ORIGINAL RESEARCH PAPER
Volume - 9 | Issue - 12 | December - 2020 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

# EFFECT OF SERUM VITAMIN D LEVEL IN PATIENTS WITH CORONARY ARTERY DISEASES IN A TERTIARY CARE HOSPITAL- MIDNAPORE MEDICAL COLLEGE AND HOSPITAL

Biochemistry

**Dr.Brahmarshi Das** Associate Professor, Department of Biochemistry, Midnapore Medical College and Hospital, Paschim Medinipur, Pin - 721101.

**Dr.Narendra Nath Hait*** Assistant Professor, Department of Gynaecology and Obstetrics, Midnapore Medical College and Hospital, Paschim Medinipur, Pin - 721101. *Corresponding Author

**Dr. Sayantan Dasgupta** Associate Professor, Dept of Biochemistry, North Bengal Medical College.

**Dr. Debarshi Jana** PhD, Young Scientist (DST), IPGMER and SSKM Hospital, Kolkata.

## ABSTRACT

The aim of study is to elucidate the status of vitamin D level in adult patients with cardiovascular disease. Measure serum 25 (OH) Vitamin D level in adult patients diagnosed with cardio vascular disease. Lipid profile level in diagnosed patients with cardio vascular disease. Blood glucose level in diagnosed patients with cardiovascular disease. Assess those patients with ECG parameters. Compare the level of serum 25 (OH) vitamin D level in adult patients of cardio vascular disease with respect to age and sex matched apparently healthy controls.

Observational, non-interventional, hospital based cross sectional study and case control studies were done for clinical correction. Patients with diagnosed Cardiovascular Disease were selected from OPD and IPD of the department of Medicine, Midnapore Medical College and Hospital, Paschim Medinipur and analysis of biochemical parameters were done in the Department of Biochemistry, Midnapore Medical College and Hospital, Paschim Medinipur.

In Control, the mean Vitamin D (mean± S.D.) of patients was 40.3500± 1.9456. The mean Vitamin D was significantly higher in cases compared to control which was statistically significant. We concluded that serum vitamin D level in patients was significantly correlated with coronary artery diseases in a tertiary care hospital.

## KEYWORDS

Effect, Serum Vitamin D Level, Coronary Artery Diseases

## INTRODUCTION

Vitamin D is considered a steroid hormone with a broad spectrum of action in the human body. Its action arises from the binding of its active metabolite (la25 - dihydroxyvitamin D) to its receptor (Vitamin D Receptor), which is present throughout the body, including vascular smooth muscle cells and cardiomyocytes. Initially, vitamin D deficiency was related only to changes in the musculoskeletal system, but in recent years, researchers have demonstrated its relationship with several pathology related to other systems, such as cardiovascular disease. This study trying review is vitamin D's pathophysiology; describe its relationship with cardiovascular diseases based on the most recent publications.[1]

The role of vitamin D in the regulation of bone metabolism has been well established. However, in recent years, many studies have demonstrated that its role extends far beyond bone health. Growing evidence has shown a strong association between vitamin D deficiency and hypertension, metabolic syndrome, diabetes mellitus and atherosclerosis. The mechanisms by which Vitamin D exerts its cardiovascular protective effects are still not completely understood, but there is evidence that it participate in the regulation of rennin-angiotensin system and the mechanisms of insulin sensitivity and activity of inflammatory cytokines, besides its direct cardiovascular actions. In this review, several studies like vitamin D deficiency with cardiometabolic risk as well as small randomized trials that have evaluated the cardiovascular effects as are presented.[2] Cardiovascular disease (CVD) is the leading cause of morbidity and mortality in the world. Although the role of traditional risk factors is already consolidated, it is known that they cannot fully explain the development of CVD, which has caused continuous search for new risk factors. Growing evidence, obtained in recent years, has suggested that vitamin D deficiency may be associated with an increased risk of CVD. Vitamin D is actually a steroid hormone primary function is the regulation of calcium and phosphorus homeostasis, through its interaction with parathyroid gland, the kidneys and intestines. Although it can be obtained through food intake, the main source of vitamin D is represented by its systems in the body itself.[3]

Vitamin D, the fourth vitamin to be described, was initially characterized as a factor capable of curing rickets, a disease characterized by bone demineralization and skeletal deformities. Currently, vitamin D comprises a group of secosteroid molecules derived from 7-dehydrocholesterol (7-DHC) that includes the active metabolite (la, 25-dihydroxyvitamin D or calcitriol), its precursors (cholecalciferol or vitamin D3, ergocalciferol or vitamin D2, and 25-hydroxyvitamin D or calcidiol), as well as its degradation products. These molecules, along with their carrier proteins and receptors, comprise an important metabolic axis; the endocrine vitamin D system.[4]

The aim of study is to elucidate the status of vitamin D level in adult patients with cardiovascular disease.

## METHODOLOGY

### PLACE OF STUDY

Department of Biochemistry Midnapore Medical College and Hospital, Paschim Medinipur.

### PERIOD OF STUDY

January 2019 to June 2020

### SAMPLE SIZE / DESIGN

A hundred patients with diagnosed cardio vascular disease and hundred numbers of age and sex matched apparently healthy controls are taken for the study. All groups were assessed for the serum Vitamin D and lipid profile, Sodium, Potassium.

A minimum of hundred age and sex matched apparently healthy controls were taken.

### a) INCLUSION CRITERIA

Age and sex matched apparently healthy individual, who have agreed to sign the informed consent form, (have no history of Cardiovascular Disease) were selected as control.

### i) EXCLUSIONG CRITERIA

ii) The patients who are bed ridden for a long time.
iii) Chronic smokers, alcoholics and diabetics.
iv) Patients with chronic protracted illness.
v) Patients with history of gastrointestinal diseases (crohn's disease, celiac disease, overgrowth syndrome), known to influence serum Vitamin-D level.
vi) Patients on antioxidants, (Vitamin-D Tablets, multivitamin medications which may affect the study parameters),

vii) Pregnancy, lactation
viii) Drugs affecting Vit-D level.

## RESULT AND ANALYSIS

Our study showed that in Case, 15(15.0%) patients were 21-30years old, 40(40.0%) patients were 31-40 years old, 34(34.0%) patients were 41-50 years old and 11(11.0%) patients were 51-60 years old. In Control, 16(16.0%) patients were 21-30years old, 63(63.0%) patients were 31-40 years old, 11(11.0%) patients were 41-50 years old and 10(10.0%) patients were 51-60 years old. Association of Age in years vs group was not statistically significant ( p=0.7344). In Case, 50(50.0%) patients were Female and 50(50.0%) patients were Male. In Control, 48(48.0%) patients were Female and 52(52.0%) patients were Male. Association of Sexvs group was not statistically significant (p=0.7772). In Case, 50(50.0%) patients were Hindu and 50(50.0%) patients were Muslim. In Control, 48(48.0%) patients were Hindu and 52(52.0%) patients were Muslim. Association of Religion vs group was not statistically significant (p=0.0372).

We found that in Case, the mean Age (mean± s.d.) of patients was 39.6300 ± 8.1236. In Control, the mean Age (mean± s.d.) of patients was 39.5600 ± 7.7320. Difference of mean Age with both Group was statistically significant (p=0.9503). In Case, the mean Total Cholesterol (mean± s.d.) of patients was 136.3400± 10.1953. In Control, the mean Total Cholesterol (mean± s.d.) of patients was 103.9400± 7.0622. Difference of mean Total Cholesterol with both Group was statistically significant (p<0.0001). In Case, the mean HDL (mean± s.d.) of patients was 42.5500± 2.9657. In Control, the mean HDL (mean± s.d.) of patients was 50.0100 ± 7.2244. Difference of mean HDL with both Group was statistically significant (p<0.0001). In Case, the mean LDL (mean± s.d.) of patients was 71.7800± 8.6077. In Control, the mean LDL (mean± s.d.) of patients was 50.4700± 4.6720. Difference of mean LDL with both Group was statistically significant (p<0.0001).

We showed that in Case, the mean VLDL (mean± s.d.) of patients was 22.2000± 3.0218. In Control, the mean VLDL (mean± s.d.) of patients was 10.7900± 2.4300. Difference of mean VLDL with both Group was statistically significant (p<0.0001). In Case, the mean TG (mean± s.d.) of patients was 112.5100± 16.5019. In Control, the mean TG (mean± s.d.) of patients was 53.8000± 12.1398. Difference of mean TG with both Group was statistically significant (p<0.0001). In Case, the mean Vitamin D (mean± s.d.) of patients was 16.9400± 2.3042. In Control, the mean Vitamin D (mean± s.d.) of patients was 40.3500± 1.9456. Difference of mean Vitamin D with both Group was statistically significant (p<0.0001).

## DISCUSSION

We found that in Case, the mean Age (mean± S.D.) of patients was 39.6300 ± 8.1236. In Control, the mean Age (mean± S.D.) of patients was 39.5600 ± 7.7320. Difference of mean Age with both Group was statistically significant (p=0.9503). In Case, 15(15.0%) patients were 21-30years old, 40(40.0%) patients were 31-40 years old, 34(34.0%) patients were 41-50 years old and 11(11.0%) patients were 51-60 years old. In Control, 16(16.0%) patients were 21-30years old, 63(63.0%) patients were 31-40 years old, 11(11.0%) patients were 41-50 years old and 10(10.0%) patients were 51-60 years old. Association of Age in years vs group was not statistically significant (p=0.7344).

Vacek JL et al [5](2012) found that the mean age was 58 ± 15 years, 71% were women (n = 7,758), and the average body mass index was 30 ± 8 kg/m2. The mean serum vitamin D level was 24.1 ± 13.6 ng/ml. Of the 10,899 patients, 3,294 (29.7%) were in the normal vitamin D range and 7,665 (70.3%) were deficient.

Anderson JL et al [6](2010) found that Vitamin D deficiency was associated with highly significant (p <0.0001) increases in the prevalence of diabetes, hypertension, hyperlipidemia, and peripheral vascular disease. Also, those without risk factors but with severe deficiency had an increased likelihood of developing diabetes, hypertension, and hyperlipidemia. The vitamin D levels were also highly associated with coronary artery disease, myocardial infarction, heart failure, and stroke (all p <0.0001), as well as with incident death, heart failure, coronary artery disease/myocardial infarction (all p <0.0001), stroke (p = 0.003), and their composite (p <0.0001). In conclusion, they have confirmed a high prevalence of vitamin D deficiency in the general healthcare population and an association between vitamin D levels and prevalent and incident CV risk factors and outcomes.

C Brewer L et al [7](2011) found that vitamin D is important in the etiology of atherosclerosis, it is unclear at what stage(s) in the atherosclerotic disease process vitamin D may exert its effects. Large-scale, well-conducted, placebo controlled clinical trials testing the efficacy of vitamin D supplementation in delaying, slowing, or reverting the atherosclerotic disease process have not yet been conducted. Until the results of these studies are available, we believe it is premature to recommend vitamin D as a therapeutic option in atherosclerosis.

McGreevy C et al [8](2011) found that prevalence of vitamin D deficiency, and discusses recent evidence for the association between hypovitaminosis D and cardiovascular disease. Few randomized, controlled trials have evaluated the effect of vitamin D replacement on cardiovascular outcomes, and the results have been inconclusive or contradictory. Carefully designed randomized, controlled trials are essential to evaluate the role of vitamin D supplementation in reducing cardiovascular disease.

Pilz S et al [9](2016) found that vitamin D supplementation with the commonly used doses, and whether vitamin D has cardiovascular effects in individuals with overt vitamin D deficiency remains to be evaluated. Here, they provide an update on clinical studies on vitamin D and cardiovascular risk, discuss ongoing vitamin D research, and consider the management of vitamin D deficiency from a cardiovascular health perspective.

Barreto DV et al [10](2009) found that Vitamin D deficiency and insufficiency were highly prevalent in this CKD cohort. Low 25D levels affected mortality independently of vascular calcification and stiffness, suggesting that 25D may influence survival in CKD patients via additional pathways that need to be further explored.

Norman PE et al [11](2014) found that the role of vitamin D supplementation in the management of cardiovascular disease remains to be established. This review summarizes the clinical studies showing associations between vitamin D status and cardiovascular disease and the experimental studies that explore the mechanistic basis for these associations.

Michos ED et al [12](2008) found that vitamin D deficiency may be a contributor to the development of cardiovascular disease potentially through associations with diabetes or hypertension.

Levin A et al [13](2005) found that Vitamin D deficiency might be an underestimated nonclassical risk factor for cardiovascular disease in CKD. Based on a review of the evidence, from both basic science and clinical studies, this article supports the possible protective role of vitamin D beyond its effect on mineral metabolism, and suggests the need for ongoing evaluation of the role of vitamin D in cardiovascular health in the CKD population.

Judd S et al [14](2008) found that vitamin D deficiency as a cardiovascular risk factor and to explore potential mechanisms for the cardio-protective effect of vitamin D.

Poole KE et al [15](2006) found that vitamin D was identified in the majority of patients with acute stroke throughout the year and may have preceded stroke. Vitamin D is a potential risk marker for stroke, and the role of vitamin D repletion in enhancing musculoskeletal health after stroke needs to be explored.

## SUMMARY AND CONCLUSION

We found that the mean Total Cholesterol was more in case compared to control which was statistically significant. It was found that the mean HDL was less in case compared to control which was statistically significant. Our study found that the mean LDL was more in case compared to control which was statistically significant. We found that the mean TG was more in case compared to control which was statistically significant.

It was found that in Case, the mean Vitamin D (mean± S.D.) of patients was 16.9400± 2.3042.

In Control, the mean Vitamin D (mean± S.D.) of patients was 40.3500± 1.9456. The mean Vitamin D was significantly higher in cases compared to control which was statistically significant. We concluded

that serum vitamin D level in patients was significantly correlated with coronary artery diseases in a tertiary care hospital.

**Table: Distribution of mean Vitamin D: Group**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Vitamin D** | Case | 100 | 16.9400 | 2.3042 | 12.0000 | 21.0000 | 17.0000 | <0.001 |
| | Control | 100 | 40.3500 | 1.9456 | 37.0000 | 46.0000 | 40.0000 | |

## REFERENCE


1. Mendis S, Puska P, Norrving B (2011). Global Atlas on Cardiovascular Disease Prevention and Control (PDF). pp. 3–18. ISBN 978-92-4-156437-3. Archived (PDF) from the original on 2014-08-17.
2. Naghavi M, Wang H, Lozano R, Davis A, Liang X, Zhou M, et al. (GBD 2013 Mortality and Causes of Death Collaborators) (January 2015). 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013". Lancet. 385 (9963): 117–71.
3. McGill HC, McMahan CA, Gidding SS (March 2008). "Preventing heart disease in the 21st century: implications of the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study". Circulation. 117 (9): 1216–27.
4. O'Donnell MJ, Chin SL, Rangarajan S, Xavier D, Liu L, Zhang H, et al. (August 2016). "Global and regional effects of potentially modifiable risk factors associated with acute stroke in 32 countries (INTERSTROKE): a case-control study". Lancet. 388 (10046): 761–75. doi:10.1016/S0140-6736(16)30506-2. PMID 27431356. S2CID 39752176.
5. Vacek JL, Vanga SR, Good M, Lai SM, Lakkireddy D, Howard PA. Vitamin D deficiency and supplementation and relation to cardiovascular health. The American journal of cardiology. 2012 Feb 1;109(3):359-63.
6. Anderson JL, May HT, Horne BD, Bair TL, Hall NL, Carlquist JF, Lappé DL, Muhlestein JB, Group IH. Relation of vitamin D deficiency to cardiovascular risk factors, disease status, and incident events in a general healthcare population. The American journal of cardiology. 2010 Oct 1;106(7):963-8.
7. C Brewer L, D Michos E, P Reis J. Vitamin D in atherosclerosis, vascular disease, and endothelial function. Current drug targets. 2011 Jan 1;12(1):54-60.
8. McGreevy C, Williams D. New insights about vitamin D and cardiovascular disease: a narrative review. Annals of internal medicine. 2011 Dec 20;155(12):820-6.
9. Pilz S, Verheyen N, Grübler MR, Tomaschitz A, März W. Vitamin D and cardiovascular disease prevention. Nature Reviews Cardiology. 2016 Jul;13(7):404.
10. Barreto DV, Barreto FC, Liabeuf S, Temmar M, Boitte F, Choukroun G, Fournier A, Massy ZA. Vitamin D affects survival independently of vascular calcification in chronic kidney disease. Clinical journal of the American Society of Nephrology. 2009 Jun 1;4(6):1128-35.
11. Norman PE, Powell JT. Vitamin D and cardiovascular disease. Circulation research. 2014 Jan 17;114(2):379-93.
12. Michos ED, Melamed ML. Vitamin D and cardiovascular disease risk. Current Opinion in Clinical Nutrition & Metabolic Care. 2008 Jan 1;11(1):7-12.
13. Levin A, Li YC. Vitamin D and its analogues: do they protect against cardiovascular disease in patients with kidney disease?. Kidney international. 2005 Nov 1;68(5):1973-81.
14. Judd S, Tangpricha V. Vitamin D deficiency and risk for cardiovascular disease. Circulation. 2008 Jan 29;117(4):503.
15. Poole KE, Loveridge N, Barker PJ, Halsall DJ, Rose C, Reeve J, Warburton EA. Reduced vitamin D in acute stroke. Stroke. 2006 Jan 1;37(1):243-5.