

Correlation of Vitamin D with Severity of Coronary Artery Disease and Short-Term Outcome in Female Versus Male Patients with Coronary Artery Disease

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Abstract

Background: Coronary artery disease (CAD) is a worldwide health concern. Cardiovascular disease (CAD) continues to be the predominant cause of mortality and morbidity, despite the enhancement of the current preventive and therapeutic strategies. **This study aimed to** correlate between vitamin D (Vit D), CAD severity and short-term outcome in females versus males with CAD. **Methods:** This cross-sectional study was conducted on 200 cases who were categorized into 2 equal groups: group A (males) and group B (females). Vit D level was evaluated in all the cases studied, and they also underwent electrocardiography, coronary angiography, and calculation of Syntax. **Results:** There was a significant negative correlation between Syntax score and Vit D level, meanwhile there was a significant positive correlation between Syntax score and number of affected vessels. **Conclusion:** There is an association between Vit D and CAD as it can predict the CAD severity at cutoff value of ≤ 20 ng/ml, with 98.86% sensitivity, 84.82% specificity, 83.7% PPV and 99.0% NPV.

Keywords: Coronary Angiography; Coronary Artery Disease; Electrocardiography; Vitamin D.

Introduction

Coronary artery disease (CAD) is a global health issue. Despite the progress made in current preventive and therapeutic methods, cardiovascular disease (CVD) remains the primary cause of morbidity and mortality in both

developed and developing countries (1). The prevention of CAD through primary

and secondary measures necessitates the risk factors identification, including male sex, old age, DM, HTN, smoking, dyslipidemia, chronic renal disease, and a positive family history. However,

conventional risk factors do not occur in all patients with CAD, which suggests that there are alternative causes of their atherosclerosis. A substantial risk of CV events, myocardial infarction (MI), and mortality is associated with reduced vitamin D (Vit D), as demonstrated by extensive cross-sectional studies that have a long-term follow-up of over seven years (2).

Vit D deficiency has become more prevalent among the general population. It has been demonstrated that vit D has the capacity to directly or indirectly affect the renin–angiotensin system, inflammation, cytokine levels, and adaptive immune responses to inflammation. (3). Deficiency of Vit D may contribute to CVD through the mechanisms of impaired insulin sensitivity, inflammation, and increased renin–angiotensin levels. In patients who have coronary arteries and endure coronary angiography, subclinical atherosclerosis and endothelial dysfunctions have been associated with vit D deficiency. Insufficient vit D levels have been shown to exacerbate atherosclerosis, as well as cause vascular disease, smooth muscle cell proliferation, foam cell formation, and endothelial dysfunction (4).

The objective of this investigation was to establish a correlation between the CAD severity and vit D, short term outcome in female versus male CAD patients, relation between Vit D, ischemic and thrombotic burden and illustrate the

extent of CAD and the gender-specific variations in vit D levels.

Patients and methods

The cross-sectional investigation was conducted on 200 patients with CAD, presented at Cardiology Department in Benha Insurance Hospital & Benha University Hospital. The patients provided written informed consent and received a confidential code number and an explanation of the study's purpose. The research was conducted with the approval of the Research Ethics Committee code MS 29-4-2024 at the Faculty of Medicine at Benha University from March 2024 to March 2025.

Inclusion criteria included adult patients aged 18 years or older, regardless of gender, who had CAD (acute ST-elevation MI, non-ST-elevation MI, old MI, and chronic stable or unstable angina pectoris).

Exclusion criteria included the administration of vit D and calcium supplements, parathyroid gland disorders, bone tumors or metabolic bone diseases, or a history of chronic kidney disease.

Grouping: Patients were assigned to two equal groups: **Group A (n=100):** including male patients. **Group B (N=100):** including female patients.

All studied cases were subjected to complete history including patient demographics, medical history and

previous medications for CAD management and symptoms review by

chest pain characteristics, symptoms of heart failure and other relevant symptoms. Risk factors including HTN which is diagnosed according to European society of cardiology (ESC) (5), symptoms suggestive of STEMI, time delay as patient related delay, diabetes mellitus, chronic kidney disease, dyslipidemia which was diagnosed according to ESC (6), smoking status, previous history of CAD according to ESC (7) and family history of CAD (8). **Complete physical examinations** including general assessment, cardiovascular, respiratory, abdominal and extremities examinations. **Laboratory investigations** were conducted, including a complete blood count, kidney function assays (serum creatinine and blood urea), international normalized ratio, prothrombin time, partial thromboplastin time, hepatic function, fasting and random blood glucose levels, lipid profile, cardiac enzymes, electrolytes, and serum vit D deficiency (optimal value 40-80 ng/mL). The participants were divided into three categories: mild (20-30 ng/mL), moderate (10-20 ng/mL), and severe (<10 ng/mL). In addition, participants were classified as normal (>30 ng/mL) (9). Radiological investigations by electrocardiography (ECG), echocardiographic examination, and coronary angiography.

The LIAISON® Vit D assay (Diasorin Inc) was employed to evaluate Vit D

levels using the chemiluminescence method. The laboratory established a normal range for 25(OH)D levels of 30 to 100 ng/ml. A value of less than 10 ng/ml was considered to indicate severe hypovitaminosis D.

A predetermined algorithm was employed to calculate syntax scores, which were subsequently supplemented with points. For this algorithm, six clinical variables were assessed: age, sex, left ventricular ejection fraction (LVEF), creatinine clearance (CrCl), chronic obstructive pulmonary disease, and peripheral artery disease. The lesions' complexity was indicated by higher scores, which ranged from 0 to over 60 (10).

12-lead surface ECG was conducted for each patient. STEMI was diagnosed based on presence of a new (or increasing) and persistent elevated ST-segment in at least two contiguous leads of ≥ 1 mm in all leads, with the potential exception of leads V2-V3. In the appropriate clinical context, the following cut-off values were applied; for males under 40 years are ≥ 2.5 mm, males over 40 years are ≥ 2 mm, and women of any age are ≥ 1.5 mm. Contiguous ECG leads were anatomically adjacent and symbolized a particular myocardial territory. Using modified Simpson methodologies, the EF% was evaluated during the echocardiographic examination. The endocardial borders were traced in both systole and diastole, and apical two-chamber and four-chamber views were

obtained. A computer algorithm partitioned the LV cavity into approximately 20 discs. LV volume was determined by the sum of the volumes of these discs. The LVEF was determined by dividing the disparity between the diastolic and systolic volumes by the diastolic volume (11).

At least one coronary stenosis that exceeded 50% was regarded as indicative of CAD. The presence of 3-vessel disease and/or left main disease was used to assess the CAD severity (12). Patients who had previously undergone percutaneous coronary interventions were believed to have a vessel that was diseased, and the treated lesion was believed to be as such. patients who had undergone prior bypass procedures were evaluated for the number of diseased vessels, as well as both native arteries and grafts, to ascertain the extent of CAD.

Statistical analysis:

SPSS v27 (IBM©, Armonk, NY, USA) was used to conduct statistical analysis. Shapiro-Wilks' test and histograms were used to verify the normality of the data distribution. The quantitative parametric data was analyzed using an unpaired student t-test, which was based on the mean and standard deviation (SD). The Mann Whitney test was used to assess quantitative non-parametric data, which was presented using the interquartile range (IQR) and median. To analyze the qualitative variables, which were reported as frequencies and percentages,

we utilized Fisher's exact test or the Chi-square test as appropriate. A two-tailed P value below 0.05 was used to assess statistical significance. Pearson or spearman correlation to measure the degree of relationship between two numerical variables. Receiver operating characteristic (ROC) curve which extends from the lowest left corner to the highest left corner and subsequently to the highest right corner. Area under the curve, also known as area under the curve (AUC), is a measure the diagnostic performance; an AUC more than 50% indicates satisfactory performance, and an AUC close to 100% indicates excellent performance.

Results

The females' age was significantly greater than that of the males, while other demographic data were insignificantly different between both groups. The risk factors, smoking, history of MI, previous percutaneous coronary intervention (PCI), history of MI, HTN, and CABG in males were significantly increased than females. There was an insignificant difference between both groups regarding the other risk factors. The medications history, the presenting symptoms and the vital signs were comparable between two groups. High-density lipoprotein (HDL) in males was significantly lower compared to females however triglycerides, total cholesterol and LDL in females were significantly higher than males. There was an insignificant difference between both groups regarding the other

laboratory investigations. The Vit D level in males was significantly decreased compared to females. In comparison to females, the Vit D levels of the moderate and severe deficiency groups were significantly elevated in males. The ECG findings on admission were significantly different between both groups. The angiographic findings, 1-vessel CAD and lesion severity <50% in females were significantly greater than males, while SYNTAX score, LAD, LCX, RCA affected vessels, lesion length, severity (50-70%, >70%), and Males had significantly higher rates of chronic occlusion than females. There was an insignificant difference between both groups regarding the EF, the other angiographic findings, and the adverse events. **Table 1**

In comparison to the normal and mild deficiency, the occurrence of smokers was elevated in the severe and moderate deficiency in both males and females, and it was significantly higher in the severe deficiency than in the moderate deficiency ($P<0.001$) among males, while smoking in females was insignificantly different. Males with severe deficiency exhibited significantly higher clopidogrel levels than those with moderate deficiency. The ASA levels in the mild and moderate deficiency groups were significantly lower than those in the normal group. The severe group exhibited a significantly higher HbA1c value than the normal, mild, and moderate deficiency groups. Notably, the moderate deficiency group exhibited a significantly higher HbA1c value than

the normal group however HbA1c was insignificantly different between the other groups and each other's. Triglycerides and LDL in severe deficiency were significantly higher than normal, mild, and moderate deficiency groups. Compared to the normal group, HDL levels were significantly lower in moderate and severe deficiency, with an insignificant difference between the other groups and each other's in female. The baseline characteristics, the other risk factors, the presenting symptoms, the other medications, and the other laboratory investigations were insignificantly different from the studied.

Table 2

Among both males and females, the ECG on admission was insignificantly different among the studied groups. The EF of females was significantly lower in the moderate and severe deficiency groups than in the normal group, and it was significantly decreased in the severe deficiency groups over the mild deficiency groups. The EF of males in the severe deficiency group was lower than that of the normal, mild, and moderate deficiency groups. was significantly lower in mild and moderate deficiency groups than normal groups. In the severe deficiency group, the syntax score was significantly greater than that of the other groups. Similarly, the moderate deficiency group exhibited a significantly higher syntax score than the normal and mild groups. The severity of CAD (50-70%, >70%) was the highest in severe groups, while the remaining angiographic findings were

insignificantly different among the studied groups. The adverse events including reinfarction and mortality in severe deficiency group were significantly higher than the other groups. Bleeding was insignificantly different among the studied groups.

Table 3

A significant relation was found between syntax score and severity, Since the syntax score was higher in patients with a severity level exceeding 70% than in those without a severity level, and those with <50%, and was insignificantly different than those with 50-70%. Syntax score was significantly higher in patients with severity 50-70% than patients with no severity and those with <50% and was significantly higher in patients with severity <50% than patients with no severity. There was an insignificant

relation between syntax score and affected vessels. **Table 4**

A significant negative correlation was observed between the Syntax score and Vit D level ($r=-0.751$, $P<0.001$), whereas a significant positive correlation was observed between the number of affected vessels and the Syntax score ($r=0.305$, $P<0.001$). **Table 5, Figure 1**

Vit D level can significantly predict the severity of CAD with AUC of 0.936, at cutoff value of ≤ 20 ng/ml, with 98.86% sensitivity, 84.82% specificity, 83.7% PPV and 99.0% NPV. **Figure 2,3.**

The multivariate logistic regression analysis showed that sex (male), smoking, chronic occlusion, Syntax score and vit D level were significant predictors for CAD severity. **Table 6**

Table 1: Baseline characteristics, risk factors, medications history, vital signs, presenting symptoms, laboratory investigation, vitamin D level, ECG on admission, ejection fraction and angiographic and adverse events findings of the studied groups

	Male group (n=100)	Female group (n=100)	P-value
Baseline characteristics			
Age (years)	58.05±4.1	69.11 ± 4.71	<0.001*
Weight (kg)	78.68±10.18	78.03 ±10.13	0.651
Height (m)	1.67 ± 0.04	1.66 ±0.04	0.146
BMI (kg/m ²)	28.36 ±3.97	28.44 ±4.14	0.878
Risk factors			
Smoking	52 (52%)	2 (2%)	<0.001*
HTN	65(65%)	47(47%)	0.044*
DM	46 (46%)	40 (40%)	0.319
Dyslipidaemias	62 (62%)	59 (59%)	0.664
Atrial fibrillation	11 (11%)	15 (15%)	0.400
Family history	38 (38%)	26 (26%)	0.068
History of MI	33 (33%)	16 (16%)	0.005*
Previous PCI	31 (31%)	18 (18%)	0.032*
Previous CABG	18 (18%)	7 (7%)	0.018*
COPD	5 (5%)	9 (9%)	0.267
Medications history			
ACE inhibitors	32 (32%)	29 (29%)	0.645
ARBs	20 (20%)	23 (23%)	0.605

Beta blockers	33 (33%)	25 (25%)	0.212
Nitrates	28 (28%)	31 (31%)	0.471
Statins	36 (36%)	29 (29%)	0.344
Calcium antagonists	18 (18%)	11 (11%)	0.159
Diuretics	13 (13%)	10 (10%)	0.638
ASA	37 (37%)	30 (30%)	0.494
Clopidogrel	12(12%)	9 (9%)	0.471
Presenting symptoms			
Chest pain	77(77%)	69(69%)	0.202
Dyspnea	23(23%)	31(31%)	
Vital signs			
HR (beats/min)	82.3 ± 7.95	82.56 ± 7.14	0.808
SBP (mmHg)	138.1 ± 10.12	137.2 ± 10.06	0.529
DBP (mmHg)	79.2 ± 7.74	80.2 ± 7.38	0.351
HR (beats/min)	82.3 ± 7.95	82.56 ± 7.14	0.808
Laboratory investigation			
Hb (g/dL)	13.45 ± 0.63	13.35 ± 0.6	0.272
WBCs (*109/L)	7.23 ± 1.1	7.46 ± 1.13	0.156
Platelets (*109/L)	300.13 ± 27.07	300.27 ± 27.33	0.971
HbA1c (%)	6.0 ± 1.17	6.03 ± 1.15	0.870
Serum creatinine (mg/dL)	0.98 ± 0.12	0.98 ± 0.12	0.793
Urea (mg/dL)	34.1± 8.67	33.99 ± 9.1	0.930
CRP (mg/dL)	1.78 ± 0.32	1.81 ± 0.31	0.530
PT (sec)	11.71 ± 0.45	11.78 ± 0.48	0.246
PTT (sec)	28.98 ± 2.94	29.96± 3.1	0.397
INR	0.99 ± 0.14	0.99 ± 0.14	0.844
ALT (U/L)	32.56 ± 4.77	32.75± 4.72	0.777
AST (U/L)	28.52 ± 6.44	29.99± 6.16	0.101
Cholesterol (mg/dL)	180.56± 20.44	157.57± 9.81	<0.001*
Triglycerides (mg/dL)	174.6± 51.25	120.72± 17.71	<0.001*
HDL (mg/dL)	39.83± 3.04	47.11± 5.15	<0.001*
LDL (mg/dL)	105.81± 17.94	86.32± 8.43	<0.001*
Vitamin D level			
Vit D level (ng/mL)	19.49 ± 12.44	23.76 ± 11.87	0.014*
Normal (>30 ng/mL)	24 (24%)	34 (34%)	0.044*
Mild deficiency (20-30 ng/mL)	16 (16%)	25 (25%)	
Moderate deficiency (10-20 ng/mL)	31 (31%)	25 (25%)	
Severe deficiency (<10 ng/mL)	29 (29%)	16 (16%)	
ECG on admission			
STEMI	14 (14%)	9 (9%)	0.005*
Non-STEMI	29 (29%)	18 (18%)	
Old myocardial infarction	13 (13%)	10 (10%)	
Chronic stable angina pectoris	12 (12%)	34 (34%)	
Unstable angina pectoris	32 (32%)	29 (29%)	
Ejection fraction and angiographic findings			
EF (%)	56.89 ± 4.79	57.1 ± 4.82	0.758
Syntax score	23.35± 11.89	19.78± 11.11	0.029*
Affected vessels			
1-vessel CAD	37 (37%)	55 (55%)	0.011*
2-vessel CAD	30 (30%)	20 (20%)	0.102
3-vessel CAD	33 (33%)	25 (25%)	0.212
Type of vessel			
LAD	57 (57%)	29 (29%)	<0.001*
LCX	46 (46%)	18 (18%)	<0.001*
RCA	34 (34%)	12 (12%)	<0.001*
OM	21 (21%)	13 (13%)	0.132
Diagonal	19 (19%)	10 (10%)	0.555
PDA	12 (12%)	9 (9%)	0.070

Lesion characteristics	Ramus intermedius	7 (7%)	4 (4%)	0.352
	Lesion length (mm)	26.13 ± 6.5	20 ± 5.85	<0.001*
	Severity <50%	7 (7%)	24 (24%)	<0.001*
	50-70	14 (14%)	7 (7%)	0.040*
	>70%	30 (30)	6 (6%)	<0.001*
Adverse events	Chronic occlusion	14 (14%)	4 (4%)	0.013*
	Thrombus	4 (4%)	2 (2%)	0.407
	Bleeding	3 (3%)	1 (1%)	0.312
	Reinfarction	8 (8%)	4 (4%)	0.233
	Mortality	5 (5%)	2 (5%)	0.445

Data presents as mean ± SD or frequency (%). *Statistically significant difference as p value <0.05. BMI: Body mass index, HTN: hypertension, DM: diabetes mellitus, MI: myocardial infarction, PCI: percutaneous coronary intervention, CABG: coronary artery bypass graft surgery, COPD: chronic obstructive pulmonary disease, ACE: angiotensin-converting enzyme, ARBs: angiotensin receptor blockers, ASA: acetylsalicylic acid, HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, Hb: hemoglobin, WBCs: white blood cells, HbA1c: glycated hemoglobin, CRP: c-reactive protein, PT: percutaneous therapy, PTT: Partial thromboplastin time, INR: international normalized ratio, ALT: alanine aminotransferase, AST: aspartate aminotransferase, HDL: high-density lipoprotein, LDL: low-density lipoprotein, STEMI: ST-elevation myocardial infarction, EF: Ejection fraction, CAD: coronary artery disease, LAD: left anterior descending artery, LCX: left circumflex artery, RCA: right coronary artery, OM: obtuse marginal, PDA: posterior descending artery.

Table 2: Baseline characteristics, risk factors, medications, presenting symptoms according to vit D status, vital signs, and laboratory investigation.

		Normal	Mild deficiency	Moderate deficiency	Severe deficiency	P value	
Baseline characteristics							
Age (years)	Males	58.21 ±4.23	56.25 ±3.86	58.71 ±4.06	58.21 ±4.07	0.267	
	Females	70.15±4.45	68.04 ±4.58	69.04 ±4.95	68.69 ±5.08	0.386	
Weight (kg)	Males	79.54 ±9.71	78.20 ±9.92	78.84 ±9.59	81.24±10.39	0.692	
	Females	77.74±10.07	78.08 ±11.75	79.52 ±9.04	76.25 ±9.74	0.791	
Height (m)	Males	1.67 ±0.05	1.67 ±0.04	1.66 ±0.04	1.67±0.04	0.651	
	Females	1.67 ±0.05	1.64 ±0.04	1.67±0.05	1.65 ±0.04	0.066	
BMI (kg/m²)	Males	28.68 ±3.83	26.14 ±4.37	28.64 ±3.78	29 ±3.82	0.106	
	Females	28.04±4.14	29.02±4.67	28.61±3.71	28.13±4.14	0.819	
Risk factors							
Smoking	Males	3 (12.5%)	3 (18.8%)	19(61.29%)	27 (93.10%)	<0.001*	P1=0.588
							P2<0.001*
							P3<0.001*
							P4<0.001*
							P5<0.001*
							P6<0.001*
HTN	Females	0 (0%)	0 (0.0%)	1 (4.0%)	1 (6.3%)	0.061	
	Males	14 (58.3%)	11 (68.8%)	16 (51.6%)	24 (82.8%)	0.071	
DM	Females	20 (58.8%)	11 (44.0%)	10 (40.0%)	6 (37.5%)	0.381	
	Males	7 (29.2%)	8 (50.0%)	19 (61.3%)	12 (41.4%)	0.111	
dyslipidaemia	Females	17 (50.0%)	7 (28.0%)	9 (36.0%)	7 (43.8%)	0.365	
	Males	14 (58.3%)	10 (62.5%)	22 (71.0%)	16 (55.2%)	0.621	
Atrial fibrillation	Females	21 (61.8%)	17 (68.0%)	16 (64.0%)	5 (31.3%)	0.098	
	Males	4 (16.7%)	1 (6.3%)	3 (9.7%)	3 (10.3%)	0.747	
Family history	Females	5 (14.7%)	3 (12.0%)	6 (24.0%)	1 (6.3%)	0.436	
	Males	10 (41.7%)	4 (25.0%)	15 (48.4%)	9 (31.0%)	0.347	
History of MI	Females	6 (17.6%)	5 (20%)	9 (36%)	6 (37.5%)	----	
	Males	11 (45.8%)	4 (25.0%)	11(35.5%)	7 (24.1%)	0.338	
Previous PCI	Females	5 (14.7%)	5 (20.0%)	3 (12.0%)	3 (18.8%)	0.867	
	Males	11 (45.8%)	5 (31.3%)	9 (29.0%)	6 (20.7%)	0.265	
Previous CABG	Females	5 (14.7%)	6 (24.0%)	4 (16.0%)	3 (18.8%)	0.817	
	Males	6 (25.0%)	3 (18.8%)	6 (19.4%)	3 (10.3%)	0.574	
COPD	Females	0 (0.0%)	4 (16.0%)	2 (8.0%)	1 (6.3%)	0.126	
	Males	0 (0.0%)	1 (6.3%)	1 (3.2%)	3 (10.3%)	0.352	
	Females	2 (5.9%)	4 (16.0%)	2 (8.0%)	1 (6.3%)	0.556	
Medications							
ACE	Males	5 (20.83%)	6 (37.5%)	14 (45.2%)	7 (24.1%)	0.180	

inhibitors	Females	10 (29.4%)	7 (28%)	8 (32%)	4 (25%)	0.969	
ARBs	Males	3 (12.5%)	3 (18.8%)	7 (22.6%)	7 (24.1%)	0.729	
	Females	7 (20.6%)	5 (20%)	9 (36%)	2 (12.5%)	0.305	
Beta	Males	8 (33.3%)	7 (43.8%)	9 (29%)	9 (31%)	0.775	
blockers	Females	8 (23.5%)	8 (32%)	6 (24%)	3 (18.8%)	0.792	
Nitrates	Males	6 (25%)	4 (25%)	12 (38.7%)	6 (20.7%)	0.438	
	Females	8 (23.5%)	6 (24%)	10 (40%)	7 (43.8%)	0.305	
Statins	Males	10 (41.7%)	5 (31.3%)	9 (29%)	12 (41.4%)	0.680	
	Females	8 (23.5%)	6 (24%)	8 (32%)	7 (43.8%)	0.458	
Calcium	Males	5 (20.8%)	2 (12.5%)	9 (29.0%)	2 (6.9%)	0.142	
antagonists	Females	4 (11.8%)	5 (20%)	1 (4%)	1 (6.3%)	0.295	
Diuretics	Males	3 (12.5%)	1 (6.3%)	6 (19.4%)	3 (10.3%)	0.585	
	Female	2 (5.9%)	4 (16%)	3 (12%)	1 (6.3%)	0.572	
ASA	Males	12 (50%)	3 (18.8%)	13 (41.9%)	9 (31%)	0.188	
	Females	17 (50%)	4 (16%)	5 (20%)	4 (25%)	0.017*	P1=0.007* P2=0.018* P3=0.095 P4=0.713 P5=0.689 P6=0.706
Clopidogre I	Males	2 (8.3%)	1 (6.3%)	8 (25.8%)	1 (3.4%)	0.038*	P1=1.00 P2=0.159 P3=0.584 P4=0.138 P5=1.00 P6=0.026*
	Females	3 (8.8%)	3 (12%)	2 (8%)	1 (6.3%)	0.929	
Symptoms	Males	17 (70.8%) 7 (29.2%)	10 (62.5%) 6 (37.5%)	26 (83.9%) 5 (16.1%)	24 (82.8%) 5 (17.2%)	0.286	
	Females	17 (50.0%) 17 (50.0%)	22 (88.0%) 3 (12.0%)	18 (72.0%) 7 (28.0%)	12 (75.0%) 4 (25.0%)	0.016*	
			Vital signs				
HR (beats /min)	Males	82.25 ±7.62	79.69±7.73	84.52 ±8.27	81.41 ±7.76	0.213	
	Females	83.03±7.022	82.24±7.73	82.04±7.65	82.88±6.11	0.950	
SBP (mmHg)	Males	137.08±10.83	138.75±9.57	135.48±9.61	141.38±9.90	0.142	
	Females	137.65±10.17	139.6±8.89	134±10.408	137.5±10.65	0.260	
DBP (mmHg)	Males	78.33 ±8.68	78.75 ±7.19	78.39±7.35	81.03±7.72	0.514	
	Females	80±7.38	80±7.638	80.8±7.59	80±7.30	0.975	
			Laboratory investigation				
HbA1c (%)	Males	5.663 ±1.04	6.12 ±1.18	6.35 ±1.22	7.81 ±1.99	0.040*	P1=0.209 P2=0.030* P3<0.001* P4=0.539 P5<0.001* P6=0.001*
	Females	5.25 ±1.14	5.716 ±1.22	6.02±1.087	6.97±1.38	<0.001*	P1=0.146 P2=0.0114 P3<0.001* P4=0.370 P5*=0.003* P6=0.018*
Serum creatinine (mg/dL)	Males	0.99± 0.11	0.92± 0.13	0.99± 0.12	0.99± 0.13	0.271	
	Females	0.99 ±0.13	1.02 ±0.12	0.95 ±0.11	0.95 ±0.13	0.207	
PT (sec)	Males	11.71± 0.42	11.54± 0.46	11.75± 0.41	11.75± 0.5	0.426	
	Females	11.87 ±0.48	11.64 ±0.39	11.77±0.52	11.82 ±0.49	0.334	
PTT (sec)	Males	29.5± 3.3	28.56± 2.83	28.81± 2.95	28.97± 2.76	0.762	
	Females	30.29± 2.89	29.96± 3.42	29.64± 3.13	29.75± 3.19	0.869	
INR	Males	1.02± 0.14	1.03± 0.15	0.95± 0.15	0.98± 0.13	0.218	
	Females	0.98 ±0.13	1.01 ±0.14	0.95 ±0.16	1.04 ±0.15	0.229	
Cholestero	Males	178.71± 19.8	176.5± 22.2	178.2± 21.4	186.6± 18.7	0.292	

I (mg/dL)	Females	148.08± 10.7	153.4± 10.7	157.98± 9.3	167.6± 8.45	<0.001*	P1=0.197 P2=0.002* P3<0.001* P4=0.370 P5<0.001* P6=0.018*
Triglycerides (mg/dL)	Males	156.25± 50	155.6± 40.8	165.5± 49.9	195.9± 53.4	0.013*	P1=1.00 P2=0.902 P3=0.024* P4=0.916 P5=0.004* P6=0.026*
	Females	110.62± 17.45	118± 15.8	119.6± 15.04	144.75± 15.14	<0.001*	P1=0.309 P2=0.155 P3<0.001* P4=0.985 P5<0.001* P6<0.001*
HDL (mg/dL)	Males	39.88± 3.07	39.56± 3.5	39.55± 2.89	38.93± 2.93		0.715
	Females	51.71± 6.55	48.72± 4.87	47.16± 4.48	44.38± 3.61	<0.001*	P1=0.144 P2=0.008* P3<0.001* P4=0.722 P5=0.055 P6=0.355
LDL (mg/dL)	Males	98.17± 17.9	98.38± 13.6	103.5± 16.4	115.6± 17.9	<0.001*	P1=1.00 P2=0.652 P3=0.002* P4=0.758 P5=0.008* P6=0.032*
	Females	82.24± 8.33	85.96± 7.88	86.88± 8.06	94.5± 4.78	<0.001*	P1=0.262 P2=0.107 P3<0.001* P4=0.974 P5=0.004* P6=0.014*

Data presents as mean ± SD or frequency (%) *statically significant as p value <0.05. P1: P Value between Normal and Mild, P2: P Value between Normal and Moderate, P3: P Value between normal and Severe. P4: P Value between Mild and Moderate, P5: P Value between Mild and Severe, P6: P Value between Moderate and Severe. Normal (>30 ng/mL), mild deficiency (20-30 ng/mL), moderate deficiency (10-20 ng/mL), and severe deficiency (<10 ng/mL).). BMI: Body mass index. HTN: hypertension, DM: diabetes mellitus, MI: myocardial infarction, PCI: percutaneous coronary intervention, CABG: coronary artery bypass graft surgery, COPD: chronic obstructive pulmonary disease, MI: myocardial infarction, ACE: angiotensin-converting enzyme, ARBs: angiotensin receptor blockers, ASA: acetylsalicylic acid, HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, HbA1c: glycated haemoglobin, CRP: c-reactive protein, PT: percussion therapy, PTT: Partial thromboplastin time, INR: international normalized ratio, HDL: high-density lipoprotein, LDL: low-density lipoprotein.

Table 3: ECG on admission, ejection fraction, syntax score, angiographic findings and adverse events

			Normal	Mild deficiency	Moderate deficiency	Severe deficiency	P value	
ECG on admission								
Male	STEMI		2 (8.33%)	1 (6.25%)	5 (16.13%)	6 (20.69%)	0.715	
	Non-STEMI		7 (29.17%)	6 (37.5%)	6 (19.35%)	10 (34.48%)		
	Old myocardial infarction		3 (12.5%)	1 (6.25%)	4 (12.9%)	5 (17.24%)		
	Chronic stable angina		4 (16.67%)	1 (6.25%)	5 (16.13%)	2 (6.9%)		
	Unstable angina pectoris		8 (16.67%)	7 (43.75%)	11 (35.48%)	6 (20.69%)		
Female	STEMI		1 (2.9%)	1 (4%)	3 (12%)	4 (25%)	0.272	
	Non-STEMI		7 (20.6%)	3 (12%)	6 (24%)	2 (12.5%)		
	Old myocardial infarction		2 (5.9%)	3 (12%)	4 (16%)	1 (6.3%)		
	Chronic stable angina		15 (44.1%)	7 (28%)	7 (28%)	5 (31.3%)		
	Unstable angina pectoris		9 (26.5%)	11 (44%)	5 (20%)	4 (25%)		
Ejection fraction EF (%)	Males		60.75± 4.95	55.38± 4.16	53.42± 4.49	49.66± 3.59	<0.001*	P1=0.001* P2<0.001* P3<0.001* P4=0.458 P5<0.001* P6=0.006*
	Females		59.44± 5.22	57.08± 3.81	54.36± 4.32	53.06± 5.77	<0.001*	P1=0.246 P2<0.001* P3<0.001* P4=0.191 P5=0.049* P6=0.832
Syntax score	Males		10.79 ±5.39	10.19 ±4.83	27.65 ±3.23	36.41 ±4.62	<0.001*	P1=0.097 P2<0.001* P3<0.001* P4<0.001* P5<0.001* P6<0.001*
	Females		12.50 ±6.82	12.40 ±6.22	27.92 ±6.04	34.06 ±7.27	<0.001*	P1=1.00 P2<0.001* P3<0.001* P4<0.001 P5<0.001* P6=0.022*
Angiographic findings								
Affected vessels	Males	1-vessel CAD	12 (50%)	5 (31.25%)	12 (38.71%)	8 (27.59%)	0.736	
		2-vessel CAD	6 (25%)	5 (31.25%)	10 (32.26%)	9 (31.03%)		
		3-vessel CAD	6 (25%)	6 (37.5%)	9 (29.03%)	12 (41.38%)		
	Females	1-vessel CAD	6 (25%)	6 (37.5%)	9 (29.03%)	12 (41.38%)	0.558	
		2-vessel CAD	19 (55.9%)	15 (60.0%)	12 (48.0%)	9 (56.3%)		
		3-vessel CAD	7 (20.6%)	6 (24.0%)	3 (12.0%)	4 (25.0%)		
Type of vessel	Male	LAD	15 (62.5%)	8 (50%)	17 (54.8%)	17 (58.6%)	0.872	
		LCX	7 (29.2%)	9 (56.3%)	18 (58.1%)	12 (41.4%)	0.140	
		RCA	8 (33.3%)	8 (50%)	8 (25.8%)	10 (34.5%)	0.430	
		OM	3 (12.5%)	3 (18.8%)	7 (22.6%)	8 (27.6%)	0.594	
		Diagonal	6 (25%)	2 (12.5%)	5 (16.1%)	6 (20.7%)	0.748	
		PDA	3 (12.5%)	2 (12.5%)	5 (16.1%)	2 (7.1%)	0.771	
		Ramus intermedius	2 (8.3%)	1 (6.3%)	3 (9.7%)	1 (3.6%)	0.821	
	Female	LAD	9 (26.47%)	5 (20%)	9 (36%)	6 (37.5%)	0.651	
		LCX	6 (17.6%)	2 (8%)	6 (24%)	4 (25%)	0.417	
		RCA	3 (8.8%)	3 (12%)	2 (8%)	4 (25%)	0.353	

Lesion characteristics	Male	OM	4 (11.76%)	3 (12%)	3 (12%)	3 (18.8%)	0.798	
		Diagonal	3 (8.8%)	1 (4%)	3 (12%)	3 (18.8%)	0.734	
		PDA	3 (8.8%)	1 (4%)	3 (12%)	2 (12.5%)	0.363	
		Ramus	0 (0%)	1 (4%)	2 (8%)	1 (6.3%)	0.445	
		intermedius						
		Lesion length (mm)	24.83 ± 7.27	25.25 ± 6.6	25.65 ± 6.13	28.21 ± 5.96	0.225	
		Severity	< 50%	1 (4.17%)	1 (12.5%)	4 (12.9%)	1 (3.45%)	0.470
	Female		50-70%	0 (0%)	0 (12.5%)	6 (19.35%)	8 (27.59%)	0.008*
			> 70%	0 (0%)	2 (12.5%)	11(35.48%)	17 (58.62%)	<0.001*
		Chronic occlusion	3 (12.5%)	4 (25.0%)	4 (12.9%)	3 (10.3%)	0.571	
		Thrombus	1 (4.2%)	2 (12.5%)	0 (0.0%)	1 (3.4%)	0.228	
		Lesion length (mm)	20.65 ± 5.29	20.36 ± 6.1	18.40 ± 5.94	20.56 ± 6.45	0.475	
		Severity	< 50%	4 (11.8%)	5 (20%)	10 (40%)	5 (31.3%)	0.072
			50-70%	1 (2.9%)	1 (4%)	1 (4%)	4 (25%)	0.023*
			> 70%	0 (0%)	0 (0%)	1 (4%)	5 (31.3%)	<0.001*
Adverse events	Chronic occlusion	2 (5.9%)	1 (4.0%)	0 (0.0%)	1 (6.3%)	0.667		
	Thrombus	0 (0.0%)	1 (4.0%)	0 (0.0%)	1 (6.3%)	0.363		
Bleeding	Males	0 (0%)	0 (0%)	1 (3.2%)	2 (6.9%)	0.431		
	Females	0 (0%)	0 (0%)	0 (0%)	1 (6.3%)	0.151		
Reinfarction	Males	0 (0%)	0 (0%)	2 (6.5%)	6 (20.7%)	0.019*		
	Females	0 (0%)	0 (0%)	1 (4%)	3 (18.8%)	0.009*		
Mortality	Males	0 (0%)	0 (0%)	0 (0%)	5 (17.2%)	0		
	Females	0 (0%)	0 (0%)	0 (0%)	2 (12.5%)	0.013*		

STEMI: ST-elevation myocardial infarction, EF: ejection fraction, LAD: left anterior descending artery, LCX: left circumflex artery, RCA: right coronary artery, OM: obtuse marginal and PDA: posterior descending artery.

Table 4:Relation between syntax score and severity and the affected vessels

		Syntax score	P-value	
Severity	No	16.61± 10.06	<0.001*	P1=0.023*
	<50%	22.35± 10.74		P2<0.001*
	50-70	29.83± 9.42		P3<0.001*
	>70%	31.59± 8.64		P4=0.033*, P5=0.001*, P6=0.911
Affected vessels	1-vessel CAD	22.00 ± 11.7	0.886	
	2-vessel CAD	21.28 ± 12.2		
	3-vessel CAD	21.12 ± 11.1		

Data was presented as statistically significant different as p value <0.05, P1: P Value between 0 and <50%, P2: P Value between 0 and 50-70%, P3: P Value between 0 and >70%, P4: P Value between <50 and 50-70%, P5: P Value between <50% and >70%, P6: P Value between 50-70% and >70%.CAD: coronary artery disease

Table 5: Correlation between Syntax score and other parameters

		Syntax score	
		r	P
Number of affected vessels		0.305	<0.001*
Vit D level (ng/ml)		-0.751	<0.001*

Data was presented as statistically significant different as p value <0.05.r: correlation coefficient

Table 6: Multivariate logistic regression analysis for prediction of CAD severity

	OR	95% CI	P value
Age (years)	0.9583	0.8759 to 1.0485	0.354
Sex	0.1513	0.0324 to 0.7073	0.016*
Smoking	0.1789	0.0505 to 0.6339	0.008*
HTN	0.6941	0.3196 to 1.5073	0.356
Cholesterol (mg/dL)	1.0149	0.9950 to 1.0351	0.144
Triglycerides (mg/dL)	1.0144	0.9946 to 1.0346	0.155
HDL (mg/dL)	1.0277	0.9825 to 1.0750	0.234
LDL (mg/dL)	0.9780	0.9419 to 1.0155	0.247
Chronic occlusion	6.535	1.7393 to 24.5544	0.005*
Lesion length	0.969	0.9116 to 1.0300	0.312
Syntax	1.0170	1.0013 to 1.0329	0.033*
Vessel affected	1.1171	0.7035 to 1.7737	0.639
Vit D (ng/ml)	1.0176	1.0018 to 1.0337	0.028*

HTN: hypertension, HDL: high density lipoprotein, LDL: low density lipoprotein, OR: odds ratio, CI: confidence interval, *: statistically significant as p value <0.05.

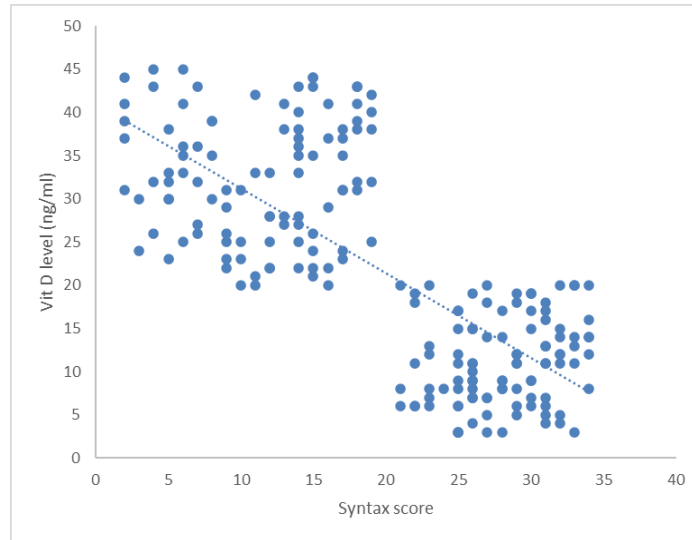


Figure 1:Correlation between Syntax score and Vit D level.

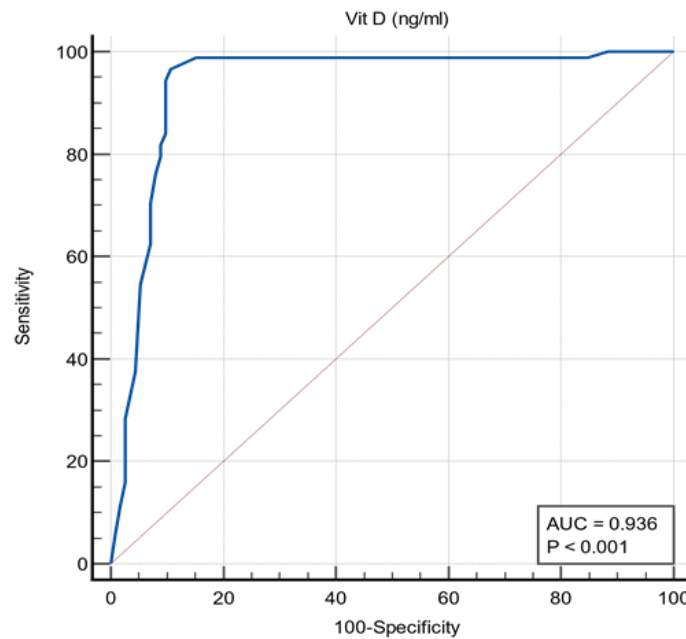


Figure 2: ROC curve analysis of Vit D level for prediction of Severity of CAD

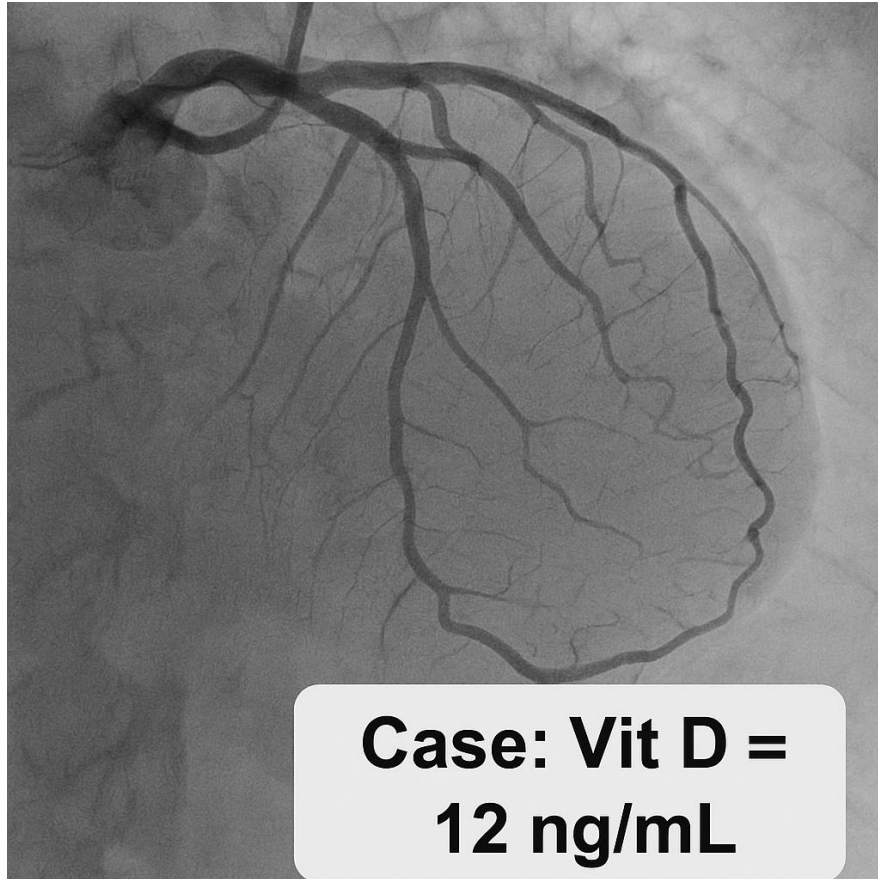


Figure 3. Representative grayscale coronary angiography image illustrating the left and right coronary arteries opacified with radiopaque contrast. The case presented shows a serum 25-hydroxyvitamin D level of 12 ng/mL, which falls within the severe deficiency range (<10–20 ng/mL), and is associated with a higher SYNTAX score and increased coronary artery disease complexity according to the study findings.

Discussion

Our research revealed that the average age of females was significantly higher than that of males ($P < 0.001$). These results are parallel to Gilabert-Garcia et al., (13) who noted that men are generally at a higher risk of CAD than women, especially in younger years, the risk increases in both sexes as they age, and by age 65 and older, the prevalence and impact of CAD are similar in both men and women.

Regarding the risk factors, smoking, history of MI, HTN, history of MI, previous PCI and CABG in males were significantly higher than females ($P < 0.05$). According to our findings, Yasmin et al., (14) proved that males are more susceptible to having a history of MI in the context of CAD than females. This is reflected in both higher incidence rates of MI in men and a greater likelihood of presenting with a history of MI compared to women.

Contrary, Jeong et al., (15) demonstrated that in comparison to non-smokers, female smokers have a 25% higher incidence of CAD than male smokers.

Regarding the results, total cholesterol, triglycerides, and LDL were significantly increased in females than males ($P<0.05$). HDL was significantly lower in males than females ($P<0.001$). Additionally, Holven & van Lennep al., (16) revealed that women have higher HDL-C levels and lower LDL-C levels, while LDL-C levels increase and HDL-C levels decrease in men from early adulthood to middle age.

According to our investigation, males showed significantly lower Vit D levels than females ($P=0.014$). The Vit D status of both groups was significantly different ($P=0.044$) as males exhibited a significantly higher prevalence of moderate and severe deficiency than females.

As well, Haitchi et al., (17) conducted a retrospective study and found that Vit D levels in males were significantly lower than those of the females.

Our study found that the ECG findings on admission were significantly different between both groups ($P=0.005$). Carbone et al., (18) confirmed our results as they showed that ECG findings in (CAD) can differ between males and females, impacting diagnostic accuracy and interpretation. While some findings are similar, others are more prevalent in one sex than the other.

Regarding the angiographic findings, SYNTAX score, LAD, LCX, RCA affected vessels, lesion length, severity (50-70%, >70%), and chronic occlusion in males were significantly higher than females ($P<0.05$) while 1-vessel CAD and lesion severity <50% were significantly higher in females than males ($P=0.011$, <0.001). These results are agreed with Chiha et al., (19) who demonstrated that women may have a higher prevalence of mild, single-vessel CAD than men. Also, severe CAD, particularly high-risk CAD like left main artery stenosis, three-vessel disease, or two-vessel disease with proximal LAD involvement, is more frequent in men.

Based on the vit D status, smoking was significantly higher in the moderate and severe deficiency than normal among males ($P<0.001$, <0.001), was significantly higher in the moderate and severe deficiency than mild deficiency ($P<0.001$, <0.001), and smoker number was significantly higher in the severe deficiency than moderate deficiency ($P<0.001$). In alignment with our study, Yang et al., (20) illustrated that smoking negatively affects circulating Vit D levels in adults.

Based on the vit D status, among females, ASA was significantly lower in mild and moderate deficiency groups than normal group ($P=0.007$, 0.018) and among males, clopidogrel was significantly higher in severe deficiency than moderate deficiency group ($P=0.026$).

Based on the vit D status, among males, Triglycerides and LDL in the severe group were significantly higher than the normal, mild, and the moderate deficiency groups ($P<0.05$), HbA1c was significantly higher in moderate deficiency than normal group ($P=0.030$). HbA1c and total cholesterol in females, were significantly higher in severe deficiency than normal, mild, and moderate deficiency groups ($P<0.05$), was significantly higher in moderate deficiency than normal group ($P<0.05$). HDL was significantly lower in moderate and severe deficiency compared to normal group ($P=0.008$, <0.001).

These findings are in accordance with Hwang et al., (21) who determined that severe Vit D deficiency is associated with a significant increase in HbA1c levels, particularly in individuals with abnormal HbA1c values. This suggests that Vit D deficiency may contribute to poorer glycemic control in diabetic men, potentially leading to higher blood glucose levels over time.

On the other hand, Autier P et al. (22) conducted a meta-analysis that revealed that, Vit D supplementation did not produce a statistically significant difference in total cholesterol, triglycerides, LDL-C, and HDL-C.

On the basis of Vit D status, the EF of males was significantly lower in the severe deficiency group than in the normal, mild, and moderate deficiency groups ($P<0.001$, <0.001 , 0.006), and in

the mild and moderate deficiency groups than in the normal group ($P=0.001$, <0.001). In the moderate and severe deficiency groups, EF was significantly lower than that of the normal group among females ($P<0.001$, 0.001), and it was significantly lower in the severe deficiency group than in the mild deficiency group ($P=0.049$).

These results are consistence by Wu et al., (23) who asserted that chronic vit D deficiency may lead to a reduced ejection fraction (EF) in specific heart failure patients, particularly those with a reduced ejection fraction (HFrEF). The syntax scores of the severe deficiency group were significantly higher than those of the other groups ($P<0.05$), and the moderate deficiency group was significantly higher than the normal and mild groups ($P<0.05$) based on the vit D status of males and females.

In terms of the angiographic findings, the severity of CAD was significantly different among the studied groups ($P<0.05$) for males and females (50-70%, $>70\%$), with the severe group exhibiting the highest severity. In agreement with the current findings, Candemir et al., (24) SYNTAX scores were independently predicted by 25(OH)D levels, as determined by a multivariable regression analysis. 25(OH)D levels were significantly reduced in the group with the highest SYNTAX scores. Both the 25(OH)D levels and the SYNTAX scores demonstrated significant negative correlations.

Regarding the adverse events, severe deficiency group had higher incidence of reinfarction and mortality than the other groups ($P<0.005$).

Similarly, Correia et al., (25) found that severe Vit D deficiency is associated with increased incidence of reinfarction and mortality in CAD patients. Specifically, individuals with severe Vit D deficiency have a significantly higher risk of in-hospital cardiovascular mortality compared to those with sufficient levels. This increased risk is independent of other cardiovascular risk factors and persists even after accounting for factors like the GRACE score and Gensini angiographic score.

Our study showed that the relation between syntax score and severity was significant ($P<0.001$), Patients with a severity of $>70\%$ exhibited a statistically higher syntax score than those with no severity or a severity of $<50\%$ ($P<0.001$, 0.001), while the difference between patients with $50-70\%$ severity was not statistically significant. The syntax score was significantly increased in patients with a severity of $50-70\%$ compared to those with no severity and a severity of $<50\%$ ($P<0.001$, 0.033), and it was also significantly elevated in patients with a severity of $<50\%$ compared to those with no severity ($P=0.023$).

These findings are assured by Rasheed et al., (26) who declared that there is a significant relationship between SYNTAX score and the CAD severity. Higher SYNTAX scores indicate more

complex and severe CAD and are associated with poorer prognosis and increased need for revascularization.

In our investigation, the Syntax score and Vit D level were found to have a significant negative correlation. The Syntax score and the number of afflicted vessels exhibited a significant positive correlation. Furthermore, Sahani and Gupta al., (27) in patients with CAD, a significant negative correlation was observed between the Syntax score and Vit D levels. This means that patients with reduced Vit D levels had elevated SYNTAX scores, resulting in more severe and complex CAD.

In our results, Vit D level can significantly predict the severity of CAD with AUC of 0.936 ($P<0.001$), at cutoff value of ≤ 20 ng/ml, with 98.86% sensitivity, 84.82% specificity, 83.7% PPV and 99.0% NPV.

In accordance with the results, Candemir et al., (24) showed that specific Vit D levels, like 13.87 ng/mL, have been shown a high sensitivity (81%) and 80.6% specificity in predicting high SYNTAX scores, in the assessment of CAD.

The limitations of the current study were single-center nature, the relatively small sample size, and the absence of a study on the therapeutic effect of Vit D deficiency on CAD outcome.

Therefore, this study recommended further investigations with larger and stratified sample size, multi-center study

and future studies assessing therapeutic effect of Vit D deficiency on CAD outcome.

Conclusions

There is an association between Vit D and as it can predict the severity of CAD at cutoff value of ≤ 20 ng/ml, with 98.86% sensitivity, 84.82% specificity, 83.7% PPV and 99.0% NPV.

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