IJFANS INTERNATIONAL JOURNAL OF FOOD AND NUTRITIONAL SCIENCES
ISSN PRINT 2319 1775 Online 2320 7876
 Journal Volume 14, Iss 02, 2025

SERUM VITAMIN D3 LEVELS IN A CROSS SECTION OF POPULATION ATTENDING OPD AT TERTIARY CARE HOSPITAL IN DEHRADUN, UTTARAKHAND.

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Abstract

Vitamin D deficiency is not very common since it can be synthesised in the body in adequate amounts by simple exposure to sunlight even for 10 minutes every day. It may be seen in people who are bedridden for long periods, strict vegetarians, chronic alcoholics and person suffering from severe liver and kidney diseases or fat Malabsorption syndromes. Vitamin D is also involved in some function in addition to calcium homeostasis and bone metabolism. Receptors for calcitriol occur in many other tissues such as the parathyroid gland, islet of pancreas, myeloid stem cells in bone marrow and keratinocytes of skin. Vitamin D seems to be involved in cell differentiation, both in normal and malignant tissues. It also stimulates the production of cytokines thus playing a role in immunomodulation. Adequate intake of vitamin D is beneficial in reducing the risk of certain autoimmune disease. In our study total 830 patients participated, there were 340 males and 490 females. Among these patients 23.73% were having adequate level, 19.27% having insufficient vitamin D levels, 53.97% having severe deficiency of vitamin D and 3.01% are having toxicity to Vitamin D. Present study concludes that Vitamin D levels was deficient in Dehradun, Uttarakhand region of India.

Keywords- Vitamin D, Calcidiol, Sun Exposure, Vitamin D binding protein.

Introduction

Vitamin D is a fat-soluble vitamin, known for its antirachitic activity.[1] Calciferols are a group of lipid-soluble compounds with a 4-ringed cholesterol backbone and refer to both, Vitamin D3, i.e., cholecalciferol and Vitamin D2, i.e., ergocalciferol.[2] Few foods naturally contain vitamin D (oily fish, such as sardines, herring, tuna, mackerel, salmon, and cod liver oil, egg yolks, shiitake mushrooms, liver or organ meats), so dermal synthesis after ultraviolet

B (UVB) radiation remains the major route to obtain vitamin D, accounting for 90% of vitamin D replenishment.[3]

Vitamin D, in general, refers to Vitamin D3. Vitamin D can be synthesized endogenously. Cholecalciferol (vitamin D3) is from animal sources and ergocalciferol (vitamin D2) is from plants. [4] About 90% of the required Vitamin D is synthesized in the skin under sun exposure. [5] UV-B photons act on pro– vitamin D3, a precursor in the cholesterol biosynthetic pathway, in the plasma membrane of epidermal cells to form pre–vitamin D3. Pre–vitamin D3 is rapidly transformed into vitamin D3 and transferred to the extracellular space where it binds to vitamin D–binding protein. [6] From there it is transported to the liver, where it is hydroxylated into 25(OH)D. [7] Cholesterol-like precursor (7-dehydrocholesterol) in skin epidermal cells can be converted after UVB radiation into pre-vitamin D, which also isomerizes to vitamin D3. Both vitamin D3 and D2 are biologically inactive. They need further enzymatic conversion to its active forms. First, it undergoes 25-hydroxylation in liver to 25(OH)D (calcidiol), the major circulating form of vitamin D, with a half-life of 2 to 3 weeks. Then it is converted in kidneys through 1-alpha-hydroxylation to its most active form, 1,25(OH)2D (calcitriol), with a half-life of 4 to 6 h. This process is driven by parathyroid hormone (PTH) and other mediators, including hypophosphatemia and growth hormone. [8,9] Vitamin D is classically known to regulate calcium and phosphate metabolism. It is needed for the maintenance of normal blood levels of calcium and phosphate that are required for normal mineralization of bone, muscle contraction, nerve conduction, and general cellular function in all cells of the body. It not only plays an essential role in maintaining healthy mineralized skeleton, but also is an immunomodulatory hormone, for inflammation, cell proliferation, and differentiation. [5,6,10,11] The active form of Vitamin D stimulates the absorption of calcium in the duodenum and increases calcium influx in distal tubules of kidney through nuclear Vitamin D receptor (VDR); latter is specifically regulated by parathormone level. [12]

At the time of hypocalcaemia, the plasma level of ionized calcium falls and this is detected by parathyroid gland calcium receptors. PTH is secreted by parathyroid gland, which stimulates 1-alpha-hydroxylation in kidneys to make more 1,25(OH)2D from circulating 25(OH)D. The elevation of 1,25(OH)2D increases calcium transport within intestines, bones, and kidneys, and further regulates the osteoblast and osteoclast activity. As plasma calcium rises back to normal, further secretion of PTH decreases. [8,9] It functions through a vitamin D receptor (VDR) that is universally expressed in nucleated cells. Both the vitamin D receptor (VDR) and metabolizing enzymes are expressed by various types of immune cells including lymphocytes, monocytes, macrophages, and dendritic cells [13,14]. Its most important biological role is promoting enterocyte differentiation and intestinal calcium absorption, facilitating calcium homeostasis. This physiologic loop of vitamin D and calcium homeostasis demonstrates that sufficient circulating 25(OH)D is essential to maintain adequate 1,25(OH)2D synthesis and plasma calcium level.[5] Dietary vitamin D is absorbed in the small intestine, incorporated into chylomicrons, and then transported to the liver bound to vitamin D–binding protein.[7] From the liver, 25(OH)D travels to the kidney bound to vitamin D–binding protein.[7,14] The kidney further hydroxylates 25(OH)D to 1,25-dihydroxyvitamin D (1,25[OH]2D), the most active form.[15] Once in the active form,

1,25(OH)2D (calcitriol) travels to the rest of the body in any cells with vitamin D receptors.[7]

Material and method

This cross-sectional study was conducted in the department of Hospital Laboratory (pathology), Military Hospital Dehradun, Uttarakhand from a period of 1-Jan-2021 to 1 Jan 2022. In this study all the 830 subjects who came for the estimation of 25 (OH)D level of both the sexes have been included. Blood samples were collected from all 830 subjects under aseptic precaution into plain vacutainers and labelled properly. Then serum 25 (OH)D level estimation was done by VIDAS® 25 OH Vitamin D Total is an automated quantitative test for the determination of 25-hydroxyvitamin D Total by using the ELFA (Enzyme Linked Fluorescent Assay) technique. Reference range for serum 25(OH) D level is 30-100ng/ml. serum vitamin D levels <20ng/ml as severe deficiency, levels <30ng/ml is insufficient and levels >100ng/ml as potential toxicity.

Inclusion criteria

- Age >18 years
- People who consented to participate in the study

Result

This cross-sectional study was carried out for period of 1 year, and a total of 830 patients were selected for the study and serum vitamin D levels were estimated. Out of total 830 patients, there were 340 males and 490 females. Among these patients 23.73% were having adequate level, 19.27% having insufficient vitamin D levels, 53.97% having severe deficiency of vitamin D and 3.01% are having toxicity to Vitamin D.

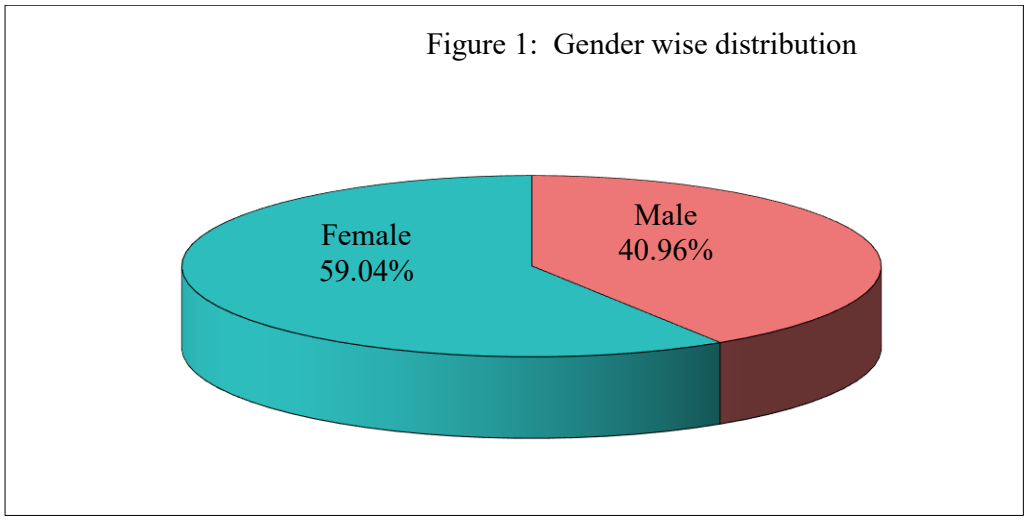

Figure 1- Distribution of patients by gender

ISSN PRINT 2319 1775 Online 2320 7876
 Journal Volume 14, Iss 02, 2025

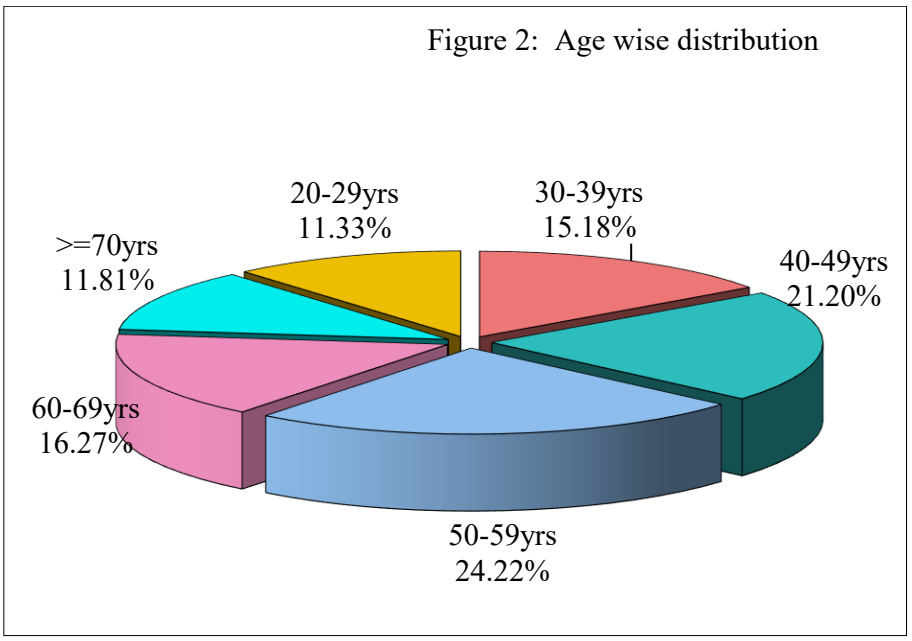

Figure 2- Distribution of patients by age group

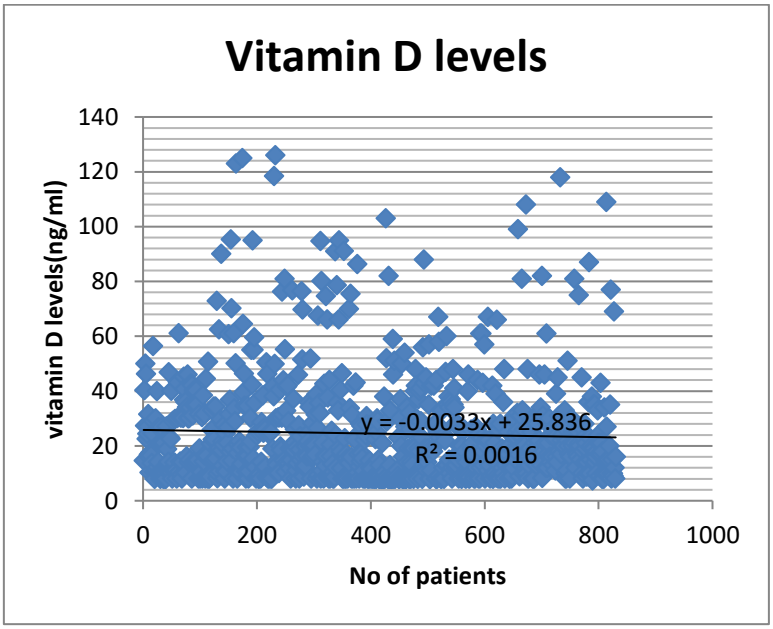

Figure 3- It is evident from the above graph that Vitamin D deficiency is quite rampant in Dehradun region of Uttarakhand, India. Apart from low intake in diet, people with liver, kidney and skin disorders also have Vitamin D deficiency. There are many reasons for it being so common in our country. Increased indoor lifestyle, thereby preventing adequate exposure to sunlight. This is mainly in the urban population due to modernization.

Discussion

Vitamin D is also known as calciferol because of its role in calcium metabolism and antirachitic factor because it prevents rickets. It is a modified steroid, synthesised in the skin under the influence of sunlight and is necessary for metabolism of calcium and phosphorus. Vitamin D undergoes hydroxylation in the liver to form 25 hydroxy vitamin D [25(OH)

vitamin D]. The two main forms are vitamin D2 (ergocalciferol) and vitamin D3 (cholecalciferol). Vitamin D2 is obtained from the diet, and is derived from ultraviolet irradiation of ergosterol, found in fungi. Both metabolites are transported in the blood bound to vitamin D binding protein (DBP). These inactive vitamin D metabolites must undergo a two-step hydroxylation process to become biologically active. Initially, vitamin D2 and D3 undergo hydroxylation in the maternal liver, via the action of vitamin D 25-hydroxylase enzyme (CYP27A1), to form the inactive steroid precursor 25-hydroxy-vitamin D (25[OH]D). 25[OH]D is the major circulating and stored form of vitamin D. Nearly 30 percent to 50 percent of people are estimated to have deficiency of vitamin D, and insufficiency and vitamin D deficiency are recognized as global health issues in the world.[16] Vitamin D is produced endogenously through exposure of skin to sunlight and it is absorbed from foods containing or supplemented with Vitamin D.[17,18] Its RDA is 400 IU or 10 mg, it binds to the receptor of target cells and regulate through gene expression.[19] Pro-vitamin D3 or 7-dehydrocholestrol, which is primarily found in the basal and spinous cell layers of the epidermis, undergoes a photochemical reaction to form pre-vitamin D3. The UV light blocking function of melanin leads to a requirement of greater UV light exposure in order to produce equivalent amounts of Vitamin D3 in dark skinned populations. In addition to its action on the kidneys, calcitriol bound to vitamin D binding protein acts by both genomic and non-genomic mechanisms on certain other target tissues like bone, intestine, and parathyroid gland that express the vitamin D receptors. [20] Vitamin D is metabolized to its biologically active form, 1,25-dihydroxyvitamin D, a hormone that regulates calcium and phosphate metabolism. Deficiency of vitamin D results in impaired bone formation and produces rickets in children and osteomalacia in adults. Poor diet, lack of sun exposure, decreased synthesis of Vitamin D and decreased renal hydroxylation of 25(OH) D due to old age are the main cause of Vitamin D deficiency.[21] However, vitamin D deficiency may result in inadequate circulating 25(OH)D, on the other hand, a low vitamin D status is emerging as a very common condition worldwide, and several studies from basic science to clinical applications have highlighted a strong association with chronic diseases, as well as acute conditions. Moreover, the large amount of observational data currently available are also accompanied by pathophysiological associations of vitamin D with energy homeostasis, and regulation of the immune and endocrine systems [22].Pollution can hamper the synthesis of Vitamin D in the skin by UV rays[23] changing food habits contribute to low dietary calcium and Vitamin D intake, Phytates and phosphates which are present in fiber rich diet, can deplete Vitamin D stores and increase calcium requirement[24] Increased skin pigmentation and application of sunscreens also affects its absorption from skin, Unplanned pregnancies in women with dietary deficit can lead to worsening of Vitamin D status in both mother and child. So adequate amount of Vitamin D should be taken as Vitamin D plays role beyond calcium homeostasis and bone formation. Keeping in mind the enormous health benefits of vitamin D, guidelines for food fortification, vitamin D supplementation and adequate calcium intake for the Indian population should be formulated and implemented. As vitamin D insufficiency and deficiency are easily preventable, the current recommendations of taking 1–1.5 g of dietary calcium and daily requirements for vitamin D are around 800–1000 IU, but larger doses are needed for patients who are already deficient. For moderate deficiency, that is 15–25 nmol/L, oral supplementation with 3000–5000 IU daily for 6–12

weeks can be used to replete stores followed by a maintenance dose of 1000–2000 IU per day. Vitamin D status should be assessed 3–4 months after commencing treatment as vitamin D is stored in fat and muscle and there is a lag time before normalisation of serum concentrations. For severe vitamin D deficiency, that is 25-hydroxyvitamin D less than 15 nmol/L, the intramuscular form of cholecalciferol 100,000 IU (megadose therapy) may be more suitable to replenish stores more quickly and effectively. This is especially pertinent for patients with malabsorption, acute medical illnesses and poor dietary compliance. Currently, such formulations are only available for specialists under a special access scheme. [25,26]

Vitamin D deficiency in the Indian scenario:

Nearly 30 percent to 50 percent of people are estimated to have deficiency of vitamin D, and insufficiency and vitamin D deficiency are recognized as global health issues in the world. [27] A community-based cross-sectional study was conducted by Palla Suryanarayana et.al among 298 urban elderly (≥60 years) by adapting a random sampling procedure. The mean ± SE plasma vitamin D and the prevalence of VDD among the urban elderly population were 19.3 ± 0.54 (ng/ml) and 56.3%, respectively. The prevalence of VDD was high among the urban elderly population in the south Indian city of Hyderabad. High BMI, MS, HT and education are significant associated factors of VDD. [28] Study conducted by Hinduja, Anupa R. A. et.al in Mumbai, India found that 57% of participants were deficient, 25% had insufficient, and 18% had adequate vitamin-D levels. There were a greater number of younger (P = 0.003) and upper-middle-class participants in the deficient group (P = 0.043816). Only 18.11% of participants had adequate vitamin-D levels. The prevalence of vitamin-D deficiency was equally distributed in both genders. Upper-middle-class participants had a higher prevalence of vitamin-D deficiency. There was no difference in the prevalence of comorbidities in vitamin-D-deficient, insufficient, and sufficient participants. [29]

Conclusion

Total 830 patients participated in this study; there were 340 males and 490 females. Among these patients 23.73% were having adequate level of Vitamin D, 19.27% having insufficient vitamin D levels, 53.97% having severe deficiency of vitamin D and 3.01% are having toxicity to Vitamin D. Our study concludes that there is high prevalence of vitamin D deficiency in Dehradun region of Uttarakhand, treating with vitamin D supplements and sun exposure maybe useful and seems to be necessary to overcome from deficiency status.

Acknowledgments

We all authors are very much thankful to all respected patients and all technical staff members for their valuable work in our study.

Ethical standard: 1) This material is the authors' own original work, which has not been previously published elsewhere.

2) The paper is not currently being considered for publication elsewhere.

3) The paper reflects the authors' own research and analysis in a truthful and complete manner.

Funding support: Nil

Conflict of Interest: Nil

Informed consent: Informed consent was taken for all the participants who was willing to take part in study.

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