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Original research article

Similar angiographic appearance on CT-Angiography of symptomatic and asymptomatic carotid near-occlusion

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Abstract
Background and objectives: The study sought to examine how symptomatic and asymptomatic carotid near-occlusion appeared on angiography.
Methods: A blinded study evaluated 635 patients for consecutive CTAs at NRI Medical College/Hospital from June 2021 to May 2022 for estimation of symptomatic and asymptomatic carotid near occlusion.
Results: 380 (60%) of the 635 patients (50% carotid stenosis) were symptomatic. Both symptomatic (112, 30%) and asymptomatic (50, 25%) cases of near-occlusion shared the same prevalence. Between symptomatic and asymptomatic cases with near-occlusion, the angiographic appearance was remarkably similar: the mean stenosis lumen diameter (0.7 mm), distal ICA diameter (2.1 mm), and ECA ratio (0.79) were the same in both groups. The average ICA ratio (0.46 and 0.48) and fraction of full collapse (46% and 40%) were fairly similar.
Conclusion: The primary conclusion of this study was that there was no change in the angiographic appearance of near-occlusions between symptomatic patients and asymptomatic individuals.
Keywords: CT-angiography, carotid stenosis, carotid near-occlusion, stroke.

Introduction

A profound carotid stenosis called carotid near-occlusion reduces the size of the ICA beyond the stenosis (collapses it). There are two categories of near-occlusions: one with partial collapse and the other with greater severity (with full collapse) [1]. Full collapse has been defined as a prognosis-based threshold (distal ICA diameter 2.0 mm and/or ICA ratio 0.42), with full collapse being associated with a high risk of early stroke recurrence (31% within 14 days of the presenting event) and full collapse being associated with a low risk (4%) of early stroke recurrence [1, 2]. There is no study that we could find that contrasted the angiographic features of symptomatic versus asymptomatic near-occlusions. The comparison of the angiographic appearance of symptomatic and asymptomatic near-occlusions was the main goal of this investigation. According to the study's premise, compared to asymptomatic individuals with 50% carotid stenosis, symptomatic patients with 50% carotid stenosis experience near-occlusion more frequently and with greater severity (smaller remaining stenosis lumen and distal ICA) [2, 3].

Material and Methods

All individuals with extra-cranial carotid stenosis of at least 50% were included. In our evaluation of the medical records, we identified people as symptomatic if they had an ischemic cerebrovascular event within six months after the examination that fit with the distribution of a stenosed artery. Reviewers were blinded to the degree of stenosis but were aware of the side of stenosis because it was necessary for data synthesis. A neurologist with substantial carotid stenosis experience evaluated all cases with ambiguous symptomatic status. Two observers were blinded to one another and to the clinical data, made the near-occlussion diagnosis as previously described, with an inter-rater observer kappa of 0.80. Discussions were used to settle disagreements. When the distal ICA collapsed, feature interpretation to diagnose a near-occlusion was employed, with the most likely explanation being a severe proximal stenosis. It was important to avoid mistaking coincident stenosis with anatomical variance for near-occlusion [3]. The diameters of the stenosis lumen, distal ICA (located far from the stenosis), and ECA were measured by one observer (EJ) (just before its terminal bifurcation behind the jaw). Without additional magnification, callipers were methodically positioned in the centre of the "fuzzy edge." Distal ICA diameter 2.0 mm and/or an ICA ratio 0.42 were used to determine full collapse. For patients with symptoms, the index side was identified as their problematic side, while for those with no symptoms, it was identified as having the most severe stenosis. We used mean, standard deviation, median, and interquartile range when appropriate. In bivariate analysis, we applied the t-test and 2-test [3, 4]. All variables having p 0.1 in the baseline assessment were employed as co-variates in multivariable analysis (linear and binary logistic regression). Statistics were deemed significant at p 0.05. For the two analyses involving multiple testing

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(Tables 1 and 2), a Bonferroni-corrected p-value threshold was computed as 0.05/m, where "m" is the number of tests. Calculations were performed using IBM SPSS 28.0.

Result

Table 1: Baseline Characteristics and CT Findings in the Entire Study Population

		Asymptomatic (*n*=255)	Symptomatic (n = 380)	pa
Age mean (SD)		65 (8)	65 (8)	0.84
Women n(%)		97 (38)	116 (31)	0.051
Previous myocardial infarction n(%) b		65 (25)	69 (18)	0.11
Current angina *n*(%)b		47 (18)	51 (13)	0.13
Current heart failure *n*(%)b		20 (8)	22 (6)	0.35
Current claudication *n*(%)b		21 (8)	24 (6)	0.21
Previous arterial revascularization *n*(%)b		90 (35)	65 (17)	<0.001
Atrial fibrillation *n*(%)b		35 (14)	36 (9)	0.046
Current smoking *n*(%)b		49 (19)	71 (18)	0.68
Hypertension *n*(%)b,c		240 (94)	345 (91)	0.011
Diabetes *n*(%)b		55 (21)	93 (24)	0.71
Total cholesterol mean (SD)d		4.3 (1.6)	4.6 (1.2)	0.28
LDL cholesterol mean (SD)d		2.9 (1.1)	2.8 (0.7)	0.024
HDL cholesterol mean (SD)d		1.28 (0.50)	1.17 (0.30)	0.007
Referred from other hospital *n*(%)		160 (60)	289 (76)	<0.001
Presenting event	Stroke *n*(%)	NA	192 (51)	–
	TIA *n*(%)	NA	133 (35)	
	Retinal *n*(%)	NA	50 (13)	
Delay presenting event CTA median (IQR) CTA indication	Cerebrovascular event, other cause n(%) e	NA 120 (47)	2 (0-6) NA	– –
	Suspected cerebrovascular event *n*(%)f	70 (28)	NA	
	Other *n*(%)g	60 (24)	NA	
CTA findings on index side	Conventional stenosis *n*(%)	200 (78)	275 (72)	0.24h
	Near-occlusion without full collapse *n*(%)	29 (11)	60 (16)	
	Near-occlusion with full collapse *n*(%)	23 (9)	40 (11)	
	Degree of conventional stenosis mean (SD)	62 (9)	65 (10)	<0.001
≥50% stenosis or occlusion on contralateral side *n* (%)		71 (27)	110 (29)	0.79

Of the 635 people we included, 380 (60%) had symptoms of carotid stenosis greater than 50%. Although some of the differences may have been false positives as a result of multiple testing, there were several baseline variations across the symptom groups. There were no differences between the three stenosis groups when comparing symptomatic and asymptomatic instances (bivariate p = 0.23; multivariable p = 0.38; data not shown), nor between near-occlusion and standard 50% stenosis (p=0.12; multivariable p=0.28; data not shown).

Table 2: CT findings among cases with near-occlusion

	Asymptomatic (*n*=50)	Symptomatic (*n*=112)	Bivariate *p*[a]	Multivariable *p*b
Stenosis Diameter mm mean(SD)c	0.7 (0.2)	0.7 (0.2)	0.75	0.59
Reduce opacity in stenosis n(%) c,d	25 (49)	48 (49)	1.0	0.27
Distal ICA diameter mm mean (SD)	2.1 (1.1)	2.1 (1.0)	0.86	0.29
ICA ratio mean (SD)e	0.46 (0.24)	0.48 (0.34)	0.63	0.71
ECA ratio mean(SD)	0.79 (0.49)	0.69 (0.61)	0.99	0.63
Full collapse *n*(%)	25 (46)	45 (40)	0.87	0.35
≥50% stenosisor occlusion on contralateral side *n*(%)	19 (38)	33 (29)	0.38	0.67

In bivariate and multivariable studies of near-occlusions, there were no differences in any parameter of how severe the near-occlusion was between symptomatic and asymptomatic instances.

Discussion

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Uncertainity in cause of strokes to occur repeatedly when there is a near-occlusion and complete collapse is estimated. A hypothesis involves cerebral hypoperfusion. Full collapse results in a significant reduction in blood flow through the ICA since ICA diameter is highly correlated with ICA flow. The dilatation of cerebral arterioli, however, will help the autoregulation system enlist collaterals. Assumably, those with excellent collateral capacity will require less dilatation than those with poor capacity [4, 5]. TCD and SPECT can be used to determine how much dilatation capacity is still available (cerebrovascular reserve). Hypoperfusion of the cerebral cortex is indicated by no more reserves (exhausted). In a SPECT research with 15 near-occlusions, some of the near-occlusions had exhausted cerebrovascular reserve and some had merely diminished reserve, but the analysis did not differentiate between symptomatic and asymptomatic near-occlusions or between with and without full collapse.
In a TCD research with 50 symptomatic near-occlusions, 22 (44%) had diminished and 11 (22%) had exhausted cerebrovascular reserve, with similar results for both full collapse and partial collapse. Contrarily, Greiner et al. reported in brief that despite 53 near-occlusions (apparently all with full collapse), the cerebrovascular reserve was always diminished but never exhausted [4, 5, 6]. Due to the frequent collateral recruitment seen in near-occlusions, some dilating is anticipated. However, many of these instances should have depleted cerebrovascular reserve if hypoperfusion is the cause of stroke in symptomatic full collapse, which occurs manifestly more frequently than both asymptomatic near-occlusion with full collapse and symptomatic near-occlusion without full collapse. This has never, however, been demonstrated. In fact, evidence suggests that the contrary is true. Further supporting this, we report no distinction in angiographic appearance between symptomatic and asymptomatic near-occlusion. It would make sense that some instances might become symptomatic as a stenosis proceeds to full collapse if cerebral hypoperfusion was the aetiology [7, 8]. If that's the case, there ought to be a lot fewer asymptomatic near-occlusions with full collapse, which we dispute. We also discovered no difference in the severity of the near-occlusion when evaluated on a spectrum, indicating that this was not a coincidental result of an unlucky threshold for full collapse (distal ICA diameter and ICA ratio) [8, 9]. Our research thus contributes to the expanding body of evidence showing that cerebral hypoperfusion is not the cause of stroke in near-occlusion with full collapse and more research is necessary. Assessment of the embolic mechanism should be part of these evaluations as well, as artery ligation has been suggested as a potential treatment for near-occlusion with full collapse if the mechanism is embolic [9].
The breadth of the sample and the thorough assessment of stenosis severity and symptom state were strengths of this study. Lack of a test for cerebrovascular reserve and a potential selection bias among asymptomatic stenoses due to the fact that many were found to be caused by cerebrovascular illness and that more symptomatic patients than asymptomatic patients were sent to the clinic are weaknesses.

Conclusion: Symptomatic and asymptomatic carotid near-occlusions seem the same on an angiogram, in conclusion. The pathophysiology of carotid near-occlusion is furthered by these studies.

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