

POSTER PRESENTATION

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Characterization of peri-infarct zone by CMR is a robust predictor of major adverse events and is strongly associated with systemic inflammatory response post-myocardial infarction

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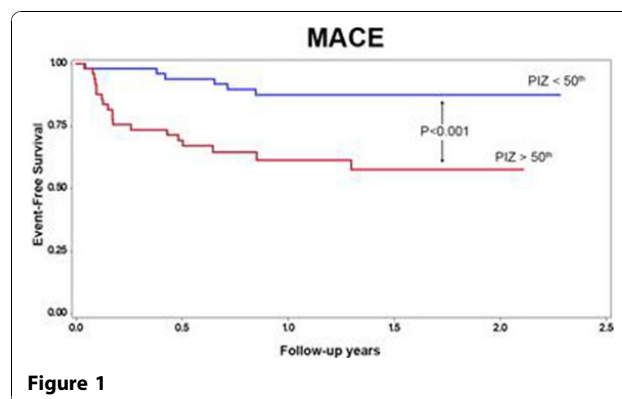
Introduction

While previous studies suggest that the peri-infarct zone (PIZ) may be an important arrhythmogenic substrate and may be associated with an unfavorable outcome post-MI, to date, there is no convincing mechanistic explanation to support that relationship. Systemic inflammatory response (SIR) in the acute phase post-MI has also been associated an adverse prognosis. Furthermore, a strong SIR may lead to greater heterogeneity of the infarcted myocardium. We hypothesized that the PIZ extent would be associated with the severity of the post-MI SIR. We further sought to determine if prognostic information provided by PIZ extent and SIR would complement classical markers of adverse outcome post-MI.

Method/results

We prospectively enrolled 102 patients (24 females, mean age 55±10) admitted with a ST elevation MI. SIR was estimated on admission (D1) and on the 5th post-MI day (D5) by levels of high-sensitivity CRP, interleukin-2 (IL-2) and tumor necrosis factor type- α (TNF- α). CMR was performed 4-weeks after MI (1.5-TGE-CV/i) including cine imaging, and LGE 10-minutes after a cumulative dose of 0.2mmol/Kg of gadolinium. PIZ, total and core infarct mass were quantified based on signal-intensity (SI) thresholds using the full-width at half-maximum method (tissue with SI>peak remote but<50%

of max was defined as PIZ). At a median follow-up of 16.1 months (IQR12.3), 25 major adverse cardiovascular events (MACE)(24%) had occurred (11 cardiac deaths, 8 MIs and 6 unstable anginas). CRP, IL-2 and TNF- α measured at D5 and the variation between D1 and D5 (delta) demonstrated a strong association with PIZ (CRP-D5, $r=0.69$, $p<0.0001$; delta-CRP, $r=0.7$, $p<0.0001$; IL-2-D5, $r=0.5$, $p<0.0001$; delta-IL-2, $r=0.6$, $p<0.0001$; TNF- α , $r=0.5$, $p<0.0001$; delta-TNF- α , $r=0.4$, $p=0.0001$). After adjustment for age, total infarct size, gender and for their respective baseline level, delta-CRP, delta-IL-2 and delta-TNF- α maintain strong association with the PIZ mass expressed as tertiles (delta-CRP, $R^2=0.5$, $p<0.0001$; delta-IL-2, $R^2=0.3$, $p=0.02$; delta-TNF- α , $R^2=0.5$, $p=0.003$). By univariable analysis, the PIZ mass, total infarct size, delta-CRP and delta-IL2 showed the strongest association with MACE(PIZ, $LR\chi^2$ 25.7, HR



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1.13, $P < 0.0001$; total infarct mass, $LR\chi^2$ 7.2, HR 1.03, $P = 0.006$; delta-CRP, $LR\chi^2$ 25.3, HR 1.15, $P < 0.0001$; delta-IL2, $LR\chi^2$ 19.3, HR 1.08, $P < 0.0001$). After adjustment for age, LVEF and delta-CRP, PIZ mass maintained a strong adjusted association with MACE (adjusted $LR\chi^2$ 8.5, HR 1.17, $P = 0.003$). Patients with PIZ mass $> 50^{\text{th}}$ demonstrated a significantly reduced MACE-free survival ($p = 0.0008$, Figure 1)

Conclusion

PIZ as evidence of infarct tissue heterogeneity provides prognostic information post-MI. In addition, the extent of PIZ by CMR is strongly associated with SIR in the subacute phase post-MI.

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