The Detection of Changes in Carboxyhemoglobin Levels in Asthmatics.

Naples R., Laskowski D., McCarthy K., Mattox E., Comhair S.A.A., Erzurum S.C.

Am J Respir Crit Care Med. 2011; 183: A4454

Rationale

Exhaled nitric oxide (NO) is increased in asthma due to induction of NO synthase (iNOS) by proinflammatory cytokines in asthma. Similarly heme oxygenase-1 (HO-1), which degrades hemoglobin into biliverdin, iron, and carbon monoxide (CO), is induced in the inflammatory environment of the asthmatic airways and results in greater exhaled CO. Since CO binds to heme with a higher affinity than oxygen, we hypothesized that carboxyhemoglobin (COHb) levels may be increased in asthma and that levels may be related to asthma severity. NO can oxidize iron in hemoglobin to produce Methemoglobin (MetHb), possibly affecting oxygenated Hb and COHb. Hence, we evaluated COHb and MetHb in asthma and controls.

Methods

COHb in asthma (n=25) and controls (n=25) were examined in arterial and venous blood by Radiometer (ABL700). COHb was also measured using pulse CO-Oximeter method (Masimo Rad-87), in which COHb levels were determined by differential absorption of visible and infrared light across a sensor on the patient's index fingertip. Cigarette smokers were excluded. Spirometry and exhaled NO (Aerocrine NIOX) were collected per ATS guidelines.

Results

Arterial COHb (COHb%: asthma 2.4 ± 2.9%, controls 1.7 ± 1.2; p=0.294) and venous COHb (COHb%: asthma 1.7 ±0.5%, controls 2.1 ±0.9%, p=0.2) were similar among asthma and controls. Arterial COHb correlated with FEV1/FVC in asthma (R=-0.44, p=0.0263). Despite higher exhaled NO, venous MetHb was lower in asthma compared to controls (MetHb%: asthma 0.77 ±0.22%, controls 0.98 ±0.23%, p=0.0084). MetHb was correlated with venous nitrate (an end product of NO) (R=+0.55, p=0.0111) but not airflow limitation or COHb (all $p>0.05$).

Conclusion

In asthma, COHb is not detectably higher in asthma than healthy individuals, although arterial COHb is related to airflow limitation. The paradoxically lower MetHb in asthma suggests upregulation of compensatory pathways in the high NO environment of asthma.