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Utility of computed tomography in assessment of pulmonary hypertension secondary to biomass smoke exposure

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Background: The aim of this study was to investigate the feasibility of main pulmonary artery diameter quantification by thoracic computerized tomography (CT) in the diagnosis of pulmonary hypertension seconder to biomass smoke exposure.

Material/Methods: One hundred and four women subjects with biomass smoke exposure and 20 healthy women subjects were enrolled in the prospective study. The correlation between echocardiographic estimation of systolic pulmonary artery pressure and the main pulmonary artery diameter of the cases were studied.

Results: The main pulmonary artery diameter was 26.9±5.1 in the control subjects and 37.1±6.4 in subjects with biomass smoke exposure. This difference was statistically significant ($p<0.001$). The systolic pulmonary artery pressure was 22.7±12.4 in the control subjects and 57.3±22 in subjects with biomass smoke exposure. This difference was statistically significant ($p<0.001$). Systolic pulmonary artery pressure was significantly correlated with the main pulmonary artery diameter ($r=0.614$, $p<0.01$). A receiver operating characteristic (ROC) curve analysis showed that a value of 29 mm of the main pulmonary artery diameter differentiated between pulmonary hypertension and non-pulmonary hypertension patients. The sensitivity of the measurement to diagnose pulmonary hypertension was 91% and specificity was 80%.

Conclusions: Our results indicate that main pulmonary artery diameter measurements by SCT may suggest presence of pulmonary hypertension in biomass smoke exposed women.

MeSH Keywords: Tomography, X-Ray Computed • Biomass • Hypertension, Pulmonary

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Background

Biological fuels that produce heat are called biomass. It is predicted that half of the world population and more than 90% of the rural population in developing countries uses biomass fuels [1]. In Turkey, biomass is used in regions with low socioeconomic levels. The most commonly utilized biomass fuel is called *turd*, a fuel type produced by dehumidifying animal waste. Turd is used for cooking bread and meal in a closed oven called a *tandir*. The tandir has no ventilation except for the entry door and a small chimney. Biomass smoke is a mixture of complex particles, gases such as carbon monoxide, sulfur oxides, nitrous oxides, aldehydes, and poly-organic matter, including polycyclic aromatic hydrocarbons [2]. These particles and gases are harmful to human health and may cause cancer [3]. The correlation between the effects of biomass smoke in respiratory functions, the structure of the lungs, and numerous pulmonary pathologies like chronic obstructive pulmonary disease, interstitial pulmonary disease, cancer, asthma, and lower respiratory tract infections has already been established [4–8].

The relation of biomass with pulmonary hypertension and cor pulmonale has been reported in several studies [2,9–13]. In Van province in eastern Turkey, we reported a higher level of pulmonary hypertension in pulmonary diseases of women exposed to biomass than in pulmonary diseases related to cigarette smoking. Previously, we reported that pulmonary hypertension could develop even without the presence of a pulmonary disease [14].

Pulmonary angiography is the "gold standard" test for the diagnosis of pulmonary hypertension**.** However, it is an invasive procedure with risks of complications. For this reason, numerous noninvasive methods are utilized for diagnosis of pulmonary hypertension, including CT. A thorax CT scan is used frequently in pulmonary diseases practice. The aim of this study was to investigate the feasibility of main pulmonary artery diameter quantification by intravenous (iv) contrast agent administered thoracic computerized tomography (CT) in the diagnosis of pulmonary hypertension secondary to biomass smoke exposure.

Material and Methods

In this study, 104 female subjects with biomass smoke exposure and 20 healthy female subjects were enrolled.

Age, biomass smoke exposure duration and density, echocardiographic estimation of sPAP, mPAD measured by thoracic CT, and diagnoses were recorded.

Biomass smoke exposure was recorded as the number of exposure-years, the number of days per week, and hours per day. The biomass smoke exposure duration was expressed as the number of years. Biomass smoke exposure density was expressed as the number of hours per year. For example, if a person is exposed to biomass smoke for 1 hour per day for a year, her biomass smoke exposure density is 1 hour/year.

Patients with the following criteria were included in the study as the biomass smoke-exposed group: (1) clinical or radiological evidence of pulmonary hypertension and cor pulmonale, (2) long-term biomass smoke exposure (at least 10 years), and (3) the presence of measurable sPAP in echocardiographic evaluation. Twenty female patients who were evaluated for suspected pulmonary embolism, but who had normal findings by echocardiography and thorax CT, served as the control group. Patients were excluded from either the biomass smoke-exposed or control groups if they had primary left ventricular dysfunction or acute cardiac illness, obesity, a history of significant organic and/or inorganic dust exposure, a history of cigarette smoking, passive cigarette smoke exposure, or a clear history of a known chronic lung disease before exposure to biomass smoke (e.g., tuberculosis, childhood respiratory diseases, thoracic structural anomalies).

The study protocol was carried out in accordance with the Helsinki Declaration as revised in 2000. The study protocol was approved by the local ethics committee, and informed consent was obtained from each subject.

The patients in the biomass smoke-exposed group were further divided into 4 subgroups for the purpose of determining the correlation of the mPAD measurements with the clinical features in each group.

Group 1 (chronic obstructive pulmonary disease group): Chronic obstructive pulmonary disease was diagnosed by the signs and symptoms of chronic bronchitis and/or pulmonary emphysema, and the presence of chronic and irreversible airflow obstruction (forced expiratory volume in 1 second (FEV1)/forced vital capacity below 70%, FEV1 below 80% predicted). Asthma was excluded, as assessed by clinical history and response to bronchodilators (12% increase in FEV1 following 400 mg of inhaled salbutamol) [15].

Group 2 (Pulmonary embolism): The criterion used for diagnosis of pulmonary embolism on iv contrast medium-administered thoracic CT was an intraluminal filling defect. A CT scan was interpreted as revealing positive findings for an embolus only if a definite filling defect was seen on more than 1 contiguous axial image.

Groups 3 and 4: These patients had normal pulmonary function tests with greater PAP values. Group 3 patients had pulmonary hypertension associated complaints and findings such

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as dyspnea and/or peripheral edema and/or cor pulmonale, while Group 4 patients did not have these same findings. We previously reported on a group of women exposed to biomass smoke; they did not have airway obstruction, but had pulmonary hypertension and cor pulmonale [15]. Therefore; Group 3 was termed the idiopathic pulmonary arterial hypertension-like group and Group 4 was termed the asymptomatic idiopathic pulmonary arterial hypertension-like group. The last 2 groups might have prominent pulmonary artery involvement that could be related to biomass smoke exposure.

Echocardiographic measurement

Echocardiography was performed by the same cardiologist (with 7 years of experience) using a Vivid 3 instrument (General Electric, USA) and by utilizing a 2-MHz probe. The gradient between the right ventricular peak systolic pressure and right atrium pressure was measured by Doppler echocardiography at rest in cases with tricuspid insufficiency. The modified Bernoulli equation was used to calculate PAP pressure: PAP=4 × (tricuspid systolic jet). The estimated sPAP was obtained by adding the right atrium mean pressure. Right atrial pressure is estimated to be 5 mmHg when the diameter of the inferior vena cava (IVC) is <1.7 cm and a 50% decrease in the diameter with inspiration, 10 mmHg when IVC is >1.7 cm and with normal inspiratory collapse (≥50%), and 15 mmHg when IVC is >1.7 cm and inspiratory collapse is less than 50% [8]. When sPAP is >35 mmHg, the presence of pulmonary hypertension is established according to the new recommendations of the Working Group on Diagnosis and Assessment of Pulmonary Arterial Hypertension in the 4th World Symposium on Pulmonary Hypertension [9].

Thoracic CT scan

A thoracic CT scan was performed using a 4-detector multislice CT scanner (SOMATOM Sensation 4, Siemens, Erlangen, Germany) by injecting contrast agents intravenously while the patient held her breath. Scanning began 20 seconds after the start of contrast material injection. The non-ionic contrast material (Ultravist 300, Schering, Berlin, Germany) at an average volume of 100 mL and concentration of 300 mg I/100 mL) was administered by a power injector at a rate of 3 mL/second via an 18-gauge plastic angiocatheter inserted into the cubital vein. Imaging parameters were 120 kV and 152 mA, with 0.5 second rotation time, 4×2 mm collimation, and 5 mm slice thickness. Data were transferred to a network computer workstation (Leonardo; Siemens Medical Systems, Germany) and diameters were measured using a window level of 400 HU, with the center at 60 HU. The widest diameter perpendicular to the long axis of the main pulmonary artery was measured with computer calipers at the level of the pulmonary artery bifurcation by mediastinal window (Figure 1).

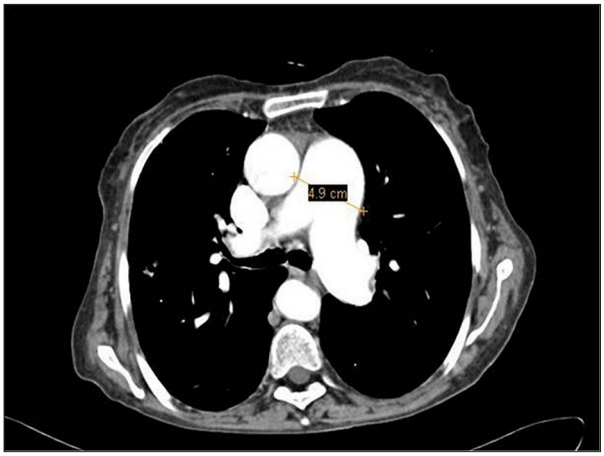

Figure 1. The widest diameter perpendicular to the long axis of the main pulmonary artery was measured with computer calipers at the level of the pulmonary artery bifurcation by mediastinal window.

Statistical analysis

Differences between subgroups were evaluated by one-way analysis of variance. Duncan's multiple range test was utilized to determine differences between group means. A Pearson correlation analysis was conducted to examine liner relationships among variables (age, years of exposure, density of exposure) with sPAP and mPAD, and between sPAP and mPAD in the exposed group and in each subgroup. The control and study groups were compared by Student's t test. Receiver operating characteristic (ROC) curve analysis was performed to find a cut-off value of mPAD for diagnostic ability to predict sPAP >35 mmHg as pulmonary hypertension. The test measurement sensitivity, specificity, positive predictive rate, negative predictive rate, and truth value were all estimated by standard formulas.

Results

The demographic characteristics of the subjects in the study are shown in Table 1. There were no statistically significant differences between subjects with biomass smoke exposure and healthy subjects with respect to age (p>0.05).

The mean mPAD and the mean sPAP were significantly higher in subjects with biomass smoke exposure than in healthy subjects (p<0.001 for both).

The biomass smoke exposure characteristics of the patients in the study subgroups are shown in Table 2. There were no statistically significant differences between the mean ages of the participants in the subgroups (p>0.05). Likewise, no significant difference was observed between the biomass smoke

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Table 1. Comparison of total biomass smoke exposed and control group properties.

	Exposed group (n=104)	Control group (n=20)
mPAD*	37.1±6.4	26.9±5.1
sPAP*	57.3±22	22.7±12.4
Age	60.3±8.3	64.4±10.9

* p<0.001. mPAD – main pulmonary artery diameter; sPAP – systolic pulmonary artery pressure

sPAP was significantly correlated with the mPAD in the total biomass smoke-exposed group and in each subgroup (r=0.614, p<0.01) (Table 3, Figure 2).

In the ROC analysis, the threshold value for predicting pulmonary hypertension at which the highest diagnostic sensitivity and specificity intersected was found when mPAD was ≥29 mm (area under the curve (AUC)=0.86). Its sensitivity, specificity, positive predictive value, negative predictive value, and accuracy were 91%, 80%, 97%, 50%, and 84%, respectively (Figure 3).

Table 2. Comparison of subgroup properties.

	N (%)	Age mean ±SD	Biomass exposure duration (year)	Biomass exposure density (hours/year)	sPAP	mPAD
COPD	44 (30.6%)	66±8.4	36.52±9.39	88.21±59.33	54.40±24.50	36.50±6.96
PE	11 (7.6%)	66±17.0	35.09±12.06	57.41±56.85	65.00±22.50	37.27±5.98
IPAHLG	25 (17.4%)	62±11.0	32.68±8.93	64.32±39.06	61.12±21.71	37.84±7.16
AIPAHLG	24 (16.7%)	64±9.7	34.00±8.05	78.05±73.06	40.08±15.00*	32.21±6.26*

* p<0.001. Values are mean ±SD. COPD – chronic obstructive pulmonary disease; PE – pulmonary embolusI; PAHLD – idiopathic pulmonary arterial hypertension-like group; AIPAHLG – asymptomatic idiopathic pulmonary arterial hypertension-like groups; PAP – systolic pulmonary artery pressurem; PAD – main pulmonary artery diameter.

Table 3. Correlation between systolic pulmonary artery pressure (sPAP) and main pulmonary artery diameter (mPAD) in the total biomass smoke-exposed group and each subgroup.

Subgroup	Correlation coefficient (r)	
COPD	0.605	p<0.01
PE	0.810	p<0.01
IPAHLG	0.432	p<0.05
AIPAHLG	0.596	p<0.01
Total group	0.614	p<0.01

exposure durations or the biomass smoke exposure densities in the subgroups (p>0.05).

The mean mPAD and the sPAP value were significantly lower in the asymptomatic idiopathic pulmonary arterial hypertension-like group compared to the other subgroups (p<0.001) (Table 2).

Twelve patient had sPAP< 35 mmHg in the biomass smoke-exposed group. Four patient had sPAP >35 mmHg in the control group.

Discussion

Biomass smoke is composed of a relatively equal mixture of coarse (2.5–3.5 μm) and ultrafine/fine (0.02–2.5 μm) particles and can penetrate deeply into the lung, producing a variety of morphologic and biochemical changes [16,17]. A recent meta-analysis, which reviewed risk of chronic obstructive pulmonary disease from exposure to biomass smoke, concluded that biomass smoke exposure is a clear risk factor for chronic obstructive pulmonary disease [18] and reported that clinical characteristics, quality of life, and mortality rates of biomass smoke-related chronic obstructive pulmonary disease cases were similar in degree to that of tobacco smokers [19–22].

The relationship between biomass smoke exposure and pulmonary hypertension and cor pulmonale has long been established [9–11]. Because the prevalence of cor pulmonale is high in female patients with ages ranging from 20 to 60 years, Padmavati et al. conducted an autopsy study in Delhi, India [9]. They reported that none of the cases had a history of cigarette smoking; however, the common anamnesis was the exposure to biomass smoke in ill-ventilated environments. They concluded that pulmonary hypertension and cor pulmonale development might have a correlation with biomass smoke exposure [9]. Furthermore, Sandoval et al. reported a clinical picture with chronic pulmonary disease

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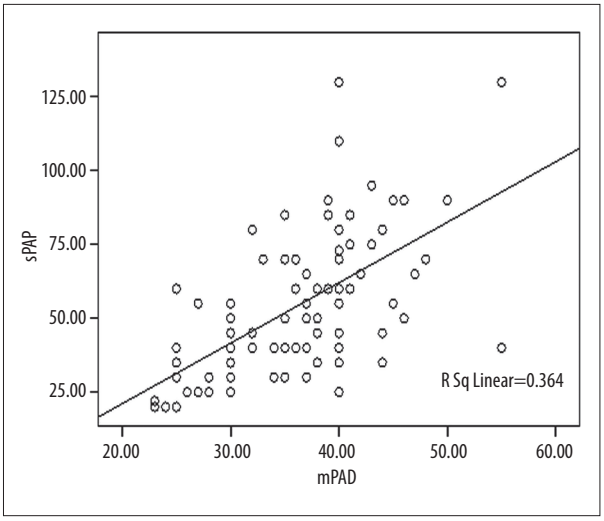

Figure 2. Correlation of systolic pulmonary artery pressure (sPAP) and main pulmonary artery diameter (mPAD) in the biomass smoke-exposed group (r=0.614, p<0.01).

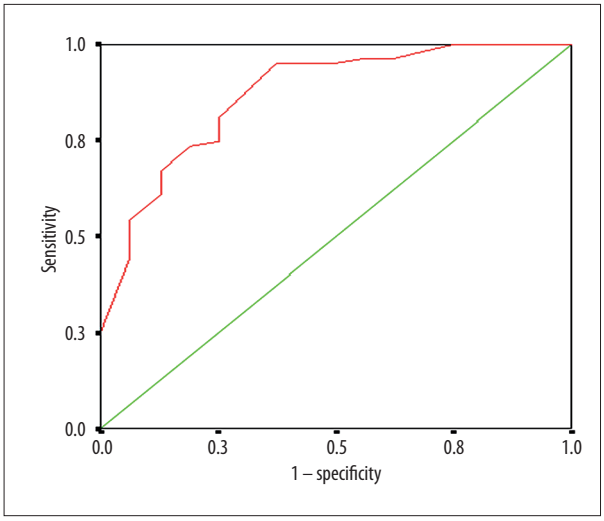

Figure 3. Receiver operating characteristic (ROC) curve analysis. The threshold value for predicting pulmonary hypertension at which the highest diagnostic sensitivity and specificity intersected was when the main pulmonary artery diameter (mPAD) was ≥29 mm. The area under the curve (AUC) was 0.86.

and significantly high pulmonary hypertension that they have frequently observed among women exposed to wood smoke [2]. Another autopsy study, which compared the chronic obstructive pulmonary disease cases caused by biomass smoke exposure and cigarette smoking, reported that vascular changes were prominent in both groups, but were more severe in the biomass smoke group, which could explain common pulmonary hypertension and cor pulmonale in women exposed to biomass smoke [13].

In our region, we frequently see female patients with symptoms suggestive of biomass smoke exposure. We investigated and compared the pulmonary hypertension prevalence between biomass smoke-exposed and non-exposed women who had no apparent diseases. We found a higher prevalence of pulmonary hypertension in the biomass smoke-exposed subjects (48% and 12%, respectively) [14].

Several studies have been conducted on the utility of the diameter of the main pulmonary artery and its main branches scanned by CT in the diagnosis of pulmonary hypertension [23–25]. These studies found an upper limit of normal mPAD of 33.2 mm, 28.6 mm, and 29 mm. They established that mPAD >29 mm and >33.2 mm have a sensitivity of 58% and 84%, and a specificity of 95% and 75%, respectively, for a pulmonary hypertension diagnosis. We found that biomass smoke-exposed subjects had increased mPAD compared with healthy subjects (p<0.001). We also found that mPAD ≥ 29 mm has a sensitivity of 91% and a specificity of 80% for a pulmonary hypertension diagnosis in the biomass smoke-exposed group.

Some studies have reported that CT can be used to differentiate the causes of pulmonary hypertension [26,27]. In our study, we evaluated pulmonary embolism, the presence of interstitial involvement, and aeration increase with CT, all of which can contribute to pulmonary hypertension.

Numerous studies have examined the correlation between mPAD measured by CT and mean PAP measured by right-sided heart catheterization with the purpose of assessing CT for pulmonary hypertension diagnosis in different diseases. A positive correlation was found between the 2 methods in studies of heart and lung diseases (r=0.83, p<0.001; r=0.74, p<0.0005; r=0.67, p<0.001), but no correlation was found in another study [24,30–32].

In thromboembolic pulmonary hypertension and primary pulmonary hypertension, 2 studies found a correlation between mPAD and mean PAP (r=0.43, p<0.01; r=0.42, p<0.001), but no correlation was found in a third study [32–34]. In contrast, we found a significant correlation between sPAP and mPAD in patients with chronic lung diseases, as well as in idiopathic pulmonary arterial hypertension-like group patients and asymptomatic idiopathic pulmonary arterial hypertension-like group patients, who may share similar pathogenesis with primary pulmonary hypertension and chronic thromboembolic pulmonary hypertension.

Burakowska et al. studied the correlation between pulmonary artery diameters measured by CT and echocardiographic sPAP in cases with acute pulmonary hypertension associated with pulmonary embolism and chronic pulmonary hypertension [35]. They reported that while the sPAP and mPAD correlation was significant in pulmonary hypertension cases developed in association with acute pulmonary embolism (r=0.487, p<0.003), this correlation was not observed in chronic pulmonary hypertension (r=0.223) [35].

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We observed a significant correlation between sPAP and mPAD in the biomass smoke-exposed group (r=0.634 p<0.01) and in the chronic obstructive pulmonary disease, pulmonary embolism, idiopathic pulmonary arterial hypertension-like, and asymptomatic idiopathic pulmonary arterial hypertension-like subgroups.

The results of most of these studies are in agreement with our findings and suggest that **CT** measurements may be used in a pre-diagnosis of pulmonary hypertension. ROC curve analysis showed that mPAD >29 mm provides the best sensitivity and specificity for detecting pulmonary hypertension by CT scan as reported in previous studies [25].

A linear correlation was established between CT mPAD and echocardiographic sPAP in the diseases related to biomass smoke exposure. The positive predictive rate of mPAD ≥29 mm was quite high. Therefore, CT mPAD should be used to evaluate the presence of pulmonary hypertension, which is common in biomass smoke-exposed women, and mPAD ≥29 mm should be a warning for the presence of pulmonary hypertension.

Conclusions

Our results indicate that main pulmonary artery diameter measurements by thoracic CT may suggest presence of pulmonary hypertension in biomass smoke-exposed women.

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