CACS <100 with the Group and CACS> 100 with potential to differentiate group all together on the Effect of Multiple Logistic Regression Analysis of Risk Factors Results

Variables independent	Odds Ratio	95% Confidence Interval	95% Confidence Interval	p-value
		Lower Limit	Upper Limit	
Age	1,055	0,981	1,135	0,149
Male factor	5,984	1,237	28,958	0,026
History of hypertension	0,467	0,125	1,744	0,257
Smoking history	0,551	0,122	2,489	0,438
Left ventricular diastolic dysfunctiondisfonksiyon	0,483	0,127	1,843	0,287
Body mass index	0,821	0,673	1,002	0,053
Total cholesterol	1,018	0,989	1,048	0,231
Low-density lipoprotein cholesterol	1,006	0,975	1,038	0,719
High-density lipoprotein cholesterol	0,968	0,896	1,045	0,398
EATV >167.3	4,682	1,298	16,892	0,018

PP-300

N Terminal pro-Brain Natriuretic Peptide Level is Associated with SYNTAX Score and Aortic Distensibility in Patients with Stable Coronary Artery Disease

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Objectıve: The high N-terminal pro-brain natriuretic peptide (NT-proBNP) level provides significant prognostic information in patients with coronary artery disease (CAD). It is unclear whether aortic distensibility (AD) which reflects aortic stiffness and extent and complexity of CAD assessed with SYNTAX score (SS) affects the secretion of NT-proBNP in stable CAD. We aimed to investigate the relation between NT-proBNP levels with AD and extent and complexity of CAD in stable CAD.
Methods: The study included 411 patients with stable CAD (Mean age=61.7±9.9 years, male/female= 247/164). The patients were divided into two groups according to the median NT-proBNP value (NT-proBNPlow group < 114 pg/ml and NT-proBNPhigh group ≥114 pg/ml). Aortic distensibility was calculated from the echocardiographically derived ascending aorta diameters and hemodynamic pressure measurements. Coronary angiography was performed and SS were determined in all patients. NT-proBNP and other biochemical markers were measured in all subjects.
Results: Aortic distensibility and ejection fraction values of NT-proBNPhigh group were lower and their SS levels were higher compared with NT-proBNPlow group ($p<0.05$, for all) (Table). NT-proBNP level was independently associated with AD ($\beta=-0.378$, $p<0.001$), SS ($\beta=0.262$, $p<0.001$) and ejection fraction ($\beta=-0.295$, $p<0.001$) on multiple linear regression analysis.
Conclusıon: NT-proBNP was independently associated with impaired elastic property of aorta and extent and complexity of CAD, as well as left ventricle systolic dysfunction.

Table. Baseline clinical and laboratory characteristics of patients

Variables	NT-proBNPlow Group (n=205)	NT-proBNPhigh Group (n=206)	p
Age, years	60.3±10.1	63.0±9.5	0.007
SYNTAX Score	14.8±7.3	21.8±9.4	<0.001
Ejection Fraction (%)	65.6±4.3	62.4±4.3	<0.001
Aortic Distensibility (cm2 dyn-1 x10-6)	3.2±1.5	1.7±0.77	<0.001

PP-301

Vitamin D Status and Clinical Severity in ST Segment Elevation Myocardial Infarction

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Purpose: Emerging data revealed the significant role of 25 (OH) vitamin D (vitD) in cardiovascular (CV) events. Clinical indices like Thrombolysis in Myocardial Infarction (TIMI) risk score, corrected TIMI frame count (CTFC) and high sensitivity cardiac troponin T (hs-cTnT) levels have short and long-term predictive values regarding CV mortality and morbidity in ST-segment elevation acute myocardial infarction (STEMI). The aim of this study was to determine the predictive value of vitD for clinical severity parameters in STEMI.
Methods: Patients with STEMI admitted to our hospital were prospectively and consecutively evaluated and proceeded to primary percutaneous coronary intervention (PCI). Patients with a previous history of coronary artery bypass graft (CABG), renal/hepatic failure and patients in need for emergency CABG were excluded from the study. 102 subjects ([mean±SD]age, 57±11 years) were enrolled in the study (female n [%]:18 [17,6%]). VitD levels were obtained on admission. VitD < 20 ng/ml was defined as vitD deficiency. CTFC were calculated after PCI for culprit lesion.
Results: VitD deficiency was detected in 63,4% (<30 ng/ml in 92,7%) of the study population. In vitD deficient group, significantly higher hs-cTnT admission values ([median] 3598 ng/L vs 2576 ng/L, p=0,015), TIMI-STEMI scores (25th-75th percentiles; 2-5 vs 1-3, p<0,001), LAD CTFC (Data±SEM; 18,4±2,3 vs 12,6±1,4 p=0,042) and RCA CTFC (27,5,4±3,6 vs 19,6±1,6 p=0,044) were detected compared with non vitD deficient group. VitD levels were inversely correlated with TIMI STEMI risk scores (r:-0,438, p<0,001). In multivariate regression analyses, vitD levels was found as an independent predictor of higher TIMI-STEMI scores after adjusting for age, gender, HT and DM (Table-1).
Conclusıons: Our findings suggest that low vitD levels might play a role in disease severity of STEMI patients by means of its independent associations with risk algorithms.

Table-1

Variables	β	p	Confidence Interval (95%)
Age (years)	0,327	< 0,04	(0,01 - 0,10)
Gender	0,139	0,44	(-1,29 - 2,91)
HT	0,059	0,78	(-1,49 - 1,97)
DM	0,082	0,66	(-1,45 - 2,26)
VitD	-0,440	0,01	(-0,16 - -0,02)
Regression model for TIMI-STEMI risk score			

PP-302

The Relationship Between Coronary Slow Flow and Urotensin II

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Objectıve: Although there have been a number of studies about coronary slow flow (CSF), the pathophysiologic mechanism still has not been fully understood. There are many proposed mechanisms such as; microvascular vasomotor dysfunction, diffuse atherosclerosis, inflammation, small vessel occlusion, increased microvascular resistance and endothelial dysfunction. Urotensin has 50 times more powerful vasoconstrictive effect than endothelin. It is not known that whether the urotensin plays a role in the pathophysiology of CSF.
Methods: This study included 32 patients with coronary slow flow and 32 patients with normal coronary arteries (NCA). Coronary flow is evaluated by TIMI (Thrombolysis In Myocardial Infarction) frame count (TFC) method. Coronary slow flow diagnosis is considered when TFCs for LAD ≥39, for Cx ≥27, and for RCA ≥24. Urotensin II levels were measured from the blood samples by Elisa method in both groups.
Results: The baseline clinical characteristics were similar in both groups. TIMI frame count was significantly higher in the CSF group than the NCA group ($p<0.001$). Urotensin II levels of SCF group were significantly higher than those of NCA group (122 pg/ml (71-831), 95 pg/ml (21-635), $p<0.001$). Additionally; the LDL levels and leukocyte count were higher in CSF group than the NCA group ($p=0.056$ and $p=0.02$) (Table 1).
Conclusıon: The higher levels Urotensin II in CSF group suggests that Urotensin II is the one of the factors that plays a role in the pathogenesis. Also, the higher white blood cells count in the slow coronary flow group makes us to think that inflammation may play a role in the pathogenesis.

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