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ORGINAL ARTICLE

The Predictive Value of P Wave Peak Time for Coronary Artery Disease Severity in Patients with Acute Myocardial Infarction

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ABSTRACT

Background: Risk stratification for patients with acute myocardial infarction (AMI) and prediction of coronary artery disease severity (CAD) add a valuable benefit to the management and prognosis.We aimed to evaluate the relationship between P wave peak time (PWPT) on surface electrocardiography and Gensini score (GS) of coronary atherosclerotic lesions in AMI patients.

Methods: A total of 76 patients presented with AMI were enrolled and were divided according to GS into two groups; GS <30 & GS ≥ 30. PWPT was obtained at both admission ECG and after 60 minutes from intervention and was compared between groups.

Results: The mean age of our patients was 53.18 ± 9.95 years, 77.6 % were males and 22.4 % were females. Significant prolongation of PWPT duration was observed on admission ECG at GS ≥ 30 (65.51±5.61 ms) vs (57.04 ± 4.33ms) at GS <30, p<0.001. Wh ile after 60 min from reperfusion, the delta change at PWPT has significantly reduced at severe CAD group with NSTEMI rather than STEMI cases. PWPT at cut off value > 60 ms with (AUC 0.859; 95% CI 0.741– 0.976; p < 0.001; sensitivity 70.5%, specificity 85.7%) was found as a good predictor of CAD severity in NSTEMI cases.

Conclusion: PWPT is associated with the severity and complexity of CAD in patients with AMI & can be used as a simple, non-invasive tool adding in patient risk stratification.

Keywords: PWPT, Acute myocardial infarction, Gensini score, Coronary artery disease severity.

INTRODUCTION

Mortality among patients with acute myocardial infarction is highly correlated with CAD severity. NSTEMI patients represent a substantial proportion of the CAD high-risk category mostly having multi-vessel disease, so early risk stratification is important for proper timely revascularization and patient prognosis as well [1].

The Gensini scoring system is among the commonly used systems to assess CAD severity, a high GS is associated with increased mortality [2].

Recently, P wave duration has emerged as a new stay of research in CAD and has been shown to be associated with reperfusion success and AF development in STEMI [3,4].

We suppose that patients with high GS would have greater jeopardized myocardium; extending beyond the ventricles to involve atrial myocardium in higher-risk patients.

Methods:

Study design

The current study is a single-centre observational cross-sectional study conducted in our institute during the period from August 2020 to October 2022. All patients gave written informed consent to participate in the study and all procedures were carried out with the agreement of our institutional review board (IRB), in accordance with the principles of the Helsinki Declaration.

Study population

A total of 76 patients with AMI (50% STEMI cases and 50% non- STEMI), underwent primary percutaneous coronary intervention (PPCI) in our Cath Lab. From the period, of August 2020 to October 2022 were enrolled in the study. Myocardial infarction was defined based on the following criteria: Ongoing ischemic symptoms (within 12 h) and typical rise in cardiac biomarkers, in STEMI patients; a new ST elevation in 2 or more

contiguous leads with leads V1 and V2 measuring at least 0.2 mV or at least 0.1 mV in the remaining leads or new developed left bundle-branch block pattern.

Patients with non-successful PPCI, previous history of CAD, cardiomyopathy, EF <50 %, valvular heart diseases, end-stage organ failure, atrial dysrhythmias including atrial fibrillation/flutter/tachycardia, left atrial diltation secondary to significant structural heart disease and those with inappropriate ECG due to poor image quality were excluded from the study.

Coronary angiography and Gensini score

Coronary angiography was performed via the femoral approach with 7-Fr, images were recorded in multiple projections on a digital system for quantitative analysis, and primary PCI for the culprit vessel was performed according to standard practice. GS was calculated to evaluate the CAD severity. The degree of stenosis and the coronary artery lesion site were scored as follows: 1,2,4,8,16,32 points for (≤ 25%, 26–50%, 51–75%, 76–90%, 91–99% narrowing and for total occlusions) respectively. Then, each lesion score is multiplied by a factor according to the lesion's position in the coronary vessel (5 for the left main, 2.5 for the proximal segment of the left anterior descending (LAD) artery, 2.5 for the proximal segment of the circumflex artery, 1.5 for the mid-segment of the LAD artery, 1.0 for the right coronary artery (RCA), the distal segment of the LAD, the posterolateral artery, and the obtuse marginal artery, and 0.5 for other segments). Finally, the summation of the individual coronary segment scores & calculation of GS. We classified our patients into two groups based on the median value of our GS: Group A (GS <30 points) & Group B (GS≥30 points) (5).

Electrocardiographic analysis

A digital 12-lead ECG which was recorded at a speed of 25 mm/s and a voltage of 10 mm/mV were obtained from all patients at admission and 60 min after PPCI. All ECG papers were analysed with digital image processing software and measurements were calculated by two independent cardiologists blinded to patients' clinical information. P wave peak time (PWPT) was defined as the duration from the beginning of P wave to its peak and measured from leads II and V1 as a mean of three consecutive beats and given as milliseconds.

STATISTICAL ANALYSIS

Data were analysed with SPSS statistical software package version 20.0. (Armonk, NY: IBM Corp) Normality of continuous variables was analysed with the Kolmogorov-Smirnov test. Continuous variables with normal distribution were expressed as mean ± standard deviation, were between two independent groups using Student's t test or Mann-Whitney U test. and those without normal distribution were expressed as median (interquartile range). Categorical variables were expressed as numbers and percentages (%) and compared using the chi-square test or Fisher's exact test. Statistical significance was assumed at a p value <0.05. Correlation between variables was obtained by The Pearson coefficient for continuous variables with normal distribution, and the Spearman's coefficient for variables without normal distribution. Multivariate logistic regression analyses were performed to identify the independent predictors of the of CAD severity. A receiver operating curve (ROC) analysis was used to define PWPT cut off value predicted CAD severity with the best specificity and sensitivity.

RESULTS

Our study recruited 76 patients, their mean age 53.18 ± 9.95 years, 77.6 % were males and 22.4 % were females. All were hospitalized for acute MI and underwent PPCI. Patients were divided into two groups based on Gensini score; GS< 30, n = 41 (53.9%). and GS ≥ 30, n = 35 (46.1%).

We observed higher prevalence of diabetes mellitus, HTN and, dyslipidaemia among patients with GS ≥ 30 (65.7%,71.4% & 71.4%) vs (26.8%, 43.9 and 39%) at lower GS<30, P value (0.001, 0.016 & 0.005 respectively). While there was no significant difference between groups in terms of other studied risk factors; age, sex, smoking or family history of IHD. (Table 1).

Furthermore, routine serum biomarkers LDL, HDL, TG, WBCs, Haemoglobin level, platelets, CK-MB isoenzyme & serum electrolytes; sodium, potassium, calcium, were all similar between groups, except for creatinine which showed higher values at GS ≥ 30 (1.33±0.9 vs 0.94±0.22, p=0.008). Echocardiographic parameters; EF %, E/A, E wave DT, LAVI &wall motion score index (WMSI) did not differ between groups. (Table 2,3)

Comparing electrocardiographic parameters; there was significant higher PWPT duration at admission ECG at GS ≥ 30 group (62.51±5.61ms) vs (57.04 ± 4.33ms) at low GS <30, p<0.001, also prolonged PWPT was strongly correlated to high GS at NSTEMI while showed moderate correlation at STEMI patients (Table 4, figure 1,2).

ECG recorded after 60 min from reperfusion, the delta changes of PWPT at severe CAD (GS≥ 30) was significantly more in NSTEMI; mean± SD, 17.47 ± 5.51, median (IQR) 18.0 (15.0 – 21.0) compared to STEMI cases; mean ± SD, 12.22 ±

6.39, median (IQR) 13.0 (10.0 – 17.0), p =0.015. (Table 5)

All parameters found to be associated with CAD severity in the univariate analyses were included in multiple logistic regression analysis. Both serum creatinine; OR: 1.113, 95% CI: (1.030 – 1.203), p = 0.007 and admission PWPT; OR: 1.300, 95% CI: (1.014 – 1.668), p = 0.039, were found to be independent predictors of having a GS ≥ 30 in STEMI cases while in NSTEMI cases PWPT stands alone as an independent predictor of severity OR: 1.381 95% CI: (1.065 – 1.791), P= 0.015. (Table 6,7).

Only, PWPT showed the highest AUC at ROC analysis. PWPT Cut-off value of >56 ms provided an appropriate diagnostic performance to detect severe CAD in STEMI, (AUC 0.749; 95% CI 0.588– 0.910 ;p =0.009; sensitivity 77.7%, specificity 70%), While its cut off at NSTEMI cases was >60 ms with (AUC 0.859; 95% CI 0.741– 0.976 ;p < 0.001; sensitivity 70.5%, specificity 85.7%) (Table 8, figure 3,4).

Table 1: Demographic and risk factors between studied groups.

Risk factors	Severity				χ^2 t	p
	Non severe (<30) (n = 41)		Severe (>30) (n = 35)			
	No.	%	No.	%		
Age mean SD years	53.80 ± 8.75		52.46 ± 11.28		0.586	0.560
Gender Male female	31(75.6%) 10n(24.4%)		28(80%) 7 (20%)		0.210	0.560
HTN	18	43.9%	25	71.4	5.823*	0.016*
Diabetes	11	26.8%	23	65.7	11.55*	0.001*
Smoker	29	70.7%	26	74.3	0.119	0.730
Dyslipidemia	16	39.0%	25	71.4	7.980*	0.005*
Family History of IHD	13	31.7%	12	34.3	0.057	0.812

HTN ; hypertension ,IHD :ischemic heart disease ;mean ±SD; mean± standard deviation, x^2: Chi square test, t; Student t-test

Table 2: Echocardiographic parameters between groups

Variables	Non severe	Severe	Test of sig.	P value
EF %	54.57 ± 7.94	53.33 ± ± 7.7	U=675	0.657
E wave DT ms	184.9 ± 76.7	180.7 40.5	T 0.285	0.777
E/A Mean ± SD median	0.89 ± 0.3 0.8	0.89 ± 0.41 0.7	U 636.5	0.395
LAVI ml/m^2	22.5±4.2	23±5.1	t 1.35	0.267
WMSI Mean ± SD median	1.4 ± 0.27 1.44	1.47 ± 0.28 1.5	U=633.5	0.379

EF; ejection fraction. DT; E wave deceleration time, WMSI; wall motion score index. LAVI ;left atrial volume index.

Table 3: laboratory findings between groups

Variables	Non severe	Severe	Test of sig. t	P value
CK-MB (ng/mL)	40.9± 10.3	41.1 ±11.2	1.32	0.052
Creatinine	0.94 ± 0.22	1.33± 0.9	5.68	0.008*
HDL (mg/dl)	50.5±5.9	51±4.7	1.23	0.189
LDL (mg/dl)	197±14.6	195.9±13.8	1.60	0.359

Variables	Non severe	Severe	Test of sig. t	P value
Triglycerides (mg/dl)	158±13.6	160±17.4	0.987	0.097
Na^+ mEq/L	137.3 ± 4.06	138.7 ± 3.88	1.48	0.143
K^+ mmol/L	4.2 ± 0.5	5.22 ±6.76	1.21	0.477
Ca^+ mg/dL	8.84± 0.48	8.87 ±0.46	0.5	0.808
HB gm/dl	13.6 ±1.5	13.6 ±1.7	1.1	0.921
WBCs µL	10.04 ±3.5	10.22 ± 4.22	0.2	0.842
Platelets (× 103/µl)	266.3 ±48	265.7 ±59.4	0.95	0.191

CK-MB; creatine kinase-MB isoenzymes, Na; Sodium, K; potassium, Ca; calcium, HB; haemoglobin, WBCs; white blood cells.U: Mann Whitney test , mEq/L; Milliequivalents per litre, g/dl **;** grams per decilitre, mmol/L ;Millimoles per liter; µL microliters.

Table 4: Distribution of PWPT according to CAD severity in studied groups.

PWPT	Severity		T	p
	Non severe (<30) Mean ± SD.	**Severe (>30) Mean ± SD.**		
Total patients **PWPT on admission (ms)** **PWPT after 60 of PCI(ms)**	 57.05 ± 4.33 45.78 ± 5.01	 62.51 ± 5.61 47.74 ± 4.85	 4.791 1.728	 <0.001 0.088
STEMI **PWPT on admission(ms)** **PWPT after 60 of PCI(ms)**	 57.0 ± 3.77 45.45 3.05	 60.72 ± 5.22 48.5 3.87	2.538* 2.712	 0.016* 0.010*
NSTEMI **PWPT on admission(ms)** **PWPT after 60 of PCI(ms)**	57.10 ± 4.90 46.10 ± 6.41	64.41 ± 5.51 46.94 ± 5.72	 4.329* 0.424	<0.001* 0.674

PWPT; P wave peak time, PCI; percutaneous coronary intervention

Table 5: Comparison between Delta change at PWPT after PPCI in STEMI and non–STEMI cases

Decrease in PWPT	NSTEMI	STEMI	U	p
Gensini Score <30	**(n = 21)**	**(n = 20)**		
Min. – Max.	0.0 – 20.0	5.0 – 26.0	200.0	0.793
Mean ± SD.	11.0 ± 4.93	11.55 ± 5.45		
Median (IQR)	10.0 (8.0 – 15.0)	11.0 (7.0 – 14.0)		
Gensini Score ≥30	**(n = 17)**	**(n = 18)**		
Min. – Max.	5.0 – 25.0	-6.0 – 21.0	80.0*	0.015*
Mean ± SD.	17.47 ± 5.51	12.22 ± 6.39		
Median (IQR)	18.0 (15.0 – 21.0)	13.0 (10.0 – 17.0)		

IQR: Inter Quartile Range SD: **Standard deviation** **U: Mann Whitney test**

Table 6: Univariate and multivariate Logistic regression analysis for CAD Severity predictors in STEMI group

	Univariate		Multivariate	
	p	**OR (95%C.I)**	**p**	**OR (95%C.I)**
Diabetic	0.013*	7.083 (1.52 – 33.032)	0.112	7.355 (0.628 – 86.143)
PWPT on admission (ms)	0.025*	1.208 (1.024 – 1.426)	0.039*	1.300 (1.014 – 1.668)
Creatinine(x100)	0.004*	1.097 (1.031 – 1.168)	0.007*	1.113 (1.030 – 1.203)

OR: Odd`s ratio, C.I: Confidence interval

Table 7: Univariate and multivariate Logistic regression analysis for CAD Severity predictors in NSTEMI group

	Univariate		Multivariate	
	p	**OR (95%C.I)**	**p**	**OR (95%C.I)**
HTN	0.004*	12.187(2.186 – 67.945)	0.171	4.603 (0.516 – 41.015)
Diabetic	0.022*	5.281 (1.270 –21.966)	0.304	0.186 (0.008 – 4.609)
Hyperlipidemia	0.004*	12.19 (2.186 – 67.95)	0.085	16.07 (0.679 – 380.5)
PWPT on admission	0.002*	1.365 (1.117 – 1.669)	0.015*	1.381 (1.065 – 1.791)

Table 8: Diagnostic performance of PWPT on admission in both groups.

Admission PWPT	**AUC**	**p**	**95% C.I**	**Cut off#**	**Sensitivity**	**Specificity**	**PPV**	**NPV**
STEMI	0.749	0.009*	0.588 – 0.910	>56	77.78	70.0	70.0	77.8
NSTEMI	0.859	<0.001*	0.741 – 0.976	>60	70.59	85.71	80.0	78.3

AUC: Area Under a Curve p value: Probability value CI: Confidence Intervals

NPV: Negative predictive value PPV: Positive predictive value

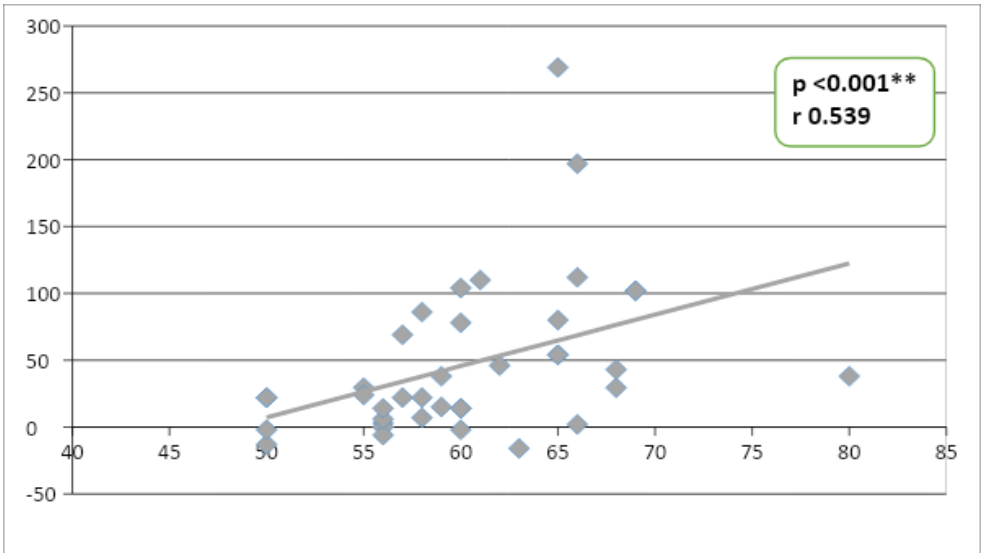

Fig (1): Correlation between admission PWPT and Gensini Score NSTEMI cases

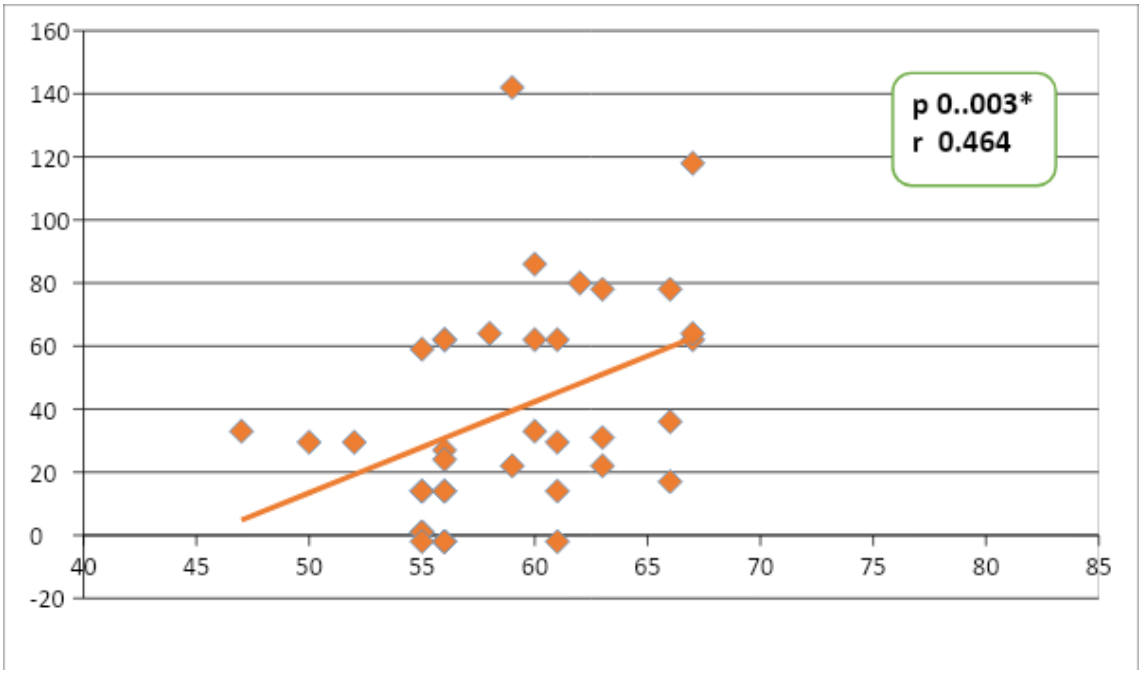

Fig (2): Correlation between admission PWPT and Gensini Score STEMI cases

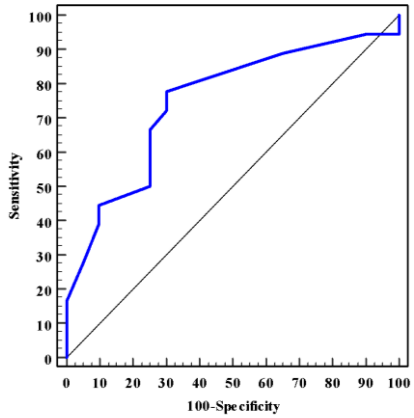

Figure (3): ROC curve of PWPT for prediction of CAD severity in STEMI group

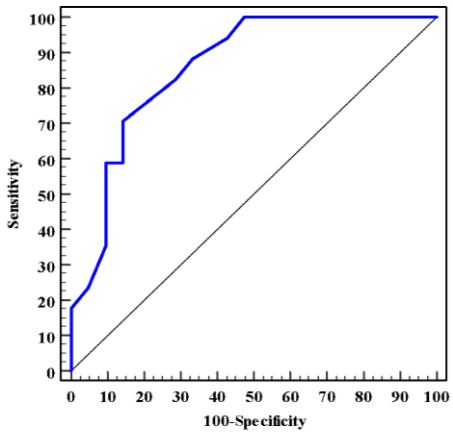

Figure (4) ROC curve of PWPT for prediction of CAD severity in NSTEMI group

DISCUSSION

It is long known that mortality among CAD patients is highly correlated with disease severity. Numerous scoring systems have emerged for CAD risk stratification; the most are invasively applied. Gensini scoring system has been related to both short as well as long-term cardiovascular risk [6].

Analysis of P on surface ECG has been handled in numerous previous studies, and proved to be associated with atrial structural deformations secondary to many cardiovascular diseases as hypertension, mitral valve disease, atrial arrhythmias [7]. Now, PWPT emerged as a recent promising tool worth research in CAD reflecting inevitable ischemic atrial electrical remodelling in response to acute severe coronary ischemia [8,9].

Our study demonstrated that prolonged PWPT obtained from surface ECG on admission was correlated to the severity and complexity of CAD assessed by GS in patients with AMI.

PWPT with a cut-off value of 56 ms with sensitivity of 77.78%and specificity of 70% and AUC 0.749, p =0.009 is an independent predictor of GS ≥ 30 in STEMI cases, while higher cut-off value of 60 ms with sensitivity of 70.59%and specificity of 85.71% & AUC 0.859, p<0.001 is an independent predictor of GS ≥30 in NSTEMI cases.

This comes in line with Bayman et al., who reported that PWPT correlated to Gensini score and can predict CAD severity in patients with NSTEMI with cut off value of 45.5 ms at 62% sensitivity of and 71% specificity [10].

Another report by Burak et al., observed that prolonged PWPT is associated with high SYNTAX score and is an independent predictor of severe CAD with a higher cut-off value of 69.6 ms at 78.9% specificity and a 58.3%. sensitivity [11].

Despite P wave duration is affected by many confounders; left atrial diameter, LA volume as well as left ventricular diastolic function, in our study we did not find any significant correlation between PWPT and the aforementioned parameters, which supports our proposal; ischemic damping of atrial perfusion affected atrial depolarization causing PWPT prolongation rather than actual diltation or elevated left ventricular diastolic pressure in response to ventricular ischemia.

This was discordant to that reported by Burak et al., and Alasga et al., who stated that PWPT was positively correlated to LA diameter, which may be attributed to different selection criteria between studies [12].

Regarding severe CAD group, we found that admission PWPT showed higher values at NSTEMI patients (64.41 ± 5.51) versus (60.72 ± 5.22) in STEMI group, p =0.025. Also, higher correlation to CAD severity at NSTEMI group r=0.539, p<0.001 while moderate correlation was observed at STEMI group, r =0.464, p =0.003, which may be explained by more diffusely affected coronaries and higher risk profile in NSTEMI cases which turns their myocardium more vulnerable to ischemic insults.

Regarding PWPT evaluation after 60 minutes from successful PPCI, it showed significant improvement irrespective of CAD severity in NSTEMI cases, 46.10 ± 6.41 vs 46.94 ± 5.72 with p =0.674. This can be explained by the importance of such simple tool in risk stratification in patients with NSTEMI, so successful reperfusion dramatically improves the vulnerable ischemic myocardium and gets PWPT down in severe group in comparable to results in less severe group, illuminating the effect of reperfusion in such risky group. This effect of revascularization was also obvious at NSTEMI rather than STEMI cases as the delta reduction of PWPT was significant at the first; mean ± SD, 17.47 ± 5.51, median (IQR) 18.0 (15.0 – 21.0) compared to STEMI cases; mean ± SD, 12.22 ± 6.39, median (IQR) 13.0 (10.0 – 17.0), p =0.015.

CONCLUSION

The current study demonstrates good correlation between PWPT evaluated on the admission ECG and the complexity and severity of CAD. However, PWPT showed effective reduction after invasive revascularization in the NSTEMI group compared to STEMI. This finding supports the valuable addition of PWPT as a simple, non-invasive bedside tool for risk classification in high risk NSTEMI cases where typical ischemic ECG changes may be absent. So rises the priority of early invasive management.

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