Title: How does CT pulmonary vascular structure relate to airways, emphysema and COPD disease severity?

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Structured Abstract:

INTRODUCTION: Chronic obstructive pulmonary disease (COPD) is a chronic and debilitating disease affecting >10% of Canadians over the age of 35. Ultimately, inflammation, remodeling and destruction of the airways and parenchyma also effects the pulmonary vascular system. Arterial wall remodeling and loss of diffusing capacity due to emphysema leads to chronic pulmonary hypertension low blood oxygen saturation and right heart failure; however, the pathophysiology of these relationships is poorly understood. Airway destruction is shown to be driven by changes to the small pulmonary vasculature. Recent work has demonstrated pruning of the small vessels of the lungs using computed tomography (CT). Accordingly, the objective of this study was to investigate relationships between CT pulmonary vessel and airway structure, emphysema and pulmonary function testing. We hypothesize that participants with COPD have significantly reduced pulmonary blood vessel volume compared to ex-smokers without COPD, and related to disease severity, emphysema and airway remodeling.

METHODS: Participants provided written informed consent to an ethics-board approved protocol and underwent thoracic CT and pulmonary function testing. CT analysis was performed using VIDA Vision software (VIDA Diagnostics Inc., Coralville, IA). Pulmonary vessels were automatically segmented from CT and total blood vessel volume (TBV) was calculated as the volume of the vascular tree. Emphysema was quantified as the area of the lung less than -950 HU (RA950). Univariate relationships were evaluated using linear regression and group differences were tested using ANOVA; covariates of age, sex, BMI and thoracic cavity volume (TCV) were included in models as necessary (SPSS 25, Armonk, NY).

RESULTS: We evaluated 86 participants including 40 ex-smokers without COPD (73±10yrs) and 46 participants with COPD (74±11yrs). TBV was significantly related to FEV1/FVC (forced expiratory volume in 1 second divided by forced vital capacity) (r2=.20, p<.01). Ex-smokers with and without COPD had significantly different FEV1 (67±25 and 103±19%pred;p<.0001), FEV1/FVC (51±13 and 79±6%;p<.0001), RA950 (12±12 and 2±4%;p<.0001) and airway lumen area (LA) (9±2 and 11±4mm2;p<.05). Further analyses of ex-smokers compared to GOLD 1, 2, and 3+4 sub-categories of COPD demonstrate a significant main effect of LA (p<.05) and TBV (p<.0001) between groups when controlling for TCV.

DISCUSSION: In a cross-sectional sample of participants with COPD and ex-smokers without COPD, pulmonary vascular volume measured using CT was related to both airway and parenchymal CT measurements and pulmonary function measurements, and significantly related to COPD severity. This provides evidence of the complex and important pathophysiological interactions between the airways, vessels and parenchyma in COPD. Future work will include a longitudinal investigation of these relationships in COPD in order to elucidate phenotypes of disease progression.