

# To Assess the Serum Vitamin D in Vitiligo Patients: SMHRC and AVBR Hospital of Vidarbha Region

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## Abstract

**Introduction:** Vitiligo is a long-term skin condition that is characterized by skin patches which lose their pigment. The affected skin patches are becoming white and typically have narrow margins. The skin hair can also turn white. Mouth and nose inside might also be involved. Occasionally, the vitiligo “comes in families,” implying a genetic origin. It ensures that the risk of other family members contracting vitiligo is not predictable. Approximately 25 to 50 per cent of people with vitiligo have a parent with vitiligo, and around 6 per cent have vitiligo siblings. Low serum levels of vitamin D have been associated with many autoimmune disorders and a number of other skin conditions. Vitiligo is an autoimmune disease which is characterized by immune processes that kill melanocytes. Melanocytes express vitamin D receptors, and vitamin D status can affect their function.

**Objective:** To assess serum Vit-D levels of vitiligo patients and contrast the outcomes and controls.

**Material and Method:** In all out, 50 vitiligo patients and 50 controls were taken on the examination. Vit-D levels were estimated from blood tests. Gathering correlations were performed utilizing fitting factual strategies.

**Observation and Results:** The patients had lower serum Vit-D levels than the controls, yet this distinction was not noteworthy ( $p = 0.570$ ).

**Conclusion:** It stays obscure whether Vit-D inadequacy causes vitiligo. Bigger controlled investigations are required to demonstrate whether low flowing VIT-D is a causative factor in vitiligo.

**Keywords:** Vitiligo, Vit-D, immune system illnesses, Etiopathogenesis.

## Introduction

Vitiligo affects approximately 2 percent of the world's population regardless of skin type, age and sex<sup>[1]</sup>. The malady may influence the two sexual orientations and all skin types<sup>[2]</sup> and may likewise be related with fundamental immune system sicknesses, for example, lupus erythematosus, scleroderma, immune system thyroiditis and alopecia areata<sup>[3]</sup>. Diminished serum Vit-D levels are found in numerous immune system illnesses including foundational lupus erythematosus, diabetes mellitus, rheumatoid joint inflammation,

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different sclerosis and alopecia areata<sup>[4]</sup>. Genetic factors play an important pathogenic factor. Multiple studies have included multiple genes in the pathogenesis of vitiligo, including studies on Jordanian population<sup>[5, 6]</sup>.

Vitiligo has a significant psychological and social effect on individuals especially affected by women, or on exposed sites and more so in people of colour (POC)<sup>[7]</sup>. Vitamin D3 is a vitamin essential to humans. The majority of its active form is obtained by activating the pre-vitamin D3 formed in the skin following exposure to sunlight, in particular UVB (290–320 nm). Diet is only a minor vitamin source. This vitamin plays an important role in immunity (innate and adaptive), calcium control, and melanin synthesis; in addition, several diseases have been associated with decreased vitamin D levels. Melanocytes express vitamin D receptors which may indicate a possible role for vitamin D in melanocyte function regulation.<sup>[8]</sup>

Vit-D is a basic hormone that is orchestrated in the skin<sup>[9]</sup>. The dynamic type of Vit-D, 1, 25-dihydroxyvitamin D3, is a hormone that manages calcium and bone digestion, controls cell multiplication and separation and furthermore displays certain immunoregulatory capacities<sup>[10]</sup>. Vit-D may influence both natural and versatile resistant reactions through receptors in T and B lymphocytes, macrophages and dendritic cells<sup>[11]</sup>. What's more, Vit-D3 increments tyrosinase action and melanogenesis by means of an atomic hormone receptor – the Vit-D receptor (VDR) in melanocytes<sup>[12]</sup>. Vit-D and its analogy are utilized to treat skin issue, including psoriasis and vitiligo<sup>[13]</sup>. Patients with vitiligo have been treated with topical calcipotriene<sup>[13]</sup>. Hardly any reports have examined the relationship among vitiligo and decreased Vit-D levels, yet these examinations give clashing outcomes<sup>[14, 15]</sup>. Point: This examination expected to decide if patients with vitiligo have lower serum Vit-D levels contrasted with controls.

### Material and Method

In all out, 50 patients determined to have vitiligo, in our outpatient division from 1 December 2013 to 31 March 2014, were taken a crack at the examination; 47 age-, sex and skin phototype-coordinated sound controls additionally took part. The patients were analyzed by a similar dermatologist and the analysis of vitiligo was made by clinical discoveries and a Wood's light assessment. Punch biopsy was performed on dubious

cases, with the conclusion at that point checked. Point by point sickness and family ancestries were gotten from all patients. Different segment and way of life factors were recorded, including age, sexual orientation, skin model and sunscreen utilization. Members with liver or kidney issue, hyperparathyroidism, hypo-parathyroidism, any metabolic bone issue (for example osteoporosis or osteopenia) or fiery ailments were rejected from the investigation, similar to those taking Vit-D-or calcium-including drugs, or any fundamental or topical treatment for vitiligo inside the earlier month. Controls were selected from the accomplices or family members of patients, if not influenced by vitiligo, to limit contrasts because of dietary admission of Vit-D. Educated assent was acquired from all members and the neighbourhood Ethics Committee affirmed the examination, which was led as per the fundamentals of the Declaration of Helsinki. Blood tests were taken in the first part of the day after a base fasting time of 8 hour. Serum free T3, free T4, thyroid animating hormone, fasting glucose, against thyroid peroxidase antibodies, hostile to thyroglobulin antibodies, Vit-B12 and Vit-D levels were estimated.

### Observation and Results

In complete, 26 (52%) guys and 24(48%) females were remembered for the investigation gathering. The benchmark group comprised of 30 (60%) guys and 20 (40%) females. The mean periods of the patient and control bunches were  $31.96 \pm 10.68$  and  $32.45 \pm 9.52$  years, separately. There was no huge distinction between the patient and controls as far as age ( $p = 0.809$ ). All patients had summed up vitiligo as controlled by the nearness of reciprocal evenly circulated depigmented macules in trademark areas. The mean age at vitiligo macules beginning was  $17.48 \pm 9.81$  years. The term of the injuries extended up to 25 years. A family ancestry of vitiligo was accounted for in one patient. No patients had diabetes mellitus or Vit-B12 lack. Immune system thyroid ailments were accounted for in 14 (28%) patients. Vit-D levels were tried throughout months, from December 2019 to May 2020. The patients' serum Vit-D levels ran from 6 to 42 ng/ml (mean:  $12.54 \pm 9.29$  ng/ml); in the benchmark group they ran from 8 to 39 ng/ml (mean:  $13.07 \pm 5.98$  ng/ml). The patients had lower coursing Vit-D levels than controls, however this distinction was not critical ( $p = 0.7352$ ).

### Discussion

In the current investigation, we discovered lower

serum Vit-D levels in patients with vitiligo comparative with controls; be that as it may, this distinction was not huge. Both the patients and controls had exceptionally low circulating Vit-D levels. This might be on the grounds that the blood tests were gathered during winter months. There are not very many examinations assessing serum Vit-D levels in vitiligo patients [14, 15]. Ustun et al. examined 25 vitiligo patients and 41 controls: deficient (< 30 ng/ml) or low (< 15 ng/ml) levels of Vit-D were seen in most of patients, yet the distinctions were not huge contrasted with controls [14]. These examiners expressed that countless examinations had announced low degrees of flowing Vit-D in immune system sicknesses; however it stays muddled whether this is a reason or consequence of immune system maladies [14]. Another examination researched 40 vitiligo patients and 40 age-and sexual orientation coordinated controls. Altogether lower serum Vit-D levels were found in the patient comparative with controls. The creators estimated on the chance of Vit-D supplementation for the treatment of vitiligo patients later on [15]. The pathogenesis of vitiligo remains to a great extent obscure. There are various hypotheses clarifying the pathogenesis of vitiligo, with all hereditary, immune system, autotoxic and neurogenic causes hypothesized. The immune system hypothesis is the best-bolstered one, since vitiligo might be related with other immune system sicknesses including malignant sickliness, hyperthyroidism, Hashimoto's thyroiditis, alopecia areata and adrenocortical disappointment. Besides, histological investigations have exhibited a high recurrence of cytotoxic T lymphocytes explicit to melanocytic antigens in vitiligo injuries, proposing an immediate, melanocyte-explicit T cell assault [16, 17]. Vit-D, which is a fat-dissolvable vitamin acquired by people through eating routine, is specifically compelling to dermatologists since it is combined in the skin by bright light. It has been utilized to treat psoriasis, vitiligo and other skin ailments for a long time [17]. The dynamic type of Vit-D, 1, 25-dihydroxyvitamin D<sub>3</sub>, directs calcium and bone digestion, yet in addition controls cell multiplication and separation and applies immunoregulatory exercises [13]. In a past report, it was accounted for those patients with comorbid immune system diseases are bound to have low serum Vit-D levels [16]. Vit-D has a nuclear receptor called Vit-D receptor (VDR). Vit-D receptors are available in the cells engaged with calcium and bone digestion, and furthermore in keratinocytes, melanocytes, fibroblasts and resistant framework cells of the skin. Polymorphisms in VDR are corresponded with expanded helplessness

to numerous sclerosis, provocative entrapment malady, rheumatoid joint pain and type 1 diabetes mellitus [19]. Vit-D applies a noteworthy impact on melanocytes and keratinocytes by means of different instruments. In vitro investigations have demonstrated that Vit-D<sub>3</sub> is related with an expansion in tyrosinase action and melanogenesis [11], which may add to re-pigmentation in vitiligo macules. Vit-D analogs, including calcipotriol and tacalcitol, are known to improve re-pigmentation in vitiligo patients [20-22]. Another investigation announced that Vit-D applies immune-modulatory impacts by hindering the declaration of interleukin (IL)- 6, IL-8, tumor necrosis factor (TNF)-  $\alpha$ , and TNF- $\gamma$  [23]. Besides, it has likewise been indicated that the dynamic type of Vit-D lessens the apoptotic action prompted by UVB in melanocytes [13].

## Conclusion

More investigation is required to outline the connection between VITAMIN D and vitiligo, to assess whether a low degree of serum Vit-D is a causative factor in vitiligo, and to learn whether Vit-D supplements are helpful for both the anticipation and treatment of vitiligo. Irreconcilable circumstance the creators proclaim no irreconcilable situation.

**Ethical Clearance:** Taken from institutional ethics committee.

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**Conflict of Interest:** Nil.

## References

1. Diala M et al. Assessment of Serum Vitamin D Levels in Patients with Vitiligo in Jordan: A Case-Control Study. *Dermatology Research and Practice*. Volume 2019, Article ID 2048409, 4 pages.
2. Nordlund J. The epidemiology and genetics of vitiligo. *Clin Dermatol* 1997; 15: 875-78.
3. Lee H et al. Prevalence of vitiligo and associated comorbidities in Korea. *Yonsei Med J* 2015; 56: 719-25.
4. Kamen D, Aranow C. The link between vitamin D deficiency and systemic lupus erythematosus. *Curr Rheumatol Rep* 2008; 10: 273-80.
5. Alkhateeb A et al. SMOC2 gene variant and the risk of vitiligo in Jordanian Arabs, *European Journal of Dermatology*, 2010; 20(6):701-704.

6. Alkhateeb A et al. Genetic association of NALP1 with generalized vitiligo in Jordanian Arabs. *Archives of Dermatological Research*. 2010;302(8):631–634.
7. Amer A et al. Quality of life in patients with vitiligo: an analysis of the dermatology life quality index outcome over the past two decades, *International Journal of Dermatology*. 2016;55(6):608–614.
8. AlGhamdi K et al. The role of vitamin D in melanogenesis with an emphasis on vitiligo, *Indian Journal of Dermatology, Venereology, and Leprology*, 2013;79(6): 750–758.
9. Lips P. Vitamin D physiology. *Prog Biophys Mol Biol* 2006; 92: 4-8.
10. Karagun E et al. The role of serum vitamin D levels in vitiligo. *PostepyDermatologii I Alergologii*. 2016;33(4):300-302.
11. Adorini L, Penna G. Control of autoimmune diseases by the vitamin D endocrine system. *Nat Clin Pract Rheumatol* 2008; 4: 404-12.
12. Oikawa A, Nakayasu M. Stimulation of melanogenesis in cultured melanoma cells by calciferols. *FEBS Lett* 1974; 42: 32-5.
13. AlGhamdi K, Kumar A, Moussa N. The role of vitamin D in melanogenesis with an emphasis on vitiligo. *Indian J Dermatol Venereol Leprol* 2013; 79: 750-8.
14. Ustun I, Seraslan G, Gokce C, et al. Investigation of vitamin D levels in patients with vitiligo vulgaris. *ActaDermatovenerol Croat* 2014; 22: 110-3.
15. Saleh H et al. Evaluation of serum 25-hydroxyvitamin D levels in vitiligo patients with and without autoimmune diseases. *Photodermatol Photoimmunol Photomed* 2013; 29: 34-40.
16. Czajkowski R, Męcińska-Jundziłł K. Current aspects of vitiligo genetics. *Postep Dermatol Alergol* 2014; 31: 247-55.
17. Laddha N et al. Role of oxidative stress and autoimmunity in onset and progression of vitiligo. *Exp Dermatol* 2014; 23: 352-3.
18. Silverberg J et al. A pilot study assessing the role of 25 hydroxy vitamin D levels in patients with vitiligo vulgaris. *J Am Acad Dermatol* 2010; 62: 937-41.
19. Ersoy-Evans S. Commentary: vitamin D and autoimmunity: is there an association? *J Am Acad Dermatol* 2010; 62: 942-4.
20. Birlea S et al. New insights on therapy with vitamin D analogs targeting the intracellular pathways that control repigmentation in human vitiligo. *Med Res Rev* 2009; 29: 514-46.
21. Parsad D, Saini R, Verma N. Combination of PUVA-sol and topical calcipotriol in vitiligo. *Dermatology* 1998; 197: 167-70.
22. Oh SH et al. Combination treatment of non-segmental vitiligo with a 308-nm xenon chloride excimer laser and topical high-concentration tacalcitol: a prospective, single-blinded, paired, comparative study. *J Am Acad Dermatol* 2011; 65: 428-30.
23. Koizumi H, Kaplan A, Shimizu T, Ohkawara A. 1, 25-dihydroxyvitamin D3 and a new analogue, 22-oxacalcitriol, modulate proliferation and interleukin-8 secretion of normal human keratinocytes. *J Dermatol Sci* 1997; 15: 207-13.