study period and of these, random selection of 10% i.e. 250 subjects were considered for the study. Baseline demographics, medical comorbidities, serum uric acid level, medication history, and information whether they were followed by rheumatologist or primary care physician (PCP) were extracted from electronic health record.

We used Stat view Version 5.01 (SAS Institute Inc. Cary, NC) for analysis. We described data with frequency terms, continuous data by mean $\pm$ standard deviation, and categorical data by percent. Univariate analysis identified predictors of interest that were later incorporated in the best fit model with logistic regression. A p value of ≤ 0.05 was considered statistically significant.

Results: The mean age of subjects was 61 $\pm$ 11 years, mean BMI was 32 $\pm$ 7 kg/m², 98% were males and 80% were African Americans. 26% of subjects had history of alcohol use, 89% had hypertension and 88% had chronic kidney disease (CKD stage ≥ 2). 86% of the subjects were followed by primary care physician (PCP), and 5% of them were followed by rheumatology and rest of the 9% were non-compliant. 30% of subjects were receiving urate-lowering therapy and 23% of patients were on gout prophylactic therapy. 21% of patients had multiple (≥ 2) visits to the ED. The mean uric acid level was 8.5 $\pm$ 2.1 mg/dl for subjects with single visit compared to 9.04 $\pm$ 2.1 mg/dl for multiple visits to the ED (P = 0.09).

In the univariate analysis, CKD (stage ≥ 2) and higher uric acid level were associated with increased ED visits (P = 0.09) and not being on urate lowering therapy was also associated with increased frequency of ED visits (P = 0.02). On logistic regression analysis, irrespective of the type of physician follow up (PCP vs rheumatologists), being on urate-lowering therapy was associated with reduced frequency of ED visits. (P=0.02).

Conclusion: Urate-lowering therapy (ULT) was associated with reduced ED visit irrespective of follow up care provided by PCP or rheumatologist. Given that only one third of our patients were on ULT, improving ULT dispensing by the physician and patient compliance with ULT can decrease health care utilization.

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AB0881

DESCRIPTIVE ANALYSIS OF PATIENTS WITH OSTEOGENESIS IMPERFECTA IN A TERTIARY HOSPITAL IN MADRID

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Background: Osteogenesis imperfecta (OI) is an inherited connective tissue disorder with an incidence of 1 per 20,000 births. It is also called "brittle bone disease" and is most commonly caused by mutations in genes encoding type I collagen or proteins involved in its posttranslational modification. Most patients have an autosomal dominant mutation in *COL1A1* or *COL1A2*. The severity of the clinical presentation depends upon the effect of the mutation that occurs, having many different phenotypic presentations. In the most severe forms, patients suffer multiple fractures with minimal or no trauma whereas mild forms may only manifest with premature osteoporosis. Attending to the clinical presentation, radiographic findings and the mutation that occurs, 11 different types have been described, with type I and III being the most prevalent. Many treatments have been studied, but none has been found to be curative. The most frequently used are bisphosphonates which try to prevent bone fragility and reduce the number of fractures but none have been approved specifically for use in either children or adults with OI.

Objectives: The aim of the study is to analyse the clinical characteristics of osteogenesis imperfecta (OI) patients followed in our hospital and to evaluate the different treatments used in their management.

Methods: A retrospective study was conducted. All patients diagnosed with OI and seen in the different departments of our hospital were included and analyzed. A database was created, including both clinical and epidemiological data and a descriptive analysis was carried out.

Results: 25 patients with OI are currently being followed up in our hospital and were included. Although most patients were being followed in both the Rheumatology (9) and Orthopedic units (9), 4 were being followed by pediatrics, 1 by endocrinology, 1 by internal medicine and 1 by

geriatrics. 72% were female (18) with a mean age at diagnosis of 17 years (range: 1 month to 67 years). All of them had had previous fractures before the diagnosis. The number of fractures during their follow-up varied according to the different types of OI, with an average of 6 fractures (range 3-24) per patient and an average of at least 4.16 orthopedic surgeries each. 12/25 patients were diagnosed in the first ten years of life, being the ones that accumulated the highest number of fractures (96 vs. 54). Only 3 patients had family background of OI, all of them being type I. Although only 9/25 patients had undergone genetic study, all 3 cases of type III, which is the most severe, debuted in the first decade of life. Phenotypically 14/25 (56%) had short stature and 18/25 (72%) had blue sclerae, being these less frequent in those patients with debut after 20 years of age, of which 57% (4/7) had normal sclerotics. Only 4 patients suffered from dentinogenesis imperfecta (16%) and 3 from otosclerosis and had hearing problems (12%). Regarding the treatment received, 60% of the patients (15/25) were on current treatment with oral calcium and 64% (16/25) with oral vitamin D supplements. On the other hand only 60% of the patients received bisphosphonates (4 were being treated with risedronate, 7 with pamidronate, and 4 had received both zoledronic and pamidronate during their lives).

Conclusion: Although a rare disease, OI has an important morbimortality in most patients. Severe cases suffer multiple fractures and undergo several orthopedic surgeries during their lives. Given the high cost of genetic analysis, this is reserved for the most severe cases which tend to debut at younger ages and are mostly type III. Treatment for this condition is not standardized and is generally reserved for type III OI patients, which is one of the most severe types. Bisphosphonates, calcium and vitamin D are usually used in order to try to prevent new fragility fractures but in most cases fracture rates remain high despite treatment.

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AB0882

ACROMEGALY DO NOT INCREASE THE RISK OF VERTEBRAL FRACTURES : A RETROSPECTIVE AND PROSPECTIVE STUDY ON 50 PATIENTS

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Background: Patients with acromegaly appear to be at an increased risk of vertebral fractures (VFs) despite normal bone mineral density (BMD)¹. However, these patients could have several endocrine deficits as hypogonadism known to increase the fracture risk independently of the GH effects. The physiopathology of GH excess on bone is unclear. In addition, patients with acromegaly have radiological deformations of the spine, called Erdheim's syndrome, which can overestimate the radiological vertebral fractures.

Objectives: Investigate the prevalence of VFs in our cohort of patients with acromegaly.

Methods: It was a monocentric, retrospective and prospective study. The rheumatologic evaluation was less than 3 years for all patients. For 40% of patients, this evaluation was prospective after the begin of the study. Acromegaly patients younger than 80 and followed at the Nantes University hospital in January 2018 were included. Patients were excluded if they had a rheumatologic or endocrinologic disease interfering with the results. The prevalence of radiological vertebral fractures was evaluated on conventional lumbar and thoracic spine radiographs using Genant's semi-quantitative assessment. We also assessed qualitative abnormalities of the spine using 3 criteria : osteophytes, disc space narrowing and cuneiform aspect of vertebrae. The X-rays were read by two rheumatologists independently. We analyzed BMD at lumbar spine and total hip, endocrine status and quality of life through 3 questionnaires (AcroQoL, specific of acromegaly; Oswestry evaluating the functional impact of pain; HAQ evaluating the functional capacity).

Results: We included 56 patients. 6 patients were excluded : 3 declined, 1 had bone metastasis and 2 had another endocrinologic disease (adrenocortical and panhypopituitarism). We analyzed the prevalence of VFs in 50 patients (19 females, 31 males, median age 53, range 28-79). The average of time between the diagnosis of acromegaly and the last rheumatologic evaluation was 9.1 years. 3 patients (6.1%) had a VF : 1 grade 1 and 2 grade 2 of Genant's assessment. 28% patients were osteopenic and 12% were osteoporotic. Among fractured patients, 2 were osteoporotic and 1 osteopenic. 26% were hypogonadal (100% substituted), 16% had central adrenal insufficiency (100% substituted). 14 women were menopausal (74% of women). Thoracic spine was deformed in 31 patients (61%) and lumbar spine in 21 patients (43%), for at least

one of the 3 criteria. Patients with spine deformation were older ($p=0.043$), with higher BMI ($p=0.004$) and had a trend to be more hypogonadal ($p=0.06$). Concerning quality of life, AcroQoL's average was 70.9% (score 0 to 100, maximal quality of life =100, range 32-98), HAQ's average was 0.18 (score 0 to 3, maximal quality of life 0, range 0-1.38) and Oswestry's average was 9.8 (score 0 to 100, maximal quality of life 0, range 0-44).

Conclusion: This study shows for the first time that acromegaly patients are not at an increased risk of vertebral fractures. This result differs from the literature that reported more than 30% of VF in this population. Our study bring several points of explanation. First, the vertebral abnormalities were frequent in our patients and can overestimate the VFs without a qualitative analysis of the X-ray. Secondly, the right endocrine balance plays an important role in osteoporosis. Our patients were well supplemented, it can reduce the risk of osteoporosis. More studies are needed to confirm this new hypothesis.

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AB0883

SYSTEMIC INFLAMMATION AND ATHEROSCLEROSIS IN PATIENTS WITH GOUT. RESULTS FROM THE NOR-GOUT STUDY

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Background: The association between gout and cardiovascular disease (CVD) is well known,¹ whereas mechanisms behind this association are poorly understood.

Objectives: This study aimed to evaluate factors associated with asymptomatic carotid atherosclerosis in patients with gout.

Methods: In this prospective study patients with crystal-proven gout were included after a recent disease flare, if the serum urate level was >360 µmol/L (>6 mg/dl). We analysed baseline data in patients without established CVD who were referred to a CVD risk evaluation, including ultrasound of the carotid arteries, blood pressure measurement and laboratory tests. Carotid atherosclerotic plaques were defined in the longitudinal view as protrusions into the lumen of ≥1.5 mm or at least 2 times the adjacent intima-media thickness according to the Mannheim criteria.

Results: Of the 79 gout patients included, approximately 10% were females, and mean (SD) age was

52.1±13.1 years. Thirty-two (40.5%) had carotid plaques (Table). Only 9.3% were current smokers, while mean (SD) body mass index was high (29.1±4.7 kg/m²). Lipids were in the normal range, with a mean (SD) total cholesterol at 5.3±1.09 mmol/L and low density lipoprotein cholesterol 3.12±0.95 mmol/L. Systolic blood pressure was in the normal range 134.0±15.1 mmHg, although 29.1% of the patients were treated with anti-hypertensive agents. In univariate analyses, higher age, hypertension and higher erythrocyte sedimentation rate (as a marker of systemic inflammation) were significantly associated with the presence of carotid plaques ($p=0.01$ and $p=0.04$, respectively) (Figure). Serum urate levels or disease duration were not associated with carotid plaques ($p=0.27$ and $p=0.44$, respectively).

Conclusion: Our results indicate an association between systemic inflammation and atherosclerosis in patients with gout. To be able to efficiently prevent CVD in this patient group, prospective studies with larger sample sizes are needed to elucidate the mechanisms behind the increased risk of CVD in gout patients.

Table. Patient characteristics

Number n (%)	79 (100.0)	32 (40.5)	47 (59.5)	-
Age (years) mean±SD	52.1±13.1	60.0±10.4	46.7±12.1	
Sex male/female n (%)	72/7 (91.1)/(8.9)	28/4 (87.5)/(12.5)	44/3 (93.6)/(6.4)	0.43
Disease duration (median IQR)	6.0 (3.0-12.0)	6.0 (3.8-14.3)	5.0 (2.0-10.0)	0.44
CV risk factors				
Smoking n (%)	7 (9.3)	2 (2.7)	5 (6.7)	0.69
BMI mean±SD	29.1±4.7	28.5±4.2	29.5±5.0	0.35
TC (mmol/L) mean±SD	5.3±1.09	5.21±1.18	5.39±1.02	0.48
LDL-c (mmol/L) mean±SD	3.12±0.95	3.24±1.07	3.04±0.84	0.41
BP systolic (mm Hg) mean±SD	134.0±15.1	138.0±16.2	131.3±13.9	0.06
BP diastolic (mm Hg) mean±SD	82.8±8.0	83.6±6.9	82.3±8.8	0.46
Co-morbidities n (%)				
Hypertension	36 (45.6)	21 (26.6)	15 (19.0)	0.01
Diabetes	4 (5.1)	3 (3.8)	1 (1.3)	0.30
Biomarkers mean±SD				
S-urate (µmol/L)	495.6±84.0	481.8±97.5	504.9±73.2	0.27
ESR (mm/h)	12.9±13.9	17.3±17.6	9.8±9.7	0.04
CRP (mg/L) median (IQR)	3.5 (2.0-7.8)	5.0 (2.0-13.5)	3.0 (1.0-6.0)	0.09

Figure. Association of carotid plaques and systemic inflammation

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AB0884

CLINICAL AND DEMOGRAPHIC CHARACTERISTICS OF PATIENTS WITH OCHRONOTIC ARTHROPATHY

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Background: Alkaptonuria (AKU) is a metabolic disorder that causes accumulation of oxidized homogentisic acid (HGA) in the connective tissues. The excessive deposition of HGA and its metabolites can cause