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Acute Coronary Syndromes

HIGHER INTER-CELLULAR-ADHESION MOLECULE 1 LEVELS IN ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION PATIENTS ASSOCIATED WITH PLAQUE VULNERABILITY IN NON-CULPRIT LESIONS AT 10 MONTH FOLLOW-UP

ACC Moderated Poster Contributions

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Background: Several studies have reported risks of adverse late and early cardiac outcomes associated with elevated levels of inter-cellular-adhesion molecule 1 (ICAM-1) in acute coronary patients. ICAM-1 cause the migration of the monocytes into the intima of the coronary artery thus playing an important role in inflammatory cascade. Plaque composition is another indicator for extent of ongoing inflammation in coronary lesion. We hypothesized that higher ICAM-1 levels could be related to plaque vulnerability in non-culprit lesions.

Methods: A total of 65 patients presenting with ST-segment elevation myocardial infarction (STEMI) at Latvian Centre of Cardiology were prospectively enrolled in this study. After thrombus aspiration, IVUS with i-Map tissue characterisation (Qivus 2.0, Medis medical imaging systems by, Leiden, the Netherlands) of the infarct-related artery followed by stent implantation was done. Fibrotic and necrotic tissue were analyzed as categorical values. As cut off points of procentual plaque composition for necrotic and fibrotic tissue were defined as 30% and 60%, respectively.

Results: Higher levels of ICAM-1 at index event were associated with necrotic tissue above 30% at distal segment (433.71 ± 250.58 vs 269.88 ± 106.97 (ng/ml), $p=0.004$) and proximal segment (359.22 ± 195.22 vs 240.94 ± 80.43 (ng/ml), $p=0.004$). Similarly, higher levels of ICAM-1 at follow-up were associated with necrotic tissue above 30% at distal segment (480.35 ± 282.20 (ng/ml) vs 263.25 ± 114.59 (ng/ml), $p=0.001$) and proximal segment (360.63 ± 208.98 (ng/ml) vs 234.81 ± 95.91 (ng/ml), $p=0.007$). Lower levels of ICAM-1 at index event were associated with fibrotic tissue above 60% at distal segment (270.79 ± 107.82 vs 403.69 ± 242.16 (ng/ml), $p=0.013$) and proximal segment (243.90 ± 83.90 vs 333.82 ± 185.11 (ng/ml), $p=0.024$). Lower levels of ICAM-1 at follow-up were associated with fibrotic tissue above 60% at distal segment (264.62 ± 115.39 vs 433.21 ± 277.57 (ng/ml), $p=0.007$) and proximal segment (236.86 ± 101.56 vs 334.13 ± 196.16 (ng/ml), $p=0.033$).

Conclusions: Higher levels of ICAM-1 were associated with plaque vulnerability in non-culprit lesions in STEMI patients at 10 month follow-up.