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HRCT evaluation of bronchial response to deep inspiration in asthma and chronic obstructive pulmonary disease (COPD)

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Prof. Adrian K Dixon
Chairman of the ESR Publications Committee

Vienna, January 2012



HRCT evaluation of bronchial response to deep inspiration in asthma and chronic obstructive pulmonary disease (COPD)

Poster No.: C-0779
Congress: ECR 2012
Type: Scientific Exhibit
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Keywords: Lung, Respiratory system, Thorax, CT-High Resolution, Computer Applications-Detection, diagnosis, Chronic obstructive airways disease
DOI: 10.1594/ecr2012/C-0779

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Purpose

In asthma and chronic obstructive pulmonary disease (COPD) functional disorder may be defined not only in terms of bronchial obstruction, but also by airway response to stimuli including deep inhalation (DI). DIs exert their effects on airways through the radial traction that is applied to the airway wall by virtue of the interdependence between intraparenchymal airways and the surrounding parenchyma. DI effect decreases with severity of diseases and correlates with changes in airway distensibility (AD) evaluated by HRCT. Our study aimed at assessing the variables that affect such AD.

Methods and Materials

We studied twelve asthmatics (M/F: 7/5) and eight COPD patients (7/1) with similar degree of obstruction. None of the study subjects were current smokers. None of them had acute exacerbation within the previous three months, and all of them were clinically stable at the time of the study. The study was performed on two days. The first visit included clinical and functional assessments. On a separate occasion, within seven days, the imaging assessment was carried out. Each subject underwent HRCT evaluation, which was performed by spiral computed tomography (multidetector 64 channels, Philips Medical System, Cleveland, OH), with a setting of 120 kVp, 200 mAs, a 0.9-mm slice thickness, a rotation time 0.5s, and a reconstruction interval of 0.45 mm, pitch 0.923, during a single breath and moved caudally. Images were reconstructed at a window level of - 600 Hounsfield units (HU) and a window width of 1600 HU. Two sets of scans were obtained: the first one was acquired at the end of the expiratory phase of the tidal volume (functional residual capacity, FRC) and the second one at maximal lung inspiration (total lung capacity, TLC). The airway diameter, airway wall thicknesses (as a fraction of airway diameter; AWF) and lung parenchyma density were analyzed using the validated Pulmonary Workstation 2 software package (VIDA Diagnostics, Inc, Iowa City, IA) (Fig. 1-2). AD was calculated as the ratio of the difference in bronchial lumen diameter at TLC and FRC over the diameter at FRC.

Images for this section:



Fig. 1: Images relevant to phases of post processing procedure of lung CT scans.

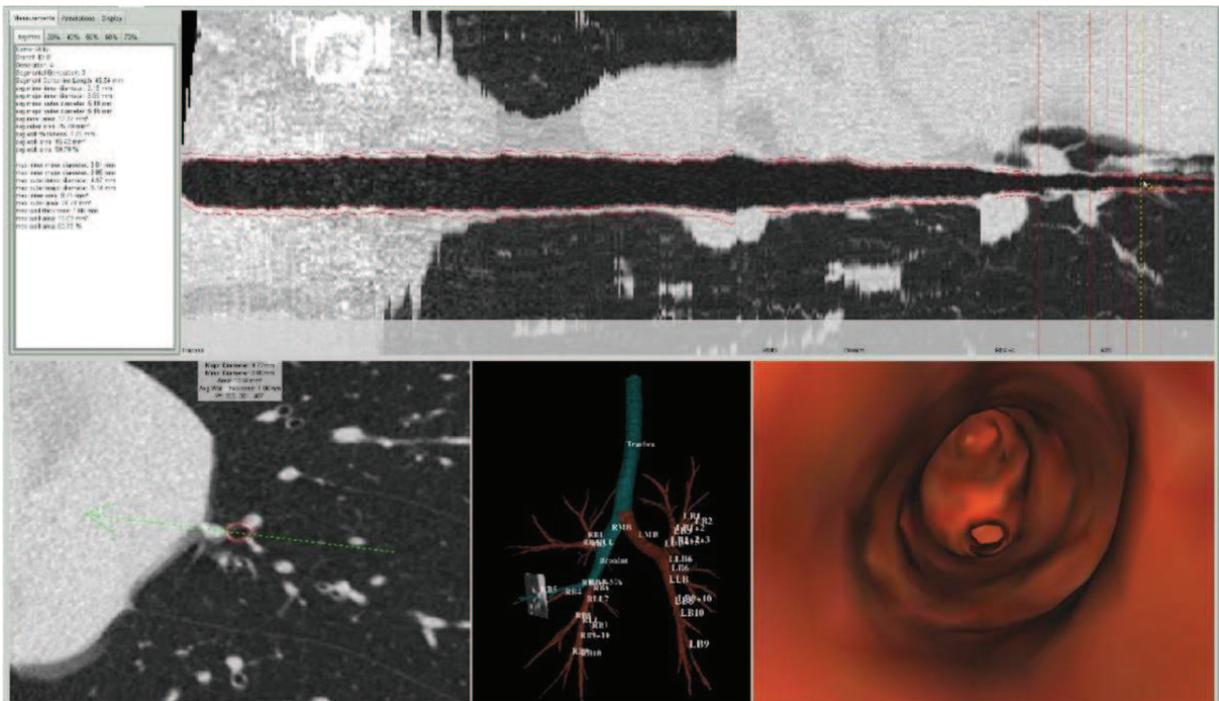


Fig. 2: Further phases of post-processing of pulmonary airways leading to virtual bronchoscopy.

Results

We evaluated 701 airways. The degree of obstruction did not differ between asthma and COPD (FEV 1% pred 69.0 +/- 4.8 vs 61.3 +/- 5.9 p= 0.31). AD did not differ between groups (14 +/- 3.5 vs 17 +/- 4.3 p=0.58). No significant differences between the two groups were detected when AD was calculated by size for the small (p=0.27), the medium (p=0.94) and the large (p=0.40) airways, respectively. We did not find a significant difference in the mean AWF between the groups (at TLC: 0.52±0.01% in the asthmatics and 0.51±0.01% in the COPD, p=0.50; at FRC: 0.55±0.01% in the asthmatics and 0.55±0.01% in the COPD, p=0.94). In asthma AD was correlated to FEV1 % pred (r²= 0.45, p=0.018) (Fig.3), while in COPD it correlated with residual volume (RV) % pred (r²= 0.51, p= 0.046) (Fig.4) and with RV/TLC% (r²= 0.68, p=0.01) (Fig.5).

Images for this section:

Asthmatics

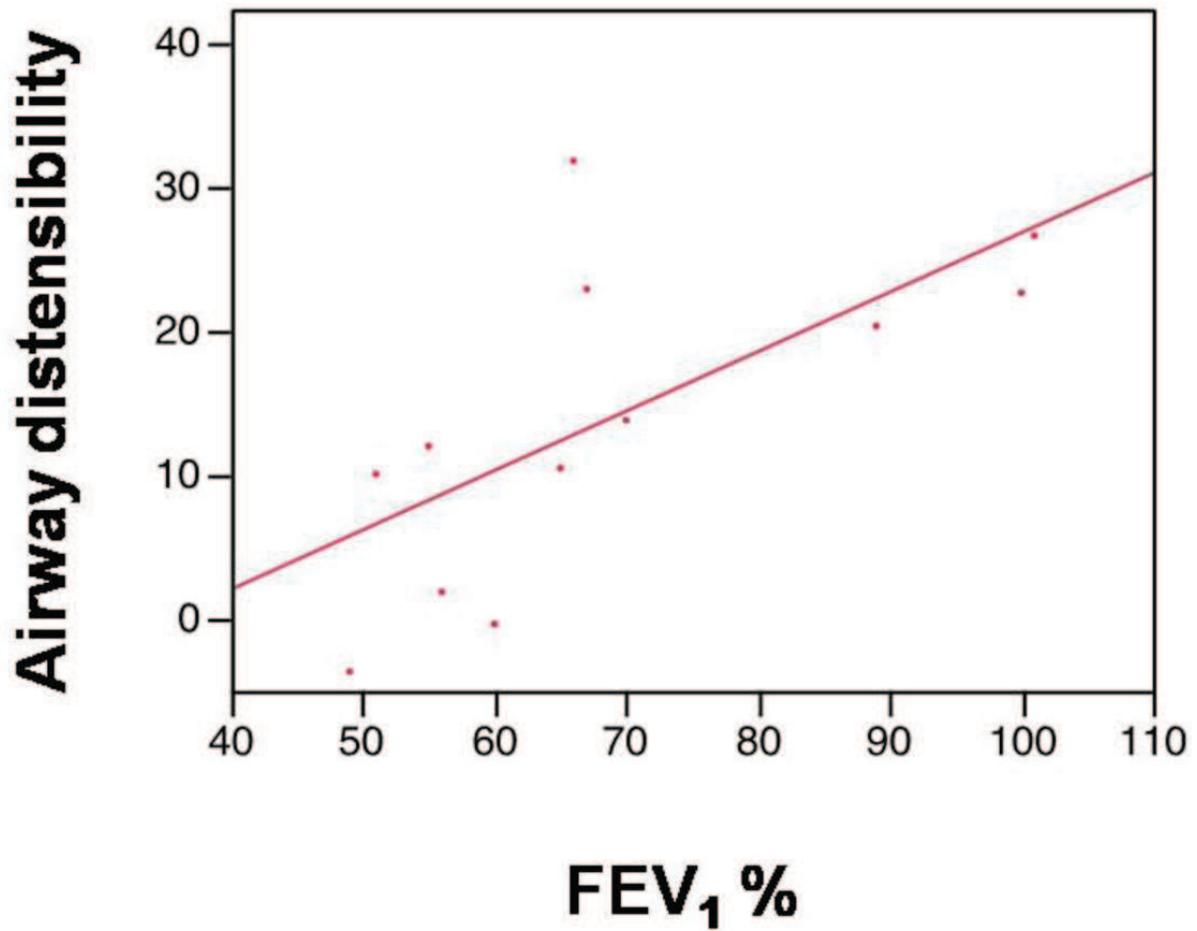


Fig. 3: In asthma AD was correlated to FEV₁ % pred ($r^2= 0.45$, $p=0.018$)

COPD

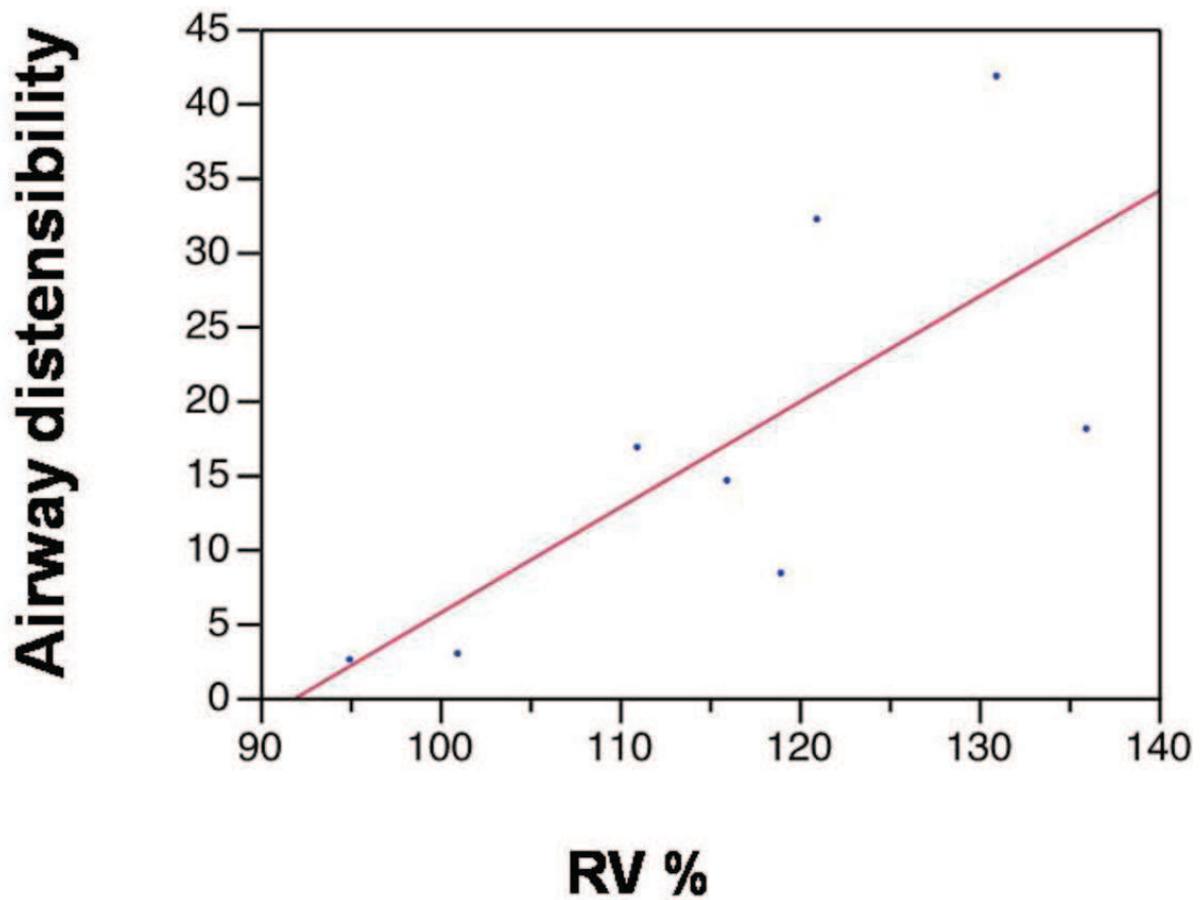


Fig. 4: In COPD AD is correlated with residual volume (RV) % pred ($r^2 = 0.51$, $p = 0.046$).

COPD

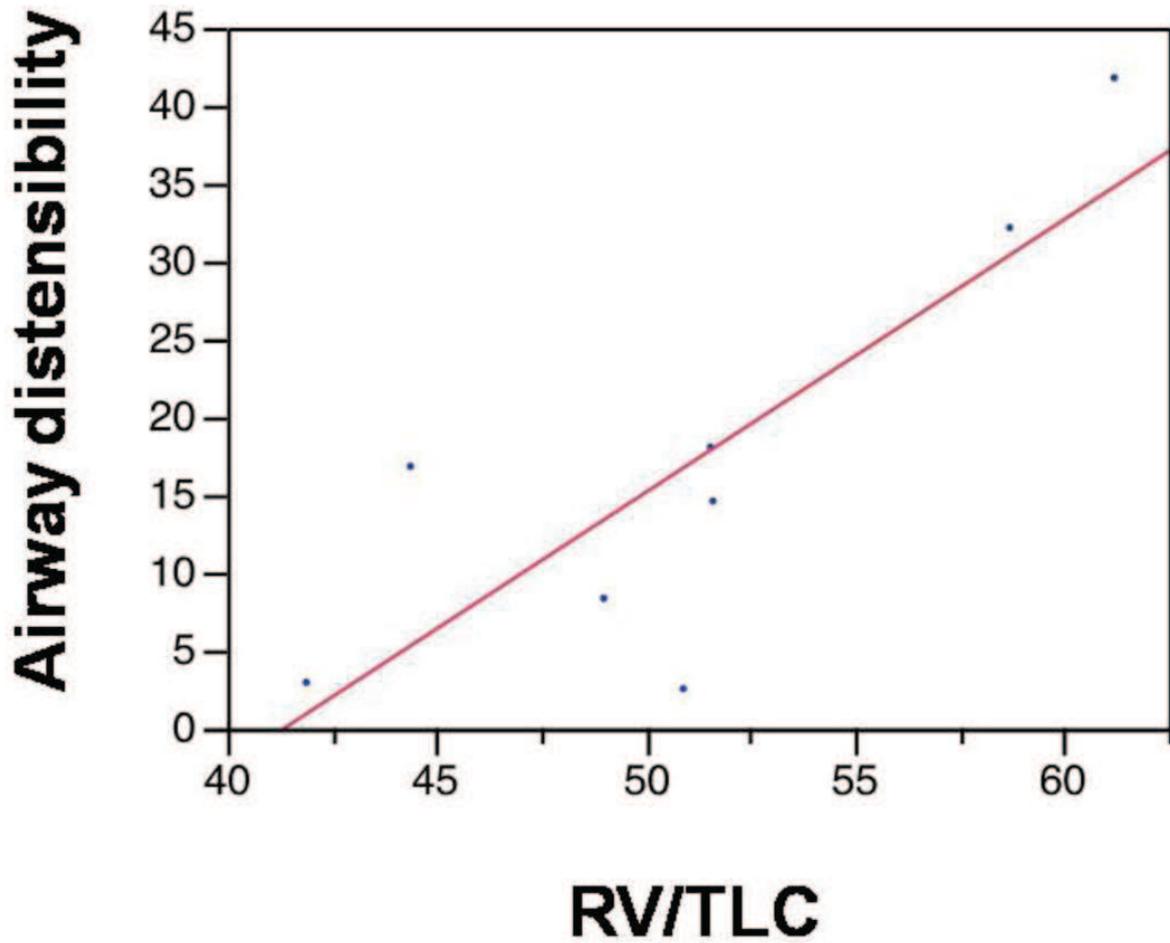


Fig. 5: In COPD AD is correlated with RV/TLC% ($r^2= 0.68$, $p=0.01$).

Conclusion

Our study demonstrated that, for similar level of airway obstruction the degree of AD induced by DI does not differ between asthma and COPD. The different correlation of AD with functional parameters may be interpreted as due to different pathogenetics mechanisms (bronchial obstruction and air trapping respectively) that influence bronchodilator response to DI in the two diseases.

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