



**Original Article**

**PREVALENCE OF VITAMIN D DEFICIENCY IN  
OUTDOOR PATIENTS**

**Publication History:**

Received 29/5/2020  
Accepted 23/6/2020  
Published 28/7/2020

**AUTHORS:**

- 1- DR. RANA GULRAIZ, MAYO HOSPITAL LAHORE
- 2- DR. AMNA AWAIS, HOLY FAMILY HOSPITAL RAWALPINDI
- 3- DR. NEHA NADEEM, HOLY FAMILY HOSPITAL RAWALPINDI

**Corresponding Author:**

Dr. Rana Gulraiz  
Mayo Hospital Lahore  
drranagulraiz1@yahoo.com



**Cite this article as:**

Gulraiz R, Awais A, Nadeem N. Prevalence of Vitamin D deficiency in outdoor patients. *Int. J. Med. Dent. Allied health sci.* 2020;2(7). 215-21

**DOI:**

10.5281/zenodo.3967757

**ABSTRACT:**

Vitamin D is a group of fat-soluble secosteroids responsible for increasing intestinal absorption of calcium, magnesium, and phosphate, and multiple other biological effects. In humans, the most important compounds in this group are vitamin D3 (also known as cholecalciferol) and vitamin D2 (ergocalciferol). This cross-sectional study was conducted in outdoor departments of different hospitals. Patients presenting with chronic diseases i.e. joint pains, hypertension, diabetes mellitus, and tuberculosis were included. A total of 136 patients were included in the study. There were 71 (52.21%) males and 65 (47.79%) female

© Copyright 2020

This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.





patients. The mean age of the patients was  $39.34 \pm 4.34$  years. The mean vitamin D levels were  $33.48 \pm 13.26$  nmol/L. Eighty-one (59.56%) patients were having normal levels of vitamin D, forty-eight (35.29%) patients had vitamin D levels lower than the normal and seven (5.15%) patients had vitamin D levels higher than normal levels.

**Keywords: Vitamin D Levels, Outdoor Patients**

**INTRODUCTION:**

Vitamin D is a group of fat-soluble secosteroids responsible for increasing intestinal absorption of calcium, magnesium, and phosphate, and multiple other biological effects. In humans, the most important compounds in this group are vitamin D3 (also known as cholecalciferol) and vitamin D2 (ergocalciferol). Vitamin D from the diet, or from skin synthesis, is biologically inactive. It is activated by two protein enzyme hydroxylation steps, the first in the liver and the second in the kidneys. As vitamin D can be synthesized in adequate amounts by most mammals if exposed to sufficient sunlight, it is not essential, so technically not a vitamin. Instead it can be considered a hormone, with activation of the vitamin D pro-hormone resulting in the active form, calcitriol, which then produces effects via a nuclear receptor in multiple locations (1, 2).



Serum concentration of 25(OH)D is the best indicator of vitamin D status. It reflects vitamin D produced cutaneously and that obtained from food and supplements and has a long circulating half-life of 15 days. 25(OH)D functions as a biomarker of exposure, but it is not clear to what extent 25(OH)D levels also serve as a biomarker of effect (i.e., relating to health status or outcomes). Serum 25(OH)D levels do not indicate the amount of vitamin D stored in body tissues. In contrast to 25(OH)D, circulating 1,25(OH)<sub>2</sub>D is generally not a good indicator of vitamin D status because it has a short half-life of 15 hours and serum concentrations are regulated by parathyroid hormone, calcium, and phosphate. Levels of 1,25(OH)<sub>2</sub>D do not typically decrease until vitamin D deficiency is severe (3, 4).

There is considerable discussion of the serum concentrations of 25(OH)D associated with deficiency (e.g., rickets), adequacy for bone health, and optimal overall health, and cut points have not been developed by a scientific consensus process. Based on its review of data of vitamin D needs, a committee of the Institute of Medicine concluded that persons are at risk of vitamin D deficiency at serum 25(OH)D concentrations <30 nmol/L (<12 ng/mL). Some are potentially at risk for inadequacy at levels ranging from 30–50 nmol/L (12–20 ng/mL). Practically all people are sufficient at levels ≥50 nmol/L (≥20 ng/mL); the committee stated that 50



nmol/L is the serum 25(OH)D level that covers the needs of 97.5% of the population. Serum concentrations >125 nmol/L (>50 ng/mL) are associated with potential adverse effects (1, 2, 5).

#### **MATERIAL AND METHODS:**

This cross-sectional study was conducted in outdoor departments of different hospitals. Patients presenting with chronic diseases i.e. joint pains, hypertension, diabetes mellitus, and tuberculosis were included. After informed consent, proper history of patients including demographic history i.e. age, gender, occupation, dietary routine, the structure of house, etc. was taken. Vitamin D levels were obtained from the laboratory. Data were analyzed using SPSS Ver. 25. Quantitative variables were presented as mean and standard deviation. Qualitative variables were presented as numbers and percentages. The relevant statistical analysis was performed.

#### **RESULTS:**

A total of 136 patients were included in the study. There were 71 (52.21%) males and 65 (47.79%) female patients. The mean age of the patients was  $39.34 \pm 4.34$  years, the mean age of male patients was  $40.12 \pm 2.32$  years and the mean age of female patients was  $38.43 \pm 2.78$  years. The mean vitamin D levels were  $33.48 \pm 13.26$  nmol/L. In males it values were 33.43



$\pm 15.51$  nmol/L and in females  $31.99 \pm 14.14$  nmol/L. Maximum levels noted were 59.12 nmol/L and minimum levels noted were 14.21 nmol/L. Eighty-one (59.56%) patients were having normal levels of vitamin D, forty-eight (35.29%) patients had vitamin D levels lower than the normal and seven (5.15%) patients had vitamin D levels higher than normal levels.

#### **DISCUSSION:**

Nutrient deficiencies are usually the result of dietary inadequacy, impaired absorption and use, increased requirement, or increased excretion. A vitamin D deficiency can occur when usual intake is lower than recommended levels over time, exposure to sunlight is limited, the kidneys cannot convert 25(OH)D to its active form, or absorption of vitamin D from the digestive tract is inadequate. Vitamin D-deficient diets are associated with milk allergy, lactose intolerance, ovo-vegetarianism, and veganism (6-8). Rickets and osteomalacia are the classical vitamin D deficiency diseases. In children, vitamin D deficiency causes rickets, a disease characterized by a failure of bone tissue to properly mineralize, resulting in soft bones and skeletal deformities. Rickets was first described in the mid-17th century by British researchers. In the late 19th and early 20th centuries, German physicians noted that consuming 1–3 teaspoons/day of cod liver oil could reverse rickets. The fortification of milk with



vitamin D beginning in the 1930s has made rickets a rare disease in the United States, although it is still reported periodically, particularly among African American infants and children. Prolonged exclusive breastfeeding without the AAP-recommended vitamin D supplementation is a significant cause of rickets, particularly in dark-skinned infants breastfed by mothers who are not vitamin D replete.

Additional causes of rickets include extensive use of sunscreens and placement of children in daycare programs, where they often have less outdoor activity and sun exposure. Rickets is also more prevalent among immigrants from Asia, Africa, and the Middle East, possibly because of genetic differences in vitamin D metabolism and behavioral differences that lead to less sun exposure. In adults, vitamin D deficiency can lead to osteomalacia, resulting in weak bones. Symptoms of bone pain and muscle weakness can indicate inadequate vitamin D levels, but such symptoms can be subtle and go undetected in the initial stages (9, 10).

**REFERENCES:**

1. Adams JS, Hewison M. Update in vitamin D. *The Journal of Clinical Endocrinology & Metabolism*. 2010;95(2):471-8.
2. Bikle DD. Vitamin D metabolism, mechanism of action, and clinical applications. *Chemistry & biology*. 2014;21(3):319-29.



3. Christakos S, Ajibade DV, Dhawan P, Fechner AJ, Mady LJ. Vitamin D: metabolism. *Rheumatic Disease Clinics*. 2012;38(1):1-11.
4. Haussler MR, Whitfield GK, Kaneko I, Haussler CA, Hsieh D, Hsieh J-C, et al. Molecular mechanisms of vitamin D action. *Calcified tissue international*. 2013;92(2):77-98.
5. Toss G, Almqvist S, Larsson L, Zetterqvist H. Vitamin D deficiency in welfare institutions for the aged. *Acta Medica Scandinavica*. 1980;208(1-6):87-9.
6. Malabanan A, Veronikis I. Redefining vitamin D insufficiency. *The Lancet*. 1998;351(9105):805-6.
7. Rosen CJ. Vitamin D insufficiency. *New England Journal of Medicine*. 2011;364(3):248-54.
8. Thacher TD, Clarke BL, editors. *Vitamin D insufficiency*. Mayo Clinic Proceedings; 2011: Elsevier.
9. Ward LM, Gaboury I, Ladhani M, Zlotkin S. Vitamin D-deficiency rickets among children in Canada. *Cmaj*. 2007;177(2):161-6.
10. Pettifor JM, Thandrayen K, Thacher TD. Vitamin D deficiency and nutritional rickets in children. *Vitamin D: Elsevier*; 2018. p. 179-201.