MEASUREMENT OF QRS AND QTC DURATION AND DISPERSION PREDICTS VENTRICULAR ARRHYTHMIAS IN THE EARLY STAGE OF ACUTE MYOCARDIAL INFRACTION

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ABSTRACT

Background

Myocardial infarction is one of the most common causes of mortality in middle and older age groups, especially in the presence of a ventricular arrhythmia.

Objectives

To determine the relationship between QRS and QTc duration and dispersion with the occurrence of ventriculricular arrhythmia in early stage of myocardial infarction.

Methods

This prospective study of 100 patients (72 males) admitted to Slemani Cardiac Hospital, Sulaimani, Kurdistan Region -Iraq with ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention. Electrocardiograms on admission and one day later were evaluated for the duration and dispersion of corrected QT interval (QTc) and QRS.

Results

Mean QTc was 453±35 ms on admission and 440±31.3 ms one day later. Mean QTc dispersion (QTC) was 69±18.4 ms on admission and 49.8±15 ms one day later. Mean QRS duration was 76.8±12.8 ms on admission and 70±10.7 ms one day later, while mean QRS dispersion (d QRS) was 29.4±14.7 ms on admission and 18.8±17.5 ms one day later. There was a significant correlation between ventricular arrhythmia and QRS, dQRS, QTc, and dQTc ≥ 60ms.

Conclusion

Patients with the increased value of corrected QT dispersion, QRS duration, and dispersion in the early stage of STEMI have a greater likelihood of developing ventricular arrhythmia.

Keywords: *Measurement of QRS, QTc Duration, Dispersion Predicts Ventricular Arrhythmias, Acute Myocardial Infraction.*

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INTRODUCTION

Myocardial infarction (MI) is one of the most common causes of mortality in the middle and older age group. [1]. The death rate has decreased in recent years since the invention of antiplatelet, β-blockers, and statins, which also reduced adverse events and the rate of recurrence [2]. The short and long-term mortality in patients with acute MI is significantly higher in the presence of ventricular tachycardia (VT) or ventricular fibrillation (VF) in the peri-infarct time [3].

In the early stage of acute coronary syndrome, arrhythmia usually represents polymorphic VT or VF, which occur in a minority of patients with acute myocardial ischemia. They usually have a genetic predisposition [4]. Numerous changes occur during acute myocardial ischemia, a precursor for ventricular arrhythmia. Anaerobic glycolysis leads to acidosis and a decrease in adenosine triphosphate (ATP) while extracellular potassium (K+) and lysophosphatidylcholine builds-up. This, in turn, leads to ionic imbalance, and shorter action potential period, and a change in resting membrane potential together with mishandling of intracellular calcium (Ca2+) [5].

Around 50% of acute MI-related deaths occur within the first hour of the event, mostly due to arrhythmias, while late deaths are attributed to cardiac rupture, electromechanical dissociation, cardiogenic shock, etc.[6]. Recognition and understanding of changes happening during acute ischemia is crucial to prevent complications. Furthermore, several investigators suggest that alterations in the level and quality of cardiac autonomic control will determine the early and long-term prognosis of the patient after cute MI [7].

Repolarization inhomogeneity in the cardiac electrical system is expressed by QTc dispersion (dQTc). QTc dispersion is the difference between the maximum and minimum corrected QT interval in different leads. In contrast, adjacent QTc dispersion is the maximum difference in QTc interval between two adjacent leads.

The latter represents an abnormality in regional repolarization and refractoriness, and hence it is considered the main factor for the occurrence of reentrant ventricular tachyarrhythmia [8].

The difference between the maximum and minimum QRS duration in standard 12-lead ECG is called QRS dispersion. QRS dispersion has been proposed as a marker of inhomogeneous depolarization of the ventricle and, if increased, could be associated with a negative prognosis [9].

This study aims to assess the relationship of occurrence of arrhythmia in the early stage of STEMI and specific electrocardiographic measurements such as the duration and dispersion of QRS complex and corrected QT interval in a cohort of patients admitted and treated in Slemani Cardiac Hospital (SCH), Sulaymaniyah, Kurdistan-Iraq given the relevant literature.

PATIENTS AND METHODS

A total of 100 patients with STEMI admitted to SCH over two months were enrolled in this prospective study. Complete history and clinical examination were performed. In addition, their electrocardiogram (done by GE MAC 1600 machine) on admission and 24 hours post primary percutaneous coronary intervention were evaluated for corrected (QTc) and QT dispersion (dQTc) together with QRS duration and dispersion (dQRS) by one investigator.

In this study, QT interval was defined as the duration from the beginning of the QRS complex to the end of the T wave at its return to the isoelectric line or the nadir between T and U waves calculated in all leads, and its average was expressed in ms [10]. QTc dispersion was defined as the difference between the maximum and minimum QTc in 12 lead ECG [9]. It was calculated by Bazett's formula from all leads, and the average was expressed in MS [11] .QRS duration was defined as the time between the initiations of the Q waves or R waves till the end of the R waves or S waves and expressed in MS [12], while QRS dispersion referred to the difference between the maximum and minimum QRS duration of the 12 lead ECG and expressed in MS[.9].

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 25. Descriptive statistics (mean, standard deviations, frequencies, and percentages) were calculated for demographic and health-related factors. In addition, Chi-Square and T-tests were used to find out the significant correlation between different data pairs.

This study protocol was approved for its ethical consideration by Kurdistan Board for Medical Specialties, Directorate of Training Affairs, Ethical and Scientific Research Units. Informed consent was obtained from all participants in the study.

RESULTS

All 100 participants completed the study with no missed cases; the age ranged from 22 to 90 years with a mean of 59.8 ±12. There were 72 males and 34 smokers. There was a significant correlation between ventricular arrhythmia and QRS, dQRS, QTc, and dQTc ≥ 60ms.

Table 1. Shows the demographic characteristics of the participants.

Parameter	Value
Mean age ± SD (years)	59.8 ± 12
Age range (years)	22-90
20-30 year	1
31-40 year	3
41-50 year	15
51-60 year	30
> 60 year	51
Total	100
Male	72
Female	28
Smokers	34

Table 2. Shows the clinical characteristics of STEMI patients.

Parameter		Value
Symptom	Chest pain	91
	Epigastric pain	6
	SOB	3
Time of presentation	< 4 hours	68
	>4 hours	32
Comorbidities	Hypertension	64
	DM	29
	IHD	11
Vital signs	Mean SBP	138.25±30.5
	Mean DBP	83.46±20
	Mean HR	95.31± 25.8 bmp
Location of AMI	Anterior	46
	Inferior	42
	Lateral	12
ECG findings	Ventricular Arrhythmia	11
	Admission QRS and dQRS	76.8±12.8 and 29.4±14.7ms
	Admission QTc and dQTc	453.3±35ms and 69±18.4ms
	Admission QTc ≥ 60 ms	73
	2nd-day QTc ≥ 60 ms	34
	2nd-day QTc and dQTc	440±31.3 and 49.8±15
	2nd-day QRS and dQRS	70±10.7 and 18.8±7.5
Heart rate	Normal	23
	Tachycardia	65
	Bradycardia	12
EF%	≥ 50	69
	40-49	23
	≤ 40	8
Number of diseased vessels	1	57
	2	39
	3	4
Culprit's vessel	LAD	46
	RCA	39
	LCX	15

Table 3. Correlates different variables with ventricular arrhythmias.

Parameter		Yes	No	P-value.
Male		10	62	0.127
Female		1	27	
Mean age		53.82	60.62	0.07
Smokers		8	26	0.07
Non-smokers		3	63	
Hypertension		6	58	0.3
		3	31	
DM		1	28	0.1
Non-diabetics		10	61	
IHD		2	9	0.1
No IHD		9	80	
Mean HR		102.45±20.8	94.4±26.4	0.063
Anterior AMI		7	39	0.4
Inferior AMI		3	39	
Lateral AMI		1	11	
Number of vessels	1 vessel	7	50	0.3
	2 vessel	3	36	
	3 vessels	1	3	
Culprit's vessels	LAD	7	39	0.3
	LCX	2	13	
	RCA	2	37	
QTc		471.27 ± 36.6ms	451±34.4ms	0.07
D QTc		94.82±17.7ms	65.97±15.9ms	0.001
QRS		91.55±5.3ms	75±12ms	0.001
d QRS		46.8±18.7ms	27.2±12.6ms	0.001
2nd-day QTc		446.18±21ms	439.2±32ms	0.001
2nd-day d QTc		66.7±11.64ms	47.7±14ms	0.001
2nd-day QRS		81±10ms	68.7±10ms	0.001
2nd-day d QRS		26.8±9.5ms	17.8±6.6ms	0.001
Admission dQTc≥60ms		11	62	0.02
2nd-day dQTc≥60ms		9	25	0.01

DISCUSSION

Cardiovascular diseases are among the leading causes of mortality globally [13]. Despite a remarkable decrease in the incidence and prevalence of cardiovascular diseases in the past three decades in the western world, there is a significant rise in the developing world. Annually, cardiovascular diseases kill approximately 17.5 million people, of which 80% are from low or mid-income countries [14].

In the current study, the age of patients with ventricular arrhythmia was younger than those without ventricular arrhythmia (53.82 vs. 60.62 years), which is similar to the finding of Rahimi et al.[15] but opposite to what is found in another study [9].

In this study, there was no significant relationship between the sex of patients, smoking, hypertension, diabetes mellitus, and the risk of developing ventricular arrhythmias.

The Physiopathology of ECG alterations during acute MI responsible for repolarization and depolarization defects and ventricular arrhythmia has been explained by several authors [16-19].

Recently, many studies have focused on ECG parameters during acute MI. A study [20] stated that QRS fragmentation during AMI is associated with a higher rate of ventricular arrhythmia in the short term. Likewise, Bayés de Luna and Elosua[21],documented that the QRS width is one of the ECG parameters closely related to sudden death during AMI and Hetland et al.[22] showed a significantly broader QRS in patients with ventricular arrhythmias 40 days after AMI. On the other side, a relatively large study involving 724 patients documented no correlation of ORS duration and dispersion with arrhythmic events or QRS dispersion with death during follow-up [23].

In this study, we found that total QRS duration was significantly associated with the occurrence of ventricular arrhythmia (91.55 ± 5.3 ms for ventricular arrhythmia vs. 75 ± 12 ms for no arrhythmia; p-value 0.001). In addition, we also found a significant association of QRS dispersion with the occurrence of ventricular arrhythmia (46.8 ± 18.7 ms for ventricular arrhythmia vs. 27.2 ± 12.6 ms; p-value 0.001); relatively similar findings were recorded in one study [9] (QRS duration 95.2 ± 17.9 ms for ventricular arrhythmia vs. 72.3 ± 5.6 ms for no arrhythmia; p<0.001), QRS dispersion 42.1 ± 24.5 ms for ventricular arrhythmia vs. 18.9 ± 7.8 ms for no arrhythmia; p-value 0.001) and also comparable with what is found by Perkiömäki and his coworker [24] (dQRS of induced VT in IHD group was 48 ± 16 ms vs. 28 ± 11 ms in healthy subjects without VT).

Higher QTc dispersion was proved in many studies as a factor to increase the liability of ventricular arrhythmia. [25- 26] In contrast, Leitch and his colleagues[27],rejected this finding, and Fiol[28] and his friends also rejected the ability of QT dispersion to predict ventricular fibrillation in AMI. Higham and his coworkers in two separate studies [29, 30] have documented that QT dispersion increases from the first moment of ischemic insult and decreases over time. In this study, we found that there was a significant association between QTc dispersion at admission and on the second day with the development of ventricular arrhythmia. (d QTc was 94.82 ± 17.7 ms in the presence of ventricular arrhythmia while it was 65.97 ± 15.9 ms in the absence of ventricular arrhythmia; p-value = 0.001). This finding was consistent with a previous study [9], (91.9 ± 23.3 ms vs. 62.5 ± 31.3 ms; p-value 0.001). Fahad et al. [31], reported higher dispersion values but with the same association and ratio (164 ± 10.4 ms in the arrhythmia group vs. 119.1 ± 18.6 ms in a non- arrhythmic group). They have also confirmed that QTc dispersion will decrease with time (125 ± 22.9 ms on the first day, 110 ± 22.6 on the second day, and 94.3 ± 16.7 ms after seven days). These findings were also observed in our study as the mean QTc dispersion on admission was 69 ± 18.4 ms and decreased to 49.8 ± 15 ms on the 2nd day .

Previous studies showed that LAD artery and anterior myocardial infarctions were the most culprit vessels and sites in generating ventricular arrhythmia inpatient with ACS. [32,33], similarly, our study showed the same result in which 63% of patients with ventricular arrhythmia had an anterior myocardial infarction in which the culprit's vessels were LAD, which can be explained by its larger size infarction, more ischemic burden, and ventricular dysfunction. Besides this, the number of diseased vessels is an important indicator for developing ventricular arrhythmia in many studies, as low as 37.5% [34]. As high as 55% [35] were recorded, we found that 37% of ventricular arrhythmia were among patients with more than two vessels disease.

In conclusion; corrected QT dispersion and QRS duration and dispersion at the time of admission is a predictor of the liability to get early ventricular arrhythmias in an inpatient with ST-elevation myocardial infarction.

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