

Usability of cardiac magnetic resonance imaging for procedural myocardial infarction undergoing rotational atherectomy

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Rotational atherectomy (RA) is a safe and effective percutaneous technique utilized in a treatment of calcified lesions to modify as a primary therapy. Even though the procedural success rate with RA is 90%, RA is associated with higher rate of no flow/slow flow which occurs as a result of micro debris and/or bubbles than other coronary revascularization procedure (1). This phenomenon must be related with procedural myocardial infarction (type 4a MI). Although type 4a MI is not associated with an increased rate of death in long-term outcomes, it is associated with increased risk for recurrent myocardial infarction (MI) (2). Recurrent MI has been shown to be associated with clinical heart failure and higher cardiac mortality (3,4), therefore, to detect type 4a MI is important for patients who were treated percutaneous coronary intervention (PCI) with RA. This article is review for the clinical prospective cohort study that was researched by McEntegart *et al.* The author enrolled 58 near-consecutive patients who were scheduled to have a PCI with RA for stable angina. The aim of the study was to determine the incidence of type 4a MI following PCI with RA and to investigate the cardiac injury or MI associated cardiac magnetic resonance (CMR) (5).

The diagnostic criteria of type 4a MI is controversial with two definitions; the Third Universal Definition (3rd UD) (6) and the Society for Cardiac Angiography and Intervention (SCAI) definition (7), which are currently

widely used. The universal definition of type 4a MI was defined as an increase in cardiac troponin (cTn) to $>5\times$ the 99th percentile of the upper reference limit (URL) during the first 48h following PCI (in patients with normal baseline cTn value), plus either: (I) evidence of prolonged chest pain (>20 min), or (II) ischemic ST-segment changes or new pathological Q waves, or (III) angiographic evidence of a flow limiting complication, or (IV) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality (WMA) (6). The SCAI definition of type 4a MI was defined as an elevation of creatine kinase-myocardial band (CK-MB) to $\geq 10\times$ upper limit of normal (ULN) or cTn to $\geq 70\times$ ULN (if CK-MB level are unavailable), or by CK-MB to $\geq 5\times$ ULN or cTn to $\geq 35\times$ ULN plus the development of new pathologic Q-waves in ≥ 2 contiguous leads or left bundle branch block (LBBB) (7). The primary difference of these definition is that the 3rd UD definition recommends the use of cTn, and the SCAI definition recommends the use of CK-MB. The diagnostic criteria of cTn value in the 3rd UD definition is low, therefore, there is a possibility of oversensitive to diagnose type 4a MI (8). Whereas, the sensitivity of SCAI definition might be low because CK-MB and cTn level of SCAI definition is high values (9). Each of these definitions has both good points and bad points. McEntegart *et al.* determined the incidence of type 4a MI after PCI with

RA using the both definitions in the clinical study. The investigator measured high-sensitivity cTn at 6–18 hours after RA (5).

Late gadolinium enhancement CMR (LGE-CMR) imaging allows assessment for the identification of myocardial infarction. LGE-CMR permits precise quantification of even small areas of myocardial necrosis, such as procedural MI (10,11). Rahimi *et al.* reported new LGE after PCI was associated with an increase of death, non-fatal myocardial infarction, or sustained ventricular arrhythmia, however there is no relationship between cTn values after procedure and primary outcome (12). It is well known that a rise in cardiac biomarkers such as cTn and CK-MB sometime occurs in patients undergoing PCI. Therefore, combining cardiac biomarkers with LGE-CMR might be increase not only sensitivity but also specificity. McEntegart *et al.* compared combinational CMR with non-combinational CMR for diagnosing type 4a MI using imaging evidence in the 3rd UD definition criteria (SCAI definition does not require imaging evidence), however, the investigator did not compare CMR with other imaging devices such as cardiac echo and cardiac scintigraphy (5). In the study, CMR was performed before RA procedure, 7-day after, and 6-month after PCI with RA (5). The protocol of CMR was composed T2 mapping for myocardial edema, cine CMR for regional WMA, perfusion CMR with adenosine stress, and LGE-CMR (5). A type 4a MI was defined that LGE was confirmed on both axial and long axis, or a new WMA was detected (5). As a result, the 3rd UD definition combined CMR could detect 24% type 4a MI patients of all, and non-combined CMR (other routine clinical practices had been done) could detect 10% patients, and SCAI definition could detect only 4% patients, which was not assessed by statistical analysis (5).

The advantage of this study is focusing on type 4a MI patients undergoing RA, and combining the 3rd UD definition of type 4a MI with CMR as imaging evidence (SCAI definition does not require imaging evidence). Previous study was reported that there was a strong correlation between the rise in cTn values at 24 hours after PCI and mean mass of new myocardial enhancement detected by LGE-CMR (13). Basically, sensitivity of the 3rd UD definition is high, but specificity and positive predictive values is low (8). Low specificity definition may lead to prolonged hospital stay and unnecessary therapeutic intervention, which may result in increased costs. Combining the 3rd UD definition with CMR have specificity (sensitivity: 100%, specificity: 90%) raise in

the study. Furthermore, it was also good point that CMR was performed before RA, 7-day and 6-month after RA procedure to assess changes over time. In consequence, there was the improvement of myocardial edema, new WMA, and new LGE between 7-day CMR after RA and 6-month (5).

However, there are some issues that are needed to be discussed. First, although McEntegart *et al.* concluded PCI with RA was associated with a significant risk of type 4a MI, the author did not compare between with and without RA in the study. Therefore, the investigator could not conclude whether RA would be significant higher risk of type 4a MI than other procedures. The assessment of incidence rate of type 4a MI undergoing RA was only thing as the author mentioned in the aim of study. In addition, previous study reported that procedural complexity, including the number of stents, total stent length, and bifurcation stenting might contribute to type 4a MI occurring (2). In order to evaluate the association with RA, these procedural characteristics had to be considered doing statistical analyses.

Second, although one of the aims of study was to investigate whether detected cardiac injury or MI due to RA would be associated with CMR, it was uncertain if CMR could contribute to the detection of type 4a MI and myocardial injury, because the investigator did not compare with other imaging devices such as cardiac echo or cardiac scintigraphy in case of evaluating imaging evidence of the 3rd UD definition. As mentioned above, the detecting rate of type 4a MI with the 3rd UD definition combined CMR is higher than non-combined CMR, however it was uncertain whether there were significant differences.

Third, there were no baseline cTn and CK-MB value which should be evaluated before procedure. Both of the 3rd UD and SCAI definition require evaluating baseline cardiac biomarker values before procedure (6,7). Especially, in terms of cTn, it is the most specific and sensitive biomarkers of myocardial cell injury, however it is well known that, in patient with renal dysfunction or septic shock, cTn elevations which cannot be linked to myocardial injury are found occasionally, which is not yet convincingly explained (14). Gustavsson *et al.* reported that cTn must be measured before and after PCI to diagnose procedure related MI to avoid false positive diagnosis (15). Only actual increases between before and after procedure could be regarded as indicating type 4a MI. Furthermore, previous study showed that patients with elevated cTn baseline levels (pre-PCI) had significant higher overall cumulative 12-month death/MI rate compared with

those non-elevated baseline levels (16). Prasad *et al.* reported that abnormal pre-procedural troponin level is a powerful independent predictor of prognosis after PCI and has a greater prognostic significance than the post-procedural biomarker levels (17). Therefore, to evaluate baseline cardiac biomarker such as cTn and CK-MB before PCI is very important. In particular, 18% of all patients have mild to moderate renal dysfunction ($30 < \text{eGFR} < 60 \text{ mL/min/1.73 m}^2$) in the study, the patients should be hence measured cTn values before procedure.

Fourth, there is different timeline between evaluating cardiac biomarkers and performing CMR. Although cardiac biomarkers were evaluated within 6–18 hours after procedure in the study, CMR was performed at 7 days after procedure. Therefore, there is possibility that CMR image of 7 days after procedure does not reflect cardiac biomarkers values which is evaluated within 6–18 hours after PCI with RA.

In summary, the clinical prospective cohort study by McEntegart *et al.* has valuable contributions to suggest that combining the diagnostic criteria of type 4a MI with CMR might be useful for diagnosing procedural MI undergoing PCI with RA. However, it is still unknown whether PCI with RA is associated with a risk of type 4a MI and whether combining the diagnostic criteria of type 4a MI with CMR increase the accuracy to diagnose procedural MI undergoing RA. In order to clarify these issues and understand long-term outcome of procedural MI undergoing RA, randomized controlled trial merits to be carried out in the near future.

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Footnote

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

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