Background: Calcium pyrophosphate dihydrate crystal-deposits (CPPD) is a common crystal disease affecting men and women equally. It is normally seen in peripheral arthritis: imaging findings. Eur Radiol. févr 2006;16(2):459–68.

**Disclosure of Interests:** Christiane 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

**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 but is more severe and exacerbates joint 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.

**Results:** A total of 79 inpatients with gout who were admitted to Guangdong 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:** 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-1β), 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-1β 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].

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**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 dose 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-β1 (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-1β, 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-1β 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 partially reversed the urate induced phenotype: lowered IL-1β 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:**

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