other disease (Prescott and Vestbo).¹ There is also a need to take into account the confounding effects of malnutrition which have been shown to be independently linked to increased mortality (Collins *et al*).² The current study investigated the influence of social deprivation on 1-year survival rates in COPD outpatients, independently of malnutrition. 424 outpatients with COPD were routinely screened for malnutrition risk using the 'Malnutrition Universal Screening Tool'; 'MUST' (Elia),³ between July and May 2009; 222 males and 202 females; mean age 73 (SD 9.9) years; body mass index 25.8 (SD 6.3) kg/m². Each individual's deprivation was calculated using the index of multiple deprivation (IMD) which was established according to the geographical location of each patient's address (postcode). IMD includes a number of indicators covering economic, housing and social issues (eg, health, education and employment) into a single deprivation score (Nobel *et al*).⁴ The lower the IMD score, the lower an individual's deprivation. The IMD was assigned to each outpatient at the time of screening and related to 1-year mortality from the date screened. Outpatients who died within 1-year of screening were significantly more likely to reside within a deprived postcode (IMD 19.7±SD 13.1 vs 15.4±SD 10.7; $p=0.023$, OR 1.03, 95% CI 1.00 to 1.06) than those that did not die. Deprivation remained a significant independent risk factor for 1-year mortality even when adjusted for malnutrition as well as age, gender and disease severity (binary logistic regression; $p=0.008$, OR 1.04, 95% CI 1.04 to 1.07). Deprivation was not associated with disease-severity ($p=0.906$) or body mass index, kg/m² ($p=0.921$) using ANOVA. This is the first study to show that deprivation, assessed using IMD, is associated with increased 1-year mortality in outpatients with COPD independently of malnutrition, age and disease severity. Deprivation should be considered in the targeted management of these patients.

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S166 THE ROLE OF CLINICAL, METABOLIC AND CARDIAC BIOMARKERS IN PREDICTING OUTCOME FROM COPD EXACERBATIONS REQUIRING HOSPITAL ADMISSION: A PROSPECTIVE OBSERVATIONAL STUDY

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Introduction Baseline hyperglycaemia along with Respiratory Rate has been shown to represent a marker for mortality and failure of Non Invasive Ventilation in COPD exacerbations complicated by decompensated respiratory failure. This study aims to determine whether the same association holds true for those COPD exacerbations admitted to hospital in the absence of respiratory acidosis and if other markers may predict outcomes in such a population.

Methodology COPD patients admitted to University Hospital Aintree with an acute exacerbation were recruited within 48 h of admission with the primary end point being 3 month mortality. Patients presenting with respiratory acidosis were excluded. Subjects underwent clinical assessment at recruitment; blood samples were drawn for Random Blood glucose (RBG), Brain Natriuretic Peptide

(BNP) level (Siemens Healthcare©). Admission ECGs were analysed in order to calculate Cardiac Infarction Injury Score (CIIS).

Results 116 patients (mean age 70 years; 55% female; FEV1 0.98 (predicted 2.49) litres; admission ABGS pH 7.40; PCO2 5.93kPa; PO2 9.04kPa) were recruited; 18 (16%) patients had died by 3 months. Hyperglycaemia (defined as RBG ≥ 7 mmol/l) was not associated with increased 3 month mortality (observed in 6/18 (33%) of deaths at 3 months vs 35/98(36%) of survivors; $p=0.85$). The Respiratory Rate (RR) measured during clinical assessment appeared to significantly higher in those who had died by 3 months (27 (SD 5) vs 24 (SD 5); $p=0.048$). No association was observed between those who had died by 3 months in terms of BNP levels (18.71 vs 24.22 pg/ml; $p=0.48$), CIIS (7.65 vs 6.25; $p=0.37$), age (71 vs 70 years; $p=0.63$), PaCO2 (6.29 vs 5.87 kPa) or serum Bicarbonate (25 vs 26 mmol/l; $p=0.64$). An inverse correlation was noted between BNP values and admission PaCO2 levels (Correlation coefficient -0.25 ; $p=0.018$) and Bicarbonate levels (Correlation coefficient -0.35 ; $p=0.001$); a positive correlation was observed between BNP levels and patient age (Correlation coefficient -0.35 ; $p=0.002$).

Discussion In patients admitted to hospital with COPD exacerbations, hyperglycaemia, BNP level and CIIS were not found to be predictors of mortality in the absence of acute respiratory acidosis. Interestingly, Respiratory Rate measured whilst in hospital appears to predict outcome at 3 months.

S167 EXPANSION OF THE RED CELL DISTRIBUTION WIDTH AND EVOLVING IRON DEFICIENCY AS PREDICTORS OF POOR OUTCOME IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Background Chronic obstructive pulmonary disease (COPD) is a multi-system disorder driven partly by diffuse inflammation, malnutrition and haematological aberrations. Because the red blood cell distribution width (RDW) is a surrogate of these anomalies, we hypothesised that it might be of prognostic importance in COPD patients. Additionally, we tested the supposition that iron deficiency (ID) per se could be a prevalent and ominous co-morbidity in these individuals.

Methods We analysed the relation of red cell indices on admission and over time with mortality in 655 consecutively eligible COPD patients (mean±SD age 77±12 y, FEV1 0.85±0.34 l, FVC 1.44±0.60 l, 54% male). Concomitant heart failure, ischaemic heart disease, and neoplasia were exclusion criteria. The combination of a high RDW and low mean cell haemoglobin (MCH) was utilised to identify ID.

Results On admission, an RDW>15%, Hb<12.5 g/dl, MCH<27, and ID were evident in 33%, 31%, 12% and 10% of patients. Compared to those with an RDW≤15%, patients with levels >15% had lower Hbs, lower FEV1s, and longer median (±IQR) hospital stays (9±11 vs 8±8 days, $P<0.001$). Over a mean period of 40±29 months, 227 (35%) patients died. On Cox proportional hazards analyses, a higher RDW predicted increased mortality (adjusted χ^2 16, $P<0.0001$) independently of age (χ^2 11, $P<0.001$), FEV1 (χ^2 5, $P<0.03$), Hb and creatinine (latter two not retained in model) and provided graded prognostic information (abstract S167 figure 1A) incremental to that of FEV1 ($P<0.05$ for change in χ^2). Over time, 63%, 72%, 65%, and 46% of patients had a rise in RDW, a fall in Hb, a fall in MCH, and evolving ID (rising RDW and falling MCH), respectively. A rising RDW predicted death (adjusted χ^2 32, $P<0.0001$) independently of baseline RDWs and changes in Hb,