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Short communication

Quantitative and qualitative assessment of acute myocardial injury by CMR at multiple time points after acute myocardial infarction☆

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ABSTRACT

Background: Recent experimental studies have shown a dynamic time course of myocardial edema with an initial wave of edematous reaction within hours after reperfusion which almost resolved at 24 h. However, this dynamic pattern appears to be absent in clinical cohort studies. Thus far, no studies have combined a quantitative and qualitative assessment of acute myocardial injury in a large clinical cohort to explain these divergent findings.
Methods: A cohort of 225 patients (59 ± 11 years, 83% men) with successfully reperfused STEMI within 12 h of symptom onset were included. Quantitative measurements of myocardial damage such as T1 mapping and T2 triple short-tau inversion recovery (STIR), contrast-to-noise ratio (CNR) and their impact on area-at-risk (AAR), infarct size (IS), and myocardial salvage index (MSI) were assessed at different time points. One-way analysis of variance (ANOVA) and linear regression analysis was used to compare myocardial damage at the different time points.
Results: A small fraction of patients underwent CMR within 24 h of reperfusion (17/225, 7.6%). No significant variations in AAR, IS, MSI, T2 STIR CNR, or native T1 maps were observed between the different time points after reperfusion. Time of CMR was not a significant predictor of AAR ($P = 0.90$), IS ($P = 0.27$), MSI ($P = 0.23$) or T2 STIR CNR ($P = 0.23$).
Conclusions: The majority of CMR exams in STEMI patients are performed outside the dynamic time window of early post-MI edema. The stable pattern of markers of acute myocardial damage at different time points suggests these markers are reliable for the prognostication of patients after STEMI.

1. Introduction

Recent experimental studies have shown a bimodal pattern of myocardial edema early post-MI with an initial wave of edema within 3-hour post-reperfusion, partial regression at 24 h, and a deferred wave peaking at 4–7 days [1,2]. This time-dependent change in edema affected CMR-based estimates of myocardial injury such as area-at-risk (AAR) and myocardial salvage index (MSI). The importance of these findings cannot be overstated as they inquire us to critically check the validity of numerous previously published clinical trials using these CMR parameters as endpoints [3,4]. However, a recent retrospective analysis of a large multicenter cohort including 795 STEMI patients did not reveal a bimodal pattern of edema early post-MI [5]. A potential explanation for this discrepancy is the different time frames at which patients were imaged. Immediately after reperfusion most STEMI patients are too unstable to safely undergo CMR and CMR scans are usually performed at least 24 h after reperfusion. Therefore, the hyperacute (<3 h) edema peak and transient decrease in edema (at 24 h) may not be observable in clinical cohorts. Thus far, no studies have examined the early time course post-MI of quantitative measurements of myocardial damage such as T1 mapping and T2 triple short-tau inversion recovery (STIR) contrast-to-noise ratio (CNR) and their impact on AAR and myocardial salvage index (MSI) assessment in a large prospectively acquired cohort of STEMI patients.

2. Methods

A total of 225 patients (59 ± 11 years, 83% men) with successfully reperfused STEMI within 12 h of symptom onset were included at the University Hospitals Leuven, Belgium [6]. All CMR imaging was performed within 7 days after reperfusion on a 1.5 T scanner (Ingenia-CX, Philips, Best, The Netherlands), using a comprehensive protocol including T2-weighed STIR imaging and T1-weighed late gadolinium enhancement (LGE). Additionally, native T1 mapping results (MOLLI 5 s(3 s) 3 s, balanced steady-state free precession

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(b-SSFP) readout, flip angle 35°) were available in a subset of 40 patients. All CMR studies were post hoc analyzed by experienced investigators blinded to the clinical data. Patients were divided into equal quartiles based on the time between reperfusion and CMR (8 h–45 h, 45 h–67 h, 67 h–96 h, and 96 h–168 h). T2 STIR CNR was calculated as signal intensity difference between infarcted and remote myocardium, divided by standard deviation of noise (air) [7]. Hypointense areas in the infarcted myocardium suggestive of intramyocardial hemorrhage (IMH) on T2-weighted images were excluded for measurements of signal intensity but included in the AAR as described before [8]. A similar

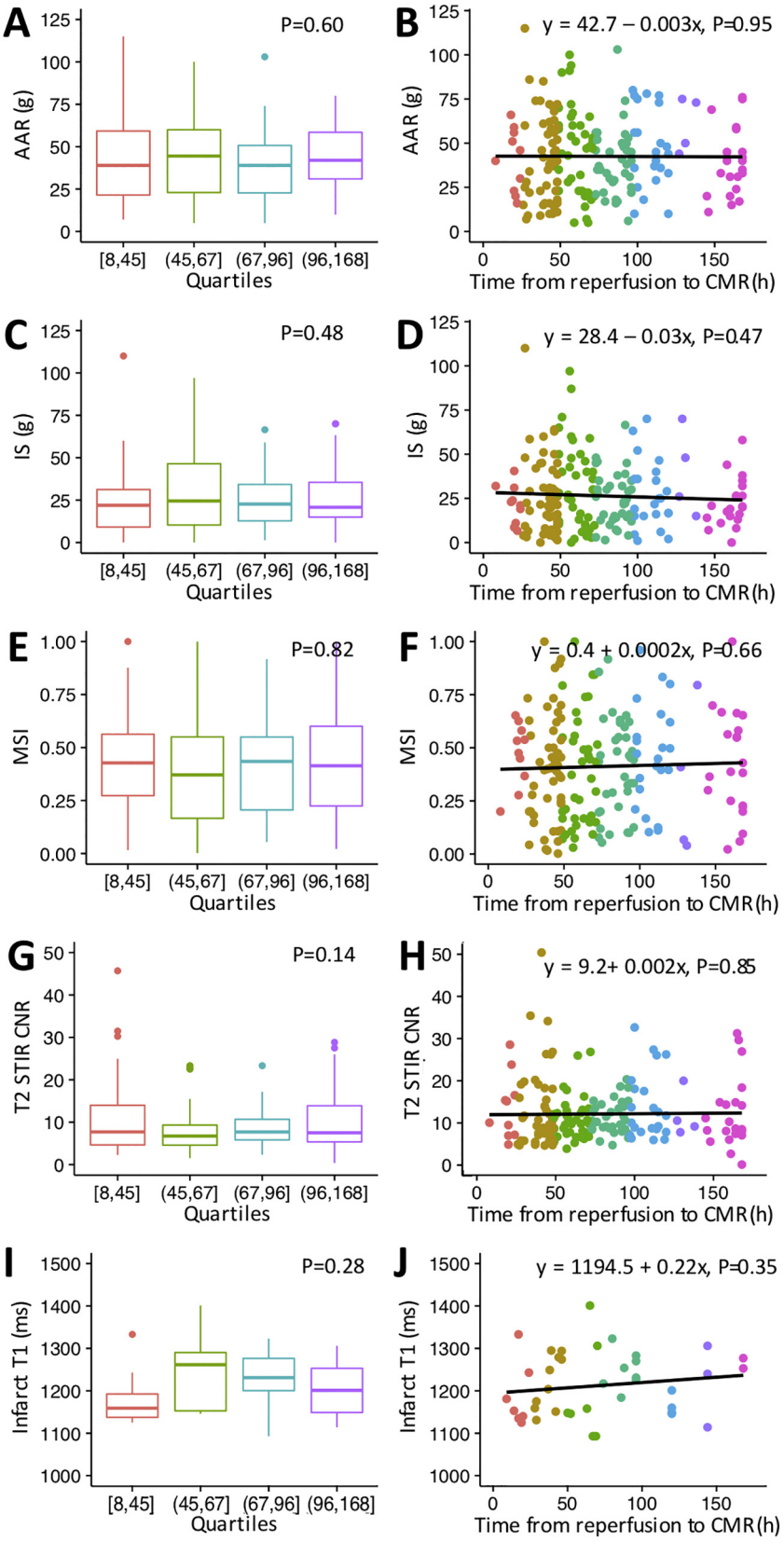

Fig. 1. Boxplots (A, C, E, G, I) and scatterplots with regression line (B, D, F, H, J) show area-at-risk (AAR) (A, B), infarct size (IS) (C, D), myocardial salvage index (MSI) (E, F), T2 STIR contrast-to-noise ratio (CNR) (G, H), and native infarct T1 map values (I, J) according to the time between reperfusion and CMR.

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