Original Article

Comparisons of Myocardial Deformation Between Cases With Normal Coronary Arteries and Patients With Coronary Slow Flow

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ABSTRACT

Background: Slow coronary flow (SCF) is a condition defined as the delayed passage of the contrast agent in the absence of angiographic coronary artery stenosis. Left ventricular (LV) systolic and diastolic dysfunction has been reported in patients with SCF, which can influence their functional capacity. This study compared myocardial deformation between cases with normal coronary arteries and patients with SCF.

Methods: This cross-sectional comparative study included 32 patients with SCF and 32 controls with normal epicardial coronary arteries (NECA). After coronary angiography, echocardiography was done for all the participants and the results were compared between the groups.

Results: A total of 64 patients were studied. The mean global longitudinal peak systolic strain (GLPS.Avg) was 16.85. SCF was significantly more frequent in the men than in the women ($P < 0.05$). Diabetes mellitus, systemic hypertension, a history of past or current smoking, and a family history of coronary artery disease (CAD) in the patients with SCF and dyslipidemia in the NECA group were more frequent, although these differences were not statistically significant. GLPS.Avg and global longitudinal peak systolic stress in the apical 4-chamber view (GLPS.A4C) in the patients with SCF were significantly lower than those in the NECA group. Global strain in the apical 2- and 3-chamber views (GLPS.A2C and GLPS.LAX), septal E, septal A, lateral E, lateral A, and right ventricular Sm (peak myocardial systolic velocity) were also nonsignificantly lower in the patients with SCF.

Conclusions: Strain imaging using 2D echocardiography was abnormal in our patients with SCF, in comparison with the NECA group. These abnormalities may represent subtle systolic and/or diastolic dysfunction in patients suffering from SCF. ***(Iranian Heart Journal 2019; 20(3): 84-90)***

KEYWORDS: Echocardiography, Myocardial deformation, NECA, Slow flow, Strain

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Received: January 1, 2019 **Accepted:** April 8, 2019

In some patients with chest pains scheduled for selective coronary angiography, a slow contrast-agent passage is observed through the epicardial coronary arteries in the absence of obvious stenosis; this condition is termed "the slow coronary flow" (SCF) phenomenon and has a prevalence rate of 2–34% among patients under coronary angiography. [1, 2] However, in the majority of previous studies, systolic and diastolic dysfunction was reported in patients with SCF. [3-10] Some controversial results also have been reported. [11, 12] SCF may affect the functional capacity of patients. [7] These patients may experience life-threatening arrhythmias and sudden cardiac death, but the pathological mechanism and the related effects on the left ventricular function are not clear yet. [4] Male gender and a high body mass index are among the contributing risk factors. [2, 12, 13] Additionally, metabolic disorders are more common among these patients. [14, 15]

The duration and extension of tissue deformation are important factors in myocardial contractility, and increased time to peak systolic strain is reported in these patients with a predictive role for worse regional myocardial contractility. [16] Albeit not established yet, endothelial and microvascular dysfunction could be among the possible etiologies of SCF. [17-20] There are some controversies regarding the coronary flow reserve (CFR). Also in some studies, the coronary risk factors in these patients were not different from normal coronary subjects. [17-20] Since only one-third of patients with positive noninvasive tests would have stenosis in coronary angiography, finding some noninvasive tests with acceptable sensitivity and specificity to assess the coronary artery is an issue of importance. Two- and 3D echocardiography and strain tests are useful tools to evaluate myocardial deformation in patients with stable coronary artery disease. [21, 22] Given the controversies in the existing literature on patients with SCF and the related effects on the left ventricular function, we carried out the present study to compare myocardial deformation between cases with normal coronary arteries and patients with SCF.

METHODS

This cross-sectional comparative study, conducted in a tertiary health care center in Tehran, Iran, from 2016 to 2017, recruited 32 consecutive patients with SCF as the case group and 32 subjects with normal epicardial coronary artery angiography (NECA) as the control group. The inclusion criterion was SCF (TIMI frame count > 27) or NECA in angiography. The exclusion criteria were comprised of a left ventricular ejection fraction < 55%, previous myocardial infarction or percutaneous coronary intervention, coronary artery disease, a history of arrhythmias, and valvular heart diseases with moderate-to-high severity.

The study protocol was approved by the local ethics committee. No additional tests were done for the patients, and nor were any extra fees charged. After angiography, the patients underwent echocardiography by blinded cardiologists. Myocardial deformation was determined based on echocardiographic diagnostic criteria and compared between the 2 groups.

Data analysis was performed on the 64 subjects, consisting of 32 subjects in the control group and 32 patients in the case group. The data analyses were performed using SPSS software, version 20.0 (Statistical Procedures for Social Sciences; Chicago, Illinois, USA). The χ^2, Fisher, Kruskal–Wallis, Kolmogorov–Smirnov, Mann–Whitney *U*, and regression tests were used for the analyses, and the results were considered statistically significant at a *P* value < 0.05.

RESULTS

In each group, 32 subjects were present (20 male and 12 female patients in the SCF group vs 12 male and 20 female subjects in the NECA group). SCF was significantly more frequent in

the men than in the women ($P < 0.05$). The mean age, the body mass index, and global strain were 51.5 years, 28.9 kg/m², and 16.85, respectively (Table 1). Diabetes mellitus, hypertension, smoking, and a family history of coronary artery disease in the patients with SCF and dyslipidemia in the NECA group were more frequent, although these differences were not statistically significant ($P > 0.05$). The serum uric acid level was significantly higher in the SCF group ($P < 0.05$). The mean global longitudinal peak systolic strain (GLPS.Avg) and global strain in the apical 4-chamber view (GLPS.A4C) were significantly lower in the SCF group. The results were the same between the 2 groups for GLPS.A2C, GLPS.LAX, septal E, septal A, lateral E, lateral A, and RV Sm ($P > 0.05$).

According to the regression analysis, SCF ($P = 0.033$) and diabetes ($P = 0.019$) independently resulted in a significant decrease in GLPS.Avg (Fig. 1). Additionally, SCF ($P = 0.022$) and diabetes ($P = 0.057$) independently resulted in a significant decrease in GLPS.A4C and diabetes resulted in a significant decrease in GLPS.LAX ($P = 0.014$). Similar associations were not seen for GLPS.A2C and also hypertension and all the values of strain ($P > 0.05$).

As is shown in Table 2 and Figure 2, across the SCF subgroups, the strain test results were the same ($P > 0.05$). In addition, according to the Mann–Whitney *U*-test, SCF showed no significant difference for strain in the right coronary and left anterior descending arteries ($P > 0.05$).

Table 1. Distribution of the variables across the groups

	NECA			SCF		
Variable	Percentile			Percentile		
	25	50	75	25	50	75
Age (y)	46.25	52.50	58.75	46.25	50.00	61.75
Weight (kg)	70.25	78.00	86.00	73.50	80.00	87.75
Height (cm)	157.0	161.0	170.5	160.5	170.0	174.7
BMI (kg/m^2)	26.59	29.10	31.04	26.76	28.11	30.64
Hgb (mg/dL)	11.9	13.4	14.5	13.0	13.7	14.7
Plt (n/µL)	187250	240500	284250	193000	231500	262000
FBS (mg/dL)	88	97	118	95	101	114
TG (mg/dL)	91	109	144	93	116	157
Chol (mg/dL)	127	145	173	117	155	167
HDL (mg/dL)	35	38	47	32	39	44
LDL (mg/dL)	65	77	100	65	89	97
Uric acid (mg/dL)	3.8	4.9	5.7	4.4	5.6	6.2
AST (IU/L)	17	19	27	17	20	22
ALT (IU/L)	16	22	34	16	22	34
Bil.T (mg/dL)	0.60	0.60	0.80	0.60	0.80	0.97
Bil.D (mg/dL)	0.12	0.20	0.20	0.10	0.20	0.20
LVEF (%)	55	55	55	50	55	55
MV.E (m/s)	0.50	0.62	0.69	0.53	0.64	0.75
MV.A (m/s)	0.48	0.59	0.76	0.55	0.62	0.73
MV.E to A	0.79	0.94	1.31	0.76	1.06	1.23
Septal Sm (m/s)	0.06	0.07	0.08	0.06	0.07	0.07
Septal E (m/s)	0.06	0.07	0.10	0.06	0.06	0.08
Septal A (m/s)	0.07	0.08	0.10	0.07	0.08	0.09
Lateral Sm (m/s)	0.07	0.08	0.09	0.07	0.08	0.10
Lateral E (m/s)	0.08	0.10	0.11	0.06	0.09	0.10
Lateral A (m/s)	0.07	0.09	0.10	0.07	0.08	0.10
RV Sm (m/s)	0.10	0.12	0.13	0.11	0.11	0.12
GLPS. LAX (%)	15.72	17.25	19.52	14.00	16.65	17.72
GLPS.A4C (%)	13.65	17.95	19.42	12.60	15.50	17.50
GLPS.A2C (%)	16.27	17.50	18.85	15.12	16.30	18.25
GLPS.Avg (%)	16.30	17.50	19.10	14.40	16.60	17.75

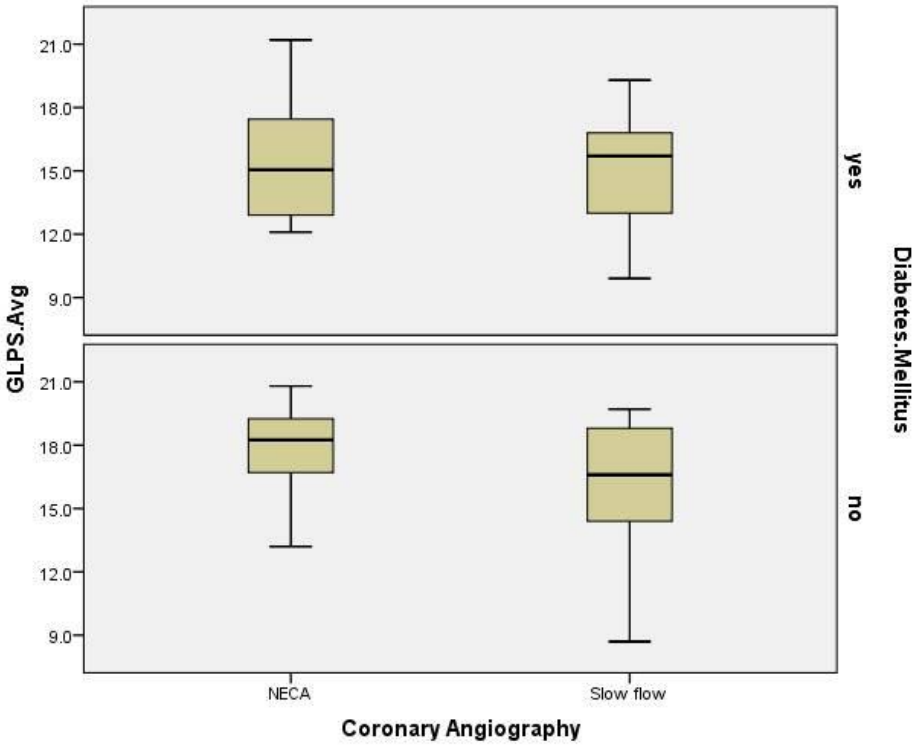

Figure 1. Association between GLPS.Avg and diabetes and SCF

Table 2. Interquartile results for the strain test across the SCF groups

SCF Subgroup			GLPS.LAX	GLPS.A4C	GLPS.A2C	GLPS.Avg
SCF LAD	N		3	3	3	3
	Percentiles	25	12.000	7.300	13.200	11.100
		50	12.500	13.900	13.400	13.000
		75	.	.	.	.
SCF RCA	N		3	3	3	3
	Percentiles	25	11.200	4.900	9.900	8.700
		50	16.500	15.600	16.400	16.600
		75	.	.	.	.
SCF LAD and LCX	N		12	12	12	12
	Percentiles	25	14.900	13.875	14.850	14.575
		50	16.600	15.850	16.000	16.600
		75	17.725	18.650	19.875	18.800
SCF LAD and RCA	N		1	1	1	1
	Percentiles	25	16.500	15.400	18.300	16.700
		50	16.500	15.400	18.300	16.700
		75	16.500	15.400	18.300	16.700
SCF LAD, LCX, and RCA	N		13	13	13	13
	Percentiles	25	14.000	12.500	15.800	14.400
		50	17.200	14.900	17.300	16.600
		75	18.150	18.000	18.850	18.650

SCF, Slow coronary flow; LAD, Left anterior descending coronary artery; RCA, Right coronary artery; LCX, Left circumflex artery

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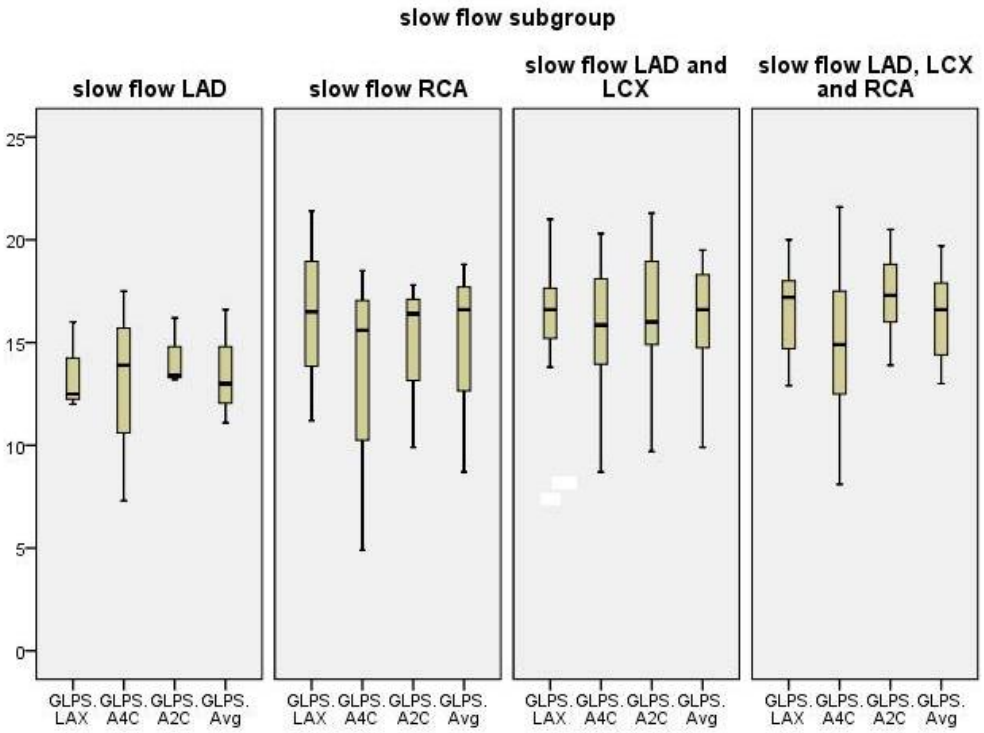

Figure 2. Interquartile results for the strain test across the SCF groups

SCF, Slow coronary flow; LAD, Left anterior descending coronary artery; RCA, Right coronary artery; LCX, Left circumflex artery

DISCUSSION

There are different reports on the effects of SCF on the LV function. [23-25] Hawkins BM et al [13] reported no significant association with diastolic function, but gender and obesity were the independent predictors for SCF. The lack of difference for the body mass index may be due to a small sample size. However, gender had a significant difference in our study. Yilmaz et al [14] reported that patients with SCF had significantly higher total cholesterol, low-density lipoprotein cholesterol, and body mass index. In our study, despite higher levels in the NECA group, there was no significant difference with the subjects suffering from SCF, which may also be due to our sample size. Dai and colleagues [15] demonstrated that smoking and diabetes had higher prevalence rates in the SCF group.

We found that male gender was related to a higher SCF rate. Diabetes mellitus, hypertension, smoking, and a family history of coronary artery disease were similar across the groups. The serum uric acid level was significantly higher in the SCF group. Baykan et al [3] reported LV systolic and diastolic dysfunction in patients with SCF and mitral annular velocities by tissue Doppler imaging were useful in this field. Li et al [4] reported similar results, in addition to a reduced mitral E velocity and E/A ratio, in patients with SCF by comparison with NECA subjects. However, in our study, septal E, septal A, lateral E, and lateral A were not significantly different between the 2 groups.

Balci et al [5] reported preserved RV systolic and diastolic functions despite a decreased left systolic function. Altunkas and colleagues [6] also reported this matter. In our study, RV Sm

was lower in the SCF group, but with no significant difference.

Elsherbiny et al [7] reported that LV systolic and diastolic dysfunction in patients with SCF would affect their functional capacity and they underscored the importance of follow-up in these patients. Gunes et al [8] also reported reduced diastolic and regional functions of the LV, which chimes in with our study. Nurkalem and colleagues [9] revealed that despite a preserved LVEF in patients with SCF, the global and regional strain rate and the longitudinal LV systolic function were abnormal in comparison with those in the control group. These findings are concordant with our findings in some aspects. In our study, the GLPS.Avg and global strain in the apical 4-chamber view (GLPS-A4C) were significantly lower in the SCF group than in the NECA group. We observed no similar associations as regards GLPS.A2C and also hypertension and all the values of strains. A study by Sezgin et al [10] reported diastolic dysfunction in the SCF group, but without a difference in systolic parameters.

CONCLUSIONS

Strain imaging via 2D echocardiography was abnormal in our patients with SCF in comparison with the NECA group. These abnormalities may represent subtle systolic and/or diastolic dysfunction in patients with SCF. Moreover, it is rational to pay more attention to the control of diabetes, hypertension, smoking, and serum uric acid status given their higher levels in patients with SCF and also the independent effect of diabetes on the LV strain.

Conflict of Interest: None

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