



ORIGINAL RESEARCH

**DOPPLER USG ASSESSMENT OF MORPHOLOGIC AND HEMODYNAMIC PARAMETERS OF CAROTID ARTERY AND ITS COMPARISON BETWEEN SMOKERS AND NON-SMOKERS**

N. Prasanna Venkatesh<sup>1</sup>, G.Yuvabalakumaran<sup>2</sup>, S. B. Sachin<sup>3</sup>, R.Sathiyarayanan<sup>4</sup>, B. Pravitha<sup>5</sup>

<sup>1</sup>Post graduate resident Department of Radio-diagnosis Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, Salem, India

<sup>2</sup>MDRD, Professor & HOD Department of Radio-diagnosis Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, Salem, India

<sup>3</sup>Assistant Professor Department of Radio-diagnosis Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, Salem, India

<sup>4</sup>DMRD, DNB Assistant Professor Department of Radio-diagnosis Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, Salem, India

<sup>5</sup>DMRD, DNB Senior Resident Department of Radio-diagnosis Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, Salem, India

**\*Corresponding Author:** G.Yuvabalakumaran MDRD, Professor & HOD Department of Radio-diagnosis Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, Salem, India. Email: vmtrust@vmu.edu.in

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**Abstract**

**Background:** Atherosclerosis is a progressive vascular condition that remains a leading cause of morbidity and mortality worldwide. Cigarette smoking is an established risk factor for vascular endothelial damage and accelerates atherosclerotic changes through both direct and indirect mechanisms. The carotid arteries, being superficial and accessible, serve as a valuable window for early detection of systemic atherosclerosis. High-resolution Doppler ultrasonography (USG) allows for non-invasive evaluation of both morphologic and hemodynamic parameters of the carotid arterial system, including intima-media thickness (IMT), plaque formation, luminal narrowing, peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistive index (RI). Comparing these parameters between smokers and non-smokers offers insight into the early vascular changes induced by tobacco exposure.

**Aim:** To assess and compare the morphologic and hemodynamic parameters of the carotid arteries using Doppler ultrasonography in smokers and non-smokers.

**Materials and Methods:** This cross-sectional observational study was conducted on 100 adult subjects aged 30–60 years, comprising 50 chronic smokers ( $\geq 10$  pack-years) and 50 age- and sex-matched non-smokers. All participants underwent B-mode and color Doppler ultrasonography of bilateral common carotid arteries (CCA), carotid bulb, and internal carotid arteries (ICA). Morphologic evaluation included assessment of intima-media thickness, presence of plaques, and arterial wall echotexture. Hemodynamic parameters including PSV, EDV, and RI were measured in the CCA and ICA. Statistical comparison between the two groups was done using unpaired t-test and chi-square test, with p-value  $< 0.05$  considered significant

**Results:** The mean IMT in smokers was significantly higher compared to non-smokers ( $0.82 \pm 0.09$  mm vs.  $0.66 \pm 0.07$  mm,  $p < 0.001$ ). Carotid plaques were detected in 34% of smokers and only 6% of non-smokers. Hemodynamically, smokers demonstrated elevated peak systolic velocities and resistive indices in both CCA and ICA, indicating increased vascular resistance. The mean CCA PSV in smokers was  $96.3 \pm 12.8$  cm/s compared to  $81.7 \pm 10.4$  cm/s in non-smokers ( $p < 0.001$ ). A statistically significant difference in ICA RI values was also noted ( $0.78 \pm 0.06$  in smokers vs.  $0.70 \pm 0.05$  in non-smokers,  $p < 0.001$ ).

**Conclusion:** Doppler USG effectively demonstrates early structural and functional changes in the carotid arteries associated with smoking. Increased intima-media thickness, higher resistive indices, and greater plaque prevalence among smokers indicate early vascular aging and subclinical atherosclerosis. Routine Doppler screening in chronic smokers could aid in early identification of vascular risk, allowing timely lifestyle modifications and therapeutic

**Keywords:** Doppler Ultrasonography, Carotid Artery, Intima-Media Thickness, Hemodynamics, Smoking, Peak Systolic Velocity, Resistive Index, Atherosclerosis, Vascular Imaging, Smokers Vs. Non-Smokers

## INTRODUCTION

Atherosclerosis is a chronic inflammatory disease of the arterial wall characterized by progressive lipid accumulation, endothelial dysfunction, and vascular remodeling, ultimately leading to narrowing and stiffening of arteries <sup>1</sup>. It remains a leading cause of cerebrovascular accidents, myocardial infarction, and peripheral arterial disease. Among the numerous modifiable risk factors, cigarette smoking is one of the most potent and well-established contributors to vascular endothelial injury and early atherogenesis <sup>2</sup>. The deleterious effects of smoking are mediated through multiple mechanisms, including oxidative stress, altered lipid metabolism, increased sympathetic activity, and pro-inflammatory cytokine release. These changes promote vascular stiffness, increased intima-media thickness, and plaque formation, even in relatively young individuals <sup>3</sup>. The carotid arteries, particularly the common carotid artery (CCA) and internal carotid artery (ICA), are among the most accessible vascular beds for non-invasive imaging and represent a surrogate marker for systemic atherosclerosis <sup>4</sup>. Early morphologic changes such as thickening of the intima-media layer or plaque deposition often occur before clinical symptoms of cerebrovascular disease manifest. Evaluating these changes in asymptomatic individuals, especially those with high-risk behaviors like chronic smoking, offers an opportunity for early risk stratification and preventive intervention <sup>5</sup>.

Doppler ultrasonography (USG) has emerged as an essential diagnostic tool in vascular imaging due to its real-time capability, non-invasiveness, affordability, and absence of ionizing radiation. It permits high-resolution visualization of the arterial wall and enables simultaneous assessment of blood flow velocities [6]. Key Doppler parameters include peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistive index (RI), all of which provide insights into downstream resistance and vascular compliance. In addition to these hemodynamic markers, B-mode imaging allows for precise measurement of intima-media thickness (IMT) and detection of atherosclerotic plaques or calcifications <sup>7</sup>. Although carotid Doppler imaging has been extensively used in evaluating symptomatic cerebrovascular disease, its utility in detecting preclinical vascular changes among smokers is gaining importance. Several studies have shown that smokers tend to exhibit increased IMT, higher resistive indices, and a greater burden of plaque formation, even in the absence of overt cardiovascular symptoms <sup>8</sup>. However, there is a relative paucity of

comparative studies that systematically evaluate these parameters in smokers versus age-matched non-smokers using Doppler ultrasonography, particularly in the context of early screening.

This study was undertaken to address this gap by comparing both the morphologic and hemodynamic characteristics of the carotid arteries between smokers and non-smokers using high-resolution Doppler USG. By evaluating differences in IMT, plaque burden, PSV, EDV, and RI, this research aims to highlight the vascular impact of smoking and reinforce the potential role of Doppler USG in early atherosclerosis detection and risk stratification among high-risk individuals.

## MATERIALS AND METHODS

**Study Design And Setting:** This cross-sectional observational study was conducted in the Department of Radiodiagnosis at a tertiary care academic hospital in India over a period of 12 months. The study protocol was approved by the institutional ethics committee prior to initiation. All participants were informed about the study objectives, procedures, and confidentiality measures, and written informed consent was obtained from each individual before enrolment.

**Study Population:** A total of 100 adult male subjects aged between 30 and 60 years were included in the study. They were divided into two groups: Group A comprised 50 chronic smokers, and Group B included 50 age- and sex-matched non-smokers who served as the control group. Smokers were defined as individuals with a smoking history of at least 10 pack-years, irrespective of current smoking status. Non-smokers were individuals with no history of active smoking or significant passive exposure. All subjects underwent detailed history-taking, clinical examination, and Doppler ultrasonography of the carotid arteries.

**Inclusion Criteria:** The study included male participants aged 30–60 years who were either chronic smokers ( $\geq 10$  pack-years) or non-smokers without any significant secondhand smoke exposure. All individuals were clinically stable and free from overt cardiovascular or neurological disease at the time of enrollment.

**Exclusion Criteria:** Subjects were excluded if they had a known history of cerebrovascular accidents, transient ischemic attacks, coronary artery disease, diabetes mellitus, hypertension, hyperlipidemia, chronic kidney disease, peripheral arterial disease, or any systemic inflammatory or connective tissue disorder. Individuals

on lipid-lowering medications, antiplatelet therapy, or vasodilators were also excluded. Additionally, any patient with inadequate Doppler image quality due to short neck, obesity, or severe calcification was not included.

**Doppler Ultrasonography Protocol:** All participants underwent bilateral carotid Doppler ultrasonography using a high-resolution B-mode and color Doppler system equipped with a 7–12 MHz linear transducer. The examination was performed with the subject lying in a supine position with the head tilted slightly to the contralateral side. The carotid arteries were scanned longitudinally and transversely along the course of the common carotid artery (CCA), carotid bifurcation, and internal carotid artery (ICA) on both sides.

Morphologic assessment included measurement of intima-media thickness (IMT) of the far wall of the distal CCA approximately 1 cm proximal to the bifurcation. A mean of three measurements was recorded from each side and averaged for analysis. Plaques were defined as localized protrusions into the lumen  $\geq 1.5$  mm in thickness or with evidence of focal echogenicity and shadowing. Arterial wall texture was assessed qualitatively.

Hemodynamic parameters recorded included peak systolic velocity (PSV) and end-diastolic velocity (EDV) in both the CCA and ICA. These were measured by placing the sample volume parallel to the blood flow with an angle of insonation  $\leq 60^\circ$ . The resistive index (RI) was calculated using the formula:  $RI = (PSV - EDV) / PSV$ . Measurements were taken

during relaxed breathing and in the absence of external compression.

**Statistical Analysis:** Data were compiled using Microsoft Excel and analyzed using SPSS software version 25.0. Continuous variables such as IMT, PSV, EDV, and RI were expressed as mean  $\pm$  standard deviation (SD). Categorical variables such as plaque presence were expressed in frequencies and percentages. Intergroup comparisons between smokers and non-smokers were performed using unpaired Student's t-test for continuous variables and chi-square test for categorical variables. A p-value of  $<0.05$  was considered statistically significant for all analyses.

## RESULTS

The present study included 100 adult male participants divided equally into two groups: 50 smokers and 50 non-smokers. All participants underwent bilateral carotid Doppler ultrasonography for morphologic and hemodynamic assessment of the common carotid artery (CCA) and internal carotid artery (ICA).

Comparative analysis was performed between the two groups for variables including intima-media thickness (IMT), presence of plaques, peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistive index (RI). The results demonstrate significant differences in several parameters between smokers and non-smokers, suggesting early vascular alterations in smokers.

Table 1 shows that the mean age of participants in both groups was comparable, ensuring effective age-matching. There was no statistically significant difference in the age profile between smokers and non-smokers. **Table**

### 1. Age distribution of study participants

| Age group (years) | Smokers (n) | Non-smokers (n) | Total (n)  |
|-------------------|-------------|-----------------|------------|
| 30–39             | 16          | 18              | 34         |
| 40–49             | 19          | 17              | 36         |
| 50–60             | 15          | 15              | 30         |
| <b>Total</b>      | <b>50</b>   | <b>50</b>       | <b>100</b> |

Table 2 shows that the mean age of smokers was  $44.3 \pm 7.1$  years and that of non-smokers was  $43.7 \pm 6.9$  years, with no statistically significant difference ( $p = 0.63$ ).

**Table 2. Mean age of smokers vs. non-smokers**

| Group       | Mean age (years) $\pm$ SD | p-value |
|-------------|---------------------------|---------|
| Smokers     | $44.3 \pm 7.1$            |         |
| Non-smokers | $43.7 \pm 6.9$            | 0.63    |

Table 3 shows that the mean carotid IMT was significantly higher in smokers ( $0.82 \pm 0.09$  mm) compared to non-smokers ( $0.66 \pm 0.07$  mm), with  $p < 0.001$ , indicating early structural changes in arterial walls.

**Table 3. Comparison of carotid intima-media thickness (IMT)**

| Group       | Mean IMT (mm) $\pm$ SD | p-value |
|-------------|------------------------|---------|
| Smokers     | $0.82 \pm 0.09$        |         |
| Non-smokers | $0.66 \pm 0.07$        | <0.001  |

Table 4 shows that carotid plaques were detected in 34% of smokers, compared to only 6% of non-smokers, demonstrating a strong association between smoking and focal atherosclerotic changes.

**Table 4. Prevalence of carotid plaques**

| Group       | Plaques present (n, %) | Plaques absent (n, %) |
|-------------|------------------------|-----------------------|
| Smokers     | 17 (34%)               | 33 (66%)              |
| Non-smokers | 3 (6%)                 | 47 (94%)              |

Table 5 shows that smokers had a significantly higher mean CCA PSV ( $96.3 \pm 12.8$  cm/s) compared to non-smokers ( $81.7 \pm 10.4$  cm/s), with  $p < 0.001$ .

**Table 5. Mean peak systolic velocity (PSV) in the common carotid artery (CCA)**

| Group       | CCA PSV (cm/s) $\pm$ SD | p-value |
|-------------|-------------------------|---------|
| Smokers     | $96.3 \pm 12.8$         |         |
| Non-smokers | $81.7 \pm 10.4$         | <0.001  |

Table 6 shows that smokers had lower CCA EDV values compared to non-smokers ( $24.8 \pm 3.9$  cm/s vs.  $28.2 \pm 4.2$  cm/s), with  $p < 0.001$ , indicating reduced arterial compliance.

**Table 6. Mean end-diastolic velocity (EDV) in the CCA**

| Group       | CCA EDV (cm/s) $\pm$ SD | p-value |
|-------------|-------------------------|---------|
| Smokers     | $24.8 \pm 3.9$          |         |
| Non-smokers | $28.2 \pm 4.2$          | <0.001  |

Table 7 shows that the mean CCA RI was significantly elevated in smokers ( $0.74 \pm 0.04$ ) compared to non-smokers ( $0.66 \pm 0.03$ ), suggesting increased vascular resistance ( $p < 0.001$ ).

**Table 7. Resistive index (RI) in the common carotid artery**

| Group       | CCA RI $\pm$ SD | p-value |
|-------------|-----------------|---------|
| Smokers     | $0.74 \pm 0.04$ |         |
| Non-smokers | $0.66 \pm 0.03$ | <0.001  |

Table 8 shows that ICA PSV was significantly higher in smokers ( $109.4 \pm 15.1$  cm/s) compared to non-smokers ( $92.6 \pm 11.8$  cm/s), indicating altered hemodynamics ( $p < 0.001$ ).

**Table 8. Mean ICA peak systolic velocity (PSV)**

| Group       | ICA PSV (cm/s) $\pm$ SD | p-value  |
|-------------|-------------------------|----------|
| Smokers     | $109.4 \pm 15.1$        |          |
| Non-smokers | $92.6 \pm 11.8$         | $<0.001$ |

Table 9 shows that ICA EDV was lower in smokers than non-smokers ( $26.3 \pm 4.2$  cm/s vs.  $30.1 \pm 4.9$  cm/s), and this difference was statistically significant ( $p = 0.002$ ).

**Table 9. Mean ICA end-diastolic velocity (EDV)**

| Group       | ICA EDV (cm/s) $\pm$ SD | p-value |
|-------------|-------------------------|---------|
| Smokers     | $26.3 \pm 4.2$          |         |
| Non-smokers | $30.1 \pm 4.9$          | $0.002$ |

Table 10 shows that ICA RI was significantly elevated in smokers ( $0.78 \pm 0.06$ ) compared to non-smokers ( $0.70 \pm 0.05$ ), further confirming increased vascular resistance ( $p < 0.001$ ).

**Table 10. Resistive index (RI) in the internal carotid artery**

| Group       | ICA RI $\pm$ SD | p-value  |
|-------------|-----------------|----------|
| Smokers     | $0.78 \pm 0.06$ |          |
| Non-smokers | $0.70 \pm 0.05$ | $<0.001$ |

Table 11 shows that bilateral plaques were present in 14% of smokers and in none of the non-smokers, reinforcing the diffuse nature of atherosclerotic involvement in smokers.

**Table 11. Distribution of bilateral plaque formation**

| Group       | Bilateral plaques present (n, %) | Absent (n, %) |
|-------------|----------------------------------|---------------|
| Smokers     | 7 (14%)                          | 43 (86%)      |
| Non-smokers | 0 (0%)                           | 50 (100%)     |

Table 12 shows a consolidated comparison of all major Doppler parameters, reaffirming statistically significant differences in IMT, PSV, EDV, and RI between smokers and non-smokers across both carotid territories.

**Table 12. Summary of all hemodynamic parameters between groups**

| Parameter      | Smokers (mean $\pm$ SD) | Non-smokers (mean $\pm$ SD) | p-value |
|----------------|-------------------------|-----------------------------|---------|
| IMT (mm)       | 0.82 $\pm$ 0.09         | 0.66 $\pm$ 0.07             | <0.001  |
| CCA PSV (cm/s) | 96.3 $\pm$ 12.8         | 81.7 $\pm$ 10.4             | <0.001  |
| CCA EDV (cm/s) | 24.8 $\pm$ 3.9          | 28.2 $\pm$ 4.2              | <0.001  |
| CCA RI         | 0.74 $\pm$ 0.04         | 0.66 $\pm$ 0.03             | <0.001  |
| ICA PSV (cm/s) | 109.4 $\pm$ 15.1        | 92.6 $\pm$ 11.8             | <0.001  |
| ICA EDV (cm/s) | 26.3 $\pm$ 4.2          | 30.1 $\pm$ 4.9              | 0.002   |
| ICA RI         | 0.78 $\pm$ 0.06         | 0.70 $\pm$ 0.05             | <0.001  |

**Table 1** shows that both groups had comparable age distribution. **Table 2** confirms no significant difference in mean age between smokers and non-smokers. **Table 3** shows a significantly higher IMT in smokers, indicating early atherosclerotic changes. **Table 4** shows that plaque formation was markedly more prevalent among smokers. **Table 5** shows elevated peak systolic velocity in the CCA among smokers. **Table 6** shows that smokers had lower end-diastolic velocity in the CCA, indicating reduced compliance. **Table 7** shows a significantly higher resistive index in the CCA of smokers. **Table 8** shows elevated ICA peak systolic velocity in smokers, while **Table 9** shows lower ICA end-diastolic velocity. **Table 10** confirms that ICA resistive index was also significantly higher in smokers. **Table 11** shows that bilateral plaque formation was seen only in smokers. **Table 12** provides a consolidated summary of all Doppler parameters, emphasizing the consistent and significant vascular differences between smokers and non-smokers.

## DISCUSSION

This study aimed to evaluate and compare the morphologic and hemodynamic characteristics of the carotid arteries in smokers and non-smokers using Doppler ultrasonography. The results clearly demonstrate that chronic smoking is associated with significant vascular alterations, both structurally and functionally, detectable through non-invasive carotid imaging<sup>9</sup>. These findings underscore the subclinical progression of atherosclerosis in smokers and highlight the diagnostic potential of Doppler USG in early risk assessment. A key structural finding in this study was the significant increase in carotid intima-media thickness (IMT) among smokers compared to non-smokers. IMT is a well-established surrogate marker for early atherosclerosis and vascular remodeling<sup>10</sup>. The observed thickening of the intima-media layer in smokers reflects endothelial dysfunction, smooth muscle proliferation, and lipid deposition occurring at the arterial wall level. This process appears to be accelerated in chronic smokers due to continuous exposure to nicotine, carbon monoxide, and oxidative agents that collectively impair vascular elasticity and promote inflammation<sup>11</sup>.

In addition to IMT changes, plaque formation was markedly more prevalent in smokers, with one-third demonstrating atherosclerotic plaques in the carotid arteries. The plaques were predominantly located near the bifurcation and extended into the proximal internal carotid artery. Notably, bilateral plaque formation was present in 14% of smokers and absent in all non-smokers, indicating a more diffuse and symmetrical pattern of vascular involvement<sup>12</sup>. The presence of plaques, even in asymptomatic individuals, is clinically relevant as it represents an advanced stage of atherosclerotic involvement and increases the risk for cerebrovascular events. Hemodynamically, the study showed consistent alterations in blood flow parameters among smokers. Peak systolic velocity (PSV) in both the common carotid artery (CCA) and internal carotid artery (ICA) was significantly higher in smokers, suggesting increased flow acceleration likely due to early luminal narrowing or wall rigidity<sup>13</sup>. Concurrently, end-diastolic velocity (EDV) values were significantly lower in smokers, reflecting reduced vascular compliance and altered flow dynamics. The resistive index (RI), a parameter reflecting peripheral resistance, was also significantly elevated in smokers for both CCA and ICA. The increase in RI indicates heightened downstream resistance, which could be secondary to vascular wall stiffness, endothelial dysfunction, or early plaque burden<sup>14</sup>.

These findings collectively support the view that smoking induces both morphologic and functional vascular changes, even before clinical symptoms of vascular insufficiency become apparent. The combination of increased IMT, elevated RI, and presence of plaques represents a constellation of Doppler features indicative of early vascular aging and subclinical atherosclerosis. Importantly, these changes were detectable in smokers without overt comorbidities, reinforcing the silent but progressive nature of tobacco-induced vascular damage<sup>15</sup>. Doppler ultrasonography, by providing real-time visualization of arterial structure and flow, proves to be a valuable tool in identifying such early changes. Its non-invasive nature, wide availability, and cost-effectiveness make it suitable for routine vascular screening in high-risk populations, including chronic smokers. Incorporating Doppler USG into preventive health check-ups can facilitate timely lifestyle interventions and targeted pharmacologic therapy to halt or reverse the progression of vascular disease<sup>16</sup>.

Despite the strengths of this study, certain limitations must be acknowledged. First, the study was limited to adult male participants aged 30–60 years, which may restrict the generalizability of findings to female or older populations. Second, the smoking group was defined based on history ( $\geq 10$  pack-years), without biochemical verification, which could introduce recall bias. Third, the study excluded individuals with known comorbidities to isolate the effect of smoking; however, this may underestimate the cumulative vascular impact seen in real-world scenarios where multiple risk factors coexist. Finally, the cross-sectional design limits the ability to determine causality or monitor the progression of vascular changes over time.

## CONCLUSION

This study demonstrates that chronic smoking is associated with significant morphologic and hemodynamic alterations in the carotid arteries, even in asymptomatic individuals. Increased intima-media thickness, elevated resistive indices, and a higher prevalence of carotid plaques were consistently observed in smokers compared to non-smokers, suggesting early-onset subclinical atherosclerosis. Doppler ultrasonography proved to be a reliable and sensitive modality for detecting these vascular changes, offering a non-invasive, accessible, and cost-effective approach for early screening. The findings underscore the importance of incorporating carotid Doppler evaluation in routine assessment protocols for chronic smokers, as early identification of vascular risk may allow timely implementation of preventive and therapeutic interventions aimed at reducing long-term cerebrovascular morbidity.

## DECLARATION

## Author Contributions

All authors contributed substantially to the study's conception, data acquisition, analysis, and interpretation. [Insert first author initials] coordinated the Doppler imaging and data interpretation. [Insert second author initials] performed the statistical analysis and clinical correlation. All authors reviewed and approved the final manuscript.

## Ethical Approval

The study was approved by the Institutional Ethics Committee.

## Informed Consent

Written informed consent was obtained from all participants included in the study.

## Declaration of Helsinki

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

## Availability of Research Data

The datasets generated and analyzed during the study are available from the corresponding author upon reasonable request.

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This study received no external funding or financial support from any agency or institution.

## Conflict of Interest Statement

The authors declare no conflicts of interest related to this study.

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