Conclusion: Hsa_circ_0012732, hsa_circ_0008961, hsa_circ_0405239 and hsa_circ_0068784 may be related to the pathogenesis of AS. Among them, hsa_circ_0012732 may be involved in AS inflammation and has the potential to participate in the judgment of disease activity.

REFERENCES:

- [1] LS, K., et al., The biogenesis, biology and characterization of circular RNAs. 2019. 20(11): p. 675-691.
- [2] J, W., et al., Non-coding RNAs in Rheumatoid Arthritis: From Bench to Bed-side. 2019. 10: p. 3129.

Figure 1 Relative expression levels of hsa_circ_0012732, hsa_circ_0008961 among the 3 groups

Disclosure of Interests: Yi-Ping Tang Grant/research support from: Science and Technology Project of Nanchong City (18SXHZ0522)., Yu-Feng Qing: None declared, Zeng-Rong Dong: None declared, Fei Dai: None declared, Jianxiong Zheng: None declared, Yi Jiang: None declared, Yi-Xi He: None declared, Quan-Bo Zhang Grant/research support from: National Natural Science Foundation of China (General Program) (81974250); Science and Technology Plan Project of Sichuan Province (2018JY0257)

DOI: 10.1136/annrheumdis-2021-eular.3412

AB0076 THE ROLE OF MACROPHAGE MIGRATION INHIBITORY FACTOR IN ACTIVITY AND PROGRESSION OF ANKYLOSING SPONDYLITIS

Y. Gazar¹, M. Hanafy¹, M. Elrefi². ¹Faculty of Medicine, AL-Azhar university, Rheumatology and Rehabilitation, cairo, Egypt; ²Military Hospitals, Rheumatology, cairo, Egypt

Background: Ankylosing Spondylitis (AS) is a chronic inflammatory form of arthritis that predominantly affects the axial joints and has a global prevalence of 1%. Disease progression is marked by osteoproliferation and bone fusion in the axial joints, causing considerable loss of mobility. Migration Inhibitory Factor (MIF) is a potent pro-inflammatory cytokine implicated in several diseases. Elevated serum MIF levels have been reported in AS the role of MIF in bone formation has been a subject of controversy.

Objectives: Detection the level of Migration Inhibitory Factor in Ankylosing Spondylitis patients and correlate the level with disease activity and progression.

Methods: This cross-sectional case-control study were concluded upon 70 randomly selected people from internal medicine department in Kobry El-kobba Military Hospital and from rheumatology and rehabilitation department in EL-Hussein university hospital. The population study were divided into two groups: Group A: (50) Patients with Ankylosing Spondylitis (AS) who met the European Spondyloarthritis Study Group (ESSG) criteria for AS (30) and Group B: (20) Healthy controls. Serum MIF by ELISA were measured. X-rays cervical & lumbosacral vertebrae (dorsal, lateral) views were obtained. m-SASS score (based on radiographic findings to detect progression) were assessed. MSK U/S on Tendo Achilles affected in AS patients were done.

Results: the mean age of all patients was (38.2 ± 8.4) years. Regarding gender of the patients, the majority (65.7%) of patients were males, while (34.3%) were females. (88%) of AS patients had cervical erosion or sclerosis, (46%) had dorsal erosion or sclerosis, (92%) had lumbosacral erosion or sclerosis, with m-SASS score of (11 ± 6.1). Regarding musculoskeletal U/S, (72%) had evidence of

inflammation and erosion. Highly significant increase in ESR, CRP and MIF levels in A group; compared to control B group; with highly significant statistical difference (p < 0.01). the increase in smoking, ESR and MIF level; had an independent effect on increasing the probability of progression occurrence; with significant statistical difference (p < 0.05 respectively). MIF level at a cutoff point (>51) predicted patients with progression, with fair accuracy (74%), sensitivity= 53% and specificity= 94% (p = 0.0056).

Conclusion: MIF play an important role in the pathogenesis of Ankylosing Spondylitis and has a link between chronic inflammation, new bone formation and disease progression and can be used as indicator for activity and disease progression.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.3421

SLE, Sjögren's and APS - aetiology, pathogenesis and animal models

AB0077

ASSOCIATION BETWEEN VITAMIN D RECEPTOR GENE POLYMORPHISMS AND SYSTEMIC LUPUS ERYTHEMATOSUS IN MALTESE PATIENTS

B. Magro¹, J. A. Grech Meli², J. Debatista³, N. Aquilina⁴, K. Gatt⁵, A. Borg⁶, C. Scerri². ¹Mater Dei Hospital, Rheumatology, Msida, Malta; ²University of Malta, Faculty of Medicine and Surgery, Msida, Malta; ³University of Malta, Faculty of Medicine and Surgery, Msida, Malta; ⁴Mater Dei Hospital, Medicine, Msida, Malta; ⁵Mater Dei Hospital, Medicine, Msida, Malta; ⁶Mater Dei Hospital, Rheumatology, Msida, Malta

Background: Vitamin D deficiency is highly prevalent in patients with systemic lupus erythematosus (SLE). Vitamin D acts through the vitamin D receptor (VDR) that is present in most cells, and it can regulate the transcription of over 200 genes. The expression of vitamin D receptors by a variety of cells belonging to the innate and adaptive immune systems has created interest with regards to the role of vitamin D in the pathogenesis of SLE. Several polymorphisms of the VDR gene have been described, namely BsmI, Apal, TaqI and FokI. A number of VDR gene polymorphism genotypes have been associated with increased risk of SLE mostly in Asians and Africans.¹

Objectives: The aim of this study was to establish whether an association was present between VDR gene polymorphisms and SLE susceptibility in a cohort of SLE patients living in Malta. A further aim was to assess the relationship between these VDR gene polymorphisms and SLE disease characteristics.

Methods: 59 SLE patients living in Malta and attending Rheumatology clinic at Mater Dei Hospital were recruited for the study after providing informed consent. The patients were over the age of 18 years and fulfilled the SLICC classification criteria for SLE. The patients were interviewed and blood samples were taken. RNA extraction was performed from whole blood. QuantiGene Plex technology was used to measure the expression of 12 interferon (IFN) signature genes in the extracted RNA. 93 cord blood samples obtained from individuals living in Malta were used as a control. DNA extraction was carried out from the blood samples obtained from the patients and controls. The VDR gene was screened and the regions containing the VDR polymorphisms were amplified for each patient. The amplified regions were then digested with their respective restriction enzymes in order to view the patient's genotype via restriction fragment length polymorphism. Statistical analysis, including odds ratio (OR), was carried out to gauge the significance in the association of these polymorphisms with SLE.

Results: 94.9% of SLE patients were female and they had a mean age of 44.5 years. All the patients were of Caucasian ethnicity. 13.6% had vitamin D deficiency (serum 25-hydroxyvitamin D <20ng/ml) and 25.4% were vitamin D insufficient (serum 25-hydroxyvitamin D 20-29ng/ml). The results showed that when Apal polymorphism was present as a homozygote for the variant allele there was a significant decrease in SLE prevalence (OR=0.39, CI 0.17-0.87, p=0.02). The results were also analysed by placing the polymorphisms into haplotypes. The haplotype containing all wild-type alleles for the VDR gene and the haplotype containing all wild-type alleles with the variant allele for FokI had an increased prevalence of SLE (OR= 1.95, CI 1.12-3.38, p=0.01 and OR= 2.36, CI 1.13-4.91, p=0.02 respectively). The patients who were homozygous for the variant allele for BsmI had a significantly higher SLE disease activity index-2K (SLEDAI-2K) (mean 5.00) compared to those that were heterozygous (mean 2.66; p=0.010). No significant difference was noted in damage, IFN signature gene expression, organ manifestation and autoantibody profile between the different genotypes for the 4 VDR polymorphisms. SLE patients who were homozygous variant for the Apal or TaqI polymorphisms had an increased prevalence of fibromyalgia (OR=7.50, CI 1.47-38.16, p=0.02 and OR=12.00, CI 1.80-80.05, p=0.02 respectively).

Conclusion: The study showed that in the Maltese population the presence of the VDR gene polymorphism haplotype containing all wild-type alleles and the haplotype containing all wild-type alleles with the variant allele for FokI are associated with an increased risk of SLE. Moreover the homozygous variant genotype for BsmI was associated with a higher SLE disease activity. The homozygous