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 31st 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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