OBJECTIVES: To assess early signs of CKD risk development in patients with gout by SENS-CRS technology.

METHODS: We enrolled 24 patients with gout. 95% were men (average age 54±9 years). Duration of gout was 8 [4; 11] years. All patients had chronic tophaceous gout, 30% of patients had tophi. SENS-CRS was carried out on a 2-detector γ-camera with simultaneous recording in 2 projections. In studies of the urinary system domestic radiopharmaceutical (RP) 99mTc-technetor with glomerulo- and 10-15% tubulotrop properties was used. The CRS study consisted of: 1) a basic 21-min functional study; 2) a delayed 21-min study (without the injection of RP) with a preliminary diuresis forcing to identify persistent urodynamic dysfunction.

RESULTS: 16 patients with gout with serum creatinine level of more than 125 μmol/l had established diagnosis of chronic pyelonephritis, glomerulonephritis, urolithiasis, etc. and were allocated into a separate subgroup. In the comparative analysis of these 16 patients and other 50 patients with gout there was reliable distinction only for one indicator D (%), the rate of RP removal from a kidney parenchyma (p < 0.05). There was no significant distinctions for glucerolar filtration rate by Reben, the nuclear indicator (Gmn) of renal parenchyma concentration function, and other indicators. Renocortical parameter D (%) is one of the earliest highly sensitive markers of intrarenal developments of stagnation at development of serious morphofunctional violations in a parenchyma. In the subgroup of 16 patients the indicator accounted on average D = 48% ± 7% (range 38-55%); in subgroup of 50 patients D = 63% ± 10% (47-89%).

According to the CKD-EPI formula and SENS-CRS technology in the subgroup of 16 patients CKD stage II-III was evaluated. In addition to the static marker of RP removal from a parenchyma (D) when comparing the 2 phases of CRS there was observed a steady sign of stagnation in a parenchyma and groups of upper and/or lower cups: ID = Gmin/GC < 1. Based on the water test which accelerates diuresis there was a sufficient regulation of urostasis in the renal pelvis revealed at the basic test (IP = Gpelv/GP > 1,0). This indicated that the functional reserve of the outflow regulation from the kidneys was preserved at these patients.

CONCLUSION: This functional diagnostics allows one-time control of hemodynamics and concentration function of the parenchyma of each kidney, quantitative and qualitative signs of urodynamic delays in the intra- and post-renal urinary tract. Differentiated analysis SENS-CRS contributes to the timely therapeutic correction as well as referral for consultation to specialists (urologists, nephrologists, gynecologists, etc.).

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Oxidative stress marker allantoin is not associated with the change of serum uric acid level in patients with systemic rheumatic diseases after abrogation of systemic inflammation by TNF inhibition

Lenka Hasikova1,2, Marketa Pavlikova3, Petr Kozik3, Kveta Kalikova3, Blanka Stiburkova1,2, Jakub Zavada1,2, Institute of Rheumatology, Prague, Czech Republic.

Department of Rheumatology, First Faculty of Medicine, Charles University, Prague, Czech Republic. 2Department of Probability and Mathematical Statistics, Faculty of Mathematics and Physics, Charles University, Prague, Czech Republic. 3Department of Analytical Chemistry, Faculty of Science, Charles University, Prague, Czech Republic.

Department of Physical and Macromolecular Chemistry, Faculty of Science, Charles University, Prague, Czech Republic. 4Department of Pediatrics and Adolescent Medicine, First Faculty of Medicine, Charles University and General University Hospital in Prague, Prague, Czech Republic.

Background: In patients with gout, the serum uric acid (SUA) is usually lower during acute gouty attacks than during intercritical periods. In a previous study, we have shown that abrogation of systemic inflammation by TNF inhibitors (TNFi) results in an increase in the levels of SUA in patients with systemic rheumatic diseases. We have not found any correlation between the magnitude of change of SUA and CRP or pro-inflammatory cytokines (MCP-1, IFN-x, IFN-y, IL-10, IL-6, IL-12, IL-17a, IL-18, IL-23, IL-33, TNF-α).1 Another possible mechanism for the lowering of SUA during inflammation may be consumption of circulating SUA in free radical reactions generated during systemic inflammation. Allantoin has been validated as a stable biomarker of oxidative stress in humans.2

OBJECTIVES: We aimed to investigate whether the magnitude of change of SUA after starting therapy with TNFi is associated with the change of oxidative stress marker allantoin in patients with systemic autoimmune rheumatic diseases: rheumatoid arthritis (RA), ankylosing spondylitis (AS), psoriatic arthritis (PsA) and juvenile idiopathic arthritis (JIA).

METHODS: A cohort of 94 patients with clinically active chronic inflammatory rheumatic diseases (31 with RA, 33 with AS, 18 with PsA, 12 with JIA) and CRP > 10 mg/L was recruited in the Institute of Rheumatology, Prague. SUA, CRP and allantoin were measured before and after 3 months of treatment with TNFi. Assessment of allantoin in plasma samples was carried out on the Agilent Infinity 1290 system coupled with a Triple Quad 6460 tandem mass spectrometer. For the statistical analysis the ratio between the values at the baseline and values after 3 months of therapy with TNFi were used. We retrieved demographic data and disease characteristics.

RESULTS: The levels of SUA have significantly increased after 3 months of treatment with TNFi (270.5 [78.8] vs. 303.0 [101.5] μmol/l, p < 0.0001), while CRP (29.4 [27.7] vs. 2.0 [3.8] mg/l, p < 0.0001) has decreased. There was no significant change of allantoin before and after 3 months of treatment with TNFi. In the linear regression model, CRP or allantoin had no effect on the magnitude of change of SUA.

Conclusion: The abrogation of systemic inflammation by TNFi results in an increase in the levels of SUA in patients with systemic rheumatic diseases, but the mechanism remains elusive. We have not observed any correlation between the magnitude of change of SUA and CRP or oxidative stress marker allantoin.

REFERENCES


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Endothelial dysfunction in patients with gout. Relationship between cardiovascular risk factors

Ekaterina Il’inykh, Maxim Eliseev, Alexander Volkov. V.A. Nasonova Research Institute of Rheumatology, Moscow, Russian Federation.

Background: Patients with gout have a high risk of developing cardiovascular disease based on atherosclerosis. An early marker, and at the same time a risk factor of cardiovascular disease, is a violation of the vasoregulating endothelial activity.

Objectives: To identify the relationship between cardiovascular risk factors and endothelial dysfunction (ED) in patients with gout.

Methods: The study included 80 pts with gout. The criteria for inclusion were 1.Male 2.Age >55 years 3.Intercritical period of arthritis 4.Absence of clinical signs of coronary artery disease 5.Absence of drug therapy. Vasoregulating endothelial activity was evaluated in all pts, 52 of them determined the carotid intima-media thickness (C-IMT). Flow-mediated (endothelium-dependent) dilatations (FMD) and nitroglycerin-induced (endothelium - independent) dilatation (EID) were assessed by highresolution ultrasonography (D. Celermaier). A non-invasive ultrasound technique was used to measure C-IMT. All patients were diagnosed with serum total cholesterol (CHOL), subtypes (LDL-C, HDL-C), glucose (GLU), uric acid (SUA), hsCRP. Cardiovascular risk (CVR) was calculated. Statistical analysis was conducted using the applied programs package of descriptive statistics STATISTICA 12.0 (StatSoft,Inc. USA).

Results: It was found that 41 (51.25%) pts had FMD less than 8%. A correlation was found between FMD and CVR (r = -0.28, p < 0.05), age (r= -0.37, p<0.001), Body mass index (BMI)
ANALYSIS OF LIFESTYLE AND CLINICAL FEATURES OF THE PATIENTS WITH GOUT IN MEIZHOU, GUANGDONG, CHINA

Yutong Jiang1, Yiquan Wen2, Zhongyu Liu1, Qiyun Chen2, Yefei Huang1, Yuedong Hospital, Meizhou, China

Background: Gout is one of the most common metabolic diseases caused by purine metabolic disorder, leading to joint destruction and kidney impairment. With the improvement of living conditions, the incidence of gout is increasing year by year, especially in the developed coastal areas of China. Previous studies found that the management of gout was unsatisfactory [1]. Besides, the development of rheumatology in Meizhou, Guangdong province is extremely slow. There are only ten rheumatologists, and the general public has a limited understanding of gout.

Objectives: Our aim was to explore the lifestyle, clinical features and risk factors of recurrent gout attacks in patients with gout in Meizhou, Guangdong province.

Methods: Demographic data, lifestyle and clinical data of 188 patients with gout in Meizhou were collected. Demographic variables included age, gender, marital status, education, BMI, smoking status, drinking status, working style, exercise habit, late sleeping habit. Gender, marital status, education, BMI, smoking status, drinking status, working style, exercise habit, late sleeping habit were recorded based on self-report. Exercise habits included not exercising, exercising at least 1 hour a week, and exercising at least 1 hour a day, exercising at least 1 hour a month. Working style included long time of sitting, standing, walking, etc. Exercise habits include not exercising, exercising at least 1 hour a day, exercising at least 1 hour a week, and exercising at least 1 hour a month. Clinical characteristics include first time and cause of gout attack, pain score, duration, alleviating methods, treatment, patients’ self-reported gout episodes (below 5 times, 5-10 times, 11-20 times, more than 20 times), tophi, family history and comorbidities. Multiple regressions analysis was used to analyze the factors of the numbers of gout attacks.

Results: Of the 188 cases, 171 (91.0%) were males and 17 (9.0%) were females. The average age was 55±16 years. 94 (50%) of the patients were drinking alcohol, and 58 (30.9%) were fond of seafood and other high-purine food. 149 (79.3%) patients had less than 1000ml of water per day, and the vast majority of patients had no exercise habit (136 (72.3%) patients had no exercise habit). The interval between the first gout attack and the first uric acid increasing was -0.4 (SD=2.5) years. The first gout attack occurred in the CVR. C-IMT correlates with an increase in CVR and with parameters reflecting the severity of gout.

REFERENCE


Disclosure of Interests: None declared


AB0875 GOUT, NOT JUST A DISEASE OF THE FOOT.
LITERATURE REVIEW OF SYSTEMIC DEPOSITION OF URATE

Ada Kumar1, Puja Khanna2, 3, Horizon Pharma, Lake Forest, United States of America; 4University of Michigan, Ann Arbor, United States of America

Background: Gout is the most common adult inflammatory arthropathy in the US. Although tophi in the extremities is a known source of the inflammatory cascade, urate deposition in organs throughout the body is not as well recognized. Patients with gout often have associated co-morbidities including renal disease, cardiovascular disease and metabolic syndrome, however, a causal role has not been established. Direct urate deposition in these organ systems may be of interest to link the causality of these systemic disorders.

Objectives: Perform a literature review including clinical exam, autopsy, pathology, and radiology imaging results demonstrating systemic deposition of urate exclusive of the extremities.

Methods: PUBMED from 1920 to 2018 was searched to identify reports of non-extremity urate deposition. Key words included: extra-articular gout, systemic deposition of urate, ocular gout, gout nephropathy, renal tophi, gouty heart, cardiac valves and urate, urate deposition in the arteries, prostate and urate, autopsy findings in gout, cutaneous urate deposits, gouty panniculitis, auricular gout, breast and urate, gastrointestinal gout, pancreas and tophus, laryngeal tophus, and spinal gout. The reference lists from these publications were also used to identify additional articles.

The literature was reviewed for organ system involvement and documented based on sites of urate deposition within an organ system.

Results: There were 249 case reports documenting non-extremity urate deposition confirmed by autopsy, biopsy, surgery, clinical exam and/or radiology imaging. Urate deposition was reported in multiple organ systems (Table 1) including the spine, integumentary, ocular, renal, cardiovascular, gastrointestinal, larynx, breast, middle ear, pancreas, nasal, prostate gland, liver, pulmonary, penis, naibled, and pelvis.

Conclusion: Numerous case reports documented systemic deposition of urate based on autopsy, pathology, imaging and clinical exam. Urate crystal deposition with the formation of tophi and micro-tophi involve multiple organ systems including cardiovascular, renal, spine, integumentary, prostate, bowel, pancreas, eyes, pelvic, breast, lungs, middle ear, larynx, liver, penis, naibled, and nose. Given the strong association of gout with various comorbidities, this demonstrates a need for further studies to determine the clinical significance of systemic urate deposition with respect to ongoing subclinical inflammation and potential end-organ damage.

REFERENCES


Abstract AB0875 Table 1

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