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Comparative Analysis of Symptomatic and Asymptomatic Carotid Plaques in Patients with Bilateral Mild Carotid Stenosis (30% - 50%)

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Abstract

Background: Nonstenosing carotid plaques are increasingly recognized as a potential cause of ischemic stroke, yet their contribution remains underappreciated in routine etiological classification. **Objective**: To compare morphological characteristics of carotid plaques between symptomatic and asymptomatic sides in patients with bilateral mild carotid stenosis (30% - 50%) and to explore their association with ischemic stroke occurrence. **Methods**: We conducted a retrospective, descriptive, single-center study in the Neurology Department of Ibn Rochd University Hospital, Casablanca, between January 2022 and December 2024. Patients presenting with acute ischemic stroke and bilateral carotid stenosis ranging from 30% to 50% were included. Carotid plaque characteristics were assessed primarily using computed tomography angiography (CTA), supplemented by Doppler ultrasound analysis for hemodynamic and echogenicity assessment. Stenosis severity was measured according to NASCET criteria. **Results**: Among 340 ischemic stroke patients, 60 were identified as having probable large-artery atherosclerotic involvement after etiological evaluation, and 10 fulfilled the inclusion criteria. The mean age was 68.5 years, with a male predominance (70%). Hypertension (80%) and diabetes mellitus (60%) were the most prevalent vascular risk factors. In this paired within-patient descriptive comparison, vulnerable plaque features appeared more frequent on the symptomatic side than on the contralateral asymptomatic side. Hypoechogenicity was observed in 5/10 symptomatic plaques versus 2/10 asymptomatic plaques, while surface irregularity was present in 5/10 versus 2/10, respectively. Mural thrombus and plaque ulceration were identified exclusively

on symptomatic plaques (2/10 vs 0/10). Ipsilateral recurrence occurred in two patients during follow-up. **Conclusion**: High-risk nonstenosing carotid plaques were more frequently identified ipsilateral to ischemic stroke, suggesting a possible association between plaque vulnerability and stroke occurrence despite mild luminal narrowing. These findings support reconsideration of stroke classification systems that rely exclusively on luminal stenosis severity.

Keywords

Ischemic Stroke, Mild Carotid Stenosis, Plaque Vulnerability, Carotid Atherosclerosis

1. Introduction

Carotid atherosclerosis is a well-established cause of ischemic stroke, traditionally associated with significant luminal stenosis. However, increasing evidence suggests that carotid plaques causing less than 50% stenosis may also play a critical role, particularly when they exhibit high-risk morphological features.

Recent studies have demonstrated that a substantial proportion of ischemic strokes may be associated with ipsilateral nonstenosing carotid plaques, raising concerns about potential misclassification of stroke etiology when relying solely on the degree of stenosis. In particular, vulnerable plaque characteristics such as fibrous cap thinning, intraplaque hemorrhage, and neovascularization may contribute to plaque instability and embolic events [1].

The present study aims to compare plaque morphology between symptomatic and asymptomatic carotid arteries in patients with bilateral mild stenosis (30% - 50%) and to assess their potential role in ischemic stroke occurrence.

2. Methods

This retrospective, descriptive, single-center study was conducted in the Neurology Department of Ibn Rochd University Hospital in Casablanca between January 2022 and December 2024.

Inclusion criteria

Patients were eligible if they:

- presented with acute ischemic stroke confirmed by brain imaging,
- had bilateral carotid stenosis ranging from 30% to 50%,
- underwent supra-aortic CTA,
- and had complete clinical and radiological records available.

Exclusion criteria

Patients were excluded in cases of:

- carotid stenosis greater than 50%,
- unilateral carotid disease,
- cardioembolic source,

- atrial fibrillation,
- arterial dissection,
- vasculitis,
- significant intracranial stenosis,
- incomplete imaging studies,
- poor-quality CTA.

Among the 60 patients identified as having probable large-artery atherosclerotic involvement after etiological evaluation, 50 were excluded because they did not meet the bilateral mild carotid stenosis criterion, had unilateral disease, stenosis greater than 50%, incomplete vascular imaging, or competing stroke mechanisms identified during etiological workup.

Baseline characteristics included age, sex, and vascular risk factors such as hypertension, diabetes mellitus, dyslipidemia, smoking status, and obstructive sleep apnea. Stroke severity at admission was assessed using the National Institutes of Health Stroke Scale (NIHSS).

The symptomatic carotid artery was defined as the artery ipsilateral to the acute ischemic infarct identified on brain imaging.

Competing stroke mechanisms were excluded through standard etiological investigations including electrocardiography, cardiac echocardiography, prolonged rhythm monitoring when indicated, intracranial vascular imaging, and laboratory evaluation according to the TOAST classification system.

Brain imaging was reviewed to determine infarct location. Vascular imaging included CTA of the supra-aortic trunks and extracranial carotid arteries.

Carotid stenosis severity was measured on CTA using NASCET criteria. The percentage of stenosis was calculated by comparing the minimal luminal diameter at the site of maximal narrowing with the normal distal internal carotid artery lumen. Measurements were independently reviewed by experienced neuroradiologists.

Plaque morphology assessment was primarily based on CTA findings, including plaque surface irregularity, ulceration, calcifications, mural thrombus, and degree of stenosis. Doppler ultrasound was additionally used to assess plaque echogenicity and hemodynamic parameters, including flow velocity and spectral broadening.

Clinical and radiological outcomes, including recurrence and vessel recanalization, were also recorded.

The median clinical follow-up duration was 12 months (range: 6 - 18 months). Stroke recurrence was assessed clinically and confirmed by repeat neuroimaging when available. Recanalization was evaluated using follow-up vascular imaging.

Given the small sample size, no inferential statistical analysis was performed and findings should be considered descriptive and exploratory.

3. Results

During the study period, 340 patients were admitted for ischemic stroke, among

whom 60 were identified as having probable large-artery atherosclerotic involvement after etiological evaluation. Of these, 10 patients fulfilled the inclusion criteria of bilateral mild carotid stenosis.

The study population had a mean age of 68.5 years and was predominantly male (70%). Cardiovascular risk factors were highly prevalent, with hypertension observed in 80% of patients and diabetes mellitus in 60%. Dyslipidemia was present in 40% of cases, while 30% of patients had obstructive sleep apnea and 50% were active smokers. A prior history of ipsilateral ischemic stroke or transient ischemic attack was documented in two patients.

The mean NIHSS score at admission was 10, indicating moderate stroke severity. Brain imaging showed that the middle cerebral artery territory was involved in 80% of cases, whereas the remaining patients presented with watershed infarctions.

In this paired within-patient descriptive comparison, vulnerable plaque features appeared more frequent on the symptomatic side than on the contralateral asymptomatic side.

Mural thrombus and plaque ulceration were identified exclusively on symptomatic plaques. An example of a vulnerable symptomatic plaque is shown in Figure 1. Hemodynamic disturbances assessed by Doppler imaging, reflected by increased flow velocities with spectral broadening, were also more frequently observed on the symptomatic side (Table 1).

During follow-up, ipsilateral recurrence occurred in two patients, and one patient demonstrated vascular recanalization on follow-up imaging.

Overall, high-risk morphological features of carotid plaques were more frequently identified on the symptomatic side compared with the contralateral side, supporting a possible association between plaque vulnerability and stroke pathogenesis despite mild luminal narrowing.

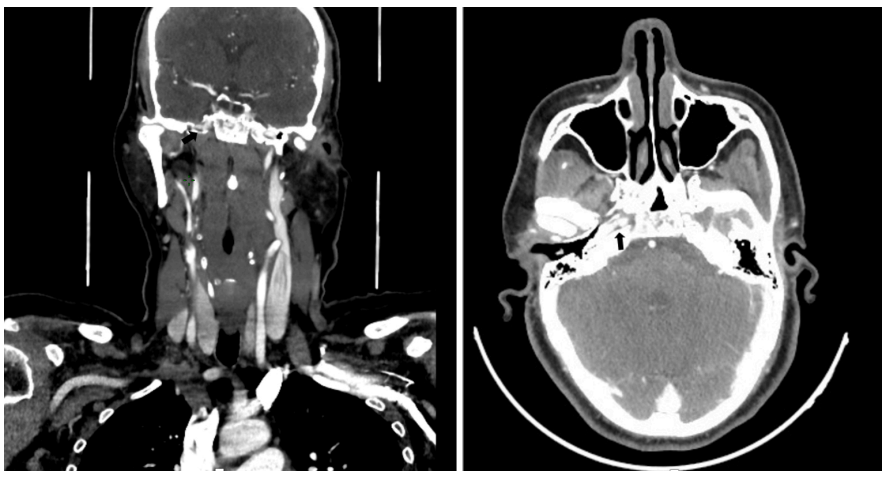

Figure 1. Computed tomography angiography (CTA) of the right internal carotid artery. Coronal (left) and axial (right) images show an atherosclerotic plaque located in the petrous segment (arrows), with irregular margins suggestive of plaque vulnerability. The lesion is associated with mild luminal narrowing, estimated approximately 35%.

Table 1. Comparison of plaque characteristics between symptomatic and asymptomatic carotid arteries in patients with bilateral mild carotid stenosis (30% - 50%). Data are presented as n (%). CTA: computed tomography angiography.

Plaque characteristic	*Symptomatic side* (*n* = 10)	*Asymptomatic side* (*n* = 10)
Hypoechogenicity (*ultrasound*)	5/10 (50%)	2/10 (20%)
Surface irregularity (*CTA*)	5/10 (50%)	2/10 (20%)
Microcalcifications (*CTA*)	6/10 (60%)	4/10 (40%)
Fibrous cap ulceration (*CTA*)	2/10 (20%)	0/10 (0%)
Mural thrombus (*CTA*)	2/10 (20%)	0/10 (0%)
Increased flow velocity with spectral broadening (*Doppler ultrasound*)	4/10 (40%)	1/10 (10%)

4. Discussion

In our study, we observed that high-risk carotid plaques were more frequently present on the ipsilateral side of a recent cerebral infarction compared to the contralateral side. These findings suggest that nonstenosing carotid plaques, even in the setting of mild luminal narrowing, may play a significant role in the occurrence of ischemic stroke.

Our results are consistent with previous studies, including American and Indian series, which have demonstrated a higher prevalence of high-risk carotid plaques ipsilateral to acute brain infarction compared to the contralateral side. This supports the hypothesis that plaque vulnerability, rather than the degree of stenosis alone, is a key determinant in stroke pathophysiology [1] [2].

A substantial proportion of ischemic strokes, particularly those classified as cryptogenic or idiopathic, may be related to mild or nonstenosing carotid atherosclerosis. This can be explained by the presence of advanced plaque lesions that undergo complex structural changes, including calcification, neovascularization, and progressive thinning of the fibrous cap, ultimately leading to plaque instability and rupture. These processes may result in artery-to-artery embolism even in the absence of significant luminal stenosis [3].

From a pathophysiological standpoint, plaque stability is largely determined by the integrity of the fibrous cap, which depends on the balance between pro-inflammatory and anti-inflammatory mechanisms within the plaque. Inflammatory mediators promote extracellular matrix degradation—particularly collagen breakdown—through the activation of matrix metalloproteinases, whereas reparative pathways involving transforming growth factor contribute to collagen synthesis. Disruption of this balance weakens the fibrous cap, increasing the risk of rupture and thromboembolic complications [4].

It is also important to note that some atherosclerotic plaques may undergo outward remodeling, allowing them to remain angiographically nonstenosing despite

significant structural vulnerability. This may explain why certain high-risk plaques are not detected when assessment is based solely on luminal narrowing [5].

With optimal medical therapy, plaque stabilization typically requires several months, usually between 6 and 12 months. In contrast, certain endovascular approaches, such as closed-cell stent placement without balloon angioplasty, may promote faster endothelialization and stabilization of the plaque, potentially reducing the risk of early recurrent embolic events. However, the role of such interventions in patients with mild but vulnerable carotid stenosis remains uncertain [6].

Therefore, randomized controlled trials are needed to evaluate the safety and efficacy of interventional strategies in selected patients with symptomatic, high-risk, and nonstenosing carotid plaques.

5. Limitations

This study has several limitations. First, its retrospective single-center design and very small sample size limit the generalizability of the findings. Second, imaging modalities were not fully standardized across all patients. Third, the absence of inferential statistical analysis prevents establishing causal relationships between plaque morphology and stroke occurrence. Therefore, these findings should be interpreted as exploratory and hypothesis-generating.

6. Conclusion

High-risk carotid plaques with less than 50% stenosis are more frequently observed on the symptomatic side in patients with ischemic stroke, suggesting a causal relationship. Reliance solely on luminal stenosis thresholds may lead to underrecognition of a significant stroke mechanism. Future research should focus on integrating plaque vulnerability into diagnostic and therapeutic algorithms.

Acknowledgements

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Ethical Approval

This retrospective observational study was conducted in accordance with institutional ethical standards and the Declaration of Helsinki. Given the retrospective nature of the study and anonymization of patient data, formal ethical committee approval was waived according to local institutional policy.

Consent

Patient data were anonymized prior to analysis. Written informed consent for publication was obtained when required by institutional policy.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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