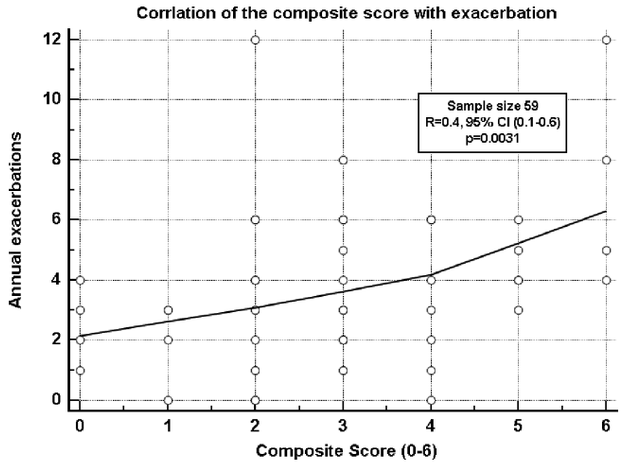
63 (55%), mean FEV1 = 2.0 L (SD1.8–2.1), FEV1% pred = 68%, and mean FEV1/FVC ratio = 71% (SD = 68–74).

A significant positive correlation between FeNO and PBE was observed (r = 0.39, p = 0.004), but not with periostin. Only FeNO significantly correlated to exacerbations (r = 0.42, p = 0.0008) and only periostin correlated significantly to ACQ7 score (r = 0.33, p = 0.0053). In addition, the biomarkers composite score significantly correlated with exacerbations (r = 0.4, p = 0.0031) (Figure 1), but not ACQ7.



Abstract P72 Figure 1 The relationship between T2 composite score and annual OCS requiring exacerbations

Conclusion In real life settings, FeNO correlated with historical exacerbations and the T2 composite score displayed a dose response correlation with exacerbations frequency. Periostin correlated with ACQ7 but not exacerbations. Further research is required to confirm these findings.

REFERENCE

1 Heaney LG, *et al*. *Thorax* 2015;**0**:1–3. doi:10.1136/thoraxjnl-2015-207326

P73 A PILOT STUDY TO INVESTIGATE THE USE OF SERUM INHALED CORTICOSTEROID CONCENTRATION AS A POTENTIAL MARKER OF TREATMENT ADHERENCE IN SEVERE ASTHMA

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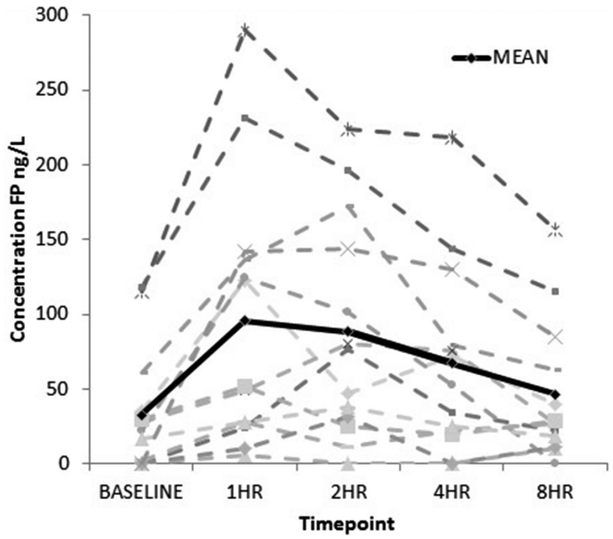
Background Inhaled anti-inflammatory therapy is fundamental to asthma management, but adherence is very poor. This increases the risk of exacerbations and poor symptom control. Currently there is no direct way of assessing adherence to inhaled steroids accurately. The primary aim of this project was to determine whether liquid chromatography tandem mass spectrometry could be used to detect fluticasone propionate (FP) in human serum as a potential aid to therapeutic monitoring. The secondary aim was to relate serum levels of FP to markers of asthma severity.

Methods We collected blood samples over an 8 hr period from inpatients with severe asthma on a stable dose of inhaled FP. Following baseline (trough) sampling, patients were observed using their inhaler, with inhaler technique documented. Subsequent samples were obtained 1, 2s, 4 and 8 hrs post inhalation. Demographic details and spirometry were also recorded.

Results Thirteen patients were recruited: 8 males, 5 females; age range 22–64 yrs; FEV1 range 53–101% predicted; 10 patients on 1000 mcg/day, and three on 2000 mcg/day FP. The mean concentration of FP at 1 hr post inhaler (peak in 7/13 patients) was 95.5 (SD 89.1) ng/L. The mean pre-dose trough concentration was 32.9 (SD 41.5) ng/L. Two patients were noted to have poor inhaler technique; these patients had some of the lowest serum FP levels recorded. The FEV1% predicted was found to be strongly correlated with the peak serum FP concentration (Pearson's r = 0.8, p = 0.001).

Conclusion We have demonstrated that FP can be detected in the blood of patients with severe asthma following directly observed therapy; this could have a potential future application as a direct measure of adherence and perhaps inhaler technique. We also demonstrated a profound effect of reduced lung function predicting low serum FP levels. Future work will explore whether poor absorption reflects relatively poor efficacy in the most severe patients.



Abstract P73 Figure 1 Serum concentration of FP at sampling timepoints; dashed lines represent individual patients; solid line group mean

P74 PREVALENCE OF SPECIFIC ANTIBODY DEFICIENCY IN SEVERE ASTHMA

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Introduction Patients with asthma are prone to recurrent infective exacerbations, due to viral or bacterial infections. We have previously presented retrospective data, demonstrating

Abstract P74 Table 1 Prevalence of specific antibody deficiency and associated patient characteristics

	Prevalence	Bronchial wall thickening on CT chest	Bronchiectasis on CT chest	≥7 Exacerbations past year	Total ITU admissions	Percentage predicted FEV1 (%)	Inhaled steroid (BDP equivalent) μg
n = 45	n (%)	n (%)	n (%)	n (%)	n (%)	Mean (SD)	Mean (SD)
Total *H. influenzae* deficient	30 (70)						
***H. influenzae* deficient alone**	13 (31)	8 (23)	5 (5) - 14	4 (3) - 17	3 (3) - 18	57 (16)	2540 (2240)
Total *S. pneumoniae* deficient	23(51)						
***S. pneumoniae* deficient alone**	5 (12)	3 (8.5)	2 (2) - 6	1 (0) - 4	0 (2) - 0	77 (8)	1933 (1101)
Combined Deficiency	17 (40)	11 (31)	6 (10) - 17	8 (3) - 33	4 (2) - 24	73 (23)	1837 (900)
Competent Specific Antibody Levels	7 (13)	2 (6)	3 (2) - 8.5	2 (3) - 6	0 (3) - 0	70 (32)	1840 (607)

significantly reduced lung function in severe asthma patients with specific antibody deficiency. The prevalence and impact of specific antibody deficiency in this patient group is not known.

Aim We aimed to determine the prevalence of specific antibody deficiency and its association with markers of disease severity.

Methods We prospectively collected data from all new patients attending the regional severe asthma clinic. We recorded demographic details and markers of disease severity including: BTS treatment step, inhaled corticosteroid (ICS) dose, spirometry, blood and sputum eosinophil count, radiological findings such as bronchiectasis and bronchial wall thickening, exacerbations in the last year and ITU admissions. Specific antibody levels to *Haemophilus Influenzae* (Streptococcus Pneumoniae (<0.35 μg/ml to at least 6 of the 12 serotypes classed as deficient) were measured.

Results Data for 53 patients (39F), mean (SD) age 49.6 (15.9) years were available. Mean (SD) FEV1 was 69 (22)% predicted, ICS dose 1914 (1337) micrograms, and BMI 31.5 (8.6) kg/m^2. All were at BTS step 3–5. Information on specific antibody levels was available for 43 and 45 patients for *H Influenzae* and *S pneumoniae* respectively and for both in 42 patients. Overall out of the 42 patients 35 (83%) were deficient to one or both the organisms. Of these 13 (31%) were deficient to *H Influenzae* alone, 5(12%) to *S pneumoniae* alone, and 17 (40.5%) to both. Looking at each organism separately 30 (70%) out of 43 were deficient to *H Influenzae* and 23 out of 45 (51%) were deficient to *S pneumoniae*. Of the 32 patients for whom data were available 20 (63%) had 7 or more exacerbations in the preceding year and two thirds of these had bronchial wall thickening on their CT scans. A third of patients (30%) reported at least one ITU admission. The presence of specific antibody deficiency did not correlate with any clinical or radiological findings.

Conclusion Specific antibody deficiency to *H influenza* and *S pneumoniae* is remarkably common in moderate to severe asthma. Further studies are required to determine the clinical significance of this finding.

P75 CLUSTER CLASSIFICATION AS A PREDICTOR OF ADVERSE RADIOLOGICAL OUTCOMES IN ALLERGIC FUNGAL AIRWAYS DISEASE (AFAD)

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Introduction The relationship between immunological biomarkers and evidence of lung damage has not been established in asthmatics that are sensitised to fungi. We sought to determine what features of allergic fungal airways disease were related to adverse radiological outcomes by the use of cluster analysis.

Method Factor analysis was used to determine the significance of different clinical and immunological variables, the number of clusters present and cluster membership (n = 423). The presence of radiological indicators of lung damage and inflammation were then assessed between these groups.

Results Three clusters were identified. Cluster 1 (37.1%) were obese, had late onset and minimal eosinophilic disease, cluster 2 (40.9%) had late onset eosinophilic disease and cluster 3 (22%) had early onset, atopic disease. Sensitisation to *A. fumigatus* was more prevalent in cluster 3 (94.2%; sIgE *A. fumigatus* 10.2 kUA/L (1.31–35.4)), compared to cluster 1 (44.9%; sIgE *A. fumigatus* 0.16 kUA/L (0.03–0.94)) and cluster 2 (45.7%; sIgE *A. fumigatus* 0.25 kUA/L (0.06–1.36)).

Cluster 3 had a greater degree of airflow obstruction ($p < 0.001$), bronchiectasis (69%, $p < 0.001$), tree in bud (32.4%, $p < 0.001$), collapse/consolidation (48.6%, p0.007) and fibrosis (31.7%, $p < 0.05$) than any of the other groups.

Conclusion This cluster analysis demonstrates that sensitisation to *A. fumigatus*, in addition to the other known clinical phenotypes, identifies asthmatics most at risk of developing fixed airflow obstruction and radiological features of airway inflammation and damage.

P76 PSYCHOGENIC VOICE DISORDER MIMICKING TREATMENT-REFRACTORY RESPIRATORY DISEASE

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Introduction and objectives Psychogenic voice disorder (PVD) is widely acknowledged by voice and ENT specialists as an important cause of dysphonia and breathlessness. The significance of abnormal vocal function in the aetiology of respiratory symptoms is under-recognised in respiratory medicine. The aim of this study is to highlight the importance of PVD as a key differential diagnosis for patients who present with respiratory symptoms and altered voice quality.