山东大学学报(医学版) 2011, 49(4) 17- DOI: ISSN: 1671-7554 CN: 37-1390/R

论文

慢性阻塞性肺疾病及糖尿病大鼠肺泡灌洗液SIgA的含量测定

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摘要：

目的 建立慢性阻塞性肺疾病（COPD）、糖尿病（DM）和慢性阻塞性肺疾病并糖尿病（COPD+DM）大鼠模型，测定其肺泡灌洗液（BALF）中分泌型免疫球蛋白A（SIgA）含量，观察上述不同疾病时BALF中SIgA含量的变化。方法 将48只大鼠随机分成COPD组、DM组、COPD+DM组和正常组，每组12只。大鼠实验环境中预饲养1周后，建立糖尿病模型，1周后用气道内滴入脂多糖(LPS)和被动吸烟的方法建立慢性阻塞性肺疾病大鼠模型。大鼠成模后，取其右侧肺组织，做常规病理检查。同时对左侧肺组织进行肺泡灌洗，并采用酶联免疫吸附测定法（ELISA）测定肺泡灌洗液中SIgA的含量。结果 ① 各组大鼠肺组织病理学改变：正常组大鼠肺泡结构正常，未见炎性细胞浸润。COPD组大鼠支气管黏膜上皮脱落，肺间质及气道管壁有炎性细胞浸润，肺泡腔扩大，部分融合成肺大疱。DM组大鼠支气管上皮纤毛有倒伏现象，基膜下有炎性细胞浸润。COPD+DM组支气管黏膜上皮脱落严重，肺间质及气道管壁有大量的炎性细胞浸润，肺泡结构破坏，部分融合成肺大疱；② 各组大鼠肺泡灌洗液中SIgA含量测定结果：与正常组比较，COPD组的SIgA含量显著下降（P<0.01）,DM组和DM+COPD组的SIgA含量升高（P<0.01）；与COPD组比较， DM组、COPD+DM组SIgA含量显著升高（P<0.01）；DM组和COPD+DM组相比，差异无统计学意义（P>0.05）。结论 SIgA在肺部黏膜免疫中发挥正向的防御作用。COPD大鼠肺灌洗液中的SIgA水平较正常组明显减低，揭示气道黏膜局部防御功能下降。DM大鼠及COPD+DM大鼠肺灌洗液中的SIgA增高，可能与晚期糖基化产物免疫应答和对病原微生物的代偿性免疫增高有关。

关键词： 慢性阻塞性肺疾病；糖尿病；模型，动物；支气管肺泡灌洗液；分泌型免疫球蛋白A；大鼠，Wistar

Determination of the concentration of SIgA in BALF in rats with chronic obstructive pulmonary disease and diabetes mellitus

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Abstract:

Objective To determine the concentration and differences of SIgA in bronchoalveolar lavage fluid (BALF) in the rat model of chronic obstructive pulmonary disease（COPD）, diabetes mellitus （DM）and chronic obstructive pulmonary disease plus diabetes mellitus（COPD+DM）. Methods 48 rats were divided into four groups： the normal group, the COPD group, the DM group, and the COPD+DM group. There were 12 rats in each group. Bred in the experimental environment for one week, the DM model was established. One week later, the COPD model was established by cigarette inhalation and intratracheal LPS exposure. After the rats were sacrificed, pathological changes of the right lung were observed, and the left lung was lavaged to determine the concentration of SIgA in BALF by ELISA. Results ① Pathological changes of lung tissue： in the normal group, the structure of alveolus was normal and no inflammatory cell infiltration was found. In the COPD group, the bronchial epithelium was ablated, and the interstitialpulmonary and airway walls were infiltrated with inflammatory cells, and the ruptured and enlarged alveolus and bullaes were identified. In the DM group, there was inflammatory cell infiltration under the basement membrane and the lodging cilias of bronchial epithelium. In the COPD+DM group, the bronchial epithelium was severely ablated, and the interstitial-pulmonary and airway walls were infiltrated with abundant inflammatory cells.The structure of the alveolus were destroyed and some formed into bullaes. ② Concentration of SIgA in BALF： Compared with the normal group, the concentration of SIgA in the COPD group decreased (P<0.01), but increased in the DM group and the COPD+DM group (P<0.01). Compared with the COPD group, the concentration of SIgA in the DM group and the COPD+DM group significantly increased (P<0.01). There was no difference in the concentration of SIgA between the DM group and the COPD+DM group (P>0.05). Conclusion SIgA plays a defensive role in pulmonary mucosal immunity. Compared with the normal group, the significant decrease of SIgA in the COPD group indicates that the pulmonary mucosal immunity declines in the COPD group. The increased concentration of SIgA in the DM and COPD+DM group may be attributed to the immune response of advanced glycosylation end products (AGEs) and the increased compensatory immunity to pathogenic microorganisms.

Keywords: Pulmonary disease, Chronic obstructive； Diabetes mellitus； Models, Animal； Bronchoalveolar lavage fluid； Immunoglobulin A, Secretory； Rats, Wistar

收稿日期 2010-06-04 修回日期 网络版发布日期

DOI:

基金项目:

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