208. PLASMA MICROPARTICLE LEVELS ARE NOT RAISED IN PATIENTS WITH ANKYLOSING SPONDYLITIS

Nicholas A. Bradley
1 School of Medicine, University of Liverpool, Liverpool, UK

Background: Microparticles are vesicles of plasma membrane formed by cellular stimulation or apoptosis. Variations in their membrane bound proteins and contents result in heterogeneity between samples, allowing for identification of their source and role in disease. Intravesicular contents such as microRNA and prothrombotic factors are implicated in the development of cardiovascular disease, and patients with inflammatory arthropathies have increased risk of adverse cardiovascular event, a proportion of which remains when traditionally described risk factors are discounted. Whilst inflammatory conditions such as RA have had extensive microparticle investigation, to date only one study describes levels of microparticles in AS, showing no significant difference in counts between cases and controls. This study aims to further characterize levels of microparticles in AS patients, and add novel data describing their cellular origin and contents (including microRNA analysis).

Methods: 11 patients diagnosed with AS under the care of University Hospital Aintree (UHA) rheumatology department were recruited in the outpatient setting, and matched for age and gender with 10 controls recruited from staff at UHA Clinical Sciences department. Mean ages were 46.5 and 41.4 in cases and controls respectively. Baseline data on disease activity, cardiovascular risk factors and demographic characteristics were obtained from case notes at recruitment. Mean disease activity at recruitment (BASDAI) was 4.3. 6 patients were receiving anti-TNF therapy. Microparticles were isolated from venous blood by serial centrifugation. These were resuspended in HBSS and analysed for count, CD surface markers and annexin-V (AV) fluorescence using flow cytometry.

Results: The data generated were not normally distributed thus figures quoted are median (IQR), and the Mann–Whitney U test used to assess statistical significance. Microparticle counts per ml of blood were $2.29 \times 10^6$ ($4.16 \times 10^6$) and $2.95 \times 10^6$ ($1.88 \times 10^6$) in cases and controls respectively ($P = 0.0843$). CD proteins CD4, CD62, CD14 and VCam1 showed increased expression in patients’ microparticles compared with controls ($P < 0.05$), whilst CD41 and CD54 showed decreased expression in patients ($P < 0.05$). AV fluorescence (given as % positive expression) was 67.58% (33.42%) vs 98.27% (4.37%) in cases and controls ($P = 0.0014$). No significant differences between high and low disease activity cohorts or between patients receiving biologic vs non-biologic therapies were observed.

Conclusion: These data show that whilst there is no significant difference in number of microparticles in AS patients compared with controls, their origin is different both in terms of the cellular source and the mechanism behind their formation; increased AV fluorescence suggests increased proportions of microparticles formed under apoptotic stimulus in cases compared with controls. Our intended recruitment of more patients to increase sample size may reveal significant results not yet described.

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