pleural tissue at each time point and summarised as Mean (+/- SD).

Pleural biopsies were obtained at MT in 8/9 patients who underwent complete CE-MRI. Paraffin-embedded tissue was available for 6/8 and stained with Factor VIII and CD34 immunostains. Blood vessel numbers and total vessel area were measured using quantitative image-analysis software (Leica Biosystems, U. K.) and correlated against contrast kinetic parameters (early SI increment (0–4.5 min) and peak SI), using Spearman’s test. Patients were followed-up in a specialist pleural clinic and survival recorded.

Results Mean age was 75 years (+/- 7). 93% (n = 14) were male. Final diagnoses were: MPM (n = 6), lung adenocarcinoma (n = 1), breast adenocarcinoma (n = 1), renal cell carcinoma (n = 1), Benign Asbestos Pleural Effusion (n = 4), rheumatoid arthritis-related effusion (n = 1) and haemothorax (n = 1).

Figure 1 demonstrates relationships identified between contrast kinetic parameters and tissue vascularity. Mean follow-up was 267 (+/- 149) days, over which time mortality for MPM patients exhibiting early peak CE was 100% (n = 2/2) vs. 0% (n = 0/1) for late peak CE (log rank p = 0.2).

Conclusions We have established a functional MRI protocol for use in MPM. Within the limitations of this pilot study, early CE kinetics appear to reflect pleural tissue vascularity. Further work is ongoing to fully assess the diagnostic, prognostic and predictive value of this imaging biomarker.

Basic mechanisms in COPD pathogenesis

PHAGOCYTOSIS BY BLOOD NEUTROPHILS IS NOT ATTENUATED IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

GM Walton, T Purvis, C Chadwick, RA Stockley, E Sapey. University of Birmingham, Birmingham, UK

10.1136/thoraxjnl-2014-206260.52

Rationale All COPD phenotypes have airway neutrophilia but, despite this, bacteria associated infections are common, relate to decline and a significant proportion of patients have persistent airway colonisation. This is suggestive of innate immune dys-function. In vitro studies have shown reduced neutrophil migratory accuracy in COPD (Sapey, Stockley et al. 2011) however, the ability of the neutrophil to contain bacterial infection upon arrival at a site of infection is poorly understood. Literature regarding the phagocytic ability of neutrophils from patients with COPD is conflicting and inconclusive. It is unclear whether responses change depending on the bacterial species present. We hypothesised that neutrophil phagocytosis during COPD is impaired, predisposing patients to increased inflammation and reduced bacterial clearance.

Methods Blood neutrophils were isolated from stable-state COPD patients and healthy age-matched controls (HC). Phagocytosis of both opsonised (with 10% pooled COPD serum) and unopsonised pHrodo<sup>™</sup>-conjugated <i>Staphylococcus aureus</i> bioparticles (SA, n = $S45$

HOW SUCCESSFUL ARE MEDICAL THORACOSCOPISTS AT PREDICTING MALIGNANCY?


10.1136/thoraxjnl-2014-206260.51

Introduction Use of medical thoracoscopy by physicians to diagnose malignant pleural disease is increasing. Thoracoscopy is also used therapeutically to pleurodese (talc poudrage), at the time of biopsy, to minimise pleural effusion recurrence. How-ever, this relies on the physician being confident of their diagnosis (out of 10), whether they predict trapped lung and if they would perform pleurodesis. Gold standard of diagnosis was the histology result.

Results Procedural survey: 16 physicians responded from 12 tertiary referral centres: 13 consultants and 3 specialist registrars. 15 (94%) had performed >10 thoracoscopies each. Four institutions (25%) perform between >10 thoracoscopies per month; 12 (75%) between 1–10 per month. Only 6 (38%) perform thoracoscopies as day cases. All perform rigid rather than flexible thoracoscopies.

Video survey: Of the 20 video clips, the mean number of correct answers was 12.4 (62%). Respondents were more confident of their answers (out of 10) when correct (7.1/10) than incorrect (6.1/10). In cases deemed malignant, 69% would have performed talc pleurodesis; however, 17% would have pleurodesed patients who had benign disease (See Table). Respondents only correctly predicted trapped lung in 2.6/20 cases (13%).

Conclusion Despite being experienced thoracoscopists, only 62% correctly diagnosed malignant or benign on video clips. The majority would appropriately perform pleurodesis, but 17% may have inappropriately pleurodesed benign disease. There are limitations to this small survey using short thoracoscopy clips, but this data suggests caution is required when considering making diagnosis on macroscopic appearance and deciding whether the lung is trapped.

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ENHANCED IL-6/CCL3 SIGNALLING IN THE PLASMA OF PATIENTS WITH COPD

Role of IL-6 in the inflammatory response

IL-6 is a pro-inflammatory cytokine that signals through soluble (sIL-6R/gp80) and membrane bound (gp80) receptors to promote recruitment of mononuclear cells. IL-6 induces expression of CCL3, a monocyte chemokine. Monocytes are precursors of macrophages and dendritic cells. They can be classified into three subtypes according to surface expression of CD14 (LPS receptor) and CD16 (Fc gammaRIII): CD14++CD16-, CD14+CD16+ and CD14-CD16++. We measured plasma levels of IL-6, sIL-6R and CCL3 and determined the chemokine receptor expression profile of circulating monocytes in COPD.

Methods 70 COPD patients and 30 healthy controls comprising 15 smokers (S) and 15 healthy non-smokers (HNS) underwent plasma sampling. Levels of IL-6, sIL-6R and CCL3 were determined by multiplex analysis (MSD) of plasma. Multi-colour flow cytometry was performed on whole blood obtained from 32 COPD patients, 8 S and 8 HNS to measure surface expression levels of chemokine receptors CCR1, CCR2, CCR7, CXCR1 and CX3CR1 on CD14++CD16-, CD14+CD16+ and CD14-CD16++ monocytes.

Results COPD patients had the greatest levels of IL-6 and sIL-6R. CCL3 was not detected in any controls, but was present in a subset of COPD patients. No surface expression of the CCL3 receptor CCR1 was detected on CD14+CD16+. Monocytes of COPD patients was greater than those of HNS (p = 0.04). There were no significant differences in expression levels of other chemokine receptors.

Conclusions We report evidence of enhanced IL-6 signalling in the plasma of COPD patients and increased plasma CCL3 in a subset of individuals from this disease group. Furthermore, there was increased CCR1 expression on COPD monocytes. Enhanced IL-6 may co-ordinate the mononuclear component of the inflammatory response in COPD.