Methods: Patients with a crystal-proven diagnosis of CPPD and age- and sex-matched disease-controls were prospectively enrolled in this cross-sectional, monocentric, case-control study. All subjects underwent a bilateral US examination of 9 hyaline cartilages (HC), 6 fibrocartilages (FC), 5 tendons, 1 joint recess and 1 ligament as follows: shoulder (glenoid FC, humeral HC and acromioclavicular FC), elbow (humeral HC and triceps tendon), wrist (triangular FC, scapho-lunate ligament, volar recess of the radio-lunate joint), hand (HC of the metacarpophalangeal joints from 2nd to 5th finger), hip (acetabular FC and femoral HC), knee (femoral condyles' HC, meniscal FC, patellar and quadriceps tendons), ankle (talar HC, Achilles tendon and plantar fascia). US assessment was carried-out by a rheumatologist blinded to clinical data. CPP deposits were identified as presence/absence, according to the OMERACT definitions [1].

Results: Ninety-five patients were enrolled: 45 CPPD patients (age: 72±10.6 years, disease duration: 5.6±7.8 years, female/male ratio: 1.3) and 50 age- and sex-matched disease-controls (18 with rheumatoid arthritis, 13 with osteoarthritis, 10 with psoriatic arthritis and 9 with gout).

The FC of the medial and lateral meniscus were the most frequently involved targets of CPP deposits in cases (81.8% and 77.3% of patients, respectively), followed by the triangular FC of the wrist (68.2%), the HC of the femoral condyles (54.5%), the scapho-lunate ligament (52.3%) and the acetabular FC (50.0%). In all these anatomical targets, US findings indicative of CPP deposits were detected in a significantly higher percentage of cases than controls ($p<0.01$).

The US scanning protocols that showed the best balance between sensitivity and specificity, the most sensitive and the most specific were shown in Table 1.

Table 1. Diagnostic performances of different US scanning protocols

Anatomical targets	SE	SP	LH+	LH-
Knee meniscal FC and wrist triangular FC	0.86 (0.76-0.96)	0.86 (0.73-0.94)	6.35 (3.17-12.72)	0.13 (0.06-0.30)
Knee meniscal FC, wrist triangular FC and hip acetabular FC	0.93 (0.82-0.99)	0.82 (0.69-0.91)	5.19 (2.85-9.42)	0.08 (0.03-0.24)
Hip acetabular FC, knee femoral condyles' HC and	0.67 (0.51-0.80)	0.96 (0.86-0.99)	16.67 (4.22-65.82)	0.35 (0.23-0.53)

wrist scapho-lunate ligament

LH: likelihood ratio, SE: sensitivity, SP: specificity, US: ultrasonography.

In all US scanning protocols, each anatomical target was assessed bilaterally.

Figure 1 includes representative pictures showing CPP crystal deposits in different anatomical targets.

A: Wrist, longitudinal scan of the triangular FC complex.

B: Knee, longitudinal scan of the lateral meniscus.

C: Hip, longitudinal scan of the acetabular labrum FC,

D: Knee, longitudinal scan of the medial femoral condyle's HC.

Arrows: CPP crystal deposits at FC, arrowhead: CPP crystal deposits at HC.

Conclusion: Our results suggest that bilateral US assessment of knee, wrist and hip provided acceptable sensitivity and specificity for diagnosing CPPD.

REFERENCES:

- [1] Filippou G, et al. Identification of calcium pyrophosphate deposition disease (CPPD) by ultrasound: reliability of the OMERACT definitions in an extended set of joints-an international multiobserver study by the OMERACT Calcium Pyrophosphate Deposition Disease Ultrasound Subtask Force. *Ann Rheum Dis.* 2018;77:1194-9.

Disclosure of Interests: Edoardo Cipolletta: None declared, Jacopo Di Battista: None declared, Walter Grassi Speakers bureau: Walter Grassi has received speaking fees from AbbVie, Celgene, Grünenthal, Pfizer and Union Chimique Belge Pharma., Emilio Filippucci Speakers bureau: Emilio Filippucci. has received speaking fees from AbbVie, Bristol-Myers Squibb, Janssen-Cilag, Novartis, Pfizer, Roche and Union Chimique Belge Pharma.

DOI: 10.1136/annrheumdis-2021-eular.496

OP0206

ASSOCIATION BETWEEN FEMALE REPRODUCTIVE FACTORS AND GOUT: NATIONWIDE POPULATION-BASED COHORT STUDY OF 1 MILLION POSTMENOPAUSAL WOMEN

Y. Eun¹, I. Y. Kim², K. D. Han³, K. Lee³, S. Y. Kang¹, S. Lee¹, H. S. Cha¹, E. M. Koh¹, J. Lee¹, H. Kim⁴, ¹Samsung Medical Center, Sungkyunkwan University School of Medicine, Department of Medicine, Seoul, Korea, c of (South Korea); ²National Police Hospital, Department of Medicine, Seoul, Korea, Republic of (South Korea); ³Soongsil University, Department of Statistics and Actuarial Science, Seoul, Korea, Republic of (South Korea); ⁴Samsung Medical Center, Sungkyunkwan University School of Medicine, Department of Medical Humanities, Seoul, Korea, Republic of (South Korea)

Background: Previous studies have shown that the incidence of gout differs according to gender, and its risk factors also differ according to gender. However, little research has been done on the association between reproductive factors and gout.

Objectives: Our study attempted to determine whether there is an association between reproductive factors and the incidence of gout in a large nationwide population-based cohort of postmenopausal women.

Methods: Postmenopausal women aged 40-69 who participated in national health screenings in 2009 were included in the study. Subjects who had been diagnosed with gout prior to medical examination were excluded from the study, and a total of 1,076,378 women were included in the study. Outcome was the occurrence of gout which was defined as a case of two outpatient visit or one hospitalization for gout using the ICD-10 code of gout (M10) in the claim database. The Cox proportional hazard model was used for the analysis, and stratified analysis according to the body mass index (BMI) and chronic kidney disease (CKD) was performed.

Results: Mean follow-up duration was 8.1 years, and incident cases of gout were 64,052. Later menarche (adjusted HR 1.10, 95% CI 1.02-1.19 in >16 years, compared with ≤12 years), earlier menopause (adjusted HR 1.12 in <40 years, 1.06 in 40-45 years, 1.03 in 45-50 years, compared with 50-55 years), and shorter reproductive span (adjusted HR 1.10 in <30 years, 1.06 in 30-35 years, compared with ≥40 years) were associated with a high risk of gout. No association between parity and gout incidence was observed. Use of oral contraceptives (OC; adjusted HR 1.03 in <1 year, 1.05 ≥1 year, compared with non-user) and hormone replacement therapy (HRT; adjusted HR 1.16 in <2 years, 1.16 in 2-5 years, 1.18 in ≥5 years, compared with non-user) were associated with an increased risk of gout. The association between reproductive factors and gout remained in the trend without statistical significance in the low BMI group, but not in the high BMI group. The effects of use OC and HRT on gout were not significant in the CKD group.

Conclusion: Shorter exposure to endogenous estrogen was associated with a high risk of gout. Conversely, exposure to exogenous estrogen such as OC and HRT was associated with an increase in gout risk. This association was not significant in subjects with high BMI or CKD.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.4169

OP0207

ASSOCIATION BETWEEN DEMENTIA AND GOUT: A NATIONWIDE POPULATION-BASED STUDY

J. Kim¹, H. M. P. Park², D. H. Yim³, I. A. Choi⁴, S. Y. Eom⁵. ¹Chungbuk National University Hospital, Internal Medicine, Division of Rheumatology, Cheongju-si, Chungcheongbuk-do, Korea, Republic of (South Korea); ²Chungbuk National University Hospital, Psychiatry, Cheongju, Korea, Republic of (South Korea); ³Chungbuk National University, Center for Environmental Medicine, Cheongju, Korea, Republic of (South Korea); ⁴Chungbuk National University College of Medicine, Division of Rheumatology, Department of Internal Medicine, Cheongju, Korea, Republic of (South Korea); ⁵College of Medicine, Chungbuk National University, Department of Preventive Medicine, Cheongju, Korea, Republic of (South Korea)

Background: Dementia is a common mental illness associated with aging. Alzheimer's disease (AD) and Vascular dementia (VaD) are the most common causes of dementia in the elderly. In previous studies, hyperuricemia was suggested to have anti-oxidant effects and possible neuroprotective effects [1,2].

Objectives: Hyperuricemia is the most important factor in the pathogenesis of gout. This study aims to investigate association between gout and dementia.

Methods: We conducted a nationwide population-based study using the NHIS-NSC database of the Korean populations consisted of 1 million individuals between 2002 and 2013. Gout patients were defined by International Classification of Diseases (ICD)-10 code for gout (M10) and prescriptions for urate lowering therapy including allopurinol, febuxostat and benzbromarone at least 30days. Dementia was also defined by ICD-10 codes for dementia; Alzheimer's disease (AD; F00), vascular dementia (VaD; F01).

Results: Excluded 40 patients with previous diagnosed dementia, total 5,496 gout patients were compared to 27,480 age- and sex-matched control group. The analysis results showed that gout patients had a lower risk of overall dementia [odds ratio (OR): 0.79; 95% confidence interval (CI): 0.63-0.99; $p < 0.05$] after adjusted for age, sex, household income, comorbidities. The risk of VaD (OR: 0.77; 95% CI: 0.32-1.9; $p=0.68$) and AD (OR: 0.8; 95% CI: 0.53-1.19; $p=0.06$) tended to be lower in gout group but these were not significant.

Conclusion: Gout patients had a lower risk of dementia. This result tended to exist for both AD and VaD type of dementia.

REFERENCES:

- [1] Hooper DC, Spitsin S, Kean RB, et al. Uric acid, a natural scavenger of peroxynitrite, in experimental allergic encephalomyelitis and multiple sclerosis. *Proc Natl Acad Sci USA*. 1998; 95: 675–80.
- [2] Bowman GL, Shannon J, Frei B, et al. Uric acid as a CNS antioxidant. *J Alzheimer's Dis*. 2010; 19: 1331–6.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.2292

OP0208

INCIDENT GOUT: RISK OF DEATH AND CAUSE-SPECIFIC MORTALITY IN WESTERN SWEDEN – A NESTED CASE CONTROL STUDY

M. Dehlin¹, T. Zverkova Sandström¹, L. T. H. Jacobsson¹. ¹Institute of Medicine, Sahlgrenska Academy, Department of Rheumatology and Inflammation Research, Gothenburg, Sweden

Background: Excess mortality in gout has been attributed to its positive association with many comorbidities. Lately, a negative association with dementia has been suggested in some cohort studies. Data is limited on how this reflects in cause-specific mortality of gout patients.

Objectives: Our aims were to determine the relative risks in incident gout patients compared to general population 1) for overall death 2) for cause-specific mortality.

Methods: All incident cases of gout (definition: $1 \geq$ ICD-10 code for gout (M10/M14.0) in the population based health care database VEGA; no gout diagnosis ≥ 5 years previously) between 2006-2015 in western Sweden were identified. Five population controls per case matched on age, sex and place of residence were identified. Comorbidities, defined by ICD-10 codes, (alcohol related disorders, hypertension, ischemic heart disease, heart failure, cerebrovascular disease, diabetes, dyslipidemia, obesity, renal disease, dementia, lung diseases and cancer) were identified for 5 years preceding index date for cases and controls in VEGA. Patients were followed until the earliest of death, migration or study end 31 of December 2017. Cause of death was identified through the national cause of death register and grouped as death due to: CVD, renal disease, dementia, infections, diabetes, diseases of the digestive system, lung diseases, cancer and other. COX regression analyses, adjusted for baseline demographics and comorbidities, were used to compare mortality in cases and controls, taking competing cause of death into account by the Fine and Gray method.

Results: From the total adult population of western Sweden between 2006 and 2015 (yearly average of 1264150 individuals), we identified 24320 cases of incident gout and 119781 controls, who were followed on average 5.3 and 5.6 years respectively. At baseline mean age was (SD) 67.3 years (15.3) and 67.6% were men. At baseline, income and level of education was significantly lower in gout cases compared to controls, whereas all comorbidities were significantly more

Table 1. Causes of death by exposure status and cause-specific mortality overall, gout cases compared to controls, adjusted hazard ratios by the Fine and Gray method, 95% confidence intervals

Causes of death	Cause of death, n (%)		Hazard ratios (95% CI)	
	Gout cases, n = 24320	Controls, n = 119781	Model 1	Model 2
Cardiovascular disease	3527 (14.5)	11369 (9.5)	1.61 (1.55-1.67)	1.20 (1.16-1.26)
Renal disease	140 (0.6)	239 (0.2)	2.79 (2.26-3.44)	1.68 (1.31-2.16)
Dementia	287 (1.2)	2922 (2.4)	0.45 (0.40-0.51)	0.68 (0.60-0.77)
Infections	437 (1.8)	1631 (1.4)	1.27 (1.14-1.41)	1.15 (1.03-1.28)
Diabetes	250 (1.0)	665 (0.6)	1.80 (1.55-2.08)	1.02 (0.87-1.20)
Diseases digestive system	226 (0.9)	725 (0.6)	1.49 (1.29-1.74)	1.36 (1.16-1.59)
Lung diseases	306 (1.3)	1216 (1.0)	1.20 (1.06-1.36)	0.89 (0.78-1.01)
Cancer	1179 (4.9)	6067 (5.1)	0.92 (0.87-0.98)	0.94 (0.88-0.99)
Other	655 (2.7)	2899 (2.4)	1.08 (0.99-1.17)	1.09 (0.99-1.19)
All-cause mortality	7007 (28.8)	27733 (23.2)	1.32 (1.29-1.36)	1.08 (1.05-1.11)

Model 1: adjusted for age and sex

Model 2: adjusted for baseline values for: age, sex, marital status, income, education, born outside of Sweden, alcohol related disorders, hypertension, ischemic heart disease, heart failure, cerebrovascular disease, diabetes, dyslipidemia, obesity, renal disease, dementia, lung diseases and cancer

common among gout cases (data not shown) except for dementia which was significantly more common in controls, 4.2% versus 2.5% in cases, $p < .0001$. The HR for overall death was increased in gout patients (HR: 1.08 (95% CI: 1.05-1.11) (Table 1). Gout cases had significantly increased risk for death by CVD, renal disease, infections, diseases of the digestive system and significantly decreased risk for death by dementia and cancer (Table 1). Point estimates were similar in men and women (data not shown).

Conclusion: This study demonstrates a modest increase in overall mortality in gout patients and a decreased risk of death from dementia reflecting its inverse relation to gout.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.454

OP0209

SONOGRAPHIC ESTIMATION OF MONOSODIUM URATE BURDEN PREDICTS THE FULFILMENT OF THE 2016 REMISSION CRITERIA FOR GOUT: A 12-MONTH STUDY

E. Cipolletta¹, A. Di Matteo¹, W. Grassi¹, E. Filippucci¹. ¹Polytechnic University of Marche, Department of Clinical and Molecular Sciences, Ancona, Italy

Background: Preliminary remission criteria for gout include the following five domains: serum uric acid (SUA) levels $< 360 \mu\text{mol/l}$, absence of subcutaneous tophi, absence of gouty flares, pain (due to gout) $< 2/10$ and patient global assessment of disease activity $< 2/10$ [1]. To achieve remission, all criteria must be fulfilled at least twice in a 12-month period [1]. Although imaging techniques allow to visualise monosodium urate (MSU) deposits and to estimate their burden, an imaging domain was not included.

Objectives: To investigate whether baseline MSU burden estimated by ultrasonography (US) predicts the achievement of remission according to these criteria [1] over 12 months.

Methods: In this 12-month prospective, observational and monocentric study, patients with gout according to the 2015 ACR/EULAR criteria and on urate-lowering therapy for at least the preceding 6 months were consecutively recruited. After the baseline clinical assessment, only patients fulfilling all domains of the remission criteria [1] (and therefore amenable to achieve the remission at 12 months) were followed up at 6-month intervals for one year.

The US findings indicative of MSU deposits were identified according to the OMERACT definitions. The following joints were included in the US scanning protocol: elbows, wrists, 2nd metacarpophalangeal joints, knees, ankles and 1st metatarsophalangeal joints. Sum scores of aggregates, double contour (DC) signs, and tophi were calculated separately, and a total score resulting from the sum of all elementary US findings was recorded.

Results: Seventy patients with gout were recruited; of these, 20 (28.6%) were excluded at baseline because 13 (18.6%) did not satisfy the SUA domain, 11 (15.7%) tophus domain, 7 (10.0%) pain domain and 5 (7.1%) disease activity domain. In the enrolled patients (Age: 59.9 ± 14.8 years, female/male ratio: 1/49, disease duration: 6.5 ± 6.6 years), remission criteria were fulfilled in 21 (42.0%) of 50 patients over 12 months.

No significant difference was found between patients fulfilling and not fulfilling the remission criteria at 12 months in all clinical and laboratory data except for the gout flare prophylaxis (7, 33.3% vs 20, 69.0%; $p=0.02$).

The baseline US MSU burden was significantly lower (total score 1.9 ± 1.8) in patients in remission than in those not in remission at 12 months (total score 5.1 ± 3.1) ($p < 0.01$). The fulfilment of the remission criteria at 12 months was recorded in 87.5% of the patients without baseline US evidence of MSU deposits and in only 33.0% of those with at least one US finding indicating MSU deposits ($p < 0.01$).

US scores and gout flare prophylaxis were the only significant predictors of remission in the univariate analyses (Table 1).

Table 1. Predictive values of baseline data for the remission at 12 months.

	OR (95%CI)	P value
Ongoing flare prophylaxis	0.23 (0.07-0.75)	0.02
Total score=0	10.83 (1.14-102.59)	0.04
Aggregate score=0	5.53 (1.34-22.76)	<0.01
DC sign score=0	7.33 (1.71-31.44)	<0.01
Tophus score=0	3.88 (1.08-13.92)	0.02

95%CI: 95% confidence interval; **OR:** odds ratio.

The risk of not fulfilling the remission criteria increased with the US burden of MSU deposits. For each 1-point increase in total score, aggregate score, DC sign score and tophus score, the risk increased by 1.81- (95%CI: 1.27-2.60), 1.73- (95%CI: 1.14-2.64), 4.16- (95%CI: 1.55-11.3) and 1.95-fold (95%CI: 1.07-3.56), respectively (Figure 1).