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Original article

Evaluation of 1-minute Heart rate variability during deep breathing as a Prognostic indicator in patients with Acute myocardial infarction

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ABSTRACT

Background and objective- Heart rate variability (HRV) is a noninvasive electrocardiographic marker reflecting the activity of the sympathetic and vagal components of the autonomic nervous system on the sinus node of the heart. Decreased HRV is a risk factor for both arrhythmic and non-arrhythmic deaths following acute myocardial infarction (MI) .Our aim was to find out the utility of heart rate variability (during deep breathing over 1 minute) in predicting end point cardiac events (risk stratification) in patients after an episode of acute myocardial infarction. Material and methods- Bedside heart rate variability (HRV) during deep breathing was assessed in 100 patients after a first episode of myocardial infarction. This test was performed between 6-48 hours after the first attack of myocardial infarction. We defined low HRV as HRV < 10 beats/minute. The patients were followed up over a duration of 3 months to document end point cardiac events. Statistical analysis was done using the student 't' test . Results- Heart rate variability (HRV) < 10 beats/minute was seen in 44 (44%) patients. 16 patients died in the follow up period. Among the 16 patients who died 15 (93.75%) patients had HRV < 10 beats/minute.Conclusion- Heart rate variability (HRV) during deep breathing is a simple, cost effective bedside test in risk stratification of patients who present with myocardial infarction.

1.Introduction:

Heart rate (HR) is not constant but varies considerably [1]. It varies as a result of interplay between many factors including physical and mental stress, exercise, respiration, thermoregulation, blood pressure regulation, and actions of renin-angiotensin system, circadian rhythms and other unknown complex mechanisms. These subtle fluctuations in sinus rhythm known as heart rate variability (HRV) provide an insight to comprehend the autonomic modulation of the heart [2]. Evidence from studies indicates a strong association between compromised autonomic nervous system (decreased vagal activity or increased sympathetic activity), sudden cardiac death and non sudden cardiac death [3].

There are several methods for measuring autonomic nervous system (ANS) activity in patients after myocardial infarction (MI) [4-8]. These include tests of HRV measured by 24 hour Holter monitoring, HRV calculated over a brief period of 2 - 15 minutes and assessment of baro-reflex activity. However these tests are expensive and require Holter monitoring with elaborate analytical systems including sophisticated computerized analysis. Most of these expensive and sophisticated studies have been carried out in the west.

During deep breathing over 1 minute (6 cycles of respiration; 5 second each for expiration and inspiration) changes in the heart rate occur primarily because of alterations of vagal-cardiac activity [9]. This simple bed side test of 1 minute heart rate variability was used by Amos Katz et al [10] to predict death in patients after myocardial infarction.

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Heart rate variability is a noninvasive electrocardiographic marker reflecting the activity of the sympathetic and vagal components of the ANS on the sinus node of the heart [11].With this background we decided to find out the utility of heart rate variability (during deep breathing over 1 minute) in predicting end point cardiac events (risk stratification) in patients after an episode of myocardial infarction . Role of other parameters like QTc interval and Left Ventricular Ejection Fraction (LVEF) as risk stratification tools post myocardial infarction was also assessed.

2.Materials and Methods

This longitudinal study was conducted in the coronary care unit of a tertiary care centre at Mangalore. Patients admitted with a diagnosis of acute MI were asked to participate in the study. Approval of Ethics committee was taken before starting the study. Written informed consent was taken from the patients .Exclusion criteria was : patients who could not perform the deep breathing test, patients with previous attack of MI, patients with other heart disease (Valvular or Cardiomyopathy), patients with unstable angina, hypertensives on beta blockers, patients with atrial flutter or atrial fibrillation, patients with Coronary Artery Bypass graft surgery within 3 months of MI and other diseases like malignancy that were liable to shorten the patient survival time .

Hundred patients were selected and they were subjected to the deep breathing test. Their Lead II ECG was recorded within 6 to 48 hours after a first attack of MI while the patients were lying supine. Before beginning the test patients were taught to breathe at a rate of 6 cycles per minute; 5 seconds for each inhalation and 5 seconds for each exhalation. During each cycle, the examiner raised his hand to signal the start of each inhalation and lowered it to signal the start of each exhalation. Lead II ECG was recorded continuously for 1 minute while the patient was breathing as instructed. The instantaneous heart rate was determined with the help of successive RR intervals using scaled caliper. The variability in the heart rate (HRV) was calculated as the difference between the shortest and the longest RR interval, measured as beats per minute. We defined low HRV as HRV < 10 beats/minute, a 'prolonged' QT c as 0.44 or above and a low LVEF as LVEF < 40 %.

Diagnosis of acute MI was confirmed in all patients with the help of the following criteria;

1. ST elevation of at least 2 mm in 2 pre-cordial leads or 1 mm in 2 limb leads.

2. Chest pain persisting for more than 30 minutes and not relieved by nitrates.

3. Elevated levels of troponin T / CPK-MB.

Other clinical variables such as age and sex, co-morbid factors such as hypertension, diabetes, ischemic heart disease and smoking were noted. Killips staging was documented. Investigations such as CPK-MB, blood sugar, complete lipid profile, and echo-cardiography were performed in all the patients. Drugs that were prescribed to the patients were noted.

HRV was calculated for all these patients using the method as described by Amos Katz et al [10]. They were followed up for a period of 3 months and any end point cardiac event (eg; a second MI, recurrent angina, congestive heart failure and death) were noted.

2.1.Statistical analysis

The collected data was analyzed using SPSS version 11.5. Students't' test was used to evaluate the significance of each of the parameters with respect to the end point cardiac event. A p value of < 0.05 was considered statistically significant.

3.Results

100 patients who had acute MI participated in the study. Patient characteristics are summarized in Table 1. 83 males and 17 females who had acute MI were enrolled. Thrombolysis was done in 52 (52%) patients. Majority of patients belonged to Killips stage 2. HRV < 10 beats/minute was seen in 44 (44%) patients. Left Ventricular Ejection Fraction (LVEF) was < 40 % in 47 (47%) patients. QT c interval was more than 0.44 seconds in 53 (53%) patients. These patients were followed up for a period of 3 months and end point cardiac events were recorded .

16 patients died in the follow up period. Cause of death is depicted in Table 2. Among the 16 patients who died 15 (93.75%) patients had HRV < 10 beats/minute, 11(68.75%) patients had Left Ventricular Ejection Fraction (LVEF) < 40 % and all 16 (100%) patients had QT c interval was more than 0.44 (Table 3). Low HRV, Prolonged QTc, Low left ventricular ejection fraction were significant risk factors for mortality (Table 4) .

Table.1. Demographic and clinical variables of the study subjects (n = 100)

Parameters	Patient characteristics	(n)
Sex	Male	83
	Female	17
Age	> 60	46
	< 60	54
Comorbidity	Diabetes	27
	Hypertension	41
	IHD	26
	Smoking	58
Site of MI	Anterior wall	68
	Inferior wall	32
Thrombolysis	Done	52
	Not done	48
Killips staging	Stage 1	19
	Stage 2	47
	Stage 3	32
	Stage 4	2
LVEF	> 40 %	53
	< 40 %	47
QT c	> 0.44	53
	< 0.44	47
Beta blockers	Administered	52
	Not administered	48
ACE inhibitors*	Administered	67
	Not administered	33

***ACE inhibitors=Angiotensin converting enzyme**

Table 2: Causes of mortality in the study group

Cause of mortality	(n)
Arrhythmia	8
Non arrhythmic (Cardiogenic shock)	7
Extradural Hematoma	1
Total	16

Table 3. Comparison of clinical and epidemiological variables with the outcome among patients with MI

Cause of mortality	Alive (n=84)	Dead (n=16
Male	71	12
Female	13	4
Diabetes	25	2
Hypertension	36	5
IHD*	20	6
Smoking	46	12
ACE inhibitors	54	13
Beta Blockers	45	7
CCB**	7	1
Nitrates	82	14
Thrombolysis	49	3
Killips 1	19	0
Killips 2	43	4
Killips 3	22	10
Killips 4	0	2
< 10 HRV	29	15
< 40 LVEF	36	11
> 0.44 QT c	37	16

*IHD=Ischemic heart disease, ** CCB=Calcium channel blockers

Table 4. Comparison of lab parameters with the outcome among patients with MI

Parameter	Alive (n = 84) Mean ± SD	Dead (n = 16) Mean ± SD	p value
Cholesterol (mg/dl)	219.26 ± 39.32	229.06 ± 31.68	>0.05
CK MB (Units/L)	68.83 ± 57.46	75.5 ± 45.83	>0.05
Triglycerides (mg/dl)	168.61 ± 41.92	168.75 ± 33.03	>0.05
LDL* (mg/dl)	147.87 ± 33.41	144.88 ± 26.49	>0.05
HDL** (mg/dl)	37.38 ± 7.32	34.69 ± 6.01	>0.05
LVEF (%)	41.99 ± 7.56	37.5 ± 6.5	<0.05
QTc (seconds)	0.438 ± 0.055	0.488 ± 0.044	<0.01
HRV (beats/minute)	13.14 ± 5.36	6.75 ± 2.32	< 0.001

*** LDL=Low density lipoprotein,**HDL=High density lipoprotein**

4.Discussion

Low HRV, Prolonged QTc and low left ventricular ejection fraction were significant risk factors for cardiac mortality post MI in our study. Among the 16 patients who died 15 (93.75%) patients had HRV < 10 beats/minute, 11(68.75%) patients had Left Ventricular Ejection Fraction (LVEF) < 40 % and all 16 (100%) patients had QT c interval was more than 0.44.

Evidence suggests that decreased HRV is associated with increased ventricular arrhythmias and mortality [12]. HRV is influenced by variables such as gender and certain medication (e.g. thrombolysis, antiarrhythmic drugs, β-blockers and ACE inhibitors) [13-16] . Heart rate variability has been used in various clinical settings, including diabetes [17], hypertension [18], sudden cardiac death [19], Congestive heart failure [20], and in obstructive sleep apnoea [21].

Patients who survive an episode of acute myocardial infarction (AMI) remain at risk of recurrent cardiac events and sudden cardiac death after discharge, despite optimal medical treatment. In population studies, decreased HRV has had predictive value for mortality among healthy adults. Decreased HRV is a risk factor for both arrhythmic and non-arrhythmic deaths following acute myocardial infarction (MI) .

Wolf et al. [22] showed the association between decreased HRV and increased mortality after myocardial infarction. Kleiger et al. [23] showed that in a large post-MI population decreased HRV predicted mortality independently of other risk factors, which were LVEF, and non sustained ventricular tachycardia.The predictive value of HRV is independent of other factors established for postinfarction risk stratification, such as depressed left ventricular ejection fraction, increased ventricular ectopic activity, and presence of late potentials [24]. For prediction of all-cause mortality, the value of HRV is similar to that of left ventricular ejection fraction, but HRV is superior to left ventricular ejection fraction in predicting arrhythmic events (sudden cardiac death and ventricular tachycardia) [25].

Analysis of HRV consists of a series of measurements of successive RR interval variations of sinus origin. The Task Force of the European Society of Cardiology (ESC) and the North American Society of Pacing and Electrophysiology (NASPE) has defined and established standards of measurement, physiological interpretation and clinical use of HRV [24]. Time domain indices, geometric measures and frequency domain indices constitute nowadays the standard clinically used parameters [11]. Recent studies have shown that other indices of HRV, such as deceleration capacity may be stronger predictors of mortality following AMI than traditional measures of HRV or even LVEF [26].

Previous studies have shown that time and frequency domain measurements of HRV are excellent predictors of death after MI. In this era of sophisticated tests of HRV, Katz et al. showed that a simple 1-minute deep breathing test of HRV in patients after myocardial infarction appeared to be a good predictor for mortality and sudden death .Our results match with the results of Katz et al .

We feel that the measurement of HRV immediately after an acute MI could equip the clinician with a useful tool for risk stratification of his patients. Special priority and intensive monitoring of patients with low HRV needs to be done in order to prevent early arrhythmic deaths and late non - arrhythmic events. Certain interventions such as thrombolysis or PTCA, done early in MI can improve HRV in such patients. The use of therapeutic agents like beta blockers might go a long way in improving HRV and altering the short and long term mortality and morbidity in MI patients with low HRV. This simple bed side HRV test provides an eye opener to the finer regulation of a complex mechanism that controls the human heart.

Our study had some limitations. The deep breathing test could not be used in patients who were on ventilator and patients with severe pulmonary edema. These patients were excluded. The follow up period was short compared to other studies.

The main strength of our study is that we used a inexpensive bed side test as a risk stratification tool in patients who had acute myocardial infarction.

5.Conclusion

HRV during deep breathing is a simple, cost-effective, reliable risk stratification tool in patients with acute MI. HRV in combination with low LVEF/ prolonged QTc or both could possibly provide a sensitive parameter to accurately predict mortality. Early interventions in patients with low HRV could revert the same and improve the prognosis in patients with acute MI.

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