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The role of revascularization on short-term Heart Rate Variability (HRV) and Signal Averaged Electrocardiogram (SAECG) in Stable Coronary Artery Disease (CAD)

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ABSTRACT

Background: Revascularization in patients with stable coronary artery disease (SCAD) can reduce myocardial ischemia and improves the autonomic nervous system, which can be measured by heart rate variability (HRV) and signal-averaged electrocardiogram (SAECG). This study aimed to investigate the effect of revascularization on HRV and SAECG in patients with SCAD.

Methods: This is a single-center prospective cohort study. Revascularization refers to percutaneous coronary intervention (PCI). The primary outcome of this study is the change in short-term HRV and SAECG from before revascularization to after revascularization. HRV and SAECG measurement was performed for 10 minutes before PCI and one-month post-PCI. Data were analyzed using SPSS version 23 for Windows.

Results: There are 30 patients included in this study, and 46.7% underwent incomplete PCI. There was no significant difference in HRV parameters in the time domain method. In the frequency domain, only the LF/HF ratio changed significantly from 3.5±2.6 before PCI to 2.16±1.9 after PCI, with a 1.33 decrease in LF/HF (p=0.007). Subgroup analysis was performed for patients receiving complete and incomplete revascularization. In patients who received complete revascularization, there was a significant difference in LF/HF ratio change of 1.6±2.28 (p=0.013). In patients that receive incomplete revascularization, there is a significant increase in HF 37.8±57.3 (p=0,028). In terms of SAECG parameters, there is no significant difference between before and after PCI in both primary and subgroup analysis.

Conclusion: Revascularization resulted in a significant reduction in LF/HF ratio assessed by short-term HRV. There was no benefit of revascularization in terms of SAECG parameters.

Keywords: SAECG, Heart Rate Variability, Revascularization, Percutaneous Coronary Intervention, Coronary Artery Disease.
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INTRODUCTION

Patients with Stable Coronary Artery Disease (SCAD) are the most common referral to cardiology practice. This population is a heterogeneous mixture of varying degrees of left ventricular dysfunction, and coronary artery obstruction, with or without prior myocardial infarction.[1] Management of SCAD comprises controlling the risk factors, optimal pharmacological intervention and revascularization. A common indication is persistent symptoms despite optimum medical treatment or to improve prognosis.[2]

Patients with SCAD often experience depolarization and autonomic system dysfunction. The autonomic nervous system can be assessed by evaluating the Heart Rate Variability (HRV), while ventricular depolarization abnormality can be assessed using a Signal-Averaged Electrocardiogram (SAECG).[1,3] HRV is the variation of RR interval between consecutive beats mainly regulated extrinsically from heart rhythm. HRV is caused by dysfunction of the cardiac autonomic nervous system and circulation. Usually, there is a balance between the sympathetic and parasympathetic nervous systems, regulating the heart rate.[3] Patients with SCAD may experience HRV alteration due to increased sympathetic activities.[4] HRV measurements have different time intervals; short-term HRV (5-10 minutes) is similar to long term HRV (24 hours) in terms of risk stratification for ischemic heart diseases.[5,6] Due to time efficiency, short-term HRV is a more attractive option for this purpose. SAECG is high-resolution electrocardiography that can measure low electrical amplitude in the myocardium and detect the ventricular depolarization area with slow conduction, known as Late Potential (LP).[7] SAECG may evaluate LP, which is a substrate for arrhythmogenesis. Abnormal SAECG is associated with a higher degree of coronary stenosis and poorer prognosis in patients with SCAD.[1]

Revascularization in patients with SCAD can reduce myocardial ischemia, improve the autonomic nervous system, and improve myocardial perfusion. This, in turn, may improve HRV and SAECG parameters in patients with SCAD. Based on those mentioned above, this study aims to investigate the effect of revascularization on HRV and SAECG in patients with SCAD.

METHODS

This is a single-center prospective cohort study in the Department of Cardiology and Vascular Medicine, RSUP Dr. R. D. Kandou Hospital, Manado, Indonesia. A consecutive sampling of adults with SCAD from both inpatient and outpatient departments was performed between May to August 2019. Revascularization refers to percutaneous coronary intervention (PCI). The study was conducted in accordance with the ethical standards of the 1964 Helsinki Declaration and its later amendments. Informed consent was not obtained due to the retrospective observational nature of the study. The inclusion criteria were: 1) Patients with stable angina pectoris with or without risk factors (diabetes, hypertension, smoking, dyslipidemia and obesity); 2) Provide consent for short term HRV and SAECG, treadmill exercise test, and inclusion for the study; 3) Patients without prior history of PCI or coronary artery bypass graft (CABG); and 4) Sinus rhythm. In addition, the exclusion criteria were: 1) Patients with resting ventricular or supraventricular arrhythmias with ECG measurement; 2) Patients with acute heart failure; 3) Patients with acute coronary syndrome requiring primary or early PCI; 4) Presence of left bundle branch block (LBBB) or ST-segment deviation > 1 mm on ECG; 5) Patients with anti-arrhythmic medication (except beta-blocker), antipsychotics, and anti-depressant; 6) Patients with a pacemaker; 7) Patients with atrial fibrillation; and 8) Uncooperative patients.

The primary outcome of this study is the change in short-term HRV and SAECG from before revascularization to after revascularization. HRV and SAECG measurement was performed for 10 minutes. For HRV, the time domain and frequency domain analyses were performed. SAECG was recorded by resting 12-lead during sinus rhythm with bipolar X, Y and Z lead. The recording was performed using Holter Biox (VasoMedical Inc) at rest for 5 minutes, and the results were analyzed using ARCS series ECG and ABP analysis and reporting software. Patients were evaluated using Holter before PCI and one 1-month post-PCI.

Statistical analysis was conducted using the Statistical Product and Service Solutions (SPSS) version 23. The target for sample size was derived from the formula for paired T-test. Descriptive analysis was performed and reported as frequency table, mean, standard deviation, and minimum-maximum. The role of revascularization on short-term HRV and SAECG were numerical data that was tested using Paired T-test (prior and after revascularization). P-values < 0.05 were considered as statistically significant.

RESULTS

There are 30 patients with SCAD who fulfilled the inclusion criteria and underwent PCI after HRV measurement, followed by HRV measurement post-PCI. The baseline characteristics of the patients were included in Table 1. Most of the patients were male. The mean syntax score was 19.7, and from all patients that underwent PCI, 16 patients (53.3%) underwent complete PCI, and 14 patients (46.7%) underwent incomplete PCI (Table 1).

HRV measurement was conducted before PCI and one month after PCI. There was no significant difference in HRV parameters in the time domain method, but there was a significant difference in the frequency domain method. From several frequency domain parameters, only LF/HF ratio changed significantly from 3.5±2.6 before PCI to 2.16±1.9 after PCI, with a 1.33 decrease in LF/HF (p=0.007) (Table 2).

Subgroup analysis was performed for patients receiving complete and incomplete revascularization. In patients who received complete revascularization, there was a significant difference in LF/HF ratio change of 1.6±2.28 (p=0.013) (Table 3). In patients that receive incomplete revascularization, there is a significant increase in HF 37.8±57.3 (p=0.028) (Table 4). In terms of SAECG parameters, there is no significant difference (p>0.05) between before and after PCI in both primary and subgroup analysis (Table 5).

DISCUSSION

This study showed a significant LF/HF ratio reduction upon short-term HRV measurement in patients with SCAD after PCI, which indicates a reduction of sympathetic nervous system activity. There was no significant difference in SAECG parameters in patients with SCAD after PCI.

The mean short-term HRV for the time domain in this study differs from that of Nunan D et al., that conduct the study on a normal population.[8] The mean RR, SDNN, and rMSSD in our population were 908.6±133.9, 55.8±86.6, and 44.2±107.3, respectively. While in Nunan et al.'s study, the RR, SDNN, and rMSSD were 926±90, 50±16, and 42±15, respectively.[8] The frequency domain LF, HF, and LF/HF ratios in this study were 701.2±751, 249.7 ±253.9, 3.5±2.62, respectively. While the LF, HF, and LF/HF ratios reported by Nunan et al. were 519±291, 657±777, and 2.8±2.6, respectively.[8]

About 33.3% of the patients have normal SAECG, and 66.6% have abnormal SAECG measured by QRS duration, RMS40, and LAS40. The mean SAECG in this study also differs from that of Danford DA et al., who measured SAECG in a healthy population.[9] The result is similar to Gomes ME et al., especially in the HRV for the frequency domain.[10] Gomes ME et al. measured the HRV change before and one month after PCI in patients who received complete revascularization exclusively and found an improvement in the LF/HF ratio.[10] They reported a significant decrease in the LF/HF ratio from 3.7±0.6 to 2.4±0.4, p < 0.05 and concluded that PCI resulted in lower sympathetic nervous system activity in patients undergoing PCI.[10] In our study, we found a significant decrease in LF/HF ratio from 3.5±2.62 to 2.16±1.9.

LF/HF ratio assesses the sympathetic and parasympathetic nervous system activity, in which sympathetic nervous system activity contributed to LF, and parasympathetic activity contributed to

Table 1. Baseline Characteristics.

Variables	N (%)	Mean± SD
Male	19 (63.0)	
Age (years)		61.1±8.1
Weight (kg)		67.8±12.4
Height (m)		1.6±0.1
Body Mass Index(kg/m^2)		25.7±3.7
Body Surface Area		1.7±0.2
Hemoglobin (g/dL)		13.2±1.4
Leukosit (/µL)		8,203.3±3,154.8
Trombosit (/µL x 1000)		263.4±812.5
Fasting Blood Glucose (mg/dL)		129.4±70.2
Random Blood Glucose (mg/dL)		163.8±69.3
HbA1C (%)		7.0±1.5
Ureum (mg/dL)		31.7±16.6
Creatinine (mg/dL)		1.4±1.7
eGFR (CKD-EPI)		77.3±23.4
Uric Acid (mg/dL)		7.2±2.0
Aspartate Aminotransferase (U/L)		30.4±24.0
Alanine Aminotransferase (U/L)		33.2±41.7
Total Cholestrol (mg/dL)		166.2±47.9
High Density Lipoprotein (mg/dL)		36.6±8.5
Low Density Lipoprotein (mg/dL)		99.0±40.1
Triglyceride (mg/dL)		163.0±78.7
Risk Factors		
Diabetes	15 (50.0)	
Hypertension	27 (90.0)	
Smoking	16 (53.0)	
Dyslipidemia	13 (43.0)	
Obesity	5 (17.0)	
Medications		
Aspirin	30 (100.0)	
Clopidogrel	18 (60.0)	
Ticagrelor	12 (40.0)	
ACE-I	10 (33.0)	
ARB	16 (53.0)	
Bisoprolol	22 (73.0)	
Metoprolol	1 (3.0)	
Statin	30 (100.0)	
Amlodipine	15 (50.0)	
Isosorbide dinitrate	3 (10.0)	
Glyceryl trinitrate	29 (97.0)	
OAD	9 (30.0)	
Insulin	5 (17.0)	
Coronary Artery Involvement		
LM	3 (10.0)	
LAD	28 (93.0)	
LCx	18 (60.0)	
RCA	20 (67.0)	
Number of Vessels Involved		
One	8 (27.0)	
Two	8 (27.0)	
Three	14 (47.0)	
Revascularization		
Complete	16 (53.0)	
Incomplete	14 (47.0)	
Ejection Fraction (%)		59.7±14.3
SYNTAX Score		19.7±12.2

ACE-I: Angiotensin-Converting Enzyme Inhibitor, ARB: Angiotensin Receptor Blocker, OAD: Oral Antidiabetic Drugs, LM: left main, LAD: Left Anterior Descending, LCx: Left Circumflex, RCA: Right Coronary Artery, SYNTAX score: Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery.

HF. By evaluating the LF/HF ratio, we can estimate the ratio between sympathetic and parasympathetic nervous system activity.[11,12] A high LF/HF ratio reflects sympathetic dominance, and a low LF/HF ratio reflects parasympathetic dominance.[5] Our study showed that the LF/HF ratio decreases after PCI. Aydinlar et al. indicate that HRV change before PCI (5 minutes) to briefly after balloon inflation (5 minutes) resulted in increased HF and decreased LF/HF ratio. This demonstrates that there has been reduced sympathetic nervous system activity after PCI.[13]

The time-domain analysis in this study differs from that of previous studies. Abrootan et al. and Aydinlar et al. showed significant improvement in SDNN 24 hours after PCI.[13,14] In our study, there was no significant difference in all time-domain HRV parameters. This may be caused due to the high rate of incomplete revascularization in our study and the temporal difference in HRV measurement.

Intermittent chronic ischemia, usually found in SCAD, may slow down electrical activity in several areas of the myocardium. SAECG in patients with coronary artery disease differs from those without coronary artery disease. Berkalp P et al. compared the SAECG before and after PCI in patients with SCAD and found QRS duration, RMS40, and LAS40 improvement.[15] That study showed that PCI could improve late potential parameters, which may decrease the risk for malignant arrhythmia, and increase the potential of high-frequency QRS, indicating reduced ischemia after PCI.[15] Our study did not demonstrate a significant difference in late potential after PCI in patients with SCAD. This result is similar to Sotomi Y et al., which reported no significant improvement in terms of late potential after revascularization.[16] Thus, the role of revascularization in SAECG parameters remains uncertain.

Myocardial revascularization using PCI aims to minimize residual ischemia.[2] Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation (COURAGE) study demonstrate added benefit in reducing mortality and myocardial infarction by reducing residual stress-induced ischemia.[17] Recurrence of symptoms or ischemia after PCI might be due to restenosis, incomplete revascularization, or disease progression.[2] In this study, 46.7% underwent incomplete revascularization, leaving residual ischemia. In the subgroup of patients undergoing incomplete revascularization, there was a significant increase in HF, indicating increased parasympathetic nervous response after PCI.

In the complete revascularization subgroup, the LF/HF ratio considerably decreases, which indicates a reduction in sympathetic nervous system activity. There was also a decrease in VLG, which demonstrated a reduction in sympathetic nervous system activity. However, it can be altered by physical activity and thermoregulatory changes.[18-20] There is a need for further research in a larger sample size with a longer follow-up duration to evaluate long term benefits and whether the improvement of the parameters leads to better clinical outcome

Table 2. Change in Short Term Heart Rate Variability.

Variables	Pre PCI		Post PCI		Pre – Post PCI		
	Mean	SD	Mean	SD	Mean	SD	p[a]
Mean RR	908.60	133.90	907.90	173.60	0.75	139.7	0.977
SDNN	55.80	86.60	51.40	86.60	4.32	22.02	0.290
SDANN	34.50	99.30	31.70	99.60	2.84	137.23	0.911
rMSSD	44.20	107.30	55.20	136.40	-10.97	36.27	0.108
pNN50 (%)	6.93	12.97	8.54	14.20	-1.60	11.85	0.465
Total Power (TP)	6,499.70	16,648.60	4,396.60	1,059.60	2,103.1	7,087.9	0.115
Very Low Frequency (VLF)	5,548.70	16,035.90	3,216.50	9,720,30	2,332.2	6,484.0	0.058
Low Frequency (LF)	701.20	751.00	680.50	1584.90	20.72	1,318.87	0.932
High Frequency (HF)	249.70	253.90	499.50	1150.70	-249.83	987.45	0.176
LF/HF	3.50	2.62	2.16	1.90	1.33	2.54	0.007*

[a]Paired T-Test: Statistically significant if p-value less than 0.05; SDNN: Standard Deviation Normal to Normal, SDANN: Standard Deviation Average Normal to Normal, rMSSD: Root Mean Square of Successive Differences, pNN50: Percentage of Successive RR Intervals

Table 3. Change in Short Term Heart Rate Variability (Complete Revascularization Subgroup).

Variables	Pre PCI		Post PCI		Pre – Post PCI		
	Mean	SD	Mean	SD	Mean	SD	*p*[a]
Mean RR	902,3	110,1	931,6	114,3	-29,3	134,0	0,395
SDNN	47,6	22,8	44,7	41,7	2,9	28,6	0,686
SDANN	18,4	12,4	46,7	135,9	-28,3	139,0	0,427
rMSSD	30,7	28,7	42,7	58,2	-11,9	36,1	0,204
pNN50(%)	5,4	7,0	9,1	12,5	-3,65	14,92	0,343
Total Power (TP)	4553,9	3881,0	3250,6	4661,4	1303,2	4187,5	0,232
Very Low Frequency (VLF)	3335,2	3158,0	1559,4	1334,4	1775,8	2319,3	0,008
Low Frequency (LF)	869,8	725,7	907,1	2113,5	-37,3	1815,2	0,936
High Frequency (HF)	348,9	283,1	784,2	1530,2	-435,2	1343,0	0,214
LF/HF	3,44	2,72	1,81	1,80	1,60	2,28	0,013*

[a]Paired T-Test: Statistically significant if p-value less than 0.05; SDNN: Standard Deviation Normal to Normal, SDANN: Standard Deviation Average Normal to Normal, rMSSD: Root Mean Square of Successive Differences, pNN50: Percentage of Successive RR Intervals.

Table 4. Change in Short Term Heart Rate Variability (Incomplete Revascularization Subgroup).

Variables	Pre PCI		Post PCI		Pre – Post PCI		
	Mean	SD	Mean	SD	Mean	SD	*p*[a]
Mean RR	916.00	161.10	880.90	225.20	35.10	143.00	0.375
SDNN	65.20	126.40	59.30	120.90	5.92	11.61	0.079
SDANN	53.10	145.40	14.60	15.90	38.50	130.80	0.291
rMSSD	59.80	155.70	69.60	192.90	-9.80	37.80	0.348
pNN50(%)	8.70	17.70	7.90	16.40	0.74	6.80	0.687
Total Power (TP)	8,723.60	24,309.50	5,706.40	14,897.20	3,017.20	9,494.70	0.256
Very Low Frequency (VLF)	8,078.60	23,435.40	5,110.50	14,194.30	2,968.00	9,314.80	0.254
Low Frequency (LF)	508.60	758.70	421.60	560.70	87.00	263.50	0.238
High Frequency (HF)	136.40	159.50	174.30	196.50	-37.80	57.30	0.028*
LF/HF	3.60	2.69	2.57	1.99	1.03	2.85	0.200

[a]Paired t-test; Statistically significant if p-value less than 0.05; SDNN: Standard Deviation Normal to Normal, SDANN: Standard Deviation Average Normal to Normal, rMSSD: Root Mean Square of Successive Differences, pNN50: Percentage of Successive RR Intervals.

Table 5. Change in Signal Averaged Electrocardiogram.

Variables	Pre PCI n(%), Mean ± SD	Post PCI n(%), Mean ± SD	Pre – Post PCI n(%), Mean ± SD	p^a
Normal SAECG	10 (33)	10 (33)		1,000
Abnormal SAECG	20 (67)	20 (67)		1,000
Total QRS duration	92,20±14,82	90,50±18,69	1,70±21,03	0,661
Under 40 Vµ	11,53±11,08	10,47±12,47	1,067±15,02	0,700
Last 40 ms	127,32±66,53	118,82±74,30	8,50±70,05	0,511

[a]Two proportion test for categorical variables and paired t-test for continuous variables

CONCLUSION

There is a significant reduction of LF/HF ratio assessed by short-term HRV in patients with SCAD who underwent PCI, indicating the reduction of sympathetic nervous system activity. However, there is no significant difference between other short-term HRV parameters and SAECG parameters after PCI.

CONFLICT OF INTEREST

There was no competing interest regarding the manuscript.

ETHICS CONSIDERATION

Ethics approval has been obtained from the Ethics Committee, Faculty of Medicine, Universitas Sam Ratulangi, Prof, Dr. R. D. Kandou, Hospital, Manado, Indonesia, prior to the study being conducted.

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AUTHORS CONTRIBUTION

EJG and ALP were involved in the design and conceptualization of the study. EJG, ALP, JP, and NCHP were involved in data curation and investigation. EJG and ALP contributed to the statistical analysis. EJG and ALP write the original draft. JP and NCHP performed critical revision and editing of the manuscript.

REFERENCES

1. Hofmann T, Burmeister A, Meinertz T. Prognostic significance of the signal averaged electrocardiogram in patients with chronic stable coronary artery disease: Analysis in the time domain and by spectral temporal mapping. Z Kardiol. 2004;93(1):32–42.
2. Neumann FJ, Sousa-Uva M, Ahlsson A, Alfonso F, Banning AP, Benedetto U, et al. 2018 ESC/EACTS Guidelines on myocardial revascularization. Eur Heart J. 2019;40(2):87–165.
3. Acharya UR, Joseph KP, Kannathal N, Lim CM, Suri JS. Heart rate variability: A review. Med Biol Eng Comput. 2006;44(12):1031–1051.
4. Mironova T, Mironov V, Antufiev V, Safronova E, Mironov M, Davydova E. Heart rate variability analysis at coronary artery disease and angina pectoris. Recent Pat Cardiovasc Drug Discov. 2009;4(1):45–54.
5. Shaffer F, Ginsberg JP. An Overview of Heart Rate Variability Metrics and Norms. Front Public Health. 2017;5:258.
6. Voss A, Schroeder R, Vallverdú M, Schulz S, Cygankiewicz I, Vázquez R, et al. Short-term vs. long-term heart rate variability in ischemic cardiomyopathy risk stratification. Front Physiol. 2013;4:364.
7. Gatzoulis KA, Arsenos P, Trachanas K, Dilaveris P, Antoniou C, Tsiachris D, et al. Signal-averaged electrocardiography: Past, present, and future. J Arrhythmia. 2018;34(3):222–229.
8. Nunan D, Sandercock GRH, Brodie DA. A quantitative systematic review of normal values for short-term heart rate variability in healthy adults. PACE - Pacing Clin Electrophysiol. 2010;33(11):1407–1417.
9. Danford DA, Stelling JA, Kugler JD, Cheatham JP, Latson LA, Gumbiner CH, et al. Signal-Averaged Electrocardiography of the Terminal QRS in Healthy Young Adults. Pacing Clin Electrophysiol. 1989;12(11):1712–1716.
10. Gomes ME, Aengevaeren WR, Lenders JW, Verheugt FW, Smits P, Tack CJ. Improving myocardial perfusion by percutaneous coronary intervention reduces central sympathetic activity in stable angina. Clin Cardiol. 2010;33(6):E16-E21.
11. Shaffer F, McCraty R, Zerr CL. A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. Front Psychol. 2014;5:1040.
12. Prabawa IPY, Lestari AAW, Muliarta IM, Mardhika PE, Pertiwi GAR, Bhargah A, et al. The Stromal Cell-derived Factor-1/CXCL12 3'A-gene Polymorphism is Related to the Increased Risk of Coronary Artery Disease: A Systematic Review and Meta-analysis. Open Access Macedonian Journal of Medical Sciences. 2020;8(F):197-202.
13. Aydinlar A, Sentürk T, Ozdemïr B, Kaderlï AA, Aydin O. Effect of percutaneous transluminal coronary angioplasty on QT dispersion and heart rate variability parameters. Cardiovasc J Afr. 2009;20(4):240-244
14. Abrootan S, Yazdankhah S, Payami B, Alasti M. Changes in Heart Rate Variability Parameters after Elective Percutaneous Coronary Intervention. J Tehran Heart Cent. 2015;10(2):80-84.
15. Berkalp B, Oral D, Caglar N, Omurlu K, Pamir G, Alpman A, et al. Effects of percutaneous transluminal coronary angioplasty on late potentials and high frequency mid-QRS potentials. Cardiology. 1994;85(3–4):216–221.
16. Sotomi Y, Okamura A, Iwakura K, Date M, Nagai H, Yamasaki T, et al. Impact of revascularization of coronary chronic total occlusion on left ventricular function and electrical stability: analysis by speckle tracking echocardiography and signal-averaged electrocardiogram. Int J Cardiovasc Imaging. 2017;33(6):815–823.
17. Shaw LJ, Berman DS, Maron DJ, Mancini GBJ, Hayes SW, Hartigan PM, et al. Optimal medical therapy with or without percutaneous coronary intervention to reduce ischemic burden: results from the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation (COURAGE) trial nuclear substudy. Circulation. 2008;117(10):1283–1291
18. Bernardi L, Valle F, Coco M, Calciati A, Sleight P. Physical activity influences heart rate variability and very-low-frequency components in Holter electrocardiograms. Cardiovasc Res. 1996;32(2):234–237.
19. Taylor JA, Carr DL, Myers CW, Eckberg DL. Mechanisms underlying very-low-frequency RR-interval oscillations in humans. Circulation. 1998;98(6):547–555.
20. Limbong M, Herry Y, Ardhianto P, Suhartono. Risk score of contrast-induced nephropathy in patients after percutaneous coronary intervention. Bali Medical Journal. 2021;10(1):400-404.