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.

REFERENCES

- [1] Neogi T. NEJM. 2011; 364:443-52
- [2] Khanna D., FitzGerald J.D., et al. *Arthritis Care Res.* 2012; 64(10):1431-1446
- [3] Fels E., Sundry J.S., et al. *Curr Opin Rheum.* 2008; 20:198-202
- [4] Kuo C.F., Grainge M.J., et al. *Clin and Epidem Res.* 2014; 0:1-8
- [5] Khanna P., et al. *Health and Quality of Life Outcomes.* 2012; 10:117

Disclosure of Interests: Megan Francis-Sedlak Shareholder of: Horizon Pharma, Employee of: Horizon Pharma, Brian LaMoreaux Shareholder of: Horizon Pharma, Employee of: Horizon Pharma, Robert J Holt Shareholder of: Horizon Pharma, Employee of: Horizon Pharma
DOI: 10.1136/annrheumdis-2019-eular.2889

SAT0430

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

LI Justin¹, Marissa Suh¹, Mark Brigham¹, Jeff Kent², Brian LaMoreaux², Richard J. Johnson³, Brian Mandell⁴, Nandini Hadker¹, Herman Sanchez¹, Kevin Francis¹, Gavin Miyasato¹. ¹Trinity Partners, New York, United States of America; ²Horizon Pharma USA, Inc., Lake Forest, United States of America; ³University of Colorado, Denver, United States of America; ⁴Cleveland Clinic, Cleveland, United States of America

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.

REFERENCES

- [1] Abbott KC, et al. *Transplantation.* 2005; 80(10):1383-1391.
- [2] Hernández-Molina G, et al. *Transplantation.* 2008; 86(11):1543-1547.
- [3] Brigham MD, et al. *Arthritis Rheumatol.* 2018; 70 (suppl 10).

Disclosure of Interests: Justin Li Consultant for: Horizon Pharma, Marissa Suh Consultant for: Horizon Pharma, Mark Brigham Consultant for: Horizon Pharma, Jeff Kent Shareholder of: Horizon Pharma, Employee of: Horizon Pharma, Brian LaMoreaux Shareholder of: Horizon Pharma, Employee of: Horizon Pharma, Richard J. Johnson Consultant for: Horizon Pharma, Brian Mandell Consultant for: Horizon Pharma, Nandini Hadker Consultant for: Horizon Pharma, Herman Sanchez Consultant for: Horizon Pharma, Kevin Francis Consultant for: Horizon Pharma, Gavin Miyasato Consultant for: Horizon Pharma

DOI: 10.1136/annrheumdis-2019-eular.2897

SAT0431

RED GINSENG EXTRACTS SUPPRESS MONOSODIUM URATE CRYSTAL INDUCED NLRP3 ACTIVATION

Jennifer Lee, Moon Young Kim, Sung-Hwan Park. *Seoul St Mary's hospital, College of Medicine, The Catholic University of Korea, Division of Rheumatology, Department of Internal Medicine, Seoul, Korea, Rep. of (South Korea)*

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.