

Distensibility coefficient of the common carotid artery in acute cerebral infarct by M-mode ultrasound: A cross-sectional study

Vishwanath T Thimmaiah, C Anupama

Department of Radiodiagnosis, JSS Medical College and Hospital, JSS University, Mysore, Karnataka, India

Abstract

Introduction: Distensibility coefficient (DC) is abnormal in most cases of acute cerebral infarct patients. Intima-media thickness (IMT) is measured by B (brightness)-mode and distensibility by M (motion)-mode ultrasound. DC is not routinely measured in clinical practice even though it is the earliest functional change to occur in common carotid arteries. IMT represents structural change and DC represents functional changes that occur in common carotid.

Aim: DC is a valid independent indicator for risk assessment of acute cerebral infarcts. The present study is aimed at knowing the association of abnormal DC with acute cerebral infarct patients.

Materials and Methods: Cross sectional study was undertaken over a period of 9 months with 210 cases of acute cerebral infarct. Distensibility of common carotid artery was measured by M-mode ultrasound. Cramer's V value ranged from 0.0 to 1.0 and $P < 0.05$ was considered statistically significant.

Results: DC was reduced in 62.8% of the total 210 cases of acute cerebral infarct. Maximum cases (38%) were in the age groups of 61–70 years. Cramer's V value ranged from <15 to $<24 \times 10^{-3}$ kPa ($P < 0.001$). There was a good association between reduced DC and acute cerebral infarct in the present study.

Conclusion: DC is a valid independent risk factor for acute cerebral infarct. DC represents functional vessel wall property and decreases with age. M-mode-derived measurements for DC are valid, and abnormal DC value is associated well with acute cerebral infarct.

Keywords: Distensibility, functional, intima-media thickness

Address for correspondence: Dr. Vishwanath T Thimmaiah, Department of Radiodiagnosis, JSS Medical College and Hospital, JSS University, Mysore - 570 004, Karnataka, India.
E-mail: vishurad@gmail.com

INTRODUCTION

The distensibility of an artery is a reflection of mechanical stress affecting the arterial wall during each cardiac cycle. Decreased arterial distensibility or increased arterial wall stiffness is a common pathological mechanism for many factors associated with cerebrovascular and cardiovascular diseases.^[1] Structural and functional changes of vessel

will do occur with aging resulting in decreased carotid distensibility and increased stiffness as the age progresses. Local distensibility of a vessel is important for protecting the arterial wall from damage during each cardiac cycle, particularly for the common carotid that is more susceptible to vascular damage. Common carotid intima-media thickness (IMT) is a strong predictor of future vascular

Access this article online

Quick Response Code:



Website:

www.wajradiology.org

DOI:

10.4103/wajr.wajr_23_17

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Thimmaiah VT, Anupama C. Distensibility coefficient of the common carotid artery in acute cerebral infarct by M-mode ultrasound: A cross-sectional study. West Afr J Radiol 2018;25:90-4.

events and acts as a surrogate marker for atherosclerosis. Although carotid IMT assesses the structural properties of the carotid artery, it does not assess the functional properties of the vessel. Distensibility coefficient (DC) assesses the functional property of the vessel and changes in arterial distensibility occurring much earlier than clinical symptoms or IMT changes alone. DC is a measure of the arterial distensibility with each cardiac pulsation. IMT and DC represent different vessel wall properties with decreased DC being common pathologic mechanism for many factors that lead to the occurrence and progression of vascular changes.^[2] Reduced common carotid DC can be used as an independent risk factor for acute cerebral infarct.^[3]

Aim

The present study was undertaken to know the association of DC of common carotid artery in acute cerebral infarct patients.

MATERIALS AND METHODS

Study design

A cross-sectional study was undertaken in the Department of Radiology, JSS Medical College and Hospital, Mysore, Karnataka, India, over a period of 9 months from August 2012 to April 2013.

Subjects

All adult cases with acute cerebral infarct who were referred for carotid Doppler examination were included in the study.

Inclusion criteria

All adults of age more than 18 years with acute cerebral infarcts diagnosed by computed tomography (CT) or magnetic resonance imaging (MRI).

Exclusion criteria

1. Pediatrics with acute cerebral infarct
2. Acute or chronic thrombus involving carotid arteries
3. Neoplastic lesions involving carotid bodies
4. Acute cerebral venous thrombosis
5. Acute intracerebral hematomas were excluded from the study.

Data collection

Acute cerebral infarct patients who were diagnosed by cross-sectional imaging were referred for carotid Doppler examination. Brachial artery blood pressure was measured noninvasively, and pulse pressure was calculated as the difference between maximal systolic and diastolic blood pressure. Patients were then placed in supine position with both shoulders over a pillow to provide adequate support. Neck was slightly hyperextended to make

Doppler examination comfortable. A high frequency probe (7–12 MHz) was placed on the common carotid artery on the side of acute cerebral infarct that was diagnosed on cross-sectional imaging with slight compression so as to obtain good longitudinal image of the artery. M-mode ultrasound was used to record the maximal change in the diameter of the common carotid artery during systole and diastole phase of each cardiac cycle; M-mode cursor was placed 2 cm proximal to common carotid bifurcation at a plaque-free site to obtain the movement of interfaces that involves serial measurements of the location of a carotid wall echo structure from periodic pulsing in a single X-axis direction of the transducer. M-mode displays the time traces of the depth of reflecting interfaces over a few cardiac cycles. Maximum and minimum differences between the traces of opposite walls are used as estimates of the systolic and diastolic diameters, respectively, on a single given image [Figure 1]. DC was calculated from the formula $(2 \times \Delta d / Dd) / \Delta P$ (10^{-3} kPa)^[4] where Δd is change in systolic and diastolic diameter, ΔP is pulse pressure, and Dd is end-diastolic diameter. DC of $<24 \times 10^{-3}$ kPa was taken as the cutoff between normal and abnormal values.^[5]

Statistical analysis

All the data were entered in Microsoft Excel sheet for analysis. Categorical variables were reported as proportions. Analysis was done using Microsoft Excel 2013 and SPSS 20.0 software (IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp). Cramer's V and P values were calculated for each age group.

RESULTS

The study comprises 210 cases of acute cerebral infarct with 82.8% males and 27.2% females. Mean age was 56.4 years for males and 54.7 years for females. The lowest age was 31 years in both males and females with highest age being 75 years and 78 years for males and females, respectively. Majority of the cases (73.3%) were having acute cerebral infarct on the right side in both males (74.1%) and females (69.4%). Maximum cases were seen between the age groups of 61–70 years (38%) [Tables 1-3]. A total of 132 (62.8%) cases showed abnormal DC ranging from >12 to $<24 \times$

Table 1: Age wise distribution of acute cerebral infarct

Age (years)	Acute cerebral infarct		Total
	Right (%)	Left (%)	
31-40	16 (66.7)	8 (33.3)	24
41-50	24 (49.0)	25 (51.0)	49
51-60	32 (74.4)	11 (25.6)	43
61-70	68 (85.0)	12 (15.0)	80
71-80	14 (100.0)	0	14
Total	154 (73.3)	56 (26.7)	210

10^{-3} kPa. There was progressive decrease in DC values as the age increases among different age group. Cramer's V value ranged from 0.021 to 0.036 showing an good association and *P* value was significant with values <0.001 [Table 4].

DISCUSSION

Acute cerebral infarct is a serious health problem with high morbidity and mortality worldwide. Arterial distensibility is defined as the ability of the artery to expand and relax with each cardiac pulsation. It represents local compliance of blood vessel and changes with arterial pressure changes.^[6] Many risk factors are known for the development of acute cerebral infarct, but they do not precisely predict which individuals will develop infarct over a period of time in future. Increased arterial stiffness or reduced DC is an intermediate factor in the pathway of development from risk factors to acute cerebral infarct. Reduced arterial DC is also a common pathologic mechanism for many associated risk factors that lead to initiation and progression of the arterial changes that predisposes to acute cerebral infarct.^[7] DC represents functional property of an artery and its impairment occurs much earlier in the atherosclerotic process than the structural wall changes or clinical symptoms.^[8] Measurements of arterial stiffness and IMT of common carotid help to identify their roles in

pathogenesis of cerebral infarcts and with early detection of these changes leading for the development of more effective strategies for the prevention of cerebral infarct. Mechanical stress or pressure effect of blood on the carotid arterial wall is the main cause for pathogenesis of increased stiffness which, in turn, reduces the local compliance of an artery during each cardiac cycle.^[9] Increased arterial stiffness also occurs as a result of damaging effect on the arterial wall over a period of time by many other associated cerebrovascular risk factors. Stiffening of the arteries is one of the earliest detectable manifestations of adverse changes that occur within the arterial wall from stress-related injuries.^[10] Arterial distensibility also depends on the age of individuals.^[11] Elastin is the basic component of elastic fibers, with its gene located on chromosome 7 in humans and forms the bulk of tunica media of large and medium-sized arteries.^[12] Aging causes loss of these elastic fibers in the arterial wall leading to an increase in arterial stiffness.

DC, compliance coefficient, pressure-strain elastic modulus, Young's modulus, and pulse wave velocity are among the several parameters used for arterial stiffness measurement. In most of these parameters, the relationships between distensibility of artery, indicated by change in lumen diameters, pulse pressure, and carotid lumen diameter are included in the study.^[13] Measurements of these variations in carotid arterial diameters during each cardiac cycle are highly valid in the study of arterial pathophysiology and mechanical properties of the arterial wall.^[14] DC is a valid indicator of these functional properties of carotid arteries and can be used as a predictor for the initiation or progression of atherosclerosis and arterial hypertension, which forms the major risk factors for the development of acute cerebral infarcts.^[15] Reduced DC

Table 2: Sex distribution of acute cerebral infarct

Sex	Acute cerebral infarcts		Total (%)
	Right (%)	Left (%)	
Male cases	129 (74.1)	45 (25.9)	174 (82.8)
Female cases	25 (69.4)	11 (30.6)	36 (17.2)
Total cases	154 (73.3)	56 (26.7)	210 (100)

Table 3: Cross-tabulation of Distensibility coefficient with age groups

Age (years)	DC		Total
	Normal	Abnormal	
31-40	20	2	22
41-50	24	31	55
51-60	15	39	54
61-70	12	44	56
71-80	7	16	23
Total (%)	78 (37.2)	132 (62.8)	210

DC – Distensibility coefficient

Table 4: Distensibility coefficient values among different age groups

Age (years)	DC	Total (n=210)	Cramer's V value	<i>P</i>
31-40	$<20-24 \times 10^{-3}$ kPa	22 (10.4)	0.021	<0.001
41-50	$<18-20 \times 10^{-3}$ kPa	55 (26.2)	0.023	<0.001
51-60	$<15-18 \times 10^{-3}$ kPa	54 (25.8)	0.028	<0.001
61-70	$<12-15 \times 10^{-3}$ kPa	56 (26.7)	0.033	<0.001
71-80	$<15 \times 10^{-3}$ kPa	23 (10.9)	0.036	<0.001

DC – Distensibility coefficient

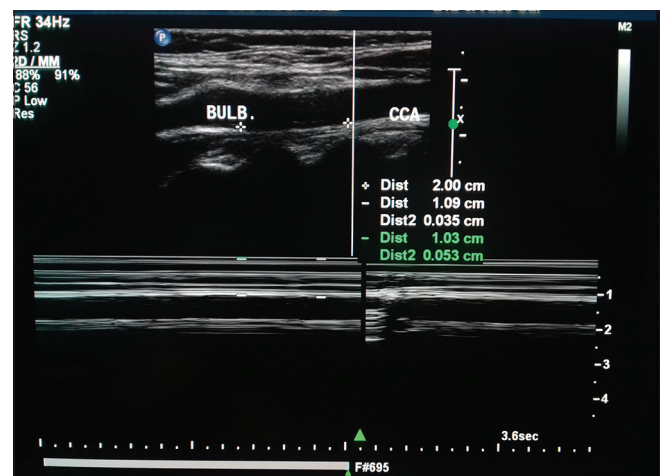


Figure 1: Systolic and diastolic measurement of common carotid artery on M-mode ultrasound

can be considered as an independent risk factor by itself for the future development of cerebrovascular disease as reduced distensibility being the common pathologic mechanism for many factors that lead to occurrence and progression of vascular changes, much earlier than increased IMT or clinical symptoms. DC is a measure of the arterial distensibility with each cardiac pulsation. IMT and DC represent different vessel wall properties with IMT representing mechanical and DC representing functional property of a vessel.^[16] The true relation between DC and increased risk for the development of acute cerebral infarct is unknown, but several hypothesis has been postulated for their association. Most important is the presence of atherosclerosis leading to both increased IMT and increased stiffness. Increased stiffness leads to reduced elasticity and increased vessel damage with both of these mechanisms applying each other for the development of acute infarct.

Arterial distensibility cannot be measured directly, but indirect measurement is possible with M-mode ultrasound by calculating dynamic measurements during each cardiac cycle.^[17] Common carotid artery DC measurement is easy to perform clinically with little additional time and is a reliable method.^[18] DC is calculated by considering maximum diameter change of lumen diameters during each cardiac cycle and blood pressure changes.^[19] Small change in diameter in carotid arteries will have big changes in arterial distensibility and reproducibility of diameter measurement by M-mode ultrasound.^[20] Measurement of distensibility will further improve the characterization of atherosclerosis in common carotid arteries and forms a more sensitive marker for progression or regression of atherosclerosis. It also helps to select those patients who will require earlier and more aggressive antiatherogenic treatment.^[21] Noninvasive imaging methods to measure arterial stiffness are available and are easy to perform clinically.^[22] Ultrasound techniques are very safe, reliable, repeatable, and cost effective.^[23] M-mode is available with all ultrasound manufactures and can be repeated many times for averaging the values for age- and sex-matched ratios. Advantage with M-mode being both systolic and diastolic lumen diameters is displayed in a single image with which DC can be calculated in single sitting. Although ultrasound has limitations such as observer dependency, two-dimensional (2D) image data, and incomplete characterization of atheromatous plaques, it can be used as a best imaging modality for distensibility measurement. CT carotid angiogram (CTA) of common carotid can also be used to measure distensibility with added advantage that noncircular cross-sections can also be measured.^[24] The disadvantages of CTA are radiation exposure, availability, repeatability, and high cost. MRI with magnetic resonance

angiogram is another noninvasive imaging method available for distensibility measurement with carotid plaque characterization. 2D dark blood T2-weighted and three-dimensional T1 CINE imaging protocols are used for distensibility measurement but have limitations of high cost, availability, and complex technology. M-mode ultrasound measures both systolic and diastolic diameters of a cardiac cycle in one single image that can be captured by placing cursor over the carotid image obtained from B-mode. Similar image by B-mode would require sequential images to be captured and compared with each cardiac cycle, thus making its complex compared to M-mode. By measuring these parameters by M-ultrasound, which can be easily performed in day-to-day practice, future predictions of cerebral infarct can be done. All cerebral infarct patients referred for carotid evaluation, along with IMT, DC measurement should be implied in routine clinical practice. DC measurements are valid and can be used as a valid independent risk factor for acute cerebral infarct. Reduced distensibility is a marker of increased cerebrovascular risk in patients who already have manifestations of vascular disease or other cerebrovascular risk factors.

Limitations

DC was measured in common carotid artery on the side of cerebral infarct. However, similar changes can occur in contralateral common carotid or internal carotid artery which was not measured in our study. Distensibility measurement of internal carotid artery is difficult in many cases when compared to that of common carotid artery.

CONCLUSION

Reduced distensibility of common carotid is an independent risk factor for acute cerebral infarct. IMT represents structural wall property and DC represents functional vessel wall property of common carotids. M-mode-derived indices are valid and decreased DC is one of the important predictor for acute cerebral infarct and when measured increases the predictive power of carotid ultrasonography in acute cerebral infarcts.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Rothwell PM. Carotid artery disease and the risk of ischaemic stroke and coronary vascular events. *Cerebrovasc Dis* 2000;10 Suppl 5:21-33.
2. Oliver JJ, Webb DJ. Noninvasive assessment of arterial stiffness and risk of atherosclerotic events. *Arterioscler Thromb Vasc Biol*

- 2003;23:554-66.
3. Giannattasio C, Mancia G. Arterial distensibility in humans. Modulating mechanisms, alterations in diseases and effects of treatment. *J Hypertens* 2002;20:1889-99.
 4. Gamble G, Zorn J, Sanders G, MacMahon S, Sharpe N. Estimation of arterial stiffness, compliance, and distensibility from M-mode ultrasound measurements of the common carotid artery. *Stroke* 1994;25:11-6.
 5. van Popele NM, Grobbee DE, Bots ML, Asmar R, Topouchian J, Reneman RS, *et al.* Association between arterial stiffness and atherosclerosis: The Rotterdam study. *Stroke* 2001;32:454-60.
 6. Christensen T, Neubauer B. Increased arterial wall stiffness and thickness in medium-sized arteries in patients with insulin-dependent diabetes mellitus. *Acta Radiol* 1988;29:299-302.
 7. Hoeks AP, Brands PJ, Smeets FA, Reneman RS. Assessment of the distensibility of superficial arteries. *Ultrasound Med Biol* 1990;16:121-8.
 8. Selzer RH, Mack WJ, Lee PL, Kwong-Fu H, Hodis HN. Improved common carotid elasticity and intima-media thickness measurements from computer analysis of sequential ultrasound frames. *Atherosclerosis* 2001;154:185-93.
 9. Carallo C, Irace C, Pujia A, De Franceschi MS, Crescenzo A, Motti C, *et al.* Evaluation of common carotid hemodynamic forces. Relations with wall thickening. *Hypertension* 1999;34:217-21.
 10. Ziemann SJ, Melenovsky V, Kass DA. Mechanisms, pathophysiology, and therapy of arterial stiffness. *Arterioscler Thromb Vasc Biol* 2005;25:932-43.
 11. Safar ME, Girerd X, Laurent S. Structural changes of large conduit arteries in hypertension. *J Hypertens* 1996;14:545-55.
 12. Fazio MJ, Mattei MG, Passage E, Chu ML, Black D, Solomon E, *et al.* Human elastin gene: New evidence for localization to the long arm of chromosome 7. *Am J Hum Genet* 1991;48:696-703.
 13. Reneman RS, Hoeks AP, Westerhoff N. Non-invasive assessment of artery wall properties in humans: methods and interpretation. *J Vasc Invest* 1996;2:53-64.
 14. Denarić N, Garić J, Chironi G, Massonneau M, Laskri F, Salomon J, *et al.* Distribution of ultrasonographically-assessed dimensions of common carotid arteries in healthy adults of both sexes. *Atherosclerosis* 2000;148:297-302.
 15. Bots ML, Hoes AW, Koudstaal PJ, Hofman A, Grobbee DE. Common carotid intima-media thickness and risk of stroke and myocardial infarction: The Rotterdam study. *Circulation* 1997;96:1432-7.
 16. Kawasaki T, Sasayama S, Yagi S, Asakawa T, Hirai T. Non-invasive assessment of the age related changes in stiffness of major branches of the human arteries. *Cardiovasc Res* 1987;21:678-87.
 17. Reneman RS, Meinders JM, Hoeks AP. Non-invasive ultrasound in arterial wall dynamics in humans: What have we learned and what remains to be solved. *Eur Heart J* 2005;26:960-6.
 18. Wada T, Kodaira K, Fujishiro K, Maie K, Tsukiyama E, Fukumoto T, *et al.* Correlation of ultrasound-measured common carotid artery stiffness with pathological findings. *Arterioscler Thromb* 1994;14:479-82.
 19. Tsigoulis G, Vemmos K, Papamichael C, Spengos K, Daffertshofer M, Cimboneriu A, *et al.* Common carotid arterial stiffness and the risk of ischaemic stroke. *Eur J Neurol* 2006;13:475-81.
 20. Godia EC, Madhok R, Pittman J, Trocio S, Ramas R, Cabral D, *et al.* Carotid artery distensibility: A reliability study. *J Ultrasound Med* 2007;26:1157-65.
 21. Smilde TJ, Wollersheim H, Van Langen H, Stalenhoef AF. Reproducibility of ultrasonographic measurements of different carotid and femoral artery segments in healthy subjects and in patients with increased intima-media thickness. *Clin Sci (Lond)* 1997;93:317-24.
 22. Arnett DK, Chambless LE, Kim H, Evans GW, Riley W. Variability in ultrasonic measurements of arterial stiffness in the atherosclerosis risk in communities study. *Ultrasound Med Biol* 1999;25:175-80.
 23. Pannier BM, Avolio AP, Hoeks A, Mancia G, Takazawa K. Methods and devices for measuring arterial compliance in humans. *Am J Hypertens* 2002;15:743-53.
 24. Zhang J, Fletcher JG, Vrtiska TJ, Manduca A, Thompson JL, Raghavan ML, *et al.* Large-vessel distensibility measurement with electrocardiographically gated multidetector CT: Phantom study and initial experience. *Radiology* 2007;245:258-66.

Author Help: Online submission of the manuscripts

Articles can be submitted online from <http://www.journalonweb.com>. For online submission, the articles should be prepared in two files (first page file and article file). Images should be submitted separately.

1) First Page File:

Prepare the title page, covering letter, acknowledgement etc. using a word processor program. All information related to your identity should be included here. Use text/rtf/doc/pdf files. Do not zip the files.

2) Article File:

The main text of the article, beginning with the Abstract to References (including tables) should be in this file. Do not include any information (such as acknowledgement, your names in page headers etc.) in this file. Use text/rtf/doc/pdf files. Do not zip the files. Limit the file size to 1 MB. Do not incorporate images in the file. If file size is large, graphs can be submitted separately as images, without their being incorporated in the article file. This will reduce the size of the file.

3) Images:

Submit good quality color images. Each image should be less than 4096 kb (4 MB) in size. The size of the image can be reduced by decreasing the actual height and width of the images (keep up to about 6 inches and up to about 1800 x 1200 pixels). JPEG is the most suitable file format. The image quality should be good enough to judge the scientific value of the image. For the purpose of printing, always retain a good quality, high resolution image. This high resolution image should be sent to the editorial office at the time of sending a revised article.

4) Legends:

Legends for the figures/images should be included at the end of the article file.