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OP0233

EFFICACY AND SAFETY OF UPADACITINIB IN PATIENTS WITH PSORIATIC ARTHRITIS AND AXIAL INVOLVEMENT

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Background: Patients (pts) with psoriatic arthritis (PsA) and axial involvement exhibit greater disease activity and quality of life impairments compared with those without axial involvement.

Objectives: To characterize PsA pts with and without axial involvement and compare efficacy of UPA vs placebo (PBO) in PsA pts with axial involvement.

Methods: In SELECT-PsA 1 (NCT03104400; N=1705, non-biologic DMARD IR) and SELECT-PsA 2 (NCT03104374; N=642, biologic DMARD IR), pts with active PsA (≥3 swollen and ≥3 tender joints), active or historical psoriasis, and on ≤2 non-biologic DMARDs were randomized to once daily UPA 15mg, UPA 30mg, adalimumab 40mg every other week (SELECT-PsA 1 only), or PBO. Efficacy was assessed in pts with axial involvement (diagnosed by investigators based on totality of information) pooled from the 2 studies. Assessments included change from BL in BASDAI, BASDAI Q2 (neck/back/hip pain) and Q3 (joint swelling/pain), and the AS Disease Activity Score (ASDAS-CRP), and percentage with BASDAI 50 response, ASDAS inactive disease (ID), ASDAS low disease activity (LDA), ASDAS major improvement (MI), and ASDAS clinically important improvement (CII). Uveitis and inflammatory bowel disease (IBD) adverse events were reviewed. Data on 24-week PBO-controlled period are presented.

Results: Prevalence of axial involvement was 31.3% in SELECT-PsA 1 and 34.2% in SELECT-PsA 2 (Table). Treatment with UPA 15mg and 30mg resulted in significantly greater improvements from BL in the BASDAI, BASDAI Q2 (neck/back/hip pain) and Q3 (joint swelling/pain) and ASDAS-CRP at weeks 12 and 24 vs PBO (Figure). Similarly, significantly higher percentages of pts on UPA 15mg and 30mg achieved BASDAI 50, ASDAS ID, LDA, MI, and CII at weeks 12 and 24 vs PBO (Figure). One pt on UPA 30mg had incident uveitis, and no IBD was reported on UPA.

Conclusion: PsA pts with axial involvement had higher BL disease burden compared with those without axial involvement. UPA was efficacious in treating axial symptoms in pts with psoriatic spondylitis.

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Table 1. Demographics and Baseline Characteristics

Parameter, mean (SD)	SELECT-PsA 1			SELECT-PsA 2		
	With Psoriatic Spondylitis (n=534)	Without Psoriatic Spondylitis (n=1170)	P value*	With Psoriatic Spondylitis (n=219)	Without Psoriatic Spondylitis (n=421)	P value*
BMI (kg/m ²)	29.9 (6.5)	30.5 (6.9)	.0810	31.6 (8.0)	31.3 (6.9)	.6226
TJC68	21.6 (15.1)	19.2 (13.5)	.0022	27.5 (18.0)	23.3 (16.2)	.0027
SJC66	11.7 (9.4)	11.0 (7.9)	.1184	12.9 (9.2)	11.7 (8.7)	.0804
Physician's Global Assessment (NRS 0–10)	6.7 (1.6)	6.5 (1.7)	.0437	6.6 (1.8)	6.5 (1.7)	.1897
HAQ-DI	1.2 (0.6) n=531	1.1 (0.6) n=1164	.0170	1.2 (0.6) n=218	1.2 (0.7) n=416	.2049
Presence of dactylitis, n (%) [†]	188 (35.2)	328 (28.0)	.0028	69 (31.5)	100 (23.8)	.0348
Presence of enthesitis, n (%) [‡]	432 (80.9)	884 (75.6)	.0147	189 (86.3)	337 (80.0)	.0125
ASDAS-CRP	3.4 (0.9) n=530	3.1 (1.0) n=1161	<.0001	3.3 (1.0) n=217	3.2 (1.1) n=416	.1032
BASDAI	5.8 (2.0) n=530	5.3 (2.2) n=1161	<.0001	6.2 (2.2) n=217	5.8 (2.2) n=416	.0673
Morning Stiffness Duration (NRS 0–10; BASDAI Q6)	5.0 (3.0)	4.7 (3.0)	.0368	5.6 (3.2)	5.1 (3.0)	.0454
Patient's Assessment of Inflammatory Neck, Back, or Hip Pain (NRS 0–10; BASDAI Q2)	n=530	n=1161	<.0001	n=217	n=416	.0001

*Calculated by t-test for continuous variables and chi-square test for categorical values. Bolded if <0.05. Defined as [†]LDI>0 and [‡]total enthesitis count >0

Figure. Integrated Analysis of Efficacy in PsA Patients With Axial Involvement

ASDAS, ankylosing spondylitis disease activity score; BL, baseline; CII, clinically important improvement; ID, inactive disease; LDA, low disease activity; MI, major improvement; MMRM, mixed-effects model repeated measurement; NRI, non-responder imputation; QD, once daily; UPA, upadacitinib. *p<0.0001, UPA vs placebo; nominal P values are presented and were not adjusted for multiple testing. MMRM analysis with unstructured variance-covariance matrix, including treatment, visit, treatment-by-visit interaction, study, and the stratification factor of current DMARD use (yes/no) as fixed factors and the continuous fixed covariates of baseline measurement. NRI analysis with nominal P value constructed using the Mantel-Haenszel estimation adjusting for study and the main stratification factor of current DMARD use (yes/no). 95% CI for response rate calculated based on normal approximation to the binomial distribution.

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Epidemiology and public health

OP0234 RISK OF ACUTE MYOCARDIAL INFARCTION AMONG NEW USERS OF CHONDROITIN SULPHATE: A NESTED CASE-CONTROL STUDY

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Background: There is some evidence from epidemiological studies suggesting that CS and glucosamine could play a role in cardiovascular disease (CVD) prevention (1-4). Studies to date have included prevalent users, therefore a bias that overestimates protection cannot be excluded.

Objectives: To test the hypothesis that chondroitin sulphate (CS) or glucosamine reduce the risk of acute myocardial infarction (AMI).

Methods: Case-control study nested in a primary cohort composed of patients aged 40 to 99 years, with at least one year of follow-up in the BIFAP database during the 2002-2015 study period. From this cohort of patients, we identified incident cases of AMI and randomly selected five controls per case, matched by exact age, gender, and index date. Adjusted odds ratios (AOR) and their corresponding 95% confidence interval (CI) were calculated through a conditional logistic regression. Only new users of CS or glucosamine were considered.

Results: A total of 23,585 incident cases of AMI and 117,405 controls were included. The mean age was 67.0 (SD 13.4) years and 71.75% were male, in both groups. 558 (2.37%) cases and 3,082 (2.62%) controls used or had used CS. The current use of CS was associated with a lower risk of AMI (AOR 0.57; 95%CI: 0.46-0.72) and disappeared after discontinuation (recent and past users). The reduced risk among current users was observed in both short-term (<365 days AOR 0.58; 95%CI: 0.45-0.75) and long-term users (>364 days AOR 0.56; 95%CI 0.36-0.87), in both sexes (men, AOR=0.52; 95%CI:0.38-0.70; women, AOR=0.65; 95%CI: 0.46-0.91), in individuals over or under 70 years of age (AOR=0.54; 95%CI:0.38-0.77, and AOR=0.61; 95%CI:0.45-0.82, respectively) and in individuals at intermediate (AOR=0.65; 95%CI:0.48-0.91) and high cardiovascular risk (AOR=0.48;95%CI:0.27-0.83), but not in those at low risk (AOR=1.11; 95%CI:0.48-2.56). In contrast, the current use of glucosamine was not associated with either increased or decreased risk of AMI (AOR= 0.86; CI95% 0.66-1.08)

Conclusion: Our results support a cardioprotective effect of CS, while no effect was observed with glucosamine. The highest protection was found among subgroups at higher cardiovascular risk.

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OP0235 PRO-INFLAMMATORY DIET AND RISK OF INCIDENT GOUT: 3 PROSPECTIVE COHORT STUDIES OF US MEN AND WOMEN

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Background: Emerging evidence suggests inflammation may drive progression from hyperuricemia to clinical gout, but the role of extrinsic, modifiable sources of chronic inflammation, such as diet, on gout risk is unknown. Notably, greater dietary inflammatory potential has been independently associated with increased risk of incident cardiovascular disease (CVD)¹ and type 2 diabetes (T2D).²

Objectives: Prospectively examine the relation between dietary inflammatory potential and risk of gout in three large cohorts of US women and men over 30 years.

Methods: Ascertaining the ACR survey criteria for gout for several decades,³ we studied gout risk among 164,090 women from Nurses Health Study I (1986-2016) and II (1989-2017) and 40,598 men from Health Professionals Follow-up Study (1986-2016), free of gout at baseline. Dietary intake and covariates were assessed by validated questionnaires every 4 years. Inflammatory potential of diet was evaluated using a food-based empirical dietary index of inflammatory potential score (EDIP) pre-defined based on circulating levels of IL-6, C-reactive protein, adiponectin, and TNF α .⁴

We assigned an EDIP score for each participant, adjusted for total energy take, and prospectively examined the association between quintiles of EDIP score and incident gout, adjusting for potential confounders. We also stratified by alcohol intake, as alcohol has anti-inflammatory properties,⁴ but is associated with a higher gout risk, particularly beer.⁵

Results: We documented 2,874 incident gout cases over 5,124,940 person-years of follow-up. In pooled multivariable-adjusted analyses, those in the highest EDIP quintile had 59% higher gout risk (multivariable RR 1.59; 95% CI 1.41-1.79), compared with the lowest (Table 1). This remained positive with further adjustment for BMI, a likely causal intermediate (RR 1.27, 1.12 to 1.42), and was stronger among non-drinkers (RR 2.37, 1.58 to 2.56) than drinkers (RR 1.57, 1.38 to 1.78) (Table 1).

Table 1. Risk Ratio (95% CI) of Gout According to Quintiles of Inflammatory Diet Score, Overall and by Alcohol Use

	Q1: lowest	Q2	Q3	Q4	Q5: highest	P for trend
Overall						
N cases	473	493	530	623	755	
Person-years	1,024,571	1,025,618	1,025,284	1,024,779	1,024,688	
Age-adjusted RR	1.00 (Ref)	1.05 (0.92, 1.19)	1.13 (0.99, 1.27)	1.33 (1.18, 1.50)	1.64 (1.46, 1.84)	<0.001
MV-Adjusted* RR	1.00 (Ref)	1.04 (0.92, 1.18)	1.12 (0.98, 1.26)	1.31 (1.16, 1.48)	1.59 (1.41, 1.79)	<0.001
MV-Adjusted** RR (+ BMI)	1.00 (Ref)	1.00 (0.88, 1.13)	1.03 (0.91, 1.17)	1.16 (1.02, 1.31)	1.27 (1.12, 1.42)	<0.001
No Alcohol Use						
N cases	26	58	84	143	251	
Person-years	118,301	189,938	249,389	313,511	396,080	
MV-Adjusted* RR	1.00 (Ref)	1.31 (0.82, 2.08)	1.37 (0.88, 2.13)	1.80 (1.18, 2.74)	2.37 (1.58, 2.56)	<0.001
MV-Adjusted** RR (+ BMI)	1.00 (Ref)	1.28 (0.80, 2.03)	1.32 (0.85, 2.05)	1.61 (1.06, 2.45)	1.85 (1.23, 2.79)	<0.001
Alcohol Use						
N cases	447	435	446	480	504	
Person-years	906,271	835,680	775,895	711,267	628,609	
MV-Adjusted* RR	1.00 (Ref)	1.04 (0.91, 1.19)	1.13 (0.99, 1.29)	1.31 (1.15, 1.50)	1.57 (1.38, 1.78)	<0.001
MV-Adjusted** RR (+ BMI)	1.00 (Ref)	1.00 (0.88, 1.14)	1.05 (0.92, 1.20)	1.17 (1.03, 1.33)	1.28 (1.12, 1.46)	<0.001

*Multivariable (MV) models adjusted for age (month), White race, smoking, menopause (women only), hormone use (women only), physical activity, history of hypertension, and diuretic use. **MV + BMI models additionally adjusted for BMI (a likely causal intermediate)