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CARTILAGINOUS AIRWAY INFLAMMATION IN SMOKERS. J.B. Mullen, B.R. Wiggs, J.L. Wright, P.D. Pare and J.C. Hogg. UBC Pulm. Res. Lab., St. Paul's Hospital, Vancouver, B.C., Canada.

To assess the role of cartilaginous airway inflammation in the pathogenesis of COPD, we studied 40 subjects 59.3 ± 9.4 (SD) years old with a smoking history of 1081.9 ± 733.4 (SD) cigarette years prior to lobectomy for coin lesions. FEV₁, FVC, MMFR, TLC, RV and FRC were measured pre-operatively and the surgical specimens were processed for quantitative morphology. Cartilaginous airways were graded for inflammation involving the mucosa, glands, gland ducts, interstitium, smooth muscle and nerves and the epithelium was graded for goblet and squamous cell metaplasia (1). Airway structural components were measured using a digitizing board. The severity of the small airway pathology was established (2) and the emphysema estimated (3). Mucosal inflammation accounted for 39%, gland 23%, gland duct 16%, interstitial 15%, smooth muscle <3%, and nerve <3% of the total inflammatory score obtained by summing all six values. The bronchial wall consisted of (mean ± SD) 74.7 ± 6.9% connective tissue, 19 ± 6% cartilage, 3.9 ± 2.3% glands, and 2.1 ± 1% muscle. The total inflammatory score correlated positively with the proportion of glands (p <.01) but not with the other structures. Airway inflammation also correlated positively with both goblet cell (p <.01) and squamous cell (p <.001) metaplasia. The severity of the inflammatory reaction in the cartilaginous airways increased in relation to the amount smoked (p <.05). There was no relationship between the severity of central airways inflammation and peripheral airways disease, severity of emphysema or tests of pulmonary function. We conclude that cigarette smoking produces an inflammatory reaction in the cartilaginous airways which is most active in the airway mucosa where it correlates with increasing goblet cell and squamous cell metaplasia as well as mucous gland size.

1. N Engl J Med 1978; 298: 1277-81.
2. Am Rev Respir Dis 1983; 127: 474-77.
3. Hum Pathol 1970; 1: 215-26.

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APPENDIX 3

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1e. Detection of Small Airways Disease

WRIGHT, L.M. LAWSON, P.D. PARÉ, S. KENNEDY, B. WIGGS, and J.C. HOGG

SUMMARY In order to investigate the relationship between pulmonary function and disease of the membranous and respiratory bronchioles, we studied 96 patients who required lobectomy for removal of a solitary pulmonary nodule. A subgroup of patients with forced expiratory volume in one second (FEV₁) greater than 80% predicted were further analyzed to determine if abnormalities in tests designed to detect peripheral airways disease actually correlated with the pathology found in these airways. Analysis of the data shows that inflammation in both respiratory and membranous bronchioles, goblet cell metaplasia of the epithelium in membranous bronchioles, and decreasing muscle in the respiratory bronchioles are the pathologic features that are associated with deterioration of the FEV₁. When the FEV₁ is greater than 80% of the predicted value, inflammation of the respiratory bronchioles and fibrosis of both membranes and respiratory bronchioles increase with decreasing FEV₁. Tests of specialized pulmonary function appear to correlate with epithelial pathologic parameters of membranous bronchioles and inflammation and fibrosis of respiratory bronchioles. When patients with FEV₁ greater than 80% predicted were subdivided according to the number of abnormal tests of small airways function, there was a significant increase in inflammation of the walls of respiratory bronchioles when 2 tests were abnormal and increases in both airway wall and intraluminal inflammatory cells as well as increased wall fibrosis when 3 tests were abnormal. We conclude that when the FEV₁ is greater than 80% predicted, abnormalities in the tests for small airway disease reflect pathologic changes in the respiratory bronchioles.

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