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Assessment of Left Atrial Volumes and Functions in Patients with Coronary Slow Flow

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Abstract

Objectives: Coronary slow flow phenomenon (CSFP) is the slow or late progression of the opaque material to the distal vascular structures during angiography in patients with normal or near-normal coronary arteries. This study aims to evaluate left atrial volumes and functions using conventional transthoracic and tissue Doppler echocardiographic parameters in patients with CSFP.

Materials and Methods: According to criteria determined by Gibson, 50 patients with slow flow in at least one coronary artery were included as cases, and 40 subjects with normal coronary flow were included as controls.

Results: In the transmitral and tissue Doppler analysis, mitral early velocity (E), mitral late velocity/mitral early velocity (E/A), and Em were significantly lower in the coronary slow flow (CSF) group. LA, Am, mitral early velocity/early

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diastolic velocity (E/Em), LAV_{max}, LAV_{min}, LAV_{preA}, index volumes, LAAEV, LATEV, and LAAEF were found to be higher in the CSF group. A significant positive correlation was observed between Frame LAD and LAAEF (r=0.66, p<0.001) and between Frame LAD and E/Em (r=0.34, p<0.001). A significant negative correlation was found between LAAEF and E/A ratio (r=-0.4, p=0.003). There was also a significant positive correlation between LAPEF and E/A (r=0.44, p<0.001) and between the mean frame and LAAEF (r=0.4, p=0.002).

Conclusion: Impaired LV diastolic functions and significant changes in LA volumes were found in patients with CSFP.

Keywords: Coronary slow flow, left atrial functions, echocardiography, left atrial volume

Introduction

Coronary slow flow (CSF) is the slow or late progression of the opaque material to the distal vascular area without coronary artery stenosis (>50%) during angiography[1]. CSF may present with chronic coronary syndrome or acute coronary syndrome[2,3]. The underlying pathophysiological causes of this phenomenon are microvascular dysfunction, vasomotor dysfunction[1,4-6], diffuse atheroma plaques that do not cause stenosis in the coronary lumen, diffuse calcification, and diffuse intimal thickening[7,8].

Previous studies have shown that CSF is an atherosclerotic process, which increases microvascular resistance and affects both small and large vessels. Left ventricular (LV) diastolic dysfunction (LVDD) is among the earliest findings of ischemic heart disease and affects left atrial (LA) functions. LA functions may also be affected by valvular diseases, coronary artery disease (CAD), heart failure, and diastolic dysfunction. LA functions are important for cardiac output, and decreased LA functions are associated with atrial arrhythmias and ischemic stroke. LV systolic and diastolic dysfunction in CSF has been shown in previous studies[9]. However, the effects of CSF on LA functions have not been well established, and only limited data are available about this condition. Therefore, this research will assess LA volumes and functions in patients with CSF using conventional transthoracic and tissue Doppler echocardiographic parameters.

Materials and Methods

Coronary angiographies were performed in patients, who had been diagnosed with stable or unstable angina with myocardial ischemia detected by noninvasive tests. According to criteria determined by Gibson et al.[10], 50 patients with CSF in at least one coronary artery were included as cases, and 40 patients without ≥50% coronary stenosis were enrolled as controls. The study design was approved by the Ethics Committee of Dicle University. The participants' demographic data, medical histories, and laboratory parameters were collected from our hospital's electronic database. The participants' physical characteristics, such as body surface area (BSA), waist circumference, weight, height, and body mass index (BMI), were measured. Individuals were excluded from the study if they had ≥50% stenosis of the coronary arteries, myocardial infarctions, significant valvular heart disease, diabetes mellitus (DM), hypertension (HT), left ventricular systolic function of <50%, left and right bundle branch block, atrial fibrillation, chronic kidney disease, or congenital heart disease.

Coronary angiographies were performed according to the Judkin's[11] standard technique. The thrombolysis in myocardial infarction (TIMI) frame count (TFC) method was employed to measure coronary flow[10]. The starting point was set to be the moment when the contrast material touched the side of the artery and began to progress. The ending point was set as the moment when the contrast agent reached the distal part of the mustard for the left

anterior descending artery (LAD), when the first side of the posterolateral artery was given for the right coronary artery (RCA), and when the longest distal branch was observed for circumflex artery (Cx). The LAD frame was standardized by dividing it by 1.7 because of its length. The number of frames obtained by shooting at 12 fps was multiplied by a constant number of 2.4, following Gibson et al.[10]. Patients with a frame number of 20.2±3.0 for RCA, 36±2.6 for LAD, and 22±4.1 for Cx were diagnosed as having CSF.

For all participants, standard M-mode, 2D images, and Doppler recordings were completed using a 3.5Mhz probe and a simultaneous echocardiogram with a GE Vingmed Vivid S-5 echocardiography device. Echocardiographic examination was performed by a single researcher.

LV M-mode parameters, LV wall thickness, LV end-systolic diameter (LVESD), and LV end-diastolic diameter (LVEDD) measurements were obtained from the parasternal long axis plane[12]. The LV ejection fraction (LVEF) was measured according to Teicholtz's formula. Pulse wave Doppler velocity findings were examined via apical four-chamber images taken from three consecutive cycles. Mitral late velocity (A), mitral early velocity (E), deceleration time (DT), and isovolumetric relaxation time (IVRT) were also evaluated. Tissue Doppler examination was performed from the apical four-chamber window by placing the sample volume on the junction of the lateral anulus and the wall. Early diastolic velocity (Em), systolic myocardial velocity (Sm), and late diastolic velocity (Am) were calculated from the mitral lateral wall with tissue Doppler evaluation. E/A and E/Em ratios were also calculated. The average of three consecutive measurements for each parameter was recorded. After standard echocardiographic evaluation, LA sizes and volumes were examined. LA sizes were measured via the parasternal long axis and the apical four-chamber images. LA volume was evaluated using the modified Simpson method from the apical four-chamber images[13,14]. Atrial volume was adjusted according to BSA in all patients. The maximum volume LA (Vol_{max}) was measured just before the mitral valve opening. The preatrial contraction volume (Vol_{pre-A}) was measured at the time of the onset of the P wave in the echocardiogram. The LA minimum volume (Vol_{min}) was measured at the time of the mitral valve's closing. The emptying volume of the LA was calculated using the available volumes[15].

LA passive emptying volume (LAPEV) = Vol_{max} - Vol_{pre-A}

LA passive emptying fraction (LAPEF) = LA_{PEV}/Vol_{max}

LA conduit volume (LACV) = LV stroke volume - (Vol_{max}-Vol_{min})

LA active emptying volume (LAAEV) = Vol_{pre-A} - Vol_{min},

LA active emptying fraction (LAAEF) = LA_{AEV}/Vol_{pre-A},

LA total emptying volume (LATEV) = Vol_{max} - Vol_{min},

LA total emptying fraction (LATEF) = LA_{TEV}/Vol_{max}

Statistical Analysis

SPSS 18 program was used for statistical evaluation. For comparing variables, the Student's t-test or Mann-Whitney U test were used. The chi-square and Fischer's Exact test were used for categorical variables. Pearson correlation analysis method was used for correlation analysis between variables. Parametric variables were expressed as mean ± standard deviation and categorical variables were expressed as percentage. Statistical significance was accepted when p-value was <0.05.

Results

The study consisted of 90 patients, 50 for the CSF group and 40 for the control group. Male gender was higher in the CSF group (p=0.02). We did not find a difference between the two groups for age, HT, DM, smoking, blood pressure, heart rate, BMI, and BSA. Hyperlipidemia was higher and statistically significant in the CSF group (p=0.01) (Table 1).

Platelet, mean platelet volume, neutrophil/lymphocyte ratio (NLR), low-density lipoprotein (LDL), and C-reactive protein were significantly higher in the CSF group (Table 2).

Table 1. Demographic characteristics of the study groups

	CSF (n=50)	Control group (n=40)	p-value
Gender, male (n, %)	40 (80)	23 (57.5)	0.020
Age (years)	48.6±12.5	47.8±6.0	NS
Body surface area (BSA)(m^2)	1.98±0.2	1.91±0.1	NS
Systolic blood pressure (mmHg)	120±11	119±13	NS
Diastolic blood pressure (mmHg)	73±8	74±9	NS
Heart rate (bpm)	72±10	78±9	NS
Body mass index (BMI) (kg/m^2)	28±3.6	27.4±3.2	NS
Hyperlipidemia (n, %)	19/38	6/15	0.010
Smoking (n, %)	22/44	14/35	NS
Hypertension (n, %)	10/20	14/35	NS
Diabetes mellitus (n, %)	3/6	3/7.5	NS

CSF: Coronary slow flow, NS: Non-significant, n: Number

Table 2. Baseline hematological and biochemical parameters in the study

	CSF (n=50)	Control group (n=40)	p-value
White blood cell count ($10^3/\mu L$)	8.7±2.2	8.2±2.0	NS
Hemoglobin (g/dL)	14±1.9	13.8±1.5	NS
Hematocrit (%)	42.4±5.3	42.2±4.5	NS
Platelet count ($10^3/mm^3$)	245±56	221±27	0.010
MPV (μm^3)	8.4±0.9	7.9±0.7	0.010
Neutrophil ($10^3/mm^3$)	5.6±2.1	4.7±1.4	0.020
Lymphocyte ($10^3/mm^3$)	2.4±0.8	2.6±0.2	NS
Neutrophil/lymphocyte ratio	2.5±1.6	1.8±0.5	0.010
RDW (%)	13.9±1.0	13.4±0.6	0.006
Creatinine (mg/dL)	0.83±0.2	0.77±0.1	NS
CRP (mg/dL)	1.6±0.4	0.9±0.2	<0.001
Glucose (mg/dL)	102±18	103±22	NS
Total cholesterol (mg/dL)	189±48	179±32	NS
LDL (mg/dL)	108±31	91±24	0.006
HDL (mg/dL)	41±10	51±14	<0.001
Triglyceride (mg/dL)	190 (110-279)	157 (96-237)	NS

Significant p-values are shown in bold.
CSF: Coronary slow flow, MPV: Mean platelet volume, RDW: Red blood cell distribution width, CRP: C-reactive protein, LDL: Low density lipoprotein, HDL: High density lipoprotein, NS: Non-significant, n: Number

We did not find statistical variation between the two groups for LVEDD, LVESD, EF, LVEDV, LVESV, A, and Sm. In Doppler analysis, E, E/A, and Em were significantly lower in the CSF group. LA, Am, E/Em, LAV_{max}, LAV_{min}, LAV_{preA}, index volumes, LAAEV, LATEV, and LAAEF were higher in the CSF group. LAPEV, LAPEF, and LAV conduit were found to be lower in the CSF group (Tables 3 and 4).

Coronary angiographic findings were statistically different between the two groups. As expected, the number of frames for CSF patients was higher than the control group (Table 5).

Table 3. Echocardiographic parameters of the patients

	CSF (n=50)	Control group (n=40)	p-value
EF (%)	60±3	60±2	NS
LVEDD (cm)	4.7±0.2	4.4±0.2	NS
LVESD (cm)	3.0±0.2	2.9±0.4	NS
IVS (cm)	1.1±0.1	1.0±0.09	NS
LVDV (mL)	73.1±21.5	71.2±13.6	NS
LVSV (mL)	34.8±9.1	30.1±6.9	NS
LA diameter (cm)	3.6±0.2	3.3±0.1	**<0.001**
Mitral-E (m/s)	0.65±0.1	0.73±0.1	**<0.001**
Mitral-A (m/s)	0.68±0.1	0.66±0.1	NS
E/A	0.99±0.2	1.16±0.3	**0.003**
Em (m/s)	0.099±0.02	0.12±0.02	**<0.001**
Am (m/s)	0.12±0.02	0.10±0.02	**0.010**
Em/Am	0.83±0.2	1.2±0.3	**<0.001**
Sm (m/s)	0.095±0.02	0.092±0.02	NS
E/E'	7.1±2.1	5.9±1.3	**0.003**

Significant p-values are shown in bold.
LV: Left ventricular, CSF: Coronary slow flow, A: Mitral late velocity, E: Mitral early velocity, Em: Early diastolic velocity, Sm: Systolic myocardial velocity, EF: Ejection fraction, LVEDD: LV end-diastolic diameter, LVESD: LV end-systolic diameter, IVS: intact ventricular septum, NS: Non-significant, n: Number

Table 4. Left atrial volumes, index volumes and fractions

	CSF (n=50)	Control group (n=40)	p-value
LAV_{max} (mL)	33±10	21.6±4.6	**<0.001**
$LAVI_{max}$ (mL/m^2)	16.6±4.6	11.3±2.1	**<0.001**
LAV_{min} (mL)	12.7±5.2	9.5±2.5	**<0.001**
$LAVI_{min}$ (mL/m^2)	6.4±3.3	5.0±1.3	**<0.001**
LAV_{preA} (mL)	25.4±7.6	13±3.5	**<0.001**
$LAVI_{preA}$ (mL/m^2)	12.8±2.8	6.8±2.6	**<0.001**
LATEV (mL)	20.3±7.5	12.1±2.8	**<0.001**
LATEVI (mL/m^2)	10.1±3.6	6.3±1.5	**<0.001**
LAPEV (mL)	7.6±3.8	8.6±2.6	**0.010**
LAPEVI (mL/m^2)	3.8±1.9	4.5±1.4	**0.010**
LAAEV (mL)	12.3±3.8	3.5±2.1	**<0.001**
LAAEVI (mL/m^2)	6.2±1.9	1.85±0.8	**<0.0001**
LACV (mL)	22.5±11.1	29±6.2	**<0.001**
LAVCVI (mL/m^2)	11.3±5.1	15.1±2.9	**<0.001**
LATEF (%)	61.4±9.4	57.3±6.4	NS
LAPEF (%)	23±9.1	39±9.6	**0.003**
LAAEF (%)	48±12.8	27±9.1	**0.008**

Significant p-values are shown in bold.
NS: Non-significant, CSF: Coronary slow flow, n: Number

A significant positive relationship was observed between Frame LAD and E/Em (p<0.001) and between Frame LAD and LAAEF (p<0.001). The graphs showing the relationship between Frame LAD and LAPEF, LAAEF, LATEF are shown in Figure 1. A significant positive relationship was observed between the Mean Frame and LAAEF (p=0.002) and between LAPEF and E/A (p<0.001). The relationship between the Mean Frame and LAAEF and the relationship between LAAEF and E/A is shown in Figure 2. Correlation analyses are shown in Table 6.

Discussion

CSF prevalence is 1% in patients with ACS[16]. In the TIMI-IIIA study, coronary arteries were found to have normal or non-significant CAD in 4% of patients with the diagnosis of unstable angina pectoris[17]. Atherosclerosis is a chronic, multifactorial and progressive process starting from early childhood. In intracoronary ultrasonography studies, atherosclerotic changes were observed in the coronary arteries of patients with CSF, and these lesions progressed to the media layer rather than the lumen.

Table 5. Coronary angiography parameters

	CSF (n=50)	Control group (n=40)	p-value
Frame LAD	36±8.9	29.5±2.5	<0.001
Frame CorLAD	20.5±5.1	17.3±1.5	<0.001
Frame Cx	24.3±7.8	20.7±2.4	0.006
Frame RCA	26±8.7	19.6±1.7	<0.001
Frame mean	28.7±5.6	23.3±1.7	<0.001

Significant p-values are shown in bold.
LAD: Left anterior descending artery, Cx: Circumflex artery, RCA: Right coronary artery, CSF: Coronary slow flow, n: Number

Table 6. Correlation analysis

	LAPEF	LAAEF	LATEF	E/A	E/Em
Frame LAD	r=-0.19 p=0.100	r=0.66 p<0.001	r=0.43 p<0.001	r=0.4 p=0.004	r=0.341 p<0.001
Frame CorLAD	r=-0.28 p=0.040	r=0.1 p=0.200	r=-0.17 p=0.900	r=0.31 p=0.024	r=0.01 p=0.900
Frame Cx	r=-0.2 p=0.100	r=0.05 p=0.600	r=-0.12 p=0.400	r=-0.27 p=0.590	r=0.06 p=0.66
Frame RCA	r=0.1 p=0.500	r=0.10 p=0.400	r=0.13 p=0.360	r=-0.1 p=0.460	r=0.08 p=0.560
Mean Frame	r=-0.16 p=0.260	r=0.42 p=0.002	r=0.24 p=0.090	r=-0.24 p=0.080	r=0.3 p=0.070
LAPEF	1	r=-0.14 p=0.330	r=0.52 p<0.001	r=0.44 p<0.001	r=0.12 p=0.380
LAAEF	r=-0.13 p=0.340	1	r=0.76 p<0.001	r=-0.40 p=0.003	r=0.14 p=0.330
LATEF	r=0.52 p<0.001	r=0.76 p<0.001	1	r=-0.05 p=0.700	r=0.17 p=0.210

LAD: Left atrial, LAD: Left anterior descending artery, Cx: Circumflex artery, RCA: Right coronary artery, A: Mitral late velocity, E: Mitral early velocity, Em: Early diastolic velocity, Sm: Systolic myocardial velocity, LAPEV: LA passive emptying volume, LAPEF: LA passive emptying fraction, LATEF: LA total emptying fraction, n: Number

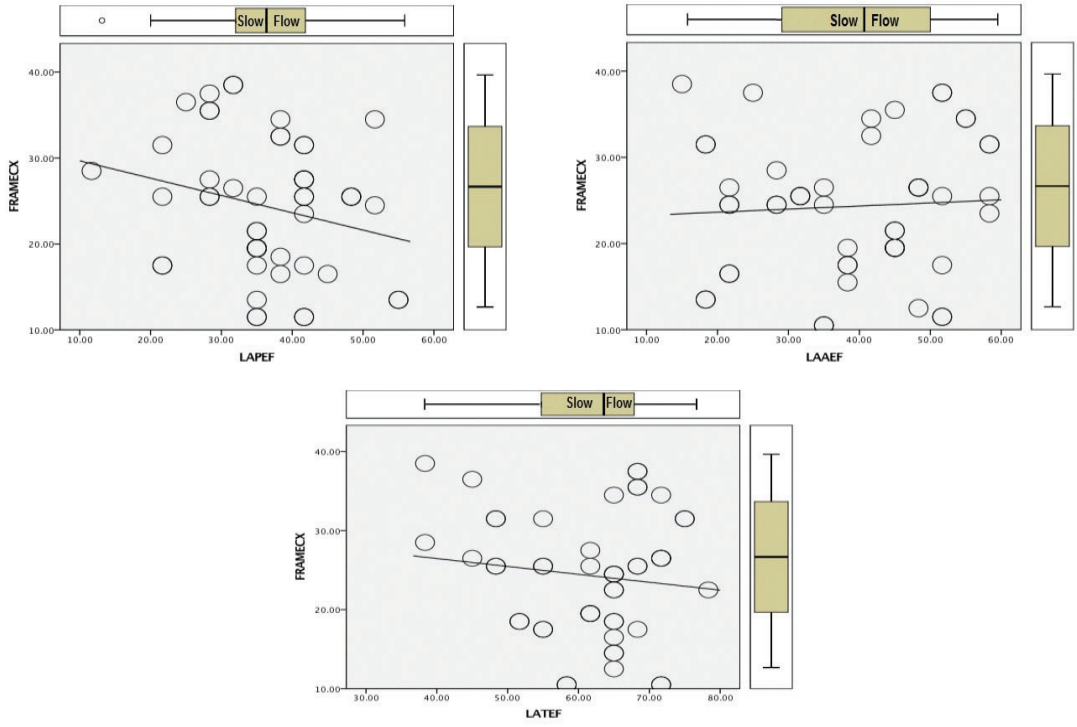

Figure 1. Charts showing correlations between Frame LAD and LAPEF, LATEF, and LAAEF

LAD: Left anterior descending artery, LAPEF: LA passive emptying fraction, LATEF: LA total emptying fraction, LAAEF: LA active emptying fraction

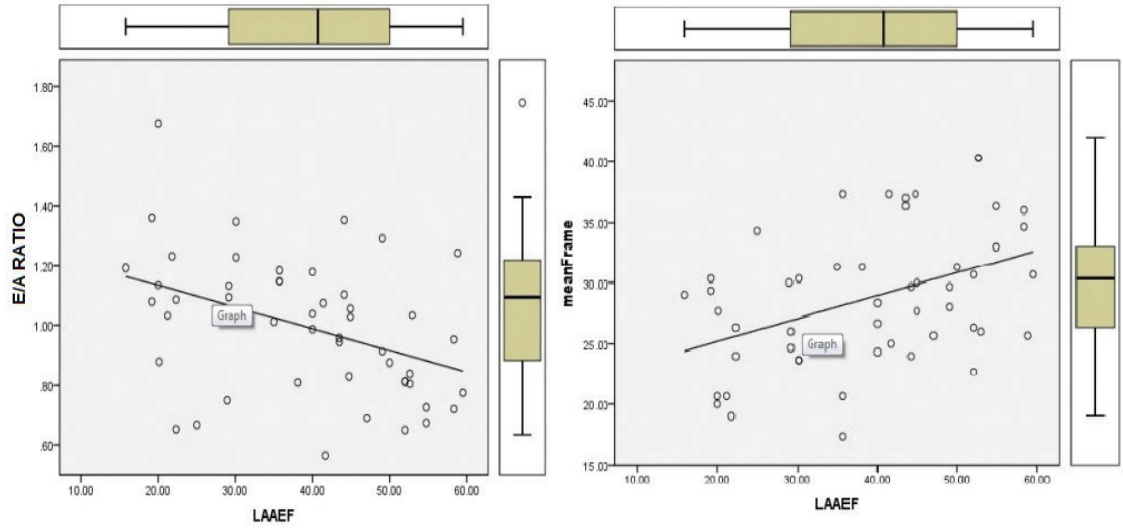

Figure 2. Correlation graphs between LAAEF and E/A, Mean Frame and LAAEF

LAAEF: LA active emptying fraction, A: Mitral late velocity, E: Mitral early velocity

Widespread calcification, intimal irregularity, atheroma plaques were observed in the vessel wall[7,8]. Therefore, CSF is believed to be an early process of atherosclerotic CAD.

Many studies have demonstrated that diastolic functions impair before LV systolic function in CAD. Although it is non-obstructive CAD, CSF affects left ventricular diastolic functions by causing ischemia at the microvascular level[18]. Li et al.[19], Wang et al.[20] and Suner et al.[9] showed that LV diastolic functions were significantly reduced in patients with CSF. Pritchett et al.[21] showed that the LA volume index indicated the severity of diastolic dysfunction. In our study, we found the E, E/A, and Em parameters to be lower in the CSF patients ($p<0.001$, $p=0.003$, $p=<0.001$, respectively). The Am was higher in the CSF group than in the control group ($p=0.01$). Em/Am was lower in the CSF group than in the control group ($p<0.001$). Our findings were consistent with studies showing LVDD in patients with CSF.

Tsang et al.[22] reported that the left atrial volume index was a sensitive morphophysiological indicator of diastolic functions and a useful parameter for future cardiovascular mortality and morbidity. LA volume and fractions may show the severity of LVDD[23]. In our study, LAV_{max}, LAV_{min}, LAV_{preA}, and index volumes were higher for the CSF group ($p<0.001$). E/Em was higher in the CSF group than in the control group ($p=0.003$). The E/Em ratio correlates with pulmonary capillary pressure and LV end-diastolic pressure. The LV filling consists of three-phase and involves the depot function of the LA during ventricular systole, active pump function in the late diastole, the passive conduit function in the early diastole. Left atrial mechanical ejaculation function is important for patients with LVDD[24]. Despite the decrease in passive filling in early diastole in cases such as ischemia, HT, and old age with which LV diastolic function impairs, active atrial pump function in late diastole has been found to maintain adequate cardiac output[25,26]. This contribution of LA to LV is explained by the Frank-Starling mechanism[27]. In our study, we found that LAPEV and fraction were low, LAAEV and fraction were increased. This can be explained as the compensation for keeping the LV stroke volume constant. While LV diastolic functions decrease, LA pump functions increase. However, this increase is not continuous, and after a certain point, LA myocyte functions may decrease due to intrinsic causes.

LA has three functions: Reservoir function, conduit function, and pump function[28]. The conduit function consists of two phases: passive discharge phase (fast filling phase) and diastasis phase. The passive phase depends on the relaxation, compliance, and viscoelastic properties of the myocardium, and the diastasis phase usually depends on LV compliance[29]. LAV conduit and index volumes were lower in CSF patients than in the control patients ($p<0.001$). This finding may support the reduction of LV compliance due to ischemia in patients with CSF.

A significant negative relationship between E/A and LAAEF was observed and it can be concluded that impaired diastolic functions will cause a rising in left atrial active functions. We found a significant positive relationship between Frame LAD and LAAEF, E/Em. There was no such relationship with RCA and Cx. These findings show that as the frame of LAD increases, left atrial contractions increase, and LA pressure increases. It can be explained by the fact that LAD feeds the larger myocardial area and causes greater microvascular ischemia.

Conclusion

Impaired LV diastolic functions and significant changes in LA volumes were found in patients with CSFP. We well know that increase in LA volumes causes high atrial fibrillation rates, stroke, and death. Therefore, maintaining the sinus rhythm in patients with CSF is becoming more important.

Ethics

Ethics Committee Approval: After the protocol for the study was prepared, approval was obtained from the Ethics Committee of Dicle University Faculty of Medicine (decision no: 190, date: 26/12/2015).

Informed Consent: All patients included in the study were informed about the study in accordance with the

ethical principles of human research, as stated in the second Helsinki declaration. All of the patients accepted to participate the present study.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: B.A., E.T., H.Ç., Concept: B.A., H.Ç., Design: B.A., T.P., H.Ç., Data Collection and Process: B.A., T.P., M.Ö., A.A., M.K., Analysis or Interpretation: B.A., E.T., Literature Search: B.A., E.T., Writing: B.A., E.T.

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