Protective effect of catechin on ischemia-reperfusion-induced renal injury in rats

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Abstract:
There is increasing evidence to suggest that toxic oxygen radicals play a role in the pathogenesis of ischemia/reperfusion (I/R) injury in the kidney. This study was designed to investigate the effects of catechin, a bioflavonoid, in I/R-induced renal failure in rats. The protective effect of catechin against the damage inflicted by reactive oxygen species (ROS) during renal I/R was investigated in Sprague Dawley rats using histopathological and biochemical parameters. In one set of experiments, animals were unilaterally nephrectomized, and subjected to 4.5 min of left renal pedicle occlusion, and in another set both the renal pedicles were occluded for 45 min followed by 24 h of reperfusion. Catechin (40 mg/kg, po) was administered twice daily for 4 days and 2 h prior to ischemia. At the end of the reperfusion period, rats were sacrificed. Thiobarbituric acid reactive substances (TBARS), reduced glutathione levels, glutathione reductase, catalase, and superoxide dismutase activities were determined in renal tissue. Serum creatinine and blood urea nitrogen concentrations were measured for the evaluation of renal function. Ischemic control animals demonstrated severe deterioration of renal function, renal morphology and a significant renal oxidative stress. Pretreatment of animals with catechin markedly attenuated renal dysfunction, morphological alterations, reduced elevated TBARS levels and restored the depleted renal antioxidant enzymes. The findings imply that ROS play a causal role in I/R-induced renal injury, and catechin exerts renoprotective effects probably by the radical scavenging and antioxidant activities.

Key words:
ischemia-reperfusion, oxidative stress, catechin