Research article

Does serum vitamin D affect lipid profile?

Monalisa Biswas, Shaik Allabhakshu, Krishnananda Prabhu

Department of Biochemistry, Kasturba Medical College, Manipal, Manipal Academy of Higher Education, Manipal, 576104, Karnataka, India

(Received: August 2021

Revised: November 2021

Accepted: December 2021)

Corresponding author: **Krishnananda Prabhu**. Email: krishnananda.prabhu@manipal.edu

ABSTRACT

Introduction and Aim: Vitamin D, besides its classical physiological functions, exerts effects on brain, prostate, breast, colon, pancreas, and immune cells due to the ubiquitous presence of its receptors. Hypovitaminosis D predisposes individuals to various micro and macro vascular complications of metabolic syndrome and diabetes mellitus. Despite speculations regarding implications of its deficiency on the cardio metabolic health of general/susceptible population, its role remains underexplored in the Indian population.

Materials and Methods: This cross-sectional study carried out in Biochemistry Department of the institution, recruited 219 patients (after obtaining written consent) of both genders above 18 years who underwent Vitamin D testing. Blood samples obtained were assayed for HbA1c, C Reactive protein, thiol and lipid. Kruskal Wallis test and Spearman correlation were employed for statistical analysis.

Results: Significantly low HDL and high atherogenic index of plasma (AIP) were observed in males with vitamin D deficiency. Significant inverse correlation of vitamin D with CRP and total cholesterol (among vitamin D deficient males) was observed. A significant inverse correlation between serum HDL and AIP and a significant direct correlation between triglyceride and AIP were observed irrespective of their vitamin D status. Heat map showed marginally elevated lipid parameters among vitamin D insufficient males.

Conclusion: Vitamin D may emerge as a surrogate marker in risk stratification of patients with diabetes and dyslipidaemia. More insights are required to assess the gender specific susceptibility to dyslipidaemia and atherosclerosis in relation to vitamin D levels.

Keywords: Atherosclerosis; dyslipidaemia; lipid metabolism; vitamin D; vitamin D deficiency.

INTRODUCTION

Vitamin D, besides its classical physiological functions, exerts its effects on brain, prostate, breast, colon, pancreas, and immune cells due to the ubiquitous presence of its receptors (1-3). Despite being located near the equatorial region; India is endemic to vitamin D deficiency with an estimated 50% of the population (or more) manifesting a varying spectrum of vitamin D insufficiency (2). Evidence suggests vitamin D deficiency might lead to beta cell dysfunction and progressive insulin resistance, increasing the susceptibility to the develop metabolic syndrome along with increase in glycated haemoglobin (1). These series of effects triggered by hypovitaminosis D predisposes individuals to various micro and macro vascular complications of metabolic syndrome and diabetes mellitus (1).

Despite being located near the equatorial region, vitamin D deficiency has assumed an epidemic the in India (an approximate 50–90% deficient population) due to the characteristic skin pigmentation, which mandates Indian subcontinent inhabitants to acquire double UVB exposure than white Europeans (3-5). India reports a high prevalence of vitamin D deficiency among all ages, genders, varying socio-economic groups, urban and rural populations,

pregnant and post-menopausal women, and healthy young adults (5-7).

Accumulating evidence from invivo as well and human research suggest hypovitaminosis D may facilitate beta cell dysfunction (8-11). Vitamin D deficiency is being enormously explored in the pathogenesis of dyslipidaemia with observational reports claiming deficiencies to be associated with perturbations in lipid profile (high triglycerides, low HDL) along with insulin resistance (2). Despite all these promising data from observational studies, interventional studies (RCTs) could not confirm the protective effect of vitamin D correction in dyslipidaemias (2). Vitamin D influences androgen synthesis, ovarian androgens may exert variable effects on glucose and lipid metabolism between genders (12). Literature evidence has claimed that female with lower tertiles of vitamin D have shown association with an increased prevalence and severity of CAD (13). A study has claimed that serum 25(OH)D levels and serum lipids associations were more pronounced in males than in females (11).

Despite the increased prevalence of metabolic syndromes, cardiovascular, ischemic heart disease and vitamin D deficiency in India, conclusive evidence of its contributory effect on cardiometabolic factors is indeed scarce (2). Hence, this study investigates the

correlation of known cardiometabolic risk factors like the antioxidant and inflammatory biomarkers (protein thiol, CRP, lipid profile, glycated haemoglobin) with the circulating vitamin D levels.

MATERIALS AND METHODS

This cross-sectional study was conducted by the Department of Biochemistry during December 2017 to June 2018. Ethical approval was obtained from the Institutional Review Board (IRB) vide Letter No. IEC No 882/2017 dated 13th December 2017 and informed written consent was taken from all participants.

Two hundred and nineteen subjects more than 18 years of age belonging to both the genders who visited Kasturba Hospital, Manipal and underwent vitamin D testing during the study period were included. The sample cohort was classified into three groups based on the serum vitamin D with levels above 30 ng/mL classified as sufficient (group-1, n=51), between 20 ng/mL to 30 ng/mL classified insufficient (group-2, n=60) and below 20 ng/mL classified as deficient (group-3, n=108). Paediatric patients (below 18 years) were excluded from the study.

Freshly obtained serum and plasma (for glycated Hb) samples were analysed in the Clinical Biochemistry Laboratory. Serum CRP was analysed by particle enhanced immunoturbidimetric assay (Roche cobas 8000), serum 25(OH) D levels by ECLIA (Electro chemiluminescence Assay: Competition principle, Roche cobas 8000), glycated haemoglobin by TINIA (Turbid metric inhibition immunoassay, Roche cobas 6000) and lipid profile was measured colorimetric enzymatic method (Roche cobas 8000). Residual

samples were aliquoted and preserved at -20°C till further bulk analysis of protein thiol by Dinitrobenzene (DTNB)- Ellman's spectrophotometric method.

Kruskal Wallis test was used to evaluate differences between the groups. P-value less than 0.05 is considered. significant. Spearman's rank correlation coefficient test was used to ascertain with relationships between target analytes/ biomarkers.

RESULTS

Out of the total two hundred and nineteen participants recruited in the study, 41.6% (n=91) were males and 58.4% (n=128) were females. Fifty one participants had sufficient levels of circulating vitamin D, while 60 participants showed insufficiency and 108 participants were deficient in vitamin D. Vitamin D was deficient among 45% of participants of both genders. 24% of the males had sufficient vitamin D and 31% showed insufficiency. Among females, vitamin D sufficiency was observed among 34% and 21% showed insufficiency. There was no significant difference in the age distribution of participants between the three groups.

Table 1 shows the comparison of target analytes among the six groups, HDL and AIP showed significant alteration in Vitamin D deficient males when compared to other groups.

Further, gender wise stratification of vitamin D sufficiency and its correlation with the target analytes was carried out (Table 2). Glycated haemoglobin and CRP showed moderate positive significant correlation in vitamin D deficient males.

Table 1: Comparison of biochemical parameters between the three groups (Original)

Biochemical Parameters	S.M. N=23	S.F. N= 51	I.M. N=28	I.F. N=31	D.M. N=41	D.F. N=67	KW	P* value
HbA1c (g%)	6.30 (5.5, 7.0)	6.3 (5.5, 7.3)	6.0 (5.5, 6.4)	5.6 (5.4, 6.7)	5.8 (5.4, 6.9)	5.6 (5.3, 6.1)	10.72	0.057
CRP (mg/L)	1.57 (0.58, 3.28)	1.8 (0.80, 4.41)	2.3 (0.81, 5.34)	1.34 (0.7, 3.39)	1.81 (0.63, 3.75)	2.34 (1.03, 5.5)	2.77	0.735
Thiol (µg/L)	602 (541, 672)	589 (522, 677)	596 (529, 737)	578 (463, 646)	586 (517, 695)	569 (502, 646)	2.12	0.831
Total Cholesterol (mg/dl)	171 (150, 198)	176 (148, 200)	170 (150, 222)	184 (150, 216)	143 (135, 191)	175 (147, 204)	6.77	0.238
Triglyceride (mg/dl)	111 (97.5, 195)	113 (90, 154)	130 (99, 196)	102 (84, 144)	145 (86, 194)	106 (67, 138)	10.08	0.073
HDL (mg/dl)	41 (33.7, 52)	45 (39, 55)	42 (32, 51)	48 (41, 60)	40 (33, 43)	50 (39, 57)	24.79	0.0002
LDL (mg/dl)	101 (78.5, 125.8)	89 (77, 116)	103 (86, 139)	108 (79, 143)	85 (65, 118)	100 (84, 130)	9.58	0.088
AIP	0.455 (0.27, 0.73)	0.389 (0.20, 0.59)	0.53 (0.38, 0.67)	0.362 (0.2, 0.53)	0.572 (0.31, 0.77)	0.323 (0.05, 0.57)	21.95	0.0005

* Kruskal Wallis test. (post hoc Dunn's Multiple Comparison Test)

Abbreviations: S.M. (Sufficient Males), S.F. (Sufficient Females), I.M. (Insufficient Males), I.F. (Insufficient Females), D.M. (Deficient Males), D.F. (Deficient Females), KW (Kruskal Wallis statistic).

Table 2: Gender wise correlation of variables with vitamin D (Original)

Biochemical parameters	Sufficient Males		Sufficient Females		Insufficient Males		Insufficient Females		Deficient Males		Deficient Females	
	R	P*	R	P*	R	P*	R	P*	R	P*	R	P*
Glycated Hb	-0.36	0.09	-0.06	0.65	-0.07	0.70	0.06	0.75	-0.31	0.04	-0.02	0.84
CRP	0.02	0.93	0.11	0.45	-0.08	0.68	0.16	0.37	-0.45	0.01	0.193	0.11
Thiol	0.03	0.88	-0.08	0.56	0.33	0.08	0.06	0.74	0.05	0.76	0.016	0.89
Total Cholesterol	-0.29	0.18	-0.11	0.46	0.08	0.65	0.09	0.60	0.13	0.42	0.171	0.16
Triglyceride	-0.03	0.89	0.12	0.40	0.12	0.52	0.08	0.68	-0.07	0.66	0.175	0.15
HDL	-0.04	0.87	-0.07	0.62	-0.01	0.97	0.10	0.58	0.20	0.21	0.067	0.58
LDL	-0.22	0.33	-0.08	0.55	0.13	0.51	0.03	0.88	0.129	0.41	0.165	0.18
AIP	0.02	0.93	0.15	0.29	0.11	0.59	0.03	0.88	-0.14	0.37	0.116	0.34

*Spearman correlation

Fig. 1 shows the percentage of participants showing altered/ abnormal lipid profile across the three groups.

Fig. 1: Gender wise comparison of percentage subjects showing elevated biomarkers in each group (based on vitamin D status) (Original). Abbreviations: CRP – C Reactive protein; TC – Total cholesterol; Tgl – Triglyceride; HDL – High Density Lipoprotein; LDL – Low Density Lipoprotein.

Fig. 2 is a heat map of assayed markers between various groups. This clearly shows an increased level of lipid profile perturbations in males having insufficient vitamin D as compared to other groups, with serum triglyceride levels systematically higher in

males with insufficient and deficient vitamin D levels when compared to males of the sufficient group as well as females of all groups.

Table 3 outlines the overall and gender wise correlation of triglyceride and HDL with AIP.

Biomarker	Sufficient Males	Sufficient Females	Insufficient Males	Insufficient Females	Deficient Males	Deficient Females
Glycated Hb	6.3	6.3	6	5.6	5.8	5.6
CRP	1.57	1.8	2.3	1.37	1.81	2.34
Thiol	602	589	596	574	586	569
Total Cholesterol	171	176	170	182	143	175
Triglyceride	111	113	130	102	145	106
HDL	41	45	42	46	40	50
LDL	101	89	102	106	84	100
AIP	0.45	0.38	0.53	0.35	0.57	0.32

Fig. 2: Heat Map (Original). Abbreviations: AIP – Atherogenic Index of Plasma**Table 3:** Correlation between biochemical parameters & AIP (Original)

Biochemical markers		Spearman rho value	P value
Triglyceride & AIP	Overall	0.89	3.162e - 77
	Males	0.83	7.8e - 25
	Females	0.90	8.87e - 56
HDL & AIP	Overall	-0.72	1.16e - 35
	Males	-0.69	1.3e - 14
	Females	-0.71	1.44e - 23

DISCUSSION

The global expression of vitamin D receptor in human body emphasizes on its myriad roles in chronic diseases (14, 15). The high prevalence of its deficiency, its contributory role in metabolic and cardiovascular diseases along with the scarcity of conclusive Indian data have intrigued us to revisit the association of vitamin D with cardiometabolic indices. Our study shows a statistically significant decreased HDL and increased AIP values (potent indicators & risk markers of cardiovascular disease) in males with vitamin D deficiency (Table 1). We also observed a marginal elevation in CRP and triglycerides in males with deficient levels of vitamin D, triglycerides and HDL showed elevations in males across all the three groups when compared to females, while total cholesterol was found to be lower among males and LDL was lower in males of insufficient and deficient group when compared to females, although these differences were not statistically significant which could be owing to the sample size and the lipid lowering therapeutic interventions which might be used by some of the participants. Vitamin D deficient females showed an opposite trend (for CRP) as that of deficient males indicating the pathophysiological differences for atherosclerosis across genders. Marginally higher CRP levels were observed among vitamin D deficient females (Table 1). A strong positive correlation was observed between triglyceride and AIP and a strong negative correlation was observed between serum HDL and AIP (Table 3). Further, heat map (Fig. 3) showed marginally elevated lipid parameters in vitamin D insufficient males, thus this group might be predisposed or having increased susceptibility to inflammation and atherosclerosis.

These observations clearly indicate an increased level of lipid profile perturbations in males having insufficient/ deficient vitamin D as compared to other groups, which elucidates the possible mechanistic role of vitamin D in atherogenesis. It also emphasizes the vulnerability of vitamin D deficient males to manifest a significantly unfavourable cardiometabolic risk of initiation and progression of atherogenic changes. Previous literature evidence has shown increased prevalence vitamin D deficiency in patients with type 2 diabetes mellitus as it plays a role in insulin secretion and action (16-19). Evidence suggest that vitamin D might inhibit triglyceride synthesis and secretion by increasing intestinal calcium absorption

(1, 2). An animal-based study reported that calcitriol treatment (at above physiological concentrations) resulted in reduced hepatic triglyceride accumulation (20). Though not through supplementation, our study clearly shows that males with vitamin D deficiency have a significantly unfavourable cardiometabolic risk profile and vitamin D supplementation could probably be effective in reversing this. It is important to note that acetyl CoA is the precursor for both fatty acid and cholesterol synthesis. A review by Dibaba *et al.*, claims the vitamin D supplementation exerts a beneficial effect on circulating levels of total cholesterol, LDL and triglyceride levels (21). However, in contrast to the above a meta-analysis of 12 RCTs showed a statistically significant adverse effect of vitamin supplementation on LDL levels especially in obese subject (22). The present study also failed to report any significant differences in serum LDL levels across all groups irrespective of their vitamin D status and gender.

Vitamin D acting like a steroid hormone, modulates the expression of various matrix metalloproteinase (MMPs), growth factors and cytokines involved in the inflammatory response thus regulating endothelial function (23). The vitamin D receptor (through which calcitriol achieves its functions) was believed to be exclusively endocrine, coupling with retinoic X receptor to form a heterodimer, subsequently binding to response elements in the promoter regions of target genes and exerting effects related to mineral metabolism (24). Recent research has shown that the vitamin D receptor is expressed in tissues of multiple origin which could explain its varying effects such as immunomodulation, cell proliferation and differentiation (25,26). Further, a cytoplasmic form of the vitamin D receptor has been identified that responds rapidly or non-genomically to calcitriol and is thought to be involved in cellular calcium handling and other processes (24,27).

It is important to identify the role of vitamin D deficiency in stimulation of inflammatory responses and in progression of atherosclerosis. The association between vitamin D and cardiovascular disease could be explained by a lipid-lowering effect of vitamin D. This has been substantiated in several cross-sectional studies among subjects with high serum vitamin D levels showing favourable serum lipid profiles (15). Vitamin D induced inhibitions in vascular wall inflammation (through down regulation of

interleukins IL-1, IL-6 and TNF- α) could possibly exert a cardio-protective effects (15). The expression of Vitamin D receptors in cardiac muscles, endothelial cells and vascular smooth muscle strongly reinforces the association of vitamin D perturbations in vascular and cardiac physiology (1,28). The negative association of serum lipids with circulating vitamin D could be possibly explained due to the decreased intestinal absorption, reduced endogenous lipid synthesis and increased lipolytic effects exerted by vitamin D (29). These evidences indicate that vitamin D deficient males have a higher predisposition for CAD/atherosclerosis/inflammation.

As vitamin D is known to influence androgen synthesis, there may be some gender specific variations in the effects of vitamin D deficiency on the various cardio metabolic risk factors (2, 30). Evidence have claimed that the relationship of cholecalciferol concentration on serum lipids/ lipoproteins is appreciably striking and marked in males when compared to females (1). This is reiterated in this present study with only vitamin D deficient males showing unfavourable triglycerides and AIP levels in comparison to females across all groups. This could be due to life style differences such as smoking, alcohol consumption, sun exposure and physical activity among males (1). Further we show a strong positive correlation among triglyceride and AIP and a strong negative correlation among serum HDL and AIP indicating atherogenic potentials of perturbed lipid profile (Table 3).

The study is limited by the cross-sectional study design and a retrospective model where concrete evidence of causality cannot be established. Further, influence of ongoing medications of circulating levels of lipoproteins has not been accounted for in this study. A large prospective cohort confounder adjusted study design with a long follow up period would be ideal to establish causal associations.

CONCLUSION

Based on the present study and existing scientific evidence of coexisting vitamin D deficiency in hyperlipidaemia, metabolic syndromes and coronary artery diseases and the potential role of vitamin D as an anti-inflammatory, hypoglycaemic and hypolipidemic agent in observational studies, vitamin D may emerge as a surrogate marker in risk stratification of patients with diabetes and dyslipidaemia. Evaluation/ correction of vitamin D deficiency might emerge as a standard of care/ good clinical practice in routine health assessment modules.

ACKNOWLEDGEMENT

The authors acknowledge Manipal Academy of Higher Education for providing a congenial and supportive platform for carrying out the research study. We also thank the University Grants Commission and the Council for Scientific and

Industrial Research for supporting our PhD scholar through their fellowship and contingency support.

CONFLICT OF INTEREST

The authors state that they have no conflicts of interest with regard to the present study.

REFERENCES

- Wang, Y., Si, S., Liu, J., Wang, Z., Jia, H., Feng, K., *et al.*, The Associations of Serum Lipids with Vitamin D Status. *PLoS ONE*. 2016; 11(10): e0165157.
- Verena Schwetz V, Schamagl H, Trummer C, Stojakovic T, Pandis M, Gröbler M. R., *et al.*, Vitamin D supplementation and lipoprotein metabolism: A randomized controlled trial. *Journal of Clinical Lipidology*. 2018; 12(3): 588-596.
- Gupta, G. R. A. Vitamin D deficiency in India: prevalence, causalities and interventions. *Nutrients*. 2014; 6(2): 729-775.
- Harinarayan, C. V., Holick, M. F., Prasad, U. V., Vani, P. S., Himabindu, G. Vitamin D status and sun exposure in India. *Dermatoendocrinol*. 2013; 5(1): 130-141.
- Aparna, P., Muthathal, S., Nongkynrih, B., Gupta, S. K. Vitamin D deficiency in India. *J Family Med Prim Care*. 2018; 7(2): 324-330.
- Kamboj, P., Dwivedi, S., Toteja, G. S. Prevalence of hypovitaminosis D in India & way forward. *Indian J Med Res*. 2018; 148: 548-556.
- Garg, R., Agarwal, V., Agarwal, P., Singh, S., Malhotra, N. Prevalence of vitamin D deficiency in Indian women. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*. 2018; 7(6): 2222-2225.
- Szymczak-Pajor, I., Śliwińska, A. Analysis of Association between Vitamin D Deficiency and Insulin Resistance. *Nutrients*. 2019; 11(4): 794.
- Cheng, Q., Boucher, B. J., Leung, P. S. Modulation of hypovitaminosis D-induced islet dysfunction and insulin resistance through direct suppression of the pancreatic islet renin-angiotensin system in mice. *Diabetologia*. 2013; 56: 553-562.
- Sung, C. C., Liao, M. T., Lu, K. C., Wu, C. C. Role of Vitamin D in Insulin Resistance. *BioMed Research International*. 2012; 2012: 634195.
- Infante, M., Ricordi, C., Sanchez, J., Clare-Salzler, M. J., Padilla, N., Fuenmayor, V., *et al.*, Influence of Vitamin D on islet autoimmunity and beta-cell function in type 1 diabetes. *Nutrients*. 2019; 11(9): 2185.
- Sanghera, D. K., Sapkota, B. R., Aston, C. E., Blackett, P. R. Vitamin D status, gender differences, and cardiometabolic health disparities. *Ann Nutr Metab*. 2017; 70:79-87.
- Verdoia, M., Schaffer, A., Barbieri, L., Di Giovine, G., Marino, P., Suryapranata H, De Luca G; Novara Atherosclerosis Study Group (NAS). Impact of gender difference on vitamin D status and its relationship with the extent of coronary artery disease. *Nutr Metab Cardiovasc Dis*. 2015; 25(5): 464-470.
- Jorde, R., Grimnes, G. Vitamin D and Lipids: Do We Really Need More Studies? *Circulation*. 2012; 126: 252-254.
- Challoumas, D. Vitamin D supplementation and lipid profile: What does the best available evidence show? *Atherosclerosis*. 2014; 235(1): 130-139.
- Bhatt, S. P., Misra, A., Gulati, S., Singh, N., Pandey, R. M. Lower vitamin D levels are associated with higher blood glucose levels in Asian Indian women with pre-diabetes: a population-based cross-sectional study in North India. *BMJ Open Diabetes Res Care*. 2018; 6(1): e000501.
- Daga, R. A., Laway, B. A., Shah, Z. A., Mir, S. A., Kotwal, S. K., Zargar, A. H. High prevalence of vitamin D deficiency among newly diagnosed youth-onset diabetes mellitus in north India. *Arq Bras Endocrinol Metab*. 2012; 56(7): 423-428.

18. Bayani, M. A., Akbari, R., Banasaz, B., Saeedi, F. Status of Vitamin-D in diabetic patients. *Caspian J Intern Med.* 2014; 5(1): 40-42.
19. Karau, P. B., Kirna, B., Amayo, E., Joshi, M., Ngare, S., Muriira, G. The prevalence of vitamin D deficiency among patients with type 2 diabetes seen at a referral hospital in Kenya. *Pan Afr Med J.* 2019; 34: 38.
20. Cheng, S., So, W. Y., Zhang, D., Cheng, Q., Boucher, B. J., Leung, P. S. Calcitriol Reduces Hepatic Triglyceride Accumulation and Glucose Output Through Ca²⁺/CaMKK β /AMPK Activation Under Insulin-Resistant Conditions in Type 2 Diabetes Mellitus. *Curr Mol Med.* 2016; 16(8): 747-758.
21. Dibaba, D. T. Effect of vitamin D supplementation on serum lipid profiles: a systematic review and meta-analysis. *Nutrition Reviews.* 2019; 77(12): 890-902.
22. Wang, H., Xia, N., Yang, Y., Peng, D. Q. Influence of vitamin D supplementation on plasma lipid profiles: a meta-analysis of randomized controlled trials. *Lipids Health Dis.* 2012; 11: 42.
23. Kim, S. H., Baek, M. S., Yoon, D. S., Park, J. S., Yoon, B. W., Oh, B. S., *et al.*, Vitamin D inhibits expression and activity of matrix metalloproteinase in human lung fibroblasts (HFL-1) cells. *Tuberc Respir Dis (Seoul).* 2014; 77(2): 73-80.
24. Pike, J. W., Christakos, S. Biology and Mechanisms of Action of the Vitamin D Hormone. *Endocrinol Metab Clin North Am.* 2017; 46(4): 815-843.
25. Umar, M., Sastry, K. S., Chouchane, A. I. Role of Vitamin D beyond the skeletal function: a review of the molecular and clinical studies. *Int J Mol Sci.* 2018; 19(6): 1618.
26. Almeida, L. F., Coimbra, T. M. Vitamin D actions on cell differentiation, proliferation and inflammation. *Int J Complement Alt Med.* 2017; 6(5): 00201.
27. Bikle, D. Vitamin D: Production, metabolism, and mechanisms of action. [Updated 2017 Aug 11]. In: Feingold KR, Anawalt B, Boyce A, *et al.*, editors. *Endotext* [Internet].
28. Mozos, I., Marginean, O. Links between Vitamin D deficiency and cardiovascular diseases. *Biomed Res Int.* 2015; 2015: 109275.
29. Kim, M. R., Jeong, S. J. Relationship between Vitamin D Level and Lipid Profile in Non-Obese Children. *Metabolites.* 2019; 9(7): 125.
30. Chaudhuri, J. R., Mridula, K. R., Anamika, A., Boddu, D. B., Misra, P. K., Lingaiah, A., *et al.*, Deficiency of 25-Hydroxyvitamin D and Dyslipidemia in Indian Subjects. *Journal of Lipids.* 2013; 2013: 623420.