



Ex-smokers with and without COPD: Investigating CT Pulmonary Vascular, Airway, Pulmonary Artery and Aorta Measurements



V Desai^{1,2}, PV Wyszkiwicz^{1,2}, AM Matheson^{1,2}, M Sharma^{1,2}, MJ McIntosh^{1,2}, HK Kooner^{1,2}, DG McCormack³ and G Parraga¹⁻⁴

Western University, Canada
¹Robarts Research Institute; ²Department of Medical Biophysics, ³Division of Respiriology, Department of Medicine, ⁴School of Biomedical Engineering, Western University, London, Canada

Introduction

- Pulmonary hypertension is characterized by increased pressure in the pulmonary artery¹
- It is a key contributor to worsening symptoms in individuals with chronic obstructive pulmonary disease (COPD)²
- The pulmonary artery to aorta diameter ratio (PA:Ao) is measured using x-ray computed-tomography (CT)³
- PA:Ao is an important biomarker for pulmonary hypertension²
- Longitudinal changes in this measurement and its relationship to pulmonary structure and airway structural changes is not well understood

Objective

To investigate longitudinal changes in PA:Ao and its relationship with CT pulmonary vascular changes, airway abnormalities, airflow limitation and exercise-capacity

Methods

- Ex-smokers with and without COPD (n=94) provided written, informed consent to an approved study protocol (NCT00279329)
- Participants completed CT, pulmonary function tests, quality-of-life questionnaires and 6-minute-walk-distance (6MWD) at baseline and follow-up (2.6 ± 0.6 years)
- PA:Ao measured using custom-built software (Shift-64 Workstation)⁴
- CT analyzed using Chest Imaging Platform (Brigham and Women's Hospital) to generate total blood volume (TBV) and the blood volume in vessels less than 5 mm² in cross-sectional-area (BV₅)
- VIDAvision used to generate airway wall area (WA) and wall thickness percent (WT%)
- Pearson or spearman correlations used to evaluate relationships between imaging and pulmonary function measurements

Results

Table 1. Demographic characteristics, pulmonary function, exercise-capacity and imaging measurements in ex-smokers with and without COPD at baseline and follow-up

Parameter Mean ± SD	Baseline (n=94)	Follow-Up (n=94)	p
Age (years)	70 ± 9	72 ± 9	-
Female n (%)	33 (35)	33 (35)	-
BMI (kg/m ²)	28 ± 4	28 ± 2	.2
FEV ₁ % _{pred}	83 ± 26	83 ± 29	.9
6MWD (m)	412 ± 76	395 ± 84	.004
PA:Ao	0.75 ± 0.10	0.74 ± 0.11	.23
TBV (mL)	260 ± 54	256 ± 51	.3
BV ₅ /TBV	0.44 ± 0.08	0.42 ± 0.07	.02
WT%	18.4 ± 0.8	18.2 ± 0.8	.04
WA (mm ²)	66.6 ± 1.9	66.3 ± 1.8	.03

Definition of abbreviations: BMI = Body mass index; FEV₁ = Forced expiratory volume in 1 second; %_{pred} = percent of predicted value; 6MWD = 6-minute-walk-distance; PA:Ao = Pulmonary artery to aorta diameter ratio; TBV = Total blood volume in pulmonary vasculature; BV₅ = Blood volume in vessels less than 5mm² cross-sectional area; WT% = Airway wall thickness percent; WA = Airway wall area; bolded values are statistically significant

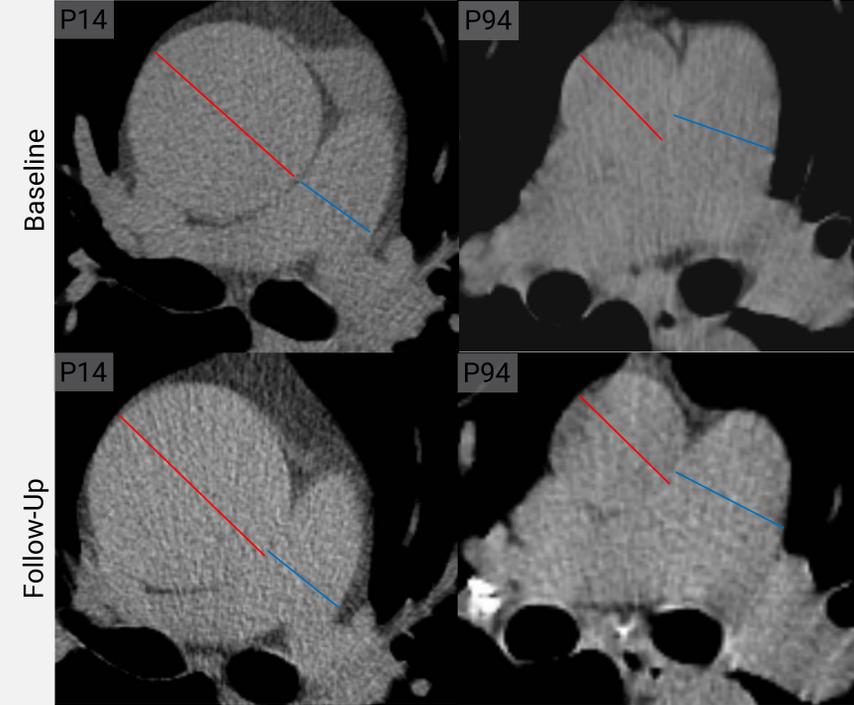


Figure 1. Measurements of PA:Ao acquired using CT in ex-smokers with and without COPD at baseline and follow-up. Red line denotes aorta diameter and blue line denotes pulmonary artery diameter.

P14: Male, 78 years old
Baseline: BMI = 31 kg/m²; FEV₁ = 140 %_{pred}; 6MWD = 456 m; PA:Ao = 0.44
Follow-up: BMI = 34 kg/m²; FEV₁ = 152 %_{pred}; 6MWD = 480 m; PA:Ao = 0.36

P94: Female, 58 years old
Baseline: BMI = 21 kg/m²; FEV₁ = 32 %_{pred}; 6MWD = 371 m; PA:Ao = 0.78
Follow-up: BMI = 19 kg/m²; FEV₁ = 32 %_{pred}; 6MWD = 342 m; PA:Ao = 0.86

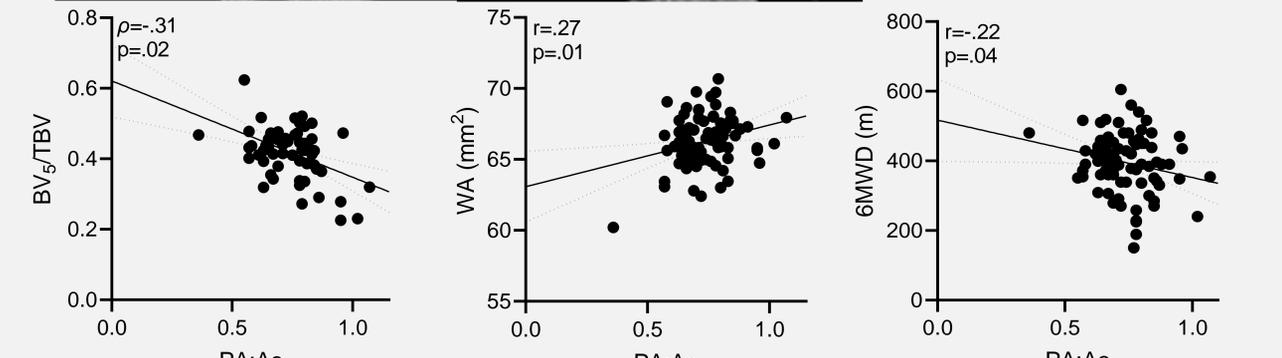


Figure 2. Correlations for PA:Ao with pulmonary blood volume measurements, airway wall measurements and exercise-capacity at follow-up

Discussion

- PA:Ao and TBV did not change longitudinally after 3 years, although BV₅/TBV (p=.02) was reduced
- This may indicate small vessel remodelling in the pulmonary vasculature
- Exercise-capacity was significantly decreased at follow-up (p=.004)
- Increased PA:Ao was correlated with greater airway wall thickening at baseline (r=.25, p=.02) and follow-up (r=.27, p=.01) and reduced small vessel blood volume at both baseline (rho=-.32, p=.01) and follow-up (rho=-.31, p=.02)
- Together, these detected airway and pulmonary vascular abnormalities may indicate blood redistribution from small vessels
- PA:Ao may serve as a key biomarker for the underlying pathophysiology that explains worsening symptoms in ex-smokers with and without COPD over time

Future Work

- To compare PA:Ao with CT pulmonary vascular changes and airway structural changes across increasing COPD severities

Conclusions

Airway and pulmonary vascular abnormalities may indicate blood redistribution from small vessels, which is an important factor to consider for subclinical pulmonary hypertension.

References

1. Chaouat *et al.* EurRespirJ (2008).
2. Kovacs *et al.* AmJRespirCritCareMed (2020).
3. Wells *et al.* NEJM (2012).
4. Iyer *et al.* Chest (2014).

Acknowledgments

CONTACT: vdesaigo@uwo.ca, gparraga@robarts.ca