IJBPAS, August, 2022, 11(8): 3515-3522

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

ASSOCIATION BETWEEN THE SERUM 25HYDROXY CHOLECALCIFEROL AND LIPID PROFILE IN THE PATIENTS OF CORONARY ARTERY DISEASE IN PUDUCHERRY POPULATION

E.VASUDEVAN[1*], B. SHANTHI[2], MARY CHANDRIKA ANTON[3] AND M. MAHALAKSHMI[4]

1: Tutor,Dept of Biochemistry, SreeBalaji Medical College and Hospital

2: Professor and Head, Department of Biochemistry, SreeBalaji Medical College and Hospital

3: Assistant Professor, Department of Biochemistry, SreeBalaji Medical College and Hospital

4: Tutor, Dept of Biochemistry, Sree Balaji Medical College and Hospital

***Corresponding Author: E.Vasudevan: E Mail:Vasukalai24@gmail.com**

Received 16th Aug. 2021; Revised 20th Sept. 2021; Accepted 08th Dec. 2021; Available online 1st Aug. 2022

https://doi.org/10.31032/IJBPAS/2021/11.8.6270

ABSTRACT

Introduction: Coronary artery disease (CAD) is the most common disease affecting both men and women world-wide. Lifestyle modifications play a major role as the causative factor for CAD. **Aims**: To determine the association between vitamin D level and serum lipid profile in patients with Coronary Artery Disease. **Methods**: This Study was conducted in Mahatma Gandhi Medical College and ResearchInstitute, Puducherry. A standardised health questionnaire was circulated among study participants - 30Coronary Artery Disease patients (cases) and 30 healthy controls. It includedinformation on current and previous medicines, hypertension, and Coronary artery participants. Subjects were selected by their answer to the questions pertaining to this study.Cases and controls had their means and standard deviations (SD) calculated. Analysis of variance (ANOVA) and Pearson's correlation were performed to determine the relationship between the parameters was used to find the statistical significance and correlation of Vitamin D and Lipid Profile among the both the groups. **Result**: The vitamin D values of cases (21.14 ± 12.9) are low when compared to controls (56.54 ± 18.88) with a significant p value. LDL of cases (129.1 ± 26.91) was found to be high than controls (99.77 ± 26.23) (105.1 ± 25.43). HDL of cases (33.83±6.82) was found to be lower than

controls (49.53±6.12).**Conclusion**: Altered lipid profile with low HDL-C, high LDL-C, high LDL-C/HDL-C suggests increased risk for CAD. Low vitamin D levels are also associated with high risk for CAD. This study shows that CAD patients were found to have low Vitamin D levels and altered lipid profile status. Correcting vitamin D deficiency will prevent Coronary artery disease.

Keywords: coronary artery disease (CAD), 25hydroxy cholecalciferol, HDL, LDL

INTRODUCTION

There is ample epidemiological evidence suggesting that high blood cholesterol levels increase cardiovascular risk, and that this risk varies depending on the amounts of the various serum cholesterol fractions **[1]**. The LDL-C/HDL-C ratio, which can be derived from a routine lipid profile and is more accurate than LDL-C or HDL-C alone as an indicator of cardiovascular risk. Determination of the LDL-C/HDL-C ratio helps in deciding at which point lipid-lowering medication should be started. The LDL-C/HDL-C ratio had more predictive value than LDL-C or HDL-C alone in the Helsinki project, a 5-year clinical trial of more than 4,000 middle-aged men with increased lipids **[2]**. The current NCEP guidelines indicate LDL-C/HDL-C ratio levels of roughly 2.5 **[3]**. According to current studies, the risk of dying from cardiovascular disease increases dramatically around the ratio of 3.3-3.7 **[4]**. Previous epidemiological studies have linked a lack of 25hydroxy cholecalciferol to myocardial infraction, heart failure, diabetic cardiovascular disease, and peripheral vascular disease. Individuals with vitamin-D insufficiency who were monitored for 4 to 5 years had a 53 percent to 80 percent increased rate of cardiovascular consequences such as death from MI or heart failure **[5]**. More recent research has found a link between hypertension, diabetes, atherosclerosis, microalbuminuria, stroke, and reduced renal function and the mean serum level of 25(OH)D **[6- 9]**. Vitamin-D deficiency is more common in most countries of the world, including India. Humans get vitamin-D via the sun or by food intake **[10]**. As a result, this study was done to examine if there was a relationship between vitamin D and serum lipid profile in coronary heart disease patients in Puducherry population.

MATERIALS AND METHODS

The research was carried out at the Department of Biochemistry, in collaboration with the Departments of General Medicine and Cardiology, at the Mahatma Gandhi Medical College and Research Institute, Pillayarkuppam, Puducherry [MGMCRI], a tertiary health care institution under the jurisdiction of Sri

BalajiVidyapeeth University Puducherry. The Institutional Research Committee and the Institutional Human Ethical Committee both authorised the project.

A standardised health questionnaire were circulated among study participants - Coronary Artery Disease patients (cases) and healthy controls. It includedinformation on current and previous medicines, hypertension, and Coronary artery participants. Subjects were selected by their answer to the questions pertaining to this study.

INCLUSION CRITERIA

- CASES -Newly diagnosed coronary artery disease patients in General medicine and Cardiology OPD at Mahatma Gandhi Medical College and Research Institute
- CONTROLS –Age matched healthy volunteers who were employed in MGMC&RI

EXCLUSION CRITERIA

- Patient who have Tuberculosis, Type-1 diabetes mellitus, Anemia, Chronic bone pain, Renal failure and Endocrine disorders
- Pregnant females
- Patients who are already on calcium, vitamin-D, multivitamin, haematinic, steroid, and hormonal treatment

SAMPLE COLLECTION:

Under aseptic precautions, by venepuncture venous blood(5ml) was drawn from all the subjects after overnight fasting of 10-12hours. From that serum was separated for analysing of serum vitamin-D levels and Lipid profile.

STUDY PARAMETERS:

1.Serum Vitamin-D(25-(OH)D)
2.Serum total cholesterol
3.Serum Triglycerides
4.High density lipoprotein(HDL)
5.Low density Lipoprotein(LDL)
8.Serum urea
9.Serum creatinine
10.Blood Glucose

Estimation of Vitamin-D was done by direct competitive chemiluminescence immunoassay (CLIA). The 25 OH vitamin D is dissociated from its binding protein during the first incubation and binds to the particular antibody on the solid phase. The tracer (vitamin D coupled to an isoluminol derivative) is added after 10 minutes. The unbound material is removed using a wash cycle after a second 10 minute incubation.The starter reagents are then introduced to start the flash chemiluminescent process. A photomultiplier measures the light signal in relative light units (RLU), which is inversely proportional to the amount of 25 OH vitamin D present in the calibrators, controls, or samples. Lipid profile parameters total cholesterol was estimated by CHOD-POD method, triglycerides by

GPO-POD method, HDL and LDL by direct method, LDL-C/HDL-C ratio by calculation.

STATISTICAL ANALYSIS:

Cases and controls had their means and standard deviations (SD) calculated. Statistical Package for the Social Sciences (SPSS) version 17 software was used to perform the statistical analysis. Analysis of variance (ANOVA) and Pearson's correlation were performed to determine the relationship between the parameterswasused to find the statistical significance and correlation of Vitamin D and Lipid Profile among the both the groups. P ≤ 0.05 was considered significant.

RESULTS

The results of this study are elaborated below.

Table 1 shows the mean and SD of both cases and control along with p value.

The vitamin D values of cases (21.14 ± 12.9) are low when compared to controls (56.54 ± 18.88) with a significant p value. Total cholesterol of cases (193.33 ± 59.2) are high when compared to controls (154.53 ± 27.07). Triglycerides of cases (126.73±44.89), LDL of cases (129.1 ± 26.91) were found to be high than controls (99.77 ± 26.23) (105.1 ± 25.43). HDL of cases (33.83±6.82) was found to be lower than controls (49.53±6.12).

Table 1: Comparison of study parameters between cases and control

PARAMETERS	CASES (MEAN ± SD)	CONTROLS (MEAN ± SD)	T-TEST	p-VALUE
Vitamin D	21.14 ± 12.9	56.54 ± 18.88	8.4776	0.0000
Cholesterol	193.33 ± 59.2	154.53 ± 27.07	3.2645	0.0018
TAG	126.73±44.89	99.77 ± 26.23	2.8410	0.0062
HDL	33.83±6.82	49.53±6.12	0.717	0.479
LDL	129.1 ± 26.91	105.1 ± 25.43	3.5508	0.0008
Urea	45.6 ± 6.36	25.97 ± 7.11	11.2746	0.0000
Creatinine	1.7 ± 0.21	0.96 ± 0.18	14.5126	0.0000
Glucose	120.37 ± 5.59	89.43 ± 10.6	14.1354	0.0000

SD – standard deviation

Table 2: Correlation Between Lipid Profile and Vitamin –D

Correlations		
		VitaminD
Cholesterol	Pearson Correlation	-.325*
	Sig. (2-tailed)	0.011
TAG	Pearson Correlation	-0.142
	Sig. (2-tailed)	0.279
HDL	Pearson Correlation	-0.016
	Sig. (2-tailed)	0.906
LDL	Pearson Correlation	-.332**
	Sig. (2-tailed)	0.01

****Correlation is significant at the 0.01 level (2-tailed).**

***Correlation is significantat the 0.05 level (2-tailed).**

DISCUSSION

The current study found that vitamin –D (25(OH)D) levels in CAD patients were [21.14 ± 12.9ng/dl], compared to [56.54 ± 18.88ng/d] in the control group. However, when compared to the control group, the reduction in vitamin D concentration was statistically significant (P<0.000).We also discovered a negative connection between serum vitamin D and coronary artery disease (CAD) in the current investigation. Previous epidemiological research revealed that myocardial infarction, heart failure, diabetic cardiovascular disease, and peripheral vascular disease are all linked to a lack of 25(OH) D. Most body cells including cardiomyocytes and vascular smooth muscle and also the endothelium of the vessels have vitamin D receptors **[11]**. The mechanism that causes vitamin D's preventative benefit against cardiovascular disease is unknown, although various theories have been offered, including the effect of vitamin D on insulin compliance, blood pressure, and parathyroid hormone levels.

Furthermore, vitamin D has anti-inflammatory properties, and existing cholesterol is cleared by macrophages, resulting in foam cell production on vending machine walls. In addition, there is an inverse relationship between vitamin D serum level and coronary artery calcification. Vitamin D deficiency is a rather prevalent condition. The risk of cardiovascular disease was nearly four times higher in persons who were vitamin D deficient compared to those who had a normal level of vitamin D.

Decholecalciferol suppresses the rennin gene, which regulates the renin-angiotensin axis. Changes in the smooth muscles of the vascular wall, as well as inflammation, are caused by 25 – OH cholescalciferol, which could explain cardiovascular complications **[11-13]**. Clinical investigations have found a cross-sectional link between low vitamin D levels and plasma renin activity, blood pressure, coronary artery calcification, and cardiovascular disease **[14, 15]**. Furthermore, experimental and clinical data suggest that vitamin D deficiency promotes the development of hypertension, suggesting another possible mechanism linking vitamin D deficiency hypertension and vascular risk **[16, 17]**. Furthermore, levels of 1,25dihydroxy cholecalciferol have been shown to be inversely associated with vascular calcification, suggesting that vitamin D may influence MI risk through its effects on vascular calcification **[14]**. Low ca++ intake, possibly in combination with vitamin D deficiency, has been linked to impaired fasting glucose and the risk of type 2 diabetes mellitus, both of which are CVD risk factors **[18]**.

CONCLUSION

Coronary artery disease is highly prevalent non communicable disease in India. Many people are getting affected in young age itself. Altered lipid profile with low HDL-C, high LDL-C, highLDL-C/HDL-C suggests increased risk for CAD. Low vitamin D levels are also associated with high risk for CAD. This study shows that CAD patients were found to have low Vitamin D levels and altered lipid profile status. Correcting vitamin D deficiency will prevent Coronary artery disease.

AUTHORS CONTRIBUTION:

This study was done byMr. E.Vasudevan under the guidance of Dr. B. Shanthi. Dr. Mary Chandrika Anton contributed in review of literature and discussion.

ACKNOWLEDGEMENT:

We acknowledge theDepartment of Biochemistry, Departments of General Medicine and Cardiology of Mahatma Gandhi Medical College and Research Institute, Puducherry for giving us support to complete this project.

ETHICAL STANDARDS

The study involved human participants following the ethical standards of the tertiary health care institution where the study was conducted.

CONFLICT OF INTEREST

Declared none by the authors.

LIMITATIONS OF THE STUDY

The study population shall be enlarged as it was relatively less.

FUNDING:

The principle investigator did not get fund from any agencies for carrying out this project.

REFERENCES:

[1] Ghada A. Soliman Dietary Cholesterol and the Lack of Evidence in Cardiovascular Disease, Nutrients. 2018 Jun; 10(6): 780. Published online 2018 Jun 16. doi: 10.3390/nu10060780

[2] Statins, high-density lipoprotein cholesterol, and regression of coronary atherosclerosis. Nicholls SJ, Tuzcu EM, Sipahi I, Grasso AW, Schoenhagen P, Hu T, Wolski K, Crowe T, Desai MY, Hazen SL, Kapadia SR, Nissen SE JAMA. 2007 Feb 7; 297(5):499-508.

[3] Lemieux I, Lamarche B, Couillard C, F. Total Cholesterol/HDL Cholesterol Ratio vs LDL Cholesterol/HDL Cholesterol Ratio as Indices of Ischemic Heart Disease Risk in Men: The Quebec Cardiovascular Study. *Arch Intern Med.* 2001;161(22):2685–2692. doi:10.1001/archinte.161.22.2685

[4] T. Montalcini, G. Gorgone, G.Sesti, F. Perticone. Relation

between serum uric acid and carotid intima-media thickness in healthy postmenopausal women. Department of clinical and experimental medicine "G. Salvatore" university of Catanzaromagna Graecia, Viale Europa, I-88 100 catanzaro, Italy.

[5] Holick MF. Vitamin D deficiency. N Engl J Med 2007; 357(3):266-81.

[6] Scragg R, Sowers M, Bell C. Serum 25-hydroxyvitamin D, ethnicity and blood pressure in the Third National Health and Nutrition Examination Survey. Am J Hypertens 2007; 20(7): 713-9.

[7] Scragg R, Sowers M, Bell C. Serum 25-hydroxyvitamin D, diabetes, and ethnicity in the Third National Health and Nutrition Examination Survey. Diabetes Care 2004; 27(12): 2813-8.

[8] Martins D, Wolf M, Pan D, Zadshir A, Tareen N, Thadhani R, *et al*. Prevalence of cardiovascular risk factors and the serumlevels of 25-hydroxyl vitamin D in the United States: data from the Third National Health and Nutrition Examination Survey. Arch Intern Med 2007; 167(11): 1159-65

[9] Kendrick J, Targher G, Smits G, Chonchol M. 25-Hydroxyvitamin D deficiency is independently associated with cardiovascular disease in the Third National Health and Nutrition Examination Survey. Atherosclerosis 2009; 205(1): 255-60.

[10] Kendricka J, Targherb G, Smitsa G, Chonchola M. 25-Hydroxyvitamin D deficiency is independently associated with cardiovascular disease in the Third National Health and Nutrition Examination Survey. Athero-sclerosis. 2009;205:255–60. [PubMed]

[11] Nibbelink KA, Tishkoff DX, Hershey SD, Rahman A, Simpson RU.1,25(OH)2-vitamin D3 actions on cell proliferation, size, gene expression, and receptor localization, in the HL-1 cardiac myocyte. J Steroid Biochem Mol Biol Mar. 2007;103:533–7.

[12] Y. Arnson, D. Itzhaky, M. Mosseri *et al*., "Vitamin D inflammatory cytokines and coronary events: a comprehensive review," *Clinical Reviews in Allergy and Immunology*, vol. 45, no. 2, pp. 236–247, 2013.

[13] C. Robinson-Cohen, A. N. Hoofnagle, J. H. Ix *et al*., "Racial differences in the association of serum 25-hydroxyvitamin D

concentration with coronary heart disease events," *Journal of the American Medical Association*, vol. 310, no. 2, pp. 179–188, 2013.

[14] J. H. Lee, R. Gadi, J. A. Spertus, F. Tang, and J. H. O'Keefe, "Prevalence of vitamin D deficiency in patients with acute myocardial infarction," *The American Journal of Cardiology*, vol. 107, no. 11, pp. 1636–1638, 2011.

[15] B. Goleniewska, M. Kacprzak, and M. Zieli'nska, "Vitamin D level and extent of coronary stenotic lesions in patients with first acute myocardial infarction," *Cardiology Journal*, vol. 21, no. 1, pp. 18–23, 2014.

[16] R. Chowdhury, S. Kunutsor, A. Vitezova *et al*., "Vitamin D and risk of cause specific death: systematic review and meta-analysis of observational cohort and randomised intervention studies," *British Medical Journal*, vol. 348, Article IDg1903, 2014.

[17] S. Pilz, R. M. A. Henry, M. B. Snijder *et al*., "Vitamin D deficiency and myocardial structure and function in older men and women: the Hoorn study," *Journal of Endocrinological Investigation*, vol. 33, no. 9, pp. 612–617, 2010.

[18] M. Sobieszcz'anska, J. Jonkisz, M. Tabin, and K. Laszki- Szcza,chor, "Osteoporosis: genetic determinants and relationship with cardiovascular disease," *Advances in Clinical and Experimental Medicine*, vol. 22, no. 1, pp. 119–124, 2013