

Prevalence of Asymptomatic Atherosclerotic Plaque at Carotid Bifurcation in Individuals of 20-70 years of Age

AMIT KUMAR CHOUBEY¹, HIRDESH SAHNI², MUKUL BHATIA³, SAMARESH SAHU⁴

ABSTRACT

Introduction: Stroke is the second most common cause of death worldwide. Among 20-30% of all ischaemic infarcts are caused by carotid artery stenosis. Computed Tomography Angiography (CTA), Magnetic Resonance Angiography (MRA), Digital Subtraction Angiography (DSA) and duplex imaging are the available modalities for evaluation of extracranial atherosclerosis. Duplex imaging for Carotid Intima-Media Thickness (CIMT) and plaque evaluation in carotid vessels is a good screening tool for atherosclerosis. No prevalence study for carotid atherosclerosis is available in Indian population.

Aim: To determine the prevalence of asymptomatic atherosclerotic plaques and asymptomatic critical stenosis at carotid bifurcation in individuals of 20-70 years of age.

Materials and Methods: The present prospective consecutive cross-sectional study was carried on 250 volunteers (125 males and 125 females) in the 20-70 years age group with no history of TIA/cerebral ischaemic stroke with 50 subjects in each

decade. The carotid arteries of volunteers were screened by duplex imaging. Descriptive and inferential statistical analysis was carried out by statistical software SPSS 15.0. Results on categorical measurements were presented in Number (%).

Results: Overall prevalence of carotid plaque was 14% in 20-70 years of age. Prevalence of plaque in Common Carotid Artery (CCA), Internal Carotid Artery (ICA) and External Carotid Artery (ECA) was 4%, 10% and 0.4% respectively. Plaque prevalence was 2%, 4%, 2%, 24% and 38% in age groups of 20-30 years, 31-40 years, 41-50 years, 51-60 years and 61-70 years respectively. Prevalence of percentage stenosis was 2% for 50-69% stenosis and 0.4% for >69% stenosis.

Conclusion: In the present study, the overall prevalence of Carotid plaque was 14% and prevalence of percentage stenosis was 0.4% for >69% stenosis. So screening of asymptomatic population for carotid atherosclerosis is not recommended in view of low prevalence rate of significant stenosis and the management will remain same based on standard preventive therapy.

Keywords: Atherosclerosis, Carotid atherosclerosis, Carotid doppler, Duplex imaging

INTRODUCTION

Stroke is the second most common cause of death and fourth most common cause of disability in world [1]. It is the most common cause of disability, second most common cause of dementia and third most common cause of death in developed nations [2]. In India, Stroke has already attained epidemic with annual incidence of 13 per lac in 1969-70 and 145 per lac per year during 2003-05 and 2005-06 [3-5]. An 87% of these strokes are ischaemic and most of these ischaemic strokes are due to embolization from an atherosclerotic plaque [6,7]. It has been found that 20-30% of all ischaemic infarcts are caused by carotid artery stenosis and stenosis of >69% of carotid vessels is a well-known risk factor for stroke and are treated with endarterectomy [8,9]. Since, atherosclerosis is the main cause of stroke and it can remain silent over several decades, it is very important to identify them in subclinical stage to have better knowledge of the disease progression and early institution of preventive measures [10]. Furthermore, it is important to know the degree of carotid stenosis for stroke evaluation. Angiography by CT, MRI, digital subtraction angiography and duplex imaging are the available modalities for evaluation of extracranial atherosclerosis [8]. Duplex imaging for CIMT and plaque evaluation in carotid vessels is a good screening tool for atherosclerosis. As far as author's knowledge, no prevalence study is available in Indian population. This prompted us to measure prevalence of asymptomatic atherosclerotic plaque at carotid bifurcation between 20-70 years of age. The study was done in wide range of age groups to know in which age groups preventive measures should be initiated. The present study may form baseline for many studies in future on carotid atherosclerosis, age of onset of carotid atherosclerosis, recommendation for carotid screening in target population, estimated workload for endarterectomy surgery etc.

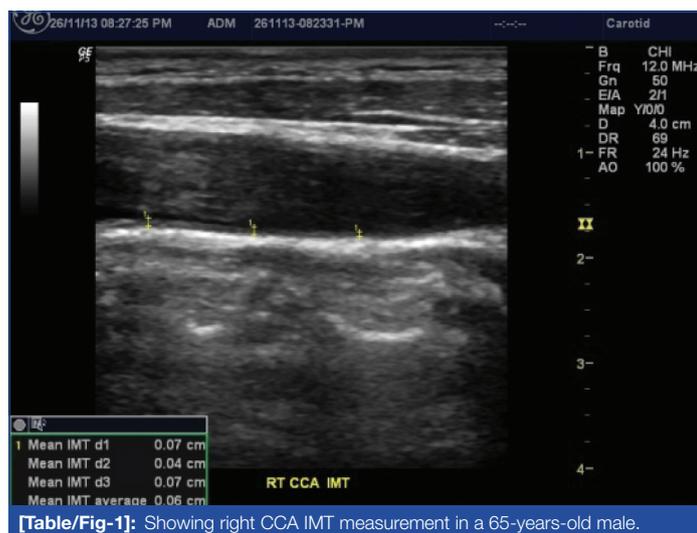
MATERIALS AND METHODS

The present prospective consecutive cross sectional study was done in a tertiary care hospital in Bangalore (India) from February 2013 to February 2014 after clearance from hospital Ethical Committee and informed written consent from volunteers. These volunteers were patients and their relatives reporting to hospital for unrelated disease and were of diverse ethnic group.

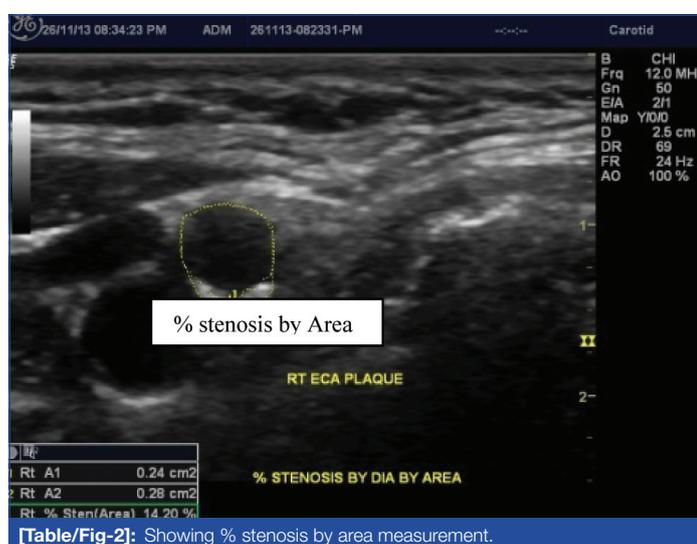
No prior information on prevalence was available and hence assumed maximum prevalence of 50% yielding maximum sample size with 95% confidence interval and 7% error of margin the required sample size was 196. However, we included 250 volunteers (125 males, 125 females) in the 20-70 years age group with no history of TIA/cerebral ischaemic stroke with 50 subjects (25 males, 25 females) in each decade.

Ultrasonographic scanning was performed with the subject in the supine position. A duplex ultrasound system (Logiq P5 GE health care) was used, with a linear transducer with 7.5-MHz scanning frequency in B-mode and 5-MHz scanning frequency in colour doppler mode. The carotid arteries were explored with longitudinal and transverse scans. CCA, ICA and ECA was evaluated on grey scale, colour doppler and spectral doppler. The CIMT was measured in CCA, ICA and ECA with in 2 cm of carotid bulb as shown in [Table/Fig-1]. IMT of 0.9 or more was taken as increased CIMT. Plaque was said to be present if 2 of the 3 criteria's were present viz., Abnormal wall thickness (defined as CIMT >1.5 mm), abnormal shape (protrusion into the lumen, loss of alignment with adjacent arterial wall boundary) and abnormal wall texture (brighter echoes than adjacent boundaries) [11,12]. If plaque was detected then it was evaluated for location of plaque in CCA/ICA/ECA, percentage stenosis of corresponding vessels by area on grey scale images

[Table/Fig-2] and stenosis was classified as <50%, 50-69%, >69% stenosis [Table/Fig-2] [8].



[Table/Fig-1]: Showing right CCA IMT measurement in a 65-years-old male.



[Table/Fig-2]: Showing % stenosis by area measurement.

STATISTICAL ANALYSIS

Descriptive and inferential statistical analysis were carried out by Statistical software SPSS 15.0. Results on categorical measurements were presented in Number (%). Significance was assessed at 5% level of significance. A p-value <0.05 was taken as significant.

The assumption was made that dependent variables are normally distributed. Chi-square was used to find the significance of study parameters on categorical scale between two or more groups.

RESULTS

The present study showed that overall prevalence of plaque was 14% {95% Confidence Interval (CI)=10.01-18.72} out of which 4% (95% CI= 2.04-7.01) was in CCA, 10% (95% CI= 6.72-14.19) in ICA and only 0.4% (95% CI= 0.02-1.95) in ECA. The average CIMT in right and left CCA, ICA and ECA in males and females are shown in [Table/Fig-3,4]. The prevalence of plaque was 2%, 4%, 2%, 24% and 38% in age group of 20-30 years, 31-40 years, 41-50 years, 51-60 years and 61-70 years respectively [Table/Fig-5]. An 18.4% of males (23 out of 125) had plaque while only 9.6% of females (12 out of 125) had plaque which was statistically significant (p-value=0.05). Gender distribution of plaques in different age groups are shown in [Table/Fig-6]. The prevalence of percentage stenosis caused by plaque is depicted in [Table/Fig-7]. The distribution of percent stenosis in different age group has been shown in [Table/Fig-8]. The distribution of

percentage stenosis of plaques in males and females is shown in [Table/Fig-9].

Age groups	Right CCA (mm)	Left CCA (mm)	Right ICA (mm)	Left ICA (mm)	Right ECA (mm)	Left ECA (mm)
20-30 yrs	0.49	0.5	0.4	0.43	0.32	0.36
31-40 yrs	0.54	0.56	0.5	0.49	0.39	0.39
41-50 yrs	0.7	0.68	0.63	0.53	0.43	0.39
51-60 yrs	0.79	0.8	0.76	0.7	0.47	0.43
61-70 yrs	0.9	1.05	0.77	0.8	0.47	0.47

[Table/Fig-3]: Showing average Carotid Intima-Media Thickness (CIMT) in right and left Common Carotid Artery (CCA), Internal Carotid Artery (ICA) and External carotid Artery (ECA) in males.

Age groups	Right CCA (mm)	Left CCA (mm)	Right ICA (mm)	Left ICA (mm)	Right ECA (mm)	Left ECA (mm)
20-30 yrs	0.49	0.49	0.42	0.40	0.34	0.29
31-40 yrs	0.62	0.6	0.47	0.48	0.38	0.36
41-50 yrs	0.62	0.6	0.57	0.54	0.38	0.36
51-60 yrs	0.61	0.66	0.56	0.6	0.41	0.4
61-70 yrs	0.76	0.8	0.62	0.67	0.38	0.41

[Table/Fig-4]: Showing average Carotid Intima-Media Thickness (CIMT) in right and left Common Carotid Artery (CCA), Internal Carotid Artery (ICA) and External carotid Artery (ECA) in females.

Plaques	Age in years				
	20-30 years	31-40 years	41-50 years	51-60 years	61-70 years
No	49 (98.0%)	48 (96.0%)	49 (98.0%)	38 (76.0%)	31 (62.0%)
Yes	1 (2.0%)	2 (4.0%)	1 (2.0%)	12 (24.0%)	19 (38.0%)
Total	50 (100.0%)	50 (100.0%)	50 (100.0%)	50 (100.0%)	50 (100.0%)

[Table/Fig-5]: Distribution of presence of plaques in different age groups. Prevalence of plaques is significantly more associated with higher age with p<0.001**.

Age groups	Males (n=35)	Females (n=35)	p-value
20-30 yrs	1 (2.86%)	0 (0%)	
31-40 yrs	0 (0%)	2 (5.71%)	
41-50 yrs	1 (2.86%)	0 (0%)	
51-60 yrs	7 (20%)	5 (14.29%)	0.055
61-70 yrs	14 (40%)	5 (14.29%)	0.032
Total	23 (65.7%)	12 (34.3%)	0.045

[Table/Fig-6]: Showing gender distribution of plaques in different age groups.

Stenosis %	Plaques: Overall	
	Overall prevalence of % stenosis (n=250)	Percentage of plaques causing %stenosis (n=35)
No stenosis	215 (86.0%)	0
<50.0%	29 (11.6%)	29 (82.9%)
50-69%	5 (2%)	5 (14.3%)
>69.0%	1 (0.4%)	1 (2.9%)
Total	250 (100.0%)	35 (100.0%)

[Table/Fig-7]: Showing prevalence of percentage stenosis caused by plaques.

Age groups	<50% stenosis (n=35)	50-69% stenosis (n=35)	>69% stenosis (n=35)
20-30 yrs	1 (2.86%)	0 (0%)	0 (0%)
31-40 yrs	2 (5.71%)	0 (0%)	0 (0%)
41-50 yrs	1 (2.86%)	0 (0%)	0 (0%)
51-60 yrs	10 (28.57%)	2 (5.71%)	0 (0%)
61-70 yrs	15 (42.86%)	3 (8.57%)	1 (2.86%)
Total	29 (82.86%)	5 (14.29%)	1 (2.86%)

[Table/Fig-8]: Showing distribution of percentage stenosis of plaques in different age groups.

Age groups	<50% stenosis	50-69% stenosis	>69% stenosis
Males	18	4	1
Females	11	1	0
Total	29	5	1

[Table/Fig-9]: Showing distribution of percentage stenosis of plaques in males and females.

DISCUSSION

The study was performed to measure CIMT by Ultrasonography (USG) and to determine the prevalence of atherosclerotic plaque at carotid bifurcation. USG was taken as modality for evaluation as it is non-invasive, cost effective, safe, easily available and all Radiologists are well versed with technical proficiency to perform the present study. The present study included 20-70 years age group population to find the age distribution of plaques.

The CIMT is a well-described surrogate marker for cardiovascular disease and increased CIMT has been associated with higher incidence and higher prevalence of CHD and stroke [13,14]. Further, statins, which reduce major adverse cardiovascular events, have been shown to stabilise and regress CIMT [13]. In the Cardiovascular Health Study, CCA IMT greater than 0.87 mm and ICA IMT greater than 0.90 mm were associated with a progressively increased risk of cardiovascular events. It was also found that for each 0.20 mm increase in CCA IMT, the risk increased by approximately 27%. For each 0.55 mm increase in ICA IMT, the risk increased by approximately 30% [14].

Katamadze N et al., on British population in 2013 found that the mean values of CIMT in CCA were 0.82 ± 0.15 mm in males and 0.79 ± 0.13 mm in females while in ICA it was 0.88 ± 0.42 mm in males and 0.77 ± 0.27 mm in females [15].

Mannami T et al., in their study on Suita population of Japan found that CIMT in 50 to 79 years was 0.92 ± 0.13 mm for men and 0.89 ± 0.11 for women [16]. On the other hand, the CIMTs in the population aged 40 to 79 years of the asymptomatic carotid artery progression study were 0.91 ± 0.22 mm and 0.92 ± 0.24 for left CCA and 0.92 ± 0.22 and 0.92 ± 0.23 for those of the right CCA for males and females respectively [17]. On the other hand, the CIMTs in the Atherosclerosis Risk in Communities (ARIC) cohort were 0.84 ± 0.26 mm and 0.75 ± 0.21 for the left CCA of men and women and 0.82 ± 0.26 and 0.76 ± 0.22 for the right CCA of men and women, respectively [18]. The present study shows that the average CIMT in Indian population is less than average CIMT of above mentioned studies. The less CIMT in our population may be due to less cardiovascular risk factors compared to above population however the present study clearly shows that CIMT progressively increases with age and Increased mean CIMT was noted in 61-70 years age group in both males and females.

The present study population had total 35 volunteers with plaque. The overall prevalence of plaque was 14%. The prevalence of plaque was 2%, 4%, 2%, 24% and 38% in age group of 20-30, 31-40, 41-50, 51-60 and 61-70 years respectively from which it is clear that as the age progresses, the prevalence of plaque increases. Atherosclerosis is a complex slowly progressive disease which starts in early teenage years and progresses over decades [8]. Studies have shown that atherosclerosis begins as early as infancy [19]. Various morphological observational studies have shown that the plaque develops due to continuous progression of an uncomplicated fatty streak. The associations with risk factors as well as topographic distribution of fatty streaks and plaques are almost same [20]. The present study shows the prevalence of 2% plaques at 20-30 years age group which means onset of atherosclerosis from transformation from fatty streak is before 20 years. The point to note here is that in 41-50 years age group, the plaque prevalence was just 2% while at 51-60 years it was 24% and 61-70 years age group it was 38%. The sudden rise in plaque prevalence at fifth decade of life needs to be evaluated to initiate early preventive measures

to avoid atherosclerotic related Cerebrovascular Accidents (CVA), Coronary Heart Disease (CHD) and Peripheral Vascular Diseases (PVDs). Similar findings were noted in the MONICA project carried out in Augsburg which showed that the prevalence of plaque is strongly correlated to age (increasing trend in both sexes, $P < 0.001$) and the process of establishing plaques starts in men earlier than in women ($p < 0.01$) [21]. In a population study by Maria TM et al., on >16 000 people, 90% of subjects with severe carotid lesions were >55 years of age [22].

Prati P et al., in their study found that the global prevalence of carotid atherosclerosis was 25.4% in men and 26.4% in women [23]. The present study revealed plaque prevalence as 18.4% in males and 9.6% in females in Indian population which clearly reveals that there is significant higher no of carotid plaque in males than females. The study by Magyar MT et al., also revealed higher prevalence of carotid stenosis in men which was 1.2% than women of 0.7% in >60 years age group [22].

Mannami T et al., in their study on Suita population of Japan Indicated that 4.4% of their subjects aged 50 to 79 years, had a stenosis level >50% [16]. In contrast, as part of the MONICA project carried out in Augsburg on 1388 male and female subjects, >75% stenosis was observed in only 0.6% of the participants aged 25 to 64 years, whereas Colgan MP et al., reported that only 4% of 348 participants had stenosis >50% and only 1% had stenosis >80% [21,24]. In the Framingham Study, 8% of the 1189 members of the cohort aged 66 to 93 years had stenosis of $\geq 50\%$ [25]. Based on various population-based studies and the accuracy of carotid duplex ultrasonography, the estimated prevalence of Carotid artery stenosis of 60%-99% in the general population older than age 65 years is about 1% [26]. The present study shows that 14.3% of total plaque has resulted in 50-69% stenosis and 2.9% had resulted in >69% stenosis. However, overall prevalence of percentage stenosis is 11.6% for <50% stenosis, 2% for 50-69% stenosis and was only 0.4% for >69% stenosis.

A meta-analytical study which included various studies published from 1966 to 2003 in which digital subtraction angiography was taken as reference standard reported that duplex ultrasonography has sensitivity and specificity of 98% and 88% respectively for CAS of 50% or greater while for CAS of 70% or more, the sensitivity and specificity was 90% and 94% respectively [27]. It clearly indicates that duplex ultrasonography is a good tool for evaluation of carotid. But due to low prevalence of CAS in general population, the possibility of false positive results will be much higher in general population based study [27].

United States Preventive Services Task Force (USPSTF) found that although there is overall reduction of strokes by 3.5% if Carotid Endarterectomy (CEA) surgery is performed by expert surgeons in a selected group of patients as compared to outdated medical management but in case of asymptomatic population with CAS, the difference in overall reduction of stroke by modern medical management and surgery will be much lower [27]. Furthermore, there are no evidence that identification of asymptomatic CAS will be of any help in changing the treatment policy beyond standard medical therapy for CVS disease prevention [27].

USPSTF also found that the diagnosis as well as treatment of CAS can cause harms. The identification of carotid stenosis by screening procedures will lead to multiple diagnostic tests and unnecessary interventions which may lead to serious harms. The 30-day stroke or mortality associated with CEA is approx. 2.45% to 5% in various centers [27]. Post CEA Myocardial infarctions are reported to be 0.8% to 2.2%. The 30-day stroke or mortality rate after carotid angioplasty and stenting is also around 3.1% to 3.8%. Thus, the overall associated harms of screening asymptomatic carotid artery followed by interventions are more than expected benefit. In view of above, it was concluded in 2014 by USPSTF with moderate certainty that the harms of screening for asymptomatic CAS outweigh the benefits [27].

Our study was done in only 250 volunteers and shows prevalence of >69% stenosis as only 0.4%. Many studies by different societies have shown that there is no benefit in treatment of patient by CEA and risk is high as compared to benefit by CEA [6,27,28,29]. So detection of carotid stenosis will lead to unnecessary investigation in patients without any benefit. Therefore till further scientific study available to substantiate significant benefits of screening, screening for carotid atherosclerosis is not recommended in asymptomatic population. However, clinicians dealing with high cardiovascular risks cases have the option to use carotid doppler study in selected cases.

LIMITATION

The study was conducted on limited sample size in a relatively healthy volunteers where socioeconomic and education status were not taken which can affect the plaque prevalence. Ultrasonography is user dependant which can affect the prevalence study and plaque analysis.

CONCLUSION

Although, overall prevalence of carotid plaque is 14% and most of these lies in ICA but >69% stenosis is present only in 0.4% of general population. The plaque prevalence is 24% and 38% in age groups of 51-60 years and 61-70 years respectively showing higher prevalence in advancing age which is statistically significant ($p < 0.001$). Presently, the risk of treatment is more than expected benefit. Screening of carotid in asymptomatic population will lead to unnecessary investigations, increased workload on health care system, legal issues, increased expenditures and apprehension for patients and may even harm the patients by these unwarranted investigations or surgeries. So screening Carotid Doppler study in asymptomatic population is not recommended in present scenario and standard preventive treatments based on traditional cardiovascular risk factors can be offered till scientific knowledge available to substantiate benefits of screening.

REFERENCES

- [1] Strong K, Mathers C, Bonita R. Preventing stroke: saving lives around the world. *Lancet Neurol.* 2007;6(2):182-87.
- [2] Furie KL, Kasner SE, Adams RJ. Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: a guideline for healthcare professionals from the American heart association/American stroke association. *Stroke.* 2011;42:227-76. Available from: <https://www.ncbi.nlm.nih.gov/pubmed/20966421>
- [3] Abraham J, Rao PS, Inbaraj SG, Shetty G, Jose CJ. An epidemiological study of hemiplegia due to stroke in South India. *Stroke.* 1970;1(6):477-81.
- [4] Das SK, Banerjee TK, Biswas A, Roy T, Raut DK, Mukherjee CS, et al. A prospective community-based study of stroke in Kolkata, India. *Stroke.* 2007;38(3):906-10. Available from: <http://stroke.ahajournals.org/content/38/3/906>
- [5] Dalal PM, Malik S, Bhattacharjee M, Trivedi ND, Vairale J, Bhat P, et al. Population-based stroke survey in Mumbai, India: incidence and 28 -day case fatality. *Neuroepidemiology.* 2008;31:254-61. Available from: <https://www.ncbi.nlm.nih.gov/pubmed/18931521>
- [6] Mozzafarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, et al. On behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics-2016 update: a report from the American Heart Association. *Circulation.* 2016;133(4):e38-360.
- [7] Golledge J, Greenhalgh RM, Davies AH. The symptomatic carotid plaque. *Stroke.* 2000;31(3):774-81.
- [8] Anne G Osborn. *Vasculopathy, Osborn's Brain: imaging, pathology and anatomy.* 1st edn. Canada. Amirsys. 2013:247-60.
- [9] Spence JD. Ultrasound measurement of carotid plaque as a surrogate outcome for coronary artery disease. *Am J Cardiol.* 2002;89(4A):10B-15B.
- [10] Ross R. Rous-Whipple Award Lecture. Atherosclerosis: a defense mechanism gone awry. *Am J Pathol.* 1993;143(4):987-1002.
- [11] Dalal P, Bhattacharya M, Vairale J, Bhat P. UN millennium development goals: Can we halt the stroke epidemic in India? *Annals of Indian Academy Neurology.* 2007;10:130-36.
- [12] Insull WJ. The pathology of atherosclerosis: plaque development and plaque responses to medical treatment. *Am J Med.* 2009;122(1 Suppl):S3-S14.
- [13] Chambless LE, Heiss G, Folsom AR. Association of coronary heart disease incidence with carotid arterial wall thickness and major risk factors: the Atherosclerosis Risk in Communities (ARIC) study, 1987-1993. *Am J Epidemiol.* 1997;(146)1997:483-94.
- [14] O'Leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson SK Jr. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. *N Engl J Med.* 1999;340(1):14-22.
- [15] Katamadze N, Berstein L, Grishkin Y. Subclinical carotid atherosclerosis in patients with traditional cardiovascular risk factors. *Eur Heart J.* 2013;34(suppl 1):46-47.
- [16] Mannami T, Konishi M, Baba S, Nishi N, Terao A. Prevalence of asymptomatic carotid atherosclerotic lesions detected by high-resolution ultrasonography and its relation to cardiovascular risk factors in the general population of a Japanese City. *Stroke.* 1997;28(3):518-25.
- [17] Espeland MA, Hoen H, Byington R, Howard G, Riley WA, Furberg CD. Spatial distribution of carotid intimal-medial thickness as measured by B-mode ultrasonography. *Stroke.* 1994;25:1812-19.
- [18] Crouse JR, Goldbourt U, Evans G, Pinsky J, Sharrett AR, Sorlie P, et al. Risk factors and segment-specific carotid arterial enlargement in the Atherosclerosis Risk in Communities (ARIC) cohort. *Stroke.* 1996;27:69-75.
- [19] Milei J, Ottaviani G, Lavezzi AM, Grana DR, Stella I, Matturri L. Perinatal and infant early atherosclerotic coronary lesions. *Can J Cardiol.* 2008;24(2):137-41.
- [20] McGill HC, McMahan CA, Herderick EE, Malcom GT, Tracy RE, Strong JP, et al. Origin of atherosclerosis in childhood and adolescence. *Am J Clin Nutr.* 2000;72(5):1307s-15s.
- [21] Gostomzyk JG, Heller WD, Gerhardt P, Lee PN, Keil U. B-scan ultrasound examination of the carotid arteries within a representative population (MONICA Project Augsburg). *Klin Wochenschr.* 1988;66(suppl 11):58-65.
- [22] Magyar MT, Szikszai Z, Balla J, Valikovics A, Kappelmayer J, Imre S, et al. Early-onset carotid atherosclerosis is associated with increased intima-media thickness and elevated serum levels of inflammatory markers. *Stroke.* 2003;34:58-63.
- [23] Prati P, Vanuzzo D, Casaroli M, Di Chiara A, De Biasi F, Feruglio GA, et al. Prevalence and determinants of carotid atherosclerosis in a general population. *Stroke.* 1992;23(12):1705-11.
- [24] Colgan MP, Strode GR, Sommer DJ, Gibbs JL. Prevalence of asymptomatic carotid disease: results of duplex scanning in 348 unselected volunteers. *J Vasc Surg.* 1988;8:674-78.
- [25] O'Leary DH, Anderson KM, Wolf PA, Evans JC, Poehlman HW. Cholesterol and carotid atherosclerosis in older persons: the Framingham Study. *Ann Epidemiol.* 1992;2(1-2):147-53.
- [26] Craig WJ. Health effects of vegan diets. *Am J Clin Nutr.* 2009;89(5):1627S-33S.
- [27] Jonas DE, Feltner C, Amick HR, Sheridan S, Zheng ZJ, Watford DJ, et al. Screening for Asymptomatic Carotid Artery Stenosis: A systematic review and meta-analysis for the US. Preventive Services Task Force. Rockville, MD: Agency for Healthcare Research and Quality; 2014
- [28] Society for Vascular Surgery. SVS Position Statement on Vascular Screenings, 2007. Accessed at http://www.vascularweb.org/_CONTRIBUTION_PAGES/Patient_Information/screenings/SVS_Position_Statement_on_Vascular_Screenings.html
- [29] Goldstein LB, Bushnell CD, Adams RJ, Appel LJ, Braun LT, Chaturvedi S et al. Guidelines for the primary prevention of stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2011;42(2):517-84.

PARTICULARS OF CONTRIBUTORS:

1. Head, Department of Radiodiagnosis, Indian Naval Hospital Ship Sanjivani, Kochi, Kerala, India.
2. Head, Department of Radiodiagnosis, Command Hospital (Air Force), Bangalore, Karnataka, India.
3. Associate Professor, Department of Radiodiagnosis, Armed Forces Medical College, Pune, Maharashtra, India.
4. Associate Professor, Department of Radiodiagnosis, Command Hospital (Air Force), Bangalore, Karnataka, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Amit Kumar Choubey,
Head, Department of Radiodiagnosis, Indian Naval Hospital Ship Sanjivani, Naval Base, Kochi-682004, Kerala, India.
E-mail: amyafmc@gmail.com

Date of Submission: **Oct 22, 2017**
Date of Peer Review: **Jan 23, 2018**
Date of Acceptance: **Feb 20, 2018**
Date of Publishing: **May 01, 2018**

FINANCIAL OR OTHER COMPETING INTERESTS: None.