

Keywords: Vitamin D; Covid-19; Disease; Corona; SARS-CoV-2

Abstract:

Vit D is a steroid hormone made either by the skin's reaction to UV light or exogenously through food or diet/food we eat. Vit D deficiency is a global public health issue that affects a lot of individuals at all stages of life. A connection between vit D deficiency and a range of illnesses, including systemic infection, has been discovered in several investigations over the last decade. Vit D deficiency affects immunological activities because it has an immunomodulatory effect, enhancing innate immunity through the production of antiviral peptides, and so improving mucosal immunity. Clinical trials have compared decreased levels of blood vit D to acute RTI.

The coronavirus disease of year 2019 (the Disease) is a global pandemic that is causing wide spread destruction on our communities. Given the pandemic's massive health and economic effect, any approach for improving patients' conditions, speeding recovery, and lowering the risk of worsening and death would be considered important clinically and economically [1]. The Coronavirus pandemic is still a serious cause of concern in most countries. In the Disease individuals, the severe acute respiratory syndrome COVID virus (SARS-CoV-2) can cause ARDS like symptoms leading to severe disease and eventually, death. SARS-outbreak CoV-2's and rapid spread pose a global health danger with an uncertain outcome. Vit D has antiviral properties and can also act as an anti-inflammatory and Immuno modulator, according to new research. During infection, SARS-CoV-2 appears to rely heavily on the immune evasion route. In some individuals, this is preceded to hyper reaction and cytokine storm, which is a known pathologic process which leads to development of ARDS-like symptoms and complications. The COVID virus gains entry via alveolar and intestinal epithelial cells via using ACE-II (angiotensin-converting enzyme) as a host receptor. The renin-angiotensin (RAAS) system may then be dysregulated, resulting in excessive cytokine production.

Introduction:

To determine various roles of vit D in the Disease in different patients according to their age, needs, current and pre-existing medical conditions keeping in mind all the side effects and complications, in order to achieve better prognosis.

Introduction:

This Narrative Review compiles data statements and compares data collection methods in various formats through PubMed [2].

Abstract:

Vit D is an immune modulator hormone that has been shown to be useful in the treatment of a variety of upper respiratory infections. Vit D can aid in the reduction of hyper-inflammatory reactions and speeding up of the healing process in affected regions, especially lung tissue. As a result, there are both ecological and mechanistic grounds to pursue vit D activity in the disease patients [3]. Because there are currently no cures for the disease, we believe that vit D's ability to change the path of disease severity should be researched. Clinical trials may be conducted to determine the aid of vit D supplementation in people who are deficient of vit D as well as at high risk. Dr. Kuruvayeshwant, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Wardha, India; E-mail: kuruvayeshwant023@gmail.com

Received: 4-Jan-2022, Manuscript No: omha-22-52387, Editor assigned

gene structures in diminished by T allele, and it was consistently linked to increased sensitivity in our meta-analysis. Moreover, the prevalence viz-a-viz. Respiratory Syncytial virus infection correlates with the global distribution of the T allele, implying a link in immune response of vitamin D and viruses which are enveloped like RSV. Vit D regulates another intrinsic antibacterial factor, defensin2, in addition to cathelicidin. Defensin 2, also known as cathelicidin, enhances host defense by boosting involvement of antiviral cytokines and chemokine's in monocyte, natural killer cell, neutrophil, and T cell recruitment.

Two 25-OHD and 1,25 (OH) 2D increase killer marker LC3 expression, which is an integral physiological interaction to vitamin D. Vit D boosts cellular clearance in the congenital part of the immune system via interactions similar to the ones triggered by antimicrobial proteins. Cathelicidin antimicrobial function is critical, but it also has a variety of other functions, such as inducing a variety of proinflammatory cytokines, stimulating neutrophil, monocyte, and T cell chemotaxis into the infection site, and inducing apoptosis and autophagy in infected epithelial cells to aid in the clearance of respiratory infections [4]. The vit D receptor (VDR) which converts 25 (OH) cholecalciferol to 1, 25 (OH) 2 cholecalciferol and is present on numerous immune cells. CYP27B1, a member of the cytochrome p450 family, may produce 1, 25 (OH) 2VitD in a range of cell types other than renal cells with the aid of TLR. By giving feedback to vit D signaling and inhibiting vit D's role pertaining to a factor regulating transcription in the application of suppression of the renin regulation genetic mechanism, RAAS can serve as an endocrine regulator that acts inversely with respect to RAAS. In VDR up regulation and overexpression in the lungs and its dynamics with cholecalciferol can prevent lung injury by suppressing the RAAS. Vit D may also impact the functional status of T lymphocytes by increasing memory and regulatory T cells (Treg), decreasing neutrophil/lymphocyte ratio, and increasing memory and regulatory T cells (Treg). Because lymphopenia and T lymphocyte dysfunction are linked to the severity of the disease, 87-89, 104 vit D may help to enhance the CD8+ T cell response and therefore better control the disease.

Vitamin D deficiency given with value of 25-OHD-cholecalciferol (30 ng/mL) was discovered in three-quarters of the total patients and 85 percent of which needed Intensive care in a survey of hospitalized the disease patients. Furthermore, according to survey vit D status in Europe, a study of the disease severity found that nations with the highest prevalence of abnormally high vit D levels are related to the high risk of illness and mortality. Furthermore, preliminary research data from the US found a substantial relationship intertwining vit D insufficiency and fatal outcomes, as well as various elements of a not so great result [5]. The ratio of having a normal result other than major result extended 7.94 X with an increase in the original variation of 25 (OH) D, whereas the chances of getting a small outcome instead of a critical result increased 19.61 times. Hence severity of disease and levels of vitamin D are related, patient death, CRP levels, and the increase in lymphocyte percentage. Vit D deficiency has also been related to a less risk of hypoxia and unconsciousness. Decrease in vitamin D affects 40% of Europeans, regardless of their age, sex, skin type, or geographic location. Similarly, European nations with severe vit D deficiency (France 27.3%, Portugal 21.2%, and Austria 19.3%) showed substantial cases of the disease. Vit D may regulate cytokine storm in the disease by regulating IL-6 levels.

Activity of cholecalciferol was enhanced by increasing the regulation of the gene called as VDR. In the bronchial epithelium and

the monocytes cells, Dexamethasone along with that reduces the levels of calcitriol-induced-cathelicidin. VDD may have a higher influence in younger patients, as evidenced by a substantial interaction with age. The occurrence of pneumonia/acute respiratory distress syndrome (ARDS), myocarditis, microvascular thrombosis, and/or cytokine storm, all of which entail underlying inflammation, often determines the severity of the illness in COVID-19 individuals. T regulatory lymphocytes are an important line of defense against excessive inflammation and viral infection in general (Tregs). In one set of COVID-19 patients, Treg levels were found to be low, and 'markedly lower in severe instances.' High Treg blood levels were shown to be associated with a lower level of respiratory viral illness in a study of elderly nursing home patients. These findings imply that increasing Treg levels could help to reduce the severity of viral illness, including COVID-19 [6]. Vitamin supplementation issue has been debated on widely with strong opinions for and against the argument. This pandemic increased the reason for its debate. It has been seen that the group of people who have always been in a deficit of this vitamin were at high risk of getting the disease and reacting to a severe stage. Some concerns were also raised regarding the fact that people had to stay indoors hence were deprived of vitamin from sunlight.

Surprisingly, numerous risk factors for vit D deficiency (defined as a 25-hydroxyvit D (25(OH) D) level less than 30 nmol/L) are also risk factors for the disease infection and poor outcomes. Being older, fat, male, and having pre-existing chronic illnesses, for example, are all risk factors for deficiency, making patients more vulnerable to the disease. The disease mortality is highest among individuals over 80 years old, e.g. >20% in Italy, and this is often the age group with the greater levels of insufficiency regardless of nation. People who live at higher latitudes or have darker skin colours (Black Asian Minority ethnics-BAME in the UK) are more likely to be affected by the disease, according to recent findings. BAME people are also more likely to have obesity, pre-existing chronic disease (such as heart disease or diabetes), and vit D deficiency. Importantly, a global link between northern latitude and increased the disease mortality has already been shown. While there might be a lot of causes for this, it does support the theory that sunlight exposure and hence vit D level may influence the severity of the disease. We think that vit D insufficiency has a role in the severity of the disease reactions, and that vit D deficiency will be strongly connected to the disease mortality in Europe. Inflammation caused by disease, on the other hand, may impair 25OHD metabolism, particularly that of its binding protein, resulting in decreased circulating levels and evaluation bias. In the disease, OHD supplementation was not linked to hospitalization, but it did appear to be a risk factor for increased in-hospital mortality. Correlating Cholecalciferol levels with ACE density in severely ill patients. The group of COVID subjects exhibited a remarkable change in cholecalciferol levels [7]. When compared to the control group, the serum ACE levels in the patients group increased significantly. Even when compared to other patients, the ACE concentration was substantially greater in deceased persons.

SARS-CoV-2 has been demonstrated to block the activation of the interferon system by a variety of mechanisms, including PRR recognition, signalling pathway inhibition, degradation of mRNA of the host, and degradation of protein. This antagonistic relationship results in abnormal inflammatory responses, which in turn leads to viral multiplication. Protease and reactive oxygen species production caused

by inflammatory cells in inflammation which induces significant damage of cells and tissue of lung, resulting in severe pathological alterations in the lungs. Shock due to endotoxins and failure of multiple organs result from an increase in cytokine levels, especially TNF-alpha.

therapy which was a study conducted at the Oxford School in the United Kingdom. The mortality rate in ventilated patients was reduced by 35%, while the rate in supplementary oxygen therapy patients was

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Vitamin D is a sec steroid hormone that was first found during the English industrial revolution to treat rickets. Since then, vitamin D has been credited with a variety of functions, including skeletal and extra skeletal. Vitamin D comes in two forms: vitamin D2 (ergocalciferol), which is mostly produced from plants, and vitamin D3 (cholecalciferol), which is found in higher animals and accounts for about 80–90% of the total. After being exposed to ultraviolet wavelength B, 7-dehydrocholesterol is transformed to cholecalciferol. Both endogenous and exogenous versions are inert, and fully active vitamin D needs dual hydroxylation stages by cytochrome enzymatic action. Vitamin D binding protein transports circulating vitamin D to the liver. The first hydroxylation occurs in the liver by cytochrome P 450 vitamin D hydroxylases at the 25-carbon position, resulting in the synthesis of calcitriol. It is employed as a general indicator of cholecalciferol levels in humans due to its increased shelf life. Calcidiol flushes the liver and is internalised into the renal system, where renal 1-hydroxylase causes it to undergo C-1 hydroxylation, which is tightly controlled by the PTH or the Para hormone. Hydroxylation in the kidneys converts vitamin D to calcitriol, which is fully functional and active.

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The main disadvantage of dexamethasone therapy is that it promotes or accelerates viral multiplication and progression, potentially worsening the disease or delaying recovery. The significance of dexamethasone in the therapeutic context of critically ill COVID subjects was recently underlined in a Randomized study of COVID-19