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	Osteoarthritis	2.35 (1.45,3.81)	2.37 (1.43,3.95)	2.39 (1.42,4.04)
Comorbidities at	Heart Failure	2.06 (1.11,3.81)	2.21 (1.17,4.17)	2.03 (1.06,3.91)
Enrollment (vs Absence)	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)	**
	Allopurinol	1.58 (1.13,2.22)	1.59 (1.13,2.23)	**
Medication Use at Enrollment (vs No Use)	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

with gout in normal and overweight categories were had higher risk vs. controls with FRS-CHD score of 8.86 ± 8.05 vs. 6.53 ± 6.41 ($p=0.03$) and 9.37 ± 7.72 vs. 7.89 ± 5.58 ($p=0.01$), respectively. No differences for 2013 ACC/AHA in global and BMI-stratified comparisons. Proportion of high-risk subjects were similar in both groups regardless of BMI category: for FRS-CHD 7.5 vs 4.7% ($p=0.06$) and 2013 ACC/AHA with 21.5 vs. 17.8% ($p=0.14$).

Conclusion: Normal BMI or overweight Mexican patients with gout may be at higher 10-year estimated cardiovascular risk compared to matched age, gender and BMI healthy controls. Obese gout subjects are at high risk with non-statistical significance scores for FRS-CHD or 2013 ACC/AHA compared to controls.

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POS1150

ANATOMICAL LOCATIONS AND CORRELATES OF CALCIUM PYROPHOSPHATE CRYSTAL DEPOSITS OF THE SPINE – PATHOLOGIC EXAMINATION OF 77 SURGICAL CASES

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Background: Spinal involvement in calcium pyrophosphate deposition disease (CPPD) is thought to be a rare occurrence and is seen infrequently as crowned dens syndrome. Furthermore, data on anatomical locations and correlates of calcium pyrophosphate (CPP) deposits in spinal CPPD are scarce.

Objectives: To describe the anatomical locations and correlates of pathologically confirmed CPPD of the spine.

Methods: Consecutive patients with spinal CPPD were identified via retrospective chart review of individuals who underwent spine surgery for intractable chronic neck or back pain at Massachusetts General Hospital between 2009 and 2014. These deposits and surrounding anatomical structures were surgically resected and confirmed to have calcium pyrophosphate deposition upon pathologic review. We reviewed musculoskeletal imaging (CT, MRI, XR) and laboratory data from these pathologically confirmed cases.

Results: From April 2009 to August 2014, we identified 77 individuals with pathologically confirmed CPPD of the spine. The mean age was 68 years; 41 (53%) were female; mean BMI was 28.7. Calcium pyrophosphate (CPP) was grossly identified intraoperatively by the surgeon in 38 cases (50%), typically as “chalky white deposits” (Figure 1). CPP deposits were seen most frequently in the ligamentum flavum (23%) and intervertebral disc (23%), followed by other less common locations (Table 1). Imaging findings in the soft tissue or intervertebral disc suggestive of CPPD were found in 5 cases (6%), whereas findings of spinal canal narrowing, facet arthropathy, or ligamentum flavum thickening were eventually correlative with CPP deposits in pathologic specimens. Only 7 (9%) experienced a prior episode of acute CPP arthritis (pseudogout). Chondrocalcinosis on x-ray was seen in 26 cases (34%), most commonly in the wrist and/or knees. Osteoarthritis was present in all spinal imaging, and 65% had comorbid scoliosis. Laboratory abnormalities associated with secondary causes of CPPD (hypercalcemia, hypomagnesemia, hyperparathyroidism) were not seen with spinal CPPD.

Conclusion: Spinal CPPD may occur more frequently than previously perceived. The ligamentum flavum and intervertebral discs were common anatomical locations for spinal CPPD. Advanced imaging of the spine showed low sensitivity for detecting spinal CPPD. Only a small minority had typical peripheral joint involvement or imaging with peripheral joint chondrocalcinosis. Thus, without pathologic confirmation, the vast majority of cases would remain unidentified. These findings call for the need to seek pathologic confirmation to determine the robust epidemiology and also raise the potential role for pre-operative CPPD treatment.

Table 1. Spinal Anatomic Locations of Pathologically Confirmed CPPD

Spinal Anatomic Location	No. of Sites (%) [*]
ligamentum flavum	29 (23)
Intervertebral Disc	28 (23)
Other Location	19 (15)
Posterior Elements	18 (15)
Facet	14 (11)
Synovium	8 (6)
Interspinous Ligament	3 (2)
Subarticular/Lateral Recess	2 (2)
Fibrocartilaginous Tissue	1 (1)
Inner Spine	1 (1)
Other Ligament	1 (1)

^{*}Some patients had more than one anatomic location where CPP was isolated

Figure 1. Gross visualization of calcium pyrophosphate deposition (black arrow)

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Infection-related rheumatic diseases

POS1151

IMMUNOGENICITY AND SAFETY OF 23-VALENT PNEUMOCOCCAL POLYSACCHARIDE VACCINE IN PATIENTS WITH SPONDYLOARTHRITIS.

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Background: Currently, for the treatment of patients with spondyloarthritis (SpA), basic anti-inflammatory drugs and biological drugs are widely used to effectively control the activity of the disease. At the same time, the use of these drugs is associated with an increased risk of developing infections, some of which can be prevented by vaccination.

Objectives: The aim of the study was to evaluate the immunogenicity and safety of the 23-valent pneumococcal polysaccharide vaccine (PPV-23) in patients with SpA.

Methods: The study included 42 patients with SpA: 30 men, 12 women, age 22-60 years, disease duration 14.8 ± 10.6 years. Ankylosing spondylitis was diagnosed in 30 patients, psoriatic spondyloarthritis – in 12 patients. Activity of diseases was assessed as high in 78% of patients (median BASDAI index was 5.3 [4.1; 6.8]). At the time of inclusion in the study, all patients received nonsteroidal anti-inflammatory drugs, 14 patients - methotrexate, 10 - sulfasalazine, 6 - glucocorticoids, 2 - leflunomide, 2 - etanercept, 2 - secukinumab. 15 patients were planned to be prescribed secukinumab, 3 – etanercept, 3 - adalimumab, 1 – golimumab. 7 patients had a history of more than 2 cases of lower respiratory tract infections, 2 patients reported a monthly exacerbation of chronic sinusitis, 1 patient – the development of otitis every 2-3 months, requiring the use of antibacterial drugs. PPV-23 was administered in the amount of 1 dose (0.5ml) subcutaneously into the deltoid muscle against the background of anti-rheumatic therapy.