P20 A DATABASE APPROACH TO DOSE SCORE CALCULATION AS A TOOL TO IDENTIFY 'AT RISK' CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS THROUGH CLINICAL RECORDS

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Establishing how best to target resources remains a challenge within COPD as this is a heterogeneous patient group with complex needs often poorly reflected by routinely collected clinical measurements such as FEV_1.

Jones *et al*. created The DOSE score (dyspnoea (MRC score), obstruction (FEV_1 percentage predicted), smoking status and exacerbation number in a year) (Table 1) a validated, clinically useful measure of risk stratification in COPD which utilises data already routinely collected in Primary Care for QOF review.

Abstract P20 Table 1 DOSE INDEX SCORING SYSTEM (Jones *et al*. AJRCCM 2009;180(12):1189–95): The DOSE Index points associated with every category of all four variables are added to build the DOSE Index score

	DOSE Index Points			
	0	1	2	3
MRC Dyspnoea Scale Score	0-1	2	3	4
FEV$_1$% predicted	>50	30-49	<30	
Smoking status	Non smoker	Smoker		
Exacerbations per year	0-1	2-3	>3	

By using a collaborative approach with informatics, statistical and clinical input we developed a database approach to calculating a DOSE score using routinely collected and coded Primary and Secondary Care data. A local NHS database holding anonymised clinical records for over one million patients was used to identify a cohort of over 13,000 patients with codes diagnostic of COPD.

Microsoft Structured Query Language Server was used to identify, cleanse and standardise the required clinical information and calculate the DOSE score, creating a series of functions that can be replicated across other database management systems.

Date of FEV_1 percentage predicted was taken as the index date for DOSE score calculation. Where only FEV_1 was recorded, a percentage of predicted FEV_1 was calculated using available height and age data.

Read codes (the routine coding system used in primary care) and ICD-10 codes were used to compile lists identifying those symptoms, diagnoses and prescriptions indicative of COPD exacerbations. These lists were applied in the year prior to the chosen FEV_1 value and, functions were written to cluster those events felt to be reflective of a single exacerbation.

Read codes reflecting MRC score and smoking status closest in time to the index FEV_1 measurement were combined with the above measurements, generating a complete score in approximately 10,000 patients. Partial scores were created for a further 1500 patients with incomplete data for the individual score components.

This approach provides a simple way for clinicians to risk stratify their COPD population without increasing their clinical workload. This gives an opportunity to identify those at highest risk of hospital admission and death and proactively allocate resources accordingly.

P21 THE APPLICABILITY OF CURRENT CARDIOVASCULAR RISK SCORES AND CARDIOVASCULAR SURROGATES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A CASE-CONTROL STUDY

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Background COPD is a complex multi-morbid disorder with significant cardiac mortality. Despite this, current cardiovascular scoring systems do not include COPD in their risk prediction models. The aims of this study were to assess whether differences in cardiovascular surrogate markers exist in COPD and to further our understanding of the relationship of COPD to cardiovascular structure and function.

Methods This post-hoc cross-sectional analysis utilised baseline data from two randomised controlled trials (n = 36 and 54). 26 COPD patients were matched for global cardiovascular risk with 26 controls with normal lung function using QRISK2, a validated scoring system for predicting the 10-year risk of cardiovascular disease in a United Kingdom population. Patients underwent cardiac magnetic resonance imaging, arterial stiffness and lung function measurements.

Results Pulse wave velocity (PWV) (mean difference +1.0 m/s, 95% CI 0.02–1.92; p = 0.045) and total arterial compliance (TAC) (mean difference -0.27 mL/m^2/mmHg, 95% CI -0.39, -0.15; p < 0.001) were adversely affected in COPD compared to the control group matched for cardiovascular risk. In the whole cohort (n = 90) QRISK2 (β = 0.046, p = 0.017) and FEV1 (β = -0.013, p = 0.022) were associated with PWV in multivariate analysis. The relationship between QRISK2 and PWV appeared to be modified by COPD, where the interaction term reached borderline significance (p = 0.060). FEV1 (β = 0.005, p = 0.004) was also associated with TAC in multivariate analysis. Cardiac chamber size and stroke volume was decreased in COPD compared to controls. The mean difference in left ventricle stroke volume index (LVSVI) and left and right end diastolic volume index was -10.3 ml/m^2 (95% CI -15.1, -5.5, p < 0.001), -14.1 ml/m^2 (95% CI -21.9,-6.3 p < 0.001) and -13.0 ml/m^2 (95% CI -23.3, -2.6 P < 0.015) respectively, which were shown to be associated with airflow limitation in multivariate models. In the COPD group associations were found with lung hyperinflation (LVSVI: β = -0.075, p = 0.032; Left atrial size: β = -0.129, p = 0.047) and fibrinogen (TAC: β = 0.716, p = 0.030).

Conclusion Surrogates for cardiovascular outcomes are adversely affected in COPD compared to a group matched for global cardiovascular risk, suggesting that current scoring systems may be suboptimal for cardiovascular risk prediction in COPD.