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 hyperparathyroidism 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 distribution 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 ferritin 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 homozygous mutations of H63D allele, heterozygous mutations for C282Y allele, and double heterozygous 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 HFE 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

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

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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 RCTs² 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 persistent 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

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Background: Several studies have suggested that the prevalence of gout has been increasing worldwide, perhaps related to lifestyle factors.¹⁻³ 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