Does Vitamin C and E Supplementation Have a Protective Role in Mild Preeclampsia?

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Abstract

Objectives: Oxidative stress has been proposed as a key factor involved in the development of pre-eclampsia. Supplementation with antioxidant vitamins has been proposed to reduce the risk of preeclampsia and perinatal complications, but the effects of this intervention are uncertain.

Aim of the Study: Was to assess if supplementing women with mild pre-eclampsia with antioxidants in the form of (Vitamin C 1000mg + Vitamin E 400IU) during the third trimester of pregnancy might help to counteract oxidative stress and thereby prevent or delay the incidence of severe pre-eclampsia?

Research Design and Methods: This research includes of 30 mild pre-eclamptic women, all were primigravidae with singleton gestation in the third trimester (28-32 weeks of gestation). Fifteen of them were supplemented with (Vitamin C 1000mg + Vitamin E 400IU) tablets) daily until delivery and were considered as the study group (PE-CE). The control group (n=15) were not supplemented with vitamin C and E tablets (PE). Measurement of serum nitric oxide level (NO) was assessed initially on the first visit and re-evaluated at the end of the study using ELISA. Follow-up of all pregnant women was done until delivery.

Results: Significant increase in serum NO levels were realized among PE-CE group compared to the controls. Also, significant decrease in serum NO levels among PE group were obtained at the end of the study; reflecting the significant decrease in oxidative stress among the pre-eclamptic group who received anti-oxidants. Significant negative correlation between serum NO level and mean arterial blood pressure was evident in PE-CE group.

Conclusion: Patients with pre-eclampsia are exposed to oxidative stress, which may have a role in the pathogenesis of the disease. Supplementation with the antioxidants vitamin C and E could be considered in the management of mild pre-eclampsia and to be routinely administered in mid trimester pregnancy.

Key Words: Vit. C – Vit. E – Preeclampsia.

Introduction

PRE-ECLAMPSIA affects between 0.4% and 2.8% of all pregnancies in developed countries and much more in developing countries. It is more prevalent in first pregnancies and is associated with the highest maternal and fetal morbidity and mortality of all pregnancy complications. The cause of pre-eclampsia remains largely unknown, but poor placentation is an important predisposing factor which is strongly supported by resolution of symptoms after delivery [1].

Primary dysfunction in pre-eclampsia is a relative deficiency of available nitric oxide (NO) secondary to oxidative degradation and excess of peroxynitrite. The body uses NO to help keeping blood vessels relaxed and blood pressure in check. Blood stores of nitric oxide are kept readily available in the form of S-nitroso-albumin; the women with pre-eclampsia had levels of S-nitroso-albumin that were two to three times higher than the normal controls. In addition, women with pre-eclampsia had low levels of vitamin C in their blood. Vitamin C helps breakdown of S-nitrosoalbumin to release nitric oxide into the blood stream [2]. So, the combination of deficiency of usable nitric oxide and increase in peroxynitrite can directly or indirectly initiate the vast majority of physiological and serological changes associated with pre-eclampsia, such as high blood pressure, decreased glomerular filtration rate, proteinuria, platelet dysfunction, increased thromboxane and endothelin and a decrease in prostacyclin [2].

The placenta appears to be the principal source of free radical synthesis but maternal leucocytes and the maternal endothelium are also likely contributors. Rajmakers et al. [3] suggested an important role for placental trophoblast nicotinamide adenine dinucleotide phosphate (NADPH) oxidase
in free radical generation in pre-eclampsia. The antioxidant vitamin E is now known to have multiple actions in addition to prevention of lipid peroxidation (i.e., inhibition of NADPH oxidase activation and the inflammatory response).

Vitamin E (alpha-tocopherol) and C (ascorbate) have important roles in the contribution they make to antioxidant potential, as alpha-tocopherol is the major lipid-soluble chain breaking antioxidant in cell-membranes. Ascorbic acid has been described as the major "front line" water-soluble antioxidant. Vitamins C and E concentration are less in preeclamptic women [4].

So, in view of the abnormally low plasma vitamin C concentration and increase free radicals in pre-eclampsia, a combination of vitamins C and E is a promising prophylactic strategy for prevention of pre-eclampsia. The aim of the current study was to investigate the role of antioxidant supplementation vitamin C and vitamin E on the clinical course of mild preeclamptic patients assessed by the measurement of the serum nitric oxide level (NO) twice at the start and at the end of the study.

**Subjects and Methods**

This is a prospective study carried out on 30 pregnant Egyptian women suffering from mild preeclampsia. They were recruited from the Obstetric and Gynecology Hospital, Ain Shams University from March 2005 to December 2007. They were matched in respect to age, socioeconomic status and weeks of gestation. All the participating women gave informed consents preliminary to the study. The design of the study was approved by the Ethical Committee of our institute (Atomic Energy Authority).

**Inclusion Criteria:** Thirty pregnant primigravidae women attended antenatal clinic, age between 18-40 years, with mild pre-eclampsia as the blood pressure was > 140/90 to ≤ 160/110, with proteinuria (1+), with or without the presence of edema; according to the criteria of the International Society of the Study of Hypertension in Pregnancy Brown et al. [5]. All were in the third trimester (gestational age 28-32 weeks assessed by the last menstrual period and confirmed by early ultrasound previously done during the first trimester of pregnancy), with a singleton viable fetus. Body mass index (BMI) was calculated as weight before pregnancy in (kg) divided by squared height (m²) [6].

**Exclusion criteria:** All the participants had no past or present history of diabetes, cerebro-vascular disease, or apparent endocrinopathy. Patients with blood pressure higher than 160/110, proteinuria > 1+, severe preeclampsia, system affection as liver, kidney disease or heart disease and hematological disease as thrombocytopenia were excluded. Smokers were also excluded as maternal smoking is known to be associated with various prenatal complications such as placental abnormalities for instance decreased intervillous blood flow and altered uteroplacental flow [7].

**The recruited women were randomly categorized into two groups:**

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**Study Group (PE-CE):** Comprised of 15 pregnant primigravidae with mild pre-eclampsia who received at the start of the study vitamin C (1000mg) tablets and vitamin E (400IU) tablets daily until delivery [8].

**Control Group (PE):** Consisted of 15 pregnant primigravidae with mild pre-eclampsia who didn't receive vitamins C and E.

**On the first visit of the study, all women were subjected to:**

- **Full medical and obstetric history:** Included antenatal care, blood pressure readings and antihypertensive treatment, with particular emphasis on symptoms of pre-eclampsia; headache, visual symptoms, epigastric pain, nausea and vomiting, decreased urine output, hematuria, or rapid weight gain, in each antenatal care visit.

- **Clinical examination:** Thorough clinical examination with stress on maternal blood pressure measured with a standard mercury sphygmomanometer on the right arm after the subjects had been resting in the supine position for at least 5 minutes. Blood pressure was measured after putting subjects in left lateral position for 10 minutes at least [9]. Physical examination was performed to exclude localized epigastric tenderness. Then albuminuria was detected and leveled by using strip urine dipsticks (score 1 + or higher on dipstick test).

- **Subjects and Methods**

The (PE-CE) group received (Vitamin C 1000mg) and (Vitamin E 400IU) tablets, with insurance that they would receive these tablets every day. The controls (PE) did not receive these tablets but took their usual multivitamins and antihypertensive medication (Aldomet tab, 250mg). Then all patients were followed-up weekly for an average of 6 to 8 weeks from the beginning of the study. Also, follow-up was done using ultrasound considering various fetal biometries as biparietal diameter, abdominal circumference, head circumference and long bones as femur length. Estimated...
fetal weight was also detected. Doppler study was done for blood flow analysis of the umbilical artery before delivery [10].

Blood sampling:

The first blood sample (at the start of the study): five ml venous blood was drawn from the forearm into empty clean tube for each patient and then centrifuged at 100 x g within 30 minutes of collection. Serum was stored in closed tubes at –70ºC for NO assay. The rest of serum was used for detection of triglycerides, cholesterol, uric acid, creatinine, serum alanine aminotransferase (ALT), aspartate aminotransferase (AST) and albumin levels.

- Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were estimated according to the method of Tekum and Timothy [11].
- Albumin level was measured according to the method of Ziyatdinova et al. [12].
- Nitric oxide (NO) levels in serum were determined using ELISA according to the method of Ghasemi et al. [13].
- Serum triglycerides and total cholesterol were estimated according the methods described by Winkler et al. [14], Moghadasian et al. [15] respectively.
- Serum urea, uric acid and creatinine were determined according to the methods of Hallet and Cook, Yi et al., [17] and Holmes et al., [18] respectively.

The series of tests and sampling were repeated after 6 weeks of receiving (Vitamin C 1000mg) and (Vitamin E 400IU) tablets by the study group. The duration of the study was about 6-10 weeks for each patient.

Follow-up of all patients was done till the time of delivery.

Statistical analysis:

All data were expressed as mean ± standard deviation in both studied groups. Statistical differences in patients before and after vitamin C and E supplementation were measured using the paired dependent "t" test. Statistical differences between 2 groups were done using simple student t test. Comparison between the 2 groups as regards the gestational age of fetus at time of delivery was done using Chi-square test. Relationships between serum levels of NO and MBP were presented by simple linear regression analysis and Pearson (r) correlation coefficients. The results were considered significant whenever p values <0.05 and highly significant when p values <0.001. The statistical calculations were done using Statistica (version 6), while the statistical representation were done using Microsoft Excel (2007) and Statistica (version 6) programs.

Results

The results are demonstrated through the following Tables (1-5) and Figs. (1-2).

Clinical parameters of the patients are shown in Table (1). Insignificant differences between the two groups are illustrated as regards age, body mass index, systolic, diastolic or mean arterial blood pressure (p>0.05), indicating that all subjects matched each other in their clinical data distributions.

Table (1): Demographic and clinical data at the start of the study using simple student "t" test.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>PE-CE n=15 Mean ± SD</th>
<th>PE n=15 Mean ± SD</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>30.8±7.4</td>
<td>29.7±6.0</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>29.9±1.4</td>
<td>30.4±1.5</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>150.3±6.4</td>
<td>152±7.0</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>99±6.8</td>
<td>101±7.3</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Mean arterial blood pressure (mmHg)</td>
<td>105.8±4.5</td>
<td>108.3±4.3</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m^2)</td>
<td>34.8±0.5</td>
<td>35.1±0.4</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>

Table (2): Comparison between the PE-CE and PE groups as regards the mean levels of serum nitric oxide and mean arterial blood pressure at the start and at the end of the study using student "t" test.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Groups</th>
<th>PE-CE n=15 Mean ± SD</th>
<th>PE n=15 Mean ± SD</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO (Umol/L)</td>
<td>Initial reading</td>
<td>33.37±14.04</td>
<td>33.35±15.73</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>48.9±22.2</td>
<td>27.7±12.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>MABP (mmHg)</td>
<td>Initial reading</td>
<td>105.8±4.5</td>
<td>108.3±4.3</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>93±6.2</td>
<td>109±7</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

From Table (2) there was no significant difference in the initial readings of NO and MABP between the control and the PE-CE groups. Whereas at the end of the study, the NO level was significantly higher in the PE-CE group when compared to the controls. Also, the MABP was highly significantly lower among the study group.
Table (3): Comparison between the initial and final readings of the mean levels of serum nitric oxide and mean arterial blood pressure in both groups using paired "t" test.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Groups</th>
<th>n</th>
<th>Initial reading</th>
<th>Final reading</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>NO (Umol/L)</td>
<td>PE-CE group</td>
<td>15</td>
<td>33.37±14.04</td>
<td>48.9±22.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>PE group</td>
<td>15</td>
<td>33.35±15.73</td>
<td>27.7±12.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>MABP (mmHg)</td>
<td>PE-CE group</td>
<td>15</td>
<td>105.80±4.50</td>
<td>93.0±6.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>PE group</td>
<td>15</td>
<td>108.30±4.30</td>
<td>109.0±7.0</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Fig. (1-A): Comparing between initial and final reading of NO in PE-CE and PE groups.

A significant increase in the mean level of NO among PE-CE group at the end of the study while significant decrease was realized in the control group (Table 3, Fig. 1A). The MABP revealed high significant decrease among the PE-CE groups and significant increase in the controls at the end of the study (Table 3, Fig. 1B).

Fig. (1-B): Comparing between initial and final reading of MABP in PE-CE and PE groups.

A very high significant negative correlation between the serum level of NO and the MABP among preeclamptic patients has been revealed.

Fig. (2): Correlation between serum NO and MABP among preeclamptic patients.

Table (4): Comparison between PE-CE and PE groups at the start and end of the study as regarding lipid profile, kidney and liver function tests using student "t" test.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PE-CE N=15</th>
<th>PE N=15</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides (mg/dl):</td>
<td>Initial reading</td>
<td>120.2±11.8</td>
<td>116.7±10.64</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>116.9±13.2</td>
<td>121.6±15.80</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl):</td>
<td>Initial reading</td>
<td>189.0±11.5</td>
<td>184.0±11.2</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>181.5±16.4</td>
<td>176.5±21.7</td>
</tr>
<tr>
<td>Serum uric acid (mg/dl):</td>
<td>Initial reading</td>
<td>4.49±0.31</td>
<td>4.60±0.25</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>4.52±0.37</td>
<td>4.72±0.30</td>
</tr>
<tr>
<td>Serum creatinine (mg/dl):</td>
<td>Initial reading</td>
<td>0.41±0.08</td>
<td>0.44±0.16</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>0.44±0.14</td>
<td>0.47±0.13</td>
</tr>
<tr>
<td>ALT (IU):</td>
<td>Initial reading</td>
<td>17.4±5.9</td>
<td>18.0±5.50</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>22.1±7.5</td>
<td>18.3±5.40</td>
</tr>
<tr>
<td>AST (IU):</td>
<td>Initial reading</td>
<td>20.1±7.4</td>
<td>20.0±5.40</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>21.9±6.1</td>
<td>20.3±6.70</td>
</tr>
<tr>
<td>Serum albumin (g/dl):</td>
<td>Initial reading</td>
<td>3.33±0.62</td>
<td>3.11±0.96</td>
</tr>
<tr>
<td></td>
<td>Final reading</td>
<td>3.46±0.57</td>
<td>3.59±0.45</td>
</tr>
</tbody>
</table>
Table (4) clarifies no significant difference between the control and the study groups at the start and at the end of the study, as regards triglycerides, cholesterol, serum uric acid, ALT, AST, serum albumin and serum creatinine.

Table (5): Gestational age of viable fetus at the time of delivery, in the control and the study groups using Chi square test.

<table>
<thead>
<tr>
<th>Gestational age of a viable fetus at time of delivery</th>
<th>PE-CE group N=15</th>
<th>PE group N=15</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;37 weeks</td>
<td>15</td>
<td>11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>%</td>
<td>100%</td>
<td>73%</td>
<td></td>
</tr>
<tr>
<td>&lt;37 weeks</td>
<td>0</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>%</td>
<td>0</td>
<td>27%</td>
<td></td>
</tr>
</tbody>
</table>

Table (5) clarified that all members of PE-CE group (n=15, 100%) achieved term and received a viable fetus, whereas 73% only of the PE group who accomplished term; reflecting that the administration of Vitamin C and E helps the preeclamptic patients to achieve full term.

Discussion

Preeclampsia is an important and a leading cause of both maternal morbidity and adverse perinatal outcomes. Despite progress in perinatal medicine for patients with an established diagnosis of preeclampsia, a therapeutic approach other than termination of pregnancy was unsuccessful. Women predisposed to preeclampsia begin pregnancy with a certain degree of endothelial dysfunction, a lesion that precedes shallow placentation [19].

The proposed sequence of events comprises endothelial dysfunction, defective trophoblast invasion and consequential impaired placental perfusion, immune maladaptation and inflammation. The possible link between these could be oxidative stress by excessive production of reactive oxygen species (ROS) coupled with inadequate or overwhelmed antioxidant defense mechanisms. These defense mechanisms, involving antioxidant vitamins and enzyme systems, may restrain the extent of damage caused by oxidative stress. Markers of oxidative stress in women with established PE were confirmed. Accordingly, these findings support an expected beneficial effect of antioxidant therapy in the prevention of PE and other pregnancy-related disorders [20].

The present study assess the effect of administration of Vitamin C and E on the serum NO levels in mild pre-eclampsia. Our results indicated that NO levels were significantly increased among mild pre-eclamptic patients who received vitamin C and E during the midtrimester stage of pregnancy compared to controls; reflecting the significant decrease in oxidative stress among the pre-eclamptic group who received anti-oxidants. A significant correlation between serum NO level and mean arterial blood pressure was evident in PE-CE group. Also, by the end of the study, those patients who received vitamin E and C achieved term.

Placenta is also highly vascular and is exposed to high maternal oxygen partial pressure. Initially the placenta has a hypoxic environment. As it matures and its vascularization develops, it changes to an oxygen-rich environment and its abundant mitochondrial mass favors the production of reactive oxygen species (ROS) [21].

Vitamin C deficiency affects placental structure, ROS production and facilitates placental infection, which result in increased risk of premature rupture of placental membranes and premature births [22]. The placenta is avid to absorb vitamin C, so when maternal plasma ascorbic acid concentration is low it is absorbed by active mechanisms. At higher plasma ascorbic acid concentrations, it enters the placenta by passive diffusion. Curiously, ascorbate is preferentially taken up in the oxidized form (dehydroascorbic acid), which more easily passes the lipid layer. However, it is transformed to its reduced form at the expense of other reducing agents and donate electrons before transferring to the fetus by active transport [23].

Vitamins C and E levels in the sera of preeclamptic women were lower and significantly different than in normal pregnancies [4]. Dehghan and Dehghanan [24] confirmed that plasma vitamin C level was significantly lower in pre-eclampsia pregnant women in the third trimester of pregnancy than in the normotensive group. Vitamin E and vitamin C are known to be powerful antioxidants. Vitamin E (α-tocopherol) is the major lipid-soluble antioxidant that protects cells against lipid peroxidation. Vitamin C is a quencher of free radicals as well as singlet oxygen. It also regenerates vitamin E.

Oxidative stress is a normal phenomenon in normotensive pregnancy, however, in pre-eclampsia, oxidative stress is exaggerated. Pre-eclampsia develops as oxidative stress reaches a threshold level/point of no return. Antioxidant vitamin C and E intervention in early pregnancy may prevent development of pre-eclampsia by enhancing maternal antioxidant capacity, inhibiting...
by enhancing cellular integrity, the antioxidants in placenta, may contribute to the endothelial dysfunction, which is proposed that they may initiate maternal vascular endothelial dysfunction and leucocyte activation, which is a recognized feature of this disorder [25]. Endothelial dysfunction underlies the pathogenesis of preeclampsia, but its mechanism has not yet been completely understood. Var et al. [26] added that elevated oxygen free radicals may partially explain the endothelial cell damage. They declared that reduced NO may increase blood pressure and ischemia in preeclamptic patients. They concluded that increased oxygen free radical levels and decreased NO levels are closely associated with preeclampsia-related endothelial dysfunction.

Plasma antioxidant activity has been reported to increase progressively throughout pregnancy [3]. Antioxidants, contained in certain vitamins, can neutralize free radicals, preventing them from causing damage to our bodies. Antioxidants, such as vitamin C and vitamin E can neutralize free radicals Kaaja, 2008 [19]. Padiayatty et al. [27] suggested that antioxidants have been proposed as a potential preventive strategy on the basis of data suggesting that endothelial dysfunction is fundamental to the development of preeclampsia and that increased oxidative stress, particularly in the placenta, may contribute to the endothelial dysfunction. Although the magnitude of the oxidative stress and of the reduction in antioxidant activity in women with preeclampsia is the subject of considerable controversy, there has been interest in the use of supplementation with vitamin C and vitamin E to reduce oxidative stress, limit the injury of endothelial cells and prevent or reduce the severity of preeclampsia. The hypothesis is that by enhancing cellular integrity, the antioxidants would protect cells from degradation by peroxides and would fortify villus development [28].

Vitamins E (alpha-tocopherol) and C, have differences in the contribution they make to antioxidant potential, as vitamin E is the major lipid-soluble chain-breaking antioxidant in cell membranes while vitamin C is an important aqueous phase antioxidant. Antioxidants may act synergistically, for instance when vitamin C regenerates alpha-tocopherol from the tocopherol radical, this ‘sacrificial’ antioxidant acts more by sparing vitamin E than by recycling. Thus, it might be important to evaluate the effectiveness of potential antioxidant defense systems in limiting scale. This study provides evidence for the relationship between plasma vitamin C levels during the third trimester and preeclampsia [24].

Vladimir et al. [2] found that S-Nitrosothiols and S-nitrosoalbumin concentrations which are the major reservoirs of NO are increased in preeclampsia plasma reflecting a decreased release of NO from these major reservoirs. These levels are two to three times higher in preeclampsia than in normal pregnancy. Because vitamin C is essential for decomposition of S-Nitrosothiols and the release of NO, they speculated that the ascorbate deficiency typical of preeclampsia plasma result in decreased rates of decomposition of S-Nitrosothiols. So, the availability of NO which is required for the normal regulation of vascular tone is decreased in preeclampsia, thus contributing to the vascular pathogenesis of this pregnancy disorder. Oxidative stress accompanied by a pronounced depletion of ascorbate is thought to contribute to the endothelial dysfunction of preeclampsia. Also, vitamin C has been shown to reverse NO dependent endothelial dysfunction in hypertension.

The antioxidants vitamins, C and E, are able to inhibit formation of free radicals thereby inhibiting the oxidation of NO (which is a potent relaxant of vascular smooth muscle) and thus maintaining the vasodilator status of blood vessels. Vitamin C has been reported to have a direct acute effect on the inhibition of the constrictor response of resistance arteries to a variety of stimulus. It is reported that there is synergism between the actions of vitamin E and vitamin C. Raijmakers et al., [3]. Dehghan and Dehghanan [24] added that vitamin C can regenerate vitamin E from the alphatocopheroxyl radical.

Chappell et al. [8] and Holmes and McCance [23] clarified that administration of 1000mg of vitamin C and 400IU of vitamin E/day, reduced the incidence of pre-eclampsia in women who were at risk, with improvement of biochemical indices of placental dysfunction and oxidative stress toward normal value due to improvement in endothelium-dependent vasodilatation. Vitamin C, as an effective antioxidant in human plasma, provides major defense against the diseases caused by oxidative stress. It is likely that severe oxidative stress in eclampsia utilizes a higher amount of vitamin C.
to fight the oxidative stress leading to a depletion of the vitamin. The higher vitamin C levels in pre-eclampsia may serve to prevent oxidation of NO (endothelium dependent vasodilator) to maintain the vasodilatation of blood vessels [24,29].

A recent review by Yu et al. [30] showed that the risk of pre-eclampsia rises dramatically as BMI increases. If pre-pregnancy BMI was 26 (over-weight), the patient would have double the risk of developing pre-eclampsia than a woman with a BMI of 21 (ideal). If BMI was 30 (obese), the patient would have triple the risk of developing the condition [6].

Lipid profile; kidney and liver function tests of both groups at the start of the study showed similar trends and were within the normal ranges of pregnant women. The same picture was observed at the end of the study in both groups which means that vitamin C and E supplementation has no effect on the patient lipid profile, kidney and liver function. This was in consistent with the results of Chappell et al. [8] who found that uric acid level is slightly increased throughout pregnancy from 20 to 36 weeks and there was no significant effect of vitamins C and E supplementation and also there were no significant differences between the control group and study group patients who were on vitamin C and E, in any of lipid parameters that were evaluated. As for ALT, AST and serum albumin, the results were non-significant with (p > 0.05) between the two groups. The vital organs as the liver and the kidney, have their own potent compensatory mechanisms and so they need a longer time of exposure to the insult to show a significant drop in their performance and this explains why blood chemistry is not commonly altered even in patients with pre-eclampsia except for severe cases as HELLP syndrome [31].

Accordingly, analysis of the results revealed that vitamin C and vitamin E supplementation could be useful in the management of patients with mild preeclampsia.

References


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