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Poster Presentation

The Effects of Captopril, as Angiotensin Converting Enzyme Inhibitor, on LPS-Induced Systemic Inflammation

Javad Boskabadi[1, 3], Mohammad Hossein Boskabady[1, 2*]

[1]Neurogeneeic Inflammation Research Centre, Mashhad University of Medical Sciences, Mashhad, Iran
[2]Department of Physiology, School of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran
[3]Student Research Committee, Department of Pharmacology, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

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Abstract

It has been shown that the renin-angiotensin system (RAS) plays key roles in the inflammation process. Imbalance in the oxidant-antioxidant system is one of the major causes of inflammation. In the present study, the effects of captopril on total and differential WBC, oxidative stress in systemic inflammation produced by lipopolysaccharide (LPS) were investigated. The rats were divided to: control (saline), LPS (1 mg/kg), 12.5, 25 or 50 mg/kg captopril treated before LPS administration (LPS-Cap12.5 , LPS-Cap 25 and LPS-Cap 50) and captopril 50 mg/kg before saline adminis tration (positive control group) groups. The levels of total and percentage of differential WBC in blood, the levels of malondialdehyde (MDA), total thiol groups, the activities of superoxide dismutase (SOD) and catalase (CAT) in the serum were evaluated. In the LPS group, total WBCs count, percent of neutrophils, basophils, eosinophils, monocytes in blood and MDA levels in serum were significantly higher than the control group ($p<0.05$ to $p<0.001$). Total WBCs count and percentage of eosinophils in the blood of LPS-Cap25 and LPS-Cap50 groups, percentage of neutrophils, monocytes, basophils in the blood and MDA levels in serum of LPS-Cap50 group were significantly decreased compared to the LPS group. Total thiol groups, activity of SOD and CAT enzymes, percentage of lymphocytes in the LPS-Cap50 group were significantly increased compared to LPS group. ($p<0.05$ to $p<0.001$). The results of this s tudy showed that captopril dose dependently reduced total and percentage of differential white blood cells in systemic inflammation induced by LPS in rats and also improved inflammatory responses and oxidative stress.

Keywords: Captopril, Inflammation, Lipopolysaccharide, Oxidative Stress

Corresponding Author: Mohammad Hossein Boskabady
E-mail: *boskabadymh@mums.ac.ir*

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