showed subjective deterioration in symptom trajectory 4 days prior to exacerbation onset ($p < 0.01$), with re-exacerbators demonstrating a higher baseline symptom burden in the post-treatment period compared to single exacerbators ($p < 0.01$).

In conclusion, salivary biomarker levels can complement patient self-assessment to provide clinically useful cues to enable earlier identification of exacerbations in COPD; salivary CRP potentially offers additional information on re-exacerbation risk. These results support opportunities for patient-reported events and salivary biomarkers to be used synergistically in future near-patient COPD diagnostics for enhanced self-management and prompt exacerbation intervention.

S67 MORTALITY IN COPD PATIENTS FOLLOWING COMMUNITY ACQUIRED PNEUMONIA: A POPULATION DATABASE ANALYSIS OF LINKED HEALTHCARE RECORDS

[1]N Williams, [2]NA Coombs, [3]M Johnson, [2]L Josephs, [4]LA Rigge, [2]DM Thomas, [5]TMA Wilkinson. *[1]Southampton NIHR Biomedical Research Unit, University Hospital Southampton NHS Foundation Trust, Southampton, UK; [2]Primary Care and Population Sciences, University of Southampton, Southampton, UK; [3]NIHR CLAHRC Wessex Methodological Hub, University of Southampton, Southampton, UK; [4]Department of Respiratory Medicine, University Hospital Southampton NHS Foundation Trust, Southampton, UK; [5]Clinical & Experimental Sciences, University of Southampton, Southampton, UK*

10.1136/thoraxjnl-2016-209333.73

Introduction Community acquired pneumonia (CAP) is a common occurrence in patients with chronic obstructive pulmonary disease (COPD), yet controversy still remains about its affect on outcome. We therefore investigated the impact of CAP on mortality in a cohort of COPD patients identified from the Hampshire Health Record analytical database, a local NHS database containing linked, anonymised primary and secondary care records.

Methods Patients were defined as having COPD if they had a diagnostic Read code in their primary care record at any time prior to the 1st January 2010 and were aged ≥40 years at the start of the study. CAP episodes occurring over a 5-year period from 1st January 2010 were identified using Read and ICD-10 codes. The outcome measure was all-cause mortality following a CAP diagnosis. Cox proportional hazard modelling was used to estimate hazard ratios (HR) and confidence intervals (CI), adjusting for age, sex, GOLD stage, smoking status and inhaled corticosteroid use (ICS)).

Results The cohort comprised 14506 COPD patients. The mean age was 70.3 (±10.8) years and 53.6% were male. 1931 (13.3%) patients had at least one CAP and 2870 (19.8%) deaths occurred over the study period. 28.2% of patients diagnosed with CAP died compared to only 9.7% of those without a CAP diagnosis ($p < 0.001$). Logistic regression analysis, controlling for potential confounders identified CAP as an independent risk factor for future mortality (odds ratio 2.72; CI 2.37–3.12, $p < 0.001$). Compared to younger individuals (40–59 years) those aged 60–79 and >80 years had the highest mortality risk following CAP (HR 2.65; CI 1.61–4.34, HR 7.03; CI 4.27–11.57 respectively, both $p < 0.001$). Concurrent use of inhaled Fluticasone or Budesonide were associated with reduced mortality risk following CAP (HR 0.82; CI: 0.68–0.98 $p = 0.029$, HR 0.55; CI: 0.39–0.76 $p < 0.001$, respectively) (Figure 1).

Conclusion CAP in COPD is associated with increased risk of mortality, especially in older individuals. Although known to increase CAP risk, ICS use appears to reduce risk of mortality following CAP. Further research to understand the mechanisms underlying CAP risk in COPD and modulating effects of ICS is key, to guide development of future, targeted preventative strategies.

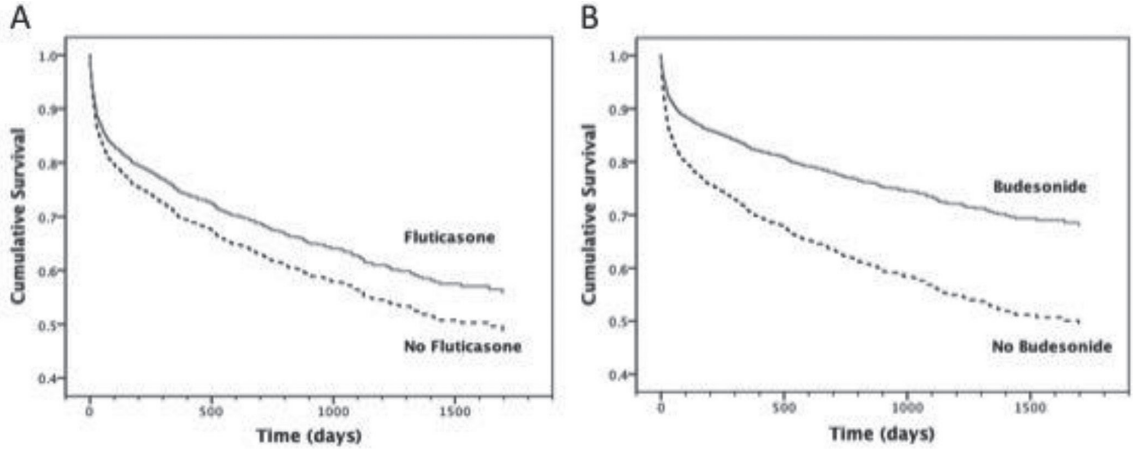
S68 COPD IN THE ED: EOSINOPHILS, TREATMENT AND OUTCOMES, DATA FROM THE PRE-AWARD STUDY

[1]REK Russell, [2]T Doggett, [1]I Pavord, [2]R Pullinger, [2]S Beer, [1]M Bafadhel. *[1]University of Oxford, Oxford, UK; [2]Oxford University Hospitals NHS Foundation Trust, Oxford, UK*

10.1136/thoraxjnl-2016-209333.74

Rationale Acute exacerbations of COPD (AECOPD) are common. Peripheral blood eosinophil count (PBE) predicts outcomes in moderate and severe exacerbations, but little is known about PBE levels and outcomes of AECOPD in the emergency department (ED).

Methods Data for all attendances to ED of a large teaching hospital throughout a 12-month period for patients attending with AECOPD were studied. Anonymised data was cleaned to remove diagnostic and data errors and analysed using GraphPadPrism6 with statistical methods suitable for the data collected. Data is



Abstract S67 Figure 1 Survival comparisons following CAP in COPD patients, stratified by Fluticasone (A) or Budesonide (B) use

Thorax: first published as 10.1136/thoraxjnl-2016-209333.74 on 15 November 2016. Downloaded from http://thorax.bmj.com/ on May 24, 2022 by guest. Protected by copyright.

presented as mean (SEM). Data collected included: demography, length of stay (LOS), vital status, initial treatment, full blood counts, renal function and CRP.

Results There were 549 patients, with 768 AECOPD events. The mean (SD) age was 71 years and 192 episodes were associated with an eosinophil count >2% (26.6%). There were 403 (56%) AECOPD episodes leading to admission; there was no difference in the eosinophil count between patients admitted or discharged from ED. Absolute and relative PBE levels were increased in patients re-attending ED (Absolute PBE mean difference 0.08, 95% CI: 0.02 to 0.13, p = 0.007; %PBE mean difference 0.6, 95% CI: 0.14 to 1.1, p = 0.001). Patients with a PBE > 2% were readmitted more often (p = 0.002, RR 1.16, 95% CI: 1.05 to 1.26). For patients admitted, mean LOS was reduced if admission %PBE levels were >2% (4.6 (0.5) vs. 5.8 (0.3) days, p = 0.012). In patients known to have received oral corticosteroids in ED, the reduction in LOS was greater still if the %PBE was >2% compared to ≤2% (mean (SD) 4.1 (1.0) vs. 6.5 (0.9), p = 0.046). In-patient mortality occurred in 35 patients and occurred more frequently in patients with a %PBE ≤ 2% (RR 1.16, 95% CI: 1.04 to 1.68, p = 0.012).

Conclusions This real-world data suggests that PBE levels may be a useful marker for predicting important clinical outcomes in AECOPD.

S69 TROPONIN LEVELS AND RISK OF DEATH FOLLOWING A MYOCARDIAL INFARCTION IN PEOPLE WITH AND WITHOUT COPD

KJ Rothnie, N Ahmed, JK Quint. *Imperial College London, London, UK*

10.1136/thoraxjnl-2016-209333.75

Introduction Myocardial Infarction (MI) is a common comorbidity and cause of death in people with COPD, and COPD is associated with increased risk of death following acute MI.[1,2] We aimed to: 1) compare levels of peak troponin following MI between people with and without COPD; and 2) investigate differences in the prognostic value of peak troponin between those with and without COPD.

Methods Patients from the Myocardial Ischaemic National Audit Project (MINAP) database who had linked Office of National Statistics (ONS) mortality data from 2003–2013 were included in the study. COPD was defined as the presence of obstructive airway disease and smoking history. We used linear regression to compare levels of peak troponin I and T between people with and without COPD followed by logistic regression to investigate the prognostic value of peak troponin level in predicting 180 day mortality separately for those with and without COPD. All models were adjusted for age, sex, smoking, peripheral vascular disease, cerebrovascular disease, chronic renal failure and previous angina.

Results We included 300,146 patients with a first MI, 34,027 (11.3%) with COPD. Peak troponin T & I was lower for those with COPD following both STEMI (troponin T: 0.51 ng/mL lower, adjusted% 17% lower (95% CI: 6–18%); troponin I: 5.49 ng/mL lower, adjusted% 12% lower (6–18%)) and non-STEMI (troponin T: 0.07 ng/mL lower, adjusted% 20% lower (95% CI: 16–25%); troponin I: 0.34 ng/mL lower, adjusted% 19% lower (15–23%)). The prognostic value of increased peak troponin was higher for COPD patients than those without COPD for troponin T, p-value for interaction = 0.02 (Table 1), but this was not apparent for troponin I p-value for interaction = 0.520.

Abstract S69 Table 1 Risk of death at 180 days after acute MI for those with and without COPD at differing troponin T levels.

Troponin level (ng/mL)	COPD adjusted OR (95% CI)	Non-COPD adjusted OR (95% CI)
<0.01	1 (reference)	1 (reference)
0.01–0.049	1.71 (0.84–3.47)	0.82 (0.62–1.08)
0.05–0.099	1.59 (0.79–3.18)	1.28 (1.00–1.65)
0.1–0.49	2.34 (1.20–4.55)	1.44 (1.34–1.82)
0.5–1.79	2.36 (1.21–4.60)	1.63 (1.29–2.06)
>1.8	2.55 (1.31–4.95)	1.94 (1.54–2.45)

Conclusion Cardiac Troponin T appears a better prognostic indicator for long-term outcome amongst COPD patients following an MI compared to Troponin I. Clinicians should not be reassured by relative lower troponin levels in COPD patients at the time of an MI.

REFERENCES

1 Rothnie KJ, *et al*. Risk of myocardial infarction (MI) and death following MI in people with chronic obstructive pulmonary disease (COPD): a systematic review and meta-analysis. *BMJ Open* 2015;**5**(9):e007824.
2 Rothnie KJ, *et al*. Closing the mortality gap after a myocardial infarction in people with and without chronic obstructive pulmonary disease. *Heart* 2015;**101**(14):1103–10.

Infections and the Impact on Childhood Respiratory Diseases

S70 NEONATAL AIRWAY EPITHELIAL CELL IL-8 RESPONSES TO INFECTION ARE REDUCED IN THOSE WHO GO ON TO WHEEZE

[1]SW Turner, [1]D Miller, [1]G Walsh, [2]U Power, [2]M Shields, [1]G Devereux. *[1]University of Aberdeen, Aberdeen, UK; [2]Queen's University Belfast, Belfast, UK*

10.1136/thoraxjnl-2016-209333.76

Introduction Airway epithelial cells (AEC) are important contributors to the innate immune system and AEC function in children with asthma differs to that of children without asthma. We recruited a birth cohort to establish whether AEC function was abnormal before the onset of asthma symptoms.

Methods Pregnant mothers were recruited and nasal AEC brushings were collected from neonates within 48 hours of birth. Cells were cultured in a submerged model and stimulated with tumour necrosis factor alpha/interleukin-1 beta (TNFa/IL1b), lipopolysaccharide (LPS) and house dust mite (HDM). The mediators in the culture supernatant were quantified by cytometric bead array or ELISA and included: interleukin (IL)−6, IL-8, granulocyte macrophage colony stimulating factor (GMCSF) and interferon gamma (IFNg). The primary outcome of interest was IL-8 expressed as median [interquartile range] in pg/mg protein. Parents returned a postal questionnaire when their child was four years old.

Results AEC were successfully cultured in 139 neonates of whom 120 were contacted and 85 questionnaires were returned. The mean age was 3.8 years, 42 were boys and 10 reported wheeze in the previous 12 months. Neonates who had recent wheeze at four years had reduced median AEC IL-8 release after exposure to TNFa/IL1b (60 [33, 71] versus 105 [64, 157], p = 0.002, see Figure 1) or to LPS (2.2 [0, 9.0] versus 10.5 [4.6, 24.0] p = 0.038) compared to those who did not wheeze. Neonatal AEC GMCSF

Thorax: first published as 10.1136/thoraxjnl-2016-209333.74 on 15 November 2016. Downloaded from http://thorax.bmj.com/ on May 24, 2022 by guest. Protected by copyright.