(43.6%) were on diuretics mainly for hypertension (64.7%), heart failure (9.4%), and renal failure (5.9%). Follow-up data was available from 209 patients (92.9%), with a median 9 months of follow-up (4–14). ULT used was allopurinol in 172 patients (82.6%), febuxostat in 34 (16.5%), and benzbromarone in only 2 cases (0.9%). Regarding the baseline characteristics, patients on diuretics were older, had higher rates of females, hypertension, diabetes, and cardiovascular disease, and showed higher SUA and lower glomerular filtration rate. The table shows the outcomes comparison according to diuretic treatment, globally and stratified by type of ULT (excluding the two cases of benzbromarone). Except for a lower achievement of SUA <5 in the allopurinol subgroup, no significant differences were found either globally or by type of ULT.

Conclusions: Despite its high rate, diuretics currently seem to not have a significant impact on the achievement of the different objectives of treatment of patients with gout.

References:

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Disclosure of Interest: None declared

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THU0438 LACK OF PREDICTIVE VALUE OF THE NIAID/FAAN CRITERIA TO IDENTIFY SUBJECTS WITH EVIDENCE OF IMMUNE ACTIVATION AFTER RECEIVING PEGLOTICASE FOR CHRONIC REFRACTORY GOUT

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Background: The NIAID/FAAN criteria have been widely applied to identify anaphylaxis despite having only modest specificity.^{1,2}

Objectives: To evaluate the predictive value of the NIAID/FAAN criteria to identify evidence of immune activation in subjects with chronic refractory gout treated with pegloticase in randomized clinical trials (RCTs) who experienced infusion reactions (IRs), by assessing the capacity of biochemical testing for immunologic activation to confirm the likelihood of anaphylaxis.

Methods: During the RCTs supporting the approval of pegloticase for treatment of chronic refractory gout, subjects remained on therapy despite becoming nonresponders, which reflected the development of anti-drug antibodies (ADA); ~60% of subjects continued to receive pegloticase despite elevated ADA titers and many had IRs. In subjects receiving pegloticase every 2 weeks (q2w), 22/85 (25.9%) had a total of 43 IRs and 34/84 (40.5%) receiving pegloticase every 4 weeks (q4w) had a total of 70 IRs. 21/22 (95.5%) and 29/34 (85.3%) subjects with IRs in the q2w and q4w groups, respectively, were nonresponders. The 113 total IRs were categorized post hoc as to whether they met NIAID/FAAN criteria for anaphylaxis. Six IRs in 6 subjects met criteria, 53 IRs in 33 subjects had only one feature and were designated as "hypersensitivity", and 54 IRs in 29 subjects had no features and were designated "other". The clinical courses of these IRs and whether they were associated with elevated tryptase levels as a measure of mast cell degranulation or complement consumption as a measure of immune complex activity were assessed.

Results: Of a total of 852 infusions in subjects receiving q2w pegloticase, 43 (5.0%) were associated with IRs as were 70 of 830 infusions (9.5%) in subjects receiving q4w pegloticase. Of the 70 IRs associated with q4w administration, 3 (4.3%) met the criteria for anaphylaxis, 31 (44.3%) for hypersensitivity and 36 (51.4%) were other. The respective values for subjects receiving pegloticase q2w were 3/43 (7.0%), 22/43 (51.1%), and 18/43 (41.9%). A total of 14 IRs (12.4%) were associated with elevated tryptase and 31 (27.4%) with decreased complement CH50 levels. Immunologic abnormalities were not significantly different in the 3 groups, with 16.7%, 17.0% and 7.4% of anaphylaxis, hypersensitivity and other IRs, respectively, having increased tryptase; and 0%, 30.2%, and 27.8%, respectively, having decreased complement. No subjects were classified as experiencing anaphylaxis by investigators, none required hospitalization, and 60.7%, continued to receive pegloticase.

Conclusions: Post-hoc designation of pegloticase-treated subjects as meeting NIAID/FAAN criteria for anaphylaxis was not associated with a higher frequency of biochemical evidence of immune activation or a more severe clinical course. In pegloticase-treated subjects, the NIAID/FAAN anaphylaxis criteria did not identify subjects with IRs associated with elevated tryptase and decreased complement CH50 levels.

References:

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[2] Campbell RL, et al. *J Allergy Clin Immunol*. 2012;129:748–752.

Disclosure of Interest: L. Calabrese Consultant for: BMS, Genentech, Horizon, Crescendo, Pfizer, GSK, Regeneron, UCB, Abbvie, Janssen, A. Kavanaugh Consultant for: Abbvie, Amgen, Janssen, BMS, UCB, Pfizer, A. Yeo Consultant for: Horizon Pharma, P. Lipsky Consultant for: AstraZeneca, Celgene, EMD Serono, GSK, Horizon Pharma, Janssen, Medimmune, Pfizer, Roche, Sanofi, UCB

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THU0439 MORE OBESITY AND HIGHER SERUM URIC ACID IN EARLY-ONSET GOUT PATIENTS IN SOUTH CHINA

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Background: Gout incidence increases in a linear fashion with age until 70 years. It was reported that there was a trend of earlier gout onset. Few studies about clinical features of early-onset gout in China are reported.

Objectives: To investigate the characteristics of early-onset gout in south China.

Methods: Patients with primary gout were recruited between June 2012 and December 2016. Face to face interview was carried out to collect demographic information, clinical characteristics of gout and comorbid diseases. The patients with gout onset before age 30 were defined as early-onset group. The other gout patients were enrolled as control group.

Results: (1) Five hundred and one gout patients were recruited, including 99 (19.8%) in the early-onset group and 402 (80.2%) in the control group. The onset age of the early-onset group was 24.2±5.8 (range 14–30) and the control group was 51.7±13.9 (range 31–84). There was no significant difference of gout duration between early-onset group and control group ($P < 0.05$, Table 1). (2) Compared with the control group, the early-onset group presented lower frequency of flare in last year, higher serum uric acid and higher proportion of sUA >10mg/dl ($P < 0.05$, Table 1). Body mass index (BMI) and prevalence of obesity were higher in the early-onset group ($P < 0.05$, Table 1). The early-onset group exhibited lower prevalence of hypertension, diabetic mellitus, urolithiasis and chronic kidney disease (CKD) ($P < 0.05$, Table 1). (3) There were 20 patients (20.2%) with tophi in the early-onset group. Compared with the 79 early-onset gout patients without tophi, the patients with tophi presented higher age (39.6±8.9 VS 29.1±6.2, $P < 0.001$), duration of gout (12.6±4.8 VS 4.5±4.8, $P < 0.001$), number of involved joints (12.5±9.3 VS 4.3±3.7, $P < 0.001$) and prevalence of CKD (30.0% VS 10.1%, $P = 0.034$), and lower BMI (23.7±3.7 VS 26.6±4.1 kg/m², $P = 0.004$). Logistic regression demonstrated that higher disease duration [OR 1.30, 95% CI (1.15, 1.46), $P < 0.001$] and lower BMI [OR 0.80, 95% CI (0.67, 0.96), $P = 0.015$] were associated with tophi in the early-onset gout patients.

Table 1. Comparison of variables between early-onset group and control group

	Early-onset group (N=99)	Control group (N=402)	P
Age, years	30.4±8.2	57.9±13.5	<0.001
Female, %	1.0%	12.7%	<0.001
Age of onset, years	24.2±5.8	51.7±13.9	<0.001
Duration of gout, years	6.2±5.8	6.2±5.6	0.953
Frequency of flare in last year	6.4±7.5	9.3±10.9	0.022
sUA, mg/dl	10.6±2.4	8.9±2.5	<0.001
>10mg/dl, %	59.6%	32.8%	<0.001
No. of involved joints	5.9±5.0	5.9±5.5	0.981
Tophi, %	20.2%	26.1%	0.397
Alcohol overuse, %	35.4%	34.1%	0.811
BMI, kg/m ²	26.1±4.2	24.8±3.9	0.004
Obesity, %	27.3%	15.5%	0.005
Dyslipidemia, %	51.5%	54.0%	0.666
Hypertension, %	14.1%	44.0%	<0.001
DM, %	6.1%	24.6%	<0.001
Urolithiasis, %	19.2%	33.6%	0.007
eGFR, ml/min/1.73m ²	84.8±51.0	62.9±19.7	<0.001
CKD, %	14.1%	45.6%	<0.001

sUA: serum uric acid; BMI: body mass index; DM: diabetic mellitus; eGFR: estimated glomerular filtration rate; CKD: chronic kidney disease.

Conclusions: Early-onset gout exhibited higher sUA and was associated with obesity but not with hypertension, diabetic mellitus and renal insufficiency. Longer disease duration and lower BMI were associated with tophi in the early-onset gout patients.

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THU0440 TEMPORAL TRENDS OF VENOUS THROMBOEMBOLISM RISK BEFORE AND AFTER DIAGNOSIS OF GOUT. A GENERAL POPULATION-BASED STUDY

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Background: Data on the risk of venous thromboembolism (VTE), including deep venous thrombosis and pulmonary embolism) in patients with gout are scarce.

Objectives: 1) To estimate the overall risk of VTE in an incident gout cohort; 2) To estimate the temporal trends of VTE before and after gout diagnosis compared with the general population.

Methods: We conducted a matched cohort study using a population based administrative health database that includes all outpatient visits, hospital admissions, vital statistics, and dispensed medications for all residents in British Columbia, Canada. Gout cases were defined using one ICD-9/10 code (274/M10) from a

physician or hospital visit. To ensure incident gout, we required that all cases have ≥ 10 years of prior registration without a gout diagnosis. Gout cases were matched 1:1 with controls on age, sex, and cohort entry time. Prior VTE events were excluded after matching. VTE was defined using ICD-9/ICD-10 codes plus use of oral anti-coagulant. We calculated incidence rate ratios (IRRs) and age, sex, and entry time matched multivariable hazard ratios (HRs) for the risk of VTE. Sensitivity analyses were conducted to assess for unmeasured confounders (e.g., obesity).

Results: Among 105,307 individuals with newly diagnosed gout (61% male, mean age of 58 yrs), we observed 1,212 VTE events (mean follow-up time of 5.3 yrs). The fully adjusted HR was 1.27 (95% CI, 1.16–1.39) (Table 1). Our results remained significant in the sensitivity analyses (OR of 2.0 between gout and unmeasured confounder and a 50% prevalence for obesity). There were 437 incident VTE events within 3 yrs prior to the gout diagnosis. Compared to controls, during the 3rd, 2nd, and 1st yrs before gout diagnosis, the fully adjusted HRs for VTE in patients with imminent gout were 1.51, 1.58 and 1.73. The corresponding HRs in the 1st, 2nd, 3rd, 4th, and 5th yrs after gout diagnosis were 1.54, 1.39, 1.38, 1.37, and 1.32, respectively.

Table 1

	Gout (N)	Control (N)	No. of VTE events		IRRs (95% CI)	Age-, sex-adjusted (95% CI)	Fully adjusted* (95% CI)
			Gout	Control			
Before gout diagnosis							
-3yrs	105,744	106,219	437	254	1.73 (1.48,2.03)	1.68 (1.44,1.96)	1.51 (1.29,1.77)
-2yrs	105,638	106,146	331	181	1.84 (1.53,2.22)	1.78 (1.49,2.14)	1.58 (1.31,1.90)
-1yr	105,493	106,055	186	90	2.08 (1.61,2.70)	2.01 (1.56,2.58)	1.73 (1.34,2.24)
After gout diagnosis							
1yr	105,307	105,965	229	116	1.99 (1.58,2.51)	1.92 (1.54,2.40)	1.54 (1.22,1.94)
2yrs	105,307	105,965	390	234	1.68 (1.43,1.99)	1.63 (1.39,1.92)	1.39 (1.18,1.64)
3yrs	105,307	105,965	540	331	1.65 (1.43,1.89)	1.60 (1.39,1.83)	1.38 (1.20,1.59)
4yrs	105,307	105,965	667	414	1.63 (1.44,1.85)	1.58 (1.40,1.79)	1.37 (1.21,1.56)
5yrs	105,307	105,965	775	506	1.55 (1.38,1.74)	1.51 (1.35,1.68)	1.32 (1.18,1.49)
Overall	105,307	105,965	1,212	858	1.44 (1.32,1.57)	1.40 (1.29,1.53)	1.27 (1.16,1.39)

*Adjusted for Charlson comorbidity index, cancer, alcoholism, hypertension, sepsis, trauma, healthcare utilization, NSAIDs, HRT, glucocorticoid and fractures, surgery within 1 year before entry cohort.

Conclusions: Patients with gout have an increased risk of VTE. The risk increases gradually before gout diagnosis, peaks in the year prior to gout diagnosis, and then progressively declines. Our findings suggest that hyperuricemia or gout associated inflammation may be a contributing factor for VTE.

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THU0441 SYNOVIAL FLUID LEUKOCYTE COUNT AND ITS ASSOCIATION WITH CRYSTAL DEPOSITION IN ASYMPTOMATIC HYPERURICEMIA: A PRELIMINARY REPORT

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Background: Joint deposits of monosodium urate (MSU) crystals in asymptomatic hyperuricemia (AH) may associate with more severe forms of atherosclerosis [1]. It likely relates with crystal-driven subclinical inflammation, a phenomenon well described in gout [2] but to date not assessed in AH.

Objectives: To assess the synovial fluid (SF) leukocytes count in patients with AH depending on the presence of crystals.

Methods: Consecutive patients with AH were selected from nephrology clinics. Hyperuricemia threshold was established at serum uric acid (SUA) $\geq 7\text{mg/dL}$; those on current urate-lowering therapy were excluded. Ultrasound (US) of knees, ankles and first metatarsophalangeal joints was performed blinded to clinical and laboratory data, in order to perform US-guided aspiration and obtain SF samples. These samples were analyzed in fresh by two observers, using a compensated polarized light microscope equipped with two viewing stations. The second observer was in addition unaware of US findings. The presence of leukocytes and crystals was established by consensus. SF leukocyte count was performed using a Neubauer counting camera. Mann-Whitney's U and Kruskal-Wallis' H were used to compare difference in leukocyte counts between groups.

Results: To end of January 2017, 30 patients have been assessed, with SF samples available from 27 (three showed no joint effusion at US). Median age was 70 years (p25–75 59.8–75.5), and 16 of them (60%) were males. Median (p25–75) SUA and estimated glomerular filtration rate were 8.1 mg/dL (7.6–8.5) and 37.0 mL/min (31.0–47.5), respectively. Seven (25%) were on diuretics. After microscopy evaluation, MSU crystals were found in four patients (14.8%), calcium pyrophosphate (CPP) crystals in five (18.5%), and no crystals in 18 (66.7%). Figure shows median leukocytes count according to crystal group: 200/mm³ (138–540) in MSU group; 60/mm³ (50–235) in CPP group; and 30/mm³ (10–53) in those with no crystal at SF. A statistically significant difference was found between the three groups (p=0.001), and comparing MSU crystals and no crystals groups (p<0.001); no difference was found between MSU and CPP groups (p=0.111).

Conclusions: In a preliminary report, MSU crystal deposition in patients with AH leads to higher SF leukocyte count, indicating low-grade inflammation. Despite low numbers and being an ongoing study, this significant finding might contribute to explain the severe atherosclerosis data recently reported in AH patients with silent crystal deposits.

References:

[1] Arthritis Rheumatol 2016;68:1531.

[2] Arthritis Rheum 1991;34:141.

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THU0442 EHEALTH SUPPORTED COLLABORATIVE CARE MODEL FOR GOUT INVOLVING RHEUMATOLOGY, PHARMACY, AND DIETETICS: PROOF-OF-CONCEPT OBSERVATIONAL STUDY

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Background: Sub-optimal quality of care for gout, the most common inflammatory arthritis, has called for novel models to improve care and outcomes of patients.

Objectives: Our objective was to evaluate the feasibility of the "Virtual Gout Clinic Study" (VGCS) a collaborative approach to gout management involving rheumatology, pharmacy, and dietetics that take advantage of eHealth, namely, shared access of electronic medical records (EMRs), to support this unique, decentralized model of care.

Methods: Design: We conducted a 1-year, proof-of-concept, longitudinal observational study with a target of 30 patients. **Eligibility:** Patients with gout were eligible if they were: 1) seen in 1 of 4 rheumatology practices with 8 rheumatologists participating across British Columbia, Canada; 2) experienced ≥ 1 flare in the past year; 3) have a serum uric acid (SUA) level $\geq 360 \mu\text{mol/L}$ within the previous 2 months. **Follow-up:** Within the VGCS, patients receive follow-up with their rheumatologist on an as needed-basis, monthly telephone consults from the study pharmacist, and 1 telephone consult with the dietician. In this decentralized model of care, health care professionals are not co-located but have access to the patient's EMR to facilitate remote communication and collaboration. **Outcomes:** Prospective data was collected at baseline, 3, 6, and 12 months. The primary outcome measure was SUA at 12-months, specifically below recommended target level of $360 \mu\text{mol/L}$. Secondary outcomes included patient reported outcome measures including the Compliance Questionnaire Rheumatology, Health Assessment Questionnaire Disability Index, and EQ-5D, as well as information on interventions provided within the VGCS.

Results: Overall, 33 individuals with gout participated in the VGCS. There were 22 males (88%) and mean age was 60.8 ± 15.3 years. 29, 28, and 25 patients have completed follow-up at 3, 6, and 12 months respectively. At baseline, 22 (67%) patients were prescribed urate-lowering therapy (ULT; 21 with allopurinol and 1 with febuxostat). The remaining 11 patients initiated ULT during the study period. Figure 1 shows mean SUA decreased over baseline, 3, 6, and 12 months from $451 \mu\text{mol/L}$, $367 \mu\text{mol/L}$, $356 \mu\text{mol/L}$, and $364 \mu\text{mol/L}$, respectively, with 67% of patients having SUA $< 360 \mu\text{mol/L}$ at 12 months. A total of 126 pharmacist and 24 dietitian consults have been provided. Pharmacist interventions included: up-titration of allopurinol dosage for 15 patients, restarting of ULT for 4 non-adherent patients, discontinuation of unnecessary medications for 12 patients, and renewals of prescription medications using solely electronic communications for 4 patients.

Conclusions: These findings provide evidence for the feasibility and effectiveness of the VGCS. With respect to feasibility, we successfully established the shared EMR framework between rheumatologists and study pharmacist and dietitian and demonstrated utility with respect to facilitating communication and collaborative