

receive active anti-cancer treatment, but the relevance of this observation is obscured by a lack of case-mix adjustment and a high proportion of unrecorded data. We have sought to examine this finding more closely on the 2010 dataset (with less unrecorded data) by performing case-mix adjustment.

Methods Details of all patients from English trusts that were submitted to the NLCA database in 2010 were obtained. We then performed logistic regression analysis based on sex, age, stage and performance status to calculate mutually-adjusted ORs for overall and specific treatments. Since a patient would have reduced opportunity to access an LCNS if their survival were short, a second model was created excluding those patients who had survival of <28 days.

Results Of 30354 in the dataset, 42 were removed due to missing sex (4), in situ disease (2) and occult stage (36). 74.8% were recorded as having been seen by a LCNS, 7.8% were not seen, and in 17.4% the outcome was not recorded. The latter two groups were combined for the remainder of the analysis. ORs for treatment if seen by a nurse in both models are shown below.

Conclusions Contact with a LCNS was associated with increased rates of active treatment, particularly chemotherapy or radiotherapy, but not surgery, and this effect was independent of sex, age, disease stage and performance status. While the LUCADA dataset does not contain detailed information on individual reasons for LCNS assessments, this should be investigated further as there may be important additions to the known benefits LCNS provide to patients. However, regardless of the explanation, all lung cancer patients should have the opportunity to benefit from the expertise of a LCNS.

COPD systemic manifestations and cardiovascular disease

S91 ASPERGILLUS FUMIGATUS SENSITISATION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

doi:10.1136/thoraxjnl-2011-201054b.91

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Background Bacteria and viruses have been implicated in exacerbations of chronic obstructive pulmonary disease (COPD) and bacteria are often isolated in stable state. Whether fungi are also commonly present and associated with clinical and pathological features of disease is uncertain.

Objectives To determine the frequency of filamentous fungal culture and sensitisation to *Aspergillus fumigatus* in COPD and its relationship to clinical outcomes.

Methods Subjects with COPD were recruited from a single centre into a 1-year observational study. Assessments of lung function, allergen testing, and sputum analysis for inflammation, bacterial and fungal cultures were undertaken in COPD subjects and in smoking healthy controls.

Results Fungi were cultured at baseline in 63/128 subjects of which 47/63 were *A. fumigatus*. A fungus was cultured in 2/11 controls (both were *A. fumigatus*). The total sputum cell count, sputum neutrophil % and inhaled corticosteroid dosage were significantly increased in COPD patients with a positive fungal culture compared to those without a fungal culture ($p < 0.05$), but the within subject repeatability of fungal culture between stable visits was low ($K = -0.04$). Sensitisation to *A. fumigatus* was present in 13% of COPD subjects and was associated with worse lung function (FEV_1 % predicted 39% vs 51%; $p = 0.01$), but not related to fungal culture. Positive fungal cultures were present in 42/110 exacerbations and were not associated with bacterial culture or severity of exacerbation.

Conclusions *A. fumigatus* sensitisation is related to poor lung function. Positive fungal culture is a common feature of COPD. The clinical significance of this remains uncertain.

S92 COGNITIVE FUNCTION & CEREBRAL WHITE MATTER TRACT MICROSTRUCTURE IN COPD

doi:10.1136/thoraxjnl-2011-201054b.92

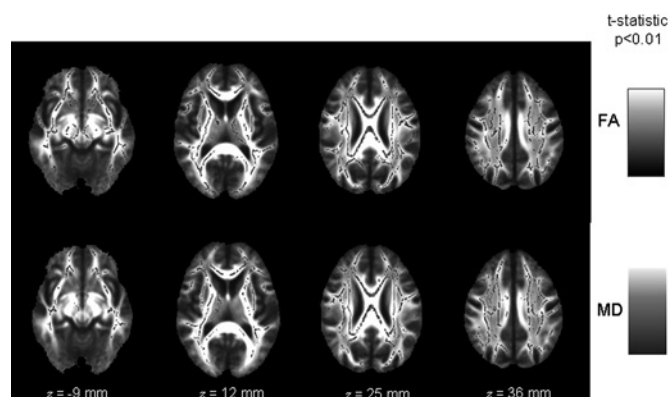
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Rationale There is evidence to suggest that COPD leads to cognitive impairment in patients both with and without hypoxaemia¹; but the pathogenesis remains poorly understood. Also relevant to potential brain pathology in COPD are common vascular comorbidities including hypertension, diabetes and older age. Diffusion tensor imaging (DTI) is a novel MRI technique sensitive to subtle changes in white matter due to vascular damage. This is the first study to investigate white matter microstructure and tract pathology in COPD.

Methods Participants (n=50) completed a full cognitive assessment (including executive function, working memory, episodic memory, processing speed, visuospatial ability) and 3T MRI scan. We compare 25 stable non-exacerbating COPD and 25 age-matched healthy controls. Volumes of grey matter (GMV), white matter (WMV), and white matter lesions (LV), were calculated. DTI data was analysed using tract based spatial statistics (TBSS).²

Results There are significant group differences between COPD patients and controls on all cognitive measures except episodic memory (executive function: $F = 15.39$, $p < 0.001$; working memory: $F = 5.94$, $p = 0.019$; episodic memory: $F = 3.91$, $p = 0.054$; processing speed: $F = 11.64$, $p = 0.001$; visuospatial ability: $F = 10.10$, $p = 0.003$). COPD patients did not differ from healthy controls on measures of normalised GMV ($t = 0.229$, $p = 0.820$) or WMV ($t = -0.727$, $p = 0.471$). Normalised Lesion Volume was significantly greater in patients vs controls ($t = -2.27$, $p = 0.029$). DTI-TBSS revealed lower fractional anisotropy (FA) and higher mean diffusivity (MD) values throughout the brain in COPD patients vs Control subjects. Group differences in white matter integrity were observed throughout the temporal, frontal, parietal and occipital lobes and amounted to 60% of the total FA skeleton. See Abstract S92 figure 1.

Conclusion This is the first paper to demonstrate that white matter integrity throughout the brain is significantly compromised in patients with COPD compared to age-matched Controls. This damage to white matter is also demonstrated by the significant group differences in white matter lesion load. No differences between patients and Controls were observed in brain volume, suggesting that group differences may be related to white matter integrity rather than atrophy.



Abstract S92 Figure 1

REFERENCES

1. Dodd JW, Getov SV, Jones PW. Cognitive function in COPD. *Eur Respir J* 2010; **35**:913–22.