CONTRIBUTION OF BONE MARROW-DERIVED FIBROCYTES TO SILICOSIS

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Introduction Exposure to free silica induces silicosis and its mechanism is less clear. Myofibroblast is regarded as a primary effector cell which is highly synthetic for collagen and lead to extensive fibrosis in lung. However, its origin is still controversial. Fibrocyte is one source of myofibroblast and proved to play a pivotal role in lung fibrogenesis, but whether fibrocyte participates in the process of silicosis is rarely reported. Therefore, the present study was designed to investigate the contribution of fibrocytes in silicosis.

Methods The rat model of silicosis was established by single intratracheal instillation of SiO2 solution (100 mg/0.5 ml/rat). HE and Masson staining were used to evaluate histopathology and collagen deposition. Flow cytometry and immunofluorescence were performed to detect number of fibrocytes and contribution to myofibroblasts.

Results During experimental silicosis (from week 1, 2, 3, 6, 9, 12), the number of fibrocyte is markedly increased in peripheral blood and lung tissue by using flow cytometry. Furthermore, the number of fibrocyte is markedly increased in peripheral blood During experimental silicosis (from week 1, 2, 3, 6, 9, 12). myofibroblasts, respectively. The trend analysis of different sources of myofibroblast during silicosis indicates that fibrocyte and lung type II epithelial cell derived myofibroblast play an important role at the early stage of silicosis (week 1 to week 3), while resident lung fibroblast-derived myofibroblast mainly do a predominant role during fibrosis formative period (week 6 to week 12).

Discussion Taken together, these data suggest that fibrocyte is involved in the pathogenesis of silicosis and it may be useful as an indicator for disease activity. Different sources of myofibroblasts play roles in different phases of silicosis.

CHRONIC OBSTRUCTIVE PULMONARY DISORDER IN CHRONICALLY EXPOSED TO SILICA: EXPERIENCE OF HOSPITAL DAS CLINICAS DA UFMG

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Introduction There is no description in literature for relationship between exposure to silica and occurrence of COPD in Brazilian population. This work aims to evaluate the importance of this exposure as a predisposing factor for chronic obstructive ventilation disorder (OVD) and associate the time of exposure with the FEV1/FVC ratio.

Methods Serie of cases with 1389 patients, from 1984 to 2017. The cases were evaluated in relation to: chest X-ray, spirometry, clinical and occupational history. The spirometry classification was based on Brazilian guidelines.

Results All patients analysed were exposed to silica (median exposure: 15 years). The median age was 46.0 years (97% male). Smokers or ex-smokers accounted 59.1%. Silicosis was diagnosed in 44.0%, current tuberculosis or sequel 12.8%, asthma 5.6%; Autoimmune diseases 2.9% and heart diseases 4.0%. Spirometrics of 975 patients were analysed (OVD: 38.3%). After exclusion of TB and asthma patients, the prevalence of OVD decreased to 33.5%. Excluding silicosis patients, the prevalence of disorder was 24.9%. In the last subgroup, excluding smokers and ex-smokers, the prevalence of OVD was 15%. A subgroup with homogeneous exposure (165 lapi- daries of semiprecious stones) was selected to evaluate the contribution of smoking (years/packet) and time of exposure to silica in the FEV1/FVC ratio. Linear regression model was applied. Each year of exposure to silica showed a worsening in FEV1/FVC ratio of 0.002 and each year/packet had a 0.003 reduction (p-value 0.034 and 0.000, respectively).

Conclusion A prevalence of 15% of OVD was demonstrated in individuals whose only risk factor was exposure to silica. In the subgroup of homogeneous exposure it was possible to establish an exposure unit that could be compared with years/packet of cigarettes. The importance of both, independently, for the occurrence of OVD has been demonstrated.