The Patterns of some Inflammatory Cytokines, Liver Enzymes, and Oxidative Markers Circulating amongst Prolonged Cigarette Smokers

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Abstract

Objective(s): The present study was designed to provide additional information on the levels of cytokine responses, endothelial injury, and oxidative stress responses during prolonged cigarette smoking using the serum levels of interleukin-1β, IL-10, CRP, liver enzymes (aspartate transaminase, alanine transaminase, alkaline phosphates), creatine kinase-MB, troponin T and malondialdehyde (MDA) on prolonged smokers.

Materials and methods: A total of 204 subjects within the ages of 18 and 55 years were randomly studied. Among these were 95 smokers (smoking at least 6 cigarettes per day for more than 6 months) and 109 nonsmoking individuals from Igbinedion University Teaching Hospital Okada Community. Absolute white blood counts were estimated using Sysmex® Automated Hematology Analyzer whereas; IL-1β, IL-10, CRP, MDA, TT, and CK-MB were estimated using enzyme-linked immunosorbent assay methods. Liver enzymes (AST and ALT) were estimated using auto-analyzer.

Results: The levels of IL-10, CRP, MDA, CK-MB, TT, liver enzymes, SBP and DBP were significantly elevated in the smokers as compared with the non-smoking subjects whereas; the levels of IL-1

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oxides and cadmium [14]. Many of these agents are known to be immunosuppressive by affecting both innate and adaptive immune response [15,16]. Also, other studies have observed that the number of interferon gamma secreting cells was extremely reduced in prolonged cigarette smokers [17,18]. T-cell activation, proliferation and expression of the cytotoxic proteins are significantly reduced by exposure to cigarette smoke [19]. Spleen cells from animals that are subjected to the heavy dose of cigarette smoke have a significant reduction in their natural killer cell-mediated lytic activity [20]. Cigarette smoke impairs NK cell-dependent tumor immune

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