Conclusions: CCR10 + CD8 + T cells have regulatory properties and differentiate Pso from PsA. Future studies are needed to uncover if aberrances in regulatory, cutaneous CD8 + T cells precede the transition to joint disease.

Disclosure of Interest: None declared

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AB0143

IMPLICATION OF OSTEONECTIN ON CARDIOVASCULAR RISK IN AXIAL SPONDYLOARTHRITIS: A SEROLOGICAL AND GENETIC STUDY

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Background: Cardiovascular (CV) disease and atherosclerosis are common causes of morbidity in axial spondyloarthritis (axSpA),¹ a disease characterised by changes in the osteoproliferative process. A dysregulation in the molecules involved in bone remodelling could also affect the atherosclerotic process, since both processes are linked.²⁻³ Osteonectin (ON), a key molecule in bone homeostasis,⁴ was associated to obesity, insulin resistance and diabetes.⁵

Objectives: Since the exact role of ON on CV risk in axSpA has not been elucidated yet, we evaluated its role in the development of subclinical atherosclerosis and its association with CV risk factors in axSpA patients at the serological and genetic level.

Methods: 171 axSpA patients fulfilling the classification criteria for axSpA^{6,7} and 84 controls were included in this study. Serum ON levels were measured by multiplex assays. Carotid ultrasound was performed to evaluate the presence of markers of subclinical atherosclerosis. Five ON polymorphisms (rs1054204 [G/C], rs11950384 [G/A], rs13182103 [A/G], rs11745387 [G/A] and rs4958487 [A/G]) were selected by tagging and genotyped using TaqMan assays.

Results: No difference was observed in ON levels between axSpA and controls. Serum ON and CRP levels at study positively correlated in axSpA ($p=0.008$). Higher ON levels were observed in men, smokers and patients with an atherogenic index indicative of dyslipidemia ($p=0.008$, 0.01 and 0.001 , respectively). No association was found between ON and subclinical atherosclerosis in axSpA. Interestingly, we disclosed that the presence of the A allele of rs13182103 and rs11950384 ON polymorphisms led to lower ON serum levels in axSpA ($p<0.05$). Furthermore, the presence of the A allele of rs13182103 was linked to a later diagnosis of axSpA when compared to those patients bearing the G allele ($p=0.002$).

Conclusions: Our results show that ON is linked to inflammation and CV risk factors in axSpA and suggest that the A allele of rs13182103 and rs11950384 ON polymorphisms may have a protective effect in axSpA, leading to reduced ON serum levels and later diagnosis of the disease. These data support an implication of ON in the development and progression of atherosclerotic disease in axSpA.

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AB0144

NMR BASED SERUM AND SYNOVIAL FLUID METABOLOMICS REVEAL SIMILAR METABOLOMIC PROFILE IN PATIENTS WITH REACTIVE ARTHRITIS AND UNDIFFERENTIATED SPONDYLOARTHRITIS

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Background: Reactive arthritis (ReA) and undifferentiated spondyloarthritis (USpA) have similar clinical picture, the former is preceded by mucosal infections and the latter do not meet criteria of well defined entity of seronegative spondyloarthritis (SSA). We have shown that both ReA and USpA have similar synovial fluid T cell response to salmonella antigens and cytokine profile.

Objectives: To investigate if the metabolic profiles of ReA in sera and synovial fluid are different from those of USpA

Methods: Standard definitions were used to classify patients as ReA² and USpA³. Metabolic profiles in sera and synovial fluid samples were measured using 1D ¹H NMR spectroscopy and analysed using CHENOMX® software. The quantitative profiles between the groups were compared using Partial Least Square-Discriminatory Analysis (PLS-DA) and Q² parameter was used to assess the metabolic differences between the groups.

Results: The study involved 19 ReA and 13 USpA patients with median age 26 (21–33.75) years. Of 32 patients: 7 (22%) were females, three (12%) had mono-phasic illness, 16 (64%) polyphasic, and six (24%) persistent arthritis (follow-up data was not available for 7). 27% had monoarticular, 59% oligoarticular and 14% polyarticular involvement. Six had inflammatory backpain, three had oral ulcers, one dactylitis, but none had mucocutaneous manifestations. Two had asymptomatic sacroiliitis on radiographs.

Compared to normal controls ($n=18$, median age=29 years, male:female=17:1), the sera of ReA/USpA patients were characterised by elevated levels of malonate, mannose, N-nitrosodimethylamine, and pyruvate, whereas 3-hydroxyisovalerate was decreased significantly (T-test p -value<0.001). PLS-DA analysis between sera and synovial fluid samples showed clear demarcation with higher pyruvate and acetoacetate and lower aspartate, methanol, ethanol, and methylsuccinate in synovial fluid samples compared to sera (t-test $p<0.01$). The PLS-DA analysis of sera showed little difference between ReA and USpA (figure 1) while that of synovial fluid showed Q²<0 for all models. Further no significant metabolic differences in sera/synovial fluid were found between HLA-B27 positive and negative groups (Q²<0 in all models).

Abstract AB0144 – Figure 1. (a) PLS-DA of sera of HLA B27 positive versus negative patients (b) PLS-DA of synovial fluid of HLA B27 positive versus negative patients

Conclusions: ReA and USpA have indistinguishable metabolomics profiles in sera and synovial fluid reflecting similar metabolite-inflammatory pathways involved.

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