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Original Research Article

COMPARISON OF SPOTTY CALCIFICATIONS ON CERVICO-CEREBRAL COMPUTED TOMOGRAPHY ANGIOGRAPHY IN PATIENTS WITH AND WITHOUT ISCHEMIC STROKE

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ABSTRACT

Background: *Ischemic stroke is a major global cause of morbidity and mortality. Cervico-cerebral computed tomography angiography (CTA) allows early visualization of vascular changes, such as spotty calcifications and small, focal calcium deposits within arterial walls, considered indicators of plaque vulnerability and stroke risk.* ***Objective:*** *To compare the mean number and distribution of spotty calcifications on CTA among patients with ischemic stroke and those without stroke but with carotid atherosclerosis in the Pakistani population.* ***Study Design:*** *Case–control study.* ***Setting:*** *Department of Diagnostic Radiology, Chaudhry Pervaiz Elahi Institute of Cardiology (CPEIC), Multan, Pakistan.* ***Duration of Study:*** *January to December 2024.* ***Methods:*** *Sixty participants aged 40–65 years were enrolled, including 30 patients with confirmed ischemic stroke (cases) and 30 age- and sex-matched controls with carotid atherosclerosis but no prior stroke. Cervico-cerebral CTA was performed using a 256-slice multidetector scanner. Spotty calcifications were defined as calcified plaques <3 mm in length and <90° arc with attenuation ≥130 Hounsfield Units. The number and vascular distribution of calcifications were assessed and compared using an independent-samples t-test. Data analysis was conducted in SPSS version 25, with a p-value ≤ 0.05 considered statistically significant.* ***Results:*** *The mean age of participants was 56.8 ± 6.9 years, with males comprising 63.3%. The mean number of spotty calcifications was significantly higher in ischemic stroke patients (6.9 ± 4.2) than in controls (3.0 ± 2.8; p < 0.001). The carotid bifurcation was the most frequent site of calcification, followed by the carotid siphon and middle cerebral artery (MCA) segments. Stroke patients showed significantly higher calcification counts in LMCA1, RMCA1, and the basilar artery (p < 0.05). Subgroup analysis revealed a higher calcification burden in stroke patients with diabetes mellitus (p = 0.002), hypertension (p = 0.003), and smoking history (p = 0.004).* ***Conclusion:*** *Spotty calcifications are significantly more prevalent and widely distributed in patients with ischemic stroke compared to those without stroke, particularly at the carotid bifurcation and MCA segments. Their strong association with diabetes, hypertension, and smoking suggests that spotty calcifications on CTA may serve as early imaging biomarkers for stroke risk, aiding in timely prevention and intervention strategies in the Pakistani population.*

Keywords: Ischemic Stroke; Computed Tomography Angiography; Spotty Calcifications; Carotid Atherosclerosis; Vascular Imaging

INTRODUCTION

Ischemic stroke is a leading cause of morbidity and mortality worldwide, significantly impacting public health. Recent advancements in imaging techniques, particularly computed tomography angiography (CTA), have enhanced our understanding of cerebrovascular diseases, enabling earlier and more accurate Diagnosis of ischemic stroke and its risk factors. Among the many factors contributing to ischemic stroke, the presence of calcifications in the cervico-cerebral arteries has attracted increasing attention. Spotty calcifications, defined as small, dispersed calcified plaques seen on imaging, have been proposed as potential indicators of vulnerable atherosclerotic plaques and correlate with the risk of ischemic events.

The existing literature highlights an association between spotty calcification and stroke risk. Liu et al. reported a higher incidence of spotty calcifications in stroke patients compared to those without a history of stroke, indicating that such calcifications may serve as predictive markers for ischemic events (1). Similarly, Homssi et al. demonstrated that, even in non-stenotic carotid plaques, spotty calcifications were significantly associated with ipsilateral ischemic stroke, underscoring their role in predisposing patients to vascular events (2). In a systematic review, Li et al. confirmed the potential of intracranial artery calcifications as independent predictors of ischemic stroke, suggesting that the extent of calcification could reflect the severity of atherosclerosis and plaque instability (3).

The association between calcification and cerebrovascular risk has been highlighted by studies examining distinct patterns of calcification. For example, Pakizer et al. found that while spotty calcifications could indicate some risk, other plaque characteristics were more strongly associated with ischemic events, offering a nuanced perspective on plaque pathology (4). This understanding of plaque characteristics is crucial for refining risk stratification in stroke and related cerebrovascular events.

Furthermore, recent findings suggest a broader implication of spotty calcifications in predicting additional patient outcomes. Zheng et al. found that the presence of spotty calcification in cervicocephalic arteries was linked not only to ischemic strokes but could also correlate with coronary atherosclerosis, thus influencing cardiovascular health more broadly (5). Corroborating this, Yang et al. indicated that calcifications in vertebral arteries may reflect systemic atherosclerotic processes, potentially linking cerebrovascular health to metabolic conditions such as diabetes (6).

A deeper understanding of the interplay between spotty calcifications and ischemic strokes could significantly improve clinical practice, especially when using high-resolution CTA to assess the cervical and cranial vascular architecture. Assessing these factors is particularly salient for the Pakistani population, where the incidence of stroke

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remains alarmingly high due to increasing rates of hypertension, diabetes, and obesity. In this context, identifying spotty calcifications not only enhances individual risk assessment but also informs public health strategies to mitigate stroke burden in Pakistan.

Thus, the existing body of research provides substantial evidence connecting spotty calcifications in the cervico-cerebral arteries with ischemic stroke risk. Understanding these associations could facilitate better prediction models for stroke risk and improve therapeutic strategies, particularly in populations with distinct epidemiological profiles such as that of Pakistan. Further studies are warranted to establish standardized protocols for assessing calcifications and their implications for stroke risk across diverse populations.

METHODOLOGY

The present study was conducted in the Department of Diagnostic Radiology at the Chaudhry Pervaiz Elahi Institute of Cardiology (CPEIC), Multan, Pakistan, following ethical approval from the institutional review committee from January 2024 to December 2024. This case–control study was designed to compare the mean number of spotty calcifications on cervico-cerebral computed tomography angiography (CTA) in patients with ischemic stroke and those without stroke but with carotid atherosclerosis. The study was carried out over a period of six months after approval of the research synopsis. A total of 60 participants were included in the study, comprising 30 patients with confirmed acute ischemic stroke (cases) and 30 participants without a history of stroke but with positive carotid atherosclerosis detected on Doppler ultrasonography (controls). The sample size was determined using Open Epi software based on previous research findings reporting mean spotty calcifications of 6.83 ± 4.34 among stroke patients and 2.98 ± 2.87 among controls. Assuming a power of 80% and a 95% confidence level, the calculated sample size was 60, equally divided between the two groups. A non-probability consecutive sampling technique was used to select participants to ensure adequate representation within the study timeframe.

Participants aged 40-65 years, regardless of gender, were eligible for inclusion. The case group consisted of patients diagnosed with acute ischemic stroke in the Department of Neurology, confirmed by CT brain with contrast showing a hypodense area corresponding to a vascular territory and clinical evidence of focal neurological deficit lasting more than 24 hours. These patients were contacted within 1 week of discharge for cervico-cerebral CT angiography. The control group included healthy participants with no history of stroke but with evidence of carotid atherosclerosis—defined as plaque formation or intimal thickening greater than 1 mm—detected during annual carotid Doppler screening. Exclusion criteria included patients with a history of previous stroke, carotid artery dissection, vasculitis, or other known cerebrovascular anomalies that could interfere with imaging interpretation. Written informed consent was obtained from all participants before inclusion in the study.

For all participants, baseline demographic and clinical information, including age, gender, diabetes mellitus, hypertension, smoking status, chronic kidney disease, and family history of stroke, was recorded using a structured proforma. Diabetes mellitus was considered present if the patient had been diagnosed and on hypoglycemic therapy for at least one year. Hypertension was considered positive if the participant was on antihypertensive medication for a minimum of one year. Smoking status was defined as a history of smoking ten or more cigarettes per day for at least two consecutive years, while chronic kidney disease was considered positive if the patient had been on renal replacement therapy for a minimum of one year. Family history of stroke was determined based on self-reporting and confirmation from medical records, where applicable.

Cervico-cerebral CT angiography was performed for all study participants using a 256-slice multidetector CT scanner following standard angiographic protocols. Scans were acquired in the axial, coronal, and sagittal planes to comprehensively visualize the arterial anatomy. All images were evaluated by a consultant radiologist with more than 5 years of post-fellowship experience in neuroimaging, who was blinded to participants' clinical status to minimize observer bias. Spotty calcifications were defined as discrete regions with a CT attenuation value of ≥130 Hounsfield Units, with a length below 3 mm and an arc of calcium deposition below 90° in the vessel wall. The total number of spotty calcifications was recorded for each patient, along with their anatomical locations. Eleven vascular segments known to be commonly affected by atherosclerosis were evaluated, including both carotid and vertebrobasilar circulations. In the carotid system, these segments comprised the left and right common carotid artery bifurcations (Lbi and Rbi), left and right carotid siphons (Lsi and Rsi), and the first segments of the left and right middle cerebral arteries (LMCA1 and RMCA1). In the vertebrobasilar system, the first and fourth segments of both vertebral arteries (LV1, RV1, LV4, RV4) and the basilar artery were examined. The number and distribution of spotty calcifications in these arterial territories were carefully documented.

All data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 25. Quantitative variables, such as age and the number of spotty calcifications, were expressed as mean ± standard deviation. In contrast, qualitative variables, such as gender, diabetes, hypertension, smoking, chronic kidney disease, and family history of stroke, were summarized as frequencies and percentages. The normality of numerical data was assessed using the Shapiro–Wilk test. The independent-samples t-test was used to compare the mean number of spotty calcifications between the case and control groups, with p-values ≤ 0.05 considered statistically significant. Stratified analyses were further conducted to explore the influence of potential confounding factors such as age, gender, diabetes, hypertension, chronic kidney disease, smoking, and family history of stroke on the relationship between spotty calcifications and stroke status. Post-stratification comparisons were performed using the independent sample t-test to determine statistical significance within subgroups.

RESULTS

A total of 60 participants were included in the study: 30 cases (patients with ischemic stroke) and 30 controls (patients without stroke but with carotid atherosclerosis). The mean age of participants was 56.8 ± 6.9 years, ranging from 42 to 65 years. The mean age among stroke cases was 58.1 ± 6.4 years, while that among controls was 55.5 ± 7.1 years. Males comprised 63.3% (n = 38) of the total cohort, whereas females accounted for 36.7% (n = 22).

Comorbid conditions such as hypertension (56.7%), diabetes mellitus (45.0%), and smoking (33.3%) were more prevalent among stroke cases compared to controls. A positive family history of stroke was found in 26.7% of the cases, while chronic kidney disease was observed in 11.7% overall. (Table 1)

The mean number of spotty calcifications (SC) was significantly higher in stroke patients (6.9 ± 4.2) than in controls (3.0 ± 2.8;$p<$ 0.001). This difference indicates a strong association between ischemic stroke and higher SC burden, consistent with the hypothesized vascular calcification pattern among Pakistani patients. (Table 2)

Segment-wise analysis showed that the carotid bifurcation (left and right) was the most frequent site of calcification in both groups, followed by the siphon and middle cerebral artery (MCA1) segments. Stroke patients exhibited a significantly higher number of calcifications in LMCA1, RMCA1, and the basilar artery segments compared to controls ($p<0.05$). (Table 3)

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After stratification, the number of spotty calcifications remained significantly higher in stroke cases across subgroups of diabetic (p = 0.002), hypertensive (p = 0.003), and smoker (p = 0.004) participants. The association was not statistically significant among participants without these comorbidities (p > 0.05). (Table 4)

Table 1: Demographic Characteristics of Study Participants (n = 60)

Variable	Overall (n=60)	Stroke Cases (n=30)	Controls (n=30)	p-value
Age (years)	56.8 ± 6.9	58.1 ± 6.4	55.5 ± 7.1	0.17
Gender				0.42
Male	38 (63.3%)	20 (66.7%)	18 (60.0%)	
Female	22 (36.7%)	10 (33.3%)	12 (40.0%)	
Diabetes mellitus	27 (45.0%)	16 (53.3%)	11 (36.7%)	0.19
Hypertension	34 (56.7%)	20 (66.7%)	14 (46.7%)	0.10
Smoking	20 (33.3%)	13 (43.3%)	7 (23.3%)	0.09
Family history of stroke	11 (18.3%)	8 (26.7%)	3 (10.0%)	0.09
Chronic kidney disease	7 (11.7%)	5 (16.7%)	2 (6.7%)	0.22
Independent sample t-test and chi-square test applied; significance set at p ≤ 0.05.				

Table 2: Comparison of Mean Number of Spotty Calcifications Between Groups

Group	n	Mean ± SD	t-value	p-value
Stroke Cases	30	6.9 ± 4.2	3.92	<0.001
Controls	30	3.0 ± 2.8		

Table 3: Segment-wise Distribution of Spotty Calcifications

Vascular Segment	Stroke Cases (n=30)	Controls (n=30)	p-value
Left bifurcation (Lbi)	23 (76.7%)	15 (50.0%)	0.03
Right bifurcation (Rbi)	21 (70.0%)	13 (43.3%)	0.04
Left siphon (Lsi)	16 (53.3%)	8 (26.7%)	0.03
Right siphon (Rsi)	14 (46.7%)	7 (23.3%)	0.05
LMCA1	12 (40.0%)	4 (13.3%)	0.02
RMCA1	10 (33.3%)	3 (10.0%)	0.03
LV1	9 (30.0%)	6 (20.0%)	0.38
RV1	7 (23.3%)	5 (16.7%)	0.52
LV4	8 (26.7%)	4 (13.3%)	0.21
RV4	7 (23.3%)	3 (10.0%)	0.19
Basilar artery	11 (36.7%)	2 (6.7%)	0.006

Table 4: Stratification of Mean Spotty Calcifications According to Risk Factors

Risk factor	Stroke Cases (Mean ± SD)	Controls (Mean ± SD)	p-value
Diabetes mellitus	7.3 ± 4.0	3.5 ± 2.6	0.002
Hypertension	6.8 ± 4.1	3.2 ± 2.9	0.003
Smokers	7.1 ± 4.5	3.1 ± 2.7	0.004
Non-smokers	6.5 ± 3.8	2.9 ± 2.6	0.005
Family history positive	7.6 ± 4.3	3.4 ± 3.0	0.01
CKD positive	8.2 ± 4.8	4.1 ± 2.9	0.03

DISCUSSION

In this study, we investigated the prevalence and distribution of spotty calcifications (SC) in cervico-cerebral arteries among patients with ischemic stroke compared to controls with carotid atherosclerosis but without stroke. Our findings reveal that the mean number of SC is significantly higher in stroke patients, aligning with prior research that establishes a link between vascular calcification and cerebrovascular events.

The age distribution of participants in both groups shows a mean age of 56.8 years, with stroke patients slightly older at 58.1 years. Similar findings have been noted in studies by Homssi et al., where age was identified as a significant factor contributing to ischemic stroke risk (7). The predominance of males (63.3%) in our cohort is consistent with literature indicating a higher stroke incidence among men, which Homssi et al. also corroborated in their studies on stroke demographics and risk factors [7]. The higher prevalence of hypertension (56.7%), diabetes mellitus (45.0%), and smoking (33.3%) in stroke patients mirrors findings from Xue et al., who reported these comorbidities as critical stroke risk factors (8).

Our study observed that the mean number of SC in stroke patients (6.9 ± 4.2) is considerably higher than in controls (3.0 ± 2.8), with a p-value < 0.001, suggesting a robust association between ischemic stroke and SC burden. This observation is supported by Pakizer et al., who noted the correlation between carotid plaques with calcifications and increased stroke risk, reinforcing our findings (9).

The segment-wise distribution of calcifications in our study demonstrates the carotid bifurcation as the most affected site, which aligns with findings reported by Jeon et al. in their research on carotid imaging and stroke (10). Specifically, we found significantly greater calcification in the left and right carotid bifurcation and several segments of the middle cerebral artery (MCA) in stroke patients compared to controls. This pattern of segmental preference has been echoed in studies that highlight that bifurcation regions are particularly vulnerable to atherosclerotic changes and subsequent ischemic events (11).

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Interestingly, we also noted a higher number of calcifications in the left middle cerebral artery (LMCA) and right middle cerebral artery (RMCA) segments among stroke patients. Our findings of localized calcification align with those of Homssi et al., who observed that atherosclerotic plaques with SC in these regions were associated with ischemic events, thereby establishing a critical correlation between anatomical locations and vascular risk in stroke populations (7).

Further stratification of the data emphasized that stroke patients with concurrent diabetes mellitus, hypertension, and smoking demonstrated significantly higher mean SC counts. Pakizer et al. have similarly highlighted the cumulative effect of these risk factors in exacerbating vascular pathology and increasing stroke likelihood (9). Our results demonstrate the importance of managing these conditions to potentially mitigate stroke risk, as supported by prior studies showing that risk factor modification can improve outcomes (12).

Interestingly, when assessing participants without these comorbid conditions, the differences in SC counts between stroke cases and controls were not statistically significant, suggesting that the presence of these comorbidities exacerbates the risk of ischemic stroke by amplifying the calcification burden. This nuanced understanding can help healthcare providers tailor prevention strategies, particularly in the Pakistani context, where these risk factors are prevalent.

Thus, the findings from our cohort study provide evidence linking spotty calcifications in cervico-cerebral arteries to ischemic stroke, underscoring the significance of these vascular changes as potential predictive markers of stroke risk. The demographic parallels with previous studies suggest that similar vascular pathologies are at play across diverse populations. As such, screening for SC in high-risk individuals, particularly in regions with elevated rates of ischemic stroke, like Pakistan, can be pivotal in stroke prevention strategies.

Our study advocates further investigation into the characteristics of vascular calcifications and their direct implications for stroke outcomes, thereby contributing to a more refined understanding of stroke pathophysiology across ethnic groups.

CONCLUSION

Spotty calcifications on cervico-cerebral CTA were significantly more common and extensive in patients with ischemic stroke than in controls with carotid atherosclerosis. The findings suggest that these calcifications may represent vulnerable plaques and serve as predictive markers of stroke risk, particularly among individuals with diabetes, hypertension, or smoking habits. Incorporating spotty calcification assessment into routine vascular imaging could enhance early detection of stroke risk and guide targeted preventive strategies in high-risk populations, such as Pakistan.

DECLARATIONS

Data Availability Statement

All data generated or analysed during the study are included in the manuscript.

Ethics approval and consent to participate

Approved by the department Concerned. (IRBEC)

Consent for publication

Approved

Funding

Not applicable

CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTION

AYESHA RAZZAQ (PGR)
Conception of Study, Development of Research Methodology Design, Study Design, Review of manuscript, and final approval of manuscript.
Manuscript drafting.
NAZAHAT PASHA (Assistant Professor)
Manuscript revisions, critical input.
Study Design, Review of Literature.
ZUNAIRA SADIQ (PGR)
Conception of Study, Final approval of manuscript.
RAWISH FATIMA (HO)
Data entry, data analysis, and drafting an article
MIAN ADNAN ASLAM JAVAID (Statistical Analyst)
Performed statistical analysis, interpreted data, and contributed to the preparation and final review of the manuscript.

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