

Results: There was male predominance in smokers with CAE. Smokers with CAE had higher MPV and WBC compared with both nonsmokers with CAE and controls (each $p < 0.05$). Platelet count was comparable in smoking and nonsmoking CAE patients. Median hs-CRP level was higher in smokers with CAE than both nonsmokers with CAE [3.2 (2.5 - 3.9) vs 2.9 (2.0 - 3.6) mg/L, $p = 0.01$] and controls [3.2 (2.5 - 3.9) vs 1.9 (1.7 - 2.5) mg/L, $p = 0.001$]. Similarly, serum fibrinogen level was higher in smokers with CAE compared with both non-smokers with CAE (373±68 vs 347±54 mg/dl, $p = 0.02$) and controls (373±68 vs 344±60 mg/dl, $p = 0.02$). Also, smokers with CAE had higher level of D-dimer compared to non-smokers with CAE (262±65 vs 229±83 µg/dl, $p = 0.01$) and controls (262±65 vs 188±61 µg/dl, $p = 0.001$).

Conclusion: Our findings suggest that smoking may induce platelet activation, inflammation and prothrombotic state in CAE patients.

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Serum Nitric Oxide Levels in Patients with Isolated Coronary Artery Ectasia

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Background: Plasma levels of nitric oxide (NO) are decreased in patients with atherosclerosis and in those with risk factors for atherosclerosis. In these patients, reduction of the serum nitric oxide levels are correlated with the severity of endothelial dysfunction and atherosclerosis. Endothelial dysfunction and diffuse atherosclerosis have been proposed for the etiology of coronary artery ectasia (CAE). The aim of this study was to evaluate the relationship between CAE and serum nitric oxide levels.

Methods: The transient and volatile nature of NO makes it unsuitable for most convenient detection methods, however, the plasma levels of nitrite plus nitrate (NOx), two breakdown products, nitrate (NO₃) and nitrite (NO₂) can be detected by photometric methods. We measured plasma levels of NO by photometric methods in 40 patients with isolated coronary artery ectasia and 20 patients with normal coronary arteries as a control group (mean age 58.2±11.7 vs 57.1±12.5, resp. $p = 0.74$).

Results: Plasma nitric oxide concentrations were significantly lower in the CAE group than control group (41.8±22.4 vs 77.3±21.9 µmol/l, $p < 0.001$). We observed statistically significant correlation between decreased level of serum nitric oxide and the presence of isolated coronary artery ectasia ($r = -0.61$, $p < 0.001$). In multivariate analysis serum NO level is a unique independent predictor of presence of coronary artery ectasia (OR=0.93; 95% confidence interval, 0.89-0.97, $p < 0.001$).

Conclusion: We found that serum NO level is decreased in patients with isolated coronary artery ectasia. These findings suggest that decreased NO level may be associated with endothelial dysfunction leading to the development of coronary artery ectasia.

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Mean Platelet Volume Associated with Aortic Distensibility, Chronic Inflammation and Diabetes in Patients with Stable Coronary Artery Disease

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Background: The patients with increased mean platelet volume (MPV) values had a higher risk of developing myocardial infarction and adverse cardiovascular events in stable coronary artery disease (CAD). In this study, we aimed to assess the effective factors on high MPV in patients with stable CAD.

Methods: In all, 411 consecutive patients (247 males and 164 females; mean age: 61.7±9.9 years) with angiographically proven CAD were included in the study. Two different groups were determined according to MPV values (MPVlow group < 9.5 fL, and MPVhigh group ≥ 9.5 fL). Aortic distensibility was calculated from the echocardiographically derived ascending aorta diameters and hemodynamic pressure measurements. Extent and complexity of CAD was calculated by the SYNTAX score. MPV, high sensitive C-reactive protein (hsCRP) and other biochemical markers were measured with an automated chemistry analyzer.

Results: SYNTAX score, hsCRP levels and frequencies of diabetes and hypertension were higher in MPVhigh group compared with MPVlow group ($p < 0.05$, for all). Aortic distensibility value and platelet count of patients with MPVhigh group were lower than patients with MPVlow group ($p < 0.05$, for all). Multivariate linear regression analysis showed that MPV was independently related with diabetes ($\beta = -0.135$, $p = 0.007$), hsCRP ($\beta = 0.259$, $p < 0.001$), platelet count ($\beta = -0.144$, $p < 0.001$) and AD ($\beta = -0.425$, $p < 0.001$). Although MPV was associated with SYNTAX score in bivariate analysis, similar relationship was not observed multivariate analysis ($\beta = -0.034$, $p = 0.579$).

Conclusion: High MPV value in patients with stable CAD is independently related with AD, as well as diabetes, hsCRP and platelet count.

Comparison of baseline, clinical, laboratory, echocardiographic and angiographic findings

Variables	MPVlow Group (n=205)	MPVhigh Group (n=206)	P value
SYNTAX score	10.2±8.0	15.9±9.3	<0.001
hsCRP (mg/dl)	0.83±0.52	1.17±0.63	<0.001
Diabetes mellitus (%)	45 (22.0%)	109 (52.9%)	<0.001
AD (10-6 dyn-1 cm2)	3.1±1.4	1.8±1.2	<0.001

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Coronary Risk Profile and Clinical Features of Subjects with Myocardial Bridging Documented with MSCT Angiography

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Aims: Myocardial bridging is a congenital abnormality of coronary arteries which may cause functionally and anatomically restriction of coronary flow. Thus it may clinically manifest with angina, ischemia, and infarction or lethal arrhythmia during extreme strenuous exercise. We aimed to define whether clinical features of those patients with MB overlap with classical coronary risk factors.

Material-Method: We evaluated medical recordings (ECG, treadmill test results, laboratory findings and coronary risk factors) of 38 subjects with myocardial bridging diagnosed with MSCT angiography.

Results: Study population (age 40.15±4.7) had laboratory findings within normal ranges and low risk profile of atherosclerotic coronary artery disease (CAD). 2 patients had previous history of follow up in ICU, 7 patients had sporadically chest pain on exertion, 21 asymptomatic patients diagnosed detected with treadmill test upon minimal STsegment or T wave changes on resting ECG. Of 30 subjects, 4, 10 and 16 subjects had minimally ST/T wave changes on resting ECG in inferior, lateral and inferior lateral derivations, respectively. Treadmill test was interpreted as equivocal or positive in 32 and 6 subjects, respectively. Of the 36 and 2 patients' bridging was located in the mid and proximal segment of coronary artery, respectively. MB was located on LAD in 32 subjects, whereas it was located on IMA, Cx and RCA in 4, 2 and 2 subjects, respectively.

Conclusion: Patients with MB may have frequently low risk profile for CAD and may clinically manifest with minimal ECG changes and equivocal TT. So it should not be ignored if the subjects have low risk profile for CAD with minimal ECG changes accompanying with angina either typical or atypical. It should certainly be reminded especially in young, young adults, professional sportsmen, military or security personnel who may participate in extremely strenuous exercise. MSCT angiography is the best way of imaging coronary artery and provides anatomically evidence of atherosclerotic disease at any stage or presence of myocardial bridging on any coronary artery and also severity of luminal narrowing due to those lesions. It was documented that those lesions may disturb coronary flow and lead anginal symptoms by limiting coronary vasodilatation or causing vasospasm even they did not critically obstruct the coronary lumen on resting state. Myocardial bridging may not be so much innocent due to its potential to induce ischemia and arrhythmia especially in young and adult subjects.

	(N=38)	Mean±SD	Minimum	Maximum
Age (year)		40.16±4.71	28.00	46.00
Height		176.37±5.07	165.00	185.00
Weight		81.11±4.52	72.00	90.00
Heart Rate		80.6±13.9	52.00	116.00
Fasting blood glucose		91.21±5.38	83.00	105.00
Total cholesterol		201.00±32.77	129.00	263.00
LDL cholesterol		125.58±32.28	68.00	186.00
HDL cholesterol		44.42±5.73	37.00	54.00
Triglyceride		151.32±46.43	55.00	268.00

Coronary risk factors	Yes	No
Family history premature CAD	4	34
Obesity	0	38
Hyperlipidemia	2	36
Hypertension	0	38
Diabetes	0	38
Smoking	4	34
ECG changes	30	8
II, III, aVF	4	
V4, V5, V6	10	
II, III, aVF, V4, V5, V6	16	
Treadmill test results	Positive 6	Equivocal 32
Localization of MB on Coronary artery		
LAD artery	32	Proximal 2 Mid segment 30
IMA artery	4	Mid 4
Cx artery	2	Mid 2
RCA artery	2	Mid 2

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The Relationship of the Glycosylated Hemoglobin A1c Levels with the Severity of the Coronary Artery Disease in Non-diabetic Stable Angina Patients

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Objective: We sought to determine the relationship between the severity of the coronary artery disease measured with the Gensini score and the hemoglobin A1c (HbA1c) levels in non-diabetic patients with stable angina pectoris.

Methods: A total of 93 patients undergoing coronary angiography were included in the study. Patients were divided into 3 groups by use of Gensini score (21 patients with normal coronary arteries, 26 patients with mild atherosclerosis and 46 patients with severe atherosclerotic lesions). The associations between severity of coronary artery disease and HbA1c levels were assessed using logistic regression analysis.

Results: The blood glucose readings were observed to be comparable between the groups (p=0.097). While the HbA1c values were higher in severe atherosclerosis group compared with mild atherosclerosis and normal coronary arteries groups (6.7±1.5, 6.0±0.8 and 5.6±0.6%, respectively, p=0.002). The HbA1c values were observed to be correlated with the Gensini score (r=0.374, p<0.001). A cutoff value of 6.0% for HbA1c predicted severe atherosclerosis with a sensitivity and specificity of 54% and 74%, respectively. In the multivariate analysis, high levels of HbA1c were observed to be independent predictors of severe atherosclerosis (OR: 1.975; 95% CI: 1.101-3.542, p=0.022).

Conclusion: Increasing levels of HbA1c in non-diabetic patients with stable angina pectoris are associated with the severe atherosclerosis that may help to predict the increased risk for coronary artery disease.

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Gender [male]	0.859 (0.369-2.000)	0.725	0.743 (0.272-2.026)	0.561
Glucose [mg/dl]	1.018 (1.000-1.036)	0.048	0.997 (0.974-1.021)	0.822
Creatinine [mg/dl]	3.736 (0.902-15.478)	0.069	2.807 (0.620-12.710)	0.180
HDL [mg/dl]	0.950 (0.908-0.994)	0.025	0.972 (0.924-1.023)	0.275
HbA1c (%)	2.163 (1.305-3.587)	0.003	1.975 (1.101-3.542)	0.022

PP-326

Can Fragmented QRS on 12 Derivation ECG Be Used As a Predictor of In Hospital Mortality in Patients Admitted with Acute ST Elevated Myocardial Infarction?

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Objective: Fragmented QRS (fQRS) is defined as QRS notchings resembling conduction delay in at least 2 neighbouring derivations on 12 derivation surface ECG in the absence of bundle branch block pattern. There are several studies that showed this ECG finding to be related with long term mortality of patients with coronary artery disease. As far as we know there's no study evaluating the relationship between fQRS presence and in hospital mortality in acute ST elevated myocardial infarction (STEMI). Aim of our study is to investigate the relationship between fQRS presence and in hospital mortality in patients admitting for the first time with acute STEMI.

Material-Methods: 248 patients admitted for to Dokuz Eylül University Hospital Cardiology Department for the first time with acute STEMI between 01 January 2009 and 01 July 2011 are included in our study. Patients having ECG findings that can be misdiagnosed as fQRS [incomplete right bundle branch block pattern in V1, pacemaker rhythm, wide QRS complex (QRS >120 ms)] and with CABG history are excluded. All 12 derivation ECG recordings on admission and in 48 hours of admission are investigated for the presence of fQRS. Presence of fQRS is defined as presence of more than one R wave pattern or notching on R or S waves in neighbouring 2 derivations (Figure 1). ECG recordings of patients died from all causes during hospital stay and patients discharged (without mortality) are compared according to presence of fQRS.

Findings: In 91 patients (36.7%) included in our study fQRS was determined. Between groups of patients with fQRS and without fQRS there was no significant difference in MI localization (anterior MI: 38.5% vs 45.2%, p>0.05). In hospital mortality was found to be significantly higher in patients with fQRS than in patients without it. (14.3% vs 4.5%, p=0.006) (Figure 2). Additionally when compared to patients without fQRS, patients with fQRS were found to have lower left ventricular ejection fraction (35±7% vs 47±6%; p<0.001), higher leukocyte counts (12.958±3.07 vs 10.780±3.38; p<0.001), higher maximum troponin levels (62.73±53.49 vs 29.71±16.17; p<0.001) and longer QRS durations (107.86±8.95 ms vs 102.77±9.21 ms; p<0.001) (Table 1).

Result: In patients with acute STEMI fQRS presence on surface ECG is not related with MI localization whereas it is related with increased in hospital mortality. Also supporting this in acute STEMI patients presence of fQRS may help to determine high risk patients with larger infarct size.

