Figure 1 Linear regression analysis of the correlation between food intake frequency and BF% and sUA in male gout patients

Food intake frequency	All patients (n=338)	Correlation with BF%				Correlation with sUA			
		Coefficient β	LCI	UCI	P	Coefficient β	LCI	UCI	P
Alcohol									
None	141(44.3)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-20g/d	89(28)	-1.0466	-2.5604	0.4711	0.1760	-0.3635	-0.9208	0.2429	0.2408
21-40g/d	39(12.3)	0.2762	-1.8168	2.3692	0.7953	0.2723	-0.5348	1.0793	0.5072
>40g/d	49(15.4)	0.7476	-1.3134	2.8086	0.4758	0.3947	-0.4607	1.1301	0.4082
Red meat									
0-100g/d	102(32.1)	0.0	NA	NA	NA	0.0	NA	NA	NA
101-200g/d	149(46.8)	-0.9870	-2.4907	0.5567	0.2126	-0.2361	-0.8253	0.3530	0.4307
201-300g/d	52(16.4)	-0.6439	-2.3878	1.0999	0.1349	0.3138	-0.4343	1.0519	0.4079
>300g/d	15(4.7)	0.9134	-2.2809	4.1076	0.5740	0.1041	-1.1282	1.3364	0.8680
Animal oil									
<1 times/w	227(71.4)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	65(20.4)	0.5580	-1.2227	2.3388	0.5378	-0.5776	-1.2646	0.1095	0.0991
3-4 times/w	15(4.7)	-0.8508	-9.9278	2.2261	0.5867	-0.7521	-9.9994	0.4916	0.2131
>4 times/w	11(3.5)	-2.5650	-6.1940	1.0628	0.1650	-10.1541	-2.5499	2.5551	0.3506
Seafish									
<1 times/w	215(67.6)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	59(18.4)	0.7886	-0.3244	2.4915	0.5672	-0.1258	-0.7203	0.5297	0.6587
3-4 times/w	25(7.8)	-0.9334	-3.3759	1.5091	0.4525	-0.0335	-0.9762	0.9092	0.9443
>4 times/w	19(6)	-2.5593	-5.3741	0.2555	0.0746	-0.1390	-1.2305	0.9525	0.8023
Veget									
<1 times/w	287(90.3)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	23(7.2)	0.7567	-1.7281	3.2415	0.5493	-0.1055	-1.0642	0.8532	0.8286
3-4 times/w	4(1.3)	-1.0208	-6.7962	4.7643	0.7248	0.1551	-2.0547	2.3650	0.8902
>4 times/w	4(1.3)	-1.0445	-6.9190	4.8299	0.7246	-0.0671	-2.3326	2.1983	0.9535
Slow-cooking soup									
<1 times/w	129(40.6)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	99(31.1)	-0.1611	-1.6797	1.3576	0.8348	-0.3682	-0.9538	0.2174	0.2169
3-4 times/w	52(16.4)	-1.1055	-3.6452	0.8342	0.2628	-0.1888	-0.9383	0.5608	0.6705
>4 times/w	38(11.5)	0.7027	-1.4212	2.8265	0.5354	-0.3409	-1.1604	0.7586	0.4135
Fructose containing beverages									
<1 time/w	168(52.8)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	44(13.8)	1.4198	-0.5328	3.3725	0.1534	0.0239	-0.7318	0.7795	0.9504
3-4 times/w	41(12.9)	2.4619	0.3580	4.5657	0.0220	-0.0888	-0.9077	0.7301	0.8311
>4 times/w	65(20.4)	2.8996	0.8266	4.9726	0.0049	0.6695	-0.021	1.4023	0.0730
Tea									
<1 times/w	147(46.2)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	34(10.7)	-0.5081	-2.7650	1.6789	0.6308	0.0261	-0.8309	0.8832	0.9522
3-4 times/w	14(4.4)	0.9783	-2.2133	4.1700	0.5407	-0.1891	-1.4205	1.0423	0.7626
>4 times/w	123(38.7)	-0.1466	-1.5524	1.2592	0.5975	0.2479	-0.2942	0.7900	0.3688
Coffee									
<1 times/w	293(92.1)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	15(4.7)	-0.9200	-4.9628	1.1228	0.2132	-0.3609	-1.2162	0.2244	0.1130
3-4 times/w	10(3.1)	-0.7054	-10.4630	11.0761	0.9112	10.1822	3.1249	5.4884	0.5894
>4 times/w	9(2.8)	-1.6474	-6.7000	2.4052	0.4243	0.2745	-1.2899	1.8388	0.7801
Milk and milk products									
<1 times/w	173(54.4)	0.0	NA	NA	NA	0.0	NA	NA	NA
1-2 times/w	95(29.4)	-0.5368	-1.9924	0.9188	0.4885	0.3462	-0.2155	0.9080	0.2760
3-4 times/w	27(8.5)	-1.7837	-4.1981	0.5407	0.320	-0.2811	-1.1810	0.6189	0.5991
>4 times/w	23(7.2)	0.9340	-2.4735	2.5519	0.965	0.0890	-0.8837	1.0436	0.8575

Multivariate^a, adjusted for potential confounding factors including age, duration of gout, family history, eGFR, comorbidities (including hypertension (yes/no), diabetes mellitus (yes/no), dyslipidemia (yes/no), metabolic syndrome (yes/no), and fatty liver (yes/no)) and the other dietary factors.

Multivariate^b, adjusted for the confounding factors mentioned above and BF%.

LCI, lower limit of confidence interval; UCI, upper limit of confidence interval; NA, not applicable; time/w, the frequency of dietary intake per week; BF%, body fat percentage; sUA, serum uric acid.

Conclusion: Our data show that body fat mediates the correlation between FCB consumption and sUA in male gout patients, which imply the importance of body composition assessment.

Acknowledgement: The present study was supported by Guangdong Natural Science Foundation, China (Grant no. 2014A030310086) to Qian-Hua Li.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.5774

SAT0409

P2X7R PROMOTE THE ATTACK OF ACUTE GOUTY ARTHRITIS IN RATS FROM CLINICAL TO PATHOLOGY

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Background: ATP may be the second causative signal for the onset of gout, which acts on P2X7R to regulate the development of acute gouty arthritis (1). P2X7R is the most relevant to the inflammatory response in the P2X family and is also an ATP-gated cation channel (2).

Objectives: The aim of this study was to activate P2X7R signaling pathway through changes in extracellular ATP concentrations, leading to the development of acute gouty arthritis and the production of pro-inflammatory.

Methods: 120 male Sprague-Dawley rats were randomly divided into 3 groups: After establishment of acute gouty arthritis model, rats were given P2X7R agonist ATP, P2X7R inhibitor BBG and PBS, respectively. The clinical manifestations of the ankle joints were evaluated at 6h, 12h, 24h, 48h and 72h before the rats were sacrificed, and rat ankle synovial slices for H&E staining. IL-1 β , IL-6 and TNF- α in the serum of rats were detected by ELISA kits.

Results: 1. P2X7R regulates the development of acute gouty arthritis: At 12h, the clinical scores of ATP group were significantly higher than those of BBG group and control group (P=0.001,0.042), and the control group was higher than BBG group (P=0.034). At 12h and 24h, the swelling index of ATP group was the most obvious than other two groups (P=0.000, 0.001; P=0.000, 0.003), followed by control group (P=0.009, 0.001); Furthermore, there was a large infiltration of

inflammatory cells in the synovial tissue of the right ankle joint of rats, at 12h, and 24h, the infiltration of mononuclear cell in ATP group was significantly higher than that in BBG group and control group (P=0.000, 0.007; P=0.000, 0.001); The neutrophils infiltration in ATP group was the highest among the three groups at 24h (P=0.001, 0.04), and the control group was higher than BBG group (P=0.04). 2. P2X7R regulates pro-inflammatory cytokines production: At 24h, the level of IL-1 β in ATP group was significantly higher than BBG group and control group (P=0.001, 0.003); At 6h, 12h and 24h, higher level of IL-6 in ATP group compared with BBG group and control group (P=0.004, 0.04; P=0.000, 0.002; P=0.001, 0.012); The level of TNF- α was obviously higher in ATP group than in BBG group and control group at 6h and 24h (P=0.007, 0.011; P=0.001, 0.018), and BBG group was lower than control group, but without statistically significant (P>0.05).

Conclusion: Activation of P2X7R can significantly promote the attack of acute gouty arthritis and the production of IL-1 β , IL-6 and TNF- α , suggesting that P2X7R affects the development of acute gouty arthritis and regulates the secretion of pro-inflammatory cytokines.

REFERENCE

- [1] Tao JH, Zhang Y, Li XP. P2X7R: a potential key regulator of acute gouty arthritis. *Semin Arthritis Rheum*, 2013, 43(3): 376-380. (2) Hechler B, Gachet C. Purinergic receptors in thrombosis and inflammation[J]. *Arterioscler Thromb Vasc Biol*, 2015, 35:2307-2315

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.7147

SAT0410

RADIOGRAPHIC EROSION SCORE IMPROVED WITH TARGETED URATE-LOWERING THERAPY IN A PROSPECTIVE GOUT VIETNAMESE COHORT

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Background Urate deposition in joints of patients with neglected gout leads to destructive arthropathy, with subchondral bone erosion, bone construction, and late joint space narrowing. Gouty erosions are believed to improve under urate lowering drugs (ULDs), but their course has been little studied. We therefore performed a systematic prospective study of a Vietnamese cohort started on allopurinol at inclusion.

Objectives To characterize radiographic outcome of gouty erosions under serum urate-targeted ULD

Methods Included patients had crystal-proven gout, eGFR > 60 ml/min and were not taking ULDs at inclusion. Allopurinol was progressively increased to reach EULAR- recommended uricemia targets and patients were prospectively followed-up, in particular with sequential hand and foot antero-posterior radiographs and MTP and knee ultrasound (US) scans. Joint erosions were scored according to a validated index1, double contour thickness was evaluated by a 4 point-score. One index tophus per patient (hand or foot) was measured by US scan.

Results 100 patients (99M) with a mean age of 45.9 + 9.9 years, and median gout duration of 8 years (range 0-33) were included. 91 had clinical tophi. Mean baseline uricemia was 492 μ mol/L. Median final allopurinol dose was 520 mg/day. 71 patients had evidence of urate arthropathy at baseline, 68 of the feet and 23 of the hands (all but 3 with foot involvement). 1st MTPs were predominantly involved (44 and 35 patients on the right and left sides respectively). Urate arthropathy at baseline correlated with gout duration (p=0.01), clinical tophi (p=0.0007), maximum double contour thickness and index tophus US measurement (p=0.03) by univariate analysis and with clinical tophi by multivariable analysis (p=0.03). Uricemia target <300 μ mol/L was obtained in 63.6, 67.8, 70.6, and 61.0 % of patients at 3, 6, 12 and 24 months respectively. Median radiological follow-up was 12 months (range 3-49). Mean radiological erosion score1 was 12.1 at baseline (n=71), with a decrease of -0.7 at 6 months (n=38,

$p=0.004$), -1.4 at 12 months ($n=47$, $p<0.0001$) and -4.2 at 24 months ($n=14$, $p=0.001$). Complete radiographic erosion healing was observed in only one foot joint in which a small baseline erosion disappeared on M33 radiograph. Erosion score decrease correlated with decrease of double contours at 12 months ($\rho=0.45$, $p=0.001$) and with mean SUA between M3 and M12 ($\rho=0.59$, $p=0.03$). A total of 41 joints (hand and foot DIPs, foot II-V PIPs) could not be scored due to score specifications. Prominent diaphysis erosions were seen in 6 patients and were not allowed scoring despite improvement. Bone construction and reappearance of bone contours were striking features of improved radiographs that were not measured by the score. Ankylosis of a toe joint was observed in 4 patients. Shrinking of 3 toes developed during dissolution of massive joint space/subchondral bone urate deposits.

Conclusion Urate-lowering improved gouty erosion score, in correlation with decrease of uricemia and urate crystal load. However, complete erosion recovery was seldom observed during a median follow-up of 12 months. Increased bone production and massive urate dissolution under ULD occasionally led to 2 undescribed features of gouty arthropathy: joint ankylosis and shrinking of involved toes respectively

REFERENCES

[1] Dalbeth et al, *Arthritis Care Res* 2007;57:1067-73.

Acknowledgement ART Viggo, Société Française de Rhumatologie

Disclosure of Interests Thomas Bardin Consultant for: Astrazeneca, Grunenthal, Horizon, Novartis, Speakers bureau: Astella, AstraZeneca, Grunenthal, Quang Dinh Nguyen: None declared, Khoi Minh Tran: None declared, Nghia Hieu Le: None declared, Duc Minh Do: None declared, Valérie Bousson: None declared, Pascal Richette Consultant for: Grunenthal, Horizon, Speakers bureau: AstraZeneca, Grunenthal, Matthieu resche-rigon: None declared

DOI: 10.1136/annrheumdis-2019-eular.7882

SAT0411

COMORBIDITY PATTERNS IN GOUT USING THE US GENERAL POPULATION – CLUSTER ANALYSIS OF THE NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY 2007-2016

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Background: Gout is associated with many metabolic and cardiorenal comorbidities. Several previous studies investigated the comorbidity patterns by cluster analysis; however, no such study has been based on

Table 1. Characteristics of Patients with Gout ($n = 8607137$)

Characteristics	Overall ($n = 8607137$)
Demographics	
Age, years (SD)	61.4 (0.6)
Body Mass Index, kg/m ² (SD)	32.0 (0.3)
Comorbidities	
Obesity (%)	4586268 (53)
Hypertension (%)	6280529 (73)
Diabetes (%)	2991109 (35)
Dyslipidemia (%)	5328364 (62)
Hypercholesterolemia (%)	5200179 (60)
Hypertriglyceridemia (%)	500382 (6)
Metabolic Syndrome (%)	4295088 (50)
Coronary Heart Disease (%)	1026990 (12)
Heart Failure (%)	976066 (11)
Cerebrovascular Disease (%)	731253 (9)
Chronic Kidney Disease (%)	1747506 (20)
Nephrolithiasis (%)	1333749 (16)
Liver Disease (%)	1559172 (18)
Malignancy (%)	1866732 (22)

the entire general population. As such, the generalizability, which is essential to these pattern analyses, remains unknown.

Objectives: We used cluster analysis on a large, representative general population sample of the United States to identify discrete phenotype patterns comorbidities observed in gout patients.

Methods: We used data from 8,607,137 participants in the 2007-2016 National Health and Nutrition Examination Survey (NHANES), a nationally representative sample of adults in the United States. Diagnosis of gout was based on survey of physician- or health professional-diagnosed gout. We used cluster analysis to identify subgroups among participants with gout with distinct comorbidity subgroups based on 9 variables: obesity, hypertension, diabetes, hypercholesterolemia, hypertriglyceridemia, coronary heart disease (CHD), heart failure (HF), chronic kidney disease (CKD), and liver disease.

Results: Cluster analysis identified 5 different subgroups (C1-C5) (Table 2). C1 had the highest proportion of patients with CKD (97%). C2 consisted of patients with gout but few other comorbidities. All patients in C3 had diabetes, but no HF, CHD, or CKD. C4 consisted primarily of patients with liver disease (85%), with high prevalences of lipid abnormalities and obesity. C5 had the highest proportion of patients with CHD and HF.

Conclusion: These findings from a nationally representative data of US adults indicate that gout comorbidities have 5 distinctive phenotypic patterns. These subgroups may have implications in pathogenesis of gout and/or for personalized care in the management of gout and comorbidities.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2019-eular.8097

Table 2. Clustering of Patients with Gout Based on Comorbidities

Characteristics	C1 ($n = 1257504$)	C2 ($n = 3455594$)	C3 ($n = 1199819$)	C4 ($n = 1430528$)	C5 ($n = 1263692$)
Demographics					
Age (Years)	68.9 (0.9)	58.5 (0.8)	62.2 (1.0)	56.2 (1.2)	66.9 (0.9)
Male gender (%)	831369 (66%)	2342105 (67%)	868002 (72%)	986610 (69%)	815468 (64%)
Body Mass Index (kg/m ²)	31.6 (0.5)	30.7 (0.3)	34.5 (0.9)	33.1 (0.8)	32.2 (0.6)
Comorbidities					
Obesity	644864 (51%)	1634488 (47%)	841074 (70%)	857774 (60%)	608068 (48%)
Hypertension	990667 (79%)	2098496 (61%)	919843 (77%)	1169545 (82%)	1101979 (87%)
Diabetes	519609 (41%)	0 (0%)	1199819 (100%)	562870 (39%)	708811 (56%)
Hypercholesterolemia	766072 (61%)	1880096 (54%)	767170 (64%)	1003052 (70%)	783789 (62%)
Hypertriglyceridemia	6584 (0.5%)	9440 (0.3%)	54026 (5%)	411456 (29%)	18876 (2%)
Coronary Heart Disease	20621 (2%)	0 (0%)	0 (0%)	66088 (5%)	940281 (74%)
Heart Failure	219615 (18%)	2434 (0.1%)	0 (0.0%)	12845 (0.9%)	741172 (59%)
Chronic Kidney Disease	1217813 (97%)	0 (0.0%)	0 (0.0%)	210085 (15%)	319608 (25%)
Liver Disease	9216 (0.7%)	80403 (2%)	33896 (3%)	1216843 (85%)	218814 (17%)

SAT0412

DOES THERAPEUTIC EDUCATION IMPROVE GOUT MANAGEMENT: THE EXPERIENCE OF LARIBOISIERE UNIVERSITY HOSPITAL PARIS-FRANCE

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Background: Gout is a frequent and curable arthritis affecting 0.9% of adults in France. Its management remains suboptimal and less than one third of patients have efficient urate-lowering treatment (ULT). However, when individualized information were given and patients engaged in their care a treat-to-target strategy permits to achieve serum urate level (SU) at target (SU < 360 µmol/L) in more than 95% of patients. Therapeutic education sessions (TES) may improve patient knowledges, gout flare and ULT management.

Objectives: To assess the percentage of patients achieving SU target at 1 year after a TES.

Methods: TES was set up in the department since January 2014. Patients willing to participate to TES responded to a questionnaire and were interviewed with a nurse assessing their knowledges, believes and gout representations. Each TES included 5 to 8 patients and was conducted according to participants' needs. We retrospectively included all patients who had attended one TES between 1st January 2014 and 31st December 2017 and who had at least one visit between 9 and 15 months later.

TES patients were matched in 1:1 with no-TES patients regarding age, sex and referent practitioner.

For all patients were collected: demographics, disease duration, SU, treatments and comorbidities (type 2 diabetes, chronic kidney disease, hypertension, and cardiovascular diseases).

Results: Overall, 54 TES patients were included and matched with 54 non-TES patients. Patients' characteristics (demographics, disease duration, SU, treatments and comorbidities) were similar at baseline except for the body mass index (BMI) and the follow-up. BMI was higher (31.0 ± 5.7 vs 28.4 ± 5.0 kg/m², p=0.03) and the final visit shorter (11 ± 3.6 vs 14.4 ± 4.8 months, p=0.001) in no-TES patients than in TES patients, respectively.

At final visit, 36 TES patients (67%) and 34 no-TES patients (63%) reached the SU target (< 360 µmol/L) (p=0.84) (Table 1). Moreover, 22 (41%) TES patients and 18 (33%) no-TES patients had a final SU < 300 µmol/L (p=0.43). There were no differences between groups (TES vs no-TES patients respectively) for: final SU (342 µmol/L ± 94 vs 338 ±

71, p=0.84), ULT (89% vs 96%, p=0.16), ULT dosage and hyperuricemic therapy (15% vs 11%, p=0.77).

Conclusion: The percentage of patients achieving SU target after one single TES was high compare to literature data. However, it was not different from usual care achieved in our department, a tertiary care center expert in gout management. The diffusion of our *savoir-faire* and the TES will improve gout management.

REFERENCE

- [1] Doherty M, Jenkins W, Richardson H, Sarmanova A, Abhishek A, Ashton D, et al. Efficacy and cost-effectiveness of nurse-led care involving education and engagement of patients and a treat-to-target urate-lowering strategy versus usual care for gout: a randomised controlled trial. *The Lancet*. 2018;392(10156):1403–1412.

Disclosure of Interests: Omar Al Tabaa: None declared, Etienne Gaix-Fontaine: None declared, Julia Herrou: None declared, Frederic Lioté Grant/research support from: institutional grants from Grunenthal, Ipsen Pharma/Menarini, Novartis, SOBI for the European Crystal Network Workshops, Consultant for: Grunenthal, Novartis, Thomas Bardin Consultant for: Astrazeneca, Grunenthal, Horizon, Novartis, Speakers bureau: Astella, AstraZeneca, Grunenthal., Pascal Richette Consultant for: Grunenthal, Horizon, Speakers bureau: AstraZeneca, Grunenthal, Aline Frazier: None declared, Hang Kong Ea: None declared

DOI: 10.1136/annrheumdis-2019-eular.4032

SAT0413

SIZE MATTERS – EXTENT OF MONOSODIUM URATE DEPOSITS BUT NOT SERUM URATE LEVEL PREDICTS COMPLETE RESOLUTION OF MONOSODIUM URATE CRYSTAL DEPOSITS IN PATIENTS WITH GOUT

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Background: Gout is based on the deposition of monosodium urate (MSU) crystals. While it is well established that life-style intervention and/or conventional urate lowering therapy can lead to a decrease in serum urate levels, the impact of such interventions to resolve already existing MSU deposits is very limited.

Objectives: In this study we wanted to determine if and to what extent MSU deposits resolve if patients follow structured life-style intervention or continuously conventional urate lowering therapy.

Methods: Subjects with diagnosis of gout and the presence of MSU deposits in the feet in baseline dual energy CT (DECT) scan, received either life-style intervention only (N=24) or additional conventional urate lowering therapy (allopurinol: N=29, febuxostat: N=22, benzbromarone N=2) for a mean period of 18 months before receiving a follow-up DECT examination. MSU deposits were quantified by volumetric measurement and semi quantitative scoring at baseline and follow up.

Results: Serum uric acid (SUA) level decreased from 7.2±1.7 to 6.7 ±1.7mg/dl with life-style intervention; from 7.0±1.5 to 5.5±1.8mg/dl with allopurinol and from 7.8±3.0 to 5.1 ± 2.5mg/dl with febuxostat. MSU volume significantly decreased in patients undergoing life-style intervention (baseline: 0.07cm³ ± 0.09; follow up: 0.05 ± 0.15cm³; p=0.007), treatment with allopurinol (0.11 ± 0.15 cm³ to follow up: 0.02 ± 0.04 cm³, p<0.001) or febuxostat (MSU volume baseline: 0.99 ± 2.8 to follow up: 0.64 ± 2.09; p=0.001). With respect to conversion from a DECT+ into a DECT- state, baseline MSU deposit burden, but not baseline SUA level or decrease in SUA level, was associated with reaching a DECT- state.

Table 1. Patient's characteristics for gout at the end of the follow-up

	Baseline		End of follow-up		p value
	TES	No-TES	TES	No-TES	
Patient with serum urate < 360 µmol/L, n (%)	0 (0%)	0 (0%)	36 (67%)	34 (63%)	0,84
Patient with serum urate < 300 µmol/L, n (%)	0 (0%)	0 (0%)	22 (41%)	18 (33%)	0,43
Serum urate concentration (µmol/L), mean (SD)	533 (105)	528 (97)	342 (94)	338 (71)	0,84
Taking ULT	21 (39%)	19 (35%)	48 (89%)	48 (96%)	0,16
Allopurinol, n (%)	16 (30%)	12 (22%)	22 (41%)	22 (44%)	0,89
Febuxostat, n (%)	5 (9%)	7 (13%)	26 (48%)	26 (52%)	0,84
ULT dose					
Allopurinol (mg/day), mean (SD)	196 (169)	183 (41)	284 (102)	241 (76)	0,22
Febuxostat (mg/day), mean (SD)	100 (30)	86 (10)	92 (26)	83 (13)	0,32
Taking hyperuricemia therapy, n (%)	13 (24%)	16 (30%)	8 (15%)	6 (11%)	0,77
Aspirine, n (%)	6 (11%)	8 (15%)	2 (4%)	2 (4%)	1
Thiazide diuretics, n (%)	5 (9%)	7 (13%)	6 (11%)	3 (6%)	0,49
Furosemide, n (%)	6 (11%)	7 (13%)	2 (4%)	2 (4%)	1

ULT = urate-lowering therapy