**Idiopathic pulmonary fibrosis: a study using volumetric imaging and functional data in a computational lung model**

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**Introduction**

Idiopathic pulmonary fibrosis (IPF) is the most aggressive and frequent form of idiopathic ?interstitial pneumonias with unknown cause. It is a lethal fibrosing lung disorder that occurs primarily in middle-aged and elderly adults. The progression of IPF is variable between individuals and no established quantitative tools exist to indicate the likely progression of the disease. The aim of this work is to develop a new quantitative method of the IPF that integrates information from volumetric imaging, pulmonary function tests, and computational models for lung function; and to conduct a quantitative assessment of IPF tissue characterisation and disease development over time in comparison to normal adults of the same age.

**Method**

Tissue regions in HRCT images from 8 patients with IPF (aged X-Y) were quantitatively analysed and compared to 30 normal subjects aged X-Y. In the IPF cohort tissue abnormalities were classified using CALIPER (Computer-Aided Lung Informatics for Pathology Evaluation and Ratings) software. Mean tissue density (TD), volume and spatial distribution of ...were calculated. The classified data were mapped to a statistical shape model, which allows consistent comparison between patients or within one patient at different time points. A computational representation airway tree was generated for each individual patient and deformed to fit the lung shape of each time point. A principal component analysis (PCA) and fractal dimension was applied to assess lung shape and tissue heterogeneity differences between IPF lungs andnormal aging lungs. These quantitative tissue biomarkers were used to drive an ununiformed tissue property based stress distribution of IPF lung, which led to a further simulation of mechanical ventilation and gas exchange in a computional model

**Result**

Fibrosis usually has consistently higher TD (0.34/0.41 for reticular/ground-glass) compared to normal tissue (0.28), and presents predominantly in lower lobes basally and peripherally. In contrast, emphysema has lower density (0.08) and appears predominantly in upper lobes. Most IPF patients experience a decrease lung volume over time (11.85% off). The lung shape between IPF lung and old normal ones are slightly different with p-values of 0.001, 0.194 and 0.454 respectively for the first three modes based on PCA analysis. Stress distribution indicates a continuous correlation over time with lung volume change and disease distribution. V-Q mismatching and impaired gas exchange occurs in IPF lungs even in CT evaluated ‘normal’ tissue.

**Conclusion**

An image based analysis of IPF tissue characterisation and distribution combined with functional modelling techniques provides a set of markers for lung function in IPF. This could be a potential tool to help with assessment of IPF progression at the early stage of this disease.