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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)