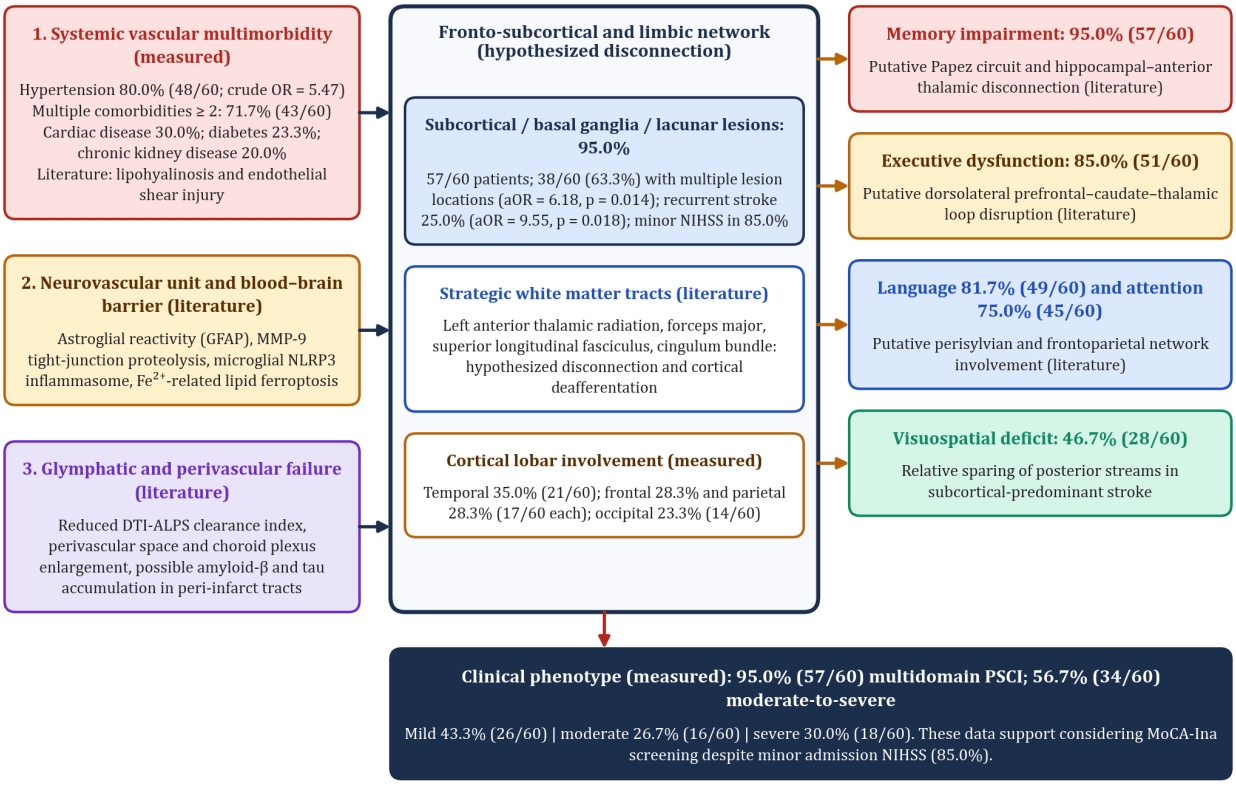


Figure 5. Conceptual (hypothesized) model of post-stroke cognitive impairment that places the present clinical and lesion data (hypertension 80.0%, multiple comorbidities 71.7%, cardiac disease 30.0%, diabetes 23.3%, renal disease 20.0%; subcortical/basal ganglia/lacunar lesions 95.0%, multifocal lesions 63.3%) alongside pathways reported in the literature (blood–brain barrier breakdown, glymphatic clearance failure, iron-related ferroptosis). The pathways were not measured in this study. They are drawn as converging on a fronto-subcortical and limbic network, with the multidomain deficits observed (95.0%) in memory (95.0%), executive function (85.0%), language (81.7%), attention (75.0%), and visuospatial processing (46.7%).

Resting-state functional MRI (rs-fMRI) and functional near-infrared spectroscopy (fNIRS) studies published between 2023 and 2026 provide supporting physiological evidence, from other cohorts, for this subcortical disconnection mechanism.[43–46,48] Han et al. (2025) used 3.0-T rs-fMRI. Patients with PSCI had significantly lower fractional amplitude of low-frequency fluctuations (zfALFF) in the left caudate nucleus, left putamen, anterior cingulate cortex, and superior and inferior temporal gyri. Regional homogeneity (SzKCC-ReHo) was also decreased in the middle frontal gyrus, and functional connectivity between the angular gyrus, precuneus, and inferior frontal gyrus was degraded. Anterior cingulate zfALFF correlated directly with MoCA scores.[43] Min et al. (2023) reported increased degree centrality of the right parahippocampal gyrus in mild subcortical stroke, and Liu et al. (2025) reported altered brain dynamics in post-stroke cognitive and motor dysfunction. Tao et al. (2026) observed aberrant hyper-connectivity across 20 cerebellocortical and fusiform network pairs after thalamic and basal ganglia infarction, which may reflect maladaptive or compensatory neural recruitment.[44–46] Liu et al. (2024) used fNIRS during working memory tasks. Patients with PSCI showed attenuated hemodynamic activation in the left prefrontal cortex and relied on inefficient compensatory recruitment of the right frontopolar and orbitofrontal cortices.[48] In our cohort, cortical involvement affected the temporal lobe in 35.0% (21/60) and the frontal and parietal lobes in 28.3% each. This cortical involvement may have disrupted perisylvian language networks and frontoparietal dorsal attentional networks, which could contribute to the 81.7% language and 75.0% attention deficits; network disruption was not measured. The lower rate of occipital lobe injury (23.3%) preserved primary and associative visual-constructional streams in more than half of the subtest evaluations (46.7% visuospatial deficit).

Focal disconnection is only part of the picture. Our multivariable model identified systemic vascular multimorbidity as the strongest systemic correlate of moderate-to-severe PSCI (≥ 2 comorbidities, present in 71.7% [43/60]; aOR = 16.92, p = 0.003). The burden was anchored by arterial hypertension in 80.0% (48/60; crude OR = 5.47, p = 0.013). Cardiac disease (30.0%, 18/60), diabetes mellitus (23.3%, 14/60), chronic kidney disease (20.0%, 12/60), and dyslipidemia (18.3%, 11/60) added to it.[49–52] At the microvascular and cellular level (Figure 5), chronic arterial hypertension exposes deep penetrating lenticulostriate and thalamoperforating arterioles to pulsatile barotrauma; this causes smooth muscle hypertrophy, lipohyalinosis, lumen narrowing, and impaired cerebrovascular autoregulation. The picture fits the 2024 meta-analysis by Huang et al., which found a significant pooled association between hypertension and PSCI (OR = 1.53, 95% CI: 1.18–1.99). It also fits the 2025 cerebral small vessel disease analysis by Cheng et

al.[49,50] Chronic hyperglycemia (Lee et al., 2023) is proposed to promote advanced glycation end-product (RAGE) signaling, endothelial nitric oxide synthase uncoupling, and basal lamina thickening. Atrial fibrillation and structural heart disease (He et al., 2025) release repetitive showers of microemboli and cause chronic cerebral hypoperfusion.[51,52] Recent translational research points to three converging molecular pathways that may link vascular multimorbidity to progressive post-stroke neurodegeneration; none was measured in the present cohort. First, blood–brain barrier breakdown with astroglial-microglial neuroinflammation. Ischemic and microvascular injury trigger matrix metalloproteinase-9 (MMP-9) proteolysis of tight junctions. Reactive astrogliosis follows, with massive release of glial fibrillary acidic protein (GFAP), as reported in the 2026 acute ischemic stroke cohort of our institution by Patricia, Laksmidewi, et al., which measured serum GFAP. Microglial NLRP3 inflammasome activation then releases neurotoxic IL-1β, TNF-α, and IL-6.[5,47,53] Second, failure of the glymphatic system. Prospective diffusion tensor imaging studies in 2025 by Wang et al. and Huang et al. reported a lower DTI-ALPS interstitial clearance index (AUC = 0.868 for discriminating early cognitive impairment after subcortical infarcts) and larger perivascular space (PVS) and choroid plexus volumes after stroke. These changes have been interpreted as impaired glymphatic clearance, with possible perivascular astrocytic aquaporin-4 (AQP4) depolarization and trapping of neurotoxic amyloid-β ($A\beta_{1–42}$) and hyperphosphorylated tau within peri-infarct white matter tracts.[39,40] Third, post-ischemic brain iron dyshomeostasis and ferroptosis. As synthesized by Guo et al. (2026), pro-inflammatory IL-6/JAK/STAT3 signaling upregulates local cerebral hepcidin. Hepcidin internalizes ferroportin-1 (FPN1) and traps ferrous iron (Fe^{2+}) in striatal and hippocampal neurons. Fenton-driven lipid peroxidation and glutathione peroxidase-4 (GPX4) depletion then cause ferroptotic neuronal death across fronto-subcortical and memory circuits.[54]

Applying these findings calls for a change in post-stroke care across Indonesian tertiary and secondary hospitals. Figure 6 sets out a three-stage clinical algorithm.[10,23,24,55,56] In Stage 1 (Early Cognitive Screening), 85.0% of PSCI patients in our cohort had minor acute stroke severity (NIHSS 1–4) and 95.0% had multidomain deficits. Routine bedside MoCA-Ina screening should therefore be considered for all stroke survivors. It should take place before hospital discharge (or within 2–4 weeks after the stroke) and be repeated at 3, 6, and 12 months in outpatient neurology polyclinics.[5,23,24,57] Clinicians should apply the standard MoCA-Ina threshold (< 26 with 1-point education adjustment) to maximize sensitivity for mild cognitive impairment (43.3% of our cohort). They should also apply the revised stroke-specific cutoff of 21/22 identified by Wei et al. (2023) to flag patients moving toward moderate-to-severe PSCI. The five subdomain scores need checking too, not only the global sum.[23,24] In Stage 2 (Clinicoradiological Risk Triage), patients with any of the four independent multivariable predictors in Table 5 could be prioritized for intensive, high-frequency neurorehabilitation pathways; these predictors are multiple comorbidities ≥ 2 (aOR = 16.92), admission NIHSS ≥ 5 (aOR = 13.75), recurrent stroke history (aOR = 9.55), and multiple lesion locations on CT/MRI (aOR = 6.18). Subcortical/lacunar topography (95.0%) and arterial hypertension (80.0%) add to the risk. In Stage 3 (Multimodal Domain-Targeted Rehabilitation), interventions should target the dominant memory (95.0%) and executive (85.0%) deficits in our cohort. Gao et al. pooled 19 randomized controlled trials (N = 875) in a 2025 meta-analysis. Computerized cognitive training (CCT) significantly improved global cognition (SMD = 0.46, $p < 0.001$), attention (SMD = −0.45, $p < 0.001$), executive function (SMD = 0.39, $p = 0.01$), and quality of life in PSCI. Short-term, high-frequency regimens worked best.[58] Brain stimulation is another option. Kreiger et al. (2025), Yang et al. (2025), and Han et al. (2023) synthesized the evidence. High-frequency (10–20 Hz) repetitive transcranial magnetic stimulation (rTMS) or anodal transcranial direct current stimulation (tDCS) over the left dorsolateral prefrontal cortex (DLPFC) restores excitatory network coupling between the CEN and DMN; it also upregulates hippocampal BDNF–TrkB–synaptophysin expression, suppresses microglial neuroinflammation, and improves working memory and executive control.[55,56,59] These cognitive and neuromodulatory approaches should be combined with strict secondary vascular prevention (blood pressure control, glycemic stabilization, statin therapy, vitamin D optimization, and cardioembolic anticoagulation). Together they form a comprehensive strategy to halt cognitive progression and preserve survivor independence.[60]

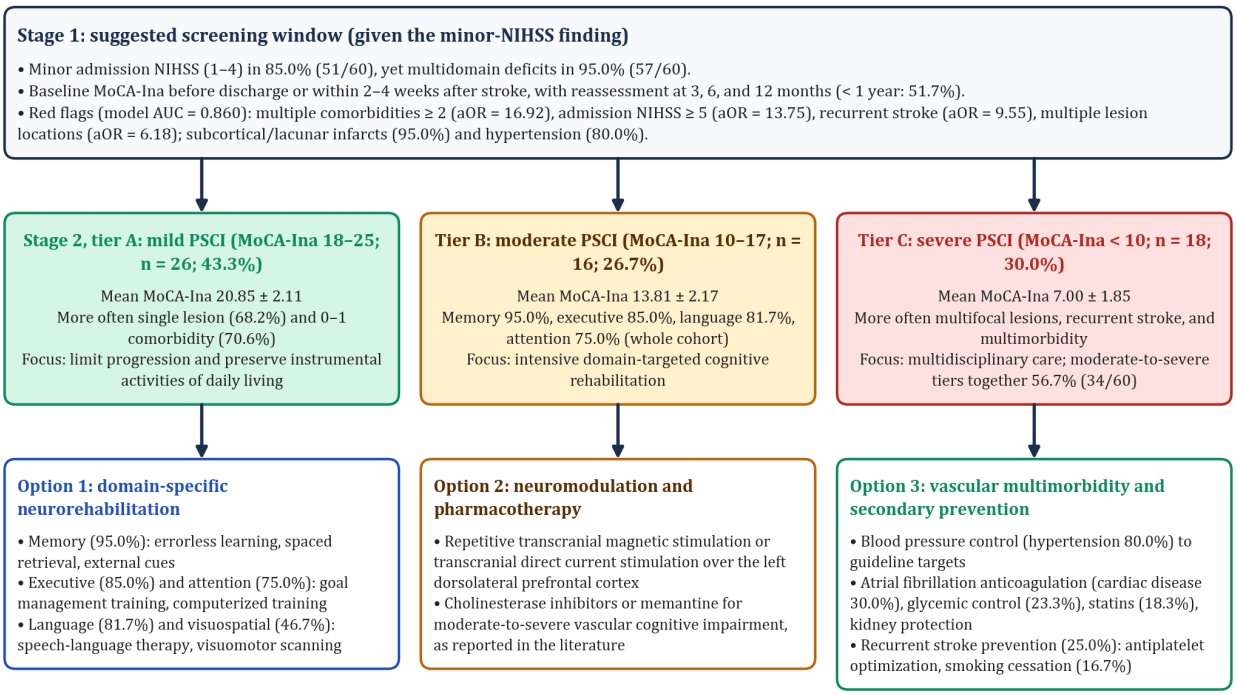


Figure 6. Proposed clinical approach (not a validated protocol) for early post-stroke cognitive screening, risk stratification, and multimodal neurorehabilitation in Indonesian tertiary stroke care, derived from the present associations and the literature. Stage 1 suggests MoCA-Ina screening before discharge and within the first year after stroke (< 1 year: 51.7%) given the minor admission NIHSS finding (85.0%). Stage 2 stratifies patients into mild (43.3%), moderate (26.7%), and severe PSCI (30.0%), guiding Stage 3 options of domain-specific cognitive rehabilitation, non-invasive neuromodulation (rTMS/tDCS), pharmacotherapy, and intensive secondary vascular prevention.

4.1. Strengths and limitations

The study has several strengths and some methodological limitations that should guide future research. Its strengths include a standardized, culturally validated psychometric instrument (MoCA-Ina) with complete five-domain decomposition. Lesion mapping was comprehensive across cortical, subcortical, and infratentorial compartments. MoCA-Ina subdomain phenotyping was standardized. The biostatistical analysis had three tiers (univariate, bivariate contingency, and IRLS multivariable logistic regression, with an ROC AUC of 0.860). There are limitations too. First, the cross-sectional design captures cognitive performance at a single outpatient evaluation; it cannot establish causal temporality or follow within-patient transitions between mild, moderate, and severe PSCI strata. Second, the study enrolled patients already diagnosed with PSCI who attended a tertiary academic neurology polyclinic (N = 60), with no parallel cognitively normal post-stroke control group. Our multivariable model therefore estimates the determinants of moderate-to-severe versus mild PSCI and not the population-level incidence of PSCI among all unselected stroke admissions. Third, the four-variable multivariable model had EPV = 6.5, below the conventional 10, and produced wide 95% confidence intervals around the adjusted odds ratios; it should be read as exploratory. Quantitative volumetric MRI metrics were also not routinely available in standard clinical records, such as automated white matter hyperintensity volume, medial temporal lobe atrophy (Scheltens scale), and DTI-ALPS glymphatic indices. Fourth, domain impairment was defined as any score below the domain maximum, the subcortical lesion category was a composite, education was not analyzed, and the data come from the records of one center, which limits generalizability. Future prospective, multicenter longitudinal cohorts across Indonesian stroke centers should combine serial MoCA-Ina domain tracking with quantitative multimodal MRI and circulating neuroinflammatory and astroglial biomarkers.

5. Conclusion

In this tertiary hospital cohort from Bali, Indonesia (N = 60), post-stroke cognitive impairment was characterized mainly by multidomain cognitive dysfunction (95.0%). Memory (95.0%) and executive function (85.0%) were the most severely affected domains, followed by language (81.7%), attention (75.0%), and visuospatial function (46.7%). Mild impairment was the most frequent presentation (43.3%). A combined 56.7% of patients had moderate (26.7%) or severe (30.0%) cognitive deficits. This burden of multidomain and moderate-to-severe cognitive morbidity occurred although 85.0% of patients had minor acute stroke severity on admission (NIHSS 1–4). Near-universal subcortical, basal ganglia, periventricular, and lacunar lesions (95.0%), multiple lesion locations (63.3%), arterial hypertension (80.0%), and systemic multimorbidity (71.7%) accompanied these findings.

Bivariate and multivariable logistic regression identified four statistically significant, independent clinicoradiological correlates of moderate-to-severe PSCI. They were multiple comorbidities ≥ 2 conditions (aOR = 16.92, p = 0.003), admission NIHSS ≥ 5 (aOR =

13.75, p = 0.044), recurrent stroke history (aOR = 9.55, p = 0.018), and multiple lesion locations (aOR = 6.18, p = 0.014). Discrimination was good (ROC AUC = 0.860). These results support routine early, domain-specific MoCA-Ina cognitive screening of all stroke survivors, especially those with minor NIHSS scores and subcortical lesions. Screening alone is not enough. It should be paired with targeted multimodal cognitive rehabilitation, non-invasive prefrontal neuromodulation, and intensive secondary vascular risk control to improve long-term functional recovery and quality of life.

6. Declarations

6.1. Ethics approval and consent to participate

The study protocol received ethical exemption from the Research Ethics Committee (Komisi Etik Penelitian) of the Faculty of Medicine, Universitas Udayana / Prof. Dr. I.G.N.G. Ngoerah General Hospital (Ethical Exemption No. 1514/UN14.2.2.VII.14/LT/2026). The study retrospectively used anonymized routine clinical and psychometric records; the committee therefore waived individual written informed consent, and all personal identifiers were removed before database lock, in line with the Declaration of Helsinki.

6.2. Consent for publication

Not applicable; no identifiable individual data or images are presented.

6.3. Availability of data and materials

All aggregated clinical, neuroanatomical, and psychometric data supporting the findings of this study are provided within Tables 1–6 and Figures 1–6; de-identified analytical records are available from the corresponding author upon reasonable request.

6.4. Competing interests

The authors declare that they have no financial or non-financial competing interests.

6.5. Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

6.6. Author contributions

A.A.A.P.L.: conceptualization, methodology, supervision, writing – original draft, writing – review & editing. I.G.A.A.A.Y.: investigation, data curation, formal analysis, validation, writing – review & editing. E.E.S.: investigation, data curation, visualization, writing – review & editing. All authors discussed the results and approved the final manuscript.

6.7. Use of generative artificial intelligence

The authors did not use generative artificial intelligence tools in the preparation of this manuscript.

6.8. Acknowledgements

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