Metadichol® Induced High Levels of Vitamin C: Case Studies

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Abstract

We recently reported that Metadichol®[1] brings about a three to four-fold increase in Vitamin C levels in patients without the use of Vitamin C supplementation. In this study of 6 patients who experienced a 5-12 fold increase in plasma Vitamin C levels higher than 80-100 u mol/L level which is the highest reported to date by oral supplementation at high doses of Vitamin C. Metadichol improved in these patients TSH levels, normalized High Blood pressure, fasting glucose levels, reduced eosinophil count, high triglycerides, body fat reduction and increased bone mass, normalized sodium levels, reducing high insulin levels, increased creatinine output in urine and also reducing of Red Cell Distribution width %. Metadichol thus serves as a surrogate for Vitamin C at doses of 5 mg per day as opposed to mega doses that are currently used.

Keywords: Vitamin C; Dehydroascorbic acid; Metadichol; Nanolipid; Long chain saturated alcohols; VDR; Inverse agonist; Glutathione; RDW; TSH; Thyroid antibodies; Gulo; Pseudo genes; Bone density; Asthma; Eosinophil count Vitamin E; G6PD; Glucose Transporters; Nuclear receptors

Introduction

Vitamin C is a ubiquitous key nutrient. Humans do not have the ability to produce and synthesize vitamin C in their bodies and must obtain it from food sources. Vitamin C synthesis is not possible in guinea pigs, monkeys, and apes. Humans lack the L-Gluconolactone oxidase (GULO) enzyme that completes the last enzymatic step of synthesis of vitamin C from glucose [2]. In the genome of humans and that of the anthropoid primates, a non-functional gene is present. This inability to synthesize vitamin C in primates is believed to have occurred about 60 million years ago [3].

Vitamin C is essential to maintain a sensitive balance in the functioning of the organs in the body. It participates in the: hydrolysis of individual amino acids, the formation of collagen necessary for body growth, tissue repair and wound healing, synthesis of the hormone adrenaline (epinephrine) and the hydroxylation of anti-inflammatory steroids in the adrenal gland, iron and copper metabolism, and folic acid metabolism. Vitamin C is related to the metabolism of the vitamins A, E, B6 and B12 [4]. The concentrations of total ascorbic acid reduced and oxidized forms of ascorbic acid decline with age [5].

A review of the health benefits of vitamin C

The role of vitamin C in thyroid disease: Dubey et al. have documented low levels of ascorbic acid in patients with hyperthyroidism [6]. These have been confirmed by Ademoglu et al. [7] and Aliciğüz et al. [8] Kumar et al. described low levels of vitamin C in hyperthyroidism, while at the same time lipid peroxides, glucose, and HbA1C levels were elevated [9].

Vitamin C and hypertension: Some epidemiological studies have shown a negative correlation between BP and vitamin C [10,11,12]. Vitamin C use as a supplement improves vascular function and reduces blood pressure in both experimental models and patients. Ascorbate improves vasodilation, probably by increasing Nitric Oxide bioavailability. Mullan et al. showed that chronic daily supplementation with 500 mg oral ascorbic acid could lower blood pressure and improve arterial stiffness in patients with type 2 diabetes (Figure 1).

Vitamin C and bone density: Low plasma levels of vitamin C leads to scurvy, which, among other ailments, causes gingiva, bone pain, and impaired wound healing. A review by Patrick et al [13] describes the importance of vitamin C as it relates to maintenance of bone tissues. Various epidemiological studies and animal models show that Vitamin C has a positive on trabecular bone formation by influencing the expression of bone matrix genes in osteoblasts.

Vitamin C and metabolic syndrome: Pearson et al. have reviewed the literature on Vitamin C and its and showed that adults with higher vitamin C levels show lower weight, BMI and waist circumference, and improved metabolic health biomarkers like HbA1c, insulin and triglycerides, that are major risk for type 2 diabetes [14].

Vitamin C and Asthma: Asthma leads to inflammation of the airways. Oxidants are one of the important factors that damage the airways. Vitamin C is a protective antioxidant of the airways and decreasing airway eosinophils in asthma is an indicator improved asthmatic conditions. Low levels of antioxidants lead to more severe asthma [15]. One needs to take mega doses of vitamin C to be effective. Typical dietary quantities and low supplemental doses do not work. [16,17].

Oral and IV Vitamin C and Plasma levels

Vitamin C concentration is controlled by intestinal absorption, tissue transport, and renal reabsorption [18]. With increasing oral doses of vitamin C up to 1000 mg, Levine et al. showed that plasma vitamin C concentration reaches a steady-state concentration (60 to 80 μ mol/L) in healthy young adults [19,20]. Once plasma ascorbic acid levels reach saturation, additional vitamin C gets excreted in the urine.

IV administration of vitamin C bypasses the intestines and high concentrations of ascorbic acid are achievable in the plasma; over time.

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But renal excretion restores vitamin C to baseline plasma levels [21]. Recent clinical reports indicate that the role of high-dose intravenous vitamin C therapy in cancer treatment could be useful [22].

The higher blood level of Vitamin C is associated with lower mortality and morbidity in several chronic conditions [23]. Metabolism of vitamin C, including absorption and its uptake by several cell types, is inhibited by increasing glucose concentration. The glucose-ascorbate antagonism (GAA) theory [24] suggests elevated glucose levels restrict vitamin C from entering cells. Dehydroascorbic acid (DHAA) transport into cells was shown to be impaired by high blood glucose level in most cell types including adipocytes, erythrocytes, neutrophils, osteoblasts and smooth muscle cell [25,26].

Metadichol® is a mixture of C-26, C-28 and C-30 straight chain alcohols. It is analogous with Vitamin D which is a cyclic C-27 alcohol. The generic name is Policosanol. Metadichol is a Nano formulation of these with a particle size of 53 nm. It is inverse agonist of Vitamin D receptor (VDR). It is the only one of its kind known in literature today. We have already documented that it increases Vitamin C levels leading to improvement in disease conditions as we show in case Studies below.

The patient information sheet detailed the procedures involved in the study (aims, methodology, potential risks, anticipated benefits) and the investigator explained these to each patient. The patient signed the consent form to indicate that the information had been explained and understood. The patient was then allowed time to consider the information presented before signing and dating the informed consent form to indicate that they fully understood the information and willingly volunteered to participate in the study. The patient was given a copy of the informed consent form for their information. The original copy of the informed consent was kept in a confidential file in the Investigators center records. Criteria for exclusion from the study included pregnant or lactating females any serious and or uncontrolled medical conditions interfering with the study or placing the patient at unacceptable risk. Only patients who fulfilled all the inclusion criteria and did not meet any of the exclusion criteria were enrolled into the study.

Subjects aged 18 years of age Various medical conditions received. Metadichol at 5 mg per day orally Subject visits were scheduled at Baseline/Day O (Visit 1), at Day 30 (Visit 2), Day 60 (Visit 3), and Day 90 (Visit 4) End of the Study. All visit occurred within Follow up visit day+6 days. The study population consisted of male or non-pregnant female patients aged 18 years of age with a previously diagnosed medical conditions. All patients provided written informed consent to participate in the study prior to being screened.

**Methods and Objective**

Non-randomized, Open study of 6 patients with various morbidities with a below normal Vitamin C at baseline study on efficacy of improved Vitamin C levels on various biomarkers with use of Metadichol @ 5 mg per day for 90 days. One patient was followed for an additional 90 days.
Results and Discussion

The results shown involved patients with a wide variety of disease symptoms. The common theme is that in all the cases, there was a significant increase of Vitamin C, 3 to 11 times, that led to normalizing key biomarkers for each patient. We have already shown how Metadichol affects various diseases. Including Type 2 diabetes [27], decreasing RDW [28], controlling of diastolic and systolic pressure, [29].

The role of Vitamin D in thyroid related diseases is well known, and the effect of, Metadichol on thyroid given its binding to VDR is not surprising [35].

The actions of Metadichol binding to VDR leads to a proposed pathway which efficiently recycles Vitamin C (Figure 7).

In addition to serving as a substitute for Vitamin D, Metadichol is also behaving as a Vitamin C substitute. It is possible to envision that increased Vitamin C levels and VDR binding leads to a synergism operating through multiple pathways to bring about homeostasis. Complex diseases need drugs acting on distinct targets, which are part of a network regulating various physiological responses. Metadichol is exhibiting a poly pharmacological phenomenon by acting on multiple targets pertaining to multiple disease pathways [36].
C as observed in plasma with Metadichol. IV infusion of Vitamin C does allow for higher levels to be reached, but that is not a practical solution and is more useful in life threatening illnesses. Metadichol by binding to VDR could serve as a useful surrogate of Vitamin C a leading to in leading to higher levels in plasma and rectifying diseases.

References

1. Raghavan PR (2017) Metadichol ® and Vitamin C Increase In Vivo, an Open-Label Study. Vitam Miner 6: 163.
2. Nobile S, Woodhill JM (1981) Vitamin C: the mysterious redox-system a trigger of life? MTP Press; Lancaster, Boston, pp: 1-185.
3. Kagawa Y (2009) Human Specific Vitamin C Metabolism and Xenobiotic Polymorphism: The Optimal Nutrition. In: Kucharski H, Zajac J (eds.) Nova Science Publishers, Inc, pp: 45-85.
4. Levy T (2002) Vitamin C, Infectious Diseases, and toxics: Curing the Incurable. Xlibris Corp, pp: 11-447.
5. Sasaki R, Tohoku J (1983) Influences of sex and age on serum ascorbic acid. J Exp Med 140: 97-104.
6. Dubey SS, Singh RP, Udupa KN (1977) Ascorbic acid status of hyperthyroid patients. Indian J Med Res 65: 865-870.
7. Ademoglu E (1998) The effect of methimazole on the oxidant and antioxidant system in patients with hyperthyroidism. Pharmacol Res 38: 93-96.
8. Aicigüzel (2001) Erythrocyte, plasma, and serum antioxidant activities in untreated toxic multinodular goiter patients. Free Radic Biol Med. 30: 665-670.
9. Mohan Kumar KM (2004) Possible link between glycated hemoglobin and lipid peroxidation in hyperthyroidism. Clin Chim Acta 342: 187-192.
10. Duffy SJ, Gokce N, Holbrook M, Liza M, Elizabeth S, et al. (2001) Effect of ascorbic acid treatment on conduit vessel endothelial dysfunction in patients with hypertension. Am J Physiol Heart Circ Physiol 280: 528-534.
11. Duffy SJ (1999) Treatment of hypertension with ascorbic acid. Lancet, 354: 2048-2049.
12. Rodrigo R, Prat H, Passalaquca W, Araya J, Guichard C, et al. (2007) Relationship between oxidative stress and essential hypertension. Hypertens Res 30: 1159-1167.
13. Mullen BA, Young IS, Fee H, McCance DR (2002) Ascorbic Acid Reduces Blood Pressure and Arterial Stiffness in Type 2 Diabetes. Hypertension 40: 804-809.
14. Aghajanian P (2015) J Bone Miner Res 30: 1945-1955.
15. Misso NL, Wildhaber J, Ray S, Vally H, Thompson PJ (2005) Plasma concentrations of dietary and non-dietary antioxidants are low in severe asthma. Eur Respir J 26: 257-264.
16. Harik-Khan RI, Muller DC, Wise RA (2004) Serum vitamin levels and the risk of asthma in children. Am J Epidemiol 159: 351-357.
17. Levine M, Padayatty SJ, Espay MG (2011) Vitamin C: a concentration-function approach yields pharmacology and therapeutic discoveries. Adv Nutr 2: 78-88.
18. Levine M (1996) Vitamin C pharmacokinetics in healthy volunteers: evidence for a recommended dietary allowance. Proc Natl Acad Sci 93: 3704-3709.
19. Levine M, Wang Y, Padayatty SJ, Morrow J (2001) A new recommended dietary allowance of vitamin C for healthy young women. Proc Natl Acad Sci 98: 9842-9846.
20. Padayatty SJ, Sun H, Wan Y, Hugh D, Hewitt S, et al. (2004) Vitamin C pharmacokinetics: implications for oral and intravenous use. Ann Intern Med 140: 533-537.
22. Padayatty SJ, Sun H, Wan Y, Hugh D, Hewitt S, et al. (2006) Intravenously administered vitamin C as cancer therapy: three cases. CMAJ: Canadian Medical Association 174: 937-942.

23. Clemens Z, Csaba T (2016) Vitamin C and Disease; Insights from the Evolutionary Perspective, Journal of Evolution and Health 1: 13.

24. Hamel EE, Santisteban GA, Ely JT (1986) Hyperglycemia and reproductive defects in non-diabetic gravidas: a mouse model test of a new theory. Life Sci 39: 1425-1428.

25. Malo C, Wilson JX (2000) Glucose modulates vitamin C transport in adult human small intestinal brush border membrane vesicles. J Nutr 130: 63-69.

26. Wilson JX (2005) Regulation of vitamin C transport. Annu Rev Nutr 25: 105-125.

27. Raghavan PR (2010) Metadichol and Type 2 Diabetes A case report. Journal of the Science of Healing Outcomes 31: 19-25.

28. Raghavan PR (2017) Systolic, and Diastolic BP Control in Metabolic Syndrome Patients with Metadichol® a Novel Nano Emulsion Lipid. J Cardiol & Cardiovasc Ther 5: 555660.

29. Chuan Fan, Theresa T (1940) Effect of Vitamin C on Creatine and Creatinine Metabolism. Exp Bio Medi 45: 90-92.

30. Isabelle C (2012) The Vitamin D Receptor in Thyroid Development and Function. Eur Thyroid J 1: 168-175.

31. Hopkins AL (2007) Network pharmacology. Nat Biotechnol 25: 1110-1111.

32. Pavlovic V (2010) A short overview of vitamin C and selected cells of the immune system. Cent Eur J Med 8: 1-10.