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Original Research Article

Cerebral small vessel disease in an Indian cohort: magnetic resonance imaging burden, clinical profiles and risk factor associations

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ABSTRACT

Background: Cerebral small vessel disease (CSVD) is a major contributor to stroke, cognitive impairment, gait disturbances, and disability. Given its high burden and multifaceted clinical impact, CSVD has emerged as a critical public health concern. There is limited understanding of how radiological markers of CSVD correlate with clinical manifestations in Indian populations, which may differ from Western cohorts due to genetic, lifestyle, and environmental influences.

Methods: This observational, cross-sectional study included 85 adults with magnetic resonance imaging (MRI) evidence of at least one CSVD marker. Clinical assessment included cognition, gait, Parkinsonism, vascular risk factors, and stroke subtypes. MRI markers were classified per STRIVE criteria, and cumulative burden quantified using the total SVD score. Associations between radiological markers and clinical outcomes were evaluated using appropriate statistical tests.

Results: Lacunes (76.5%) and WMH (75.3%) were the most frequent markers, followed by CMB (42.4%), PVERSUS (30.6%), and RSSI (14.1%). Dementia was significantly more common in patients with lacunes (26.1% versus 5%; p=0.043) and WMH (25.6% versus 4.7%; p=0.034). CMB were strongly associated with Parkinsonism (36.1% versus 14.3%; p=0.019) and gait abnormalities (27.8% versus 10.2%; p=0.036). Higher SVD scores correlated with cerebral atrophy (68.9% versus 32.5%; p=0.001) and greater clinical impairment. Acute infarcts clustered with RSSI (50% versus 21.9%) and high SVD burden.

Conclusion: This study demonstrates substantial CSVD burden in an Indian cohort and highlights strong clinical–radiological correlations. Findings reinforce CSVD as a whole-brain, cumulative microvascular disorder with significant cognitive and motor implications.

Keywords: Cerebral small vessel disease, MRI markers, Cognitive impairment

INTRODUCTION

Cerebral small vessel disease (CSVD) is a major cause of stroke, cognitive decline, gait impairment, mood disturbances, and functional dependency worldwide.[1] Indian magnetic resonance imaging (MRI) studies show that 30–35% of asymptomatic adults have at least one CSVD marker, and the prevalence rises to 50–80% in stroke and cognitive impairment cohorts.[2] CSVD involves pathological processes affecting perforating arterioles, capillaries, and venules, leading to chronic ischemia, endothelial dysfunction, vessel wall thickening, and impaired autoregulation.[1]

Blood–brain barrier (BBB) disruption and inflammation are key mechanisms linking vascular injury to white matter damage.[3] Neuroimaging features, standardized under STRIVE criteria, include lacunes, white matter hyperintensities (WMH), cerebral microbleeds (CMB), enlarged perivascular spaces (EPVERSUS), and recent small subcortical infarcts (RSSI).[4] WMH indicate chronic ischemic demyelination, lacunes are healed infarcts, and CMB signals suggest small-vessel fragility.[5]

Modern neuroimaging shows CSVD as a whole-brain network disease, not focal.[6] WMH, lacunes, and CMB disconnect large networks, affecting executive function, attention, gait, and mood.[6] Microstructural issues go beyond visible lesions into normal white matter, with diffuse cerebrovascular reactivity impairment, supporting CSVD as a system-wide vascular pathology.[7,8]

Age and hypertension are the main factors of CSVD burden. Midlife hypertension speeds up WMH progression and cognitive decline, with diabetes, dyslipidaemia, smoking, and kidney disease also involved. Persistent endothelial dysfunction, BBB leakage, and impaired glymphatic clearance may cause CSVD to progress despite good risk management.[9,10]

Despite global research, data from India are limited. India has high hypertension, diabetes, and early vascular aging, with stroke occurring a decade earlier than in Western populations.[11] There is a lack of population-specific CSVD characterization, despite rising dementia and cerebrovascular disease. Understanding CSVD features in Indian cohorts is crucial for early diagnosis, risk assessment, and prevention of long-term disability.[12] This study aims to evaluate the radiological spectrum of CSVD using MRI, assess clinical–radiological correlations, such as cognitive and gait associations, evaluate risk factors associated with CSVD markers, and integrate findings into modern CSVD pathophysiology, highlighting its whole-brain disease nature.

METHODS

Study design

This was a cross-sectional, observational study conducted in the Department of Neurology at Indraprastha Apollo Hospital, New Delhi. Patients presenting with clinical features suggestive of CSVD and undergoing MRI brain evaluation were consecutively recruited over the study period. The study adhered to institutional ethics guidelines, and written informed consent was obtained from all participants.

Eligibility criteria

Patients aged ≥18 years with either sex, patients with MRI brain showing at least one CSVD marker (lacunes, white matter hyperintensities, cerebral microbleeds, enlarged perivascular spaces, or recent small subcortical infarcts), and clinically relevant symptoms (stroke/TIA, cognitive impairment, gait abnormalities, neurobehavioral symptoms) were included in this study.

Patients with evidence of large-vessel territorial infarcts, intracerebral hemorrhage >1.5 cm, CNS infections, demyelinating disorders, neurodegenerative diseases, vasculitis, or autoimmune encephalopathies, intracranial tumors, or structural lesions unrelated to CSVD, and patients with severe motion artifacts limiting interpretation were excluded from this study.

Study method

All patients underwent detailed neurological evaluation including history, vascular risk factor profiling, and comprehensive physical and neurological examination. Specific data recorded included; demographics details including age, sex, duration and type of presenting symptoms, vascular risk factors like hypertension, diabetes, dyslipidaemia, smoking, ischemic heart disease, renal dysfunction, and history of prior stroke, blood pressure and anthropometric parameters, cognitive profile; assessed via screening tools, gait abnormalities, falls, balance impairment, and stroke syndromes, categorized using established clinical criteria for lacunar stroke presentations.

Neuroimaging protocol

All participants underwent MRI brain on a 1.5T/3T scanner. The protocol included; T1-weighted imaging, T2-weighted imaging, (fluid-attenuated inversion recovery (FLAIR), susceptibility-weighted imaging (SWI) for microbleeds, diffusion-weighted imaging (DWI), ADC maps, and gradient echo sequences (if applicable).

Images were interpreted jointly by two experienced radiologists/neurologists blinded to clinical data. Any discrepancies were resolved by consensus, and if disagreement persisted, a third senior reviewer adjudicated.

CSVD markers (STRIVE criteria)

Imaging findings were categorized according to published STRIVE standards.

White matter hyperintensities (WMH)

Graded using Fazekas scores (periventricular and deep WMH). Classified as mild, moderate, or severe.

Lacunes

Round/ovoid lesions 3–15 mm in diameter, CSF-like intensity, located in deep grey/white matter.

Cerebral microbleeds (CMB)

Hypointense lesions ≤10 mm on SWI; categorized by location (deep, lobar, infratentorial).

Perivascular spaces (PVERSUS)

Linear/round structures <3 mm; graded in basal ganglia and centrum semiovale.

Recent small subcortical infarcts (RSSI)

Acute lacunar infarcts confirmed on DWI/ADC.

Total SVD score

Each of the following received one point to generate a 0–4 composite SVD score; presence of lacunes, presence of ≥1 microbleed, moderate–severe basal ganglia PVERSUS, and severe WMH (Fazekas 2–3 PVWM or 3 DWM).

This composite score was used to capture cumulative CSVD burden.

Laboratory investigations

Standard laboratory tests were performed, including fasting blood glucose, HbA1c, lipid profile, renal function tests, complete blood counts, and thyroid function tests.

Endpoints

The primary endpoint was to assess the radiological burden like frequency and severity of individual CSVD markers.

The secondary endpoint was to assess the clinical features like cognitive impairment, gait abnormalities, and lacunar stroke subtypes. The risk factor associations like hypertension, diabetes, dyslipidaemia, smoking, and age were also assessed.

Statistical analysis

Data were analyzed using statistical package for the social sciences (SPSS) (version as in thesis). Continuous variables were shown as mean±SD, categorical as percentages. T-tests compared groups for continuous data; chi-square for categorical; ANOVA for multiple radiological severity categories. P<0.05 was significant. Effect sizes, correlations, and stratified analyses examined associations between CSVD markers and clinical variables, but no multivariate modeling was done due to cohort size.

RESULTS

Demographic and clinical profile

The study included 85 patients with a mean age of 69.6 years and a male predominance of 62.4%. The average body mass index was 27.8 kg/m^2. Regarding comorbidities, diabetes mellitus was present in 55.3% of the cohort and hypertension in 50.6%. A history of previous ischemic stroke was noted in 11.8%, and previous hemorrhagic stroke in 2.4% of patients.

In terms of clinical presentation, 38.8% of patients presented with a cerebrovascular accident. Parkinsonism was identified in 23.5%, dementia in 21.2%, and gait abnormalities in 17.6%.

Neuroimaging demonstrated a substantial burden of small vessel disease burden. White matter hyperintensities were common: 49.4% had Fazekas grade 1, 23.5% grade 2, and 2.4% grade 3. Lacunes were most frequent, in 76.5%. Recent small subcortical infarcts occurred in 14.1%, microbleeds in 42.4%, and perivascular spaces in 30.6%. The small vessel disease score distribution was: 15.3% scored 1, 31.8% scored 2, and 30.6% scored 3. Cerebral atrophy was in 51.8%. Acute findings included hemorrhages in 4.7% and infarcts in 25.9%. Data are summarized in Table 1.

White matter hyperintensities and associated clinical profile

Compared with patients without white matter hyperintensities (WMH), those with WMH had dementia significantly more frequently, occurring in 26.56% of patients with WMH compared to 4.76% without WMH (p=0.034). Cerebral atrophy also showed a strong association with WMH, present in 67.19% of patients with WMH versus only 4.76% without (p<0.001).

White matter hyperintensities and associated clinical profile

Compared with patients without WMH, those with WMH had dementia significantly more frequently, occurring in 26.56% of patients with WMH compared to 4.76% without WMH (p=0.034). Cerebral atrophy also showed a strong association with WMH, present in 67.19% of patients with WMH versus only 4.76% without (p<0.001). Other clinical variables showed expected trends but did not reach significance. Parkinsonism was in 25% of WMH patients and 19.05% without WMH (p=0.577). Gait abnormalities occurred in 18.75% versus 14.29% (p=0.641), depression in 10.94% versus 4.76% (p=0.400). Cerebrovascular accident history was in 35.94% with WMH and 47.62% without WMH (p=0.341). Lifestyle factors were higher in WMH group: smoking in 28.13% versus 9.52% (p=0.081), alcohol abuse in 28.13% versus 19.05% (p=0.41), not statistically significant. Large-artery atherosclerosis was more in patients without WMH (38.10%) than with WMH (18.75%), but not significant (p=0.070). These data are summarized in Table 2.

Lacunes and associated clinical profile

Patients with lacunes had higher dementia rates (26.15% versus. 5%, p=0.043) and were more likely to smoke (29.23% versus. 5%, p=0.025) than those without lacunes. Cerebral atrophy (53.85% versus. 45%) and acute infarcts

(27.69% versus. 20%) were more common in lacune-positive patients. Large-artery atherosclerosis was also more frequent in lacune-positive patients (27.69% versus. 10%), though not statistically significant. Details are in Table 3.

Cerebral microbleeds and associated clinical profile

Parkinsonism showed a significant association with the presence of CMB, occurring in 36.1% of patients with CMB compared with 14.3% of those without (p=0.019). Gait abnormalities were also significantly more frequent among individuals with CMB, seen in 27.8% of CMB-positive patients versus 10.2% of those without CMB (p=0.036). Cerebral atrophy demonstrated one of the strongest associations, occurring in 72.2% of patients with CMB, compared with 36.7% of those without CMB (p=0.001). This represents a large and statistically robust difference, suggesting a strong relationship between microbleed burden and structural brain degeneration. These data are summarized in Table 4.

SVD score and associated clinical profile

Upon comparison of patients with low SVD burden (score ≤2) and high SVD burden (score >2), cerebral atrophy showed the strongest association, occurring in 68.9% of patients with higher SVD scores compared with 32.5% of those with lower scores (p=0.001). This represents a highly significant and clinically robust relationship, indicating that structural brain degeneration increases substantially with cumulative SVD burden. Dementia demonstrated a notable upward trend in the higher SVD group, occurring in 28.9% of patients with SVD scores >2 compared with 12.5% of those with scores ≤2, representing a large absolute difference even though statistical significance was not reached (p=0.065). Smoking also showed a meaningful difference, being more frequent among patients with higher SVD burden (31.1% versus 15%), consistent with vascular risk clustering, although not statistically significant (p=0.080). These data are summarized in Table 5.

Recent small subcortical infarct and associated clinical profile

Under the category RSSI, comparison of patients with and without RSSI (yes=12; no=73) revealed several clinically meaningful differences despite the absence of statistically significant p values. Cerebral atrophy showed one of the most prominent differences, being present in 75% of patients with RSSI compared with 47.9% of those without RSSI, suggesting a substantial association between RSSI and global brain tissue loss. Acute infarcts were also notably more common in the RSSI-positive group (50%) than in the RSSI-negative group (21.9%), reflecting a large absolute difference and indicating that RSSI clusters with other acute ischemic events.

Large-artery atherosclerosis was significantly more common among patients with RSSI (41.7%) than among those without (20.5%), suggesting a potential interaction between small- and large-vessel pathology. Dementia occurred in 33.3% of patients with RSSI versus 19.2% of those without, representing a sizeable difference and suggesting that RSSI may contribute to cognitive impairment through disruption of strategic white-matter and subcortical circuits. Previous ischemic stroke also appeared more frequent in the RSSI-positive group (25%) compared with the RSSI-negative group (9.6%), indicating a possible cumulative vascular burden. Smoking showed a higher prevalence among RSSI patients (33.3%) compared with those without (21.9%), consistent with established microvascular pathology risks. These data are summarized in Table 6.

Perivascular space and associated clinical profile

Upon comparison of patients with and without enlarged perivascular spaces (PVERSUS), dementia was significantly more common among patients with PVERSUS, occurring in 34.6% of the PVERSUS-positive group compared with 15.3% of those without PVERSUS (p=0.044). This represents the only statistically significant association in this subgroup. Several other variables demonstrated large numerical differences that, although statistically non-significant, remain clinically relevant in the context of cerebral small vessel disease.

Cerebral atrophy showed a notably higher frequency among individuals with PVERSUS, present in 65.4% versus 45.8% in those without PVERSUS, suggesting an important association between PVERSUS burden and global neurodegeneration. Alcohol abuse also appeared more common among PVERSUS-positive patients (34.6%) compared with PVERSUS-negative patients (22%), reflecting a clinically meaningful upward trend. Acute infarcts were identified in 26.9% of those with PVERSUS versus 25.4% without PVERSUS, indicating a slight but relevant elevation. Smoking prevalence was also somewhat higher in the PVERSUS group (26.9%) compared with the non-PVERSUS group (22%), consistent with vascular risk clustering. These data are summarized in Table 7.

Table 1: Demographic characteristics of patients.

Parameters		Mean (SD)/N (%)
Age (years)		69.6 (11.3)
Sex	Male	53 (62.4)
	Female	32 (37.6)
Blood pressure	SBP	137.7 (19.6)

Continued.

Parameters		Mean (SD)/N (%)
	DBP	80.8 (13.0)
BMI (kg/m²)		27.8 (3.5)
Comorbidities	Diabetes	47 (55.3)
	Hypertension	43 (50.6)
	Previous hemorrhagic stroke	2 (2.4)
	Previous ischemic stroke	10 (11.8)
	Coronary artery disease	19 (22.4)
	Chronic kidney disease	5 (5.9)
	Alcohol abuse	22 (25.9)
	Smoking	20 (23.5)
Clinical profile	Cerebrovascular accident	33 (38.8)
	Parkinson	20 (23.5)
	Dementia	18 (21.2)
	Gait abnormality	15 (17.6)
	Depression	8 (9.4)
	Asymptomatic	1 (1.1)
	Other	29 (34.1)
MRI features	WMH (Fazekas grade 1)	42 (49.4)
	WMH (Fazekas grade 2)	20 (23.5)
	WMH (Fazekas grade 3)	2 (2.4)
	Lacunas	65 (76.5)
	Recent small subcortical infarct	12 (14.1)
	Cerebral microbleeds	36 (42.4)
	Perivascular space	26 (30.6)
SVD score	1	13 (15.3)
	2	27 (31.8)
	3	26 (30.6)
	4	16 (18.8)
	5	3 (3.5)
	Cerebral atrophy	44 (51.8)
	Acute hemorrhage	4 (4.7)
	Acute infarct	22 (25.9)

SBP: Systolic blood pressure, DBP: diastolic blood pressure, BMI: body mass index, MRI: magnetic resonance imaging, WMH: white matter hyperintensities, SVD: small vessel disease

Table 2: White matter hyperintensities and associated clinical profile.

Variable	WMH: yes (64) (%)	WMH: no (21) (%)	P value
Dementia	17 (26.56)	1 (4.76)	0.034
Depression	7 (10.94)	1 (4.76)	0.400
Parkinsonism	16 (25.00)	4 (19.05)	0.577
Gait Abnormality	12 (18.75)	3 (14.29)	0.641
Cerebrovascular accident	23 (35.94)	10 (47.62)	0.341
Previous ischemic stroke	9 (14.06)	1 (4.76)	0.251
Coronary artery disease	16 (25.00)	3 (14.29)	0.306
RWMA	22 (34.38)	4 (19.05)	0.186
Diabetes	34 (53.12)	13 (61.9)	0.483
Hypertension	34 (53.12)	09 (42.85)	0.414
Smoking	18 (28.13)	2 (9.52)	0.081
Alcohol abuse	18 (28.13)	4 (19.05)	0.41
Cerebral atrophy	43 (67.19)	1 (4.76)	<0.001
Acute infarct	13 (20.31)	9 (42.86)	0.041
Large artery atherosclerosis	12 (18.75)	8 (38.10)	0.070

WMH: White matter hyperintensities, RWMA: regional wall motion abnormality

Table 3: Lacunes and associated clinical profile.

Variable	Lacunes: yes (65) (%)	Lacunes: no (20) (%)	P value
Dementia	17 (26.15)	1 (5)	0.043
Depression	08 (12.31)	0 (0)	0.189
Parkinsonism	16 (24.62)	4 (20)	0.771
Gait abnormality	12 (18.46)	3 (15)	1.000
Cerebrovascular accident	24 (36.92)	9 (45)	0.517
Previous ischemic stroke	06 (9.23)	4 (20)	0.236
Coronary artery disease	13 (20)	6 (30)	0.368
Diabetes	38 (58.46)	9 (45)	0.290
Hypertension	33 (50.76)	10 (50)	0.952
Smoking	19 (29.23)	1 (5)	0.025
Alcohol abuse	19 (29.23)	3 (15)	0.204
Cerebral atrophy	35 (53.85)	9 (45)	0.489
Acute infarct	18 (27.69)	4 (20)	0.492
Large artery atherosclerosis	18 (27.69)	2 (10)	0.379

Table 4: Cerebral microbleeds and associated clinical profile.

Variable	CMB: yes (36) (%)	CMB: no (49) (%)	P value
Dementia	10 (27.8)	08 (16.3)	0.202
Depression	03 (8.3)	05 (10.2)	1.000
Parkinsonism	13 (36.1)	07 (14.3)	0.019
Gait abnormality	10 (27.8)	05 (10.2)	0.036
Cerebrovascular accident	10 (27.8)	23 (46.9)	0.073
Previous ischemic stroke	03 (8.3)	07 (14.3)	0.507
Coronary artery disease	07 (19.4)	12 (24.5)	0.581
Diabetes	21 (58.3)	26 (53.1)	0.629
Hypertension	19 (52.7)	24 (48.9)	0.729
RWMA	13 (36.1)	13 (26.5)	0.344
Smoking	10 (27.8)	10 (20.4)	0.429
Alcohol abuse	11 (30.6)	11 (22.4)	0.399
Cerebral atrophy	26 (72.2)	18 (36.7)	0.001
Acute infarct	06 (16.7)	16 (32.7)	0.096
Large artery atherosclerosis	10 (27.8)	10 (20.4)	0.429

CMB: Cerebral microbleeds, RWMA: regional wall motion abnormality

Table 5: SVD score and associated clinical profile.

Variable	SVD score ≤2 (40) (%)	Variable	P value
Dementia	05 (12.5)	13 (28.9)	0.065
Depression	04 (10.0)	04 (8.9)	1.000
Parkinsonism	08 (20.0)	12 (26.7)	0.470
Gait abnormality	06 (15.0)	09 (20.0)	0.546
Cerebrovascular accident	17 (42.5)	16 (35.6)	0.512
Previous ischemic stroke	04 (10.0)	06 (13.3)	0.743
Coronary artery disease	11 (27.5)	08 (17.8)	0.283
Diabetes	21 (52.5)	26 (57.7)	0.625
Hypertension	19 (47.5)	24 (53.3)	0.591
RWMA	10 (25.0)	16 (35.6)	0.292
Smoking	06 (15.0)	14 (31.1)	0.080
Alcohol abuse	08 (20.0)	14 (31.1)	0.243
Cerebral atrophy	13 (32.5)	31 (68.9)	0.001
Acute infarct	11 (27.5)	11 (24.4)	0.748
Large artery atherosclerosis	08 (20.0)	12 (26.7)	0.470

DISCUSSION

This study provides an integrated clinical–radiological characterization of CSVD in an Indian tertiary-care neurology population. By examining patients with symptomatic CSVD, this study demonstrated a high prevalence of radiological markers, particularly lacunes (76.5%) and WMH (75.3%), along with relevant clinical manifestations, including cognitive impairment, Parkinsonism, gait dysfunction, and lacunar stroke syndromes. These findings align with international literature and reinforce the concept of CSVD as a progressive, diffuse, whole-brain disease.

This study found strong associations between cognitive impairment and both lacunes and WMH. Patients with lacunes had significantly higher dementia prevalence (26.1% versus 5%), while WMH was associated with both dementia (25.6% versus 4.7%) and reduced MMSE scores (24.9 versus 27.7). These findings are consistent with global data indicating that WMH and lacunes, particularly when confluent or strategically located, disrupt frontal and subcortical networks underlying executive function and information processing speed.[13] Tuladhar et al demonstrated that WMH burden correlates with global structural network inefficiency, which in turn strongly predicts cognitive decline.[14] Likewise, lacunes in the basal ganglia and thalamus impair attentional control and psychomotor speed.[15] This study confirms these mechanisms in an Indian cohort, emphasizing the cumulative cognitive effects of mixed SVD markers.

Parkinson was present in 23.5% of patients and was significantly associated with CMB (36.1% versus 14.3%). CMB was also associated with gait abnormalities (27.8% versus 10.2%). These findings are physiologically plausible. Microbleeds in the basal ganglia and brainstem may disrupt motor pathways and predispose to gait freezing, bradykinesia, and postural instability.[16] Sundaresan et al. reported that CMB reflect vessel fragility and localized hemosiderin deposition in motor-regulating nuclei.[17] Nearly 39% of the cohort presented with cerebrovascular events. Lacunes, WMH, and CMB were especially common in patients with acute infarcts or TIA. This aligns with existing literature describing CSVD as a major cause of lacunar stroke and a contributor to hemorrhagic risk.[18] Vermeer et al. emphasized that silent infarcts frequently seen in CSVD, strongly predict future symptomatic stroke.[19]

Smoking emerged as a significant risk factor for lacunes (29.2% versus 5%). Smoking accelerates endothelial dysfunction, oxidative stress, and microvascular wall damage, explaining its association with lacunar infarction.[20] The ARIC-MRI study demonstrated that a higher number of smoking pack-years was associated with a greater risk of WMH progression, whereas time since quitting and age at smoking initiation showed no significant association with WMH progression.[21]

Modern neuroimaging research underscores that CSVD affects not only discrete regions but the entire structural and functional connectome.[22] Dementia was significantly more frequent in patients with lacunes (26.1% versus 5%) and WMH (25.6% versus 4.7%), and WMH was associated with lower MMSE scores (24.9 versus 27.7). Motor dysfunction showed a stronger relationship with CMB than WMH, with Parkinsonism (36.1% versus 14.3%) and gait abnormality (27.8% versus 10.2%) significantly more common in patients with microbleeds. Stroke patterns also reflected cumulative microvascular injury, with acute infarcts more frequent in patients with RSSI (50% versus 21.9%) and high SVD scores (50% for mSVD >2). Collectively, these findings reinforce CSVD as a whole-brain disease, in which cumulative microvascular pathology better explains clinical deficits than isolated lesions.

Limitations

This study had several notable limitations. First, being a single-centre study from a tertiary care hospital, the findings may not be generalizable to broader community. The modest sample size (n=85) limited the ability to perform robust multivariate modelling and reduced the statistical power to detect weaker associations. Cognitive assessment was based solely on the mini-mental state examination (MMSE), which, although practical, lacks sensitivity for detecting subtle executive dysfunction or early cognitive decline commonly seen in CSVD, thereby introducing potential measurement bias. Similarly, MRI-based classification of CSVD markers was extracted from clinical reports, and the absence of dual or blinded image reading raises the possibility of reader-dependent variability and misclassification bias, particularly for subjective components such as Fazekas WMH grading, identification of lacunes versus enlarged perivascular spaces, and quantification of cerebral microbleeds. Such measurement biases may have diluted or exaggerated some associations.

Strengths

CSVD appears particularly relevant to India due to the region's high prevalence of hypertension and early vascular aging, rising dementia burden, high rates of uncontrolled diabetes, and increased stroke incidence in midlife. This study fills an important gap by presenting systematic CSVD characterization in an Indian cohort. Early identification of high SVD burden, especially WMH grade, microbleeds, and composite scores, may help in proactive management of vascular risk factors to prevent cognitive and motor decline.

Research gaps and future scope

This study identifies research gaps, such as the need for longitudinal, multi-centre Indian cohorts to establish causal pathways and improve generalizability, since the current cross-sectional design limits temporal inferences.

Standardized, blinded imaging and advanced MRI techniques are needed to reduce bias and improve diagnosis. More sensitive cognitive and neuropsychiatric tests are required to better characterize CSVD-related deficits beyond MMSE and PHQ-9. Future research should explore biomarkers, endothelial dysfunction, and metabolic factors specific to India, and validate SVD scores for prognosis. Larger, rigorous studies combining imaging, clinical data, and interventions are essential to advance CSVD research in India.

CONCLUSION

This study demonstrates a high burden of CSVD markers, particularly lacunes, white matter hyperintensities, and cerebral microbleeds in an Indian symptomatic cohort and identifies robust clinical–radiological associations with cognitive impairment, Parkinsonism, gait dysfunction, and overall SVD burden. These associations, while not implying causality due to the cross-sectional observational design, are consistent with the contemporary understanding of CSVD as a diffuse, whole-brain disorder influenced by vascular aging, hypertension, metabolic factors, and endothelial dysfunction. The study further suggests that composite SVD scoring may provide meaningful prognostic insight and could be considered in routine clinical evaluation, although confirmatory longitudinal studies are required. Overall, the findings underscore the importance of early detection, comprehensive vascular risk factor management, and the need for population-specific CSVD research in India to better characterize disease patterns and progression.

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