

## Correlation of Carotid Atherosclerosis with Different Types of Ischemic Strokes Determined by Ultrasound of Neck Vessels.

Syed Maqbool Ahmad , Irfan Ahmad Bhat , Samia Rashid MD.

### Abstract

#### Aims and objectives

Extra cranial carotid atherosclerosis particularly at the bifurcation and the beginning of the internal carotid artery is a known cause of stroke and transient ischemic attacks. Carotid angiography is invasive and expansive, neck vessel doppler provides adequate information.

#### Methods

Carotid bifurcation of 100 patients with recent stroke or TIA and bilateral plaques were evaluated by B mode real-time ultrasonography.

#### Results

Patients with stroke had total occlusion, narrower minimum residual lumen and thicker plaques on the ipsilateral side, while patients with TIA had more echo lucent plaques and significant longitudinal plaque motion.

**Conclusion** These data demonstrate a significant relationship between carotid artery ultrasound plaque characteristics and type of ischemic cerebrovascular events.

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### Introduction

Extra cranial carotid atherosclerosis particularly at the bifurcation and the beginning of the internal carotid artery is known for decades together as a cause for cerebrovascular events like ischemic stroke and TIA. Hultquist in 1942 described the clinic pathologic correlation in his extensive monograph after studying the entire carotid system in 1400 autopsies[1]. Although atherosclerosis affects almost all the arteries supplying the brain ranging from fine penetrating branches to large extra cranial vessels, it is the carotid bifurcation and the beginning of the internal carotid artery which is the commonest and the most severely affected site[2]. Carotid atherosclerosis accounts for about 5% of ischemic strokes and TIA by virtue of luminal stenosis and plaque related thromboembolism[3]. Clinical examination like carotid bruits and carotid pulse is insensitive for detection of presence and severity of the disease in the neck vessels[4]. Carotid angiography although the reference standard and highly specific and sensitive for detection of luminal stenosis, does not provide as good a data of the plaque characteristics and the arterial wall. It is also invasive expansive and associated with adverse outcomes in 1-2% of patients[5]. B mode real time ultrasonography a non-invasive inexpensive and easily approachable imaging modality provides detailed information of the vessel wall, atherosclerotic plaque and luminal stenosis with enough sensitivity and specificity[6].

### Materials and Methods

This study comprised of about 100 patients either admitted or outpatients of Govt Medical College Srinagar with history of C.T proven stroke or clinically a TIA in the month preceding the B-MODE REAL TIME SCAN. Out of the 100 patients 65 had stroke and 35 had TIA. Only those patients who had bilateral plaques were selected for the study. Cardio embolic stroke was excluded by detailed transthoracic and transesophageal echo. Patients were excluded if they had bilateral stroke

### Author Affiliations

Syed Maqbool Ahmad DM, DNB, FNB;  
Irfan Ahmad Bhat MD, DM; Dr Samia Rashid MD.  
Government Medical College,  
Srinagar-Kashmir.

### Correspondence

Dr. Syed Maqbool Ahmad  
Consultant Cardiology  
Email:- syedmaqbool2000@gmail.com  
(m) 9906723332

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### Keywords

Carotid bifurcation ,atherosclerotic  
plaque ,minimum residual lumen , plaque  
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TIA or if they had a stroke/TIA with undetermined hemispheric involvement. A matched case control design was used to compare clinically affected brain hemisphere (case) with the unaffected contralateral hemisphere (control) with regard to the characteristics of the atherosclerotic plaque and the luminal reduction caused by it. A B- mode ultrasound scan was carried with Philips ATL 7.5-12 MHz transducer with an axial resolution of 0.5mm by experienced sonologists. These sonologists were unaware of the history, physical examination and investigations of the patient. Pulsed Doppler and spectral analysis were used to differentiate external from internal carotid arteries. Carotids were examined bilaterally at the level of bifurcation, internal carotid and common carotid from transverse and longitudinal orientation. Gain setting and continuous angling adjustments were made to optimize the image quality. Following features were looked for.

1. Plaque; was defined as arterial wall lesion projecting into the lumen.
2. Residual lumen diameter; Defined as lumen intima arterial wall boundaries was looked for at minimum residual lumen and the reference point ( a point 6mm above the flow divider),this was to look for extension of the plaque into the internal carotid artery.
3. Lesion width (plaque thickness); Refers to arterial wall thickness and was operationally defined as the distance between lumen intima and media adventitia interface. Total plaque thickness was taken as sum of near and far wall thickness.
4. Ultrasonic reflectivity; Grey scale intensity of the lesions (echogenicity) was assessed as being on average as low(blood like) or intermediate/high (intense bright echogenicity).
5. Plaque motion; Radial wall motion defined as changes in vessel wall diameter during cardiac cycle. It was defined as either present or absent
6. Longitudinal plaque motion; Defined as the apparent distal shift of the plaque axis during cardiac cycle was assessed at the site of minimum residual lumen and recorded as either present or absent.

**Results**

Patients who experienced stroke, out of 65, seven had total occlusion of the ipsilateral carotid(P-Val 0.03) while as only 2 patients out of 35 TIA patients had total

occlusion. Patients with stroke had a residual lumen diameter of 3.61±0.18 on the ipsilateral side and 4.61±0.15 on the contralateral asymptomatic side.

Distribution of age and sex of the patients who participated in the study

| Sex    | Number%   | Age(years)mean ±s.d | P - val |
|--------|-----------|---------------------|---------|
| Male   | 59(59.00) | 65.68±7.20          | 0.173   |
| Female | 41(41.00) | 63.00±5.83          | 0.173   |

Presence of occlusion in carotid arteries in patients with recent stroke or transient ischemic attack with bilateral carotid lesions

| Variable      | Total | Absent | Bi-lateral | Only contralateral   | Only ipsilateral | P-val |
|---------------|-------|--------|------------|----------------------|------------------|-------|
|               |       |        |            | To hemisphere damage |                  |       |
| RECENT STROKE | 65    | 57     | 1          | 0                    | 7                | 0.03  |
| RECENT TIA    | 35    | 29     | 1          | 1                    | 4                | 0.40  |

Patients who experienced a recent stroke had a higher prevalence of occlusion in the ipsilateral carotid arteries (P-value = 0.03), while as any TIA patients did not show significant association

Plaque thickness at the site of minimum residual lumen was 4.32±0.18 on the ipsilateral side and 3.27±0.11 on the contralateral side (P-Val 0.000). Residual lumen diameter at the reference point in stroke patients on the ipsilateral side was (5.12±0.16) and on the contralateral side was 5.63±0.13 (P-Val 0.00). Plaque thickness at the reference point in stroke patients on the ipsilateral side was 1.74±0.13 and on the contralateral side was 1.32±0.09(P-Val 0.00). In stroke patients radial motion proximal to the lesion was seen in 53 patients bilaterally ,only contralateral in 3 and only ipsilateral in 1(p val-0.60).Radial wall motion distal to the lesion was seen bilaterally in 50 patients ,only contra laterally in 2 and only ipsilateral in 1 (P-Val 0.96).longitudinal plaque motion was absent in 46, bilateral in 6, only contralateral in 2 and only ipsilateral in 3( P-Val 0.94). plaque reflectivity in patients with stroke was moderate and seen in 20 patients bilaterally, 6 patients contra laterally and 4 patients only ipsilateral. It was high bilaterally in 15 patients, only contralateral in 10 patients and only ipsilateral in 8 patients. Low reflectivity was seen in 8 patients bilaterally, only contralateral in 10 and in only ipsilateral in 8. Residual lumen diameter at MRL site in TIA patients was 3.81±0.27 on ipsilateral side and 3.96±44 on the contralateral side (P-Val 0.20). Residual lumen diameter at the reference point in TIA patients was 4.92±0.14 ipsilateral and 5.10±0.21 on the contralateral side (P-Val 0.06). Plaque thickness at MRL site in patients with TIA was 4.39±0.08 ipsilateral and 3.87±0.09 contralateral (P-Val 0.000). plaque thickness at the reference point in TIA patients was 2.42±0.19 ipsilateral and 2.25±0.18 contra laterally (P-Val 0.01).

Plaque reflectivity in TIA patients was high in 5 patients bilaterally, contralateral in 7 and ipsilateral in 6. It was moderate bilaterally in 10, contralateral in 9 patients and ipsilateral in 6 patients. Low reflectivity was seen bilaterally in 3, contralateral in 2 and ipsilateral in 10 patients.

Statistical analysis of the data was done by test statistic students t-test for the difference of means, McNemar's chi-square test for nominal data and odds ratio for strength of association. Any p value less than 0.05 was taken significant. The analysis of the data was performed by using SPSS version 10.0.

### Discussion

As age advances the prevalence of extra cranial carotid atherosclerosis increases steeply and becomes highly prevalent in elderly population, but despite this high prevalence extra cranial carotid atherosclerosis accounts for about 5% of ischemic strokes only. Plaques are found not only on symptomatic side but also both in asymptomatic and symptomatic patients on their contralateral asymptomatic side as well. Question arises what sort of plaques cause symptoms. About luminal stenosis there is conflicting evidence. Lennihnen et al[7] in a retrospective study of 200 consecutive endarterectomies demonstrated that patients with stenosis >75% were more likely to be symptomatic than patients with <75% of stenosis. On the other hand Imparato et al[8] reported 82% of patients with focal symptoms had stenosis greater than 70% on the ipsilateral side, while only 53% of patients with focal symptoms had stenosis greater than 70% on the contralateral asymptomatic side. Duplex USG also has produced conflicting results, Leahy et al[9] found no difference when comparing percent stenosis between ipsilateral and contralateral asymptomatic sides. In contrast Dempsey et al[10] found that percent stenosis is an independent risk factor for ipsilateral stroke. In our study occlusion of the carotid artery showed significant association with stroke, but this association was insignificant in patients with TIA. Plaque rupture and sudden thrombotic occlusion might be the cause. Borrenstein and Norris[4] followed 40 patients with unilateral carotid stenosis and found that occurrence of clinical symptoms was related to the time of total occlusion. Similar results were found by Fischer<sup>1</sup> and Ojemann Iannuzzi et al[11] while studying 534 patients with recent CVA that occlusion of the carotids was significantly associated with stroke and that there was no such association with TIA.

Lesion thickness in our study showed significant association with both stroke as well as TIA. Thicker lesions are supposed to be unstable, rupture prone and can reduce the lumen significantly. Bassiouny et al[12] found that thicker plaques are more unstable and rupture prone. Cardiovascular health study concluded that risk of stroke is better predicted by wall thickness than percent stenosis. Patients with TIA had thicker plaques on the ipsilateral side compared to the contralateral side (P-Val 0.00). Ricotta et al[14] also showed that B-mode USG plaque thickness

measurement was quite compatible with endarterectomy specimens and concluded that B- mode USG was superior to angiography for lesion width measurement[14].

Residual lumen diameter in our study showed significant association with stroke. The lumen in stroke patients was 1mm narrower on ipsilateral side at minimum residual lumen site and about 0.5mm narrower at the reference point. Dempsey et al[10] found that residual lumen predicts stroke better, while as thick lesions predict TIA better.

Longitudinal plaque motion was shown to be more prevalent in ipsilateral plaques in patients with TIA. Patients with stroke however did not show a significant association. This apparent plaque motion could represent the effect of pressure variability during the cardiac cycle on the boundary interfaces between the plaque and other arterial wall tunics. Atherosclerotic lesions produce angiogenic factors that induce proliferation and penetration of the adventitial vasa vasorum through tunica media into the base of the plaque. Longitudinal plaque motion may cause a shearing effect at the base of the lesion resulting in rupture of the vasa vasorum leading to a big hemorrhage in the plaque making it unstable and rupture prone. Lannuzzi et al[11] also demonstrated that longitudinal plaque motion was significantly associated with TIA. Radial wall motion in our study did not show any association with either stroke or TIA.

Echogenicity of the plaque; Our study demonstrated a high prevalence of echolucent plaques in patients with TIA, while as no such association was found in patients with stroke. Tromso study[11] demonstrated that B mode USG can reliably pick echogenicity of the plaque. Lengsfeld et al[10] showed that echolucent plaques were associated with events more than echogenic plaques. Grey Wheale et al[16] showed that echolucent plaques are cholesterol rich, may contain big hemorrhages and are quite unstable. Joseph F Pollock[17] also demonstrated that echolucent plaques are highly associated with clinical events. Marry louis et al[18] demonstrated that B -mode USG predicts lipid rich plaques which appear echolucent and that it can be a potential diagnostic tool in prevention of neurological events.

### Conclusion

Our study demonstrated that stroke patients showed significant association with total occlusion, lesion width and minimum residual lumen, while as TIA patients showed significant association with wider plaques, echolucency and longitudinal plaque motion. These data demonstrate significant relationship between carotid artery ultrasound plaque characteristics and ischemic cerebrovascular events. These findings encourage further prospective studies in asymptomatic subjects focused on ultrasonic plaque characteristics as predictors of subsequent TIA and stroke.

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