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A Study to Assess the Prevalance of Vitamin-D Deficiency in Patients with Left Ventricular Dysfunction and Its Correlation with Conventional Echocardiographic Parameter

Dr. Apurba Bikash Pramanik, [1] Dr. Pulakesh Sinha,[2] Dr. Saroj Mandal, [2] Dr. Amit Kumar Jha, [2] Dr. Aditya Verma, [2] Dr. Debarshi Jana [2]
[1] *Nil Ratan Sircar Medical College and Hospital*
[2] *Institute of Post-Graduate Medical Education and Research and SSKM Hospital*
Corresponding Author: Dr. Debarshi Jana

Abstract: *Clinical studies have reported cross-sectional associations between lower vitamin D levels and blood pressure, coronary artery calcification, and prevalent cardiovascular disease like coronary artery disease, chronic left ventricular dysfunction other than musculo-skeletal disease.*
Aim of our study is to assess the prevalence of vitamin D deficiency in patients with chronic left ventricular dysfunction (LVEF<50%).
50 patients attending at cardiology outdoor and admitted at cardiology ward of IPGME&R and SSKM Hospital were included in the study from one year (April 2016-March 2017).
Our study demonstrate that the prevalence of vitamin D deficiency (< 20ng/ml) in patients with chronic left ventricular dysfunction is significantly more (72%) than general population (40 to 50%) though the prevalence of hypovitaminosis D (30ng/ml) is similar in both groups. (84% vs 90%.
Low vitamin D level (<20 ng/ml) appears to be associated with worse systolic functions in terms of high end systolic volume (ESV) and left ventricular end systolic dimension(LVIDs) ,end diastolic volume (EDV), left ventricular end diastolic dimension (LVIDs), low left ventricular ejection fraction (LVEF), low fractional shortening (FS).
Key Words: *Cardiovascular Disease, Vitamin-D Deficiency, Left Ventricular Dysfunction, Conventional Echocardiographic Parameter*

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I. Introduction

Vitamin D is endogenously synthesized in human beings from photo conversion of 7-dehydrocholestrol in the skin to cholecalciferol on exposure to ultraviolet radiation of sun. In a tropical country like India, where sunlight exposure is abundant, vitamin D deficiency seems unlikely. However, as opposed to this, various studies have highlighted that 70-100% Indians in different age groups are vitamin D insufficient or deficient [1]. Vitamin D deficiency is highly prevalent worldwide [2], and is also noted to be high in India [3,4]. Approximately 90% of chronic HF patients have hypovitaminosis D [5], even in sunny climates. Low levels of 25(OH)D, the principle circulating storage form of vitamin D, is present in as many as one third to one half of otherwise healthy middle aged to elderly population [2,6-8]. Limited cutaneous synthesis due to inadequate sun exposure or pigmented skin and inadequate dietary intake are the principle causes of low 25(OH)D levels. Although most consequences of vitamin D deficiency involve the musculoskeletal system, there is a growing body of evidence suggesting that low levels of vitamin D may adversely affect the cardiovascular system [9]. A serum 25-hydroxyvitamin D level below 75 nmol/l (30 ng/ml) is generally regarded as vitamin D insufficiency in both adults and children, while a level below 50 nmol/l (20 ng/ml) is considered deficiency in both populations [10,11,5]. Vitamin D deficiency, which is affected by multiple factors , appears to have an association with diverse cardiac diseases starting with its direct effect on the cardiac cell, its association with coronary artery disease (CAD), and its risk factors such as diabetes and hypertension (HTN); ending at last and probably not least in its relation with congestive heart failure (CHF). Similarly, there is some evidence that links vitamin D deficiency to increased risk of stroke. Myocardium is an important target tissue for vitamin D mediated effects on a genomic and non-genomic level. Cardiomyocytes express the vitamin D receptor, and studies in rodents have shown that vitamin D protects against cardiac hypertrophy and myocardial dysfunction.[12] The association between vitamin D and cardiovascular-disease events is widely debated and analyzed in the literature. In a cross-sectional study, Pilz et al. measured 2 5-hydroxy vitamin D (25 (OH) vitamin D) levels in 3299 Caucasian patients who were routinely referred for coronary angiography.[13] They found that vitamin D deficiency is associated with prevalent

myocardial dysfunction, heart failure, and sudden cardiac death. Although the link between vitamin D deficiency and cardiovascular disease may be, in part, mediated through elevated PTH and calcium-phosphate metabolism, recent scientific evidence showed that vitamin D has 3 major potential protective mechanisms. First, experimental studies indicate that 1.25-(OH) Vitamin D could directly suppress rennin gene expression. Second, is the presence in the cardiac muscle cells of vitamin D receptors, a calcitriol-dependent Ca2+ binding protein and a calcitriol-mediated rapid activation of voltage-dependent Ca2+ channels. Third, vitamin D deficiency triggers secondary hyperparathyroidism, which then directly promotes cardiac hypertrophy (the direct PTH toxicity hypothesis)[14]. Routine digital gray scale 2-D , tissue doppler cine loops and pulsed-wave doppler-derived transmitral flow profile, and digital color tissue doppler-derived mitral annular velocity were obtained from the apical 4-chamber view were obtained, to study the relation between serum 25-hydroxy vitamin D levels and echocardiographic parameters of cardiac systolic and diastolic functions in patients with Left Ventricular Dysfunction. Clinical studies have reported cross-sectional associations between lower vitamin D levels and blood pressure, coronary artery calcification, and prevalent cardiovascular disease like coronary artery disease, chronic left ventricular dysfunction other than musculo-skeletal disease.

Objectives are to assess the prevalence of vitamin D deficiency in patients with chronic left ventricular dysfunction(LVEF<50%) and to evaluate the correlation between vitamin D level and conventional echocardiographic parameter of chronic left ventricular systolic as well as left ventricular diastolic function. Other objective is to determine the demographic profile of patients presenting with chronic left ventricular dysfunction & vitamin D deficiency and to evaluate the prevalence of risk factor of chronic left ventricular dysfunction.

II. Material And Methods

Patients attending at cardiology outdoor and admitted at cardiology ward of IPGME&R and SSKM Hospital were included in the study from one year (April 2016-March 2017). Total 50 patients suffering from chronic Left Ventricular Dysfunction (LVEF<50%) was included in this study.

Inclution critaria:

(a) Age ≥ 18 years.
(b) Either Sex.
(c) All cases of chronic left ventricular dysfunction (LVEF<50%) with NYHA Class2, 3,4.

Exclution criteria:

(a) Age <18 years
(b) Patients having congenital heart disease
(c) Patients with chronic kidney disease.
(d) Patients with previous vitamin D supplementations.
(e) Patients with conditions known to cause Vit. D deficiency e.g.malignancies, Chronic liver disease, Bowel disease causing malabsorbtion , patient on Hemodyalysis.
(f) Patients with poor echogenicity.

Parameter to be studied:

(a) Trans-thoracic echocardiographic study was performed for all patients with commercially available echocardiography systems equipped with a 5- MHz multifrequency phased array transducer (GE, Vivid 6).
(b) Age, sex, Body weight, Height, Body mass index,History of other disease, history of smoking, Alcohol intake, dyslipidemia, Diabetes Mellitus, Hypertension, NYHA Class of Heart failure, Dialated cardiomyopathy, Coronary artery disease, Rheumatic heart disease.
(c) Venous blood sampling was taken and sent for laboratory quantitative measurements of serum levels of 25 (OH) vit. D (by using ELISA technique) . The low vitamin D level (with more link to cardiovascular diseases) is considered <20 ng/ml as reported by the National Health and Nutritional Examination Surveys (NHANES) (1988–1994, 2000– 2004).4,6

Transthorasic echocardiography:

Trans-thoracic echocardiographic study was performed for all patients with commercially available echocardiography systems equipped with a 5 MHz multifrequency phased array transducer(GE,Vivid 6). Digital routine gray scale 2-dimensional and tissue Doppler cine loops from 3 consecutive beats were obtained at end-expiratory apnea from standard apical views at depths of 12–20 cm. Gain settings were adjusted for routine gray scale 2D imaging to optimize endocardial definitions. Routine digital gray scale 2-D and tissue Doppler cine loops were obtained, including mid-LV short axis views at the level of the papillary muscle and standard apical views (4-chamber, 2-chamber, and long-axis). Sector width was optimized to allow for complete

visualization while maximizing the frame rate. LV end-diastolic volume, end-systolic volume (ESV), and ejection fraction were obtained with the modified biplane Simpson's method from the apical 2- and 4-chamber images using the biplane Simpson's technique. All measurements were made in >3 consecutive cardiac cycles and in >5 cycles if the patient's rhythm was AF and average values were used for the final analyses. The pulsed-wave Doppler-derived transmitral flow profile, and digital color tissue Doppler-derived mitral annular velocity were obtained from the apical 4-chamber view. The mitral flow early diastolic wave velocity (E), late diastolic atrial contraction wave velocity (A), and the E-wave deceleration time (E-DcT) were measured; spectral pulsed-wave tissue Doppler-derived peak systolic velocity (s0), early diastolic velocity (el), late diastolic velocity (al), and the E/el ratio were calculated to estimate the LV filling pressure for all patients.

Analysis of data:

Data are presented as Mean± Standard deviation for continuously distributed variables, and in absolute numbers and percentages for the discrete variables. Student's t test was performed to compare parametric variables between two groups. Pearson's correlation coefficient was used to examine the relation between vitamin D level and several study variables including different echocardiographic variables. Linear regression analysis was performed with Echocardiographic parameter as dependent variables and vitamin D as independent variables. Level of p value< 0.05 were considered statistically significant. Data was stored by XLSTAT and MS EXCEL.

III. Result And Analysis

It was found that total chronic left ventricular dysfunction patients into two groups . Group 1 has vitamin D level <20ng/dl & group 2 has vitamin D level ≥20ng/ml. There is no statistically significant difference in age, height, body weight, BMI between two sub group of chronic left ventricular dysfunction patient. Two groups are matched are evenly matched with respect to baseline variable. We found that association between sex vs. vitamin-D in two groups was not statistically significant (p=0.1050) but prevalence is more in female than male. It was found that student s t test was performed to show statistical significance against the null hypothesis that there was no difference in laboratory parameter among the two groups other than urea as indicated by p value against each difference. We found that patient s with vitamin D < 20ng/ml has significantly Higher Urea level [28.92+-7.23 vs. 22.86+-8.15; p value<0.05] than patients with vitamin D >-20ng/ml.

It was found that student s t test was performed to show statistical significance against the null hypothesis that there was no difference in component of lipid among the two groups. We found that echocardiographic diastolic component of left ventricular function. Student s t test was performed to show statistical significance against the null hypothesis that there was no difference in echocardiographic diastolic component among the two groups. It was found that student's t-test was performed to show statistical significance against the null hypothesis that there was no difference in echocardiographic diastolic component among the two groups. There is statistically significant difference between the two sub-groups regarding the LVIDD, EDV, LVIDS, ESV, LVEF and FS as indicated by p-value against each difference.

We found that patients with vitamin D <20ng/ml had significantly high ESV(118.3+-29.39 vs 84.35+-23.38), ESD(49.78+-5.43 vs 41.57+-4.38, p-value 0.0001), EDV(179.92+-38.99 vs 144.14+- 31.37, p-value 0.0036) , EDD(60.08+-5.49 vs 53.28+-4.82,p value 0.0002) ,compared to patients with vitamin D ≥20ng/ml. It was found that patients with vitamin D ≥20ng/ml had significantly low ,FS(17.22+-3.48 vs 21.07+-3.05, p-value 0.007), LVEF(33.89+-6.07 vs 44.07+-3.34. p-value 0.0001) compared to patients with vitamin D <20ng/ml. We found that among chronic left ventricular dysfunction patient 56% are diabetic, 16% are alcoholic,42% hypertensive,46% smoker, family history of CAD 34%, CAD 68%,DCM 12%,RHD 12%.Other than this 38 % present with NYHA class 2,44% present with NYHA class 3, 18 % present with class 4. It was found that at the univariate analysis(table) the some echocardiographic parameter of systolic function correlate with vitamin D level .Vitamin D is negatively correlated with LVIDd(r = -0.531; p -value< 0.001), LVIDs (r= -0.616; p -value<0.001), EDV (r= -0.387;p value 0.005), ESV(R= - 0.45; p - value 0.001). This negative correlation is statistically significant. Vitamin D is positively correlated with LVEF(r= 0.654; p - value<0.001), FS(r= 0.421; p value 0.002).

IV. Discussion

Although most consequences of vitamin D deficiency involve the musculoskeletal system, there is a growing body of evidence suggesting that low levels of vitamin D may adversely affect the cardiovascular system 9. A serum 25-hydroxyvitamin D level below 75 nmol/l (30 ng/ml) is generally regarded as vitamin D insufficiency in both adults and children, while a level below 50 nmol/l (20 ng/ml) is considered deficiency in both populations [15,16]. So vitamin D level <30ng/ml is regarded as Hypovitaminosis D.[10] Our study shows that the prevalence of vitamin D deficiency (< 20ng/ml) in patients with chronic left ventricular dysfunction is

significantly higher than general population though the prevalence of hypovitaminosis D (<30ng/ml) is similar in both groups. Our study demonstrated that the prevalence of hypovitaminosis in patients with left ventricular dysfunction is 88% & vitamin D deficiency is 72% which corroborate with the findings of study done by Kim DH, Sabour S, et al that shows hypovitaminosis D is present in 90% of chronic heart failure patients even in sunny climate [17] . This is supported by the study conducted by Rudrajit Paul et al in Kolkata [18] that shows that vitamin D deficiency is present in 47.5 % of general population hypovitaminosis D is present 87.5% of general population. This is further supported by study conducted by Rachana Bachel et all from Amritsar [19] shows that vitamin D deficiency is present in 40% of general people and hypovitaminosis D is present in 84% of general people. Our study also shows that vitamin D deficiency is more prevalent in female than male population (75% vs 70.4%) that is also supported by two previous study. Our study shows that vitamin D level is significantly correlated with some echocardiographic parameter of LV systolic function. This was proven when we found that ESV(118.3+-29.39 vs 84.35+-23.38), LVIDs(49.78+-5.43 vs 41.57+-4.38, p value 0.0001), EDV(179.92+-38.99 vs 144.14+- 31.37,p value 0.0036) , LVIDd(60.08+-5.49 vs 53.28+-4.82,p value 0.0002) , all are negatively correlated with serum vitamin D levels. Moreover, vitamin D level is also positively correlated with FS(17.22+-3.48 vs 21.07+-3.05, p-value 0.007), LVEF(33.89+-6.07 vs 44.07+-3.34.p value 0.0001) It seems that the deficiency of vitamin D (vitamin D <20 ng/ml) weakens its protective effects of vitamin D against fibrosis, myocardial fiber thickening and anti-apoptosis, resulting in wall thickening and eventually dilatation. Our findings are consistent with previous cross-sectional studies conducted by Shane E et al [20] showing vitamin D deficiency associated with worse LV function. Another cross sectional study conducted by Jegger d et al[21] also showed that patients with 25-OH D <25 nmol/L had larger end-systolic diameters and reduced fractional shortening, compared with heart failure patients with 25-OH D ≥ 25 nmol/L, an observation that is also consistent with our cross-sectional analyses. Another study conducted by Mohammed Ahmed Abdel Rahman et al[22] showed that low vitamin D level is associated with worse left ventricular systolic function that is high LVIDD,LVIDV & left ventricular wall thickness . Ameri P, Ronco D, Casu M, et al [23] shoes high prevalence of vitamin D deficiency and its association with left ventricular dilation in elderly patients with chronic heart failure. This study also shows that higher vitamin D levels were associated with lower average myocardial early diastolic tissue velocity (el), higher E/el ratio, and longer IVRT, which are all linked to worse diastolic functions. Another study conducted by Anil Pandit et al showed that there is no correlation between vitamin D level & left ventricular diastolic function but our study does not show any such correlation of vitamin D level with Echocardiographic parameter of diastolic function. The speciality of our study is that we have shown that low vitamin D level is positively correlated with FS & LVEF. Vitamin D deficiency has been observed to induce myocardial hypertrophy and extracellular matrix production and deposition in myocardial tissue. Mediated by matrix metalloproteinase, extracellular matrix remodelling may be involved in progressive LV remodeling, dilatation, and heart failure. At the molecular level, vitamin D also has intriguing immunoregulatory and DNA protective properties. Other than these finding our study shows prevalence of coronary artery disease and its risk factor in heart failure patient is significantly higher than general population ie in chronic left ventricular dysfunction patient 56% are diabetic , 46% are smoker, 42% are hypertensive, 16 % are alcoholic. Among chronic heart failure patients family history of CAD present in 34% cases , RHD present in 12% & DCM present in 12% patient. Prevalence of hypercholesterolemia is 26%. The study conducted by Gupta et al from Jaipur in 2002 [24] shows prevalence of CAD was 6.2% in men and 10.8% in women, hypertension (36.9%), tobacco use (23.9%), obesity (63%) and Shashank R. Joshi shows Prevalence of hypercholesterolemia is [25] (39.1%). Diabetes was prevalent in 12.2% of the cases in general population. Our study shows prevalence of risk factor of CHD is high than general population.

V. Conclusion

Our study suggest that reduced vitamin D level (<20 ng/ml) appears to be associated with worse systolic functions in terms of high end systolic volume (ESV) and left ventricular end systolic dimension(LVIDs) ,end diastolic volume (EDV), left ventricular end diastolic dimension (LVIDs), low left ventricular ejection fraction (LVEF), low fractional shortening (FS). But there is no statistically significant differences in diastolic function between two groups of patients ie those with vitamin D level < 20ng/ml and those with vitamin D level >= 20ng/ml.

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Table 1: Distribution of mean Age (Yrs), Height (cm), Body Weight (Kg), BMI, Hb, ESR, UREA, CREAT, FBS, PPBS, HbA1C, T/Ch, LDL, HDL, VLDL, TG in two groups.

		Number	Mean	SD	Minimum	Maximum	Median	p- value
Age (Yrs)	<20	36	58.8611	10.2933	35.0000	85.0000	57.5000	0.4703
	≥20	14	61.0714	7.6205	43.0000	72.0000	62.0000	
Height (cm)	<20	36	164.6667	5.5857	153.0000	174.0000	164.5000	0.3447
	≥20	14	166.4286	6.5482	156.0000	174.0000	168.0000	
Body Weight (Kg)	<20	36	60.2778	5.8437	49.0000	72.0000	61.0000	0.3851
	≥20	14	61.9286	6.3302	53.0000	77.0000	60.5000	
BMI	<20	36	22.1956	2.1080	17.4000	28.0400	21.8500	0.8010
	≥20	14	22.3607	1.9602	19.2000	25.7500	21.6000	
Hb	<20	36	12.5333	1.6699	9.2000	16.0000	12.5000	0.9488
	≥20	14	12.5000	1.5566	10.0000	16.0000	12.0000	
ESR	<20	36	23.2500	5.1235	15.0000	34.0000	24.0000	0.3761
	≥20	14	24.9286	7.7902	12.0000	36.0000	28.0000	
UREA	<20	36	28.9167	7.2284	12.0000	48.0000	27.5000	0.0134
	≥20	14	22.8571	8.1510	14.0000	36.0000	20.5000	
CREAT	<20	36	1.1528	.2261	0.7000	1.6000	1.2000	0.8089
	≥20	14	1.1357	.2134	0.7000	1.4000	1.2000	
FBS	1	36	99.6389	13.0541	69.0000	122.0000	99.5000	0.8910
	2	14	99.0714	13.1586	82.0000	122.0000	97.0000	
PPBS	<20	36	139.6389	18.2211	102.0000	178.0000	138.0000	0.9595
	≥20	14	139.3571	15.4303	122.0000	184.0000	136.5000	
HbA1C	<20	36	6.8556	1.6171	5.5000	11.4000	6.0000	0.1371
	≥20	14	6.1571	.9493	5.5000	9.2000	5.8500	
T/Ch	<20	36	184.5000	30.9769	121.0000	253.0000	187.5000	0.4006
	≥20	14	194.9286	55.1731	114.0000	363.0000	183.5000	
LDL	<20	36	121.3611	23.8485	67.0000	158.0000	128.0000	0.4215
	≥20	14	130.0000	51.9141	53.0000	291.0000	122.0000	
HDL	<20	36	31.6667	10.0029	17.0000	71.0000	30.5000	0.3262
	≥20	14	34.7857	9.9319	20.0000	49.0000	39.0000	
VLDL	<20	36	31.0556	7.4141	19.0000	56.0000	29.5000	0.8573
	≥20	14	30.6429	6.7779	19.0000	43.0000	29.0000	

TG	<20	36	155.3333	36.9788	95.0000	280.0000	147.5000	0.9025
	≥20	14	153.9286	34.0350	95.0000	215.0000	145.0000	

Table 2: Distribution of mean E, A, E/A, DT, E (cm/s), A(cm/s), E/E, IVRT, IVRT, IVSd, LVIDd, LVPWd, IVSs, LVEF, FS, SV, LVdMass, EDV, ESV in two groups

		Number	Mean	SD	Minimum	Maximum	Median	p- value
E	<20	36	.0897	.1220	0.0500	0.8000	0.0700	
	≥20	14	.0686	.0110	0.0600	0.1000	0.0700	0.5230
A	<20	36	.0819	.0086	0.0600	0.0900	0.0800	
	≥20	14	.0800	.0068	0.0700	0.0900	0.0800	0.4507
E/A	<20	36	1.2594	.4655	0.5500	1.9700	1.3900	
	≥20	14	1.3036	.4322	0.5700	1.8200	1.3100	0.7604
DT	<20	36	182.0556	20.3343	143.0000	215.0000	179.5000	
	≥20	14	179.5714	20.0680	154.0000	214.0000	174.5000	0.6988
E(cm/s)	<20	36	64.9444	20.1096	34.0000	93.0000	66.5000	
	≥20	14	72.5000	19.1462	39.0000	94.0000	78.0000	0.2329
A(cm/s)	<20	36	53.7222	11.2443	31.0000	74.0000	55.0000	0.2898
	≥20	14	57.3571	9.4267	42.0000	72.0000	56.5000	
E/E	<20	36	9.2022	2.3883	5.4200	12.8300	9.7250	0.0776
	≥20	14	10.6057	2.6792	6.5000	13.5000	11.0050	
IVRT	<20	36	94.2222	11.9645	74.0000	119.0000	91.5000	0.2528
	≥20	14	90.0000	10.4808	78.0000	112.0000	89.0000	
IVSd	<20	36	8.5278	1.1585	6.0000	11.0000	9.0000	0.0168
	≥20	14	9.3571	.7449	8.0000	10.0000	9.5000	
LVIDd	<20	36	60.0833	5.4948	51.0000	77.0000	58.5000	0.0002
	≥20	14	53.2857	4.8267	48.0000	67.0000	53.0000	
LVPWd	<20	36	8.5000	1.4442	6.0000	12.0000	9.0000	0.0179
	≥20	14	9.5000	.7596	8.0000	11.0000	9.5000	
IVSs	<20	36	9.3056	6.4090	6.0000	46.0000	8.0000	0.8290
	≥20	14	8.9286	1.2688	6.0000	11.0000	9.0000	
LVIDs	<20	36	49.7778	5.4254	38.0000	63.0000	50.0000	<0.0001
	≥20	14	41.5714	4.3803	37.0000	53.0000	41.0000	
LVPWs	<20	36	8.5833	1.2277	6.0000	11.0000	8.5000	0.0083
	≥20	14	9.7143	1.4899	6.0000	12.0000	10.0000	
LVEF	<20	36	33.8889	6.0653	16.0000	48.0000	33.0000	<0.0001
	≥20	14	44.0714	3.3389	37.0000	49.0000	44.0000	
FS	<20	36	17.2222	3.4815	12.0000	26.0000	17.0000	0.0007
	≥20	14	21.0714	3.0500	14.0000	25.0000	22.0000	
SV	<20	36	61.8056	13.1297	41.0000	89.0000	59.5000	0.4422
	≥20	14	58.7143	11.3166	47.0000	90.0000	57.0000	
LVdMass	<20	36	240.7753	40.5479	137.6000	321.1800	240.4300	0.6088
	≥20	14	234.2507	39.3038	170.6500	309.2800	234.5400	
EDV	<20	36	179.9167	38.9875	123.0000	316.0000	167.0000	0.0036
	≥20	14	144.1429	31.3733	108.0000	228.0000	142.0000	
ESV	<20	36	118.3056	29.3976	69.0000	202.0000	114.5000	0.0003
	≥20	14	84.3571	23.3752	59.0000	138.0000	79.0000	

Table 3: Distribution of DM, ALCOHOL, SMOKING, FAMILY HIST, HTN, DCM, NYHA, CAD, RHD.

		Frequency	Percent
DM	Absent	22	44.0%
	Present	28	56.0%
ALCOHOL	Absent	42	84.0%
	Present	8	16.0%
SMOKING	Absent	27	54.0%
	Present	23	46.0%
FAMILY HIST	Absent	33	66.0%
	Present	17	34.0%
HTN	Absent	29	58.0%
	Present	21	42.0%
DCM	Absent	44	88.0%
	Present	6	12.0%
NYHA	2	19	38.0%
	3	22	44.0%
	4	9	18.0%
CAD	Absent	16	32.0%
	Present	34	68.0%
RHD	Absent	44	88.0%
	Present	6	12.0%

Table 4: Correlation of IVSd, LVIDd, LVPWd, IVSs, LVIDs, LVPWs, LVEF, FS, SV, LVd Mass, EDV and ESV with VIT-D

	CORRELATION COEFICIENT	VIT-D	
IVSd	Pearson Correlation Coefficient (r)	-.124	Negative Correlation
	p-value	.390	Not Significant
	Number	50	
LVIDd	Pearson Correlation Coefficient (r)	-.531**	Negative Correlation
	p-value	<0.001	Significant
	Number	50	
LVPWd	Pearson Correlation Coefficient (r)	-.007	Negative Correlation
	p-value	.962	Not Significant
	Number	50	
IVSs	Pearson Correlation Coefficient (r)	-.007	Negative Correlation
	p-value	.963	Not Significant
	Number	50	
LVIDs	Pearson Correlation Coefficient (r)	-.616**	Negative Correlation
	p-value	<0.001	Significant
	Number	50	
LVPWs	Pearson Correlation Coefficient (r)	.066	Positive Correlation
	p-value	.649	Not Significant
	Number	50	
LVEF	Pearson Correlation Coefficient (r)	.654**	Positive Correlation
	p-value	<0.001	Significant
	Number	50	
FS	Pearson Correlation Coefficient (r)	.421**	Positive Correlation
	p-value	.002	Significant
	Number	50	
SV	Pearson Correlation Coefficient (r)	-.145	Negative Correlation
	p-value	.314	Not Significant
	Number	50	
LVd Mass	Pearson Correlation Coefficient (r)	-.142	Negative Correlation
	p-value	.325	Not Significant
	Number	50	
EDV	Pearson Correlation Coefficient (r)	-.387**	Negative Correlation
	p-value	.005	Significant
	Number	50	
ESV	Pearson Correlation Coefficient (r)	-.450**	Negative Correlation
	p-value	.001	Significant
	Number	50	

Dr. Apurba Bikash Pramanik. "A Study to Assess the Prevalance of Vitamin-D Deficiency in Patients with Left Ventricular Dysfunction and Its Correlation with Conventional Echocardiographic Parameter." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 4, 2019, pp 52-58.