

NS Gale¹, M Munnery², A Albarrati¹, DJ Shale¹, JR Cockcroft².

1) School of Healthcare Sciences, Cardiff University, UK, CF14 4XN 2) School of Health Sciences Cardiff Metropolitan University

BACKGROUND

Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory multisystem disease. In addition to progressive loss of lung function, patients experience increased risk of cardiovascular (CV) disease, metabolic abnormalities, skeletal muscle dysfunction and osteoporosis. Patients with COPD and a low BMI have poorer health outcomes¹, while obesity may increase CV risk.²

Cross-sectional studies in COPD have identified increased arterial stiffness as measured by aortic pulse wave velocity (aPWV)³. Aortic PWV is an independent predictor of CV events, however, its use as a prognostic CV risk predictor in COPD is at present unknown.

AIM

► The aim of this analysis was to explore BMI, CV risk (aPWV), systemic inflammation and exercise capacity in COPD.

METHOD

Patients with COPD (n=524) (confirmed with spirometry) and 143 comparator subjects (current or ex-smokers free from respiratory disease) were recruited from the ARCADE (Assessment of Risk in Chronic Airways Disease Evaluation) study. Assessments included lung function (forced expiratory volume in 1 second (FEV₁)), smoking history, BMI, aPWV (SphygmoCor device), blood pressure (BP), 6-minute walking distance (6MWD). Inflammation was measured by high sensitivity C-reactive protein (HsCRP) and fibrinogen. Patients were classified by BMI as follows: low (<19.9 kg/m²), healthy (20-24.9kg/m²), overweight (25-29.9kg/m²), obese (>30kg/m²).

RESULTS

There was no difference in gender, age, lung function or smoking history between patients grouped according to BMI. However, there was a difference in PWV, systolic BP, 6MWD and inflammation between the groups (p<0.05). The difference in PWV remained after adjustment for age and mean BP (Table 1). Overweight and obese patients (BMI <25) had greater PWV and inflammation, while obese patients had the poorest 6MWD.

In COPD, BMI related to aPWV r=0.178, 6MWD r=-0.264, CRP rs=0.276 and Fibrinogen rs=0.115, (all p<0.05), but not age, lung function, mean BP or smoking. A stepwise linear regression revealed that aPWV was predicted by age, mean BP and BMI in COPD R²=0.264 (p<0.01).

Table 1 Anthropometric and haemodynamic data in patients with COPD across BMI categories and comparators compared to combined data for patients with COPD

Table 1	BMI<19.9 n=29	BMI 20-24.9 n=121	BMI 25-29.9 n=201	BMI >30 n=173	p	Comparator n=143	p
Male: female†	12:17	61:30	106:95	93:80	0.640	70:73	0.531
Age (years)	62.2 ± 7.8	65.8 ± 7.7	66.8 ± 7.4	66.3 ± 7.5	0.023	64.8 ± 7.5	0.059
FEV ₁ (L)	1.35 ± 0.64	1.31 ± 0.57	1.46 ± 0.61	1.47 ± 0.59	0.073	2.72 ± 0.67	<0.001
FEV ₁ % predicted	56 ± 22	55 ± 19	60 ± 19	59 ± 18	0.137	105 ± 14	<0.001
Smoking (Pack Years)	33.3 ± 14.9	39.8 ± 23.5	39.2 ± 23.5	43.8 ± 29.4	0.115	21.1 ± 17.2	<0.001
Aortic PWV (m/s)	8.9 ± 2.0	9.5 ± 2.2	10.2 ± 2.61	10.1 ± 2.3	<0.001	8.4 ± 1.8	<0.001
Aortic PWV adj (m/s)	9.4 ± 2.2	9.5 ± 2.2	10.2 ± 2.2	10.1 ± 2.2	<0.001	68 ± 10	<0.001
Systolic BP (mmHg)	144 ± 21	144 ± 19	149 ± 19	144 ± 17.4	0.079	140 ± 18	<0.001
Diastolic BP(mmHg)	82 ± 12	81 ± 10	82 ± 10	83 ± 11	0.604	81 ± 9	0.190
Mean BP (mmHg)	99 ± 16	97 ± 12	98 ± 11	98 ± 12	0.721	95 ± 11	0.003
6MWD (m)	344 ± 104	362 ± 110	353 ± 123	291 ± 1306	0.004	503 ± 86	<0.001
HsCRP (mg/L) #	1.4 (0.6-2.9)	2.3 (1.3-5.1)	3.6 (1.8-7.7)	4.6 (2.4-7.8)	<0.001	1.6 (0.8-3.8)	0.006
Fibrinogen (g/L)#	3.0 (2.5-3.5)	3.4 (2.8-4.1)	3.5 (2.9-4.1)	3.5 (3.0-4.2)	0.012	3.0 (2.6-3.7)	0.103

Data are mean SD or median (interquartile range), ANOVA, † chi-square or # Kruskal-Wallis test for difference

CONCLUSION

► The findings suggest obese patients with COPD have greater CV risk which may be a cause or consequence of poorer physical capacity and greater inflammation. Optimisation of BMI in COPD may improve outcomes.

► ARCADE is the first longitudinal study of CV risk factors and other systemic manifestations in patients with COPD. Further follow-up will evaluate the prognostic utility of haemodynamic measurements

REFERENCES

- 1) Landbo, et al. Am JRespirCrit Care Med 1999;160:1856-61.
- 2) Mannino, et al. Eur Respir J. 2008;32(4):962-969.
- 3) Sabit, et al. Am J Resp Crit Care Med 2007 175:1259-65

ACKNOWLEDGEMENTS

Thanks to all our volunteers and GlaxoSmithKline who supported the study.