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 <i>H. influenzae</i> deficient	30 (70)						
<i>H. influenzae</i> deficient alone	13 (31)	8 (23)	5 (5) - 14	4 (3) - 17	3 (3) - 18	57 (16)	2540 (2240)
Total <i>S. pneumoniae</i> deficient	23(51)						
<i>S. pneumoniae</i> 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/m². 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.