Table.

B (95% confidence interval per one SD of the comorbidity index/subscale) are reported

	NRS hand pain	NRS pain all joints	AUSCAN hand pain	WOMAC knee/hip pain	Painful joints	PPT Trapezium	PPT Tibialis Anterior	TS
<i>Comorbidity Index</i>	0.76 (0.12, 0.24)	0.76 (0.12, 0.24)	1.61 (0.15, 0.37)	2.76 (0.26, 0.50)	2.76 (0.42, 0.88)	-0.38 (-0.15, -0.04)	-0.25 (-0.13, 0.01)	0.25 (0.02, 0.11)
Disease	0.69 (0.24, 0.52)	0.56 (0.16, 0.46)	0.89 (0.22, 0.75)	1.34 (0.45, 1.02)	2.19 (0.66, 1.76)	-0.34 (-0.32, -0.06)	-0.24 (-0.29, 0.04)	0.23 (0.02, 0.23)
Treatment	0.56 (0.18, 0.48)	0.58 (0.18, 0.49)	0.80 (0.19, 0.76)	1.29 (0.46, 1.06)	1.79 (0.47, 1.65)	-0.31 (-0.32, -0.44)	-0.19 (-0.28, 0.07)	0.22 (0.01, 0.24)
Limitations	0.69 (0.32, 0.70)	0.86 (0.44, 0.81)	1.16 (0.51, 1.20)	1.58 (0.78, 1.53)	3.30 (1.72, 3.13)	-0.41 (-0.47, -0.13)	-0.26 (-0.41, 0.02)	0.26 (0.04, 0.33)
Depression	0.96 (0.29, 1.63)	0.66 (-0.04, 1.36)	1.74 (0.46, 3.02)	0.99 (-0.38, 2.35)	3.43 (0.78, 6.09)	-0.36 (-1.00, 0.29)	-0.44 (-1.25, 0.37)	0.02 (-0.50, 0.53)
Back pain	0.89 (0.38, 1.40)	1.10 (0.57, 1.63)	1.04 (0.08, 2.00)	2.90 (1.87, 3.93)	2.64 (0.65, 4.64)	0.08 (-0.41, 0.56)	0.23 (-0.38, 0.84)	0.22 (-0.17, 0.61)
Hypertension	0.64 (0.05, 1.22)	0.45 (-0.17, 1.06)	1.00 (-0.12, 2.11)	1.32 (0.13, 2.52)	2.74 (0.44, 5.05)	-0.32 (-0.88, 0.23)	-0.28 (-0.97, 0.40)	0.17 (-0.28, 0.62)

comorbidity other than OA. Mean (SD) comorbidity index value was 7.73 (4.25). Higher values were statistically significantly associated with greater pain severity by each pain questionnaire, lower PPT trapezium (borderline significant for PPT tibialis anterior), and greater TS (Table). Similar results were found for subscales. Depression, back pain and hypertension associated with most measures of pain. (Table). Included in the same model, results remained similar (not shown). No statistically significant association between individual comorbidities and pain sensitization (Table).

Conclusion: Higher burden of comorbidities was associated with greater pain intensity and central pain sensitization. Hypertension, back pain and depression were associated with most pain measures. The lack of relation between individual comorbidities and pain sensitization suggest the burden of comorbidity is more important.

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OP0088

A COHORT STUDY ON THE BIDIRECTIONAL RELATIONSHIP BETWEEN FIBROMYALGIA AND PERIODONTITIS OVER A 15-YEAR FOLLOW-UP

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Background: Fibromyalgia has been proposed to be driven by chronic inflammation and infections that are associated with early rheumatoid arthritis and inflammatory arthritis. Other than the central sensitization model, our knowledge of fibromyalgia pathogenesis has been expanded with an inflammation-dependent theory, which is stimulated by prolonged spinal cord hyperexcitability.

Objectives: To determine the relationship between periodontitis and fibromyalgia.

Methods: In this cohort study, 196,428 periodontitis patients and 196,428 propensity score-matched non-periodontitis controls were enrolled. A Cox proportional hazard model was utilized to estimate the risk of fibromyalgia and survival analysis was adopted to assess the time-dependent effect of periodontitis on fibromyalgia. Subgroup analyses stratified by age, gender, and follow-up years were conducted to identify susceptible populations. A symmetrical cohort was designed to ascertain the relationship between fibromyalgia and the risk of periodontitis.

Results: Patients with history of periodontitis were more likely to develop fibromyalgia than non-periodontitis controls (HR =1.42, 95% CI =1.39–1.44, P < 0.001), which persisted in the survival analysis (log-rank test P < 0.0001). This effect was significant in both genders and all age subgroups,

and was particularly evident in males (HR=1.52, 95% CI = 1.48–1.56, P < 0.001) and younger periodontitis patients (HR= 1.55, 95% CI =1.50–1.60, P < 0.001). Fibromyalgia patients who never had periodontitis presented with higher risk for periodontitis over time (HR = 1.43, 95% CI = 1.40 - 1.45, P < 0.001).

Conclusion: This is the first longitudinal study that addresses the bidirectional relationship between fibromyalgia and periodontitis, in which periodontitis may serve as a risk factor or early sign of fibromyalgia. Based on the observed relationship between fibromyalgia and periodontitis, regular follow-ups and patient education are recommended for patients with either disease.

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OP0089

DO PAIN CATASTROPHIZING REDUCE THE LIKELIHOOD OF REMISSION IN PATIENTS WITH CHRONIC INFLAMMATORY JOINT DISORDERS?

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Background: Rheumatoid arthritis (RA), psoriatic arthritis (PsA) and axial spondyloarthritis (ax-SpA) may have a devastating impact on patients' lives, including chronic and relapsing pain. One factor contributing to bio-psycho-social model of pain in these diseases, is pain catastrophizing (PC) defined as tendency to report pain experience in exaggerated terms, to ruminate more or to feel helpless about it. In one RA study PC has been shown to be associated with patient reported outcomes e.g. reduced likelihood of remission using composite measures.[1]

Objectives: Main objective of this study was to explore if pain catastrophizing score is associated with remission rates in the chronic inflammatory joint disorders RA, PsA and ax-SpA.

Methods: Patients were recruited during routine assessment at an outpatient clinic in Norway. Variables collected included demographics, treatment, disease duration, global pain VAS, pain catastrophizing score (PCS), and disease activity scores: DAS28-CRP for RA, BASDAI for ax-SpA and DAPSA for PsA. Cut off definitions of remission was for DAS28 <2.6, for BASDAI <4.0 and for DAPSA ≤4.0. Two questions from Coping Strategies Questionnaire as recommended by Jensen et al [2] were used to describe PC, range PC total score (PCS) 0–6. Patients with PCS ≥ 4 were defined as high pain catastrophizers. Statistics included Chi-Square test and adjusted logistic regression (enter procedure).

Results: For RA (N=580), PsA (N=394) and ax-SpA (N=225) mean age (SD) was 61.5 (13.1), 54.4 (12.9) and 47.5 (12.9) years, mean disease duration 12.6 (10.9), 10.2 (8.4) and 12.2 (10.7) years and percentage of women 66.7%, 47.0% and 38.4%. Mean (SD) DAS28-CRP in RA was 2.3 (1.0), BASDAI for ax-SpA 3.7 (2.3) and DAPSA for PsA 10.8 (8.5). Mean (SD) global pain (VAS 0–100mm) was for RA 32.0 (25.6), for PsA 35.5 (25.4) and for ax-SpA 39.4 (25.4) mm.

Conventional synthetic DMARDs were used by 65.0% of RA patients, 54.8% by PsA and 9.8% in ax-SpA and for biologic DMARDs the corresponding numbers were 36.7%, 43.9%, 65.9%, respectively.

Percentage of patients in remission was significantly lower in high pain catastrophizers compared with low pain catastrophizers in RA (34.1% vs 70.3%, $p<0.01$) and ax-SpA patients (17.9% vs 64.5%, $p<0.01$) but not in PsA (10.3% vs 26.7%, $p=0.06$).

In analysis adjusting for age, gender, disease duration and global pain (Tab. 1), PC was independently associated with lower remission rate in RA (OR 0.79 (95% CI 0.63, 0.99), $p=0.04$) and in ax-SpA (OR 0.65 (95% CI 0.46, 0.93), $p=0.02$) but not in PsA (OR 0.83 (95% CI 0.55, 1.25), $p=0.37$).

Conclusion: Our data indicates that high level of patient's PC may reduce likelihood of achieving remission in RA and ax-SpA. Interestingly, this was not shown in PsA patients. Further studies are needed to reveal the importance of PC and its influence on composite disease activity measures used to define disease status e.g. remission.

Table 1. Variables and their associations with remission rates in RA, PsA, ax-SpA in logistic regression analysis.

	RA remission		PsA remission		ax-SpA remission	
	OR (95%CI)	p	OR (95%CI)	p	OR (95%CI)	p
PCS	0.79 (0.63, 0.99)	0.04	0.83 (0.55, 1.25)	0.37	0.65 (0.46, 0.93)	0.02
Age	0.99 (0.97, 1.01)	0.17	1.00 (0.96, 1.04)	0.85	0.98 (0.94, 1.02)	0.27
Gender	0.87 (0.52, 1.45)	0.59	0.37 (0.15, 0.93)	0.03	0.92 (0.40, 2.14)	0.85
Disease duration	1.03 (1.00, 1.05)	0.03	0.95 (0.90, 1.00)	0.06	1.04 (0.99, 1.09)	0.09
Global pain (VAS)	0.96 (0.95, 0.97)	<0.01	0.85 (0.81, 0.89)	<0.01	0.90 (0.88, 0.92)	<0.01

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Infection-related rheumatic and orphan diseases

OP0090

CLASSIFICATION OF PATIENTS WITH RELAPSING POLYCHONDRIITIS BASED ON SOMATIC MUTATIONS IN UBA1

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Background: Somatic mutations in ubiquitin activating enzyme 1 (UBA1) cause a newly defined syndrome known as VEXAS. [1] More than fifty percent of patients currently identified with VEXAS meet diagnostic criteria for relapsing polychondritis (RP).

Objectives: To determine the prevalence VEXAS within a cohort of patients with RP, to compare their clinical, laboratory, and immunologic features and to develop a clinical algorithm to inform genetic screening for VEXAS among patients with RP.

Methods: Exome and targeted sequencing of the UBA1 gene was performed in a prospective observational cohort of patients with RP. Clinical and immunological characteristics of patients with RP were compared based on presence or absence of UBA1 mutations. Random forest was used to derive a clinical algorithm to identify patients with UBA1 mutations. Immune populations were quantified by multipanel flow cytometry. Categorical and continuous variables were compared using the chi square or Kruskal-Wallis test. $P<0.05$ defined statistical significance.

Results: Seven of 92 patients with RP (7.6%) were confirmed to have UBA1 mutations (VEXAS-RP). Six additional patients with VEXAS-RP from other cohorts were included for subsequent analyses. Patients with VEXAS-RP were all male, older at disease onset, and commonly had fever, ear chondritis, skin involvement, deep vein thrombosis, and pulmonary infiltrates. Patients with RP as compared with VEXAS-RP had a significantly higher prevalence of airway chondritis, costochondritis and tenosynovitis/arthritis. (Table). Mortality was significantly greater in VEXAS-RP than RP (27% vs 2% $p=0.01$). Maximum ESR, CRP, and mean corpuscular volume (MCV) values were significantly greater in VEXAS-RP. Absolute monocyte, lymphocyte, and platelet counts were significantly lower in VEXAS-RP. A decision tree based on male sex, MCV>100 fl and Platelet count<200 K/ul classified between VEXAS-RP and RP with 100% sensitivity and 96% specificity.

Conclusion: Mutations in UBA1 are causal for disease in a subset of patients with RP. These patients are defined by disease onset in the fifth decade of life or later, male sex, ear/nose chondritis and hematologic abnormalities. Early identification is important in VEXAS given the associated high mortality rate.

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Table 1. Clinical Characteristics of patients with RP vs VEXAS-RP

	All Patients n=98	RP n=85	VEXAS-RP n=13	p value
Demographic Characteristics				
Race, White n (%)	90 (92)	77 (91)	13 (100)	0.59
Sex, Male n (%)	26 (27)	13 (15)	13 (100)	<0.0001
Age, Symptom onset, years, Median (IQR)	38 (30-47)	37 (28-43)	56 (54-64)	<0.0001
Clinical Symptoms				
Fever n (%)	33 (34)	20 (24)	13 (100)	<0.0001
Ear chondritis n (%)	61 (62)	48 (56)	13 (100)	0.0015
Nose chondritis n (%)	83 (85)	71 (84)	12 (92)	0.68
Airway chondritis n (%)	37 (38)	37 (44)	0 (0)	0.0015
Tenosynovitis/arthritis n (%)	83 (85)	77 (91)	6 (46)	0.0005
Skin involvement n (%)	33 (34)	22 (26)	11 (85)	<0.0001
Laboratory Values				
ESR, mm/hr, median (IQR)	12 (6-22)	11 (5-19)	66.5 (42-110)	<0.0001
CRP, mg/L, median (IQR)	2.9 (0.8-9.6)	1.9 (0.6-6.3)	17.7 (9.6-99.5)	<0.0001
Platelet count (k/uL)	246(201-299)	258 (227-312)	145 (100-169)	<0.0001
MCV fL	93.05 (90-98)	92.2 (89-95)	105 (102-115)	<0.0001
Absolute lymphocyte count	1.6 (1.1-2.3)	1.78(1.4-2.4)	0.92 (0.5-1.2)	<0.0001
CT scan abnormalities				
Pulmonary infiltrates n (%)	16 (16.33)	6 (7.06)	10 (77)	<0.0001
Complications				
Death n (%)	6 (6)	3 (4)	3 (23)	0.029
Unprovoked DVT	12 (12)	4 (5)	8 (62)	<0.0001

N number; IQR = interquartile range

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