18.1.4 - Vulnerable Plaque

Blood iron level and vulnerable coronary atherosclerotic plaques

Oleksiak A.[1]; Kruk M.[2]; Rucinska KI.[3]; Marcinkiewicz K.[1]; Demkow M.[2]; Kepka C.[2]

[1]National Institute of Cardiology, Department of Intensive Cardiac Therapy, Warsaw, Poland
[2]National Institute of Cardiology, Department of Coronary and Structural Heart Diseases, Warsaw, Poland
[3]Medical University of Warsaw, Warsaw, Poland

Funding Acknowledgements: Type of funding sources: Public grant(s) – National budget only. Main funding source(s): National Science Centre, Poland

BACKGROUND: Vulnerable plaque rupture is one of the causes of acute coronary syndromes. Preliminary research indicate that iron might accelerate the oxidation of low density lipoproteins (LDL) which can then be taken up by the LDL receptor on macrophages leading to their development into foam cells. Foam cell infiltration and necrotic core expansion are key events in atherogenesis and vulnerable plaque formation. However, the potential pathophysiological roles of iron in plaque development remain uncertain.

PURPOSE: The aim of the study was to investigate the relationship between iron and the type and composition of atherosclerotic plaques in the coronary arteries.

METHODS: In 200 patients with ≥1 stenosis ≥50% in computed tomography coronary angiography (CTCA) made for clinical indications we assessed: free iron level, the presence of high-risk plaque features: low-attenuation plaque (LAP), napkin-ring sign (NRS), positive remodeling (PR) and spotty calcium (SC) (CT Coronary, Syngo, Siemens), type of plaque (calcified, mixed, non-calcified) and their composition (calcified, fibrous, fibro-fatty, necrotic core) (QAngioCT, Medis). Fibro-fatty and necrotic core were analyzed together as vulnerable plaque component. The study was financed by the National Science Centre (2016/21/N/NZ5/01450).

RESULTS: Of 200 patients (125 men, 66 ± 10 years), the mean iron level (μg/dl) was 91 ±30 for women and 103 ±33 for men ($p = 0.5$). 3 patients had iron deficiency and 2 patients had iron overload. In CTCA analysis there were 815 calcified, 344 non-calcified and 438 mixed plaques. There was a trend in correlation between iron level and non-calcified plaque presence ($p = 0.06$). LAP was detected in 56 patients, NRS in 83, PR in 132, and SC in 125. Patients with LAP had higher iron levels (113 vs 93 μg/dl; $p < 0.001$). There was no association between iron and NRS, PR or SC ($p > 0.05$). In univariate regression analysis, the predictors of LAP were iron ($p < 0.001$) and male gender ($p = 0.01$). In multivariate regression analysis, iron was an independent predictor of LAP ($p < 0.001$; OR 1.02; 95%CI 1.01-1.03). Higher iron levels correlated with more fibro-fatty ($p = 0.009$) and necrotic core ($p = 0.02$); less calcified ($p = 0.04$); and with no relation to fibrous ($p = 0.9$), thus higher iron levels were associated with greater vulnerable plaque component ($p = 0.003$).

CONCLUSIONS: Higher iron levels are more likely to be associated with low-attenuation plaque and a greater vulnerable component of atherosclerotic plaques.