Effects of curcumin and captopril on the functions of kidney and nerve in streptozotocin-induced diabetic rats: role of angiotensin converting enzyme 1

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Abstract:

Oxidative stress and inflammation are involved in the development and progression of diabetes and its complications. The renin-angiotensin system also plays an important role in the pathogenesis of diabetes and its complications. We hypothesized that curcumin and captopril would restore the kidney and nerve functions of diabetic rats through their angiotensin converting enzyme 1 (ACE1) inhibiting activity as well as their antioxidant and anti-inflammatory effects. Diabetes was induced by a single intraperitoneal injection of streptozotocin (100 mg·kg⁻¹ body weight). One week after induction of diabetes, rats were treated with 100 mg·kg⁻¹·day⁻¹ curcumin or 50 mg·kg⁻¹·day⁻¹ captopril orally for 6 weeks. Compared with diabetic control rats, curcumin- or captopril-treated diabetic rats had significantly improved blood glucose, lipid profile, kidney/body weight ratio, serum creatinine, blood urea nitrogen (BUN), and pain thresholds assessed by Von Frey filaments, hot plate test, and tail-flick test. Diabetic control rats showed increased levels of total peroxide, renal and neural tumor necrosis factor-α and interleukin-10, and renal ACE1 compared with nondiabetic rats. Although treatment with either curcumin or captopril restored the altered variables, captopril was more effective in reducing these variables. ACE1 was positively correlated with BUN and creatinine and negatively correlated with paw withdrawal threshold, hot plate reaction time, and tail-flick latency, suggesting a possible causal relationship. We conclude that curcumin and captopril protect against diabetic nephropathy and neuropathy by inhibiting ACE1 as well as oxidation and inflammation. These findings suggest that curcumin and captopril may have a role in the treatment of diabetic nephropathy and neuropathy.

Keywords:

diabetes, curcumin, captopril, ACE1

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