Computed tomography plaque analysis identifies residual inflammation in recent acute myocardial infarction patients

X. Wang¹, M.S. Yew², P.D. Adamson³, M.Y.Y. Chan⁴, N.A.S. Raffiee⁵, H.T. Yap⁶, Y.T. Loong⁵, A. Elankovan⁵, S. Leng⁵, W. Huang⁶, H.K. Lee⁷, L.L.S. Teo⁴, L. Zhong¹, D.J. Hausenloy¹, L. Baskaran⁵

¹Duke-NUS Graduate Medical School Singapore, Singapore, Singapore
²Tan Tock Seng Hospital, Department of Cardiology, Singapore, Singapore
³University of Otago Christchurch, Department of Medicine, Christchurch, New Zealand
⁴National University Heart Centre, Singapore, Singapore
⁵National Heart Centre Singapore, Singapore, Singapore
⁶Agency for Science, Technology and Research, Institute of Infocomm, Research, Singapore, Singapore
⁷Agency for Science, Technology and Research, Bioinformatics Institute, Singapore, Singapore

Funding Acknowledgements: Type of funding sources: Private company. Main funding source(s): Astra Zeneca

Introduction: Residual inflammatory risk (RIR) following acute myocardial infarction (AMI) patients is associated with recurrent major adverse cardiovascular events. Atherosclerotic plaque quantification using computed tomographic coronary angiography (CTCA) can specifically identify inflamed plaque characteristics. We compared plaque burden and tissue composition in AMI patients with those of stable coronary artery disease (CAD) patients to identify imaging markers of RIR.

Purpose: We aimed to identify signs of residual inflammation in recent AMI patients via CTCA atheroma quantification. We hypothesised that recent AMI patients have plaque characteristics suggestive of inflammation.

Methods: 108 patients recruited from multiple sites with AMI < 1 month were propensity-matched via CAD risk factors (age, gender, diabetes, hypertension, hyperlipidaemia, smoking and family history of CAD) with stable CAD patients in a 1:1 ratio. Propensity matched recent AMI and stable CAD patient cohorts had similar profiles: age (60±9.7 vs 57±10.0 years), gender (91.7% vs 92.6% male), diabetes (13.9% vs 15.7%), hypertension (44.4% vs 50.9%) and hyperlipidaemia (50.9% vs 63.9%, p>0.05 for all). Total plaque burden (total lesion volume/total vessel volume) and plaque composition (fibrous fatty [fibrofatty, FF, 31–130 HU], necrotic core [NC, -30–30 HU], fibrous [131–350 HU] and calcium [>350 HU]) were extracted from CTCA scans and aggregated per patient. Culprit lesions of recent AMI patients were excluded from the analysis due to the fact that coronary stents would affect plaque characterisation. Multivariate linear regression, corrected for CAD risk factors, was used to compare these parameters between the two cohorts.

Results: Recent AMI patients and stable CAD patients had similar total plaque burden (β=0.001±0.013, p=0.950, Figure 1a). Patients with recent AMI had higher non-calcified plaque volume ratio (β=0.071±0.026, p<0.05, Figure 1b) due to higher FF (β=0.094±0.019, Figure 1d) and NC (β=0.025±0.005, Figure 1e) plaque volume (p<0.05 for all), but less fibrous plaque volume (β=-0.048±0.019, p<0.05, Figure 1c).

Conclusions: Recent AMI patients had a greater non-calcified plaque burden than stable CAD patients due to larger FF and NC plaque burden. Since FF and NC components indicate ongoing inflammation in the plaque, while fibrous content indicates resolved inflammation; increased NC and FF burden suggest increased coronary plaque inflammation in non-culprit lesions of recent AMI patients, supporting the presence of residual inflammation in recent AMI patients.