Conclusions Individuals with HHT may be protected against primary and/or metastatic lung cancer. This does not appear to be due to reduced smoking habit.

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P10 CARBOPLATIN AND GEMCITABINE IMPAIR NEUTROPHIL PHAGOCYTIC FUNCTION

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Background Lung cancer causes 6% of UK deaths with 20% due to infection[1,2]. The reason is multifactorial with myelosuppression secondary to chemotherapy being a contributor. However, the effect of chemotherapy drugs on the function of peripheral neutrophils has rarely been explored. It is proposed carboplatin and gemcitabine may decrease the function of circulating neutrophils that are present, causing a poorer response to infection by neutrophils that are present. Granulocyte-macrophage colony-stimulating factor (GM-CSF) is used to stimulate neutrophil production in febrile neutropenic patients on chemotherapy, however studies show it also improves mature neutrophil function[3,4]. The aim was to research the effect of carboplatin and gemcitabine on neutrophil phagocytic function and to determine whether GM-CSF reverses any deficit seen in phagocytosis.
Methods Neutrophils isolated from healthy donor blood by dextran sedimentation followed by discontinuous Percoll gradient. Untreated neutrophils compared with those pre-incubated with carboplatin, gemcitabine or both drugs (10^{-4} mM). Phagocytosis assessed by ingestion of serum-opsonised zymosan. Repeated with 30 minute GM-CSF (5 ng/ml) pre-incubation. Apoptosis analysed by flow cytometry. All cells incubated for 2 hours untreated or with single or both chemotherapeutic agents (10^{-4} mM) or phorbol 12-myristate 13-acetate (PMA) for 30 minutes. Cells stained with APC-Annexin V to detect apoptosis and propidium iodide to distinguish early and late apoptotic cells.
Results A significant decrease in percentage phagocytosis occurred with chemotherapy drugs compared to untreated neutrophils: carboplatin $p = < 0.001$, gemcitabine $p = < 0.01$, carboplatin and gemcitabine $p = < 0.05$. GM-CSF caused a significant increase in percentage phagocytosis when gemcitabine caused a deficit ($p = < 0.05$) with a 9–13% increasing trend seen in other conditions. No difference in apoptosis occurred between untreated (median 3.9%) and chemotherapy treated neutrophils (median: carboplatin 4.4%, gemcitabine 4.9%, carboplatin and gemcitabine 3.6%).
Conclusions This study demonstrated that phagocytosis is impaired by carboplatin and gemcitabine in healthy volunteer blood. Therefore it is proposed a reduced response to infection may also occur in lung cancer patients given these drugs, contributing to mortality. There was no difference in apoptosis, suggesting an alternative mechanism of action. Further study is required to explore the mechanism of action and the effect of GM-CSF on phagocytosis following chemotherapy.

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P11 TYROSINE KINASE INHIBITOR USE IN PULMONARY ADENOCARCINOMA

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Introduction Personalised treatments are becoming increasingly important in medicine, and the use of tyrosine kinase inhibitors (TKI) in the management of the subset of patients with pulmonary adenocarcinomas which express a mutation in the epidermal growth factor receptor (EGFR), is one example of this approach. However, although this cell type is common, in the UK only a small proportion of patients express the EGFR mutation and there is little published data on the number of patients receiving TKI therapy. Since 2010 we have routinely performed EGFR testing on pulmonary adenocarcinoma samples at our large cancer unit and were interested to assess our use of TKIs.
Methods We reviewed all cases of pulmonary adenocarcinoma since EGFR testing began, looking for the proportion EGFR positive, their performance state (PS), and what treatments were offered.
Results Of 241 cases of primary pulmonary adenocarcinoma, 54 (22%) had insufficient material available for mutation testing. Of the remaining 187, 23 (12%) were EGFR positive (mean age 76 [range 47–92], 18 female). Although 16 were PS ≤2, only 5 patients had stage 1A cancer with 13 having advanced disease (stage 3B/4). Seven patients underwent attempted curative surgery and 4 patients with poor PS were managed with best supportive care only. Twelve patients (52% of the EGFR positive group and 6.4% of the tested cohort) received a TKI (mean age 74 [50–92], PS ≤2 in 8, 10 female), and in 3 of these it was the only treatment modality offered (1%).
Conclusions We have shown that our cohort of patients with adenocarcinoma had a higher rate of EGFR mutation than expected, and that half of these received targeted biological therapy with tyrosine kinase inhibitors. Importantly TKI therapy was the only treatment modality available for patients who would have previously been untreated. This work emphasises the need to obtain a histological diagnosis in patients with lung cancer, to ensure that all possible treatment modalities can be considered.

P12 TEMPORAL TRENDS, CAUSES & RISK FACTORS FOR HOSPITAL ADMISSIONS IN INCURABLE LUNG CANCER

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Introduction Little is known about hospital admissions (HAs) following a diagnosis of incurable lung cancer (LC). This study sets out to identify temporal trends for HA in this group of vulnerable patients in addition to exploring the reasons behind and potential risk factors for HA.
Methods All new LC diagnoses for 2009–2011 (n = 565) were identified, from which 1:4 were selected randomly (n = 142). Records were reviewed and those patients treated with curative intent or diagnosed and died in same HA were excluded from analysis. Basic demographic data were collected including comorbidity score, stage, histology and LC was classified as either central (mediastinum to origin of lobar bronchi and vessels) or