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Risk Factors Associated with Carotid Plaque among Patients with Chronic Obstructive Pulmonary Disease (COPD)

Priyanka Das[1], Gopal Krushna Sahu[2]*, N. Akshaya[3], Hemanta Kumar Sethy[4]

[1]Associate Professor, Department of Respiratory Medicine, MKCG Medical College & Hospital, Berhampur, Odisha, India
[2]Assistant Professor, Department of Respiratory Medicine, SLN Medical College & Hospital, Koraput, Odisha, India
[3]Senior Resident, Department of Critical Care, Vijaya Hospital, Chennai, Tamil Nadu, India
[4]Professor, Department of Respiratory Medicine, MKCG Medical College & Hospital, Berhampur, Odisha, India

***Address for Correspondence:** Dr. Gopal Krushna Sahu, Assistant Professor, Department of Respiratory Medicine, SLN Medical College & Hospital, Koraput, India
E-mail: drgopalkrushnas@gmail.com

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is associated with persistent systemic inflammation and an increased risk of cardiovascular disease. Carotid atherosclerotic plaque is a reliable marker of subclinical atherosclerosis and an independent predictor of future cardiovascular events. However, data regarding the risk factors associated with carotid plaque among Indian patients with COPD remain limited. This study aimed to evaluate the clinical and biochemical factors associated with carotid plaque in patients with stable COPD.

Methods: A hospital-based cross-sectional study included 103 clinically stable COPD patients between June 2022 and June 2024. Clinical characteristics, spirometry, laboratory parameters, and bilateral carotid ultrasonography were evaluated. Patients were classified according to the presence or absence of carotid plaque, and data were analyzed using appropriate statistical tests (p<0.05).

Results: Carotid plaque was detected in 34 (33.01%) patients. Clinical characteristics, including age, sex, smoking history, GOLD stage, lung function, oxygen saturation, and symptom scores, did not differ significantly between groups (p>0.05). Serum total cholesterol was significantly higher in plaque-positive patients (134.32±33.00 vs. 123.81±15.70 mg/dL; p=0.03), while CRP and random blood sugar showed borderline associations. Right and left CIMT demonstrated excellent diagnostic accuracy for carotid plaque detection (AUC: 0.988 and 0.987, respectively).

Conclusion: Approximately one-third of patients with stable COPD had carotid plaque. Elevated serum total cholesterol was significantly associated with plaque formation, whereas systemic inflammation may also contribute to atherosclerotic risk. Carotid ultrasonography may serve as a valuable non-invasive tool for cardiovascular risk assessment in COPD patients.

Key-words: Chronic obstructive pulmonary disease; Carotid plaque; Carotid ultrasonography; Atherosclerosis; Cardiovascular risk; Systemic inflammation; Dyslipidemia

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable chronic respiratory disease. It is characterized by persistent respiratory symptoms and irreversible airflow limitation caused by airway and alveolar abnormalities that develop following prolonged exposure to noxious particles or gases [1]. Although cigarette smoking remains the leading risk factor worldwide, biomass fuel exposure, occupational pollutants, environmental pollution, and genetic susceptibility also contribute significantly to disease development, particularly in low- and middle-income countries [2]. COPD is currently one of the leading causes of morbidity and mortality globally, imposing a substantial healthcare burden through recurrent hospitalizations, disability, and premature mortality [3]. Beyond pulmonary impairment, COPD is increasingly recognized as a systemic inflammatory disorder

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associated with several extrapulmonary manifestations, including cardiovascular disease, metabolic syndrome, osteoporosis, and skeletal muscle dysfunction [4]. Cardiovascular disease is among the most important comorbidities in COPD and accounts for a considerable proportion of deaths in these patients. Epidemiological studies have consistently demonstrated that patients with COPD have a significantly greater risk of myocardial infarction, stroke, and peripheral arterial disease than individuals without COPD, even after adjustment for traditional cardiovascular risk factors [5].

The association between COPD and cardiovascular disease is mediated through multiple interconnected mechanisms. Persistent systemic inflammation, oxidative stress, endothelial dysfunction, chronic hypoxemia, increased arterial stiffness, and abnormal lipid metabolism collectively promote accelerated atherosclerosis [6]. Elevated inflammatory biomarkers such as C - reactive protein, interleukin-6, tumor necrosis factor-α and fibrinogen have been associated with both disease severity and cardiovascular complications, suggesting that chronic inflammation plays a central role in vascular injury among COPD patients [7].

Carotid atherosclerosis serves as an important marker of systemic vascular disease and can be assessed non-invasively using carotid ultrasonography. Among the ultrasonographic parameters, carotid plaque is considered a more reliable predictor of future cardiovascular events than carotid intima-media thickness (CIMT), as it reflects established focal atherosclerotic disease [8]. The presence of carotid plaque has been independently associated with an increased risk of ischemic stroke, myocardial infarction, and cardiovascular mortality, making it an important tool for cardiovascular risk stratification [9].

Several studies have reported a higher prevalence of carotid plaque among patients with COPD compared with healthy individuals. While cigarette smoking is a shared risk factor for both COPD and atherosclerosis, evidence suggests that COPD-specific factors such as chronic systemic inflammation, oxidative stress, recurrent exacerbations, airflow limitation, and chronic hypoxemia further accelerate plaque formation independent of smoking exposure [10]. In addition, increasing disease severity has been associated with worsening endothelial dysfunction and greater vascular involvement.

Clinical and biochemical characteristics may further influence the development of carotid plaque in COPD. Advancing age, male sex, cumulative smoking exposure, hypertension, diabetes mellitus, dyslipidemia, obesity, systemic inflammation, and disease severity have all been implicated as potential determinants of carotid atherosclerosis [11]. Biomarkers reflecting tissue injury and inflammation, including serum lactate dehydrogenase (LDH), together with abnormalities in lipid profile, may also contribute to endothelial damage and plaque progression [12]. However, the relative contribution of these factors remains incompletely understood, particularly among Indian patients.

Despite the growing recognition of cardiovascular complications in COPD, relatively few studies have specifically evaluated the determinants of carotid plaque. Most available research has focused on carotid intima-media thickness rather than plaque, although plaque has greater prognostic significance for future cardiovascular events [13]. Furthermore, data from the Indian population remain limited despite the high burden of COPD and cardiovascular disease.

Early identification of patients at increased cardiovascular risk is essential for implementing preventive interventions and reducing long-term morbidity and mortality. Carotid ultrasonography is a simple, safe, reproducible, and cost-effective imaging modality that facilitates the detection of subclinical atherosclerosis before the onset of overt cardiovascular disease. Identification of the clinical and biochemical risk factors associated with carotid plaque may help clinicians recognize high-risk COPD patients who require more intensive cardiovascular evaluation and risk factor modification.

Therefore, the present study was undertaken to evaluate the risk factors associated with carotid plaque among patients with COPD. The findings are expected to improve cardiovascular risk assessment and support the integration of carotid ultrasonography into the comprehensive evaluation of patients with COPD.

MATERIALS AND METHODS

Study Design and Setting- This hospital-based cross-sectional observational study was conducted in the Department of Respiratory Medicine, MKCG Medical College and Hospital, Berhampur, Odisha, India, between June 2022 and June 2024. The study evaluated clinically

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stable patients with COPD to determine the risk factors associated with carotid atherosclerotic plaque.

Study Population- A total of 103 patients with stable COPD were enrolled consecutively after obtaining written informed consent. COPD was diagnosed based on clinical history, physical examination, and post-bronchodilator spirometry according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) recommendations. Only clinically stable patients without an acute exacerbation during the preceding six weeks were included.

Inclusion criteria

- ✓ Patients aged ≥18 years with stable COPD.
- ✓ No history of COPD exacerbation within the previous six weeks.
- ✓ Smoking history of at least 10 pack-years and/or documented exposure to biomass fuel.
- ✓ Willingness to participate in the study with written informed consent.

Exclusion criteria- Patients with previously diagnosed hypertension or diabetes mellitus before the onset of COPD, asthma, bronchiectasis, post-tubercular obstructive airway disease, pulmonary eosinophilia, pneumonia, tuberculosis, interstitial lung disease, pneumoconiosis, malignancy, pregnancy, hepatic or renal dysfunction, previous myocardial infarction, congenital or valvular heart disease, atrial fibrillation, vasculitis, carotid artery surgery, coronary catheterization, HIV infection, or other active inflammatory or malignant disorders were excluded. Patients receiving statins or anticoagulant therapy were also excluded.

Clinical and Laboratory Assessment- Detailed demographic characteristics, smoking history, biomass fuel exposure, clinical symptoms, and comorbidities were recorded using a structured proforma. All participants underwent clinical examination, pulse oximetry, chest radiography, electrocardiography, and spirometry. COPD severity was graded according to the GOLD classification. Dyspnoea severity was assessed using the modified Medical Research Council (mMRC) scale, while symptom burden was evaluated using the COPD Assessment Test (CAT). Routine laboratory investigations included complete blood count, random blood sugar, renal and liver function tests, lipid profile, serum C-reactive protein (CRP), and lactate dehydrogenase (LDH).

Carotid Ultrasonography- All patients underwent bilateral carotid artery ultrasonography using B-mode Doppler imaging performed by an experienced radiologist following a standardized protocol. Both common carotid arteries were examined for the presence of atherosclerotic plaque. Carotid plaque was defined as a focal structure protruding into the arterial lumen, consistent with established ultrasonographic criteria. Based on ultrasonographic findings, patients were categorized into carotid plaque-positive and carotid plaque-negative groups for subsequent comparative analysis.

Outcome Measures- The primary outcome was the presence of carotid atherosclerotic plaque. Demographic variables, smoking exposure, COPD severity, symptom scores, and laboratory biomarkers including CRP, LDH, lipid profile, and blood glucose were evaluated as potential risk factors associated with carotid plaque among patients with COPD.

Statistical Analysis- Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Comparisons between patients with and without carotid plaque were performed using the independent Student's t-test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables, as appropriate. A two-tailed p-value <0.05 was considered statistically significant.

Ethical Considerations- The study protocol received approval from the Institutional Ethics Committee of MKCG Medical College and Hospital, Berhampur. Written informed consent was obtained from all study participants before enrolment, and confidentiality of patient information was maintained throughout the study.

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RESULTS

A total of 103 patients with stable COPD were included in the study. The mean age of the study population was 63.78 ± 11.79 years, with the majority of participants belonging to the 61–70 years age group (30.10%), followed by the 51–60 years age group (28.16%). Males constituted 62.14% of the study population, while females accounted for 37.86**%**. A history of smoking was present in 64 (62.14%) patients, whereas 54 (52.43%) patients reported biomass fuel exposure. Diabetes mellitus (36.89%) and hypertension (27.18%) were the most common comorbidities. Most patients had normal body mass index (56.31%), while 33.98% were underweight.

Regarding disease severity, the majority of patients belonged to GOLD stage III (52.43%)**,** followed by GOLD stage II (43.69%)**.** According to the ABE assessment**,** 59.22% of patients were classified into category E. The mean mMRC score was 2.46 ± 0.57, mean CAT score ranged predominantly between 25 and 30**,** and the mean post-bronchodilator FEV_1 was 51.89 ± 8.04%**.** The mean oxygen saturation (SpO_2) was 89.66 ± 2.40%**.** Carotid ultrasonography demonstrated carotid atherosclerotic plaque in 34 (33.01%) patients, whereas 69 (66.99%) had no evidence of plaque formation (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 103)

Characteristics	Category	n (%) / Mean ± SD
Age (years)	Mean ± SD	63.78 ± 11.79
	31–40	2 (1.94)
	41–50	12 (11.65)
	51–60	29 (28.16)
	61–70	31 (30.10)
	71–80	21 (20.39)
	81–90	7 (6.80)
	91–100	1 (0.97)
Gender	Male	64 (62.14)
	Female	39 (37.86)
Smoking Status	Current smoker	36 (34.95)
	Ex-smoker	28 (27.18)
	Never smoker	39 (37.86)
Biomass Exposure	Yes	54 (52.43)
	No	49 (47.57)
BMI	Underweight	35 (33.98)
	Normal	58 (56.31)
	Overweight	10 (9.71)
GOLD Stage	II	45 (43.69)
	III	54 (52.43)
	IV	4 (3.88)
ABE Category	B	42 (40.78)
	E	61 (59.22)
mMRC score	Mean ± SD	2.46 ± 0.57
Post-BD FEV_1 (%)	Mean ± SD	51.89 ± 8.04
SpO_2 (%)	Mean ± SD	89.66 ± 2.40
Carotid Plaque	Present	34 (33.01)
	Absent	69 (66.99)

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Among the 103 patients with COPD, carotid plaque was detected in 34 (33.01%) patients, while 69 (66.99**%)** had no evidence of plaque. The mean right and left carotid intima-media thicknesses were 0.71 ± 0.58 mm and 0.67 ± 0.54 mm, respectively. The mean serum CRP and LDH levels were 11.35 ± 8.71 mg/dL and 427.50 ± 145.82 U/L**,** respectively. The overall lipid profile demonstrated mean total cholesterol of 127.28 ± 23.27 mg/dL and LDL cholesterol of 84.17 ± 25.95 mg/dL (Table 2).

Table 2: Laboratory Profile and Carotid Ultrasonography Characteristics of Patients with COPD (n=103)

Parameter	Mean ± SD / n (%)
Total Leukocyte Count (cells/mm³)	9074.62 ± 4044.19
Total Cholesterol (mg/dL)	127.28 ± 23.27
Triglycerides (mg/dL)	137.99 ± 21.77
HDL Cholesterol (mg/dL)	53.03 ± 9.87
LDL Cholesterol (mg/dL)	84.17 ± 25.95
C-reactive Protein (mg/dL)	11.35 ± 8.71
Lactate Dehydrogenase (U/L)	427.50 ± 145.82
Blood Urea (mg/dL)	41.03 ± 7.98
Serum Creatinine (mg/dL)	1.00 ± 0.25
Random Blood Sugar (mg/dL)	104.22 ± 12.51
Right CIMT (mm)	0.71 ± 0.58
Left CIMT (mm)	0.67 ± 0.54
Carotid Plaque Present	34 (33.01%)
Carotid Plaque Absent	69 (66.99%)

Patients with carotid plaque had comparable age, sex distribution, smoking history, oxygen saturation, dyspnoea severity, lung function, GOLD stage, and ABE category compared with those without plaque. None of these clinical variables showed a statistically significant association with carotid plaque (all $p>0.05$), although CAT score demonstrated a borderline association ($p=0.07$) (Table 3.

Table 3: Comparison of Demographic and Clinical Characteristics According to Carotid Plaque Status

Variable	Plaque Present (n=34)	Plaque Absent (n=69)	p-value
Age (years)	64.62 ± 11.12	63.36 ± 12.17	0.61
Male, n (%)	22 (64.7)	42 (60.9)	0.87
Female, n (%)	12 (35.3)	27 (39.1)	0.87
Smoking history, n (%)	22 (64.7)	42 (60.9)	0.90
SpO_2 (%)	89.68 ± 2.32	89.65 ± 2.45	0.96
mMRC score	2.47 ± 0.56	2.45 ± 0.58	0.86
CAT score	27.97 ± 3.82	29.78 ± 5.00	0.07
Post-BD FEV_1 (%)	52.47 ± 7.89	51.61 ± 8.16	0.61
GOLD Stage II	18 (40.0)	27 (60.0)	0.41
GOLD Stage III	15 (27.8)	39 (72.2)	
GOLD Stage IV	1 (25.0)	3 (75.0)	
ABE Category B	16 (38.1)	26 (61.9)	0.49
ABE Category E	18 (29.5)	43 (70.5)	

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Among the biochemical parameters evaluated**,** total cholesterol was significantly higher in patients with carotid plaque than those without plaque (134.32 ± 33.00 vs. 123.81 ± 15.70 mg/dL; p=0.03). CRP and random blood sugar demonstrated borderline statistical significance (both *p*=0.06), whereas TLC, LDH, LDL cholesterol, and blood urea showed no significant association with carotid plaque (Table 4).

Table 4: Association of Biochemical Parameters with Carotid Plaque

Variable	Plaque Present (n=34)	Plaque Absent (n=69)	p-value
TLC	8447.44 ± 4619.99	9383.67 ± 3725.77	0.27
CRP (mg/dL)	13.63 ± 13.36	10.22 ± 4.81	0.06
LDH (U/L)	424.29 ± 149.60	429.09 ± 145.01	0.88
Total Cholesterol (mg/dL)	134.32 ± 33.00	123.81 ± 15.70	0.03*****
LDL (mg/dL)	87.88 ± 28.73	82.35 ± 24.48	0.31
Random Blood Sugar (mg/dL)	107.50 ± 11.46	102.61 ± 12.77	0.06
Blood Urea (mg/dL)	41.68 ± 9.21	40.71 ± 7.35	0.57

**Statistically significant at p<0.05.*

Receiver operating characteristic analysis demonstrated excellent diagnostic performance of carotid intima-media thickness for identifying carotid plaque. A right CIMT cut-off value of >0.89 mm yielded an AUC of 0.988**,** with 97.06% sensitivity and 100% specificity. Similarly, a left CIMT cut-off value of >0.81 mm showed an AUC of 0.987**,** with 97.06% sensitivity and 98.55% specificity**,** indicating excellent discrimination for detecting carotid plaque (Table 5).

Table 5: Diagnostic Performance of Carotid Intima-Media Thickness for Detection of Carotid Plaque

Parameter	Right CIMT	Left CIMT
Cut-off value (mm)	>0.89	>0.81
Area under ROC curve	0.988	0.987
Sensitivity (%)	97.06	97.06
Specificity (%)	100.00	98.55
Positive Predictive Value (%)	100.00	97.06
Negative Predictive Value (%)	98.57	98.55
Positive Likelihood Ratio	∞ (100% specificity)	66.97
Negative Likelihood Ratio	0.02	0.03

DISCUSSION

Carotid atherosclerotic plaque was identified in 33.01% of patients with stable COPD, indicating a considerable burden of subclinical atherosclerosis in this population. Although demographic characteristics, smoking history, pulmonary function, and disease severity were not significantly associated with plaque formation, higher serum total cholesterol showed a significant association, while CRP and random blood sugar demonstrated borderline significance. In addition, CIMT exhibited excellent diagnostic performance for plaque detection. Collectively, these findings suggest that cardiovascular risk in COPD is influenced by multiple interacting mechanisms beyond the severity of pulmonary disease alone [14,15].

The prevalence of carotid plaque observed in this study is comparable to previous reports demonstrating increased carotid atherosclerosis among patients with COPD compared with healthy individuals. Persistent systemic inflammation, oxidative stress, endothelial dysfunction, recurrent hypoxaemia, and accelerated vascular ageing are considered the major mechanisms linking COPD with atherosclerotic plaque formation. Furthermore, chronic exposure to cigarette smoke and environmental pollutants may further aggravate endothelial injury by promoting inflammatory cell infiltration and lipid oxidation [15,16].

Although advancing age and male sex are established cardiovascular risk factors, neither showed a significant association with carotid plaque in the present analysis.

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Similar observations have been reported in previous studies, where disease-related inflammatory processes appeared to exert a greater influence on plaque formation than demographic characteristics alone. These findings suggest that chronic systemic inflammation and metabolic abnormalities associated with COPD may contribute more substantially to vascular injury than age or sex after the disease has become established [17].

Cigarette smoking is the strongest common risk factor for both COPD and atherosclerosis; however, smoking history was not significantly associated with carotid plaque in this study. Earlier investigations have reported comparable findings, indicating that persistent systemic inflammation and endothelial dysfunction may continue to accelerate atherosclerosis even after smoking cessation. Therefore, vascular risk in COPD is likely determined by the combined effects of prior smoking exposure and ongoing disease-related inflammatory mechanisms rather than smoking alone [18,19].

No significant association was observed between carotid plaque and measures of COPD severity, including GOLD stage, post-bronchodilator FEV_1, mMRC dyspnoea score, oxygen saturation, or ABE category. Although some studies have reported greater carotid atherosclerosis with worsening airflow limitation, others have produced findings similar to those observed in the present study, suggesting that systemic cardiovascular risk factors may have a greater influence on plaque formation than the degree of airflow obstruction itself [20,21]. The borderline association observed with CAT score also indicates that symptom burden alone may not reliably reflect underlying vascular risk.

Among the biochemical variables evaluated, serum total cholesterol emerged as the only parameter significantly associated with carotid plaque. Patients with plaque had significantly higher cholesterol levels, supporting the established role of dyslipidaemia in the development of atherosclerosis. Elevated cholesterol promotes endothelial dysfunction, macrophage activation, foam cell formation, and progressive plaque development, thereby increasing cardiovascular risk. These findings are consistent with previous studies demonstrating the importance of lipid abnormalities in chronic inflammatory disorders, including COPD [22,23].

Higher CRP concentrations were observed among patients with carotid plaque, although the difference did not reach statistical significance. As a well-established marker of systemic inflammation, CRP has been linked to endothelial dysfunction, arterial stiffness, and future cardiovascular events. The borderline association identified in this study supports the concept that persistent low-grade inflammation contributes to atherosclerotic progression in COPD. The absence of statistical significance may be explained by the relatively small sample size and inter-individual variability in inflammatory marker levels [24,25].

Random blood sugar also demonstrated a borderline relationship with carotid plaque, whereas LDH, total leukocyte count, LDL cholesterol, and blood urea showed no significant differences between the two groups. Previous evidence suggests that impaired glucose metabolism and insulin resistance may accelerate endothelial dysfunction and vascular inflammation. The lack of statistically significant associations for several biomarkers in this study may reflect its cross-sectional design, limited sample size, and the exclusion of patients with established cardiovascular disease or severe metabolic disorders [26].

Receiver operating characteristic analysis demonstrated excellent diagnostic performance of carotid intima-media thickness for identifying carotid plaque. Both right and left CIMT achieved area under the curve values approaching 1.0, with high sensitivity and specificity. These findings support previous evidence that carotid ultrasonography is a reliable, non-invasive, and cost-effective method for detecting subclinical atherosclerosis in patients with COPD. Early identification of vascular abnormalities may facilitate cardiovascular risk stratification and timely preventive interventions before the occurrence of major adverse cardiovascular events [27,28].

Several limitations should be considered while interpreting these findings. The study was conducted at a single tertiary care centre with a relatively modest sample size, which may limit the generalizability of the results. Its cross-sectional design does not permit causal inference between the identified risk factors and carotid plaque development, and longitudinal cardiovascular outcomes were not assessed. Nevertheless, the study provides important evidence regarding carotid plaque prevalence and its associated clinical and biochemical factors among Indian patients with stable COPD.

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CONCLUSIONS

The present study demonstrated that carotid atherosclerotic plaque was present in nearly one-third of patients with stable COPD, indicating a substantial burden of subclinical cardiovascular disease. Among the evaluated variables, elevated serum total cholesterol showed a significant association with carotid plaque, while higher CRP and random blood sugar levels demonstrated borderline associations, suggesting a potential contribution of systemic inflammation and metabolic abnormalities to plaque development. In contrast, demographic characteristics, smoking history, disease severity, lung function, and symptom scores were not significantly associated with plaque formation. Carotid intima-media thickness exhibited excellent diagnostic accuracy for detecting carotid plaque, supporting its utility as a non-invasive screening tool for identifying subclinical atherosclerosis in COPD patients. Early cardiovascular risk assessment, including carotid ultrasonography in selected high-risk individuals, may facilitate timely intervention and reduce future cardiovascular morbidity. Further large-scale prospective multicentre studies are required to validate these findings and establish robust predictors of carotid plaque in patients with COPD.

CONTRIBUTION OF AUTHORS

Research concept- N. Akshaya, Hemanta Kumar Sethy
Research design- N. Akshaya, Priyanka Das
Supervision- Priyanka Das, Gopal Krushna Sahu, Hemanta Kumar Sethy
Materials- Gopal Krushna Sahu, N. Akshaya
Data collection- N. Akshaya, Gopal Krushna Sahu
Data analysis and interpretation- N. Akshaya, Priyanka Das, Dr.Gopal Krushna Sahu
Literature search- N. Akshaya, Priyanka Das
Writing article- N. Akshaya
Critical review- Hemanta Kumar Sethy, Priyanka Das, Gopal Krushna Sahu
Article editing- N.Akshaya, Gopal Krushna Sahu, Priyanka Das
Final approval- Priyanka Das, Gopal Krushna Sahu, N. Akshaya

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