CORRELATION OF EXHALED NITRIC OXIDE WITH NITROGEN OXIDES AND SELECTED CYTOKINES IN INDUCED SPUTUM OF STABLE COPD PATIENTS

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Exhaled NO (eNO) may play an important role as a non-invasive marker of airway inflammation, especially in bronchial asthma. However, little is known about eNO in COPD patients and the relationship between eNO and other sputum inflammatory mediators is not well recognized.

Aim: The aim of the study was to assess eNO in patients with COPD and to correlate the level of eNO with sputum inflammatory mediators.

Material and methods: Forty two COPD patients (aged 59 ±12 yr) and 13 healthy nonsmoking control subjects were included in this study. In all patients and controls eNO measurements were performed by an on-line method using a chemiluminescent NO analyzer. Cytokine (IL-8, TNF- α , TGF- β 1, TGF- β 2, GM-CSF, Eotaxin) concentrations were measured in induced sputum using an ELISA method and nitrogen oxides (NOs, as nitrite or nitrate) were assayed colorimetrically by the Griess reaction.

Results: The mean eNO (10.4 \pm 6.1 ppb) and NOs levels (15.9 \pm 15.3 µmol/L) in COPD patients did not differ significantly from those in controls (9.4 \pm 6.4 ppb and 9.0 \pm 6.4 µmol/L, respectively). There was no significant correlation between eNO and sputum NOs. The mean sputum concentrations of IL-8 (4949.5 pg/ml), Eotaxin (16.0 pg/ml) TGF- β 2 (653.8 pg/ml), and TNF- α (16.3 pg/ml) were higher in COPD patients than in controls (1243.1, 9.9, 301.2, and 1.5, respectively). The mean concentrations of TGF- β 1 (11.8 pg/ml) and GM-CSF (4.9 pg/ml) in COPD were significantly (P<0.01) lower than in controls (71.4 pg/ml and 12.6 pg/ml, respectively). No relationships between the eNO level and the cytokines examined were observed. However, NOs correlated positively with IL-8 level (r=0.50, P<0.01) and the spirometric FEV1 correlated negatively with the NOs and IL-8 levels (r=-0.34 and r=-0.49, P<0.05) in the COPD group.

Conclusions: eNO, sputum NOs, and other sputum inflammatory cytokines offer separate and additive information about the pathophysiological condition of patients with COPD.