

Normative and Comparative CT-Derived Airway Indices in Asthmatics and Smokers in Rivers State, Nigeria: An Ambispective Study

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ABSTRACT

Background: Quantitative computed tomography (QCT) provides objective morphometric measures of the airway and has increasingly been investigated as a non-invasive means of assessing and characterizing airway remodelling. However, locality-specific normative data are scarce in Nigeria. This study therefore established CT-derived airway indices among participants with normal airway findings and compared them with corresponding measurements in asthmatics and smoking adults in Rivers State, Nigeria. **Methods:** An ambispective cross-sectional study was carried out among 110 adults aged 18–75 years who underwent chest CT at a diagnostic centre in Rivers State, between March 2025 and March 2026. Participants comprised 63 adults with normal airway findings, 37 asthmatic adults, and 10 smokers. CT examinations were acquired at full inspiration and analyzed using an AI-assisted airway inspector on the Chest Imaging Platform. Airway wall thickness (AWT), airway lumen area (ALA) and wall area percentage (WA%) were extracted. Descriptive statistics, Shapiro–Wilk and Levene tests, one-way analysis of variance (ANOVA), Kruskal–Wallis testing with post-hoc comparisons, multiple linear regression, and an F-test were applied, with $p < 0.05$ considered statistically significant. **Results:** Mean AWT was 1.95 ± 0.30 mm among participants with normal airway findings, 2.79 ± 0.44 mm among asthmatics, and 3.45 ± 0.57 mm among smokers. ANOVA demonstrated a highly significant group difference ($F = 103.30$, $p < 0.001$). Tukey comparisons showed that AWT was greater in asthmatics than in participants with normal airway findings by 0.844 mm and greater in smokers than in participants with normal airway findings by 1.503 mm. The smoker–asthma difference was 0.659 mm; however, the non-parametric Dunn comparison did not demonstrate a statistically significant difference between these two groups (adjusted $p = 0.121$). Mean ALA was 20.0 ± 7.06 mm² in participants with normal airway findings, 19.1 ± 9.64 mm² in asthmatics, and 23.5 ± 7.79 mm² in smokers, with no significant overall difference ($p = 0.339$). WA% was $67.1 \pm 7.21\%$, $76.9 \pm 8.84\%$, and $76.8 \pm 9.11\%$ for participants with normal airway findings, asthmatics and smokers, respectively, with a significant group difference ($p < 0.001$). Regression analysis

showed that asthma and smoking were the principal predictors of AWT ($R^2 = 0.671$) and WA% ($R^2 = 0.298$), whereas age, gender, and weight were not significant predictors in the pooled models. Smokers had approximately 3.67 times the variance in AWT observed among participants with normal airway findings ($F = 3.67$, $p = 0.001$). Conclusion: This study provides preliminary locality-specific CT-derived airway reference values for participants with normal airway findings and demonstrates measurable airway remodelling in asthma and smoking. AWT and WA% appear more discriminating than ALA in this cohort. The findings support further multi-centre Nigerian studies recruiting a larger and gender-balanced sample size.

Keywords: airway wall thickness; airway lumen area; wall area percentage; quantitative computed tomography; asthma; smoking; airway remodelling; Nigeria.

INTRODUCTION

The conducting airways form a branching system responsible for conveying inspired and expired gases between the atmosphere and the respiratory surfaces. Beyond their role in ventilation, the airway epithelium, smooth muscle, connective tissue and vascular structures participate in host defense, mucociliary clearance and regulation of airway calibre. According to Ball et al. (2021), the lower respiratory tract includes the trachea, main bronchi, lobar and segmental bronchi, bronchioles and distal respiratory structures. Alterations in airway structure can therefore directly affects airflow and respiratory function.

Asthma is a chronic heterogeneous respiratory disease characterized by airway inflammation, variable airflow limitation and bronchial hyper responsiveness. Moreover, persistent inflammatory activity can lead to structural remodelling involving epithelial changes, subepithelial fibrosis, smooth-muscle hypertrophy and alterations in airway wall thickness (Holgate and Polosa, 2006; Lemanske and Busse, 2010; Kaur and Chupp, 2019). The global burden of asthma remains substantial, and a systematic analysis by Adeboye et al. (2022) demonstrated a considerable burden across low- and middle-income settings. In Nigeria, the burden is particularly relevant because underdiagnosis, unequal access to respiratory services, and environmental exposures may influence disease expression.

Tobacco smoking represents another major source of chronic airway injury. Tobacco smoke contains numerous toxic and carcinogenic constituents capable of inducing epithelial injury, inflammatory-cell recruitment, oxidative stress, and structural remodelling (Hecht, 2003; Drope et al., 2018). Donohue et al. (2012) examined a multi-ethnic cohort and reported associations between smoking exposure and thicker airway walls, while Dijkstra et al. (2013) demonstrated that airway wall measurements obtained from low-dose CT were associated with respiratory symptoms in heavy smokers. These observations indicate that quantitative airway measurements may reveal structural abnormalities even when conventional clinical assessments are less sensitive.

Quantitative CT has consequently been investigated as a means of objectively measuring airway morphology. Airway wall thickness (AWT), Airway lumen Area (ALA) and wall area percentage (WA%) can be obtained after segmenting the airway tree (Martinez et al., 2012; Kirby and Smith, 2023). Automated or semi-automated platforms can reduce the labour associated with manual measurements and can facilitate reproducible analysis. The Chest Imaging Platform and related airway-segmentation approaches have expanded the feasibility of quantitative airway analysis in research.

Despite the growing literature, much of the evidence originates from Europe, North America, and Asia. The present dissertation identified a paucity of comparable Nigerian reference data and specifically highlighted the absence of established locality-specific measurements for apparently healthy adults, asthmatics, and smokers in Rivers State. The present manuscript therefore investigates CT-derived AWT, ALA, and WA% across these groups and evaluates their potential value as comparative structural biomarkers.

PATIENTS AND METHODS

Study design and setting

An ambispective cross-sectional study was conducted at Image Diagnostic and Clinical Support Centre, Eligbam, Obio-Akpor Local Government Area, Rivers State, Nigeria, between March 2025 and March 2026.

The study was based on the parent dissertation dataset and was also designed to compare quantitative airway measurements among participants with normal CT airway reports, adults with asthma and smokers.

Participants and sampling

A total of 110 participants aged 18–75 years were included: 63 participants with normal CT airway reports (57.3%), 37 asthmatic participants (33.6%), and 10 smokers (9.1%). Participants were recruited using purposive and convenience approaches according to the inclusion criteria in the parent study. The participants with normal airway findings, as reported and signed by a consultant radiologist, were those referred for CT scans for other clinical indications involving the chest region. Asthma and smoking status were established by self-declaration based on the study recruitment criteria.

CT acquisition and quantitative airway analysis

Chest CT examinations were performed using a calibrated Siemens Somatom Emotion 64-slice CT scanner. Images were acquired with participants in the supine position and at full inspiration. The CT scanning parameters were standardized and audited by the department. The images were reconstructed at 1.0 mm slice thickness, with 1 mm increment, 512 mm × 512 mm matrix, viewed at a window level of –450 HU and a window width of 1500 HU (Ma et al., 2023). The bronchial wall metrics were obtained in cross-sectionally reformatted images for all segmental and subsegmental bronchi. The images of each bronchus examined were approximately perpendicular to the bronchial axis. The Chest Imaging Platform AI tool (Airway Inspector) was used to extract airway centre-lines. The airway boundary line was detectable when no adjacent tissue of similar CT density was present and was considered acceptable when it was at least 25% of the circumference detected at a specific location (Ma et al., 2023). Airways with an internal perimeter of 10 mm (Pi10) were measured by a single observer. The airway inspector software generated quantitative measurements including AWT, ALA and WA%. The approach is consistent with contemporary quantitative CT methodology, in which airway segmentation permits objective morphometric assessment (Kirby and Smith, 2023).

Outcome measures

The primary airway indices were AWT, ALA and WA%. AWT represents the thickness of the bronchial wall; ALA represents the internal calibre of the measured airway; and WA% represents the proportion of the airway cross-sectional area occupied by the wall. These measures were selected because they capture complementary aspects of airway remodelling.

Statistical analysis

Data were analysed using R statistical software. Descriptive statistics were presented as mean ± standard deviation and medians where appropriate. Distributional assumptions were assessed using the Shapiro–Wilk test and homogeneity of variance using Levene's test. Between-group comparisons were examined using one-way ANOVA and supported with Kruskal–Wallis testing because of the small smoker group and non-normality in some variables. Tukey and Dunn post-hoc procedures were also used as appropriate. Multiple linear regression was used to investigate the independent association of group, age, gender, and weight with each airway index. An F-test compared the variance in AWT and ALA between smokers and participants with normal airway findings. Statistical significance was set at $p < 0.05$.

RESULTS

Table 1. Summary of the descriptive analysis

Variable	All Participants	Participants with Normal Airway Findings	Asthmatic	Smokers
n (%)	110 (100%)	63 (57.3%)	37 (33.6%)	10 (9.1%)
Sex				
Male	35 (31.8%)	19 (30.2%)	6 (16.2%)	10 (100%)

Female	75 (68.2%)	44 (69.8%)	31 (83.8%)	0 (0%)
Age (yrs)				
Mean $\pm$ SD	50.3 $\pm$ 14.38	51.5 $\pm$ 14.9	49.7 $\pm$ 13.8	44.4 $\pm$ 13.1
Median	49	49	48	45.5
Weight (kg)				
Mean $\pm$ SD	78.2 $\pm$ 12.4	79.5 $\pm$ 12.0	74.7 $\pm$ 11.6	83.2 $\pm$ 15.2
Median	76	80	72	86
Airway wall thickness (mm)				
Mean $\pm$ SD	—	1.95 $\pm$ 0.299	2.79 $\pm$ 0.435	3.45 $\pm$ 0.572
Median	—	1.92	2.69	3.4
Min–Max	—	1.22–2.94	2.83–3.91*	2.68–4.59
Airway lumen Area (mm²)				
Mean $\pm$ SD	—	20.0 $\pm$ 7.06	19.1 $\pm$ 9.64	23.5 $\pm$ 7.79
Median	—	19.4	16.4	22.6
Min–Max	—	5.57–37.4	7.42–41.6	8.85–33.8
Airway wall area percentage (%)				
Mean $\pm$ SD	—	67.1 $\pm$ 7.21	76.9 $\pm$ 8.84	76.8 $\pm$ 9.11
Median	—	65.6	78.1	77.9
Min–Max	—	54.1–85.2	46.7–90.1	58.4–94.0

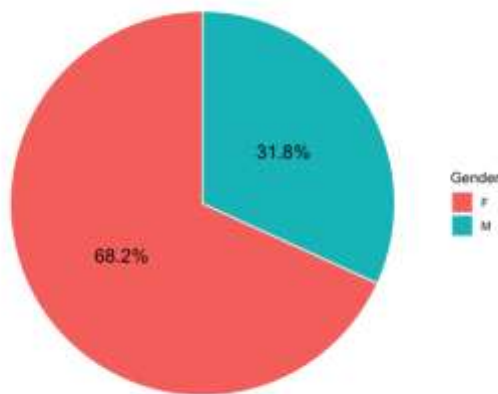


Figure 1: Pie Chart showing gender of all participants in the study

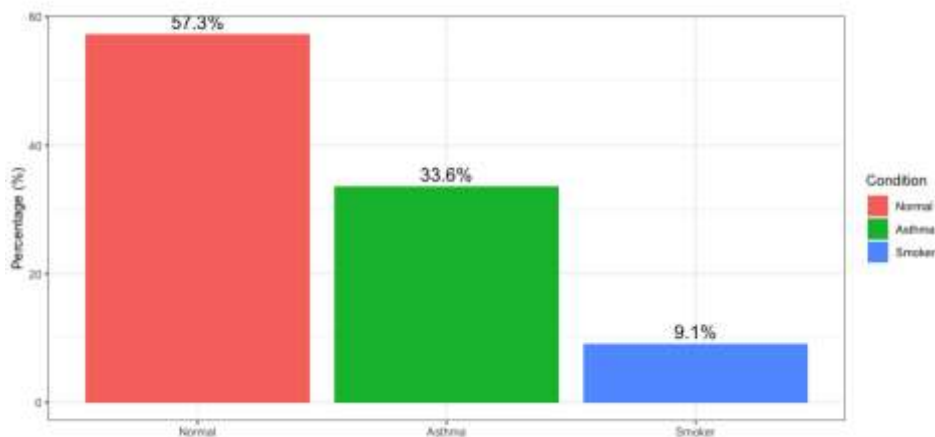


Figure 2: Bar chart showing the distribution of all participants viz: apparently healthy(normal), asthma, and smoker

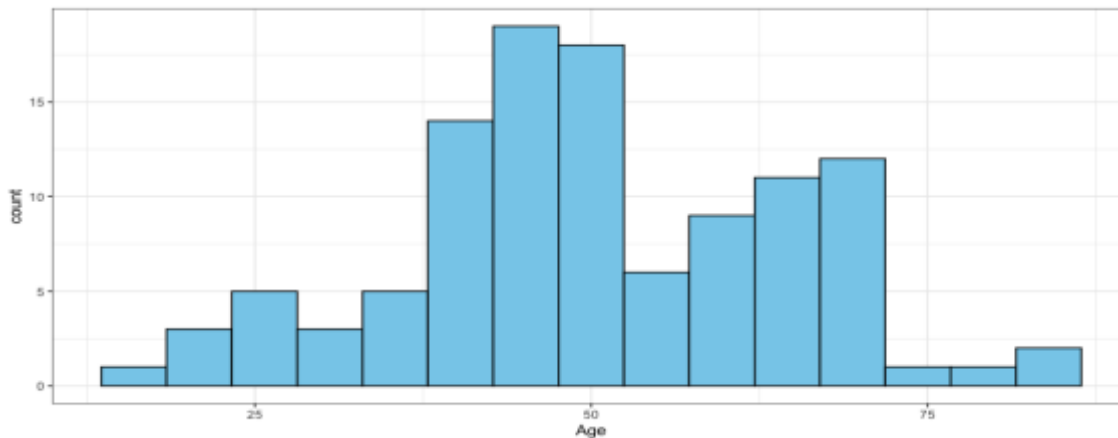


Figure 3. Distribution of age of all participants

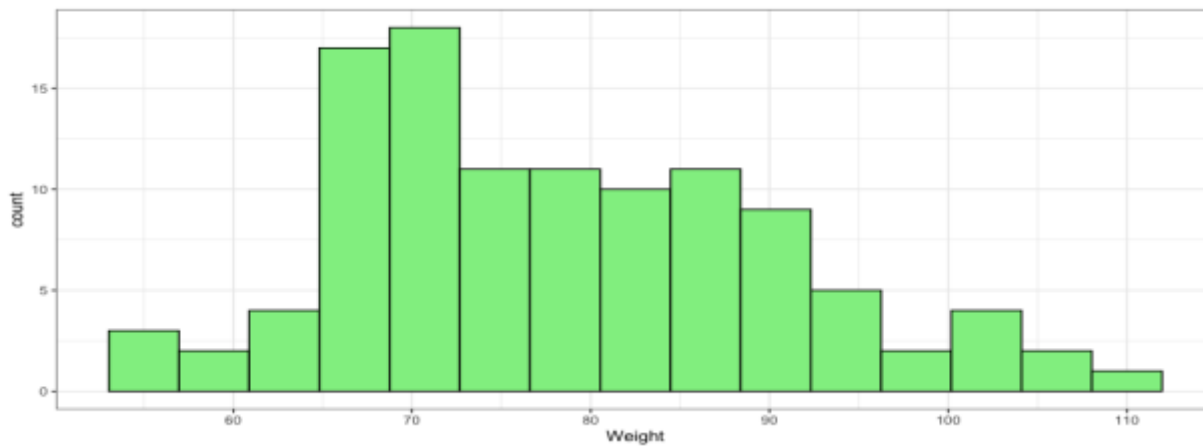


Figure 4: Weight distribution of all participants

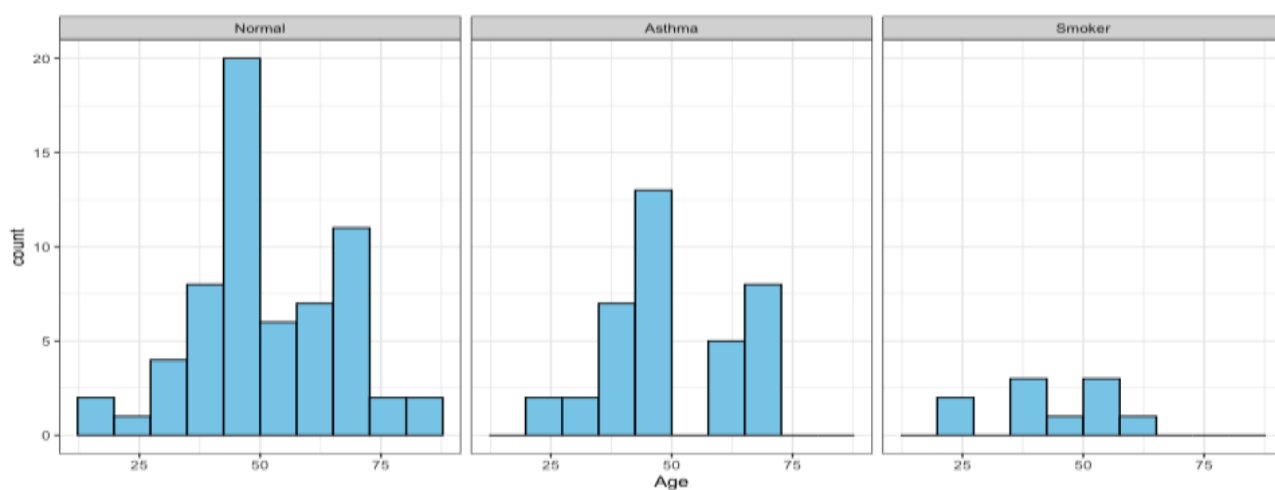


Figure 5: Age distribution among apparently healthy, asthmatic and smoker participants

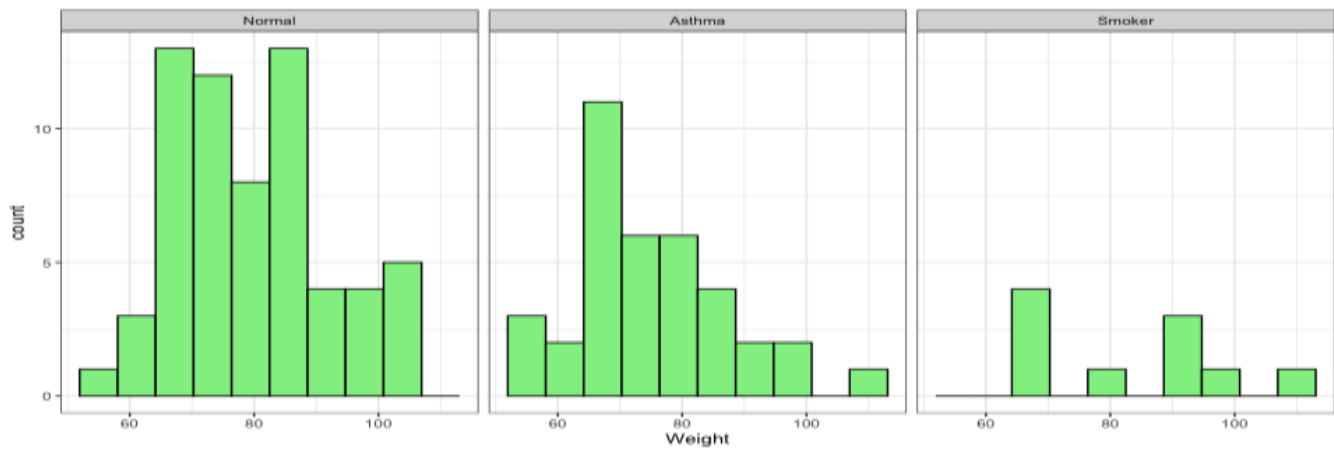


Figure 6: Weight distribution among participants with normal airway findings, asthmatics and smokers

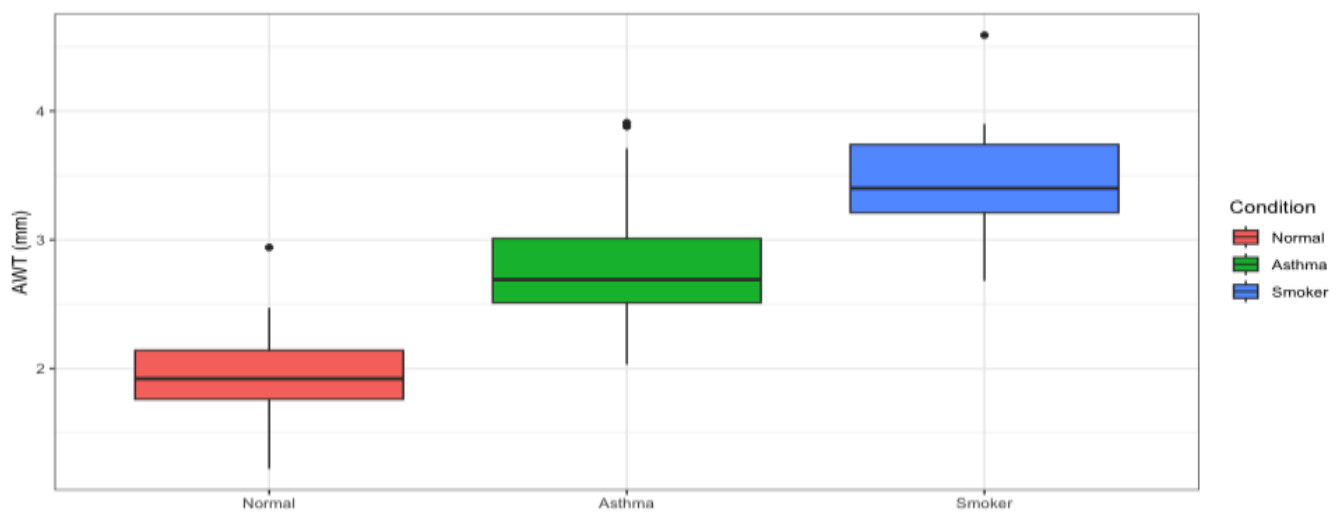


Figure 7: Boxplot of airway wall thickness

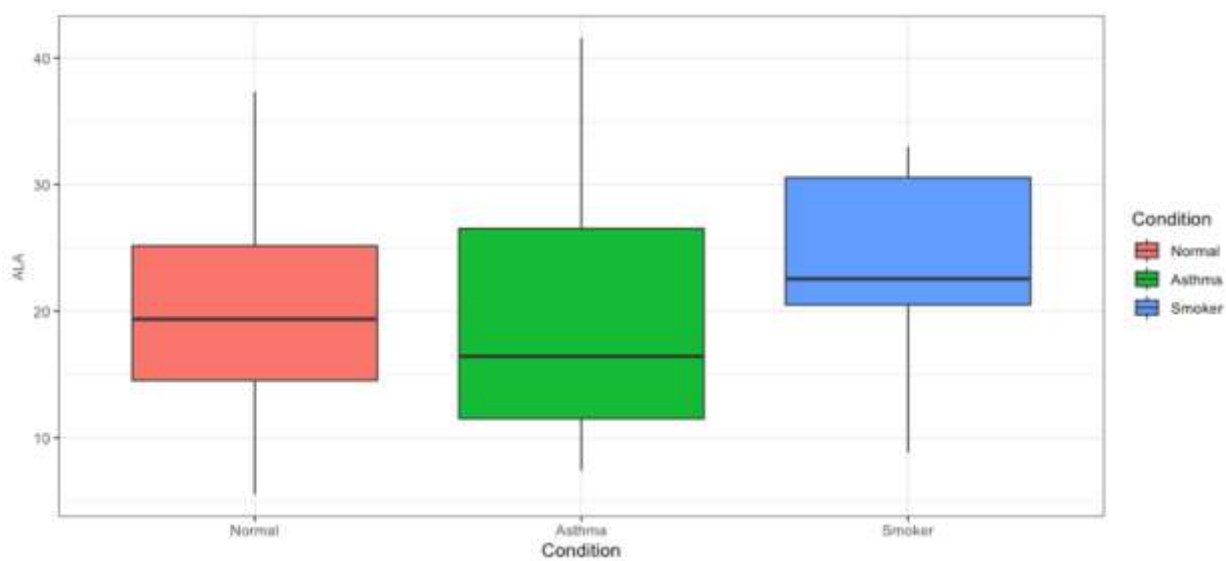


Figure 8: Airway Lumen Area boxplot

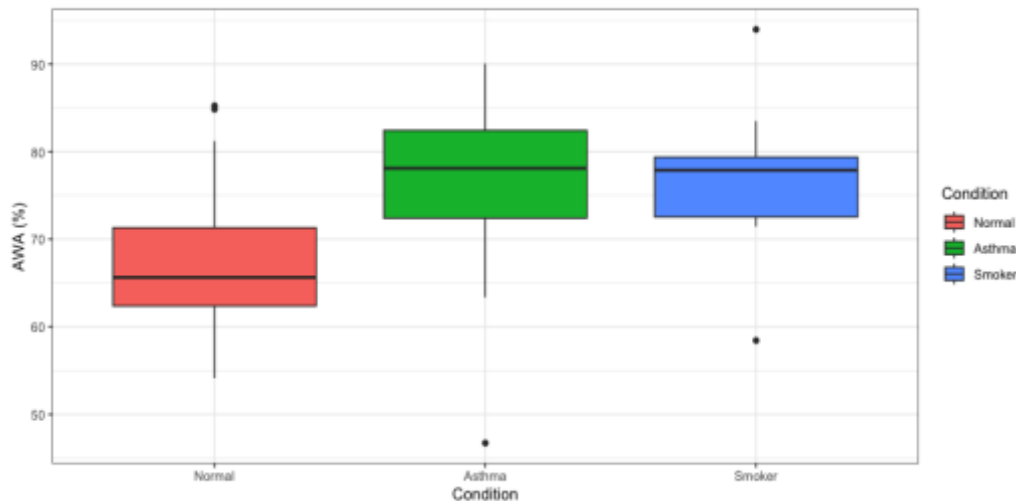


Figure 9: Airway Wall Area Percentage boxplots

The study included 110 participants: 63 participants with normal airway findings, 37 asthmatic, and 10 smokers. Overall, 75 participants (68.2%) were female, and 35 (31.8%) were male. The mean age was 50.3 ± 14.4 years, and the mean weight was 78.2 ± 12.4 kg. The participants with normal airway findings had a mean age of 51.5 ± 14.9 years and mean weight of 79.5 ± 12.0 kg; the asthma group had a mean age of 49.7 ± 13.8 years and mean weight of 74.7 ± 11.6 kg; and the smoker group had a mean age of 44.4 ± 13.1 years and mean weight of 83.2 ± 15.2 kg. The smoker group was entirely male, whereas females predominated among participants with normal airway findings and among asthmatic participants.

CT-derived airway indices

Mean AWT increased progressively from participants with normal airway findings to asthmatic participants and smokers. The corresponding values were 1.95 ± 0.30 mm, 2.79 ± 0.44 mm and 3.45 ± 0.57 mm.

Model statistics: $F(5.104) = 42.38$, $p < 0.001$, $\eta^2 = 0.671$, Adjusted, $\eta^2 = 0.655$. Mean ALA was 20.0 ± 7.06 mm², 19.1 ± 9.64 mm² and 23.5 ± 7.79 mm², respectively. Mean WA% was $67.1 \pm 7.21\%$ among participants with normal airway findings, $76.9 \pm 8.84\%$ among asthmatics and $76.8 \pm 9.11\%$ among smokers.

Table 4.2: Multiple linear regression analysis of factors associated with airway wall thickness (AWT)

Predictor	Estimate (β)	95% Confidence Interval	Standard Error	p-value
Intercept	1.64	1.098, 2.181	0.273	<0.001
Asthma (vs participants with normal airway findings)	0.853	0.693, 1.012	0.08	<0.001
Smoker (vs Participants with normal airway findings)				
Age (years)	1.549	1.257, 1.840	0.147	<0.001
Male (vs Female)	-0.001	-0.006, 0.005	0.003	0.819
Weight	-0.095	-0.276, 0.086	0.091	0.298
	0.005	-0.001, 0.011	0.003	0.127

AWT differed significantly across the three groups (ANOVA $F = 103.30$, $p < 0.001$). Tukey post-hoc testing demonstrated significant differences between participants with normal airway findings and asthmatics (mean difference 0.844 mm, 95% CI 0.659–1.031, $p < 0.001$), participants with normal airway findings and smokers (mean difference 1.503 mm, 95% CI 1.198–1.809, $p < 0.001$), and asthmatics and smokers (mean difference 0.659 mm, 95% CI 0.338–0.979, $p < 0.001$). Because AWT was not normally distributed in the asthma group and the smoker sample was small, a Kruskal–Wallis test was performed. It remained significant ($\chi^2 = 74.178$, $df = 2$, $p < 0.001$), while the Dunn procedure showed significant differences between participants with normal airway findings and asthmatics (adjusted $p < 0.001$) and between participants with normal airway findings and

smokers (adjusted $p < 0.001$), but not between asthmatics and smokers (adjusted $p = 0.121$). ALA did not differ significantly among the three groups overall ($p = 0.339$). In contrast, WA% differed significantly across groups ($\chi^2 = 32.782$, $df = 2$, $p < 0.001$). Dunn post-hoc testing demonstrated significantly higher WA% in asthmatic and smoker participants compared with participants with normal airway findings, while the difference between asthmatic and smoker participants was not statistically significant.

Regression analysis

The multivariable model for AWT was statistically significant, $F(5,104) = 42.38$, $p < 0.001$, and explained 67.1% of the observed variability ($R^2 = 0.671$; adjusted $R^2 = 0.655$). Relative to participants with normal airway findings, asthma was associated with an adjusted increase of 0.853 mm ($p < 0.001$) and smoking with an adjusted increase of 1.549 mm ($p < 0.001$). Age, gender and weight were not statistically significant predictors. The model for ALA was not statistically significant, $F(5,104) = 0.867$, $p = 0.506$, with $R^2 = 0.04$. The WA% model was statistically significant, $F(5,104) = 8.839$, $p < 0.001$, explaining 29.8% of variability ($R^2 = 0.298$; adjusted $R^2 = 0.264$). Asthma and smoking were significant predictors, whereas age, gender and weight were not. Among participants with normal airway findings analyzed separately, correlations between age or weight and the airway indices were weak. Weight showed the strongest observed association with WA% ($r = 0.273$).

DISCUSSION

The present study established CT-derived airway indices for participants with normal airway findings in Rivers State and demonstrated measurable differences associated with asthma and smoking. The central finding was the progressive increase in AWT from participants with normal airway findings through asthmatics to smokers. This pattern is biologically plausible because both chronic airway inflammation in asthma and tobacco-related injury can produce epithelial changes, inflammatory infiltration, smooth-muscle alteration and remodelling (Holgate and Polosa, 2006; Savin et al., 2023).

The participants with normal airway findings mean AWT of 1.95 ± 0.30 mm provides a locality-specific benchmark for this study population. Direct comparison with published values requires caution because airway generation, CT spatial resolution, reconstruction kernel, lung inflation, segmentation method and the precise definition of the measured airway can materially affect measurements (Matsuoka et al., 2005; Zach et al., 2012; Kirby & Smith, 2023). Nevertheless, the direction of difference between the groups is consistent with previous research. Patyk et al. (2020) examined patients with asthma and reported greater airway wall thickness across several bronchial generations, while Telenga et al. (2017) found increased AWT among current smokers.

Smoking produced the greatest mean AWT in the present cohort. This finding is in concord with the study of Donohue et al. (2012), who investigated smoking-related airway morphology and found that increasing cigarette exposure was associated with thicker airway walls. Dijkstra et al. (2013) similarly examined heavy smokers and found that greater airway wall thickness was associated with respiratory symptoms. Charbonnier et al. (2019) further demonstrated that airway wall thickness changed with smoking status, supporting its potential value as an imaging biomarker of smoking-related airway injury.

The absence of a statistically significant asthma-versus-smoking difference after non-parametric adjustment should be interpreted carefully. The smoker group contained only 10 participants and was entirely male, while the asthma group was predominantly female. Thus, the study was better powered to identify differences between the disease groups and the participants with normal airway findings than to distinguish asthma from smoking. The discrepancy between Tukey and Dunn post hoc analyses reinforces the need to avoid over-interpreting the apparent numerical difference between asthmatic and smoker participants.

Airway lumen area did not differ significantly among groups. This finding differs from studies that reported airway lumen narrowing in asthma or smoking, including Patyk et al. (2020), Chae et al. (2021) and Donohue et al. (2012). A possible explanation is that airway narrowing may be generation-dependent and more pronounced in smaller distal airways than in the segmental and subsegmental airways emphasized in the present analysis. Lung inflation, airway orientation and segmentation methodology may also influence lumen measurements (Huang et al., 2019; Kirby and Smith, 2023).

WA% was significantly higher in both asthmatic and smoker participants than in participants with normal airway findings. This result is important because WA% incorporates both wall and lumen dimensions and can therefore provide a relative measure of airway remodelling. Aysola et al. (2008) examined airway remodelling in severe asthma and demonstrated the usefulness of CT-derived airway measures as structural correlates of disease. Similarly, quantitative CT studies in smokers have associated airway wall measures with clinical and functional abnormalities (Grydeland et al., 2010; Lynch et al., 2015).

The regression results further demonstrated that disease or exposure status was more strongly associated with AWT and WA% than age, gender or weight within this dataset. The AWT model explained 67.1% variability, indicating a substantial contribution from group membership. This result differs from some larger studies in which demographic and anthropometric characteristics contributed materially to airway dimensions. Kim et al. (2011), for example, examined a large smoking cohort and identified gender and body-size variables as important determinants of airway dimensions. The absence of a significant gender effect in the present study should therefore not be interpreted as evidence that gender is biologically irrelevant. Rather, the strong imbalance in gender distribution, particularly the all-male smoker group, makes independent separation of gender from smoking difficult.

Similarly, the weak correlations observed among participants with normal airway findings suggest that age and weight alone were not strong determinants of the measured airway indices in this sample. Dudurych et al. (2024) and other population-based investigations have nevertheless shown that age, gender, body size and smoking history can influence quantitative airway measurements. The present results therefore support local reference standards while also demonstrating the need for larger Nigerian datasets that permit stratification by gender, age, height, body mass index and smoking exposure.

CONCLUSION

This ambispective study established preliminary CT-derived airway reference values for participants with normal airway findings in Rivers State and demonstrated significantly greater airway wall thickness and wall area percentage among asthmatic and smoking adults. Smoking was associated with the highest mean AWT. ALA did not differ significantly among the groups. The findings indicate that AWT and WA% may be more sensitive structural markers of airway remodelling than ALA in this cohort. However, the unequal group sizes and gender distribution warrant cautious application.

RECOMMENDATIONS

Larger multi-centre Nigerian studies should establish population-specific reference ranges for AWT, ALA and WA%. Future studies should recruit substantially larger and gender-balanced smokers and asthma groups. Quantitative CT indices should be correlated with spirometry, symptom severity, asthma duration, treatment and cumulative smoking exposure. CT acquisition, reconstruction and airway segmentation protocols should be standardized to improve inter-centre comparability. Automated airway analysis should be validated against manual measurements and alternative segmentation platforms before routine clinical implementation.

LIMITATIONS

The study was conducted at a single centre; it involved an unequal group structure, with only 10 smokers. Gender distribution was markedly unbalanced, particularly because all smokers were male. This observational design limits causal inference. Spirometric correlation was not performed; also, asthma duration, severity and detailed smoking exposure were not sufficiently incorporated into the reported models. Accordingly, the reference values should be regarded as preliminary and locality-specific rather than definitive national standards.

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