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Comparative evaluation of predictive value of heart rate variability and QT dispersion in patients with acute STEMI underwent pPCI or thrombolysis.

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Abstract

Objectives: This study sought to evaluate prognostic value of HRV and QT dispersion in patients with STEMI treated by pPCI versus thrombolysis and further evaluate prognostic value of HRV in pPCI stratified according to Syntax Score-II.

Methods: 410 consecutive patients admitted with STEMI undergoing pPCI (n=200) or thrombolysis (n=210) within 12 hours of symptom onset. Patients underwent in pPCI further stratified according to syntax score-II score. The primary endpoint was all-cause mortality during hospital stay. Secondary endpoints were all cause mortality and MACE at 30 days.

Results: The HRV were significantly lower in patients underwent thrombolysis in comparison to pPCI and the patients having high SS-II score in pPCI group in comparison to intermediate or low score. The patients in high SS-II score group also had significantly higher risk of MACE and all cause mortality in comparison to intermediate or low SS-II score. The mean QT dispersion was not significantly different between two groups, however QTd were significantly higher in patients died during study period in comparison to patients survived at 30 days.

Conclusions: The SS-II and HRV can predict short-term adverse outcome in patients underwent pPCI. The high SS-II and SDNN less than 40 ms can serve as cut-off value to identifying patients at risk.

Keywords: Heart rate variability, Primary PCI, Syntax Score II.

Abbreviations

HRV- Heart rate variability
QTd- QT dispersion
p-PCI- Primary angioplasty
STEMI- ST-segment elevation myocardial infarction
SS II- Syntax score-II score
AMI- Acute Myocardial Infarction
CPK-MB- Creatine kinase-MB
DAPT- Dual antiplatelets therapy
IRA- Infract related artery
SDNN- SD of all normal – normal beat intervals in 24 h

SDNNi- Mean of the SDs of all 5-min normal – normal intervals in 24 h

PVBs- Premature ventricular beats

Introduction

Cardiovascular diseases have become the leading cause of mortality in world. STEMI comprises approximately 25% to 40% of all patients admitted with acute coronary syndrome, having 6% in-hospital mortality and 18% mortality at one year [1-4]. The mortality in acute phase of AMI associated with electrical or mechanical complications, and related to duration as well as severity of myocardial ischemia. Mechanical complications including ventricular free wall or papillary muscle rupture, pericardial tamponade, and ischemic valvular dysfunction can produce profound hemodynamic derangements or death that may mimic sudden arrhythmic death[5]. The arrhythmic complications result of electrical heterogeneity cause by myocardial ischemia, also due to alteration in autonomic tone and increase intracellular Ca^{2+} create autonomic triggered activity [6].

The AMI induce state of nuero-hormonal stress that impaired cardiac autonomic function and lead to decrease in heart rate variability (HRV) and increase QTd. In past factors such as left ventricular systolic functions, heart rate variability and QT dispersion all shown as useful predictive tool in patients reperfused with thrombolytic drugs to identify patients at higher risk for adverse cardiovascular outcome [7-11] and successful lysis was associated with significant improvement in HRV, with significant lower incidence of ventricular arrhythmias and mortality [12 13]. However, in modern era of pPCI predictive value of HRV largely unknown in STEMI due to routine use of dual antiplatelets, beta-blockers and use of primary PCI resulting early and successful reperfusion of infract related artery (IRA).

The SYNTAX (Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery) score developed as a comprehensive tool to quantified severity coronary disease with respect to their number, location, and complexity of lesion. It was initially developed to evaluate anatomical severity of coronary artery disease in patient with stable coronary artery disease and recently, it is also validated for risk stratification in STEMI patients undergoing primary PCI, and it was also suggested, syntax scores ability can be further improved by combining anatomical score with clinical variables therefore support role of SS-II score for risk stratification in patients undergoing p-PCI.

The study aimed to assess the prognostic value of HRV and QT dispersion in patients with a STEMI treated by pPCI or thrombolytic drugs. Furthermore, HRV stratified according to SS-II score in patients underwent pPCI to evaluate their correlation and their combined value in predicting short-term adverse outcome.

Methods

Study groups- Acute myocardial infarction **(**AMI) was diagnosed in patients presented with prolonged (≥30 min) chest pain and persistent ST-segment elevation (≥1 mm) in two or more ECG leads and was confirmed by typical variations in serum cardiac enzymes and /or regional wall motion abnormality on echocardiography. All the enrolled patients were divided in two groups. The PCI group included 200 consecutive patients of STEMI underwent to primary PCI within 12 hours of onset symptoms. Patients underwent rescue PCI were excluded from study. The thrombolysis group included 210 consecutive patients of STEMI underwent thrombolysis with intravenous thrombolytic drug within 12 hours of symptom onset.

Study protocols

The routine investigations was done at enrolment and CPK-MB with standard 12 lead ECG was done at enrollment, at 90 minutes post primary PCI or post thrombolysis, after 12 hrs, 24 hrs and on day of discharge on ECG strips running at speed of 25 mm/sec and at a setting of 1mv=10mm. For each patient, we use ECG to further categorized STEMI into AWMI or non-AWMI. Total ischemia time also calculated as total time spend between from onset of symptoms to reperfusion.

The patients in PCI group, in addition to aspirin (300 mg), a loading dose of clopidogrel 600 mg or prasugrel 60 mg or ticagrelor 180 mg was administered if the patient was not pretreated. Patients were thereafter maintained on daily dose of aspirin 150 mg and clopidogrel 75 mg or prasugrel 10 mg or ticagrelor 180 mg. The patients in thrombolysis group, loading dose of aspirin (300 mg) and clopidogrel 300 mg, were administrated if the patient was not pretreated and maintain on asprin150 mg and clopidogrel 75 mg[15].

All PCI procedures performed by experienced interventional cardiologists using a femoral or radial approach. Patients undergoing PCI were administered 100 IU/kg heparin during the procedure. The dose of heparin was reduced to 70 IU/kg if a glycoprotein IIb–IIIa inhibitor was used. The use of DAPT, Glycoprotein IIb–IIIa inhibitor, thrombus aspiration, pre or post dilatation and stent selection were left on the operator's discretion. Angiographic success defined as a residual stenosis <20% of the vessel diameter and achieving TIMI flow Grade III[16].

Syntax score

From the angiogram, each coronary lesion causing ≥50% diameter stenosis in a vessel with a caliber ≥1.5 mm was scored to yield the overall anatomical syntax score, as no valid method described to calculate syntax score in primary PCI, in our study if the IRA was total occluded we scored it as an occluded artery of less than 3 months duration.

Syntax Score II consists of anatomical syntax score and six clinical variables (age, creatinine clearance, LVEF, sex, COPD, and peripheral vascular disease), which was calculated with the SS-II online calculator[17]. The SS-II divides into three groups as $SS\text{-}II_{low} \leq 22$, $SS\text{-}II_{intermediate}$ 23-32, $SS\text{-}II_{high} \geq 33$.

QT dispersion

The QT dispersion will be calculated as QTd = QTmax – QTmin, QT interval was measured manually from the onset of QRS complex to the end of T wave. The end of T wave was considered the point of return to the isoelectric line. ECGs in which the QT interval was not measurable in more than 8 leads were excluded from the study. If U waves were present then QT interval was taken from the beginning of QRS complex to the lowest point between T and U waves[18].

For each patient, two independent experienced cardiologists blinded to clinical data computed all angiographic variables involved in the calculation of SS-II and QTd.

Holter monitoring

All patients underwent 24-h ECG Holter recording with in first 24 hours after AMI using three-channel digital Holter recorders (BPL TRAK-48, from BPL medical technologies pvt Ltd). All recordings were analysed using the BPL ECG lab software. HRV was measured over the entire 24 hours both in the time-domain and in the frequency-domain. As a measure of long- and short-term time domain HRV, we obtained the stander deviation (SD) of all normal – normal beat intervals in 24 h (SDNN) and the mean of the SDs of all 5-min normal – normal intervals in 24 h (SDNNi),

respectively.

In the frequency-domain, HRV was assessed in the 0 – 0.5-Hz range of frequencies, using a fast Fourier transform spectral analysis algorithm, with a spectral resolution of 0.0005Hz. Data were analyzed in 10-min epochs throughout 24 h and the results from all epochs were averaged to form a composite spectrum. As a measure of long- and short-term frequency domain HRV parameters, we considered the amplitude value of 0.15-0.40 Hz RR interval changes in the high frequency (HF) and value of 0.04 – 0.15 Hz in the low frequency (LF) spectrum, respectively. LF/HF ratio was calculated that indicates correlation between sympathetic and parasympathetic activity. The arrhythmia indices such as number of premature ventricular beats (PVBs), couplets of PVBs and episodes of non-sustained ventricular tachycardia (defined as ≥ 3 PVBs with a rate ≥ 100 b/min) were also measured.

Patients with cardiac rhythm abnormalities (e.g., atrial fibrillation, pacemaker rhythm, frequent premature supraventricular beats) and/or taking anti-arrhythmic drugs including digoxin, which made unreliable HRV analysis, were excluded from study. The informed consent taken from all participants in both groups at enrolment. Left ventricular ejection fraction (LVEF) was assessed before discharge in all patients by two-dimensional echocardiography, using the Simpson method. The institute ethical committee approved the study.

Study endpoints

The primary endpoint was all-cause mortality during hospital stay. Secondary endpoints were all cause mortality and major adverse cardiac and cerebrovascular events (MACE) defined as a composite of any ACS, repeat revascularization include infarct related or non-infarct related vessel underwent revascularization by PCI or CABG by ischemia driven symptoms and stoke at 30 days.

Statistical analysis

Continuous Data are reported as mean ± SD and were compared by the unpaired Student's t-test for 2 group comparisons or ANOVA for more than 2 groups. Categorical data was expressed as percentage and were compared by the Chi square test or Fischer exact test as applicable. $P < 0.05$ was considered statistically significant. Statistical analyses were performed using SPSS statistical software, version 21.

Results

Our study is a single center prospective trial conducted from October 2015 to July 2017. All patients admitted with acute coronary syndrome in our institute was screened. Consecutive patients of STEMI meet study inclusion criteria enrolled in thrombolysis (n=210) and p-PCI group (n=200). Out of 410 enrolled patients approximately two third were male (M: F ratio 2:1) with mean age in p-PCI group was 55.46 ± 28 years and 56.33 ± 26.62 years in thrombolysis group. In patients enrolled patient in both the groups 60.5% patients had AWMI; 30% had IWMI, and 20 % patients had Killip class II or more at enrollment. The total ischemia time was 354 ± 293.7 minutes in thrombolysis group in compare to 336.2 ± 275.9 minutes in p-PCI group with mean door to balloon time was 73 ±28 minutes. The baseline characteristics including risk factors, uses of DAPT and beta-blockers were comparable in both the groups (table-1).

Heart rate variability assessed by 24 hours Holter monitoring (table-2) was significantly lower in patients underwent thrombolysis in comparison to p-PCI (SDNN 61.64 ± 24.5, vs 47.01 ± 17.03, $p<0.001$ and SDNNi 49.51 ± 23.64 vs 37.76 ± 15.61, $p<0.01$ in pPCI

and thrombolysis group respectively), and the LF/HF ratio was also significantly lower in patients in thrombolysis group (4.65 ± 1.14 in p-PCI vs 3.13 ± 1.32 in thrombolysis; p<0.01). QTd were measured at 0 hours, at 12 hours, at 24 hours tended to be lower in patients underwent p-PCI but the difference was not statically significant.

The patients enrolled in p-PCI group were further stratified according to total SS-II for PCI, and patients with high SS-II score had significantly lowered heart rate variability in comparison to patients in having low or intermediate SS-II scores (table-3). Similarly, heart rate variability was also significantly lower in patients with AWMI (SDNN in AWMI 53.6 ± 22.07 compare to 65.8 ± 20.9 in IWMI; p<0.05) as they have higher syntax score and lower LVEF then patients with IWMI. The total MACE (58% vs 18.6% vs 6%, p = <0.001), all cause as well as cardiac deaths (14.5% vs 4.6% vs none, P<0.05) and QTd were significantly higher in patients with high SS-II score with significant lower HRV in comparison to intermediate and low SS-II score group.

Total thirty patients were died during hospital stay (11 patients in p-PCI group and 19 patients in thrombolysis group) out of them, 19 patients were having refractory cardiogenic shock, eight patients were having severe mitral regurgitation with refractory heart failure, one patient had post thrombolysis intracranial hemorrhage, two patients who underwent pPCI in with intermediate SS-II group were expired due to sepsis and acute pancreatitis with multi-organ failure respectively. All the patients died had significant lower HRV and higher QTd (Table-4) in comparison to patients survive at 30 day.

Discussion

To the best of our knowledge, the relationship between SS-II score and HRV has been not previously evaluated in patients underwent pPCI. In our study we demonstrate heart rate variability is an important prognostic marker of cardiac autonomic function that significantly improve after complete revascularization of infarct related artery achieved by primary PCI in comparison to reperfusion achieved by thrombolytic drugs. The arrhythmia indices such as PVBs was significantly lower in patients underwent pPCI; however it was not associated with decrease in clinical significant episodes of sustain or ill sustain ventricular arrhythmias.

Syntax Score was developed in syntax trial [19](n=1800) and validated in DELTA register [20](n=2891), although role of syntax score was initially not validated in patients undergoing primary PCI as these patients were excluded from original syntax trial. The syntax score in p-PCI first evaluated by Garg et al. [22] (n=807) in prospective study, demonstrated syntax score had significant predictive value for assessing short term clinical outcome in patients with STEMI undergoing primary PCI and study also suggested syntax scores ability can be further improved with inclusion of clinical variables supports role of SS-II in p-PCI. The SS-II score consists of anatomical syntax score and six clinical variables that improve prognostic value of SS-II score over original Syntax Score in predicting mortality after PCI or CABG[21].

Our study shows patients with higher SS-II scores had significantly higher incidence of MACE, cardiac as well as all-cause mortality and associated with significantly lower HRV, which attributed to greater alteration of autonomic functions cause by higher ischemic burden, multivessel involvement and lower left ventricular systolic functions in these group of patients in comparison to low SS-II scores.

The results from previously published studies were variable regarding effect of infarct site on HRV [23-25], in our study HRV was significantly lower in patients enrolled with AWMI in early acute phase as they have higher ischemic burden and higher SS-II score in compare to patients with IWMI.

Larosa et al[26] in a prospective trial evaluated effect of primary percutaneous coronary intervention on ventricular arrhythmias, heart rate variability and failed to find significant improvement in HRV in patients treated by primary PCI, these contrast finding can explain by use of newer generation fibrinlytic agents (either recombinant tissue-type plasminogen activator) and early enrolment of patients within 6 hours on onset of symptoms leading to higher successful lysis in these patients. In contrast, our study, patients were enrolled twice as late (up to 12 hours) in comparison to Larosa et al and majority of patients were thrombolysed with non-fibrin specific thrombolytic agents (84%) that may affected the outcome of reperfusion in our study population. Only 63% of patients underwent thrombolysis had successful ST resolution (≥70%) at 90 minutes suggest partial recanalization in remaining patients producing greater ischemic stress lead to lower HRV in comparison to Larosa et al[26].

The study demonstrate heart rate variability still can serve as important predictive tool to identifying patients at higher risk of fatal or non fatal adverse outcome at 30 days in modern era of pPCI and its predictive value further enhance by with use of SS-II score. In our study all the patients with cardiac deaths were having high SS-II score and SDNN were less than 40 ms, these findings were in accordance of past trial done in thrombolytic era [27] and these cut-off values can served as important tool to identifying patients likely to have short-term adverse outcome. The mean QT dispersion was highest at time of enrollment and progressively decreases over period 24 hours. The mean QT dispersion was not significantly different between p-PCI or thrombolysis group, however QTd were significantly higher in non-survivor compare to survivor patients suggest its role of identifying patients with at higher risk for fatal outcome.

In study by Vaishnav S et al[28], evaluated effect of beta blocker on HRV showed no significant effect of beta-blocker on the distribution of HRV suggest it unlikely to affect our finding.

Limitations

This study is limited by relatively small sample size of the current study reiterates the need to validate these findings in a larger patient cohort. Secondly, currently no method validated of calculating the Syntax score in patients with STEMI undergoing pPCI, in our study we scored total occluded IRA as an occluded artery of less than 3 months duration.

Conclusion: Our study demonstrates predictive value of SS-II and HRV in current era of primary PCI. To the best of our knowledge, the relationship between SS-II and HRV has not previously been investigated. The Syntax Score-II and heart rate variability can predict 30 days adverse outcome. The high SS-II and SDNN less than 40 ms can serve as cut-off value to identifying 30-day adverse outcome in patents underwent p-PCI.

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Legend Tables

Table 1: Baseline clinical characteristics in both the groups of patients.

	Primary PCI (n=200)	Thrombolysis (n=210)	P-value
PVB/24 hours PVB >	221 ± 106	338 ± 219 10%	<0.01
10/hour (%) NSVT	13%	13% 10%	0.65
Sustain VT	11%		0.82
	6%		0.43
Age (years)	55.46 ± 28.4	56.33 ± 26.62	<0.82
Sex Male	69%	66%	0.76
Female	31 %	34 %	
Cardiovascular risk factors-			
Diabetes	30%	32%	0.87
Hypertension	25%	28%	0.74
Tobacco use	83%	87%	0.55
Hyperlipidemia	19%	23%	0.60
F/H CAD	13%	9%	0.49
Clinical profile-SBP, mm Hg	128.6±15.3	131±17.4	0.30 0.20
Heart rate Killip class ≥2	88±29.2	83±26.3	0.59 0.29
Creatinine (mg/dL)Ejection fraction (%)	18%	22%	0.57
	1.1±0.7	1.0±0.65	
	42.3±15.8	41.2±11.9	
Type of STEM AWMI	63%	58 %	0.76
IWMI ± RV/PW MI	28 %	32%	
Others	9%	10%	
Total Ischemia time (min)	336.2 ± 275.9	354 ± 293.7	0.65
Door to balloon time (min)	73 ±28	-	

In-hospital medication	Aspirin	100%	100%	-
	Clopidogrel / prasugrel	100%	98%	0.47
	GP IIb-IIIa inhibitors	58%	-	- -
	Statins	100%	100%	1.0
	Beta blockers	82%	81%	0.84
	ACE inhibitors/ARB	86%	84%	0.67
	Inotropic drugs	12%	15%	0.86
	Diuretics	19%	21%	

Table 2: Ventricular arrhythmias and heart rate variability results in p-PCI

	Primary PCI (n=200)	Thrombolysis group (n=210)	P-value
SDNN, ms	61.64 ± 24.5	47.01 ± 17.03	<0.01(S)
SDNN-i, ms	49.51 ± 23.64	37.76 ± 15.61	<0.01(S)
LF/HF ratio	4.35 ± 2.14	3.23 ± 1.82	<0.01(S)
QT 0 hr	118.9 ± 21.27	123.2 ± 24.14	0.18
QT 12 hr	102.8 ± 19.17	108.1 ± 21.27	0.06
QT 24 hr	89.1 ± 17.5	93 ± 20.54	0.15

Table 3: Heart rate variables and clinical outcome according to Syntax Score-II

HRV	LOW SS-II (n=66)	INTERMEDIATE SS-II (N=86)	HIGH S-II (N=48)	P value
SDNN, ms	70.01±26.63	59.78 ± 22.77	43.72 ± 15.84	< 0.01(S)
SDNN-i, ms	57.1 ± 17.28	51.01 ± 22.22	36.26 ± 15.13	< 0.01(S)
LF/HF	4.97 ± 1.538	4.18.062 ± 1.88	2.764 ± 1.968	0.034(S)
All-cause mortality	0	4(4.6%)	7(14.5%)	0.031(S)
Cardiac death	0	2(2.3%)	7(14.5%)	0.023(S)
Repeat revascularization	2(3%)	8(9.3%)	14(29.1%)	0.018(S)
Acute coronary syndrome		4(4.6%)	6(12.5%)	0.020(S)
Re-hospitalization	4(6%)	8(9.3%)	14(29.1%)	0.021(S)
MACE	4(6%)	16(18.6%)	18(58%)	< 0.01(S)

Table 4: heart variable and QTd in survivor and non-survivors

HRV	Survived (N=380)	Death (N=30)	P value
SDNN, ms	59.53 ± 22.33	36.22 ± 8.78	0.012(S)
SDNN-i, ms	48.71 ± 21.35	30.96 ± 4.48	0.023(S)
LF/HF	4.846 ± 1.819	2.814 ± 1.19	0.018(S)
QT 0 hr	113.9 ± 19.2	143.2 ± 21.1	0.019(S)

QT 12 hr	102.8 ± 18.1	128.1 ± 18.2	0.021(S)
QT 24 hr	89.1 ± 15.5 1	104 ± 20.54	<0.034(S)