

Vitamin E and regression of hypercholesterolemia-induced oxidative stress in kidney

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Abstract Hypercholesterolemia (HC) is an independent risk factor for the onset and progression of renal disease. HC induces oxidative stress (OS) in the kidney; Vitamin E (Vit.E), an antioxidant, slows the progression of OS in the kidney. This study was to investigate if Vit.E regresses the HC-induced OS, and the regression is associated with an increase in the antioxidant reserve (AR). The studies were carried out in four groups of rabbits. The kidneys were removed under anesthesia. OS and AR in the renal tissue were assessed by measuring malondialdehyde (MDA) and chemiluminescent (CL) activity, respectively. High-cholesterol diet elevated the serum total cholesterol (TC), and the regular diet with or without Vit.E following a high-cholesterol diet reduced the serum TC to control levels. HC increased the MDA levels of kidney by 5.54-fold compared to control. The MDA contents of the kidneys in groups on regular diet with or without Vit.E were, respectively, 56 and 53 % lower than the control group. The CL activity in the control group was $12.15 \pm 0.73 \times 10^6$ RLU/mg protein. The CL activity in HC group was 45.26 % lower than that in control, indicating an increase in AR. The regular diet with or without Vit.E following high-cholesterol diet normalized the CL activity/AR. In conclusion, HC increases OS in the kidney; reduction of serum cholesterol by regular diet regresses the renal OS but Vit.E does not regress HC-induced OS in kidney.

Keywords Chemiluminescent activity · Hypercholesterolemia · Malondialdehyde ·

Regression of oxidative stress · Renal oxidative stress · Vitamin E

Introduction

There is a large amount of data demonstrating the role of oxidative stress (OS) in renal disease [1]. Hypercholesterolemia (HC) has been reported to be an independent risk factor for the onset [2] and progression [3] of renal disease. HC can induce and worsen renal glomerular, interstitial, and vascular damage [4, 5]. HC is known to generate reactive oxygen species (ROS) through various mechanisms [6]. HC increases the production of superoxide anion from endothelial cells [7] and polymorphonuclear leukocytes [8]. HC is associated with the increased formation of oxidized low-density lipoprotein (LDL) [9] and lipid peroxidation product, malondialdehyde (MDA) [10–13], a measure of OS. It has been reported that xanthine oxidase which combines with xanthine to produce ROS is activated in HC in swine [14]. Increase in the ROS levels could also be due to decreases in the antioxidants, enzymatic [superoxide dismutase, catalase and glutathione peroxidase] and nonenzymatic [reduced glutathione, Vitamin E, Vitamin C, selenium, and magnesium] antioxidants. Vitamin E, a nonenzymatic antioxidant [15], has been reported to slow down the progression of HC-induced OS in kidney [16]. To be of potential benefit to patients with HC-induced OS, Vitamin E should be able to regress the OS in kidney. However, it is not known if Vitamin E regresses the established HC-induced OS in the kidney. The objectives of this study were to investigate: if Vitamin E regresses HC-induced OS in kidney, and if regression of OS is associated with decrease in the serum levels of total cholesterol (TC) and increase in the antioxidant reserve

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