

The effect of antioxidant vitamin E in patients with hyperlipidemia in Tikrit city

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Abstract

The cross sectional study was designed to investigate the effect of antioxidant vitamin E 400mg daily dose in patients with hyperlipidemia for nine months. The study was performed on 90 patients (36 males and 54 females) from medical clinic in the Al-Qadsia area/Tikrit city.

Results: The study was showed the level of triglycerides significantly decreased after three months of treatment in both female and male, the level of total cholesterol was significantly decline after three months of treatment in female and male, the level of HDL-cholesterol is significant decline after six months of treatment in female and after nine months in male, the level of VLDL-cholesterol is significantly decreased in female after nine months of treatment and after three months in male, while the level of LDL -cholesterol is significantly decreased after three months of treatment in both female and male. There is some side effect appear in some patients during the course of treatment in study as abdominal pain, gastrointestinal disturbance and fatigue.

CONCLUSION: This study was recorded the significantly decline in triglycerides, total cholesterol, LDL-cholesterol, followed by HDL and VLDL levels in females patients with hyperlipidemic, on the other hand, total cholesterol, triglycerides, VLDL levels showed the greatest reduction followed by LDL, and HDL-cholesterol levels in male patients with hyperlipidemic after using vitamin E in a dose 400mg/day for nine months,

Key words: hyperlipidemia patients, vitamin E tablets, kit manufacturing .

Introduction

Hyperlipidemia is one of a metabolic disorder that involves elevation in plasma concentration of any of the lipoprotein aspects(1-3), such as total cholesterol, triglycerides, cholesterol esters and phospholipids(4) when estimated after 12 hour fasting(5-7).

Antioxidant is the mechanism for defense of tissue against peroxidation of polyunsaturated fatty acid contained in cellular and subcellular membrane phospholipids by preventing the initiation of free radical chain reaction(8).

Lipids are a heterogeneous group of water-insoluble (hydrophobic) organic molecules that can

be extracted from tissues by nonpolar solvents. Lipids are transported in the blood as large 'lipoproteins'(9).

The Pathology of hyperlipidemia can caused by primary (genetic predisposition) or secondary (diet, medication, or underlying disease)(10-15). The primary clinical sign of hyperlipidemia is usually develop xanthomas, which are tumor like collection of lipid (triglycerides and cholesterol) that can arise in the tendons, joints of continued trauma, e.g. knee, elbow, as well as palm and vascular disease. Diagnosis of hyperlipidemia estimation, which is done after a 12 hour fast includes plasma triglycerides, total cholesterol, and HDL-cholesterol is used to determine

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hyperlipidemia(16-17). Reliance on the total cholesterol value alone may be misleading because some patients with high LDL-cholesterol levels may have total plasma cholesterol within the normal range and rare case may have elevated plasma total cholesterol due to increase HDL-cholesterol concentration(18-19).

Oxidation and antioxidant of lipid: -

Peroxidation in which lipids (auto oxidation) exposed to oxygen, is responsible not only for deterioration of food but also for damage to tissue in vivo, where it may be a cause of cancer, inflammatory disease, atherosclerosis, aging, etc(20). Oxidation of LDL is a lipid peroxidation chain reaction initiated by so called free radicals. Radicals are molecules with an unpaired free electron, making them high reactive. In particular, oxygen radicals (ex. Hydroxyl-, hydroperoxyl-, superoxide radical), which are produced in the cells as by products of oxidative metabolic processes are of importance. The free radical removes a hydrogen atom from polyunsaturated fatty acid molecules in the LDL particle. Polyunsaturated fatty acids are highly susceptible to lipid peroxidation, because the susceptibility of fatty acid to oxidation increases with its number of double bonds. Lipid peroxy radicals are formed which in turn can-initiated oxidation in neighboring fatty acid. This process leads to a breakdown of polyunsaturated fatty acid, yielding variety of reactive aldehyde, ketones, and other products, some of which form covalent bonds with LDL, furthermore, the LDL particles are exposed to variety of free radical species and oxidative enzyme produced by arterial wall cells. If LDL is exposed to pro-oxidative condition, it becomes depleted of its antioxidants, with-tocopherol being the first to be lost. Those found in the naturally occur polyunsaturated fatty acid. Lipid peroxidation is a chain reaction providing continuous supply of free radicals that initiate = Triglycerides: Cat. No. TR 213(30).

further peroxidation(21-23). Vitamin E appears to be first line of defense against peroxidation of polyunsaturated fatty acids contained in cellular and subcellular membrane phospholipids(24). Vitamin E act in the lipid phase of membrane through out cells, it protects against the effect of toxic radical(25-26), vitamin E stabilizes the lipid portion of the cell membrane by preventing oxidation of polyunsaturated fatty acid phospholipids, thereby maintain the integrity of cell membrane(27-29).

The aim of the present study: -

- To determine the effect of antioxidant vitamin E on the hyperlipidemia.

PATIENTS AND METHODS

This work was conducted in the medical clinic in Al-Qadsia area/Tikrit city/Salahudeen Governorate. The work was carried out from the November/2011 until October/2013. The patients were firstly checked to isolated only hyperlipidemia. 90 patients underwent hyperlipidemia in both male and female with age range from 20- 55 years were include in this study (36 males and 54 females). The patients were treated with vitamin E 400mg daily for nine months and check lipid profile every three months. All these checked do by using procedures were performed according to the recommendation of the kit manufacturing company. Samples were taken by vein puncture after 12 hours fasting. All data were presented as a mean and standard deviation. Comparisons were performed by using student-test (t-test).

Calculation: -

A sample

Triglyceride concentration = ----- X concentration of standard

A standard

Concentration of standard = 2.29 mmol/l

= Cholesterol (Enzymatic Endpoint Method): Cat. No. CH 201(30).

A sample

Concentration of cholesterol in sample = ----- X concentration of standard

A standard

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Concentration of standard = 5.17 mmol/l

= HDL-cholesterol (precipitant): Cat. No. CH 203(31).

A sample

Concentration of HDL-cholesterol in supernatant = ----- X concentration of standard

A standard

Concentration of standard = 1.3 mmol/l

= LDL-cholesterol calculation(32):

To calculate the LDL-cholesterol by equation:

In mmol/l = Total cholesterol – triglycerides/2.2 – HDL-cholesterol

= VLDL calculation(30):

To calculate the VLDL by equation:

In mmol/l = Triglyceride / 2.2

RESULTS

This study was designed to investigate the effect of antioxidant vitamin E on the triglycerides, total cholesterol, HDL-cholesterol, VLDL and LDL-cholesterol levels in 90 patients with hyperlipidemia (36 males and 54 females).

The effect of vitamin E on hyperlipidemia of female patients:

As shown in table 1, vitamin E in a dose of 400mg daily for three months significantly decrease the level of triglycerides, total cholesterol ($P < 0.005$) from the three months of treatment in hyperlipidemic female patients.

However, LDL-cholesterol level was significantly decreased after the three months of treatment ($P < 0.05$) and further decline was noticed when the treatment was continued for nine months ($p < 0.025$), while HDL-cholesterol level was not changed in the first three months of treatment but it significantly declined after the six months of treatment while VLDL level was not changed in the first six months but significantly decline after nine months.

The effect of vitamin E on hyperlipidemia of male patients:

Table 2 shows that the level of triglycerides, total cholesterol and VLDL significantly declined ($P < 0.005$) after three months of treatment with vitamin E 400mg daily in hyperlipidemic male patients, however, no further decline was noticed when the treatment continued for nine months.

The LDL-cholesterol level was significantly decreased after the three months of treatment ($P < 0.05$) and further decline was noticed when the

treatment was continued for six months ($p < 0.025$), while HDL-cholesterol level significantly ($P < 0.05$) declined after the nine months of the treatment.

DISCUSSION

Many researchers have worked on the effect of antioxidant vitamin E on the hyperlipidemic patients, as it is considered to be one of the most important drug group because of the effectiveness and minimal side effects.

The results of this study showed that vitamin E significantly decreased lipid profile, these results were in agreement with Howard, and many authors who found that vitamin E significantly reduced cholesterol, triglycerides, LDL levels(33-34), and also in agreement with the results of Fuller who found that vitamin E reduced triglycerides after 6th week(35).

These effects could occur because vitamin E the first line of defense against peroxidation of polyunsaturated fatty acid containing in cellular and subcellular phospholipids, thereby maintaining the integrity of cell membrane, reduce the risk of heart disease by decreasing lipid peroxidation and reducing platelet adhesives(36), the phospholipids of mitochondria, endoplasmic reticulum and plasma membrane posses affinities for alpha-tocopherol and the vitamin appeared to concentrate at this site, it also protects against the toxic effect of free radicals (37).

Furthermore, vitamin E inhibition of fatty acid peroxidation with less formation of malonaldehyde and large amount of active (n-3) fatty acid in their

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site of action in the liver, resulting in a greater decrease in the synthesis of triglyceride (37-38).

CONCLUSIONS

This study throws some light on the effect of antioxidant vitamin E on lipid profile, of both male and female patients with hyperlipidemia .

However, with the using of vitamin E in a dose 400mg/day for nine months, the main reduction was recorded in total cholesterol, triglycerides, LDL level followed by HDL-cholesterol and VLDL levels in females hyperlipidemic patients, on the other hand, total cholesterol, triglycerides, VLDL showed the greatest reduction followed by LDL and HDL-cholesterol levels in male hyperlipidemic patients.

RECOMMENDATIONS

From the outcome of the present study the following recommendations may be suggested:

- 1- Other-antioxidant drug might be studied to show their effect in hyperlipidemia.
- 2-Also antioxidants drug can be used prophylactically with other antihyperlipidemic drugs to study results.

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Table 1: The mean $\pm$ SD of triglycerides, total cholesterol, HDL-cholesterol, VLDL and LDL-cholesterol in 54 female patients of hyperlipidemic treated with vitamin E in a dose of 400mg daily for nine months.

Type of investigation	Normal value mmol/L	Before treatment	Three month treatment	Six month treatment	Nine month treatment
Triglycerides mmol / l	0.9 – 2.4	2.76 $\pm$ 0.42	2.53 $\pm$ 0.70 ***	2.38 $\pm$ 0.48 ****	2.31 $\pm$ 0.47 *****
Cholesterol mmol / l	3.9 – 6.5	7.02 $\pm$ 0.52	6.37 $\pm$ 0.58 ****	6.33 $\pm$ 0.79 ****	6.30 $\pm$ 0.47 ****
HDL mmol / l	0.9 – 1.4	0.64 $\pm$ 0.10	0.62 $\pm$ 0.16 NS	0.59 $\pm$ 0.20 *	0.58 $\pm$ 0.15 **
VLDL mmol / l	<0.53	1.22 $\pm$ 0.19	1.21 $\pm$ 0.30 NS	1.18 $\pm$ 0.25 NS	1.11 $\pm$ 0.19 **
LDL mmol / l	1.8 – 4.3	4.96 $\pm$ 0.52	4.63 $\pm$ 0.86 *	4.65 $\pm$ 0.65 **	4.66 $\pm$ 0.74 **

* P < 0.05

** P < 0.025

*** P < 0.01

**** P < 0.005

NS non significant

Table 2: The mean $\pm$ SD of triglycerides, total cholesterol, HDL-cholesterol, VLDL and LDL-cholesterol in 36 male patients of hyperlipidemia treated by vitamin E in a dose of 400mg for nine months.

Type of investigation	Normal value mmol/L	Before treatment	Three month treatment	Six month treatment	Nine month treatment
Triglyceride s mmol / l	0.9 – 2.4	2.73 $\pm$ 0.34	2.22 $\pm$ 0.79 ****	2.20 $\pm$ 0.73 ****	2.18 $\pm$ 0.75 ****
Cholesterol mmol / l	3.9 – 6.5	6.67 $\pm$ 0.36	6.20 $\pm$ 0.82 ****	5.90 $\pm$ 1.11 ****	5.85 $\pm$ 1.11 ****
HDL mmol / l	0.9 – 1.4	1.31 $\pm$ 0.19	0.61 $\pm$ 0.13 NS	0.56 $\pm$ 0.15 NS	0.54 $\pm$ 0.15 *
VLDL mmol / l	<0.53	1.31 $\pm$ 0.19	1.12 $\pm$ 0.32 ****	1.14 $\pm$ 0.18 ****	1.11 $\pm$ 0.39 ****
LDL mmol / l	1.8 – 4.3	4.70 $\pm$ 0.57	4.78 $\pm$ 0.68 *	4.53 $\pm$ 0.62 **	4.49 $\pm$ 0.82 **

* P < 0.05

** P < 0.025

*** P < 0.01

**** P < 0.005

NS non significant.