Abstract AB0865 Table 1. Clinical & management of gout in inpatients for CVDs.

	Gout (n=40)
Prior diagnosis at records	14 (35.0)
-No	20 (50.0)
-Clinical diagnosis	6 (15.0)
-Proven by crystals	
Serum urate levels (mg/dl) at admission, mean (SD)	7.1 (±2.6)
Median serum urate levels in the previous 5 years (mg/dl), mean (SD)	6.8 (±1.6)
Current serum urate <6 mg/dl	8 (33.3)
Years since the first flare, median (IQR)	15 (10-30)
Number of flares, median (IQR)	3 (1-9)
Number of involved joints, median (IQR)	2 (1-3)
Presence of tophi	3 (7.7)
Urate-lowering agents at some point	28 (70.0)
Current use of urate-lowering agents	20 (50.0)
Use of prophylactic colchicine	5 (23.8)

Data shown as n (%), unless otherwise specified.

Conclusion: Gout is prevalent in hospitalised patients for CVDs, affecting one in seven cases. The management of gout in these patients appears suboptimal, with serum uric acid levels above recommended targets and a significant percentage of no urate-lowering treatment.

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AB0866

METABOLIC SYNDROME IN PATIENTS WITH GOUT ATTENDED IN A SPECIALIZED OUTPATIENT UNIT IN SPAIN. COMPARISON WITH GENERAL POPULATION AND CARDIOVASCULAR IMPLICATIONS

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Background: Metabolic syndrome (MS) is a cluster of interrelated components: central adiposity or higher waist circumference (WC), high values of triglycerides (TG), elevated blood pressure (BP), impaired fasting glucose and decreased HDL-cholesterol (HDL-c). It is associated with a higher incidence of developing diabetes (DM), as well as with other cardiovascular diseases (CVD). A direct relationship between serum uric acid (sUA) and the risk of develop MS has been reported in several studies of patients with hyperuricemia and gout.

Objectives: To study the prevalence of MS and associated CVD in patients with gout attended in a specialized outpatient unit, comparing with other local and national studies.

Methods: Retrospective observational study with consecutive patients diagnosed with gout according to EULAR/ACR criteria between August and December 2018. We analyzed the presence of MS according to the 2015 International Diabetes Federation criteria (central obesity as BMI ≥ 30 kg/m² or WC ≥ 94 cm (≥ 80 in women), with ≥ 2 of the following: TG ≥ 1.71 mmol/l, HDL-c < 1.04 mmol/l (< 1.3 mmol/l in women), hyperglycemia ≥ 5.55 mmol/l (or T2DM previously diagnosed or hypoglycaemic treatment), hypertension (HT; $\geq 130/85$ mmHg or use of antihypertensive drugs). Treatments received and family history (FH) of gout and CVD were recorded. Demographic, anthropometric, clinical and analytical variables were analyzed. All data were compared with those obtained in three Spanish studies of the general population: DARIOS, ENRICA, SIMETAP.

Results: 57 patients with gout were included. Average age 62 years (39-90), 48 years (18-90) at the time of gout onset. 94.7% males. 40.3% with FH of gout, tophi in 52.6%. Clinical pattern: monoarticular 15.8%, oligoarticular 56.1%, polyarticular 28.1%. 68.4% received urate-lowering therapy: 45.6% Allopurinol, 22.8% Febuxostat. sUA at the time of inclusion 6.7 mg/dl (2.6-11.3). Total cholesterol 186 mg/dl (4.84 mmol/l); 96-350 mg/dl/2.5-9.1 mmol/l). TG 185 mg/dl (2.11 mmol/l); 68-742 mg/dl/0.78-8.46 mmol/l). BMI 31.8 kg/m² (24-58.1). MS was found in 66.7% and pre-morbid metabolic syndrome (pMS; excluding patients with previously diagnosed CVD or DM) in 26.3%. Obesity 57.9% and overweight 38.6%. HT 80.7%; 10.5% diagnosed in our unit. DM 22.8%. Dyslipidemia (DL: hypercholesterolemia or high levels of TG) 64.9%; 14% diagnosed in our unit. 29.8% smokers, 49.1% formers. Alcohol consumers 54.4%. Chronic kidney disease (CKD) 42.1%; 12.3% diagnosed in our unit. Ischemic heart disease (IHD) 14%. Stroke 24.6%. FH of CVD 24.6%. Comparing with

the general population the prevalence of MS and pMS was higher (DARIOS: 31% and 24%, respectively; ENRICA: 22.7% and 16.9%; SIMETAP: 41% and 25%), as well as with the presence of CKD (11.5% in SIMETAP) and CVD (SIMETAP: HT 38%, T2DM 16%, obesity 28%, IHD 4.8% and stroke 3.8%).

Conclusion: There is a very significant percentage of MS and pMS in patients with gout compared to the general population, with important presence of CKD and CVD, sometimes underdiagnosed. Nursing guidelines were established with healthy lifestyle and periodic controls directed at all patients, with special emphasis on those newly diagnosed of HT or DL, as well as those of higher cardiovascular risk.

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AB0867

DEPRESSIVE SYMPTOMS INFLUENCE SUCCESS OF ALLOPURINOL AS URATE LOWERING THERAPY

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Background: Elevated levels of serum urate (sUA) is the known precursor of gout and urate lowering therapies (ULT) are the backbone of successful gout treatment. However, less is known about factors that influence the effectiveness of ULTs. Depression has shown strong relationships with treatment noncompliance, which may translate into reduced effectiveness of treatment with allopurinol. Little is known about any effects depression may produce on the observed serum urate lowering efficacy of ULTs (e.g.; allopurinol).

Objectives: To evaluate the role of depressive symptoms in the efficacy of allopurinol for lowering sUA in the context of a clinical trial.

Methods: Within a larger cross-over clinical trial of 300 daily mg of allopurinol vs. placebo for urate lowering and its effect in ambulatory blood pressure (1), 67 patients had complete data for depressive symptoms at the beginning of each treatment period, as well as sUA before and after a 4-week treatment period with allopurinol and a 4-week placebo period (order of conditions was randomized). Depressive symptoms were assessed with the Center for Epidemiologic Studies Depression Scale-10 (CESD-10) (2). Paired samples t-test evaluated change in sUA from pre- to post-treatment during active treatment and placebo. Then, linear regressions predicted change in sUA over each treatment period from pre-treatment depressive symptoms, adjusting for sex and race which were associated with baseline sUA levels.

Results: The 67 patients had average age 27.01 years (SD=6.5, range 18-40), 39% were African-American, and 64% were males. Over the 4-week active treatment period, sUA levels decreased from average 5.8 mg/dL (SD=1.2) (345.7 μ mol/L) to 4.4 mg/dL (SD=1.2) (261.7 μ mol/L), $p < .001$. However, sUA did not change during the 4-week placebo condition (both 5.8 mg/dL (345.7 μ mol/L), SD=1.1 and 1.3, $p = 0.71$). Pre-treatment depressive symptoms ranged from "no symptoms" (0) to "severe symptoms" (16 or 20), with mean in the "no to mild" range (M=4.6, SD=4.1 before active treatment; M=6.1, SD=4.8 before placebo). After adjusting for pre-treatment sUA, sex and race, pre-treatment depressive scores predicted higher levels of sUA at the end of the active treatment period ($b = .07$, $\beta = .25$, $p = .028$) but not at the end of the placebo period ($b = .03$, $\beta = .11$, $p = .102$). After 4 weeks of allopurinol, the estimated difference in sUA between individuals with pre-treatment depressive scores of 0 vs. 16 was 1.12 mg/dL (66.6 μ mol/L), which was similar to the average treatment effect of 1.4 mg/dL (88.3 μ mol/L).

Conclusion: Even in the absence of clinical diagnosis of depression, depressive symptoms are associated with reduced efficacy of urate-

lowering with allopurinol. This could have important implications for gout treatment, as it would suggest that screening for depressive symptoms might be indicated for treatment success. Additional analyses will address whether this effect can be explained by treatment noncompliance.

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AB0868

ULTRASOUND MONOSODIUM URATE CRYSTAL DEPOSITS IN THE JOINTS OF GOUT PATIENTS CORRELATE WITH THE WORSENING OF THE HEART SYSTOLIC FUNCTION

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Background: The connection between gout and cardiovascular (CV) complications has been investigated a lot. Recent studies demonstrate that higher body urate load is an indicator of increased CV risk.

Objectives: To determine whether ultrasound deposits of monosodium urate (MSU) crystals in the joints of gout patients (pts) correlate with the worsening of the heart systolic function and left ventricular hypertrophy.

Methods: This was a single-center cross-sectional study including 56 consecutive gout pts, 40 males and 16 females, in a mean age 58.9±13.2 years with disease duration 6.42±6.93 years. All of them underwent transthoracic echocardiography and ultrasound examination of the joints of the hands, elbows, knees, ankles and feet. By transthoracic echocardiography, conducted with 2.5 MHz transducer phased array working with pulse Doppler frequency of 2.5 MHz, were measured parameters which are independent predictors of CV risk: left ventricular mass index (LVMI), ejection fraction (EF), fractional shortening (FS) and systolic motion of the myocardium (Sm). Ultrasound studies of the joints were performed with a high-frequency, linear transducer, 4-15 MHz. The existence of double contour sign, tendon MSU deposits, snow storm, tophi and tophi with erosions or a combination of these ultrasound features was assessed. Examinations were done in accordance with the latest published guidelines. Data were analyzed by Kolmogorov-Smirnov, Mann-Whitney, t-test, Kruskal Wallis test and Spearman correlation.

Results: 49(87.5%) of the pts had ultrasound evidence of crystal deposits. Serum uric acid levels were equal in pts with and without MSU deposits in the joints, (p=0.569). Among individuals without ultrasound MSU deposits, pts with MSU findings in one joint area and in those with crystal deposits in two or more joint areas we did not find a difference in the mean values of LVMI (mean±SD; 126.86±21.95 g/m² vs 120.09±31.50 g/m² vs 128.68±35.19 g/m², p=0.761), FS (mean±SD; 37.73±4.62% vs 38.25±3.51% vs 35.40±6.57%, p=0.205) and Sm (mean±SD; 0.27±0.35 m/s vs 0.11±0.02 m/s vs 0.10±0.02 m/s, p=0.266). A tendency of lower EF without reaching significance was detected in subjects with crystal deposits in two or more joints, (p=0.071). The values of LVMI (p=0.295), EF (p=0.396), FS (p=0.566) and Sm (p=0.154) were similar in pts without MSU findings, subjects who had a double contour sign, those with tendon MSU deposits, pts with snow storm, individuals with tophi and in pts with tophi and erosions. However, pts with crystal deposits in the knees and in the tibiotalar joints had lower EF (p=0.047; p=0.049, respectively) and lower FS (p=0.036; p=0.016, respectively) compared to those who had no crystal deposits in these joint areas. Further a weak negative correlation was detected between the number of joints with crystal deposits and EF (r= -0.294, p=0.029) and FS (r= -0.292, p=0.030). Regarding LVMI and Sm no correlation was established with the presence of crystal deposits in the joints (r=0.079, p=0.568 and r= -0.242, p=0.076).

Conclusion: Despite the lack of difference in serum uric acid between pts with and without MSU deposits in the joints, those with MSU findings

in the knees and in the tibiotalar joints had more deteriorated systolic function of the heart in comparison to pts without crystal deposits in these joint areas. A negative correlation existed between the number of joints with crystal deposits and functional pumping indices of the heart. On the other hand, MSU deposits in the knees and in the tibiotalar joints are associated with higher urate load than deposits in the small joints. We suggest that the higher urate load is connected to the worsened pumping function of the heart.

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AB0869

SERUM LEVELS OF ROS PRODUCTS, NO AND ASCORBATE RADICALS IN GOUT PATIENTS ARE NOT ASSOCIATED WITH ARTERIOSCLEROTIC COMMON CAROTID ARTERIES CHANGES AND USE OF XANTHINE OXIDASE INHIBITORS

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Background: Oxidative stress along with chronic inflammation and hyperuricemia links gout to arteriosclerotic vascular changes. Studies examining the connection between the level of oxidative stress and arteriosclerotic carotid arteries changes in gout patients (pts) are not enough.

Objectives: To establish the association between the serum levels of reactive oxygen species (ROS) products, nitric oxide (NO) radicals and ascorbate radicals with intima-media thickness (IMT) and common carotid artery resistive index (CCARI) in gout pts, and to find out whether the connection is more pronounced when tophi are present.

Methods: This was a cross-sectional study including 71 gout pts in a mean age 56.86±11.95 years, 61 males and 10 females (45 without tophi pts and 26 gouty tophi pts). Serum levels of ROS products, NO radicals and ascorbate radicals were determined by ex vivo electron paramagnetic resonance (EPR) study. All EPR measurements were performed on an X-band EMXmicro, spectrometer Bruker, Germany, equipped with Standard Resonator. Spectral processing was done by using Bruker WIN-EPR and Sinfonia software. By applying ultrasound of the common carotid arteries, conducted with 10 MHz linear transducer working with pulse Doppler frequency of 5 MHz, were measured IMT and CCARI. Statistical analyses were done by One-Sample Kolmogorov-Smirnov, Chi-Square, Fisher's exact test, Mann-Whitney, t-test and Pearson correlation.

Results: Gouty arthritis without tophi and gouty tophi pts were age-matched, (p=0.309). The mean values of serum uric acid (p=0.569) and distribution of the subjects with gout attack (p=0.173), smoking (p=0.828), arterial hypertension (p=0.735), dyslipidemia (p=0.646), chronic renal failure (p=0.233) and obesity (p=0.623) was equal in the groups. In the tophi group CRP and the number of pts who had suffered a cardiovascular event were higher (p=0.048; p=0.031). Serum levels of ROS products, NO radicals and ascorbate radicals were comparable in gouty arthritis without tophi and in gouty tophi pts (p=0.783; p=0.521; p=0.651). In the groups no difference was registered in CCARI (p=0.273) but intima-media was thicker in the presence of tophi, (p=0.027). No correlation existed between ROS products, NO radicals and ascorbate radicals with IMT (r= -0.100, p=0.405; r= -0.186, p=0.121; r=0.154, p=0.201). ROS products, NO radicals and ascorbate radicals did not correlate with CCARI (r= -0.110, p=0.359; r= -0.066, p=0.587; r=0.094, p=0.436). Among treated and untreated with Allopurinol pts no difference was found in the mean values of ROS products (p=0.169), NO radicals (p=0.167), ascorbate radicals (p=0.460), IMT (p=0.873) and CCARI (p=0.930). Among treated and untreated with Febuxostat pts the mean values of ROS products (p=0.546), ascorbate radicals (p=0.309), IMT (p=0.842) and CCARI (p=0.100) were similar. A tendency of higher serum NO radicals was established in pts taking Febuxostat in comparison to those not treated with it (p=0.076).

Conclusion: Although between the earlier and the later stage of the disease there was no difference in the level of oxidative stress, the level of chronic inflammation was higher in gouty tophi pts. No connection was found between serum ROS products, NO radicals and ascorbate radicals with arteriosclerotic changes of the carotid arteries and use of xanthine oxidase inhibitors. We suggest that in gout pts chronic inflammation has an important role in the process of atherogenesis.