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Background: Vaspin is a novel anti-inflammatory adipokine associated with cardiovascular (CV) disease and inflammation in chronic inflammatory conditions different from axial spondyloarthritis (axSpA).¹ Given the high incidence of CV disease (mainly due to accelerated atherosclerosis) exhibited by axSpA patients,² we wondered if vaspin could also be a key molecule in this process. However, data on the role of vaspin regarding atherosclerotic disease in the context of axSpA is scarce.³

Objectives: To evaluate the implication of vaspin, at the genetic and serological level, in subclinical atherosclerosis and CV risk in axSpA.

Methods: 510 patients who fulfilled the ASAS criteria for axSpA⁴ were included in this study. Carotid ultrasound (US) was performed to evaluate the presence of subclinical atherosclerosis. Three vaspin gene variants (rs2236242 T/A, rs7159023 G/A and rs35262691 T/C) were genotyped by TaqMan probes. Serum vaspin levels were assessed by Enzyme-Linked Immunosorbent Assay. Analysis was performed using a statistical software.

Results: Serum vaspin levels were significantly higher in female patients than in males and also in obese patients when compared to those with normal weight ($p < 0.05$). At the genetic level, we disclosed that the minor allele of rs2236242 (A) was associated with lower serum vaspin levels in axSpA, while the rs7159023 minor allele (A) was linked to higher serum levels ($p < 0.05$). When the three polymorphisms assessed were combined conforming haplotypes, we disclosed that the TGC haplotype related to high serum levels of vaspin ($p = 0.01$). However, no statistically significant association was observed between vaspin and markers of subclinical atherosclerosis, both at the genetic and serological level.

Conclusion: Our results revealed that vaspin is linked to CV risk factors that may influence on the atherosclerotic process in axSpA. Additionally, we disclosed that serum vaspin concentration is genetically modulated in a large cohort of patients with axSpA.

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Acknowledgements: Personal funds: RL-M: Miguel Servet type I CP16/00033 (ISCIII-ESF); SR-M: RD16/0012/0009 (ISCIII-ERDF); VP-C: PREVAL18/01 (IDI-VAL); LL-G: INVAL20/06 (IDIVAL).

Disclosure of Interests: Javier Rueda-Gotor: None declared, Raquel López-Mejías: None declared, Sara Remuzgo-Martínez: None declared, Verónica Pulito Cueto: None declared, Alfonso Corrales: None declared, Leticia Lera-Gómez: None declared, Virginia Portilla: None declared, Iñigo González-Mazón: None declared, Ricardo Blanco Speakers bureau: Abbvie, Pfizer, Roche, Bristol-Myers, Janssen and MSD, Consultant of: Abbvie, Pfizer, Roche, Bristol-Myers, Janssen and MSD, Grant/research support from: Abbvie, MSD and Roche, Rosa Expósito: None declared, Cristina Mata: None declared, Javier Llorca: None declared, Vanessa Hernández-Hernández: None declared, Carlos Rodríguez-Lozano: None declared, Nuria Barbarroja Puerto: None declared, Rafaela Ortega Castro: None declared, Noelia García Castañeda: None declared, Cristina Fernández-Carballido: None declared, María Paz Martínez-Vidal: None declared, David Castro-Corredor: None declared, Joaquín Anino-Fernández: None declared, Diana Peiteado: None declared, Chamaida Plasencia: None declared, E Galindez: None declared, María L. García Vivar: None declared, Oreste Gualillo: None declared, Juan Carlos Quevedo-Abeledo: None declared, Santos Castañeda: None declared, Iván Ferraz-Amaro: None declared, Miguel A González-Gay Speakers bureau: Pfizer, Abbvie, MSD, Grant/research support from: Pfizer, Abbvie, MSD, Fernanda Genre: None declared

DOI: 10.1136/annrheumdis-2021-eular.839

AB0071

RELATIONSHIP BETWEEN PRO-INFLAMMATORY CYTOKINE AND ATHEROGENIC INDEXES IN AXIAL RADIOGRAPHIC SPONDYLOARTHRITIS

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Background: The cardiovascular burden in inflammatory rheumatic diseases is well recognized. This burden has been reported in spondyloarthritis. Atherogenic indexes are known with their role of predicting cardiovascular risk.

Objectives: The aim of our study was to determine the relation between pro-inflammatory cytokines and atherogenic indexes in spondyloarthritis.

Methods: We performed a cross sectional study including 38 patients with spondyloarthritis according to ASAS criteria. For each patient we measured interleukin (IL)1, IL6, IL17, IL23 and tumor necrosis factor (TNF) alpha, total Cholesterol (CT), Triglycerides (TG), High density lipoprotein Cholesterol (HDLc) and Low density lipoprotein cholesterol (LDLc). We also calculated the following ratios: CT/HDLc, TG/HDLc, LDLc/HDLc, and LogTG/HDLc.

Disease activity was measured using ASDAS (Ankylosing Spondylitis Disease Activity Score) and BASDAI (Bath Ankylosing Spondylitis Disease Activity Index). Statistical analysis was performed using IBM SPSS Statistics 25.

Results: The mean age were 45.4±12.5 years. There were 33 Male (sex ratio were 6.6).

The mean C-reactive protein(CRP) were 29.6 ±40.34mg/L, mean erythrocyte sedimentation rate(ESR) were 41±33.9mm.

The mean ASDASCRP, BASDAI were 2.8±1.24, 3.7±2.12, respectively.

Active disease were noted in 68 % of patients using ASDASCRP score.

The mean IL1, IL6, IL17, IL23, TNF alpha were 11.6 ±25pg/ml, 15.4±45.9pg/ml, 84.6±77.9pg/ml, 15.3±15.26pg/ml, 25.3±47.9pg/ml respectively.

The mean CT,HDLc,LDLc, TG were 4.47±0.93mmol/l, 1.09±0.3mmol/L, 2.77±0.78mmol/L, 1.29±0.54mmol/L respectively.

The mean CT/HDLc, TG/HDLc, LDL/HDLc, LogTG/HDLc were 4.28±1.26, 1.22±0.6, 2.7±1.06, 0.03±0.23, respectively.

Correlations were found between TG/HDLc ratio and IL1 ($p < 0.01$, $r = 0.515$), IL6($p < 0.05$; $r = 0.407$), Log TG/HDLc and IL1 ($p < 0.05$, $r = 0.369$), and IL6($p < 0.05$; $r = 0.333$)

However, no correlations were noted between atherogenic indexes and IL7, IL17, IL23, TNFalpha.

Conclusion: Our study showed a correlations between atherogenic indexes and both IL1 and IL6.

These findings suggests that serum IL-6 and IL1 levels in spondyloarthritis contribute to the development of cardiovascular disease atherosclerosis.

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Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.1281

AB0072

SERUM AMYLOID A PROTEIN (SAA) IN ANKYLOSING SPONDYLITIS AND THE RELATIONSHIP WITH SYSTEMIC INFLAMMATION

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Background: Serum amyloid A protein (SAA) likely has a critical role in control and possibly propagation of the primordial acute phase response and is the precursor of AA amyloid fibrils. Prolonged elevations in SAA are the major inciting factor for AA amyloidosis developing in chronic inflammatory diseases. In Russia 2-4% of patients with ankylosing spondylitis (AS) have secondary (AA) amyloidosis.

Objectives: To study the level of SAA in AS its relationship with indicators of disease activity.

Methods: 124 patients with AS (according to mNYC 1984) 70 men, 54 women, of whom HLA B 27 positive 91.1% mean age 38.1 (± 12.9), age at the onset of the disease 23.5 (± 9.9) consecutively admitted to the clinic of the Research Institute rheumatology from February to November 2020. In addition to the standard examination (the median CRP 6.7 mg/l [1.4; 24.9], ESR 13 mm/h [7; 27], SAA was studied in all patients by the nephelometric method.

Results: The median SAA in 124 patients was 12.5 mg/l [4;71.6]. Among them, 31% had normal SAA level (<5 mg/l), and 69 % - more than 5 mg/l. In 21 (17.5 %) cases, the level of SAA was increased at normal CRP levels, and only in 2 cases an increase in the level of CRP at normal SAA levels; 50 patients (40.3 %) with normal ESR had elevated SAA levels, and 7 (5.6%) - ESR exceeded the upper limits of the norm with normal SAA levels. Comparison of the average values of the levels of SAA, CRP, ESR in men and women did not reveal significant differences between them. The SAA level was weakly correlated with ESR ($r = 0.2$; $p = 0.002$) and BASDAI ($r = 0.3$; $p = 0.002$), moderately with ASDAS-CRP ($r = 0.5$; $p < 0.0001$), but showed a strong association with CRP ($r = 0.80$; $p < 0.00001$). Patients with elevated SAA levels (>5 mg/l) had a shorter disease duration (10 and 12 year; $p < 0.0004$), higher ASDAS-CRP (2.9 and 2.4; $p < 0.003$), blood CRP level (14.6 and 1.3; $p < 0.00001$), and significantly more peripheral arthritis (60% and 39%; $p < 0.05$) than patients with normal indicators.

Conclusion: The level of SAA correlates well with indicators of AS activity, especially with the level of CRP, and can be used as an alternative indicator of disease activity.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.1782