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Relationship between vitamin D deficiency and thrombus load in patients with ST-elevation myocardial infarction

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Abstract. – OBJECTIVE: Clinical studies detecting the increase in thrombotic events with vitamin D deficiency note the relationship between vitamin D and thrombosis. This study aims at evaluating the relationship between serum vitamin D levels and coronary thrombus burden.

PATIENTS AND METHODS: We retrospectively evaluated 77 patients with ST-elevated myocardial infarction (STEMI). Serum vitamin D levels, degree of coronary thrombus, Thrombolysis in Myocardial Infarction (TIMI) frame count and the extent and severity of atherosclerosis in coronary arteries were also measured in all cases. Patients were divided into 2 groups, according to thrombus load.

RESULTS: The rate of vitamin D deficiency in the study population was 79.22% (< 20 ng/mL). Vitamin D levels were significantly higher in patients with a mild thrombus load than in patients with a severe thrombus load (16 *vs.* 13.95 $p = 0.018$). Gensini scores were significantly higher in patients with a severe thrombus burden than in patients with a mild thrombus burden (42 *vs.* 54.5 $p = 0.014$). There was a low negative correlation between vitamin D levels and thrombus burden classification grades ($r = -0.304$, $p = 0.007$), Cx TIMI frame counts ($r = -0.402$, $p < 0.001$), and RCA TIMI frame counts ($r = -0.479$, $p < 0.001$). There was a moderate negative correlation between serum vitamin D levels and LAD TIMI frame count ($r = -0.507$, $p < 0.001$).

CONCLUSIONS: The results of our study showed that low 25(OH)D3 levels are an independent predictor of high coronary artery thrombus load and post-procedural TIMI frame count increase in patients with STEMI undergoing primary percutaneous coronary intervention.

Key Words:
Vitamin D, ST elevated, Thrombus burden.

Introduction

ST elevation acute myocardial infarction continues to be the primary cause of mortality and morbidity worldwide, despite advances in pharmacological therapy and mechanical reperfusion therapy[1]. The main cause of STEMI is coronary thrombus that results from atherosclerotic plaque rupture and the consequent cessation of coronary blood flow. Infarct-related coronary artery thrombus load determines the clinical presentation of the patient with acute coronary syndrome, from unstable angina to STEMI[2]. Primary percutaneous coronary intervention (PCI) is the therapy of choice for patients with STEMI. In the early revascularization and responsible lesion, TIMI III degree flow is the main goal for survival. The presence of thrombus in patients with STEMI undergoing PCI has been reported to increase the incidence of major adverse cardiac and cerebrovascular events (MACCE), and a high thrombus load has been reported to be independently associated with stent thrombosis, distal embolization, and no-reflow[3].

Vitamin D is a steroid hormone. A prominent form of vitamin D, which circulates in the blood is 25(OH)D3. Vitamin D receptors are present in a wide variety of tissues, including vascular endothelium, cardiomyocytes, and lymphocytes. 25(OH)D3 deficiency plays a very important role in both chronic inflammatory diseases and in the cardiovascular system[3]. Vitamin D suppresses the renin-angiotensin system, improves endothelial function, and causes anticoagulant effects by increasing the level of thrombomodulin and decreasing the tissue factor[4]. Intracoronary thrombus and the amount of thrombus load are important prognostic determinants in STEMI[5]. Clinical studies[6] detecting the increase

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in thrombotic events with vitamin D deficiency have noted the relationship between vitamin D and thrombosis.

This study aims at evaluating the relationship between serum Vitamin D levels, the degree of coronary thrombus, the TIMI frame count, and the extent and severity of atherosclerosis in coronary arteries in patients with acute STEMI undergoing PCI.

Patients and Methods

Study Population

In this retrospective study, patients who were admitted within the first 12 hours after the onset of chest pain between July 2019 and October 2019, having no history of thrombolytic therapy, and at least a 1 mm (2 mm for V1-V3) ST-segment elevation in two or more consecutive leads on electrocardiography (ECG) were included in this retrospective study. We included 77 patients with acute STEMI, 17 of whom were women, who underwent PCI. All patients were informed about the study and their written consent was obtained. The research was conducted in accordance with the principles set out by the Declaration of Helsinki, the International Good Clinical Practice guidelines and all applicable legal requirements. The study protocol was approved by the Bursa City Hospital Medical Ethics Committee (No. 2021-1/25).

Patients with a prior history of ischemic heart diseases, such as acute myocardial infarction, coronary intervention or coronary artery bypass grafting, chronic liver (aminotransferase activity > 40 IU/L), and kidney failure (glomerular filtration rate [GFR] < 60 mL/min/1.72 m^2), patients with a history of active infection or malignancy, patients with known bleeding, coagulation and platelet dysfunction, patients with chronic systemic and inflammatory diseases, patients with a history of anticoagulant and antithrombotic drug use, patients with known vitamin D metabolism disease, patients who had received thrombolytic therapy prior to coronary angiography and who had undergone thrombus aspiration and Glycoprotein IIb/IIIa inhibitors in coronary angiography were excluded from the study.

Study Protocol

The demographic information and cardiovascular risk factors of all patients were recorded. The left ventricular ejection fraction was measured in the first 24 hours after primary PCI with the modified Simpson method using an echocardiography device (Vivid 7, GE VingmedSound Horten, Norway), according to the recommendations of the American Society of Echocardiography[7].

Gensini scores, TIMI frame rates and serum 25(OH)D levels were measured for all cases. The angiographic severity of coronary artery disease was determined according to the Gensini score. We divided the patients into two groups according to the thrombus burden classification as mild thrombogenicity (Grade 2 and 3) - group A, and severe thrombogenicity (Grade 4 and 5) - group B.

Angiographic Analysis

Emergency coronary angiography and PCI were performed by an experienced interventional cardiologist using a monoplane Innova GS 330 cardiac angiography system (GE Company, USA) with a 6 or 7-F guiding catheter and a femoral approach. Iopromide (Ultravist 370, Schering AG, Berlin, Germany) was used as a contrast agent during the coronary angiography in all patients. Coronary angiograms were recorded in the right and left oblique planes at a rate of 30 frames/s using cranial and caudal angulations.

All patients were administered a 300 mg acetylsalicylic acid, 600 mg clopidogrel loading dose, and an intravenous bolus of heparin (100 U/kg) during coronary angiography to homogenize antiplatelet efficacy in accordance with the 2017 guidelines of the European Society of Cardiology in relation to STEMI management[8].

Routine stenting was done in relation to the artery responsible for the acute myocardial infarction using a drug-eluting stent following direct or balloon angioplasty using various guide-wires.

Coronary angiographic analysis was performed by two expert invasive cardiologists, who did not know the patient's identity and clinical and laboratory characteristics. The evaluation of angiographic thrombus burden was performed according to the classification obtained from the TIMI-14 study (Table I)[9].

Gensini Score

In this method, narrowing of the coronary artery lumen is 1 for 1% to 25% stenosis, 2 for 26% to 50% stenosis, 4 for 51% to 75% stenosis, 8 for 76% to 90% stenosis, and 16 for 91% to 99% stenosis. and scored 32 for complete occlusion. The calculated score is then multiplied by a

Table I. The TIMI thrombus burden classification.

Grade 0	No angiographic evidence of thrombus
Grade 1	Angiographic features suggestive of thrombus (decreased contrast density, haziness of contrast, irregular lesion contour, a smooth convex meniscus at the site of a total occlusion, suggestive, but not firmly diagnostic of thrombus)
Grade 2	Definite thrombi present in multiple angiographic projections (marked irregular lesion contour with a significant filling defect – the greatest dimension of thrombus is < 1/2 vessel diameter)
Grade 3	Definite thrombus appears in multiple angiographic views (greatest dimension from > 1/2 to < 2 vessel diameters)
Grade 4	Definite large size thrombus present (greatest dimension > 2 vessel diameters)
Grade 5	Definite complete thrombotic occlusion of a vessel (a convex margin that stains with contrast, persisting for several cardiac cycles); angiographic detection of a grade 5 TIMI thrombus leads to further exploration of the occlusive thrombotic content. Either a PCI guidewire or a small balloon is advanced across the thrombotic total occlusion. Crossing the thrombus results in restoration of antegrade flow in the treated vessel. Consequently, the ensuing coronary angiogram enables restratification of the underlying residual thrombus (final TIMI thrombus grade).

factor representing the importance of the location of the lesion in the coronary artery system. The multiplication factor for the left main coronary lesion is 5; it is 2.5 for the proximal left anterior descending artery (LAD) and proximal circumflex artery (LCX) lesions; 1.5 for middle LAD lesions; and 1 for lesions of the distal LAD, middle/distal circumflex artery, and right coronary artery (RCA). The multiplication factor for other branches is 0.5[10].

TIMI Frame Count

The TIMI frame number is determined as the sum of the cineangiographic frames during the staining period from the *ostium* to the distal level of the coronary artery. The corrected TIMI frame count in the responsible artery and other coronary arteries after PCI was calculated for each patient. The TIMI frame count was calculated by dividing the value by 1.7, since the LAD is longer compared to either the RCA or the LCX. The proposed normal TIMI frame count levels were 21 ± 2 for LAD, 22 ± 4 for LCX, and 20 ± 3 for RCA[11].

Laboratory Analyses

After a 12-h overnight fast, venous blood samples were taken and collected. The serum 25(OH) D levels were measured by radioimmunoassay, and vitamin D deficiency was defined as a blood 25(OH)D3 level < 20 ng/mL. All inter- and intra-assay coefficients of variation were lower than 5%.

Statistical Analysis

All analyses were performed on SPSS v. 21 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk's test was used to determine whether variables were normally distributed. Data were given as mean ± standard deviation or median (minimum-maximum) for continuous variables, according to normality of distribution and frequency (percentage) for categorical variables. Normally distributed variables were analyzed with the Independent Samples *t*-test. Non-normally distributed variables were analyzed with the Mann-Whitney U test. Categorical variables were analyzed with the Chi-Square test or Fisher's exact test. Spearman correlation coefficients were calculated to evaluate relationships between variables. Two-tailed *p*-values lower than 0.05 were considered statistically significant.

Results

77 patients (60 males and 17 females) were included in the study; mean age was 56.00 ± 11.11 (38-82). A summary of patients' characteristics, biochemical measurements, echocardiography measurements and a summary of coronary artery disease scores and biochemical measurements with regard to thrombus severity are shown respectively in Tables II, III, and IV.

The thrombus loads of patients, who underwent primary PCI, were evaluated. According to the thrombus burden classification, 30 (38.96%) patients were grade 5, 18 (23.38%) patients were grade 4, 17 (22.08%) patients were grade 3 and 12 (15.58%) patients were grade 2. Patients were divided into 2 groups according to thrombus load. Vitamin D levels between group A (Grade 2 and 3) with mild thrombogenicity (48 patients), and group B (Grade 4 and 5) with severe thrombogenicity (29 patients);

Table II. Summary of patients' characteristics.

	Thrombus burden classification severity			
	Mild (grade 2 and 3) (n = 29)	Severe (grade 4 and 5) (n = 48)	Total	*p*-value
Age	56.31 ± 9.72	55.81 ± 11.97	56.00 ± 11.11	0.617
Gender				0.735
Male	22 (75.9%)	32 (79.2%)	60 (77.92%)	
Female	7 (24.1%)	8 (20.8%)	17 (22.08%)	
Heredity	18 (62.07%)	39 (81.25%)	20 (25.97%)	0.063
Smoking	22 (75.86%)	29 (60.41%)	51 (66.23%)	0.627
Diabetes Mellitus	8 (27.58%)	14 (29.16%)	22 (28.57%)	0.165
Hypertension	13 (44.82%)	18 (37.5%)	31 (40.26%)	0.525
Hyperlipidemia	7 (24.13%)	8 (16.66%)	15 (19.48%)	0.423
Chronic Renal Failure	0 (0.00%)	0 (0.00%)	0 (0.00%)	N/A
Drugs				
Acetylsalicylic acid	14 (48.27%)	20 (41.66%)	34 (44.16%)	0.571
Beta-blocker	5 (17.24%)	4 (8.33%)	9 (11.69%)	0.238
ACE Inhibitor	9 (31.03%)	14 (29.16%)	23 (29.87%)	0.862
Spironolactone	0 (0.00%)	0 (0.00%)	0 (0.00%)	N/A

Data are given as mean ± standard deviation for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables.

a comparison of post-procedural TIMI frame count and Gensini score for each coronary artery is shown in Table V.

The rate of vitamin D deficiency in the study population was 79.22% (< 20 ng/mL, 61 patients). Vitamin D levels were significantly higher in patients with a mild thrombus load than in patients with a severe thrombus load (16 *vs.* 13.95, $p = 0.018$). In addition, Gensini scores were significantly higher in patients with a severe thrombus burden than in patients with a mild thrombus burden (42 *vs.* 54.5, $p = 0.014$) (Table VI).

There was a low negative correlation between vitamin D levels and thrombus burden classifi-

Table III. Summary of biochemical measurements.

	MI type			
	Anterior (n = 37)	Inferior (n = 40)	Total	*p*-value
B12	311 (100-2,000)	288 (100-623)	292 (100-2,000)	0.494
Folate	7.36 (3.27-15.7)	5.81 (1.79-14.5)	6.27 (1.79-15.7)	0.028
Homocysteine	12.75 (5.7-62.3)	14.6 (6.9-49)	13 (5.7-62.3)	0.356
Vitamin D	17 (5.4-47.4)	13.9 (5.14-27.9)	15.1 (5.14-47.4)	0.082
WBC (×1000)	11 (5.76-24.25)	11.3 (7.7-15.5)	11.1 (5.76-24.25)	0.943
Hemoglobin	14.5 (9.7-16.9)	13.75 (8.1-16.4)	14 (8.1-16.9)	0.227
Platelet (×1000)	259 (185-441)	261.5 (154-474)	261 (154-474)	0.585
Glucose	164 (87-395)	133 (86-446)	152 (86-446)	0.383
Urea	28.12 ± 8.43	26.38 ± 12.15	27.22 ± 10.50	0.469
Creatinine	0.89 (0.61-2)	0.83 (0.56-1.52)	0.85 (0.56-2)	0.414
Sodium	136.76 ± 3.52	137.80 ± 3.09	137.30 ± 3.32	0.170
Potassium	4.12 ± 0.43	4.11 ± 0.36	4.11 ± 0.39	0.945
Total Cholesterol	182.91 ± 39.30	188.56 ± 40.46	185.77 ± 39.71	0.553
LDL	115.95 ± 37.18	123.34 ± 33.64	119.70 ± 35.37	0.383
HDL	42 (25-70)	39.5 (28-68)	40 (25-70)	0.162
Triglyceride	105 (36-254)	115.5 (29-356)	106 (29-356)	0.469
TSH	1.26 (0.46-3.83)	1.23 (0.42-2.66)	1.26 (0.42-3.83)	0.942

Data are given as mean ± standard deviation or median (minimum-maximum) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables.

Table IV. Summary of echocardiography measurements.

	MI type			
	Anterior (n = 37)	Inferior (n = 40)	Total	*p*-value
Left Ventricle (in diastole)	4.6 (4.1-5.5)	4.5 (4.1-5.2)	4.5 (4.1-5.5)	0.124
Left Ventricle (in systole)	3.1 (2.1-4.3)	3 (2.1-4.1)	3 (2.1-4.3)	0.102
Interventricular septum	1 (0.9-1.1)	1 (0.8-1.2)	1 (0.8-1.2)	0.190
Posterior Wall	1 (0.9-1.1)	1 (0.8-1.2)	1 (0.8-1.2)	0.070
Left Atrium	3.9 (3.1-4.9)	3.7 (3.1-4.4)	3.9 (3.1-4.9)	0.635
Mitral Insufficiency	11 (29.73%)	11 (27.50%)	22 (28.57%)	1.000
Tricuspid Insufficiency	17 (45.95%)	19 (47.50%)	36 (46.75%)	1.000
Aortic Insufficiency	15 (40.54%)	11 (27.50%)	26 (33.77%)	0.333
Ejection Fractional	40 (20-60)	45 (35-60)	45 (20-60)	< 0.001
E wave	0.9 (0.5-1)	1 (0.6-1.2)	0.9 (0.5-1.2)	0.161
A wave	0.9 (0.5-1.2)	0.9 (0.5-1.3)	0.9 (0.5-1.3)	0.929
E/A Ratio	0.9 (0.67-2)	1.11 (0.64-2)	0.91 (0.64-2)	0.311

Data are given as median (minimum - maximum) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables.

cation grades (r = -0.304, p = 0.007), Cx TIMI frame counts (r = -0.402, $p < 0.001$), and RCA TIMI frame counts (r = -0.479, $p < 0.001$). In addition, there was a moderate negative correlation between serum vitamin D levels and LAD TIMI frame count (r = -0.507, $p < 0.001$) (Figures 1-4). All TIMI frame counts (LAD, Cx and RCA) were significantly correlated to each other. There was a significant positive correlation between Gensini scores and thrombus burden classification grades (r = 0.279, p = 0.014), LAD TIMI frame count (r = 0.267, p = 0.020), and RCA TIMI trame count (r = 0.233, p = 0.043) (Table VI).

Discussion

The results of our study showed that low 25(OH)D3 levels are an independent predictor of high coronary artery thrombus load and post-procedural TIMI frame count increase in patients with STEMI undergoing PCI, although there was a negative correlation between vitamin D level and Gensini score, with which we evaluated the relationship between the extent and severity of atherosclerosis in the coronary arteries.

The low vitamin D (< 20 ng/mL) level in the general population is approximately 50%[12]. In our

Table V. Summary of coronary artery disease scores and biochemical measurements with regard to thrombus severity.

	Thrombus burden classification severity		
	Mild (grade 2 and 3) (n = 29)	Severe (grade 4 and 5) (n = 48)	*p*-value
TIMI Frame Count			
LAD	32 (24-38)	32 (24-48)	0.131
Cx	24 (16-38)	23 (16-46)	0.992
RCA	20 (16-28)	22 (14-48)	0.271
Gensini Score	42 (18-86)	54.5 (24-144)	0.014
SYNTAX Score I	11 (3-21.5)	13.75 (3-24.5)	0.160
SYNTAX Score II	24 (11.8-42.7)	24.85 (11.7-51.7)	0.549
B12	290 (100-2,000)	293 (100-492)	0.862
Folate	6.92 (4.62-15.6)	6.06 (1.79-15.7)	0.024
Homocysteine	12.75 (6.9-37.9)	13.3 (5.7-62.3)	0.102
Vitamin D	16 (5.14-47.4)	13.95 (5.4-24.8)	0.018

Data are given as median (minimum-maximum) for continuous variables according to normality of distribution and as frequency (percentage) for categorical variables.

Table VI. Correlations between coronary artery disease scores and biochemical measurements.

		Burden classification	TIMI frame count (LAD)	TIMI frame count (Cx)	TIMI frame count (RCA)	Gensini Score	SYNTAX Score I	SYNTAX Score II
Vitamin D	*r*	-0.304*	-0.507*	-0.402*	-0.479*	-0.176	-0.104	-0.007
	p	0.007	< 0.001	< 0.001	< 0.001	0.128	0.381	0.956
Burden Classification	r		0.188	0.048	0.195	0.279*	0.194	0.091
	p		0.101	0.675	0.089	0.014	0.100	0.445
TIMI Frame Count (LAD)	*r*			0.433*	0.333*	0.267*	0.198	0.098
	p			< 0.001	0.003	0.020	0.092	0.411
TIMI Frame Count (Cx)	*r*				0.391*	-0.034	0.045	0.066
	p				< 0.001	0.774	0.705	0.577
TIMI Frame Count (RCA)	*r*					0.233*	0.167	0.047
	p					0.043	0.159	0.690
Gensini Score	*r*						0.634*	0.334*
	p						< 0.001	0.004
SYNTAX Score I	*r*							0.602*
	p							< 0.001

r: Spearman correlation coefficient. *: Correlation is significant at the 0.05 level (2-tailed).

study population, the rate of patients with low vitamin D levels was 79.22%. In patients with STEMI, a higher proportion with low vitamin D levels compared to the general population was found. In the study of Ng et al[4] in 2013, the rate of low vitamin D was found to be approximately 74% in patients who had had acute myocardial infarction.

In acute STEMI that develops as a result of complete thrombotic occlusion of the coronary artery, the aim of PCI is to provide TIMI III degree flow early, to maximize myocardial salvage and to improve the outcomes of patients after STEMI[1]. However, PCI does not always provide a good result. One of the most common complications of PCI is the no-reflow phenomenon. Moreover, intracoronary thrombus and the density of the thrombus burden are important prognostic determinants in STEMI[6]. Vitamin D receptors are found in a wide variety of tissues, including the vascular endothelium, cardiomyocytes, and lymphocytes[3]. Literature examining the increase in thrombotic events where vitamin D deficiency is present indicate the relationship between vitamin D and thrombosis. Although thrombosis can be in the arterial or venous system, it can cause many serious complications, including myocardial infarction, stroke, ischemia, and venous thromboembolism[5]. In the study of Jorde et al[13], a negative correlation was found between serum 25(OH) vitamin D and Plasminogen activator inhibitor 1 (PAI-1), tissue type plasminogen activator (tPA Ag) and hs-CRP. According to the results of this study, the serum level of vitamin D appears to be

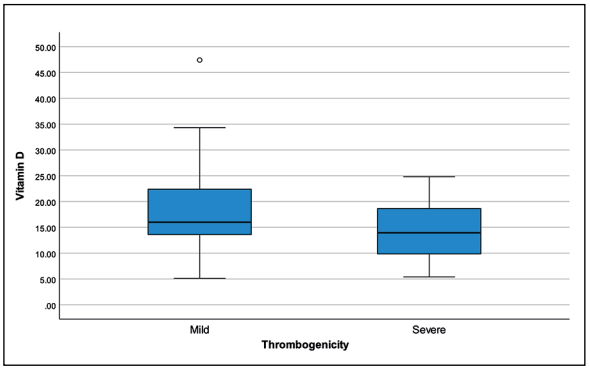

Figure 1. Vitamin D levels with regard to thrombogenicity.

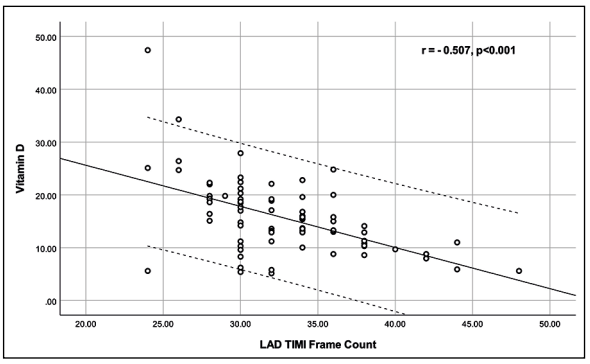

Figure 2. Scatter plot of Vitamin D levels and LAD TIMI frame counts.

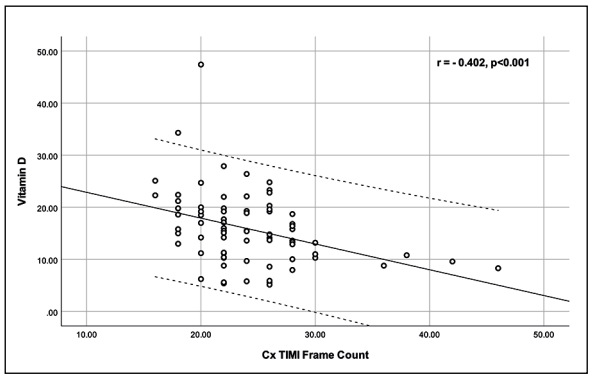

Figure 3. Scatter plot of Vitamin D levels and Cx TIMI frame counts.

associated with fibrinolytic activity and the integrity of the vascular endothelium. Thrombus formation occurs when tissue factor released from damaged endothelial cells and factor VIIa initiate the coagulation cascade. In addition, decreased fibrinolysis and increased platelet adhesion facilitate thrombus formation. Experimental studies[14] have shown that vitamin D has anticoagulant activity. A positive correlation was found between 25(OH)D3 and the tissue factor pathway inhibitor (TFPI) in a study conducted by Topaloglu et al[14]. Therefore, severe deficiency of Vitamin D, which acts as an anticoagulant, may cause an increased thrombus load even in patients with no atherosclerotic burden.

Although the TIMI flow grade is a well-known technique for assessing epicardial blood flow, the TIMI frame count method plays an important role not only in understanding coronary artery blood flow, but also in understanding microvascular function in the presence and absence of acute myocardial infarction. Detection of low TIMI frame counts in the responsible artery after acute myocardial infarction has been shown to be associated with a good prognosis[15]. In our study population, a moderate negative correlation between vitamin D levels and LAD TIMI frame counts and a low negative correlation between Cx TIMI frame counts and RCA TIMI frame counts were present. In their study, Gibson et al[9] showed that TIMI frame count numbers could differ even in the presence of TIMI III flow in patients with acute myocardial infarction, and that lower TIMI frame counts were detected in patients with successful reperfusion.

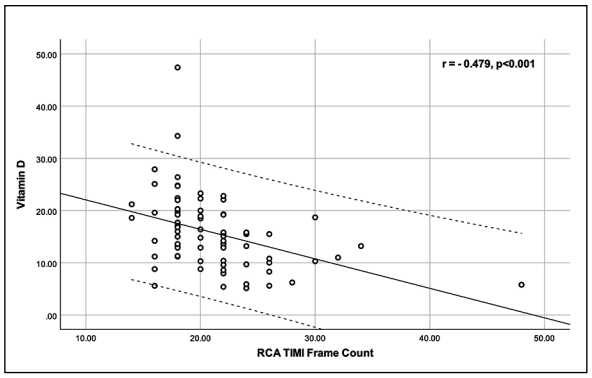

Figure 4. Scatter plot of Vitamin D levels and RCA TIMI frame counts.

Vitamin D deficiency may lead to worse outcomes, even if patients who undergo angiographically successful percutaneous coronary intervention. In a study conducted by Oz et al[16], a strong correlation between vitamin D deficiency, coronary slow flow and endothelial dysfunction was found.

In one study[17], vitamin D3 supplementation was found to have remarkable effects on inflammation levels (mainly interlukin-6 levels) in hospitalized patients with STEMI. In another study[18], similar findings on CRP levels were shown in patients with non-ST-elevation acute coronary syndrome. A recent study[19] has found a relationship between low vitamin D levels and atherosclerotic risk factors, plasma renin activity, hypertension, coronary artery calcification, and cardiovascular diseases. This study has also clarified the role of vitamin D in the pathogenesis of cardiovascular disease through its direct involvement in plaque formation and progression.

In our study, the Gensini scoring system was used to evaluate the severity and extent of coronary artery disease. The purpose of this was to test the relationship between the atherosclerotic burden and vitamin D deficiency. In our study, Gensini scores were found to be significantly higher in patients with STEMI with a severe thrombus load than in patients with a mild thrombus load, while there was a negative correlation between Gensini scores and vitamin D levels, but no statistically significant difference was found. However, vitamin D deficiency may contribute to the progression of atherosclerosis.

Study Limitations

A few limitations need to be mentioned before interpreting the results of this study. Firstly, the study was mono-centric, the patient population was relatively small, and the study was not prospective. It is also important to note that vitamin D levels can change due to a variety of factors,

including seasonal variation, geographic location, exposure to sunlight, dietary habits, and clothing style. In order to see the reflection of the results of our study in the clinic, prospective character studies based on thrombotic events as endpoints are needed and this study is a pioneer study for this.

Conclusions

Vitamin D deficiency is more common in patients with STEMI undergoing PCI than in a healthy population. Patients with low vitamin D levels have a higher thrombus burden and slower coronary flow numbers after the procedure. This results in a worse microvascular performance. This may be related to the prognosis of the patients. No statistical correlation was found between the severity and prevalence of coronary artery disease and vitamin D levels. Vitamin D may be the essential hormone for PCI procedural success in acute STEMI patients, and vitamin D deficiency appears to be a cause of poor prognosis in STEMI patients. Routine measurement of vitamin D levels can be included in the routine laboratory examination of patients with coronary artery disease, and more attention should be paid to vitamin D replacement in cases of deficiency.

Conflict of Interest

The Authors declare that they have no conflict of interests.

Funding

This study received no financial assistance.

Ethics Approval

The study protocol was approved by the Bursa City Hospital Medical Ethics Committee (No. 2021-1/25).

Authors' Contribution

Concept – BU, AK; Design – BU, AK, DT, IZ, Supervision – SKT, MAY, DT,AK; Funding – BU, SCO, IZ, Materials – BU, SCO, IZ, DT, AK; Data Collection and/or processing – BU, AK, SKT, MAY; Analysis &/or interpretation – AK, BU, IZ, DT, SKT, Literature search – BU, IZ; Writing – BU, AK, SCO, DT; Critical review – BU, AK, SKT, MAY, DT.

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