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Radiological evaluation of the impact of chronic obstructive pulmonary disease on ascending aortic diameter in hypertensive patients

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ABSTRACT

Aims: This study aims to radiologically evaluate the impact of chronic obstructive pulmonary disease (COPD) on the diameter of the AsAo in individuals diagnosed with hypertension and to assess potential structural effects of COPD on the cardiovascular system.

Methods: This retrospective cross-sectional study was conducted at Yozgat Bozok University Research and Training Hospital. Clinical and radiological data of 60 hypertensive individuals who underwent thoracic computed tomography (CT) between January 1, 2023, and June 1, 2025, were analyzed. Participants were divided into two groups based on the presence or absence of a COPD diagnosis. AsAo and aortic arch (Arcus Ao) diameters were measured from CT images. Clinical variables, comorbidities, and lipid profiles were statistically evaluated.

Results: The mean age of participants was significantly higher in the COPD group (73.8±7.92 years) compared to the control group (67.93±12.59 years) (p=0.036). The mean AsAo diameter was 40.40±4.90 mm in the COPD group and 36.01±3.79 mm in the control group, revealing a statistically significant difference (p=0.00028). No significant difference was found between the groups regarding Arcus Ao diameter (p=0.103). Among lipid parameters, there were no significant differences in HDL, LDL, or total cholesterol levels; however, triglyceride levels showed a trend toward significance (p=0.069).

Conclusion: The findings suggest that COPD is not merely a respiratory condition but also a systemic disease with notable vascular implications. The significant increase in AsAo diameter in the COPD group may be attributed to chronic inflammation, endothelial dysfunction, and degeneration of elastic fibers associated with the disease. The absence of a significant difference in Arcus Ao diameter may be related to the anatomically stable structure of this segment. The higher comorbidity burden and older age observed in COPD patients further support the systemic nature of the disease. Therefore, COPD should be regarded as a potential vascular risk modifier to be considered in hypertensive individuals.

Keywords: Ascending aortic diameter, chronic obstructive pulmonary disease (COPD), hypertension

INTRODUCTION

Hypertension (HT) is recognized as one of the leading causes of cardiovascular morbidity and mortality worldwide. According to the 2018 guidelines jointly issued by the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH), HT is defined as a systolic blood pressure ≥140 mmHg and/or a diastolic blood pressure ≥90 mmHg.[1] Over time, HT may lead to degeneration of elastic fibers and accumulation of collagen in the aortic wall, resulting in structural changes—particularly in the ascending aorta—which can ultimately cause aortic dilatation. This pathological process increases the risk of life-threatening complications such as aortic dissection and rupture.[2]

A meta-analysis has reported the prevalence of proximal aortic dilatation in hypertensive individuals to range between 6.1% and 19%.[2] Furthermore, a Mendelian randomization study revealed that genetically predicted increases in systolic and diastolic blood pressure were significantly associated

with greater ascending thoracic aorta diameter.[3] This finding highlights the pronounced effect of HT on aortic morphology. A 2025 position paper also emphasized that HT may lead to remodeling of the proximal aortic wall, which is closely linked to clinically significant outcomes such as dissection and aneurysm formation.[4]

Chronic obstructive pulmonary disease (COPD), particularly in individuals with emphysematous phenotype, can also contribute to structural changes in the aortic wall. This is primarily due to systemic inflammation, oxidative stress, and increased proteolytic enzyme activity, which together result in elastic fiber loss and vascular remodeling.[5] In the large-scale, multi-center MESA (Multi-Ethnic Study of Atherosclerosis) study conducted in 2021, individuals with COPD were shown to have significantly larger ascending aortic diameters compared to those without the disease.[5]

The coexistence of COPD and HT is commonly observed in clinical practice. A large cohort study covering the years 1999 to 2018 reported a HT prevalence of 58.5% among individuals with COPD, with a particularly higher rate among adults under 60 years of age and heavy smokers.[6] In addition, frequently observed features in COPD—such as arterial stiffness, endothelial dysfunction, and increased pulmonary vascular resistance—may contribute to a compounded cardiovascular burden through parallel dysfunctions in both systemic and pulmonary vascular systems.[8]

The coexistence of HT and COPD may serve as a dual risk factor for ascending aortic dilatation. In a retrospective study on individuals with COPD, the prevalence of aortic aneurysm was reported as 14.3%, and 56% of those affected also had HT.[7] Nevertheless, studies specifically focusing on the impact of COPD on ascending aortic diameter in hypertensive individuals remain limited. Given the high likelihood of coexistence between COPD and cardiovascular disease—and the potential for more severe clinical outcomes—exploring this relationship in greater detail is essential for effective cardiovascular risk management.[9]

Recent radiological and prospective studies have demonstrated that COPD can lead to systemic vascular effects beyond the pulmonary system, such as increased aortic inflammation and arterial sclerosis.[10] Moreover, systemic inflammation, hyperinflation, and intrathoracic pressure changes associated with COPD may increase wall stress in large vessels, including the ascending aorta, thereby promoting dilatation.[5] Population-based imaging studies have further shown that advanced age, male sex, smoking, dyslipidemia, and obesity are significantly associated with age-related vascular remodeling processes and with ascending aortic enlargement.[11,12]

In this context, radiological evaluation of the effect of COPD on ascending aortic diameter in individuals diagnosed with HT may enhance our understanding of the combined impact of HT and respiratory disease on aortic morphology. Furthermore, it may contribute to the development of more personalized and targeted cardiovascular risk stratification strategies by incorporating individual risk factors.[13,14]

METHODS

Ethics

The study was conducted with the approval of the Non-interventional Clinical Researches Ethics Committee of Yozgat Bozok University Faculty of Medicine (Date: 02.07.2025, Decision No: 2025-GOKAEK-2513_2025.07.02_540). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. As a retrospective study, no diagnostic or therapeutic intervention was made in the course of patient care, and all data were obtained from existing clinical and radiological records.

Study Design and Participants

This study aimed to radiologically evaluate the impact of COPD on ascending aortic diameter in hypertensive individuals who presented to the Radiology, Pulmonology, and Cardiology departments of Yozgat Bozok University Research and Training Hospital. The study was based on clinical and radiological evaluations performed between January 1, 2023, and June 1, 2025.

The study sample consisted of individuals diagnosed with HT who had undergone thoracic CT. Participants were categorized into two groups based on the presence or absence of a clinical diagnosis of COPD. Only individuals aged 18 years and older were included. Patients with active infections, malignancies, systemic connective tissue diseases, a history of immunosuppressive therapy, or current use of pharmacological agents that could affect immune function were excluded.

Imaging Protocol

Measurements of the AsAo diameter were retrospectively evaluated using thoracic CT images obtained for clinical indications. CT scans were performed with a Philips Brilliance 64 Slice CT scanner using a thoracic protocol with 1-mm slice thickness, 120–80 kV, and 180 mAs dose modulation, and were acquired without contrast. All scans were performed with ECG gating. Individuals with available imaging were included in the study. Measurements were performed using standard techniques at the level of the pulmonary artery bifurcation, with the maximum anteroposterior diameter on the axial plane taken as the reference. All measurements were independently conducted by a cardiovascular surgeon and a radiologist, both with clinical and radiological experience, and no significant interobserver variability was observed between the measurements.

Clinical and Demographic Data

Data were retrieved digitally through the hospital information management system (HIMS). Systematic records were collected for all participants, including age, sex, smoking status, and cardiovascular risk factors such as HT, diabetes mellitus (DM), dyslipidemia, obesity, and family history. In addition to clinical data, laboratory parameters were reviewed; however, the primary focus of the study was to examine the relationship between radiological measurements and accompanying clinical variables.

Statistical Analysis

The data analyses were performed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean±standard deviation (SD), and categorical variables as numbers and percentages (%). The distribution of variables was assessed using the Kolmogorov-Smirnov test. Independent samples t-test was used to compare normally distributed groups, while the Mann-Whitney U test was applied for non-normally distributed data. Categorical variables were analyzed using the Chi-square test. Pearson or Spearman correlation tests were employed to assess the relationship between the presence of COPD, ascending aortic diameter, and cardiometabolic risk factors. A p-value <0.05 was considered statistically significant.

RESULTS

Of the 60 individuals included in the study, 51.7% (n=31) were male and 48.3% (n=29) were female, indicating a relatively balanced gender distribution. No statistically significant difference was observed between the groups in terms of gender (p=1.000). The participants' ages ranged from 38 to 92 years. The mean age in the COPD group was 73.8±7.92 years, whereas it was 67.93±12.59 years in the non-COPD group. The independent samples t-test revealed a statistically significant age difference between the groups (p=0.036, p<0.05).

When evaluating comorbidities among participants, DM was the most commonly observed condition. DM was present in 43.3% of the cohort, while heart failure (HF) was identified in 13.3% of individuals.

Lipid parameters were compared between individuals with and without COPD. The mean HDL cholesterol level was 45.96±11.55 mg/dl in the COPD group and 42.31±11.67 mg/dl in the control group. LDL levels were 97.76±32.45 mg/dl and 110.09±40.12 mg/dl, respectively. The mean triglyceride levels were 128.83±60.03 mg/dl in the COPD group and 159.63±68.50 mg/dl in the non-COPD group. Total cholesterol was measured at 167.65±37.24 mg/dl and 184.35±49.42 mg/dl, respectively. According to independent samples t-tests, there were no statistically significant differences in HDL (p=0.229), LDL (p=0.195), triglyceride (p=0.069), or total cholesterol (p=0.145) levels between the groups (p>0.05) (**Table 1**).

Table 1. Comparison of lipid profiles

Parameter	COPD (+) mean±SD	COPD (–) Mean±SD	p-value
HDL (mg/dl)	45.96±11.55	42.31±11.67	0.229
LDL (mg/dl)	97.76±32.45	110.09±40.12	0.195
Triglycerides (mg/dl)	128.83±60.03	159.63±68.50	0.069
Total cholesterol (mg/dl)	167.65±37.24	184.35±49.42	0.145

HDL: High-density lipoprotein, LDL: Low-density lipoprotein, SD: Standard deviation, COPD: Chronic obstructive pulmonary disease

The AsAo diameter was compared between individuals with and without a diagnosis of COPD. In the COPD group, the mean AsAo diameter was measured as 40.40±4.90 mm, whereas in the non-COPD group it was 36.01±3.79 mm. This difference was evaluated using the independent Samples t-test and was found to be statistically significant (p=0.00028) (**Figure, Table 2**).

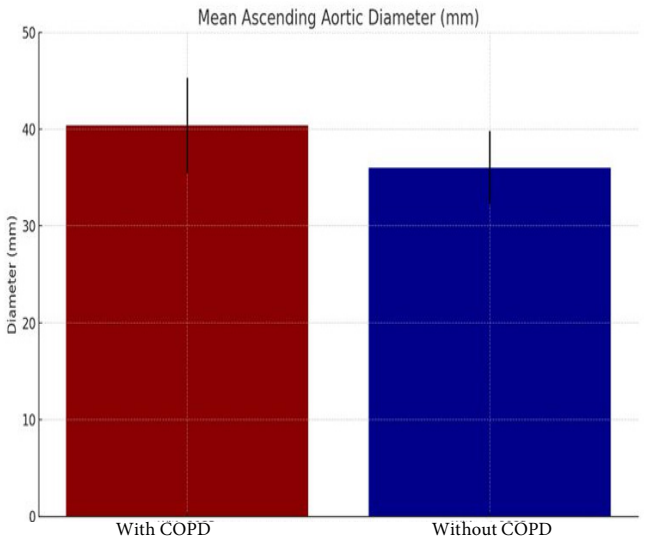

Figure. Comparison of ascending aortic diameter based on the presence of COPD
COPD: Chronic obstructive pulmonary disease

Table 2. Comparison of ascending aortic diameter (AsAo)

Group	AsAo diameter (mm)±SD	p-value
COPD present	40.40±4.90	0.00028
COPD absent	36.01±3.79	0.00028

COPD: Chronic obstructive pulmonary disease, AsAo: Ascending aorta, SD: Standard deviation

In this study, the diameter of the Arcus Ao was compared between individuals with and without a diagnosis of COPD. The mean Arcus Ao diameter in the COPD group was calculated as 32.32±3.48 mm, whereas in the non-COPD group it was 30.89±3.20 mm. The statistical significance of this difference was evaluated using an independent Samples t-test, yielding a p-value of 0.103 (**Table 3**).

Table 3. Comparison of AsAo and aortic arch diameters by COPD group

Aortic segment	COPD present (mm)±SD	COPD absent (mm)±SD	p-value	Statistical significance
AsAo	40.40±4.90	36.01±3.79	0.00028	Significant (p<0.05)
Aortic arch (Arcus Ao)	32.32±3.48	30.89±3.20	0.103	Not significant (p>0.05)

COPD: Chronic obstructive pulmonary disease, AsAo: Ascending aorta, Arcus Ao: Aortic arch, SD: Standard deviation

DISCUSSION

Among the 60 individuals included in this study, 51.7% (n=31) were male and 48.3% (n=29) were female. No statistically significant difference was found in gender distribution between those with and without COPD (p=1.000), indicating that gender was not a significant determinant of COPD status in this sample. However, previous literature widely recognizes male sex as a risk factor for the development of COPD.[14] Furthermore, studies have shown that ascending aortic diameters are significantly greater in men than in women.[12] In our study, due to the relatively balanced gender distribution, the effect of gender on outcomes was not prominently observed.[13]

Participants' ages ranged from 38 to 92 years. The mean age was 73.8±7.92 years in the hypertensive group with COPD and 67.93±12.59 years in the group without COPD. The age

difference between the groups was statistically significant (p=0.036), suggesting that individuals diagnosed with COPD were older, and that age may play a key role in the development of COPD.[6] Piccari et al.[8] reported that age is not only associated with COPD but also with cardiovascular complications. Similarly, Guo et al.[13] highlighted a significant relationship between age and AsAo diameter, underscoring age-related vascular changes.

In the assessment of comorbidities among hypertensive individuals, DM was the most frequently reported condition, with a notable proportion of COPD patients also having DM. Additional comorbidities included congestive HF, asthma, coronary artery disease, hypothyroidism, benign prostatic hyperplasia, and chronic kidney disease. The higher number of comorbidities observed in the COPD group likely reflects the systemic inflammatory burden of COPD, which can affect multiple organ systems. These findings are in line with those of Ando et al.,[7] who reported frequent co-occurrence of aortic aneurysm, HT, and HF in COPD patients. Polman et al.[9] also described a strong association between COPD and cardiometabolic disorders. Piccari et al.[8] further emphasized that vascular dysfunction in COPD may contribute to the overall comorbidity burden.

Regarding lipid profiles, no statistically significant differences were observed between groups in HDL, LDL, or triglyceride levels. In the COPD group, mean HDL was 45.96±11.55 mg/dl, LDL was 97.76±32.45 mg/dl, and triglycerides were 128.83±60.03 mg/dl. In the non-COPD group, these values were 42.31±11.67 mg/dl, 110.09±40.12 mg/dl, and 159.63±68.50 mg/dl, respectively. A borderline significance was observed for triglyceride levels (p=0.069), which may indicate a clinical tendency toward dyslipidemia in COPD. Polman et al.[9] pointed to the high prevalence of metabolic syndrome among individuals with COPD. Fisk et al.[10] proposed that vascular inflammation and atherosclerotic mechanisms contribute to dyslipidemia in COPD. Labaki et al.[24] also demonstrated that CT imaging can help identify dyslipidemia and other chronic diseases in COPD. Nonetheless, some studies have reported inconclusive effects of COPD on lipid profiles.[25]

When examining the AsAo, the diameter was significantly greater in individuals with COPD (40.40±4.90 mm) compared to those without COPD (36.01±3.79 mm) (p=0.00028). This suggests that COPD may contribute to ascending aortic dilatation. Known mechanisms in COPD, such as chronic hypoxemia, oxidative stress, endothelial dysfunction, and elastic fiber degeneration, may promote vascular remodeling.[5,8,14] Fujikura et al.[5] observed that thoracic aortic diameters are notably larger in individuals with COPD, potentially due to emphysematous lung changes. Xiong et al.[15] reported similar findings for abdominal aortic enlargement in COPD. Ng et al.[16] proposed that the pulmonary artery-to-aorta ratio may serve as an indicator of vascular alterations in COPD.

Regarding the Arcus Ao, the average diameter was 32.32±3.48 mm in the COPD group and 30.89±3.20 mm in the non-COPD group. However, the difference was not statistically significant (p=0.103). This suggests that the Arcus Ao may be less affected by COPD-related changes. Anatomically, the aortic arch is more stable and may carry less hemodynamic burden compared to the AsAo.[12,14,17] Hatemi et al.[14] found generalized aortic dilatation in individuals with COPD, but the changes were most prominent in the ascending segment. Similarly, Mensel et al.[12] noted that Arcus Ao diameter is relatively stable despite age and other risk factors.

The overall findings of this study support the concept that COPD is not only a pulmonary disease but also a systemic and cardiovascular condition. In particular, the observed increase in AsAo diameter may reflect the vascular burden imposed by COPD. Additionally, studies have shown that the pulmonary artery-to-aorta ratio is associated with right heart function, underscoring the value of vascular imaging in this population.[19,23] Labaki[24] and Rueda-Ochoa et al.[25] have clearly demonstrated associations between thoracic aortic diameter and both cardiovascular events and mortality.

Limitations

This study has several limitations. First, its retrospective design and relatively small sample size limit the generalizability of the results. In addition, the absence of longitudinal follow-up data and the inability to account for potential confounding factors such as smoking pack-years and medication use should be considered when interpreting the findings. Future prospective studies with larger cohorts are warranted to validate these results and to determine whether COPD should be incorporated into cardiovascular risk stratification models for hypertensive patients. In conclusion, the coexistence of COPD and HT should be carefully evaluated in terms of their effects on cardiovascular structures, and multidisciplinary follow-up strategies should be developed for these individuals.

CONCLUSION

In hypertensive individuals, the presence of COPD appears to negatively affect cardiovascular structures, particularly through the significant enlargement observed in the ascending aorta. This finding underscores that COPD is not solely a respiratory disease, but rather a systemic condition with notable vascular implications. The absence of a significant difference Arcus Ao diameter may be attributed to the anatomically more stable nature of this segment. Continued radiological monitoring and a multidisciplinary approach are essential in individuals with COPD for the early detection of potential cardiovascular complications and for improving clinical outcomes.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the approval of the Non-interventional Clinical Researches Ethics Committee of Yozgat Bozok University Faculty of Medicine (Date: 02.07.2025, Decision No: 2025-GOKAEK-2513_2025.07.02_540).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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