analogues, 32 (40%) were taking analgesics, although 65 (80%) suffered from pain (71% articular pain, 22% bone pain and 20% enthesitic pain). Of the 81 patients, 71 (88%) presented with structural damage including 44 (63%) with syndesmophytes or ossifications. The average number of syndesmophytes/ossifications was 4.1±2.6 per patient in this subgroup. Most of them were localised at the cervical spine (82%) and the lumbar spine (62%). In addition, these ossifications were fine in 59% (syndesmophytes-like) and coarse in 41%. The other localizations were the ischial region (40%), iliac crests (37%) and the cotyles (90%). Calcanei were visible on 52 radiographs and showed coarse ossifications for 22 of them (42%). 46 patients (65%) had already hip osteoarthritis and 26 (37%) had at least one osteophyte on the spine mainly on the thoracic spine (58%). We found complete ankylosis of sacro-iliac joints in 12 patients (17%).

Conclusions: This observational study shows that 88% of adult XLH patients have structural damages and at least one ossification on the spine and/or on peri-articular joint (specially on the cotyles). Of importance, these ossifications were as frequent and as severe in patients not treated with phosphate and vitamin D analogues than in untreated patients. Knowing that this disease can be misdiagnosed and eventually diagnosed only at the adult age, attention must be paid to ossification of the entheses, in particular of the common anterior vertebral ligament and Achilles tendons. The mechanisms of formation of these enthesopathies remain to be determined.

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SAT0366

SECULAR TRENDS IN THE INCIDENCE AND PREVALENCE OF GOUT IN DENMARK FROM 1995–2015: A NATIONWIDE REGISTER-BASED STUDY

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Background: Gout is perceived as the most prevalent inflammatory arthritis in the western world – but with great regional and ethnical variation. The incidence and prevalence is thought to be increasing but it's based on few population studies with limited calendar time periods.^[1] We took advantage of the unique Danish health registries to examine the incidence and prevalence of gout in a nationwide cohort covering the last two decades.

Objectives: To examine temporal trends in the incidence and prevalence of gout in the adult Danish population.

Methods: Using the nationwide Danish National Patient Registry (DNPR), we calculated the incident rate of hospitalised gout patients (per 1 000 000 person-years) within each 1 year period from 1995 to 2015, and prevalence of gout requiring hospitalisation in 2000 and 2015. Additionally, the age- and gender-specific incidence rates of gout from 1995 to 2015 were calculated. Incidence rates were standardised according to the NORDCAN standard population. Confidence intervals (CI) were calculated according to a Poisson distribution. Both incidence rates based on diagnosis from all specialties and rheumatologists, respectively, were calculated.

Results: In the 20 year study period we identified 45 685 newly diagnosed cases of gout. We found an increase in overall incidence, from 32.26/100,000 [95%CI 30.7 to 33.1] in 1995 to 57.5/100,000 [95%CI 55.6 to 59.5] in 2015 (p<0.001). The age and sex specific incidence rates increased progressively with increasing age among both sexes, most markedly in patients aged >60 years. Similar trends were observed for cases diagnosed at rheumatology departments. We likewise observed an increase in the prevalence of gout from 0.29% [95%CI 0.29 to 0.30] in 2000 to 0.68% [95%CI 0.68 to 0.69] in 2015.

Conclusions: We found a 1.8-fold increase in the incident rate of hospitalised gout patients in the period 1995–2015 in Denmark among both men and women. Similarly we found a 2.2-fold increase in the prevalence from 2000 to 2015. Further research is needed to elucidate the causes underlying the observed increase in the burden of gout in Denmark

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SAT0366

SERUM URIC ACID INCREASES AFTER ABROGATION OF SYSTEMIC INFLAMMATION BY TNF INHIBITION

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Background: In patients with gout, the serum uric acid (SUA) is usually lower during acute gouty attacks than during intercritical periods. It has been suggested that systemic inflammatory response can cause this phenomenon.

Objectives: We aimed to investigate whether SUA levels are associated with levels of pro-inflammatory cytokines in patients with systemic autoimmune rheumatic diseases: rheumatoid arthritis (RA), ankylosing spondylitis (AS), psoriatic arthritis (PsA) and juvenile idiopathic arthritis (JIA) and whether the initiation of a therapy with TNF inhibitors (TNFi) would change levels of SUA.

Methods: A cohort of 128 patients with clinically active chronic inflammatory rheumatic disease (44 with RA, 45 with AS, 23 with PsA, 16 with JIA) and CRP ≥ 10 mg/L was recruited in the Institute of Rheumatology, Prague. SUA, CRP, creatinine, MCP-1, IFN-α2, IFN-γ, IL-1β, IL-6, IL-8, IL-10, IL-12, IL-17a, IL-18, IL-23, IL-33 and TNF-α were measured in serum before and after 3 months of treatment with TNFi. We employed bead-based immunoassays to quantify the cytokines and retrieved demographic data, BMI and co-medications. We present the data as mean ± standard deviation or mean and 95% CI.

Results: SUA was significantly lower before initiation of TNFi (288.5 ± 78.7 μmol/l) than after 3 months of treatment (307.7 ± 81.7 μmol/l, p=0.0002). The level of CRP (38.9 ± 30.3 vs 5.0 ± 7.6 mg/L, p<0.0001), IL-6 (172.8 ± 339.0 vs 24.2 ± 32.5 pg/ml, p<0.0001) and MCP-1 (1062 ± 554.7 vs 959.2 ± 508.9 pg/ml, p=0.015) significantly decreased during treatment. When we grouped our patients according to sex and diagnosis, the SUA levels significantly increased in the male patients (311.4 ± 79.5 vs 343.9 ± 74.3 μmol/l, p<0.0001), patients with AS (298.4 ± 70.0 vs 326.4 ± 73.1 μmol/l, p=0.0009) and with PsA (302.6 ± 75.3 vs 334.6 ± 74.3 μmol/l, p=0.033), but not in patients with RA or JIA. There was no correlation between the magnitude of the change of SUA, CRP and pro-inflammatory cytokines or the reported use of NSAIDs. However, when we classified our cohort according to increase or decrease of SUA after initiation of TNFi, we observed statistically significant differences in the changes of IL-1β (1.5, –10.8 to 13.9, vs 29.4, 5.4 to 53.1 pg/ml, p=0.023), IL-23 (0.7, –40.0 to 41.3 vs 102.0, 5.5 to 198.6 pg/ml, p=0.023), IL-33 (–1.2, –26.7 to 24.3 vs 48.8, 3.8 to 93.9 pg/ml, p=0.039) and TNF-α (–0.8, –6.1 to 4.5 vs 10.5, –0.4 to 21.32 pg/ml, p=0.037) between these two groups.

Conclusions: We have shown that abrogation of systemic inflammation by TNFis results in an increase in the levels of SUA within 3 months, especially in male patients with AS or PsA. Our results support the hypothesis that systemic inflammation is associated with a decrease in SUA concentrations. The mechanism remains elusive. However, IL-1β, IL-23, IL-33 and TNF-α are candidate players. The difference between the male and female patients argue for a role of the sex hormones. We cannot exclude the possibility that an underreported differential in the use of NSAIDs, known for their uricosuric effect, may have partly biased our observations.

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