

## Prevalence of Vitamin-D deficiency in Ulcerative Colitis

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### ABSTRACT:

**INTRODUCTION:** Ulcerative colitis is a chronic inflammatory disease of the colon of unclear etiology with an increasing incidence in India. Immunomodulatory effects of vitamin-D has been linked to autoimmune disease such as ulcerative colitis and deficiency of vitamin-D has been found to correlate with severity and activity of disease. In our study we evaluated the prevalence of vitamin-D deficiency in mild, moderate, and severe ulcerative colitis.

**AIM:** To Study the prevalence of vitamin-D level and deficiency in Ulcerative colitis patient at our centre.

**METHODS:** Retrospective data of 70 Patients with Ulcerative colitis diagnosed by clinical, endoscopic and histopathologic evaluation from 2015-2017 at our centre were included in the study and they are classified into mild, moderate, severe by Truelove and Witts criteria. Serum vitamin-D levels were measured in all patients and 70 healthy controls. Patients with mean age of 22-55 years and no comorbid illness such as Diabetes Mellitus, Systemic Hypertension, Chronic kidney disease were included in the study. Vitamin-D levels were defined as Sufficient (>30ng/mL), Insufficient (20-30ng/mL), Deficient (<20ng/mL).

**RESULTS:** 70 patients (male=43, female=27) with mean age of 40 years and average symptom duration of 6 months prior to our hospital visit were reassessed during follow up at our hospital. They were classified into mild UC group n=23(32.8%), moderate UC group n=31(44.28%) and severe UC group n=16(22.85%). Among study group 18(78.26%) patients in mild UC 26(83.87%) patients in moderate UC and 14(87.5%) patients in severe UC group were vitamin-D deficient. Among 70 healthy controls serum vitamin-D levels were deficient in 18(25.71%) patients and insufficient in 22(31.42%) patients.

**CONCLUSIONS:** We conclude that immunomodulatory property of serum vitamin-D might be a contributory factor to disease activity of ulcerative colitis, however other factors such as infections, poor compliance to treatment and steroid resistance should also be considered.

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### I. Introduction:

Vitamin D is very important in calcium homeostasis, controlling the formation and resorption of bone. Vitamin D is acknowledged as an immune system regulator. T-Cell mediated immunity is under modulatory control of 1,25-dihydroxycholecalciferol, the active form of vitamin D. The absence of vitamin D was shown to blunt the T-cell mediated immune responses. Most body stores come from vitamin D<sub>3</sub> which is synthesised by the direct action of sunlight on the skin. A small proportion of total vitamin D is obtained from the diet. The pathogenesis of IBD involves a complex interplay between genetic, environmental and immunological factors. It has been determined that in both forms of IBD, the tissue damage results from an inappropriate or exaggerated immune response to luminal antigens of gut microflora. T-cells overproduce inflammatory mediators like interleukin-17 (IL-17), tumour necrosis factor  $\alpha$  (TNF  $\alpha$ ) and interferon gamma (IFN- $\gamma$ ). The tissue damage occurs in areas that are heavily infiltrated with activated CD4+ lymphocytes. A recent hypothesis linking vitamin D deficiency to development of immune-related disorders has been proposed. This argues that decreased outdoor activity and vitamin D poor diets have led to fluctuations in vitamin D status in developed countries, particularly those that experience long winter seasons. For people living in northern climates, vitamin D deficiency is relatively common. In particular, patients with IBD are likely to have a high incidence of hypovitaminosis D. This could be due to a number of different mechanisms, including decreased intestinal absorption, increased intestinal loss through a protein losing enteropathy, and decreased exposure to sunlight. This has led some to believe that vitamin D deficiency may play a role in the pathogenesis of IBD. The aim of this study was to know the prevalence of vitamin D for treating inflammatory bowel disease.

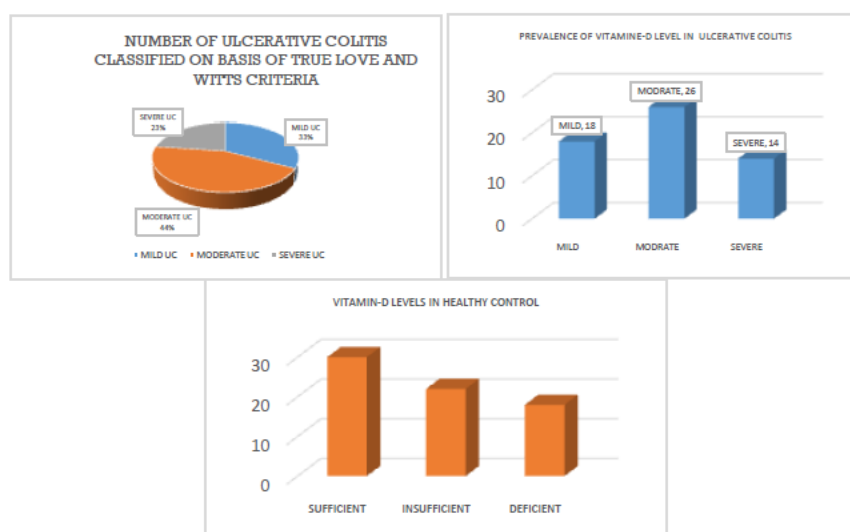
### II. Materials and methods:

Retrospective data of 70 Patients with Ulcerative colitis diagnosed by clinical, endoscopic and histopathologic evaluation from 2015-2017 at our Centre were included in the study and they are classified into mild, moderate, severe by Truelove and Witts criteria. Serum vitamin-D levels were measured in all patients and 70 healthy controls. Patients with mean age of 22-55 years and with no comorbid illness such as Diabetes Mellitus, Systemic Hypertension, Chronic kidney disease were included in the study. Serum vitamin D levels are measured in all patients and Vitamin-D levels were defined as Sufficient (>30ng/mL), Insufficient (20-30ng/mL), Deficient (<20ng/mL).

### III. Results:

70 patients (male=43, female=27) with mean age of 40 years and average symptom duration of 6 months prior to our hospital visit were reassessed during follow up at our hospital. They were classified into mild UC group n=23(32.8%), moderate UC group n=31(44.28%) and severe UC group n=16(22.85%). Among study group 18(78.26%) patients in mild UC

26(83.87%)patients in moderate UC and 14(87.5% )patients in severe UC group were vitamin-D deficient. Among 70 healthy controls serum vitamin-D levels were deficient in 18(25.71%) patients and insufficient in 22(31.42%) patients.



#### IV. Discussion

The prevalence of vitamin D deficiency in adults with ulcerative colitis has been reported to be as high as about 45–50% and has been attributed to various factors including decreased sunlight exposure, low oral vitamin D intake, disturbed entero hepatic circulation and increased loss of vitamin D as the result of protein-losing enteropathy. However, it is unclear if vitamin D deficiency is an environmental trigger for autoimmunity in IBD or if IBD directly causes vitamin D deficiency. Vitamin D deficiency in IBD patients has also been shown to be an independent risk factor for higher disease activity scores and increased frequency of corticosteroid use. Vitamin D affects both the innate and adaptive immune systems and leads to immune-tolerance of self-structures. Vitamin D through its receptors influences the maturation and differentiation of antigen presenting cells, dendritic cells and macrophages, resulting in the decreased activation of T cells and suppression of inflammatory cytokines and may thereby reduce disease activity in IBD. Despite the evidence suggesting an association between vitamin D and IBD, there have been no prospective human studies looking at the effects of vitamin D3 in patients with ulcerative colitis. The concordance estimates of UC between twins is 20% or less, suggesting non-genetic factors also play a significant role in the pathogenesis of UC. Some environmental factors that have been associated with IBD include cigarette smoking, oral contraceptive use, vitamin D levels, dietary factors, stress, non-steroidal anti-inflammatory drugs, physical activity and duration of sleep. Vitamin-D is increasingly being identified as an important environmental factor influencing many chronic diseases including cancers, cardiovascular disease, and autoimmune diseases such as multiple sclerosis and IBD.

In our study group 18(25.71%)patients in mild UC 26(83.87%)patients in moderate UC and 14(87.5% )patients in severe UC group were vitamin-D deficient, when compared to healthy controls 18(25.71%). This prevalence shows vitamin-D might be contributory to disease activity and also be risk factor for development of disease, however more studies are required for the pathogenesis, disease activity and genetic association to establish strong relationship between Vitamin-D and IBD.

#### V. Conclusions

We recommend Vitamin -D level estimation for all ulcerative colitis patients and should be planned for supplementation though evidence is lacking and need further studies to suggest role of vitamin-D in activity of disease.

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