using an automated analyser (Abbot Cell-Dyne 3700 System Abbott Diagnostics, Santa Clara, USA). CFR was calculated as the ratio of hyperemic to baseline peak diastolic velocities after dipyridamole infusion by transthoracic pulsed wave Doppler echocardiography. A CFR value ≥2.5 was accepted as normal.

Results: CFR inversely correlated with WBC (r = −0.219 P = 0.009), neutrophil (r = −0.230 P = 0.006) and monocytes (r = −0.188 P = 0.026) counts. WBC, neutrophil and monocytes counts were higher in impaired CFR group than in normal CFR group (Table).

Conclusion: Increased WBC, neutrophil and monocytes counts in patients with impaired CFR delineate the inflammatory milieu in these patients.

Table: White blood cell and subsets characteristics of the subjects with or without impaired coronary flow reserve

	Impaired CFR (n = 82)	Normal CFR (n = 62)	P
WBC	7220±1644	6601±1672	0.029
Neutrophil	4171±1232	3738±1190	0.039
Lymphocyte	2376±685	2199±654	0.123
Monocytes	494.3±139.8	438.7±135.7	0.020
C-reactive protein	2.7 (1.4–5.0)	1.3 (0.7–3.2)	0.036

WBC: white blood cell count; CFR: coronary flow reserve.

OP-062
INCREASED CIRCULATING SOLUBLE CD40 LEVELS IN PATIENTS WITH SLOW CORONARY FLOW PHENOMENON

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Objective: Slow coronary flow (SCF) is an angiographic finding characterized with delayed opacification of epicardial coronary arteries without obstructive coronary disease. CD40/CD40 ligand (CD40L) signaling seems closely related to atherosclerosis due to increased inflammation and prothrombotic state. We investigated whether soluble CD40 (sCD40), an indirect marker of CD40/CD40L dyad, is related to SCF.

Methods: The present study was cross-sectional and observational, consisting of seventy individuals who underwent coronary angiography with suspicion of CAD and had angiographically normal coronary arteries of varying coronary flow rates. The relationship between sCD40, C-reactive protein (CRP) and SCF phenomenon was investigated. Fifty patients with isolated SCF (mean age: 56±10 years) and 20 age- and gender-matched control participants with normal coronary flow (NCF) and normal coronary arteries (NCA), (mean age: 55±10 years) were included in the study.

Results: The clinical characteristics were not statistically significant different between SCF and NCA group. Serum CRP levels was also similar between two groups. Serum sCD40 level was significantly higher in the SCF group compared to control group (74±31 vs. 59±16 pg/mL, p = 0.014). In multivariate analyses, mean coronary diameter strongly and sCD40 weakly predicted SCF.

Table: The independent relationship of soluble CD40 with slow coronary flow phenomenon in 70 individuals

Variable	OR (95% CI)	p value
sCD40 (pg/ml)	1.044 (1.006–1.084)	0.023
Ln (sCD40)	18.11 (1.694–193.5)	0.017
Mean coronary diameter	7.358 (1.990–27.20)	0.003

*Logistic regression analyses with enter method for independent variables which were included if they were significantly different in the univariate analyses.

Conclusion: This study revealed, for the first time, significantly increased serum sCD40 levels in patients with SCF. Although we cannot conclude the underlying pathological process of SCF, we believe that these findings may be pivotal for further studies searching the specific roles of CD40/CD40L signaling on SCF phenomenon in coronary vasculature.

OP-063
THE RELATIONSHIP BETWEEN β-FIBRINOGEN −455 G>A GENE POLYMORPHISM AND SLOW CORONARY FLOW

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Background: Slow coronary flow (SCF) is an angiographic finding characterized by delayed distal vessel opacification in the absence of significant epicardial coronary disease, and is an important clinical entity because it may be the cause of angina at rest or during exercise, acute myocardial infarction, and hypertension. The pathophysiological mechanisms of the SCF remain undetermined. Endothelial dysfunction and microvascular dysfunction have been suggested as underlying mechanisms. Slow coronary flow is considered to be a form of early phase atherosclerosis in some studies. β-fibrinogen −455 G/A genotypes have been described to be associated with coronary artery disease (CAD) and myocardial infarction. Although various gene polymorphsims have been studied in patients with SCF, β-fibrinogen gene polymorphisms have not been studied previously. We investigated relationship between β-fibrinogen −455 G>A gene polymorphism and SCF.

Methods: Thirty five patients with SCF and otherwise normal coronary arteries (mean age 52±8 years) and 30 patients with normal coronary angiograms (mean age 52±8 years) were included in the study. TIMI frame count ≥40 frames for the left anterior descending artery was considered as SCF. The types of β-fibrinogen −455 G>A gene polymorphisms were detected by the polymerase chain reaction method. For each polymorphic position, one of three possible patterns may be obtained: Normal, Heterozygous, or Homozygous mutant genotype. Demographic characteristics and major risk factors for atherosclerosis were evaluated in the study groups.

Results: There was no significant difference with respect to age and gender between groups. The genotype distribution in SCF group was as follows: normal genotype frequency was 18 (51%) and heterozygous genotype frequency was 17 (49%). Homozygous genotype was not detected in SCF group. The genotype distribution in control group was as follows: normal genotype frequency was 12 (40%), heterozygous genotype frequency was 17 (57%) and homozygous genotype frequency was 1 (3%).

Conclusions: In this study, we found that homozygous genotype of β-fibrinogen −455 G>A polymorphism was higher in patients with SCF compared to control subject. However, further large-sized studies are required for determining relationship between β-fibrinogen −455 G>A gene polymorphisms and SCF.

OP-064
SERUM PROLIDASE ACTIVITY IN PATIENTS WITH NONATHEROSCLEROTIC CORONARY ARTERY ANEURYSM

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Objective: Coronary artery aneurysms are defined as a dilatation in the diameter of a coronary artery segment to more than 1.5-fold normal size. The underlying etiology of coronary artery