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**SPUTUM EOSINOPHILS, EXHALED NITRIC OXIDE, DURING LATE ASTHMATIC REACTION IN PATIENTS WITH WESTERN RED CEDAR ASTHMA.**

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We investigated changes in sputum eosinophils and exhaled nitric oxide (NO) before and at intervals after inhalation challenge tests with plicatic acid (the compound responsible for occupational asthma due to Western red cedar). Of the 17 subjects who underwent challenge, 9 had a positive asthmatic reaction (responders) while 8 had a negative reaction (nonresponders). At 6 and 24 hours after challenge with plicatic acid, there was a significant increase in eosinophils in induced sputum among responders; the changes in eosinophils was inversely related to changes in FEV1 at 6 hours. Levels of exhaled NO increased at 24 hours after challenge with plicatic acid in both responders and nonresponders, significant only in nonresponders. Such changes were not found after methacholine challenge in both responders and nonresponders. No correlation was found between the increase in exhaled NO and the magnitude of functional changes in the airways. There were significant correlations between the degree of sputum eosinophilia and the level of exhaled NO before and after plicatic acid as well as after methacholine challenge.

We conclude that eosinophilic airway inflammation occurs during late asthmatic reaction induced by plicatic acid in patients with Western red cedar asthma as reflected by increased in sputum eosinophils. The usefulness of measuring these 2 parameters in the clinical evaluation of patients with suspected occupational asthma caused by low molecular weight compounds has yet to be determined.

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**DO NEUTROPHILS PLAY A ROLE IN THE BRONCHIOLITIS OBLITERANS SYNDROME POST-LUNG TRANSPLANT?**

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Although lung transplantation is an accepted therapeutic strategy for many end-stage pulmonary diseases, Bronchiolitis Obliterans Syndrome (BOS), a form of chronic lung rejection, remains the major constraint on its long-term success. Neutrophils have been associated with fibrosing lung conditions and are increased in the bronchoalveolar lavage (BAL) of BOS patients. This study examines neutrophil accumulation in the BAL, airway wall and lung parenchyma, as well as BAL IL-8 levels, in normal controls and lung transplant recipients (LTR) with and without BOS. Bronchoscopy involved endobronchial biopsy (EBB), BAL, and transbronchial biopsy (TBB) sampling. Tissue neutrophils were identified by neutrophil elastase staining on 3 µm paraffin biopsy sections and quantified by computerized image analyzer. IL-8 levels were measured in unconcentrated BAL by ELISA (Amersham, UK). Compared with controls, airway wall neutrophilia was increased in both stable and BOS LTR ( $p < 0.05$ ). BAL neutrophils and IL-8 levels were also increased in both LTR groups ( $p < 0.01$ ) compared to controls, with levels being significantly higher in the BOS group ( $p < 0.01$ ). Neutrophil numbers were not significantly different in the lung parenchyma between the two LTR groups. In conclusion, the increase in neutrophils in the airway wall and BAL from LTR patients with BOS supports the concept that neutrophils plays a significant role in the pathogenesis of BOS in post-lung transplantation.

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