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ORIGINAL ARTICLE

Right ventricular function in coronary slow flow: A two-dimensional speckle-tracking echocardiographic study

Koroner yavaş akım fenomeninde sağ ventrikül fonksiyonu: İki boyutlu speckle-tracking ekokardiyografik çalışma

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ABSTRACT

Objective: Coronary slow-flow phenomenon (CSFP) is described as protracted passage of angiographic contrast agent to the distal portion of the epicardial coronary arteries in the absence of stenosis. Few studies have addressed the effects of this condition on right ventricular (RV) dysfunction. The present objective was to assess RV function in CSFP via 2-dimensional speckle-tracking echocardiography (2DSTE).

Methods: A total of 29 patients with CSFP and 29 participants with normal coronary flow were compared regarding RV systolic and diastolic functions. Participants were matched for age, sex, hypertension, and diabetes mellitus. RV systolic and diastolic functions were evaluated with pulsed-wave tissue Doppler echocardiography and 2DSTE.

Results: There were no statistically significant differences between the CSFP group and the control group regarding tissue Doppler echocardiographic and 2DSTE-derived indices.

Conclusion: CSFP was not associated with tissue Doppler echocardiographic and 2DSTE-derived indices of RV systolic and diastolic function.

ÖZET

Amaç: Koroner yavaşakım fenomeni (KYAF) stenoz yokluğunda anjiyografik kontrast maddenin epikardiyal koroner arterlerin distal kısmına geçişinde uzama olarak tanımlanır. Az sayıda çalışma bu durumun sağ ventrikül (SğV) işlev bozukluğuna etkilerini ele almıştır. Burada amaç KYAF'de 2 boyutlu speckle-tracking ekokardiyografi (2BSTE) kullanarak KYAF'de SağV fonksiyonlarını değerlendirmekti.

Yöntemler: Koroner yavaşakım fenomenili toplam 29 hasta ile normal koroner akımı olan 29 katılımcı SğV sistolik ve diyastolik fonksiyonlar açısından karşılaştırıldı. Katılımcılar yaş, cinsiyet, hipertansiyon ve diabetes mellitus açısından eşleştirildi. Sağ ventrikül sistolik ve diyastolik fonksiyonları atım dalgalı doku Doppler ekokardiyografisi ve 2BSTE ile değerlendirildi.

Bulgular: Doku Doppler ekokardiyografisi ve 2BSTE indeksleri açısından KYAF grubuyla kontrol grubu arasında istatistiksel açıdan anlamlı farklılıklar yoktu.

Sonuç: Koroner yavaşakım fenomeni, SağV'nin sistolik ve diyastolik fonksiyonuna ilişkin doku Doppler ekokardiyografisi ve 2BSTE'nin indeksleri ile ilişkili değildi.

Coronary slow-flow phenomenon (CSFP) is defined as the late filling of the distal part of the coronary artery in the presence of normal coronary artery disease.[1] CSFP is seen in about 1% of coronary angiographies,[2] and can present with acute coronary syndrome[3] and ventricular arrhythmia.[4] There have also been reports of QRS changes in patients with CSFP,[5,6] who tend to require more hospital readmissions than normal coronary patients.[7] In this condition, there is endothelial function disorder, decrease in plasma nitric oxide level,[8] and rise in endothelin level.[9] In addition, inflammatory cytokines are involved in its pathogenesis.[10] Right ventricular (RV) myocardial biopsies reveal patchy fibrosis and myocardial hypertrophy.[11] The literature contains studies, albeit limited, on left ventricular (LV) function, evaluated via echocardiographic modalities, with divergent results.[12–14] Even less information is available regard-

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ing the right ventricle evaluated by echocardiography.[15–17] It should, therefore, come as no surprise that data concerning the effect of CSFP on RV myocardial function is very limited.

Abbreviations:

2DSTE	*Two-dimensional speckle-tracking echocardiography*
CSFP	*Coronary slow-flow phenomenon*
LAD	*Left anterior descending artery*
LCX	*Left circumflex artery*
LV	*Left ventricular*
RCA	*Right coronary artery*
RV	*Right ventricular*
SS	*Systolic strain*
SRS	*Systolic strain rate*
SRE	*Early diastolic strain rate*
SRA	*Late diastolic strain rate*
TAPSE	*Tricuspid annular plane systolic excursion*
TIMI	*Thrombolysis in myocardial infarction*

The aim of the present study was to evaluate RV function using 2-dimensional speckle-tracking echocardiography (2DSTE) in patients with CSFP, and to compare it with that of a control group matched regarding age, sex, diabetes mellitus, and hypertension.

METHODS

Study population

A review of the angiography database, conducted between April 2011 and September 2014, showed that 28595 selective coronary angiographic procedures had been performed at our center. All selective coronary angiographies were performed due to presence of acute coronary syndrome, noninvasive tests suggestive of significant coronary artery disease, and high clinical probability of severe coronary artery disease. A total of 200 patients were identified as having CSFP with no coronary stenosis or <50% coronary stenosis. Coronary angiographic procedures showing CSFP were assessed to identify patients that would fulfill the present criterion: slow-flow pattern in all coronary arteries (see the coronary angiography subsection below for the definition).

From the same angiography database, a control group was selected. Participants had normal coronary arteries or coronary artery stenosis <50%, with normal coronary flow, identified within the same time period. Selected subjects were matched according to age (±5 years), sex, hypertension, and diabetes mellitus. Other inclusion criteria were normal sinus rhythm, LV ejection fraction >55%, tricuspid annular plane systolic excursion (TAPSE) >17 mm, and velocity of s' wave in the tricuspid lateral annulus acquired by pulsed-wave tissue Doppler (RV Sm) >10 cm/s. Exclusion criteria consisted of pulmonary hypertension (estimated systolic pulmonary artery pressure >35 mmHg by echocardiography), coronary ectasia, dilated and hypertrophic cardiomyopathy, pericardial disease, congenital heart disease, any degree of valvular heart stenosis, more-than-mild valvular heart regurgitation, history of myocardial infarction, history of inflammatory disease, history of chemotherapy or mediastinal radiotherapy, liver or kidney disease, bundle branch block, pacemaker rhythm, history of cardiac surgery or percutaneous coronary intervention, hyperthyroidism, hypothyroidism, and low-quality echocardiographic images.

Those who qualified for the patient and control groups were asked to attend clinical visits and undergo echocardiography. A total of 29 patients elected to participate. Histories were obtained, and physical examinations and transthoracic echocardiography were performed between October and November 2014. Subjects newly diagnosed with hypertension or diabetes mellitus were excluded. All echocardiographic examinations were performed by the same echocardiologist, who was blinded to clinical and coronary angiographic data. Written informed consent was obtained from each subject, and the study was approved by the Cardiology Department Research Committee.

Coronary angiography

Selective coronary angiography was performed by femoral artery approach, using the Judkins technique with manual contrast injection. Diagnosis of CSFP was made in accordance with a study by Gibson et al.[18] Briefly, the time between manifestation of contrast agent in the proximal portion of the coronary artery, and its arrival into the distal part of the coronary artery was registered in a cineframe count. Distal bifurcation of the left anterior descending artery (LAD), left circumflex artery (LCX), and the first posterolateral branch of the right coronary artery (RCA) were defined as the distal section. Thrombolysis in myocardial infarction (TIMI) frame count for each coronary artery was obtained by subtracting the distal frame count from the first-appearance frame count. Corrected LAD TIMI frame count was calculated by dividing LAD TIMI frame count by 1.7. Cut-off points of the TIMI frame count for normal coronary flow, in accordance with a study Gibson et al.,18 were 36.2±2.6 for the LAD (corrected cutoff value for the

LAD: 21.1±1.5), 22.1±4.1 for the LCX, and 20.4±3 for the RCA.

Mean TIMI frame count for each subject was calculated by averaging values for the LAD, LCX, and RCA. In the present study, normal coronary flow was defined as values less than the mean, and CSFP was considered values more than 2 SDs above the mean of the reported values in any coronary artery. TIMI was evaluated by 2 cardiologists, and if there were discrepancies, a third cardiologist resolved the issue. In our hospital, selective coronary angiographic images are acquired at 15 frames per second, so frames counted were multiplied by 2 for adjustment.

Standard echocardiography

Transthoracic echocardiography was preformed for all participants in the left lateral decubitus position. A commercial-setting 2–4-MHz probe (Medical Systems S5; GE Healthcare, Little Chalfont, UK) was used to obtain and analyze standard echocardiography and pulsed-wave tissue Doppler imaging. LV internal diameters, LV wall thickness, diastolic RV diameter in the basal and mid cavities, diastolic longitudinal RV diameter, right atrial area, TAPSE, and RV fractional area change were measured according to the guidelines of the American Society of Echocardiography.[19] Tricuspid flow waves, including E and A waves and deceleration time of the E wave, were measured from apical 4-chamber view by pulsed-wave Doppler, in accordance with the guidelines of the American Society of Echocardiography.[20] Subsequently, the average of the peak velocity of these waves in 5 cardiac cycles was measured.

Tissue Doppler echocardiography

Tricuspid annular velocities were provided from apical 4-chamber view using pulsed-wave tissue Doppler imaging. Sample volume (2–4 mm) was placed on the lateral annulus of the tricuspid valve with maximal alignment to the RV free wall. Gain and filter were adjusted to minimize noise. A horizontal sweep rate of 50–100 mm/s and a velocity scale of -20 cm/s and 20 cm/s were chosen. One systolic positive wave (s') and 2 negative waves in early (e') were recorded. Mean peak velocity of these waves in 5 cardiac cycles was recorded. Isovolumic contraction time was defined as the time between the end of the a' wave and beginning of the s' wave. Isovolumic relaxation time was defined as the time between the end of the s' wave and beginning of the e' wave, and the duration of the s' wave was defined as ejection time. These intervals were measured in the lateral annulus of the tricuspid valve. Myocardial performance index, defined as the ratio of isovolumic relaxation time plus isovolumic contraction time to ejection time, was calculated, as was the ratio of peak tricuspid E wave to peak e'wave.

Two-dimensional speckle-tracking echocardiography

Another commercial-setting, 2—4-MHz probe (EKO 7; Samsung Medison, Seoul, South Korea) was used to obtain images and analyze the longitudinal deformation of the RV by 2DSTE. Three consecutive cardiac cycles at end-expiration from apical 4-chamber view at 40–80 frames per second were stored in this setting. Longitudinal deformation analysis was performed on the stored images in the memory of this setting. The end of ventricular systole was automatically determined by software. Endocardial RV border tracings were manually performed at end-systole, and the epicardial borders were automatically defined. The free wall of the RV was automatically divided into 3 segments: basal, mid, and apical. Region of interest was manually adjusted. When at least 1 myocardial segment was visible in the RV free wall, but with poor image quality after several efforts, the patient was excluded from the study. Each RV free-wall segment was individually analyzed, and deformation indices were calculated by averaging values of the 3 segments. Level zero was considered the beginning of the QRS. RV strain curve included 1 negative predominant wave throughout the cardiac cycle, so the peak of this wave was measured as peak systolic strain (SS). The systolic strain rate (SRS) curve of the RV comprised 1 negative systolic wave and 2 positive waves in early (SRE) and late (SRA) diastole (Figure 1).

Interobserver variability was calculated as coefficient variation. It was evaluated in 9 patients for the SS, SRS, SRE, and SRA of the RV, and was 8.8%, 8.1%, 7.2%, and 7.1% after 3 months, respectively. Intraobserver variability was calculated as coefficient variation. It was evaluated in 9 patients for the SS, SRS, SRE, and SRA of the RV, and was 3.6%, 5.3%, 5.1%, and 7.5% after 3 months, respectively.

Statistical analysis

Categorical variables were summarized as frequen-

cies and percentages. Continuous variables without normal distribution were reported as median and interquartile range boundaries. Continuous variables with normal distribution were reported as mean±SD. Kolmogorov-Smirnov test was used to evaluate the distribution of continuous variables. Continuous data were compared using unpaired t-test (if normally distributed) and Mann-Whitney U test (if not normally distributed). Chi-square test or Fisher's exact test were used to compare categorical variables, which were appropriated, and p values <0.05 were considered statistically significant. SPSS software (version 21.0; SPSS Inc., Chicago, IL, USA) was used for statistical analyses.

RESULTS

Presently recruited were 29 patients with CSFP and 29 participants with normal coronary flow. Baseline clinical characteristics, laboratory data, and angiographic profiles of the CSFP and control groups are summarized in Table 1. In the CSFP group, 15 (52%) patients had normal electrocardiography, 10 (35%) had insignificant ST-T changes (ST depression <0.05 mm, flat T wave, or T wave inversion <1 mm), and 4 (13%) had significant ST-T changes (ST depression >0.05 mm or T wave inversion >1 mm). Both groups were similar in terms of clinical and lab data, with the exceptions of body mass index (30.8±4.6 vs 28.0±4.1, p=0.019) and high-density lipoprotein levels (36.9±8.1 vs 42.4±10.1, p=0.031). TIMI frame count was greater in the CSFP group than in the control group: corrected LAD (41.2 [34.7–51.2] vs 16.5 [12.4–20.0]; p<0.001), LCX (40.0 [34.0–57.0] vs 16.0 [12.0–20.0]; p<0.001), and RCA (28.0 [24.0–41.5] vs 12.0 [9.0–18.0]; p<0.001). The RCA had a slow-flow pattern in all patients. Table 2 presents standard echocardiographic data in the CSFP and normal groups. Baseline echocardiographic data were similar in both groups. Results of 2DSTE indices and tissue Doppler imaging data are summarized in Table 3. There were no statistically significant differences between the 2 groups, regarding these indices.

DISCUSSION

In the present study, the RV function of the CSFP group was compared to that of the control group, which comprised individuals with normal coronary flow. The groups were matched for age, sex, hypertension, and diabetes mellitus in a 1:1 patient:control design. Analysis showed no statistically significant differences between the 2 groups regarding tricuspid pulsed-wave, RV tissue Doppler, and RV deformation indices.

Only a few studies have addressed RV function in patients with CSFP, and somewhat discrepant results have been reported.[15–17] While some studies have shown no differences between systolic and diastolic parameters, evaluated by tissue Doppler imaging,[16,17] 1 showed that e' and E/e' ratio of the CSFP group were different from those of the control group.[15] In the only existing study in which RV function was evaluated with 2DSTE, Wang et al.15 found out that SRE in CSFP patients was different from that of the control group, though no other statistically significant differences were found in other 2DSTE indices. The present findings are in agreement with certain findings of Wang and et al., as no detectable difference in RV systolic functions were found using tissue Doppler echocardiography and 2DSTE. However, the present findings regarding RV diastolic function diverge. In order to account for this discrepancy, it should be noted that unlike the study conducted by Wang et al., the present included a 1:1 patient:control matching design regarding age, sex, hypertension, and diabetes mellitus. Moreover, the RCA presently displayed a slow-flow pattern in all patients. Furthermore, it seems that patients with gross RV dysfunction (detected by TAPSE or RV Sm) were included in the investigation byWang et al., while these individuals were excluded from the present study.

Evaluation of LV function with tissue Doppler echocardiography and 2DSTE has been performed in a limited number of studies, with certain discrepancies. Some have demonstrated differences in systolic or diastolic function between CSFP and control groups, while others have reported none.[12–15] It seems that available information on LV function in CSFP is still a matter of controversy.

2DSTE is a relatively new echocardiographic modality that allows for quantitative assessment of global and regional myocardial function. It is free from insonation angle dependency and cardiac translational motion.[21–24] However, although this modality was initially used to analyze LV function, it is currently applicable to the evaluation of RV function in many

conditions such as RV myocardial infarction and coronary artery disease.[25–28]

A recent study demonstrated that in patients with CSFP, nicorandil treatment raised nitric oxide levels,

Table 1. Clinical and laboratory characteristics of patient and control groups

Characteristics	Patient Group (n=29)	Control Group (n=29)	*p*
Age (y)	54.2±7.9	54.0±9.2	0.950
Sex (male), n (%)	20 (69.0)	20 (69.0)	
Body mass index (kg/m^2)	30.8±4.6	28.0±4.1	0.019
Hypertension, n (%)	10 (35)	10 (35)	
Diabetes, n (%)	7 (24)	7 (24)	
Dyslipidemia, n (%)	13 (45)	13 (45)	
Smoking, n (%)	10 (35)	8 (28)	0.570
Family history of CAD, n (%)	8 (28)	5 (17)	0.345
Symptoms			
Asymptomatic, n (%)	0 (0.0)	1 (3)	
Atypical chest pain, n (%)	13 (45)	9 (31)	0.279
Typical chest pain, n (%)	14 (48)	14 (48)	
Dyspnea on exertion, n (%)	2 (7)	5 (17)	0.423
Aspirin, n (%)	22 (76)	17 (59)	0.162
Statin, n (%)	21 (72)	19 (66)	0.570
ACE or ARB, n (%)	12 (41)	7 (24)	0.162
Nitrate, n (%)	3 (10)	2 (7)	1.000
Diuretics, n (%)	3 (10)	3 (10)	
Calcium channel blocker, n (%)	2 (7)	2 (7)	
Fibrate, n (%)	1 (3)	1 (3)	
Oral antidiabetic agents, n (%)	5 (17)	6 (21)	0.738
Insulin, n (%)	1 (3)	1 (3)	
Beta blocker, n (%)	11 (38)	11 (38)	
Fasting blood sugar (mg/dL)	96.5 (89.2–113.2)	104.0 (91.5–119.0)	0.404
Triglyceride (mg/dL)	153.0 (110.0–192.2)	123.0 (91.0–169.0)	0.186
Cholesterol (mg/dL)	169.0±39.4	177.7±42.2	0.445
High-density lipoprotein (mg/dL)	36.9±8.1	42.4±10.1	0.031
Low-density lipoprotein (mg/dL)	109.5 (83.0–125.70)	103.0 (85.5–135.0)	0.755
Hemoglobin	15.3±1.3	14.7±1.5	0.081
Creatinine	0.8±0.2	0.8±0.2	0.602
Heart rate (/min)	71.0±12.0	74.0±13.8	0.283
Systolic blood pressure (mmHg)	126.0±13.3	122.5±14.4	0.353
Diastolic blood pressure (mmHg)	80.8±9.6	76.0±11.1	0.086
Left anterior descending artery (frame)	41.2 (34.7–51.2)	16.5 (12.4–20.0)	<0.001
Left circumflex artery (frame)	40.0 (34.0–57.0)	16.0 (12.0–20.0)	<0.001
Right coronary artery (frame)	28.0 (24.0–41.5)	12.0 (9.0–18.0)	<0.001
Mean total frame count (frame)	37.8 (32.0–46.8)	15.0 (13.0–17.5)	<0.001

CAD: Coronary artery disease; ACEI/ARB: Angiotensin-converting enzyme inhibitor/Angiotensin receptor blocker.

Table 2. Standard echocardiographic data in the patient and control groups

Variables	Patient Group (n=29)	Control Group (n=29)	*p*
Left ventricular end-diastolic dimension (cm)	4.7±0.5	4.5±0.5	0.156
Left ventricular end-systolic dimension (cm)	3.2±0.5	3.0 ±0.4	0.137
Interventricular septum (cm)	0.8±0.1	0.9±0.1	0.819
Posterior wall (cm)	0.8±0.1	0.8±0.1	0.649
Left ventricular ejection fraction (%)	61.0±4.2	63.0±4.4	0.095
Right ventricular basal cavity diameter (cm)	2.8±0.4	2.8±0.3	0.578
Right ventricular mid-cavity diameter (cm)	2.8±0.3	2.7±0.4	0.681
Right ventricular longitudinal diameter (cm)	6.2±0.7	6.0±0.8	0.427
Tricuspid annular plane systolic excursion (cm)	2.2±0.4	2.2±0.3	0.912
Systolic pulmonary artery pressure (mmHg)	26.0±4.0	26.0±4.0	0.423
Right ventricular end-diastolic area (cm^2)	13.7±1.5	13.6±1.9	0.607
Right ventricular end-systolic area (cm^2)	7.5±0.8	7.3±1.0	0.518
Right ventricular fractional area change (%)	45.0±6.0	45.0±7.0	0.700
Right atrium area (cm^2)	15.3±1.6	14.9±2.0	0.427
Tricuspid E (cm/s)	44.0±12.0	44.0±10.0	0.948
Tricuspid A (cm/s)	40.0±11.0	40.0±13.0	0.952
Tricuspid E /A ratio	1.1 (0.8–1.4)	1.2 (1.0–1.2)	0.692
Tricuspid deceleration time	186.0±47.0	202.0±33.0	0.165

Table 3. Tissue Doppler imaging and two-dimensional speckle-tracking data in the patient and control groups

Variables	Patient Group (n=29)	Control Group (n=29)	*p*
Right ventricle e' (cm/s)	8.5±2.2	9.4±2.0	0.095
Right ventricle a' (cm/s)	13.0±4.0	14.0±3.0	0.132
Right ventricle s' (cm/s)	12.5±2.0	12.4±2.0	0.820
Right ventricle e'/a'	0.64 (0.55–0.74)	0.62 (0.53–0.73)	0.713
Tricuspid E/e'	5.7 (3.8-6.9)	4.6 (3.9–5.7)	0.219
Right ventricle isovolumic contraction time (ms)	70.0±11.0	66.0±9.0	0.149
Right ventricle isovolumic relaxation time (ms)	0.0 (0.0–67.0)	0.0 (0.0–78.0)	0.782
Right ventricle ejection time (ms)	276.0±32.2	278.4±41.2	0.806
Right ventricle myocardial performance index	0.26 (0.22–0.39)	0.27 (0.21–0.48)	0.911
Strain (%)	-20.6 (-24.6– -16.8)	-24.3 (-27.6– -20.0)	0.062
Systolic strain rate (1/s)	-1.8 (-2.1– -1.4)	-1.9 (-2.1– -1.4)	0.388
Early diastolic strain rate (1/s)	1.5 (1.3–2.0)	1.8 (1.3–2.3)	0.222
Late diastolic strain rate (1/s)	1.1 (0.8–1.6)	1.2 (1.0–1.4)	0.591

decreased endothelin levels, and increased mitral E/A ratio.[29] The results of that study also showed that while systolic longitudinal strain rate in some LV segments was increased, there was no change in other LV segments. The authors concluded that the change in nitric oxide and endothelin levels may not be parallel to the change in function of all LV segments. While previous studies showed a drop in

plasma nitric oxide levels[8] and a rise in endothelin levels,[9] the present findings demonstrated no statistically significant differences in RV systolic and diastolic functions between the CSFP and control groups.

In one study, RV endomyocardial biopsies obtained from patients with CSFP showed diseased small coronary arteries, with hyperplasia of the fibromuscular layer, hypertrophy of the media layer, proliferation of the myointimal layer, degeneration of endothelial cells, and swelling of the endothelial cells of the capillaries, encroaching on the lumen. Hypertrophy of the myocardium, deposition of lipofuscin, and patchy fibrosis were also reported.[11]

In spite of these pathological changes in the RV structure, systolic and diastolic dysfunction of the RV was not detected using various echocardiographic modalities in the majority of CSFP studies. It is possible that either these pathological changes result in subtle changes in RV myocardial function that are lower than the detection level of these modalities, or that these pathological changes are mechanically insignificant. These explanations become more comprehensible when viewed in light of the following facts. The mass of the right ventricle is less than that of the left.[30] In addition, the right ventricle contracts against a low-impedance, high-distensible, low-pressure pulmonary vascular bed.[31] Moreover, the right ventricle is more compliant than the left.[32] The extensive coronary collateral network, lower O_2 consumption, enhanced ability for O_2 extraction,[33] and right coronary perfusion in systole and diastole should also be taken into consideration.[34]

Another salient point is the complex shape of the right ventricle. In the 2DSTE assessment of RV function, the right ventricle is evaluated in only 1 section: apical 4-chamber view. In addition, there is a high degree of variability in the longitudinal deformation indices of the right ventricle. As 3-dimensional echocardiography can provide better anatomical delineation of the RV, it is a current method in RV echocardiography. Nonetheless, it will be a long time before its widespread use in routine clinical work. It seems that a more comprehensive evaluation of the RV with other modalities, including 3-dimensional speckle-tracking echocardiography or tagged cardiac magnetic resonance imaging, may be able to detect subtle myocardial dysfunction.

Study limitations

Small sample size and retrospective design were the primary present limitations. In addition, we used 2DSTE software, which was designed for the left ventricle.

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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. Crossref
2. Goel PK, Gupta SK, Agarwal A, Kapoor A. Slow coronary flow: a distinct angiographic subgroup in syndrome X. Angiology 2001;52:507–14. Crossref
3. Beltrame JF, Limaye SB, Horowitz JD. The coronary slow flow phenomenon--a new coronary microvascular disorder. Cardiology 2002;97:197–202. Crossref
4. Saya S, Hennebry TA, Lozano P, Lazzara R, Schechter E. Coronary slow flow phenomenon and risk for sudden cardiac death due to ventricular arrhythmias: a case report and review of literature. Clin Cardiol 2008;31:352–5. Crossref
5. Cakmak HA, Aslan S, Gul M, Kalkan AK, Ozturk D, Celik O, et al. Assessment of the relationship between a narrow fragmented QRS complex and coronary slow flow. Cardiol J 2015;22:428–36. Crossref
6. Yilmaz H, Gungor B, Kemaloglu T, Sayar N, Erer B, Yilmaz M, et al. The presence of fragmented QRS on 12-lead ECG in patients with coronary slow flow. Kardiol Pol 2014;72:14–9.
7. Yilmaz H, Demir I, Uyar Z. Clinical and coronary angiographic characteristics of patients with coronary slow flow. Acta Cardiol 2008;63:579–84. Crossref
8. Sezgin N, Barutcu I, Sezgin AT, Gullu H, Turkmen M, Esen AM, et al. Plasma nitric oxide level and its role in slow coronary flow phenomenon. Int Heart J 2005;46:373–82. Crossref
9. Pekdemir H, Polat G, Cin VG, Camsari A, Cicek D, Akkus MN, et al. Elevated plasma endothelin-1 levels in coronary sinus during rapid right atrial pacing in patients with slow coronary flow. Int J Cardiol 2004;97:35–41. Crossref
10. Ucgun T, Başar C, Memişoğulları R, Demirin H, Türker Y, Aslantaş Y. Serum visfatin and omentin levels in slow coronary flow. Rev Port Cardiol 2014;33:789–94. Crossref
11. 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. Crossref

12. Elsherbiny IA. Left ventricular function and exercise capacity in patients with slow coronary flow. Echocardiography 2012;29:158–64. Crossref
13. Nurkalem Z, Gorgulu S, Uslu N, Orhan AL, Alper AT, Erer B, et al. Longitudinal left ventricular systolic function is impaired in patients with coronary slow flow. Int J Cardiovasc Imaging 2009;25:25–32. Crossref
14. Zencir C, Cetin M, Güngör H, Karaman K, Akgüllü C, Eryılmaz U, et al. Evaluation of left ventricular systolic and diastolic functions in patients with coronary slow flow phenomenon. [Article in Turkish] Turk Kardiyol Dern Ars 2013;41:691–6. Crossref
15. Wang Y, Ma C, Zhang Y, Guan Z, Liu S, Li Y, Yang J. Assessment of left and right ventricular diastolic and systolic functions using two-dimensional speckle-tracking echocardiography in patients with coronary slow-flow phenomenon. PLoS One 2015;10:e0117979. Crossref
16. Altunkas F, Koc F, Ceyhan K, Celik A, Kadi H, Karayakali M, et al. The effect of slow coronary flow on right and left ventricular performance. Med Princ Pract 2014;23:34–9. Crossref
17. Balci MM, Arslan U, Kocaoğlu I, Kafes H, Balci KG, Kisacik HL. Right ventricular functions in patients with slow coronary flow. Acta Cardiol 2013;68:161–6.
18. 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. Crossref
19. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440–63. Crossref
20. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 2010;23:685–713; quiz 786–8. Crossref
21. Perk G, Tunick PA, Kronzon I. Non-Doppler two-dimensional strain imaging by echocardiography--from technical considerations to clinical applications. J Am Soc Echocardiogr 2007;20:234–43. Crossref
22. Dandel M, Lehmkuhl H, Knosalla C, Suramelashvili N, Hetzer R. Strain and strain rate imaging by echocardiography - basic concepts and clinical applicability. Curr Cardiol Rev 2009;5:133–48. Crossref
23. Blessberger H, Binder T. NON-invasive imaging: Two dimensional speckle tracking echocardiography: basic principles. Heart 2010;96:716–22. Crossref
24. Geyer H, Caracciolo G, Abe H, Wilansky S, Carerj S, Gentile F, et al. Assessment of myocardial mechanics using speckle tracking echocardiography: fundamentals and clinical applications. J Am Soc Echocardiogr 2010;23(4):351–69; quiz 453–5. Crossref
25. Lemarié J, Huttin O, Girerd N, Mandry D, Juillière Y, Moulin F, et al. Usefulness of Speckle-Tracking Imaging for Right Ventricular Assessment after Acute Myocardial Infarction: A Magnetic Resonance Imaging/Echocardiographic Comparison within the Relation between Aldosterone and Cardiac Remodeling after Myocardial Infarction Study. J Am Soc Echocardiogr 2015;28:818–27. Crossref
26. Huttin O, Lemarié J, Di Meglio M, Girerd N, Mandry D, Moulin F, et al. Assessment of right ventricular functional recovery after acute myocardial infarction by 2D speckle-tracking echocardiography. Int J Cardiovasc Imaging 2015;31:537–45.
27. Hutyra M, Skála T, Horák D, Köcher M, Tüdös Z, Zapletalová J, et al. Echocardiographic assessment of global longitudinal right ventricular function in patients with an acute inferior ST elevation myocardial infarction and proximal right coronary artery occlusion. Int J Cardiovasc Imaging 2015;31:497–507.
28. Rallidis LS, Makavos G, Nihoyannopoulos P. Right ventricular involvement in coronary artery disease: role of echocardiography for diagnosis and prognosis. J Am Soc Echocardiogr 2014;27:223–9. Crossref
29. Chen Z, Chen X, Li S, Huo X, Fu X, Dong X. Nicorandil improves myocardial function by regulating plasma nitric oxide and endothelin-1 in coronary slow flow. Coron Artery Dis 2015;26:114–20. Crossref
30. Lorenz CH, Walker ES, Morgan VL, Klein SS, Graham TP Jr. Normal human right and left ventricular mass, systolic function, and gender differences by cine magnetic resonance imaging. J Cardiovasc Magn Reson 1999;1:7–21. Crossref
31. Dell'Italia LJ. The right ventricle: anatomy, physiology, and clinical importance. Curr Probl Cardiol 1991;16:653–720.
32. Gaasch WH, Cole JS, Quinones MA, Alexander JK. Dynamic determinants of letf ventricular diastolic pressure-volume relations in man. Circulation 1975;51:317–23. Crossref
33. Haupt HM, Hutchins GM, Moore GW. Right ventricular infarction: role of the moderator band artery in determining infarct size. Circulation 1983;67:1268–72. Crossref
34. Kinch JW, Ryan TJ. Right ventricular infarction. N Engl J Med 1994;330:1211–7. Crossref

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