

## Microvascular obstruction is associated with greater extracellular matrix remodelling in the remote myocardium after infarction: A T1-mapping study

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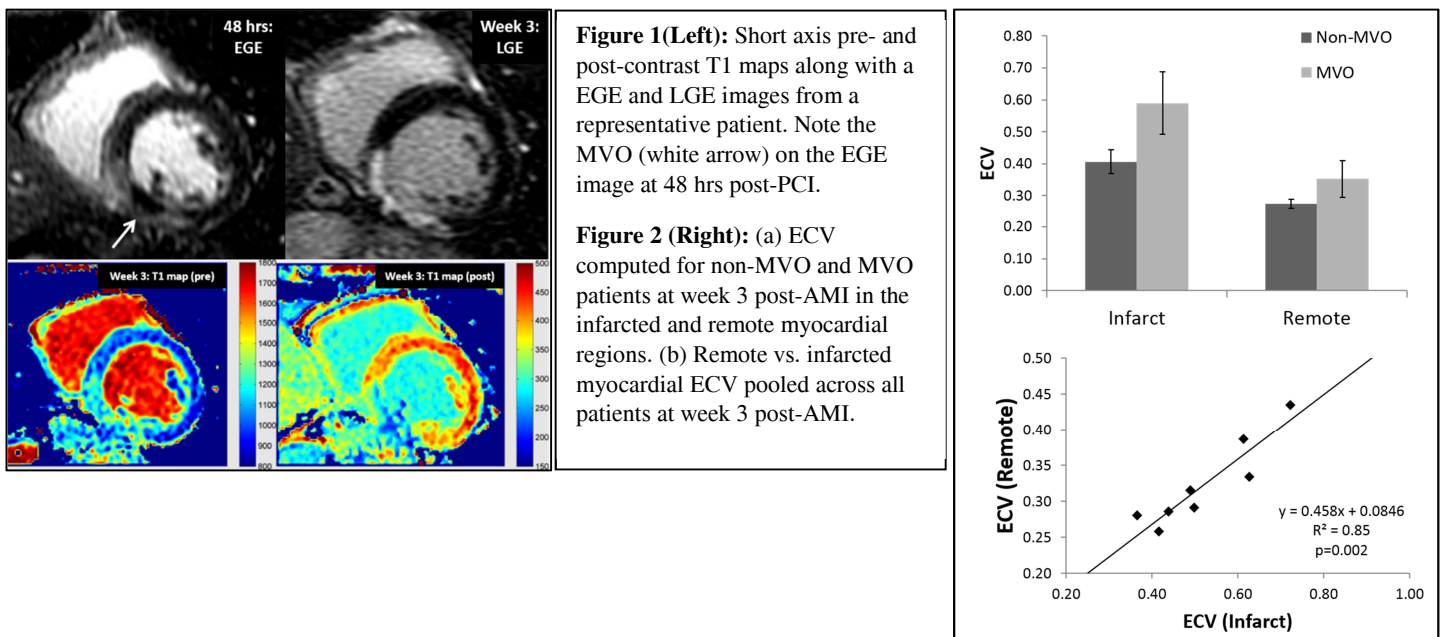
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**Introduction:** Recent studies have shown that extracellular matrix alterations can occur in both infarcted as well as remote myocardium post acute myocardial infarction (AMI) [1,2]. In particular, the remote matrix has been shown to increase in the sub-acute stage and may remain expanded in the chronic condition. However, previous studies have not assessed the relationship between ECV and the severity of infarction. It would be beneficial to study the relationship between infarcts with microvascular obstruction (MVO) and the alterations in the ECV, since MVO has been associated with long-term cardiovascular complications. In this study, our objective was to assess the impact of microvascular obstruction (MVO) on ECV in the myocardium of patients' post-AMI. In particular, our hypothesis was that ECV alterations in the remote myocardium would be more prominent in patients with MVO.

**Methods:** 8 patients with STEMI underwent a CMR exam on a 1.5T scanner (GE Healthcare) at 48 hrs and 3 week time-points post-PCI. Infarction and MVO was assessed using early and late gadolinium enhancement (EGE, LGE) imaging. Pre- and post-contrast T1 was quantified using a Modified Look-Locker Inversion recovery (MOLLI) sequence with heart rate correction. The MOLLI sequence was acquired with two inversion schedules in a 3-5 pattern with 3 pausing heart-beats acquiring 8 images in 11 heart-beats. Reconstruction was done using the RUSL (Relaxometry Using Sequence SimuLation) method ensuring heart-rate insensitivity [3]. ECV fraction was estimated at week 3 from the relation:  $(1 - \text{hematocrit}) * (1/T1_{\text{myo, post}} - 1/T1_{\text{myo, pre}}) / (1/T1_{\text{blood, post}} - 1/T1_{\text{blood, pre}})$ . Infarcted and remote myocardial segments were chosen based on LGE images. T2 mapping was performed using a spiral-T2prep sequence to evaluate edema. Given its association with MVO, hemorrhage was also assessed from T2\* maps obtained using a multi-echo gradient-echo acquisition.

**Results:** MVO was demonstrated in 5/8 patients at 48 hrs post-PCI; low T2\* indicated the presence of hemorrhage in these patients. Fig. 1 shows representative T1 maps and the corresponding LGE image. In the MVO group, ECV was found to be significantly higher than the non-MVO group in both infarcted ( $0.59 \pm 0.1$  vs.  $0.41 \pm 0.04$ ,  $p=0.01$ ) and remote tissue ( $0.35 \pm 0.06$  vs.  $0.27 \pm 0.01$ ,  $p=0.04$ ) at week 3 post-AMI (Fig. 2a). When values were pooled across all patients, ECV in the remote tissue progressively increased with ECV in the infarcted myocardium with a high degree of correlation (Fig. 2b). We noted an increase in T2 values in the MVO group, in both infarct ( $56.3 \pm 5.1$  vs  $44.6 \pm 5.7$ ,  $p=0.04$ ) and remote myocardium ( $40.2 \pm 0.9$  vs  $38.0 \pm 0.5$ ,  $p=0.04$ ).

**Conclusions:** Prior studies have shown that remote myocardial segments are affected post-AMI; in the sub-acute phase, severe infarcts with MVO and hemorrhage exhibit elevated T2 in association with vasodilator dysfunction [4]. Our pilot observations additionally suggest that extracellular matrix expansion might be greater for high-risk patients in both infarcted and especially remote myocardium. This also confirms that early changes in remote myocardium may potentially be indicative of long-term adverse remodeling post-AMI.



- References:**
1. Ugander M, et al. Eur Heart J. 2012,33:1268.
  2. Chan W, et al. JACC- Cardiovasc Imaging 2012,5:884.
  3. Ramanan V et al. ISMRM 2013 #1418.
  4. Ghugre NR, et al. Mag Res Med 2012,70:1095.