CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.3604137

Available online at: http://www.iajps.com Research Article

ANALYSIS OF HEART RATE CHANGES IN ANTERIOR AND INFERIOR WALL ST ELEVATION MYOCARDIAL INFARCTION

Maria Noor, Abdullah Bilal, M. Zarrar Arif Butt

Article Received: November 2019 **Accepted:** December 2019 **Published:** January 2020

Abstract:

An inferior wall myocardial infarction also known as IWMI, or inferior MI, or inferior ST segment elevation MI, or inferior STEMI occurs when inferior myocardial tissue supplied by the right coronary artery, or RCA, is injured due to thrombosis of that vessel. The basic aim of the study is to analyse the heart rate changes in anterior and inferior wall ST elevation myocardial infarction. This descriptive study was conducted in Health department Punjab during 2019. The data was collected from 100 patients of both genders. All patients of anterior and inferior wall ST elevation myocardial infarction only who got thrombolytic therapy with age range 30-60 of both gender were included. The data was collected from 100 patients of both male and female. Patients with NSTEMI were older than those with STEMI, and presented more often history of hypertension, previous MI and coronary revascularization procedures, and clinical signs of metabolic syndrome. Patients with NSTEMI had greater number of critical coronary stenoses, revascularization was more often incomplete, and such patients presented more often with symptoms of heart failure on initial admission to the coronary care unit. It is concluded that there is no difference among the heart rate variability indices in different type of MI, age group and gender. Data from this study show two major important results, first, that SA can detect differences in cardiac autonomic modulation after primary PCI.

Corresponding author:
Maria Noor,

QR code

Please cite this article in press Maria Noor et al., ***Analysis Of Heart Rate Changes In Anterior And Inferior Wall St Elevation Myocardial Infarction..,*** *Indo Am. J. P. Sci, 2020; 07(01).*

INTRODUCTION:

An inferior wall myocardial infarction also known as IWMI, or inferior MI, or inferior ST segment elevation MI, or inferior STEMI occurs when inferior myocardial tissue supplied by the right coronary artery, or RCA, is injured due to thrombosis of that vessel. When an inferior MI extends to posterior regions as well, an associated posterior wall MI may occur. Heart rate variability (HRV) has been known as a measurable parameter of the cardiac autonomic function. The cardiac autonomic innervation is heterogeneous and hence leads to different patterns of autonomic modulation. The normal pattern of autonomic modulation is altered in the case of myocardial infarction, the pattern of alteration is not uniform, and it depends on the infarcted wall or region of the heart. This altered autonomic modulation starts within a few hours after the acute event [1]. In ST-segment elevation myocardial infarction (STEMI) patients, cardiac autonomic modulation is predominantly characterized by activated sympathetic and withdrawn parasympathetic activity in the early hours after STEMI. It is worth mentioning that this autonomic modulation shows a difference according to the location of the infarction, with the inferior/posterior/right ventricular infarctions showing a more pronounced vagal/vaso-depressive response while the anterior infarctions showing a more pronounced sympathetic response [2].

However, the effect of treatment of the acute MI whether by fibrinolysis or primary percutaneous coronary intervention (PCI) has not been adequately studied regarding its effect on recovery of the normal pattern of autonomic cardiac modulation [3]. The importance of this point arises from the effect of the autonomic modulation on contributing to reperfusion injury and arrhythmias such as non-sustained ventricular tachycardia [4].

An anterior wall myocardial infarction also known as anterior wall MI, or AWMI, or anterior ST segment elevation MI, or anterior STEMI occurs when anterior myocardial tissue usually supplied by the left anterior descending coronary artery suffers injury due to lack of blood supply. When an AWMI extends to the septal and lateral regions as well, the culprit lesion is usually more proximal in the LAD or even in the left main coronary artery. This large anterior myocardial infarction is termed an extensive anterior [5].

Inferior wall myocardial infarction (MI) occurs from a coronary artery occlusion with resultant decreased perfusion to that region of the myocardium. Unless there is timely treatment, this results in myocardial ischemia followed by infarction. In most patients, the inferior myocardium is supplied by the right coronary artery. In about 6-10% of the population, because of left dominance, the left circumflex will supply the posterior descending coronary artery. Approximately 40% of all MIs involve the inferior wall. Traditionally, inferior MIs have a better prognosis than those in other regions, such as the anterior wall of the heart. The mortality rate of an inferior wall MI is less than 10%. However, several complicating factors that increase mortality, including right ventricular infarction, hypotension, bradycardia heart block, and cardiogenic shock [6].

Aims and objectives

The basic aim of the study is to analyse the heart rate changes in anterior and inferior wall ST elevation myocardial infarction.

MATERIAL AND METHODS:

This descriptive study was conducted in Health department Punjab during 2019. The data was collected from 100 patients of both genders. All patients of anterior and inferior wall ST elevation myocardial infarction only who got thrombolytic therapy with age range 30-60 of both gender were included. Those patients who were not willing, autonomic neuropathy, thyrotoxicosis, hypothyroidism, diabetes mellitus, renal failure, previous history of MI, late presentation (after 24 hrs), cardiogenic shock, or known case of valvular heart disease, ventricular arrhythmia or atrial fibrillation, second or third degree AV nodal block, frequent PVCs (10/min), bigeminy or trigeminy and cerebrovascular accident were excluded. Those who required cardiopulmonary resuscitation during Holter monitoring or problem in computer processing (difficult analysis due to signal artifact, sinus beat 15%, and recording.

Statistical analysis

The data was collected and analysed using SPSS version20.0. All the values were expressed in mean and standard deviation.

RESULTS:

The data was collected from 100 patients of both male and female. Patients with NSTEMI were older than those with STEMI, and presented more often history of hypertension, previous MI and coronary revascularization procedures, and clinical signs of metabolic syndrome. Patients with NSTEMI had greater number of critical coronary stenoses, revascularization was more often incomplete, and such patients presented more often with symptoms of heart failure on initial admission to the coronary care unit.

Table 1: Main data of patients, reported for the whole group and for patients with ST-elevation myocardial infarction and non-ST-elevation myocardial infarction

	All patients	**STEMI**	**NSTEMI**	**P[1]**	**P[2]**
Age, yr	63.5 ± 12.1	61.3 ± 12.5	67.4 ± 10.4	< 0.001	
Previous AMI, n (%)	60 (18)	21 (10)	39 (33)		< 0.001
Previous stroke, n (%)	11 (3)	5 (2)	6 (5)		0.193
Total cholesterol (under treatment), mg/dL	124.3 ± 26.0	123.4 ± 26.3	125.8 ± 25.5	0.424	
Metabolic syndrome, n (%)	204 (62)	124 (60)	80 (68)		0.011
BMI	27.2 ± 4.3	26.9 ± 3.7	27.9 ± 5.2	0.090	
AMI characteristics					
Anterior, n (%)	171 (52)	138 (66)	33 (28)		< 0.001
Inferior, n (%)	86 (26)	66 (32)	20 (17)		0.003
Other, n (%)	69 (21)	4 (2)	65 (55)		< 0.001
Coronary vessels with critical lesions, n	2.05 ± 0.85	1.94 ± 0.84	2.25 ± 0.85	0.002	
Incomplete revascularization, n (%)	151 (46)	87 (42)	64 (54)		0.031
Left ventricle ejection fraction, %	47.2 ± 10.3	47.8 ± 9.2	46.4 ± 12.0	0.222	
Patients with LVEF < 40%, n (%)	85 (26)	43 (21)	41 (35)		0.006
Patient with heart failure at initial admission, n(%)	37 (11)	15 (7)	22 (19)		0.002
Time before Holter, d	16.2 ± 9.6	15.6 ± 9.5	17.4 ± 9.8	0.117	
Therapy at time of discharge from hospital (number of cases, %)					
Aspirin	314 (96)	202 (97)	112 (95)		0.469
Clopidogrel	302 (93)	192 (92)	110 (93)		0.458
Warfarin	38 (12)	22 (11)	16 (14)		0.399
β-blocker	290 (89)	189 (91)	101 (86)		0.198
Ca-antagonist	38 (12)	18 (9)	20 (17)		0.022
ACE-inhibitor	264 (81)	180 (86)	84 (71)		0.001
AT-II-antagonist	43 (13)	16 (8)	27 (23)		< 0.001
Statin	314 (96)	201 (97)	113 (96)		0.893
Diuretic(s)	140 (43)	75 (36)	65 (55)		0.001
HRV parameters					
Mean heart rate, bpm	68.1 ± 10.0	69.1 ± 10.1	66.2 ± 9.7	0.016	

DISCUSSION:

HRV is accepted as clinical test when it was 13 confirmed that it is one of the strong and independent risk factor for cardiac arrhythmias and sudden death especially after acute myocardial infarction [7]. Sympathetic surge after 14 acute myocardial infarction is strong predictor of malignant arrhythmias and sudden death while parasympathetic activity has protective effect. Low heart rate variability shows sympathetic over activity which is one the powerful and independent risk for malignant arrhythmias and sudden death after acute myocardial infarction [8].

Previous studies referred to autonomic alterations in STEMI patients with very few actually studying the effect of revascularization on the pattern of autonomic modulation. Vagal overactivity is well known to be more frequent in inferior STEMI compared to sympathetic overactivity in anterior STEMI, this can be explained by the preferential distribution of vagal afferents to the inferoposterior wall of the left ventricle. Thus, the effect of revascularization whether by primary PCI or by fibrinolysis is supposed to be associated with different cardiac autonomic patterns of recovery depending on the site of STEMI [9]. Primary PCI offers the gold standard therapy by restoring the flow in the IRA according to the guidelines of treatment of STEMI, while its effect on restoring the normal autonomic modulation pattern remains unclear.

Lotze et al studied HRV changes using time domain method, they showed that inferior STEMI treated with thrombolysis showed an autonomic modulatory pattern characterized by initial vagal hyperactivity then followed by sympathetic predominance within few hours [10].

CONCLUSION:

It is concluded that there is no difference among the heart rate variability indices in different type of MI, age group and gender. Data from this study show two major important results, first, that SA can detect differences in cardiac autonomic modulation after primary PCI as an applicable and noncomplicated method, and second that the pattern of autonomic modulation after revascularization by primary PCI shows a predominant sympathetic activity in inferior STEMI in contrast to a predominantly vagal modulation in anterior STEMI.

REFERENCES:

1. Erdogan A, Coch M, Bilgin M, Parahuleva M, Tillmanns H, Waldecker B, Soydan N. Prognostic value of heart rate variability after acute myocardial infarction in the era of immediate reperfusion. Herzschrittmacherther Elektrophysiol. 2008;19:161–168.
2. Wiliński J, Sondej T, Kusiak A, Wiliński B, Kameczura T, Bacior B, Czarnecka D. Heart rate variability in the course of ST--segment elevation myocardial infarction treated with primary percutaneous transluminal coronary angioplasty in elderly and younger patients. Przegl Lek. 2014;71:61–65.
3. Larosa C, Sgueglia GA, Sestito A, Infusino F, Niccoli G, Lamendola P, Mariani L, Santangeli P, Lombardo A, Crea F, et al. Predictors of impaired heart rate variability and clinical outcome in patients with acute myocardial infarction treated by primary angioplasty. J Cardiovasc Med (Hagerstown) 2008;9:76–80.
4. Brateanu A. Heart rate variability after myocardial infarction: what we know and what we still need to find out. Curr Med Res Opin. 2015;31:1855–1860.
5. Huikuri HV, Raatikainen MJ, Moerch-Joergensen R, Hartikainen J, Virtanen V, Boland J, Anttonen O, Hoest N, Boersma LV, Platou ES, et al. Prediction of fatal or near-fatal cardiac arrhythmia events in patients with depressed left ventricular function after an acute myocardial infarction. Eur Heart J. 2009;30:689–698.
6. Stein PK, Domitrovich PP, Huikuri HV, Kleiger RE. Traditional and nonlinear heart rate variability are each independently associated with mortality after myocardial infarction. J Cardiovasc Electrophysiol. 2005;16:13–20.
7. Steg PG, James SK, Atar D, Badano LP, Blömstrom-Lundqvist C, Borger MA, Di Mario C, Dickstein K, Ducrocq G, Fernandez-Aviles F, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J. 2012;33:2569–2619.
8. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, Bax JJ, Borger MA, Brotons C, Chew DP, et al. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC) Eur Heart J. 2016;37:267–315.
9. Greco C, Cacciatore G, Gulizia M, Martinelli L, Oliva F, Olivari Z, Seccareccia F, Temporelli PL, Urbinati S. [Selection criteria for referral to cardiac rehabilitation centers] Monaldi Arch Chest Dis. 2011;76:1–12.
10. Coviello I, Pinnacchio G, Laurito M, Stazi A, Battipaglia I, Barone L, Mollo R, Russo G, Villano A, Sestito A, et al. Prognostic role of heart rate variability in patients with ST-segment elevation acute myocardial infarction treated by

primary angioplasty. Cardiology. 2013;124:63–70.