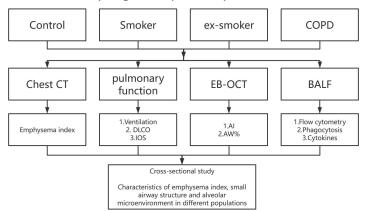
Alveolar macrophage function correlated with small airway remodeling in COPD

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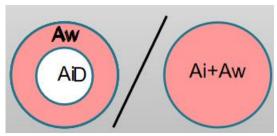
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Objective: Chronic obstructive pulmonary disease is characterized by airway inflammation and remodeling, small airway lesions are the most obvious and earliest involved. However, the association between airway inflammation and airway remodeling remains unclear. To explore the correlation of alveolar macrophages (AM) with small airway structure and its potential role in COPD pathogenesis.

Methods: We performed endobronchial optical coherence tomography (EB-OCT) and bronchoalveolar lavage (BAL) form patients with COPD, heavy-smokers with small airway disorders and healthy controls. The luminal area (Ai) and airway wall area% (Aw%) from the 3rd to 9th generation of bronchi were measured using EB-OCT. BAL cells were stained with anti-CD68 for total macrophages, anti-CD163 for M2, and anti-CD68 for M1 macrophages, respectively.



Multiplex ELISA was conducted to measure cytokines in BALF (IL-1 β).



Results: Patients with COPD had higher expression of IL-1β than control, but similar with that of heavy smokers (2.5±0.6 vs 11.1±4.5 vs 4.8±0.3). The M1/M2 ratio were comparable among COPD, and control (78.3±37.5 heavy-smoker 189.0±89.0 vs 6.9±4.2). Further, the EB-OCT measurement indicated that patients with COPD had greater small airway wall thickening (Aw%7-9), but not smaller airway caliber (Ai7-9), than that in heavy-smokers and control subjects (43.8±0.8 vs 33.1±7.4 vs 55.3±5.5). It was worth noting that Aw%7-9, rather than Ai7-9, was negatively associated with M1/M2 ratio (r=0.888, P=0.001), whereas the correlation with IL-1\beta did not achieve statistical difference.

Conclusion: The magnitude of small airway remodeling, assessed by EB-OCT measurement, was correlated with the phenotype and function of alveolar macrophage in patients with COPD.