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(EGFR) effecting approximately 26%. These constitutive activating mutations promote tumour cell proliferation and survival, and are inevitably refractory to tyrosine kinase inhibitors (TKIs) as well as current classes of immune checkpoint immunotherapy (ICI). Third generation TKI Osimertinib offers prolonged disease-free survival, however the reasons for the lack of efficacy of ICI in EGFR mutant tumours remain unclear. The cellular immune composition of the tumour microenvironment is increasingly being recognised as a major factor in host-tumour responses. The use of multispectral immunohistochemistry (IHC) is a valuable tool to investigate the cellular composition of these tumours. Here we compare a cohort of EGFR exon 19 deletion (LREA) tumours to stage matched EGFR wild type (WT) tumours by multispectral IHC. Automated staining was performed for CD8, FoxP3, PD-1, CD68, PD-L1 and cytokeratin markers using MOTiF™ PD-1/PD-L1 lung cancer kit, and slides imaged by Vectra®Polaris<sup>™</sup>. Image analysis was performed using QuPath, and marker counts were obtained within tumour and adjacent microenvironment compartments. Immune cells marked by CD8, FoxP3, PD-1, CD68 composed 5%-30% of total cells in the WT tumours, while LREA tumours contained 5% to 15% of total cells (t-test p=0.17). While these immune cells trended to be lower in LREA tumours, further investigation into their distribution in a larger cohort of patients is required. The LREA cohort displayed poorer progression free survival (PFS) following resection and platinum therapy than the WT cohort (468 days vs 622 days, p=0.2), indicating tumour intrinsic mechanisms that result in poorer hosttumour responses. One such mechanism is the immunosuppression of the local microenvironment, and we aim to expand this work to encompass a wider cohort of patient tumours and cellular markers. Disclosure: No significant relationships.

## 7

### Correlation of 18F-FDG-PET/CT metabolic parameters with PD-L1 tumour proportion score (TPS) in resected non-small cell lung cancer (NSCLC)

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**Introduction:** Immune checkpoint inhibitors targeting the PD-1/PD-L1 axis play a significant role in the adjuvant and metastatic treatment of non-small cell lung cancer (NSCLC). PD-L1 is an important biomarker but is dynamic and heterogeneously expressed. Metabolic parameters of 18F-FDG-PET/CT, including SUVmax and total lesion glycolysis (TLG), have been shown to correlate with PD-L1 expression in advanced NSCLC. However, the relationship in early stage NSCLC remains unclear.

**Methods:** This retrospective study evaluated pre-surgery 18F-FDG-PET/CT scans in a cohort of NSCLC patients who underwent primary tumour and hilar/mediastinal nodal resection at a London cancer centre. Pre-surgery scans were assessed for tumoural maximum standardised uptake value (SUVmax), SUVmean, SUVpeak, SULpeak, metabolic tumour volume (MTV), TLG and SUV-based heterogeneity index (HISUV=x/y). PD-L1 TPS was assessed in resected primary tumour samples using 22C3 pharmDx DAKO assay. Unpaired t-test, one-way ANOVA or Kruskall-Wallis tests were applied to determine the relationship between 18F-FDG-PET/CT metabolic parameters and primary PD-L1 TPS score.

**Results:** 100 consecutive NSCLC patients with pre-surgical 18F-FDG-PET/CT and primary NSCLC resection specimens were included. Patients were grouped by primary tumour PD-L1 TPS of <1% (n=45), 1-49% (n=37) and  $\geq$ 50% (n=18). There was no statistical difference between PD-L1 TPS groups for primary tumour SUVmax (p=0.38), SUVmean (p=0.48), SUVpeak (p=0.91), SULpeak (p=0.88), MTV (p=0.06), TLG (p=0.21) and HISUV (p=0.07). Non-significance also held using PD-L1 TPS cut offs of < or  $\geq$ 1% and < or  $\geq$ 50%.

**Conclusions:** In this preliminary study, there was no correlation between 18F-FDG-PET/CT metabolic parameters and PD-L1 TPS score of primary tumour in resected NSCLC. Further work will investigate the use of 18F-FDG-PET/CT in determining PD-L1 expression of nodal metastases in early resected NSCLC.

Disclosure: No significant relationships.

8 Pre-treatment serum albumin is a prognostic biomarker for locally advanced non-small cell lung cancer: extended follow up of patients treated with concurrent chemo-radiotherapy

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Introduction: Serum albumin concentration is a predictor of survival in metastatic non-small cell lung cancer (NSCLC). We present extended follow-up data on a cohort of patients who received radical treatment for locally-advanced NSCLC and the prognostic value of pre-treatment serum albumin concentration. Methods: 93 patients who received cisplatin and vinorelbine or carboplatin and paclitaxel chemotherapy as part of concurrent chemo-radiotherapy for NSCLC at the Edinburgh Cancer Centre between October 2015 and December 2017 were identified using the Chemocare electronic prescribing system. Baseline clinical, demographical and treatment details were obtained from Chemocare and TRAK electronic medical notes. The median time to follow up was 52 months. Overall survival and disease-free survival were assessed as from the date of the last day of treatment to death, and to radiological progression respectively. Kaplan-Meier survival analysis for overall survival and disease-free survival was carried out comparing the low (<35g/L) and normal albumin concentration groups. Statistical significance and hazard ratios were assessed using the Log-rank method.

**Results:** Median overall survival in the low (n=32) vs. normal (n=61) albumin group was 17.5 vs. 46.4 months (HR: 2.446, 95% CI: 1.377–4.344, P=0.0003). Median disease-free survival in the low vs. normal albumin group was 6.3 vs. 36.0 months (HR: 2.165, 95% CI: 1.181–3.971, P <0.0022). Comparing the low and normal albumin groups; median age was 66.5 vs. 64, median BMI was 23.8 vs. 25.2, median performance status 1 in both groups and median stage was IIIA vs. IIIB.

**Conclusions:** In patients who receive radical chemo-radiotherapy for locally-advanced NSCLC, those with pre-treatment serum



Fig. 1 (abstract 8). Overall survival.