DIRECTLY MEASURED INFLAMMATION IN PSORIASIS SKIN ASSOCIATES WITH VASCULAR INFLAMMATION BEYOND TRADITIONAL RISK FACTORS

Poster Contributions
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Background: Inflammation is critical to development and progression of atherosclerosis. Psoriasis, a chronic inflammatory disease characterized by skin and systemic inflammation, is associated with increased cardiovascular diseases (CVD). We have previously demonstrated increased vascular inflammation by FDG PET CT (18F-fluorodeoxyglucose positron emission tomography/computed tomography) in psoriasis. It is currently unknown whether directly-quantified skin inflammation is related to CVD. We hypothesized that increased skin inflammation is associated with increased vascular inflammation persisting beyond adjustment for CVD risk factors.

Methods: Clinical and laboratory measures of inflammation were prospectively evaluated in 41 consecutive adult psoriasis patients (NCT01778569). Psoriasis severity was assessed using Psoriasis Area Severity Index (PASI). Aortic vascular inflammation was assessed using a validated measurement from clinical trials, the target-to-background ratio (TBR), assessed by FDG PET/CT. Multivariate linear regression was used to assess for association between TBR and PASI, adjusting for age, sex, hypertension, hyperlipidemia, diabetes, tobacco use, alcohol and body mass index. Analyses were conducted using STATA 12.0 software.

Results: No statistically significant difference was seen in demographic or metabolic risk factors between psoriasis severity groups. Framingham 10-year risk scores were low in our cohort (males mean 6.9, SD 5.4; females mean 4.5, SD 4.3; p=0.11). In unadjusted analyses, hsCRP (ρ=0.34, p=0.03) and psoriasis body surface area (ρ=0.79, p<0.001) were significantly associated with PASI. As expected, CVD risk factors were each associated with TBR (p<0.001 for all). Furthermore, as PASI increased, we observed increased TBR (unadjusted β=0.007, p<0.01) beyond CVD risk factors (adjusted β=0.002, p=0.012).

Conclusions: Directly assessed skin inflammation independently associated with vascular inflammation beyond CVD risk factors. These findings suggest that inflammation in vivo in humans heightens the risk for CVD. Whether treatment of this inflammation modulates CVD risk is unknown, and is topic of ongoing study.