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Original Article

Assessment of Left Ventricular Functions by Two-Dimensional Speckle Tracking Echocardiography in Patients with Coronary Slow-Flow Phenomenon.

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ABSTRACT

Background: Coronary slow flow phenomenon (CSFP) is a medical challenge, especially regarding its diagnosis. The use of speckle tracking seems to provide a clue for diagnosis. However, its role is not well addressed.

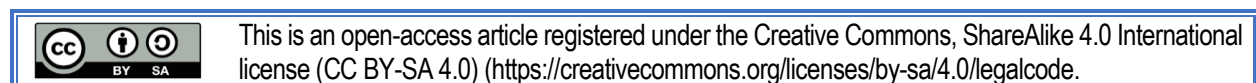
Aim of the work: To assess the value of the left ventricle's global longitudinal strain by 2D speckle tracking to detect subclinical left ventricular systolic dysfunction in patients with the slow coronary flow.

Patients and methods: This study was performed between May and September 2020 at the cardiology department, Al-Azhar University Hospital (New Damietta). Thirty patients are known to have slow coronary flow based on a previous coronary angiographic examination, and 30 healthy participants with the same demographic match (age and sex) as a control group were included. Patients were classified into two groups: Group (A): Included 30 patients with a coronary slow flow. Group (B): Included 30 normal healthy adult persons served as a control group. All were assessed clinically and by radiological investigations. In addition, speckle tracking echocardiography was done for all patients.

Results: In Doppler flow measures, E wave and E/A ratio showed significantly lower values in CSFP when compared with the control group ($p=0.02$, 0.003 respectively). According to tissue velocity imaging (TVI), there is a similarity between both groups without any significant difference. Speckle tracking measurements demonstrate significantly lower value in patients with CSFP than the control group ($p=0.003$).

Conclusion: In patients with CSF, LV systolic function detected during both conventional and tissue Doppler echocardiographic examinations was not affected, but 2D longitudinal strain demonstrated that LV systolic function was impaired. CSFP could impair LV diastolic function.

Keywords: Speckle tracking; Echocardiography; Left ventricle; Coronary Slow-Flow Phenomenon.



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INTRODUCTION

Coronary slow-flow phenomenon (CSFP) is an angiographic diagnosis characterized by a low rate of flow of contrast agent in the normal or near-normal epicardial coronary arteries. Many patients with CSFP may experience recurrent acute coronary syndromes ⁽¹⁾. CSFP is characterized by delayed opacification of coronary arteries during angiography. The frequency of CSFP is approximately 1% to 7% in patients undergoing coronary angiography. More than 80% of patients with CSFP often experience recurrent chest pain; almost 20% of whom require readmission following the same diagnosis ⁽²⁾.

CSFP mimics various clinical presentations, such as unstable angina, acute myocardial infarction, and ventricular tachycardia ⁽³⁻⁵⁾.

Although it has been well known to cardiologists for decades, the disease's etiology and pathophysiologic mechanisms have not been well understood. Whether the left ventricular (LV) and/or right ventricular (RV) functions are affected by CSFP, and to what extent, is still not precisely known. Using tissue Doppler imaging (TDI), several studies found that LV diastolic and systolic functions were impaired and that CSFP did not affect RV function ⁽⁶⁾.

Tissue Doppler signal has angle dependency and may be influenced by global heart motion, such as translation, torsion, and rotation ⁽⁷⁾. Two-D-STE is an emerging technology that measures strain and strain rate by tracking speckles in 2D grayscale echocardiographic images ⁽⁸⁾. It can measure myocardial motion in any direction irrespective of the direction of the beam and provides strain in all dimensions (longitudinal, radial, and circumferential). This objective, comprehensive, and noninvasive method can detect and assess myocardial performance ⁽⁹⁾.

Abnormalities of strain and strain rate can be found early in the development of many pathophysiologic states, thus providing a sensitive means for detecting myocardial dysfunction ⁽¹⁰⁾.

Global longitudinal strain (GLS) by two-dimensional (2D) speckle tracking echocardiography is an accurate measure of myocardial deformation. It is a superior predictor of outcomes to either the EF or wall motion score index and may become the optimal method for assessing global LV systolic function ⁽¹¹⁾.

AIM OF THE WORK

The study aims to assess the value of the left ventricle's global longitudinal strain by two-dimensional (2D) speckle tracking to detect subclinical left ventricular systolic

dysfunction in patients with a coronary slow flow.

PATIENTS AND METHODS

This study was performed between May and September 2020, at cardiology department, Al-Azhar University Hospital (New Damietta). Thirty patients known to have coronary slow flow based on previous coronary angiographic examination (group A), in addition to 30 healthy participants with the same demographic match (age and sex) as a control group (group B), were included in the study population.

Inclusion criteria: Patients with coronary slow flow with normal LV ejection fraction (EF).

Exclusion criteria: Evidence of prior myocardial infarction (assessed by history, electrocardiographic findings, and echocardiographic findings). Significant coronary artery stenosis more than or equal to 50% in a patient with CSF and stenosis more than 25% in control subjects. Acute coronary syndrome and previous percutaneous coronary intervention (PCI). Atrial fibrillation with heart rate >100. Poor echo window. Significant (more than mild) valvular stenosis and/or regurgitation. Previous heart surgery, congenital heart disease, and pericardial diseases. Cardiomyopathy or left ventricular EF less than 55%. Left bundle branch block or pacemaker implantation.

All patients signed informed consent to participate in the study, full history taking with an emphasis on age, gender, risk factors for CAD: Diabetes mellitus was defined as having a fasting blood glucose of 126mg/dl or greater on two occasions or a random plasma glucose \geq 200mg/dl with classic diabetes symptoms (increased urination, increased thirst and unexplained weight loss), or glycosylated hemoglobin > 6.5% (according to the American Diabetes Association), or the use of blood glucose-lowering medications (Insulin or oral drugs). Hypertension was defined as a systolic blood pressure \geq 140mmHg and/or diastolic blood pressure \geq 90mmHg or use of medication prescribed for hypertension. Current smoking was defined as having smoked a cigarette in the last 30 days. All were submitted to full clinical examination, and twelve lead ECG to exclude ischemia and/or serious arrhythmia.

Transthoracic echocardiography to assess the left ventricular function. With a 3.5 MHz transducer in a left lateral decubitus position during normal respiration. According to the American Society of Echocardiography recommendations, right and left heart images and measurements were acquired from standard views, and at least three consecutive cardiac cycles were recorded ⁽¹²⁾.

M-mode parameters were taken from parasternal long-axis view, LV end-diastolic dimension, interventricular

septal thickness, and posterior wall thickness were measured during LV end-diastole immediately before aortic valve opening. LV end-systolic dimension was measured during LV end-systole. LVEF was measured using the M-Mode method. Pulsed-wave Doppler was performed in the apical four-chamber view to obtain mitral inflow velocities to assess LV filling. Early diastolic mitral inflow velocity (E wave) and late diastolic mitral inflow velocity (A wave) were recorded. Pulsed wave TDI images were acquired by activating the TDI functions of the echocardiography unit. The tissue Doppler average signal of the septal side of the mitral annulus was acquired. The following measurements from the mitral annulus were made from the TDI recordings: mitral MV-Sa, MV-Ea, and MV-Aa. Dynamic 2D ultrasound images of three cardiac cycles from apical two-, three-, and four-chamber views were acquired using conventional ultrasound, with a frame rate of 57 to 72 frames per second, to measure strain. All images were obtained during breath-hold and stored in cine loop format from three or more consecutive beats. The Frame rate of images was between 60 and 80 frames/s.

Global longitudinal strain analysis (GLS): Digitally stored clips were analyzed offline using commercial imaging analysis software Q lab 10.4. For each of the three apical views, 3 points were identified on the endocardial surface: two on each side of the mitral valve and a third at the left ventricle's apex. The software automatically detected the endocardium at end-systole, tracked myocardial motion during the entire cardiac cycle, and created inverted U-shaped regions of interest (ROI) that encompassed basal, middle, and apical segments of 2 opposite LV walls. Tracking quality was assessed by the operator and scored by the software. The software automatically calculated the peak longitudinal strain for each individual segment in a 17-segment LV model, expressed as bull's eye, and calculated global longitudinal strain (GLS) by averaging local strains along the entire left ventricle. The software provided the strain curves for the 16 myocardial segments (excluding the apical cap).

Statistical Analysis: Data was presented as Mean and Standard deviation (SD) for quantitative parametric data and the median and interquartile ranges for quantitative nonparametric data. Frequency and percentage were used for presenting qualitative data. The suitable analysis was done according to the type of data obtained. Student T-test or Mann Whitney test was used to analyze quantitative data while the chi-square test and Fisher exact test was used to analyze qualitative data. P-value <0.05 was considered statistically significant.

RESULTS

Study population: The present study included (60) individuals divided into two groups, group (A) 30 patients with slow coronary flow (CSF) and group (B) 30 normal

adult healthy persons serve as a control group.

Demographic Characters: Group A included 16 males (53.3%), the mean age was (56.4±12.6) years, 14 (46.7%) were hypertensives, 3 patients (10%) were diabetics, 11 (36.7%) were smokers. In group- B, 19 people were males represent 63.3% of all control groups, the mean age was 55.2±12.2 years, 12 (40%) were hypertensives, 4 (13.3%) were diabetics, 6 (20%) were smokers. There were no significant differences between both groups regarding demographic data (Table 1)

Clinical data: Heart rate (HR) of group-A ranged between 65 and 90 bpm with mean ±SD= 75±14 bpm, SBP ranged between 110 and 150 with mean ±SD= 127±11.6mmHg, DBP ranged between 70 and 90 mmHg with mean ±SD =79.1±9.8mmHg. Otherwise, in group B the HR ranged between 70 and 90 bpm with mean ±SD= 78±9.2 bpm, SBP ranged between 115 and 145 with mean ±SD= 125±14.1 mmHg, DBP ranged between 65 and 90 mmHg with mean ±SD =77±9 mmHg; there was no statistically significant difference between both groups regarding clinical data (Table 2).

Echocardiographic parameters:

M-Mode parameters: In group A mean EF was 62.93± 8.84%, mean FS was 26.7±1.9%, mean LVEDD was 47.20±6.26 mm, mean LVESD was 29.47±7.1mm, mean intraventricular septum was 8.80±2.180mm, and mean Posterior wall thickness was 8.11±1.78mm. In the group-B, the mean EF was 65.26±5.96%, mean FS was 27.8 ± 1.6%, mean LVEDD was 47.20±6.26 mm, mean LVESD was 3.3±0.4mm, mean Intraventricular septum was 8.19±1.75mm, and mean posterior wall thickness was 7.94±1.60 mm; there was no statistically significant difference between both groups (Table 3).

Doppler flow measures:

In Group A mean velocity of E wave was 67±13cm/sec, the mean velocity of A wave 67±15cm/sec, the mean E/A ratio was 0.96±0.36, and the mean DT was 176.12±35.84ms. In group B, the mean velocity of E wave was 74±13 cm/sec, the mean velocity of A wave 65±16 cm/sec, mean E/A ratio was 1.22±0.35, and DT was 183±12ms. There was a statistically significant difference between both groups regarding early diastolic velocity and E/A ratio (p = 0.02, 0.003), respectively (table 3)

Tissue velocity imaging: As regard tissue velocity, imaging of septal MV annulus revealed that in group A mean velocity of MV-Sa was 6.76±1cm/sec, the mean velocity of MV-Ea was 7±2.94cm/sec, the mean velocity of MV-Aa 7.17±1.40cm/sec, and E/e` ratio 10±2.53. In group B mean velocity of MV-Sa was 6.98± 1.95 cm/sec, the mean velocity of MV-Ea was 7.94± 1.47cm/sec, the mean

velocity of MV-Aa 7.13 ± 1.17 cm/sec, and the E/e' ratio 10 ± 2.1 . There was no significant difference between both groups (Table 3)

Two D speckle tracking: LV-GLS was significantly of higher value in group B when compared to group A ($-19.32 \pm 2.20\%$ vs $-18.23 \pm 2.22\%$) $p = (0.03)$ (Table 3).

Table (1): Table (1): Demographic Characters of study groups

	Group A (n=30)		Group B (n=30)		P-value
Age	56.4±12.6		55.2±12.2		0.43
Sex	Male	Female	Male	Female	0.13
	16 (53.3%)	14 (46.7%)	19 (63.3%)	11 (36.7%)	
Hypertension	14 (46.7%)		12 (40%)		0.73
DM	3 (10%)		4 (10.3%)		0.31
Smoking History	11 (36.7%)		6 (20%)		0.1

Table (2): Clinical data among study groups

	Group A	Group B	P-value
Heart Rate	75±14	78±9.2	0.45
SBP mm Hg	127±11.6	125±14.1	0.36
DBP mm Hg	79.1±9.8	77±9	0.24

Table (3): Echocardiographic parameters:

	Group A	Group B	P-value
EF (%)	62.93±8.84	65.26±5.96	0.61
LVEDD (mm)	47.20±6.26	47.27±4.82	0.92
LVESD (mm)	29.47±7.1	28.12±4.34	0.68
Intraventricular septum (mm)	8.80±2.180	8.19±1.75	0.73
Posterior wall thickness (mm)	8.11±1.78	7.94±1.60	0.62
FS (%)	26.7 ± 1.9	27.8 ± 1.6	0.85
E wave (cm/sec)	67±13	74±13	0.02*
A wave (cm/sec)	67±15	65±16	0.62
E/A ratio	0.96±0.36	1.22±0.35	0.003*
MV-EDT (ms)	176.12±35.84	183±12	0.26
MV-Sa (cm/sec)	6.76±1	6.98±1.95	0.59
MV-Ea (cm/sec)	7±2.94	7.94±1.47	0.34
MV-Aa (cm/sec)	7.17±1.40	7.13±1.17	0.28
E/e' ratio	10±2.53	10±2.1	0.72
LV-GLS (%)	-18.23±2.22	-19.32±2.20	0.03*

EF; Ejection Fraction, FS; Fractional Shortening, LVEDD: Left Ventricular end-diastolic Diameter and LVESD for Left Ventricular end-systolic Diameter. E wave; Early diastolic velocity, A wave; late diastolic velocity and DT for Deceleration time, *; significant. MV-Sa; systolic annular velocity, MV-Ea; early diastolic annular velocity, MV-Aa; late diastolic annular velocity, e': early diastolic annular velocity, LV-GLS; left ventricular global longitudinal strain.

DISCUSSION

Coronary slow-flow phenomenon (CSFP) is an angiographic diagnosis characterized by a low rate of flow of contrast agent in the normal or near-normal epicardial coronary arteries. Many patients with CSFP may

experience recurrent acute coronary syndromes (1). The pathophysiologic mechanisms of the disease have not been well understood. Whether the left ventricular (LV) and/or right ventricular (RV) functions are affected by CSFP, and to what extent, is still not precisely known. Using tissue Doppler imaging (TDI), several studies found that LV

diastolic and systolic functions were impaired and that CSFP did not affect RV function⁽⁶⁾. Tissue Doppler signal has angle dependency and may be influenced by global heart motion, such as translation, torsion, and rotation⁽¹³⁾.

The main objective of this study was to evaluate LV functions in patients with CSFP. The main finding of our study was CSFP affected LV functions. In our work, there was no statistically significant difference between both groups regarding EF, FS, A wave, and DT. The E wave and E/A ratio showed lower values in the CSFP group than the control group with statistically significant differences ($p=0.02, 0.003$, respectively). In **Zencir *et al.***⁽¹⁴⁾ study, 60 patients with CSFP were compared with 30 normal individuals, left ventricular systolic and diastolic functions were assessed by conventional echo and found that there were no statistically significant differences between both groups regarding EF, E wave, A wave, E/A ratio, and DT ($p=0.67, 0.05, 0.63, 0.19, 0.06$ respectively). We found disagreement with **Zencir *et al.***⁽¹⁴⁾ study regarding E wave and E/A ratio in comparison to our work. This disagreement may be due to the difference of demographic characters between our work and **Zencir *et al.*** study (e.g.: mean age of CSFP group is 56 ± 12.6 vs. 52.4 ± 12.2 respectively).

In **Li Y, *et al.***⁽¹⁵⁾ study, 22 patients with CSFP were compared with 22 patients with normal coronary angiography, left ventricular systolic and diastolic functions were assessed by conventional echo and found that there were statistically significant differences between both groups regarding E wave and E/A ratio ($p=0.01, 0.001$ respectively), while there were no significant differences between both groups regarding EF and A wave. That comes in line with our work. **Wang *et al.***⁽⁶⁾ studied 63 patients with CSFP and 45 patients as a control group, found that there were no significant differences between both groups regarding EF ($p=0.10$), A wave ($p=0.17$), and DT ($p=0.23$). Simultaneously, the E wave and E/A ratio showed lower values in the CSFP group than the control group with statistically significant differences ($p=0.02, 0.003$ respectively). This results in agreement with our work.

Narimani *et al.*⁽¹⁶⁾ studied 36 patients with CSFP and 36 patients as a control group, found that there were no significant differences between both groups regarding EF ($p=0.99$), A wave ($p=0.17$), and DT ($p=0.23$).

Our work's tissue velocity imaging parameters showed that there were no statistically significant differences between groups regarding MV-Sa, MV-Ea, MV-Aa and E/e'. That comes in line with many studies and work of **Zencir *et al.***⁽¹⁴⁾.

In **Baykan *et al.***⁽¹⁷⁾, 50 patients with CSF were compared with 28 patients with normal coronary angiography, and they found that MV-Ea and MV-Sa were

significantly lower in the CSF group than the control group ($p=0.002, 0.005$), respectively, and this result disagrees with our work. While there were no significant differences between groups regarding MV-Aa ($p=0.4$). This disagreement of the TDI parameter may be due to the Tissue Doppler signal having angle dependency and may be influenced by global heart motion, such as translation, torsion, and rotation⁽¹³⁾.

In **Zencir *et al.***⁽¹⁴⁾ study regarding TVI, they found no significant differences between both groups regarding MV-Sa, MV-Ea, and MV-Aa ($p=0.52, 0.32, 0.29$ respectively). This result shows a similarity to our finding. **Wang *et al.***⁽⁶⁾ demonstrated that there were no statistically significant differences between both groups regarding MV-Sa, MV-Ea, and MV-Aa ($p=0.69, 0.24, 0.88$ respectively), and this result is in line with our study.

In **Narimani *et al.***⁽¹⁶⁾ study regarding TVI, they found that there were no significant differences between either group regarding MV-Sa ($p=0.06$), MV-Ea ($p=0.16$), and MV-Aa ($p=0.16$). This results in agreement with our work.

According to speckle tracking measurements, LV-GLS showed a significantly lower value in patients with CSFP than the control group ($-18.23 \pm 2.22\%$, $-19.32 \pm 2.20\%$, respectively) ($p=0.003$). These results showed agreement with **Wang *et al.***⁽⁶⁾. In their study in 2015, using 2D-STE to assess LV systolic function in the CSF patients group compared with the control group found that peak systolic longitudinal strain (LS) was lower in patients with CSFP group than in the control group ($P=0.004$).

Narimani *et al.*⁽¹⁶⁾ in 2015 studied the effect of coronary slow flow on the longitudinal left ventricular function using 2D-STE. In disagreement with our study, they found no statistically significant difference between both groups regarding LV longitudinal strain ($p=0.51$). This difference between both studies could be explained by the different number of patients, study designs, and differences in the patients' demographic characters. **Nurkalem *et al.***⁽¹⁸⁾ in 2008 studied longitudinal LV systolic function in 35 patients with coronary slow flow and 21 patients with normal coronary angiography using conventional echo and STE. They found that There was a significant difference in peak systolic strain between both groups ($P=0.0001$). This result showed agreement with our work.

Conclusion: In patients with CSF, LV systolic function detected during both conventional and tissue Doppler echocardiographic examinations were not affected, but 2D longitudinal strain demonstrated that LV systolic function was impaired. CSFP could impair LV diastolic function.

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