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Prognostic significance of carotid artery stenosis in anterior circulation ischemic stroke patients

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Abstract

Background The carotid artery stenosis (CAS) is a notable risk element for ischemic stroke, accounting for 10 to 20% of such cases. There is a deficiency in research studies exploring the prognostic impact of CAS specifically in the anterior circulation acute ischemic stroke (AIS). Therefore, we conducted this study to evaluate the prognostic impact of CAS in individuals with anterior circulation AIS. Patients were categorized into two groups, patients with both anterior circulation AIS and CAS in comparison to patients with anterior circulation AIS without CAS (control group). We compared patient characteristics, neuroimaging, and outcome evaluations using a modified Rankin Scale (mRS) and the National Institute Health Stroke Scale (NIHSS) in both groups. P -value < 0.05 meaning statistical significance.

Results Fifty patients were involved in the current study; of them, 25 patients with both anterior circulation AIS and CAS and 25 patients with anterior circulation AIS without CAS. Both groups were comparable concerning age, gender, co-morbidities of diabetes, hypertension, smoking, and dyslipidemia with p -value > 0.05. The mean NIHSS was significantly elevated the CAS group compared to the control group ($p = 0.037$). Our results showed that patients with CAS $\geq 70\%$ were associated with an mRS > 2 and a significantly higher NIHSS compared to control. The regression analysis showed that occlusion > 1/3, CAS $\geq 70\%$, and higher NIHSS score were considered independent predictors of mRS > 2. The mortality rate was significantly elevated in the CAS group versus the control group ($p < 0.05$). Both bilateral and unilateral CAS groups were comparable regarding NIHSS and mRS values.

Conclusion Patients with anterior circulation AIS who had CAS of 70% or more, whether unilateral or bilateral, showed a higher likelihood of increased mortality and more severe strokes, as evidenced by elevated NIHSS and mRS scores.

Keywords Anterior circulation, Ischemic stroke, Carotid stenosis, NIHSS, mRS

Background

Stroke continues to be a significant factor contributing to both disability and mortality worldwide. Despite notable advancements in preventing and treating its acute phase, the impact of stroke remains a significant global health concern [1]. Among stroke types, ischemic strokes constitute a significant majority, ranging from 80 to 87% [2], with approximately 20–25% of ischemic strokes resulting from large-artery atherosclerosis [3, 4]. Carotid artery stenosis (CAS) stands out as an important risk factor

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for ischemic stroke, accounting for around 10 to 20% of cases [5]. The level of stenosis is a significant risk factor for ipsilateral ischemic stroke [6]. Some ischemic strokes associated with CAS are due to reduced blood flow, while most of these strokes seem to arise from embolization originating from a susceptible atherosclerotic plaque or sudden blockage of the carotid artery with subsequent thrombus propagation [7, 8].

Standard and more advanced imaging methods can offer insights into the underlying mechanism causing ischemic stroke [9]. The significance of extracranial carotid ultrasound examinations emerges as pivotal in stroke management and prognostication, because it is cost-effective and safe. Within the landscape of studies addressing the prognostic effects of CAS, studies focusing on anterior circulations are lacking [10].

Our study was designed to precisely examine the prognostic significance of CAS in patients with anterior circulation AIS. We aimed to draw comparisons with a counterpart group experiencing anterior circulation AIS devoid of CAS. We employed the National Institute of Health Stroke Scale (NIHSS) [11] and modified Rankin Scale (mRS) [12] as prognostic tools lend precision for ischemic stroke severity.

Methods

This was a cross-sectional study, involved patients admitted to the neurology department. The study protocol received approval from the ethical committee, and prior written informed consent was acquired from all participating patients. For patients with disturbed conscious level, we obtained consent from a legally authorized representative or guardian who can make informed decisions on behalf of the patient.

We included 50 patients, divided into two equal groups: the control group ($n=25$) with anterior circulation AIS without CAS and the CAS group ($n=25$) with anterior circulation AIS and associated CAS. The included patients aged >18 years and diagnosed with anterior circulation AIS, confirmed by magnetic resonance imaging (MRI) brain, and magnetic resonance angiography (MRA) brain and neck. We excluded patients with posterior circulation infarction, hemorrhagic stroke, cerebral venous thrombosis, transient ischemic attack, or had a history of previous disability. A simple random sampling method was employed. The two groups were matched for age, sex, and risk factors. A sample of 50 individuals with AIS was chosen, following the prevalence data from a prior study. This selection was made using OpenEpi software, ensuring a 95% confidence level and a 5% margin of error.

Patients underwent comprehensive assessments, encompassing a comprehensive medical history, overall

medical examination, and neurological assessment, utilizing the NIHSS and mRS. Routine investigations included lipid profile, coagulation profile, complete blood picture, liver function tests, and kidney function tests. Imaging studies comprised routine computed tomography (CT) brain, extracranial carotid artery duplex, MRI brain, and MRA brain and neck.

Upon admission to the stroke unit, all patients received a standardized neurological evaluation, and stroke severity was quantified using the NIHSS and mRS [13]. Hypertension was defined for patients receiving antihypertensive treatment or those with average blood pressure readings equal to or exceeding 140/90 mmHg during the initial 48 h of their hospital stay were included in the study [14]. Hypercholesterolemia was assigned to patients receiving statin treatment or with total cholesterol levels ≥ 200 mg/dL [15]. Diabetes classification included individuals undergoing antidiabetic treatment or those with fasting blood glucose levels equal to or exceeding 126 mg/dL [16]. Smoking status was classified as follows: individuals classified as ex-smokers had a history of smoking at least 100 cigarettes in their lifetime but had refrained from smoking in the month preceding the stroke. On the other hand, current smokers were defined as those who had smoked at least 100 cigarettes and had smoked within the last month [17].

The main outcome measure was the occurrence of CAS at a level of $\geq 50\%$ and $\geq 70\%$. Secondary outcomes include identifying factors that predict CAS at this threshold and examining the correlation between CAS $\geq 50\%$ and $\geq 70\%$ and functional outcomes as defined by NIHSS and mRS.

The CAS was demarcated as a stenosis in the internal carotid artery of 50% or more [18]. Medical records on the degree of CAS, as evaluated by radiologists, were utilized. The CAS severity was graded using the European Carotid Surgery Trial (ECST) method [19], calculating the percentage of stenosis. Abnormalities in the Doppler investigation were noted for increased peak systolic velocities (≥ 130 cm/s) or disturbed, demodulated, reversed, or missing flows at the carotid level [20].

The recorded data underwent analysis through SPSS version 27.0. Descriptive statistics were employed for quantitative data, as mean \pm standard deviation (SD), while representing qualitative variables using frequency and percentages. The normality of the data distribution was assessed using Kolmogorov–Smirnov and Shapiro–Wilk tests. For comparisons between two normally distributed means, the independent-samples t -test was utilized. In the case of non-parametric data, the Mann–Whitney U test was applied for two-group comparisons. The Chi-square (χ^2) test was used to compare proportions among qualitative parameters. To assess and

estimate the dependence of a quantitative variable on one or more independent variables, multivariate regression was conducted. A statistical significance was considered when a p -value was below 0.05.

Results

Fifty adult patients were included; of them, 25 were in the CAS group (nine cases had bilateral CAS, and 16 had unilateral CAS) and 25 cases were in the control group. The mean age was 69.84 ± 11.25 years in the CAS group and 68.08 ± 11.95 years in the control group. There were 18 (72.0%) females in the CAS group and 12 (48.0%) females in the control group. In the CAS group, 16 patients had diabetes mellitus, 18 had hypertension, and 18 had dyslipidemia. Both compared groups were comparable regarding mean ages ($p=0.59$), diabetes ($p=0.77$), hypertension ($p=0.54$), smoking ($p=0.74$), and dyslipidemia ($p=0.15$). Table 1 shows the demographic and comorbidities data in the CAS and control groups.

Our results showed that the mean NIHSS score was significantly higher in the CAS group (bilateral and unilateral) compared to the control group (10.36 ± 6.64 versus 7.08 ± 4.02 , respectively, $p=0.037$). While no significant differences were observed in the size and location of cerebral infarction, a significant difference was found regarding the deep branches of the middle cerebral artery ($p=0.021$). Regarding, the mRS after 1 month, the CAS group exhibited a higher proportion having an mRS 6 ($p=0.020$). The control group showed a higher proportion of patients with moderate disability (mRS 1) after

1 month ($p=0.049$). The mortality rate was significantly higher in the CAS group ($p=0.018$). Table 2 shows the comparison between cases with or without CAS regarding NIHSS and cerebral infarction.

When comparing bilateral CAS versus control, the mean age was comparable between the groups (67.67 ± 6.86 for CAS versus 68.08 ± 11.95 for control, $p=0.882$). The gender did not differ significantly between the groups ($p=0.697$). No significant differences were observed regarding the prevalence of diabetes ($p=0.724$), hypertension ($p=0.886$), smoking history ($p=0.944$), or dyslipidemia ($p=0.855$) between the two groups. Further, there were no significant differences in the size of cerebral infarction ($p>0.05$), NIHSS ($p=0.073$), or distribution of major artery occlusions ($p>0.05$) between the groups.

The CAS group had a notably greater percentage of patients with an mRS score of 4 ($p=0.003$) and 6 ($p=0.017$). Conversely, the control group displayed a greater percentage of patients with an mRS score of 1 ($p=0.039$). The mortality rate was significantly higher in the bilateral CAS group compared to the control group ($p=0.015$). Table 3 shows the comparison between patients with or without bilateral symptomatic CAS across various demographic and clinical parameters.

When comparing unilateral CAS versus control, the numbers of patients with an mRS score of 4 (moderate disability, unable to walk without assistance) was notably elevated in the CAS group (25.0%) compared to none in the control group ($p=0.009$). A similar trend was observed in mRS scores of 6 (dead) where 18.8% of patients with CAS had this outcome compared to none in the control group ($p=0.026$). The mortality rate was significantly higher in the group with unilateral symptomatic CAS (18.8%) compared to the control group (0.0%) ($p=0.025$). Table 4 shows the comparison between patients with and without unilateral symptomatic CAS based on mRS scores after 1 month.

When comparing bilateral versus unilateral CAS, both groups were comparable regarding mean NIHSS scores (11.89 ± 8.18 versus 9.50 ± 5.72 , respectively, $p=0.346$). Moreover, both groups were comparable in other mRS categories (1 through 6) between the two groups. Table 5 shows the comparison between symptomatic bilateral ($n=9$) and unilateral CAS ($n=25$) regarding NIHSS and mRS.

The results showed a statistically significant association between mRS with occlusion $> 1/3$, lacunar infarct, anterior cerebral artery, and CAS $\geq 70\%$, with p -value < 0.05 in patients with CAS. Table 6 shows the association between mRS levels according to all parameters in the CAS group. Regression analysis was conducted for the prediction of mRS > 2 using occlusion $> 1/3$, lacunar infarct, anterior

Table 1 Demographic data in carotid stenosis and control groups

Variables	CAS (unilateral and bilateral) (n=25)	Control (n=25)	p-value
Age (years)			
Mean \pm SD	69.84 \pm 11.25	68.08 \pm 11.95	0.594
Gender			
Female	18 (72.0%)	12 (48.0%)	0.083
Male	7 (28.0%)	13 (52.0%)	
Co-morbidities			
DM	16 (64.0%)	15 (60.0%)	0.771
HTN	18 (72.0%)	16 (64.0%)	0.544
Smoking			
Non-smoker	20 (80.0%)	18 (72.0%)	0.739
Ex-smoker	3 (12.0%)	5 (20.0%)	0.627
Smoker	2 (8.0%)	2 (8.0%)	0.472
Dyslipidemia	18 (72.0%)	13 (52.0%)	0.145

CAS carotid artery stenosis, DM diabetes mellitus, HTN hypertension, SD standard deviation, independent t-test was used for comparing continuous data, Chi-square test was used for comparing dichotomous data, * p -value < 0.05 was considered statistically significant

Table 2 Comparison between cases with or without carotid stenosis regarding NIHSS and cerebral infarction

Variables	CAS (unilateral and bilateral) (n = 25)	Control (n = 25)	p-value
NIHSS			
Mean ± SD	10.36 ± 6.64	7.08 ± 4.02	0.037*
Size of cerebral infarction			
Cerebral infarction > 1/3 of territory	12 (48.0%)	11 (44.0%)	0.777
Cerebral infarction < 1/3 of territory	9 (36.0%)	9 (36.0%)	1.000
Lacunar infarct	4 (16.0%)	5 (20.0%)	0.713
Major artery occlusion			
ACA	2 (8.0%)	1 (4.0%)	0.552
MCA			
Cortical	6 (24.0%)	5 (20.0%)	0.733
Deep	14 (56.0%)	6 (24.0%)	0.021*
Complete	8 (32.0%)	5 (20.0%)	0.333
Watershed	3 (12.0%)	0 (0.0%)	0.074
mRS after 1 month			
0	2 (8.0%)	4 (16.0%)	0.389
1	3 (12.0%)	9 (36.0%)	0.049*
2	4 (16.0%)	10 (40.0%)	0.061
3	1 (4.0%)	1 (4.0%)	1.000
4	7 (28.0%)	0 (0.0%)	0.005*
5	3 (12.0%)	1 (4.0%)	0.302
6	5 (20.0%)	0 (0.0%)	0.020*
Mortality			
Died	5 (20.0%)	0 (0.0%)	0.018*

CAS carotid artery stenosis, NIHSS National Institute of Health Stroke Scale, mRS modified Rankin scale, MCA middle cerebral artery, ACA anterior cerebral artery, SD standard deviation, independent t-test was used for comparing continuous data, Chi-square test was used for comparing dichotomous data, *p-value < 0.05 was considered statistically significant

cerebral artery, CAS $\geq 70\%$, and NIHSS score. Higher occlusion > 1/3, CAS $\geq 70\%$, and NIHSS score were considered independent predictors of mRS > 2. Table 7 shows the multivariate analysis for the prediction of mRS > 2 among the CAS group.

Discussion

Carotid artery stenosis can contribute to ischemic stroke through several mechanisms, including embolism, thrombotic blockage, arterial dissection, or reduced blood flow. This study revealed that patients with CAS and ischemic stroke were linked with a higher mRS and NIHSS scores, indicating a poorer functional result in those individuals. Moreover, those patients encompassed higher mortality rates compared to patients without CAS. Identifying a CAS stenosis of 70% or more as a prognostic factor for an adverse functional outcome in anterior AIS is a novel discovery that has not been documented previously. These results came in line with a previous study by Muscari and colleagues [20], showing that ipsi- or contralateral CAS of 60% or more continued to be linked with an mRS > 2. Similarly, the findings of Soliman

and colleagues revealed that carotid stenosis $\geq 50\%$ had a negative effect on stroke severity and disability [21].

The current study showed no significant differences in the size or location of cerebral infarctions between the compared groups. The difference in NIHSS scores, despite similar infarction sizes and locations, may be attributed to the variability in collateral circulation. Effective collaterals can mitigate the impact of stroke on neurological function, while inadequate collaterals can exacerbate functional deficits. Therefore, the higher NIHSS scores in the CAS group might reflect inadequate collateral support, leading to more severe functional impairment despite similar infarction characteristics [22]. Additionally, comorbidities such as hypertension, diabetes, or cardiovascular diseases can affect stroke outcomes and recovery. These comorbidities can exacerbate the effects of a stroke and impede rehabilitation, contributing to worse NIHSS scores [23].

In our sample, regression analysis revealed that higher occlusion > 1/3, CAS $\geq 70\%$, and NIHSS score showed a significant association with poor prognosis. This likely resulted from the primary connection between the severity of cerebral impairment and

Table 3 Comparison between patients with stroke and bilateral symptomatic CAS (n=9) and those with stroke without CAS (n=25) across various demographic and clinical parameters.

Variables	Bilateral CAS (n=9)	Control (n=25)	p-value
Age (years)			
Mean ± SD	67.67 ± 6.86	68.08 ± 11.95	0.882
Gender			
Female	5 (55.6%)	12 (48.0%)	0.697
Male	4 (44.4%)	13 (52.0%)	
Co-morbidities			
DM	6 (66.7%)	15 (60.0%)	0.724
HTN	6 (66.7%)	16 (64.0%)	0.886
Non smoker	6 (66.7%)	18 (72.0%)	0.240
Ex-smoker	2 (22.2%)	5 (20.0%)	0.341
Smoker	1 (11.1%)	2 (8.0%)	0.944
Dyslipidemia a	5 (55.6%)	13 (52.0%)	0.855
Size of cerebral infarction			
Infarction >1/3	5 (55.6%)	11 (44.0%)	0.551
Infarction <1/3	3 (33.3%)	9 (36.0%)	0.886
Lacunar Infarct	1 (11.1%)	5 (20.0%)	0.549
NIHSS			
Mean ± SD	11.89 ± 8.18	7.08 ± 4.02	0.072
Distribution			
ACA	1 (11.1%)	1 (4.0%)	0.437
MCA			
Cortical	2 (22.2%)	5 (20.0%)	0.888
Deep	2 (22.2%)	14 (56.0%)	0.082
Complete	4 (44.4%)	5 (20.0%)	0.154
Watershed	0 (0.0%)	0 (0.0%)	1.000
mRS after 1 month			
0	2 (22.2%)	4 (16.0%)	0.680
1	0 (0.0%)	9 (36.0%)	0.039*
2	1 (11.1%)	10 (40.0%)	0.117
3	0 (0.0%)	1 (4.0%)	0.549
4	3 (33.3%)	0 (0.0%)	0.003*
5	1 (11.1%)	1 (4.0%)	0.444
6	2 (22.2%)	0 (0.0%)	0.017*
Mortality			
Died	2 (22.2%)	0 (0.0%)	0.015*

CAS carotid artery stenosis, NIHSS National Institute of Health Stroke Scale, mRS modified Rankin scale, MCA middle cerebral artery, ACA anterior cerebral artery, DM diabetes mellitus, HTN hypertension, SD standard deviation, Independent t-test was used for comparing continuous data, Chi-square test was used for comparing dichotomous data, *p-value <0.05 was considered statistically significant

prognosis. This suggests that stenoses of 70% or more, regardless of their location in the main carotid axis, indicate a significant compromise in cerebral circulation or reflect a general susceptibility to atherosclerosis. These findings represent important prognostic implications for stroke patients.

Table 4 Comparison between patients with stroke and unilateral symptomatic carotid stenosis (n= 16) and those with stroke without carotid stenosis (n=25) based on mRS scores after 1 month

Variables	Unilateral CAS (n= 16)	Control (n= 25)	p-value
Age (years)			
Mean ± SD	71.06 ± 13.16	68.08 ± 11.95	0.406
Gender			
Female	13 (81.3%)	12 (48.0%)	0.033*
Male	3 (18.8%)	13 (52.0%)	
NIHSS			
Mean ± SD	9.50 ± 5.72	7.08 ± 4.02	
mRS after 1 month			
0	0 (0.0%)	4 (16.0%)	0.096
1	3 (18.8%)	9 (36.0%)	0.244
2	3 (18.8%)	10 (40.0%)	0.160
3	1 (6.3%)	1 (4.0%)	0.742
4	4 (25.0%)	0 (0.0%)	0.009*
5	2 (12.5%)	1 (4.0%)	0.314
6	3 (18.8%)	0 (0.0%)	0.026*
Mortality			
Died	3 (18.8%)	0 (0.0%)	0.025*

CAS carotid artery stenosis, NIHSS National Institute of Health Stroke Scale, mRS modified Rankin scale, MCA middle cerebral artery, ACA anterior cerebral artery, DM diabetes mellitus, HTN hypertension, SD standard deviation, Independent t-test was used for comparing continuous data, Chi-square test was used for comparing dichotomous data, *p-value <0.05 was considered statistically significant

Table 5 Comparison between patients bilateral symptomatic carotid stenosis (n= 9) and unilateral carotid stenosis (n= 25) regarding NIHSS and mRS

Variables	Bilateral CAS (n= 9)	Unilateral CAS (n= 16)	p-value
NIHSS			
Mean ± SD	11.89 ± 8.18	9.50 ± 5.72	0.346
mRS after 1 month			
0	2 (22.2%)	0 (0.0%)	0.054
1	0 (0.0%)	3 (18.8%)	0.174
2	1 (11.1%)	3 (18.8%)	0.622
3	0 (0.0%)	1 (6.3%)	0.451
4	3 (33.3%)	4 (25.0%)	0.664
5	1 (11.1%)	2 (12.5%)	0.919
6	2 (22.2%)	3 (18.8%)	0.842
Mortality			
Died	2 (22.2%)	3 (18.75%)	0.712

CAS carotid artery stenosis, NIHSS National Institute of Health Stroke Scale, mRS modified Rankin scale, SD standard deviation, independent t-test was used for comparing continuous data, Chi-square test was used for comparing dichotomous data, *p-value <0.05 was considered statistically significant

Table 6 Association between mRS level according to all parameters in the CAS group

Variables	mRS ≤ 2 (n = 9)		mRS > 2 (n = 16)		p-value
	No	%	No	%	
Gender					
Female	7	77.8%	11	68.8%	0.629
Male	2	22.2%	5	31.3%	
Age (years)					
Mean ± SD	73.33 ± 9.80		67.88 ± 11.83		0.253
Co-morbidities					
DM	7	77.8%	9	56.3%	0.282
HTN	8	88.9%	10	62.5%	0.158
Smoking					
Non-smoker	8	88.9%	12	75.0%	0.369
X-smoker	0	0.0%	3	18.8%	
Smoker	1	11.1%	1	6.3%	
Dyslipidemia	6	66.7%	12	75.0%	0.656
Size of cerebral infarction					
Occlusion > 1/3	1	11.1%	11	68.8%	0.006*
Occlusion < 1/3	4	44.4%	5	31.3%	0.509
Lacunar Infarct	4	44.4%	0	0.0%	0.004*
Major artery occlusion					
ACA	2	22.2%	0	0.0%	0.049*
MCA					
Cortical	1	11.1%	5	31.3%	0.258
Deep	4	44.4%	2	12.5%	0.073
Complete	1	11.1%	7	43.8%	0.093
Watershed	1	11.1%	2	12.5%	0.918
Mortality					
Died	0	0.0%	5	31.3%	0.061
Carotid stenosis %					
≥ 50%	6	66.7%	15	93.8%	0.076
≥ 70%	2	22.2%	12	75.0%	0.011*
NIHSS score					
Mean ± SD	5.22 ± 3.03		13.25 ± 6.39		< 0.001**

CAS carotid artery stenosis, mRS modified Rankin Score, NIHSS National Institute of Health Stroke Score, MCA middle cerebral artery, ACA anterior cerebral artery, SD standard deviation, DM diabetes mellitus, HTN hypertension, independent t-test was used for comparing continuous data, Chi-square test was used for comparing dichotomous data, *p-value < 0.05 was considered statistically significant

It is crucial to note that the development of carotid atherosclerosis and the eventual occurrence of a stroke require a considerable duration for manifestations to appear [24]. Those who ultimately experience a stroke due to carotid atherosclerosis likely belong to the subgroup with the highest comorbidities in the carotid atherosclerosis category. Many of the patients with CAS included in our study may have developed the condition

Table 7 Multivariate analysis for prediction of mRS > 2 among patients group

Variables	β	p-value	OR	95% C.I. for OR	
				Lower	Upper
Occlusion > 1/3	2.868	0.016*	7.600	1.709	18.290
Lacunar infarct	- 4.366	0.999	1.683	1.212	2.912
ACA	- 3.030	0.999	1.552	1.117	2.685
Carotid stenosis ≥ 70%	2.351	0.017*	4.500	1.514	7.811
NIHSS score	0.709	0.030*	2.032	1.073	3.849

NIHSS National Institute of Health Stroke Scale, mRS modified Rankin scale, β Regression coefficient, OR odds ratio, CI confidence interval, ACA: anterior cerebral artery, *p-value < 0.05 was considered statistically significant

prior to the initiation of risk factor reduction, given that atherosclerosis formation spans multiple decades. The overall compromised functionality of patients with CAS and their elevated risk of cardiovascular disease may account for the poorer outcomes observed in these individuals. This suggests that CAS might serve as a symptom rather than the direct cause of the adverse outcomes. Additionally, the presence of CAS could be attributed to a low socioeconomic status, which is associated with overall worse outcomes [25, 26].

The current study showed that deep branches of MCA was significantly higher in the CAS group. The deep MCA territory infarcts are often linked to embolic phenomena or compromised cerebral perfusion related to ICA stenosis. This relationship underscores the potential role of ICA stenosis in the pathogenesis of ischemic strokes affecting deep brain structures [27]. ICA stenosis can lead to reduced cerebral blood flow and alter hemodynamics, which may predispose the deep MCA territory to ischemic damage. Additionally, impaired collateral circulation due to ICA stenosis could exacerbate ischemia in these critical regions. The presence of ICA stenosis may also indicate a higher risk of embolic events originating from the carotid plaque, which could further contribute to deep MCA infarcts y[28].

The primary limitation lies in the exclusion of patients with transient ischemic attack. The reason for the absence of information about the status of CAS is unclear. This lack of clarity could lead to an overestimation of the prevalence of CAS. One potential explanation for the missing information is the attribution of stroke to atrial fibrillation, where the degree of CAS is frequently not examined. Further, the small number of participants in our study poses a challenge as it may affect the applicability and generalizability of the results. In addition, our analysis did not assess plaque features such as irregularities, softness, hemorrhage, or detailed evaluation of collateral circulation, including

ophthalmic reversal flow. Moreover, this study did not report data on patients who received recombinant tissue plasminogen activator or mechanical thrombectomy. Future studies should consider incorporating these additional variables to enhance the evaluation of carotid plaque characteristics and their impact on stroke prognosis.

Conclusion

The current study showed that the CAS significantly influenced the prognosis of anterior circulation AIS, affecting NIHSS and mRS scores. Our findings emphasized the importance of rigorous CAS management in AIS to mitigate post-stroke morbidity and mortality. We recommend widespread carotid duplex assessments for AIS patients to precisely determine the location, criteria, and percentage of CAS, thereby informing targeted therapeutic interventions.

Abbreviations

CAS	Carotid artery stenosis
AIS	Acute ischemic stroke
mRS	Modified Rankin Scale
NIHSS	National Institute of Stroke Scale
CT	Computed tomography
MRI	Magnetic resonance imaging
MRA	Magnetic resonance angiography
ECST	The European Carotid Surgery Trial
SPSS	Statistical Package for the Social Sciences
SD	Standard deviation

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Not applicable.

Author contributions

AZA analyzed and interpreted the patient included data. MSHA performed the statistical analysis and was a major contributor in writing the manuscript. ARS implemented the study design and manuscript writing. HMA contributed in the statistical analysis and manuscript writing. HMZ implemented the study design and proofreading the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study protocol obtained approval from the ethical committee at Faculty of Medicine, Ain Shams University (in July 2021). Written consent was obtained from patients.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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References

- Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson AP, et al. Heart disease and stroke statistics-2019 update: a report from the American Heart Association. *Circulation*. 2019;139(10):e56–528.
- Kuriakose D, Xiao Z. Pathophysiology and treatment of stroke: present status and future perspectives. *Int J Mol Sci*. 2020. <https://doi.org/10.3390/ijms21207609>.
- Grau AJ, Weimar C, Buggle F, Heinrich A, Goertler M, Neumaier S, et al. Risk factors, outcome, and treatment in subtypes of ischemic stroke: the German stroke data bank. *Stroke*. 2001;32(11):2559–66.
- Lange MC, Cabral NL, Moro CH, Longo AL, Gonçalves AR, Zétola VF, et al. Incidence and mortality of ischemic stroke subtypes in Joinville, Brazil: a population-based study. *Arq Neuropsiquiatr*. 2015;73(8):648–54.
- Barrett KM, Brott TG. Stroke caused by extracranial disease. *Circ Res*. 2017;120(3):496–501.
- Nicolaides AN, Kakkos SK, Griffin M, Sabetai M, Dhanji S, Tegos T, et al. Severity of asymptomatic carotid stenosis and risk of ipsilateral hemispheric ischaemic events: results from the ACSRS study. *Eur J Vasc Endovasc Surg*. 2005;30(3):275–84.
- Pessin MS, Hinton RC, Davis KR, Duncan GW, Roberson GH, Ackerman RH, et al. Mechanisms of acute carotid stroke. *Ann Neurol*. 1979;6(3):245–52.
- Mechtouff L, Rasclé L, Crespy V, Canet-Soulas E, Nighoghossian N, Millon A. A narrative review of the pathophysiology of ischemic stroke in carotid plaques: a distinction versus a compromise between hemodynamic and embolic mechanism. *Ann Transl Med*. 2021;9(14):1208.
- Fabiani I, Palombo C, Caramella D, Nilsson J, De Caterina R. Imaging of the vulnerable carotid plaque: role of imaging techniques and a research agenda. *Neurology*. 2020;94(21):922–32.
- Mayer L, Grams A, Freyschlag CF, Gummerer M, Knoflach M. Management and prognosis of acute extracranial internal carotid artery occlusion. *Ann Transl Med*. 2020;8(19):1268.
- Chalos V, van der Ende NAM, Lingsma HF, Mulder M, Venema E, Dijkland SA, et al. National Institutes of Health Stroke Scale: an alternative primary outcome measure for trials of acute treatment for ischemic stroke. *Stroke*. 2020;51(1):282–90.
- Wilson JT, Hareendran A, Grant M, Baird T, Schulz UG, Muir KW, et al. Improving the assessment of outcomes in stroke: use of a structured interview to assign grades on the modified Rankin Scale. *Stroke*. 2002;33(9):2243–6.
- Lyden P, Lu M, Jackson C, Marler J, Kothari R, Brott T, et al. Underlying structure of the National Institutes of Health Stroke Scale: results of a factor analysis. *NINDS tPA Stroke Trial Investigators*. *Stroke*. 1999;30(11):2347–54.
- Unger T, Borghi C, Charchar F, Khan NA, Poulter NR, Prabhakaran D, et al. 2020 International Society of Hypertension Global Hypertension Practice Guidelines. *Hypertension*. 2020;75(6):1334–57.
- Agabiti Rosei E, Salvetti M. Management of hypercholesterolemia, appropriateness of therapeutic approaches and new drugs in patients with high cardiovascular risk. *High Blood Press Cardiovasc Prev*. 2016;23(3):217–30.
- Shaw JE, Zimmet PZ, McCarty D, de Courten M. Type 2 diabetes worldwide according to the new classification and criteria. *Diabetes Care*. 2000;23(Suppl 2):B5–10.
- Manczuk M, Cedzynska M. Features of ex-smokers. In: Patel VB, Preedy VR, editors. *Handbook of substance misuse and addictions: from biology to public health*. Cham: Springer International Publishing; 2022. p. 851–71.
- van Velzen TJ, Kuhrij LS, Westendorp WF, van de Beek D, Nederkoorn PJ. Prevalence, predictors and outcome of carotid stenosis: a sub study in the Preventive Antibiotics in Stroke Study (PASS). *BMC Neurol*. 2021;21(1):20.
- Wardlaw JM, Lewis S. Carotid stenosis measurement on colour Doppler ultrasound: agreement of ECST, NASCET and CCA methods applied to ultrasound with intra-arterial angiographic stenosis measurement. *Eur J Radiol*. 2005;56(2):205–11.

20. Muscari A, Bonfiglioli A, Magalotti D, Puddu GM, Zorzi V, Zoli M. Prognostic significance of carotid and vertebral ultrasound in ischemic stroke patients. *Brain Behav.* 2016;6(6): e00475.
21. Soliman RH, Oraby MI, Fathy M, Essam AM. Risk factors of acute ischemic stroke in patients presented to Beni-Suef University Hospital: prevalence and relation to stroke severity at presentation. *Egypt J Neurol Psychiatr Neurosurg.* 2018;54(1):8.
22. Zhang L, Ma J, Wang M, Zhang L, Sun W, Ji H, et al. The association between national institutes of health stroke scale score and clinical outcome in patients with large core infarctions undergoing endovascular treatment. *Neurol Therapy.* 2024;13(3):563–81.
23. Lip GYH, Lane DA, Lenarczyk R, Boriani G, Doehner W, Benjamin LA, et al. Integrated care for optimizing the management of stroke and associated heart disease: a position paper of the European Society of Cardiology Council on Stroke. *Eur Heart J.* 2022;43(26):2442–60.
24. Dempsey RJ, Vemuganti R, Varghese T, Hermann BP. A review of carotid atherosclerosis and vascular cognitive decline: a new understanding of the keys to symptomology. *Neurosurgery.* 2010;67(2):484–93; discussion 93–4.
25. Schultz WM, Kelli HM, Lisko JC, Varghese T, Shen J, Sandesara P, et al. Socioeconomic status and cardiovascular outcomes: challenges and interventions. *Circulation.* 2018;137(20):2166–78.
26. Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WB, et al. Heart disease and stroke statistics—2012 update. *Circulation.* 2012;125(1):e2–220.
27. Hartkamp NS, Hendrikse J, De Cocker LJ, de Borst GJ, Kappelle LJ, Bokkers RP. Misinterpretation of ischaemic infarct location in relationship to the cerebrovascular territories. *J Neurol Neurosurg Psychiatry.* 2016;87(10):1084–90.
28. Ismail A, Ravipati S, Gonzalez-Hernandez D, Mahmood H, Imran A, Munoz EJ, et al. Carotid artery stenosis: a look into the diagnostic and management strategies, and related complications. *Cureus.* 2023;15(5): e38794.

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