sections were incubated with JIA sera from children with and without uveitis (1:50 in PBT) for 1h.

Secondary antibody (goat anti-human IgG-488).DAPI counterstaining was used

Sections analysed by EE, using a Leica SP8 confocal microscope using LAS software. The operator was blinded to the status of the patient

Results: Sera from 13 patients were analysed: 5 with uveitis and 8 with no uveitis. Six had oligo JIA; 3 extended Oligo; 4 Poly JIA. Four were ANA -ve; 8 ANA+ve Max titre1/80 (using HEp2 cells); 1 unknown. Treatment: Seven NSAID; 1 MTX; 2 MTX and Pred; 3 Biologics. 5 Topical steroids

All tissue sections were analyzed blindly. Of the 5 patients with Uveitis 3 had antibodies against ciliary antigens and 2 did not. Of the 8 patients without uveitis 2 had antibodies against ciliary antigens and 6 did not. Fig 1 demonstrates immune histochemical findings from 2 typical patient's sera. No patient had high titres of antinuclear antibodies and there was no association between ANA positivity and ciliary body positivity. Fluorescent intensity was also higher in the Uveitis +ve patients Fig 2.

Fig 1 Ciliary body antibody detection in serum of patients with and without uveitis

Fig 2 Mean fluorescent intensity between Uveitis and Non Uveitis labelling

Conclusion: This preliminary work suggests that antibodies to ciliary body proteins may be valuable in identifying patients with JIA at risk of developing uveitis with 60% of those with uveitis being antibody positive compared with only 25% of those without uveitis. Concurrent treatment may alter the presence of antibodies in the serum and may also have suppressed the development of uveitis in those patients who were antibody positive yet had not developed uveitis. Antibody patterning within the ciliary body may also provide further useful information. We are currently studying a further group of patients who have not received immunosuppressive drugs

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THE ABSOLUTE RISK OF CLINICALLY DIAGNOSED GOUT BY CLUSTERS OF GOUT ASSOCIATED COMORBIDITIES AND LIFESTYLE FACTORS – RESULTS FROM 30 YEARS FOLLOW-UP OF THE MALMÖ PREVENTIVE PROJECT COHORT IN SOUTHERN SWEDEN

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Background: Clinical gout is predicted by a number of factors. Several of these (comorbidities and lifestyle) have been shown to cluster in gout

patients^{1,2} indicating several different pathophysiological pathways, but it is not known if and to what extent such clusters predict gout.

Objectives: To examine; 1) the prevalence of comorbidity clusters in the population and 2) the long-term absolute and relative risks for developing gout in these clusters defined at baseline in subjects of Malmö Preventive Project (MPP).

Methods: The MPP is a screening program for cardiovascular risk factors, alcohol abuse and breast cancer in Malmö, Sweden. A total of 33,346 individuals (67% male, age=45.7 years, mean follow-up=28.2 years), screened between 1974 and 1992, participated. Date of gout diagnosis was defined as the first visit with gout (using ICD-codes) to physicians within primary or specialized care, through linkage of the MPP cohort with regional and national health care registers. End of follow-up was defined as the date of first gout diagnosis, death, moving from the area or December 31st, 2014. Hierarchical clustering was performed in a subset of 22,057 individuals (screening period: 1975-1992) to group observations. Variables clustered included obesity, renal dysfunction, diabetes mellitus (DM), hypertension, cardiovascular disease (CVD), dyslipidemia, pulmonary dysfunction (PD), smoking and use of diuretics. For the five identified clusters, their population prevalence as well as the incidence and hazard ratio (HR) for gout were computed using cox-proportional hazard analysis in Rv3.5.2.

Results: Cluster-1 (C1) with “few comorbidities” was by far the most common in the population (73%), followed by cluster-4 (C4) characterized by “obesity and hyperlipidaemia” (17%). These two pathways included 86% of incident gout cases during the follow-up. The four clusters (C2-5) identified by different comorbidity patterns all resulted in a 2-3-fold increased risk for incident gout. The highest incidence for gout was seen for cluster-2 (C2) characterized by “renal dysfunction” and cluster-3 (C3) characterized by “cardiovascular disease” (Table).

Table Incidence and hazard ratios for gout in observation-clusters of MPP cohort

Cluster name	Main characteristics	Occurrence, n (%)	Incident gout (n)	Incidence* (per 100,000 person-years at risk)	HR (95% CI) ^o
C1 Few comorbidities	No history of diabetes, CVD or renal disease.	16063 (72.8)	551	119	1
C2 Renal dysfunction	Renal dysfunction (100%), higher age at inclusion	750 (3.4)	53	269	2.31 (1.73-3.07)
C3 Cardiovascular disease and alcohol risk behaviour lifestyle	High frequency of CVD (100%), smoking (74%), alcohol risk behaviour (40%) & PD (22%)	528 (2.4)	26	224	2.41 (1.63-3.58)
C4 Obesity and hyperlipidaemia	High frequency of obesity (34%) & hyperlipidemia (74%)	3673 (16.7)	235	235	2.02 (1.73-2.35)
C5 Diabetes mellitus and hypertension	High frequency of DM (51%), hypertension (53%) & diuretics use (52%)	1043 (4.7)	45	184	1.98 (1.46-2.68)

*Incidence per 100,000 person-years at risk, ^oAdjusted for age, sex

Conclusion: In a population-based study, we identified five clusters based on gout-related comorbidities. Most gout cases occurred in the cluster characterized by few comorbidities. Such cases may have predisposing factors not captured by comorbidity patterns, e.g. genetics related to serum urate. In addition, we identified four pathways each defined by different comorbidity patterns and contributing to increased risk for gout, pathways that may require partly individual interventional strategies.

REFERENCES:

- [1] Richette P, et al. Ann Rheum Dis 2015; 74(1):142-47.
- [2] Megan B, et al. Rheumatol 2018; 57(8):1358-63.

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