Original Article

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Decoding the Clinical Patterns and Underlying Risk Factors of Posterior Circulation Stroke: A Cross-sectional Study

Others Section

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ABSTRACT

Introduction: Posterior Circulation Stroke (PCS) constitutes a substantial proportion of ischemic strokes. It frequently exhibits non specific clinical features, making diagnosis difficult and potentially delayed. Hence, adequate knowledge of the clinical trends and risk factors is essential for early detection and improved outcomes.

Aim: To assess the demographic features, clinical manifestations and vital vascular risk factors in PCS patients.

Materials and Methods: This hospital-based cross-sectional study was conducted at the General Medicine Department in a Tertiary Care Centre at Kanchipuram, Chennai, Tamil Nadu, India, from July 2024 to July 2025. A total of 50 patients diagnosed with PCS were included in the study. The assessment of patients was done via brief clinical history, neurological examination and investigation of the laboratory and imaging data. A structured proforma was utilised to record demographic data, risk factors pertaining to vasculature and clinical characteristics of the PCS patients. Descriptive statistical analysis was done to evaluate the collected data, with results expressed as mean, Standard Deviation (SD), frequencies and percentages.

Results: The mean age was 58.6±12.4 years. The male population constituted 32 (64%). The average BMI was 26.1±3.8kg/m^2, indicating that the mean BMI was within the overweight range. The predominant risk factor was hypertension with 31 (62%), followed by smoking with 28 (56%), diabetes mellitus with 24 (48%), hyperlipidaemia with 20 (40%) and alcohol consumption with 18 (36%). Smoking was significantly associated with brainstem involvement on univariate analysis (p=0.048) and remained an independent predictor after multivariable adjustment {Adjusted Odds Ratio (AOR) 3.21; 95% CI: 1.09–9.42; p=0.034}. The p-value <0.05 was considered statistically significant.

Conclusion: In this cohort, PCS was more frequently observed among older adults and males, with a substantial burden of vascular risk factors. Even though statistical associations were not identified with any of the modifiable cardiovascular risk factors except smoking, the potential outcomes of these risk factors cannot be overlooked. Early diagnoses of these risk determinants play a critical role in mitigating complications and optimising clinical outcomes.

Keywords: Atherosclerosis, Hypertension, Ischaemic stroke, Posterior circulation, Risk factors

INTRODUCTION

Stroke is a highly prevalent condition associated with significant long-term disability and contributes significantly to the increasing mortality rate. Stroke is a serious neurological disorder resulting from abrupt interruption of cerebral blood flow. Ischemic stroke and haemorrhagic stroke are the two pathological forms of stroke depending on the mechanism of occurrence, with the majority of cases being ischaemic stroke. Stroke can also be categorised based on the vascular territory involved into Anterior Circulation Stroke (ACS) and Posterior Circulation Stroke (PCS). ACS involves the carotid arterial system, including the Anterior Cerebral Artery (ACA) and Middle Cerebral Artery (MCA) territories, whereas PCS involves the vertebrobasilar system [1-3].

The PCS refers to ischaemic events transpiring in the vascular region of the brain supplied by vertebral, basilar and posterior cerebral arteries. These arteries perfuse crucial brain structures such as the cerebellum, brainstem, thalamus and occipital lobes. PCS accounts for approximately 20-25% of all ischaemic strokes and remains less readily recognised than ACS because of its diverse and often non specific clinical presentation [4-6]. PCS are frequently correlated with morbidity and mortality owing to the complex anatomy of the vertebrobasilar system and the involvement of critical neuroanatomical structures. The clinical presentation of PCS is highly varied and often non specific. The predominant symptoms reported in PCS include dizziness, vertigo, headache, ataxia, dysarthria, limb weakness, nausea, vomiting, visual disturbances and altered consciousness, with the clinical presentation varying according to the specific vascular territory involved [7]. PCS contributes to a higher risk of misdiagnosis compared to ACS due to the similarity of symptoms with various neurological or vestibular disorders. This diagnostic challenge may lead to delay in ideal intervention for PCS, ultimately causing poor clinical outcomes [7,8].

Multiple aetiological and vascular risk factors contribute to the development of PCS. The classic risk factors of cardiovascular diseases are hypertension, diabetes mellitus, dyslipidaemia, smoking and cardiac diseases. These aforementioned risk factors are significant in the progression or pathogenesis of vertebrobasilar ischaemia [9,10]. Among patients with symptomatic vertebrobasilar disease, hypertension and hyperlipidaemia have been reported as highly prevalent risk factors [9]. Moreover, vertebral artery hypoplasia, vertebrobasilar stenosis, arterial dissection and dolichoectasia may be structural vascular abnormalities that are likely to predispose to impaired blood flow and ischaemic events in the posterior circulation [10,11].

Recent evidence has explored the clinical manifestations and vascular risk factors associated with PCS. They have reported a wide spectrum of clinical manifestations along with frequent coexistence of vascular disorders [12-14]. However, considerable variations in symptom profiles and risk factor distribution have been reported across different populations and healthcare settings. Furthermore, limited data are available regarding the association between clinical presentation and vascular risk factors among patients with PCS in the Indian population [13,14]. Improved insight into the clinical patterns and risk factors underlying it could ensure timely diagnosis,

appropriate management and effective preventive interventions. Therefore, the present study was undertaken to evaluate the clinical patterns and distribution of vascular risk factors among patients with PCS admitted to a tertiary care hospital and to assess the association between clinical presentation and risk variables.

MATERIALS AND METHODS

A hospital-based cross-sectional study was performed in the General Medicine Department in a Tertiary Care Centre at Kanchipuram, Chennai, Tamil Nadu, India, from July 2024 to July 2025. The study population comprised patients who were admitted to the Intermediate Medical Care Unit (IMCU) and Intensive Coronary Care Unit (ICCU) with the clinical features indicative of stroke of the posterior circulation. The Institutional Ethics Committee (IEC) provided the ethical clearance before the study commenced (Ethical clearance No. MMCH and RI IEC/PG/20/MAY/24).

Inclusion and Exclusion criteria: The study included patients aged ≥18 years with a diagnosed with PCS. The patients who presented within 48 hours of the onset of symptoms were the target population. The 48-hour time window was adopted to ensure recruitment of patients during the acute phase of stroke and to minimise potential recall bias regarding symptom onset and clinical presentation. Patients who reported after 48 hours of interval and who were not in a stable condition to give informed consent were excluded from the study.

Study sample: The study included 50 patients diagnosed with PCS. A convenience sampling method was used, and all eligible patients admitted during the study period were included. No a priori sample-size calculation or power analysis was performed.

Study Procedure

Demographic characteristics, vascular risk factors, clinical manifestations, and neuroimaging findings were systematically recorded using a standardised predesigned proforma. Routine laboratory and cardiovascular investigations were performed as part of standard clinical care and were reviewed for diagnostic and aetiological assessment.

i **Demographic and previous history:** The patients with clinical manifestations indicative of a PCS and confirmed by neuroimaging were included for evaluation. The clinical evaluation that led to the diagnosis of PCS was confirmed by clinical analysis backed by imaging data that revealed the infarction in the vertebrobasilar arterial territory. Patients with neuroimaging showing either intracranial haemorrhage or infarcts that were confined to the anterior circulation territory were not considered for the study. Age and gender were considered as detailed demographic information on all participants. A brief history of the clinical details was also taken, including the time when the stroke developed, how symptoms progressed, and the chronological order of the development of symptoms, including motor weakness, sensory problems, speech disorders, visual symptoms, instability symptoms, memory problems, or changes in behaviour. The previous occurrence of disease, hypertension, diabetes mellitus and other risk factors were also taken.

ii **Clinical features:** All the included patients were advised for a comprehensive clinical evaluation during admission. General physical examination involved evaluation of vital signs, pulse rate, blood pressure and evaluation of carotid pulse, body mass index and cutaneous signs including xanthomas. In each case, a detailed neurological examination was done, which involved assessment of higher mental functions, examination of cranial nerves, assessment of the motor system, sensory examination, cerebellar examination, as well as speech and level of consciousness. The signs indicative of involvement of the brainstem and cerebellum, such as ataxia, vertigo, dysarthria, diplopia, and cranial nerve deficits, were specifically focused on. The study focused primarily on the clinical profile and vascular risk factors of PCS; hence standardised stroke severity scales such as NIHSS and mRS were not included in the assessment protocol.

iii **Laboratory investigations:** All patients underwent routine laboratory and cardiovascular investigations as part of the standard diagnostic evaluation of stroke. These included complete blood count, blood glucose profile, renal function tests, lipid profile, electrocardiography, echocardiography, and additional investigations when clinically indicated. The findings of these investigations were utilised for diagnostic confirmation, assessment of vascular risk factors, and etiological evaluation. Only variables relevant to the study objectives were included in the statistical analysis and are presented in the results section.

iv **Radiological investigations:** Neuroimaging was involved in making the diagnosis and ascertaining the topographical location of infarction. Computed Tomography (CT) of the brain was done for all the patients during admission or as soon as possible after the occurrence of the symptoms to eliminate intracranial haemorrhage. The MRA and MRI of the brain were done to detect infarcts in the vertebrobasilar territory and determine vascular abnormalities. Doppler ultrasonography of carotid and vertebral arteries was also done to assess extracranial vascular pathology when necessary. The infarct location in the posterior circulation was categorised as a result of imaging findings and included the brainstem, cerebellum, thalamus, or occipital lobes. For analytical purposes, patients were categorised based on the presence or absence of brainstem involvement on neuroimaging. Brainstem involvement was defined as infarction affecting the pons, medulla, or midbrain. Patients with brainstem infarction were included in the brainstem involvement group irrespective of concomitant involvement of other posterior circulation territories. Patients without brainstem involvement were classified as having other posterior circulation infarctions.

STATISTICAL ANALYSIS

Data were analysed using Statistical Package for Social Sciences (IBM SPSS) statistics version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarise demographic characteristics, clinical features, vascular risk factors, and neuroimaging findings. Continuous variables were expressed as mean±SD, while categorical variables were presented as frequencies and percentages. Associations between categorical variables were assessed using the Chi-square test or Fisher's-exact test, where appropriate. Variables showing potential association with brainstem infarction were further evaluated using multivariate logistic regression analysis to identify independent predictors. AORs with 95% Confidence Intervals (CIs) were calculated. Model performance was assessed using the Nagelkerke R^2 statistic and Hosmer-Lemeshow goodness-of-fit test. A p-value of <0.05 was considered statistically significant.

RESULTS

The baseline demographic characteristics and the vascular risk factors of the patients included in the study are presented in [Table/Fig-1]. A total of 50 patients diagnosed with PCS were included, with a mean age of 58.6 ± 12.4 years, indicating that the age group was middle-aged to elderly. The largest percentage was in the 51-60 years (28%), then 61-70 years (24%). Males constituted the majority of the study population 64% and the average BMI was 26.1±3.8 kg/m^2, indicating most of the patients were overweight. Hypertension was the most prevalent vascular risk factor, occurring in 31 (62%) of the patients, followed by smoking 28 (56%) and diabetes mellitus 24 (48%). Atrial fibrillation was identified in 11 (22%) of patients, while 9 (18%) had a previous history of transient ischaemic attack or stroke.

Variables	n (%)
Age group distribution (in years)	
<40	6 (12.0%)
41-50	10 (20.0%)
51-60	14 (28.0%)
61-70	12 (24.0%)
>70	8 (16.0%)
Age (years) (Mean±SD)	58.6±12.4
Gender	
Male	32 (64.0%)
Female	18 (36.0%)
Body Mass Index (kg/m²) (Mean±SD)	26.1±3.8
Co-morbidities	
Hypertension	31 (62.0%)
Diabetes mellitus	24 (48.0%)
Hyperlipidaemia	20 (40.0%)
Smoking history	28 (56.0%)
Alcohol consumption	18 (36.0%)
Atrial fibrillation	11 (22.0%)
Previous TIA/Stroke	9 (18.0%)
Family history of stroke/CVD	13 (26.0%)

[Table/Fig-1]: Baseline demographic characteristics and vascular risk factors of the study population with PCS (N=50).

The clinical features of PCS are summarised in [Table/Fig-2]. Nausea/vomiting 34 (68%) and giddiness/vertigo 32 (64%) were the most common presenting symptoms. Motor weakness was observed in more than half of the patients 27 (54%), while ataxia was present in nearly half of the cohort 24 (48%), highlighting the frequent involvement of motor and cerebellar pathways in PCS. Other commonly reported manifestations included dysarthria 18 (36%), headache 16 (32%), diplopia 15 (30%), and cranial nerve deficits 14 (28%). Visual disturbances 12 (24%), sensory deficits 13 (26%), altered sensorium 10 (20%), and dysphagia 9 (18%) were observed less frequently.

The distribution of infarct locations based on neuroimaging findings is shown in [Table/Fig-3]. A total of 26 patients demonstrated brainstem involvement, including pontine 15 (30%), medullary 6 (12%), and midbrain infarctions 5 (10%). Cerebellar 11 (22%), occipital lobe

Clinical features	n (%)
Nausea/Vomiting	34 (68.0%)
Giddiness/Vertigo	32 (64.0%)
Motor weakness	27 (54.0%)
Ataxia	24 (48.0%)
Diplopia	15 (30.0%)
Dysarthria	18 (36.0%)
Visual disturbances	12 (24.0%)
Altered sensorium	10 (20.0%)
Headache	16 (32.0%)
Sensory deficits	13 (26.0%)
Dysphagia	9 (18.0%)
Cranial nerve deficits	14 (28.0%)

[Table/Fig-2]: Clinical presentation of the study population with PCS during admission (N=50).

infarctions 8 (16%), and thalamic infarctions 5 (10%) were also observed. Multiple posterior circulation territory involvement was noted in 9 (18%) of patients; therefore, infarct location categories were not mutually exclusive. Vertebrobasilar large-vessel pathology and cardioembolic sources were identified in 13 (26%) and 10 (20%) of patients, respectively.

Infarct location	n (%)
Brainstem infarction	
Pontine infarction	15 (30.0%)
Medullary infarction	6 (12.0%)
Midbrain infarction	5 (10.0%)
Cerebellar infarction	11 (22.0%)
Occipital lobe infarction	8 (16.0%)
Thalamic infarction	5 (10.0%)
Multiple posterior circulation territories involved	9 (18.0%)
Large vessel vertebrobasilar involvement (MRA/Doppler)	13 (26.0%)
Cardioembolic source detected on echocardiography	10 (20.0%)

[Table/Fig-3]: Neuroimaging findings and distribution of infarcts in posterior circulation territory (N=50).

The association between infarction patterns and major vascular risk factors are presented in [Table/Fig-4]. Patients with brainstem infarction demonstrated a higher prevalence of hypertension (69.2%), smoking (69.2%) and diabetes mellitus (57.7%) compared with those with infarcts at posterior circulation sites. Among the evaluated risk factors, only smoking showed a statistically significant association with brainstem infarction (p=0.048).

Multivariate logistic regression analysis was conducted to analyse the independent predictors of brainstem involvement in the occurrence of stroke in the posterior circulation, as seen in [Table/Fig-5]. Smoking was significantly associated with brainstem involvement

Risk factors	Brainstem infarct (n=26)	Other posterior territory infarcts (n=24)	p-value
Hypertension	18 (69.2%)	13 (54.2%)	0.27
Diabetes mellitus	15 (57.7%)	9 (37.5%)	0.16
Hyperlipidaemia	12 (46.2%)	8 (33.3%)	0.35
Smoking	18 (69.2%)	10 (41.7%)	0.048
Alcohol use	11 (42.3%)	7 (29.2%)	0.32
Atrial fibrillation	8 (30.8%)	3 (12.5%)	0.11
Previous TIA/stroke	6 (23.1%)	3 (12.5%)	0.33

[Table/Fig-4]: Association between major vascular risk factors and brainstem involvement in posterior circulation stroke.

Variables	Adjusted Odds Ratio (AOR)	95% Confidence Interval (CI)	p-value
Age (per year increase)	1.03	0.99 - 1.08	0.11
Gender- Male	1.42	0.52 - 3.87	0.49
Hypertension	1.86	0.64 - 5.38	0.25
Diabetes mellitus	2.14	0.78 - 5.84	0.13
Hyperlipidaemia	1.58	0.55 - 4.53	0.39
Smoking	3.21	1.09 - 9.42	0.034*
Alcohol consumption	1.37	0.46 - 4.04	0.56
Atrial fibrillation	2.76	0.79 - 9.61	0.11
Previous TIA/Stroke	1.98	0.53 - 7.38	0.31

[Table/Fig-5]: Multivariable logistic regression analysis determining the independent predictors of brainstem involvement in PCS (N=50).

on univariate analysis (p=0.048) and remained independently associated after multivariable adjustment (AOR: 3.21; 95% CI: 1.09-9.42; p=0.034). Hypertension, diabetes mellitus, hyperlipidaemia, atrial fibrillation and previous stroke showed AORs greater than 1; however, the corresponding 95% CIs included 1 and the associations were not statistically significant. The regression model showed moderate explanatory power (Nagelkerke R^2=0.34) and an acceptable goodness-of-fit (Hosmer–Lemeshow p=0.62).

DISCUSSION

The PCS accounts for approximately 20 to 25% of all ischemic strokes. The non specificity of the clinical patterns is due to the

extensive and functional territories supplied by the vertebrobasilar system. The complex vascular anatomy, including the collateral circulation and anatomical variations, demonstrated the clinical severity. The clinical patterns and identification of key vascular risk factors was supremely associated with PCS.

On evaluating age, the average age was identified to be 58 years, with the majority of patients being middle-aged to elderly, with a predominance of the male population. This finding aligns with the previous findings by Amin-Hanjani S et al., who identified that PCS was more prevalent in older patients with vascular co-morbidities [9]. The reason behind the association between age and PCS is that advancing age is a non modifiable risk factor. As age progresses, there will be atherosclerotic changes in the arteries with arterial stiffening accompanied by endothelial dysfunction, which is more susceptible to ischaemia [11]. The male population is attributed to the higher risk of PCS, especially in India, owing to cultural differences. Men are more involved in deleterious habits such as alcohol consumption or smoking, whereas women are not permitted to do so. Although there are now changing trends in these cultural differences, one other major reason is hormonal differences, which offer more protection for females than males. More oestrogen exerts increased vasoprotective effects, particularly in premenopausal women [15,16].

The present study identified the most frequently observed risk factors in the study cohort as hypertension (62%), smoking (56%), diabetes mellitus (48%), and hyperlipidaemia (40%). Similar findings have been reported by Mehndiratta MM et al., in a prospective study of posterior circulation ischemic stroke, in which hypertension was observed in 63.75% of patients, highlighting its central role in the pathogenesis of vertebrobasilar Ischaemia [17]. O'Donnell MJ et al., found hypertension as the leading risk factor, followed by smoking, diabetes and dyslipidemia in ACS and PCS [18]. Chronic hypertension causes structural changes in the cerebral vessels because of elevated pressure on the walls of the vessels. This damages the vascular endothelium, promoting plaque formation and paving the way for ischaemic events.

Atrial fibrillation was observed in 22% of the study patients, and this implies that cardioembolic processes play a major role in PCS. Another notable cause of ischaemic stroke in the posterior circulation has been identified as cardioembolism, yet the prevalence can differ according to various populations. Sposato LA et al., found that atrial fibrillation is a significant aetiology for embolic stroke in posterior circulation territories and concluded that it is often underdiagnosed in stroke patients [19]. This could be because of blood stasis by ineffective arterial contractions. This promotes the chance for thrombus formation that can embolise in the vertebrobasilar system. The direct embolic occlusion of the cerebral arteries further activates ischaemic events [1].

Nausea, vomiting and giddiness or vertigo were the most common symptoms in the current study. The same was observed in previous studies, which have identified vestibular symptoms such as vertigo, dizziness, imbalance, and nausea as frequent clinical manifestations due to involvement of the brainstem, cerebellum and vestibular pathways [12,14]. These symptoms are indicative of damage to the posterior circulation-supplied organs of the vestibular pathways and cerebellar structures. Also, the present study identified motor weakness, cerebellar ataxia and dysarthria as a clinical presentation of PCS, but least common. Similar observations have been reported in previous studies, which demonstrated that PCS may present with a combination of cerebellar signs, brainstem dysfunction, and long-tract neurological deficits depending on the site and extent of infarction. Cranial nerve involvement, including diplopia and dysphagia, further supports the frequent involvement of brainstem structures in PCS [7].

On neuroimaging, brainstem involvement was the most common anatomical pattern, occurring in 26 (52%) patients, followed by cerebellar infarction in 11 (22%), occipital lobe infarction in 8 (16%), and thalamic infarction in 5 (10%). Among the brainstem lesions, pontine infarction was observed in 15 (30%) patients. Similar patterns have been reported in the New England Medical Center Posterior Circulation Registry (NEMC-PCR), which demonstrated that the brainstem and cerebellum are among the most frequently affected regions in posterior circulation ischaemic stroke. The predominance of brainstem involvement observed in the present study is therefore consistent with the established topographical distribution of vertebrobasilar ischaemia reported in large registry-based studies [20]. Involvement of the brainstem is a known characteristic of vertebrobasilar stroke because the basilar and its branches serve essential areas like the pons, medulla and midbrain. Amin-Hanjani S et al., in their two studies found on vertebrobasilar disease, also revealed that the usual causes of Ischaemia in the brainstem region are the stenosis or occlusion of the vertebral or basilar arteries [9,10]. Also, in this study, patients had large-vessel engagement of the vertebrobasilar circulation was seen. As studied by Dinc Y et al., structural abnormalities or atherosclerotic alterations in vertebral and basilar arteries are significant contributors to the occurrence of posterior circulation Ischaemia [21]. One plausible reason for this pattern is the anatomical vulnerability. Basilar and vertebral arteries have small perforating branches, which are the end arteries with limited collateral circulation. Occlusion directly causes reduced perfusion of the brainstem, leading to Ischaemia [22].

Analysis of this current study exhibited hypertension and diabetes mellitus to be frequently observed in patients with brainstem infarction, yet, similarly, the correlation was not significant. Smoking was observed to have a significant association with brainstem infarction compared to posterior circulation infarct types. The multivariate logistic regression analysis exhibited smoking as a standout predictor of brainstem infarction with an aOR of 3.21 (95% CI: 1.09 to 9.42). This observation underscores the pivotal role of modifiable lifestyle factors in determining patterns of strokes. Smoking promotes hypercoagulability and platelet aggregation, increasing the risk of thrombus formation and subsequent ischaemic events. Furthermore, existing literature has focused on the critical importance of vascular risk factors in mitigating the burden of ischaemic stroke.

The predominance of vertigo, nausea, and vomiting observed in the present study highlights the diagnostic challenges associated with PCS. These symptoms should not be overlooked, as they may represent early manifestations of vertebrobasilar Ischaemia. Because such presentations closely resemble benign vestibular disorders, including Benign Paroxysmal Positional Vertigo (BPPV), vestibular neuritis and labyrinthitis, PCS may be initially misdiagnosed, resulting in delayed recognition and treatment. Early identification of vascular risk factors, particularly hypertension, smoking, diabetes mellitus, and atrial fibrillation, may facilitate timely diagnosis and appropriate management. Therefore, a comprehensive evaluation of demographic characteristics, vascular risk factors, clinical manifestations, and neuroimaging findings is essential for improving diagnostic accuracy, guiding preventive strategies, and optimising clinical outcomes in patients with PCS.

Limitation(s)

The present study has certain limitations. It was conducted at a single tertiary care centre with a relatively small sample size, which may limit the generalisability of the findings. The use of convenience sampling may have introduced selection bias, and the cross-sectional design precludes causal inferences. Standardised stroke severity and functional outcome measures such as NIHSS and mRS were not assessed, as the study primarily focused on clinical characteristics and vascular risk factors. In addition, long-term outcomes and recurrence rates were not evaluated, and paroxysmal atrial fibrillation may have been under-detected due to the lack of uniform prolonged

cardiac monitoring in all patients. Although family history information was collected, it was not included in the risk factor analysis because the data were self-reported and inconsistently documented across participants, limiting their reliability. Therefore, family history was not included in the final analysis.

CONCLUSION(S)

Data from the current cohort reveals a high-risk clinical and vascular profile attributed to the PCS. Middle-aged to elderly men with evident cardiovascular risk factors are disproportionately affected. Hypertension, smoking, diabetes mellitus, hyperlipidaemia, alcohol consumption and atrial fibrillation were commonly observed among the study participants; however, only smoking demonstrated a statistically significant association within this cohort. The existence of atrial fibrillation underscores the multifactorial nature of the disease. Targeted preventive strategies, including lifestyle modifications, minimise the burden, morbidity and mortality linked with PCS, thereby improving quality of life. Future prospective multicentre studies incorporating larger cohorts, detailed vascular imaging, stroke severity measures and long-term outcome assessments are needed to better elucidate the mechanisms, predictors and prognostic significance of PCS and to guide evidence based prevention and management strategies.

Authors' contribution: AV: Conceptualisation, study design, data collection, data analysis and manuscript preparation; CTM: Study supervision, interpretation of data and critical revision of the manuscript; JPK: Data collection, literature review and manuscript preparation; MP: Study supervision, critical review and final approval of the manuscript.

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