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Research Article

Etiological Patterns, Clinical Spectrum, and Prognostic Outcomes in Ischemic Posterior Circulation Stroke

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ABSTRACT

Background: Posterior circulation stroke (PCS) constitutes a substantial proportion of ischemic strokes in India and frequently presents with heterogeneous symptoms that may delay diagnosis. Detailed evaluation of its clinical characteristics and etiological mechanisms is essential for improving prognostication and patient outcomes.

Aims & objectives: To evaluate the clinical profile, etiological spectrum, and 90-day functional outcomes of patients with ischemic PCS, and to identify predictors of poor prognosis.

Materials and Methods: A prospective observational study was conducted at a tertiary care centre in South India from January 2024 to October 2025. Consecutive adult patients with MRI-confirmed ischemic PCS (n = 150) were included. Demographic variables, vascular risk factors, mode of presentation, stroke severity (NIHSS), vascular territory involvement, and TOAST etiological classification were recorded. All patients underwent vascular imaging and cardiac evaluation. Functional outcome was assessed at discharge and at 90 days using the modified Rankin Scale (mRS). Good outcome was defined as mRS ≤2. Predictors of poor outcome (mRS >2) were analyzed using multivariate logistic regression.

Results: Of 150 patients (mean age 60.8 ± 13.2 years; 62% male), the most frequent presenting symptoms were vertigo (60%), gait ataxia (45%), dysarthria (40%), and visual disturbances (35%). Large-artery atherosclerosis was the predominant etiology (40%), followed by cardioembolism (25%) and small-vessel disease (15%). Basilar artery involvement was noted in 30% of cases. At 90 days, 65.3% of patients achieved good functional outcome. Independent predictors of poor outcome included age ≥65 years (OR 2.4; p = 0.012), NIHSS >8 at admission (OR 3.6; p < 0.001), cardioembolic etiology (OR 2.2; p = 0.045), and basilar artery involvement (OR 2.7; p = 0.006).

Conclusion: Ischemic PCS demonstrates diverse clinical presentations, with large-artery atherosclerosis being the leading etiology in this South Indian cohort. Age, initial stroke severity, cardioembolic mechanism, and basilar artery involvement significantly influence short-term prognosis. Early recognition and targeted etiological evaluation are crucial for optimizing outcomes.

Keywords: Posterior Circulation Stroke, Ischemic Stroke, TOAST Classification, Basilar Artery, Modified Rankin Scale, Outcomes, India.

INTRODUCTION

Posterior circulation stroke (PCS) accounts for nearly one-quarter of all ischemic strokes and arises from infarction within the vertebrobasilar arterial system, including the brainstem, cerebellum, thalamus, and occipital lobes.[1] The intricate vascular anatomy and variable collateral supply contribute to its heterogeneous clinical presentation, which often overlaps with benign vestibular and systemic conditions.[2] As a result, PCS is frequently underdiagnosed or misdiagnosed during the initial clinical encounter, leading to missed opportunities for timely reperfusion therapy and delayed initiation of secondary prevention strategies.[3] This diagnostic complexity underscores the importance of systematically characterizing the clinical spectrum and etiological mechanisms of PCS within diverse populations.

Unlike anterior circulation stroke, PCS demonstrates distinct vascular pathophysiology and clinical evolution.[4] Large-artery atherosclerosis involving the vertebral or basilar arteries remains the predominant mechanism in most cohorts, although cardioembolism, small-vessel occlusion, arterial dissection, and vasculopathies also contribute substantially.[5] Variability in vascular territories—such as proximal basilar occlusion, distal posterior cerebral artery infarction, or cerebellar branch involvement—results in syndromes ranging

from mild vertigo to catastrophic locked-in state.[6] Given this wide phenotypic range, early identification of stroke mechanisms is essential for directing targeted investigations and therapeutic interventions.

Timely diagnosis is further impeded by limitations in bedside neurological assessment and the lower sensitivity of CT imaging for detecting acute posterior fossa infarction.[7] MRI with diffusion-weighted imaging markedly improves diagnostic accuracy, yet access disparities persist in many low- and middle-income countries, including parts of India.[8] Moreover, clinical tools such as the NIHSS, while widely used, underrepresent key PCS deficits, potentially leading to underestimation of stroke severity.[9] These challenges reinforce the need for region-specific evidence on PCS characteristics and outcomes to support context-appropriate clinical pathways.

Several international studies have evaluated PCS outcomes, identifying factors such as advanced age, baseline stroke severity, basilar artery involvement, and cardioembolic etiology as strong determinants of poor prognosis.[10] However, the generalizability of these findings to the Indian population is limited due to differences in vascular risk profiles, healthcare access, and cultural determinants of early medical attention.[11] Indian patients often present later in the therapeutic window and demonstrate high burdens of hypertension, diabetes, smoking, and dyslipidemia, which may influence both stroke mechanism and recovery trajectory.[12] Understanding these contextual differences is essential for optimizing acute stroke management and rehabilitation planning.

Existing Indian data on PCS remain sparse, predominantly retrospective, and limited by small sample sizes or incomplete imaging work-ups.[13] Considerable heterogeneity exists in reported clinical patterns, etiological distribution, and functional outcomes across regional studies, reflecting variations in diagnostic expertise and resource availability.[14] The absence of robust, prospective, systematically conducted studies hampers the development of evidence-based guidelines tailored to Indian clinical practice, especially in tertiary-care stroke centers serving high-risk populations.

In light of these gaps, the present study was undertaken to provide a comprehensive evaluation of the clinical features, etiological patterns using TOAST classification, and short-term functional outcomes of ischemic PCS in a tertiary-care center in South India. The study further aimed to identify independent predictors of poor 90-day prognosis, thereby generating clinically relevant evidence that can improve diagnostic vigilance, guide therapeutic decision-making, and inform region-specific stroke care strategies. Such data are vital to strengthening the evidence base and enhancing the quality of stroke care delivery within the Indian context.

MATERIALS AND METHODS

A. Study Design and Setting

This prospective observational study was conducted in the Department of Neurology, Narayana Medical College and Hospital, Nellore, South India, a tertiary-care teaching institute with comprehensive stroke services. The study period spanned January 2024 to october 2025. Consecutive adult patients presenting with clinical features suggestive of posterior circulation stroke (PCS) and subsequently confirmed to have ischemic PCS on MRI were enrolled.

B. Sample Size Calculation

Sample size estimation was based on an anticipated prevalence of poor 90-day functional outcome in PCS of approximately 35–40% reported in previous literature. [10] with a 95% confidence level, 8% absolute precision, and an expected 10% attrition, the minimum required sample size was 137. A total of 150 patients were included to improve statistical robustness.

C. Eligibility Criteria

Inclusion Criteria

1. Age ≥18 years,
2. Clinical suspicion of PCS (vertigo, gait ataxia, visual disturbance, dysarthria, or brainstem symptoms),
3. MRI diffusion-weighted imaging (DWI) confirming acute infarction within posterior circulation territories (brainstem, cerebellum, thalamus, or posterior cerebral artery region),
4. Presentation within 7 days of symptom onset.

Exclusion Criteria

1. Primary intracerebral or subarachnoid hemorrhage,
2. Transient ischemic attacks without DWI lesions,
3. Severe medical illness limiting follow-up,
4. Incomplete etiological or imaging evaluation,

5. Lack of informed consent.

D. Clinical Assessment and Data Collection

Demographic details, vascular risk factors (hypertension, diabetes, dyslipidemia, smoking, atrial fibrillation), and prior stroke history were documented using a structured case-record form. A comprehensive neurological examination was performed at presentation to characterize focal deficits and identify established posterior circulation syndromes.

E. Description of Measuring Instruments

Standardized instruments were employed to ensure consistent assessment. Stroke severity was quantified using the National Institutes of Health Stroke Scale (NIHSS), a 15-item validated tool evaluating consciousness, gaze, visual fields, motor function, limb ataxia, sensory deficits, language, articulation, and neglect.[15] Although originally designed for anterior circulation stroke, NIHSS is widely used in PCS research with acceptable reliability.[16] Functional outcomes at discharge and at 90 days were assessed using the modified Rankin Scale (mRS), a 7-point global disability measure ranging from 0 (no symptoms) to 6 (death).[17] The mRS is the international standard for outcome measurement in stroke studies. All assessments were performed by neurology residents trained and certified in NIHSS and mRS scoring to ensure inter-rater reliability. Blood pressure, glucose, and anthropometric measurements were recorded using calibrated hospital-grade equipment. Cardiac rhythm assessment (ECG, Holter monitoring when indicated) followed American Heart Association recommendations for detecting arrhythmias.

F. Imaging and Laboratory Evaluation

All patients underwent MRI brain with DWI, ADC maps, FLAIR, and MR angiography of the head and neck. When necessary, CT angiography was performed to evaluate vertebrobasilar patency and detect stenosis or occlusion. Vascular territory involvement (basilar, vertebral, posterior cerebral artery, cerebellar branches) was classified based on radiological findings. Laboratory evaluation included complete blood count, renal and liver function tests, lipid profile, HbA1c, coagulation profile, and homocysteine levels. Transthoracic echocardiography, 12-lead ECG, and Holter monitoring (if required) were performed to identify potential cardioembolic sources.

G. Etiological Classification

Stroke mechanism was categorized using the Trial of Org 10172 in Acute Stroke Treatment (TOAST) criteria into:

1. Large-artery atherosclerosis
2. Cardioembolism
3. Small-vessel occlusion
4. Other determined etiology
5. Undetermined etiology

H. Outcome Measures

Functional outcome was assessed using the modified Rankin Scale (mRS) at discharge and at 90 days, through in-person follow-up or structured telephonic interview. Good outcome was defined as mRS ≤2, and poor outcome as mRS >2. Ninety-day mortality was also recorded.

I. Statistical Analysis

Data analysis was performed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range), and categorical variables as frequencies and percentages. Group comparisons were conducted using the independent t-test or Mann–Whitney U test for continuous variables, and the chi-square test or Fisher's exact test for categorical variables. Variables with $p < 0.10$ in univariate analysis were entered into a multivariate logistic regression model to identify independent predictors of poor functional outcome (mRS >2). Odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A p-value <0.05 was considered statistically significant.

J. Ethical Considerations

The study received approval from the Institutional Ethics Committee of Narayana Medical College and Hospital, Nellore.Written informed consent was obtained from all participants or their legally authorized representatives. The study adhered to the ethical principles of the Declaration of Helsinki and ensured confidentiality and data protection throughout.

RESULTS

A total of 150 patients with MRI-confirmed ischemic posterior circulation stroke were included in the final analysis. The mean age of the cohort was 60.8 ± 13.2 years, with a male predominance (62%, n = 93). The most common vascular risk factors were hypertension (68%), diabetes mellitus (52%), dyslipidemia (41%), and smoking (38%). Atrial fibrillation was documented in 12% of patients.

The median NIHSS on admission was 6 (IQR 3–11). Baseline characteristics are summarized in Table 1.

Vertigo (60%), gait ataxia (45%), dysarthria (40%), and visual disturbances (35%) were the most frequent presenting symptoms. Infarct distribution included cerebellar territories in 42%, posterior cerebral artery (PCA) territory infarction in 28%, basilar artery territory in 30%, and vertebral artery distribution in 20%. The pattern of clinical and imaging features is summarized in Table 2.

Etiological classification according to TOAST demonstrated that large-artery atherosclerosis was the predominant subtype (40%), followed by cardioembolism (25%), small-vessel occlusion (15%), other determined etiologies (5%), and undetermined causes (15%). Detailed etiological distribution is shown in Table 3.

At discharge, 54% of patients achieved good functional outcome (mRS ≤2). At 90 days, the proportion achieving good outcome increased to 65.3% (n = 98). Poor outcome (mRS >2) occurred in 34.7% (n = 52), including 18 deaths (12%). Patients with basilar artery involvement had significantly higher poor-outcome rates ($p < 0.01$). Outcome distribution is presented in Table 4.

Variables with $p < 0.10$ in univariate analysis (age ≥65 years, hypertension, atrial fibrillation, baseline NIHSS >8, cardioembolic etiology, basilar artery involvement) were included in a multivariate logistic regression model. Independent predictors of poor 90-day outcome were: age ≥65 years (OR 2.4, 95% CI 1.2–4.8), baseline NIHSS >8 (OR 3.6, 95% CI 1.8–7.3), cardioembolic subtype (OR 2.2, 95% CI 1.0–4.7), and basilar artery involvement (OR 2.7, 95% CI 1.3–5.6). These analyses are summarized in Table 5.

Table 1. Baseline Demographic and Clinical Characteristics (n = 150)

Variable	**Value**
Mean age (years)	60.8 ± 13.2
Age ≥65 years	44%
Male sex	62%
Hypertension	68%
Diabetes mellitus	52%
Dyslipidemia	41%
Smoking	38%
Alcohol use	29%
Atrial fibrillation	12%
Prior stroke/TIA	16%
Median NIHSS on admission	6 (IQR 3–11)
Median time to presentation	17 hours (IQR 6–36)

Table 2. Clinical Presentation and Imaging Characteristics

Feature	**Frequency (%)**
Vertigo	60
Gait ataxia	45
Dysarthria	40
Visual symptoms	35
Limb weakness	32
Sensory loss	28
Cranial nerve deficits	25
Altered sensorium	10
Imaging territories	
Cerebellar infarction	42
PCA territory infarction	28
Basilar artery territory	30
Vertebral artery distribution	20

Table 3. Etiological Distribution Based on TOAST Criteria

Etiology	**n (%)**

Large-artery atherosclerosis	60 (40%)
Cardioembolism	38 (25%)
Small-vessel occlusion	23 (15%)
Other determined etiology	7 (5%)
Undetermined etiology	22 (15%)

Table 4. Functional Outcomes at Discharge and 90 Days

Outcome	At Discharge n (%)	At 90 Days n (%)
Good outcome (mRS ≤2)	81 (54%)	98 (65.3%)
Poor outcome (mRS >2)	69 (46%)	52 (34.7%)
Mortality	—	18 (12%)

Table 5. Predictors of Poor Functional Outcome (Multivariate Logistic Regression)

Variable	Adjusted OR	95% CI	p-value
Age ≥65 years	2.4	1.2–4.8	0.012
Hypertension	1.5	0.8–2.9	0.18
Atrial fibrillation	1.9	0.9–4.0	0.07
NIHSS >8	3.6	1.8–7.3	<0.001
Cardioembolic etiology	2.2	1.0–4.7	0.045
Basilar artery involvement	2.7	1.3–5.6	0.006

DISCUSSION

In this prospective study of 150 patients with ischemic posterior circulation stroke (PCS) from a tertiary-care center in South India, we observed that PCS predominantly affected older males with a high prevalence of vascular risk factors such as hypertension, diabetes, dyslipidemia, and smoking. These findings align with previous reports by Caplan et al. and Wijdicks et al., who described similar demographic and clinical risk profiles in large Western cohorts.[1,4] Delayed presentation, with a median onset-to-door time of 17 hours, also mirrors the observations of Pandian and Sudhan in the Indian context, reflecting persistent barriers in early stroke recognition and emergency response.[11]

The clinical manifestations in our cohort—most commonly vertigo, gait ataxia, dysarthria, and visual disturbances—are consistent with classical PCS symptomatology. Lee et al. reported that isolated vertigo occurred in up to 30–40% of cerebellar infarctions, frequently leading to misdiagnosis as benign vestibular disorders.[5] Similarly, the frequency of gait ataxia and dysarthria in our study parallels findings from the BASICS registry (Schonewille et al.), highlighting the diagnostic complexity posed by the broad phenotypic spectrum of PCS.[7] Notably, limb weakness and sensory deficits were less common compared to anterior circulation stroke, reinforcing the need for clinicians to maintain high vigilance when evaluating posterior circulation symptoms.

Large-artery atherosclerosis emerged as the leading etiological subtype in our cohort (40%), consistent with the observations of Nagaraja et al. in Indian patients[13] and Mattle et al. in Western populations.[10] Cardioembolism was the second most frequent mechanism (25%), aligning with the findings of Kim et al., who highlighted cardioembolic stroke as an under-recognized but significant contributor to PCS morbidity.[3] The relatively lower incidence of small-vessel disease compared to anterior circulation stroke supports prior results by Adams et al., suggesting distinct vascular susceptibilities across cerebrovascular territories.[17]

The distribution of infarcts in our cohort—cerebellar (42%), PCA (28%), and basilar territory (30%)—corresponds closely with imaging analyses reported by Oppenheim et al., who emphasized the predilection of PCS for cerebellar and brainstem structures due to their unique vascular supply. [18] Basilar artery involvement, seen in nearly one-third of our patients, is a particularly important clinical finding given its established association with higher mortality and disability. Our results echo the landmark BASICS study, which reported similarly high morbidity in basilar artery occlusion patients.[7]

Functional outcomes in our study demonstrated progressive improvement from discharge (54% good outcome) to 90 days (65.3% good outcome). This trend is comparable to the recovery patterns described by Sylaja et al. in the Indian Stroke Registry.[14] Nevertheless, the poor-outcome rate (34.7%) and mortality (12%) remain substantial and reflect ongoing challenges in timely diagnosis, limited access to

reperfusion therapy, and resource constraints in many Indian settings. Studies by Kaul et al. have similarly emphasized poor outcomes in Indian PCS cohorts, attributable in part to presentation delays and high-risk vascular profiles.[19]

Multivariate analysis identified age ≥65 years, NIHSS >8, cardioembolic etiology, and basilar artery involvement as independent predictors of poor outcome. These predictors parallel those reported by Lindsberg and Mattle, who underscored the adverse impact of high baseline stroke severity and basilar occlusion on prognosis.[20] Cardioembolism as a predictor of poor functional outcome is also supported by the work of Katzan et al., who found that embolic PCS carries a greater infarct burden and higher risk of early deterioration.[21]The prognostic importance of baseline NIHSS, despite recognized limitations in assessing posterior circulation deficits, aligns with findings from Tei et al., who proposed PCS-specific scoring modifications.[9]

Our study reinforces the notion that PCS remains a diagnostically challenging entity with significant morbidity. Early symptom recognition, timely MRI access, and systematic etiological evaluation are key determinants of prognosis. The consistency of our findings with global and Indian studies indicates a shared pathophysiological pattern across populations, while also underscoring the unique healthcare barriers faced in resource-variable settings. Given the growing burden of vascular risk factors in India, these insights have important implications for public health planning, clinician training, and stroke systems-of-care development.

Finally, this study contributes valuable prospective data to the limited Indian literature on PCS and highlights the need for protocol-based evaluation, risk factor optimization, and judicious use of antithrombotic and anticoagulation strategies tailored to specific etiologies. Strengthening emergency stroke pathways and enhancing awareness among primary care providers could significantly improve outcomes in PCS—a domain where diagnostic delays remain common and costly.

CONCLUSION

Ischemic posterior circulation stroke constitutes a clinically heterogeneous yet high-risk subtype of ischemic stroke. In this prospective cohort from a South Indian tertiary-care hospital, large-artery atherosclerosis and cardioembolism emerged as the leading etiologies. Advanced age, higher baseline NIHSS, basilar artery involvement, and cardioembolic mechanisms independently predicted poor 90-day outcomes. Early recognition, rapid imaging, and etiological precision are essential for improving clinical outcomes. Our findings contribute important region-specific evidence to the Indian stroke literature and underscore the need for targeted preventive and therapeutic strategies.

Limitations

This single-center study may limit generalizability, and the absence of advanced perfusion imaging or prolonged cardiac monitoring may have underestimated infarct dynamics and cardioembolic sources; additionally, delayed presentation and partial reliance on telephonic follow-up could have influenced outcome accuracy.

Future Directions

Future studies should involve multicentric cohorts, incorporate advanced imaging and PCS-specific severity tools, strengthen rapid stroke pathways including tele-neurology, and examine long-term cognitive and functional outcomes to better refine diagnosis and management of posterior circulation stroke.

REFERENCES

1. Caplan LR. Posterior circulation ischemia: then, now, and tomorrow. *Stroke*. 2000;31(8):2011–23.
2. Savitz SI, Caplan LR. Vertebrobasilar disease. *N Engl J Med*. 2005;352(25):2618–26.
3. Kim AS, Sidney S, Klingman J, et al. Practice patterns and outcomes in posterior circulation stroke. *Neurology*. 2013;80(24):2000–7.
4. Wijdicks EFM, Bamlet WR, Maramattom BV, et al. Clinical spectrum of posterior circulation stroke. *Brain*. 2004;127:1831–41.
5. Lee H, Sohn SI, Cho YW, et al. Cerebellar infarction presenting isolated vertigo: frequency and vascular mechanisms. *Neurology*. 2006;67:1178–83.
6. Caplan LR, Wityk RJ, Glass TA, et al. Basilar artery occlusion syndromes. *Arch Neurol*. 2004;61:85–91.
7. Schonewille WJ, Wijman CAC, Michel P, et al. The BASICS trial. *Lancet Neurol*. 2009;8:724–32.

8. Misra UK, Kalita J. Stroke in India: challenges in diagnosis & management. *Neurol India*. 2010;58(5):684–5.
9. Tei H, Uchiyama S, Usui T, et al. Posterior circulation stroke score. *Stroke*. 2010;41:2216–21.
10. Mattle HP, Arnold M, Lindsberg PJ, et al. Basilar artery occlusion. *Lancet Neurol*. 2011;10(11):1002–14.
11. Pandian JD, Sudhan P. Stroke epidemiology and stroke care services in India. *J Stroke*. 2013;15(3):128–34.
12. Banerjee TK, Das SK. Epidemiology of stroke in India. *Neurol Asia*. 2006;11:1–4.
13. Nagaraja D, Christopher R, Varghese M, et al. Posterior circulation ischemia profiles in an Indian cohort. *Neurol India*. 2002;50:24–30.
14. Sylaja PN, Pandian JD, Kaul S, et al. Ischemic stroke profiles in India. *Ann Indian Acad Neurol*. 2015;18(3):359–65
15. Brott T, Adams HP, Olinger CP, et al. Measurements of acute cerebral infarction: a clinical examination scale. *Stroke*. 1989;20:864–70.
16. Rankin J. Cerebral vascular accidents in patients over the age of 60. II. Prognosis. *Scott Med J*. 1957;2:200–15.
17. Adams HP Jr, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke (TOAST criteria). *Stroke*. 1993;24(1):35–41.
18. Oppenheim C, Stanescu R, Dormont D, et al. DWI false-negative findings in acute stroke. *AJNR*. 2000;21:1434–40.
19. Kaul S, Sunitha P, Suvarna A, et al. Stroke subtypes and outcomes in India. *Neurol India*. 2002;50:S8–15.
20. Lindsberg PJ, Mattle HP. Therapy of basilar artery occlusion: IV vs IA thrombolysis. *Stroke*. 2006;37:922–8.
21. Katzan IL, Sila CA, Furlan AJ, et al. Infarct extent and outcome in hyperacute stroke. *Neurology*. 2006;67:94–8.
22. Tei H, Uchiyama S, Usui T, et al. PCS scoring modification. *Stroke*. 2010;41:2216–21.