



Is it important to differentiate between peri-procedural myocardial injury and persistent myocardial scar?

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With the growth of complex high-risk percutaneous coronary intervention (PCI) it is of increasing importance to be able to quantify procedural risk and describe the consequence of peri-procedural complications. With the increasing prevalence of calcific coronary artery disease (CAD) there is a growing need for adjunctive calcium modifying techniques, with rotational atherectomy (RA) the most commonly used method. In this context, we conducted a cohort study of patients having PCI with RA to determine the incidence of peri-procedural myocardial infarction (PMI), perceived to be the most frequently associated complication. We applied the two current definitions of PMI, the 3rd universal definition (3rd UD) and Society for Cardiovascular and Interventions (SCAI) definition. Cardiac magnetic resonance (CMR) imaging is the reference standard method for myocardial tissue characterization and myocardial infarct assessment. We therefore used CMR imaging to assess the accuracy of each definition of PMI, and to determine the mid-term persistence of any detected myocardial injury (1). The multiparametric CMR protocol included cine imaging to detect new regional wall motion abnormalities (WMA), parametric T2 mapping for acute myocardial oedema, and late gadolinium enhancement (LGE) imaging to detect myocardial scar.

We would like to thank Januszek *et al.* and Jinnouchi *et al.* for their review of the wider literature and interesting commentary on the study. As stated by Januszek *et al.* (2), Lim *et al.* previously reported that CK-MB defined type 4a MI correlated better with CMR detected infarct, while cardiac troponin was over-sensitive (3). Despite this, with the wide adoption of high-sensitivity troponin in the diagnosis of MI, and with CK-MB no longer routinely available, we elected to use troponin to facilitate translation of the data directly into clinical practice.

A troponin >5× upper reference limit (URL) was detected in 68% of our cohort. On applying the 3rd UD (also requiring >20 mins of chest pain, ECG ST-segment changes or new Q waves, angiographic deterioration in flow, or imaging evidence of loss of viable myocardium or new wall motion abnormality), the incidence of type 4a MI was 10%. With the addition of CMR imaging the incidence of PMI was 24%, i.e., CMR was able to detect acute myocardial injury in an additional 14% of patients which was not apparent on the post-procedural ECG or echocardiogram. The prevalence of PMI decreased to 14% on the 6-month follow-up CMR. Contrary to the comment by Januszek *et al.*, and as acknowledged by Jinnouchi *et al.*, we had anticipated the incidence of PMI would increase

with the addition of the CMR data to the 3rd UD, as ECG and echocardiography are known to have limited sensitivity particularly for small subendocardial infarcts (2,4).

As commented by Jinnouchi *et al.*, the comparison of the day 7 and 6-month CMR data, showing complete resolution of myocardial oedema and new WMAs but persistence of LGE, highlights the difference between transient myocardial injury (i.e., myocardial stunning) and persistent myocardial scar. This draws attention to the potential issue in analyses by other groups, which by including all 3rd UD type 4a MI, combined patients with better and poorer prognoses into one cohort.

We agree with Jinnouchi *et al.* that our data does not support routine usage of CMR imaging for the diagnosis of PMI, while its prediction of clinical outcomes remain unclear. In particular, the prognostic relevance of CMR detected LGE and its use to stratify patients with 3rd UD PMI requires further investigation.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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