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Title: Expansion of alveolar-lymphoid interfaces in lungs of patients with COPD

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Body: Rationale: Although adaptive immune responses are critical for combating distal airway infections in COPD lungs, the structural basis for alveolar antigen uptake has remained poorly investigated. This study investigates the interface between alveolar lumen and the adaptive immune system at different severities of COPD. Methods: Lung resections (n=31) from mild (GOLD I), moderate-severe (GOLD II-III), and very severe (GOLD IV) COPD were subjected to detailed histological assessment of components of the adaptive immune system in distal lung. Never-smokers and non-COPD smokers served as controls (n=15). Results: In COPD, both numbers and mean size of lymphoid aggregates were increased in small airways, pulmonary vessels and the alveolar parenchyma. Irrespective of anatomical localization, the vast majority (88%) of the aggregates had direct contact with alveolar luminal spaces (37% of the aggregate perimeter). In advanced COPD, the epithelium at alveolar-lymphoid interfaces had transformed into a significantly higher columnar phenotype (p=0.02) that, apart from expressing immune-regulatory molecules, contained increased numbers of langerin-positive dendritic cells (p=0.02). Also, the total alveolar-lymphoid interface and interface-associated dendritic cells were increased (p=0.02 and p=0.002, respectively). Conclusions: The progression of COPD is linked with an expansion and remodeling of alveolar lumen-lymphoid interfaces. These alterations, which predict an increased capacity to respond to alveolar antigens, correlated with lung function parameters and may thus contribute to the aggravated inflammation in COPD lungs.