

Koroner yavaş akım fenomeni saptanan hastalarda sol ventrikül sistolik ve diyastolik fonksiyonların değerlendirilmesi

Evaluation of left ventricular systolic and diastolic functions in patients with coronary slow flow phenomenon

Dr. Cemil Zencir, Dr. Mustafa Çetin,# Dr. Hasan Güngör, Dr. Kayıhan Karaman,* Dr. Çağdaş Akgüllü, Dr. Ufuk Eryılmaz, Dr. Mücahit Avcil†

Adnan Menderes University, Faculty of Medicine, Department of Cardiology, Aydın

Adıyaman University, Faculty of Medicine, Department of Cardiology, Adıyaman

*Gazi Osman Paşa University, Faculty of Medicine, Department of Cardiology, Tokat

†Adnan Menderes University, Faculty of Medicine, Department of Emergency Medicine, Aydın

ÖZET

Amaç: Bu çalışmada, koroner yavaş akım fenomeni (KYAF) olan hastalarda geleneksel ve doku Doppler ekokardiyografisi ile ölçülen sol ventrikül sistolik ve diyastolik fonksiyon parametreleri kontrol grubuyla karşılaştırıldı.

Çalışma planı: Çalışmaya koroner anjiyografi sırasında KYAF saptanan 60 hasta (49 erkek; ortalama yaş 52.4±12.1) ve normal koroner arterler saptanan 30 gönüllü (21 erkek; ortalama yaş 50.2±12.1) alındı. Koroner yavaş akım fenomeni tanısı TIMI kare sayısı (TKS) yöntemiyle konuldu. Her bir koroner arterin TIMI kare sayısı TKS yöntemiyle hesaplandı. Geleneksel ve doku Doppler ekokardiyografi ile sol ventrikülün sistolik ve diyastolik fonksiyonları değerlendirildi. Diyastolik fonksiyon parametreleri ile TKS bağlantısı araştırıldı.

Bulgular: Gruplar arasında bazal demografik ve laboratuvar sonuçları arasında anlamlı fark yoktu. TKS, KYAF grubunda yüksekti (p<0.001). Geleneksel ekokardiyografik incelemede sol ventrikül ejeksiyon fraksiyonu

ABSTRACT

Objectives: In this study, systolic and diastolic function parameters were measured with conventional and tissue Doppler echocardiography in patients with coronary slow flow phenomenon (CSFP), and compared to those of a control group.

Study design: Sixty patients (49 males; mean age 52.4±12.1yrs) in whom CSFP was detected during coronary angiography study, and 30 volunteers with normal coronary arteries (21 males; mean age 50.2±12.1 yrs) were included in this study. CSFP was determined using the TIMI frame count (TFC) method. TIMI frame count was calculated for each coronary artery using the TFC method. Left ventricular systolic and diastolic functions were assessed by conventional echocardiography and tissue Doppler imaging. Correlation between TFC, and diastolic function parameters was investigated.

Results: Baseline demographic and laboratory results did not differ significantly between the groups. TIMI frame counts were greater in the CSFP group compared to controls (p<0.001). Left ventricular ejection fraction (65.93±8.06

Submitted on: 05..29. 2013 *Accepted for publication on:* 07.17..2013

Address of correspondence: Dr. Cemil Zencir. Adnan Menderes Üniversitesi Uygulama ve Araştırma Hastanesi, Kardiyoloji Kliniği, 09100 Aydın. Phone: +90 256 444 12 56 e-mail: czencir@yahoo.com

(EF), E/A oranı ve izovolemik gevşeme zamanı (IVRT) benzerdi (sırasıyla, %65.93±8.06 ve 66.63±5.96, 1.11±0.36 ve 1.22±0.33, 85±17 cm/sn ve 84±13 cm/sn, tüm p değerleri >0.05). Doku Doppler ile ekokardiyografik incelemede grupların Em, Am ve E/Em oranı benzer idi (sırasıyla, 7.0±2.1cm/s ve 7.4±1.7, 7.4±2.0 cm/sn, 7.0±1.4 cm/sn, 10±3 ve 10±1, tüm p değerleri >0.05).Sol inen koroner arterin (LAD) düzeltilmiş TKS (cLAD) ve ortalama TKS ile E/A oranı, DT, IVRT ve E/Em oranı arasında anlamlı bağıntı saptanmadı.

Sonuç: Çalışmamızda koroner yavaş akım fenomeni olan hastalardaki sol ventrikülün sistolik ve diyastolik fonksiyonlarının korunmuş olduğu saptandı.

vs 66.63±5.96%), E/A ratio (1.11±0.36 vs 1.22±0.33), and isovolumetric relaxation time (IVRT) (85±17 cm/s vs 84±13 cm/s) measured with conventional echocardiography showed no significant difference between the two groups. Em (7.0±2.1 cm/s vs 7.4±1.7 cm/s), Am (7.4±2.0 cm/s vs 7.0±1.4 cm/s) and E/Em (10±3 vs 10±1) measured with tissue Doppler echocardiography showed no significant difference between the two groups. Corrected TIMI frame count for the left descending coronary artery (cLAD) and mean TFC were not correlated with the E/A ratio, deceleration time (DT), IVRT or E/Em ratio.

Conclusion: Left ventricular systolic and diastolic functions were preserved in CSFP.

Abbreviations:

<i>Am</i>	<i>Late diastolic velocity</i>
<i>A</i>	<i>Late mitral annular peak velocity</i>
<i>Cx</i>	<i>Circumflex coronary artery</i>
<i>DT</i>	<i>Deceleration time</i>
<i>E</i>	<i>Early diastolic mitral inflow velocity</i>
<i>Em</i>	<i>Early peak diastolic velocity</i>
<i>IVRT</i>	<i>Isovolumetric relaxation time</i>
<i>CAG</i>	<i>Coronary angiography</i>
<i>CSFP</i>	<i>Coronary slow flow phenomenon</i>
<i>LDA</i>	<i>Left descending artery</i>
<i>RCA</i>	<i>Right coronary artery</i>
<i>Sa</i>	<i>Systolic myocardial velocity</i>
<i>TFC</i>	<i>TIMI frame count</i>

Coronary slow flow phenomenon (CSFP) is defined as delayed coronary opacification of distal coronary arteries during coronary angiography (CAG) in patients without any angiographically detected coronary artery abnormalities or critical obstructive coronary artery disease. It is firstly described by Tambe et al in 1972.[1] In its etiology many factors including vascular, inflammatory, endothelial, and vasomotor disorders have been implicated.[2-6] CSFP might be an early sign of atherosclerosis.[7] Besides in these patients, coronary artery flow

reserve was impaired.[8] Many literature studies have investigated the probable mechanisms of action of CSFP on systolic, and diastolic functions.[9-11] However in our literature review, we couldn't encounter any study which demonstrated the presence of intact left ventricular diastolic function parameters in patients with CSFP using conventional, and tissue Doppler echocardiographic modalities.

In this study, we have measured left ventricular systolic, and diastolic function parameters in patients with CSFP,

and compared them to those of the control group.

PATIENTS AND THE METHOD

The study included patients who had undergone CAG with the suspicion of coronary artery disease as detected by using noninvasive tests (exercise test, and nuclear myocardial perfusion scanning). Sixty patients with coronary slow flow phenomenon determined using the TIMI frame count (TFC) during angiography, and 30 volunteers with normal coronary arteries were included in the study. The patients with coronary artery ectasia, congestive heart failure, previous history of coronary artery angioplasty and/or stenting, pulmonary hypertension, hepatic, and renal failure, moderate or severe degrees of valvular disease, cardiomyopathy of any etiology, implantation of permanent or temporary pacemaker, atrial fibrillation, chronic obstructive pulmonary disease were not included in the study. After a 12-hour fasting period using standard laboratory techniques, blood samples were drawn from the antecubital vein. Blood samples were centrifuged. Fasting plasma levels, lipid profile, renal, and hepatic function tests were measured using Beckman Coulter Synchron LX20 Clinical System device, and kits. Whole blood count was evaluated using ABBOTT CELL-DYN System 1200®. The local ethics committee approved compliance of the study with WMA Declaration of Helsinki -

Ethical Principles for Medical Research Involving Human Subjects. Verbal, and written informed consent forms were obtained from all study participants.

Coronary angiography

All patients underwent CAG using standard exposures, and Philips® brand INTEGRIS H 5000 model or Toshiba® Infinix CC-i monoplanar cardiac angiography devices. Selective CAG was employed using Judkins technique with 6 or 7 French (F) catheters through right or left femoral approaches. As an opaque material Iopromide (Ultravist-370®) or Iohexol (Omnipaque® 350 mg/ml) was used. For each exposure an average of 6-8 ml opaque material was injected, **cranial, and caudal right, and left oblique (angulated from the perpendicular) views** were recorded at 25 frames / sec.

TIMI frame counts

For grading, and detection of coronary slow flow rate, corrected TFC method was used.[12] We assessed the first frame of opacification when the arterial lumen of the first branch was filled completely with antegrade flow, and the last frame counts were calculated when the opaque material reached to the most distal part of the artery. For the evaluation of the left anterior descending artery (LAD) apical “forking “ branch of LAD, and the most dilated segment of the circumflex coronary artery (Cx) or distal obtuse marginal branch of Cx, and for the right

coronary artery (RCA) the take-off of the first major branch or its posterolateral projection were used. To estimate corrected TIMI frame count (cLAD), TFC of LAD was divided by the coefficient 1.7. Normal mean TIMI frame counts of coronary arteries were accepted as 36 ± 2.5 for LAD, 22 ± 4.1 for Cx, and 20.4 ± 3.1 for RCA. Mean TFC was calculated by dividing the sum of all TFCs for LAD, Cx, and RCA by the number three. Values two standard deviations above these estimates for at least one coronary artery was accepted as coronary slow flow phenomenon [12]

Echocardiography

All of the study group underwent transthoracic echocardiographic examinations 6-12 hours after CAG. Transthoracic echocardiographic examination was performed with the aid of Vivid 7 Dimension® (GE Vingmed Ultrasound AS N-3190 Horten, Norway) echocardiograph, and 2.5 MHz transducer used by a single cardiologist. The patients were evaluated in the left oblique decubitus position after 5 minutes of rest as recommended by American Society of Echocardiography through standard acoustic windows; M-mode, left ventricular end-diastolic, and end-systolic diameters, thickness of septum, and posterior walls were measured. EF was measured using M-mode Teichholz method [13]. Early (E), and late (A), mitral inflow velocities, isovolumetric

relaxation time (IVRT), and E-wave deceleration time (DT) were assessed. Pulsed wave-Doppler velocity recordings were made from apical 4-chamber views, and placing sampling volumes on the tips of the mitral valves, and then images of three successive cycles were analyzed. Tissue Doppler analysis was performed from apical 4-chamber views, and sampling volume was placed on the junction of mitral annulus, and wall to obtain recordings of the left ventricle, and lateral wall annulus. Using tissue Doppler assessments systolic myocardial velocity (Sm), early (Em), and late (Am) diastolic velocities were calculated. E/A, and E/Em ratios of the patients were also calculated. For each parameter, average of three successive measurements was estimated. Single-channel echocardiography recordings were obtained from each patient. Intraobserver correlation coefficient was 0.95.

Statistical analysis

Analysis of the study data was performed using Windows SPSS 20.0 (SPSS Science, Chicago, IL, USA) statistics software program. Continuous variables were indicated as means \pm standard deviation. Non-continuous variables were expressed as percentages. Intergroup comparisons of continuous, and non-continuous variables were performed using Student's *t*-test and *chi*-square test, respectively. Correlation between continuous data was analyzed

using two-tailed Pearson test. $P < 0.05$ was accepted as statistically significant.

RESULTS

Sixty patients with CSFP with a mean age of 52.4 ± 12.1 years (49 men, and 11 women), and as a control group 30 volunteers (21 men, and 11 women) with normal coronary arteries (mean age, 50.1 ± 11.9) years were included in the study.

Basic characteristics of the study group are presented in Table 1. In the CSFP group TIMI frame counts were significantly higher relative to the control group. [cLAD: (33.3 ± 9 ve 17.2 ± 3), Cx (34 ± 13 ve 18.3 ± 4), RCA (29 ± 17 ve 17 ± 3), and mean TFC (40 ± 12 ve 21 ± 3)] ($p < 0.001$) (Table 2).

Table 1. Clinical, an demographic characteristics of the groups

	CSFP (n=60)			Control (n=30)			
	n	%	Mean \pm SD	n	%	Mean \pm SD	<i>p</i>
Mean age (yrs)			52.4 ± 12.1			50.2 ± 12.1	0,403
Male/female	49/11			21/9			0,282
Systolic blood pressure (mm Hg)			123 ± 16			129 ± 11	0,068
Diastolic blood pressure (mm Hg)			77 ± 9			79 ± 9	0,295
Hypertension	32	53		14	46		0,551
Diabetes mellitus	7	12		6	20		0,289
Hyperlipidemia	10	17		4	13		0,683
Smoking	22	36		6	20		0,107
Heart rate (bpm)			79 ± 14			81 ± 12	0,458
Body mass index (kg/m^2)			$27,3 \pm 3$			$26,2 \pm 3$	0,170
Hemoglobin (g/dl)			$14,5 \pm 1,4$			$14,0 \pm 1,6$	0,165
Fasting blood glucose (mg/dl)			112 ± 47			108 ± 41	0,752
Total cholesterol (mg/dl)			181 ± 34			$182,4 \pm 35,0$	0,864
HDL-cholesterol (mg/dl)			$37,7 \pm 6,5$			$41,1 \pm 10$	0,060
LDL-cholesterol (mg/dl)			111 ± 29			$108,6 \pm 28,2$	0,704
Triglyceride (mg/dl)			165 ± 84			$155,7 \pm 85,8$	0,603
Blood urea nitrogen			16 ± 6			15 ± 5	0,344
Serum creatinine (mg/dl)			$1,0 \pm 0,3$			$0,9 \pm 0,2$	0,310

CSFP, coronary slow flow phenomenon; SD, standard deviation; HDL, High-density lipoprotein; LDL, Low-density lipoprotein

Comparative results obtained in the conventional, and tissue Doppler study groups are shown in Table 3. A statistically significant difference was not detected between systolic, and diastolic function parameters. A significant correlation was not found between cLAD, and mean TFC, DT, IVRT, and E/Em ratio (Table 4).

DISCUSSION

In our study, we have found that systolic and diastolic function parameters

Table 2. TIMI frame scores of the groups

	CSFP (n=60) Mean ± SD	Control (n=30) Mean ± SD	<i>p</i>
Left descending coronary artery corrected TFC	33,3±9	17,2±3	<0,001
Left descending coronary artery	57,5±16	28,3±6	<0,001
Circumflex coronary artery	34±13	18,3±4	<0,001
Right coronary artery	29±17	17±3	<0,001
Mean	40±12	21±3	<0,001

CSFP, Coronary slow flow phenomenon; SD, standard deviation TFC, TIMI frame count; Mean= (LAD + Cx + RCA)/3

It is seen in nearly 1 % of the patients who had undergone CAG.[14] This clinical condition has been defined very well in many studies, and it has been suggested that it might be a subgroup of cardiac syndrome X.[5]

In its etiology, many factors including vascular, inflammatory, endothelial, genetic, and vasomotor disorder have been blamed.[2-6,15] The presence myocardial ischemia has been shown in many studies. The effect of this condition on left ventricular functions

detected during both conventional, and tissue Doppler echocardiographic examinations were not unfavourable affected. Besides, we couldn't find a significant correlations between diastolic function parameters, and TFC. CSFP is defined as delayed washout of opaque agent during routine CAG from coronary artery without any evidence of stenosis, thrombus, spasm or dissection which cause significant occlusion of the coronary arteries.

is not known, yet.[16,17] The effect of these culprit factors in the etiology of CSFP on ventricular systolic, and diastolic functions has not been elucidated clearly. In many studies, assessments of left ventricular systolic function by conventional echocardiography in patients with CSFP could not demonstrate any significant difference relative to the control group .[9,11,18-20] Also in our study left ventricular functions were similar in CSFP, and control groups, respectively.

Table 3. Conventional, and tissue Doppler echocardiographic parametres of the groups

	CSFP (n=60) Mean ± SD	Control (n=30) Mean ± SD	<i>p</i>
Left ventricular end-systolic diameter (mm)	29,12±7,1	28,47±4,43	0,648
Left ventricular end-diastolic diameter (mm)	46,20±6,16	46,27±4,56	0,958
Septum thickness (mm)	9,85±2,10	10,13±1,79	0,530
Posterior wall thickness (mm)	10,11±1,70	9,96±1,60	0,690
Ejection fraction (Teichholz) (%)	65,93±8,06	66,63±5,96	0,675
Mitral inflow E velocity (cm/sec)	70±13	76±13	0,051
Mitral inflow A velocity (cm/sec)	67±15	65±14	0,634
E/A ratio	1,12±0,36	1,22±0,33	0,193
IVRT (cm/sec)	85±17	84±13	0,745
E wave deceleration time (m/sec)	193±12	188±11	0,067
Lateral wall Sm (cm/sec)	6,4±2,0	6,1±1,5	0,523
Lateral wall Em (cm/sec)	7,0±2,1	7,4±1,7	0,324
Lateral wall Am (cm/sec)	7,4±2,0	7,0±1,4	0,291
E/Em ratio	10±3	10±1	0,726

CSFP, Coronary slow flow phenomenon; SD, standard deviation; Am Late diastolic myocardial velocity; Em, Early diastolic myocardial velocity; Sm, Early systolic myocardial velocity; IVRT, isovolumetric relaxation time

Table 4. Correlation between cLAD, median TCS, and echocardiographic parametres

	cLAD		Average TFC	
	R	P	R	PO
E/A ratio	0,193	0,14	0,217	0,09
DT (m/sec)	0,129	0,32	0,122	0,35
IVRT (cm/sec)	-0,063	0,63	0,025	0,85
E/Em ratio	0,070	0,59	0,142	0,27

DT, Ewave deceleration time (msec); IVRT, Isovolumetric relaxation time; TFC, TIMI frame count; Average TCF (LAD + Cx+ RCA)/3; cLAD: Left descending coronary artery corrected TCF

In the early phase of myocardial ischemia in the coronary artery disease before derangement of the systolic function, diastolic dysfunction develops. Left ventricular diastolic functions are more helpful in predicting ischemia than systolic

functions.[21] Some studies have demonstrated the presence of left ventricular dysfunction in patients with CSFP. Sevimli et al.[11] demonstrated decreases in E, Sm and Am, and prolongation of DT, and IVRT in the

CSFP group. Still in this study E/A, and E/Em ratio was significantly correlated with CSFP. Sezgin et al.[9] used pulsed Doppler to demonstrate impaired left ventricular filling decreased E/A ratio and prolongation of IVRT in cases with CSFP. Ekiz et al.[22] detected decreased E/A ratio, significantly lower Em, and Em/Am ratio estimated for lateral, and septal mitral annulus, prolonged IVRT, and DT without any significant change in Sm values. Tanriverdi et al.[23] found lower peak E, and A velocities, and E/A ratios, and significantly increased number of patients with diastolic dysfunction.

Demographic, and clinical features of the patients included in the study conducted by Baykan et al[19] were similar to ours. Conventional echocardiographic parameters (E, A, E/A, DT, and IVRT) analyzed in this study were not different between CRFP, and the control groups. This result was in line with our findings. However in their tissue Doppler assessments, contrary to our study, Em, Em/Am, and Sm values were significantly lower than those of the control group. In our study these values were almost alike.

In our study we evaluated left ventricular diastolic functions in patients with CSFP, pulsed, and tissue Doppler US using the following parameters: early mitral inflow velocity (E), late mitral velocity (A), E/A ratio, IVRT, E wave DT, parietal wall Sm, parietal wall Em, parietal

wall Am, and E/Em ratio. In our study any significant difference was not found between CSFP, and the control groups as for E, A velocities, E/A ratio, DT, IVRT, Em, Am, Sm velocity, and E/Em ratio. Besides, a significant correlation was not detected among cLAD, mean TCF, E/A ratio, DT, IVRT, and E/Em ratio.

The probable reasons for the differences between the results of our study, and those of the previous studies: Differences between demographic (mean age, male/female ratio, systolic, and diastolic blood pressure), and medical history (hypertension, smoking status, diabetes mellitus, and obesity) characteristics of our study group, and other comparator studies might account for diversities between study results [9,11,22,23]. Sevimli et al.[11], and Ekiz et al.[22] included patients with unstable angina pectoris in their studies. Patients with unstable angina pectoris were not included in our study which might account for different study results relative to those of the studies we encountered in our study.

Limitations of our study

Scarce number of study population, and inability to use some advanced echocardiographic technologies (strain, strain rate, and speckle tracking etc.) for the evaluation of both systolic, and diastolic parameters can be enumerated among limitations of our study.

Conclusion

In our study, left ventricular systolic, and diastolic functions in CSFP patients were found to be within normal limits. Contrary to other studies, in our study echocardiographically detected similarities between the CSFP, and the control groups as for left ventricular systolic, and diastolic functions suggest that in this patient group echocardiography fails to discriminate between two entities. Since different results have been obtained in many studies performed in this field, further larger scale studies have to be conducted on this subject.

Conflict of Interest: None declared.

REFERENCES

1. Tambe AA, Demany MA, Zimmerman HA, Mascarenhas E. Angina pectoris and slow flow velocity of dye in coronary arteries-a new angiographic finding. *Am Heart J* 1972;84:66-71.
2. Mosseri M, Yarom R, Gotsman MS, Hasin Y. Histologic evidence for small-vessel coronary artery disease in patients with angina pectoris and patent large coronary arteries. *Circulation* 1986;74:964-72.
3. Sezgin AT, Sigirci A, Barutcu I, Topal E, Sezgin N, Ozdemir R, et al. Vascular endothelial function in patients with slow coronary flow. *Coron Artery Dis* 2003;14:155-61.
4. Li JJ, Qin XW, Li ZC, Zeng HS, Gao Z, Xu B, et al. Increased plasma C-reactive protein and interleukin-6 concentrations in patients with slow coronary flow. *Clin Chim Acta* 2007;385:43-7.
5. Beltrame JF, Limaye SB, Horowitz JD. The coronary slow flow phenomenon-a new coronary microvascular disorder. *Cardiology* 2002;97:197-202.
6. Li JJ, Xu B, Li ZC, Qian J, Wei BQ. Is slow coronary flow associated with inflammation? *Med Hypotheses* 2006;66:504-8.
7. Cin VG, Pekdemir H, Camsar A, Çiçek D, Akkus MN, Parmaksız T, et al. Diffuse intimal thickening of coronary arteries in slow coronary flow. *Jpn Heart J* 2003;44:907-19.
8. Erdogan D, Caliskan M, Gullu H, Sezgin AT, Yildirim A, Muderrisoglu H. Coronary flow reserve is impaired in patients with slow coronary flow. *Atherosclerosis* 2007;191:168-74.
9. Sezgin AT, Topal E, Barutcu I, Ozdemir R, Gullu H, Bariskaner E, et al. Impaired left ventricle filling in slow coronary flow phenomenon: an echo-Doppler study. *Angiology* 2005;56:397-401.
10. Gunes Y, Tuncer M, Guntekin U, Ceylan Y, Sahin M, Simsek H. Regional functions of the left ventricle in patients with coronary slow flow and the effects of nebivolol. *Ther Adv Cardiovasc Dis* 2009;3:441-6.
11. Sevimli S, Büyükkaya E, Gündoğru F, Arslan Ş, Aksakal E, Gürlertop Y, et al. Left ventricular function in patients with coronary slow flow: a tissue Doppler study. *Turk Kardiol Dern Ars* 2007;35:360-5.
12. Gibson CM, Cannon CP, Daley WL, Dodge JT Jr, Alexander B Jr, Marble SJ, et al. TIMI frame count: a quantitative method of assessing coronary artery flow. *Circulation* 1996;93:879-88.
13. Schiller NB, Shah PM, Crawford M, DeMaria A, Devereux R, Feigenbaum H, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. *J Am Soc Echocardiogr* 1989;2:358-67.
14. Singh S, Kothari SS, Bahl VK. Coronary slow flow phenomenon: an angiographic curiosity. *Indian Heart J* 2004;56:613-7.
15. Ozdogru I, Zencir C, Dogan A, Orscelik O, Inanc MT, Celik A, et al. Acute effects of intracoronary nitroglycerin and diltiazem in coronary slow flow phenomenon. *J Investig Med* 2013;61:45-9.
16. Beltrame JF, Limaye SB, Wuttke RD, Horowitz JD. Coronary hemodynamic and metabolic studies of the coronary slow flow phenomenon. *Am Heart J* 2003;146:84-90.
17. Cakmak M, Tanriverdi H, Cakmak N, Evrengul H, Cetemen S, Kuru O. Simvastatin may improve myocardial perfusion abnormality in slow coronary flow. *Cardiology* 2008;110:39-44.
18. Elsherbiny IA. Left ventricular function and exercise capacity in patients with

slow coronary flow. Echocardiography 2012;29:158-64.

19. Baykan M, Baykan EC, Turan S, Gedikli O, Kaplan S, Kiriş A, et al. Assessment of left ventricular function and Tei index by tissue Doppler imaging in patients with slow coronary flow. Echocardiography 2009;26:1167-72.

20. Barutcu I, Sezgin AT, Sezgin N, Gullu H, Esen AM, Topal E, et al. Elevated plasma homocysteine level in slow coronary flow. Int J Cardiol 2005;101:143-5.

21. Labovitz AJ, Lewen MK, Kern M, Vandormael M, Deligonal U, Kennedy HL. Evaluation of left ventricular systolic and diastolic dysfunction during transient myocardial ischemia produced by angioplasty. J Am Coll Cardiol 1987;10:748-55.

22. Ekiz O, Avsar O, Batyraliev T. Koroner yavaş akımın sol ventrikül diyastolik fonksiyonu üzerine etkisi. TGKD 2009;13:51-5.

23. Tanriverdi H, Evrengul H, Kilic ID, Taskoylu O, Dogan G, Alpsoy S. Aortic pressures, stiffness and left ventricular function in coronary slow flow phenomenon. Cardiology 2010;116:261-7.

Anahtar sözcükler: Ekokardiyografi, Doppler; esneklik görüntüleme teknikleri/yöntemler; ventrikül fonksiyonu, sol; yavaş akım fenomeni.

Key words: Echocardiography, Doppler; elasticity, imaging techniques/methods; ventricular function, left; slow flow phenomenon.