

Cerebral Small Vessel Disease Burden in Saudi Arabia: Epidemiology, Risk Determinants, and Neuroimaging Characteristics

Thamer S Alhawaish^{1,2}, Khalid Bin Aziz^{2,3}, Basel Al Anazi^{2,4}, Fahad Alhargan^{1,2}, Alaa Mathkour⁵, Elan Homoud^{2,4}, Amjad M Alakel^{2,3}, Eman Barjas Alotaibi^{2,3}, Nedaa AlAmri^{1,2}, Noura AlRaeesi^{2,4}, Sameeha Fallatah^{2,4}, Ajmal Anversha^{1,2}

¹Department of Neurology, King Abdulaziz Medical City, Ministry of National Guard Health Affairs, Riyadh, Saudi Arabia; ²King Abdullah International Medical Research Center, Riyadh, Saudi Arabia; ³College of Medicine, King Saud bin Abdulaziz University for Health Sciences, Riyadh, Saudi Arabia; ⁴Department of Radiology, King Abdulaziz Medical City, Ministry of National Guard Health Affairs, Riyadh, Saudi Arabia; ⁵Public Health Administration, Jazan Specialist Hospital, Jazan Health Cluster, Jazan, Saudi Arabia

Correspondence: Thamer S Alhawaish, Department of Neurology, King Abdulaziz Medical City, Ministry of National Guard Health Affairs, Riyadh, Saudi Arabia, Email Thamersa18@gmail.com

Background: Cerebral small vessel disease (CSVD) is a major contributor to stroke, cognitive impairment, and gait disturbance. Data describing its burden and determinants in Middle Eastern populations remain limited. This study aimed to estimate the prevalence, clinical predictors, and neuroimaging characteristics of CSVD among adults undergoing brain MRI in Saudi Arabia and to examine associations with vascular risk factors and antihypertensive therapy.

Methods: We conducted a retrospective cross-sectional study of adults aged 40–70 years who underwent brain MRI at King Abdulaziz Medical City, Riyadh, between January 2018 and January 2019. CSVD markers were identified according to the STAndards for ReportIng Vascular changes on nEuroimaging (STRIVE) criteria, and white matter hyperintensities were graded using the Fazekas scale. Multivariable logistic regression models were constructed to identify factors independently associated with CSVD.

Results: Among 644 participants undergoing brain MRI, CSVD was identified in 36.5% of patients. Prevalence increased markedly with age, rising from 18.2% in individuals aged 40–50 years to 54.1% in those aged 60–70 years ($p < 0.001$). In the multivariable etiological model, hypertension emerged as the strongest independent vascular predictor of CSVD. White matter hyperintensities were the most frequent imaging manifestation and increased in severity with advancing age. Among hypertensive patients, certain antihypertensive classes, particularly calcium channel blockers and ACE inhibitor/ARB monotherapy, were associated with lower odds of CSVD compared with untreated hypertension.

Conclusion: In this MRI-based clinical cohort from Saudi Arabia, CSVD was present in more than one-third of adults aged 40–70 years undergoing neuroimaging. Disease prevalence increased substantially with advancing age and vascular comorbidities. These findings highlight the importance of vascular risk factor control and provide important regional data on the clinical and imaging profile of CSVD in Middle Eastern populations.

Keywords: cerebral small vessel disease, lacunar infarction, white matter hyperintensities, diabetes mellitus, hypertension, stroke prevention, Saudi Arabia

Introduction

Cerebral small vessel disease (CSVD) encompasses a spectrum of pathological changes affecting the brain's small perforating vessels, including arterioles, capillaries, and venules. These microvascular alterations underlie a wide range of clinical outcomes such as cognitive decline, stroke, gait disturbances, and neuropsychiatric symptoms.^{1–3} CSVD is strongly linked to aging and chronic vascular conditions, particularly hypertension and diabetes, and represents a major cause of neurological morbidity and cognitive impairment in older adults.^{3–6}

Recent advances in high-resolution neuroimaging have substantially improved the *in vivo* characterization of CSVD, enabling detailed visualization of its structural manifestations. On brain magnetic resonance imaging (MRI), CSVD is characterized by a spectrum of imaging markers, including white matter hyperintensities (WMH), lacunes, cerebral microbleeds (CMBs), enlarged perivascular spaces, and brain atrophy.^{6–8} Among these, WMH represent one of the most prominent and widely studied features, with their severity commonly graded using the Fazekas scale. Serial MRI examinations further allow longitudinal assessment of lesion progression and provide insight into the dynamic nature of microvascular injury.^{8,9}

Despite these advances in imaging-based characterization, the underlying biological mechanisms driving CSVD remain incompletely understood. Growing evidence suggests that endothelial dysfunction, disruption of the blood–brain barrier, and perivascular inflammation play central roles in the pathogenesis of small-vessel injury and the development of CSVD-related brain changes.^{6,10,11}

Globally, CSVD is highly prevalent, affecting approximately 5% of individuals aged 50 years and nearly all those over 90 years.¹² Modifiable risk factors include hypertension (HTN), diabetes mellitus (DM), dyslipidemia (DLP), smoking, and prior cerebrovascular disease. In addition, genetic predisposition and lifestyle-related factors including physical inactivity and dietary patterns may further influence disease susceptibility and progression.^{11,12} Population-based studies have demonstrated substantial geographic variability in the prevalence and imaging manifestations of CSVD, likely reflecting differences in genetic background, vascular risk profiles, environmental exposures, and health-care systems. For example, community-based cohorts in China have reported CSVD imaging markers in approximately 30% of adults aged 50–75 years,¹³ Similarly, the Rotterdam Scan Study in Europe identified a high prevalence of silent brain infarcts and white matter lesions among older adults.¹⁴ In contrast, recent data from Colombia reported even higher rates of CSVD markers, approaching 56%, further underscoring the marked regional heterogeneity in the burden and expression of this disease.¹⁵

To date, no neuroimaging-based studies have reported the prevalence of CSVD in Saudi Arabia. However, regional stroke data suggest that small vessel pathology constitutes an important component of the local cerebrovascular disease burden. In a Saudi cohort of young adults with stroke, small vessel disease accounted for 31.7% of cases, while a hospital-based study found that lacunar stroke represented up to 35.5% of ischemic stroke presentations.^{16,17} These findings indirectly underscore the likely clinical significance of CSVD in the region.

Despite increasing recognition of CSVD as a major contributor to stroke, cognitive impairment, gait dysfunction, and functional decline, systematic data on its prevalence, risk factors, and neuroimaging characteristics in Middle Eastern populations remain scarce. Addressing this gap is essential for defining regional disease patterns, improving risk stratification, and informing targeted prevention strategies.

Accordingly, this study aimed to evaluate the prevalence, clinical predictors, and neuroimaging characteristics of CSVD among adults undergoing brain MRI at King Abdulaziz Medical City in Riyadh. By characterizing the clinical and radiological profile of CSVD in this cohort, we sought to identify modifiable vascular risk factors and contextualize Saudi data within the broader global landscape of CSVD research.

Methods

We conducted a retrospective cross-sectional study at King Abdulaziz Medical City (KAMC), Riyadh, Saudi Arabia, using data extracted from the electronic medical records (EMR) and the Picture Archiving and Communication System (PACS). The study included all adults aged 40–70 years who underwent brain magnetic resonance imaging (MRI) between January 1, 2018, and January 1, 2019, yielding a final cohort of 644 participants.

Eligible participants were adults aged 40–70 years at the time of MRI who had sufficient demographic, clinical, and radiological data available in the medical record. MRI studies were required to include the sequences necessary for the evaluation of CSVD markers according to the STandards for ReportIng Vascular changes on nEuroimaging (STRIVE) criteria. Imaging protocols included T1-weighted, T2-weighted, fluid-attenuated inversion recovery (FLAIR), and susceptibility-weighted imaging (SWI) or gradient-echo (GRE) sequences. WMH were assessed on FLAIR images and graded using the Fazekas scale, while CMBs were identified on SWI or GRE sequences depending on the scanner type, in accordance with STRIVE criteria.

Patients were excluded if they had conditions that could substantially confound the assessment of white matter abnormalities, including large intracranial tumors, demyelinating disorders with significant white matter involvement, infectious or inflammatory central nervous system diseases, genetic or metabolic neurological disorders, or incomplete MRI studies lacking essential sequences for CSVD evaluation. All MRI examinations were performed on 3-Tesla scanners at KAMC.

All scans were independently reviewed by two board-certified neuroradiologists who were blinded to the clinical data and assessed for CSVD markers according to STRIVE definitions. Any discrepancies were resolved by consensus. Formal inter-rater reliability measures were not calculated. CSVD was defined as the presence of at least one STRIVE-defined imaging marker, including WMH, lacunes, recent subcortical infarcts, or CMBs. Participants were subsequently classified as CSVD-positive or CSVD-negative according to the presence or absence of these imaging findings. Comparisons between groups were then performed to identify demographic and clinical factors associated with CSVD and to characterize the prevalence and severity of its neuroimaging manifestations.

Extracted variables included demographic characteristics (age, sex, body mass index, and smoking status), comorbidities (DM, HTN, DLP, malignancy, atrial fibrillation, coronary artery disease, congestive heart failure, chronic kidney disease (CKD), liver cirrhosis, patent foramen ovale, and prior stroke or transient ischemic attack), and neuroimaging findings including WMH, lacunar infarcts, recent subcortical infarcts, and CMBs. Glycemic control was assessed using hemoglobin A1c (HbA1c) values recorded in the electronic medical records. Clinical and laboratory variables were extracted based on measurements obtained closest to the MRI date. Laboratory values were included if available within one year prior to the MRI or up to six months after the MRI, with preference given to values closest to the imaging date. Controlled glycemia was defined as HbA1c <7.5%. Comorbidities were defined based on documented diagnoses in the medical record at the time of imaging.

Categorical variables were summarized as frequencies and percentages. Comparisons between CSVD-positive and CSVD-negative groups were conducted using the chi-square test or Fisher's exact test, as appropriate. Two binary logistic regression models were constructed to address the study's primary and secondary research objectives.

Model 1 (Primary Etiological Model) was designed to identify independent biological and demographic predictors of CSVD and was applied to all patients with complete data ($N = 428$). The model included age group (40–50, 51–60, and 61–70 years; reference: 40–50 years), sex, diabetes mellitus, hypertension, chronic kidney disease, dyslipidemia, congestive heart failure, coronary artery disease, history of stroke or transient ischemic attack, migraine, HbA1c control status, LDL control status, hemoglobin group, platelet group, and echocardiographic findings. Medications and anti-hypertensive therapies were intentionally excluded from this model to avoid collinearity with their corresponding disease diagnoses and to allow unbiased estimation of independent vascular risk factor effects.

Model 2 (Antihypertensive Class Model) was restricted to patients with hypertension ($n = 300$; 296 with complete data) and was constructed to evaluate whether antihypertensive medication class influences CSVD risk. Untreated hypertension served as the reference category. The model included age group, sex, diabetes mellitus, chronic kidney disease, dyslipidemia, history of stroke or transient ischemic attack, and antihypertensive medication class (calcium channel blocker (CCB) monotherapy, ACE inhibitor/ARB monotherapy, combined CCB + ACEI/ARB therapy, beta-blocker therapy, and diuretic therapy). Patients receiving dual antiplatelet therapy ($n = 20$) were excluded from multi-variable models because near-complete separation precluded stable logistic regression estimation; these patients are reported descriptively. Model fit was evaluated using the Hosmer–Lemeshow goodness-of-fit test and discriminative ability was assessed using the area under the receiver operating characteristic curve (AUC). Model explanatory power was quantified using the Nagelkerke R^2 statistic. All analyses were performed using IBM SPSS Statistics version 28, and statistical significance was defined as a two-tailed p -value <0.05 . Variables were categorized before analysis to facilitate clinical interpretation and to ensure compatibility with logistic regression modelling.

The primary etiological model demonstrated acceptable discrimination (AUC = 0.758) and good calibration (Hosmer–Lemeshow $p = 0.641$), whereas the antihypertensive class model demonstrated stronger discrimination (AUC = 0.805) with adequate model fit (Hosmer–Lemeshow $p = 0.751$).

Results

Study Population

A total of 644 patients were included in the analysis. The cohort consisted of 46.6% males, with the largest proportion of participants aged 51–60 years. Nearly half of the cohort was classified as obese (47.8%), while 10.2% were documented smokers. The most prevalent comorbidities were DM (48.0%), HTN (46.6%), and DLP (40.1%), while 22.0% of patients had a history of non-CNS malignancy. The most common indications for brain MRI were headache (23.3%), evaluation for suspected brain mass or malignancy (20.8%), and motor symptoms (20.2%).

Prevalence and Clinical Associations of CSVD

Among the 644 participants, 235 patients (36.5%) demonstrated MRI evidence of CSVD, whereas 409 patients (63.5%) had no radiological evidence of CSVD. (Figure 1) Patients with CSVD were significantly older than those without CSVD ($p < 0.001$) and had higher rates of HTN, CKD, DLP, and prior stroke or transient ischemic attack (TIA) (all $p < 0.05$). Neurological presentations such as motor deficits, speech disturbances, gait abnormalities, and altered level of consciousness (LOC) were also more frequent among patients with CSVD (all $p < 0.05$).

DM was significantly associated with CSVD ($p < 0.001$), with approximately half of diabetic patients demonstrating imaging evidence of disease. Lower rates of CSVD were observed among individuals with controlled HbA1c levels, suggesting that poorer glycemic control may be associated with increased CSVD burden. An inverse pattern was observed for lipid control: low-density lipoprotein (LDL) levels were more frequently controlled in the CSVD-positive group ($p < 0.001$). In addition, hematologic and cardiovascular variables differed between groups. Higher hemoglobin levels were associated with lower odds of CSVD. Abnormal echocardiographic findings were also more frequently observed among patients with CSVD. These associations may suggest potential links between systemic physiological status, underlying cardiovascular disease, and the development of cerebral microvascular pathology (Table 1).

Medication Patterns

Among patients with hypertension ($n = 300$), 26% were treated with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers (ACEI/ARB), 19.7% with CCB, and 16.3% with a combination of both therapies.

The crude prevalence of CSVD differed across antihypertensive treatment groups. The highest prevalence was observed among patients not receiving antihypertensive therapy (66.3%), whereas the lowest prevalence was seen among CCB users (23.7%; $p < 0.001$). Beta-blocker users and patients receiving combined therapy also showed relatively high crude CSVD prevalence (84.8% and 77.6%, respectively). Because crude prevalence comparisons may be influenced by clinical differences between treatment groups, multivariable logistic regression was subsequently performed to evaluate independent associations between antihypertensive class and CSVD.

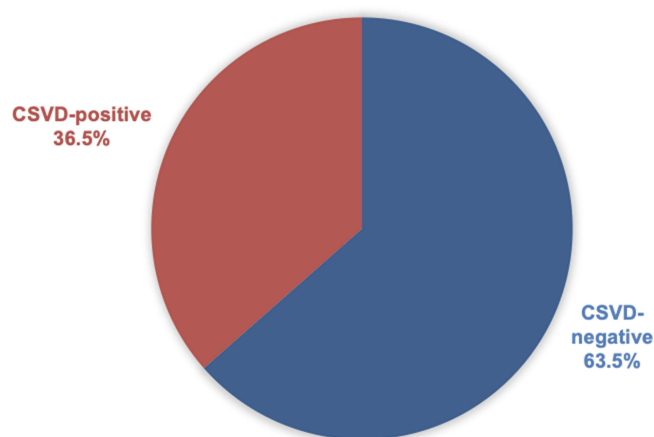


Figure 1 Proportion of patients with and without cerebral small vessel disease (CSVD) in the study cohort.

Table 1 Demographic, Clinical, and Radiological Characteristics of the Study Population

Item	Total Patients (n=644)	CSVD-Negative Group (n=409)	CSVD-Positive Group (n=235)	P-value
Age group (years)				
40–50	209 (32.5%)	171 (81.8%)	38 (18.2%)	<0.001
51–60	263 (40.8%)	159 (60.5%)	104 (39.5%)	
61–70	172 (26.7%)	79 (45.9%)	93 (54.1%)	
Gender				
Male	300 (46.6%)	179 (59.7%)	121 (40.3%)	0.059
Female	344 (53.4%)	230 (66.9%)	114 (33.1%)	
BMI group				0.025
Underweight	6 (0.9%)	5 (83.3%)	1 (16.7%)	
Normal weight	143 (22.2%)	84 (58.7%)	59 (41.3%)	
Overweight	174 (27%)	116 (66.7%)	58 (33.3%)	
Obese	308 (47.8%)	191 (62%)	117 (38%)	
N/A	13 (2%)	13 (100%)	0 (0%)	
Nationality				
Saudi	605 (93.9%)	376 (62.1%)	229 (37.9%)	0.005
Non-Saudi	39 (6.1%)	33 (84.6%)	6 (15.4%)	
Smoking	66 (10.2%)	38 (57.6%)	28 (42.4%)	0.291
DM	309 (48%)	158 (51.1%)	151 (48.9%)	<0.001
HTN	300 (46.6%)	143 (47.7%)	157 (52.3%)	<0.001
CKD	38 (5.9%)	15 (39.5%)	23 (60.5%)	0.002
Migraine	30 (4.7%)	27 (90%)	3 (10%)	0.002
Cancer	142 (22%)	98 (69%)	44 (31%)	0.123
Dyslipidemia	258 (40.1%)	144 (55.8%)	114 (44.2%)	0.001
CHF	28 (4.3%)	11 (39.3%)	17 (60.7%)	0.006
CAD	38 (5.9%)	14 (36.8%)	24 (63.2%)	0.001
PVD	2 (0.3%)	1 (50%)	1 (50%)	0.395
History of stroke/TIA	85 (13.2%)	31 (36.5%)	54 (63.5%)	<0.001
Reason of MRI				
Motor	130 (20.2%)	60 (46.2%)	70 (53.8%)	<0.001
Sensory	94 (14.6%)	61 (64.9%)	33 (35.1%)	
Speech	58 (9%)	24 (41.4%)	34 (58.6%)	0.001
Neuro-ophthalmology	40 (6.2%)	20 (50%)	20 (50%)	0.160
Seizure	64 (9.9%)	38 (59.4%)	26 (40.6%)	0.669

(Continued)

Table I (Continued).

Item	Total Patients (n=644)	CSVD-Negative Group (n=409)	CSVD-Positive Group (n=235)	P-value
Neuropsychiatric and cognitive	16 (2.5%)	7 (43.8%)	9 (56.3%)	0.218
Gait	21 (3.3%)	5 (23.8%)	16 (76.2%)	0.001
Vertigo	64 (9.9%)	36 (56.3%)	28 (43.8%)	0.383
Headache	150 (23.3%)	102 (68%)	48 (32%)	0.392
Malignancy workup	134 (20.8%)	103 (76.9%)	31 (23.1%)	0.001
CNS infection	10 (1.6%)	10 (100%)	0 (0%)	0.046
Change in mental status	41 (6.4%)	17 (41.5%)	24 (58.5%)	0.009
ENT causes: hearing, tinnitus, ear infection	20 (3.1%)	11 (55%)	9 (45%)	0.634
Cerebellar	13 (2%)	6 (46.2%)	7 (53.8%)	0.386
Urinary and fecal incontinence, Trigeminal neuralgia	26 (4%)	21 (80.8%)	5 (19.2%)	0.158
Movement disorder	13 (2%)	9 (69.2%)	4 (30.8%)	0.830
Dysphagia, voice changes	11 (1.7%)	10 (90.9%)	1 (9.1%)	0.154
Baseline Hb (g/L)				
Low	210 (32.6%)	136 (64.8%)	74 (35.2%)	0.019
Normal	374 (58.1%)	229 (61.2%)	145 (38.8%)	0.019
High	25 (3.9%)	23 (92%)	2 (8%)	
N/A	35 (5.4%)	21 (60%)	14 (40%)	
Baseline platelet count (x10³ cells/μL)				
Low	43 (6.7%)	21 (48.8%)	22 (51.2%)	0.168
Normal	525 (81.5%)	342 (65.1%)	183 (34.9%)	0.168
High	46 (7.1%)	27 (58.7%)	19 (41.3%)	
N/A	30 (4.7%)	19 (63.3%)	11 (36.7%)	
Controlled HbA1c	339 (52.6%)	227 (67%)	112 (33%)	
Controlled LDL	209 (32.5%)	107 (51.2%)	102 (48.8%)	<0.001
ECHO findings				
Normal	283 (43.9%)	176 (62.2%)	107 (37.8%)	0.031
Abnormal	32 (5%)	14 (43.8%)	18 (56.3%)	0.031
Not done	329 (51.1%)	219 (66.6%)	110 (33.4%)	

Notes: Data are presented as n (%) unless otherwise specified. Bold values indicate statistical significance ($P < 0.05$).

Abbreviations: BMI, body mass index; DM, diabetes mellitus; HTN, hypertension; CKD, chronic kidney disease; PFO, patent foramen ovale; CHF, congestive heart failure; CAD, coronary artery disease; PVD, peripheral vascular disease; TIA, transient ischemic attack; LOC, level of consciousness; Hb, hemoglobin; HbA1c, hemoglobin A1c; LDL, low-density lipoprotein.

Table 2 Medication Use Among Study Participants

	Total Patients	CSVD-Negative Group	CSVD-Positive Group	P-value
HTN medications	(n=300)	(n=143)	(n=157)	
None	98 (32.7%)	33 (33.7%)	65 (66.3%)	0.001
CCB	59 (19.7%)	45 (76.3%)	14 (23.7%)	<0.001
ACEIs/ARB	78 (26%)	45 (57.7%)	33 (42.3%)	0.033
Combined CCB + ACEIs/ARB	49 (16.3%)	11 (22.4%)	38 (77.6%)	<0.001
BB	33 (11%)	5 (15.2%)	28 (84.8%)	<0.001
Diuretic	45 (15%)	16 (35.6%)	29 (64.4%)	0.056
Other	7 (2.3%)	1 (14.3%)	6 (85.7%)	0.069
	(n=309)	(n=158)	(n=151)	
DM medications	263 (85.1%)	146 (55.5%)	117 (44.5%)	<0.001
	(n=644)	(n=409)	(n=235)	
Statin	286 (44.4%)	148 (51.7%)	138 (48.3%)	<0.001
Antiplatelet				
SAPT	180 (28%)	90 (50%)	90 (50%)	<0.001
DAPT	20 (3.1%)	2 (10%)	18 (90%)	
Anticoagulant	23 (3.6%)	8 (34.8%)	15 (65.2%)	<0.001

Notes: Data are presented as n (%). Bold values indicate statistical significance ($P < 0.05$).

Abbreviations: CCB, calcium channel blocker; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BB, beta-blocker; SAPT, single antiplatelet therapy; DAPT, dual antiplatelet therapy; HTN, hypertension; DM, diabetes mellitus.

Among individuals with diabetes mellitus, 85.1% were receiving antidiabetic medications. Statins were prescribed in 44.4% of the total cohort, while single antiplatelet therapy (SAPT) and dual antiplatelet therapy (DAPT) were used in 28% and 3.1% of patients, respectively. A higher prevalence of CSVD was observed among patients receiving beta-blockers, diuretics, statins, and antiplatelet therapy (all $p < 0.001$) (Table 2).

MRI Characteristics of CSVD

Within the CSVD-positive group, the most frequent MRI marker was WMH, present in 99.6% of cases. Other markers included lacunar infarcts (36.6%), recent subcortical infarcts (22.6%), and cerebral microbleeds (7.2%). The severity of WMH according to the Fazekas classification was distributed as grade 1 in 43%, grade 2 in 37.4%, and grade 3 in 19.6% (Figure 2, Table 3).

Relation of Fazekas Grade to Age and MRI Markers

Increasing Fazekas grade was significantly associated with older age ($p = 0.006$) and with other MRI markers of small vessel disease, including lacunes and CMBs (all $p < 0.05$) (Figure 3, Table 4). Higher WMH severity was observed with advancing age. Among patients aged 40–50 years, 55.3% had Fazekas grade 1 lesions, 34.2% had grade 2 lesions, and 10.5% had grade 3 lesions. In contrast, among patients older than 60 years, the distribution shifted toward more severe disease (35.5%, 33.3%, and 31.2%, respectively) (Figure 4).

The prevalence of CMBs increased progressively with WMH severity, occurring in 11.8% of patients with Fazekas grade 1, 35.3% with grade 2, and 52.9% with grade 3 ($p = 0.001$). Similarly, lacunar infarcts were more frequent in

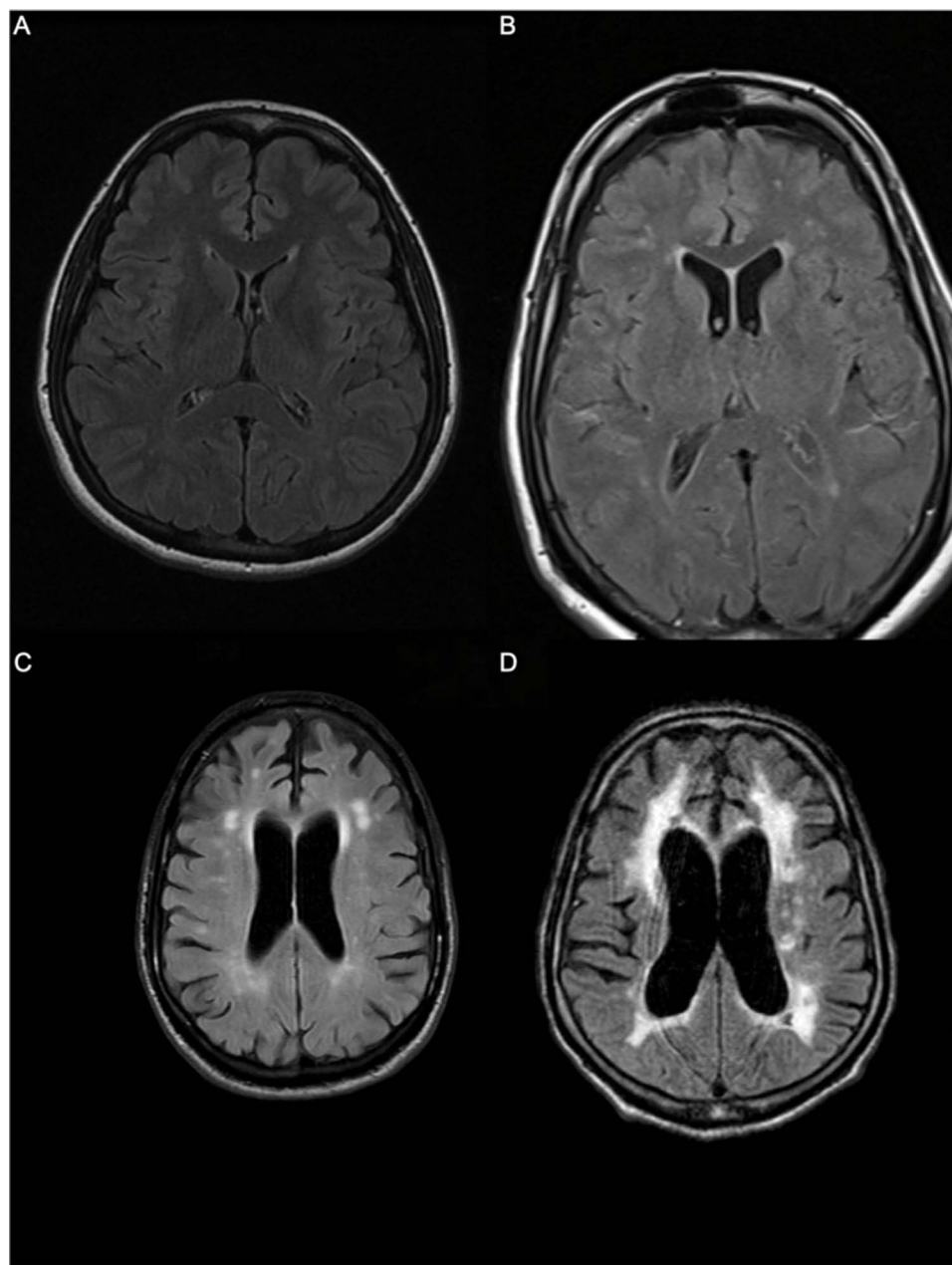


Figure 2 Fazekas classification of white matter hyperintensities (WMH) on axial T2-FLAIR MRI. Representative images demonstrate increasing WMH burden across Fazekas grades: **(A)** Grade 0—normal appearance with no WMH; **(B)** Grade 1—scattered punctate hyperintensities in the periventricular and deep white matter; **(C)** Grade 2—smooth periventricular hyperintense rim with early confluence of deep white matter lesions; **(D)** Grade 3—extensive confluent periventricular and deep white matter hyperintensities with extension into the subcortical regions.

higher Fazekas grades ($p < 0.001$), whereas recent subcortical infarcts were not significantly associated with Fazekas grade ($p = 0.221$).

Logistic Regression Analysis

In the primary etiological model Model 1 (Table 5) including all patients with complete data ($N = 428$), hypertension emerged as the strongest independent vascular risk factor for CSVD (adjusted OR 2.07, 95% CI 1.25–3.43; $p = 0.004$). Migraine demonstrated an independent inverse association with CSVD (adjusted OR 0.18, 95% CI 0.04–0.84; $p = 0.029$). High hemoglobin levels were also independently associated with lower odds of CSVD (adjusted OR 0.11, 95% CI

Table 3 MRI Markers of Cerebral Small Vessel Disease

Item	CSVD-Positive Group (n=235)	95% CI
White Matter Hyperintensities	234 (99.6%)	98.7–100.0
Lacunar infarct	86 (36.6%)	29.27 to 45.2
Recent subcortical infarct	53 (22.6%)	16.89 to 29.5
Cerebral microbleed	17 (7.2%)	4.21 to 11.58
Fazekas grade		
Grade 1	101 (43%)	35.01 to 52.22
Grade 2	88 (37.4%)	30.03 to 46.14
Grade 3	46 (19.6%)	14.33 to 26.11

Note: Data are presented as n (%) with 95% confidence intervals (CI).

0.02–0.50; $p = 0.004$). Controlled LDL levels were paradoxically associated with increased CSVD odds (adjusted OR 1.56, 95% CI 1.00–2.43; $p = 0.050$). DM demonstrated a strong association with CSVD in univariate analysis but did not remain statistically significant after adjustment in the multivariable model.

In the antihypertensive class model Model 2 (Table 6) restricted to hypertensive patients ($N = 296$), older age, female sex, diabetes mellitus, and prior stroke/TIA were independently associated with CSVD. CCB monotherapy and ACEI/ARB monotherapy were associated with significantly lower odds of CSVD compared with untreated hypertension, whereas beta-blocker therapy demonstrated a non-significant trend toward higher CSVD risk.

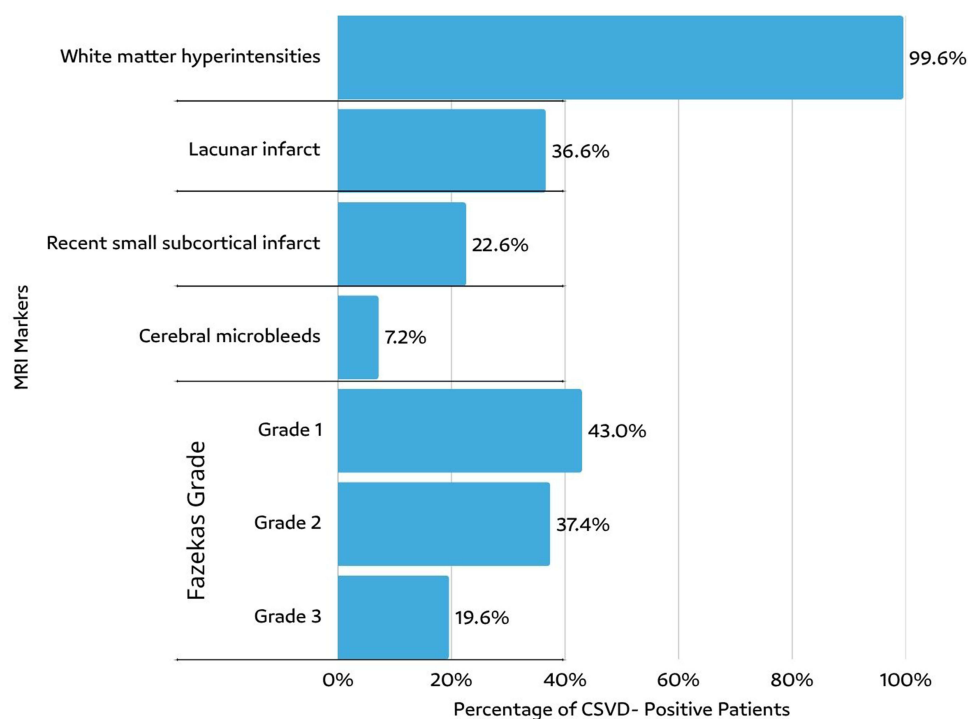


Figure 3 Distribution of MRI markers of cerebral small vessel disease (CSVD) and white matter hyperintensity severity based on the Fazekas scale among CSVD-positive patients.

Table 4 Relation Between Fazekas Grade and Age, CSVD MRI Markers and Outcomes

Item	Fazekas 1 (n=101)	Fazekas 2 (n=88)	Fazekas 3 (n=46)	P-value
Age group (years)				
40–50	21 (55.3%)	13 (34.2%)	4 (10.5%)	0.006
51–60	47 (45.2%)	44 (42.3%)	13 (12.5%)	
>60	33 (35.5%)	31 (33.3%)	29 (31.2%)	
White Matter Hyperintensities	100 (42.7%)	88 (37.6%)	46 (19.7%)	>0.999
Cerebral microbleed	2 (11.8%)	6 (35.3%)	9 (52.9%)	0.001
Lacunar infarct	23 (26.7%)	33 (38.4%)	30 (34.9%)	<0.001
Recent subcortical infarct	18 (34%)	21 (39.6%)	14 (26.4%)	0.221

Notes: Data are presented as n (%). Bold values indicate statistical significance ($P < 0.05$).

Discussion

This study provides a comprehensive evaluation of CSVD among MRI-referred patients in a Saudi clinical cohort, demonstrating a prevalence of 36.5% among adults aged 40–70 years who underwent brain MRI. This prevalence exceeds that reported in a Chinese population-based cohort (30.5%) but remains lower than estimates from a Colombian cohort,^{13,15} highlighting the substantial geographic variability of CSVD across different populations and healthcare settings. Importantly, because the present study included patients referred for MRI rather than a population-based sample, the observed prevalence should be interpreted as reflecting the burden of CSVD among MRI-referred individuals rather than the general population. As expected, CSVD prevalence increased markedly with advancing age, rising from 18.2% among individuals aged 40–50 years to 54.1% among those older than 60 years, consistent with the well-established role of vascular aging in driving microangiopathic injury.^{12,18,19} Sex differences were not prominent in the overall descriptive analyses. However, in the antihypertensive-class model restricted to hypertensive patients, female sex was independently associated with higher odds of CSVD. This finding does not align with results from several large epidemiologic studies that reported no significant sex differences in CSVD prevalence. The reasons for this association remain uncertain and may reflect differences in vascular risk profiles, treatment patterns, healthcare utilization, or residual confounding.^{13,20}

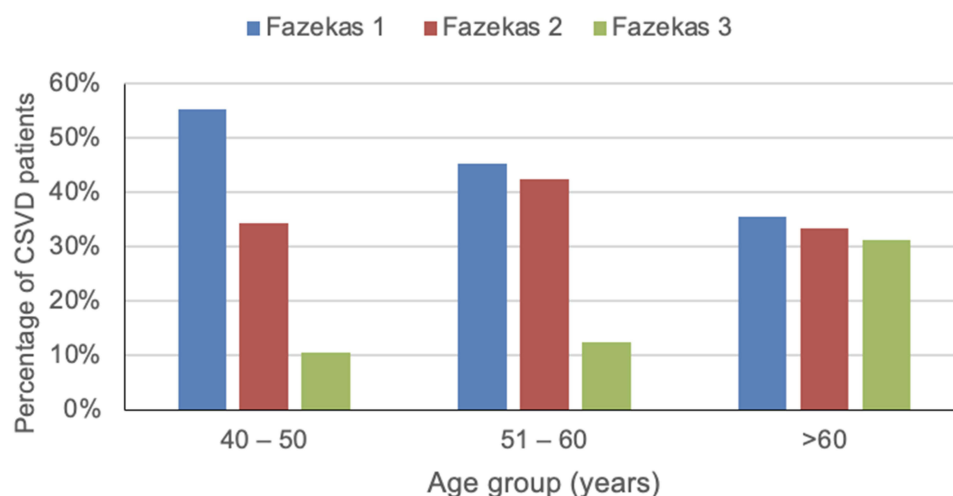


Figure 4 Distribution of Fazekas grades across age groups among patients with cerebral small vessel disease (CSVD).

Table 5 Unadjusted and Adjusted Odds Ratios for Independent Predictors of CSVD — Primary Etiological Model (All Patients)

Variable	Unadjusted Analysis			Multivariable Analysis		
	OR	95% CI	p-value	aOR	95% CI	p-value
Age group (ref: 40–50 years)						
51–60 years	2.94	1.92–4.52	<0.001*	1.46	0.81–2.63	0.203
61–70 years	5.30	3.34–8.41	<0.001*	1.85	0.98–3.50	0.060
Sex (ref: Male)						
Female	0.73	0.53–1.01	0.059	1.26	0.79–2.00	0.333
Diabetes mellitus (ref: No)						
Yes	2.84	2.04–3.97	<0.001*	1.35	0.76–2.38	0.303
Hypertension (ref: No)						
Yes	3.74	2.67–5.26	<0.001*	2.07	1.25–3.43	0.004
Chronic kidney disease (ref: No)						
Yes	2.85	1.46–5.58	0.002*	1.40	0.57–3.43	0.463
Dyslipidemia (ref: No)						
Yes	1.73	1.25–2.40	<0.001*	0.87	0.55–1.38	0.553
Congestive heart failure (ref: No)						
Yes	2.82	1.30–6.13	0.009*	1.27	0.37–4.41	0.702
Coronary artery disease (ref: No)						
Yes	3.18	1.61–6.29	<0.001*	1.51	0.61–3.78	0.376
History of stroke/TIA (ref: No)						
Yes	3.61	2.24–5.81	<0.001*	1.68	0.92–3.08	0.092
Migraine (ref: No)						
Yes	0.18	0.05–0.61	0.006*	0.18	0.04–0.84	0.029
Controlled HbA1c (ref: No)						
Yes	0.41	0.28–0.61	<0.001*	0.64	0.38–1.06	0.084
Controlled LDL (ref: No)						
Yes	1.96	1.36–2.83	<0.001*	1.56	1.00–2.43	0.050
Hemoglobin group (ref: Normal)						
Low	0.86	0.60–1.22	0.398	0.63	0.39–1.02	0.058
High	0.14	0.03–0.59	0.008*	0.11	0.02–0.50	0.004
Platelet group (ref: Normal)						
Low	1.96	1.05–3.66	0.035*	1.79	0.73–4.42	0.205
High	1.32	0.71–2.43	0.382	0.95	0.39–2.29	0.903

(Continued)

Table 5 (Continued).

Variable	Unadjusted Analysis			Multivariable Analysis		
	OR	95% CI	p-value	aOR	95% CI	p-value
Echocardiography (ref: Normal)						
Abnormal	2.11	1.01–4.43	0.047*	1.05	0.33–3.41	0.931
Not done	0.83	0.59–1.15	0.260	0.82	0.52–1.31	0.415

Notes: N = 428; events = 171. Reference categories are shown in parentheses. AUC = 0.758; Hosmer–Lemeshow $p = 0.641$; Nagelkerke $R^2 = 0.258$. Bold values indicate statistically significant p-values ($p < 0.05$).

Abbreviations: OR, odds ratio; aOR, adjusted odds ratio from multivariable logistic regression; CI, confidence interval.

Table 6 Unadjusted and Adjusted Odds Ratios for CSVD Predictors Among Hypertensive Patients — Antihypertensive Class Model

Variable	Unadjusted Analysis			Multivariable Analysis		
	OR	95% CI	p-value	aOR	95% CI	p-value
Age group (ref: 40–50 years)						
51–60 years	1.57	0.81–3.05	0.178	1.98	0.90–4.35	0.089
61–70 years	2.31	1.18–4.51	0.014*	2.62	1.15–5.99	0.022
Sex (ref: Male)						
Female	1.30	0.82–2.06	0.258	2.30	1.29–4.11	0.005
Diabetes mellitus (ref: No)						
Yes	1.94	1.17–3.25	0.011*	2.09	1.07–4.11	0.032
Chronic kidney disease (ref: No)						
Yes	1.78	0.85–3.74	0.129	1.59	0.62–4.04	0.334
Dyslipidemia (ref: No)						
Yes	1.04	0.66–1.64	0.863	0.71	0.39–1.27	0.248
History of stroke/TIA (ref: No)						
Yes	3.21	1.75–5.91	<0.001*	2.41	1.18–4.91	0.016
Antihypertensive class (ref: None — untreated hypertension)						
CCB monotherapy	0.13	0.06–0.28	<0.001*	0.10	0.04–0.23	<0.001
ACEIs/ARB monotherapy	0.24	0.12–0.49	<0.001*	0.20	0.09–0.44	<0.001
CCB + ACEIs/ARB combined	1.20	0.53–2.72	0.663	1.17	0.47–2.87	0.736
Beta-blocker	2.84	1.01–8.04	0.049*	2.69	0.90–7.99	0.075
Diuretic	0.29	0.11–0.76	0.012*	0.25	0.09–0.72	0.010

Notes: Restricted to hypertensive patients (N = 296). Reference category for medication class: untreated hypertension (no antihypertensive therapy). AUC = 0.805; Hosmer–Lemeshow $p = 0.751$; Nagelkerke $R^2 = 0.360$. OR = odds ratio; aOR = adjusted odds ratio; CI = confidence interval. All medication ORs are compared with untreated hypertension (reference group). Bold values indicate statistically significant p-values ($p < 0.05$).

Abbreviation: CCB, calcium channel blocker; ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker.

Vascular and metabolic comorbidities were strongly associated with CSVD in this study. HTN, DM, CKD, DLP, heart failure, coronary artery disease, and prior stroke or transient ischemic attack were all significantly more frequent among patients with CSVD. In the multivariable etiological model, hypertension emerged as the strongest independent vascular predictor of CSVD, highlighting the central role of chronic blood pressure–related small vessel injury.

DM demonstrated a strong association with CSVD in univariate analyses, consistent with its established contribution to microvascular injury. However, this association did not remain independently significant after adjustment for other vascular risk factors in the multivariable model, suggesting that its effect may be partially mediated through coexisting conditions such as hypertension and dyslipidemia. Chronic hyperglycemia contributes to endothelial dysfunction, oxidative stress, and advanced glycation of vascular proteins, which collectively impair microvascular autoregulation and promote white matter injury, providing a plausible biological explanation for white matter disease in CSVD.^{21,22}

Interestingly, the apparent protective association observed with antidiabetic therapy may suggest that adequate glycemic control could attenuate microangiopathic progression. However, this finding should be interpreted cautiously, as treatment patterns in observational studies may reflect differences in disease severity, duration of diabetes, or healthcare access. This observation may also reflect earlier disease onset, suboptimal glycemic control, or complex metabolic–vascular interactions that have been increasingly recognized in Middle Eastern populations.^{17,23} A notable finding in our analysis was that controlled LDL levels were associated with higher odds of CSVD. This paradoxical association likely reflects intensified lipid-lowering therapy among patients with established vascular disease or prior cerebrovascular events rather than a causal relationship between LDL control and CSVD risk. In this context, better LDL control may represent a marker of secondary prevention in individuals with higher baseline vascular risk rather than a direct contributor to CSVD development.

Another notable finding of this study was the differential association between antihypertensive medication classes and CSVD. In the multivariable antihypertensive-class model, CCB monotherapy, ACEI/ARB monotherapy, and diuretic therapy were independently associated with lower odds of CSVD compared with untreated hypertension. In contrast, beta-blocker therapy demonstrated a non-significant trend toward higher odds of CSVD, and combination CCB + ACEI/ARB therapy showed no independent association after adjustment.

These findings are broadly consistent with prior evidence, including the SPRINT-MIND trial, in which antihypertensive regimens incorporating CCBs or ACE inhibitors were associated with slower progression of WMH, potentially mediated by improved cerebral perfusion and reduced arterial stiffness.²⁴ However, in SPRINT-MIND, this effect was attenuated after adjustment for baseline WMH burden, suggesting that the observed benefit may reflect improved hemodynamic control rather than a direct class-specific neuroprotective effect.

Importantly, these medication-related associations should be interpreted with caution given the observational design of the study. Differences across antihypertensive classes are likely influenced by confounding by indication, whereby treatment selection reflects underlying disease severity, duration of hypertension, and overall cardiovascular risk profile. In this context, the higher crude prevalence of CSVD observed among patients receiving beta-blockers or combination therapy may reflect a subgroup with more advanced or complex vascular disease requiring intensified treatment, rather than a direct adverse pharmacologic effect.

Although CCB monotherapy demonstrated the lowest adjusted odds of CSVD, the present study was not designed to compare antihypertensive classes directly, and superiority of any specific class cannot be inferred. Rather, these findings suggest that effective blood pressure control, particularly in treated versus untreated hypertension, may be the primary driver of reduced CSVD burden, with observed differences between medication classes likely reflecting a combination of treatment effects and residual confounding.

Similarly, the observed association between CSVD and the use of antiplatelet or anticoagulant therapy likely reflects treatment initiated after prior vascular events or established cardiovascular disease. These therapies are therefore best interpreted as clinical markers of underlying vascular pathology rather than causal contributors to CSVD development, consistent with the potential for confounding by indication in observational studies.

The neuroimaging findings in this study were broadly consistent with the established radiologic spectrum of CSVD. WMH were by far the most prevalent imaging marker, present in nearly all CSVD cases, and higher Fazekas grades were associated with increasing age and with other markers of small vessel disease, including lacunar infarcts and cerebral

microbleeds. These associations reinforce the concept that CSVD represents a continuum of small-vessel injury, in which chronic ischemic damage and microhemorrhagic processes occur in parallel as manifestations of diffuse vascular pathology.^{4,7} CSVD was defined as the presence of at least one STRIVE-consistent imaging marker. As not all features were systematically evaluated, future studies incorporating composite burden scores may better capture overall disease severity.

Beyond conventional vascular risk factors, our analysis also identified associations between CSVD and several clinical and hematologic variables. An inverse association was observed between migraine and CSVD, likely reflecting a younger and lower-risk subgroup undergoing MRI for non-ischemic indications. Hematologic parameters also appeared to influence CSVD burden. Higher hemoglobin levels were associated with lower CSVD prevalence in our cohort, which may reflect the adverse impact of anemia on cerebral microvascular health. Prior studies have suggested a possible nonlinear (U-shaped) relationship, in which both low and excessively elevated hemoglobin levels may contribute to white matter injury through impaired oxygen delivery or increased blood viscosity.^{24,25} Also, prior studies have suggested that platelet activation and increased mean platelet volume may be stronger indicators of CSVD severity.^{24,26} These observations highlight the potential role of hematologic homeostasis in maintaining cerebral microvascular integrity.

From a clinical perspective, CSVD was most strongly associated with gait disturbance and motor dysfunction, underscoring its impact on physical function and disability beyond overt stroke. These findings emphasize that CSVD should not be considered merely an incidental radiologic finding but rather a condition with meaningful functional consequences. Given the high prevalence of vascular risk factors in Saudi Arabia, the burden of CSVD-related cognitive and motor impairment is likely to increase with population aging. Early identification and targeted management of modifiable risk factors including optimized glycemic control, effective blood pressure management, and appropriate cardiovascular risk reduction may therefore play a critical role in limiting disease progression.^{13,20,24}

Strengths and Limitations

This study has several strengths. It included a relatively large, well-characterized patients and employed rigorous neuroimaging evaluation based on STRIVE criteria, with MRI scans independently reviewed by two neuroradiologists. In addition, the study integrated detailed clinical, laboratory, and imaging data, allowing for a comprehensive assessment of CSVD determinants. Nevertheless, several limitations should be acknowledged. The retrospective cross-sectional design limits causal inference, and the single-center setting may restrict the generalizability of the findings. Furthermore, because the study population consisted of patients referred for brain MRI, the reported prevalence reflects MRI-referred individuals rather than the general population and may therefore be influenced by referral or indication bias. The absence of longitudinal follow-up precluded assessment of cognitive outcomes or progression of CSVD over time. Additionally, continuous clinical measurements such as longitudinal blood pressure data were not consistently available in the retrospective records. Antidiabetic therapies were analyzed as a combined variable because many patients were receiving multiple agents simultaneously, limiting reliable subclassification by individual drug class. Finally, although MRI scans were independently reviewed by two neuroradiologists with discrepancies resolved by consensus, formal inter-rater reliability statistics were not calculated.

Conclusion

In this MRI-based clinical cohort from Saudi Arabia, cerebral small vessel disease was present in more than one-third of adults aged 40–70 years undergoing neuroimaging, underscoring its substantial clinical burden. CSVD prevalence increased markedly with advancing age and vascular comorbidities, with hypertension emerging as the strongest independent vascular predictor.

Certain antihypertensive classes, particularly calcium channel blocker and ACEI/ARB monotherapy, were associated with lower odds of CSVD among hypertensive patients, although these findings should be interpreted cautiously given the observational design. The observed associations between CSVD and gait or motor dysfunction further emphasize its clinical relevance beyond a silent radiologic finding.

Collectively, these results reinforce the importance of aggressive vascular risk factor control and early identification of individuals at risk for cerebral microvascular disease.

Future Direction

Our findings suggest a potentially important and clinically actionable signal: CCBs may be associated with a lower burden of CSVD. This observation opens an exciting avenue for future research to determine whether CCBs exert protective effect on the cerebral microvasculature beyond their role in blood pressure reduction. If confirmed, such findings could help reshape antihypertensive management toward a more disease-targeted approach for CSVD prevention. As a next step, we plan to conduct a longitudinal follow-up study in the same cohort, incorporating cognitive assessments to better characterize the long-term radiological, functional, and cognitive impact of MRI-positive CSVD.

AI Use Statement

During the preparation of this work the author(s) used AI to assist with language editing and text organization. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Abbreviations

CSVD, cerebral small vessel disease; MRI, magnetic resonance imaging; WMH, white matter hyperintensities; CMB, cerebral microbleeds; HTN, hypertension; DM, diabetes mellitus; DLP, dyslipidemia; STRIVE, Standards for Reporting Vascular Changes on Neuroimaging; CNS, central nervous system; CKD, chronic kidney disease; HbA1c, hemoglobin A1c; TIA, transient ischemic attack; LOC, level of consciousness; LDL, low-density lipoprotein; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; SAPT, single antiplatelet therapy; DAPT, dual antiplatelet therapy.

Ethical Approval

This study was approved by the Institutional Review Board of King Abdulaziz Medical City (IRB #NRR24/061/12). Patient consent was waived due to the retrospective design. All data were anonymized prior to analysis, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

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Disclosure

The author declares no conflicts of interest.

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