



Supplementation effects of vitamin C and vitamin E on oxidative stress in post menopausal diabetic women

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ABSTRACT

Introduction: Menopause is a natural event of women with cessation of menstrual cycle. Menopause is associated with a wide variety of physical and psychological symptoms. The antioxidant enzyme systems seem to be affected in this phase due to deficiency of estrogen, which has got antioxidant properties. Oxidative stress occurs at menopause because of loss of estrogens, which have antioxidant effect on low-density lipoproteins. Diabetes is also a degenerative disease usually accompanied by increased production of free radicals or impaired antioxidant defenses. Keeping these two real fact on the mind the present study has been design, if menopausal women will suffer from the type II diabetes this problem of high production of free radical become very complicated or leading to death. **Objective:** the main objective of the study is asses the supplementation effect of vitamin E and Vitamin C on oxidative stress markers and antioxidant enzyme level with and without type II diabetic menopausal women. **Material and Method:** Blood sample of menopausal women age group with or without diabetes was collected from different hospitals of Allahabad. Serum antioxidant enzyme -Glutathione reductase, superoxide dismutase and catalase was estimated. Serum Melondialdehyde was also estimated as strees marker. **Results:** it is reveals from the study that, supplementation of vitamin C and vitamin E reduces the oxidative stress and increases the antioxidant serum enzyme level. Data shows that vitamin E is most effective than vitamin C. **Conclusion:** it is concluded from the study that diabetic menopausal women, with supplementation of vitamin E have less risk of developing oxidative stress. © 2012 Trade Science Inc. - INDIA

KEYWORDS

Menopause;
Oxidative stress;
Antioxidant enzyme;
Vitamin C;
Vitamin E.

INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia and insufficiency of secretion or action of endogenous insulin. Although the etiology of this disease is not well defined, viral infection, autoimmune disease, and environmental factors have been implicated^[1-5] Worldwide, there were approximately 194 million adults aged 20–79 years with diagnosed diabetes mellitus (DM) in 2003 (with type 2 diabetes accounting for 90–95% of all diagnosed cases), and that number is expected to increase to 333 million over the next 20 years^[6]. Diabetes is associated with increased coronary artery, cerebrovascular and peripheral vascular disease, with up to 80% of deaths in people with diabetes caused by cardiovascular disease^[7] Diabetes is usually accompanied by increased production of free radicals^[8-11] or impaired antioxidant defenses^[12-14] Menopause is associated with a wide variety of physical and psychological symptoms. It is a gradual three-stage process that concludes with the end of periods and reproductive life. Women experience menstrual bleeding during menopause and perimenopause. When a woman's menstruation has ceased

Spontaneously at least for a year it is post menopause^[15]. In post-menopause, ovaries stop making estrogen hormone. The antioxidant enzyme (AOE) system seems to be affected in this phase due to deficiency of estrogen, which has got antioxidant properties. The beneficial effects of estrogens might be attributable to their free radical scavenging

Structures^[16]. Oxidative stress occurs at menopause because of loss of estrogens, which have antioxidant effect on low-density lipoproteins. Estrogens confer cardio protection by lowering protein oxidation and antioxidant properties^[17]. Diminished antioxidant defense is associated with osteoporosis in post-menopause. Modulation of the estrogen receptors α and β has been reported to be effected in vitro by oxidative stress^[18]. A currently favored hypothesis is that oxidative stress, through a single unifying mechanism of super oxide production, is the common pathogenic factor leading to insulin resistance, β -cell dysfunction, impaired glucose tolerance (IGT) and ultimately to type 2 DM (T2DM)^[19] Increased oxidative stress is a widely accepted participant in the development and pro-

gression of diabetes and its complications^[20-22]. Overproduction of free radicals can cause oxidative damage to bimolecular, (lipids, proteins, DNA), eventually leading to many chronic diseases such as atherosclerosis, cancer, diabetes, rheumatoid arthritis, post-ischemic perfusion injury, myocardial infarction, cardiovascular diseases, chronic inflammation, stroke and septic shock, aging and other degenerative diseases in humans^[23] intake of natural antioxidants has been reported to reduce risk of cancer, cardiovascular diseases, diabetes and other diseases associated with aging, there is considerable controversy in this area^[24].

MATERIALS AND METHOD

Subjects

The case group consisted of 130 postmenopausal women with pre-existing type II diabetes. Out of 129 case subjects 80 subjects are randomly selected for supplementation study with vitamin E and vitamin C, remaining 49 case subjects are treated as positive control group (group-III). All 80 subjects were divided into two groups- group I consist of 40 subjects consuming Vitamin E and group II consist of 40 subjects consuming vitamin C. Age range for all groups was 55-70 years.

Blood sampling and biochemical analyses

Five milliliters of blood were collected between 0800-0900 h in the morning from postmenopausal women of all groups into non-heparinized bottle for the measurement of biomolecules in serum. The blood was allowed to clot, retract and the serum separated by centrifugation at room temperature (20°C). The serum was stored at -20°C till needed Then, The blood samples were analyzed for antioxidant enzymes like glutathione reductases^[25], catalase^[26] and superoxide dismutase^[27] by auto pack kit method. (Span / Diagnostic Ltd.), and MDA was estimated by using the Nadigar *et al* method with thiobarbituric acid^[28]

Statistical analysis

Differences of data between supplementation group and the control group were tested with Student's t-test. A two-sided p value <0.0001 was the level of statistically significance. All

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data were expressed as mean±SD. Statistical computations were Calculated using SPSS 9.0 for windows software (SPSS Inc, Chicago, IL, USA).

RESULTS

In the present study evaluation of effect of vitamin C and vitamin E on serum oxidative stress marker (MDA) and antioxidant enzymes such as SOD, CAT and GPX were done in post menopausal women having type II diabetes. TABLE 1 shows effect of vitamin C and vitamin E on anthropometry parameters and biochemical parameters of post menopausal women having diabetes. There is no significant difference between three groups with respect to age, weight and BMI. Serum MDA of the group one significantly differs from group II and group III ($p < 0.0001$). In addition there no significant difference between of serum MDA in group II and group III. On the other hand with reference to an-

tiioxidant enzymes there is also a significant decrease in serum SOD and GPX and significant increase in CAT enzyme in group I as compare to group II and group III. Supplementation with vitamin E and vitamin C shows that there is a significant difference between antioxidant enzymes of group II and group III. There is a significant decrease in SOD and GPX and significant increase in CAT enzyme in group II which was supplemented with vitamin E as compare to group II which was supplemented with vitamin C.

DISCUSSION

Menopausal phase in a woman's life is an important physiological phenomenon, which is associated with cessation, of menstrual cycle due to loss of ovarian function. The deficiency of estrogen in postmenopausal women develops oxidative stress, due to release of free radical or reactive oxygen species (ROS) and becomes the cause of various pathologies like development of hypertension. Nonenzymatic sources of oxida-

TABLE 1 : Showing effect of vitamin C and E on different biochemical parameters.

Types	Control (n=50) Group-I	Supplementation with vitamin C (n=40) Group - II	Supplementation with vitamin E (n=40) Group-III	p value
Age (year)	60.00±10.50	59.86±11.25	60.80±14.90	i-ii NS i-iiiNS ii-iiiNS
Body weight (kg)	80.20±9.80	81.10±10.90	79.70±8.70	i-iiNS i-iiiNS ii-iiiNS
BMI (Kg/m ²)	24.80±4.90	23.86±5.80	24.20±5.90	i-ii < 0.0001 i-iii < 0.0001 ii-iii NS
MDA levels (nmol/dl)	1.9 ± 0.4	1.1 ± 1.04	1.03 ± 1.1	i-ii < 0.0001 i-iii < 0.0001 ii-iii NS
Superoxide dismutase (SOD) U/mg	5.09 ± 1.94	6.46 ± 0.256	6.86 ± 1.1	i-ii < 0.0001 i-iii < 0.0001 ii-iii < 0.0001
Catalase (CAT) U/mg	3.08 ± 1.05	3.37 ± 2.1	3.98 ± 1.9	i-ii < 0.0001 i-iii < 0.0001 ii-iii < 0.0001
Glutathione reductase (Moles of GSSH/mg)	0.62 ± 1.004	0.86 ± 1.00	0.98 ± 1.2	i-ii < 0.0001 i-iii < 0.0001 ii-iii < 0.0001

tive stress originate from the oxidative biochemistry of glucose. Hyperglycemia can directly cause increased ROS generation. Glucose can undergo autoxidation and generate OH radicals, glucose reacts with proteins in a nonenzymatic manner leading to the development of Amadori products followed by formation of AGEs. ROS is generated at multiple steps during this process. In hyperglycemia, there is enhanced metabolism of glucose through the polyol (sorbitol) pathway, which also results in enhanced production of O₂⁻.

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