Abstract P50 Table 1 CTPA usage 2009–2014

Year	Annual total no. of CTPAs (% change from 2009)	No. of CTPAs in 6-week reference period (% of annual total)	No. of technically inadequate CTPAs (% of reference scans)	No. of scans positive for acute PE (% of technically adequate reference scans)	No. of scans negative for any diagnosis (% of all reference scans)
2009	1973 (n/a)	242 (12.3%)	19 (7.9%)	51 (22.9%)	39 (16.1%)
2010	2317 (+17.4%)	249 (10.7%)	11 (4.4%)	41 (17.2%)	42 (16.9%)
2011	2561 (+29.8%)	320 (12.5%)	20 (6.3%)	60 (20.0%)	42 (13.1%)
2012	2759 (+39.8%)	329 (11.9%)	25 (7.6%)	57 (18.8%)	43 (13.1%)
2013	3017 (+52.9%)	348 (11.5%)	18 (5.2%)	67 (20.3%)	52 (14.9%)
2014	3302 (+67.4%)	394 (11.9%)	20 (5.1%)	77 (20.6%)	55 (14.0%)

Clinical Studies in COPD

P51 CLINICAL EFFECTIVENESS OF PROCALCITONIN BASED PROTOCOLS TO GUIDE THE ADMINISTRATION OF ANTIBIOTICS IN PATIENTS PRESENTING WITH COPD EXACERBATIONS: SYSTEMATIC REVIEW AND META-ANALYSIS

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Background Different types of acute exacerbations of COPD (AECOPD) have been described, such as those driven by bacteria, viruses or by enhanced eosinophilic inflammation. Bacterial infections, where antibiotics are indicated, appear to account for approximately half of COPD exacerbations. However, challenges in differentiation of the aetiology of AECOPD have led to significant overuse of antibiotics. Serum procalcitonin, released in response to bacterial but not viral infections or non-specific inflammation, could possibly identify AECOPD requiring antibiotics. In this meta-analysis we assessed safety and clinical effectiveness of procalcitonin-based protocols to initiate or discontinue antibiotics in patients presenting with AECOPD.

Methods Based on a prospectively registered protocol, we systematically reviewed the literature and selected randomised or quasi-randomised controlled trials comparing procalcitonin based protocols to initiate or discontinue antibiotics in patients presenting with AECOPD versus standard care. We followed Cochrane's and GRADEs guidance to assess risk of bias, quality of evidence and to perform meta-analyses.

Results Eight completed trials evaluating 1,062 patients with AECOPD met the inclusion criteria. Procalcitonin-based protocols used in all included trials were similar. Antibiotics were recommended for procalcitonin levels >0.25 μg/L and discouraged for lower levels. All studies were open-labelled or single blinded and the final decision to administer or withhold antibiotic was left to the responsible clinician, who could deviate from the protocol. Adherence to procalcitonin-based protocols ranged

Abstract P51 Table 1 Clinical effectiveness of procalcitonin-based protocols to initiate or discontinue antibiotics in patients presenting with acute exacerbations of COPD

Outcomes	No of participants (studies) Follow-up	Quality of the evidence (GRADE)	Relative effect (95% CI)	Anticipated absolute effects	
				Risk with standard care	Risk difference with Procalcitonin-guided protocols
Treatment failure for the index exacerbation.	834 (5 RCTs)	⊕⊕◯◯ LOW [1,2]	**RR 0.81** (0.62 to 1.06)	206 per 1,000	**39 fewer failures per 1,000** (78 fewer to 12 more)
Length of hospital stay for the index exacerbation	1062 (8 RCTs)	⊕⊕⊕◯ MODERATE [1]	**MD −0.76** (−1.95 to 0.43)	Mean length of hospital stay was **8.55** days	MD **0.76 days lower** (1.95 lower to 0.43 higher)
Proportion of patients who were prescribed antibiotics on admission	984 (7 RCTs)	⊕⊕⊕◯ MODERATE [1]	**RR 0.56** (0.43 to 0.73)	791 per 1,000	**348 fewer prescriptions per 1,000** (451 fewer to 214 fewer)
Mean duration of the course of antibiotics	776 (6 RCTs)	⊕⊕◯◯ LOW [1,2]	**MD −3.83** (−4.32 to −3.35)	Mean duration of course of antibiotics was **8.27** days	MD **3.83 days lower** (4.32 lower to 3.35 lower)
Exacerbation recurrence rate at longest follow-up	496 (3 RCTs)	⊕⊕◯◯ LOW [1,2]	**RR 0.96** (0.69 to 1.35)	205 per 1,000	**8 fewer recurrences per 1,000** (63 fewer to 72 more)
Re-hospitalisation rate at longest follow-up	398 (3 RCTs)	⊕⊕◯◯ LOW [1,2]	**RR 1.45** (0.92 to 2.29)	116 per 1,000	**52 more admissions per 1,000** (9 fewer to 150 more)
Rate of re-hospitalisation due to an exacerbation at longest follow up	298 (2 RCTs)	⊕⊕◯◯ LOW [1,2]	**RR 1.22** (0.71 to 2.09)	135 per 1,000	**30 more admissions per 1,000** (39 fewer to 147 more)
Overall mortality at longest follow up	1062 (8 RCTs)	⊕⊕◯◯ LOW [1,2]	**RR 0.99** (0.57 to 1.70)	41 per 1,000	**0 fewer deaths per 1,000** (18 fewer to 29 more)

***The risk in the intervention group** (and its 95% confidence interval) is based on the assumed risk in the comparison group and the **relative effect** of the intervention (and its 95% CI). **CI:** Confidence interval; **RR:** Risk ratio; **MD:** Mean difference.

between 61.3% and 98.1%. Procalcitonin-based protocols decreased antibiotic prescription (RR 0.56 [0.43, 0.73]) and the total antibiotic exposure (MD −3.83 [−4.32, −3.35]), without affecting clinical outcomes such as rate of treatment failure (RR 0.81 [0.62, 1.06]), length of hospitalisation (MD −0.76 [−1.95, 0.43]), exacerbation recurrence rate (RR 0.96 [0.69, 1.35]) or mortality (RR 0.99 [0.57, 1.70]). However, the quality of the available evidence is low to moderate because of methodological limitations and small overall study population.

Conclusion Procalcitonin-based protocols to guide the administration of antibiotics in patients presenting with AECOPD appear safe and clinically effective. The quality of the available evidence is low-to-moderate because of methodological limitations and small overall population. Thus, additional appropriately designed and powered confirmatory randomised controlled trials are required.

P52 COPD AND PERIODONTITIS: CO-MORBIDITY YES OR NO?

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Introduction Chronic obstructive pulmonary disease (COPD) is an inflammatory disease associated with comorbidities including periodontitis.[1–2] Periodontitis is characterised by plaque build-up, anaerobic bacterial overgrowth and gingival inflammation which promotes recruitment and activation of neutrophils leading to alveolar bone destruction and tooth loss. However, the characterisation of periodontitis varies between studies causing some uncertainty of any association.

Aim To determine whether clinical indices of periodontitis affects its prevalence in COPD patients with and without Alpha-1-antitrypsin deficiency (AATD) and any association with lung function.

Methods 108 COPD and 63 PiZ AATD patients underwent dental examinations and lung function testing as part of an EU FP7 cross sectional study.

Varying definitions of periodontitis used in previous publications were applied; including criteria from the Centres for Disease Control and Prevention in collaboration with the American Academy of Periodontology CDC-AAP (CDC-AAP) and 5th European Workshop in Periodontology.

Periodontal indices of probing depth (PD – depth from gingival margin to the base of periodontal pocket) and clinical attachment level (CAL – distance from the cemento-enamel junction to the gingival margin plus probing depth) were then compared to lung function parameters.

Results The prevalence of periodontitis varied depending on the definition used.

Prevalence ranged from 0.7–98.6% for the whole cohort, with the lowest prevalence for average probing depth >4 mm, but CDC-AAP criteria gave a prevalence of 84.2% and 98.6% with the 5th European workshop criteria.

Lung function was significantly correlated with indices of periodontitis for AATD patients; see Table.

Conclusions The prevalence of periodontitis depends on the definition used. PD is a marker of current status, whilst CAL represents cumulative disease activity, rather like current lung function parameters.

Abstract P52 Table 1 Relationships between clinical indices of periodontitis and lung function in COPD and AATD

	Spearman's Rho and p-value			
	Average Probing Depth (PD)		Average Clinical Attachment Level (CAL)	
	COPD	AATD	COPD	AATD
% predicted FEV1	0.085	−0.42	−0.03	−0.52
	p = NS	p < 0.01	p = NS	p < 0.001
% predicted TLCO	−0.06	−0.34	0.01	−0.51
	p = NS	p < 0.01	p = NS	p < 0.001
% predicted KCO	−0.10	−0.30	−0.04	−0.42
	p = NS	p < 0.05	p = NS	p < 0.01

Periodontal indices are correlated with lung function parameters in AATD patients which could reflect the inflammatory and predominantly neutrophilic pathophysiology leading to excessive tissue destruction in both diseases.

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P53 PREDICTING POOR OUTCOMES IN COPD PATIENTS DEEMED 'LOW RISK' BY DOSE SCORE

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Introduction COPD continues to cause a substantial symptom, mortality and financial burden in the UK. Current treatment strategies are predominantly reactive as insufficient evidence exists to successfully target clinical resource into pre-emptive 'early interventions'. The DOSE (dyspnoea, obstruction, smoking status and exacerbation) score has been validated as a risk predictor for mortality, hospitalisation and poorer health status. However, only a small proportion of COPD patients with poor outcomes have high DOSE scores. We sought to establish if clinical characteristics can be used to pre-emptively identify those COPD patients vulnerable to future poor health status by using an electronic database of anonymised patient records-the Hampshire Health Record Analytical Database (HHRA).

Methods Within our HHRA database COPD cohort, we identified a cohort of 6890 patients who fell into the 'low risk' category by DOSE score (<4). Within this group, a subset met the criteria for poor COPD outcomes over the next four years, defined as; death (all cause), COPD related hospital admission, a DOSE score increase of ≥2 points or a subsequent DOSE score of ≥4 (high risk). We used logistic regression analysis to examine the association between demographic and clinical characteristics documented by Read code at baseline and those who subsequently fell into the poor outcomes subgroup.