66 missings. 90% of the participants rated app usability good to excellent and 70% would recommend the app to other patients.

Twelve out of thirty patients generated a total amount of 174 alerts where four patients with a suspicion of gout were responsible for 148 alerts (85%). These patients scored three out of four criteria as they had warm, swollen and painful joints but, after consultation with the clinician, their symptoms were not regarded as a gout flare.

The 174 alerts belonged to 23 (possible) flares with a median duration of 5 days [IQR 3.5 – 7.5]. Twenty-one pro-active telephone calls were made which resulted in four visits to the clinic within 48 hours. Clinical guidance over the phone consisted of checking in on patient's symptoms, giving advice and ten medication adjustments.

Conclusion: This prospective study shows feasibility of a smartphone app for home-monitoring gout flares for patients because of high usability scores and low attrition rates. The app has added value for gout care because it enables clinicians to act on flares as they occur. The next step is to further implement the app whilst perpetuating investigation into the added value for patients and clinical practice alike.

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Acknowledgements: This study was funded by AbbVie and Menarini.

Disclosure of Interests: : Bart Pouls: None declared, Charlotte Bekker: None declared, Bart van den Bemt Grant/research support from: UCB, Pfizer and Abbvie, Consultant of: Delivered consultancy work for UCB, Novartis and Pfizer, Speakers bureau: Pfizer, AbbVie, UCB, Biogen and Sandoz., Angelo Gaffo Grant/research support from: Received a research grant from AMGEN, Marcel Flendrie Grant/research support from: M. Flendrie has received grants from Menarini and Grunenthal., Consultant of: M. Flendrie has received consultancy fees from Menarini and Grunenthal.

DOI: 10.1136/annrheumdis-2020-eular.3643

THU0448

NO DIFFERENCE IN GOUT FLARES AFTER INITIATION OF URATE LOWERING THERAPY BETWEEN ONCE OR TWICE DAILY 0.5 MG COLCHICINE PROPHYLAXIS

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Background: Initiation of urate lowering therapy (ULT) is associated with an increase in gout flares¹. Therefore, guidelines advice prophylactic therapy during the first six months of ULT². Colchicine 0.5-1 mg/day is recommended. Whether 1 mg/day colchicine is superior to 0.5 mg/day is unknown.

Objectives: To examine the difference in gout flares in the first six months after initiation of ULT between 0.5 and 1.0 mg/day colchicine prophylaxis.

Methods: Patients with clinical diagnosis of gout, a first outpatient visit between January 2010 and March 2018 and a follow-up of at least 6 months were included in a retrospective cohort study, conducted in two rheumatology centres in the Netherlands. Within this cohort, patients starting ULT and colchicine prophylaxis were selected. Difference in gout flare incidence density (ID) in the first six months after start of ULT between colchicine 0.5 and 1.0 mg/day was analysed using Poisson regression, corrected for confounders. Secondary analyses included the proportion of patients reaching a target of serum urate acid (SUA) of < 0.36 mmol/l within the first six months.

Results: Of 2108 gout patients, 379 patients started ULT and used colchicine prophylaxis (table 1). ID of flares was 2.8 and 2.6 per patient year on colchicine 1.0 and 0.5 mg/day respectively, resulting in an incidence rate ratio of 1.05 (95% CI 0.86 – 1.27). Within the first six months 68% in the colchicine 1mg/day and 63% in the colchicine 0.5 mg/day reached their target (difference 5%, 95% CI -0.08 – 0.18).

Table 1. Baseline patient, disease and treatment characteristics

	Colchicine prophylaxis	
	0.5 mg/day (n=275)	1 mg/day (n=104)
Men, no. (%)	226 (82%)	88 (85%)
Age, years	67.3 (58.8-76.3)	65.3* (54.4-71.9)
BMI, kg/m ²	29.0 (26.1-32.1)	29.5 (27.9-31.6)
Comorbidity no. (%)		
Hypertension	146 (53%)	54 (52%)
Diabetes Mellitus	66 (24%)	24 (23%)
Hypercholesterolemia	43 (16%)	15 (14%)
Renal insufficiency	56 (20%)	18 (17%)
Crystal-confirmed diagnosis, no. (%)	197 (72%)	77 (74%)
Serum urate acid at start ULT (mmol/l), mean ± SD	0.51 ± 0.11	0.50 ± 0.11
ULT medication, no. (%)		
Allopurinol	272 (99%)	103 (99%)
Benzbromaron	2 (0.6%)	
Febuxostat	1 (0.4%)	1 (1%)

*Compared to colchicine 0.5 mg/day, P<0.05.

**No SD possible, n = 1.

Conclusion: Use of 1 mg/day colchicine is not superior to 0.5 mg/day as prophylaxis for ULT induced gout flares. For generalisability it should be noted that flare rates were not very high, probably due to the background ULT being characterised by a "start low, go slow" approach. In this context colchicine 0.5 mg/day is sufficient as prophylaxis.

References:

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Disclosure of Interests: : Frouwke Veenstra: None declared, L.M. Verhoef: None declared, Lieke Nieboer: None declared, Alfons den Broeder: None declared, Wing-Yee Kwok: None declared, Inger Meek: None declared, Frank van den Hoogen: None declared, Noortje van Herwaarden: None declared, Marcel Flendrie Grant/research support from: M. Flendrie has received grants from Menarini and Grunenthal., Consultant of: M. Flendrie has received consultancy fees from Menarini and Grunenthal.

DOI: 10.1136/annrheumdis-2020-eular.3635

THU0450

OPTIMISTIC STATUS ASSOCIATES WITH COMPLIANCE TO URATE-LOWERING THERAPY IN GOUT PATIENTS

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Background: Compliance to urate-lowering therapy (ULT) is poor in gout patients, which contributes to increased frequency of acute gout attacks, deposition of tophi and urate nephropathy [1]. Optimistic status is probably a potential and considerable factor affecting compliance to ULT in gout patients.

Objectives: To compare optimistic status between gout patients and healthy controls, and also between gout patients with good and poor compliance. Relationship between optimistic status and compliance to ULT, sUA target achievement of gout patients were assessed as well.

Methods: This was a monocentric and observational study which was performed from August 2018 to December 2019. Adult patients who met the 2015 gout classification criteria were included in this study. The healthy controls were individuals who were free of gout, hyperuricemia and other rheumatic diseases from the physical examination center of our hospital. Demographic data, including age, gender and education were collected from all individuals. Serum uric acid (sUA) were collected from gout patients at enrollment and again after 3 months. Disease duration of gout, visual analogue scale (VAS) of pain were also assessed for gout patients at enrollment. Compliance to ULT was measured using the medication possession ratio (MPR) in the following 3 months. Poor compliance was defined as MPR<0.8 and good compliance was defined as MPR≥0.8. All subjects completed the life orientation test-revised (LOT-R) for optimistic status assessment.

Results: Five hundred and thirty gout patients and 307 healthy controls matched by age (41.4±12.3 vs. 42.1±9.3 years), gender (male 97.1% vs. 95.1%) and education (college graduated 54.2% vs. 58.0%) were included in this study. Of the 530 gout patients, the mean disease duration was 5.7±4.9 years, and 292 (55.1%) patients' MPR were lower than 0.8. There was no statistic difference in LOT-R between gout patients and healthy controls (19.0±2.4 vs. 19.2±2.5, P>0.05) (Table 1). Gout patients with poor compliance (MPR<0.8) had higher level of sUA (525.5±138.0 vs. 471.2±152.5 μmol/L, P<0.05), followup sUA (450.1±154.5 vs. 361.6±120.0 μmol/L, P<0.05) and higher LOT-R (19.6±2.6 vs. 17.8±1.7, P<0.05) than those with good compliance (MPR≥0.8). Of the 292 gout patients with poor compliance, there were only 83 (28.4%) patients achieved sUA target after 3 months, and their LOT-R were significantly lower than

those did not achieve sUA target (18.8±2.1 vs. 19.6±2.4, P<0.05). Finally, LOT-R correlated positively with sUA (r=0.131, P<0.05) and followup sUA (r=0.09, P<0.05), but negatively with MPR (r=-0.473, P<0.05) of gout patients (Table 2).

Table 1 Demographic and optimistic status of gout patients and healthy controls

Variables	Gout patients (n=530)	Controls (n=307)	P
Age (years)	41.4±12.3	42.1±9.3	0.116
Male gender, n (%)	511 (96.4)	292 (95.1)	0.368
College graduated, n (%)	287 (54.2)	178 (58.0)	0.312
LOT-R	19.0±2.4	19.2±2.5	0.189

LOT-R: life orientation test-revised

Table 2 Correlation analysis between LOT-R and clinical variables in gout patients

Variables	LOT-R	P
Age	0	0.994
Disease duration	-0.058	0.182
VAS	0.034	0.432
sUA	0.131	0.003*
Followup sUA	0.126	0.016*
MPR	-0.393	<0.001*

LOT-R: life orientation test-revised, VAS: visual analogue scale, sUA: serum uric acid, MPR: medication possession ratio

*P<0.05

Conclusion: Gout patients share similar optimistic status to healthy controls. However, optimistic status relates to compliance to ULT and sUA target achievement of gout patients.

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Disclosure of Interests: : None declared

DOI: 10.1136/annrheumdis-2020-eular.6202

THURSDAY, 04 JUNE 2020

Basic and translational pain science

THU0451

CELL-MATRIX ADHESION OF BONE MARROW STROMAL CELLS IN MODIC TYPE 1 CHANGES IS INCREASED AND RELATES TO INCREASED EXPRESSION OF INTEGRIN β 1

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Background: Modic type 1 changes (MC1) are vertebral bone marrow lesions associated with non-specific low back pain (LBP). The pathophysiology of MC1 includes inflammation, fibrosis, and high bone turnover. Bone marrow stromal cells (BMSCs) are key regulators of these processes: BMSCs contribute to inflammation by regulating myelopoiesis/osteoclastogenesis; BMSCs can differentiate into osteoblasts contributing to high bone turnover, and BMSCs can differentiate into pro-fibrotic myofibroblasts.

Objectives: To identify dysregulated biological processes in MC1 BMSCs contributing to the pathobiology of MC1.

Methods: Bone marrow aspirates were obtained from LBP patients with MC1 undergoing lumbar spinal fusion. Aspirates were taken prior to screw insertion. From each patient, a MC1 and a healthy control (HC) aspirate from the adjacent vertebral body was collected. BMSCs were isolated by plastic adherence and expanded. RNA from BMSCs passage 3 was sequenced (n=3+3) (Illumina Novaseq) and gene ontology of significantly dysregulated genes (p-value < 0.05) was analyzed. Specificity and rate of BMSC matrix adhesion were quantified: BMSCs (n=8+8) were seeded on fibronectin-coated, collagen-I-coated, and non-coated plastic dishes. BMSC adhesion was evaluated

from 15min to 30min (Δ 30min - 15min). Percentage of adherent cells of MC1 and HC BMSC was compared with paired t-test. In order to identify integrins responsible for dysregulated cell-matrix adhesion, gene expression of 15 relevant integrins was measured by quantitative real-time PCR (qPCR). Normalized expressions were compared between MC1 and HC BMSC with paired t-test. Integrin β 1 protein level was semi quantitatively analyzed by Western Blot (n = 5+5) and normalized to β -Actin expression.

Results: By RNA sequencing, 154 genes were differentially expressed between MC1 and HC BMSCs (p-value $\leq$ 0.01; log₂-ratio $\geq$ 0.5). Gene ontology enrichment analysis revealed an overrepresentation of the biological process "cell-matrix adhesion" among all significantly regulated genes (p-value < 9.3e-13). A change in cell adhesion was corroborated with adhesion assay. Binding (Δ 30min - 15min) to collagen I (MC1+16%, HC +10%, p-value = 0.10), fibronectin (MC1+17%, HC +6%, p-value = 0.03), and non-coated surface (MC1+46%, HC +35%, p-value = 0.05) was increased in MC1 (Figure 1). Integrin gene expression analysis revealed significant upregulation of integrin beta-1 gene (*ITGB1*) in MC1 vs. HC (fold change = 1.24, p-value = 0.047), whereas there was no significant difference between the other integrins tested. On protein level, integrin β 1 was upregulated in MC1 in four out of five patients (Figure 2).

Figure 1. Adhesion assay.

Figure 2. Western Blot analysis.

Conclusion: Adhesion of BMSCs to matrix and integrin β 1 expression are increased in MC1. Integrin β 1 is essential for cell-matrix adhesion and an important contributor to the initiation and progression of tissue fibrosis, a hallmark of MC1. Therefore, BMSCs and integrin β 1 might be relevant novel targets for the treatment of MC1.

Disclosure of Interests: : Irina Heggli: None declared, Susanne Epprecht: None declared, Tamara Mengis: None declared, Astrid Juengel: None declared, Michael Betz: None declared, Jose Spirig: None declared, Florian Wanivenhaus: None declared, Florian Brunner: None declared, Mazda Farshad: None declared, Oliver Distler Grant/research support from: Grants/Research support from Actelion, Bayer, Boehringer Ingelheim, Competitive Drug Development International Ltd. and Mitsubishi Tanabe; he also holds the issued Patent on mir-29 for the treatment of systemic sclerosis (US8247389, EP2331143)., Consultant of: Consultancy fees from Actelion, Acceleron Pharma, AnaMar, Bayer, Baecor Discovery, Blade Therapeutics, Boehringer, CSL Behring, Catenion, ChemomAb, Curzion Pharmaceuticals, Ergonex, Galapagos NV, GSK, Glenmark Pharmaceuticals, Inventiva, Italfarmaco, iQvia, medac, Medscape, Mitsubishi Tanabe Pharma, MSD, Roche, Sanofi and UCB, Speakers bureau: Speaker fees from Actelion, Bayer, Boehringer Ingelheim, Medscape, Pfizer and Roche, Stefan Dudli: None declared

DOI: 10.1136/annrheumdis-2020-eular.2833