Abstract S107 Table 1

Variable	Exacerbation Start (No viral symptoms) n=23	End Exacerbation (No viral symptoms) n=23	p Value	Start Exacerbation (Viral symptoms) n=17	End Exacerbation (Viral symptoms) n=17	p Value
24 h sputum volume (ml)	15 (10–15)	3 (0–10)	<0.0001	20 (10–20)	5 (2–8)	<0.0001
%(n) purulent sputum	95.6 (22)	26.1 (6)	<0.0001	82.3 (14)	0 (0)	<0.0001
ISWT (m)	210 (80–350)	350 (140–430)	<0.0001	300 (100–410)	380 (255–585)	<0.0001
ESR (mm/hr)	26 (14–44)	18 (9–41)	0.04	14 (8.5–25)	12 (7.5–14)	0.01
CRP (mg/l)	14 (10–33)	5 (2–15)	<0.0001	14 (8–26.5)	5 (2–8)	0.002
WCC ($\times 10^9$) (range 4–11)	9.3 (7.3–12)	8.6 (7.2–9.7)	0.005	8 (6–12.2)	7.1 (5.4–8.75)	0.04
Neutrophil count ($\times 10^9$) (range 2–7.5)	6.42 (5.5–8.41)	5.22 (4.64–6.76)	0.02	5.44 (3.5–8.86)	3.85 (3.06–6.2)	0.07
Lymphocyte count ($\times 10^9$) (range 1.5–4)	1.76 (1.08–2.18)	1.71 (1.32–2.13)	0.55	1.82 (1.41–2.63)	1.76 (1.52–2.16)	0.8
Pathogens isolated from sputum % (n)	100 (23)	30.4 (7)	0.009	100 (17)	23.5 (4)	<0.0001

CRP, C reactive protein; ESR, Erythrocyte Sedimentation Rate; WCC, White cell count.

bacteriology; sputum colour; 24 h sputum volume; respiratory viral PCR [Influenza A and B; Respiratory Syncytial Virus; Parainfluenza Type 1, 2, 3; Adenovirus]; incremental shuttle walk test; total white cell count; lymphocyte count; neutrophil count; C reactive protein; Erythrocyte Sedimentation Rate. Data is presented as median (IQR) and groups compared using the Mann–Whitney U Test.

Results 40 patients were included. 17 reported viral symptoms. Of the 17 patients who reported viral symptoms, only 2 had positive viral PCR [Influenza Type B (n=1) and Respiratory Syncytial Virus (n=1)]. No patient in the group not reporting viral symptoms had a positive viral PCR. At the start of the exacerbation, there was no significant difference in any parameter between the groups and both groups had a similar and positive impact with 2-week course of antibiotic therapy. Abstract S107 table 1 details the measurements at the start and end of the exacerbation for each group.

Conclusion There was a low prevalence of positive virology despite the presence of viral symptoms and outcomes were similar in patients with and without viral symptoms. Larger cohort studies are needed.

S108 EXPERT CONSENSUS ON DIAGNOSTIC CRITERIA AND TERTIARY SERVICE REQUIREMENTS FOR BRONCHIECTASIS

doi:10.1136/thoraxjnl-2011-201054b.108

[1]J Holme, [2]S Bianchi, [3]I Clifton, [4]A De-Soyza, [2]F Edenborough, [3]D Peckham, [2]O Pirzada, [5]P Walker, [6]M Walshaw, [1]R Niven. *[1]University Hospital of South Manchester NHS Foundation Trust, Manchester, UK; [2]Sheffield Teaching Hospitals NHS Foundation Trust, Sheffield, UK; [3]Leeds Teaching Hospitals NHS Trust, Leeds, UK; [4]Newcastle Hospitals NHS Foundation Trust, Newcastle, UK; [5]Aintree University Hospitals NHS Foundation Trust, Liverpool, UK; [6]Liverpool Heart and Chest Hospital NHS Foundation Trust, Liverpool, UK*

Introduction There are no agreed diagnostic criteria for bronchiectasis and no stated minimum requirements for a tertiary service. The Northern Bronchiectasis Group aimed to use the validated RAND technique to form consensus opinions on these issues.

Method Following literature review, a questionnaire was devised containing 89 statements relevant to the topics above. Eight expert members rated their level of agreement with the statements from 1 (not relevant) to 9 (mandatory). Following a group debate about these statements, the experts re-structured some statements then re-rated the questionnaire. Consensus agreement, indifference or disagreement was reached if 7/8 members' scores were in the 7–9, 4–6 or 1–3 ranges respectively.

Results There was consensus agreement for 31/89, consensus indifference for 5/89 and consensus disagreement for 12/89 statements. Consensus was not reached for 41/89 statements. It was agreed daily sputum production would prompt investigation for bronchiectasis, CT was always necessary, and the following factors, support the diagnosis: bronchoarterial ratio >1.0, non-tapering bronchi, thickened airway walls, irreversible changes. It was agreed that a tertiary service should provide: access to HRCT, spirometry, routine and fungal sputum cultures, ciliary function testing; functional antibodies and immunoglobulins for all, antibiotic & hypertonic saline nebuliser challenges, nebuliser loan and maintenance, home iv antibiotic service (preferably by patients in their own homes), portacath insertion, physiotherapy at least annually, access to a dietician, immunologist, microbiologist with an interest in bronchiectasis and pulmonary rehabilitation. Specialist nurses could see selected patients and separate clinics are desirable for patients colonised with pseudomonas. There was indifference to the availability of telephone consultations, posted sputum analysis, iv antibiotic service based in the community (not in patients' home), a patient support group and patient educational sessions. Consensus was not reached regarding if respiratory infection, bronchoarterial ratios of >1.5 or >2.0 or abnormal spirometry are necessary to diagnose bronchiectasis; or if a consultant should see patients at most visits, iv antibiotics could be given by a nurse in the patients home and if access to palliative care was necessary in a tertiary service.

Conclusion Comprehensive consensus statements regarding the diagnostic criteria for bronchiectasis and tertiary service requirements have been formed.

ILD mechanisms

S109 CONTRIBUTION OF ABERRANT MONOCYTE-NATURAL KILLER T (NKT) CELL AXIS TO IMMUNE-PATHOLOGY IN SARCOIDOSIS

doi:10.1136/thoraxjnl-2011-201054b.109

A P Crawshaw, W L Kok, L Denney, L-P Ho. *Human Immunology Unit, Weatherall Institute of Molecular Medicine, University of Oxford, Oxford, UK*

Introduction Sarcoidosis is a multisystem disorder characterisedby an overactive CD4 (T-helper 1) cell response to an undefined antigen, macrophage activation and granuloma formation. It has also been shown that monocytes (precursors to macrophages) are increased in sarcoidosis. We have shown that NKT cells, a specialised subset of immuneregulatory T cells, are deficient in sarcoidosis, and that in NKT knock-out mice, monocytes accumulate at sites of inflammation in both models of influenza A infection and multiple sclerosis. Here, we hypothesise that NKT cells control monocyte function and