BIOCHEMICAL RELATIONSHIP BETWEEN VITAMIN A CONTENT IN LIVER AND NUTRITION STATE

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ABSTRACT

The aim of the present study was to investigate the biochemical changes in serum and liver retinol, serum nitric oxide (NO), total protein, serum protein electrophoresis, albumin, blood hemoglobin, glucose -6- phosphate dehydrogenises (G6PD), and antioxidant enzymes activities: glutathione peroxidase activity (GSH-PX), glutathione reductase (GR ase), activity reduced glutathione (GSH), superoxide dismutase activity (t-SOD) and catalase (CAT) in experimental under the effect of administration of ordinary, double, toxic doses of vitamin A in rats. In order to achieve this aim 80 male Sprague-Dawely rat 3-6 weeks old weighting 120-250 grams were used in the experimental investigation of this study. The animals were divided into 4 groups each 20 rats that were orally supplemented with retinol palmitate (2500, 5000, 10000 IU/Kg/day) and the fourth group served as control. The result of the present study showed a significant association between vitamin A supplementation and low level of liver and serum retinol, low nitric oxide level, high G6PD level, high catalase activity, low super oxide dismutase activity, low glutathione reductase and peroxidase activity, low reduced glutathione level. These parameters may all be regarded as risk factors for exposure to high doses of vitamin A. Vitamin A can potentially promote liver damage. When you consume large amounts of vitamin A, your body stores the excess vitamin within your liver. After a very high dose or long-term consumption of moderately high doses vitamin A can form toxic accumulations in your liver, leading to liver swelling and damage. In addition, the toxicity can cause skin peeling, kidney damage.

KEY WORDS: antioxidants, free radical, G6PD, NO, oxidative stress, protein electrophoresis, retinol palmitate, and vitamin A.

1- INTRODUCTION

Vitamin A (retinol) is an essential nutrient needed in small amounts by humans for the normal functioning of the visual system; growth, development, maintenance of epithelial cellular integrity, immune function, and reproduction. These dietary needs for vitamin A are normally provided as preformed retinol (mainly as retinyl ester) and pro-vitamin A carotenoids [1].

Vitamin deficiencies are especially common in patients with cirrhosis and result both from reduced intake with the diet and, at least for some vitamins, from reduced absorption of those vitamins that are ingested. One important example is the vitamin A deficiency frequently found in patients with cirrhosis. Vitamin A (retinol) is essentially obtained directly from the diet or can be produced in the body from a precursor compound called beta-carotene [2].

Vitamin A deficiency can impair the ability of the eye to adjust to dark conditions (i.e., causing night blindness) and can result in other eye disorders. In the liver, reduced vitamin A levels can change the structures of components of cells, and these changes