Angiotensin converting enzyme inhibition ameliorates hyperlipidemia, inflammatory and angiogenic factors in hypercholesterolemic rats

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Introduction: Renin – angiotensin system (RAS) can be activated during hyperlipidemia. Angiotensin II increases the migration of monocytes, cytokine level and gene expressions of VEGF and VCAM-1. We aimed to investigate the effect of angiotensin converting enzyme (ACE) inhibition on VEGF, VCAM-1 and nitric oxide (NO) levels in hypercholesterolemic rats.
Methods: Forty male Wistar rats were divided into 4 groups including; normal diet + saline (control); hypercholesterol diet + saline injection; normal diet + captopril injection and hypercholesterol diet+captopril injection. Before and after the diet and after the treatment, serum levels of cholesterol, triglycerides, LDL, HDL, and NO were measured Finally gene expressions of VCAM-1 and VEGF were determined.
Results: Hypercholesterolemic diet increased the serum levels of cholesterol, LDL ($p <$ 0.001) and triglycerides ($p < 0.01$); and decreased HDL ($p < 0.001$). Captopril reduced the serum levels of cholesterol, LDL ($p < 0.001$) and triglycerides ($p < 0.05$) as well as well as increased HDL level ($p < 0.01$). However the serum level of NO was decreased after hypercholesterolemic diet ($p < 0.001$) but no significant change was observed after treatment. Increased gene expressions of VEGF ($p < 0.05$) and VCAM-1 ($p < 0.01$) in hypercholesterolemia were regressed in captopril treated rats, $p < 0.01$ and $p < 0.05$, respectively.
Conclusion: ACE inhibitors like Captopril improve hyperlipidemia, prevent over-expression of some genes (e.g. VEGF and VCAM-1) that could be implicated in the inflammation and angiogenesis.

Keywords: Hypercholesterolemia; Renin – angiotensin system; VCAM-1; VEGF; Nitric oxide