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Mini Review

Protective Effects of Antioxidants on Kidney Disease

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Oxidative stress is an imbalance between the amount produced and the removal of oxidants produced. In other words, the increase in reactive oxygen species (ROS) or reactive nitrogen species (RNS) and/or the reduction of the antioxidant/intrinsic/extrinsic is the main cause of this problem [1]. This is one of the most important causes of acute and chronic kidney disease [2]. Increasing the accumulation of free radicals within the kidney resulting from the malfunctioning of the antioxidant defense will ultimately lead to acute renal tubule necrosis, impaired function, and ultimately reduced glomerular filtration (GFR) [3]. The important factors of ROS molecules are hypochlorous acid, superoxide anion, hydrogen peroxide and hydroxyl radical. Because ROS molecules are very active, they quickly destroy molecules such as fats, DNA and proteins [4]. The factors that cause elevation of ROS and its products in the kidney cells are inflammatory cytokines, angiotensin II, mechanical pressure, hyperglycemia, protein kinase C (PKC), free fatty acids, TGF-beta1 and NADPH oxidases enzymes (NOXs). NADPH oxidases (NOXs) are enzymes that produce ROS. The NOX family contains seven subgroups (NOX1- 5 and DUOX 1 and 2) [5]. Of these seven types the NOX4 sever express in tubular cells, mesangial cells and podocytes. Increases of NOX4 expression has been shown in diabetic nephropathy, and on the contrary, shutting it off will protect the kidneys. Accumulation of P -Cresyl sulfate in tubular cells causes over activation of PI3-K and PKC then trigger expression of Nox4- and p22 ^{phox} and subsequently production of ROS. Enhancement of ROS production increase inflammatory cytokines and resulting tubular cell injury [6]. Oxidative stress induces apoptosis in renal tissue [7]. There are proteins that regulates Nox4 activity, such as Poldip2 (first known as NoxR1), and the p47phox-related adaptor protein Tks5 [8]. High glucose concentrations induce Nox4 protein. In cultured mesangial cells, high glucose in culture media stimulates a rapid expression of Nox4 protein, mitochondrial fraction, which is related to an increase of ROS inside the cell [9]. Although their performance on Nox4-mediated renal cell injury is unknown. Another important group that reduces or decreases the activity of antioxidants is RNS group. The main important RNS molecules are consisted of peroxynitrite, nitrite, and nitrate [10]. Antioxidants are divided into two important groups according to their mechanism of action, breaking and preventive antioxidants [11]. Preventive antioxidants are able to dispose free oxygen and subsequently decrease the rate of chain initiation by break down of metals and finally subtractive hydroperoxides. Chain breaking antioxidants produce a stable product by donating or taking electrons from a free radical [11]. Medicinal herbs and some materials like Propolis are phytochemical compounds such as phenolic compounds or carotenoids that have potent antioxidant activity [12]. These natural materials protect cell membrane in the kidneys by reducing the lipid peroxidation (LPO) and increasing the endogenous antioxidants. Use of pargyline reduced apoptosis, necrosis, and fibrosis in kidney disease. This effect was related to reduction of the expression of collagen types I, III, IV, TGF-β1, and induction of SOD1, catalase, and inflammatory factors [13]. The family of vitamin E is able to reduce the damage to proximal tubules by inhibiting the lipid peroxidation enzyme, increasing levels of GSH and activating the catalase. In addition, the NO2-/NO3 ratio improves. Etramethylpyrazine which found in grain and grain products is able to protect kidneys from ischemia/reperfusion injury. This protection is carried out by increasing the level of SOD enzyme, reduces malondialdehyde activity and then reduces the production of ROS [14]. Another group of compounds that is rich in tea, coffee, some fruits, cereals and vegetables, has a very powerful antioxidant activity by increasing the activity of Cu/Zn SOD, glutathione peroxidase and catalase and to decrease the level of MDA [15]. Another antioxidant is α-Lipoic acid. It is containing sulfur coenzyme which involved dehydrogenase reactions in mitochondria finally leading to (ATP) formation. In diabetic neuropathy, α-lipoic acid through various mechanisms, they will protect the kidneys. Such as increase blood level and decrease ROS production in the distal nerves, reduces vascular endothelial growth factor (VEGF) protein in early diabetic nephropathy, and hypoxia in the retina. α-lipoic acid prevent renal insufficiency, glomerular mesangial matrix expansion, and glomerulosclerosis by restoring glutathione and reducing MDA levels [16].

Conclusion

Oxidative stress is one of the most important factors in kidney damage. Generally, they act by increasing the production of oxidants or reducing the antioxidant defense of the cytosol, blood plasma,

or through the failure to protect the cell membrane from lipid peroxidation. Antioxidants protect cells from damage by eliminating oxidative stress products as well as enhancing antioxidant defense.

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