

Protective Role of Glycyrrhizae Radix from Peroxynitrite-induced Renal Oxidative Damages

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[Purpose]

Since peroxynitrite (ONOO^-) formed by superoxide (O_2^-) and nitric oxide (NO) is a major factor in oxidative stress-related pathological conditions including renal failure, the recent great efforts to develop the effective and safe agents with protective activity from ONOO^- has attempted us to elucidate the protective potential of Glycyrrhizae Radix from ONOO^- -induced oxidative damages.

[Methods]

After administration of 30 consecutive days of Glycyrrhizae Radix extract at an oral dose of 30 or 60 mg/body weight/day, renal ischemia was performed for 60 min and at 50 min during ischemia lipopolysaccharide (LPS) was injected. Six hours after LPS challenge, the samples of plasma and tissue were prepared.

[Results and Conclusion]

The LPS plus ischemia-reperfusion resulted in the elevations of plasma 3-nitrotyrosine level, renal myeloperoxidase activity and nitrated tyrosine protein concentration in kidney as the indicators of ONOO^- generation. However, the administration of Glycyrrhizae Radix extract decreased the levels significantly and dose-dependently. In addition, it inhibited the reactive oxygen generation (ROS) and also showed the significant scavenging effect of NO but not that of O_2^- . Moreover, western blot analysis showed that the increased levels of inducible NO synthase (iNOS) and cyclooxygenase-2 (COX-2) in kidney led to the declines by the extract, suggesting the inhibition of NO and ROS generations derived from iNOS and COX-2. The protective roles of Glycyrrhizae Radix extract from ONOO^- were also observed in the elevation of the levels of glutathione and total-sulfhydryl, and reduction in the lipid peroxidation. Furthermore, the impairment of renal function induced by ONOO^- ameliorated significantly assessed by the measurement of renal functional parameters, urea nitrogen and creatinine in serum. The present study indicates that Glycyrrhizae Radix would be contributed to the attenuation of ONOO^- -induced renal oxidative damages.