

## Defining COPD phenotypes through imaging

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Chronic Obstructive Pulmonary Disease (COPD) is a complex disease in which airflow obstruction can be caused by different mechanisms, including the one brought about by bronchial wall thickening (chronic bronchitis), parenchymal destruction (emphysema), or mixed type emphysema-bronchitis. Different COPD phenotypes accounts for the great heterogeneity in the clinical presentation, progression, and pharmacological treatment response. Imaging can provide a significant contribution to the identification of COPD phenotypes with potential prognostic and therapeutic implications. CT semiotics of basic alterations is useful for identifying COPD phenotypes and the significance of their quantitative assessment. Emphysema is classified as centrilobular, panlobular, and paraseptal. Moreover, a sub-group in which paraseptal emphysema is combined with fibrosis (CPFE) has been characterized. Quantitative CT assessment of emphysema provides an objective measure of the extent of such parenchymal disease, correlates well with the anatomic-pathologic data, and is predictive of the degree of obstruction of expiratory airflow. The progression of the emphysema's severity quantitatively defined with CT is associated with a deterioration of the health status and with an increase in mortality.

The second group of alterations related to the pulmonary disease has its epicenter in the airways such as trachea, main bronchi, segments and subsegments observable and measurable with CT up to the fifth and sixth generation: among them are bronchiectasis, the thickening of the bronchial walls, bronchial diverticula, saber-sheath trachea and tracheobroncomalacia. Bronchiectasis can be classified as cylindrical, varicose or cystic. The thickening of the bronchial walls is caused by inflammation and remodeling of the airway walls, by squamous metaplasia of the bronchial epithelium, by the thickening of basement membrane and smooth muscle, by the hyperplasia of submucosal cells and glands, and by a cartilage deficit.

'Bronchitis' phenotypes are: small airway predominant disease (air trapping, mosaic perfusion, micronodular opacities), predominant bronchial disease (bronchi from the third to the sixth generation), or large bronchi predominant disease (trachea, main and lobar bronchi); bronchial diverticula are signs of severity. Thickening of proximal airway walls assessed by quantitative CT is inversely related to pulmonary function, and correlate with the burden of the small airway disease and of exacerbation frequency. These correlations are stronger if fourth- and fifth-generation segmental bronchi are considered. Moreover, quantitative CT imaging can adequately assess low attenuation areas, which provide a measure of air trapping correlated with the involvement of the small airways.

Emphysema susceptible smokers (SS) phenotype as opposed to normal smokers with no CT emphysema can be identified by changes of heterogeneity of pulmonary perfused blood volume (PBV) assessed via DECT dual energy CT (DECT) after sildenafil (1).

Multi detector-row computed tomography (MDCT) showed that tiotropium dilated the non-small-airway in COPD patients, an effect which is increased by adding budesonide/formoterol combination. CT imaging can evaluate drug therapeutic effect and may provide additional insights into pharmacotherapy for COPD (2).

Imaging techniques provide a powerful non-invasive tool for identifying structural COPD phenotypes and are potentially useful for assessing their response to pharmacological treatment.

### References

1. Hoffman E et al. Scientific Session 13-4 3<sup>rd</sup> World Congress of Thoracic Imaging June 8-11; 2013
2. Yasui H et al. *Pulm Pharmacol Ther* 2013;26:336-341.