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| **Increased PAFR and ICAM-1 in IPF: implications for microbial pathogenesis** |
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| **Introduction/Aim:** Idiopathic pulmonary fibrosis (IPF) is an incurable lung condition characterised by excessive scarring, and its origins remain unknown. The presence of both bacterial and viral co-infections can worsen the progression of the disease. These pathogens use adhesion molecules like the platelet-activating factor receptor (PAFR) and intercellular adhesion molecule 1 (ICAM-1) to access and infect cells, contributing to the development of infections. This study aims to determine the expression of PAFR and ICAM-1 in small airways and lung parenchyma of patients with IPF compared to normal lungs to explain if IPF patients are more susceptible to infections.**Methods:** We conducted immunohistochemically staining for PAFR and ICAM-1 on surgically resected lung tissue obtained from IPF patients (n = 11) and normal controls (n = 12). The assessment of PAFR and ICAM-1 expression was presented as a percentage in the small airway epithelium, type 2 pneumocytes and alveolar macrophages within the lung parenchymal area. All image analysis was carried out using Image Pro Plus 7.0 software.**Results:** Compared to normal controls, there was a significant increase in PAFR expression in the small airway epithelium (*p* < 0.0001), type 2 pneumocytes (*p* < 0.05) and alveolar macrophages (*p* < 0.05) in IPF. Similarly, a comparable pattern was observed for ICAM-1 expression in the small airway epithelium (*p* < 0.0001), type 2 pneumocytes (*p* < 0.0001) and alveolar macrophages (*p* < 0.0001) from IPF patients compared to normal controls. Furthermore, the ratio of positively expressed type 2 pneumocytes and alveolar macrophages was higher in IPF than in normal controls.**Conclusion:** This study presents the first evaluation of PAFR and ICAM-1 expression within small airway epithelium, type 2 pneumonocytes and alveolar macrophages in individuals with IPF. These findings may offer insights into mitigating the microbial impact and enhancing the ability to manage the progression of the disease. **Grant Support:** Clifford Craig Foundation and Lung Foundation Australia.  |