Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Conclusion: There is concern for increased risk and severity of CIP after COVID-19 infection due to 'priming.' A similar case report mentioned rapidly progressive, fatal CIP in a patient with small cell lung cancer on PD-1 immunotherapy following COVID-19. Our patient's case was further complicated by hypogammaglobulinemia, leading to a persistent COVID-19 infection and inflammatory state. IVIG has been described as a potential treatment for refractory CIP with low level supportive evidence. This case report is among the first to describe severe CIP with COVID-19 which had a dramatic response to IVIG therapy resulting in significantly decreased pulmonary fibrosis and oxygen requirements. **Keywords:** checkpoint inhibitor pneumonitis, NSCLC, covid-19

P34.06
Antiproliferative Effects of Paclitaxel on PC9-MET Cells During the Coronavirus Disease 2019 Pandemic

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Introduction: Coronavirus disease 2019 (COVID-19) poses a great challenge for the treatment of cancer patients. It presents as a severe respiratory infection in aging individuals, including lung cancer patients. COVID-19 may be linked to the progression of aggressive lung cancer. In addition, the side effects of chemotherapy, such as chemotherapy resistance and the acceleration of cellular senescence, can worsen COVID-19. Given this situation, we investigated the role of paclitaxel (a chemotherapy drug) in the cell proliferation, apoptosis, and cellular senescence of gefitinib-resistant non-small-cell lung cancer (NSCLC) cells (PC9-MET) to clarify the underlying mechanisms. **Methods:** PC9-MET cells were treated with paclitaxel for 72 h and then evaluated by a cell viability assay, DAPI staining, Giemsa staining, apoptosis assay, a reactive oxygen species (ROS) assay, SA-β-Gal staining, a terminal deoxynucleotidyl transferase dUTP nick-end labeling assay and Western blotting. **Results:** Our results revealed that paclitaxel significantly reduced the viability of PC9-MET cells and induced morphological signs of apoptosis. The apoptotic effects of paclitaxel were observed by increased levels of cleaved caspase-3 (Asp 175), cleaved caspase-9 (Asp 330) and cleaved PARP (Asp 214). In addition, paclitaxel increased ROS production, leading to DNA damage. Inhibition of ROS production by N-acetylcysteine attenuates paclitaxel-induced DNA damage. Importantly, paclitaxel eliminated cellular senescence, as observed by SA-β-Gal staining. Cellular senescence elimination was associated with p53/p21 and p16/pRb signaling inactivation. **Conclusion:** Given these findings, paclitaxel may be a promising anticancer drug and offer a new therapeutic strategy for managing gefitinib-resistant NSCLC during the COVID-19 pandemic. **Keywords:** Paclitaxel, PC9-MET, covid-19

P35 MESOTHELIOMA, THYMOMA AND OTHER THORACIC MALIGNANCIES - BASIC RESEARCH FOR PLEURAL MESOTHELIOMA

P35.01
YB-1 is a Key Player in Aggressive Behaviour and Chemoresistance in Mesothelioma

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Introduction: Malignant pleural mesothelioma (MPM) is characterised by aggressive growth and frequent resistance to chemotherapy, poor prognosis for patients and limited therapeutic options. To establish potential new therapy targets, a better understanding of the MPM biology underlying these malignant behaviours is crucial. One potential candidate is the multifunctional oncoprotein YB-1, which is often overexpressed in various cancers and associated with aggressiveness, metastasis and poor outcome. **Methods:** YB-1 expression was analysed by qPCR or western blot in cell lines, and in patient material by immunohistochemistry. YB-1 overexpression was achieved by a doxycycline-inducible, retroviral construct, which was stably introduced into MPM cell lines. YB-1 knockdown was performed using YB-1 specific siRNA. For inhibition of YB-1 phosphorylation, the RSK inhibitor BI-D1870 was used. Effects of drug interaction (CI values) were calculated using the CompuSyn software. Cell migration was assessed by live cell videomicroscopy followed by manual single cell tracking and analysis using ImageJ and DiPer software, respectively. For the zebrafish model, RFP-expressing MPM cells were injected into 48 hour old larvae, imaged after 1 and 2 days and the number of cells in the tail was manually counted. **Results:** We previously reported that YB-1 is up-regulated in MPM cell lines compared to non-malignant controls. In this study we analysed MPM tissue (n>120) and normal pleura (n=3) specimens for YB-1 expression. While all control samples were negative, tumours showed a heterogeneous YB-1 expression. YB-1 knockdown decreases MPM cell migration and invasion, hence we evaluated the impact of YB-1 overexpression on these phenotypes. MPM cell lines (n=6) which overexpress YB-1 in a doxycycline-inducible manner showed significantly increased cell scattering and migration. Furthermore, YB-1 overexpression lead to significantly higher migratory capacity in vivo using a zebrafish xenograft model. Additionally, when tumour spheroids were co-cultured with endothelial cells, YB-1 overexpression led to a significantly more extensive formation of gaps in the endothelial layer. Finally, since we found that YB-1 knockdown not only decreases cell growth in vitro and in vivo but also decreases the expression of LRP1 and ABCC1, two genes involved in drug resistance, we combined YB-1 knockdown as well as inhibition of YB-1 phosphorylation via an RSK inhibitor with cisplatin chemotherapy. Our data showed highly synergistic combination effects (CI values: 0.1 — 0.5) for several cisplatin doses in combination with either YB-1 siRNA or the RSK inhibitor. **Conclusion:** In this study we report a deregulated YB-1 expression in MPM tumor specimens compared to non-malignant controls. Our data show an important role of YB-1 in the regulation of cell migration and invasion in vitro and in vivo, which are key characteristics of MPM. Additionally, YB-1 knock down as well as pharmacological inhibition of YB-1 phosphorylation not only reduce MPM cell growth but also sensitise cells to cisplatin chemotherapy. These findings contribute to a better understanding of the biology of MPM and highlight YB-1's potential as a therapeutic target. **Keywords:** YB-1, Mesothelioma

P36 MESOTHELIOMA, THYMOMA AND OTHER THORACIC MALIGNANCIES - CASE REPORTS IN MESOTHELIOMA AND THYMOMA

P36.01
Primary Acinic Cell Carcinoma of Bronchial Ground Origin: A Case Report

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Introduction: Primary acinic cell carcinoma of the lung is an extremely rare disease. Also known as Fechner's tumor, this condition was first reported by Fechner et al. in 1972. The accurate diagnosis of primary pulmonary acinic cell carcinoma is difficult and relies on comprehensive evaluation of clinical, histological and immunohistochemical