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Soluble urokinase plasminogen activator receptor and vulnerable plaque: high suPAR is associated with larger coronary plaque necrotic core and dense calcium in non-obstructive coronary artery disease

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Background: Soluble urokinase-type plasminogen activator receptor (suPAR) is a marker of inflammatory and immune activation and is strongly predictive of adverse cardiovascular and renal outcomes. However, the relationship between elevated suPAR levels and coronary plaque vulnerability remains unknown. We hypothesized that suPAR levels will be associated with greater coronary plaque burden and the "vulnerable plaque" phenotype defined by intravascular ultrasound (IVUS).

Methods: We measured plasma suPAR by ELISA in 62 patients with non-obstructive coronary artery disease (CAD, fractional flow reserve <0.75) who underwent radiofrequency IVUS (VH-IVUS). The association between suPAR levels and VH-IVUS-defined plaque burden (plaque area/vessel area × 100) and composition (necrotic core, dense calcium, and fibrous and fibrofatty tissue) was characterized using univariable and multivariable analyses. Multivariable analyses were adjusted for age, sex, eGFR, diabetes, and hypertension.

Results: Median age was 57 (IQR 48, 63) years, 51% men, 23% diabetic, and 71% hypertensive. Median suPAR level was 2.81 (IQR 1.99, 3.44) ng/mL and median plaque burden was 38 (IQR 27, 48)%. In univariable analyses, suPAR levels correlated with percent necrotic core (r=0.30, p<0.01) and percent dense calcium (r=0.27, p=0.04) among plaque components. These associations remained significant in multivariable analyses adjusting for the aforementioned factors (β=0.24 [0.01, 0.49], p<0.047 and β =0.03 [0.03, 1.07], p=0.038, respectively). There was no correlation between suPAR levels and plaque burden.

Conclusions: In patients with non-obstructive CAD, higher suPAR levels were associated with a larger plaque necrotic core and dense calcium. It is likely that our previous findings of increased cardiovascular event rate in those with higher suPAR levels is secondary to increased plaque vulnerability (high risk plaque) in those with elevated suPAR levels.

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Effect of early pitavastatin therapy on coronary fibrous-cap thickness assessed by optical coherence tomography in patients with acute coronary syndrome: the ESCORT study

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Purpose: To investigate the role of MAIR-II in MI pathophysiology.

Methods and results: In flow cytometric analysis, MAIR-II+ myocardial cells were abundant from post-MI days 3 to 5 in injured hearts induced by permanent ligature of coronary artery. To address the role of MAIR-II in myocardial cell function in vivo, effects of MAIR-II deficiency were investigated. In echocardiography, MAIR-II knockout (KO) mice had thicker left ventricle posterior walls and higher ejection fractions compared to wild-type (WT) mice. This indicates that MAIR-II deficiency leads to favorable post-MI remodeling. After further investigation, we found that MAIR-II KO hearts had less macrophage influx and more neutrophil infiltration after MI. Moreover, M1 and M2 macrophages are known to have inflammatory and healing roles after MI respectively. In MAIR-II KO mice, there was less IL1β inflammatory gene expression, less Tgfβ and collagen type I α 2 fibroblast gene expressions, and more CD206+ M2 macrophages in the infarcted heart compared to WT.

Conclusion: MAIR-II+ myocardial cells cause an early influx of inflammatory M1 macrophages and suppress both anti-inflammatory M2 macrophage and neutrophil infiltration. This in turn, leads to increased inflammation and fibrosis and thus, poor remodelling and unfavourable prognosis after MI.