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Association between oxidative stress in COPD patients, smokers and non-smoker and serum cytokine levels in North Indian population

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Background: Cigarette smoking is a major risk factor for chronic obstructive pulmonary disease (COPD). Oxidative stress has been known for having a key role in pathogenesis of many diseases. The aim of this study was to investigate the serum cytokines levels in [Interleukin-1 β (IL-1 β), Interleukin-6 (IL-6), Tumor necrosis factor- α (TNF- α)] and their correlation with smoking categories, oxidative stress and also the relationship between antioxidant system statuses and lung function in patients with COPD and smokers and non-smokers subjects. Diagnosis was made by spirometry.

Methods: Total 100 newly diagnosed patients from 30-80 years were included in the study. Participants diagnosed as COPD were selected for the systemic serum cytokines levels. 5 ml of venous blood were collected from each patient in a plain vial and centrifuged for 10 minutes at 2655 g at 40 C, serum collected; it was stored at -80° C for further analysis. For oxidative stress 30 subjects with COPD, 30 smokers and 30 healthy non-smokers participated in this study. The investigation included determination of the lung function and the measurements of total antioxidant capacity and also erythrocyte glutathione peroxidase, glutathione reductase, superoxide dismutase and catalase.

Results: The results shows that among the smoking categories there was significant difference in the level of TNF- α (p=0.03), IL-6 (p=0.01) and IL-1 β (p=0.02) among the different categories of smoking habit. The pos-hoc analysis revealed TNF- α (pg/ml) was significantly (p<0.05) higher among ex-smoker than non-smokers. Erythrocyte glutathione peroxidase and glutathione reductase were not significantly different between the studied groups. Subjects with COPD and smokers had lower catalase and superoxide dismutase activity (P<0.001) than the non-smoker group. Levels of antioxidant capacity were significantly lower in subjects with COPD and smokers than in the non-smoker group (P<0.001). Regression analysis revealed no correlations between antioxidant status and spirometric data.

Conclusion: Decreased total antioxidant capacity in plasma of subjects with COPD and smokers suggests an increased oxidative stress in this group. However, no relationship was found between lung function and antioxidant systems status in COPD subjects. Smoking may influence TNF- α mediated systemic inflammation, which, in turn, may account for some of the benefits observed in patients with COPD who stop smoking.

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