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CARDIAC AUTONOMIC DYSFUNCTION AFTER ACUTE MYOCARDIAL INFARCTION: A HEART RATE VARIABILITY STUDY USING 24-HOUR HOLTER MONITORING

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ABSTRACT

Background: Myocardial infarction (MI) produces complex autonomic disturbances characterised by increased sympathetic outflow and reduced vagal tone. Heart rate variability (HRV) is a simple, non-invasive tool that can detect these maladaptive changes before overt clinical manifestations appear and can aid cardiac risk stratification. The objective is to assess cardiac autonomic function in post-MI patients using 24-hour Holter-derived HRV analysis and to compare HRV parameters across clinically relevant subgroups.

Materials and Methods: In this analytical cross-sectional study, 30 age- and BMI-matched patients with ST-elevation myocardial infarction admitted to the intensive coronary care unit of a tertiary care hospital underwent 24-hour Holter monitoring on the fifth post-MI day. Time-domain (mean RR, mean HR, SDNN, SDANN, RMSSD, NN50, pNN50%) and frequency-domain (total power, ULF, VLF, LF, HF, LFnu, HFnu, LF/HF) HRV indices were derived per Task Force of the European Society of Cardiology guidelines. Patients were stratified by left ventricular ejection fraction (LVEF ≥40% vs <40%), anatomical infarct site (inferior, anterior, posterior wall), and number of vessels occluded (single, double, triple vessel disease). Groups were compared using the Mann-Whitney U test (two groups) or one-way ANOVA (three groups); $p<0.05$ was considered significant.

Results: Patients with LVEF <40% had significantly higher mean heart rate and significantly lower SDNN, SDANN, RMSSD, NN50, pNN50%, total power, LF, HF, and HFnu than patients with LVEF ≥40% (all $p<0.05$). Across infarct sites, all time-domain indices except pNN50%, and total power, ULF, VLF, and LF among frequency-domain indices, differed significantly, with the poorest HRV in posterior wall MI and the best in inferior wall MI. Across vessel-disease subgroups, mean RR, mean HR, SDNN, and SDANN, together with total power, ULF, LF, LFnu, HFnu, and LF/HF, differed significantly, with triple-vessel disease showing the greatest autonomic imbalance.

Conclusion: Reduced HRV correlates with lower LVEF, anterior/posterior infarct location, and multivessel disease, confirming its value as a marker of post-MI cardiac autonomic imbalance. Twenty-four-hour HRV analysis merits inclusion in routine post-MI risk stratification and follow-up.

Keywords: Heart rate variability; myocardial infarction; cardiac autonomic dysfunction; Holter monitoring; ejection fraction; risk stratification.

INTRODUCTION

Cardiovascular disease is the leading noncommunicable cause of death worldwide, responsible for an estimated 19.8 million deaths in 2022.[1] Myocardial infarction (MI) remains a major public health problem, with more than one million hospitalisations annually in the United States alone,

and its burden continues to rise in developing countries.

Following MI, complex autonomic changes occur, marked by increased cardiac and peripheral sympathetic outflow and reduced vagal tone. While these changes are initially adaptive, sustaining blood pressure, their persistence increases myocardial oxygen demand and promotes adverse cardiac remodelling.[2] Heart rate variability (HRV)—the beat-to-beat variation in heart rate and in the interval between successive R-R complexes—offers a simple, non-invasive window into this autonomic balance: sympathetic stimulation raises heart rate and lowers HRV, whereas parasympathetic stimulation has the opposite effect. Because reduced HRV after MI has been consistently linked to adverse cardiac outcomes, it has attracted interest as a tool for cardiac risk stratification.[3]

This study aimed to assess cardiac autonomic changes after myocardial infarction using 24-hour Holter-based HRV analysis, and to compare HRV parameters across patients stratified by left ventricular ejection fraction (LVEF), anatomical infarct site, and number of coronary vessels occluded.

MATERIALS AND METHODS

Study design and setting: This analytical cross-sectional study was conducted in the Intensive Coronary Care Unit (ICCU), Department of Cardiology, of a tertiary care hospital over a period of one year, after approval from the Institutional Ethics Committee.

Study population: Thirty ST-elevation myocardial infarction (STEMI) patients receiving standard treatment protocol in the ICCU were enrolled. Patients with atrial flutter, atrial fibrillation, or paced rhythm; sinus rhythm for <80% of the 24-hour recording; prior regular beta-blocker use before MI; non-cooperation; significant comorbid valvular, pulmonary, hepatic, renal, or endocrine disease; or other severe concomitant non-cardiovascular illness were excluded. Age- and BMI-matched patients were analysed for each comparison.

Patients were classified into three subgroups for comparison: (i) LVEF ≥40% versus LVEF <40%; (ii) anatomical infarct site — inferior, anterior, or posterior wall MI; and (iii) number of coronary vessels occluded — single-, double-, or triple-vessel disease.

HRV recording: Twenty-four-hour ambulatory Holter recordings were obtained on the fifth day after acute MI. Six unipolar chest electrodes were placed at standard precordial positions (V1–V6), and limb electrodes were placed using the modified Mason-Likar configuration (arm electrodes below the clavicles, leg electrodes on the lower abdomen), which reduces movement artefact and improves patient comfort compared with conventional limb placement.

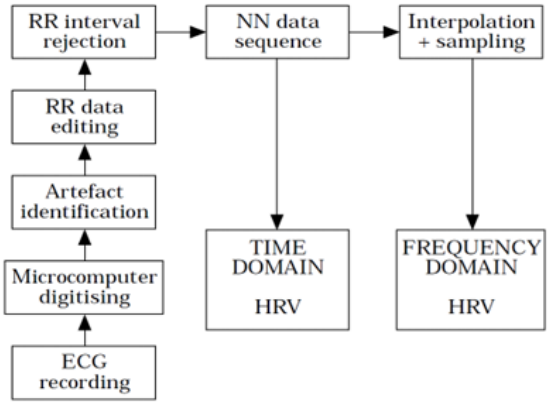

Figure 1: Steps of ECG signal recording and processing for HRV analysis.

HRV analysis: HRV was analysed in the time and frequency domains in accordance with Task Force of the European Society of Cardiology guidelines.2 Time-domain measures included mean R-R interval, mean heart rate, SDNN (standard deviation of all NN intervals), SDANN (standard deviation of 5-minute mean NN intervals), RMSSD (root mean square of successive NN interval differences), NN50, and pNN50%. Frequency-domain measures, derived by fast Fourier transform, included

Statistical analysis: Data were analysed using SPSS version 20. Because of the wide spread of HRV values, results are expressed as median (interquartile range). The LVEF subgroup (two groups) was compared using the Mann-Whitney U test; the infarct-site and vessel-disease subgroups (three groups each) were compared using one-way ANOVA. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Thirty age- and BMI-matched post-MI patients underwent 24-hour HRV analysis and were compared across the three predefined subgroups.

LVEF ≥40% versus LVEF <40% [Table 1]

Table 1: Comparison of HRV indices between patients with LVEF ≥40% and LVEF <40%

Parameter	LVEF ≥40% Median (IQR)	LVEF <40% Median (IQR)	Mann-Whitney U; p value
Mean RR (ms)	799.28 (690.7–890.3)	715.64 (652.9–794.4)	U=15; p=0.009**
Mean HR (bpm)	78 (69–82)	92 (79–98.5)	U=8.5; p=0.001**
SDNN (ms)	112 (85–161)	54.5 (49–65)	U=0; p=0.0001**
SDANN (ms)	109 (77–124)	50 (39–79)	U=1; p=0.0002**
RMSSD (ms)	31 (24–55)	23 (15.5–31)	U=19; p=0.02*
NN50	7027 (2807–12041)	3539 (472–7469)	U=23; p=0.04*
pNN50%	10 (3–21)	4 (1.5–8.5)	U=21.5; p=0.03*
Total power (ms²)	1067 (540–2997)	766 (420–1119)	U=20; p=0.02*

ULF (ms²)	24.4 (7.6–41.4)	17.5 (2.8–27.7)	U=31.5; p=0.17
VLF (ms²)	523.4 (369–780.5)	468 (143–745)	U=35; p=0.27
LF (ms²)	258.4 (166.5–977.5)	161 (113–259)	U=16; p=0.01*
HF (ms²)	178.6 (118.5–631.8)	68 (41–160)	U=9; p=0.002**
LFnu	0.5 (0.5–0.7)	0.68 (0.48–0.81)	U=27; p=0.08
HFnu	0.4 (0.29–0.46)	0.28 (0.16–0.4)	U=17; p=0.01*
LF/HF	1.4 (1.1–2.3)	2.1 (0.9–4.3)	U=27.5; p=0.09

SDNN: standard deviation of NN intervals; SDANN: standard deviation of averaged NN intervals; RMSSD: root mean square of successive differences; ULF/VLF/LF/HF: ultra-/very-low-/low-/high-frequency power; nu: normalised units. *p<0.05, **p<0.01.

Patients with LVEF <40% had a significantly faster mean heart rate and lower SDNN, SDANN, RMSSD, NN50, and pNN50% than patients with LVEF ≥40% (all p<0.05). Among frequency-domain measures, total power, LF, HF, and HFnu were significantly lower in the LVEF <40% group; ULF, VLF, LFnu, and LF/HF did not differ significantly.

Anatomical infarct site [Table 2]

Table 2: Comparison of HRV indices by anatomical site of infarction

Parameter	Inferior wall Median (IQR)	Anterior wall Median (IQR)	Posterior wall Median (IQR)	ANOVA F; p value
Mean RR (ms)	874.5 (748.3–890.3)	801.9 (711.7–864.2)	690.8 (628.9–716.7)	F=18.02; p=0.00005**
Mean HR (bpm)	76 (67–80)	80 (71–96)	93 (90–101)	F=12.84; p=0.0003**
SDNN (ms)	117.5 (100–163)	62 (54–77)	53 (47.5–56.5)	F=41.68; p<0.00001**
SDANN (ms)	112 (95.5–124.5)	44 (40–65)	53 (38–89)	F=33.10; p<0.00001**
RMSSD (ms)	32.5 (24–68)	24 (19–29)	13 (10–31)	F=4.42; p=0.02*
NN50	8766 (2947–15413)	4564 (2514–6842)	381 (212–7530)	F=4.69; p=0.02*
pNN50%	10.5 (3–29.5)	5 (2–8)	1 (0–9)	F=3.24; p=0.06
Total power (ms²)	1175 (859–3424)	825.3 (742.6–1348.4)	505.2 (237.2–669.2)	F=4.49; p=0.02*
ULF (ms²)	26 (12.9–46.8)	28.3 (22.7–29.3)	9 (2.3–16.8)	F=9.28; p=0.001**
VLF (ms²)	670 (442–914)	607.3 (460.9–802.3)	259.6 (80.5–414.1)	F=13.63; p=0.0002**
LF (ms²)	293 (213–1148)	167.6 (134.5–269.1)	115.2 (53.7–162)	F=5.17; p=0.01*
HF (ms²)	189.4 (132.2–981.2)	57.8 (49.2–152.5)	38.2 (14.7–144.3)	F=2.94; p=0.07
LFnu	0.5 (0.5–0.7)	0.6 (0.5–0.8)	0.6 (0.5–0.8)	F=0.89; p=0.4
HFnu	0.4 (0.2–0.4)	0.3 (0.1–0.4)	0.3 (0.1–0.4)	F=0.89; p=0.4
LF/HF	1.4 (1–2.4)	2.3 (1.1–4.6)	1.8 (1.1–5.2)	F=1.46; p=0.2

*p<0.05, **p<0.01.

All time-domain indices except pNN50% differed significantly across infarct sites, with the highest values (best HRV) in inferior wall MI and the lowest in posterior wall MI. Among frequency-domain indices, total power, ULF, VLF, and LF differed significantly, again favouring inferior wall MI; HF, LFnu, HFnu, and LF/HF showed no significant difference, although patients with inferior wall MI trended toward higher HFnu and lower LFnu and LF/HF, suggesting relatively preserved parasympathetic tone.

Number of vessels occluded [Table 3]

Table 3: Comparison of HRV indices by number of coronary vessels occluded

Parameter	Single-vessel Median (IQR)	Double-vessel Median (IQR)	Triple-vessel Median (IQR)	ANOVA F; p value
Mean RR (ms)	799.2 (764.8–891.2)	715.3 (652.9–853.7)	711.8 (626.8–761.8)	F=10.0; p=0.001**
Mean HR (bpm)	75.5 (67–80)	91 (83–97)	93 (83–94.5)	F=6.07; p=0.009**
SDNN (ms)	122 (104–165)	58 (50.5–54.5)	54 (49–69)	F=40.7; p=0.00001**
SDANN (ms)	114 (96–125)	65 (41–45.5)	53 (39–74)	F=24.9; p=0.00001**
RMSSD (ms)	31 (24–81)	24 (12–15.5)	28 (14.5–30.5)	F=2.71; p=0.09
NN50	7572 (2837–18786)	5551 (355–1539)	2807 (330–6841)	F=2.42; p=0.11
pNN50%	10 (3–38)	6 (0.5–1.5)	3 (1–8.5)	F=2.44; p=0.11
Total power (ms²)	1175 (837–3852)	740.4 (522.9–569.3)	556.4 (258–1382.8)	F=5.31; p=0.01*
ULF (ms²)	27.2 (21–52)	18.2 (9.9–12.3)	3.3 (2.3–26)	F=8.61; p=0.002**
VLF (ms²)	670 (442–1047)	435.5 (314.3–380.9)	146.9 (80.1–776.6)	F=3.48; p=0.05
LF (ms²)	292 (204–1319)	156.3 (118.4–142.5)	167.6 (69.5–284.9)	F=5.01; p=0.01*
HF (ms²)	182.9 (118–1330)	57.8 (26.1–43.7)	152.5 (30.4–266.9)	F=2.66; p=0.09
LFnu	0.5 (0.4–0.7)	0.6 (0.62–0.66)	0.6 (0.4–0.7)	F=4.34; p=0.02*
HFnu	0.4 (0.2–0.5)	0.3 (0.15–0.18)	0.3 (0.2–0.5)	F=4.34; p=0.02*
LF/HF	1.2 (0.9–2.4)	2.3 (1.7–1.9)	1.5 (0.7–2.7)	F=4.43; p=0.02*

*p<0.05, **p<0.01.

Mean RR, mean HR, SDNN, and SDANN differed significantly across vessel-disease subgroups, with single-vessel disease showing the best and triple-vessel disease the poorest time-domain HRV; RMSSD, NN50, and pNN50% showed no significant difference. Among frequency-domain measures, total power, ULF, LF, LFnu, HFnu, and LF/HF differed significantly, with single-vessel disease again showing the most favourable profile. Mean LVEF was highest in single-vessel disease (48%±8%),

intermediate in double-vessel disease (45%±9%), and lowest in triple-vessel disease (37%±6%).

DISCUSSION

Cardiac autonomic imbalance following myocardial insult is an established contributor to adverse cardiovascular outcomes, as shown in large cohorts such as the Framingham Heart Study.[4] The present findings extend this evidence by demonstrating a graded relationship between HRV

Patients with LVEF <40% showed a faster heart rate and lower time- and frequency-domain HRV than those with preserved LVEF, consistent with reduced baroreflex afferent activity and heightened efferent sympathetic drive that accompany low cardiac output.[5] This mirrors the ATRAMI study, an international multicentre study of 1284 post-MI patients, which linked low SDNN and reduced baroreflex sensitivity to LVEF below 35% and established that autonomic markers carry independent prognostic value after MI.[6] Similarly, Song et al. identified LVEF and time-domain HRV as complementary predictors of risk after acute MI,[7] while Huikuri and Stein highlighted HRV's role in predicting fatal and near-fatal arrhythmias in patients with reduced LVEF,[8] and Curtis and O'Keefe described abnormal autonomic tone as the most sensitive predictor of arrhythmic and cardiac mortality in the setting of left ventricular dysfunction.[9]

HRV also varied by infarct location, with the best autonomic profile in inferior wall MI and the worst in posterior wall MI. This is consistent with reports that inferior wall MI carries a more favourable in-hospital prognosis, with lower rates of heart failure and conduction defects, than anterior wall MI.[10] A proposed mechanism is that chemosensitive vagal afferents are concentrated in the inferior left ventricular wall, so ischaemia there triggers a reflex sympatho-inhibition, whereas anterior wall ischaemia produces a reflex sympatho-excitation that worsens autonomic balance and LVEF.[11] Poor outcomes in posterior wall MI have similarly been attributed to a delay in electrocardiographic diagnosis — because posterior infarction produces only reciprocal ST depression rather than elevation — which may allow a larger infarct to develop before treatment.

Autonomic imbalance was also greatest in patients with triple-vessel disease and least in single-vessel disease, paralleling the graded fall in LVEF across these groups and the historically poorer long-term survival reported with multivessel disease, likely reflecting larger infarct size and a higher burden of complications such as heart failure and arrhythmia.

CONCLUSION

Twenty-four-hour Holter-derived HRV analysis reveals a consistent gradient of cardiac autonomic imbalance after myocardial infarction: it is greatest in patients with LVEF <40%, posterior wall infarction, and triple-vessel disease, and least in patients with preserved LVEF, inferior wall infarction, and single-vessel disease. As a simple, non-invasive, and reproducible measure that reflects the same physiological continuum as LVEF and angiographic extent of disease, HRV merits inclusion in routine risk stratification and follow-up of post-MI patients to help guide lifestyle modification, follow-up intensity, and treatment adherence, and thereby to reduce the risk of fatal arrhythmia and sudden cardiac death.

This study was limited by its small, single-centre sample and cross-sectional, one-time design; larger, longitudinal studies with serial HRV assessment are needed to confirm these findings and clarify their prognostic value.

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