Table 1

	SAP (N:93)	ACS (N:58)	p value
age	59,54+/-12,5	61,2+/- 14,9	0,375
sex (F/M)	47/46	24/34	0,316
WC	90,6+/-9,6	88,5+/-8,4	0,480
BMI	27,7+/-3,5	26,9+/-1,92	0,169
HT(%)	53,8	62,1	0,398
DM(%)	29	39,7	0,214
smoker(%)	49,5	56,9	0,406
FH(%)	32,3	25,9	0,467
HPL (%)	51,6	58,6	0,502

Baseline characteristics of the study population. p<0,05 is considered significant. SAP:stable angõna pectoris, ACS: acute coronary syndrome, BMI:body mass index, WC:waist circumference, HT:hypertension, DM: diabetes mellitus, FH:family history, HPL: hyperlipidemia

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Relationship between Aortic Valve Calcification and the Development of Coronary Collateral in Patients with Coronary Artery Disease

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Introductıon: Recent data suggest that angiogenesis have an important role in valve diseases. Aortic valve calcification considered as active athero-inflammatory disease which is characterized by the accumulation of inflammatory cells and neovascularization of the valves. In the literature,studies that show that some of the mediators involved in the development of aortic valve calcification is also associated with the development of coronary collateral. The aim of this study was to investigate the presence of aortic valve calcification on the development of coronary collateral.
Methods: In our study, 44 patient who underwent coronary angiography in our department and at least one major epicardial coronary artery with complete occlusion or stenosis of 90% or higher and have an aortic valve calcification in echocardiography were included. As a control group of 52 patients with aortic valve calcification was elected with the same specifications and coronary anatomy were selected. Collateral classified according to the classification of Rentrop as 0,1,2,3.
Results: In aortic valve calcification group, age (72.1±9.2 and 68.6±10.3, p=0.09), LDL (168.4±41.6 and 143.1±43.1, p=0.08), CRP (2.4±1.9 and 1.5±1.4, p=0.02) was found to be higher than the group without aortic valve calcification. Multivessel disease was significantly higher in the group with aortic calcification (p=0.001). Also development of collateral was greater in the group of aortic valve calsification (p=0.001).; When the group of collateral compared with group of without collateral, aortic calsification (p=0.008), and one or more vessels ≥90% stenosis rates (p=0.04) were found to be more than the group without collateral. In the regression analysis, the presence of aortic calcification (β=0.3, t=3.9, p=0.01), and ≥1 vessels> = 90% stenosis (β=0.5, t=5.6, p=0.001) seen as two independent parameters affecting the development of collateral.
Conclusıon: In our study, the presence of aortic valve calcification is associated with the development of coronary collateral. Given athero-inflammatory etiopathogenesis of aortic valve calcification, in this process increased tissue cover inflammatory factors were thought to be induced coronary collateral development.

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Association Between Neutrophil/Lymphocyte Ratio and the Development of Coronary Collateral Circulation in Patients with Stable Coronary Artery Disease

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Objectıve: Several studies have established the important role of CRP in the development of coronary collateral circulation. The correlation between neutrophil/lymphocyte (N/L) ratio and collateral formation in patients with stable coronary artery disease (CAD) has not been reported.
Methods: We investigated the association between N/L ratio and the development of coronary collaterals in a cohort of 152 patients who had high-grade coronary stenosis or occlusion on their angiograms. To classify coronary collateral circulation, we used the Rentrop classification.
Results: Patients with poorly developed coronary collateral circulation had significantly higher N/L ratio compared with those with well-developed coronary collateral circulation, (4.2±4.1 vs. 3.1±2.6, p=0.039), whereas mean platelet volume (MPV), red blood cell distribution width (RDW) and uric acid were not significantly different. Logistic regression analysis showed that N/L ratio was an independent predictor of poorly developed coronary collateral circulation (odds ratio 0.752, 95% confidence interval 0.593–0.993).
Conclusıon: An elevated level of N/L ratio is independently associated with a significant impairment in coronary collateralization; patients with poorly developed collaterals tend to have a higher N/L ratio.

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Is Serum S100 Protein Associated with the Angiographic Severity of Coronary Artery Disease in Acute Coronary Syndromes?

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Background: S100, a calgranulin family protein released from white blood cells, is involved in inflammatory cardiovascular disease. It was hypothesized that the plasma level of S100 can be used to predict outcome in patients with chronic coronary artery disease (CAD). We aimed to determine the relationship between S100 protein levels and angiographic SYNTAX score, which gives information about the severity and complexity of CAD in patients with acute coronary syndromes.
Methods: This pilot study included 77 patients who were admitted to the emergency room for the evaluation of the angina pectoris. According to the clinical status and cardiac enzyme levels the patients had undergone coronary angiography. The serum S100 protein levels were measured at the administration. The independent association between serum S100 protein and the severity of CAD was statistically evaluated using PASW Statistics 18 for Windows.
Results: Mean age of the study population was 61.27±13.50 years, of whom 39 were female (50.6%) and 38 male (49.4%). Of the patients, 23.4% had diabetes mellitus, 63.6% had hypertension, 44.2% had hyperlipidemia, and 39.0% were smokers. Mean SYNTAX score was 12.5±12.2. According to SYNTAX scores, 59 of the patients (76.6%) had no significant CAD or normal coronary arteries (SYNTAX score:0-22), 18 of the patients (23.4%) had moderate to severe CAD (SYNTAX score ≥23). Mean serum S100 protein values were 0.37±0.90 µg/l in the group that had normal coronary arteries, 0.20 ± 0.46 µg/l in the group with NSTEMI, and 0.11±0.12 µg/l in the group with STEMI. According to Spearman analysis, no correlation was found between serum S100 protein and SYNTAX score (p=0.284, r=0.124). Also, there was no statistically significant correlation between s100 and troponin-t levels (p=0.051, r=0.256).
Conclusıons: Previously, it was reported that, rising levels of serum S100 protein was a specific and sensitive clinically relevant marker of acute coronary syndromes. Contrary to the literature, we did not determine any correlation between S100 protein levels and SYNTAX score. It can be explained by the small-scale of the study. Larger-scale studies should be performed to shed light on this topic.

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The Association between Coronary Flow Rate and Impaired Heart Rate Recovery in Patient with Metabolic Syndrome

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Objectıve: The aim of this study is to evaluate heart rate recovery at various time intervals, and the association between coronary flow rate and impaired heart rate recovery in patients with metabolic syndrome who had morphologically normal coronary angiogram. To our knowledge there is no published data indicating this association in metabolic syndrome patients.
Material-Methods: The study population included 43 patients with metabolic syndrome and 37 control subjects without metabolic syndrome. All patients were selected from the individuals who had recently underwent coronary angiography in our hospital with a suspicion of coronary artery disease and diagnosed as having angiographically normal coronary arteries. Exercise stress test results of the patients obtained prior to the coronary angiography were evaluated for calculating heart rate recovery values and other parameters. In addition, coronary flow was objectively evaluated for each major coronary artery in each subject using the TIMI frame count method.
Results: Baseline clinical characteristics of patients with MS and control patients were presented in Table 1. In our study, all heart rate recovery values calculated were detected significantly lower in the metabolic syndrome group compared to the control group (heart rate recovery first: 32±9 vs 37±10; p=0,01, heart rate recovery second: 46±11 vs 52±11; p=0,03, heart rate recovery third: 51±12 vs 59±12; p=0,00, heart rate recovery fourth: 54±13 vs 61±2; p=0,02) (Table 2). The TIMI frame counts for each major epicardial coronary artery and mean TIMI frame count were also found to be significantly higher in the metabolic syndrome group compared to the controls (Left anterior descending artery: 51±24vs 39±15; p=0,009, Left circumflex artery: 32±11 vs 24±7; p=0,001, Right coronary artery: 33±14 vs 24±10; p=0,003, mean

TIMI frame count: 38±15 vs 29±9; p=0,002) (Tabe 2). Additionally, a significant negative correlation was also detected between mean TIMI frame count and heart rate recovery first minute in patients with metabolic syndrome (p=0,01) (Table 3). None of metabolic syndrome parameters did not affect the heart rate recovery values, however mean TIMI frame count independently associated with the heart rate recovery first minute (p=0,04) in patients with metabolic syndrome (Table 4).

Conclusions: There was a significant negative correlation between mean TIMI frame count and heart rate recovery first minute, and that mean TIMI frame count was the only parameter independently associated with the heart rate recovery first minute in patients with metabolic syndrome. Therefore, impaired coronary blood flow occured in metabolic syndrome might be a clue of the autonomic dysfunction in addition to previously known endothelial dysfunction.

Table 1. Demographic and laboratory findings of the patients with metabolic syndrome and the controls.

	Control group n=37	Metabolic syndrome n=43	P value
Age	51±8	55±10	0,68
Gender(male/female)	22(59%)/15(41%)	22 (51%)/21(49%)	0,34
Smoking	19/18 (51%)	16/27 (37%)	0,14
Body mass index	27±2	30 ±3	**0,00**
Waist circumference (cm)	89±8	100±8	**0,00**
Svstolic BP (mm/hg)	128±25	141±20	**0,01**
Diastolic BP (mm/hg)	77±12	82 ±13	**0,05**
Resting HR (beats/minute)	74±13	78 ±13	0,19
Peak exercise HR (beats/minute)	158±17	151±18	0,10
Exercise capacity (METs)	11,9±2,7	9,4±3,3	**0,02**
Fasting glucose (mg/dL)	97±13	115±29	**0,01**
Total cholesterol (mg/dL)	204±49	188±44	0,13
LDL cholesterol (mg/dL)	137±42	129±32	0,36
HDL cholesterol (mg/dL)	44±12	35 ±7	**0,01**
Triglvceride (mg/dL)	113±33	157±48	**0,06**
Hemoglobin (g/dL)	14±1	14 ±1	0,71
Creatinine (mg/dL)	0,8±0,1	0,8 ±0,1	0,18
Insulin resistance (HOMA-R)	2,3±1,8	3,5 ±2,1	0,13

BP:Blood pressure, HR: Heart rate, LDL: Low dansity lipoprotein. HDL: High dansity lipoprotein. METS: Metabolic equivalent units.

Table 2. Comparison of the heart rate recovery and the TIMI frame counts values between two groups.

	Control group n=37	Metabolic syndrome n=43	P value
HRR 1st min (beats/minute)	37±10	32±9	**0,01**
HRR 2nd min (beats/minute)	52±11	46±11	**0,03**
HRR 3rd min (beats/minute)	59±12	51±12	**0,00**
HRR 4th min (beats/minute)	61±2	54±13	**0,02**
LAD	39±15	51±24	**0,009**
LCx	24±7	32±11	**0,001**
RCA	24±10	33±14	**0,003**
Mean TIMI frame count	29±9	38±15	**0,002**

HRR: Heart rate recovery LAD: Left anterior descending coronary artery, LCx: Left circumflex coronary artery, RCA: Right coronary artery.

Table 3. Correlation between mean TIMI frame count and heart rate recovery values in patients with metabolic syndrome.

	Mean TIMI frame count	
	Correlation coefficients (r)	P value
HRR 1st min (beats/minute)	-0,38	**0,01**
HRR 2nd min (beats/minute)	-0,22	0,15
HRR 3rd min (beats/minute)	-0,24	0,12
HRR 4th min (beats/minute)	-0,28	0,06

LAD: Left anterior descending coronary artery,
LCx: Left circumflex coronary artery,
RCA: Right coronary artery

Table 4. Role of mean TIMI frame count and each of the parameters of metabolic syndrome on heart rate recovery 1st minute in two groups.

	Control group Coefficients (r)	P value	Metabolic syndrome Coefficients (r)	P value
Mean TIMI frame count	-0,02	0,9	-0,27	**0,04**
Waist circumference (cm)	-0,14	0,5	-0,33	0,85
Svstolic BP (mm/hg)	-0,17	0,08	-0,10	0,17
Diastolic BP (mm/hg)	0,36	0,08	0,18	0,17
Fasting glucose (mg/dL)	-0,15	0,27	-0,03	0,96
HDL cholesterol (mg/dL)	0,25	0,83	1,7	0,40
Triglyceride (mg/dL)	0,02	0,9	0,39	0,35

HDL: High dansity lipoprotein

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Relationship between Coronary Collateral Circulation and Left Ventricular End Diastolic Pressure and Plasma Levels of N-Terminal Pro-B-Type Natriuretic Peptide in Patients with Chronic Total Occlusion

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Objective: Numerous investigations have shown that well-developed coronary collaterals exert protective effect on left ventricular functions. But, relationship between collateral grade and left ventricular end diastolic pressure has not been studied in chronic total occlusion patients. Also, there are conflicting data regarding to effect of collaterals on NT-proBNP levels, which has important diagnostic and prognostic utility in coronary heart disease and left ventricular dysfunction. The aim of our study is to evaluate the relationship between coronary collateral circulation and left ventricular end diastolic pressure and NT-proBNP levels.

Methods: Study group was retrospectively selected from patients who had coronary angiography at our center between June 2011 and March 2013. Clinical, biochemical, angiographic and haemodynamic data of 199 stable patients having at least one totally occluded main coronary artery were evaluated. Coronary collateral circulation was graded according to Rentrop classification. While Rentrop grade 3 was defined as good, all remaining collateral grades regarded as poor collaterals. We used Chi-square, Student t and Mann-Whitney U tests for statistical analysis.

Results: Overall 87 patients were found to have good collaterals and 112 patients had poor collaterals. There were no significant difference between patients having good or poor coronary collaterals regarding to left ventricular end diastolic pressure (16,96 ± 5,59mmHg vs 15,61 ± 6,06mmHg, p=0,379) and NT-proBNP levels (765,84 ±1417,31 pg/ml vs 994,79 ± 1787,90 pg/ml, p=0,486).

Conclusion: Even well-developed coronary collaterals may be incapable of protecting the rise of left ventricular end diastolic pressure and NT-proBNP levels which are reliable markers of the left ventricular dysfunction.

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