Association Between Early Q Waves and Reperfusion Success in Patients With ST-Segment–Elevation Myocardial Infarction Treated With Primary Percutaneous Coronary Intervention

A Cardiac Magnetic Resonance Imaging Study

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Background—Pathological early Q waves (QW) are associated with adverse outcomes in patients with ST-segment–elevation myocardial infarction (STEMI). Primary percutaneous coronary intervention (PCI) may therefore be less beneficial in patients with QW than in patients without QW. Myocardial salvage index and microvascular obstruction (MVO) are markers for reperfusion success. Thus, to clarify the benefit from primary PCI in STEMI patients with QW, we examined the association between baseline QW and myocardial salvage index and MVO in STEMI patients treated with primary PCI.

Methods and Results—The ECG was assessed before primary PCI for the presence of QW (early) in 515 STEMI patients. The patients underwent a cardiac magnetic resonance imaging scan at day 1 (interquartile range [IQR], 1–1) and again at day 92 (IQR, 89–96). Early QW was observed in 108 (21%) patients and was related to smaller final myocardial salvage index (0.59 [IQR, 0.39–0.69] versus 0.65 [IQR, 0.46–0.84]; $P<0.001$) and larger MVO (1.4 [IQR, 0.0–5.4] versus 0.0 [IQR, 0.0–2.4]; $P<0.001$) compared with non-QW. QW remained associated with both final myocardial salvage index ($\beta=-0.12$; $P=0.03$) and MVO ($\beta=0.18$; $P=0.001$) after adjusting for potential confounders.

Conclusions—Patients presenting with their first STEMI and early QW in the ECG had smaller myocardial salvage index and more extensive MVO than non-QW despite treatment within 12 hours after symptom onset. However, final myocardial salvage index in patients with QW was substantial, and patients with QW still benefit from primary PCI.

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Key Words: catheterization ■ infarction ■ magnetic resonance imaging ■ myocardium ■ reperfusion

ST-segment–elevation myocardial infarction (STEMI) is treated with timely primary percutaneous coronary intervention (PCI) to improve the prognosis by reducing final infarct size and improving myocardial salvage.[1] In STEMI patients, cardiac magnetic resonance (CMR) has become an important method to quantify area at risk,[2–5] infarct size,[5,6] and myocardial salvage index.[2,5,7] CMR does also allow for assessment of microvascular damage (microvascular obstruction [MVO]) within the infarct area.[8,9] Myocardial salvage index and MVO are both predictors for outcome in STEMI patients treated with primary PCI and therefore considered markers of successful reperfusion.[10]

A pathological Q wave (QW) in the ECG before reperfusion (early QW) is a frequent observation in patients presenting with STEMI.[11] The presence of QW is considered a surrogate for transmural infarction, whereas the absence of QW is interpreted as a sign of subendocardial infarction.[12–15] However, previous studies have found transmural infarction in some patients

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WHAT IS KNOWN

- The presence of a pathological Q wave (QW) before primary percutaneous coronary intervention (PCI), early QW, is an observation in around 1 in 5 patients with ST-segment–elevation myocardial infarction (STEMI) and is associated with larger infarct size and adverse prognosis.
- Patients with early QW and STEMI benefit to a lesser extent from primary PCI compared with thrombolysis.
- Thus, the effect of primary PCI in patients with early QW and STEMI is not well described.

WHAT THE STUDY ADDS

- STEMI patients with early QW treated with primary PCI have smaller myocardial salvage and larger microvascular obstruction as assessed by cardiac magnetic resonance imaging.
- Myocardial salvage, however, was still >50%, indicating that this patient group benefits from primary PCI and should undergo immediate angioplasty.

without QW and nontransmural infarction in some patients with QW.[16,17] Several studies using a postreperfusion ECG (late QW) to evaluate QW have shown that QW is related to larger myocardial infarction size than non-QW myocardial infarction.[16,18,19] Furthermore, the presence of early QW in patients with STEMI treated with reperfusion (thrombolysis or primary PCI) is related to worse clinical outcome.[11,20–23] Interestingly, the benefit from reperfusion with primary PCI compared with thrombolysis seems to be attenuated in patients with early QW.[24] It may therefore be speculated that patients with QW STEMI benefit to a lesser extent from primary PCI than non-QW. Evaluating the association between QW and myocardial salvage index and MVO may help to clarify the benefit from primary PCI in STEMI patients with baseline QW. However, to the best of our knowledge, there exist no data on the association between early QW and myocardial salvage index and MVO after primary PCI. Thus, the aim of this study was to evaluate the association between early QW and myocardial salvage index and MVO assessed by CMR in a contemporary STEMI cohort treated with primary PCI.

Methods

Study Population

Patients were included in the randomized study DANAMI-3 (The Third Danish Study of Optimal Acute Treatment of Patients With ST-Segment–Elevation Myocardial Infarction).[25] The DANAMI-3 trial program encompasses 3 randomized trials evaluating DANAMI-3-iPOST (ischemic postconditioning),[25] DANAMI-3-DEFER (deferred stenting),[26] and DANAMI-3-PRIMULTI (multivessel revascularization).[27] In both DANAMI-3-iPOST and DANAMI-3-DEFER, the treatment allocation had a neutral effect on the primary end point.[25,26] Patients with their first STEMI treated with primary PCI constituted the study population and were studied prospectively by ECG and CMR. The inclusion criteria were patients aged >18 years and arrival to the catheterization laboratory within 12 hours from symptom onset of STEMI. STEMI was defined as ST elevation ≥1 mm in at least 2 contiguous leads in I, II, III, aVF, aVL, V_4 through V_6 and ST elevation ≥2 mm in at least 2 contiguous leads in V_1 through V_3.

The exclusion criteria to this study were in accordance with DANAMI-3 trial.[25] In addition, the exclusion criteria in this study were previously known myocardial infarction, contraindication toward CMR, and any cardiac disease that may cause QW (hypertrophic cardiomyopathy, left bundle branch block, Wolff–Parkinson–White syndrome, left ventricular (LV) hypertrophy, and dilated cardiomyopathy).

The patients were pretreated with aspirin (300 mg orally or 500 mg intravenously), clopidogrel (600 mg orally) or prasugrel (60 mg orally), and unfractioned heparin (10 000 U intravenously) in the prehospital setting or at the referring hospital. When arriving to the hospital, the patients were randomized, and subsequently coronary angiography was performed to identify the culprit lesion. Thrombectomy, stents, bivalirudin and glycoprotein IIb/IIIa receptor antagonists was used at the operators discretion. Stenting during the initial PCI was not allowed for the patient randomized to deferred stenting. If contraindications for bivalirudin were not present, it was given to the patient before or during PCI. All patients were post-treated with aspirin (75 mg daily and lifelong) and clopidogrel (75 mg in 12 months) or prasugrel (10 mg in 12 months). All patients gave written informed consent before inclusion in the study,[26] and the study was approved by the national ethical committee. The study was done in accordance with the Helsinki declaration.

ECG Analysis

An observer blinded for CMR data or any other clinical or paraclinical data performed the ECG analysis manually. On arrival at the catheterization laboratory, continuous 12-lead ST monitoring was

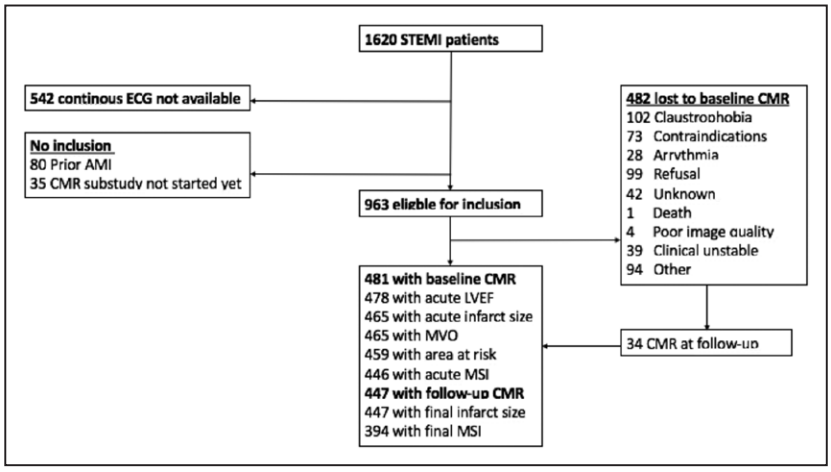

Figure 1. Flow chart of patient inclusion. AMI indicates acute myocardial infarction; CMR, cardiac magnetic resonance; LVEF, left ventricular ejection fraction; MSI, myocardial salvage index; MVO, microvascular obstruction; and STEMI, ST-segment–elevation myocardial infarction.

Table 1. Baseline Clinical, Angiographic, and Procedural Characteristics

	No-QW (pre-PCI; n=407)	QW (pre-PCI; n=108)	*P* Value
Age, y	59 (±7)	59 (±7)	0.56
Male	321 (79%)	86 (80%)	0.86
Cardiovascular risk factors			
Diabetes mellitus	33 (8%)	13 (12%)	0.20
Hypertension	136 (34%)	35 (32%)	0.83
Hypercholesterolemia	127 (31%)	39 (36%)	0.33
Current smoker	221 (54%)	69 (64%)	0.08
Multivessel disease	176 (43%)	53 (49)	0.29
Previous PCI	6 (2%)	4 (4%)	0.14
Symptom onset to wire, min	169 (127–268)	202 (132–340)	0.006
First ECG with STEMI to wire	87 (69–116)	99 (74–127)	0.09
Thrombectomy	246 (60%)	68 (63%)	0.63
TIMI (pre-PCI)			<0.001
0/1	223 (55%)	83 (77%)	
2/3	184 (45%)	25 (23%)	
TIMI (post-PCI)			0.013
0/1	6 (1.5%)	0 (0%)	
2/3	400 (98.5%)	108 (100%)	
Killip class at presentation			0.003
I	361 (96%)	90 (87%)	
II	15 (4%)	13 (13%)	
III	0 (0%)	0 (0%)	
IV	1 (0.3%)	1 (1%)	
Heart rate on admission, bpm	72 (60–85)	79 (67–87)	0.06
Systolic blood pressure on presentation, mm Hg	132 (117–149)	129 (108–149)	0.18
Anterior infarct location	177 (44%)	44 (41%)	0.58

Data are presented as n (%), mean±SD or median (interquartile range). PCI indicates percutaneous coronary intervention; QW, Q wave; STEMI, ST-segment–elevation myocardial infarction; and TIMI, Thrombolysis in Myocardial Infarction.

initiated, which was continued for a minimum of 90 minutes after the primary PCI (LIFEPAK 12; Medtronic Emergency Response Systems, Redmond, WA). The software generated a complete 12-lead ECG when the ST deviated with >0.1 mm for at least 2.5 minutes. The ST elevation analysis was measured by a computer software program (CODE-STAT Suite version 6.1; Medtronic Emergency Response Systems) in the J point +1/16 of the RR interval. ST elevation, resolution, and QW were analyzed in the single lead with highest ST elevation before PCI. The ECG on arrival at the catheterization laboratory before primary PCI was used to evaluate the presence of a QW defined according to the modified Minnesota criteria (QW duration ≥40 ms and a QW depth ≥25% of the R wave in the same lead in at least 2 contiguous leads).[18] The patients were divided into 2 groups (non-QW and QW).

CMR Imaging

Two CMR scans were performed, the initial scan during the index admission with median of 1 day (interquartile range, 1–1) after the primary PCI and the second scan 3 months after primary PCI (median, 92 days; interquartile range, 89–96). Both scans were performed on a 1.5-T scanner (Avanto or Espree scanner; Siemens, Erlangen, Germany).

To quantify area at risk, we assessed myocardial edema using T2-weighted images on the first scan. Acute infarct size was assessed on the first scan and final infarct size on the second scan with late gadolinium-enhanced images obtained 10 minutes after intravenous injection of 0.1 mmol/kg body weight gadolinium-based contrast (Gadovist; Bayer Schering, Berlin, Germany) using an ECG-triggered inversion recovery sequence. The inversion time was continuously adjusted to null the signal from the normal myocardium. All images were obtained in the short-axis plane from the atrium–ventricle plane to the apex with 8 mm slice thickness and 0 mm slice gap covering the entire LV.

Table 2. ST-Segment Resolution at 60 Minutes After Reperfusion for Patients With and Without Pathological QW in the ECG Before PCI

	No-QW (pre-PCI; n=398)	QW (pre-PCI; n=107)	*P* Value
70% ST resolution (yes)	153 (38)	46 (43)	0.39
ST elevation			0.004
<0.1 mV	262 (66)	52 (48)	
0.1–0.2 mV	94 (23)	40 (37)	
>0.2 mV	45 (11)	16 (15)	
Presence of QW 60 min after reperfusion (yes)	62 (16)	102 (94)	<0.001
ST-segment–elevation, mV	0.27 (0.14–0.46)	0.37 (0.21–0.72)	<0.001

Data are presented as n (%) or median (interquartile range). PCI indicates percutaneous coronary intervention; and QW, Q wave.

Image Analysis

The CMR images were analyzed by an observer blinded to all data using CVI42 (Circle Cardiovascular Imaging Inc, Calgary, Canada). A second observer reviewed all the CMR analyses. Any major discrepancies were reviewed and discussed and if necessary adjusted until consensus. The epicardial and endocardial contours were manually traced in all images, incorporating the papillary muscles as part of the LV cavity. Infarct size was measured on both scans as hyperenhanced myocardium (signal intensity >5 SDs of the signal intensity in the normal remote myocardium) on the late gadolinium enhancement images and was expressed in grams (absolute infarct size) and as percentage of LV mass (relative infarct size).[28] The infarct transmurality was calculated for each segment in the AHA16 segment model, and the average transmurality was calculated as sum of transmurality for all segments with positive late gadolinium enhancement divided by numbers of segments with positive late gadolinium enhancement. MVO was assessed on the first scan as hypointensive areas within the infarct zone on the late gadolinium enhancement images and was expressed as categorical variable and as percentage of LV. The area at risk was defined as myocardium with signal intensity >2 SDs of the signal intensity in the normal remote myocardium on the T2-weighted images defined. Hypointensive areas within the area at risk were regarded as a part of the area at risk, and diffuse hyperintensive areas in the remote myocardium were excluded.[3] Myocardial salvage index was calculated as follows: (area at risk−infarct size)/area at risk.[29] LV ejection fraction was measured on both scans using short-axis plane cine images and calculated as ([LV end-diastolic volume−LV systolic volume]/LV end-diastolic volume) %.

Statistical Analysis

The statistical comparisons were performed between patients with QW and non-QW in the baseline ECG. Continuous variables were tested for normal distribution by histograms of the standardized residuals and compared using either Mann–Whitney or Student *t* test. Categorical variables were compared with the χ^2 test or Fisher exact test. The relation between QW and final myocardial salvage index and MVO was adjusted for potential confounders in multivariable linear regression analyses using any variable with P <0.05 in the univariable analysis and any baseline variable with P ≤0.20 for the difference between patients with QW and non-QW. A 2-sided probability value <0.05 was considered statistically significant. All statistical analyses were performed with SPSS software version 20.0 (SPSS Inc, Chicago, IL).

Results

Clinical Characteristics

A flow chart of patient inclusion is shown in Figure 1. A total of 515 patients underwent at least 1 CMR and were thus included in this study. The baseline characteristics, stratified by the presence of early QW, are depicted in Table 1. Patients with early QW were more likely to have pre-PCI Thrombolysis in Myocardial Infarction 0/1 flow and a higher Killip class on admission. Also, duration of symptom onset to wire was longer in QW patients compared with non-QW patients. We found no statistically significant associations between the presence of QW and age, sex, or any cardiovascular risk factors.

QW and ECG

The presence of QW before PCI was also associated with the presence of QW 60 minutes after first wire, but some patients with non-QW developed QW after 60 minutes, and 5 patients with early QW had QW resolution (Table 2). QW was related to impaired ST-segment resolution expressed as magnitude of the remaining ST elevation 60 minutes after first wire but not in terms of numbers of patients achieving 70% ST-segment resolution (Table 2). Moreover, presence of QW was associated with larger ST elevations before PCI.

QW and CMR

CMR results are shown in Tables 3 through 5. Patients with early QW had larger infarct size and lower LV ejection fraction in the acute phase and at follow-up when compared with the non-QW patients. The area at risk was also larger among patients with early QW, and accordingly both the acute and final myocardial salvage index were smaller compared with the non-QW patients (Table 3; Figure 2A and 2B). The QW group did also have a higher incidence and greater extent of MVO (Table 3; Figure 3). In terms of patients lost to CMR at follow-up with available baseline CMR, there was no difference between the patients with and without QW (17% versus 12%; P=0.23). However, the importance of QW was most pronounced in patients with anterior infarct location compared with noninfarct location (Table 4). Whereas the presence of QW remained associated with outcome when stratifying the patients according to symptom onset to wire <3 and >3 hours (Data Supplement). Furthermore, the association between QW and myocardial salvage index and the extent of MVO (% LV) remained statistically significant when adjusting for potential confounders in multivariable regression models, including infarct location (Table 5). Owing to linearity between first

Table 3. CMR Outcome for Patients With and Without Pathological QW in the ECG Before PCI

	n	No-QW (pre-PCI)	n	QW (pre-PCI)	*P* Value
Acute CMR d 1 (IQR, 1–1)					
Infarct size acute (%)	369	15 (8 to 25)	96	22 (15 to 31)	<0.001
LVEF acute (%)	380	53 (45 to 58)	98	48 (42 to 57)	0.024
Average transmurality acute (%)	362	30 (17 to 41)	95	37 (29 to 45)	<0.001
Presence of MVO, n (%)	369	117 (47%)	96	65 (64%)	0.002
MVO (%LV)	369	0.0 (0.0 to 2.4)	96	1.4 (0.0 to 5.4)	<0.001
Area at risk (%)	367	32 (25 to 39)	92	37 (30 to 45)	<0.001
Myocardial salvage index acute	355	0.48 (0.32 to 0.68)	91	0.38 (0.21 to 0.52)	<0.001
Follow-up CMR d 92 (IQR, 89–96)					
Myocardial salvage index final	320	0.65 (0.46 to 0.84)	74	0.59 (0.39 to 0.69)	0.001
Average transmurality final (%)	341	28 (15 to 39)	86	38 (28 to 46)	<0.001
LVEF follow-up (%)	357	60 (54 to 65)	90	57 (48 to 63)	0.003
Infarct size follow-up (%)	354	11 (4 to 18)	89	17 (10 to 25)	<0.001
Differences over time from baseline to follow-up CMR					
Infarct size (%)	407	–4 (–7.70 to –1.10))	108	–4.70 (–8.90 to –0.10)	0.702
LVEF (%)	407	7 (1.50 to 12.0)	108	5.20 (1.50 to 11.40)	0.660
Myocardial salvage index (%)	407	0.16 (0.06 to 0.26)	108	0.16 (0.05 to 0.20)	0.571

Data are presented as n (%) or median (interquartile range). CMR indicates cardiac magnetic resonance; IQR, interquartile range; LVEF, left ventricular ejection fraction; MVO, microvascular obstruction; PCI, percutaneous coronary intervention; and QW, Q wave.

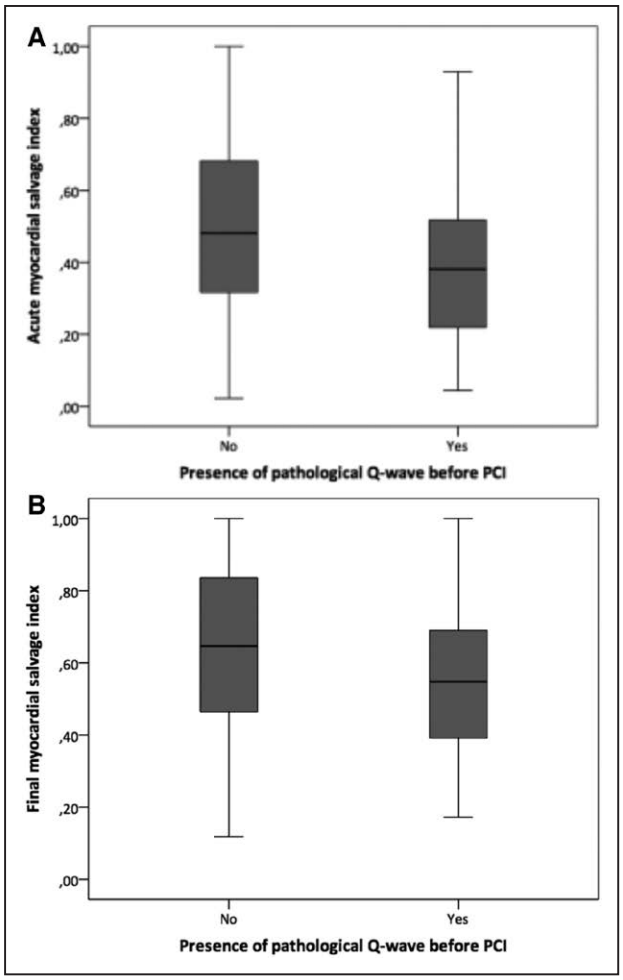

Figure 2. Association between Q wave (QW) and myocardial salvage index. Association between the presence of pathological QW in the ECG before percutaneous coronary intervention (PCI) and acute myocardial salvage index (**A**) and final myocardial salvage index (**B**).

ECG with STEMI to wire and symptom onset to wire, only the latter variable was used in the multivariate models. QW had a stronger association with myocardial salvage index and MVO than time from onset of symptoms onset to wire. There was no interaction between QW and treatment allocation (ischemic postconditioning and deferred stenting).

Discussion

The main finding in this study was that the presence of early QW in patients presenting with a first time STEMI selected for primary PCI within 12 hours after onset of symptoms was related to smaller myocardial salvage index and larger MVO assessed by CMR. However, final myocardial salvage was still substantial in patients with early QW, indicating that patients with STEMI and early QW frequently have favorable outcome after reperfusion despite presumed transmural and irreversible myocardial damage. The presence of early QW in patients presenting with significant ST-segment–elevations within 12 hours after onset of clinical relevant symptoms should therefore not exclude patients from treatment with primary PCI.

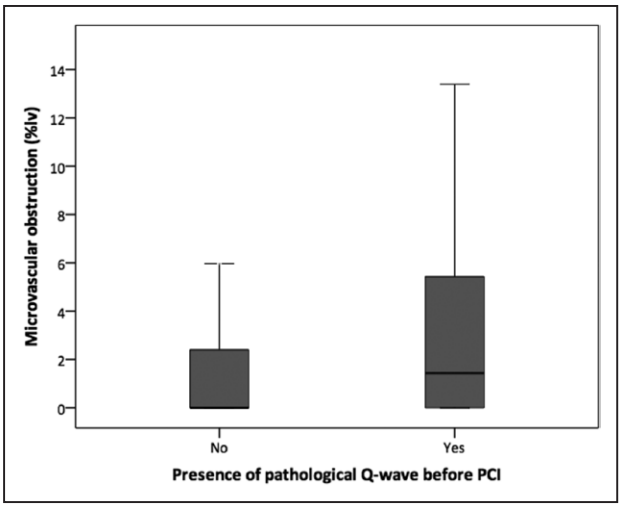

Figure 3. Association between the presence of pathological Q wave in the ECG before percutaneous coronary intervention (PCI) and microvascular obstruction. LV indicates left ventricle.

This study is the first to evaluate the association between early QW and myocardial salvage index and MVO in STEMI patients treated with primary PCI. A previous study assessed QW 60 minutes after the PCI and found that the presence of QW at this time point was significantly associated with MVO.[30] However, a post-PCI ECG will include any reperfusion injury induced QW by PCI itself and can therefore not be used to accurately assess the importance of pre-PCI QW and infarct extension. Moreover, as demonstrated in this study, some patients develop QW after PCI, and others have QW resolution. Previous studies have shown that QW during the course of acute coronary syndrome may be transient manifestations of severe ischemia and regress over time.[18,31,32] Thus, early QW is a sign of severe ischemia rather than irreversible myocardial damage, which is perfectly in line with findings in this study. In accordance with our findings, a previous study assessed QW before primary PCI and found a strong correlation with infarct size and LV ejection fraction measured by CMR.[18] But, it has previously been unknown whether the association between QW and infarct size was the result of larger area at risk leading to larger infarct size or the result of smaller myocardial salvage. In this regard, this study demonstrates that early QW is related to larger infarct size through both larger area at risk and smaller myocardial salvage.

Patients with the presence of QW on presentation did also have longer duration of symptom onset to wire and higher grade of pre-PCI Thrombolysis in Myocardial Infarction 0/1 flow, indicating that these patients have longer duration of ischemia and less spontaneous reperfusion. These factors are also related to larger myocardial damage and smaller salvage and may thus explain some of the findings in this article. Nevertheless, the association of QW with reperfusion success (myocardial salvage index and MVO) remained statistically significant in a multivariate analysis adjusting for other important factors, including symptom onset to wire and pre-PCI Thrombolysis in Myocardial Infarction flow. The results in this article did also not change when the patients were stratified according to the symptom onset to wire <3 or >3 hours. Thus, the presence of QW is related to adverse outcome

Table 4. CMR Outcome for Patients With and Without Pathological QW in the ECG Before PCI According to Anterior or Nonanterior Infarct Location

	n	No-QW (pre-PCI)	n	QW (pre-PCI)	*P* Value
Nonanterior infarct location					
Infarct size acute (%)	154	13 (7–22)	39	17 (13–25)	0.009
Infarct size follow-up (%)	149	10 (4–17)	37	14 (8–19)	0.006
Myocardial salvage index acute	150	0.49 (0.35–0.69)	38	0.41 (0.29–0.57)	0.041
Myocardial salvage index final	131	0.64 (0.46–0.81)	31	0.61 (0.47–0.71)	0.13
MVO (%LV)	154	0.0 (0.0–2.3)	39	0.7 (0.0–3.0)	0.072
Anterior infarct location					
Infarct size acute (%)	213	18 (9–32)	57	29 (22–40)	<0.001
Infarct size follow-up (%)	203	12 (3–22)	52	23 (16–30)	<0.001
Myocardial salvage index acute	203	0.46 (0.29–0.68)	53	0.34 (0.18–0.45)	0.001
Myocardial salvage index final	187	0.65 (0.46–0.87)	43	0.51 (0.29–0.63)	0.002
MVO (%LV)	213	0.7 (0.0–2.9)	57	3.2 (0.9–6.5)	<0.001

Data are presented as n (%) or median (interquartile range). CMR indicates cardiac magnetic resonance; LV, left ventricle; MVO, microvascular obstruction; PCI, percutaneous coronary intervention; and QW, Q wave.

beyond longer duration of ischemia and less spontaneous reperfusion.

Traditionally, time from symptom onset to wire is considered as duration of ischemia,[29] and early QW occurs more frequently in those with delay presentation (>3 hours from symptom onset to wire).[11] Accordingly, QW was in this article related to more extensive myocardial damage, less myocardial salvage, and higher grade of transmurality, as well as longer time from symptom onset to wire. However, symptoms onset to wire was not significantly related to MVO in the multivariate model in this study adjusting for QW, thus QW may represent a more accurate estimate of the success from primary PCI than time from symptom onset to wire. This is in accordance with the previous findings that the benefit from reperfusion by primary PCI compared with thrombolysis seems to be more pronounced among patients with non-QW.[24] Nevertheless, the presence of early QW was in this study still associated with an average transmurality <50% and final myocardial salvage >50%. In addition, LV ejection fraction increased substantially between the acute phase and follow-up at 3 months later in the QW group. Therefore, a considerable myocardial salvage after primary PCI is possible in patients with early QW, and these patients should not be rejected from immediate angioplasty.

Study Limitations

As the study uses the data from DANAMI-3 trials, this study population is already selected for primary PCI based on ECG and clinical presentation leading to a certain risk of selection bias, which is the most important limitation to this study. Also, we used the Minnesota criteria as definition of QW and analyzed the ECG manually (modified Minnesota). Despite the fact that this criterion has a strong association with infarct size,[18] the results could have been different using another definition of QW. In addition, further characterization of the QW in terms of

Table 5. Adjusted Association Between QW and Final Myocardial Salvage Index and MVO

	Acute Myocardial Salvage Index		Final Myocardial Salvage Index		MVO (%LV)	
	β	*P* Value	β	*P* Value	β	*P* Value
Diabetes mellitus	0.04	0.44	0.11	0.039	0.02	0.75
Current smoker	0.00	0.99	0.00	0.98	−0.01	0.83
Previous PCI	−0.03	0.56	−0.08	0.16	−0.05	0.38
Heart rate upon admission	−0.09	0.07	−0.11	0.06	0.04	0.50
Symptom onset to wire	−0.10	0.045	−0.12	0.024	0.09	0.08
Killip class	−0.01	0.81	−0.05	0.35	0.08	0.10
TIMI flow 2/3 vs 0/1 (pre-PCI)	0.42	<0.001	0.36	<0.001	−0.26	<0.001
Anterior infarct location	−0.15	0.004	−0.11	0.05	0.19	<0.001
QW (pre-PCI)	−0.12	0.021	−0.12	0.036	0.18	0.001

LV indicates left ventricular; MVO, microvascular obstruction; PCI, percutaneous coronary intervention; QW, Q wave; and TIMI, Thrombolysis in Myocardial Infarction.

width, height, and duration would be interesting, but this was not done in this study because the aim of this study was to assess the presence of QW in the referring ECG in the most clinically relevant manner in order for the results to be easily translated in daily clinical practice. Patients with known previous myocardial infarction were excluded from this study, but inclusion of patients with unknown previous myocardial infarct cannot be precluded. Moreover, QW is a relatively inaccurate method for assessing reperfusion success, and adding more extensive ECG analyses could help to improve the diagnostic performance of the baseline ECG to evaluate reperfusion success.[24,33] On the other hand, assessing the presence of QW is a simple and an easy method readily useful in clinical practice. Patients with known previous myocardial infarction were excluded from this study, but inclusion of patients with unknown previous myocardial infarction cannot be precluded. Finally, the excluded patients constitute a risk of selection bias because patients with conditions related to larger infarct size and MVO, a smaller myocardial salvage index, and LV ejection fraction could result in some of the reasons for exclusion in Figure 1.

Conclusions

Patients presenting with their first STEMI treated with primary PCI within 12 hours after symptom onset and early QW had smaller myocardial salvage index and more extensive MVO than patients without QW. Thus, for the first time, we show that final myocardial salvage in patients with early QW was substantial and that patients with QW may still benefit from primary PCI. Hence, these patients should not be rejected from primary PCI.

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Disclosures

None.

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Association Between Early Q Waves and Reperfusion Success in Patients With ST-Segment–Elevation Myocardial Infarction Treated With Primary Percutaneous Coronary Intervention: A Cardiac Magnetic Resonance Imaging Study

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Supplemental Table. CMR outcome for patients with and without pathological QW in the ECG with < 3 hours from symptom onset to wire or > 3 hours from symptom onset to wire.

	n	NO-QW (pre-PCI)	n	QW (pre-PCI)	P-value
<3 hours					
Infarct size acute (%)	188	14.9 (8-24)	43	22 (12-31)	0.008
Infarct size follow-up (%)	182	10 (3-17)	38	17 (12-23)	<0.001
Myocardial salvage index acute	177	0.49 (0.35-0.69)	38	0.40 (0.29-0.54)	0.015
Myocardial salvage index final	162	0.67 (0.52-0.88)	31	0.55 (0.48-0.68)	0.004
MVO (%LV)	188	0.0 (0.0-2.7)	43	1.6 (0.0-6.0)	0.018
>3 hours					
Infarct size acute (%)	157	17 (10-26)	51	22 (15-31)	0.002
Infarct size follow-up (%)	150	13 (6-20)	48	19 (8-27)	0.003
Myocardial salvage index acute	155	0.42 (0.28-0.59)	51	0.36 (0.19-0.50)	0.020
Myocardial salvage index final	138	0.59 (0.43-0.75)	41	0.51 (0.38-0.70)	0.114
MVO (%LV)	157	0.0 (0.0-2.4)	51	1.5 (0.9-5.6)	0.007

Data are presented as n (%), mean+/-SD or median (interquartile range).

CMR, cardiac magnetic resonance; ECG, electrocardiogram; PCI, percutaneous coronary intervention.