The Influence of Vitamin E to Protect Myocard Damage Due to the High Fat Diet

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ABSTRACT

Eighteen male rats, strain Sprague Dawley, were used as experimental animals to study the protective effects of vitamin E on the incidence of myocard damage due to high fat diet. The rats were randomly alloted into 3 groups of six each. All of the rats were treated high fat diet. Group I as control was fed diet containing high fat. Group II was fed containing 30 IU of vitamin E, and group III was fed containing 180 IU of vitamin E. After 60 days, the blood sample were collected from the hearts for detected specific heart protein by troponin T analysis. The statistical analysis for cardiac troponin T, data showed that there were no significant differences between group I and group II . However, there were significant differences between group I and group III or between group II and group III. This result indicated that vitamin E have some effect to protect myocard damage from group III ($p<0,05$) that containing 180 IU of vitamin E, but not from group control (I) and group II ($p>0,05$) that containing 30 IU of vitamin E.

Key words; Sprague Dawley ; vitamin E ; cardiac troponin T.

INTRODUCTION

Cardiovascular disease that caused by atherosclerosis is among the most serious and costly health problems in industrialized nation. There is substantial and increasing evidence from basic and clinical research that atherosclerosis can be prevented, that progression of atherosclerosis can be retarded, and that regression of atherosclerosis can be effected, with reduction in coronary risk (Wenger and Schlant, 1994). Cardiovascular disease is a slowly progressive disease of the large arteries that begins early in life but rarely produces symptoms untill middle age. Often the disease goes undetected untill the time of the first heart attack, and this first heart attack is often fatal (Steinberg, 1985). A number of risk factors have been identified as strongly associated with coronary heart disease. Cigarette smoking, high blood pressure, and high blood cholesterol levels are the most clearly established of these factors. Risk is greater in men, increases with age, and has a strong genetic component. Obesity, diabetes mellitus, physical inactivity, and behavior pattern are also risk factor (Steinberg, 1985).

Tocopherol (vitamin E) as the Greek name implies, was discovered because of its role in reproduction (Chandra, 1980). Vitamin E was first isolated from wheat germ oil in 1936. Although a-tocopherol is the active compound most often identified as vitamin E_1, there are seven other naturally occuring tocopherols. Differences in activity among the tocopherols are probably a function of the ability of cellular components to distinguish and remove them at different rates. Like all the fat-soluble vitamins, tocopherol is dependent on a functional lipid transport system in the gut for absorption (Stinnet, 1983). Vitamin E is the most important lipid soluble antioxidant in the body, it works to quench free radicals and acts as a terminator of lipid peroxidation (Ingold et al. 1987). At the cellular level, vitamin E appears to protect cellular and subcellular membranes from deterioration by scavenging free radicals that contain oxygen. In the absence of vitamin E, free radicals catalyze peroxidation of the polyunsatu-

rated fatty acids (PUFAs) that constitute structural components of the membranes (Mahan and Arlin, 1992).

In the present study, the influence of vitamin E to protect myocard damage due to the high fat diet was examined, based on immunological assay by troponin T as a specific cardiac marker, using rats as animal models.

MATERIALS AND METHODS

Eighteen male Sprague Dawley rats, 150-200 grams of body weight were used as experimental animals. They were housed individually, and then randomly assigned to three diets group with six rats in each group. Tap water was freely available. Group I as control was fed diet containing high fat, group II and group III were high fat diet containing 30 IU and 180 IU vitamin E respectively. After 60 days on experimental diet, blood sample were withdrawn for cardiac troponin T (TnT) analysis from all animals.

Analytical methods

Qualitative immunological detection of cardiac troponin T in blood was assayed by uysing commercial TROPT RAPID ASSAY KIT produced by Boehringer Mannheim UK (Diagnostics & Biochemicals Ltd., Bell Lane, Lewes, East Sussex, BN7 1LG, GB). A positive result (appearance of 2 lines, control and signal lines) indicates that the troponin T concentration of the sample is above the test·s treshold value of 0,2 ng/ml. This confirm the diagnosis "myocardial lesion". A negative result (appearance of 1 line) showed that myocard were normally. However, negative result due to the release kinetics of troponin T, within the first hours of the onset of symptoms does not allow to rule out myocardial infarction with certainly. If myocardial infarction is still suspected, the test needs to be repeated at appropriate intervals.

Data of the experiments were analyzed statistically using chi-aquare method design. The difference was considered to be significant if $p<0.05$.

RESULT AND DISCUSSION

The Result after 60 days on experimental diets on rats cardiac troponin T are presented on Table 1. Statistical analysis showed that there were no significant differences ($p>0,05$) between group I as control and group II that was fed containing 30 IU of vitamin E. However, there were significant differences ($p<0,05$) between group I and group III, and between group II and group III ($p<0.05$) that was fed containing 180 IU of vitamin E.

Table 1. **The result after 60 days on experimental diets on rats cardiac troponin T among group I, group II, and group III.**

Number	Group I	Group II	Group III
1	Positive (+)	Positive (+)	Negative (-)
2	Positive (+)	Positive (+)	Positive (+)
3	Positive (+)	Positive (+)	Negative (-)
4	Positive (+)	Positive (+)	Negative (-)
5	Positive (+)	Positive (+)	Negative (-)
6	Positive (+)	Negative (-)	Negative (-)

Cardiac troponin T analyses from group I and group II showed that all result were positive, that means showed the incidence of myocard damage, except one from group II showed the negative result that mean myocard cell were normal. Analysis from group III showed that 5 from 6 rats were negative result.

In the present study shows that administration of high fat to rats caused the incidence of myocard damage. Almost previous study have suggested that high fat intake increases atherogenesis and mortality from coronary heart disease (Berner, 1993; Criqui, 1986, Sugano and Imaizumi, 1995).

In this study showed that incidence of myocard damage can reduce by giving diet containing 180 IU of vitamin E. However, from group II (fed containing 30 IU of vitamin E) have no effect to protect myocard damage. The previous report saying that vitamin E is a lipid-soluble antioxidant that protects the polyunsaturated fatty acids (PUFAs) and other components of cell and organelle membranes from oxidation by reactive free radicals (Tappel, 1962). In addition, vitamin E may have important roles in biological processes that do not necessarily relate to its antioxidant function such as in DNA synthesis, the stimulation of the immune response, and the suppression of inflammation (Duthie, 1993). Consequently, the increase vitamin E intakes may inhibit

the progression of many diseases including coronary heart disease, arthritis, malignant hyperethermia, Parkinson disease, and tardive dyskinesia (Duthie, 1993, Packer, 1993).

In the present study, the statistical analysis for cardiac troponin T indicated that vitamin E have no effect to protect myocard damage from diet high fat that containing 30 IU of vitamin E. The result probably caused by overabundance of polyunsaturated fatty acids (PUFA) in the diet and relatively little vitamin E were given in this experimental. According to Stinnet (1983) vitamin E nutriture is determined by plasma tocopherol levels with correlates well with the level of plasma total lipids (in normal circumstances). There is some indication that dietary requirement for vitamin E increases as the intake of polyunsaturated fats increases.

Statistical analysis showed there were significant differences between group II (30 IU of vitamin E) and group III (180 IU of vitamin E). The previous study saying that the assessment of vitamin E requirements in human is complicated by the infrequent occurrence of clinical signs of deficiency, which ussualy develop only in premature infants or adults with malabsorption (Gutcher et al., 1984). The requirement value do not take into account the growing epidemiological evidence that intakes of vitamin E and other antioxidants are beneficial in limiting oxidative damage that may be relevant to the prevention of cataract formation, cardiovascular diseases, and several cancers (Gey, 1992). Willett's group (Stamper et al, 1993) reported a protective effect of vitamin E in prospective studies of adults with large and possibly pharmacological doses. Thus the true needs for vitamin E maybe appreciably greater than currently cited and depend on the degree of prevailing oxidative stress in one or more organs. In the previous study, most individual s while taking large dose of vitamin E have not shown toxic effect. This is fortunate considering the large amounts (100-800 mg/ day) with which many people medicate themselves (Mahan and Arlin, 1992).

The bewildering collection of symptoms reported to be associated with vitamin E deficiency have given few concrete clues as to its precise role. Nonetheless, there are few items about which there is fairly good agreement. As long as there is not an overabundance of polyunsaturated fatty acids (PUFA) in the diet, relatively little vitamin E is required (Stinnet, 1983). An important new area of vitamin E research concerns the assesment of the need for an antioxidant defense system to protecet the body from free radical-induced damage (Asim et al, 1994). The essentiality of vitamin E (predominantly as alpha-tocopherol) relates in part to its action as an antioxidant preventing the lipid peroxidation of cellular PUFA. It is considered the primary lipid – soluble antioxidant, especially with respect to terminating the lipid free radical chain reaction (Bankson et al, 1993). The carotenoid compund that include beta-carotene are also good scavengers of peroxyl radicals, especially at the low oxygen tension found in the body. Lycopene is one of the most potent carotenoid antioxidants; it is found in high concentrations in tomatoes. Another lipid – soluble antioxidant is ubiquinol-10, the reduce form of coenzyme-Q. Compared with vitamin E, it is more effective as a lipid peroxyl radical scavenger. In addition, ubiquinol-10 functions to regenerate vitamin E after it has been oxidized (Bankson et al., 1993).

CONCLUSION

Based upon the experimental result, it can be concluded that fed diet containing 180 IU of vitamin E (group III) have the greatest effect to protect myocard damage from rats after 60 days on experimental diet comparing group II that fed diet containing 30 IU of vitamin E. However, the effect protect of myocard damage it is also depend to the intakes of polyunsaturated fatty acids on the experimental diets.

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