Effect of vitamin C supplementation on lipid profile and blood sugar in normal and obese male albino rats

May N. Al Muammar¹, Islam Abdul Rahim Saad el Dien el Redh², Nawal Abdulla Al Bader², Mervat M. El-Sayed³*, Eman K. El. Gabrý³, Dina M. Trabzuni², Shaista Arzoo²

¹Department of Clinical Nutrition, College of Applied Medical Sciences, King Saud University, Riyadh, Saudi Arabia
²Department of Food and Nutrition Sciences, College of Food and Agriculture Sciences, King Saud University, Riyadh, Saudi Arabia
³Department of Medical Biochemistry, National Nutrition Institute, Egypt

Key words: Lipid profile, Vitamin C, Obesity, High density lipoprotein, Triglycerides.

http://dx.doi.org/10.12692/ijb/15.5.506-510 Article published on November 28, 2019

Abstract

Vitamin C is a vital antioxidant in humans and plays significant role in various metabolic activities. Connotation has been reported between vitamin C and atherosclerosis that assess the relationship between vitamin C and cholesterol levels. The effect of oral administration of vitamin C on serum lipids and glucose level were investigated in albino rats of wistar strain. Sixteen male albino rats were randomly divided by weight into four groups. Group 1-3 consisted of rats with normal weight (150-300gm) and 4th group consisted of obese rats (350-400 gm). Control group received via oral route placebo 4 ml distilled water and Test group T1 and T2 (normal weight rats) received 2.5 ml and 5 ml vitamin C respectively and test group T3 (obese rats) also received 5 ml vitamin C orally. Each tablet of vitamin C (500 mg) was dissolved in 125 ml of distilled of distilled water. The administration of vitamin C for 30 days produced significant (p≤0.05) decrease in triglycerides in test group vs control but reduction was not significant between test group and control for total cholesterol, LDL, HDL and glucose. The outcome of this study shows that supplementation of vitamin C to healthy wistar rat was found to be effective but statistically insignificant (except for triglycerides) in decreasing lipid profiles and glucose levels.

*Corresponding Author: Mervat M. El-Sayed mervatelsayed@gmail.com
Introduction

Vitamin C, an aqueous phase antioxidant, is a vital antioxidant in humans (Dousdampanis et al., 2014) and is capable of scavenging oxygen-derived free radicals (Garg et al., 2005). Its antioxidant activity has been found to be the major defense mechanism, in the aqueous phase, against the harmful effect of free radicals (Chaudiere and Ferrari-Iliou, 2005). Since it is structurally similar to glucose, so it can replace it in many chemical reactions, and thus is effective in prevention of non-enzymatic glycosylation of proteins (Bhatt et al., 2012). Although its storage forms are not found in human tissues but its high concentrations can be found in “metabolically highly active” organs such as liver, adrenal cortex, corpus luteum (Chatterjee and Shinde, 2002) and vegetables and fruits are its major dietary sources (Annette and John, 1985). Almost all plants and animals except guinea pigs and primates can synthesize vitamin C. Its protracted deficit in humans results to disease known as scurvy characterized by impaired collagen formation and hemorrhages (White et al., 1978).

Vitamin C plays significant role in various metabolic activities such as in formation of ferritin as cellular antioxidant, formation of active tetrahydrofolate, electron transport system, tryptophan metabolism, catecholamine synthesis, iron absorption (Chatterjee and Shinde, 2002; Imam et al., 2017). It is required for the maintenance of immune system and healthy body tissues.

The level of vitamin C has been found to be lower in patients with diagnosis of cardiac infarction and diabetes mellitus (Chatterjee and Shinde, 2002; Padayatty et al., 2003).

In various studies a connotation has been reported between vitamin C and atherosclerosis that assessed the relationship between vitamin C and cholesterol levels (Dubic and Hunter, 1987; Hillstrom et al., 2003; Padayatty et al., 2003). The aim of this study was to find the effect of vitamin C supplementation on lipid profile and blood sugar in normal and obese male Albino rats.

Materials and methods

Experimental animals

Sixteen male albino rats were provided by Experimental Animal Care and Experimental Surgery Center at the Faculty of Medicine, King Saud University, Saudi Arabia. This study is in accordance with the Animal Ethics Committee of the University.

The rats were randomly divided by weight into four groups. Group 1-3 consisted of rats with normal weight (150-250gm) and 4th group consisted of obese rats (350-400gm). They were housed individually in stainless steel cages under controlled temperature (25 ± 2°C) and relative humidity (50 ± 5%), with a 12-h light/dark cycle.

The Experimental Animal Care and Experimental Surgery Center at the Faculty of Medicine; King Saud University, Saudi Arabia provided the basal diet.

Control group received via oral route placebo -4 ml distilled water. Test group T1 and T2 (normal weight rats) received 2.5 ml and 5 ml vitamin C respectively and test group T3 (obese rats) also received 5 ml vitamin C orally. Each tablet of vitamin C (500 mg) was dissolved in 125 ml of distilled of distilled water.

Collection of blood

At the end of the experiment, on the 30th day, animals were food deprived overnight and anesthetized under chloroform. Blood was collected from the retro-orbital plexus in the heparinized tube and centrifuged at 3500 rpm for 15 min for plasma separation and stored at 5-7°C for further analysis.Kits from United Diagnostic Industry (UDI) were used to assess fasting serum levels of glucose (REF 037L), lipids profile [TG (UI59L), HDL-C (UI41HD), and TC (UI 24)].

Statistical analysis

The data are presented as the average of replicates ± SD. The data was subjected to statistical analysis by analyzing variance (ANOVA), using the SPSS software package (version 9.0). The significant differences identified using Turkey HSD tests, and p-values of < 0.05 were considered significant.
**Results and discussion**

In control group the mean values of cholesterol, triglycerides, HDL, LDL and glucose, were 164.30±56.15, 161.95±23.96, 85.61±54.57, 46.29±27.89 and 104.72±62.21 respectively (Table 1). The values for treatment groups T1, T2 and T3 for cholesterol were 168.14±22.99, 154.44±55.63 and 191.02±33.64 respectively. Those for triglycerides were 139.21±66.19, 65.57±28.31 and 151.14±35.66 respectively while those for HDL were 93.86±56.29, 101.09±52.69 and 93.26±31.69 for T1, T2 and T3 respectively. Similarly the values for treatment groups T1, T2 and T3 for LDL were 46.45±53.86, 40.24±30.29 and 67.53±22.32 and for glucose it was 77.14±34.10, 80.9±39.69 and 83.36±11.25 respectively. Vitamin C supplementation has been found to be effective in reducing triglyceride significantly (p≤0.05). Even though reduction has been observed in total cholesterol, LDL and glucose level in treatment group but when compared to control the reduction was insignificant. It can be depicted from Table 1 that vitamin C supplementation lead to increase in HDL level but statistically this increase was insignificant (p≥0.05). Lowest level of cholesterol, triglycerides, and LDL and highest level of HDL has been observed in T2 group.

**Table 1.** Effect of oral administration of vitamin C on lipid parameters and glucose level of Albino Wistar rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>164.30±56.15</td>
<td>161.95±23.96</td>
<td>154.44±55.63</td>
<td>191.02±33.64</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>161.95±23.96</td>
<td>139.21±66.19</td>
<td>65.57±28.31</td>
<td>151.14±35.66</td>
</tr>
<tr>
<td>HDL</td>
<td>85.61±54.57</td>
<td>93.86±56.29</td>
<td>101.09±52.69</td>
<td>93.26±31.69</td>
</tr>
<tr>
<td>LDL</td>
<td>46.29±27.89</td>
<td>46.45±53.86</td>
<td>40.24±30.29</td>
<td>67.53±22.32</td>
</tr>
<tr>
<td>Glucose</td>
<td>104.72±62.21</td>
<td>77.14±34.10</td>
<td>80.9±39.69</td>
<td>83.36±11.25</td>
</tr>
</tbody>
</table>

Data are expressed as the mean ± standard deviation; Model ANOVA, p values < 0.05 are significant. Superscript a,b indicates significant differences among various groups as indicated by ANOVA followed by Turkey HSD test. Test group T1 and T2 (normal weight rats) received 2.5 ml and 5 ml vitamin C respectively and test group T3 (obese rats) received 5 ml vitamin C orally.

Biochemically vitamin C is known as an antioxidant which removes the free radicals produced in the body. The results show that vitamin C improved the glycemic status and lipid profile by reducing the glucose level and lipid fraction up to some extent but not up to a statistically significant level. Similarly, previous studies have also revealed that supplementation of vitamin C to healthy individual was found to be effective but statistically insignificant in decreasing lipid profile (Satinderet al., 1987; Sharmaet al., 1988). A meta-analysis of 13 RCT shows that supplementation with at least 500 mg/d of vitamin C, resulted in a significant reduction in serum triglyceride and LDL cholesterol concentrations, though; there was an insignificant raise of serum HDL cholesterol has been observed (McRae MP., 2008). A possible explication for the perceived hypocholesterolaemic effect of vitamin C is that it thwarts LDL-cholesterol from oxidative damage and aids in degradation of cholesterol. Other reason might be that vitamin C is required by the enzyme 7α-hydroxylase in the first step of bile acid synthesis and it activates the enzyme 7α hydroxylase which increases the conversion of plasma cholesterol into bile acid henceforth causing reduction in serum levels of cholesterol (White et al., 1994). Scarcity of vitamin C impedes 7α hydroxylase which in turn led to blocking of bile acid synthesis and accumulation of cholesterol in serum (Mayes 1996; Chambialet al., 2013).

Even though in this study no significant changes has been detected with respect to HDL but previous studies shows that vitamin C protects HDL cholesterol from lipid oxidation thus letting it to be involved in the process known as reverse cholesterol transport (Hillstrom, 2003) and in preserving the cardio-protective knack of this lipoprotein fraction to
check atherogenic modification of LDL (Robert et al., 2003).

Oxidative stress is an imbalance between oxidants and antioxidants in favour of the former, potentially leading to cell damage and destruction (Sies, 1997). Vitamin C is structurally similar to glucose and can replace it in many chemical reactions and thus is effective for prevention of non-enzymatic glycosylation of protein (Ardekani and Ardekani, 2007). They observed that daily supplementation of 1000 mg vitamin C may be helpful in reducing blood glucose and lipids in patients with type 2 diabetes. It decreases glucose toxicity and contributed in part to the prevention of a decrease of β cell mass and insulin content.

The possible explanation for the beneficial effect of vitamin C on blood glucose level is that it seems to play a role in the modulation of insulin action. Vitamin C-mediated increase in insulin action is mainly due to an improvement in non-oxidative glucose metabolism (Eriksson et al., 1997; Ceriello and Motz 2004.).

The blood glucose-lowering effect of Vit-C may be attributed to its inhibition of oxidative stress; that is, Vit-C scavenging of ROS within the aqueous system of the body, protecting protein and DNA from oxidative damage (Franke et al., 2005). Hence, vitamin C reduced blood glucose toxicity and prevented damage to beta-cell mass and insulin content.

Conclusion
The outcome of this study shows hypcholesterolaemic effect. It has been observed that supplementation of vitamin C to healthy wistar rat significantly decreased triglyceride. HDL level increased and cholesterol, LDL and glucose level decreased with supplementation of vitamin C to healthy wistar rat but the differences was statistically insignificant. Short term supplementation and lower dose might be the reason, so further investigation with higher dose and longer duration are recommended.

References


http://dx.doi.org/10.3181/00379727-184-42457


http://dx.doi.org/10.1016/j.mrgentox.2005.03.001

http://dx.doi.org/10.1002/art.20781

http://dx.doi.org/10.1093/jn/133.10.3047

http://dx.doi.org/10.3390/nu9070671


http://dx.doi.org/10.1016/j.jcme.2008.01.002

http://dx.doi.org/10.1080/07315724.2003.10719272

http://dx.doi.org/10.1093/jn/133.10.3047


