leucocytes in synovial fluid plus presence of X-ray chondrocalcinosis in at least one joint) and controls (patients with synovial effusion shown to have no CPP crystals and no chondrocalcinosis in hands and knee X-rays), paired by age and gender. Patients with hemochromatosis or primary hyperparathyroidsm were not included. General variables were included along with plausible metabolic variables (Ca, P, Mg, iPth, iron saturation [satFe%], ferritin, diuretics and type of diuretic, and HFE genotype), and distributon of joint involvement (mono-oligo-polyarticular) and clinical manifestations (acute [A-CPPCA] and chronic inflammatory [CI-CPPCA]), as in EULAR recommendations.

Results: 340 patients and 316 controls were recruited, 53% were men, age at inclusion was 67 ± 10 year (IC range 62–75), time from onset of symptoms 5.2 ± 5.3 year (IC range 1–8). Regarding cases, A-CPPCA was present in 147 (43.2%), CI-CPPCA in 193 (56.8%), with monoarticular involvement in 102 (30.0%), oligoarticular in 176 (51.8%), and polyarticular in 62 (18.2%). Patients showed higher serum ferritine levels and lower Mg levels than controls (253 and 2.00 vs 204 and 2.08, respectively), along with higher rate of HFE gene mutations (Odds ratio 2.30, 95% CI: 1.66 to 3.20). Genotypes including homozygotic mutations of H63D allele, heterozygotic mutations for C282Y allele, and double heterozygotic mutations for C282Y and H63D were statistically associated with higher frequency of polyarticular involvement and with CI-PPA (figure 1). Clinical variables were also associated with higher SatFe% levels, but not Mg or ferritin levels. S65C gene mutations were not increased in patients compared to controls and did not show any association with clinical phenotype.
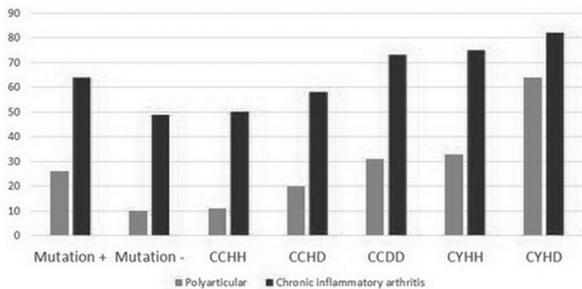
Abstract OP0183 – Table 1 Rate (%) of polyarticular and chronic inflammatory clinical pattern impact of presence of any mutation and genotypes of FHE genes

Conclusions: Patients with definite CPPCA show differences in serum Mg and Ferritin levels compared to that of controls, and may contribute, along with other factors, to development of CPPCA. Nevertheless, only presence of some HFE genotypes involving C282Y and H63D genes were associated with more severe phenotype of clinical involvement.

Disclosure of Interest: None declared

OP0184   **THE FREQUENCY OF FLARES IN SUBJECTS WITH CHRONIC REFRACTORY GOUT TREATED WITH PEGLOTICASE IS RELATED TO VARIATION IN THE LEVEL OF PLASMA URATE**

L. Calabrese[1], T. Fields[2], A. Yeo[3], P. Lipsky[4]. *[1]Cleveland Clinic, Cleveland; [2]Weill Cornell Medical College, New York; [3]Horizon Pharma, Lake Forest; [4]AMPEL BioSolutions LLC, Charlottesville, USA*

Background: Lowering plasma urate in subjects with gout is associated with an increase in flares because of an altered equilibrium between soluble urate and uric acid crystals.[1] Details of the relationship between plasma urate lowering and gout flares have not been fully delineated. Pegloticase is a mammalian recombinant uricase conjugated to polyethylene glycol approved for the treatment of chronic gout refractory to conventional oral urate lowering therapy.[2–4] Results from the two 6 month randomised clinical trials (RCTs) of pegloticase showed that during months 1–3, there was an increase in pegloticase-treated subjects experiencing gout flares vs those receiving placebo. However, the reported analysis did not separate results for subjects who did and did not respond to pegloticase with a sustained reduction in plasma urate levels or relate flares to plasma urate levels.

Objectives: To determine factors associated with the occurrence of flares in subjects who did and did not respond to pegloticase in the two RCTs for this therapy.

Methods: This analysis used results from 2 pivotal RCTs[2] of pegloticase (8 mg every 2 weeks [q2] or every 4 weeks [q4]) vs placebo in subjects with chronic refractory gout. A responder was defined as a subject with plasma urate <6.0 mg/dL≥80% of the time from the week-9 infusion to just prior to the week-13 infusion and from the week-21 infusion to the week-25 final study visit. Gout flares were defined as acute joint pain and swelling requiring treatment. Gout flare prophylaxis

(colchicine, 0.6 mg once or twice daily, or a NSAID) was initiated 1 week before the first infusion and continued throughout the study.

Results: There were increases in the frequency of subjects experiencing flares and the mean flare frequency in the pegloticase group vs placebo during the first 3 months of the trial (figure 1A). In contrast, there were marked reductions in both outcomes during months 3–6 (figure 1B). The increase in flares in the first 3 months was most evident (p=0.0006) and the decrease during the second 3 months was least marked (p=0.0006) in subjects receiving q4 pegloticase. Previous analysis indicated that q2 pegloticase caused persistently lowered plasma urate in responders, whereas q4 administration resulted in a more saw tooth pattern.[5] Multivariate linear regression analysis indicated that the only variable associated with flares in the treated subjects was the absolute change in plasma urate prior to these events. Assessment of plasma urate at both 2 weeks and immediately before flares indicated that larger differences between these two values were associated with increased flare occurrence (p=0.002).
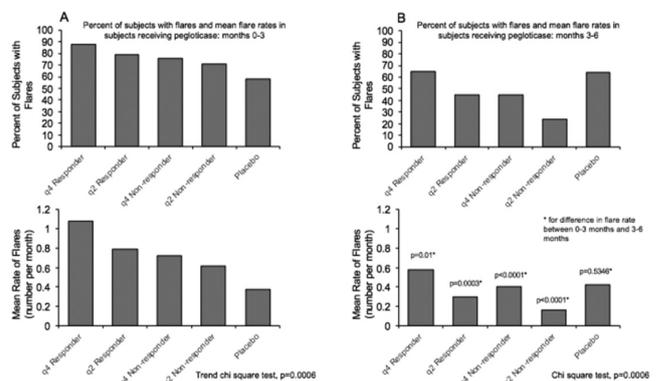
Conclusions: Flares associated with pegloticase treatment in subjects with chronic refractory gout were increased most markedly in responders to monthly administration and were significantly associated with fluctuations in plasma urate levels. Such fluctuations are much more common with monthly administration of pegloticase vs the recommended biweekly treatment regimen.

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OP0185   **EMERGENCY DEPARTMENT VISITS FOR GOUT: A DRAMATIC INCREASE IN THE PAST DECADE**

A. Mithal[1], G. Singh[2]. *[1]ICORE; [2]Stanford University, Woodside, USA*

Background: Several studies have suggested that the prevalence of gout has been increasing worldwide, perhaps related to lifestyle factors.[1–2] In the US, the NHANES study found a significantly higher age-adjusted prevalence (3.9%) in 2007–2008 than the estimate in 1988–1994 (2.9%). This trend paralleled an observed increase in hyperuricemia.[1]

Objectives: To study emergency department visits due to gout in the US over 9 years (2006 to 2014).

Methods: The Nationwide Emergency Department Sample (NEDS) is the largest all-payer emergency department (ED) database in the United States, yielding national estimates of hospital-based ED visits. It contains information from 31 million ED visits at 945 hospitals in 34 states that approximate a 20-percent stratified sample of U.S. hospital-owned EDs. Weighted, it estimates roughly 143 million ED visits. We studied all ED with primary diagnosis of Gout (ICD9 code 274.xx) from 2006–2014 and calculated prevalence in different age groups. Population data was obtained from US census bureau.

Results: Over last 9 years, 1.7 million people visited EDs with a primary diagnosis of acute gout. The total number of ED visits per year increased from 1 68 580 in 2006 to 2 13 780 in 2014, an increase of 26.8%. The prevalence of emergency room visits with primary diagnosis of gout increased from 56.5/100,000 population

in 2006 to 67/100,000 in 2014 (p<0.001). The largest increase in ED visits was a 28% increase in prevalence among the 45–64 years age group from 91/100,000 to 116/100,000 (27%, p<0.001). Men were responsible for 78% of the ED visits in both 2006 and 2014.

Age in years	Total number of ED visits in 2006	Prevalence of ED visits/100,000 population in 2006	Total number of ED visits in 2014	Prevalence of ED visits/100,000 population in 2014
18–44	49 265	44	55 216	48
45–64	68 544	91	97 081	116
65–84	44 358	137	53 206	133
85+	6242	128	8126	132
All ages	2 05 905	56.5	2 13 780	67

Conclusions: Emergency Department visits have increased dramatically in the US over the last 9 years, and this increase is mostly in the 45–64 years age group perhaps reflecting the undermanaged burden of uncontrolled gout. Education, improved recognition and long-term management as well as increased use of preventive strategies is needed.

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OP0186   **NEPHROLITHIASIS AS A COMPLICATION OF GOUT: A CROSS-SECTIONAL STUDY WITH HELICAL COMPUTED TOMOGRAPHY**

F.P. Ramos, A.T. Barbosa, R.B. Souza, F. Seragioli, R.D.N. Giorgi, R.F. Rosa. *Rheumatology, IAMSPE, São Paulo, Brazil*

Background: Gout is the most prevalent inflammatory arthritis, secondary to persistent hyperuricemia. It has been associated with development and progression of cardiometabolic diseases and chronic kidney disease. Several studies have shown a relationship between hyperuricemia and history of nephrolithiasis, although cut-off levels for uric acid and other related risk factors are still not well established.
Objectives: To determine the frequency of nephrolithiasis (NL) detected by helical computed tomography (h-CT) and its associated risk factors in patients with gout in a tertiary hospital of São Paulo, Brazil.
Methods: This cross-sectional study, conducted from 2016 to 2017, included 80 patients with a diagnosis of gout, according to the criteria of the ACR/EULAR-2015. They were questioned about the previous history of NL and submitted to h-CT for NL. Two groups were established: with and without NL; and later, unilateral and bilateral lithiasis. Anthropometric data, disease duration, serum uric acid (UA), creatinine, urinary pH(pH) and urinary UA of groups were compared. Statistical analysis included: mean, standard deviation, relative percentages, t-student test, chi-square test and ROC curve. Comparison of time to event-rate was performed by Kaplan-Meier method with log rank test. p<0.05 was considered statistically significant.
Results: NL was confirmed by h-CT in 30% of patients. However, only 16% reported previous history of NL. Groups with NL and without NL were similar in mean age (65,96±5,54 and 68,89±9,58 years, p=0.147), disease duration (16,63 ±11,49 and 11,77±9,74 years, p=0.056) and BMI (29.72±5.09 and 28.82±5,08, p=0.470). The NL group had higher pre-treatment UA compared with patients without NL (9,36±1,09 and 8,80±1,08 mg/dL, p<0.05) and the most acidic pH (5,26±0,42 and 5.74±0.62, p<0.05). In addition, patients with bilateral NL presented higher BMI than unilateral patients (p=0.036). According to ROC curve analysis, the best cutoff value for pre-treatment UA was 8.5 mg/dL, yielding sensitivity and specificity of 75% and 50%, respectively, for predicting NL events in this study. Kaplan-Meyer analysis showed that after 20 years of disease, 55% of patients with pre-treatment AU ≥8.5 mg/dL had NL, while only 18% of patients with pre-treatment AU <8.5 mg/dL.

ROC curve
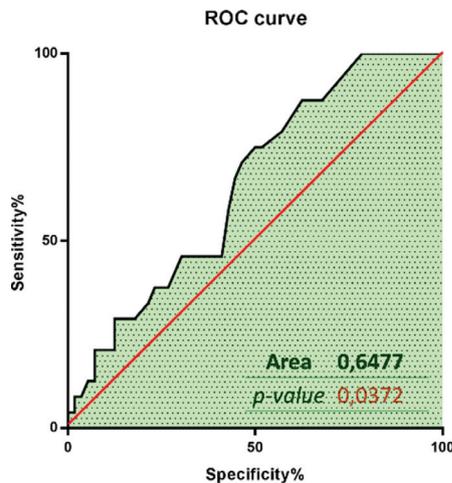
AU ≥ 8,5mg/dL		
Sensitivity	75%	95% IC: 53% - 90%
Specificity	50%	95% IC: 36% - 64%

	Without NL	With NL
AU < 8,5mg/dL	27	6 (18%)
AU ≥ 8,5mg/dL	29	18 (38%)

Conclusions: Since prevalence of NL in gout patients cannot be determined reliably from the clinical history, an active screening test for NL should be performed in these patients. Our study suggests that urine acidification and UA ≥8.5 mg/dL are associated with an increased risk of NL during follow-up of gout patients and should be corrected in their treatment.

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OP0187   **FRUCTOSE-CONTAINING BEVERAGES IS AN INDEPENDENT RISK FACTOR FOR GOUT EARLY-ONSET IN SOUTH CHINA**

Q. Li[1], S.-H. Zeng[2], X.-Y. Du[1], Y.-H. Xu[1], K.-M. Yang[1], J.-J. Liang[1], Y.-Q. Mo[1], L. Dai[1*]. *[1]Department of Rheumatology, Sun Yat-Sen Memorial Hospital, Sun Yat-Sen University; [2]Zhongshan School of Medicine, Sun Yat-Sen University, Guangzhou, China*

Background: A trend of earlier onset of gout has been reported even though its incidence increases in a linear fashion with age until 70 years. Dietary factors have been supposed to be contributed to the early onset of gout.
Objectives: To investigate diet characteristics of gout and their impact on the early onset of gout.
Methods: Consecutive gout patients who fulfilled the 2016 ACR/EULAR classification criteria were recruited between Dec 2016 and Dec 2017 A cross-section survey on dietary factors before gout onset was conducted with semi-quantitative diet questionnaire. The questionnaire included alcohol, fructose-containing beverages, soup, animal organs, sea-foods, hotpot, tea and coffee, which impact on