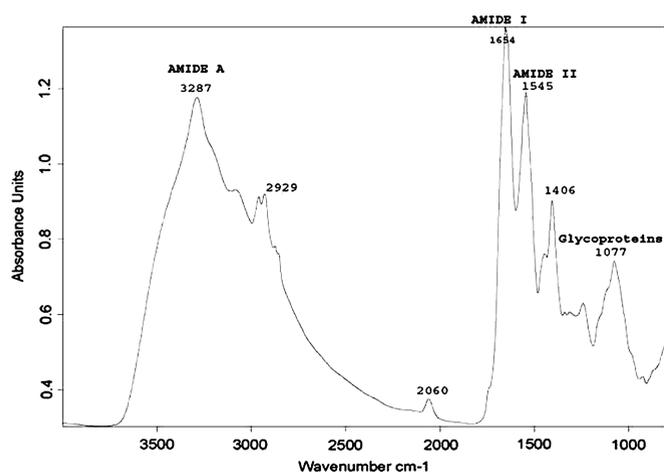


sputum profiling enables non-invasive rapid sampling and evaluation, producing spectral signatures that can differentiate COPD status or predict exacerbation presence.



Abstract P111 Figure 1 Example of a typical IR absorption spectrum for COPD sputum.

P112

ASSOCIATION OF SPUTUM PSEUDOMONAS AERUGINOSA (PSA) ISOLATION AND LENGTH OF HOSPITAL STAY IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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Introduction and objectives A lot of effort and investment has been made to improve the length of stay (LOS) in COPD related admissions with variable success. It is known that numbers of hospital admissions and re-admissions have increased. Bacterial infections contribute to acute exacerbation of COPD (AECOPD) in 50% of cases; PsA is a probable pathogenic organism (PPM) causing acute or chronic infection in severe COPD patients. We set out to examine whether PsA in sputum influenced the length of stay as this PPM is not covered by the routinely used first line antibiotics.

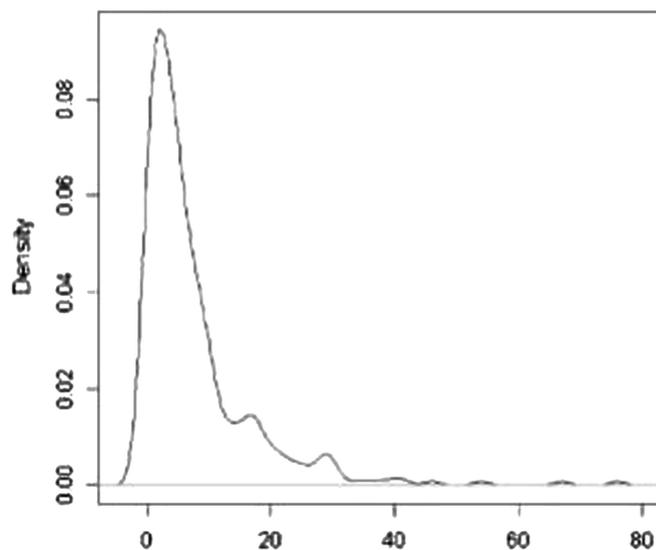
Method A retrospective audit was carried out examining the sputum culture results and LOS in all COPD admissions in an acute hospital from 1 January 2009 to 31 December 2009, patients were identified from coding (ICD: J44). Sputum cultures were attempted (and collected in all sputum producers) by a dedicated respiratory team as a routine for all COPD admissions throughout that period, which was established as part of service improvement well before the audit. Results were obtained from Pathology department database. Patients were divided into sputum producing group and non-producers, Sputum producers were further divided into patients with positive sputum culture of PPM and non-pathogenic organisms. PsA growers were identified. Median LOS was calculated to be 5 days and all groups were compared with this duration as a standard for their LOS.

Results Total admissions with COPD were 332; 203 were sputum producers and 122 had bacterial isolates. There were 37 PsA growers. LOS was seen to be 5 or more days in: 121 sputum producers vs 47 non-producers, $p=0.000046$. 79 PPM culture positive patients vs 42

without bacterial growth, $p=0.067$; PsA growers vs. all admissions, $p=0.000744$, PsA vs all sputum producers $p=0.0126$, PsA vs all PPM, $p=0.041$.

Conclusions PsA infection is associated with significantly longer LOS in hospital. All COPD patients should possibly be screened for PsA infection (and possibly underlying bronchiectasis) to inform the selection of antibiotics, which is likely to reduce LOS and may reduce re-admission rates.

Estimated Distribution of Length of Stay



P113

PROCALCITONIN (PCT) IS A SAFE AND RELIABLE BIOMARKER OF BACTERIAL INFECTION IN EXACERBATIONS OF COPD—SO WHY IS IT SO CHALLENGING TO INTRODUCE IT INTO A LARGE UK HOSPITAL?

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Background Antibiotics have only marginal efficacy in treating acute exacerbations of COPD (AECOPD). There is a large body of evidence that supports the use of PCT as a marker of bacterial infection. In York hospital patients with AECOPD account for a significant proportion of acute admissions and many are treated with antibiotics without strong evidence of a bacterial cause.

Objective To conduct a service evaluation of antibiotic prescribing following the introduction of PCT in patients with AECOPD. To observe attitudes towards and uptake of the test, and identify barriers to implementation.

Methods The evaluation ran from November 2009 to June 2010. Information on PCT was introduced to all physicians, and an algorithm for use was provided. Medical notes were reviewed from patients where PCT was requested. Evidence of documentation of PCT and whether it had been acted upon were recorded along with antibiotic use, length of stay and readmission within 1 month of discharge.

Results 54 PCT tests were performed and 49 were included in the final analysis. 32 (65%) of samples were below the cut-off for antibiotics, but were still prescribed in 11 cases. Of those above the threshold for treatment three did not receive antibiotics. PCT was documented and acted upon in only 12 (24%) patients. Overall, antibiotics were used in 25 (51%) patients. Two patients were readmitted with AECOPD within 1 month of discharge having been managed according to PCT results, with one receiving antibiotics. 24 (49%) samples were not reported on the day they were taken.

Conclusion 11 RCTs from different countries enrolling over 3500 patients have demonstrated the feasibility and safety of PCT in

guiding antibiotic prescribing. Our service evaluation showed that despite this, introducing it into a hospital setting is challenging. If the algorithm had been followed correctly 65% of patients would not have received antibiotics. Physicians often rely on clinical judgement and well-known markers of infections such as CRP and WCC. Education of physicians along with timely availability of the test play significant roles. We intend to persevere and report our results aiming to improve the use of PCT within our hospital.

P114 C-REACTIVE PROTEIN AT ONSET PREDICTS SYMPTOM RESOLUTION IN ACUTE EXACERBATIONS OF COPD

doi:10.1136/thx.2010.150987.15

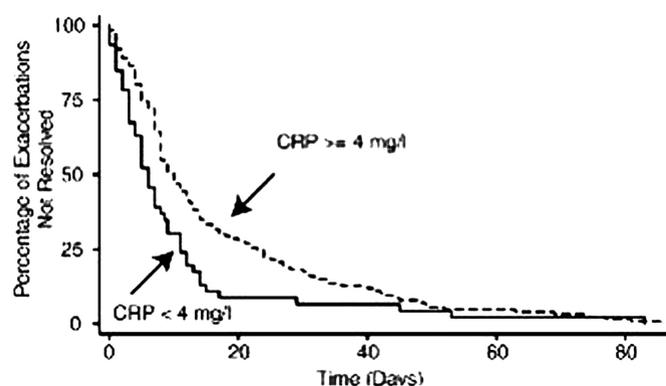
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Introduction Patients with chronic obstructive pulmonary disease (COPD) may not recover swiftly from exacerbations. In 23% of patients, symptoms do not recover within 35 days.¹ It would be valuable for clinicians to have a systemic biomarker that taken at initial consultation for an exacerbation could predict exacerbation length. We hypothesised that inflammation, measured by serum C-reactive protein (CRP) predicts the time to symptomatic resolution.

Methods We analysed daily diary cards prospectively collected from the London COPD cohort between October 1995 and January 2010. All patients had an FEV₁ ≤ 80% predicted and an FEV₁/FVC ratio ≤ 70%. An exacerbation was defined as an increase for 2 consecutive days in respiratory symptoms, with at least one major symptom (dyspnoea, sputum purulence or volume) plus another major or minor symptom (wheeze, cold sore throat and cough). Exacerbations were separated by five symptom free days. Patients were assessed and sampled at clinic visits within a median of 2 days of exacerbation onset. Exacerbation length was defined as the number of days from onset that increased airway symptoms were still recorded, and resolution time was defined as the time from clinic assessment to symptom cessation.

Results 268 patients with ≥ 365 days of diary card data experienced at least one exacerbation. Median baseline CRP was 4 mg/l. CRP at onset was available for 172 recovered exacerbations (Abstract P114 Figure 1). Mean age was 68.0 years (SD 8.3), with mean FEV₁ 1.11 l (0.45), FEV₁ predicted 44.6% (16.2). Patients with a CRP < 4 mg/l at initial clinic review had a significantly shorter resolution time than patients with CRP ≥ 4 mg/l, median 6 (IQR 3–11; n=46) vs 10 (5–23; n=126) days, p=0.0017. Cox proportional hazard risk models with adjustment for repeated measures showed that CRP predicted both exacerbation length (p=0.039) and resolution time (p=0.029).

Conclusions COPD exacerbations can occur without an elevated systemic inflammatory response. These events are associated with a



Abstract P114 Figure 1 Kaplan-Meier plot of symptom resolution by high and low CRP at exacerbation onset.

short resolution time. Thus, using CRP as a biomarker, it may be possible to identify patients that have a short recovery period and potentially may not require systemic treatment for their exacerbation.

REFERENCE

1. **Perera, et al.** Inflammatory changes, recovery and recurrence at COPD exacerbation. *ERJ* 2007;**29**:527–34.

P115 VITAMIN D BINDING PROTEIN IN COPD EXACERBATIONS

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Introduction Vitamin D binding protein (DBP) plays a role in macrophage activation and neutrophil chemotaxis, both functions being relevant to COPD and airway infection. We have shown previously that DBP is present in the COPD lung and relates directly to macrophage activation.¹ Transcription of DBP is stimulated by inflammatory cytokines,² which tend to be raised in exacerbations. DBP is also cleaved from its binding site on neutrophils by the action of neutrophil elastase.³ We reasoned that DBP would be elevated in the lung and systemic circulation during exacerbations of COPD compared to the stable state.

Methods 20 patients with a known diagnosis of COPD were studied in the stable state and at the start of an exacerbation. Circulating CRP, elastase, AAT and DBP were measured at each time point, together with sol phase DBP, AAT and CRP, where sufficient sample was available (n=9). All clinical parameters were studied in the stable state only. Relationships between exacerbation and stable state were sought using pair-wise tests for each individual, whilst group relationships between DBP, CRP and elastase were sought using Spearman's rank correlation.

Results No significant differences between stable state and exacerbation were seen for circulating or sol phase DBP (both p>0.4). Circulating CRP and AAT were significantly higher in exacerbation (mean difference 3.95, p<0.0001 and 137.39, p=0.03 respectively), as were sol phase CRP and AAT (mean difference 18.29, p=0.003 and 0.60, p=0.009 respectively). DBP did not relate to elastase in either stable state (p=0.61) or exacerbation (p=0.17), nor to CRP (p=0.71 and 0.98, respectively).

Conclusions The level of local and systemic inflammation, with consequent elastase release, during a COPD exacerbation is insufficient to alter DBP levels. As such it is unlikely that DBP-derived macrophage activation is important in their pathogenesis or resolution.

REFERENCES

1. **Wood, et al.** *Thorax* 2009;**54**(Suppl IV):A3.
2. **Guha, et al.** *Hepatology* 1995;**21**:1675–81.
3. **DiMartino, et al.** *J Immunol* 2001;**166**:2688–94.

P116 SERUM PARC (CCL18) AND EXACERBATION FREQUENCY IN COPD

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Introduction Patients with Chronic Obstructive Pulmonary Disease (COPD) experience intermittent exacerbations. The frequency of exacerbations varies amongst patients but the 'Frequent Exacerbator' appears to be an independent disease phenotype. As frequent exacerbations are associated with more rapid lung function decline, a validated biomarker is needed to identify those susceptible, prompting rigorous medical treatment and aiding selective