International Journal of Cardiology 297 (2019) 1–7

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Relationship between admission Q waves and microvascular injury in patients with ST-elevation myocardial infarction treated with primary percutaneous coronary intervention

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ARTICLE INFO

Article history:
Received 6 August 2019
Received in revised form 12 September 2019
Accepted 3 October 2019
Available online 12 October 2019

Keywords:
ST-Segment elevation myocardial infarction
Q waves
Microvascular injury
Cardiac magnetic resonance imaging

ABSTRACT

Background: Using comprehensive cardiac magnetic resonance (CMR) imaging in patients suffering from ST-elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (pPCI), we sought to investigate the association of admission Q waves with microvascular injury (microvascular obstruction (MVO) and intramyocardial haemorrhage (IMH)).

Methods: This prospective observational study included 195 STEMI patients treated with pPCI. Admission 12-lead electrocardiography was evaluated for the presence of pathological Q waves, defined as a Q wave duration of >30 ms and a depth of >0.1 mV. CMR was performed at 3 (interquartile range: 2–5) days after pPCI to determine infarct characteristics including MVO (late gadolinium enhancement) and IMH (T2* mapping).

Results: Admission Q waves were observed in 53% of patients (n = 104). These patients had a significantly lower BMI (p = 0.005), more frequent left anterior descending artery as culprit lesion (p = 0.005), were less frequent smokers (p = 0.048) and had higher rates of pre-interventional TIMI flow 0 (p = 0.018). Patients with Q waves showed a significantly larger infarct size (19%vs.12% of left ventricular mass,p < 0.001), lower ejection fraction (49%vs.54%,p = 0.001), worse global strain parameters (all p < 0.005) and more severe microvascular injury (MVO: 68%vs.34%,p < 0.001; IMH: 40%vs.20%,p = 0.002). Q waves remained associated with both MVO (odds ratio: 5.23, 95% confidence interval: 2.58 to 10.58,p < 0.001) and IMH (odds ratio: 3.94, 95% confidence interval: 1.83 to 8.46,p < 0.001) after adjusting for potential confounders (culprit lesion, pre-interventional TIMI flow 0, total ischemia time, ST-segment elevation).

Conclusions: Admission Q waves, derived from the readily available ECG, emerged as independent early markers of CMR-determined microvascular injury in STEMI patients undergoing pPCI.

1. Introduction

In patients with acute ST-elevation myocardial infarction (STEMI), immediate reperfusion, preferably with primary percutaneous coronary intervention (pPCI), is vital due to the close relationship of ischemia time with the extent of the ischemic wavefront and subsequently worse clinical outcome [1]. In clinical routine, time since symptom onset is most often used to estimate total ischemia time and thus qualification for reperfusion therapy. However, time from symptom onset is a subjective variable that may be imprecise in certain circumstances and does not fully consider dynamic factors of coronary occlusion (e.g. complete/incomplete spontaneous reperfusion) and coronary collaterals [2,3].

Alternatively, the presence of Q waves represents an objective indicator of infarct transmurality and size [4,5] and is associated with worse clinical outcome [6,7]. A recent study indicated that baseline Q waves were more closely associated with cardiac

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https://doi.org/10.1016/j.ijcard.2019.10.009
0167-5273/

magnetic resonance (CMR) - derived infarct size than symptom onset to reperfusion [2]. In addition, it has been suggested that baseline Q waves may exceed time from symptom onset as a prognostic marker in STEMI patients undergoing pPCI [6]. Besides infarct size, microvascular injury as detected by CMR is increasingly recognized as crucial marker of infarct severity and subsequently clinical outcome [8,9]. In fact, recent studies indicate that microvascular injury might be even the most potent CMR predictor of hard clinical events [10]. Microvascular obstruction (MVO) and intramyocardial haemorrhage (IMH) are the two major underlying pathologies in patients with microvascular injury that can be accurately visualized by CMR [11]. While the association between early Q waves and infarct size is well established [4,5], only few studies addressed the relationship between Q waves and microvascular injury. Nijveldt et al. could not illustrate an association between Q waves and MVO [12], whereas a more recent study suggested that patients with early Q waves show more extensive MVO [5]. Besides this ambiguity, another relevant limitation of previous studies include not using specific CMR sequences to detect IMH (a consequence of very severe microvascular injury). IMH, as specifically detected by T2* mapping, is more closely associated with adverse outcome after STEMI than MVO [13,14]. Hence, further studies are necessary to clarify the association between early Q waves and microvascular injury in STEMI patients undergoing pPCI.

The objective of the present study was therefore to comprehensively investigate the association of admission Q waves with microvascular injury (MVO and IMH) determined by CMR imaging in STEMI patients treated with contemporary pPCI.

2. Methods

2.1. Study design and clinical measurements

This prospective, observational study included 195 STEMI patients admitted to the coronary care unit of Innsbruck University Hospital between 2015 and 2018. The following inclusion criteria were applied: first STEMI diagnosed in accordance with the redefined ESC/ACC committee criteria [15], revascularization by pPCI within 24 h after onset of symptoms, an estimated glomerular filtration rate >30 ml/min/1.73 m^2 and Killip class <3 at time of CMR. Exclusion criteria were age <18 years, bundle branch block or fascicular block, any history of a previous myocardial infarction or coronary intervention and any contraindication to CMR (pacemaker, orbital foreign body, any aneurysm clips, claustrophobia, known or suggested contrast agent allergy to gadolinium). Creatine kinase and high-sensitivity cardiac troponin T (hs-cTnT) concentrations were assessed via peripheral venipuncture on hospital admission as well as once daily up to 92 h post-pPCI, as described previously [16]. Primary endpoint of the study was to evaluate the independent association between admission Q waves and microvascular injury (MVO and IMH). Main objective of this study was to disclose the independent association of admission Q waves in comparison with other early available predictors of microvascular injury. An exploratory clinical endpoint was the occurrence of major adverse cardiovascular events (MACE), defined as composite of all-cause death, myocardial re-infarction and new congestive heart failure.

For electrocardiographic (ECG) analyses, a standard 12-lead surface ECG (voltage: 10 mm/mV; speed: 25 mm/s) was performed on admission (before pPCI). For the analysis of duration and depth of Q waves, the "worst lead approach" was applied [5]. The presence of Q waves was defined as duration >30 ms and depth >0.1 mV as described previously [12,17]. During hospitalization, detailed medical history and physical examinations were performed. Written informed consent was obtained from all participants prior to inclusion. The local research ethics committee approved the study in conformity with the Declaration of Helsinki.

2.2. Cardiac magnetic resonance imaging

All CMR scans were performed on a 1.5 T Magnetom AVANTO-scanner (Siemens, Erlangen, Germany). The detailed imaging protocol and post-processing of our research group was published in detail previously [16]. In brief, left ventricular (LV) morphology and function were conducted on short-axis (10–12 slices) cine images using breath-hold, retrospective ECG-triggered trueFISP bright-blood sequences and for post-processing, standard software (ARGUS, Siemens, Erlangen, Germany) was used. Papillary muscles were included into the LV volume.

Late gadolinium enhancement (LGE) images were acquired approximately 15 min after the application of a 0.2 mmol/kg bolus of contrast agent (Gadovist®, Bayer Vital, Leverkusen, Germany) by an ECG-triggered phase-sensitive inversion recovery sequence (PSIR) with full coverage of left ventricle short-axis slices. Each slice of the LGE extent was determined quantitatively by using a commercially available workstation (IMPAX®, Agfa HealthCare, Bonn, Germany). "Hyperenhancement" was defined as a threshold of +5 standard deviations above the signal intensity of remote myocardium in the opposite myocardial segment of the LV [18]. MVO was characterized as persisting area of "hypoenhancement" within the infarcted territory, manually contoured on LGE images [19]. Infarct size and MVO are expressed as percentage of LV myocardial mass. IMH was assessed by T2* quantification using a breath-hold, cardiac gated gradient echo sequence before administration of the contrast medium [13]. A motion correction algorithm was utilized to reduce movement artefacts and motion corrected, colour-coded T2*-maps were automatically inline generated by fitting signal intensities at each image pixel with an exponential model for the given echo times. IMPAX EE workstation was appplied for evaluation of the maps by manually outlining endo- and epicardial contours with taking care to exclude subendocardial blood and subepicardial tissue to avoid partial volume effects [13]. IMH was defined as region of hypointense core within the infarcted myocardium with reduction of T2* signal intensities below 20 ms, as reported previously [13,14,20]. LV strain analyses were performed using a commercially available Tissue Tracking software (Circle Cardiovascular Imaging Inc®, Calgary, Canada). Both short- and long-axis images were available for 158 patients. Endocardial and epicardial borders were manually traced at end-diastole in short- and long-axis by retrospective motion tracking of steady-state free precession cine images. Short-axis series were divided into basal, mid-ventricular and apical position for tracing. Long-axis cine images were traced in 2-chamber, 3-chamber and 4-chamber images. Global longitudinal, radial and circumferential strain parameters were assessed based on the 16-segment model to calculate 2D peak strains and subsequently by averaging the peak values, the global strain parameters. All CMR images were analyzed by experienced readers, blinded to clinical data as well as ECG results.

2.3. Statistical analysis

SPSS Statistics 24.0 (IBM, Armonk, NY, USA) and MedCalc Version 15.8 (Ostend, Belgium) were used for statistical analyses. Continuous variables were expressed as median with corresponding interquartile ranges (IQR). Differences in continuous variables between two groups were evaluated by Mann–Whitney *U* test. Categorical variables were presented as absolute numbers with corresponding percentages. Chi-square test was used to assess differences in categorical variables. The

relation of Q waves with MVO and IMH was adjusted for potential confounders in uni-, and multivariable regression analysis for the difference in patients with presence or absence of Q waves. Early available variables of the baseline characteristics with a p-value <0.20 were first tested in univariable logistic analysis, as described previously [5]. Any variable with $p < 0.10$ in univariable analysis was further included in multivariable analysis. MACE-free survival was estimated and illustrated by means of the Kaplan-Meier method while log-rank test was used for evaluating differences in MACE-free survival. For all analyses, a two-tailed p-value of <0.05 was considered as statistically significant.

3. Results

3.1. Study population and patient characteristics

The present investigation included 195 revascularized STEMI patients with a median treatment delay of 183 (IQR: 120–287) minutes. The mean age of the overall cohort was 57 (IQR: 51–66) years.

All baseline characteristics, dichotomized by the presence (n = 104, 53%) or absence (n = 91, 47%) of Q waves, are provided in detail by Table 1. Patients with Q waves showed a significantly lower body mass index (BMI) ($p = 0.005$), lower systolic blood pressure ($p = 0.050$) and lower rates of current smokers ($p = 0.048$). Presence of Q waves was more frequently associated with left anterior descending artery as culprit lesion ($p = 0.005$), pre-interventional TIMI flow of 0 ($p = 0.018$) and tended to have a greater extent of ST-segment elevation ($p = 0.058$). Furthermore, higher levels of enzymatic infarct size, including peak creatine kinase (2481 vs. 1268 U/l) and hs-cTnT (6441 vs. 3117 ng/l), were significantly associated with presence of Q waves (both $p < 0.001$). No differences could be observed regarding presence of Q waves and diabetes mellitus ($p = 0.405$).

3.2. Q waves and CMR parameters

CMR imaging was performed at a median of 3 (IQR: 2–5) days after STEMI. No differences were observed regarding time point of CMR examination and microvascular injury (median 3 days, IQR: 2–5 days, $p = 0.524$) as well as presence of Q waves ($p = 0.199$). CMR findings and their relation with Q waves are also depicted in Table 1.

Q waves were significantly associated with lower LV ejection fraction ($p < 0.001$), worse global longitudinal ($p < 0.001$), global radial ($p = 0.003$), global circumferential strain ($p < 0.001$). Moreover, patients presenting with Q waves showed larger infarct size (19% vs. 12% of LV myocardial mass, $p < 0.001$). Microvascular injury, including MVO and IMH, was more severe in patients with Q waves (both $p < 0.001$). The relation between Q waves and microvascular injury is further illustrated in Fig. 1 and a representative ECG of admission Q waves with corresponding microvascular injury is provided by Fig. 2.

Table 1
Patient characteristics.

Characteristic	Total population (n = 195)	Q waves (n = 104, 53%)	No Q waves (n = 91, 47%)	p-value
Age, years	57 [51–66]	57 [51–67]	56 [50–66]	0.417
Female, n (%)	37 (19)	20 (19)	17 (19)	0.922
Body mass index, kg/m^2	26 [24–28]	25 [24–28]	27 [25–29]	**0.005**
Hypertension, n (%)	83 (43)	45 (43)	38 (42)	0.831
Systolic blood pressure, mmHg	140 [120–158]	135 [113–151]	140 [125–160]	**0.050**
Diastolic blood pressure, mmHg	86 [75–99]	84 [75–99]	89 [77–100]	0.240
Heart rate, min	72 [62–85]	74 [63–87]	72 [60–85]	0.435
Current smoker, n (%)	114 (59)	54 (52)	60 (66)	**0.048**
Hyperlipidaemia, n (%)	103 (53)	54 (52)	49 (54)	0.788
Diabetes mellitus, n (%)	21 (11)	13 (13)	8 (9)	0.405
Culprit lesion, (%)				**0.005**
RCA	80 (41)	42 (40)	38 (42)	
LAD	87 (45)	55 (53)	32 (35)	
LCX	26 (13)	7 (7)	19 (21)	
RI	2 (1)	0 (0)	2 (2)	
TIMI flow 0 pre-pPCI, n (%)	118 (61)	71 (68)	47 (52)	**0.018**
TIMI flow 3 post-pPCI, n (%)	174 (89)	93 (89)	81 (89)	0.926
Total ischemia time, min	183 [120–287]	193 [124–336]	162 [114–261]	0.120
ST-segment elevation, mV	0.3 [0.2–0.5]	0.4 [0.2–0.5]	0.3 [0.2–0.4]	0.058
Admission Q wave duration, ms	40 [30–50]	40 [40–65]	30 [0–30]	<**0.001**
Admission Q wave depth, mV	0.2 [0.1–0.5]	0.5 [0.2–0.9]	0.1 [0.0–0.1]	<**0.001**
Discharge Q wave duration, ms	40 [30–60]	40 [40–70]	30 [20–40]	<**0.001**
Discharge Q wave depth, mV	0.3 [0.1–0.7]	0.5 [0.2–0.9]	0.2 [0.1–0.3]	<**0.001**
Peak hs-cTnT, ng/l	4666 [1960–8625]	6441 [3424–10667]	3117 [1326–5873]	<**0.001**
Peak creatine kinase, U/l	1921 [930–3388]	2481 [1444–4130]	1268 [562–2383]	<**0.001**
CMR parameters				
LV end-diastolic volume, ml	147 [125–172]	151 [126–178]	142 [123–166]	0.183
LV end-systolic volume, ml	72 [56–91]	78 [57–101]	64 [55–84]	**0.011**
LV ejection fraction, %	52 [43–58]	49 [39–56]	54 [45–60]	**0.001**
LV global longitudinal strain, %	−13.4 [-15.4 to −11.1]	−12.0 [-14.3 to −10.0]	−14.1 [-15.9 to −12.5]	<**0.001**
LV global radial strain, %	29.4 [22.5–33.8]	27.6 [21.1–31.4]	31.8 [25.4–35.5]	**0.003**
LV global circumferential strain, %	−14.4 [-16.2 to −11.9]	−12.8 [-15.3 to −11.1]	−15.4 [-16.5 to −14.0]	<**0.001**
IS, % of LVMM	15 [7–25]	26 [17–34]	12 [5–17]	<**0.001**
MVO, n (%)	102 (52)	71 (68)	31 (34)	<**0.001**
MVO, % of LVMM	0.3 (0.0–2.3)	1.1 [0.0–3.5]	0.0 [0.0–0.8]	<**0.001**
IMH, n (%)	60 (31)	42 (40)	18 (20)	**0.002**

RCA = Right coronary artery; LAD = Left anterior descending artery; LCX = Left circumflex artery; RI = Ramus intermedius; pPCI = Primary percutaneous coronary intervention; hs-cTnT = high-sensitivity cardiac troponin T; LV = Left ventricular; IS = Infarct size; LVMM = Left ventricular myocardial mass; MVO = Microvascular obstruction; IMH = Intramyocardial haemorrhage.

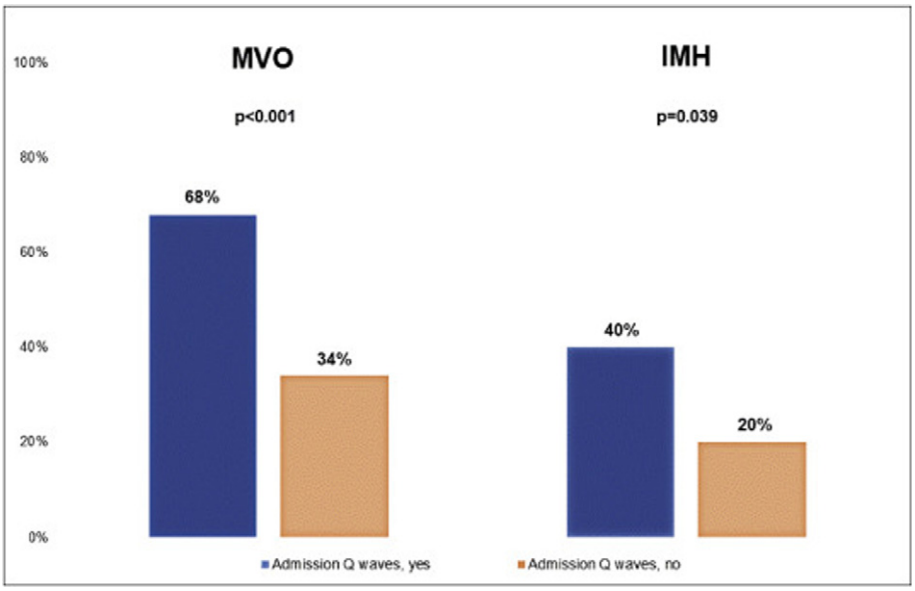

Fig. 1. Association of admission Q waves with MVO and IMH. Abbreviations: MVO = Microvascular obstruction; IMH = Intramyocardial haemorrhage.

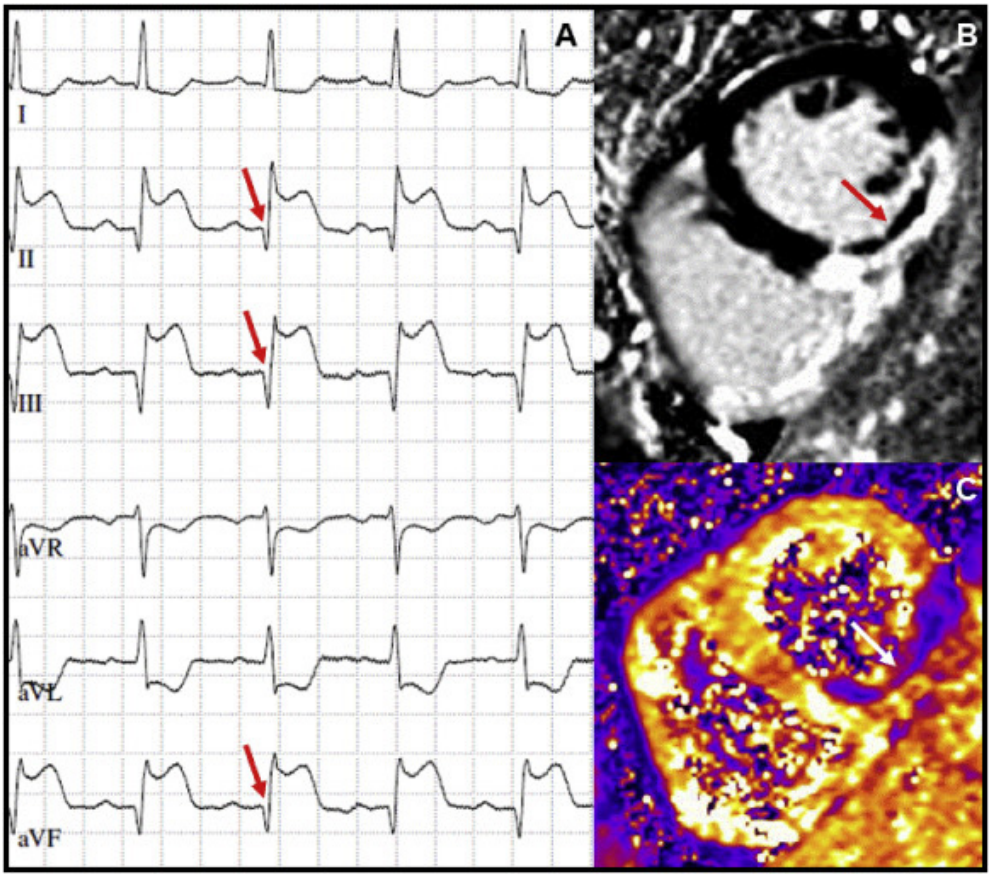

Fig. 2. Representative admission ECG (lead III as "worst lead" for Q waves (A)) with corresponding MVO (B) and IMH (C) after inferior wall STEMI. Abbreviations: ECG = Electrocardiogram; MVO = Microvascular obstruction; IMH = Intramyocardial haemorrhage; STEMI = ST-elevation myocardial infarction.

In multivariable logistic regression analysis, admission Q waves remained independently associated with both MVO and IMH after adjustment for culprit lesion, pre-interventional TIMI flow 0, total ischemia time, ST-segment elevation, systolic blood pressure and BMI (Table 2).

Furthermore, the presence of Q waves remained independent associates of microvascular injury even when stratifying the patients according to total ischemia time <3 and >3 h (Table 3, supplement).

Table 2
Binary logistic regression analysis for the prediction of microvascular injury.

	Univariable analysis		Multivariable analysis	
	OR (95% CI)	p-value	OR (95% CI)	p-value
MVO				
Admission Q wave	4.16 (2.29–7.58)	<**0.001**	5.23 (2.58–10.58)	<**0.001**
Culprit lesion	1.95 (1.29–2.96)	**0.002**	2.16 (1.36–3.44)	**0.001**
Pre-interventional TIMI flow 0	2.45 (1.36–4.42)	**0.003**	1.84 (0.93–3.64)	0.081
Total ischemia time, min	1.00 (1.00–1.00)	0.605	–	–
ST-elevation, mV	5.35 (1.12–25.48)	**0.035**	2.98 (0.61–14.43)	0.176
Systolic blood pressure, mmHg	0.99 (0.98–1.00)	0.251	–	–
Body mass index, kg/m^2	1.00 (0.93–1.09)	0.919	–	–
IMH				
Admission Q wave	2.75 (1.44–5.25)	**0.002**	3.94 (1.83–8.46)	<**0.001**
Culprit lesion	2.60 (1.65–4.10)	<**0.001**	3.32 (1.99–5.54)	<**0.001**
Pre-interventional TIMI flow 0	2.55 (1.30–5.00)	**0.007**	2.24 (1.06–4.73)	**0.034**
Total ischemia time, min	1.00 (1.00–1.00)	0.980	–	–
ST-elevation, mV	1.89 (0.44–8.17)	0.392	–	–
Systolic blood pressure, mmHg	1.00 (0.99–1.01)	0.957	–	–
Body mass index, kg/m^2	0.97 (0.89–1.06)	0.470	–	–

OR = Odds ratio; CI = Confidence interval; MVO = Microvascular obstruction; IMH = Intramyocardial haemorrhage.

Q waves determined at discharge were also associated with microvascular injury, including MVO ($p = 0.003$) and IMH ($p = 0.029$). AUC values of admission and discharge Q waves were comparable both for the prediction of MVO (AUC = 0.67 vs. 0.61, $p = 0.128$) and IMH (AUC = 0.62 vs. 0.58, $p = 0.268$).

3.3. Q waves and clinical outcome

In an exploratory analysis, 188 patients (96%) were followed for MACE at 11 (IQR: 5–13) months after STEMI. Eight patients (4%) experienced an event (1 death; 3 myocardial re-infarctions; 4 new congestive heart failures). As illustrated by the Kaplan-Meier curve, patients presenting with Q waves on admission significantly showed higher MACE rates ($p = 0.006$, Fig. 3). In addition, patients with larger infarct size also showed lower MACE-free survival ($p < 0.001$).

4. Discussion

This is the first CMR study evaluating the association of admission Q waves with microvascular injury (MVO and IMH). Admission Q waves emerged as independent associates of both MVO and IMH even after adjustment for potential confounders including culprit lesion, pre-interventional TIMI flow 0, total ischemia time, ST-segment elevation, systolic blood pressure and BMI. Consequently, the present observation highlights the value of admission Q waves as very early and objective risk marker of microvascular injury in STEMI patients referred for pPCI. Whether this subset of high-risk patients benefit from intensive therapeutic measures specifically targeting microvascular injury warrants further validation and research.

Early Q waves are frequently observed in STEMI patients [2,6,7]. In line, our findings confirm that early Q waves remain common in contemporary treated STEMI with a prevalence of approximately 50%. However, until now, only a small number of studies have addressed the value of Q waves as a distinct ECG metric of microvascular injury in patients with acute STEMI treated with pPCI. Topal et al. found that early Q waves are related to larger MVO, however, CMR imaging was performed one day after pPCI and might overestimated MVO [5]. In fact, the preferred imaging time point for MVO assessment should be between day 3–7 according to a recent expert consensus [21]. In contrast, Nijveldt et al. could not find an association between early Q waves and MVO [12]. They compared the number of Q waves with MVO, instead of one pre-defined Q wave like in the present study. Moreover, they evaluated Q waves on the post-PCI ECG. Other studies also assessed Q waves after PCI and illustrated significant associations with MVO [22]. However, Q waves assessed post-PCI will include reperfusion injury and can therefore not accurately reflect the microvascular status as early Q waves might offer. Another relevant limitation of all previous investigations is the missing information of IMH, which may be more closely related with adverse outcomes than MVO as indicated by recent investigations [13,14]. As such, this study is the first to comprehensively evaluate the association between admission Q waves and both MVO and IMH in acute STEMI patients. We found strong association between admission Q waves and MVO as well as IMH. Moreover, early Q waves remained independently related to MVO and IMH even after adjustment of other early available clinical parameters such as culprit lesion, pre-interventional TIMI flow, total ischemia time, ST-segment elevation, systolic blood pressure as well as BMI. Consequently, the presence of admission Q waves not only signifies more extensive myocardial infarction, but more importantly also inadequate microvascular tissue perfusion. In addition, global strain parameters (longitudinal, radial, circumferential) were significantly related with Q waves. This finding further emphasizes the association of Q waves with microvascular injury since myocardial deformation is closely related with both MVO and IMH [23].

Although patients with diabetes mellitus frequently suffer from coronary microvascular dysfunction [24], this thematic is still controversially discussed in the current literature. A large study by Reinstadler et al. could not demonstrate any differences in STEMI patients suffering from diabetes and the presence of microvascular obstruction [25]. This is in line with the present data, where no relation between admission Q waves and diabetes mellitus could be observed. However, in the present cohort only 11% suffered from diabetes and therefore, this cohort is not powered for this question.

Admission Q waves can therefore be considered as a very robust and easy obtainable clinical marker for early identification of patients at risk for severe myocardial damage including microvascular injury. This is of relevance since recent data foregrounded that microvascular injury is of higher predictive value for MACE than infarct size itself [26]. Lately, CMR studies also indicate that microvascular injury (especially IMH) evolves progressively during the first days after myocardial infarction and thus may be amendable to specific therapeutic interventions [27].

Whether STEMI patients with admission Q waves might benefit from adjunctive therapeutic measure to prevent or limit microvascular injury is unknown so far but merits further evaluation in dedicated studies.

Previous studies highlighted the association of Q waves with time from symptom onset [5,6]. However, in the present study no relation between total ischemia time and presence of Q waves could be illustrated. This controversy mainly could be explained due to the fact that symptom onset is a very subjective parameter and often imprecise. Indeed, Topal et al. also described that symptom onset to treatment was not significantly associated with MVO in multivariable analysis when adjusted for Q waves [5]. This is concordant with our study and prior observations showing that Q waves are independent from time to symptom onset and thus may represent more accurately the wavefront of myocardial damage [28]. In addition, the relation of Q waves with microvascular injury did not change even when patients were stratified according to total ischemia time <3 and >3 h.

Several previous analyses illustrated the prognostic impact of baseline Q waves in acute STEMI patients [7,29]. In accordance with those, we could also demonstrate significant relation between admission Q waves and worse clinical outcome. Besides, the presence of baseline Q waves, rather than time from symptom onset, were significantly associated with an increased rate of adverse in-hospital rates [29]. Hence, patients presenting with Q waves on admission are related to adverse clinical outcome beyond longer total ischemia time [5].

4.1. Clinical implications

The present data revealed a significant relation between admission Q waves and microvascular injury, one of the major prognosis markers in STEMI patients [13,30]. Considering the restricted availability of CMR for microvascular injury assessment and the globally availability of ECG, admission Q waves might offer a high clinical potential for improved early risk stratification in daily clinical routine. Besides risk assessment, the present findings might also become relevant for therapeutic consideration. Microvascular injury is also closely associated with adverse remodeling after STEMI [31]. One might therefore speculate that patients with Q waves might benefit from more intensive secondary prevention measures (e.g. anti-remodeling treatment) as well as a closer follow-up. However, this needs evaluation in prospective studies. Over and above established treatments, new therapeutic approaches for the prevention of microvascular injury have been proposed over the last years [32]. However, currently no promising treatment for clinical application could be found to protect microcirculation [32]. Therefore, future randomized trials are required to test novel therapy strategies against microvascular injury. Admission Q waves might help to select the appropriate patients with high burden of microvascular injury to test novel therapeutic approaches to potentially improve long-term prognosis after STEMI.

4.2. Limitations

The present study has some limitations. We included relatively stable STEMI patients with Killip class <3 in order to perform high-quality CMR imaging and therefore our findings are not generalizable for unstable patients with ongoing cardiogenic shock. Nevertheless, the vast majority of the overall STEMI population presents with Killip class <3 [33]. Also we used the latest consensus document of ECG criteria for the definition of Q waves, the results may be different using other definitions of Q waves [34].

5. Conclusion

Admission Q waves emerged as independent associates of CMR-determined microvascular injury in patients with acute STEMI revascularized by pPCI. Therefore, our findings emphasize the value of admission Q waves as an effective and simple clinical tool for the very early risk assessment for microvascular injury in patients presenting with STEMI.

Conflict of interest

The authors declare that there is no conflict of interest.

Acknowledgments

This study was supported by grants from the 'Austrian Society of Cardiology', the "Tiroler Wissenschaftsfonds", the Austrian Science Fund (FWF): KLI 772-B and by an intramural funding program of the Medical University Innsbruck for young scientists MUI-START, Project 2015-06-01-013.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2019.10.009.

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