Results: Total number of patients with gout was 113. Among these, 83 patients remain under rheumatology follow up, 30 were discharged within the 1 year period. 113 were considered were audit standard 1 and 2, and 83 were considered for standard 3.

Audit standard	Expected	Achieved
Urate lowering therapy should be discussed with all patients diagnosed with Gout.	100%	100% (113/113)
Strongly advised in patients with recurrent attacks (>2 over 12 months), chronic gouty arthritis, tophi, renal impairment (eGFR <60), patients on diuretics and primary gout at an early age	90%	92.9% (105/113)
Aim of urate lowering to reduce serum urate to <300µmol/l	80%	36% (30/83)

Among the 113 patients being followed up in Rheumatology, 83% were on Allopurinol, 19 were on Febuxostat, 1 patient was on Benzbromarone. 10 patients were not on Urate lowering therapy. However, 2 among them had first attack of gout without any sign of chronicity, organ damage or tophi, so not started on ULT.

- Median Allopurinol dose was 300mg. (IQR 200mg–700mg).
- 25 patients among the 113 had tophaceous gout, and 5 of them achieved BSR treatment target
- 22/83 (26.5%) patients on Allopurinol achieved BSR treatment target of <300µmol/l. 7/19 (37%) patients on Febuxostat achieved BSR treatment target of <300µmol/l

Conclusion: It is clear that a “treat to target” approach have not been adopted for x% of the patients. Gout has been shown to have better outcomes with treat to target approach. (3)

Adequate escalation of ULT is essential to achieve target serum urate level, where as the median dose of Allopurinol here is 300mg.

Gout remains inadequately treated, even in secondary care

- A consistent “Treat to target” approach needed for treatment of gout.
- Annual gout review clinic may be an effective strategy and is now being implemented as a result of this audit.

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AB0865 THE EFFECT OF ZOLEDRONIC ACID ON CHARCOT NEUROARTHROPATHY

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Background: The Charcot foot syndrome is a serious neuroarthropathy that may threaten the functional prognosis of diabetic patient. Some studies had emphasized the beneficial effect of bisphosphonates such as pamidronate and alendronate on the reduction in bone turnover markers in this disease. Though, the use of Zoledronic acid in the treatment of Charcot foot was only evaluated in two clinical trials and its efficacy remains controversial (1, 2).

Objectives: The aim of this study was to investigate the clinical and radiological efficacy of Zoledronic acid in patients with diabetes and acute Charcot neuroarthropathy.

Methods: A prospective 2-year follow-up study was carried out on eight patient with type 2 diabetic and acute Charcot neuroarthropathy. All our patients had a physical examination and podiatry exam. The stage of the neuroarthropathy has been assessed according to the clinical and the radiographic findings (Eichenholtz Classification). All patients were assigned to intravenous infusions of 4 mg Zoledronic acid.

Results: The mean age of the study group was 61.4 +/- 4.5 years. The mean age of the onset of foot pain was 18.3 +/- 1.5 years. The midfoot involvement was bilateral in 6 patients and unilateral in the other two. A

biological inflammatory syndrome was found in 3 patients with a mean erythrocyte sedimentation rate (ESR) level at 43 mm after one hour. Foot X-rays were abnormal at all cases. The clinical outcomes at one month after Zoledronic acid infusions were the complete disappearance of acute symptoms in 6 patients and the improvement of the pain in one patient with a decreased Visual Analogue Scale (VAS) for pain by 30%. One patient had a persistence of the pain (VAS for pain at 5mm). A radiological Control was performed after 6 months for 5 patients. All of them had bone consolidation and remineralization.

Conclusion: Our study showed that the Zoledronic acid, given as a single dose, had a beneficial effect on clinical and radiological features in diabetic patients with acute Charcot neuroarthropathy. Despite our encouraging outcomes, further prospective and randomized studies are needed to confirm our preliminary results.

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AB0865 GOUT IN HOSPITALISED PATIENTS FOR CARDIOVASCULAR DISEASES: PREVALENCE AND MANAGEMENT STATUS

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Background: Gout is an independent risk factor for cardiovascular diseases (CVDs), where the urate crystal-led inflammatory state likely has a key role. Crystal dissolution may carry with cardiovascular benefits, so optimal management of gout patients is essential, especially in high-risk individuals, such as those hospitalised for CVDs. Although the prevalence of gout in Western country adults is about 2-4% [1], the rate and characteristics in inpatients with CVDs remains to be defined.

Objectives: To determine the prevalence of gout, characteristics and management in a hospitalised population for CVDs.

Methods: Observational, descriptive, cross-sectional study. Patients admitted for CVDs in the Cardiology, Neurology and Vascular Surgery units of a tertiary centre were recruited following a non-consecutive, systematic sampling up to reaching the estimated minimum sample size. A face-to-face interview and a review of electronic health records were performed, in order to collect clinical, laboratory and management data regarding CVD and gout. Gout diagnosis was established in the interview using ACR/EULAR 2015 criteria. In addition, prior clinical or crystal-proven diagnoses at records were also registered. 95% confidence intervals (95%CI) were calculated for primary variable (gout prevalence), and comparisons were performed by Student's t, chi-squared and Fisher's exact tests.

Results: 299 patients were interviewed, 33 were excluded, and the final study sample was 266 participants. They were predominantly males (69.9%) and Caucasians (96.6%) with a mean age of 68 years (SD±12). The CVDs leading to admission were acute coronary syndrome (18.8%; n=50), heart failure (13.2%; n=35), stroke or transient ischemic attack (20.7%; n=55) and peripheral artery disease (47.4%; n=126).

Gout was identified in 40 individuals (prevalence 15.0%; 95%CI 10.9-19.2). Prior gout diagnosis in records was found in only two-thirds of them, mostly clinical (Table).

Patients with gout were older (72±9 vs 68±13 years, p=0.026) and showed higher rates of chronic kidney disease (55.0% vs 23.0%, p<0.001) and use of diuretics (55.0% vs 38.5%, p=0.05), with no differences in other variables.

The disease was long-standing though low numbers of flares and involved joints were referred (Table). Tophi were seen in about 8%. The serum urate levels were not properly controlled, both at the time of the CVD and as a median of the previous five years, and only one-third of the patients were on target (<6 mg/dl). Despite 70% of patients having been treated with urate-lowering agents at some point, at admission only half remained treated (38.5% of them were on target). Nearly a quarter of patients used prophylactic colchicine.

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 premorbid 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-