to quantify the severity of patient general condition and DBI is an integral indicator of changes in the number of fatty acids and body general condition.

Conclusion: The presence of AH in patients with gout makes the clinical course of the disease more severe and exacerbates lipid disorders that can make significant changes in the formation of cardiovascular complications in this category of patients. DBI can be used as an additional criteria in laboratory diagnostics and monitoring to develop adequate treatment tactics.

Disclosure of Interests: Margarita Gromova Speakers bureau: Speaker for Biethnos, Boehringer-Ingelheim, Vladimir Tsurko Speakers bureau: Speaker for Boehringer-Ingelheim, Berlin-chemie, Oksana Kisliak Speakers bureau: Speaker for Berlin-chemie, KRKA, Elena Kiseleva: None declared

DOI: 10.1136/annrheumdis-2020-eular.2213

AB0929

CALCIUM PYROPHOSPHATE DIHYDRATE CRYSTAL-DEPOSITS: REMEMBER THE SPINE!

D. Khalifa¹, K. Baccouche¹, N. El Amri¹, H. Zeglaoui¹, E. Bouajina¹. ¹Farhat Hached Hospital, Rheumatology Department, Susah, Tunisia

Background: Calcium pyrophosphate dihydrate crystal-deposits (CPPD) is a common crystal disease affecting men and women equally. It is normally seen in peripheral joints. Spine involvement is rare and may mislead the diagnosis.

Objectives: To describe clinical, radiological and therapeutic findings of CPPD of the spine.

Methods: A retrospective descriptive study was conducted in the rheumatology department of Farhat Hached Hospital, including patients diagnosed with CPPD of the spine over a period of 20 years (1998-2018). Data concerning clinical, radiological and therapeutic aspects of CPPD of the spine were collected from their medical files.

Results: Twelve patients had a spinal localisation of CPPD. The mean age was 59.91±16.21 years. Patients were mainly women with a sex ratio men/women of 1:3. Mean duration of symptoms before the diagnosis was 27.08±25.69 months. Spinal presentation was the revealing symptom of CPPD in 45.5% of the cases. It affected the cervical spine in 66.7% and the lumbar spine in 33.3% of the cases. Spinal localisation of CPPD was associated with a peripheral arthropathy in 75% of the cases (the knees in 41.7%, wrists and hands in 66.6%, shoulders in 25% and the hips in 16.7%). Discovery of spinal CPPD was coincidental on plain radiographs in 16.7% of the cases. When symptomatic, the disease manifested itself as inflammatory pain in 72.7%, mechanical pain in 9.1% and both diurnal and nocturnal pain in 18.2% of the cases. Physical examination revealed stiffness of the spine in 58.3% of the patients. Fever was noted in 8.3% and deterioration of general condition with anorexia was reported in 25% of the patients. Neurological complications were represented by cervical myelopathy in 25%, sciatica in 8.3% and a case of unilateral ulnar nerve neuropathy was reported. Laboratory tests revealed inflammation in 50% of the cases. However, crystals were present in synovial fluid in only 25% of the patients. Spinal CPPD was visible on plain radiographs in 83.3% of the cases, showing calcified discs in 75% and signs of destruction in 16.7% of the cases. Peripheral calcifications were present in the wrists in 33.3% and the knees in 58.3% of the cases. Crow dens syndrome was reported in 16.7% of the patients. CT-scan helped the diagnosis of calcifications and crown dens syndrome when performed. MRI was prescribed in 58.3% of the patients and characterized the cervical myelopathy. Treatment relied on analgesics in all patients, NSAIDs were prescribed in 83.3%, colchicine in 66.7% and general corticosteroids in 25% of the cases. Spinal immobilization was recommended in 25% of the cases for a short period. Spinal CPPD was idiopathic in 75%, secondary to hyperparathyroidism in 8.3% of the cases and familial cases were detected in 16.7% of the patients.

Conclusion: Axial CPPD is rare and is an under-recognized entity that should be considered in elderly patients with neck or back pain. It can involve the discs or ligaments. The crown dens syndrome is quite suggestive of the diagnosis. If spinal CPDD is suspected, colchicine therapy could be a good therapeutic test and would avoid unnecessary further tests.

References:

- [1] Ferrone C, Andracco R, Cimmino MA. Calcium pyrophosphate deposition disease: clinical manifestations. *Reumatismo*. 19 janv 2012;63(4):246-52.
- [2] Feydy A, Lioté F, Carlier R, Chevrot A, Drapé J-L. Cervical spine and crystal-associated diseases: imaging findings. *Eur Radiol*. févr 2006;16(2):459-68.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.4762

AB0930

URATE INDUCED PRIMING OF HUMAN MONOCYTES IS MEDIATED VIA THE TGF- β PATHWAY

V. Klück¹, L. Mies¹, R. Bakker², T. Crisan³, L. Joosten^{1,3}. ¹Radboudumc, Internal Medicine, Nijmegen, Netherlands; ²Radboudumc, Rheumatology, Nijmegen, Netherlands; ³Iuliu Hațieganu University of Medicine and Pharmacy, Medical Genetics, Cluj Napoca, Romania

Background: Hyperuricemia, elevated serum urate levels, is the main risk factor for gout, but is also associated with higher incidence of comorbidities such as cardiovascular disease, type 2 diabetes, metabolic syndrome and chronic kidney disease[1]. Crisan *et al.* showed that urate leads to increased production of interleukin (IL)-1 β , a pro-inflammatory cytokine, and downregulation of IL-1 receptor antagonist (IL-1Ra), the natural inhibitor of IL-1, in human monocytes[2]. This imbalance between IL-1 β and IL-1Ra is mediated by epigenetic reprogramming of innate immune cells[2]. RNA sequencing in urate-treated monocytes demonstrated that the TGF- β signalling pathway was differentially expressed[3].

Objectives: The objective of this study is to further explore the role of TGF- β in urate induced priming of human monocytes.

Methods: Human peripheral blood mononuclear cells (PBMCs) were isolated from healthy volunteers, adhered to a flat bottom plate, and treated for 24h with a dosing range of urate after which mRNA was isolated. For validation experiments, PBMCs from 9 gout patients and 7 healthy controls were isolated and adhered to a flat bottom plate for 4h after which cells were stored for RNA isolation. qPCR primers designed for TGF- β , TGF- β receptor I and II, MMP9, SMAD7 and ITGAV were used to assess expression levels of TGF- β pathway in these adherent monocytes. For priming experiments, adherent monocytes were primed for 24h with urate and/or recombinant TGF- β 1 (R&D systems) with or without a TGF- β receptor II antibody (R&D systems), cells were washed and restimulated with LPS for 24h. Cytokine levels in supernatant were determined by ELISA for IL-1 β , IL-6 and IL-1Ra.

Results: mRNA expression of TGF- β and its downstream targets were upregulated in urate treated monocytes and in gout patients compared to healthy controls. Moreover, urate levels significantly correlated to TGF- β in individuals with gout. Both urate and TGF- β priming increased the release of IL-1 β and IL-6 after LPS stimulation in human monocytes. We did not observe a synergistic effect between the two and therefore hypothesized that urate induced inflammation is mediated via TGF- β . Blocking the TGF- β receptor II partly reversed the urate induced phenotype: lowered IL-1 β and IL-6 production and restored levels of IL-1Ra. Further validation experiments are ongoing.

Conclusion: Urate induced priming of human monocytes is at least partly mediated via the TGF- β pathway. This study contributes to the understanding of the pathways involved in urate induced inflammatory status and might in the future provide a mechanistic explanation for the occurrence of some comorbidities in patients with gout. Additionally, as TGF- β is a major player in the pathogenesis of systemic sclerosis, this study might give a rationale for treatment of hyperuricemia in this population.

References:

- [1] Bardin, T. and P. Richette, *Impact of comorbidities on gout and hyperuricaemia: an update on prevalence and treatment options*. BMC Med, 2017. **15**(1): p. 123.
- [2] Crisan, T.O., *et al.*, *Soluble uric acid primes TLR-induced proinflammatory cytokine production by human primary cells via inhibition of IL-1Ra*. Ann Rheum Dis, 2016. **75**(4): p. 755-62.
- [3] Crisan, T.O., *et al.*, *Uric acid priming in human monocytes is driven by the AKT-PRAS40 autophagy pathway*. Proc Natl Acad Sci U S A, 2017. **114**(21): p. 5485-5490.

Disclosure of Interests: Viola Klück: None declared, Linda Mies: None declared, René Bakker: None declared, Tania Crisan: None declared, Leo Joosten Consultant of: SAB member of Olatec Therapeutics LLC

DOI: 10.1136/annrheumdis-2020-eular.5548

AB0931

THE QUALITY OF LIFE IN GOUT PATIENTS WITH ULCERATION OVER TOPHI

X. Liu^{1,2}, Z. Huang¹, Q. Huang¹, Z. Zhong^{1,2}, W. Zhao^{1,3}, T. Li^{1,2}. ¹Guangdong Second Provincial General Hospital, Department of Rheumatology and Immunology, Guangzhou; ²Southern Medical University, The Second School of Clinical Medicine, Guangzhou, China; ³University of South China, Hengyang, China

Background: The prevalence of gouty patients with ulcerations over tophi are increasing over time and it has been reported that gouty patients have significantly poor quality of life (QoL) compared to those healthy controls. [1, 2] there is no study on comparison of the QoL in patients with or without ulceration over tophi.

Objectives: To compare the QoL in gout patients with or without ulcerations over tophi.

Methods: A total of 79 inpatients with gout who were admitted to Guangdong Second Provincial General Hospital from January 2019 to January 2020 were included. Among them, ulcerations were identified in 28 patients. Short Form-36 Scales (SF-36) were chosen to assess patients. Differences between the groups were tested with Student's t test or Mann-Whitney U test for continuous variables, and Chi-square tests for categorical variables.

Results: Of 79 subjects, 74 were male, mean age was 54.23±14.3 years, disease duration was 10.94±7.06 years. Gouty patients had moderate- to high-quality levels in general health, vitality, social functioning, role emotional, and