Endothelial

Subsequently, inflammation is a leading causative factor in the pathogenesis of COPD. This local inflammation produces cytokines that reach the circulation resulting in systemic inflammation. The characteristics of systemic inflammation during exacerbations of COPD are well described. Data on systemic inflammation in patients with COPD that are clinically stable are scarce.

The objective of this study was to define key inflammatory mediators that are elevated in the systemic circulation in patients with clinically stable COPD. Methods: This was a secondary data analysis of the ongoing study titled Impact of Oxidative Stress on HIV-Induced Lung Disease study database. The Luminex High Sensitivity multiplex assay was used to measure picogram levels of the following cytokines in plasma: TNF-α, IFN-γ, IL-1β, IL-6, IL-17A, IL-17B, IL-12/70, IL-10, IL-8, IL-4, IL-5, IL-6, IL-10, IL-17A, IL-17B, IFN-γ, GM-CSF, and Fractalkine. Patients with stable COPD were compared to healthy controls. Results: A total of 37 patients were evaluated, 11 with COPD and 26 healthy controls. Significantly higher levels were identified in 13 of the 22 cytokines for COPD patients (TNF-α, IL-1β, IL-6, IL-17A, IL-17B, IL-12/70, IL-10, IL-8, IL-4, IL-5, IL-6, IL-10, IL-17A, IL-17B, IFN-γ, GM-CSF, and Fractalkine). Patients with COPD are prone to developing exacerbations that may be severe enough to require hospitalization. There are multiple studies describing an upregulation of a number of cytokines in the circulation of COPD patients which is well described in the literature, including interleukin 6 (IL-6), tumor necrosis factor alpha (TNF-alpha), and IL-10. However, there is scarce data on the level of these inflammatory mediators in the systemic circulation in the stable clinical phase of COPD. Previous studies are also limited in the number of different cytokines measured.

In addition to smoking cessation which remains the single most important intervention to decrease progression of COPD, it is important to explore medical options that may target this underlying pathophysiology directly. For example, a currently studied antibiotic, doxycycline, has been known to possess anti-inflammatory and immunomodulatory properties. A study of the effect of doxycycline on stable COPD patients has shown possible contribution to the improvement of patients’ pulmonary functions, symptoms, and COPD level of severity.

Therefore, the more information gained about systemic inflammation in stable COPD patients, the better our understanding will be of COPD pathogenesis and, in so doing, possibly facilitate future target therapy research. This is the objective of this study was to define the key inflammatory mediators that are elevated in the systemic circulation in patients with clinically stable COPD.

This was a secondary data analysis of the ongoing study titled Impact of Oxidative Stress on HIV-Induced Lung Disease study database. COPD was defined as a diagnosis documented by a physician in the medical records as well as through pulmonary function tests.

Venous blood of all subjects was collected using sodium citrate Vacutainer tubes. Following centrifugation at 300 x g for 10 min, the plasma was separated by aspiration, aliquoted and stored frozen at -80°C until assayed.

The concentrations of the following 21 cytokines and chemokines in plasma were determined using Milliplex MAP Multiplex kits (BMD Millipore, Billerica, MA) according to the manufacturer’s instructions: TNF-α, IL-6, IL-1β, IL-17A, IL-17B, IFN-γ, GM-CSF, and Fractalkine.

The plasma concentrations of the cytokines in patients with stable COPD were compared to healthy controls.