



COLOUR DOPPLER EVALUATION OF CAROTID ARTERIES IN ISCHEMIC STROKE PATIENTS

Radiology

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ABSTRACT

Objective: To evaluate common carotid artery (CCA) and internal carotid artery (ICA) to assess the severity of carotid artery stenosis in ischemic stroke patients. **Materials And Methods:** Total 72 patients, who were clinically diagnosed as having suffered from ischaemic cerebrovascular (CV) stroke, were recruited. Each patient was studied in detail with relevant clinical history and Examination; risk factors like hypertension, diabetes mellitus, smoking & hyperlipidemia were documented. Ultrasound Scan and colour Doppler of carotid arteries was done. **Results:** Hemiparesis was the most common presenting clinical symptom, 34 (47.2%), followed by hemiplegia 11 (15.2%), giddiness 7 (9.7%), hemi-sensory disturbances (HSD) 6 (8.3%). 44 patients were smokers (61%), 32 patients were hypertensive (44.4%), 28 patients were diabetic (38.8%), 18 (25%) patients had hyperlipidemia and 15 patients had a previous history of stroke (20.8%). Doppler ultrasound, 52 out of 72 (72.2%) patients had some evidence of ipsilateral/bilateral carotid stenosis (plaque + increased CIMT) and 35 out of 72 (48.6%) had plaque ipsilateral/bilateral. **Conclusion:** Color Doppler of carotid artery plays an important role in identification of plaques and quantification of degree of Stenosis in stroke patients and identification of "at risk" patients who may develop further stroke.

KEYWORDS

carotid doppler, atheromatous plaque, peak systolic velocity, end diastolic velocity, ischemic stroke, stenosis

INTRODUCTION

Stroke is an injury of central nervous system that is characteristically abrupt in onset and is due to a vascular insult which can be secondary to ischemia or hemorrhage. Pathological studies indicate that 80-85 % of strokes are due to cerebral infarctions. Atherosclerotic disease of the extracranial carotid arteries has long been recognized as the most common source of emboli that travel to the brain causing stroke [1], often with carotid plaque as the source of embolus [1]. Therefore, early detection of these atheromatous changes in carotid artery will help in reducing stroke related morbidity and mortality.

Although cerebral DSA (digital subtraction angiography) is the gold standard for identifying and quantifying atherosclerotic stenosis [2], it is somewhat insufficient in determination of plaque morphology (sensitivity 46%, specificity 74%) [3]. In spite of constant improvement of angiographic technique, it is sometimes followed by some complications, which is the main reason for use of non-invasive techniques such as color Doppler sonography, transcranial Doppler sonography, magnetic resonance angiography, and computerized tomographic angiography.

Sonographic evaluation is inexpensive, non-invasive and can provide functional and anatomical information about vessel stenosis and plaque morphology. On Gray scale, we evaluate increased IMT (intima media thickness) in Common Carotid Artery which is considered by many as the earliest change of the atheromatous process. Colour Doppler evaluation shows areas of absent flow, especially useful in hypoechoic plaques not detectable on gray scale and allows detection of abnormal flow patterns. Spectral Doppler helps us to evaluate the waveform in carotid vessels, find peak Systolic velocity (PSV), end diastolic velocity (EDV), and abnormal characteristics like spectral broadening due to turbulent flow. Power Doppler helps us detect colour streaks and helps differentiate between near occlusion and occlusion.

Trials like NASCET (North American Symptomatic Carotid Endarterectomy Trial) [4] and ECST (European Carotid Surgery Trialists collaborative group) [5] showed a definite benefit of carotid endarterectomy for patients with luminal narrowing of >70% rather than for those below it.

MATERIALS & METHODS

This Study was done in Department of Radio diagnosis, Patients who were clinically diagnosed as having suffered from ischemic CV stroke, were recruited after the permission from Institutional Review Board (IRB) - IRB (HEC-Human Ethics committee) No.- 470/2014, Radiodiagnosis No. 06/2014. Each patient was studied in detail with relevant clinical history and Examination, risk factors like hypertension, diabetes mellitus, smoking &, hyperlipidemia were

documented. All examinations were performed by the same operator.

Inclusion Criteria

- Ischemic stroke proved by CT/MRI

Exclusion Criteria

- Patients with haemorrhagic stroke.
- Patients with H/O (history of) head injury
- Pregnancy and postpartum stroke
- Systematic illness like hemodynamically unstable patients, posterior circulation infarcts, history of connective tissue disorders/vasculitis, malignancy, unconscious patients, stroke like syndromes like TB (tuberculosis), metabolic emergencies and poor general condition.

OUTCOME MEASURES

- Peak systolic velocity of CCA & ICA
- Intima media complex thickness of CCA & ICA
- Plaque characteristics as seen on the real time image
- Velocity ratio between CCA & ICA and spectral broadening.

Statistical Analysis

Statistical analysis was done after collecting all necessary data and for each imaging modality descriptive frequencies were calculated for each positive result and was analyzed using a percentage calculation.

Patient Position:

Supine position, A small pad may be placed under the shoulders to provide better exposures of neck, neck exposure can be enhanced by tilting and rotating the head (hyperextended & rotated 45°) away from the side being examined and by having the patient drop the ipsilateral shoulder as far as possible.

Transducer Position:

Posterolateral and far posterolateral positions for long axis (longitudinal) tracings anterior, lateral transducer positions for short axis (transverse) tracings.

TECHNIQUE

Initially, gray scale examination was done, intima media thickness of Common Carotid Artery was measured in longitudinal position, Presence, location, characterization (homogenous, hyperechoic or hypoechoic, heterogeneous/calcified) and surface (smooth, irregular, or ulcerated) of plaques were assessed, During the initial scanning, optimal insonation angles were determined for the estimation of respective plaque heights, and the measurements were performed on the frozen frame, perpendicular to the vascular walls. The diameter of the residual lumen and the external diameter of the artery at the same level were measured and the degree of stenosis was calculated using

the following relationship:

Percent stenosis = $C-B.100/C$

(where C is vessel wall-to-wall diameter and B is patent vessel diameter, see Fig.1).

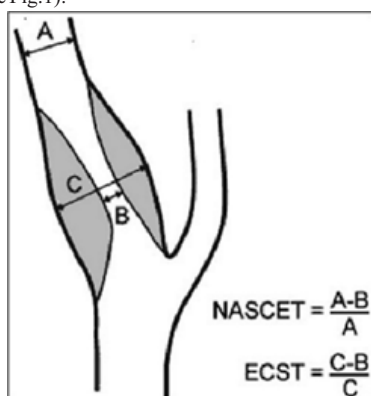


Fig.1- Method of measuring the degree of stenosis in NASCET [4] trial, and in ECST[5] trial

(Stenosis of vessel lumen can be determined by combined cross-section area, diameter measurements of the vessel lumen and also by spectral Doppler velocimetric studies.)

The gold standard has been angiography and the parameter that angiography provides is diameter stenosis and hence, in ultrasound, we also used diameter stenosis. Stenosis was classified as mild (<50%), moderate (50-69%) and severe (>70%) (Table 1). Spectral Doppler analysis was done in Common carotid artery and internal carotid artery and measurements of Peak systolic velocity, end diastolic velocity were obtained on a frozen image. After this, Colour Doppler exam was done to substantiate the previous findings, identify regions of turbulence, any undetected areas of plaque or plaque ulceration. Depending upon the need, Power Doppler may or may not be employed to differentiate near complete obstruction from complete obstruction. A plaque was defined as a focal thickening greater than the surrounding area or CIMT (carotid IMT) greater than 1.5 mm (millimeter).

Table 1- Criteria for classification of internal carotid artery disease by duplex scanning with spectral waveform analysis of pulsed Doppler signals North American Symptomatic Carotid Endarterectomy Trial (NASCET) [4] & European Carotid Surgery Trialists collaborative group (ECST) [5]

Degree of stenosis, %	ICA PSV, cm/s (centimeter per second)	Plaque estimate, %	ICA/CCA PSV ratio	ICA EDV, (centimeter per second)
Normal	<125	0	<2	<40
<50	<125	<50	<2	<40
50-69	125-230	>50	2-4	40-100
>70	>230	>50	>4	>100
Subtotal occlusion	Variable	>50 narrow lumen	Variable	>0
Total occlusion	0	100	<1	0

COLOR FLOW IMAGING

(1) Ensure focus is at the region of interest and adjust gain to optimize colour signal.

(2) Use probe positioning/beam steering to obtain satisfactory beam/vessel angle.

(3) Adjust pulse repetition frequency to suit the flow conditions. Low pulse repetition frequencies are more sensitive to low flows/velocities but may produce aliasing. High pulse repetition frequencies reduce aliasing but are less sensitive to low velocities.

(4) Set the colour flow region to appropriate size. A smaller colour flow 'box' may lead to a better frame rate and better colour resolution/sensitivity.

SPECTRAL IMAGING

(1) Position the pulse wave Doppler cursor on the vessel to be investigated.

- (2) Adjust gain so that the sonogram is clearly visible and free of noise.
- (3) The beam/vessel angle should be 60° or less if velocity measurements are to be made.
- (4) Adjust the scale and baseline.
- (5) Gate size: should be optimal, a large gate may include signals from adjacent vessels.

RESULTS

Smoking (61%), hypertension (HTN, 44.4%) and Diabetes Mellitus (DM, 38.8%) were the three most common risk factors.

Carotid Doppler Findings: 27.8% patients had no stenosis on carotid Doppler ultrasound, 72.2% patients had evidence of ipsilateral/bilateral carotid stenosis (plaque + increased CIMT) and 48.6% had plaque, ipsilateral/bilateral. Stenosis due to plaque of carotid system was found in 48.6% patients; being unilateral in 77% patients and bilateral in 23%. Plaques were most commonly found on right side 53.5% cases followed by left 46.5%.

Large infarction was present in 59.7%, and small infarction in 40.3% patients. 51.3% patients had bilateral/unilateral increased CIMT (> 0.9 mm). There was a significant association between abnormal CIMT and size of brain infarction and 58.8% patients (only increased CIMT with no plaque) had large brain infarcts.

Regarding Plaque Morphology And Surface: According to plaque characterization on sonography, majority of the plaques were hypoechoic plaques (48.6%), followed by hyperechoic (31.4%) and calcified (20%). There was a significant association between large sized infarction and ulcerated surfaced plaques (10 out of 10 ulcerated plaques, 100%).

Observation was also done between intima-media thickness (CIMT) and risk factors. More than 0.9 mm was considered abnormal; Patients with carotid plaque had significantly increased CIMT when compared to patients without plaques.

DISCUSSION

The main role of color Doppler sonography is detection of occlusive lesions in the extra cranial course of common carotid and ICA. Kreh and Giyanani[6] also advised the use of high-frequency transducer for imaging of carotid vessels.

Randomized clinical trials- The North American Symptomatic Carotid Endarterectomy Trial (NASCET)[4] study, European carotid surgery trial (ECST)[5] and the Asymptomatic Carotid Atherosclerosis Study (ACAS) as quoted by Biller, William et al. [7] have demonstrated that the surgical treatment of endarterectomy should be reserved for those patients with carotid stenosis of more than 70%, and lack of any benefit for surgery on lesions of 30% diameter.

The present study comprised of 72 cases of which 45 were males representing 62.5% and 27 were females representing 37.5% of cases. Overall men are more prone to atherosclerosis than women. This might be explained based on protective role of the female hormones. After menopause, when this effect wanes, the rates of diseases become similar, males exceeding females in all age groups and these finding are similar to that seen by Alexander et al. [8].

Smoking and hypertension were more frequent in patients with higher degrees of stenosis (50-69% and ≥ 70%) than in milder degree (< 50% stenosis) Smoking causes a reduction of HDL (high density lipoprotein) levels and fibrinogenemia and thus resulting in increased (low density lipoprotein) LDL/HDL ratio, causing increased tendency of atherosclerosis[10]. Hypertension produces a continuous trauma to endothelium and predisposes to the early stage of atherogenesis. Incidence of stroke in diabetics has been found to be 2 to 3 times higher than in general population, Executive committee for the Asymptomatic Carotid Atherosclerotic Study, and study conducted by Su et al[11] have suggested an association between hyperlipidemia and extracranial carotid stenosis. Several studies have shown an increased risk of thrombotic stroke with low HDL[11] (Table 2).

Table 2- Risk Factors

Workers	Risk factors			
	smoking	hypertension	DM (diabetes mellites)	Hyperlipid emia
Sanjeev Sehrawat et al [9]	55	37.5	45	30
Present study	61	44.4	38.8	25

The bifurcation of the common carotid artery is the commonest site of plaque in the extracranial carotid artery, which is in accordance with Philips et al[13], where they found that the areas of transient reversal of flow, flow separation and eddy formation after referred as “boundary layer separation” corresponds to segments in the carotid bifurcation where atherosclerosis developed first (Table 3).

Table 3– Site Of Plaque

Workers	Findings
Rajagopal et al (2000) [12]	Common carotid bifurcation >CCA >ICA
Philips et al [13]	Commonest site common carotid bifurcation
Present study	Common carotid bifurcation >CCA >ICA

In present study, homogenous hypoechoic plaques were more frequent in patients with large size infarcts (17 plaques out of 18 plaques, Table 4, fig.2 & fig.3). Regarding plaque surface, most of the significant large stenosis were found to be in patients with ulcerative surface (all 10 plaques), which support the suggestion that the more friable, lipid containing soft plaques are more likely to result in plaque disruption and produce symptoms more than that of firmer more fibrous and coherent plaques[15]. This was in agreement with results of previous studies by Carra [16] and Tegos [17]. Thus, above observations made us to thought that ulcerated plaques give rise to large emboli that produce infarcts larger than that produced from irregular plaques because of smaller surface defect. This observation confirms the Handa et al [18] suggestion about the important role of plaque surface in pathogenesis of cortical infarcts, as he found that patients with ulcerated plaques have seven folds increased risk for stroke.

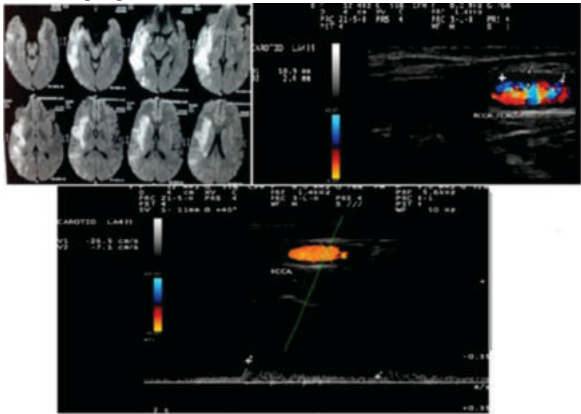


Fig.2- MRI plate showing acute ischaemic infarct involving right side, Colour Doppler sonography image showing homogenous hypoechoic smooth plaque in Right CCA, Colour Doppler sonography image of same case showing spectral broadening

Table 4- Plaque Morphology (see fig. 2 & 3)

Workers	Finding (%)		
	Stenosis	Homogenous hypoechoic	Smooth surface
Laith Ahmed et. Al [14]	86.6	33.3	33.3
Present study	82.8	45.7	40

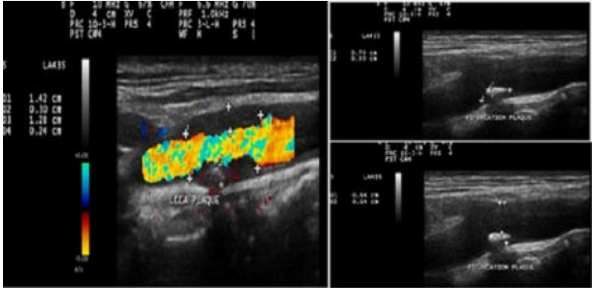


Fig.3- Colour doppler image showing calcified ulcerated plaque in Left CCA, Gray scale sonography image showing calcified smooth plaque at bifurcation, Gray scale sonography iamge showing diameter method for calculation of stenosis(C-B/Cx100)

In present study, it was observed that most of the calcified plaques were associated with small infarct, which is suggestive of their chronic nature and hemodynamic adaptation. The reason is lesser chance of calcified plaque being dislodged and causing thromboembolism, which was also found in studies of Carroll et al [19] and Taylor et al [20].

Percent Stenosis

Among the study group of 72 patients, 24 patients had <50% diameter stenosis with no significant hemodynamic changes and 11 patients had more than 50% diameter stenosis with significant hemodynamic changes, which is correlating with ICA/CCA ratio of more than 1.8.

This was in accordance with other studies done in past, this was comparable to the findings of Zhu et al [21]. Of the 35 patients with stenosis, 24 (68.5%) had <50% stenosis which was approximately similar to previous studies by Adetiloye et al [22], and Carroll et al [19]. The prevalence of patients with >50% stenosis was 11 (31.4%) which was approximately similar to that reported by Alexander et al [8] (24.3%). While the prevalence of $\geq 70\%$ stenosis was 6 patients (17.1%), which was approximately similar to that reported by Brown et al [23](20%).

One patient misdiagnosed as total occlusion on colour Doppler, was correctly diagnosed as near occlusion (with streak of residual lumen: string sign) on power Doppler (fig.4). This is because power Doppler is more sensitive for detecting extremely low-amplitude, low-velocity flow as the colour signal in power Doppler ultrasound is generated from the integrated power Doppler spectrum. Power Doppler ultrasound uses a larger dynamic range with a better signal-to-noise (S/N) ratio than colour Doppler ultrasound.

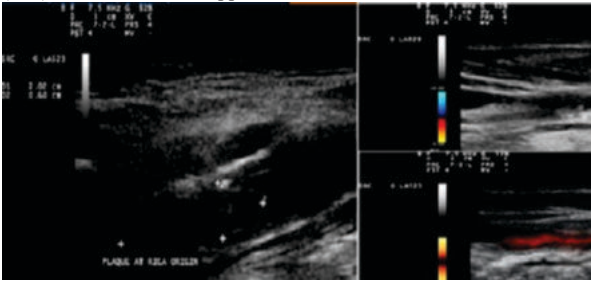


Fig.4- Gray scale sonography image showing hypoechoic irregular plaque at Right ICA origin, Colour doppler image showing absent colour flow in distal part, Same image on power doppler showing presence of colour flow.

Bluth et al [24] found the PSV ratio (fig.5) more accurate than PSV (fig.5) measurements, which is in accordance with present study (Table 5).

Table 5 - Peak Systolic Velocity (PSV) and Peak Systolic Velocity (PSV) Ratio

(PSV) and (PSV) Ratio	Bluth et al [24]	Present study
<50% stenosis	PSV ratio of, >1.8 as an indicator of 60% or greater and a ratio of 3.7 as an indicator of more than 80% diameter stenosis	PSV-20-80 cm/s, PSV ratio was <1.8,
50-70% stenosis		PSV-40-126 cm/s, PSV ratio was >1.8,
>70% stenosis		Variable velocity and ratio.

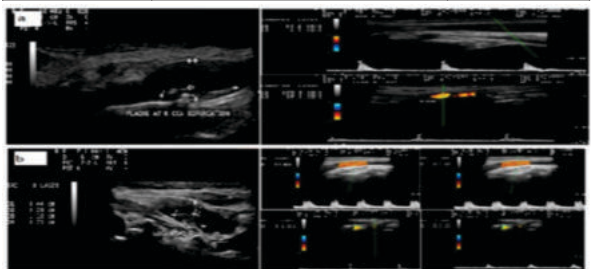


Fig.5- (a) Gray scale sonography image showing hyperechoic smooth

plaque at right carotid bifurcation stenosis <50%, Spectral wave doppler image showing PSV & EDV of Right CCA, Spectral wave doppler image showing PSV & EDV of Right ICA, showing PSV & EDV ratio of (ICA/CCA) (<1.8 & <2.4 respectively). (b) Gray scale sonography image showing hyperechoic irregular plaque in Left ICA, stenosis 50-70%, Spectral wave doppler image showing PSV of Left CCA, Spectral wave doppler image showing EDV of Left CCA, Spectral wave doppler image showing PSV of Left ICA, Spectral wave doppler image showing EDV of Left ICA, showing PSV & EDV ratio of (ICA/CCA) (>1.8 & >2.4 respectively).

End Diastolic Velocity (EDV) and End Diastolic Velocity (EDV) ratio

In present study it was observed that EDV (fig.5) of cases with <50% stenosis had range of 2-34 cm/s, EDV ratio (fig.5) was <2.4, while with 50-70% had range of 10-40 cm/s, ratio was >2.4, and >70% had variable velocity and ratio.

According to Zweibel et al [25], EDV <40 cm/s does not yield any accurate information about degree of stenosis. If the EDV is more than 40 cm/s but <100 cm/s we can estimate the stenosis to be ranging from 50% to 80%. Bluth et al [24], found EDV of ICA to ratio >2.4 predicts 60% or greater stenosis. velocity ratio of ICA/CCA define the percentage of significant stenosis as this is more accurate according to a study conducted by Bluth et al [24]. These features correlated in our patients with significant stenosis with hemodynamic changes.

Carotid intima-media thickness (CIMT)

Of all 72 patients, 20 (27.7%) had no stenosis of ipsilateral CCA and ICA, 37 (51.4%) had increased CIMT and 35 (48.6%) had different degrees of stenosis; this was comparable to the findings of Zhu et al [21].

We also observed that increased CIMT in Carotid artery was present in significant number of stroke patients (37, 51.4%) and more frequent in patients with stenosis (20, 27.7%) as compare to patients without stenosis (17, 23.6%). It was comparable to studies by Toubou et al [26] and Bots et al [27], who reported a significantly higher incidence of stroke in patients with increased CIMT, suggesting a type of relationship with brain infarction related to atherosclerosis. The statistically significant association of the known risk factors of stroke with increased CIMT in our study is supported by other studies.

Regarding cardiovascular risk factors, an increased age has the most impact on an increased CIMT: depending on different studies, age may explain 50%–80% of the variability for an increased CIMT. It has been described an annual increase of CIMT in the CCA about 0.007 mm [28], in present study it was observed that 25 (67.5%) patients out of 37 patients with increased CIMT were of age >50 years and 12 (32.5%) patients were of age <50 years.

Out of 32 patients with HTN 17 (53.1%) had abnormal CIMT, 14 (50%) out of 28 patients with DM and 9 (50%) out of 18 with hyperlipidemia had abnormal CIMT. These findings were correlated with Sun, Cheng-Huai et al [29], who stated that higher CIMT was associated with blood pressure, fasting blood sugar, and that plaque prevalence was positively associated with CIMT and blood pressure, fasting blood sugar were independent risk factors related to both carotid atherosclerosis and thick CIMT and same was observed in our study.

Our findings also correlate with those of Jadhav and Kadam [30], who found CIMT >1.1 mm in diabetics in Indian population, this also reflects the increased prevalence of carotid stenosis in ischemic stroke patients.

In our study most of the findings were in conformity with the literature, the difference observed in some observation may be due to differences in the study population.

In conclusion, Carotid Doppler is an important non-invasive diagnostic tool, it can be used for screening in high-risk asymptomatic patients, patients with history of cerebrovascular events and for determining treatment protocol. Thus, it should be used as a first line investigation in these patients supplemented by Magnetic Resonance Angiography whenever required and angiography should be used only in equivocal cases.

REFERENCES:

1. Eliasziw M, Kennedy J, Hill MD, Buchan AM, Barnett HJM- Early risk of stroke after a transient ischemic attack in patients with internal carotid artery disease. Can Med Assoc

- J 2004; 170: 1105-9.
2. Barlinn K, Alexandrov A-V. Vascular imaging in stroke: comparative analysis. Neurotherapeutics 2011; 8(3):340-8.
3. Randoux B, Marro B, Koskas F, Duyme M, Sahel M, Zouaoui A, et al. Carotid artery stenosis: prospective comparison of CT, three-dimensional gadolinium-enhanced MR, and conventional.
4. Moneta et al, Correlation of North American Symptomatic Carotid Endarterectomy Trial (NASCET) angiographic definition of 70% to 99% internal carotid artery stenosis with duplex scanning. Journal of Vascular Surgery, 17 (1), 1993, 152-159.
5. P M Rothwell and C P Warlow, Prediction of benefit from carotid endarterectomy in individual patients: a risk-modelling study. European Carotid Surgery Trialists Collaborative Group, Lancet, 353(9170), 1999, 2105-2110.
6. Krebs CA, Giyanani VL, Eisenberg RL. Ultrasound atlas for vascular diseases. 1st ed. 1999. p. 53-102.
7. Biller J, William FM, Castaldo JE, Whittemore AD, Harbaugh RE, Dempsey RJ, et al. Guidelines for carotid endarterectomy: A statement for healthcare professionals from a special writing group of the stroke council, American Heart Association. Circulation 1998; 97:501-9.
8. Alexander T, Gerald D, Gabriel R, et al. Stroke with Internal Carotid Artery Stenosis. Arch Neurol. 2001; 58:605-609.
9. Sehrawat, S., Thind, S. S., Singh, V., Kuber, R., Naware, S., & Shrotri, H. (2012). Colour Doppler evaluation of extracranial carotid artery in patients presenting with features of cerebrovascular disease: A clinical and radiological correlation. Medical Journal of Dr. DY Patil University, 5(2), 137.
10. Whisnant JP, Homer D, Ingall TJ, Baker HL Jr, O'Fallon WM, Wievers DO. Duration of cigarette smoking is strongest predictor of severe extracranial carotid artery atherosclerosis. Stroke 1990; 21:707-14.
11. Su TC, Jeng TS, Chien KL et al. Hypertension status is the major determinant of carotid atherosclerosis: a community-based study in Taiwan. Stroke 2001; 32: 2265-71.
12. Rajagopal KV, Lakhkar BN, Banavali S, Singh NK. Pictorial essay- color duplex evaluation of carotid occlusive lesions. Ind J Radiol Imag 2000; 10: 4.
13. Philips DJ, Green FM Jr, Langlois Y, Roederer GO, Strandness DE Jr. Flow velocities in carotid bifurcation of young presumed normal subjects. Ultrasound Med Biol 1983; 9:39-9.
14. Laith Ahmed et al Carotid Doppler Study in Patients with Cerebral Infarction J Fac Med Baghdad Vol.53, No.4, 2011.
15. Allen P. The Carotid & Vertebral Arteries & Transcranial Doppler. In: Allen P.L, Dubbins P.A, Pozniak A & McDicken W N (eds.) Clinical Doppler Ultrasound. 1st ed. London, Churchill Livingstone 2002; 39-59.
16. Carra G, Visona A, Bonanome A, et al. Carotid plaque morphology and cerebrovascular events. Int Angiol. 2003; 22(3):284-9.
17. Tegos T, Sabetai M, Nicolaides A, et al. Patterns of brain computed tomography infarction and carotid plaque echogenicity. J Vasc Surg. 2001; 33(2):334-9.
18. Handa N, Matsumoto M, Maeda H, et al. Ischemic stroke events and carotid atherosclerosis. Results of the Osaka Follow-up Study for Ultrasonographic Assessment of Carotid Atherosclerosis (the OSACA Study). Stroke, 1995; 26(10):1781-6.
19. Carroll BA. Carotid ultrasound. Neuroimaging Clin N Am 1996; 6:875-97.
20. Taylor, D. C. and Strandness, D. E. (1987), Carotid artery duplex scanning. J. Clin. Ultrasound, 15: 635-644. doi: 10.1002/jcu.1870150906.
21. Zhu C & Norris J. Role of carotid stenosis in ischemic stroke. Stroke 1990; 21(8):1131-1134.
22. Adetiloye VA & Al Damegh S. Sonographic evaluation of plaque morphology in hemodynamic and non-hemodynamic symptomatic carotid artery stenosis. Afr J Med Sci. 2003; 32(4): 381-5.
23. Brown PB, Zweibel, WJ, Call GK. Degree of cervical carotid artery stenosis and hemispheric stroke: duplex ultrasound findings. Radiology 1989; 170: 541-3.
24. Bluth EI, Stavros AT, Marich KW, Wetzner SM, Aufrecht D, Baker JD. Carotid duplex sonography: a multicenter recommendation for standardized imaging and Doppler criteria. RadioGraphics 1988; 8:487-506.
25. Zweibel WJ, Knighton R. Duplex examination of carotid arteries. Seminars in Ultrasound, CT and MR 1990; 11: 97-135.
26. Touboul P, Elbaz A, Koller C, et al. Common Carotid Artery Intima-Media Thickness and Brain Infarction. Circulation 2000; 102:313-318.
27. Bots M, Hoes A, Koudstaal P, et al. Common carotid intima-media thickness and risk of stroke and myocardial infarction: the Rotterdam Study. Circulation 1997; 96:1432-37.
28. Bauer M, Möhlenkamp S, Lehmann N, Schermund A, Roggenbuck U, Moebs S, et al. The effect of age and risk factors on coronary and carotid artery atherosclerotic burden in males – results of the Heinz Nixdorf Recall Study. Atherosclerosis. 2009; 205(2):595-602.
29. Sun Y, Lin CH, Lu CJ, Yip PK, Chen RC. Carotid atherosclerosis, intima media thickness and risk factors-an analysis of 1781 asymptomatic subjects in Taiwan. Atherosclerosis 2002; 164:89-94.
30. Jadhav UM, Kadam NN. Carotid Intima-Media thickness as an independent predictor of coronary artery disease. Indian Heart J 2001; 53:458-62.