of gout (p=0.003); they were more likely to have subcutaneous tophi than pts with CKD C2 (p=0.007). The frequency of diagnosing DM, hypertension, as well as the sUA level in the groups did not differ. The levels of sUA, GFR in patients with pre-therapy CKD C2 stratified by level of GFR at baseline and after 26 weeks of febuxostat therapy, are given (Table 1). The target sUA level was achieved in 33 (87%) patients with DM (the probability did not depend on the initial GFR value). Achievement of target sUA level was registered in 84% of general sample: in 83% of pts with CKD C0-1, 89% - CKD C2, 82% - CKD C3, 81% of pts with CKD C4. Mean GFR values increased in all groups, but significant differences registered in patients with CKD C0-1 (p=0.002) only.

Conclusion: The ability to achieve the target sUA level while taking febuxostat in patients with gout does not depend on renal function, exceeding 80% in patients with CKD C4. The drug is well tolerated regardless of renal function.

Disclosure of Interests: Maxim Eliseev Speakers bureau: Berlin Chemie Menarini Group, Sobi, EGIS, CSC, Mosfarma, Alrik Group, Olga Sheliabina Speakers bureau: Sobi; Berlin Chemie Menarini Group, Maria Chikina; None declared. Eugenia Markelova: None declared, Irina Kirillova: None declared.


AB1047  EFFECT OF THERAPY ON SUBCLINICAL CAROTID ATHEROSCLEROSIS IN PATIENTS WITH CALCIUM PYROPHOSPHATE DEPOSITION DISEASE (PILOT STUDY)

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Background: Supposedly, anti-inflammatory therapy in calcium pyrophosphate deposition disease (CPPD) pts may reduce the progression of atherosclerosis.

Objectives: Assessment of the dynamics of atherosclerosis development based on changes of carotid artery intima-media thickness (caIMT) in pts with CPPD receiving anti-inflammatory therapy (colchicine, methotrexate, hydroxychloroquine).

Methods: The prospective study included 26 pts aged ≥18 years with a crystal-verified diagnosis of CPPD and osteoarthritis (OA). Exclusion criteria were age >65 years, presence of cardiovascular disease, carotid artery (CA) atherosclerosis according to ultrasound results, high or very high SCORE (Systematic Coronary Risk Evaluation) index. The examination included anthropometric parameters, information about the affected joints and the time of onset of symptoms. Laboratory tests included determination of the following in blood serum: hs-CRP, lipid levels; SCORE index was calculated for all pts; CA Doppler ultrasonography (DU) was performed using the Esatec MyLab Twice ultrasound system (Italy). The manifestation of subclinical atherosclerosis was diagnosed in case of caIMT increase >0.9mm. The criteria for the presence of an atherosclerotic plaque in the CA was a local caIMT increase of more than 50% compared to the surrounding areas or an caIMT increase >1.3mm. CAIMT was measured at the first visit, then, pts with CPPD were administered methotrexate 15mg/week or hydroxychloroquine 200mg/day or colchicine 0.5mg twice daily. Pts could take NSAIDs to relieve pain. After 26-28 weeks, a second examination was carried out. Statistica 12.0 package was used for statistical data processing.

Results: The baseline values of caIMT in pts with CPPD and OA did not differ. 22 pts with CPPD and 19 with OA were examined in dynamics. Baseline caIMT ≥0.9mm was detected in 11 of 22 (50%) pts with CPPD and in 8 of 19 (42%) pts with OA (p=0.39). CAIMT ≥0.9mm was associated with an increase in hs-CRP levels ≥0.2mg/l in 8 pts with CPPD, 9 of 19 (47%) pts with OA demonstrated an increase in caIMT averages, the values in other pts remained unchanged. By the end of the study, 14 of 22 (64%) pts with CPPD had a decrease in the average values of caIMT, in 2 (9%) pts - an increase, in 6 pts the average values of caIMT did not change. 7 of 11 pts with CPPD showed normalization of caIMT, 5 of these pts had a decrease in serum hs-CRP ≥2mg/l. A decrease in the number of pts with CPPD and caIMT ≥0.9mm from 11 (42%) to 4 (18%) pts was found. At the same time, in 8 pts with CPPD the serum hs-CRP level significantly decreased: baseline 6.02 [0.69; 6.4] mg/l vs at the end of study period 1.71 [0.78; 2.25] mg/l, p=0.043. Pts with OA demonstrated the constant levels >0.2

Conclusion: Therapy with colchicine, methotrexate and hydroxychloroquine in pts with CPPD leads to regression of early signs of atherosclerosis. This result can be achieved by suppressing chronic inflammation.

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AB1048  ESTIMATED GLOMERULAR FILTRATION RATE CHANGES IN UNCONTROLLED GOUT PATIENTS CO-TREATED WITH PEGLOTICASE AND METHOTREXATE: A RETROSPECTIVE CASE SERIES

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Background: Gout patients are at increased risk for developing chronic kidney disease (CKD) and hyperuricemia is an independent risk factor for CKD worsening, particularly in women. As a result, renal function is of concern in uncontrolled gout patients. Pegloticase, a recombinant PEGlYlated uricase, can rapidly decrease serum uric acid levels (sUA) in uncontrolled gout patients, but with pegloticase monotherapy <50% have sustainedurate-lowering during Month 6 of treatment. Pegloticase treatment response rate is markedly higher when immunomodulating therapies such as methotrexate (MTX) are co-administered, but MTX use can be limited by renal impairment. Clinical trials excluded CKD patients, but real-world published cases of immumomodulation-pegloticase co-therapy have included patients with pre-therapy eGFR <60ml/min/1.73m².

Objectives: This study examined pooled case data from prior studies, focusing on renal function changes during MTX-pegloticase co-treatment in patients with and without pre-therapy CKD.

Methods: This retrospective study examined deidentified case data collected for prior retrospective studies. All patients who underwent MTX-pegloticase co-therapy were included and categorized as CKD (baseline eGFR <60ml/min/1.73m²) or non-CKD (baseline eGFR ≥60ml/min/1.73m²). sUA, renal function, blood cell counts, and liver function were closely monitored during therapy. Patient characteristics, pegloticase treatment parameters, proportion of treatment responders (≥12 infusions received and sUA <6mg/dL at infusion 12 [ongoing patients with <12 infusions excluded]), renal function changes (eGFR, CKD stage), and adverse events were examined.

Results: 15 uncontrolled gout patients with CKD (9 stage 3a, 4 stage 3b, 2 stage 4; pre-therapy mean ±SD) eGFR: 43.2±11.3ml/min/1.73m², sUA: 8.5±2.2mg/dL and 27 without CKD (pre-therapy eGFR: 82.9±19.0ml/min/1.73m²; sUA: 9.5±1.7mg/dL) were included. Patient characteristics and comorbidity profiles were similar, but CKD patients were older (72.0±9.9 vs. 52.3±14.3 yrs) and more often female (33% vs. 7%). On average, MTX was initiated ~4 wks before pegloticase treatment. patient characteristics, pegloticase treatment parameters, proportion of treatment responders (≥12 infusions received and sUA <6mg/dL at infusion 12 [ongoing patients with <12 infusions excluded]), renal function changes (eGFR, CKD stage), and adverse events were examined.