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

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ABSTRACT

INTRODUCTION: By the right coronary artery, when the inferior myocardial tissue is supplied, or RCA is injured due to that vessel thrombosis, an inferior wall myocardial infarction (IWMI), inferior ST segment elevation MI, inferior MI, or inferior STEMI, develops. **OBJECTIVES**: The study's main goal is to examine heart rate alterations in patients with anterior and inferior wa ll ST elevation myocardial infarction. **METHODOLOGY**:From January to August 2021, a desc riptive study was done at CMClarkana. The information was gathered from 100 patients of both s exes. Only patients with anterior and inferior wall ST elevation myocardial infarction who receiv ed thrombolytic treatment and were between the ages of 30 and 60 were included in the study.

RESULTS: The information was gathered from 100 male and female patients. NSTEMI patients were older than STEMI patients and had a higher rate of hypertension, prior MI ad coronary revascularization treatments, and clinical symptoms of metabolic syndrome. On initial admission to the coronary care unit, patients with NSTEMI had a higher number of sig nificant coronary stenoses, revascularization was more commonly inadequate, and they came wit h symptoms of heart failure. There is no change in heart rate variability indices between different types of MI, age groups, or genders, according to the findings. **CONCLUSION**:The findings of t his study demonstrate two significant findings: first, SA can detect variations in cardiac autonom ic modulation following primary PCI.

KEY WORDS: Heart Rate, Inferior and Anterior Wall ST Elevation, Myocardial Infarction

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INTRODUCTION

By the right coronary artery, when the inferior myocardial tissue is supplied, or RCA is injured due to that vessel thrombosis, an inferior wall myocardial infarction (IWMI), inferior ST segment elevation MI, inferior MI, or inferior STEMI, develops. An associated posterior wall MI may arise w hen an inferior MI extends to posterior regio ns as well. HRV is a quantifiable characteris tic of cardiac autonomic function that has be en recognised for a long time. The cardiac a utonomic innervation is diverse, resulting in a variety of autonomic modulation patterns. In the case of myocardial infarction, the typi cal pattern of autonomic modulation is disru pted; however, the pattern is not uniform an d varies depending on the infarcted wall or r egion of the heart. Within a few hours of the acute incident, the altered autonomic regulationbegins[1].

In the early hours following an ST segment elevation myocardial infarction (ST EMI), cardiac autonomic regulation is chara cterised by engaged sympathetic and retracte d parasympathetic activity.

It's worth noting that autonomic modulation varies depending on the location of the infar ction, with inferior/posterior/right ventricula r infarctions displaying a stronger vagal/vas odepressive response and anterior infarction s showing a stronger sympathetic response[2].

However, the effect of acute MI treatment, whether by fibrinolysis or primary percutane ous coronary intervention (PCI), on the reco very of the normal pattern of autonomic card iac regulation has not been well investigated [3].

The impact of autonomic regulation on reperfusion injury and arrhythmias such non sustained ventricular tachycardia[4] underscores the importance of this topic. The culprit lesion is frequently more proximal in the LAD or even in the left main coronary artery when an AWMI extends to the septal and lateral regions as well. An extensive anterior myocardial infarction is a significant anterior myocardial infarction [5]. A coronary artery obstruction causes a decrease in perfusion to the inferior wall of the heart, resulting in a myocardial infarction [6].The study's main goal is to examine heart rate alterations in patients with anterior and inferior wall ST elevation myocardial infarction.

METHODOLOGY

From January to August 2021, a descriptive study was done at Chandka Medical College Hospitallarkana.

The information was gathered from 100 patients of both sexes. Only patients with anterior and inferior wall ST elevation myocardial infarction who received thrombolytic treatment and were between the ages of 30 and 60 were included in the study. Patients with autonomic neuropathy, hypothyroidism, diabetes mellitus, renal failure, previous history of MyocardialInfarction, late presentation (after 24 hours), cardiogenic shock, or defined case of valvular heart disease, atrial fibrillation orventricular arrhythmia, second or third degree AV nodal block, frequent PVCs (10/min), trigeminyorbigeminy and cerebrovascular accident were all excluded. Those who needed cardiopulmonary resuscitation during Holter monitoring or had computer processing issues (difficult analysis due to signal artefact, 15% sinus beat, and recording.

Statistical analysis

SPSS version 22.o was used to analyze the data.The mean and standard deviation were used to express all of the data.

RESULTS

The data was gathered from 100 male and femalepatients.NSTEMI patients were older than STEMI patients and had a higher rate of hypertension, prior MI and coronary revascularization treatments, and clinical symptoms of metabolic syndrome. On initial admission to the coronary care unit, patients with NSTEMI had a higher number of significant coronary stenoses, revascularization was more commonly inadequate, and they came with symptoms of heart failure.

Table1: Patients' main characteristics, presented for the entire group as well as for patients with ST-elevation and non-ST-elevation myocardial infarction.

	All patients	**STEMI**	**NSTEMI**	**p1**	**p2**
Age, yr	61.6 ± 11.2	63.4 ± 11.6	65.6 ± 11.3	<0.001	
Previous AMI, n (%)	61 (18)	22 (11)	38 (32)		< 0.001
Previous stroke, n (%)	12 (4)	3 (2)	7 (6)		0.182
Total cholesterol (under treatment),	123.2 ± 27.5	122.6 ± 27.1	126.7 ± 26.8	0.302	
Metabolic syndrome, n (%)	211 (64)	125 (62)	81 (69)		0.012
BMI	25.3 ± 3.6	25.8 ± 3.8	28.3 ± 5.3	0.086	
AMI characteristics					
Anterior, n (%)	172 (54)	139 (67)	34 (29)		< 0.002
Inferior, n (%)	88 (28)	66 (34)	22 (19)		0.003
Other, n (%)	68 (22)	5 (3)	64 (56)		< 0.002
Coronary vessels with critical lesions,	2.06 ± 0.84	1.93 ± 0.83	2.24 ± 0.84	0.001	
Incomplete revascularization, n	153 (45)	86 (42)	63 (55)		0.029
Left ventricle ejection fraction, %	46.7 ± 11.5	48.9 ± 8.9	47.5 ± 13.2	0.221	

Patients with LVEF < 40%, n (%)	84 (24)	42 (22)	43 (34)		0.007
Patient with heart failure at initial admission,	33 (12)	13 (8)	23 (18)		0.001
Time before Holter, d	16.1 ± 9.7	15.4 ± 9.6	17.5 ± 9.7	0.116	
Therapy at time of discharge from hospital (number of cases, %)					
Aspirin	313 (97)	201 (98)	111 (94)		0.468
Clopidogrel	303 (94)	193 (94)	112 (91)		0.457
Warfarin	37 (13)	23 (12)	17 (13)		0.398
β-blocker	291 (88)	187 (92)	102 (86)		0.197
Ca-antagonist	37 (13)	17 (8)	21 (18)		0.024
ACE-inhibitor	266 (83)	182 (88)	83 (72)		0.002
AT-II-antagonist	44 (12)	15 (9)	26 (24)		< 0.003
Statin	315 (97)	202 (98)	112 (95)		0.894
Diuretic(s)	142 (42)	74 (37)	64 (56)		0.003
HRV parameters					
Mean heart rate, bpm	64.2 ± 10.5	68.2 ± 10.4	67.4 ± 9.8	0.017	

DISCUSSION

HRV was accepted as a clinical test after 13 studies found it to be an independent and strong and factor of risk for sudden death and cardiac arrhythmias, and particularly after an acuteMI[7].

After 14 acute MI, a powerful predictor is sympathetic surge of abrupt mortality and malignant arrhythmias but parasympathetic activity is protective. Sympathetic over activity is indicated by low heart rate variability, which is an independent and strong factor of risk for abrupt death and malignant arrhythmias following a heart attack[8].

Previous research has mentioned autonomic abnormalities in STEMI patients, but few have looked at the influence of revascularization on autonomic modulation patterns. Vagal over activity is more common in inferior STEMI than sympathetic over activity in anterior STEMI, which can be explained by vagal afferents preferred distribution to the left ventricle's inferior posterior wall. Thus, depending on the site of STEMI, the effect of revascularization, whether by primary PCI or fibrinolysis, is thought to be related with various cardiac autonomic patterns of recovery [9].

Primary PCI is the gold standard therapy for STEMI, restoring flow in the IRA according to therapeutic recommendations, although its effect on restoring normal autonomic modulation pattern is unknown.

Using the time domain technique, Lotze et colleagues discovered that inferior STEMI patients treated with thrombolysis had an autonomic modulatory pattern defined by initial vagal hyperactivity followed by sympathetic predominance within a few hours [10].

CONCLUSION

There is no change in heart rate variability indices between different types of MI, age groups, or genders, according to the findings. The findings of this study show two major findings: first, that SA can detect differences in cardiac autonomic modulation after primary PCI as a simple and straightforward method, and second, that the pattern of autonomic modulation after primary PCI shows a predominant sympathetic activity in inferior STEMI versus a predominantly vagal modulation in anterior STEMI.

ETHICS APPROVAL: The ERC gave ethical review approval

CONSENT TO PARTICIPATE: written and verbal consent was taken from subjects and next of kin

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CONFLICT OF INTEREST: No competing interest declared.

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