

Alterations in Serum Biochemical Parameters, Lipid Profile Markers, and Atherogenic Index in Patients with Vitamin D Deficiency

¹Sara Ghrib and ^{1,2}Samir Derouiche

¹Department of Cellular and Molecular Biology, Faculty of Natural Sciences and Life, University of El-Oued, El-Oued 39000, Algeria

²Laboratory of Biodiversity and Application of Biotechnology in the Agricultural Field, Faculty of Natural Sciences and life, University of El Oued, El-Oued 39000, Algeria

ABSTRACT

Background and Objective: Vitamin D deficiency is a global health problem caused mainly by obesity, lifestyle changes, and reduced sun exposure. This study aimed to evaluate the effect of vitamin D deficiency on lipid profile and biochemical biomarkers in men and women. **Materials and Methods:** The experimentation was carried out on 24 voluntary individuals who were divided into four groups: Two groups of healthy men and women (controls), and the other two groups were men and women vit D deficiency. All the volunteers were subjected to an estimation of the lipid profile, biochemical parameters. Data are presented as mean \pm SEM and compared using Student's t-test in Minitab; $p < 0.05$ was considered significant. **Results:** There was a significant increase ($p < 0.001$) in TC, LDL-C, and Atherogenic index (AI) were highly significantly raised ($p < 0.01$). At the same time, the HDL-C level is significantly decreased ($p < 0.001$) for vit D deficiency groups compared to the control one. However, the biochemical parameters didn't change. The biochemical results demonstrated that there was a significant change ($p < 0.05$) in Blood glucose, urea, and creatinine levels in vit D deficiency patients compared to the controls. **Conclusion:** The vitamin D deficiency may induce severe alterations in lipid profiles which cause an increase in cardiovascular risk for this population.

KEYWORDS

Vitamin D deficiency, lipid profile, atherogenic index, biochemical markers

Copyright © 2025 Ghrib and Derouiche. This is an open-access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

Vitamin D (25-hydroxyvitamin D (25 (OH) D)), the sunshine vitamin, is now recognized not only for its importance in bone health in Children and adults, but also for other health benefits, including reducing the risk of chronic diseases, including autoimmune diseases, common cancer, and cardiovascular disease¹. Vitamin D (which includes both D2 (ergocalciferol) and D3 (cholecalciferol)) carries out essential biologic functions through both an endocrine mechanism and an autocrine mechanism. It is found in all animals, and in humans, it is made in skin exposed to ultraviolet (UV)-B radiation^{2,3}. There are many sources of vitamin D, including vitamin D3 (calcifediol), which is mainly found in foods of animal origin, and vitamin D2 in foods of plant and yeast. In addition, there is UVB light, dietary supplements, and drugs⁴.



Vitamin D deficiency is a global health problem caused mainly by insufficient exposure to sunlight. The main causes of this deficiency are obesity, lifestyle changes, and reduced sun exposure. It is estimated that 1 billion people have vitamin D deficiency or insufficiency worldwide, particularly prevalent among elderly people^{5,6}. Many of the recent landmarks in scientific research have shown that in human beings, Oxidative stress is an important factor causing metabolic and physiological alterations and various diseases in the body⁷. The inflammatory cells are then a source of free radicals in the form of reactive oxygen and nitrogen species. They are constantly generated inside cells following exposure to xenobiotics in our ambient environment. It can affect the cardiovascular system, the respiratory system, the oral cavity, and teeth⁸. The present study was undertaken to assess changes in Serum Biochemical and lipid profile Markers in patients with vitamin D deficiency.

MATERIALS AND METHODS

Ethical approval: Ethical approval was sought and approved by the Ethical Committee of the Department of Cellular and Molecular Biology, Faculty of Natural Science and Life, University of El Oued, Algeria.

Study area: Randomly enrolled 24 volunteer men and women who visited the endocrinology service of Hospital Bachir Bennaceur in El-Oued State, located in Southeast of Algeria, from January 02, 2025, until February 18, 2025.

Sampling: The current study was conducted on 24 male and female volunteers. The men were aged between 19 and 71 years and were divided into two groups: A group of two healthy men as a control group with an average age of 50.5 ± 9.19 years, and another group of men suffering from vitamin D deficiency with an average age of 36.75 ± 23.44 years. While the women were aged between 27 and 70 years and were also divided into two groups: A group of healthy women as a control group with an average age of 50.5 ± 11.73 years, and another group of women suffering from vitamin D deficiency with an average age of 46.15 ± 11.9 years. All volunteers live in the El-Oued Region.

Inclusion criteria: Patients who had clinical diagnosis and laboratory findings of vitamin D deficiency for more than a month were evident.

Exclusion criteria: To eliminate the factors that might affect serum biochemical parameters, we excluded all anemia and other chronic diseases from the patient groups and healthy controls.

Methods and laboratory investigations: Fasting blood samples were collected and placed into tubes. Blood was transferred into HEPA tubes for Serum Biochemical studies, and the serum was obtained after centrifugation at $3000 \times g$ for 5 min, removed and retained for assay of the level of glucose, biochemical parameters. Serum samples were stored at -20°C until analysis. Serum Biochemical analysis is performed by the biochemical automated analyzer. Triglyceride (TG), Total Cholesterol (TC), Density Lipoprotein-cholesterol (HDL-C), urea, creatinine, and Calcium concentrations were measured using commercial kits (Spin React). Low Density Lipoprotein-cholesterol (LDL-C), Very Low-Density Lipoprotein-cholesterol (VLDL, C) were calculated by using formula:

$$\text{LDL-C} = \text{TG}/5$$

$$\text{LDL-C} = \text{TC} - (\text{HDL-C} + \text{TG}/5)^9$$

$$\text{Atherogenic index (AI)} = \text{TC}/\text{HDL-C}^{10}$$

Statistical analysis: The reported data are the means of measurements and their standard error of mean (SEM) values. The results of cases and controls were compared by student's test using Minitab software. Differences were considered statistically significant at $p < 0.05$.

RESULTS AND DISCUSSION

Biochemical markers: Current results for biochemical parameters (Table 1) revealed a significant decrease ($p<0.001$) in vitamin D levels in both men and women with vitamin D deficiency compared to controls. Results also showed a significant change in blood glucose levels ($p<0.01$) in men with vitamin D deficiency compared to controls, and in women, glucose levels were also higher ($p<0.05$) compared to control women. Similarly, for urea, we recorded a significant change ($p<0.05$) in men and women with vitamin D deficiency compared to controls. Our results are consistent with those reported by Xiao¹¹. Serum vitamin 25 (OH) D levels were significantly associated with age, sex, history of diabetes, estimated glomerular filtration rate, fasting blood glucose, glycated hemoglobin (HbA1c), creatinine clearance rate, and blood urea nitrogen. Lower serum 25 (OH) D concentrations appear to be associated with a high blood glucose levels¹².

The results of the current study showed a clear association between low levels of vitamin D and significant changes in biochemical analysis parameters and blood lipid levels (Table 2). There was a significant increase ($p<0.001$) in TC, LDL-C, and TG in vitamin D-deficient individuals compared to controls. Meanwhile, HDL-C levels were significantly decreased ($p<0.001$) in vitamin D-deficient groups compared to controls. Serum cholesterol, triglycerides, and Low-Density Lipoprotein (LDL) levels were significantly elevated in patients with vitamin D deficiency or insufficiency, while High-Density Lipoprotein (HDL) levels were significantly decreased^{13,14}. This study provides evidence of a significant relationship between serum vitamin D levels and LDL cholesterol levels in patients. When we analyzed, we found that as serum vitamin D levels increased, the average LDL cholesterol level decreased significantly¹⁵. Deficiency of 25(OH)D affects the levels of TC, LDL-C, and TG¹⁶.

Atherogenic index: The Atherogenic index (AI) of Women vit D deficiency was found to be statistically significantly elevated ($p<0.01$) compared to controls, as demonstrated in Fig. 1. While The Atherogenic index (AI) of men with vitamin D deficiency was not significantly changed ($p>0.05$) compared to control Men. These results are consistent with what was indicated by Lhilali I, Based on the Atherogenic indices, this study indicates that vitamin D below 20 ng/mL may increase the risk of cardiovascular disease in adult

Table 1: Comparison of biochemical parameters between control and vitamin D-deficient subjects

| Paramètre | Control | | Vit D deficiency | |
|---------------------|-------------|-------------|------------------|---------------|
| | Men | Women | Men | Women |
| Age (y) | 50.5±9.19 | 50.5±11.73 | 36.75±23.44 | 46.15±11.9 |
| Serum Vit D | 43.95±10.67 | 36.0±8.56 | 22.62±3.04*** | 16.87±6.17*** |
| Blood glucose (g/L) | 1.385±0.42 | 1.35±0.66 | 0.88±0.14** | 1.10±0.36* |
| Serum urea | 0.26±0.09 | 0.26±0.055 | 0.20±0.02* | 0.24±0.09* |
| Serum creatinin | 6.155±0.96 | 6.23±0.821 | 7.16±1.42 | 5.20±0.65 |
| Serum calcium | 94.32±6.6 | 94.73±47.37 | 95.63±1.12 | 92.14±4.05 |

Values are expressed as mean±SEM, $p<0.05$ (*), $p<0.01$ (**), and $p<0.001$ (***) indicate statistically significant differences compared to respective control groups

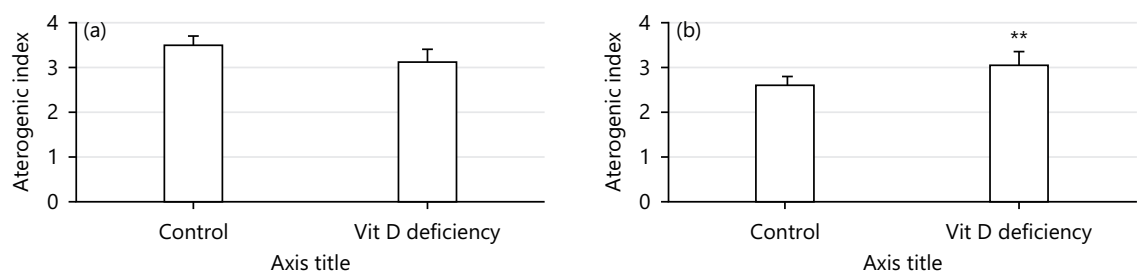


Fig. 1: Variation of the mean atherogenic index (AI) in (a) Men and (b) Women vit D deficiency and control groups

Values are mean±SEM and ** $p<0.01$: significantly different compared to the control group

Table 2: Lipid profile comparison between control and vitamin D-deficient subjects

| Parameter | Control | | Vit D deficiency | |
|--------------------------|------------|------------|------------------|-------------------------|
| | Men | Women | Men | Women |
| Serum HDL (g/L) | 0.39±0.02 | 0.77±0.4 | 0.53±0.05* | 0.59±0.2 ^{NS} |
| Serum triglycerids (g/L) | 1.165±0.06 | 0.93±0.27 | 0.79±0.25** | 1.05±0.5* |
| Serum TC (g/L) | 1.37±0.39 | 2.025±0.15 | 1.66±0.31* | 1.82±0.34 ^{NS} |
| serum VLDL (g/L) | 0.23±0.012 | 0.18±0.05 | 0.15±0.05* | 0.21±0.1 ^{NS} |
| Serum LDL (g/L) | 0.75±0.3 | 1.07±0.1 | 0.98±0.04* | 1.02±0.2 ^{NS} |

Values are expressed as mean±SEM, p<0.05 (*), p<0.01 (**), and "NS" indicates non-significant difference compared to respective control groups

women¹⁷. In a study, the prevalence of low Serum 25 (OH) D3 levels was considerably high in patients. Therefore, the augmentation of total cholesterol and the decrease of HDL-C make an increase of Atherogenic index, which may be predictive of cardiovascular risk in various clinical settings^{18,19}. The association between 25 (OH) D and various CVD risk markers suggests that 25 (OH) D might help in the prediction of CVD risk with CVD risk factors²⁰. Low vitamin D levels favor atherosclerosis, enabling vascular inflammation, endothelial dysfunction, the formation of foam cells, and the proliferation of smooth muscle cells²¹. It also supports existing evidence suggesting that adequate vitamin D status could reduce the risks of overt adult²². The present study indicated that AIP might be a strong marker for predicting the risk of CAD in postmenopausal women with cardiovascular disease²³. It was observed, however, that the increase of 25 (OH) D level results in an increased number of patients without significant lesions in the coronary arteries¹⁶.

CONCLUSION

The results of this study demonstrated a statistically significant association between vitamin D deficiency and significant changes in biochemical markers, lipid profile, and atherosclerosis in patients with this deficiency. Low vitamin D levels were associated with a significant increase in total cholesterol, triglycerides, and Low-Density Lipoprotein (LDL) cholesterol levels, along with a decrease in High-Density Lipoprotein (HDL) cholesterol levels. This supports the hypothesis that vitamin D deficiency contributes to cardiovascular risk and the development of atherosclerosis. These findings highlight the importance of routine assessment of vitamin D levels as an essential part of clinical follow-up, especially in groups at risk for cardiovascular disease. They also highlight the need to include strategies to correct vitamin D deficiency in prevention and therapeutic intervention programs to limit the development of metabolic disorders and reduce the associated health burden.

SIGNIFICANCE STATEMENT

Vitamin D deficiency is increasingly recognized as a contributing factor to metabolic and cardiovascular disorders, yet it often remains undiagnosed in clinical practice. This study reveals significant alterations in biochemical and lipid parameters among vitamin D-deficient individuals, particularly in those at higher risk of cardiovascular disease. These findings underscore the clinical value of routine Vitamin D screening and highlight the need for targeted correction strategies. Integrating vitamin D assessment and management into prevention and treatment programs could help mitigate metabolic complications and reduce long-term health burdens.

ACKNOWLEDGMENT

The author thanks the staff of El-Medjed Medical Analysis Laboratory for providing research facilities to carryout present work.

REFERENCES

1. Holick, M.F., 2009. Vitamin D status: Measurement, interpretation and clinical application. *Ann. Epidemiol.*, 19: 73-78.
2. Lappe, J.M., 2011. The role of vitamin D in human health: A paradigm shift. *J. Evidence-Based Complementary Altern. Med.*, 16: 58-72.

3. Hossein-Nezhad, A. and M.F. Holick, 2013. Vitamin D for health: A global perspective. *Mayo Clinic Proc.*, 88: 720-755.
4. Benedik, E., 2022. Sources of vitamin D for humans. *Int. J. Vitam. Nutr. Res.*, 92: 118-125.
5. Sahota, O., 2014. Understanding vitamin D deficiency. *Age Ageing*, 43: 589-591.
6. de Souza de Santana, K.V., S.L. Oliver, M.M. Mendes, S. Lanham-New, K.E. Charlton and H. Ribeiro, 2022. Association between vitamin D status and lifestyle factors in Brazilian women: Implications of sun exposure levels, diet, and health. *eClinicalMedicine*, Vol. 47. 10.1016/j.eclinm.2022.101400.
7. Boulaares, I., S. Derouiche and I.Y. Guemari, 2024. Impact of doxorubicin chemotherapy on oxidative stress status in heart and liver: An experimental study on rats. *Pharm. Sci. Anal. Res. J.*, Vol. 6.
8. Chetehouna, S., S. Derouiche, I. Boulaares and Y. Réggami, 2024. Phytochemical profile, anti-inflammatory analysis and cytotoxic activity of *SmE-SeNPs* against breast (MCF-7) cancer cells. *Biocatal. Agric. Biotechnol.*, Vol. 57. 10.1016/j.bcab.2024.103122.
9. Friedewald, W.T., R.I. Levy and D.S. Fredrickson, 1972. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin. Chem.*, 18: 499-502.
10. Sastre-Alzamora, T., P.J.T. López, Á.A. López-González, D. Vallejos, H. Paublini and J.I.R. Manent, 2024. Usefulness of atherogenic indices for predicting high values of avoidable lost life years heart age in 139,634 Spanish workers. *Diagnostics*, Vol. 14. 10.3390/diagnostics14212388.
11. Xiao, X., Y. Wang, Y. Hou, F. Han, J. Ren and Z. Hu, 2016. Vitamin D deficiency and related risk factors in patients with diabetic nephropathy. *J. Int. Med. Res.*, 44: 673-684.
12. Valladares, T., M.R. Cardoso and J.M. Aldrighi, 2019. Higher serum levels of vitamin D are associated with lower blood glucose levels. *Menopause*, 26: 781-784.
13. Bashir, N.A., A.A.M. Bashir and H.A. Bashir, 2019. Effect of vitamin D deficiency on lipid profile. *Am. J. Lab. Med.*, 4: 11-18.
14. Han, Y.Y., S.H.J. Hsu and T.C. Su, 2021. Association between vitamin D deficiency and high serum levels of small dense LDL in middle-aged adults. *Biomedicines*, Vol. 9. 10.3390/biomedicines9050464.
15. Gholamzad, A., N. Khakpour, T. Kabipour and M. Gholamzad, 2023. Association between serum vitamin D levels and lipid profiles: A cross-sectional analysis. *Sci. Rep.*, Vol. 13. 10.1038/s41598-023-47872-5.
16. Dziedzic, E.A., S. Przychodzeń and M. Dąbrowski, 2016. The effects of vitamin D on severity of coronary artery atherosclerosis and lipid profile of cardiac patients. *Arch. Med. Sci.*, 6: 1199-1206.
17. Lhilali, I., N. Zouine, L. Godderis, A. El Midaoui, S. El Jaafari and Y. Filali-Zegzouti, 2024. Relationship between vitamin D insufficiency, lipid profile and atherogenic indices in healthy women aged 18-50 years. *Eur. J. Invest. Health Psychol. Educ.*, 14: 2337-2357.
18. Sara, C., A. Ouidad, B. Islam, G.I. Yousra and D. Samir, 2020. The effect of chronic tobacco smoking on atherogenic index and cardiovascular diseases risk in El-Oued (Algeria) men. *Asian J. Res. Chem.*, 13: 489-493.
19. Atoussi, O., S. Chetehouna, I. Boulaares, Y.G. Imane and S. Derouiche, 2021. Analysis of blood pressure, lipid profile and hematological biomarkers in men addicted to tobacco chewing. *Res. J. Pharmacol. Pharmacodyn.*, 13: 1-4.
20. Mahmoodi, M.R. and H. Najafipour, 2022. Associations between serum vitamin D₃, atherogenic indices of plasma and cardiometabolic biomarkers among patients with diabetes in the KERCADR study. *BMC Endocr. Disord.*, Vol. 22. 10.1186/s12902-022-01043-1.
21. Mozos, I. and O. Marginean, 2015. Links between vitamin D deficiency and cardiovascular diseases. *BioMed Res. Int.*, Vol. 2015. 10.1155/2015/109275.
22. Sonuga, O.O., O.O. Eluyera and A.A. Sonuga, 2019. Influence of vitamin D status on atherogenic profile of apparently healthy young adults. *Appl. Med. Res.*, Vol. 6. 10.5455/amr.20190409125737.
23. Wu, T.T., Y. Gao, Y.Y. Zheng, Y.T. Ma and X. Xie, 2018. Atherogenic index of plasma (AIP): A novel predictive indicator for the coronary artery disease in postmenopausal women. *Lipids Health Dis.* Vol. 17. 10.1186/s12944-018-0828-z.