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

PP-265

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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