Available online at www.medicinescience.org

ORIGINAL RESEARCH

Medicine Science International Medical Journal

Medicine Science 2019; ( ):

The evaluation of the effect of coronary collateral formation on left ventricular function by using strain echocardiography

[1]Nuray Kahraman Ay ORCID:0000-0003-3355-0925
[2]Cihan Orem ORCID:0000-0001-6591-7336

[1]Bezmialem Vakıf University Faculty of Medicine, Department of Cardiology, Istanbul, Turkey
[2]Karadeniz Technical University Faculty of Medicine, Department of Cardiology, Trabzon, Turkey

Received 01 December 2018; Accepted 12 Febuary 2019
Available online 22.02.2019 with doi:10.5455/medscience.2018.07.9000

Abstract

The aim of this study is to compare the strain echocardiography method, that we think, evaluate ventricular functions more accurately, and the other echocardiographic methods in patients with or without angiographically developed collaterals. Non acute coronary syndrome 45 patients with determined occlusion of the left anterior descending artery (LAD) over 90% were included in this study. Collateral vessel devellopping according to Rentrop classification, grade 0-1 patients were determined as Group I (n=24) , grade 2-3 patients determined as Group II (n=21). PW doppler, tissue doppler and strain echocardiography was applied. The peak strain levels in basal anteroseptal, midseptal and apical septal segments were significantly lower in Group I (p=0.04, p=0.01 and p=0.02 respectively). Also €peak I value was significantly lower in Group I (p=0.02). In Group I, postsystolic shortening index (PSSI) was found to be positively correlated with left ventricle end diastolic volume (LVEDV) (r=0.4, p=0.05), left ventricle end systolic volume (LVESV) (r=0.47, p=0.01) and wall motion score index(WMSI) (r=0.64, p=0.001) and negatively correlated with ejection fraction(EF) (r= -0.57, p=0.03) and mitral septal annulus systolic velocity (MSA S) (r= -0.49, p=0.01). In Group II, PSSI was positively correlated with left ventricle end diastolic diamater (LVESD) (r=0.5, p=0.01) and LVESV (r=0.5, p=0.01) and negatively correlated with MSA S (r= 0.45, p=0.03). In patients who have better developed collaterals have better left ventricle functions that were observed both by strain echocardiography and conventional echocardiographic methods.

Keywords: Left ventricular function, collateral circulation, echocardiography

Introduction

Collateral vessel development in coronary artery disease (CAD) has been known for a long time. After total or subtotal occlusion of a coronary artery, the perfusion of the ischemic myocardium may be provided by the formation of collateral vessels connecting the epicardial vessels [1,2].

In coronary artery disease contraction decreases in the ischemic myocardium and completely disappears in the necrotic region. These regions with decreased contraction function are observed as regional wall motion abnormalities in echocardiography. The accurate assessment of regional wall motion is crucial in the diagnosis and treatment of CAD. Myocardial functions are determined by the visual evaluation of regional wall motion and wall thickening as two-dimensional images in conventional echocardiographic methods. Measurement and quantitative expression of the finding are the main conditions of accurate evaluation. Numerical expression of the measurements such as velocity, motion, deformation and deformation velocity in parametric imaging techniques aim at alleviating the limitations of segmentary wall motion evaluation via conventional two-dimensional (2D) images which originate from operator dependence, subjectivity and semi-quantitativeness [3]. Strain echocardiography is essentially a tissue doppler based method and measures tissue deformation (length changes through time, as percentage). Regional wall motion can be accurately and reproducibly determined quantitatively with strain imaging method. Moreover, it is possible to assess myocardial motion in three dimensions and examine the tissue motion in radial, longitudinal and circumferential axes. Velocities used in tissue doppler are affected by passive movement of the segment, strain and strain rate are not affected by passive movements and indicate active contraction.

The functional aspect of the collaterals is controversial despite theoretical knowledge. Whereas there are numerous studies reporting the beneficial effects of coronary collateral circulation on LV functions, myocardial viability and functional recovery, there is also literature suggesting otherwise [4-7]. This inconsistency may have resulted from methodological problems. One

***Coresponding Author:** Nuray Kahraman Ay, Bezmialem Vakıf University Faculty of Medicine, Department of Cardiology, Istanbul, Turkey
E-mail: drnuraykahraman@hotmail.com

methodological issue is the echocardiographic method used to evaluate ventricular function. Thus, this study aims at comparing the strain echocardiography method, which we think assesses ventricular functions more accurately, to other echocardiographic methods in cases with and without angiographically shown collateral circulation.

Material and Methods

Subjects and study design

The study is designed as a cross-sectional empirical and controlled study. 45 patients undergoing diagnostic coronary angiography in Karadeniz Technical University Hospital between January 2007 and September 2009 with determined occlusion of the left anterior descending artery (LAD) over 90% were included in this study. Mean age of patients was 63±10,8 and nine of the patients were female and 36 male. Ethics committee approval was obtained. Selective coronary angiography with Siemens Axiom Artis coronary angioraphy device, using judkins or sonos method was performed after local anaesthesia. Coronary arteries were visualized in right or left oblique positions using cranial or caudal angulations. Patients with 90% or more occlusion of the LAD together with normal or at most noncritically occluded right coronary artery (RCA) and circumflex artery (CA) were included in the study. Collateral circulation was classified semi-quantitatively between 0-3 according to Rentrop classification [8]. In Rentrop classification the grading was done as follows: Grade 0: no collateral circulation, Grade 1: Side branches of the recipient arteries are filled without epicardial artery visualization, Grade 2: Epicardial segments are partially filled via collateral vessels, Grade 3: epicardial segment of the recipient artery is completely filled via collateral vessels. The assessment was done under the supervision of two coronary angiography specialists. The patients were divided into two groups according to collateral development. The group in which collateral vessel development was grade 0 and 1 was designated as Group I (n=24) and the group with grade 2 and 3 collateral vessel development was designated as Group II (n=21).

Patients with a history of unstable angina, myocardial infarction(MI) in last three weeks, coronary artery bypass graft operation or coronary angioplasty and patients with moderate to severe valve disease, atrial fibrillation, hypertrophic or restrictive cardiomyopathy, congenital heart disease, considerable endocrine, renal or hepatic disease and suboptimal echocardiographic image quality were excluded from the study. Informed consent was obtained from all patients and the study was approved by the local Ethical Committeee. The demographic characteristics of the patients are presented in Table 1.

Table 1. Basic characteristic of the patients

Variables	Group I (n= 24)	Group II (n=21)	P
Age, Mean±SD, years	64.08 ± 11.67	62.05 ± 10.9	NS
Sex, n			
Female/Male	5/19	4/17	NS
Body Mass Index, Mean±SD	26 ± 4.2	28 ± 6.5	NS
Heart rate, Mean±SD	66.88 ± 14.18	68.71 ± 13.18	NS
Systolic blood pressure	120 ± 20.6	125 ±19.6	NS
Diastolic blood pressure	73 ± 11.9	77.7 ± 12.6	NS
Hypertension, n (%)	1 (45.8)	5 (71.4)	NS
Hyperlipidemia, n (%)	10 (41.7)	4 (19)	NS
Smoking, n (%)	5(20.8)	3 (14.3)	NS
Diabetes, n (%)	6(25)	0	0.02*
Stabile angina pectoris, n (%)	18(75)	18 (85.7)	NS
Medical treatment			NS
Beta blocker, n (%)	13 (54.2)	14 (66.7)	NS
ACE inhibitor, n (%)	12 (50)	15 (71.4)	NS
Nitrate, n (%)	5 (20.8)	10 (47.6)	NS
Aspirin, n (%)	16 (66.7)	18 (85.7)	NS
Statin, n (%)	11 (45.8)	9 (42.9)	0.003
MI time, months	18	29	**

Values are given as mean±SD and number (percent). NS: Not significant, ACE: Angiotensin converting enzyme, MI: Myocardial infarction, * p<0.05, ** p<0.01

Echocardiographic evaluation

Echocardiographic examination was done in left lateral position with Vivid 7 Digital ultrasound device (Vingmed Ultrasoung, GE) using 2.5-3.5 MHz transducer from parasternal long axis and 2 and 4 cavity images. Echocardiographic measurements were based on the criteria recommended by American Echocardiography Association [9]. Endocardial borders were drawn in apical 4 cavity views from the end of diastole and systole and end systolic and end diastolic volumes and ejection fraction(EF) were calculated using modified simpson method. The segments showing the LAD area were evaluated in parasternal long axis and 2 and 4 cavity views. The areas perfused by LAD were separated into 9 segments as: basal anteroseptal and mid anteroseptal from parasternal long axis, midseptum, apical septum and apical anterolateral from apical 4 cavity, basal anterior, mid anterior, apical anterior and apical

inferior from apical 2 cavity. Wall motion score index was calculated by dividing the total motion scores of the 9 segments showing the LAD area by 9 (Table 2).

Table 2. Scoring of the left anterior descending artery segments according to regional wall motion

Standard score		Optional score
	0	Hyperdynamic
Normal	1	
	1,5	Mildly hypokinetic
Hypokinetic	2	
	2,5	Severely hypokinetic
Akinetic	3	
Dyskinetic	4	
Aneursym	5	
	6	Akinetic in presence of scar
	7	Dyskinetic in presence of scar

Strain echocardiography

Recording was done while the 2D gray scale view was at 150-200/second frame rate. The recording was done at the end of expirium whenever possible in order to prevent the records from being affected by global cardiac motion. Sample volumes were placed inside the myocard in order to minimize the angle between doppler flow and longitudinal shortening direction. The interval between aortic valve opening and closure was determined, from with pulse wave(PW), left ventricle(LV) outflow tract flow and was recorded. Regional functions were assessed in each segment showing LAD area with strain echocardiography program [10].

Peak strain ($€_{peak}$) value was measured in each segment (Figure 1). In segments in which $€_{peak}$ was observed after aortic valve closure (AVC) end systolic strain ($€_{es}$), postsystolic shortening index (PSSI) and time between AVC and $€_{peak}$ were calculated (Figure 2). Significant postsystolic shortening was accepted as $€_{peak}$: the biggest strain value in the RR interval, $€_{es}$: the strain value recorded during AVC, PSSI: $€_{peak}$ -$€_{es}$ /$€_{peak}$, $T_€$: time between AVC and $€_{peak}$, $€_{peak}$< -% 15, PSSI > 0,25 and $T_€$ > 100 ms. These patients were designated as postsystolic shortening index (PSSI) (+).$€_{peak}$ values from 9 segments were added together and strain index ($€_{peak\ I}$) was calculated by averaging the 9 values. Strain measurements were recorded as to include at least 3 cardiac cycles. Values obtained during 3 consecutive sinus beats were recorded for each parameter and the average values were used in statistical analysis. Strain echocardiography findings were compared to conventional echocardiographic methods (EF, left ventricle volumes, wall motion score index, diastolic function parameters etc.) and Doppler parameters.

Statistical analysis

Continous variables were given as mean ± standard deviation. Kolmogorov Smirnow test was used to determine whether the variables conformed to normal distribution. Chi-square test was used in the analysis of categorical variables. In Groups I and II unpaired t test was used in the comparison of continuous varibles conforming to normal distribution and Mann Whitney test in variables not conforming to normal distribution. Correlations between parameters were determined using Pearson or Spearman tests. P<0.05 was accepted as statistically significant. All statistical analyses were made in SPSS 10 software.

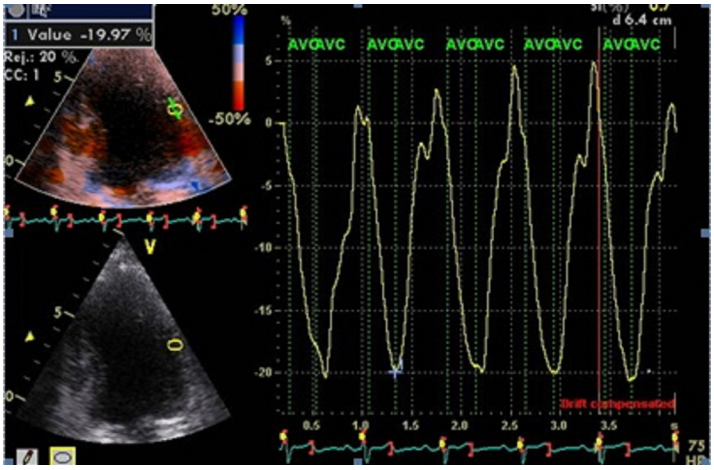

Figure 1. Calculation of the peak systolic strain (€peak) (from a segment without postsystolic shortening)

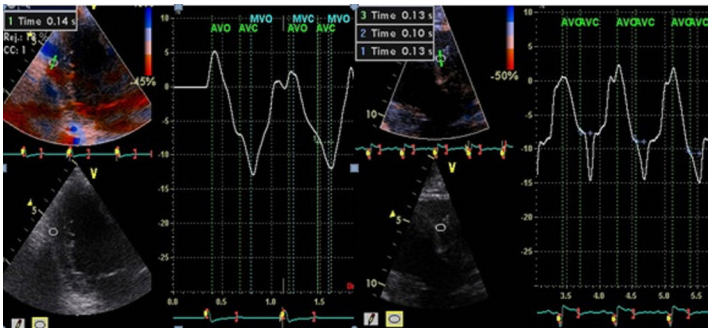

Figure 2. Calculation of the postsystolic shortening index (Endsystolic strain ($€_{es}$), postsystolic shortening index (PSSI) and time between aortic valve closure (AVC) and $€_{peak}$ ($T_€$) were calculated in the segments in which peak systolic strain corresponding to AVC was observed.)

Results

Demographic characteristics

There was no statistically significant difference between the groups regarding age, sex, body mass index, heart rate, blood pressure levels, hypertension, hyperlipidemia, presence of stable angina, smoking and medical treatment. Whereas 6 patients with diabetes mellitus were present in Group I, there were no diabetic patients in Group II (p=0,02) (Table 1). Patients in Group II had myocardial infarction previously (29 months versus 18 months, p= 0.003) (Table 1).

Echocardiographic findings

The evaluation of the echocardiographic findings of the groups is given in Table 3. Left ventricle end diastolic diamater (LVESD) was observed to be significantly lower (p:0,04) and EF to be significantly better (p:0,03) in Group II in which collateral development was good. Mitral lateral annulus deceleration time(MLA DT) (p=0,004) and Mitral septal annulus deceleration time(MSA DT) (p=0,03) were significantly longer in Group I. Wall motion score index(WMSI) was higher in Group I patients with poor collateral development (2,1±0,5 versus 1,7±0,3; p= 0,005). Peak strain levels were lower in almost all segments in Group I and were found to be significantly lower in basal anteroseptal (p=0,04), midseptal (p=0,01) and apical septal

(p=0,02) segments. €peak I value was significantly lower in Group I (p=0,02).

Table 3. Echocardiographic characteristics of the patients

	Group I (n=24)	Group II (n=21)	P
LA, mean±SD, cm	3.6 ± 0.6	3.7 ± 0.4	NS
IVS, mean±SD, cm	1.1 ± 0.1	1.2 ± 0.1	NS
LVEDD, mean±SD, cm	5 ± 0.6	4.7 ± 0.5	NS
LVESD, mean±SD, cm	3.4 ± 0.7	2.9 ± 0.7	0.04
LVEDV, mean±SD, ml	99.8 ± 42.2	87.2 ± 25.6	NS
LVESV, mean±SD, ml	54.8 ± 30.0	42.8 ± 19.1	NS
EF, mean±SD, %	45.3 ± 8.7	51.4 ± 9.5	0.03
M E/A, mean±SD	0.9 ± 0.4	0.8 ± 0.3	NS
M EDT	215.8 ± 53.9	246.8 ± 58.3	NS
M IVRT, mean±SD	109 ± 26.0	110.9 ± 26	NS
LV MPI, mean±SD	109.5 ± 26.3	111.1 ± 26.1	NS
MLA E/A, mean±SD	0.7 ± 0.3	0.8 ± 0.4	NS
MLA DT, mean±SD	150.4 ± 52.3	110.4 ± 31.1	0.004
MLA IVRT, mean±SD	94.9 ± 24.6	94.1 ± 30.8	NS
MLA MPI, mean±SD	95.3 ± 24.6	94.4 ± 30.8	NS
MSA E/A, mean±SD	0.6 ± 0.2	0.7 ± 0.2	NS
MSA DT, mean±SD	153 ± 43.0	126.9 ± 33.8	0.03
MSA IVRT, mean±SD	111.0 ± 35.4	94.6 ± 29.5	NS
MSA MPI, mean±SD	114.4 ± 35.4	95.0 ± 29.3	NS
LAD WMSI, mean±SD	2.1 ± 0.5	1.7 ± 0.3	0.005
Basal anteroseptal $€_{peak}$ (%)	-18.67 ± 4.79	-21.11 ± 2.51	0.04
Basal anterior $€_{peak}$ (%)	-16.78 ± 4.1	-16.75 ± 4.1	NS
Mid anteroseptal $€_{peak}$ (%)	-15.63 ± 5.7	-16.73 ± 4.8	NS
Mid anterior $€_{peak}$ (%)	-14.07 ± 4.3	-13.75 ± 0.3	NS
Mid septal $€_{peak}$ (%)	-16.39 ± 4.7	-19.53 ± 3.7	0.01
Apical septal $€_{peak}$ (%)	-11.84 ± 3.6	-14.1 ± 2.9	0.02
Apical anterior $€_{peak}$ (%)	-6.98 ± 6.6	-9.01 ± 3.1	NS
Apical lateral $€_{peak}$ (%)	-9.09 ± 6.12	-10.08 ± 3.0	NS
Apical inferior $€_{peak}$ (%)	-11.11 ± 3.9	-13.44 ± 4.6	0.07
$€_{peak}$ I	13.4 ± 2.9	14.9 ± 1.3	0.02

NS; Not significant, LA: Left atrium, IVS; Interventricular septum, LVEDD; Left ventricle end diastolic diameter, LVESD: Left ventricle end systolic diameter, LVEDV; Left ventricle end diastolic volume, LVESV: Left ventricle end systolic volume, EF; Ejection fraction, M EDT; Mitral E wave decelaration time, IVRT; Isovolumetric relaxation time, MPI; Miyocardial performance index, MSA; Mitral septal annulus, MLA; Mitral lateral annulus, DT; Deceleration time, LAD; Left anterior descending artery, WMSI; Wall motion score index, $€_{peak}$: The biggest strain value in the RR interval

When the presence of postsystolic shortening(PSS) was compared between two groups, postsystolic shortening was found to be present in a significantly higher number of patients in almost all segments (Table 4). PSS was present in a significantly higher number of patients in the group with poor collateral development compared to the group with good collateral development in midseptal (20,8% in Group I versus 0% in Group II; p=0,05), apical septal (70,8% in Group I versus 38,1% in Group II; p=0,04) and apical inferior (66,7% in Group I versus 33.3% in Group II; p=0,04) segments (Table 4).

The correlation of €peak I with other echocardiographic parameters was checked in both groups. In Group I, postsystolic shortening index(PSSI) was found to be positively correlated with left ventricle end diastolic volume (LVEDV) (r=0.4, p=0.05), left ventricle end systolic volüme(LVESV) (r=0.47, p=0.01) and WMSI (r=0.64, p=0.001) and negatively correlated with EF (r= -0.57, p=0.03) and mitral septal annulus systolic velocity (MSA S) (r= -0.49, p=0.01). In Group II, PSSI was positively correlated with left ventricle end systolic diamater(LVESD) (r=0.5, p=0.01) and LVESV (r=0.5, p=0.01) and negatively correlated with MSA S (r= -0.45, p=0.03)(Table 5).

Table 4. The association between collateral development and postsystolic shortening

Variables	Group I (n=24)	Group II (n=21)	P
Basal anteroseptal, n (%) PSS (+)	3 (12.5)	0	NS
Basal anterior PSS (+)	2 (8.3)	4 (19)	NS
Mid anteroseptal, n (%) PSS (+)	7 (29.2)	2 (9.5)	NS
Mid anterior, n (%) PSS (+)	8 (33.3)	4 (19)	NS
Mid septal, n (%) PSS (+)	5 (20.8)	0	0.05
Apical septal, n (%) PSS (+)	17 (70.8)	8 (38.1)	0,04
Apical anterior, n (%) PSS (+)	12 (50)	7 (33.3)	NS
Apical lateral, n (%) PSS (+)	7 (29.2)	8 (38.1)	NS
Apical inferior, n (%) PSS (+)	16 (66.7)	7 (33.3)	0.04

Variables are given as number (percent). PSS; Postsystolic shortening

Table 5. Correlation between echocardiographic parameters

			LVEDV	LVESV	WMSI	EF	MSA S
Group I	PSSI						
		p	0.05	0.01	0.001	0.03	0.01
		r	0.40	0.47	0.64	-0.57	-0.49
Group II	PSSI						
		p	0.01	0.01			0.03
		r	0.50	0.50			-0.45

PSSI ; postsystolic shortening index, LVEDV; Left ventricle end diastolic volume, LVESV: Left ventricle end systolic volume, WMSI: Wall motion score index, EF: Ejection fraction, MSA S; Mitral septal annulus.

Discussion

Angiographically visible collateral vessels are believed to preserve myorardial function during rest. Studies indicate that collateral circulation decreases myocardial ischemia, is the sole source of perfusion of the distal myocardial bed in patients who have a MI, reduces the infarct area, beneficially increases LV functions, decreases ventricular aneurysm formation and most importantly, increases survival [4, 11]. Weisman et al. [12] suggested that the presence of collaterals in CAD preserve LV functions. There are studies indicating that ischemia is decreased during percutaneous coronary angioplasty in cases in which there are collaterals in the concerned vessel [13, 14]. Although there are studies reporting beneficial effects of coronary collateral circulation on myocardial viability and functional recovery, studies suggesting contradictory findings are also present [4-7]. Many researchers suggested that the presence of collaterals do not have a protective effect on LV functions [15-19]. The inconsistency among studies may be a result of methodological problems (nonuniform patient population, multiple vessel disease, the method used to assess collaterals).

Single vessel disease was chosen this study in order to overcome these problems and make an accurate evaluation of the effects of collaterals on LV functions. There was no statistically significant difference between the two groups regarding the risk factors except diabetes frequency. All the cases were chosen among patients who had previous myocardial infarctions because LV functions would be different in MI and non-MI patients disregarding the presence or absence of collateral circulation.

Another factor which may cause negative results in previous studies might be the inadequacy in detecting collaterals. Angiographically visible collateral comprise only a small part of the total collateral network. It is not possible to detect intramural collaterals angiographically [19]. For collaterals to be angiographically visible they should exceed a diameter of 100 mm. Smaller diameter collaterals can not be observed angiographically [19]. Rentrop classification was used in this study. In recent studies regarding collateral evaluation, new measurement parameters were developed, which correlate well with Rentrop classification but include more quantitative data and give information about myocardial perfusion.

In this study LVESD and WMSI were observed to be lower, EF to be higher and DT in the septal annulus to be shorter in patients with good collateral development. In patients with good collateral development segmentary and global functions were found to be better in conventional and tissue Doppler echocardiography.

Regional wall motion abnormalities evaluated with echocardiography in CAD are usually assessed visually and are defined as normal, hypokinetic, akinetic, dyskinetic and aneurysmatic [10]. This is a rather crude method of decision. Visual assessment has myriad limitations. The ideal method of distinguishing the ischemic tissue is to use methods which can directly give quantitative values about the ischemic region. The classical and earliest method which can give quantitative information in the evaluation of regional contraction is Doppler echocardiography. It is possible to evaluate the regional and global functions of the ventricles and demonstrate the ischemic region more easily and sharply with 'Pulsed' Doppler and colour Doppler methods [10]. Although the use of velocities obtained from tissue Doppler give valuable information in the evaluation of ischemic response, in some cases velocity values may be normal in severely ischemic or infarcted segments. Normally contracting segments may affect the velocities of the damaged segments. During the assessment of tissue velocities with tissue Doppler, global heart movement, cardiac rotation and contraction in neighbouring tissues frequently affect the velocity values. 'Strain' and 'strain rate' methods are helpful in overcoming such limitations. That is why in this study strain echocardiography was used to evaluate the effects of collateral development on LV functions. In studies, PSSI>0,25 is accepted as significant for ischemia [20]. In this study, the presence of PSS was accepted as pathological in cases with PSSI>0,25 and used these values for statistical analysis. The results suggest that there may be more ischemia in patients with poor collateral development.

Peak strain value and peak strain index were higher and postsystolic shortening frequency was lower in the group with good collateral development. It was observed that peak strain index was correlated with many other echocardiographic function parameters especially in patients with poor collateral development which were expected to have poor LV functions. Kukulski et al. [20], in their study evaluating the acute ischemic changes of 73 patients with strain echocardiography during coronary angioplasty, compared the patients with grade 2-3 collateral development (7 patients) with grade 0 (9 patients) and grade 1 (29 patients) as a separate subgroup analysis. They found the peak strain value to be higher, PSS and PSSI to be lower in the well collateralized group and concluded that collaterals have beneficial effects on myocardial functions. The sample of the present study differs in that it is comprised of chronically ischemic patient.

Limitations

Studies indicate that systolic thickening/shortening decreases and postsystolic thickening/shortening increases as ischemia increases [21,22]. In a long follow-up study PSS was observed in 60% of infarcted segments, 29% of peri-infarct border segments and 5% of normal segments [23]. That is to say that PSS presence does not have the necessary sensitivity and specificity to determine infarcted segments. Ischemic infarct border segments can exhibit PSS. Therefore SR and strain evaluation should be made in conjunction to distinguish between infarcted and ischemic regions. The sample of the present study was comprised of patients with MI and strain echocardiography was evaluated singly. Thus the inability of exactly distinguishing between ischemia and infarct and the absence of stress tests which may support the findings are important limitations of this study. Echocardiographic parameters evaluated simultaneously by two observers and decision was made following consensus , but anyways intra and inter observer variabilities probably have been for the echocardiographic parameters.

Conclusion

The findings of the present study suggest that grade 2-3 collateral circulation, according to Rentrop classification has demonstrable beneficial effects on LV functions and together with a smaller ischemic area in both conventional and tissue Doppler and strain echocardiography methods in CAD patients in the chronic period.

Competing interests
The authors declare that they have no competing interest.

Financial Disclosure
The financial support for this study was provided by the researchers themselves.

Ethical approval
Study protocol was approved by Karadeniz Technical University ethics committee.

References

1. Helisch A, Schaper W. Arteriogenesis: the development and growth of collateral arteries. Microcirculation 2003;10:83-97.

2. Wolf C, Cai WJ, Vosschulte R, et al: Vasculer remodelling and altered protein expression during growth of coronary collateral arteries. J Mol Cell Cardiol. 1998;30:2291-305.

3. Dagdeviren B. Strain imaging. Clinical Echocardiography and other imaging methods. Edition. Erol O, Ozkan M. I. baskı. Ankara. MN Medical & Nobel 2007. p.71-92.

4. Vanoverschelde JL, Wijns W, Depré C, et al. Mechanism of chronic regional postischemic dysfunction in humans: new insights from the study of noninfarcted collateral-dependent myocardium. Circulation. 1993;87:1513-23.

5. Matsuda Y, Tananamito Y, Kobayashi Y, Hayashi K, Masuda T, Sakata K. Left ventricular motion after bypass operation for coronary artery disease with collaterals. Ann Thorac Surg. 1994;58:795-8.

6. Rentrop KP, Thornton JC, Feit F, Van Buskirk M. Determinants and protective potential of coronary arterial collaterals as assessed by an angioplasty model. Am J Cardiol. 1988;61:677-84.

7. Werner GS, Richartz BM, Gastmann O, Ferrari M, Figulla HR. Immediate changes of collateral function after successful recanalization of chronic total coronary occlusions. Circulation. 2000;102:2959-65.

8. Rentrop KP, Cohen M, Blanke H, Phillips RA. Changes in collateral channel filling immediately after coronary artery occlusion by an angioplasty balon in human subjects. J Am Coll Cardiol. 1985;5:587-92.

9. Schiller NB, Shah PM, Crawford M, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American society of echocardiography committee on standarts, subcommitee on quantitation of two-dimensional echocardiograms. J Am Soc Echocardiogr. 1989;2:358-67.

10. Ozer N. The role of Doppler echocardiography in the evaluation of myocardial ischemia and regional function abnormalities in patients with coronary artery disease. Anadolu Kardiyol Derg. 2008;8:23-8.

11. Charley R, Cohen M. The role of the coronary collateral circulation in limiting myocardial ischemia and infarct size. Am Heart J. 1993;126:937-45.

12. Weisman HF, Bush DE, Mannisi JA, Weisfeldt ML, Healy B. Cellular mechanisms of myocardial infarct expansion. Circulation. 1988;78:186-201.

13. Mizuno K, Horiuchi K, Matui H, et al. Role of coronary collateral vessels during angioplasty assessed by hemodynamic, electrocardiographic and metabolic changes. J Am Coll Cardiol. 1988;12:624-8.

14. de Bruyne B, Meier B, Finci L, Urban P, Rutishauser W. Potential protective effect of high coronary wedge pressure on left ventricular function after coronary occluson. Circulation. 1988;78:566-72.

15. MacDonald RG, Hill JA, Feldman RL. ST segment response to acute coronary occlusion: coronary hemodynamic and angiographic determinants of direction of ST segmentshift. Circulation. 1986;74:973-9.

16. Carroll RJ, Verani MS, Falsetti HL. The effect of collateral circulation on segmental left ventricularcontraction. Circulation. 1974;50:709-813.

17. Charney R, Cohen M. The role of the coronary collateral circulation in limiting myocardial ischemia and infarctsize. Am Heart J. 1993;126:937-45.

18. Lee CW, Park SW, Cho GY, et al. Pressure-derived fractional collateral blood flow: a primary determinant of left ventricular recovery after reperfused acute myocardial infarction. J Am Coll Cardiol. 2000;35:949-55.

19. Hirai T, Fujita M, Nakajima H, et al. Importance of collateral circulation for prevention of left ventricular aneurysm formation in acute myocardial infarction. Circulation. 1989;79:791-6.

20. Kukulski T, Jamal F, Herbots L, et al. Identification of acutely ischemic myocardium using ultrasonic strain measurements. A clinical study in patients undergoing coronary angioplasty. J Am Coll Cardiol. 2003;41:810-9.

21. Gallagher KP, Matsuzaki M, Koziol JA, Kemper WS, Ross J. Regional myocardial perfusion and wall thickening during ischemia in conscious dogs. Am J Physiol. 1984;247:727-38.

22. Jamal F, Kukulski T, Strotmann J, et al. Quantification of the spectrum of changes in regional myocardial function during acute ischemia in closed chest pigs. An ultrasound strain rate an strain study. J Am Soc Echocardiogr. 2001;14:874-84.

23. Ingul CB, Stoylen A, Slordahl SA. Recovery of stunned myocardium in acute myocardial infarction quantified by strain imaging: a clinical study. J Am Soc Echocardiogr. 2005;18:401-10.