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Original Article

Effect of Oral Vitamin C Supplementation on Serum Interleukin 6, Hepcidin and Iron Status in Type 2 Diabetes Patients with Metabolic Syndrome

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ABSTRACT

Background and Aim: Type 2 Diabetes (T2DM) with Metabolic Syndrome (Mets) is a chronic inflammatory state with an increased expression of inflammatory cytokines such as interleukin 6 (IL-6). Iron regulatory peptide hepcidin secretion is also increased as IL-6 causes hepcidin secretion, leading to iron dysregulation. Vitamin C (L ascorbic acid) has potential effects in alleviating the inflammatory status. This study aimed to investigate the effect of vitamin C supplementation on serum interleukin 6 (IL-6), hepcidin, and iron status in T2DM patients with Mets. **Methods:** Total 76 patients with the age between 40-60 years according to Adult Treatment Panel (ATP) III were selected and randomly assigned to supplement (vitamin C or placebo) groups using block randomization. Before and after 8-week supplementation of 250 mg vitamin C/placebo tablets for two times per day, serum Iron status, IL-6 and hepcidin levels were measured, using the colorimetric and the Enzyme-Linked Immunosorbent Assay. **Results:** In both groups, serum IL-6 level was significantly increased, ferritin level was significantly decreased after supplementation while changes in hepcidin levels were not statistically significant. Moreover, percent changes of all variables were not significantly different between groups. **Conclusion:** Vitamin C 250 mg two times per day for 8-week supplementation with could not provide beneficial effect to explore the anti-inflammatory effect in T2DM patients with metabolic syndrome.

Keywords: Vitamin C, Interleukin 6, Hepcidin, Iron Status and T2DM Patients with Mets.

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) with Metabolic Syndrome (MetS) is a chronic low grade inflammatory disorder and its worldwide prevalence is increasing fast among developing countries. In a chronic inflammatory condition, there is imbalance production and secretion of pro-inflammatory and anti-inflammatory cytokines from the inflammatory tissues and hence increased systemic pro-inflammatory cytokines, Interleukin-6 (IL-6) level is seen in obesity, glucose intolerance, insulin resistance, and T2DM.[1] The chronic inflammatory diseases interfere with the body's ability to use stored iron and absorb iron from the diet and it is leading to derangement in iron metabolism.[2] In healthy humans, the concentration of iron in plasma and extracellular fluid is maintained in a relatively narrow range of 10-30 μM, assuring that adequate iron is available for essential cellular functions without incurring iron toxicity. Plasma iron concentration is controlled by the hepatic peptide hormone hepcidin which regulates iron flows into plasma.[3] The sole known molecular target of hepcidin is the protein ferroportin which functions as a transmembrane conduit for the transfer of cellular iron to plasma. The binding of hepcidin to ferroportin on the membranes of iron-exporting cells such as macrophages, duodenal enterocytes and hepatocytes induces the endocytosis and proteolysis of ferroportin and thereby decreasing iron delivery from cells to plasma.[4] Thus, an increase in hepcidin could lead to decrease serum iron level and increase intracellular iron store. Hepcidin is increasingly being recognized in a systemic inflammatory state due to upregulation by inflammatory cytokines.[5,6]

Previous study conducted in Myanmar found that serum ferritin level was significantly higher in T2DM with MetS than those without MetS and iron status was in the lower normal range in T2DM with and without MetS.[7] In that study, the level of inflammatory cytokines and/or hepcidin were not determined to explain the mechanism of action of hyperferritinemia in low/normal iron parameters. A population study reported that subjects with MetS have increased serum hepcidin level, and that in subjects of both sexes hepcidin increased linearly with

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increasing number of the five classical MetS features.[8] They also found a strong association between hepcidin and ferritin.

Vitamin C (L ascorbic acid) is an essential dietary nutrient. It is a water-soluble reducing agent and antioxidant due to its characteristic of donating an electron.[9] Vitamin C contributes to maintain the redox integrity of cells and thereby protects them against reactive oxygen species generated during the respiratory burst and in the inflammatory response.[10] Moreover, some studies illustrated that vitamin C has potential effects in alleviating inflammatory status. According to Ellulu *et al.*, oral consumption of ascorbic acid (500 mg twice daily) for 8 weeks has potential effects in improving inflammatory status by reducing hs-CRP, IL-6, and Fasting Blood Glucose (FBG) in hypertensive and/or diabetic obese patients.[11] *In vitro* research demonstrated that 50 and 100 μg/mL of vitamin C directly inhibits hepcidin expression and enhances erythropoietin receptor expression in HepG2 cells cultures after 6 hr.[12] Taken together these literatures, it could be assumed that the oral vitamin C supplementation would decrease the inflammatory markers as well as hepicidin level and therefore, the present study aims to investigate the effect of vitamin C supplementation on serum IL-6, hepcidin and iron status among the type 2 diabetes patients with metabolic syndrome.

MATERIALS AND METHODS

Study Design and Population

A double-blind randomized placebo-controlled study was conducted in 76 T2DM patients with Mets who visit Primary and Sub-Health Diabetes Day Care Centers, North Okkalapa Township, Yangon, Myanmar. Patients were recruited according to inclusion and exclusion criteria. Inclusion criteria were both male and female patients of age 40-60 years who were taking any oral hypoglycemic drugs and those met any three out of five features of Mets according to Adult Treatment Panel (ATP) III criteria. Patients who need insulin injection for diabetic controlled, with diabetic complication such as diabetic nephropathy, known haematological, chronic inflammatory or liver diseases, with current acute illness or other medical illness in the past two weeks, being vegetarian, current smoker, drinking alcohol and taking anti-inflammatory drugs were excluded. After obtaining written informed consent from each patients, detailed procedure was explained, history taking and physical examination were conducted according to proforma. This study was approved by the Ethics Review Committee of the University of Medicine 1, Yangon, M.

Participants Allocation to Groups

There were two groups: vitamin C supplement group and the placebo supplement group. The selected patients were given supplement tablets, either vitamin C or placebo, according to a random allocation sequence. If the selected patients who had missed for daily taking of tablets or no longer wanted to take the drug, they were allowed to withdraw from the study. They were regularly checked at follow-up visit for every two weeks and no subjects withdrew from this study. The coding of vitamin C tablets and placebo tablets was done first and separated into identical plastic containers. These plastic containers were labeled with code numbers. These procedures were overseen by the supervisor. Subjects were randomly assigned supplements (vitamin C or placebo) using block randomization. One group received vitamin C (250) mg tablets for two times per day, while the other group received a placebo for eight weeks.

Procedure

At the first place, vitamin C and placebo preparations were done before doing patients recruitment. Vitamin C (250 mg) and placebo tablets with same colour, mass, weight to vitamin C (standard inactive) were prepared at Research Drug Production Department, Pharmacology Research Division, No. 1 Myanmar Pharmaceutical Factory, Yangon, Myanmar. At first time visit, baseline 5 mL of venous blood was collected from a peripheral vein under aseptic condition using a disposable syringe and needle for each subject after completing patient selection. The blood was collected in serum separator test tubes and transported to the Post-Graduate Research Laboratory, Department of Physiology, University of Medicine 1, Yangon, Myanmar in a cold-box. The blood was then centrifuged for 10 min at 2000 rpm and the serum was separated and stored in separate screw-tight bottles at -20°C until analysis at the Post-Graduate Research Laboratory, Department of Physiology, University of Medicine 1, Yangon, Myanmar. Serum iron and Total Iron-Binding Capacity (TIBC) were measured within one week after collection by colorimetric method and serum transferrin saturation was calculated from serum iron and TIBC concentration. Serum ferritin, serum IL-6, serum hepcidin levels were determined by Enzyme-Linked Immunosorbent Assay (ELISA) method. After completing the 8-week intervention period, a second time venous blood sample was collected and determination of same parameters were done again at the Post-Graduate Research Laboratory, Department of Physiology, University of Medicine 1, Yangon, Myanmar.

Statistical Analysis of Data

Data entry and analysis were done by SPSS software (Statistical Package for Social Sciences) version 16.0. Data was described by Mean±SD and median, Interquartile Range (IQR). Paired *'t'* test was used to compare serum IL-6 level, serum hepcidin level, serum iron status (serum iron, TIBC, transferrin saturation and serum ferritin) in each group before and after intervention. Mann-Whitney test was used for comparison of before and after percent changes in measuring parameters (serum IL-6 level, serum hepcidin level, serum iron status). *P* value <0.05 was accepted as significant level.

RESULTS

The baseline characteristics of the study groups are presented in Table 1. A total of 76 participants were enrolled, with 38 subjects each in the vitamin C and placebo groups. The mean age of participants in the vitamin C group was 50.71±6.76 years, while that of the placebo group was 51.95±6.65 years, and the difference was not statistically significant (P =0.424). Similarly, there were no significant differences between the groups in terms of Body Mass Index (BMI), waist circumference, blood pressure, fasting blood glucose, HbA_{1C}, and lipid profile, indicating comparability between the groups at baseline. However, significant group differences were observed in height (P = 0.041), weight (P = 0.022), and waist circumference (P = 0.022), with participants in the vitamin C group showing slightly higher values.

Figures 1 and 2 show comparison of serum IL-6 level before and after 8-week of vitamin C/placebo supplement in vitamin C and placebo supplement groups. Serum IL-6 levels were significantly increased after vitamin C/ placebo supplementation (P< 0.05). Figures 3 and 4 show comparison of serum Hepcidin level before and after 8-week of vitamin C/placebo supplement in vitamin C and placebo supplement groups. Serum Hepcidin levels were not significantly different after vitamin C/placebo supplementation (P<0.05).

Table 2 shows the comparison of serum iron status before and after 8-weeks vitamin C/placebo supplement in vitamin C and placebo supplement group. There was no significant difference in serum iron, TIBC and transferrin saturation (P>0.05) whereas a significant decrease in serum ferritin level was found after vitamin C supplementation (P<0.05). There was no significant difference in serum iron status between before and after placebo supplementation (P>0.05). The percent changes of all parameters were not significantly different after vitamin C/ placebo supplementation in vitamin C and placebo supplement group was shown in Table 3.

Table 1: General characteristics of study groups.

Parameters	Vitamin C group (n=38)	Placebo group (n=38)	P value
Age (year)	50.71±6.76	51.95±6.65	0.424
Height (m)	1.53±0.05	1.50 ± 0.05	0.041*
Weight (kg)	67.20±9.74	62.47±7.84	0.022*
BMI (kg/m^2)	28.75±3.63	27.73±3.63	0.226
Waist Circumference (cm)	100.11±7.90	96.03±7.25	0.022*
Resting SBP (mmHg)	119.74±5.92	118.42±6.38	0.354
Resting DBP (mmHg)	79. 74±4.34	80.26±6.36	0.675
Total Cholesterol (mg/dL)	210.32±25.37	201.84±26.02	0.155
Triglyceride (mg/dL)	162.24±8.98	161.21±9.52	0.630
RBS (mg/dL)	161.03±22.05	154. 11±20.98	0.165
HbA_{1C} (%)	6.98±0.27	6.92±0.25	0.294

Values are presented as mean±standard deviation. Independent sample *t*-test was used for comparison between groups.
P<0.05 considered statistically significant. BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; RBS: Random blood sugar; HbA_{1C}: Glycated hemoglobin.

Table 2: Comparison of serum iron status before and after 8 weeks of vitamin C /placebo supplement in vitamin C and placebo supplement groups.

Iron status	Vitamin C supplement group (n=38)			Placebo supplement group (n=38)		
	Before supplementation	After 8-week supplementation	p value	Before supplementation	After 8-week supplementation	p value
Serum iron (ug/dL)	95.28±14.69	94.62±12.61	0.805	95.99±14.83	94.52±14.47	0.482
TIBC (ug/dL)	288.30±11.56	289.58±13.50	0.469	290.68±21.24	291.30±19.89	0.853
Transferrin saturation %	33.05±5.01	32.71±4.38	0.735	33.09±5.17	32.51±5.04	0.472
Ferritin (ng/mL)	93.68±51.28	86.46±48.18	0.006*	78.70±49.98	74.73±47.47	0.270

Values are presented as mean±standard deviation. Within-group comparisons were performed using paired t-test. p<0.05 considered statistically significant. TIBC: Total iron binding capacity.

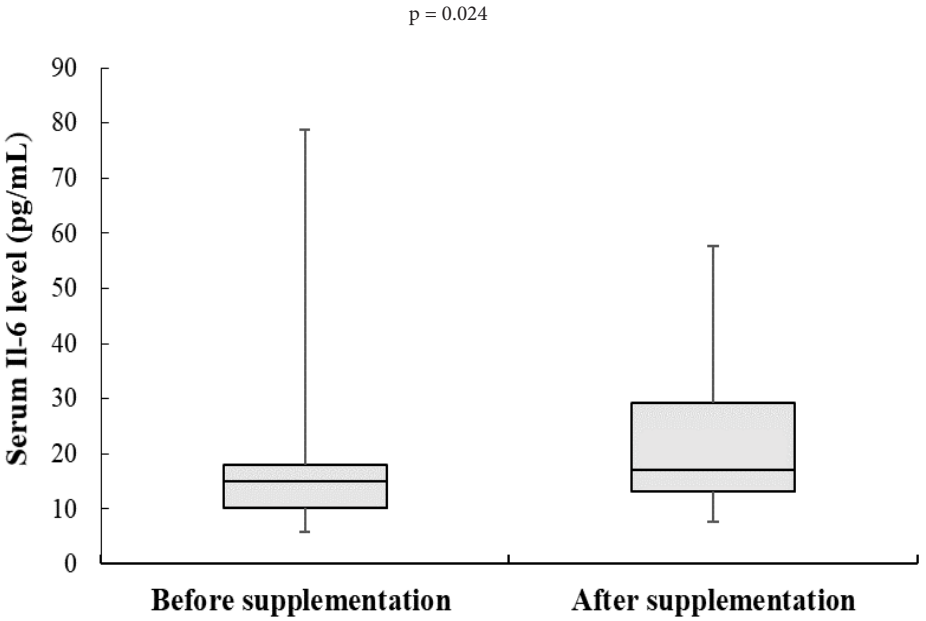

Figure 1: Serum IL-6 level before and after 8-week vitamin C supplementation. IL-6: Interleukin-6.

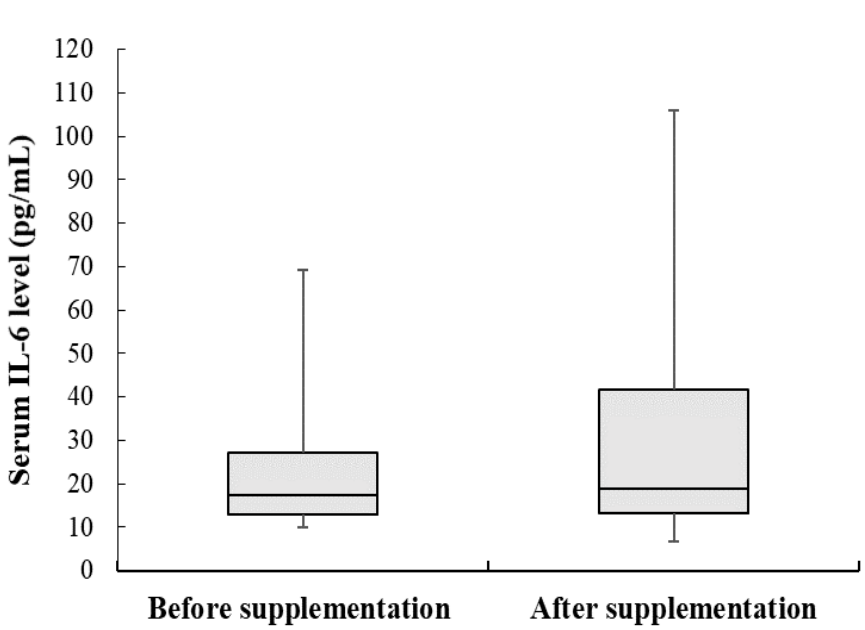

Figure 2: Serum IL-6 level before and after 8-week placebo supplementation. IL-6: Interleukin-6.

Table 3: Percent changes in biochemical variables between vitamin C and placebo groups.

Parameters	Vitamin C (n=38)	Placebo (n=38)
IL - 6	29.13 (-17.65 to 77.70)	6.01 (-13.05 to 87.50)
Hepcidin	-1.73 (-9.57 to 6.76)	0.28 (-11.89 to 10.05)
Serum iron	-0.38 (-10.43 to 14.77)	-3.62 (-9.78 to 6.14)
TIBC	0.65 (-2.40 to 2.54)	1.13 (-3.69 to 5.09)
Transferrin saturation	-2.54 (-14.50 to 16.65)	-3.94 (-12.70 to 9.28)
Ferritin	-8.33 (-16.31 to 1.53)	-4.07(-17.46 to 9.51)

Values are expressed as median (interquartile range, IQR). Between-group comparisons were performed using the Mann-Whitney U test. IL-6: Interleukin-6; TIBC: Total iron binding capacity.

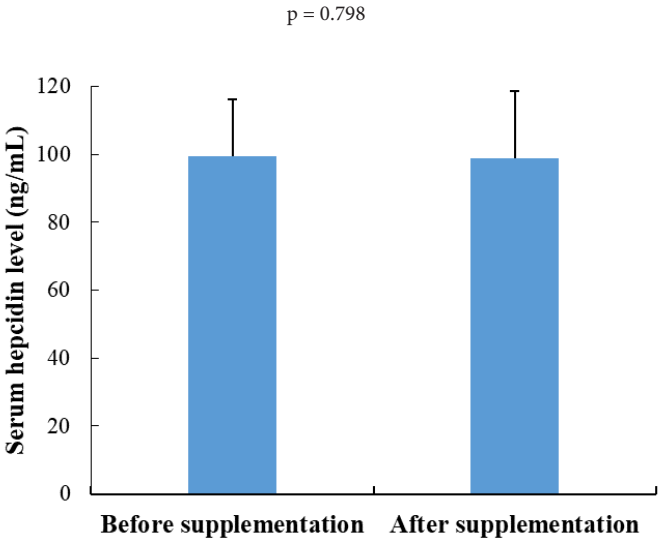

Figure 3: Serum hepcidin level before and after 8-week vitamin C supplementation.

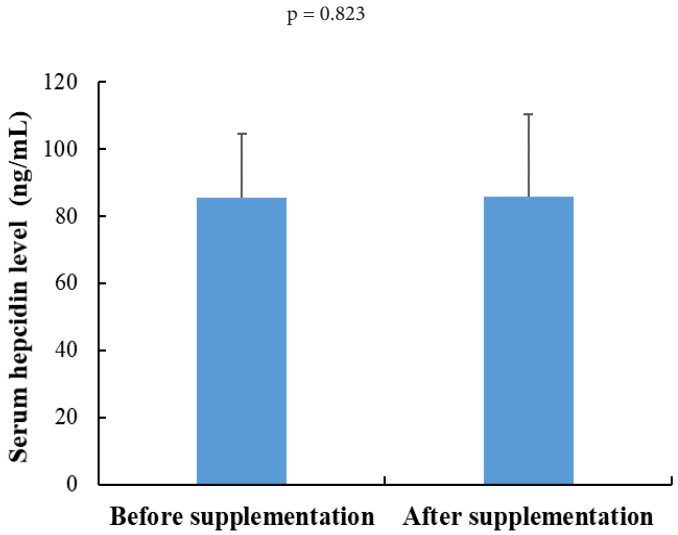

Figure 4: Serum hepcidin level before and after 8-week placebo supplementation.

DISCUSSION

According to the general characteristics of patients, BMI was not significantly different (P>0.05) but height, body weight and waist circumference were significantly higher in vitamin C group (P<0.05). The significant difference in general characteristics of patients were unpredictable data because the present study design was double blind placebo controlled study in which patients were taken drug or placebo by using random allocation sequence according to random letter table. In the present study, serum IL-6 (median, IQR) level after taking vitamin C was significantly higher than that before taking vitamin C [before: 14.94 pg/mL (10.09 - 18.06) vs after: 17.16 pg/mL (13.27 - 29.12)] (P<0.05) and also in placebo group [before: 17.40 pg/mL (12.86 - 19.64) vs after: 18.82 pg/mL (13.19 - 29.58)] (P<0.05). However, percent changes in serum IL-6 levels were not significantly different between vitamin C and placebo groups before and after respective supplementation (P>0.05). The findings of the present study were in accordance with the report of Antoniades *et al*., 2004 and Lu and colleagues, 2005.[13,14] In those studies, serum IL-6 levels of diabetes patients were also significantly increased after vitamin C supplementation and the amount of vitamin C supplementation was greater than that of the present study (2 g/day for 4 weeks and 1g × 3 times/day for 2 weeks respectively). Actually, generalized inflammatory processes are continuing along with increasing level of inflammatory cytokines during the disease progression.[13] In inflammatory responses, antioxidant vitamins are used up to neutralize the free radicals formed by glucose oxidation, non-enzymatic glycation of proteins and subsequent oxidative degradation of glycated proteins.[15] Therefore, the findings of the present study as well as these previous studies indicated that higher doses and longer duration of antioxidants might be required in diabetic patients for supporting the anti-inflammatory effect. However, the value of very high supra-physiological doses of ascorbic acid used in some acute studies has been controversial. Moreover, mechanism of action of that vitamin depends on the actual plasma concentrations.[14] As a limitation in the present study, the plasma level of vitamin C was not determined to reveal whether plasma vitamin level is increased to attain the efficient antioxidant status. The findings of the present study were contrary to those of randomized placebo-controlled study done by Ellulu *et al*., which stated that effect of vitamin C in alleviating inflammatory status by reducing the IL-6 level.[11] In that study, 31 numbers of hypertensive and/or diabetic patients were taken vitamin C (500 mg twice daily for 8 weeks) and the majority of participants were instructed to undertake physical activity during the trial period. The discrepancy in the findings might probably be due to different intervention plan since their plan was taking higher dose of antioxidant plus regular exercise whereas the present one was only vitamin C supplementation with lower dosage. Therefore, daily vitamin C 500 mg supplementation for 8 weeks could not have noticeable effect on anti-inflammation. In the present study, serum hepcidin (mean±SD) level was not significantly decreased after vitamin C supplementation (before: 99.38±16.80 ng/mL vs after: 98.82±19.66 ng/mL) and after placebo supplementation (before: 85.28±19.23 ng/mL vs after: 85.75±24.58 ng/mL). Percent changes in serum hepcidin levels were not significantly different between vitamin C and placebo groups before and after respective supplementation (P>0.05). The effect of vitamin C on serum hepcidin level was not found after 8-week supplementation in the present study, although a study found that hepcidin expression was directly inhibited by vitamin C (50 and 100 μg/mL) in HepG2 cells cultures after 6 hours.[12] Moreover, oral vitamin C (500 mg per day for three months) supplementation on hemodialysis patients with iron deficiency anaemia showed that serum hepcidin level in vitamin C group was statistically significant reduced than that of control group.[16] The

study of Lee *et al.*, stated that IL-1 plays a more important role in stimulation of hepcidin expression than does IL-6 in the liver.[17] That study demonstrated that incubating murine hepatocytes with IL-6, IL-1α and IL-1β strongly stimulates hepcidin transcription but IL-6 encoding gene disrupted mice respond to endotoxin by increasing the expression of hepcidin transcripts in the liver. In addition, Siemonsma *et al.*, found that Hepcidin was not associated with markers of inflammation but was strongly associated with all other iron indices. In that study, serum hepcidin concentrations were similar in all the groups Gambian women {lean women (n=42, Body Mass Index (BMI)=20.9 kg/m2), women with obesity (n=48, BMI=33.1 kg/m2) and women with obesity-T2D (n=30, BMI=34.5 kg/m2)}, while women with obesity and obesity-T2D showed elevated levels of inflammatory markers (IL-6), higher transferrin saturation and higher serum iron concentration.[18] In the present study, serum IL-6 levels were significantly increased in both groups after respective supplementation whereas serum hepcidin levels were not significantly increased it might be due to the presence of other confounding factors which promote the hepcidin expression in diabetes, such as inflammatory cytokines like IL-1, TNFα etc. and other microbial products. Therefore, the inhibitory effect of vitamin C on hepcidin secretion might probably depend on vitamin C dose and duration of supplementation.

In the present study, there was no significant changes in serum iron, TIBC, and transferrin saturation in both groups, but serum ferritin level was significantly decreased ($p<0.05$) after vitamin C supplementation. The ferritin is the major iron storage protein and it is also regarded as well-known inflammatory marker. Since transferrin is bound with free iron in the blood and glycation of transferrin decreases its ability to bind free iron (Fe^{2+}) and hence increased free iron pool in turn facilitates ferritin synthesis.[15] A double blind placebo controlled study of Dakhale *et al.*, showed that Fasting Blood Glucose (FBG) and Post-Meal Blood Glucose (PMBG) were significantly reduced after oral vitamin C (500 mg × b.d for 12 weeks) supplementation with metformin.[19] Moreover, serum ferritin is well correlated with baseline serum glucose level and beta cell function.[20] Therefore, it is probable that vitamin C effect on serum ferritin might be indirect effect through reducing blood glucose level, rather than its anti-inflammatory effect.

CONCLUSION

The present findings suggested that the chosen dose of vitamin C did not have anti-inflammatory effect. In spite of vitamin C supplementation, generalized inflammatory processes were continually occurring and Inflammatory cytokine (IL-6) was increasingly produced in T2DM with MetS. Therefore, future research with higher doses and longer duration of antioxidant vitamin C should be done to explore the anti-inflammatory effect of vitamin C in T2DM with MetS.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

ATP III: Adult Treatment Panel III; **BMI:** Body Mass Index; **ELISA:** Enzyme-Linked Immunosorbent Assay; **FBG:** Fasting Blood Glucose; **HbA_{1C}:** Glycated Hemoglobin; **IL-1:** Interleukin-1; **IL-6:** Interleukin-6; **MetS:** Metabolic Syndrome; **PMBG:** Post-Meal Blood Glucose; **ROS:** Reactive Oxygen Species; **T2DM:** Type 2 Diabetes Mellitus; **TIBC:** Total Iron-Binding Capacity.

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