

Poster sessions

AUC for 30 day mortality for the acute score was 0.84 (95%CI 0.80–0.88).

The strongest predictors of 1 year mortality were age >80 years HR 1.25 (95%CI 1.00–1.64)–1 point, neoplastic disease 1.91 (95%CI 1.37–2.65)–1 point, MRC dyspnoea grade 4 or 5 (3.82 95%CI 2.61–5.58)–2 points, FEV₁ <30% predicted 2.61 (95%CI 1.99–3.43)–1 point, long term oxygen therapy 1.60 95%CI 1.11–2.29)–1 point and a history of myocardial infarction or heart failure 2.76 95%CI 1.70–4.47)–1 point. The AUC for 1 year mortality of the chronic score was 0.76 (95%CI 0.73–0.80).

The resulting 4 stage model identifies different outcomes within each subgroup (See Figure 1). Furthermore the 4 stage model predicted 30-day mortality AUC 0.76 (95%CI 0.72–0.79), 1 year mortality 0.72 (95%CI 0.70–0.74) and readmissions 0.74 (95%CI 0.72–0.76) better than GOLD 2011 criteria.

Conclusion A multidimensional prognostic index can predict both short and long term outcomes after COPD exacerbations, and divides patients into clinically useful subgroups based on exacerbation severity and chronic health status.

P137 INTERNAL VALIDATION OF THE DECAF SCORE AS A PREDICTOR OF INPATIENT MORTALITY IN ACUTE EXACERBATIONS OF COPD

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Introduction In patients presenting with an acute exacerbation of COPD (AECOPD), accurate prediction of in-hospital mortality may help inform the most appropriate place and level of care. The DECAF score was developed for this purpose and designed to be simple to apply at the bedside using variables that are routinely collected on admission: Dyspnoea, Eosinopenia, Consolidation, Acidaemia and atrial Fibrillation. Whilst the performance of the tool within the derivation cohort was strong,^[1] before recommending use in clinical practice further validation is required.

Methods Both external and internal validation of the DECAF score are currently in progress; for each, a cohort of 840 patients consecutively admitted with AECOPD is being recruited. To optimise data capture, patients are identified by screening admissions units and coding records. Indices that comprise the DECAF score and other independent predictors of mortality and readmission are collected. Dyspnoea is assessed using the extended MRC dyspnoea score.^[1] Inclusion criteria are: primary diagnosis of AECOPD, age 35 or older, 10 pack years or more smoking history, and obstructive spirometry. Exclusion criteria are: other illness likely to limit survival to less than one year (principally metastatic malignancy) and previous inclusion in the validation study. We present an analysis of the performance of DECAF in the first 623 patients recruited to the internal validation cohort.

Results

Abstract P137 Table 1.

DECAF AUROC = 0.82 (95% 0.76 to 0.87)

Derivation DECAF AUROC = 0.86 (95% CI 0.82 to 0.89)

p value using Fisher's exact test Internal validation DECAF AUROC = 0.82 (95% 0.76 to 0.87) Derivation DECAF AUROC = 0.86 (95% CI 0.82 to 0.89)

Discussion As in the derivation study, DECAF is a good predictor of inpatient mortality (AUROC = 0.82), with a stepwise

increase in mortality with DECAF score. The DECAF score accurately identifies low risk (DECAF score 0–1) and high risk patients (3 or greater) admitted with an exacerbation of COPD, potentially helping select patients for early supported discharge schemes, or for intensified medical treatment or early palliation.

REFERENCES

1. Steer, J., J. Gibson, and S. C. Bourke, The DECAF Score: predicting hospital mortality in exacerbations of chronic obstructive pulmonary disease. *Thorax*, 2012. 67(11): p. 970–6.

P138 MODERATE-INTENSITY ACTIVITY IS ASSOCIATED WITH REDUCED CARDIOVASCULAR RISK FACTORS IN COPD

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Background Steps measurement is a common measure of daily physical activity, but cannot define the intensity of physical activity. Moderate intensity activity is recommended type to maintain a basic level of health (1), but in patients with COPD, the effect of moderate intensity activity on systemic manifestations has not been studied. We hypothesised that patients with higher moderate activity time would have lower frailty, inflammation and cardiovascular risk.

Methods As part of a longitudinal study in COPD (ARCADE), daily physical activity was recorded over seven days using a multi-sensor armband (SenseWear Pro armband) in 75 stable patients with COPD. Spirometry, body composition, aortic stiffness, comprehensive geriatric assessment, C-reactive protein (CRP) and fibrinogen were also determined. Moderate-intensity activity was determined by the monitor for activity between 3 to 6 METs.

Results Patients (42 males) mean (SD) age was 66 (7) years, BMI 27.5 (5.2) Kg/m², FEV₁ predicted 55 (17)% and moderate activity time 1.46 (1.23) hours. The time spent on moderate activities was not related to age or FEV₁pred. The moderate activity time related to BMI (r = -0.41), fat mass (r = -0.38) and fat percentage (r = -0.37), all p < 0.001, but not with fat free mass. However, none of these parameters related to the number of steps. Moderate activity time was inversely associated with aortic stiffness, r = -0.31, p < 0.01, CRP, fibrinogen (r = -0.26; r = -0.24, respectively) and frailty score all p < 0.05. However, the number of steps only related to the inflammatory markers.

Conclusion The changes in frailty and cardiovascular risk factors including adipose tissue, aortic stiffness and inflammatory markers are linked to the proportion of time in moderate activities as their predominant form of activity.

REFERENCES

1. Elsayy, B and Higgins, K. (2010) *Am Fam Physician* 1;81(1):55–59.

Mechanisms of lung injury

P139 PULMONARY SURFACTANT PROTECTS AGAINST SILVER NANOPARTICLE-INDUCED INFLAMMATION IN THE PERIPHERAL HUMAN LUNG

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