

SARS-Cov Treatment: Natural chemistry of vitamin c

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Abstract

L-ascorbic acid's (L-ascorbic corrosive) job as a cell reinforcement and cofactor in an assortment of metabolic exercises has a long history and is notable today. Many issues about its technique for activity and the advantages it gives to human wellbeing stay unanswered. This is valid for the predetermined dosages as well as for the conveyance course. Besides, there are numerous unanswered vulnerabilities about L-ascorbic acid's restorative adequacy in an assortment of human (irresistible) messes, as well as its safe framework movement and antiviral potential. L-ascorbic acid's capacity to work as both a cancer prevention agent and a prooxidant stresses its oxidation-decrease (redox) potential, in actuality, circumstances. The impact of intravenous L-ascorbic acid supplementation in patients with SARS-CoV-2 warrants explicit thought today. To more readily fathom the current new troubles related with L-ascorbic acid, this survey will feature well established realities in regards to L-ascorbic acid and its methods of activity.

Keywords: viral cells; clinical illnesses; cytokines; vitamin-c; SARS-CoV 2; therapeutic.

Introduction

Yet again with the presentation of another infection from the beta Covid family known as SARS-CoV-2, the antiviral capacities of L-ascorbic acid have been raised doubt about. Covids of the beta family (- CoVs) are single-abandoned RNA infections that are encased and positive-sense [1]. They basically taint bats, yet they can likewise contaminate different species like rodents and people. The Middle East Respiratory Syndrome Covid (MERS-CoV) and the Severe Acute Respiratory Syndrome Covids SARS-CoV-1 and SARS-CoV-2 have both delivered human flare-ups. Covid Disease 2019 is the aftereffect of a contamination brought about by the infection SARSCoV-2 (COVID-19). SARS-CoV-2 contaminates lung endothelial cells through restricting to the angiotensin-changing over compound 2 (ACE2) receptor by means of Spike Glycoprotein (S) (ECS). ACE2 receptors can be found in an assortment of tissues, including hematopoietic cells, kidneys, and digestive organs. The presence of SARS-CoV-2-tainted ECs in different organs of expired patients upholds this hypothesis. Brokenness, lysis, and passing are generally indications of tainted ECs. Besides, through delivering leukocyte attachment particles, ECs increment irritation in harmed tissues, actuating leukocyte development and extravasation. The actuation of leukocytes causes a cytokine storm. Enacted neutrophils and macrophages produce an overabundance of receptive oxygen and nitrogen species subsequent to being drawn to the lungs [2].

From one perspective, ROS and RNS direct safe reaction by actuating record factors that control the statement of fiery cytokines and chemokines through oxidant-incited initiation. ROS, then again, can act as oxidants, annihilating viral cells as well as lung (heart) cells, further causing EC brokenness and, at last, lung tissue obliteration. The deficiency of

microvascular boundary work in the lungs occurs in instances of broad endothelial cell injury, bringing about expanded vascular penetrability. Besides, SARS-CoV-2 restricting to the ACE2 receptor keeps ACE2 from corrupting angiotensin II, causing the angiotensin-vasopressor framework to breakdown. Expanded vascular penetrability is likewise a consequence of diminished ACE2 action. EC contractility is additionally supported by safe cells, incendiary cytokines, and vasoactive substances. Liquid leaks and fills alveolar sacks because of these occasions. At last, the cytokines IL-1 and TNF cause liquid maintenance in the lungs (aspiratory edema). High measures of cytokines, which are continually created as a response to viral contamination, intensify these manifestations - EC disappointment, irritation, vasodilation, and the arrangement of blood clusters. The COVID-19 can show in an assortment of ways, from asymptomatic to serious pneumonia. Sepsis happens when the body's reaction to contamination makes harm its tissues and organs. Pneumonia is the most pervasive irresistible reason for sepsis. Intense Respiratory Distress Syndrome (ARDS) and septic shock are brought about by alveolar brokenness and serious lung injury, which can advance to various organ disappointments and demise. Because of liquid development in the lungs, ARDS keeps required oxygen from entering the lungs, coming about in hypoxic respiratory disappointment. Therefore, ARDS and septic shock, as well as simultaneous clinical sicknesses like hypertension, cardiovascular and cerebrovascular issues, and diabetes, are the main sources of ICU confirmation and mortality in COVID-19 patients.

The deficiency of microvascular hindrance work in the lungs occurs in instances of broad endothelial cell injury, bringing about expanded vascular porousness. Besides, SARS-CoV-2 restricting to the ACE2 receptor keeps ACE2 from debasing angiotensin II, causing the angiotensin-vasopressor framework to glitch. Expanded vascular porousness is likewise a consequence of diminished ACE2 action. EC contractility is likewise supported by safe cells, incendiary cytokines, and vasoactive substances. Liquid leaks and fills alveolar sacks because of these occasions. At long last, the cytokines IL-1 and TNF cause liquid maintenance in the lungs (aspiratory edema). High measures of cytokines, which are continually created as a response to viral disease, enhance these manifestations - EC disappointment, irritation, vasodilation, and the arrangement of blood clumps [3]. The COVID-19 can show in an assortment of ways, from asymptomatic to extreme pneumonia. Sepsis happens when the body's reaction to contamination makes harm its tissues and organs. Pneumonia is the most predominant irresistible reason for sepsis. Intense respiratory trouble disorder (ARDS) and septic shock are brought about by alveolar brokenness and extreme lung injury, which can advance to numerous organ disappointments and passing. Because of liquid development in the lungs, ARDS keeps required oxygen from entering the lungs, coming about in hypoxic respiratory disappointment. Therefore, ARDS and septic shock, as well as simultaneous clinical sicknesses like hypertension, cardiovascular and cerebrovascular issues, and diabetes, are the main sources of ICU confirmation and mortality in COVID-19 patients [4-5]. A comparable remedial methodology, like HD-IVC in malignant growth treatment, could highlight L-ascorbic acid's favorable to oxidant action as a possible instrument of activity in SARS-CoV-2.

Conclusion

Vitamin C is the most widely used supplement. It was once used in the same way as aspirin. In good as well as terrible health. The COVID-19 epidemic persists today, regardless of supplementing technique, dosage, or duration. We could say it's been reborn as a Phoenix, but it's probably more fair to say it's always been a Phoenix. A redox Phoenix was used long before we knew what it was or how it operated in vivo.

REFERENCES

1. [King CG, Waugh WA. The isolation and identification of vitamin C. J Biol Chem. 1932;97\(1\):325–31.](#)
2. [Erol A. High-dose intravenous vitamin C treatment for COVID-19. 2003;56\(5\):114-51.](#)
3. [Hemilä H. A brief history of vitamin C and its deficiency, scurvy. 2012;14\(1\):1–15.](#)
4. [Szent-Györgyi A. Observations on the function of peroxidase systems and the chemistry of the adrenal cortex: description of a new carbohydrate derivative. Biochem J. 1928;22\(6\):1387–409.](#)
5. [Schraufstatter IU, Hinshaw DB, Hyslop PA, et al. Glutathione cycle activity and pyridine nucleotide levels in oxidant-induced injury of cells. J Clin Invest. 1985;76\(1\):1131–9.](#)