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THE SIGNIFICANCE OF Q WAVE ON ELECTROCARDIOGRAM AT THE PRESENTATION OF ANTERIOR WALL ST SEGMENT ELEVATION MYOCARDIAL INFARCTION FOR PATIENTS UNDERGOING PRIMARY PERCUTANEOUS INTERVENTION

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ABSTRACT

Background and Hypothesis:The presence of initial Q waves in the presenting ECG of ST segment elevation myocardial infarction (STEMI) patients was associated with adverse prognostic outcomes compared to those without Q waves regardless of the reperfusion method. Those adverse findings can be due to a number of other associated high risk variables related to poor clinical class upon presentation and/or inadequate revascularization outcome (TIMI flow less than III or inadequate ST segment resolution). Whether a similar adverse impact is going to be found in relatively lower risk STEMI patients with initial Q waves on their presenting ECG is questionable.

Aim of work: Comparative evaluation of the in hospital and intermediate outcome of primary PCI for anterior wall STEMI according to presence or absence of pathological Q waves on initial ECG analysis in selected group of patients with; Killip I clinical class on presentation, post procedure TIMI III flow, ST segment resolution (STR) > 70% and absent Q waves on the post procedural ECG.

Methods:This is a prospective, comparative study which included 191 patients; diagnosed with anterior wall STEMI, within 12 hours of onset of symptoms and underwent primary PCI according to the ACCF/AHA guidelines for management of STEMI (ACCF/AHA, 2013). Patients were divided into 2 groups according to the absence (Group A) or presence (Group B) of pathological Q waves on their initial ECG tracing analysis.

Results:Presence of baseline Q wave (group B) was associated withhigher occurrence of congestive heart failure; both during in-hospital stay [11.34%(group B) vs. 3.19% (group A), p value 0.031] and at 6 months follow up period [28.88% (group B) vs. 8.79% (group A), p value <0.001]. Patients with baseline Q waves had also worse echocardiographic indices during in-hospital stay and 6 months follow up; with significantly lower ejection fraction (EF)[In-hospital: 45.19 ±7.43% (group B) vs. 51.56 ±8.82% (group A), p value <0.001 and At 6 months: 46.14 ±14.56% (group B) vs. 55.72 ±15.53% (group A), p value <0.001)],higher Left ventricular end-systolic volume (LVESV) [In-hospital: 70.4 ±18.44 ml (group B) vs. 62.75 ±18.96 ml (group A), p value 0.005 and At 6 months: 59.59 ±22.77 ml (group B) vs. 48.81 ±18.39 ml (group A), p value <0.001]and higher (worse) wall motion score index (WMSI) [In-hospital:1.57 ±0.27 (group B) vs. 1.38 ±0.18 (group A), p value <0.001 andAt 6 months: 1.27 ±0.43 (group B) vs. 0.95 ±0.25 (group A), p value <0.001].We did not find any impact for baseline Q waves on the mortality, whether in-hospital [2.06% versus 1.06%, p value 0.579, in the Q wave (group B) vs. non Q wave (group A), respectively]orat 6 months follow up [5.26% versus 2.15%, p value 0.26, in the Q wave (group B) vs. non Q wave (group A), respectively].Multivariate analysis for the predictors of total mortality showed that advanced age was the only independent predictor of total mortality (B coefficient 0.168, 95% CI 0.62 to 0.274, p value <0.002).

Conclusion: The study adds prospective confirmation about the prognostic value of baseline Q waves in patients with anterior ST-segment elevation myocardial infarction, undergoing primary PCI. Presence of baseline Q wave is a poor prognosticfindingassociated with increased congestive heart failure, decreased LVESV, higher WMSI and lower EF, whether in-hospital or at 6 month follow up. However, we demonstrated that baseline Q waves did not have any impact on mortality, whether in-hospital or at 6 months follow up period.

INTRODUCTION

The electrocardiogram provides a simple, practical, and cost-effective method for assessment of the efficacy of a reperfusion strategy (Kumar et al., 2009). The presence of Q waves on the presenting

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electrocardiograph (ECG) can be a physiologic proxy of the stage of infarct evolution. Q waves can develop early in STEMI and do not imply irreversible myocardial necrosis(Raitt et al., 1995).

Q waves on presentation are independent of symptom duration in predicting epicardial and microvascular reperfusion in STEMI treated with thrombolysis(Wong et al., 2006 and Armstrong et al., 2009).Primary percutaneous coronary intervention (PPCI) for STEMI is superior to thrombolysis in establishing coronary blood flow, with lower rates of death, reinfarction, stroke, and heart failure(Kumar et al., 2009). Evidence on the prognostic value of Q waves on presentation in patients undergoing PPCI for STEMI is emerging (Armstrong et al., 2009).

HYPOTHESIS & AIM OF WORK

HYPOTHESIS: The presence of initial Q waves in the presenting ECG of STEMI patients was associated with adverse clinical and prognostic outcomes - regardless of the reperfusion method - compared to those without Q waves(Wong et al., 2011). Those adverse findings can be due to a number of other associated high risk variables related to poor clinical class upon presentation and/or inadequate revascularization outcome (TIMI flow less than III or inadequate ST segment resolution).Whether a similar adverse impact is going to be found in lower risk STEMI patients with initial Q waves on their presenting ECG is questionable.

AIM OF WORK: Comparative evaluation of the in-hospital and intermediate outcome of primary PCI for anterior wall ST segment elevation myocardial infarction (STEMI) according to presence or absence of pathological Q waves on initial ECG analysis in selected group of patients with; Killip I clinical class on presentation, post procedure TIMI III flow, ST segment resolution (STR) > 70% and absent Q waves on the post-proceduralECG.

STUDY DESIGN AND METHODOLOGY

STUDY DESIGN: At Dar Al Fouad Hospital (Cairo, Egypt), 261 patients were prospectively enrolled in our study over a period of 19 months (June, 2013 - December, 2014). All included patients suffered anterior wall ST elevation myocardial infarction (STEMI) and were indicated for primary percutaneous coronary intervention (PPCI) according to the 2013 ACCF/AHA guidelines for management of STEMI.

Excluded patients were those who presented after 12 hours from symptom onset (6 patients), those refereed for rescue PCI (3 patients), patients with Killip clinical class > I on presentation (8 patients) and patients with confounding features on their electrocardiogram such as bundle branch block (3 patients) or paced rhythm (1 patient). Patients with old ECG tracing that shows prior pathological Q waves in the infarct related ECG leads or history of previous myocardial infarction (MI), previous revascularization with PCI, or coronary artery bypass surgery were also excluded (12 patients) to enable best correlation of Q-wave status with infarct artery regional wall motion (Table 1).

nted after 12 hours of symptom onset
ous PCI and/or previous CABG
ardial infarction diagnosed by new or presumably new LBBB or other forms of bundle branch block
l rhythm
CG tracing that shows prior pathological Q waves in the infarct related ECG leads or history of previous ardial infarction
:cessful thrombolytic therapy prior to presentation, refereed for rescue PCI
clinical class > I on presentation
(post procedural) TIMI flow less than III
Procedural ECG with ST segment resolution (STR) <70%
opment of in-hospital Q waves on the post procedural and/or pre-discharge ECG

Table 1: Exclusion criteria for the study [Left bundle branch block (LBBB) – Electrocardiogram (ECG) – Percutaneous coronary intervention (PCI) – Coronary artery bypass grafting (CABG) - Thrombolysis in myocardial infarction (TIMI)]

The remaining 228 patients were refined according to the final TIMI flow post procedure, ST segment resolution in post procedural ECG, development of new pathological Q waves during in-hospital stay and compliance to 6 months follow up (Figure 1). Patients excluded during the course of in-hospital admission were

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those with; final (post procedural) TIMI flow less than III (12 patients), post procedural ECG with ST segment resolution (STR) <70% (6 patients), those who developed Q waves on the post procedural and/or pre-discharge ECG (15 patients) and patients who failed to comply with the 6 months follow up (4 patients).

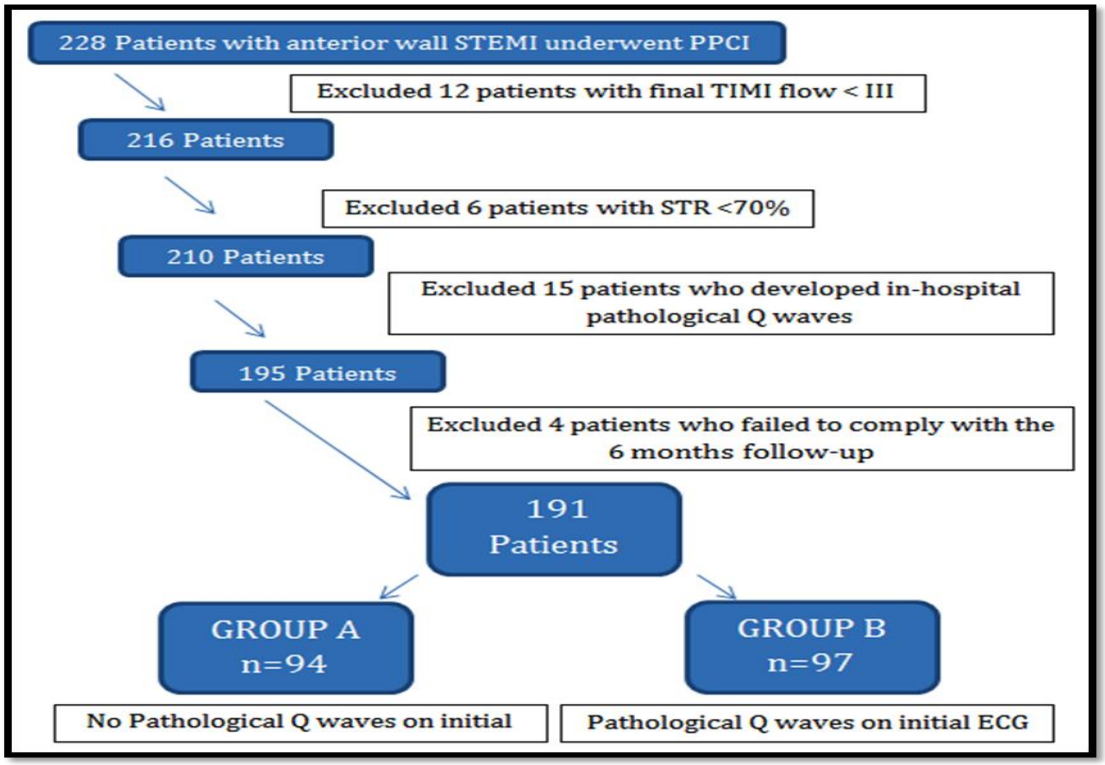

Figure 1: Flow chart for patients included in the study.[ST segment Elevation Myocardial Infarction (STEMI) - Primary Percutaneous coronary intervention (PPCI) – Thrombolysis in myocardial infarction (TIMI) - ST segment Resolution (STR) – Electrocardiogram (ECG)]

The final population included 191 patients who were divided into 2 groups according to the absence (Group A: 94 patients) or presence (Group B: 97 patients) of pathological Q waves on their initial ECG tracing analysis.

METHODOLOGY: All patients received aspirin, clopidogrel, statin, Beta blockade, and angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists unless contraindicated. All patients were subjected to thorough analysis of the following:

A)HISTORY AND EXAMINATION: Age, gender, onset of symptoms and risk factors for coronary artery disease including; diabetes mellitus, dyslipidemias, hypertension, smoking and family history of premature coronary artery disease.All patients were stratified based on the clinical evidence of left ventricular failure using Killip clinical classification(Killip et al., 1967).

B) ELECTROCARDIOGRAM (ECG): All patients underwent ECG recording at a speed of 25 mm/s using standard calibration (1 mm = 0.1 mV). The ECGs were done at presentation, immediately after revascularization and at 6 months follow up with special emphasis on pathologic Q-wave and ST segment resolution (STR).The criteria for pathological Q wave were defined according to the 2007 Joint ESC/ACCF/AHA/WHF Task Force [Table 2] (Thygesen et al. 2007).

The height (in mm) of ST segment elevations was measured 20 ms after the J-point, in leads I, aVL and V1 through V6. The degree of ST-segment resolution was assessed within 30 minutes after revascularization using Schroder's method (Schroder et al., 1995), in which the degree of ST-segment resolution was calculated relative to the baseline ST-segment elevation: complete resolution(≥70%), partial resolution (30% to 70%), and no resolution (≤30%). Only patients with complete ST segment resolution were included in the study.

Pathol	e ≥20 msor

Q	mplex
I, aVL or	e ≥30 ms and ≥0.1 mV deep or mplex
Q Equi	e ≤0.1 mV and ≤10 ms

Table 2: Definition of pathologic Q wave or Q-wave equivalent

C) CORONARY ANGIOGRAPHY AND PRIMARY PERCUTANEOUS CORONARY INTERVENTION (PPCI): All the patients with analyzed coronary angiograms had final TIMI III flow at the end of revascularization procedure. Balloon time was marked during the procedure for accurate assessment of door to balloon time. All cine-angiograms of both groups were analyzed by qualitative (eye ball angiographic analysis) and quantitative (computer-based) methods.

1. QUALITATIVE (EYE BALL ANGIOGRAPHIC ANALYSIS)

- Single vessel (left anterior descending artery) involvement versus multivessel affection.
- Level of occlusion within the vessel (proximal, mid or distal) was identified by the proximal portion of the lesion, even if the lesion extended to a more distal segment.
- Thrombus grading according to the TIMI thrombus scale [Table 3] (Gibson et al., 2001).

THROMBUS	IPTION
0	giographic evidence of thrombus
1	graphic features suggestive of thrombus ased contrast density ess of contrast lar lesion contour oth convex meniscus at the site of a total occlusion stive, but not firmly diagnostic of thrombus
2	te thrombus present in multiple angiographic projections d irregular lesion contour with a significant filling defect – the bus' greatest dimension is <1/2 vessel diameter
3	te thrombus appears in multiple angiographic views est dimension from >1/2 to <2 vessel diameters
4	te large size thrombus present est dimension >2 vessel diameters
5	te complete thrombotic occlusion of a vessel vex margin that stains with contrast, persisting for several cardiac

Table 3 Thrombus grading according to the TIMI thrombus scale (Gibson et al., 2001)

- Total atherosclerotic burden in the culprit vessel was calculated using Gensini score from the coronary arteriogram by assigning a severity score to each coronary stenosis according to the degree of luminal narrowing and its geographic importance (Figure 2). Reduction in the lumen diameter, and the roentgenographic appearance of concentric lesions and eccentric plaques were evaluated as follows:

- Reductions of 25%, 50%, 75%, 90%, 99%, and complete occlusion were given Gensini scores of 1, 2, 4, 8, 16, and 32, respectively.

- Each principal vascular segment was assigned a multiplier according to the functional significance of the myocardial area supplied by that segment: LM ×5; the proximal segment of LAD ×2.5; the proximal segment of LCx ×2.5; the mid-segment of the LAD ×1.5; the RCA, the distal segment of the LAD, the posterolateral artery and the obtuse marginal artery ×1; and others ×0.5 (Gensini et al., 1983).

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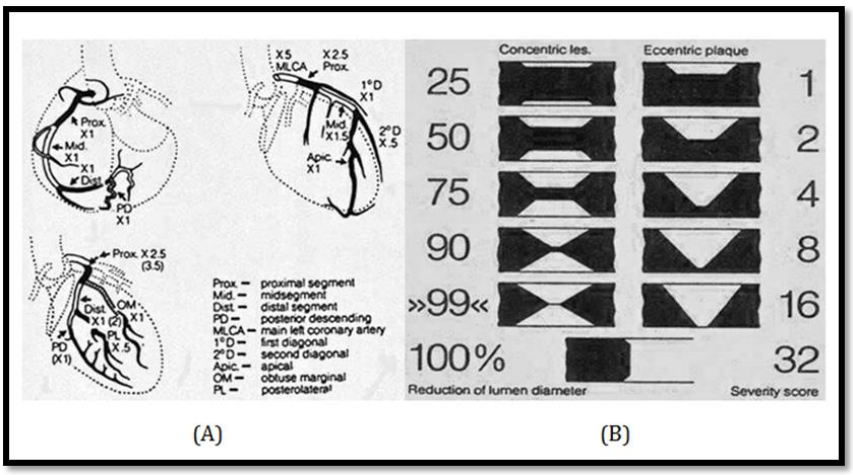

Figure 2: Gensini scoring system for angiographic assessment of atherosclerotic burden (A) Multiplier index in accordance with the functional significance of the myocardial area supplied by that segment. (B) Severity score according to luminal diameter reduction.

- Assessment of TIMI flow before and after primary PCI (Table 4) and myocardial blush grade(MBG) (Table 5).

	TIMI FLOW (IPTION
0	rfusion; no antegrade flow beyond the point of occlusion.
1	ration without perfusion; contrast material passes beyond the area of obstruction but o opacify the entire coronary bed distal to the obstruction for the duration of the cine-graphic filming sequence
2	l perfusion; contrast material passes across the obstruction and opacifies the coronary distal to the obstruction. However, the rate of entry of contrast material into the distal to the obstruction or its rate of clearance from the distal bed (or both) is otibly slower than its flow into or clearance from comparable areas not perfused by the usly occluded vessel (e.g., opposite coronary artery or the coronary bed proximal to struction)
3	lete perfusion; antegrade flow into the bed distal to the obstruction occurs as promptly tegrade flow into the bed proximal to the obstruction, and clearance of contrast ial from the involved bed is as rapid as clearance from an uninvolved bed in the same or the opposite artery

Table 4 Thrombolysis in Myocardial Infarction (TIMI) flow grading (Gibson et al. 1996)

	MYOCARDIAL BLUSH (IPTION
0	ocardial blush (or contrast density) or persisting blush (staining)
1	al myocardial blush or contrast density
2	ate myocardial blush or contrast density (less than that obtained g angiography of a contralateral or ipsilateral non-infarct -related ary artery)
3	al myocardial blush or contrast density (comparable with that ed during angiography of a contralateral or ipsilateral non-t-related coronary artery)

Table5 Myocardial blush grading (Hof et al. 1998)

2. QUANTITATIVE CORONARY ANGIOGRAPHY (QCA) ANALYSIS

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The analysis was carried out by DICOM compliant software operated on an AXIOM ARTIS FC/BC - AXA 4 Siemens device. QCA analysis was carried out before and after revascularization. Analysis included several steps; selecting a scene/image, calibration, selecting the diseased segment of the artery, artery contour detection, performing analysis and final reporting. Of many variables displayed we selected the following variables:

- Minimal Luminal Diameter (MLD) [mm]: The minimum vessel diameter at the position of the most severe stenosis (Reiber et al., 1985).
- Percentage of diameter stenosis (%DS): The percentage of the diameter of the most severe stenosis compared with the reference diameter [Stenosis diameter/reference diameter x 100%] (Harrison et al., 1984).
- Lesion length [mm]: Length of the analyzed vessel segment (Reiber et al., 1985).
- Reference Vessel Diameter (RVD): [mm]: Diameter of the vessel at the reference point (Harrison et al., 1984).

D) ECHOCARDIOGRAPHY: All patients underwent echocardiographic examination within 2 hours post revascularization and again at 6 months follow up with analysis of4 parameters; ejection fraction (EF), left ventricular end-diastolic volume (LVEDV), left ventricular end systolic volume (LVESV) and wall motion score index (WMSI).

- Calculation of Ejection Fraction (EF) and Left Ventricular Volumes:

- Measurements for the left ventricular (LV) volumes and the left ventricular ejection fraction (EF) were done from the two-dimensional (2D) echocardiographic images of the LV. Ejection fraction was calculated using the disk summation or biplane Simpson method. The LV endocardial border was traced from two orthogonal apical views (apical four-chamber [A4C] and apical two-chamber [A2C]) at end diastole and at end-systole(Schiller et. al, 1989).

- Quantitative assessment of the regional wall motion abnormalities was done by calculating Wall Motion Score Index (WMSI):

- WMSI = Sum of wall motion scores / 16 (Number of myocardial segments visualized) [Figure 3]
- Wall motion score:
  1 = Normal Kinesis with > 5mm myocardial thickening.
  2 = Hypokinesis with 2-5 mm myocardial thickening.
  3 = Akinesis with < 2mm myocardial thickening.
  4 = Dyskinesis, showing outward motion of myocardial segment (Negar et al., 2007).

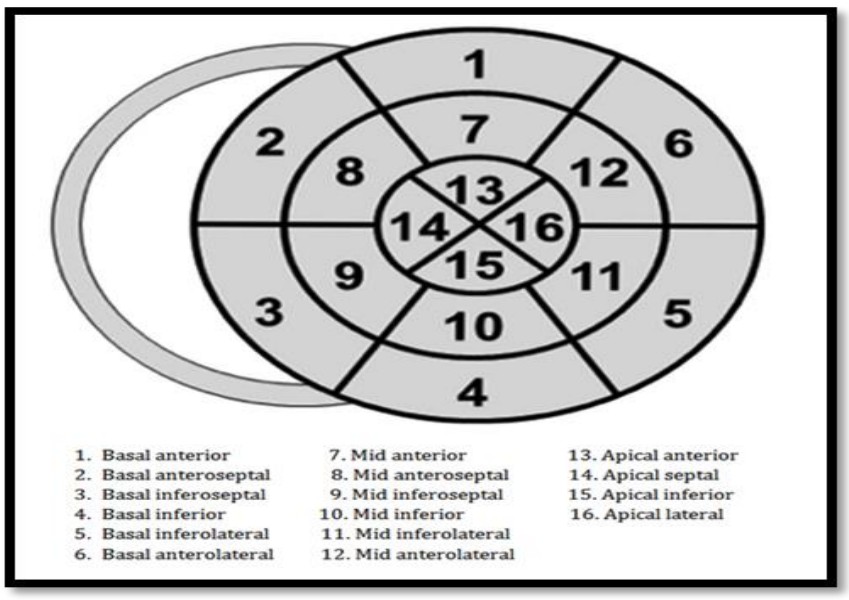

Figure 3: Diagram of the "bulls-eye" plot representing the 16 left ventricular segments used to calculate wall motion score index (WMSI)

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E) LABORATORY WORKUP: Additionally, biomarkers of myocardial necrosis (creatine kinase [CK], creatine kinase myocardial band [CK-MB] and troponin-I) were obtained; at presentation, after primary PCI (peak values) and at day 3 post myocardial infarction.

F) FOLLOW UP

All patients were followed up after a period of 6 months for history analysis, clinical examination, resting electrocardiogram and echocardiography.

G) END POINTS:

A. PRIMARY ENDPOINTS

The primary endpoints were assessed during in-hospital stay and at 6 months follow up including; cardiac mortality, re-infarction (in-hospital)/recurrent infarction (at 6 months follow up),congestive heart failure (CHF) and target vessel revascularization (TVR). Target vessel revascularization – whether repeat PCI or coronary artery bypass surgery - was assessed only at the 6 months follow up period (Table 6).

Reinfarction was defined by recurrence of ischemic chest pain with new ST-segment deviation or T-wave abnormalities and re-elevation of cardiac biomarkers ≥ 20% in two repeated measurements, sampled 6-8 hours apart (assessed during in hospital stay and the first 28 days following incident MI). On the other hand recurrent MI was diagnosed if the characteristics of MI occurred after 28 days following an incident MI (assessed at 6 months follow up).The above criteria for defining reoccurrence of MI are in accordance with the 2008-2009 revision of the WHO (World Health Organization) definition of myocardial infarction (Mendis et al., 2011). Heart failure was defined as evidence of pulmonary venous and/or peripheral congestion on history, examination, and/or imaging that required at least 40 mg of furosemide for treatment(Kumar et al., 2009).

B. SECONDARY ENDPOINTS

The secondary endpoints included; left ventricular end diastolic volume (LVEDV), left ventricular end systolic volume (LVESV), ejection fraction (EF) and wall motion score index (WMSI). These echocardiographic parameters were examined within 2 hours of revascularization (in-hospital) and again at 6 months follow up period (Table 6).

C. COMPOSITE ENDPOINTS OF MAJOR ADVERSE CARDIAC EVENTS (MACE)

Composite endpoints of major adverse cardiac events (MACE) were also assessed during the in-hospital stay and at 6 months follow up. They included cardiac mortality, re-infarction (in-hospital)/recurrent infarction (at 6 months follow up)and congestive heart failure (CHF) [Table 6].

spital and at 6 months follow up	
ıry Endpoints	ıc Death
	arction **(in-hospital)**/Recurrent infarction **(at 6 months follow up)**
	stive Heart failure
	t vessel revascularization **(only at 6 months follow up)**
dary Endpoints	entricular End Diastolic Volume (LVEDV) [ml], mean ±SD
	entricular End Systolic Volume (LVESV) [ml], mean ±SD
	ɔn fraction (EF) [%]
	Motion Score Index (WMSI)
ined MACE	ıc Death
	arction **(in-hospital)**/Recurrent infarction **(at 6 months follow up)**
	stive Heart failure

Table 6: In-hospital and 6 months follow up [Major adverse cardiac events (MACE) – milliliter (ml) - Standard Deviation (SD]

H) STATISTICAL ANALYSIS: The values were presented as means +SD or as numbers and proportions, as appropriate. The relations between qualitative variables were evaluated by Chi-square test or Fisher's exact test, as indicated. Means were compared with Student's test. Variables that were statistically significant in univariate analysis were introduced in a logistic regression model to detect independent predictors of in-hospital as well as 6 months MACE and mortality. All tests were bilateral and a P value of 5% was the limit of statistical significance. Analysis was performed by statistical package software IBM- SPSS for MAC, version 23.

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RESULTS Among 191 patients who underwent primary percutaneous coronary intervention (PPCI) for anterior wall SETMI; there were 94 patients who did not have Q waves on their initial ECG analysis (group A) versus 97 patients with pathological Q waves (group B).

ble	P A wave)	P B ve)	ıe
ears]	86	0.43	
	.1%)	.9%)	
tension	.7%)	.3%)	
tes Mellitus	..2%)	.8%)	
idemia	.6%)	.4%)	
ing	..5%)	.5%)	
r Premature IHD	.5%)	.5%)	
of Symptoms to Time of Presentation[minutes]	55.6	111	**1**
to Balloon time [minutes]	3	4	**1**
of symptoms to balloon [minutes]	60	109	**1**
Hospital Stay [Days]	1.14	.8	**1**
tay [Days]	.75	.3	**1**

Table 7: Comparative analysis for age, gender, risk factor profile, time from onset of symptoms to presentation/to balloon dilatation, door to balloon time and duration of CCU/hospital stay - Values are means ±SD (Standard Deviation) or frequency (percentage) of patients. [Family History (F.H.) - Ischemic Heart Disease (IHD) – Coronary Care Unit (CCU)]

Time from symptom onset to presentation and to balloon dilatation was significantly longer in patients with initial Q waves (group B) [276 ±111 minutes and 363 ±109 minutes, respectively] compared to those without Q waves (group A) [148 ±55.6 and 225 ±60 minutes, respectively] (both p values were < 0.001). Also the door to balloon time (DTBT) was found to be longer in those with initial Q waves (group B) [87 ±14 minutes] versus those without Q waves (group A) [77 ±13 minutes] (p value was < 0.001) [Table 7].

Both the duration of coronary care unit (CCU) admission and that of the total hospital stay were found to be longer in patients with Q waves (group B) [3.7 ±1.3 and 5.5 ±1.8 days, respectively] versus those without Q waves (group A) [3.2 ±0.75 and 4.39 ±1.14 days, respectively] (p value < 0.001). There was no statistical significance between both groups regarding the age, gender and risk factor profile [Table 7].

ble		P A wave)	P B ve)	ıe
Vessel (LAD) Involvement		50.7%)	49.3%)	1
of Involvement	mal	45.8%)	54.2%)	5
		63.2%)	36.8%)	
nbus Burden (0-5)		83.3%)	6.7%)	001
		50%)	50%)	
		00%)		
		00%)		
		3.8%)	76.2%)	
		43%)	57%)	
l TIMI Flow (0-III)		40.2%)	59.8%)	001
		3.3%)	6.7%)	
		69.8%)	30.2%)	
		00%)		
l MBG Grade (0-2)		41%)	59%)	001
		81.4%)	8.6%)	
			00%)	
Flow Before Stenting (I-III)			00%)	001
		43.8%)	56.2%)	
		78.9%)	1.1%)	
MBG Grade (1-3)			100%)	001
		58.6%)	41.4%)	
		85.7%)	4.3%)	

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Table 8: Comparative results for number of vessels involved, level of involvement within the LAD, thrombus burden, TIMI flow and myocardial blush grade - Values are frequency (percentage) of patients. [Left Anterior Descending (LAD) – Thrombolysis in Myocardial Infarction (TIMI) – Myocardial Blush Grade (MBG)]

Thrombus grading according to TIMI thrombus scale revealed a significantly higher thrombus burden in patients with Q waves (group B) [76.2% of patients with grade 4 and 57% with grade 5] versus non Q wave group (group A) [23.8% of patients with grade 4 and 43% with grade 5] (p value <0.001) [Table 8].

Patients with Q waves (group B) showed lower initial TIMI flow grade [59.8% of patients with TIMI 0 and 66.7% with TIMI I flow] as compared to patients with absent Q waves (group A) [40.2% of patients with TIMI 0 and 33.3% with TIMI I flow](p value <0.001) [Table 8].

Initial myocardial blush grade (MBG) was significantly lower in patients with Q waves (group B) [Initial MBG 0, 1: 59%, 18.6%, respectively] versus patients with absent Q waves [Initial MBG 0, 1: 41%, 81.4%, respectively](p value < 0.001). On the other hand, final MBG was considerably higher in patients with absent Q waves (group A) [Final MBG 2, 3: 58.6%, 85.7 %, respectively] when compared to those with Q waves (group B) [Final MBG 2, 3: 41.4%, 14.3%, respectively](p value <0.001) [Table 8].

ole	)UP A Q wave) 94	)UP B vave) 97	alue
ni Score e PTCA	±29	:23	)01
ni Score for LAD e PTCA	:29	:23	)01
ni Score PTCA	:8.53	±7.63	2
ni Score for LAD PTCA	±7.66	:7.36	7
Percentage of Diameter Stenosis (%DS) e stenting [%]	4 ±17	±12.13	4
Percentage of Diameter Stenosis (%DS) stenting [%]	2 ±9.37	7 ±10	4

Table 9: Comparative results of the atherosclerotic burden using Gensini score (total score for the three vessels and recalculated score for the LAD vessel alone) and the percentage of diameter stenosis (%DS) assessed by QCA - Values are means ±SD. [Percutaneous Transluminal Coronary Angioplasty (PTCA) – Left Anterior Descending – Quantitative Coronary Analysis (QCA) – Standard Deviation (SD)]

The initial atherosclerotic burden (before percutaneous transluminal coronary angioplasty [PTCA]) as assessed by Gensini scoring model was significantly higher in patients with initial Q waves (group B) [65 ±23 points] compared to those without Q waves (group A) [51.6 ±29 points] (p value <0.001). Reanalysis of the score for the culprit (LAD) vessel alone showed the same statistical significance between Q wave (group B) [64 ±23 points] and non Q wave (group A) [51 ±29 points] patients (p value <0.001) [Table 9].

iable	Sample er	P A wave)	)UP B vave) 97	alue
Thrombus Aspiration Device		..1%)	68.9%)	)01
Balloon Angioplasty		.8%)	42.2%)	)7
Thrombus Aspiration Device and n Angioplasty		3.15%)	51.85%)	)3
t Stenting		..73%)	48.27%)	1

Table 10: Comparative results for the type of percutaneous intervention carried out for the patients - Values are frequency (percentage) of patients.

All our 191 patients underwent final stenting at the end of revascularization procedure.As regard the types of intervention carried out for the patients prior to final stenting; 23.56% of all patients underwent thrombus aspiration alone prior to stenting, without balloon angioplasty. Thrombus aspiration devices were more used in

patients with initial Q waves (group B) [68.9%] compared to patients without Q waves (group A) [31.1%] (P value <0.001) [Table 10].

	iable	)UP A Q wave) 94	)UP B vave) 97	ıe
ntation	ıl CK [U/L]	±400	± 1293	
	MB [U/L]	:51	±182	
	ɔonin-I [ng/ml]	±0.87	±1.15	
Revascularization Value)	ıl CK [U/L]	2 ±1493	7 ±2341	1
	MB [U/L]	±160	±245	1
	ɔonin-I [ng/ml]	' ±1.94	; ±2.37	
s	ıl CK [U/L]	±195	±251	
	MB [U/L]	:8	:18	1
	ɔonin-I [ng/ml]	±1.07	±1.3	

Table 11: Comparative results for the cardiac enzymes (Total CK, CK-MB and Troponin-I) sampled at presentation, after revascularization and after 3 days - Values are means ±SDs. [Primary Percutaneous Coronary Intervention (PPCI) – Creatine Kinase (CK) – Myocardial Band (MB)].

Comparison between both groups for results of sampled total creatine kinase (CK), CK-MB and Troponin-I levels showed that; patients with Q waves (group B) had higher levels for cardiac biomarkers elevation and also higher values for peak cardiac biomarker sampled after revascularization [Table 11].

	Va	GR(	GR(	p
				In-Ho
Primary Endp	Cardiac	1 (1 (Valid n	2 (2 (Valid n	
	Re-infa	3 (3 (Valid n	5 (5 (Valid n	
	Congestive Heart f	3 (3 (Valid n	11 (11 (Valid n	
Combined	Cardiac Death, myocardial re-infarctic congestive heart fa	7 (7 (Valid n	18 (18 (Valid n	
Secondary Endp	LVEDV (ml), mea	128.89 ± (Valid n	127.54 (Valid n	
	LVESV (ml), mea	62.75 ± (Valid n	70.4 ± (Valid n	
	Ejection fraction (E	51.56 (Valid n	45.19 (Valid n	<
	Wall Motion Score Index (\	1.38 (Valid n	1.57 (Valid n	<
				Six Month Follo
Primary Endp	Cardiac	2 (2 (Valid n	5 (5 (Valid n	
	Recurrent myocardial infa	1 (1 (Valid n	3 (3 (Valid n	
	Target Vessel Revasculari	1 (1 (Valid n	3 (3 (Valid n	
	Congestive Heart f	8 (8 (Valid n	26 (28 (Valid n	<
Combined	Cardiac Death, recurrent myoc infarction and congestive heart fa	11 (12	34 (37	<
Secondary Endp	LVEDV (ml), mea	119.83 ± (Valid n	117.97 ± (Valid n	
	LVESV (ml), mea	48.81 ± (Valid n	59.59 ± (Valid n	<
	Ejection fraction (E	55.72 ± (Valid n	46.14 ± (Valid n	<
	Wall Motion Score Index (\	0.95 (Valid n	1.27 (Valid n	<

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Table 12: Comparative results for primary/secondary endpoints and combined MACE - Values are means ±SDs or frequency (percentage) of patients. [Left ventricular end-diastolic volume (LVEDV) - Left ventricular end systolic volume (LVESV) - Standard Deviation (SD) - Major adverse cardiac events (MACE)]

Patients with Q waves (group B) showed higher occurrence of congestive heart failure; both during in-hospital stay [11.34% in group B versus 3.19% in group A](p value 0.031) and at 6 months follow up period [28.88% in group B versus 8.79% in group A](p value <0.001). There were no other statistically significant variations between both groups as regards the other primary endpoints (in-hospital re-infarction and cardiac death as well as recurrent infarction, cardiac death and target vessel revascularization at 6 months follow up) [Table 12].

Combined MACE was higher in patients with Q waves (group B) during in-hospital stay [18.55% patients in group B versus 7.44% patients in group A] (p value 0.023) and was significantly higher during the 6 months follow up period [37.48% patients in group B versus 12.04% patients in group A] (p value <0.001) [Table 12].

Analysis of the secondary endpoints during in-hospital stay showed that patients with initial Q waves (group B) had significantly lower ejection fraction [45.19 ±7.43% versus 51.56 ±8.82% in group A patients](p value <0.001), higher Left ventricular end-diastolic volume (LVEDV) [70.4 ±18.44 ml versus 62.75 ±18.96 ml in group A patients](p value 0.005) and higher (worse) wall motion score index (WMSI) [1.57 ±0.27 versus 1.38 ±0.18 in group A patients](p value <0.001) [Table 12].

Similarly at the 6 months follow up period; patients with Q waves at presentation (group B) had significantly lower ejection fraction [46.14 ±14.56% versus 55.72 ±15.53% in group A patients](p value <0.001), higher Left ventricular end-diastolic volume (LVEDV) [59.59 ±22.77 ml versus 48.81 ±18.39 ml in group A patients](p value <0.001) and higher (worse) wall motion score index (WMSI) [1.27 ±0.43 versus 0.95 ±0.25 in group A patients](p value <0.001) [Table 12].

Va	GR((No Q	GR((Q	p
In-Hospital Mo	1 (1 (Valid n	2 (2 (Valid n	
Mortality At 6 months Foll	2 (2 (Valid n	5 (5 (Valid n	
Total Mo	3 ((Valid n	7 ((Valid n	

Table 13: Comparative results for mortality

As mentioned earlier there were no statistically significant differences between both groups of patients regarding mortality; whether in-hospital mortality (2.06% versus 1.06%, p value 0.579, in the Q wave versus non Q wave, respectively) or mortality at 6 months follow up (5.26% versus 2.15%, p value 0.26, in the Q wave versus non Q wave, respectively). Further analysis of total mortality came up with the same result (70% versus 30%, p value 0.212, in the Q wave versus non Q wave, respectively) [Table 13].

	ctors Detected on Univariate Analysis	ıe
pital MACE	Age	
	r Total Hospital stay	1
	CCU stay	1
	ıal Segment Involvement	
	Myocardial Blush Grade (0-1)	
	ts with Utilized Thrombus Aspiration	
	ts not receiving Intracoronary IIb/IIIa inhibitors	
	Gensini Score Before PTCA	
	LVESV at 2 hours post revascularization	
	EF at 2 hours post revascularization	1
	WMSI at 2 hours post revascularization	
	CK-MB Level after Revascularization (Peak value)	
	Total CK level at 3 days	
	Troponin I level after Revascularization	
At 6 months follow up	Thrombus grade TIMI thrombus grade 5	
	TIMI flow grade (0-1)	
	Myocardial Blush Grade (0-1)	
	Myocardial Blush Grade (1-2)	1
	ce of In-hospital congestive heart failure	1
	r Total Hospital stay	1
	r Time from Onset of symptoms to presentation	1
	r Time from Onset of symptoms to balloon	1
	Gensini Score Before PTCA	1
	LVESV at 2 hours post revascularization	1
	EF at 2 hours post revascularization	1
	WMSI at 2 hours post revascularization	1
	LVESV at 6 months	1
	EF at 6 months	1
	WMSI at 6 months	1

Table 14: Predictors of combined MACE (In-Hospital and 6 months) on univariate analysis - [Coronary Care Unit (CCU) – Percutaneous Transluminal Coronary Angioplasty (PTCA) - Left ventricular end systolic volume (LVESV) – Ejection Fraction (EF) – Wall Motion Score Index (WMSI) - Creatine Kinase (CK) – Myocardial Band (MB) - Thrombolysis in Myocardial Infarction (TIMI)]

Multiple factors were found to be affecting the in-hospital combined MACE on univariate analysis (Table 14). These factors were introduced in a stepwise logistic regression model (multivariate analysis) which showed 4 independent predictors for in-hospital combined MACE. Those independent predictors were; lack of administration of intracoronary glycoprotein IIb / IIIa(B coefficient – 2.715, 95% confidence interval [CI] –5.431 to 0.001, p value 0.046), longer total hospital stay (B coefficient 1.224, 95% CI 0.586 to 1.862, p value <0.001), longer CCU stay (B coefficient 1.541, 95% CI 0.419 to 2.663, p value <0.006) and lower EF at 2 hours post revascularization (B coefficient –0.35, 95% CI –11.016 to 10.316, p value <0.001) [Table 15].

	Independent Predictors on Multivariate An	B Coeff	Confidence Interv	p
In-Hos Comb	Patients not receiving Intracoronary IIb/IIIa inhi	–	–5.431 to	
	Longer Total Hospit		0.586 to	<
	longer CC		0.419 to	
	Lower EF at 2 hours post revasculari		–11.016 to 1	<
6 months Comb	Absence of In-hospital congestive heart f		–4.136 to –	<
	Lower EF at 6 n	–	–0.367 to –	<
	Higher WMSI at 6 n		0.508 to	<

Table 15: Independent predictors of combined MACE (In-Hospital and 6 months) on multivariate analysis - [Major Adverse Cardiac Events (MACE) - Coronary Care Unit (CCU) –Ejection Fraction (EF) – Wall Motion Score Index (WMSI)]

Predictors of Total Mortality on Univariate An	p
Old	
Longer Door to Balloon	

Table 16 Predictors of total mortality on univariate analysis

Independent Predictor of Total Mor on Multivariate An	B Coeff	Confidence Interv	p
Advance		0.62 to	

Table 17: Independent predictor of total mortality on multivariate analysis

Other several factors were found to be affecting the 6 months follow up MACE on univariate analysis (Table 14). These factors were introduced in a stepwise logistic regression model (multivariate analysis) which showed that; absence of in-hospital congestive heart failure (B coefficient –2.35, 95% confidence interval [CI] –4.136 to –0.564, p value 0.008), lower EF at 6 months (B coefficient –0.263, 95% CI –0.367 to –0.159, p value <0.001)and higher WMSI at 6 months (B coefficient 3.242, 95% CI 0.508 to 5.976, p value <0.018) [Table 15].

Univariate analysis for the predictors of total mortality was carried out showing that mortalities; were significantly older (p value 0.001) and had longer door to balloon time (p value 0.013)[Table 16]. The 2 variables were introduced in a stepwise logistic regression model, which showed that advanced age of the patient was an independent predictor of total mortality (B coefficient 0.168, 95% CI 0.62 to 0.274, p value <0.002) [Table 17].

DISCUSSION

Our current study adds to the growing body of evidence of the importance of assessing Q wave status in the risk stratification of a patient with ST segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PPCI) and questions the impact of initial Q waves on cardiac mortality in this subset of patients.

Prognostic value of Q waves has been studied extensively over time regardless to the reperfusion method, whether it was thrombolytic therapy or PPCI. In the era of pharmacological reperfusion, the presence of Q waves carried poor prognostic outcomes. New Q waves were associated with a lower ejection fraction, a larger end systolic volume index (Andrews et al., 2000), increased incidence of congestive heart failure (Waks et al., 2014) and increased cardiac mortality at 30 days (Andrews et al., 2000, Wong et al., 2006 and Waks et al., 2014).

After establishing solid evidence that the outcome of mechanical reperfusion in STEMI patients was more favorable than that found with thrombolytic therapy, there were reasonable expectations that the prognostic impact of Q waves would therefore be more favorable with PPCI. However, the data regarding Q wave prognosis in the PPCI era were not concurring with such hypothesis and the outcomes were almost as poor as those extrapolated in the thrombolysis era. Patients with initial baseline Q waves showed higher CHF and mortality at 90 days (Armstrong et al., 2009 and Bao et al., 2014) and at 1 year follow up (Kumar et al., 2009) versus those without baseline Q waves.

Further analysis of the studies carried out in the PPCI era showed considerable variation in the inclusion criteria and baseline characteristics of the included patients. In our current study we tried to narrow the scope

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of high risk baseline characteristics - in this high risk category of anterior wall STEMI patients - to the least possible assuming that we might reach different results.

RESULTS CONCORDANT WITH PRIOR STUDIES

The results of our study showed that patients with initial Q waves on presentation had; more time from onset of symptoms to presentation, more door to balloon time (DTBT) and hence more total ischemic time than patients without initial Q waves. This finding was concordant with 2 prior large sample population studies, both studies enrolled patients from the APEX-AMI trial (Assessment of Pexelizumab in Acute Myocardial Infarction) and the PLATO trial (Platelet inhibition and patient outcome), respectively (Armstrong et al., in 2009 and Siha et al., 2012). The increased incidence of Q wave formation with prolonged total ischemic time appears to be a logical finding. Having insufficient myocardial perfusion due to complete, prolonged and unrelieved blockage of myocardial blood supply, directs QRS complex away from the involved region, resulting in formation of Q waves (Sclarovsky et al., 1999). The above finding was even more pronounced in our study as we only included patients with incident myocardial infarction; those patients are usually not protected by any form of ischemic preconditioning whether at the epicardial (collateral flow) or cellular levels (myocardial oxygen demand).

We also found that patients with Q waves had higher levels of baseline cardiac biomarkers (total CK, CK-MB and troponin-I levels) and higher peak cardiac biomarkers sampled after revascularization than those without Q waves. These findings were consistent across all prior studies carried out on in the PPCI era (Armstrong et al., in 2009, Kumar et al., 2009 and Pieter et al., 2009).The extent of necrosis expressed in the level of elevation of cardiac biomarkers is strongly influenced by the duration of symptoms. Patients with initial Q waves have longer periods of insufficient myocardial perfusion, leading to more cellular damage, expressed in higher levels of cardiac biomarkers elevation. Time will always serve as a key clinical proxy for the stage of evolution of STEMI (Armstrong et al., 2009).

The results from our angiographic data showed that patients with Q waves had significantly worse grade (0/1) initial TIMI flow as well as higher incidence of grade 0 initial myocardial blush and grade 1 final myocardial blush. These findings were similar in prior studies that showed lower pre-PCI TIMI flow of (0/1) (Armstrong et al., in 2009 and Pieter et al., 2009) and worse (grade 1) myocardial blush in patients with baseline Q waves when compared to those without Q waves (Pieter et al., 2009).

This correlation between increased incidence of baseline Q waves and the poor pre PCI culprit flow; whether epicardial TIMI flow or myocardial blush grade is probably due to the longer overall ischemic time, less optimal myocardial perfusion, more extensive microvascular damage and hence larger areas of jeopardized myocardium. All those factors will probably lead to more incidence of Q wave occurrence.

Our data analysis showed no statistical significance between Q wave and non Q wave groups regarding; in-hospital myocardial re-infarction, recurrent myocardial infarction at 6 months follow up and occurrence of target vessel revascularization (TVR) at 6 months follow up. The above findings were concordant with a prior study which evaluated in-hospital re-infarction and one year follow up of recurrent infarction/repeat revascularization (Kumar et al., 2009). The most reasonable explanation for this finding is that the clinical efficacy of primary PCI with the use of intracoronary stents limited the risks of early re-occlusion and late restenosis when compared to primary PCI with balloon angioplasty. This eventually led to significantly lower incidence of re-infarction and target vessel revascularization (Nordmann et al., 2005).

Patients with initial Q waves showed lower ejection fraction (EF), higher left ventricular end systolic volumes (LVESV) and higher wall motion score index (WMSI); this significant variation was found in both, in-hospital and 6 months echocardiographic examination. A similar observation was seen in the study carried out by Kumar et al. in 2009 who found lower values of EF and higher incidence of resting wall motion abnormalities (ranging between hypokinetic to dyskinetic segments) in patients with initial Q waves (Kumar et al., 2009). Although the methods of assessment for RWMA in such study was more subjective compared to a quantitative wall motion index score analysis, however the results of both studies elaborate the deleterious effect of presence of Q waves on the left ventricular function.

The incidence of congestive heart failure was higher during the in-hospital assessment and significantly higher at the 6 months follow up in patients with initial Q waves versus the non Q wave group. This finding is concordant with almost all the studies examining the prognostic impact of initial Q waves in STEMI patients

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regardless to the method of revascularization (Armstrong et al., in 2009, Kumar et al., 2009 and Pieter et al., 2009).

The pronounced impact for initial Q waves in STEMI patients on the higher incidence of congestive heart failure can be explained by the strong association between Q waves and lower left ventricular systolic function as assessed by left ventricular ejection fraction (LVEF) and left ventricular end systolic volume (LVESV).

The composite MACE [including cardiac mortality, repeat myocardial infarction and congestive heart failure] was higher in the Q wave group during the in-hospital assessment and significantly higher at the 6 months follow up analysis. This finding was also similar to that seen by Kumar et al. in 2009 for the in-hospital combined MACE (16.7% and 4.5% in Q wave versus non Q wave patients, respectively, p value of <0.001) and also at one year follow up period (25.7% and 7.2% in Q wave versus non Q wave patients, respectively, p value of <0.001) (Kumar et al., 2009).

One of the interesting observations detected in our study analysis was the close pattern of statistical significance between composite MACE and congestive heart failure results. This observation appeared relatively reasonable upon comparing the univariate and multivariate predictors for congestive heart failure and composite MACE, where both endpoints had the same exact predictors. This strongly suggests that the poor outcome in Q wave patients regarding composite MACE is secondary to the adverse outcome of congestive heart failure.

Another interesting point was seen during the multivariate analysis; we found that the absence of in-hospital CHF was one of the independent predictors for increased incidence of CHF assessed at the 6 months follow up. The suggested explanation for such finding is probably the lack of optimization of the anti-failure treatment during in-hospital stay by the treating physician and the lack of patient's compliance in receiving the treatment and doing the post discharge follow up. On the other hand patients who suffered manifestations of in-hospital congestive heart failure, received more adequate and detailed plan from the treating physician regarding their anti-failure management whether medical treatment or participation in a post discharge heart failure program.

DEBATABLE WITH PRIOR STUDIES

There was no statistical significance between both groups regarding age, gender and risk factors for coronary artery disease (diabetes mellitus, hypertension, dyslipidemia and smoking). Comparing our results regarding the baseline characteristics with prior studies did not come up with a uniform result. One study concurred with our results (Kumar et al., 2009); this study shared a closer sample number compared to our study (232 patients vs. 191 in our study). While another study with a significantly larger sample number (4,530 patients) found that patients with Q waves showed more incidences of male gender and diabetes mellitus (Armstrong et al., 2009). The most apparent reason for having such discrepancy was the large difference in sample number.

RESULTS DISCONCORDANT WITH PRIOR STUDIES

The results of our study did not find any effect for initial Q waves on mortality in STEMI patients undergoing primary PCI; whether in-hospital or at 6 months follow up. Further analysis for predictors of total mortality was carried out using univariate analysis and showed that mortalities were significantly older and had longer door to balloon time. Those 2 variables were introduced in a stepwise logistic regression model (multivariate analysis), which showed that advanced age of the patient was the only independent predictor of total mortality.

The above data doubts a considerable finding from prior studies assessing the impact of initial Q waves in STEMI patients on mortality. Our results are discordant with a number of studies carried out in the era of primary PCI (Armstrong et al., 2009, Kumar et al., 2009 and Pieter et al., 2009).The most pronounced variation between our current study and those prior studies is confined to the selection criteria of studies, mainly involving three considerable differences.

The first difference is the clinical status of the included patients; our study excluded all patients with a Killip clinical class higher than one. On the other hand patients with Killip class > I were included in prior studies (Armstrong et al., 2009).

A considerable number of prior studies revealed that, the higher the Killip class on presentation, the greater the subsequent mortality (Wu et al., 2002, Steg et al., 2004 and De Luca et al., 2004). A study from the Second National Registry of Myocardial Infarction (NRMI-2) included 190,518 patients with acute MI, of whom 36,303

(19%) had Killip class II or III HF on admission. These patients had significantly higher in-hospital mortality than those without HF [21.4% versus 7.2%] (Wu et al., 2002).

Similar findings were noted in an analysis of international data on 4830 patients with STEMI from the GRACE (Global Registry of Acute Coronary Events) registry. Sixteen percent had Killip class II or III HF on admission; patients with HF had increased mortality in-hospital (17% versus 4%) as well as from discharge to six months [20% versus 3%] (Steg et al., 2004). In one series of 1548 patients undergoing primary angioplasty, increasing Killip class was associated with increasing mortality at one year, an association that appears to have been due to a higher incidence of suboptimal myocardial perfusion (De Luca et al., 2004).

The other two differences were concerning the adequacy of revascularization post procedure; obtaining a final TIMI III flow at the end of the procedure and having ≥ 70% ST segment resolution in the post procedural ECG. All our study patients showed final TIMI III flow, whereas other studies included patients with TIMI flow < III [27.1% in the Q wave arm and 7.1% in the Non Q wave arm, p value < 0.001] (Pieter et al., 2009).

Suboptimal reperfusion, as estimated in part from the post-PCI TIMI flow grade, is an important determinant of prognosis after PPCI. Mortality is worse after PPCI in the small number of patients who do not attain TIMI 3 flow (Berger et al., 1999, Mehta et al., 2003 and Ernst et al., 2005).

In a review from the PAMI (Primary stenting in Acute Myocardial Infarction) trials, 232 of 3362 patients (6.9%) did not attain TIMI 3 flow; the adjusted one-year mortality in those with and without TIMI 3 flow was 3% versus 14%; most of this difference occurred within the first 30 days. Lack of attainment of TIMI 3 flow was a significant independent predictor of mortality (Mehta et al., 2003).

Also we excluded those patients with an ST segment resolution of less than 70% in the post procedural ECG, which was not the case in other two studies with different mortality outcome, including patients not achieving ≥ 70% ST segment resolution in the post procedural ECG. One study had 75% in the Q wave arm and 56% in the non q wave arm [p value <0.001] (Kumar et al., 2009); the other study included 57% patients in the Q wave arm and 38.3% patients in the non Q wave arm [p value < 0.001] (Armstrong et al., 2009).

One study showed that inadequate STR immediately after PCI was one of the multivariable predictors of the composite end points, both in-hospital and at 1 year follow up (including death, repeat MI or heart failure) [p <0.001] (Kumar et al., 2009).

The other study suggested that the strong relationship between lesser ST-segment resolution after PCI and Q-wave presence indicates less successful myocardial reperfusion with PCI and is further supported by the greater peak biomarkers for myocardial necrosis. Hence, such patients would be expected to have less salvageable myocardium and worse clinical outcomes (Armstrong et al., 2009).

LIMITATIONS

Our study had a considerable number of limitations including, the observational nature of the study, being a single-center study and having a limited sample number. However, despite the limited sample size, we have observed statistically significant and clinically relevant differences between patients with and without baseline Q waves.

The ECG entry and the trial inclusion criteria, although broad, are not necessarily generalizable to all patients with STEMI. This is due two main factors. The first factor is exclusion of a considerable number of patients who had previous MI, stenting, or bypass surgery to enable best correlation between Q-wave status and infarct wall motion, making the results applicable only to those presenting with an incident STEMI.

The second factor is performing the study on patients with anterior wall STEMI only (excluding inferior and posterior infarctions). We did so to also have a better evaluation of Q wave impact on the outcome by choosing a population that suffered STEMI involving the larger area of myocardium; however this also confines our results to patients with larger area of jeopardized myocardium and should not apply to all STEMI patients.

CONCLUSIONS&RECOMMENDATIONS

Baseline Q waves in STEMI patients is a simple, readily accessible and cost-effective method of assessing short-term (in-hospital) and intermediate-term (6 months) MACE. The study adds prospective confirmation

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about the prognostic value of baseline Q waves in patients with anterior ST-segment elevation who presented within 12 hours of the onset of symptoms. Presence of baseline Q wave is a poor prognostic sign associated with increased congestive heart failure, decreased LVESV, higher WMSI and lower EF, whether in-hospital or at 6 month follow up. Independent predictors for in-hospital composite MACE were exactly the same predictors for in-hospital congestive heart failure. Those independent predictors were; lack of administration of intracoronary glycoprotein IIb / IIIa, longer total hospital stay, longer CCU stay and lower EF at 2 hours post revascularization.

We did not find any impact for baseline Q waves on the mortality, whether in-hospital or at 6 months follow up period. This finding was extrapolated in patients with a lower risk of clinical presentation (Killip class I) and in those achieving adequate revascularization (final TIMI III flow and STR ≥ 70%). The only independent factor for mortality was advanced age of the patient.

These findings suggest that Q waves do not impact 6-month survival, and may reflect the beneficial impact of current medical and reperfusion strategies. However the deleterious effects for Q waves on the left ventricular function and its high association with increased incidence of congestive heart failure should withdraw attention towards the importance of optimizing anti-failure management for this category of patients.

We recommend that future studies challenge our results regarding the Q wave impact on cardiac mortality. The assessment of Q waves in STEMI patients with relatively lower clinical risk and better reperfusion outcomes should be carried out with inclusion of more STEMI patients. This can be more applicable with collaboration of more than one center with primary PCI capabilities, to enhance pooling together all the necessary data for extrapolating more statistically powerful results.

LIST OF TABLES

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ABBREVIA	
	Perc
	Two-dimen
	Apical two-ch
	Apical four-ch
	American College of Cardi
	American College of Cardiology Foun
APEX-AM	Assessment of Pexelizumab in Acute Myocardial Infa
	Coronary Artery Bypass Gr
	Coronary Car
	Congestive Heart F
	Creatine
C	Creatine Kinase-Myocardia
	Diameter St
	Door To Balloon
	Electrocardio
	Ejection Fra
	Family H
GRACE reg	Global Registry of Acute Coronary
IBM corpor	International Business Ma
	Ischemic Heart D
	Pota
	Left Anterior Desce
	Left Bundle Branch
	Left Circu
	Lef
	Left Ve
L	Left Ventricular End Diastolic v
L	Left Ventricular End Systolic vo
	Media Access C
	Major Adverse Cardiac
	Myocardial Blush
	Myocardial Infa
	Minimal Luminal Dia
	Mi
	Milli
	Millis
	M
	Samp
	Nano
N	National Registry of Myocardial Infarctio
PAM	Primary Angioplasty in Myocardial Infa
	Percutaneous Coronary Interv
PLATO	PLATelet inhibition and patient Outc
	Primary Percutaneous Coronary Interve
	PercutaneousTransluminal Coronary Angio

	Quantitative Coronary Ar
	Right Coronary A
	Reference Vessel Dia
R	Regional Wall Motion Abnorma
	Standard Dev
	Statistical Package for the Social Sc
	ST segment Reso
	Thrombolysis In Myocardial Infa
	Target Vessel Revasculari
	Unit per
	World Heart Fede
	Wall Motion Score

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