Crystal diseases, metabolic bone diseases other than osteoporosis

AB1035 END STAGE RENAL DISEASE IN PATIENTS WITH GOUT AND CONTROLS, ETIOLOGICAL DIFFERENCES?

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Background: The association between gout and renal disease is well known. However, whether hyperuricemia causes renal disease, or if the association is explained by comorbidities, is still up for debate. Studies on the effects of urate and gout on chronic kidney disease (CKD) show conflicting results. There is a lack of data on the underlying causes of CKD in gout patients.
Objectives: To compare causes of end stage renal disease (ESRD) in patients with gout and population controls.
Methods: This is a population-based and register-based case-control study. Gout cases, aged 18 years or older, were identified by at least one visit to a physician either an in- or outpatient clinic with a registered ICD-10 code for gout (M10) in the Western Regional Healthcare Contact Register (VEGA), between January 1st 2001 through December 31st 2018. For each case five controls without gout were matched by sex, age, and county/place of residence at year of first diagnosis. Cases and controls were excluded if they prior to the first recorded ICD-10 code for gout, had a record of end stage renal disease, defined as codes for renal transplantation or dialysis either in VEGA or the Swedish Kidney Register (SNR). In this cohort, all cases and controls developing ESRD during the follow-up period (2001 to 2018) were identified, and the registered causes of ESRD were compared across the groups.
Results: In total, 48594 gout cases (mean age 68.4 years; 67% male), and their 150618 matched controls (mean age 63.3 years; 67% male) were included. Of the cases and controls, 529 (1.1%) and 128 (0.1%) developed ESRD, respectively. At the time-point of active uremia treatment start (as in start of dialysis or kidney transplantation), the cardiovascular comorbidity burden was higher for the gout patients (Table 1). There was no significant difference in the distribution of ESRD-causes between patients with gout and their controls (Figure 1).

Table 1. Baseline data

	Gout	Controls
Number (% of total)	529 (80.5)	128 (19.5)
Age at gout diagnosis, mean (median)	64.2 (65)	63.3 (65)
Age at ESRD, mean (median)	69.2 (72)	68.9 (71)
Male sex, n (%)	389 (73.5)	109 (85.2)
No renal failure at baseline, n (%)	239 (44.6)	77 (60.2)
Diabetes, n (%)	215 (40.6)	44 (34.4)
Hypertension, n (%)	473 (89.4)	110 (85.9)
Ischemic heart disease, n (%)*	206 (38.9)	26 (20.3)
Cerebrovascular disease, n (%)	89 (16.8)	15 (11.7)
Hematologic malignancy, n (%)	11 (2.1)	6 (4.7)
Dermatologic malignancy, n (%)	12 (2.3)	3 (2.3)
Other malignancy, n (%)	65 (12.3)	16 (12.5)

*p<0,001

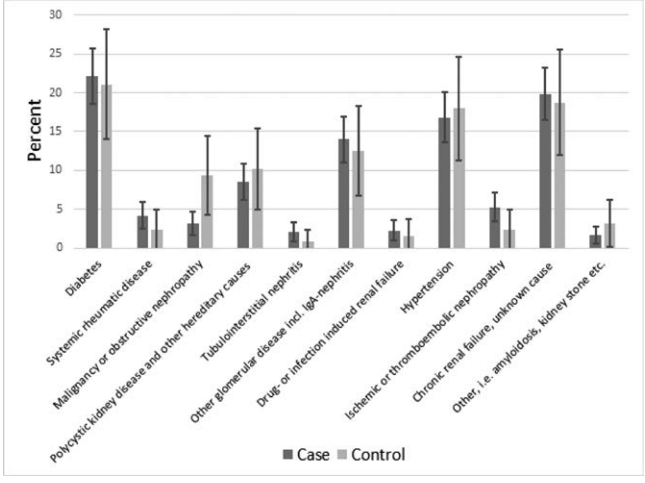

Figure 1.

Conclusion: In this study, including 48594 cases with gout, of which 529 developed ESRD, there was no apparent difference in the cause of ESRD compared to matched controls. This suggests similar pathophysiological mechanisms of ESRD development in patients with gout and the general population.
Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2022-eular.109

AB1036 IS HEMODIALYSIS ENOUGH TO CONTROL GOUT IN CHRONIC TERMINAL RENAL DEFICIENCY PATIENTS? A TEN YEARS RETROSPECTIVE STUDY

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Background: Gout is the most common inflammatory rheumatism of industrials countries and chronic renal deficiency is one of the most usual risk factor(1). Uncontrolled gout can cause articular impairment but is also associated with a global and cardiovascular excess mortality, especially in dialysis population(2). International guidelines on gout management remain currently unclear for this particular population(3,4) and current literature data are contradictory on the effectiveness of hemodialysis alone in stemming gout flares.
Objectives: The objective of this study was to analyze the rate of gout flares, during 5 years on gouty patients who starts hemodialysis, treated or not by hypouricemic drugs.
Methods: We performed an observational, retrospective and multicentric study on gouty patients who started hemodialysis between 2005 and 2015 in two nephrology unit. We recorded demographic, clinical and therapeutic data at the start of hemodialysis and throughout 5 years of follow up. A gout flare was defined as presence of uric acid crystal in articular punction when it was performed, or clinical diagnosed as such with a colchicine prescription. The effectiveness of dialysis was measured by the KT/V ratio measured during gout flares and 3 times per year for each patient correlated with the serum uric acid level. According to the French legislation, no ethics committee approval was requested for such a retrospective survey. Survival analysis by Kaplan - Meier method and bivariate analysis have been performed to study gout flares and their association with clinical or biological factor.
Results: One hundred eighty two patients have been included, 98/182 (53, 8%) had long term hypouricemic treatment: 88/98 (89%) by allopurinol and 10/98 (11%) by febuxostat. Mean age at dialysis initiation was 68.6 years with a male/female sex ratio of 2.7:1. Sixty-four/182 patients (35.16%) presented at least one gout flare during the follow-up: 42 patients (65%) without hypouricemic treatment and 22 patients (45%) with hypouricemic treatment. Patients on hypouricemic treatment since hemodialysis beginning had significantly less gout flares compared to those without (p = 0.0009) **(graphic 1).** There was no significant KT/V ratios difference between the 2 populations at the time of gout flares. Hypouricemic treatment was a protective factor for the occurrence of gout attacks (HR: 0.42, 95% CI 0.25 – 0.71) (Table 1**).**

Table 1. Associated factor of gout flares after the beginning of hemodialysis on gouty patients

	HR	95% CI
Current or active smoking	0.92	0.46-1.86
Male	0.82	1.48-1.42
Caucasian ethnic	0.83	0.64-1.75
Hypouricemic treatment	**0.42**	**0.25-0.71**
High Blood Pressure	1.21	0.44-3.36
Obesity	0.84	0.47-1.51
Dyslipidemia	0.79	0.47-1.30
Diabetes	1.28	0.78-2.11
Cardiac failure	1.42	0.73-2.81

HR: Hazard Ratio; CI: Confidence interval

Conclusion: Therefore hypouricemic treatment at the initiation of hemodialysis seems to be a protective factor against gout flares, which may persist in dialysis patients. Prospectives studies should be performed to confirm these results for this particular gouty population.
REFERENCES:
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