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ORIGINAL ARTICLE

Correlation between Gensini Score and in Hospital Outcome in ST-Elevation Myocardial Infarction Patients with Pathological Q Wave

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ABSTRACT

Background: Q waves on the electrocardiogram (ECG) are associated with a more advanced degree of myocardial infarction (MI) and are an effective predictor of worse outcomes. **[1]** The quantity, location, and degree of stenosis of coronary artery lesions as shown in **Figure 1** can be identified using the Gensini score (GS), which was developed for assessing the severity of coronary artery disease. **[2]** It is believed that GS in addition to Q waves on the ECG increases the risk of significant heart damage.

The aim of the work was to assess the correlation between GS and in-hospital outcome in STEMI patients with pathological Q wave in the presenting ECG.

Methods: This study included 142 patients with STEMI and pathological Q wave at the presenting ECG. All patients were recruited from the cardiology department at Zagazig University and assigned into two groups: Group 1 included patients with GS ≥ 20 and Group 2 with GS > 20. All patients were subjected to Full history, demographic data, ECG in admission, Laboratory investigations, Echocardiography and invasive coronary angiography.

Results: There was a statistically significant difference between studied groups regarding Family History, Hypertension, Diabetes Mellitus (DM), Hyperlipidemia and Smoking, cardiac enzymes, Ejection Fraction (EF), TIMI flow before pPCI and GS were statistically correlated with in-hospital outcome.

Conclusions: The combination of Q wave at the presenting ECG with a high Gensini score in STEMI patients is associated with severe coronary artery disease and poor in-hospital outcome.

Keywords: Gensini score, STEMI, Q wave, Outcome.

INTRODUCTION

STEMI is the most prevalent type of MI, resulting in 74.15% of all acute coronary diseases and being the primary cause of mortality among cardiac patients. [3] The Gensini score, which is a more scientific assessment tool for coronary artery stenosis, includes the number, site, and grade of stenosis of coronary artery lesions. [4] Angiographic scoring systems are highly associated with one another and with the presence of atherosclerotic plaques. As a result, scoring systems promise to provide a reliable assessment of the CAD. [5] Once the infarct proceeds to severe tissue damage, Q waves appear with the elevated ST segments. The detection of Q waves has been correlated with severe myocardial damage.

PATIENT AND METHODS

This study was a comparative cross-sectional study that included 142 patients who presented with acute STEMI with Q waves treated by primary PCI at Zagazig University Hospital from August 2020 to April 2023. The study was approved with IRB: 9650 / 2-10-2022. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans. Written consent was obtained from all participants. The patients were assigned into two groups according to Gensini score: Group 1 included patients with Gensini score ≥ 20 and Group 2 included patients with Gensini score > 20. Calculation of the Gensini score is initiated by giving a severity score to each coronary stenosis as follows: 1 Point for ≤25% narrowing, 2 Points for 26 to 50% narrowing and 4 Points for 51 to 75% narrowing, 8 points for 76 to 90% narrowing, 16 points for 91 to 99% narrowing, and 32 points for total occlusion. Each principal vascular segment will be assigned a multiplier in accordance with the functional significance of the myocardial area supplied by that segment, that is: The left main will be assigned the significant multiplier × 5, the proximal segment of the LAD will be given × 2.5, the proximal segment of the LCX will be weighted by a factor of × 2.5, the mid segment of the LAD will be assigned a factor of × 1.5, the RCA, the distal segment of the LAD, mid and distal segment of LCX, the posterior descending artery, and the obtuse marginal artery will be all given × 1, all other areas will be assigned a factor of × 0.5. Figure 1, [4]

Inclusion Criteria: The study included patients with patients with STEMI and Q wave in the presenting ECG managed by primary PCI within 12 hours of chest pain onset. The diagnostic criteria were used to diagnose STEMI: persistent cardiac pain that persisted for more than 30 minutes, ST-segment elevation (> 0.2 mV) in ≥ 2 contiguous leads on the 12-electrodes ECG or (> 0.1 mV) in the right chest electrodes and Increased cardiac enzyme (troponin).

Pathological Q waves were defined by ESC guidelines 2018 as: [18]

- Any Q wave in leads V2 – V3 > 0.02s or QS complex in leads V2 – V3.
- Q wave ≥ 0.03s and ≥ 1mm deep or QS complex in leads I, II, aVL, aVF, or V4 – V5 in any two leads of a contagious lead grouping as (I, aVL; V1 – V6; II, III, aVF).
- R wave > 0.04s in V1 – V2 and R/S > 1 with a concordant positive T wave in absence of conduction defect.

Exclusion Criteria: Patients managed by thrombolytic therapy, patients with old MI, cardiomyopathies, chronic renal or hepatic diseases, and patients with structural heart disease.

All patients were subjected to the following:

- Complete history taking and risk factors, General and local examination.
- Laboratory tests including cardiac enzymes and Troponin (highest levels of cardiac enzymes were collected), renal function tests, and lipid profile.
- A 12-lead ECG was measured with a calibration of 10 mm/mV, a speed of 25 mm/s, and ST deviation (at the J point)
- Echocardiography: within hospital stay, a 2-dimensional (2D) Echocardiography was performed to determine left ventricular (LV dimensions) and ejection fraction (EF %) using modified biplane Simpson's method. Every examination was done in the routine left lateral posture.
- Invasive Coronary angiography invasive coronary angiography and primary percutaneous coronary intervention (pPCI) were performed to every participant.
- Follow-up: consistent following-up the participants was done during in-hospital stay after perfusion and catheterization. During the hospital stay, treatment plans and medication prescriptions were handed over to the treating cardiologists and resident physicians.

Statistical Analysis

Statistical analysis was conducted using SPSS v28 (IBM©, Chicago, IL, USA). Shapiro-Wilks test and histograms were applied to assess if the data distribution was normally distributed. The unpaired student t-test was utilized to examine the quantitative parametric data, which were displayed as mean and standard deviation (SD). The Mann Test was used to evaluate quantitative non-parametric data, which were displayed as the median and interquartile range (IQR). The frequency and percentage (%) of the qualitative factors were reported, and when applicable, the Fisher's exact test or the Chi-square tests were used for analysis. A two-tailed P value < 0.05 was considered statistically significant. Kaplan Meier curve was used to show the time to the incidence of heart failure. Logistic regression was also used to estimate the relationship between a dependent variable and one or more independent variables. Multiple regression was used to analyze the relationship between a single dependent variable and several independent variables.

RESULTS

142 patients presented with STEMI with pathological Q wave at the presenting ECG, (95 male and 16 female) were enrolled in the study. There were 111 patients with GS ≥ 20, mean age, 57.04±10.09, and 31 patients with GS > 20 mean age, 53.26±10.74. Regarding demographic data and risk factors of the studied groups; there was a statistically significant difference between the 2 groups regarding, Family History, Hypertension, DM, Hyperlipidemia and Smoking while no significant difference regarding age, gender and body mass index (BMI). Table (1) Regarding laboratory data: CK-MB and Troponin I were statistically significantly higher in GS ≥ 20 than GS > 20 groups with P values (0.046 and 0.044, respectively) Figure (2), also lipid profile (LDL, HDL and Triglyceride) was statistically significantly different (P value= 0.016, <0.001 and 0.019 respectively).Table (1) Regarding echocardiographic data: EF was statistically significantly different between the 2 groups (GS ≥ 20 mean 40.77±9.35, GS > 20 mean 45.04±6.67) with P value <0.001. Also, LVEDV and LVESV were statistically significant differences between the 2 groups (P value 0.021 and <0.001 respectively).Table (1) regarding angiographic data: According to

culprit lesion, Patients with GS ≥ 20 were 96 (86.5%) with LAD as a culprit, 9 (12.3) with LCX, and 6 (5.4%) with RCA. While GS > 20 patients were 21 (67.7%) with LAD as a culprit, 1 (1.11) with LCX, 8 (25.8%) with RCA. Table (2) TIMI flow before PPCI was statistically significantly different between the 2 groups with P value = 0.024. TIMI flow post-PPCI showed; that TIMI 3 was lower in GS ≥ 20 patients 72 (57.1%) compared to GS > 20 patients 78 (86.8%) and TIMI 0 was 6 (4.8%) in GS ≥ 20 patients and no patient reported with TIMI 0 in GS > 20 patients. Table (2) Gensini score was statistically significantly different between the 2 groups which was high in patients with Gensini score ≥ 20 (mean 55.33±21.92) compared to patients with Gensini score > 20 (mean 12.77±3.65) with P value <0.001.Table (2) Following the patient's in-hospital stay we reported that patients with Gensini score ≥ 20 complicated with HF and mortality higher than Gensini score > 20 patients. Table (3) the incidence of heart failure was significantly higher in patients with Gensini score >20 compared to patients with Gensini score <20 with (HR= 2.0147 (95%CI) 1.0726 to 3.7845) (P = 0.029). Table (4), Figure (3)

Table (1):Demographic, Laboratory and Echocardiography data of the study population.

		GS >20 (n=111)	GS <20 (n=31)	P value
Age (years)		57.04±10.09	53.26±10.74	0.071
Body mass index		29.99±5.01	29.37±4.28	0.536
Gender	Male	95 (85.6%)	29 (93.5%)	0.362
	Female	16 (14.4%)	2 (6.5%)	
Hypertension		59 (53.1%)	6 (19.4%)	**0.001***
Diabetes mellitus		35 (31.5%)	3 (9.7%)	**0.020***
Smoking		72 (64.9%)	8 (25.8%)	**<0.001***
Hyperlipidemia		47 (42.3%)	9 (29.0%)	**<0.001***
Family History		14 (12.70)	2 (5.56)	**0.047***
Creatinine (mg/dl)		1.04±0.2	0.96±0.11	**0.034***
LDL (mg/dl)		98.27±49.74	75.42±27.62	**0.016***
HDL (mg/dl)		44.0±4.01	61.55±3.83	**<0.001***
Triglyceride (mg/dl)		98.5±40.94	79.9±27.61	**0.019***
CK-MB (U/L)		90.6±85.7	58.9±32.2	**0.046***
Troponin I (ng/mL)		1202.5±1654.7	583.94±680.33	**0.044***
EF		40.77±9.35	45.04±6.67	**<0.001***
LVESV		62.66±14.54	55.38±11.18	**<0.001***
LVEDV		103.78±15.1	99.15±12.23	**0.021***

GS: Gensini score, ***:** statistically significant as **P value** <0.05, **EF:** ejection fraction, **LVESD**: left ventricular end systolic diameter, **LVEDD**: left ventricular end systolic volume, left ventricular end diastolic volume, **GS:** Gensini score, ***:** statistically significant as **P value** <0.05, **HDL**: high density lipoprotein, **LDL**: low density lipoprotein, **CKMB**: creatine kinase-myocardial band.

Table (2): CoronaryAngiographic data findings

		GS >20 (n=111)	GS <20 (n=31)	P value
culprit vessel	**LAD**	96 (86.5%)	21 (67.7%)	**<0.001***
	LCX	9 (12.3)	1 (1.11)	
	RCA	6 (5.4%)	8 (25.8%)	
Gensini score		55.33±21.92	12.77±3.65	**<0.001***
TIMI before	**0**	102 (81%)	45 (50%)	**0.024***
	1	18 (14.3%)	32 (13.2%)	
	2	6 (4.8%)	13 (14.7%)	
TIMI after	**0**	6 (4.8%)	----------	0.122
	1	6 (4.8%)	1 (1.2%)	
	2	42 (33.3%)	11 (12.2%)	
	3	72 (57.1%)	78 (86.8%)	

GS: Gensini score, ***:** statistically significant as **P value** <0.05, **LAD:** left anterior descending artery**, RCA:** right coronary artery, **LCX:** left circumflex artery, **TIMI:** thrombolysis in myocardial infarction, ***:** statistically significant as **P value** <0.05

Table (3) in hospital complications:

	GS >20 (n=111)	GS <20 (n=31)	P value
HF	(69) 55%	(23) 26%	**0.001***
Mortality	(15) 12%	(6) 6.66%	**0.047***

GS: Gensini score, **HF**: heart failure.

Table (4) : incidenceof heart failure outcome in the studied groups:

	Mean	SE	95% CI for the mean
Gensini score >20	24.503	0.676	23.178 to 25.828
Gensini score <20	29.323	0.264	28.806 to 29.839

SE: standard error**, CI:** confidence interval.

Figure (1):Method of calculating Gensini score. **(Gensini G. 1983)**

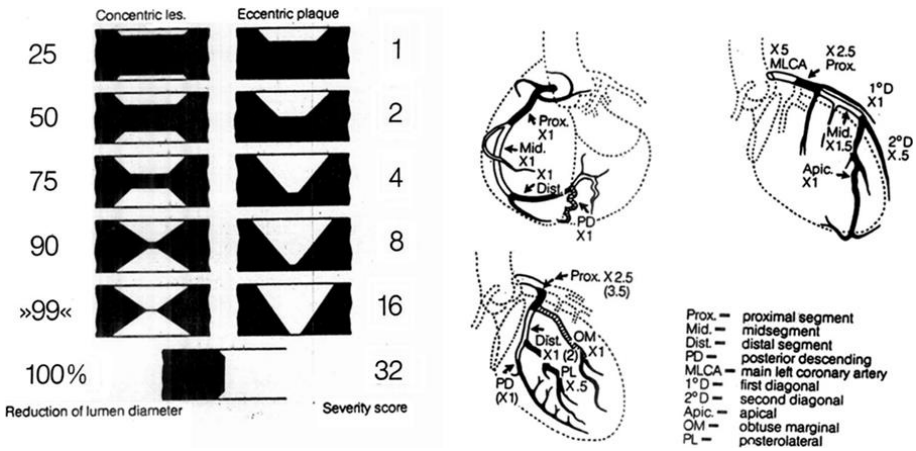


Figure (2): Troponin level of the studied groups.

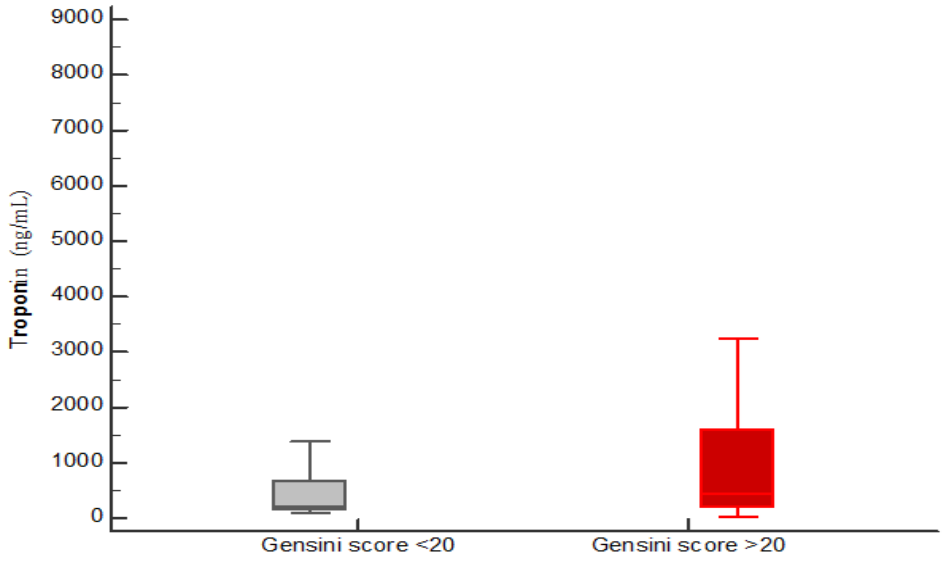
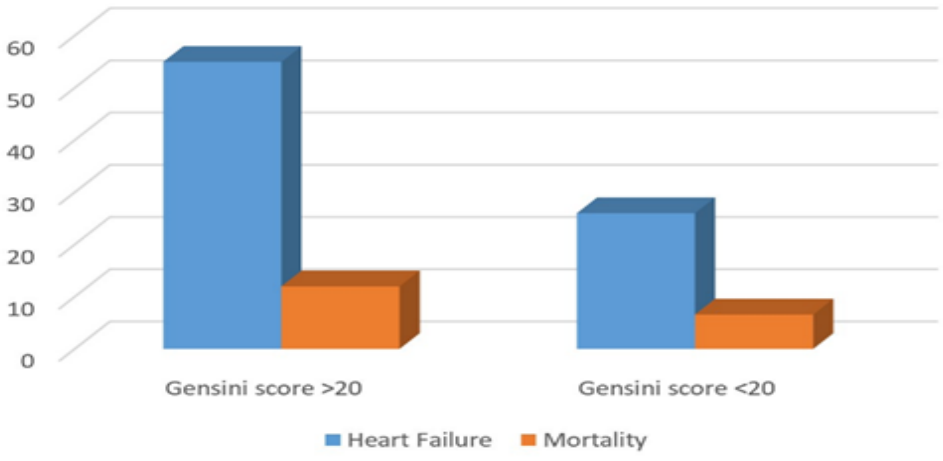


Figure (2): in hospital outcome.



DISCUSSION

Pathological Q waves on the ECG are thought to signify more widespread cardiac damage in STEMI patients and have been demonstrated to be an independent predictor of poor clinical outcomes. (1) Gensini score is one of the most important scoring systems for predicting outcome and the requirement for revascularization, as well as determining the extent and severity of CAD. [4] There are limited studies available to correlate the presence of pathological Q wave at the presenting ECG of STEMI patients and the Gensini score for assessing myocardial infarction severity and in-hospital outcome. Therefore; our study aimed to assess myocardial infarction severity and in-hospital outcome using the Gensini score combined with the presence of pathological Q wave in the presenting ECG of STEMI patients. So, 142 patients were enrolled in this study presented with STEMI with pathological Q wave of them; 111 patients with Gensini score ≥ 20, and 31 patients with Gensini score > 20. We reported that patients with Gensini score ≥ 20 were associated with risk factors such as Hypertension, Hyperlipidemia and DM, smoking, and a family history of CAD. These results indicate that the presence of risk factors is associated with a high risk of CAD whereas age, gender, and BMI were with no statistical significant difference. Charach et al. reported no relationship between GS and cardiovascular risk variables such as hypertension, smoking, BMI, and waist circumference, although it was well-correlated with age, total cholesterol, and diabetes. [6] Also, we reported that cardiac biomarkers (CK-MB and Troponin) were statistically significant higher in patients with Gensini score ≥ 20 than patients with Gensini score > 20 (P value=0.046, 0.044 respectively) which indicates that high Gensini score is associated with severe myocardial damage which was against Charach, et al. who reported a non-significant correlation between GS and cardiac troponin, While correlates with Peppes, et al. who reported that higher myocardial enzyme serum levels were related to increased severity of CAD assessed by Gensini score. [7]

Triglyceride, LDL, and HDL were significantly higher in patients with Gensini score ≥ 20 than patients with Gensini score > 20. This means that lipid profile is a critical risk factor associated with CAD severity which correlates with Gao, et al. who reported that TG and LDL, are positively correlated with the Gensini score. [8] Regarding echocardiographic data; patients with GS ≥ 20 were associated with low EF%, LVEDD, and LVESD than patients with GS > 20 which donates that the presence of Q wave with high Gensini score is associated with a severely impaired myocardial function that correlates with an earlier study of Yildirim, et al. who reported that high Gensini score is associated with Lower Left Ventricular EF% in STEMI patients undergoing pPCI and Delewi, et al. reported the same results. [9-10] Regarding angiographic data; Patients with Gensini score ≥ 20 showed lower TIMI flow post-pPCI (57.1%) than patients with Gensini score > 20 (86.8%) and the result of TIMI 0 post-pPCI was only reported in patients with GS ≥ 20. According to Li et al., the no-reflow event, which is determined by TIMI flow or myocardial blush grade following PCI, can be predicted by the Gensini score. [11] Matos, et al. discovered that TIMI 0 may be independently predicted by the Gensini score. [12] Modolo et al. demonstrated that people with no reflow (TIMI 0/1) had higher overall

Gensini scores as well as higher Gensini scores for the culprit artery than did people with optimum reperfusion. [13].

According to Elrayes et al. there was a strong correlation between NR and the existence of a pathogenic Q wave. For the NR phenomenon, the strongest association was found between TIMI flow and thrombus burden. Thus, we may state that after percutaneous coronary intervention, patients with high GS and Q waves are more likely to experience inadequate reperfusion. [14] Regarding in-hospital follow-up; Patients with Gensini score ≥ 20 were associated with higher rates of HF and mortality than patients with Gensini score > 20 patients (P value 0.001, 0.047 respectively) which agree with older studies assessing short and long-term outcome as Wang et al. showed that the Gensini score is a reliable indicator of long-term bad prognosis in patients with CAD patients undergo PCI, while Huang et al. reported that the Gensini score is linked with short-term major adverse cardiac events. [2, 15] Leivo et al. reported that pathological Q waves in the presenting ECG during STEMI are strong indicators of higher death, cardiogenic shock, and congestive heart failure. They also predict larger infarcts and impaired reperfusion. While the presence of Q waves in previous studies correlates with high major adverse cardiac events (MACE). (16) Kosmidou et al. found that regardless of the amount of time to reperfusion, patients exhibiting Q waves on their baseline ECG had a markedly elevated risk for cardiac death. De Framond, et al. Persistent Q waves during reperfusion were found to be four times as likely to result in heart failure or death in a group of anterior individuals with STEMI as non-Q-wave MI. The pathophysiology of heart failure and ischemic heart disease is expanding. The term "coronary vascular dysfunction" refers to the incapacity of the coronary circulation to meet the metabolic demands of the heart. This can result in tissue death, fibrosis, and hypoxia, which can cause heart failure. Thus, in individuals who had STEMI with Q wave, we could show a positive connection between high Gensini score and a poor in-hospital outcome. [1, 17]

CONCLUSION

Our findings suggest that the combination of Q wave at the presenting ECG with a high Gensini score in STEMI patients is associated with severe coronary artery disease and poor in-hospital outcome. The incidence of heart failure in-hospital in patients with a Gensini score ≥ 20 is significantly earlier than in patients with a Gensini score > 20.

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