Research Article

QRS Duration and Spatial Dispersion in Non-St Elevation Myocardial Infarction: Correlation with Angiographic Anatomy and in-Hospital Outcome

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Abstract

Background: 40% of patients with non-ST elevation myocardial infarction (NSTEMI) have multi-vessel disease with 11-13% rate of in-hospital emergent bypass surgery. So, rapid triage is critical for optimal management. We aimed to evaluate correlation between QRS duration/dispersion at emergency room and angiographic anatomy as well as in-hospital outcome. **Methods:** 192 patients with NSTEMI were included. QRS duration and dispersion at admission were measured. SYNTAX score (SXscore) was calculated for each patient. **Results:** SXscore was significantly and positively correlated with age, admission heart rate (HR), GRACE score, peak HsTnT level, QRS duration and QRS dispersion. By multivariate logistic regression, male gender (OR 5.042, 95% CI 1.633 –15.567, P <0.005), admission HR (OR 1.088, 95% CI 1.024 –1.157, P= 0.007) and QRS dispersion (OR 1.020, 95% CI 1.003 –1.037, P= 0.018) were independent predictors of SXscore > 33. QRS dispersion > 10 ms predicted SXscore > 33 with a sensitivity and specificity of 45.8% and 70.5% respectively (AUC 0.658, 95% CI 0.554-0.753, P= 0.0029). Patients with QRS dispersion > 10ms had significantly higher incidence of Killip class > 1 (P< 0.001), GRACE score (P< 0.001), more incidence of systolic (P=0.025)/ severe diastolic left ventricular dysfunction (P= 0.03), in-hospital recurrent ischemia (P= 0.003), and serious ventricular arrhythmias (P= 0.01). **Conclusion:** In NSTEMI, SXscore was significantly and positively correlated with QRS duration and dispersion; the latter being independent predictor of Sxscore >33. In addition, QRS dispersion >10 was associated with in-hospital Killip class >1, systolic/diastolic left ventricular dysfunction, recurrent ischemia, serious ventricular arrhythmias.

Key words: QRS duration; QRS dispersion; non-ST elevation myocardial infarction

Introduction

More than 40% of patients with an NSTEMI have multi-vessel disease[1] with the rate of in-hospital emergent bypass surgery ranging from 11-13 %[2]. Therefore, NSTEMI patients should be rapidly risk-stratified. This stratification gauges not only the timing of invasive strategy[3] and subsequent possibility of surgical revascularization but also the level of care (intermediate versus intensive)[4]. Many scoring systems (e.g. Global Registry of Acute Cardiac Events (GRACE) score) are available but there is still a clinical need for rather simple and rapid scoring to be applied[5].

QRS duration and dispersion, defined as the difference between maximum and minimum QRS durations in standard 12-lead ECG, are simple electrocardiographic markers with potential value in many clinical conditions [6-12]. In experimental acute coronary occlusion, Elmberg et al[13] demonstrated that the difference between QRS durations in leads with maximum and minimum ST segment deviation was probably correlated with severity of ischemia. Chavez-Gonzalez et al[14] demonstrated that increased QRS dispersion was associated with a greater likelihood of ventricular arrhythmias in early stages of acute myocardial infarction.

Due to scarcity of data on QRS measurements in NSTEMI, we conducted this study to investigate the correlation between QRS duration/dispersion and angiographic anatomy as well as in-hospital outcome in NSTEMI patients.

Patients and Methods

We recruited patients presenting to the emergency room with suspected non-ST elevation ACS. After cardiac enzyme assay, 192 patients with guideline-defined NSTEMI

were included in the study. Written informed consent was obtained from all patients.

Exclusion criteria:

- Previous myocardial infarction or revascularization.
- Patients with QRS duration > 120 ms or paced rhythm.
- Patients taking medications that could affect QRS interval such as amiodarone and digitalis,
- Patients with hypo- or hyperkalemia,
- Patients with cerebrovascular disease or significant renal impairment

History taking and clinical examination. With emphasis patients' hemodynamics. Killip class [15] and GRACE score [16] were assessed as previously described.

Electrocardiography (ECG):

After the diagnosis of NSTEMI was confirmed in the emergency room, a 12-lead ECG was done in accordance with American heart association (AHA) specifications[17] with paper speed set at 50 mm/sec and voltage gain of 10 mm/mV. ECG papers were scanned and analyzed using digital image processing software (http://imagej.nih.gov/ij/). ECGs with signal noise, wandering baseline, frequent ventricular premature beats were excluded from analysis. QRS and corrected QT (QTc) durations were measured as previously described [18,19] by 2 independent observers blinded to patients' clinical and angiographic data.

QRS dispersion (in ms) was calculated as the difference between the maximum QRS duration and the minimum QRS duration of the 12 leads. Interobserver variability calculated using Bland-Altman blot was non-significant (p value 0.50 for QRS maximum duration and 0.54 for QRS minimum duration).

Laboratory work up

High sensitive-Troponin T (HsTnT) was measured on admission and serially every 6 hours to determine the peak level. A 20% or greater elevation of HsTnT level from the previous sample is considered reinfarction when associated with chest pain with or without dynamic ECG changes[20]. Creatinine and serum Na and K levels were measured at admission.

Trans–thoracic echocardiography (TTE)

TTE was done within the first 24 hours of admission. Dimensions of left ventricle at end-diastole (LVEDD) and end-systole (LVESD) as well as LV ejection fraction (LVEF) using modified Simpson's method were measured in accordance with recommendations of the American Society of Echocardiography. The ratio of trans-mitral E peak velocity to tissue-Doppler derived E' peak velocity at lateral mitral annulus (E/E' ratio) was calculated as an index of diastolic function. Severe diastolic dysfunction was defined as an E/E' ratio >15 [21].

Coronary angiography (CAG):

All patients underwent CAG (the timing and method of revascularization were left to the discretion of the attending physician) using Judkins technique. Left and right systems were evaluated in at least 4 and 2 standard angiographic views respectively. SYNTAX score (SXscore) was used to evaluate the severity of CAD. Using the algorithm described on www.syntaxscore.com, each lesion with ≥ 50% diameter stenosis in vessels ≥ 1.5 mm was scored separately and added together to provide the overall SXscore. SXscore > 33 was used as an indicator of severe CAD. Scoring was done by 2 interventionists blinded to the study.

Statistical Analysis

SPSS 17.0 (SPSS Inc., Chicago, IL, USA) was used. Continuous variables were presented as mean±SD while categorical variables were presented as percentages. Correlation between SXscore and other variables was done using Spearman's test. Multivariate logistic regression analysis was performed for potential independent predictors of SXscore > 33. Receiver operating characteristic (ROC) curve analysis was performed for variables with significant correlation. According to calculated cut-off value of QRS dispersion, we divided patients into 2 groups. Data of both groups were compared by Mann Whitney U test (for continuous variables) and Chi-square test (for categorical variables). P values < 0.05 were considered statistically significant

Results

Baseline characteristics

Our study included 192 patients (126 males with mean age of 57.4±6.8 years). Hypertension was the most common CAD risk factor

(45.8%). Means of QRS duration, QRS dispersion and QTc dispersion were 92.6±14.2 ms, 19.9±8.6ms, and 37.1±12.6ms; respectively. On angiography, mean SXscore 24.5± 8.6. **Table (1)** summarizes baseline characteristics of the study group.

Table 1: baseline characteristics of the study group

Variable	Value (%)
Age (years)	57.4 ± 6.8
Male Gender	126 (65.6%)
Hypertension	88 (45.8%)
Diabetes Mellitus	48 (25%)
Dyslipidemia	40 (20.8%)
Smoking	80 (41.7%)
Admission HR (bpm)	79 ± 16
Grace score (points)	102.6 ± 18.1
Killip class > I	80 (41.7%)
Creatinine (mg/dL)	1.15 ± 0.38
Na level (mmol/L)	139.3 ± 3.0
K level (mmol/L)	3.6 ± 0.33
Peak HsTnT (ng/L)	268.4 ± 224.0
QRS duration (ms)	92.6±14.2
QRS dispersion (ms)	19.9±8.6
QTc dispersion (ms)	37.1 ± 12.6
E/E' > 15	72 (37.5%)
LVEDD (mm)	53.2 ± 9.7
LVESD (mm)	36.4 ± 10.4
LVEF (%)	58.8 ± 11.7
SXscore (points)	24.5 ± 8.6

Correlation between SXscore and other variables

Using Spearman's rank correlation, we found highly significant positive correlations between SXscore and admission HR as well as peak HsTnT level. Significant positive correlations between SXscore and age, QRS duration, QRS dispersion, LVESD as well as Grace score were found. On the other hand, significant negative correlation between SXscore and LVEF was found **(table 2)**.

Table (2): Correlation between SXscore and other variables

Variable	r value	P value
Age (years)	0.262	**0.015**
Admission HR (bpm)	0.541	**< 0.001**
Grace score	0.302	**0.003**
QRS duration (ms)	0.341	**0.001**
QRS dispersion (ms)	0.248	**0.015**
QTc dispersion (ms)	-0.065	0.529
LVEDD (mm)	0.088	0.393
LVESD (mm)	0.252	**0.013**
LVEF (%)	-0.304	**0.003**
Creatinine (mg/dL)	-0.003	0.980
Peak HsTnT level	0.523	**<0.001**

Multivariate logistic regression

Taking SXscore > 33 as an indicator of severe CAD, multivariate logistic analysis demonstrated that male gender (OR 5.042, 95% CI 1.633 –15.567, P <0.005), admission HR (OR 1.088, 95% CI 1.024 –1.157, P= 0.007) and QRS dispersion (OR 1.020, 95% CI 1.003 – 1.037, P= 0.018) were independent predictors of SXscore > 33 **(table 3)**.

Table 3: Multivariate logistic regression for prediction of SXscore > 33

SXscore > 33	Coefficient	Standard error	Wald	Degree of freedom	P	OR	95% CI	
							Lower	upper
Age (yrs)	0.048	0.041	1.368	1	0.242	0.953	0.878	1.033
Male gender	1.618	0.575	7.912	1	**0.005**	5.042	1.633	15.567
HR (bpm)	0.085	0.031	7.355	1	**0.007**	1.088	1.024	1.157
Grace score	0.961	1004.834	0.000	1	0.999	2.615	0.000	.
QRS duration (ms)	0.044	0.492	0.925	1	0.702	0.915	0.909	0.982
QRS dispersion (ms)	0.020	0.008	5.576	1	**0.018**	1.020	1.003	1.037
LVESD (mm)	0.005	0.070	0.005	1	0.942	1.005	0.877	1.152
LVEF (%)	-0.029	0.062	0.217	1	0.641	0.972	0.861	1.096

ROC curve analyses for prediction of SXscore > 33

ROC analyses were done for variables showing significant correlation with SXscore **(table 4)**.

Table 4: ROC curve analyses for prediction of SXscore > 33.

Variables	SN % (95% CI)	SP % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	AUC (95% CI)	p-value
Age > 61 years	66.67% (51.6 – 78.6)	33.33% (20.4 – 48.4)	50% (37.2 – 62.8)	50% (31.9 – 68.1)	0.618 (0.606–0.821)	0.0495
Admission HR >80 (bpm)	75% (60.4 – 86.4)	75% (60.4 – 86.4)	75% (60.4 – 86.4)	75% (60.4 – 86.4)	0.736 (0.513–0.755)	<0.001
QRS duration >100 ms	100% (92.6 – 100)	8.33% (2.3 – 20)	52.2% (41.5 – 62.7)	100% (39.8 – 100)	0.542 (0.437–0.644)	0.0387
QRS dispersion >10 ms	45.8% (31.4 – 60.8)	70.5% (74.8 – 95.3)	78.6 % (59.0 – 91.7)	61.8 % (49.2 – 73.3)	0.658 (0.554–0.752)	0.0029
LVESD > 45 mm	83.33% (69.8 – 92.5)	58.33% (43.2 – 72.4)	66.7% (53.3 – 78.3)	77.8% (60.8 – 89.9)	0.660 (0.556–0.753)	0.0056
LVEF ≤ 40 %	83.33% (69.8 – 92.5)	58.33% (43.2 – 72.4)	66.7 % (53.3 – 78.3)	77.8 % (60.8 – 89.9)	0.663 (0.560–0.756)	0.0052
Peak HsTnT > 1105	94% (89.6 – 100)	79.33% (70.5 – 87.2)	72.9% (63.4 – 86.5)	90% (78.6 – 98)	0.566 (0.481–0.702)	0.0358
Grace Score > 112	80% (71.4 – 89.4)	73% (61.6 – 85.4)	79% (64.4 – 88.9)	76% (63.1–85.3)	0.710 (0.513–0.715)	<0.001

Comparison between groups

According to the calculated cut-off value for QRS dispersion, we divided the study population into 2 groups: the first with QRS dispersion ≤ 10 ms (136 patients) and the second with QRS dispersion > 10 ms (56 patients). Higher incidence of Killip class > 1 as well as LV diastolic and systolic dysfunction was observed in the group with QRS dispersion > 10 msec. Biomarkers of myocardial injury, GRACE score, in-hospital recurrent ischemia, and serious ventricular arrhythmia were significantly higher in the group with QRS dispersion > 10 ms (table 5).

Table (5): Comparison between groups

Variables	QRS dispersion ≤10 136 (70.8%)	QRS dispersion >10 56 (29.2%)	p-value
Age (yrs)	57.7 ± 7.1	56.5 ± 5.9	0.662
Male Gender	86 (63.2%)	38 (67.9%)	0.24
Hypertension	60 (44.1%)	28 (50%)	0.559
Diabetes Mellitus	32 (23.5%)	16 (28.6%)	0.604
Dyslipidemia	28 (20.6%)	12 (21.4%)	0.512
Smoking	54 (39.7%)	26 (46.4%)	0.544
HR (bpm)	77.4 ± 9.6	80.2 ± 19.9	0.952
Killip class > I	48 (35.3%)	32 (57.1%)	**<0.001**
Grace score (points)	87.7 ± 10.8	117.5 ± 9.4	**<0.001**
Peak HsTnT level	802.3 ± 238.5	1368.5 ± 391.0	**0.02**
Creatinine (mg/dL)	1.14 ± 0.39	1.15 ± 0.37	1.00
Na level (mmol/L)	139.4 ± 1.9	139.1 ± 3.9	0.685
K level (mmol/L)	3.6 ± 0.28	3.7 ± 0.36	0.05
E/E' >15	42 (31%)	30 (54%)	**0.03**
LVEDD (mm)	51.1 ± 8.6	55.3 ± 10.3	**0.029**
LVESD (mm)	34.3 ± 8.8	38.4 ± 11.5	0.318
LVEF (%)	62.3 ± 9.4	55.3 ± 12.8	**0.025**
SXscore (points)	10.4 ± 6.9	23.0 ± 8.3	**0.001**
≥ 2 angina episodes	31 (23.5 %)	34 (60.7 %)	**0.003**
Reinfarction	2 (1.5 %)	2 (3.6 %)	0.07
Serious ventricular arrhythmia	4 (2.9 %)	6 (10.7 %)	**0.01**
Sudden cardiac death	4 (2.9 %)	2 (3.6 %)	0.063

Discussion

To the best of our knowledge, this is the first study correlating QRS dispersion with angiographic anatomy and in-hospital outcome in NSTEMI patients. The main findings in our study are:

1. Higher SXscores are associated with longer QRS duration and QRS dispersion; the latter being independent predictor of SXscore > 33.
2. Patients with QRS dispersion > 10 ms had higher incidence of Killip class > 1 as well as LV systolic and diastolic dysfunction.
3. Patients with QRS dispersion > 10 ms had significantly higher incidence of in-hospital recurrent angina episodes and serious ventricular arrhythmias.

Correlation between QRS duration and dispersion and severity of ischemia:

In our study, we found that QRS duration and dispersion showed significant positive correlations with SXscore. Significantly higher HsTnT peak levels were also found in patients with QRS dispersion > 10 ms.

Recently, Hussien et al.,[22] studied 150 patients with NSTE-ACS and found that patients with left main or three-vessel disease were associated with longer QRS duration.

Following ischemia, electrical conduction velocity in the ischemic myocardium initially increases during the first 2 minutes then subsequently decreases causing delayed activation of the ischemic tissues. In addition, there is a decrease in resting membrane potential and action potential magnitude and upstroke velocity [23]. Possible reasons for such changes are enhanced outward potassium currents and inward sodium and calcium currents. These ionic changes decrease both excitability and conductivity in purkinje fibers producing what is called peri-ischemic block[24]. Tsukahara et al.,[25] reported that such changes might be rapidly reversed by successful reperfusion.

Our finding on the positive correlation between QRS dispersion and SXscore in NSTEMI is fairly unique and is not previously reported.

Association between QRS dispersion and Killip class >1 as well as LV systolic and diastolic dysfunction in NSTEMI:

In our study, we found statistically significant association between QRS dispersion and left ventricular systolic/diastolic function.

This finding is partly supported with numerous studies correlating QRS duration to presence and severity of acute heart failure in ACS. Shah et al.,[26] studied 1544 NSTEMI patients and found that patients with a QRS duration >90 ms had lower ejection fraction at the time of discharge (46±15% vs. 51±13%; p<0.001). Furthermore, on follow up, a QRS duration < 90ms predicted a marginal improvement in LVEF (1.27±10%; p=0.049) in contrast to patients with QRS duration > 90ms where no improvement was seen (0.05±11%; p=0.937). Similar results were found in STEMI patients[27].

The correlation between prolongation of QRS duration and left ventricular failure in NSTEMI has many explanations:

1. QRS prolongation is more common with left ventricular dilatation implicating longer activation pathways[28].
2. QRS prolongation is more commonly correlated with extensive ischemia[22].
3. QRS prolongation (especially ventricular activation time) is more common in hypertensive patients with diastolic dysfunction[29]. This group is particularly prone to heart failure in the setting of ACS.
4. QRS prolongation is more commonly associated with poor microvascular perfusion [30] as well as lower Rentrop garde of coronary collaterals[31].

What is unique in our study is that QRS dispersion was found to have the same predictive value for LV systolic and diastolic dysfunction.

Association between QRS dispersion and in-hospital outcome in NSTEMI:

Serious ventricular arrhythmias (pulseless VT and VF) occurred more common in patients with QRS dispersion > 10ms.

The association between QRS duration and dispersion and the development of ACS-related life-threatening ventricular arrhythmias has been previously studied. Jiménez-Candil et al.,[32] studied 502 patients with NSTEMI and found that QRS duration > 90 ms was an independent predictor of in-hospital sudden cardiac death as well as long-term MACE. Bayés de Luna and Elosua [33] found that QRS duration is one of the ECG parameters associated with in-hospital sudden cardiac death in patients with myocardial infarction. Brilakis et al.,[34] showed a significant association between QRS duration, without bundle branch block, and late cardiovascular death. Chavez-Gonzalez et al.,[14] studied 209 patients with STEMI and found that increased QRS duration and dispersion at admission implied a greater likelihood of ventricular arrhythmias in early stages of myocardial infarction than increased duration and dispersion of QTc.

Being regional in nature, ischemia is expected to prolong QRS width with variable degrees in different surface ECG leads with the result of high QRS dispersion. Since QRS is part of QT interval, dispersion in QRS will lead to QT dispersion which facilitates the appearance of serious ventricular arrhythmias[35].

Kirchhof et al.,[36] found that prolonged QRS duration increases QT dispersion but does not relate to arrhythmias in survivors of acute myocardial infarction. This disagreement can be explained by methodological differences, using Bazett's formula for correction with heart rate being a confounder, and possible ethnic variations in ventricular repolarization.

Conclusions

In the current study, we found that higher SXscores were associated with longer QRS duration and QRS dispersion; the latter being independent predictor of SXscore > 33. Moreover, patients with QRS dispersion > 10 ms had higher incidence of Killip class > 1, LV systolic and diastolic dysfunction and significantly higher incidence of in-hospital recurrent angina episodes and serious ventricular arrhythmias.

We, therefore, recommend measuring QRS dispersion at time of admission in NSTEMI patients. Patients with QRS dispersion > 10 ms should be referred to early invasive strategy andshould receive intensive level of care with continuous arrhythmia monitoring.

Study Limitations

1. Lack of standard values of normal versus abnormal QRS dispersion. Also, QRS dispersion may change with the time of evolution of ischemia.
2. The small number of the study and, consequently, patients who had in-hospital major adverse cardiac events. This may reduce the statistical power of our analysis.
3. Lack of follow-up of QRS dispersion after successful revascularization; either by PCI or CABG.
4. Lack of long-term follow-up of patients.

References

1. Brener SJ, Milford-Beland S, Roe MT, et al., Culprit-only or multivessel revascularization in patients with acute coronary syndromes; an American College of Cardiology National Cardiovascular Database Registry report. Am Heart J 2008; 155:140–146.
2. Parikh SV, de Lemos JA, Jessen ME, et al., (CRUSADE and ACTION Registry–GWTG Participants) Timing of in-hospital coronary artery bypass graft surgery for non–ST segment elevation myocardial infarction patients. JACC: Cardiovascular Interventions 2010; 3(4): 419-427
3. Mehta SR, Granger CB, Boden WE, et al., Early versus delayed invasive intervention in acute coronary syndromes. N Engl J Med 2009; 360:2165-2175
4. Roffi M, Patrono C, Collet JP, et al., 2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J 2016; 37: 267–315.
5. Ferreira D: Risk stratification after acute coronary syndromes: Scores, scores and yet another score. Rev Port Cardiol. 2017; 36(2): 85-87
6. Anastasiou-Nana MI, Nanas JN, Karagounis LA, et al., Relation of dispersion of QRS and QT in patients with advanced congestive heart failure to cardiac and sudden death mortality. Am J Cardiol. 2000; 85:1212-7.
7. Yamada T, Shimonagata T, Misaki N, et al., Usefulness of spatial dispersion of QRS duration in predicting mortality in patients with mild to moderate chronic heart failure. Am J Cardiol. 2004;94:960-3.
8. Kountouris E, Korantzopoulos P, Karanikis P, et al., QRS dispersion: an electrocardiographic index of systolic left ventricular dysfunction in patients with left bundle branch block. Int J Cardiol. 2004; 97: 321-2.
9. Xiao SN, Qin YW, Zhang HC, et al., QRS dispersion in assessment of left ventricular systolic function and asynchronous left ventricular wall motion in patients with left buddle branch block. Acad J Second Mil Med Univ. 2010; 31(12): 1326-9.
10. Chávez GE, Alonso HA, Carmona PR, et al., QRS dispersion as an index of dyssynchrony in left bundle branch block and of synchrony after cardiac resynchronization therapy: A variable of successful response. Cor Salud. 2015; 77(22): 106-16.
11. Chávez-González E, Moreno-Martínez FL. QRS dispersion is better than QRS duration for predicting response to cardiac resynchronization therapy. Hell J Cardiol. 2016; 57(5): 366-67.
12. Ma N, Cheng H, Lu M, et al., Cardiac magnetic resonance imaging in arrhythmogenic right ventricular cardiomyopathy: Correlation to the QRS dispersion. Magn Reson Imaging. 2012; 30(10): 1454-60.
13. Elmberg V, Almer J, Pahlm O, et al. A 12-lead ECG-method for quantifying ischemia-induced QRS prolongation to estimate the severity of the acute myocardial event. Journal of Electrocardiography 2016; 49 (3): 272-277
14. Chavez-Gonzalez E, Rodriuez Jiménez AE, Moreno-Martinez FL. QRS duration and dispersion for predicting ventricular arrhythmias in early stage of acute myocardial infraction. Med Intensiva. 2017; 41(6):347-355
15. Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. Am J Cardiol. 1967;20(4):457-64.
16. Granger CB, Goldberg RJ, Dabbous O, et al., Global Registry of Acute Coronary Events Investigators. Predictors of hospital mortality in the global registry of acute coronary syndromes. Arch Intern Med., 2003; 163: 2345–2353.
17. Kligfield P, Gettes LS, Bailey JJ, et al., Recommendations for the Standardization

and Interpretation of the Electrocardiogram Part I: The Electrocardiogram and Its Technology: A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society Endorsed by the International Society for Computerized Electrocardiology. Circulation 2007;115: 1306-1324

18. Gadaleta FL, Llois SC, Sinisi VA, et al., Prolongación del intervalo QT corregido: nuevo predictor de riesgo cardiovascular en el syndrome coronario agudo sin elevación del ST. Rev Esp Cardiol. 2008; 61:572--8.
19. Bazett HC. An analysis of the time-relations of electrocardiograms. Heart. 1920; 7: 353-70.
20. Thygesen K, Alpert JS, Jaffe AS, et al., the Writing Group on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction: Third universal definition ofmyocardial infarction. European Heart Journal 2012; 33: 2551–2567
21. Nagueh SF, Smiseth OA, Appleton CP, et al., Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2016; 29: 277-314.
22. Hussien A, Battah A, Ashraf M, et al., Electrocardiography as a predictor of left main or three-vessel disease in patients with non-ST segment elevation acute coronary syndrome. The Egyptian Heart Journal 2011; 63 (2): 103-107.
23. Wilde AAM, Aksnes G. Myocardial potassium loss and cell depolarization in ischaemia and hypoxia. Cardiovascular Research 1995; 29: 1-15.
24. Pudil R, Feinberg MS, Hod H, et al., The prognostic significance of intermediate QRS prolongation in acute myocardial infarction. Int J Cardiol 2001;78:233-239
25. Tsukahara K, Kimura K, Kosuge M, et al., Clinical implications of intermediate QRS prolongation in the absence of bundle-branch block in patients with ST-segment-elevation acute myocardial infarction. Circ J 2005; 69: 29-34.
26. Shah M, Maludum O, Bhalla V, et al., QRS duration and left ventricular ejection fraction (LVEF) in non-ST segment elevation myocardial infarction (NST EMI).Int J Cardiol. 2016; 221: 524-8.
27. Rodríguez Jiménez AE, Inerarity HC, Arias BV, et al., QRS duration as a predictor of low ejection fraction in the ST-segment elevation myocardial infarction. Cor Salud 2018; 10(1) :13-20
28. Ferrans VJ, Massumi RA, Shugoll GI, et al., Ultrasound studies of myocardial biopsies in 45 patients with obstructive or congestive cardiomyopathy. In: Brink A, Bajusz S, eds. Recent Advances in Studies of Cardiac Structure and Metabolism, Cardiomyopathies. New York: University Park Press, 1973:231-72.
29. Boles U, Almuntaser I, Brown A, et al. Ventricular activation time as a marker for diastolic dysfunction in early hypertension. Am J Hypertens 2010; 23(7):781–785.
30. Karahan Z, Yaylak B, Uğurlu M, et al., QRS duration: a novel marker of microvascular reperfusion as assessed by myocardial blush grade in ST elevation myocardial infarction patients undergoing a primary percutaneous intervention. Coronary Artery Disease 2015,26:583–586
31. Karahan Z, Altıntas B, Ugurlu M, et al., The association between prolongation in QRS duration and presence of coronary collateral circulation in patients with acute myocardial infarction. Journal of the Royal Society of Medicine Cardiovascular Disease 2016; 5: 1–4
32. Jiménez-Candil J, González IC, Martín F, et al., Relationship between QRS duration and prognosis in non-ST-segment elevation acute coronary syndrome. International Journal of Cardiology 2008; 126: 196–203
33. Bayés de Luna A. and Elosua R.: Sudden death. Rev Esp Cardiol. 2012; 65: 1039-52.
34. Brilakis ES, Mavrogiorgos NC, Kopecky SL, et al., Usefulness of QRS duration in the absence of bundle branch block as an early predictor of survival in non-ST elevation acute myocardial infarction. Am J Cardiol 2002; 89(9): 1013–8.
35. Castro-Torres Y, Carmona-Puerta R, Katholi RE. Ventricular repolarization

markers for predicting malignant arrhythmias in clinical practice. World J Clin Cases. 2015; 3: 705-20.
36. Kirchhof P, Eckardt L, Arslan O, et al., Prolonged QRS duration increases QT dispersion but does not relate to arrhythmias in survivors of acute myocardial infarction. Pacing Clin Electrophysiol. 2001; 24:789-95.