patients with at least 1 gout ICD 10/ICD 9 diagnosis code (N=539,802) and 90 days of continuous urate-lowering therapy within 1 year of diagnosis. Two cohorts of patients were categorized according to their sUA levels (≥ 1 test) after at least 90 days of gout therapy: sUA<6.0 mg/dL (controlled) and sUA ≥ 8 mg/dL (uncontrolled).

Results: The controlled gout group (sUA<6 mg/dL) included 5,473 patients and the uncontrolled gout group (sUA ≥ 8 mg/dL) had 1,358 patients. The two groups were comparable in terms of demographic features. Chronic kidney disease (CKD) was a common comorbidity overall in this gout population with higher prevalence in the uncontrolled gout cohort (49.4% of uncontrolled vs. 32.4% of controlled population; OR 2.04; 95% CI of 1.808 to 2.301, $p<0.001$). The most frequent hospitalization codes were similar between the uncontrolled and controlled patients with the exception of congestive heart and acute kidney failure. 20% of uncontrolled patients were hospitalized for congestive heart failure vs. 7% in controlled (OR 3.16, 95% CI: 2.674 to 3.739, $p<0.001$), and 20% of uncontrolled patients were hospitalized for acute kidney failure vs. 8% in controlled (OR 2.95, 95% CI: 2.497 to 3.480, $p<0.001$).

Conclusion: Gout patients frequently suffer from cardiovascular and renal diseases. This large retrospective analysis suggests that when divided based on uric acid levels attained, uncontrolled gout patients are more likely to suffer from CKD and also more likely to be hospitalized for acute renal failure than controlled gout patients. Whether hyperuricemia in uncontrolled gout causes the development of specific cardiovascular and renal comorbidities, or if specific cardiovascular and renal diseases lead to hyperuricemia and uncontrolled gout is not fully established.

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SAT0430

IMPACT OF GOUT ON ALL-CAUSE MORTALITY AMONG MEDICARE BENEFICIARIES WITH A HISTORY OF KIDNEY TRANSPLANTATION: A RETROSPECTIVE COHORT STUDY

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Background: Gout is a frequent comorbidity among kidney transplant recipients. A recent analysis estimated that 13% of kidney transplant recipients had an active diagnosis of gout. The clinical impact of comorbid gout in this population is not well understood, including if gout as a co-morbidity associates with higher mortality rates among kidney transplant recipients.

Objectives: This retrospective patient claims analysis was performed to determine whether an association between gout and mortality exists in the prevalent kidney transplant population.

Methods: A retrospective study using administrative claims from the Medicare fee-for-service (FFS) Limited Data Set (LDS) was conducted. Multivariable Cox proportional hazards regression assessed the relationship between gout and all-cause mortality. Given the ambiguity of the causal relationship between gout and comorbidities that make up the Charlson

Comorbidity Index (CCI), three analyses were conducted assuming: 1) gout and certain comorbid conditions are associated by way of a common pathogenetic root, 2) gout is a precursor for developing these comorbid conditions, and 3) these conditions modify the effect of gout on mortality.

Results: Gout was associated with higher unadjusted risk of all-cause mortality (hazard ratio, HR: 1.44, 95% CI: 1.27-1.63). After adjusting for baseline demographics and time from transplantation, the HR risk with gout was attenuated but still statistically significant (HR: 1.16, 95% CI: 1.02-1.32). Further adjustment for baseline CCI found gout was not a significant risk factor (HR: 1.03, 95% CI: 0.90-1.17). Stratified models show gout among baseline CCI=0 score was associated with a 3.5-fold increased risk of all-cause mortality (HR: 3.48, 95% CI: 1.27-9.57).

Conclusion: The presence of gout was not an independent predictor of all-cause mortality among Medicare beneficiaries with a history of kidney transplantation. That gout in a subset of beneficiaries without baseline comorbidities was a predictor may suggest that gout serves as an early indicator of declining health in the larger prevalent kidney transplant population. Further research is needed to understand the relationship of gout and mortality in the kidney transplant population.

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SAT0431

RED GINSENG EXTRACTS SUPPRESS MONOSODIUM URATE CRYSTAL INDUCED NLRP3 ACTIVATION

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Background: It is well known that red ginseng extracts (RGE) has anti-inflammatory properties.

Objectives: We aimed to investigate if RGE could suppress monosodium urate crystal (MSU) - induced NLRP3 activation and thus could be a potential therapeutic for gout.

Methods: Acute air-pouch model was used to investigate *in vivo* effect on acute gouty inflammation of RGE in mice. Human monocyte cell line -THP-1- was stimulated with MSU in the presence of RGE and expression of NLRP3, ASC, caspase-1, IL-1 β was measured by PCR, immunoblotting and ELISA. Twenty four gout patients in intercritical period were randomized either to red ginseng tablet or placebo receiving group in a double-blind manner. After 3months of taking red ginseng tablets (RGT)/placebo, expression of NLRP3 and inflammatory cytokines of peripheral blood mononuclear cell was addressed by PCR.

Results: RGE sufficiently inhibited acute gouty inflammation in air-pouch mouse model, represented by reduced number of white blood cells in air pouch lavage fluid. *In vitro*, RGE dose-dependently suppressed MSU-induced IL-1 β production of THP-1 cells. RGE did not affect NLRP3 or pro IL-1 β expression. However, ASC oligomerization was inhibited and suppressed NLRP3 inflammasome assembly. Patients taking RGT for 3 months showed significantly reduced NLRP3 expression compared to baseline, which was not observed in the control group.

Conclusion: RGE successfully suppressed MSU-induced acute inflammation in mouse model. Also, RGT reduced NLRP3 expression in intercritical gout patients. These results suggest that red ginseng can be implicated as a therapeutic agent for gout.

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SAT0432 GOUTY ARTHRITIS AND DRUG-INDUCED LIVER INJURY AFTER THE TREATMENT WITH NSAIDS

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Background: In order to relieve the pain during acute gout attacks, most patients take high doses of nonsteroidal anti-inflammatory drugs (NSAIDs), which can provoke the hepatotoxicity, or DILI (drug-induced liver injury).

Objectives: To define the timing of DILI formation after the treatment with NSAIDs and its severity in patients with gouty arthritis (GA).

Methods: 738 patients with GA from our database who meet the criteria of the ACR (1977) were included into the study. NSAIDs-induced liver damage is known to be mainly hepatocellular. The dynamic ALT concentration in blood before the start of NSAIDs and during the treatment has been assessed. Hepatocellular toxicity was determined according to the DILI classification criteria based on the blood ALT level increased > 2 times to the upper limit of norm (42 U/l for men and 35 U/l for women). Inclusion criteria for the study group (n = 88) were: normal ALT before the treatment with NSAIDs, its increase during the treatment and return to the norm after the treatment.

Exclusion criterion was chronic hepatitis of any etiology. The comparison group (n = 650) consisted of patients with normal blood ALT level throughout the treatment.

Results: Among 738 patients with GA, 11.9% (n = 88) developed the hepatotoxicity following the NSAIDs therapy. No significant differences in age (54 (44-59.5) and 57 (52-63) years; p > 0.05) and gender (men 93.3% and 87.6%; p > 0.5) between the study and comparison groups were found. In the study group, the duration of NSAIDs administration was 10 (6-14) days, which did not differ from that in the comparison group — 9.5 (7-12) days (p > 0.05).

In patients with DILI, the elevated ALT was observed as follows: 2-3 times in 74 patients (84.1%); 3 to 5 times in 10 (11.4%); more than 5 times in 4 patients (4.5%). So, minimal cytolysis was presented more frequently than more severe forms (p < 0.05).

In the subgroup with the ALT concentration 3-5 times (n = 10), 6 patients received Diclofenac in high doses, 2 patients were treated with Nimesulide, 1 patient with Etodolac and 1 with Dexamethasone during the observation period.

The elevation of ALT concentration 5 times was observed in 3 patients taking Diclofenac i/m and Nimesulide per os, simultaneously, and in 1 patient taking Diclofenac in high doses.

Patients with DILI were taking the following medications: Diclofenac 48.9% (n = 43); Nimesulide 18.2% (n = 16); Meloxicam 5.7% (n = 5),

the combination of NSAIDs 20.5% (n = 18) without statistical differences (p > 0.05).

The number of patients who abused alcohol in DILI and control groups did not differ significantly — 53.4% and 48.2%, respectively (p > 0.05).

Conclusion: In patients with GA, the hepatocellular DILI was observed in 11.9% cases after the treatment with NSAIDs during 10 days (from 6 to 14 days). Among patients with DILI, 84.1% (n = 74) had NSAIDs-induced hepatitis with minimal cytolysis. Mild cytolysis was seen in 10 (11.4%), moderate in 4 patients (4.5%). In our study, no severe or fatal DILI has been noted. No significant differences for particular drugs in the hepatotoxicity incidence have been found (p > 0.05).

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SAT0433 AN ANALYSIS OF PRESCRIPTION RECORDS OF GOUT PATIENTS IN EUROPE: EVIDENCE OF SUBOPTIMAL MANAGEMENT AND CLINICAL INERTIA

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Background: Urate-lowering therapy (ULT) should be prescribed to people with recurrent gout flares and tophi, and offered to people with first-onset gout. Despite effective drugs, gout flares are common. Reasons for this include lack of prescription of ULT, under-dosing of ULT by physicians (clinical inertia), and poor adherence to ULT.

Objectives: To ascertain the quality of gout care delivered by general practitioners (GPs), prescribing patterns for ULT were analysed in 4 western European countries.

Methods: Data for this retrospective study were obtained from IQVIA's Real-World Data Longitudinal Prescription databases of GPs from France, Italy, Spain and United Kingdom (UK). The databases contain anonymised patient prescription records, including demographics, dispensing details (pharmacy, prescription date), and medication (name, dose, therapy duration). Data for patients with gout with or w/o ULT were analysed from July 2015 to June 2016.

Results: Crude prevalence of gout was 0.7% (UK) to 1.1% (France, Italy, and Spain) [Table 1]. Only about half (France, Italy, UK) to 2/3 (Spain) of diagnosed patients were on ULT. Between 19.9% (France) and 56.4% (Spain) of people on ULT had serum urate (sUA) measurements recorded within the year. Only 26.6% (Italy) to 45.6% (France) of patients with a recorded sUA level were at target (<6.0 mg/dL). The most common 1st-line treatment was allopurinol (ALLO), almost always at a dose ≤300 mg/d. Febuxostat (FBX) was prescribed as a 1st-line alternative in France and Italy. Switch to 2nd-line ULT, such as FBX, was uncommon, especially in Spain and UK. Uricosurics in monotherapy were not used. Average time on ULT ranged from 57.3% (Italy) to 72.6% (France) of the assessment year. At least one comorbidity (CM) was present in >78% of patients, the most common being hypertension, dyslipidemia, diabetes, chronic kidney disease, and obesity.

Conclusion: In the study period, management of patients with gout in 4 EU countries was suboptimal. Nearly half of diagnosed patients were not prescribed ULT. sUA levels were not being monitored regularly and mean sUA levels were above target. ALLO as the most common 1st-line ULT was generally prescribed at sub-therapeutic doses. Initiation of 2nd-line therapy was infrequent indicating a status quo and/or other reasons and medication adherence was poor indicating low patient self-management