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-