Table 1. Radiographic and clinical features of the examined patients

	CPPD N=181	Seropositive RA N=142	Seronegative RA N=129	Gout N=30	PMR N=30
% women	77,8	76,2	75,3	28,1	44,1
Mean age (years)	73,8± 9,8	58,8± 13,0	58,9± 14,7	56,6± 14,7	65,7± 8,9
Symptom duration (years)	0,3±0,7	0,4± 0,7	0,4± 0,9	1,0±3,0	0,4±1,7
Chondrocalcinosis (radiography, %)	93,0	35,7	34,6	12,5	11,8
Erosions (radiography, %)	50,8	42,9	32,1	50	8,8
Clinical involvement of hands (%)	83,2	76,2	72,8	25	50
Clinical involvement of feet (%)	24,3	33,3	32,1	62,5	11,8
Clinical involvement of knee (%)	42,7	39,3	42	59,4	32,4
Symmetric arthritis (%)	64,6	67,9	74	65,6	44,1
Acute attacks, sudden onset (%)	60,5	40,5	40,7	65,6	38,2
Both diagnoses RA and CPPD in record (%)	27,6	29,7	32,1	6,25	14,7
Therapy with MTX or another csDMARD (%)	13,0	52,4	42,0	3,1	23,5
Therapy with Hydroxychloroquine (%)	7,0	4,8	7,4	0	0
Therapy with Colchicine (%)	43,8	9,5	9,9	75	5,9

Conclusion: There were a lot of similarities but also some important differences between patients with CPPD and RA with no major differences between seropositive and seronegative RA. Of interest, radiographic chondrocalcinosis was seen in more than a third of RA patients. Importantly, clinical symmetry of arthritis and involvement of hands did not differentiate between CPPD and RA, mainly the acuteness of attacks did. Co-occurrence of both diseases was frequently observed. There was no major difference between seropositive and seronegative RA.

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POS1146 NONCODING RNA CONTRIBUTE TO PATHOGENESIS IN PRIMARY GOUTY ARTHRITIS

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Background: Gout is an arthritic disease caused by the deposition of monosodium urate crystal (MSU) in the joints, which can lead to acute inflammation and damage adjacent tissue [1].Over the past decade, noncoding RNAs (ncRNAs) have been shown to have crucial importance in health and disease[2,3]. However, studies evaluating the function of ncRNAs in gout are scarce, and current knowledge of the role of ncRNAs in gout is still limited.

Objectives: To assess the contribution of noncoding RNAs to gout and the clinical importance of these genes in primary gouty arthritis (GA).

Methods: The mRNA expression levels of noncoding RNAs (LINC00173, LINC00963, LINC01330 and miRNA-182-5p) were measured in peripheral blood mononuclear cells (PBMCs) from 60 gout patients(including 30 acute gout patients, 30 intercritical gout patients) and 40 healthy subjects. The relationship between noncoding RNA expression levels and laboratory features was analyzed in GA patients.

Results: The expression levels of LINC00173, LINC00963 and miRNA-182-5p were much lower in the AG and IG group than in the HC groups (p<0.05), and no significant difference was detected between AG and IG groups(P>0.05). The expression levels of LINC01330 were much lower in the AG group than in the IG and HC groups (p<0.05), and no significant difference was detected between AG and IG groups(P>0.05). In GA patients, the levels of noncoding RNAs mRNA correlated with laboratory inflammatory and metabolic indexes.

Conclusion: Altered noncoding RNAs expression suggests that noncoding RNAs is involved in the pathogenesis of GA and participates in regulating inflammation and metabolism.

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Figure 1. Relative Expression of noncoding RNAs in the PBMCs of Patients.

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POS1147 COSTS AND FACTORS AFFECTING HEALTH SERVICE UTILISATION IN PATIENTS WITH GOUT: A LONGITUDINAL, POPULATION-LEVEL LINKED DATA STUDY IN WESTERN AUSTRALIA

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Background: Evidence suggests that gout is associated with high health care costs and that many inpatient admissions are preventable (1). Understanding the driver of health care costs will allow more targeted intervention.

Objectives: To examine factors associated with high health service utilisation and cost in patients admitted to hospital with gout, using whole-population linked hospital, WA cancer registration, Emergency Department (ED) and death data (2).

Methods: The study included patients (18 to 84 years) who had been admitted to hospital with a primary or co-diagnosis of gout for the first time between 1 Jan 02 and 31 Dec 09 Hospital costs were calculated per patient using DRG codes and ED costs were calculated from URG codes. Costs are presented in Australian dollars. Follow-up was completed at five years post their initial gout hospitalisation, at death, or at the 31th of December 2014. Both univariable and multivariable analysis was conducted for each patient characteristic. Independent variables were assessed for collinearity. Collinearity was assumed present where the correlation co-efficient was greater than 0.7.

Results: 4,379 individuals were included. In the following five years, there was 22,222 ED attendances (median cost, \$1826 per patient (IQR: \$433 - \$4,414)), and 58,920 hospital admissions, (median cost, \$25,009 per patient (IQR: \$6,844 - \$60,535)). 4,059 (18.3%) ED attendances and 3,834 (6.5%) hospital admissions were potentially preventable. Gout was not a major driver of events, with 341 (1.5%) ED attendances and 620 (1.1%) hospital admissions coded with a primary diagnosis of gout. In the univariable analysis (Table 1), Aboriginality and smoking were associated with an increased number of both ED attendances and hospital admissions. Increased socio-economic status was associated with a reduction in ED attendances, however, this was not reflected in hospital admissions.

Conclusion: Patients admitted to hospital with gout are highly likely to be re-admitted or attend ED in the following 5 years. Many of these contacts are preventable, but are usually driven by comorbidities rather than gout.

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Table 1: Univariable incident risk ratio (95% confidence intervals) for factors associate with health service utilisation in the five years following initial gout hospitalisation.

	ED presentations			Hospital admissions		
	All presentations	PPE	Cost	All presentations	PPE	Cost
Age at 1 st presentation	1.00 (1.00, 1.00)	0.96 (0.96, 0.97)	1.01 (1.01, 1.02)	1.01 (1.00, 1.01)	1.04 (1.03, 1.04)	1.03 (1.02, 1.03)
Sex (base: male)	1.32 (1.15, 1.45)	1.22 (0.97, 1.51)	1.28 (1.11, 1.49)	1.34 (1.01, 1.28)	1.60 (1.36, 1.89)	1.24 (1.08, 1.42)
Aboriginality	3.47 (2.90, 4.03)	12.98 (0.80, 17.20)	2.34 (1.75, 2.80)	1.51 (1.25, 1.82)	1.39 (1.07, 1.80)	1.00 (0.79, 1.25)
Socio-economic status (SES)						
- 0 to 20% (lowest SES)	Base	Base	Base	Base	Base	Base
- 21 to 40%	0.53 (0.44, 0.65)	0.29 (0.21, 0.42)	0.68 (0.50, 0.92)	0.90 (0.70, 1.14)	1.03 (0.74, 1.44)	0.99 (0.75, 1.31)
- 41 to 60%	0.45 (0.38, 0.53)	0.10 (0.07, 0.14)	0.70 (0.54, 0.93)	1.16 (0.93, 1.44)	1.06 (0.78, 1.44)	1.24 (0.96, 1.60)
- 61 to 80%	0.37 (0.31, 0.45)	0.10 (0.07, 0.14)	0.56 (0.42, 0.75)	1.01 (0.80, 1.26)	0.97 (0.71, 1.33)	1.07 (0.82, 1.39)
- 81 to 100% (highest SES)	0.27 (0.23, 0.33)	0.02 (0.01, 0.03)	0.48 (0.36, 0.64)	1.12 (0.89, 1.41)	0.85 (0.62, 1.17)	1.23 (0.96, 1.63)
Smoking (ever)	1.79 (1.64, 1.95)	1.78 (1.46, 2.16)	1.68 (1.48, 1.92)	1.53 (1.38, 1.70)	2.12 (1.82, 2.46)	1.48 (1.31, 1.67)
Comorbidities						
- CVD	1.87 (1.71, 2.03)	0.87 (0.72, 1.06)	2.37 (2.09, 2.69)	3.06 (2.78, 3.38)	6.60 (5.71, 7.64)	2.68 (2.39, 3.01)
- Diabetes	1.74 (1.59, 1.92)	1.39 (1.12, 1.71)	1.83 (1.59, 2.11)	2.43 (2.18, 2.71)	4.02 (3.48, 4.64)	1.84 (1.62, 2.11)

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POS1148

RISK FACTORS FOR POLYARTICULAR GOUT FLARES—ANALYSIS OF A LONGITUDINAL ONLINE GOUT FOLLOW-UP STUDY

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Background: There are known and established risk factors for gout flares; however, no study has examined the factors specifically associated with a polyarticular gout flare.

Objectives: Evaluate risk factors of a polyarticular gout flare in a longitudinal study of individuals with gout.

Methods: We used data from the Boston Online Gout Study, a longitudinal internet-based case-crossover study, where gout patients with at least one flare within the past year were enrolled across the US. Participants (N=903) were followed prospectively for 1 year via the internet to collect information on gout flares and related data including number and specific joints involved, medical comorbidities, diet, exercise and medications.

A polyarticular gout flare was defined as having 3 or more joints involved during a patient-reported flare period. Individuals with at least one flare (to determine flare pattern) during follow-up were included in the analysis. Univariable associations were first assessed to determine factors were associated with polyarticular joint flares. was used to adjust for confounders. When assessing the association of each risk factor with prevalent polyarticular joint involvement, we used multivariable logistic regression, adjusting specific confounders guided by causal diagram.

Results: There were 724 participants with at least one flare with a mean and median age of 55 years. The cohort was 78% male and 89% White with a mean gout disease duration of 8.1 years (median 5). 197 (27%) had a polyarticular gout flare. In univariable analysis (Table 1), female sex, osteoarthritis, obesity, heart failure, hypertension, chronic kidney disease and gout related medication use were each associated with higher odds of polyarticular gout flare, whereas higher education, alcohol use at study enrollment and aspirin use were associated with lower odds. In multivariable analysis, obesity, osteoarthritis and heart failure remained associated with a higher odds of polyarticular gout flare, whereas education remained inversely associated.

Conclusion: In this prospective cohort of gout patients, obesity, heart failure and osteoarthritis were independently associated with higher odds for polyarticular gout flares. Female sex was also positively associated, whereas higher education was protective. The positive association with gout-related medications likely reflects residual confounding by indication. If confirmed, these findings would help identify those at risk for polyarticular flares to help improve clinical care.

Table 1. Associations with Polyarticular Gout Flare (≥ 3 joints)

Risk Factor		Univariable OR (95% CI)	Age- and Sex- Adjusted OR (95% CI)	Multivariable OR* (95% CI)
Age ≥ 55 years (vs < 55)		0.82 (0.59,1.14)	0.77 (0.56,1.08)	0.70 (0.49,1.01)
Female Sex		1.51 (1.04,2.22)	1.55 (1.03,2.32)	1.21 (0.78,1.88)
White Race (vs Non-White)		0.89 (0.54,1.48)	0.93 (0.56,1.54)	0.75 (0.44,1.28)
Education (vs High School or Less)	Some college	0.63 (0.37,1.07)	0.53 (0.37,1.07)	0.68 (0.39,1.17)
	College	0.45 (0.25,0.78)	0.46 (0.26,0.81)	0.49 (0.27,0.88)
	Post-graduate	0.42 (0.25,0.72)	0.44 (0.26,0.76)	0.48 (0.28,0.84)
Body Mass Index (kg/ m ²) at Enrollment (vs < 25)	25-29.9	1.69 (0.85,3.33)	1.77 (0.89,3.51)	1.82 (0.90,3.65)
	≥ 30	2.38 (1.24,4.58)	2.30 (1.19,4.43)	2.19 (1.12,4.28)
Alcohol Use at Enrollment (vs. Non-Use)		0.60 (0.40,0.89)	0.60 (0.40,0.91)	**
Gout Duration ≥ 5 years (vs < 5)		0.85 (0.61,1.18)	0.93 (0.66,1.31)	0.95 (0.66,1.35)
Presence of Comorbidities at Enrollment (vs Absence)	Osteoarthritis	2.35 (1.45,3.81)	2.37 (1.43,3.95)	2.39 (1.42,4.04)
	Heart Failure	2.06 (1.11,3.81)	2.21 (1.17,4.17)	2.03 (1.06,3.91)
	Hypertension	1.61 (1.08,2.40)	1.69 (1.10,2.60)	**
	Chronic Kidney Disease	1.97 (1.03,3.78)	2.11 (1.09,4.11)	**
Medication Use at Enrollment (vs No Use)	Allopurinol	1.58 (1.13,2.22)	1.59 (1.13,2.23)	**
	Diuretics	1.53 (1.06,2.21)	1.59 (1.06,2.38)	**
	Oral Steroids	2.21 (1.16,4.18)	2.26 (1.19,4.31)	**
	Aspirin	0.68 (0.47,0.98)	0.72 (0.49,1.07)	**

*Mutually adjusted for the variables in this column**Not included in model

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POS1149

CARDIOVASCULAR RISK STATUS AND OBESITY IN GOUT COMPARED TO HEALTHY MEXICAN SUBJECTS: A CASE-CONTROLS STUDY WITH A PROPENSITY SCORE-MATCHED ANALYSIS

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Background: Atherosclerotic cardiovascular diseases (ASCVD), metabolic syndrome and hyperuricemia due to an increment in their incidence world-wide, but information regarding the 10-year estimated risk in gout vs. healthy subjects is scarce.

Objectives: To assess baseline the 10-year cardiovascular risk and high-risk status in gout patients compared to healthy controls.

Methods: Data from 494 gout paired to controls in a ratio of 1:1 matched with age, sex and body mass index (BMI) and stratified by normal (≤ 24.9 Kg/m²), overweight (25 - 29.9 Kg/m²) and obese (≥ 30 Kg/m²). Controls were asymptomatic subjects attending a preventive clinic in Mexico City. We used the Framingham Risk Score for Coronary Heart Disease (FRS-CHD) and the 2013 American College of Cardiology/American Heart Association (2013 ACC/AHA). Additionally, high risk status was defined according to pre-defined cutoff points for FRS-CHD ($\geq 20\%$) and ACC/AHA 2013 ($\geq 7.5\%$).

Results: Data from 494 gout and 494 controls. Gout patients were male 97.4%, 47 (SD ± 13) years, mean BMI of 28.4 (SD ± 4.31) Kg/m². Age, sex, and BMI and subgroups were equilibrated (p=NS). Gout patients had higher 10-year estimated risk vs. controls nevertheless, only FRS-CHD reached statistically significant difference with 9.38 vs. 7.5 ± 5.74 (p<0.001); For 2013 ACC/AHA 4.94 ± 6.16 vs. 5.23 ± 6.78 , (p=0.49). Further stratification by BMI groups revealed that subjects