

Airway dimensions in asthma and COPD in high resolution computed tomography: can we see the difference?

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Abstract

Background: Airway remodeling in asthma and COPD results in bronchial wall thickening. The thickness of the bronchial wall can be measured in high resolution computed tomography (HRCT).

The objectives of the study were: 1) to assess the bronchial luminal and wall dimensions in asthma and COPD patients in relation to the disease severity, and 2) to compare the airway dimensions in patients with asthma and COPD.

Material and Methods: 10 asthma patients and 12 COPD patients with stable, mild to moderate disease were investigated. All patients underwent chest HRCT (window level - 450 HU, window width 1500 HU). Cross-sections of bronchi (external diameter 1.0-5.0 mm) were identified on enlarged images; the following variables were measured: external (D) and internal diameter (L), wall area (WA), lumen area (A_L), total airway area (A_O), WA% - the percentage of airway wall area, wall thickness (WT) and WT/D ratio. Separate sub-analyses were performed for airways with $D \leq 2.0$ mm and $D > 2.0$ mm.

Results: 261 and 348 cross-sections of small airways were measured in patients with asthma and COPD, respectively. There was a significant difference in WT and WA which were both greater in asthmatics than in COPD patients. In bronchi with $D > 2.0$ mm all measured parameters were significantly higher in asthma than COPD. In individual asthmatics the airway wall thickness was similar in all the assessed bronchi, while in COPD it was related to the external airway diameter.

Conclusions: Our results indicate that bronchial walls are thicker in asthmatics than in patients with COPD. It seems that airway wall thickness and the luminal diameter in patients with asthma are related to disease severity. There is no such a relationship in COPD patients.

HRCT may be a useful tool in the assessment of airway structure in obstructive lung disease.

Key words

asthma, COPD, airway thickness, airway remodeling, high resolution computed tomography

Introduction

Our knowledge on chronic inflammation and airway structural changes in asthma and chronic obstructive pulmonary disease (COPD) is mainly based on autopsy and bronchoscopic studies. The introduction of high resolution computed tomography (HRCT) offered new possibilities in the qualitative and quantitative assessment of airway (even as small as 1 mm in internal diameter) and lung tissue remodeling in asthma [1,2] and COPD [1,3]. There is evidence on the relationship between airway diameters measured in HRCT and results of other studies quantifying airway structure and function [4-7]. Moreover, HRCT seems a reliable tool in studying the changes in airway dimensions after nonspecific challenge tests [8,9], the effect of bronchodilators [6,10] and the consequences of airway remodeling e.g. air trapping [11].

The objectives of our study were: 1) to assess the bronchial luminal and wall dimensions in asthma and COPD patients in relation to the disease severity and 2) to compare airway dimensions in patients with asthma and COPD.

Material and Methods

The study was performed in patients with stable, mild-to-moderate asthma (n=10) and COPD (n=12). The study protocol was approved by the Institutional Review Board and all patients had signed an informed consent form.

The diagnosis and severity of asthma and COPD were assessed in accordance with the Global Initiative for Asthma [12] and the Global Initiative for Chronic Obstructive Lung Disease [13] guidelines, respectively. Inclusion criteria for asthmatic patients were as follows: 1) symptoms consistent with mild to moderate asthma according to GINA [12], 2) spirometric features of airway obstruction with a positive bronchial reversibility test (salbutamol 400 µg) and postbronchodilator $FEV_1/FVC > 70\%$, 3) positive result of methacholine challenge test. Diagnosis of COPD was based on: 1) a positive history of smoking (a minimum smoking history of 10 pack-years) and 2) symptoms

indicating COPD (productive cough, progressive exertional dyspnoea) and 3) FEV1/FVC<70 % in post-bronchodilator spirometry, 4) negative bronchial reversibility test.

Since the aim was to compare the radiographic features of airway remodeling in the natural course of the disease, only patients who had not been receiving inhaled corticosteroids for at least three months preceding the study onset were included. Therefore, only patients with newly recognised asthma were enrolled.

In all patients chest radiograph, pre- and postbronchodilator spirometry (Lung Test 1000, MES, Poland) according to the ERS statement [14], methacholine challenge test [15] and lung HRCT were performed. The demographic and clinical data of the asthma and COPD patients are shown in Table 1 and Table 2.

CT scanning was preceded by inhalation of a short acting beta-2 agonist (salbutamol, 400 µg via spacer) in order to achieve maximal bronchodilation. Chest CT scans were performed with 16-row CT scanner (LightSpeed 16 General Electric, USA) using: 1.25-mm collimation, 140 kV peak, 250 mA current and matrix size 512 x 512. Supine, end-inspiratory scans were performed with no contrast administration, at five selected lung levels: 1. superior margin of the aortic arch 2. the tracheal bifurcation 3. 1 cm below the tracheal bifurcation, 4. inferior pulmonary veins, 5. 2 cm above the dome of the right hemidiaphragm. The CT image data were reconstructed with a high spatial frequency algorithm and analyzed at a window level of – 450 HU and a window width of 1500 HU [16].

The CT images were enlarged (magnification x 10), and regions of interest were traced manually; the internal (L) and external diameter (D) were assessed by standard software analysis for distance measurement expressed in millimeters (mm). Then, cross-sections of bronchi with external diameter between 1.0 and 5.0 mm were identified. In order to select cross-sections perpendicular to the long airway axis, only airway cross-sections with ratio of the largest luminal diameter to the largest luminal diameter perpendicular to it ≤ 1.2 were submitted for further analysis [17].

Airway wall dimensions were measured by a radiologist blinded to the patient's diagnosis using a validated method described in earlier studies [9,18]. The interpretation of the radiological image and all the measurements were consulted with a pulmonologist. After D and L measurements, the following variables were calculated:

1. WT - wall thickness. With the assumption that the bronchial wall thickness is constant on the cross section: $WT = (D - L)/2$.
2. BWT - bronchial wall thickness expressed as the ratio of wall thickness and the external diameter; $BWT = WT/D$.
3. A_O (total bronchial area) = $\pi(D/2)^2$.
4. A_L (luminal area) = $\pi(L/2)^2$.
5. WA (airway wall area) = $A_O - A_L$.
6. WA% (the percentage of airway wall area) = $[WA/A_O] \times 100\%$.

The measurements are depicted on Figure 1. WT, BWT, WA and WA% were used to assess airway wall thickness, D and A_O defined the airway caliber while L and A_L estimated the bronchial lumen.

Two approaches were used to compare the results in asthmatics and COPD patients. First - mean values for all parameters were calculated in the individual patient and then asthma patients were compared to COPD patients. Second - all values measured in asthma patients were compared to respective values in patients with COPD. Separate sub-analyses for airways with $D \leq 2.0$ mm and $D > 2.0$ mm were performed in both groups, respectively.

All statistical calculations were performed using Statistica 6.0 software (StatSoft Inc., USA). Data are presented as mean \pm standard deviation. Mann-Whitney U-test was applied to compare two unrelated samples, while Spearman's rank correlation coefficient was utilized to test potential correlations between different variables. P values below 0.05 were considered statistically significant.

Results

We identified 261 and 348 bronchial cross-sections eligible for measurements in patients with asthma and COPD, respectively. The mean number of cross-sections evaluated in each patient was 25 ± 6 in asthma and 29 ± 6 in COPD group, $P > 0.05$. At each of the five evaluated lung levels mean 5 ± 2 bronchi were measured.

Cross-section airway dimensions in asthma patients

There were no differences in the relative airway wall thickness (expressed as mean BWT and WA%) measured at different lung levels. We found significant differences between the airway wall thickness in patients with mild (5 subjects, 119 airways) and moderate asthma (5 patients, 142 assessed bronchi). WA, A_0 , BWT and WA% were significantly larger, whereas the internal diameter and lumen area were significantly smaller in patients with moderate compared to mild asthma (Table 3).

There was no relationship between BWT or WA% and the duration of asthma symptoms or age. WA% and BWT calculated for airways with $D \leq 2$ mm and $D > 2$ mm were similar (Table 4).

Cross-section airway dimensions in COPD patients

There were no differences in the relative airway wall thickness measured at different lung levels. The airway wall thickness and airway caliber in 7 patients with mild (207 assessed bronchi) and 5 patients with moderate (141 bronchi) COPD were comparable.

The walls of smaller airways ($D \leq 2.0$ mm) were significantly thicker than the walls of larger bronchi ($D > 2.0$) (Table 4). However, there was no relationship between airway wall thickness (BWT or WA%) and the duration of symptoms or the patients' age. Interestingly, a significant positive correlation was found between the number of pack-years and WA ($r=0.68$) and WT ($r=0.82$).

Comparison of airway dimensions in asthma and COPD patients

Wall thickness (WT) and wall area (WA) of asthmatic airways were significantly greater than the respective values for the COPD airways. Cross-sections of asthmatic airways were also characterized by higher WA% and BWT than those found in COPD but the difference did not reach statistical significance (Table 5). The mean values of airway wall thickness parameters (WT, BWT, WA and WA%) in individual asthma patients also tended to be higher than those in individual COPD patients, but the differences were not statistically significant (Table 5).

Parameters defining the airway caliber (D , A_O) were slightly but not significantly higher in asthma than in COPD. Statistical significance was reached only for the comparison of A_O measurements in all the bronchi (Table 5). Airway lumen (expressed as A_L) was virtually the same in the asthmatic and COPD airways.

Analysis of the dimensions of the bronchi with external diameter larger than 2.0 mm revealed significant differences between asthmatic and COPD airways in all the measured parameters. The calculated dimensions were significantly greater in asthmatics when all assessed bronchi were compared (Table 6).

Discussion

We applied HRCT of the lung in a comparative evaluation of cross-sectional dimensions of the airways in patients with asthma and COPD. The results of our study suggest that the majority of cross-sectional airway dimensions is greater in asthmatics than in COPD patients. This finding particularly refers to the total bronchial area (A_O) and airway wall thickness (WA and WT). The mean values of these parameters were significantly higher in asthmatic than COPD airways when the results of all individual airway measurements were analyzed. Other parameters describing external airway dimension and wall thickness were also greater in asthmatics than COPD patients but the difference was not significant. Since, airway wall thickness (WT) and wall area (WA) do not change during bronchoconstriction, these variables are regarded as more reliable measures of airway thickening than WA% [17,19]. Despite the fact that the wall thickness was greater in asthmatics, the airway lumen

was similar in both studied groups when all the bronchi were analyzed. This interesting finding may lead to the conclusion that greater airway wall thickness is not necessarily associated with a smaller airway lumen. This was even more significant when only bronchi with $D > 2.0$ mm were compared in both groups. In this segment of airways all three: external bronchial dimensions, bronchial wall thickness but also bronchial lumen were greater in asthmatic than in COPD airways.

To our knowledge, there were only few HRCT studies directly comparing airway dimensions in asthma and COPD. Similarly to our findings, Simpson et al. observed an increased bronchial wall thickness in patients with neutrophilic asthma as compared to patients with COPD or smoking controls [20]. Kurashima et al. found that airway caliber in asthma was smaller than in COPD [21].

More studies addressed differences between airway dimensions in patients with asthma or COPD and healthy controls. Significant airway thickening in both diseases as compared to healthy subjects has been documented [3-5,22]. Airway wall thickening was observed in mild, moderate and severe asthma [4,5,18,22,23], but also in patients with cough-variant asthma [24]. Awadh et al. found that patients with moderate and severe asthma had greater airway wall thickening than those with mild disease [4]. It has also been reported that structural changes (airway thickening) in both large and small airways may develop in spite of optimal asthma treatment [23]. Gupta et al. demonstrated that HRCT measures of proximal airway remodeling is associated with impaired lung function [22].

Several studies showed an increased airway wall thickness in COPD patients (3,25,26). Moreover, airway wall thickening was also found in asymptomatic smokers [25]. In a study by Deveci et al., CT measurement of airway dimensions has been found useful for assessment of lung function in patients with COPD. This study showed that airway wall thickening was inversely related to the degree of airflow obstruction [3]. Similarly to significant correlation between number of pack-years and airway wall thickness parameters found in our study, a recent study by Donohue et al. showed an increase in airway wall thickness (calculated for an airway with an internal perimeter of 10 mm, P_{i10}) as a function of the number of smoking pack-years (mean increase 0.002 mm per ten pack-years) [27].

The relationship between the cigarette smoke exposure and airway wall thickness measured as Pi10 has also been reported by Grydeland et al. [28]

Based on the results of previous HRCT studies, a normal bronchial wall thickness in healthy subjects ranges between 0.18 and 0.23, and the normal range for percentage wall area is between 44 to 70% [3,4,17,25]. In our study BWT and WA% exceeded these values in both asthmatic and COPD patients. Thus, albeit we had not performed airway measurements in a control group, we may suppose that our patients with asthma and COPD showed an increased airway wall thickness. This is consistent with the results of several other studies [3,4,18,29,30].

In our previous paper, in which we compared the histopathological differences in the airway structure in asthma and COPD, we found a significantly greater basement membrane thickness in asthmatics [31]. Other differences, such as muscle layer thickening [32] and increased number of blood vessels in asthmatic airways, were also reported [33]. These findings support the hypothesis that airway remodeling results in more pronounced bronchial wall thickening in asthma than in COPD. However, one has to bear in mind that these studies were performed on samples from lobar and segmental bronchi, which are easily accessible by fiberoptic bronchoscopy. Data on histopathological changes in more distal airways are scarce. In our present analysis, we found that in COPD patients, airway wall thickness was significantly greater in airways with D between 1.0 and 2.0 mm than in larger bronchi. This might partially explain the significant difference between larger airway dimensions and lack of differences between small airway dimensions in asthma and COPD patients.

Our study showed that airway wall thickness was greater in moderate than in mild asthma. This was associated with a significantly smaller airway lumen area in patients with moderate as compared to patients with mild disease. Similar differences between airway wall thickness in patients with mild and moderate asthma were reported by Awadh et al. Interestingly, these authors did not find differences between airway dimensions of patients with moderate and severe disease [4]. Several other reports suggested a relationship between asthma severity and airway wall thickness [1,17,18].

These results are not consistent with histological findings, as airway basement membrane thickening was observed in the initial stages of asthma and did not seem to increase considerably with age or disease severity [34]. In our earlier studies, which were designed to evaluate the relationship between HRCT airway dimensions and lung function parameters as well as the airway inflammatory markers in asthma and COPD, we found a significant correlation between the inner airway diameter and eosinophil count in bronchoalveolar lavage fluid (BALF) but no significant correlation between airway dimensions and postbronchodilator FEV₁ [35,36]. There was, however, a significant negative correlation between airway luminal area (particularly when related to body surface area) and airway resistance (Raw) ($r=-0.75$ and $r=-0.92$, respectively) [35].

Some authors have observed a relationship between airway wall thickening and asthma duration [1,17]. Our study did not reveal any relation between the airway thickness and duration of symptoms and these findings are consistent with observations published by Little et al. [18].

Contrary to asthma patients, in COPD we found no correlation between airway wall thickness and severity of the disease. Similarly, Deveci et al. showed the same degree of airway thickening in patients with moderate and severe COPD [3]. It can be explained by different mechanisms of airway obstruction in asthma and COPD. In the latter loss of elastic load seems to play a major role.

We found more pronounced airway wall thickening in the smaller airways ($D \leq 2$ mm) as compared to larger airways in COPD patients. There is evidence that airways smaller than 2 mm internal diameter are the main site of airway obstruction in COPD, and the decline in FEV₁ is related to small airway wall thickening [37] and the loss of elastic load. Such a relationship has not been observed in patients with asthma. Our results are in agreement with those reported by Deveci et al. who also found a greater wall thickening in the distal airways of COPD patients and asymptomatic smokers [3].

We realize some significant limitations of our study. A direct comparison of asthma and COPD in the same severity stage may raise doubts because different criteria are applied for the

classification of severity in these diseases (symptoms, FEV₁, PEF variability in asthma vs. FEV₁ only in COPD). Therefore, mild or moderate asthma may not correspond with similar stage of COPD. Moreover, the comparison of airway dimensions in asthma and COPD could have been biased by the difference in the patients' age. It should be stressed, however, that albeit our asthmatics were significantly younger than COPD patients, they had a much longer duration of the disease. This may be an example of difficulties in appropriate study group matching when comparing asthma and COPD.

Another shortcoming of our study might be the lack of the control group of healthy subjects. This control group had not been planned because the study was aimed to compare different aspects of airway disease in asthma and COPD. However, the inclusion of healthy control subjects would probably add relevant information to our study, enabling direct comparison of airway dimension in obstructive airway diseases and healthy controls.

The role of cigarette smoke exposure not only in COPD patients, but also in asthmatics is yet another controversial point in our study. Cigarette smoke exposure in patients with asthma could potentially affect some measurements. This could be particularly important in comparisons which showed only a trend but not statistical significance. In COPD patients, airway structural changes could have been related to COPD, but also to tobacco smoke exposure. A study by Berger et al. showed that some HRCT parameters which reflect airway thickening might be significantly higher in smokers with normal lung function than in healthy non-smoking controls [38]. As we have not studied the group of asymptomatic smokers we are not able to differentiate between airway changes related to smoke exposure itself and those associated with COPD. Thus, the interpretation of our results might be somewhat ambiguous.

The number of patients in each group was relatively small. However, there were more than 200 and 300 individual airways assessed in asthma and COPD group, respectively. The main limiting factors affecting sample sizes was the requirement not to use glucocorticosteroids in the pre-study

period and the patient's consent to exposure to radiation during HRCT. The measurements were performed by one radiologist but consulted with a pulmonologist. In some previous CT studies, airway dimensions were also assessed by one radiologist and a good intra- and interobserver reproducibility was documented [17]. The results obtained in studies involving one radiologist [17] were comparable to those in which two specialists participated [3,4,18,27].

On the other hand, we made efforts to eliminate all the factors, which could potentially influence the study results. We applied approved methods and paid special attention to the inclusion criteria. As earlier studies revealed that inhaled corticosteroid therapy results in a significant decrease in basement membrane thickness [39] and airway wall thickness [2] the asthma patient study group was limited to those subjects who had not been treated with steroids.

Conclusions

Our results suggest that airway walls in asthma are thicker than in COPD, and this difference is more pronounced in airways with an external diameter greater than 2 mm. It seems that airway wall thickness and the luminal diameter in patients with asthma are related to the severity of the disease, while this is not the case in COPD patients.

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Figure legend

Figure 1. HRCT airway cross section measurements of D – external diameter, L – internal (luminal) diameter, WT – wall thickness, A_O – total airway area, A_L – luminal area, WA – wall area.

Table 1

	Asthma	COPD	P
Sex (M/F)	4/6	7/5	0.6
Age (years)	36 (27-47)	58.5 (49-64)	0.002
BMI (kg/m ²)	25.2 (23.0- 27.7)	25.1 (23.2-30.0)	0.23
Onset of symptoms (age)	8 (3-30)	55 (43-60.5)	0.0001
Duration of symptoms (years)	17 (5-31)	3 (1.5-8)	0.021
Atopy n (%)	7 (70%)	2 (16%)	0.0007
Never smokers/past smokers/ current smokers	6/3/1	0/5/7	0.0047
Pack-years	0 (0-1.5)	37.5 (30-45.5)	0.005
Severity of disease: mild (n)/moderate (n)	5/5	7/5	0.8
Postbronchodilator FEV ₁ L (% predicted)	2.95 (2.4-3.7) 87 (82-95)	2.3 (1.6-2.8) 74.5 (63.5-86)	0.2

Table 1. The characteristics of the study groups.

Table 2

	Asthma			COPD		
	mild	moderate	P	mild	moderate	P
Number of patients	5	5	NS	7	5	NS
Age (years)	37(31-41)	35(27-47)	NS	62(49-63)	55(53-67)	NS
Sex (M/F) n	3/2	1/4	NS	5/2	3/2	NS
Height (cm)	182(163-186)	167(162-167)	NS	169(164-175)	172(160-180)	NS
Body weight (kg)	93(62-96)	68(65-72)	NS	72(65-94)	73(60-83)	NS
BMI (kg/m ²)	26.3(23.3-27.7)	24(23-27.1)	NS	25.1(24.2-40)	25(21.5-28.4)	NS
Onset of symptoms (age)	4(3-30)	12 (4-30)	NS	59 (40-60)	51 (48-61)	NS
Duration of symptoms (years)	17 (9-33)	17 (5-18)	NS	3 (2,5-7,5)	2(1-6)	NS
Atopy n (%)	4(80%)	3(60%)	NS	0	1(20%)	NS
Never smokers/past smokers/ current smokers	3/1/1	3/2/0	NS	0/4/3	0/1/4	NS
Pack-years	0(0-5)	0(0-1)	NS	35(30-43)	40(33-47)	NS
Postbronchodilator FEV ₁ (% predicted)	95(90-99)	83(82-84)	0.04	86(78-87)	64(55-70)	0.01

Table 2. Comparative characteristics of patients with mild and moderate asthma and COPD.

Table 3

	Mild asthma (N = 119) mean ± SD	Moderate asthma (N = 142) mean ± SD	P
L (mm)	1.5 (1.0-1.9)	1.0 (0.8-1.5)	0.00001
WT (mm)	0.8 (0.6-1.0)	0.7 (0.5-1.0)	0.06
BWT	0.27 (0.23-0.29)	0.29 (0.26-0.31)	0.0001
A _O (mm ²)	7.5 (4.1-11.3)	4.1 (2.5-10.2)	0.002
A _L (mm ²)	1.8 (0.8-2.8)	0.8 (0.5-1,8)	0.0001
WA (mm)	5.5 (3.1-9.1)	3.5 (2.0-8.0)	0.002
WA%	78 (72-83)	82 (78-86)	0.0001

Table 3. Airway dimensions in asthma with regard to disease severity, N – number of assessed bronchi. Abbreviations: D – external diameter, L – internal (luminal) diameter, WT – wall thickness, BWT = WT/D, A_O – total airway area, A_L – luminal area, WA – wall area, WA% = [WA/A_O] x 100%

Table 4

	Airway wall thickness variables	Airways with D ≤ 2mm	Airways with D > 2mm	P
Asthma	BWT	0.28 (0.26-0.31)	0.28 (0.25-0.3)	0.1
	WA%	80 (78-85)	81 (75-84)	0.1
COPD	BWT	0.28 (0.25-0.31)	0.27 (0.24-0.3)	0.01
	WA%	80 (75-86)	78 (72-85)	0.01

Table 4. Comparison of mean values of BWT and WA% calculated for airways with D ≤ 2 mm and > 2 mm in asthma and COPD airways. Abbreviations: BWT = WT/D, WA%= [WA/A₀] x 100%.

Table 5

	Asthma n=10 patients mean±SD	COPD n=12 patients mean±SD	P		Asthma N = 261 airways mean±SD	COPD N = 348 airways mean± SD	P
D (mm)	3.1(2.3-3.8)	2.7(2.4-3.1)	0.29		2.8(1.9-3.7)	2.5(2.0-3.2)	0.27
L (mm)	1.4(0.9-1.7)	1.2(1.0-1.4)	0.53		1.2(0.8-1.7)	1.1(0.8-1.5)	0.11
WT (mm)	0.78(0.66-1.04)	0.74(0.6-0.8)	0.33		0.75(0.55-1.05)	0.7(0.55-0.85)	0.005
BWT	0.27(0.26-0.3)	0.27(0.26-0.28)	0.8		0.28(0.25-0.3)	0.27(0.24-0.31)	0.12
A_O (mm²)	8.0(4.5-11.5)	6.3(4.8-7.7)	0.35		6.1(2.8-10.7)	4.9(3.1-8.0)	0.02
A_L (mm²)	1.8(0.8-2.3)	1.4(1.0-1.8)	0.62		1.1(0.5-2.3)	0.95(0.5-1.8)	0.11
WA (mm²)	5.9(3.6-9.3)	4.8(3.7-5.9)	0.29		4.6(2.3-8.7)	3.9(2.5-6.2)	0.01
WA%	78(77-83)	78(76-80)	0.6		81(75-84)	79(73-85)	0.12

Table 5. Comparison of the mean airway dimensions in asthma and COPD. Left columns present comparison between patients (n = number of patients) while right columns show comparison between bronchial dimensions (N = the total number of bronchi measured).

Abbreviations: D – external diameter, L – internal (luminal) diameter, WT – wall thickness, BWT = WT/D, A_O – total airway area, A_L – luminal area, WA – wall area, WA% = [WA/A_O] x 100%.

Table 6

	Asthma n=10 patients mean±SD	COPD n=12 patients mean±SD	P		Asthma N = 183 airways mean±SD	COPD N = 258 airways mean± SD	P
D (mm)	3.4(2.9-3.8)	3.0(2.7-3.1)	0.12		3.3(2.7-3.9)	2.8(2.3-3.5)	0.1
L (mm)	1.5(1.2-1.7)	1.4(1.2-1.6)	0.62		1.5(1.1-1.8)	1.4(1.0-1.6)	0.008
WT (mm)	0.9(0.8-1.0)	0.8(0.7-0.8)	0.06		0.9(0.7-1,1)	0.7(0.6-0.9)	0.00001
BWT	0.27(0.26-0.29)	0.27(0.25-0.28)	0.84		0.28(0.25-0.3)	0.27(0.24-0.3)	0.047
A_O (mm²)	9.8(7.0-11.5)	7.6(5.9-8.1)	0.07		8.5(5.7-11.9)	6.2(4.2-9.6)	0.0001
A_L (mm²)	1.9(1.4-2.3)	1.6(1.2-2.2)	0.37		1.8(0.95-2.5)	1.4(0.78-2.0)	0.008
WA (mm²)	7.7(5.5-9.3)	5.5(4.7-6.2)	0.03		6.8(4.4-9.7)	4.9(3.5-7.1)	0.00001
WA%	79(77-83)	78(74-80)	0.7		81(75-84)	78(72-85)	0.047

Table 6. Comparison of the dimensions of bronchi with external diameter greater than 2 mm. Left columns - comparison between patients (n = number of patients); right columns – comparison between dimensions of all measured bronchi (N= the total number of bronchi measured). Abbreviations: D – external diameter, L – internal (luminal) diameter, WT – wall thickness, BWT = WT/D, A_O – total airway area, A_L – luminal area, WA – wall area, WA%= [WA/A_O] x 100%.

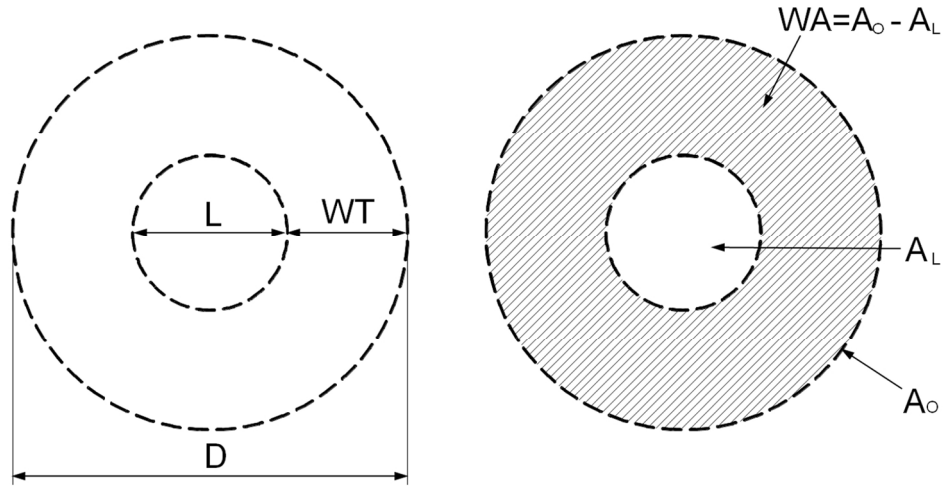


Figure 1. HRCT airway cross section measurements of D – external diameter, L – internal (luminal) diameter, WT – wall thickness, A_o – total airway area, A_L – luminal area, WA – wall area.
130x80mm (300 x 300 DPI)