VASCULAR INFLAMMATION IN THE AORTA IS RELATED TO CORONARY PLAQUE BURDEN IN PSORIASIS

Poster Contributions
Poster Hall B1
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Background: Psoriasis, a chronic inflammatory skin disease, is associated with increased vascular inflammation (VI) by [18]-fluorodeoxyglucose (FDG) PET/CT. Psoriasis also increases the risk of myocardial infarction, which may be due to inflammatory, lipid-rich coronary plaque. Whether VI in the aorta is related to coronary plaque burden is not known and is a critical step in understanding local effects of vascular inflammation. Therefore, we examined the relationship between VI by FDG PET/CT and coronary plaque burden by quantitative CT angiography in a well-phenotyped psoriasis cohort.

Methods: Psoriasis patients (N=67) and healthy controls (N=21) underwent coronary CT angiography (Toshiba 320 slice) and FDG PET-CT imaging (Siemens Biograph). Coronary plaque was assessed using QAngio CT (Medis, The Netherlands). Total (TB), dense calcium (DCB), and non-calcified burden (NCB) plaque indices were calculated by dividing total vessel plaque volume by total vessel length. VI was assessed using dedicated image analysis software (Phillips Healthcare). Target-to-background Ratio (TBR) was calculated as the ratio of arterial and venous standardized uptake values (SUV). Cardiometabolic parameters including lipid particles, HDL efflux, and homeostatic model assessment-insulin resistance (HOMA-IR) were also assessed.

Results: The population had a low Framingham Risk Score (median 4%, IQR 2-7%) but had high VI (1.82 vs 1.68 in psoriasis vs healthy controls, respectively; p<0.001). In univariate regression, an increase in aortic VI was associated with an increase in coronary TB (β=0.80, p<0.001) and NCB (β=0.71, p<0.001) even after adjustment for cardiovascular risk factors (β=0.56, p=0.02; β=0.47, p=0.02, respectively). Both VI and coronary plaque burden were associated with HDL function (β= -0.57, p<0.001; β= -0.89, p=0.002, respectively) and HOMA-IR with VI (β=0.04, p<0.001).

Conclusion: We show that VI is increased in PSO and that this VI is associated with coronary plaque burden. Vascular inflammation in the aorta therefore may be a strong surrogate marker for subclinical atherosclerosis.