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Investigating the Association Between Alcohol Consumption and Spinal Radiographic Progression in Axial Spondyloarthritis: A Longitudinal Cohort Analysis over a Period of 6 Years

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Diagnosis, Manifestations, & Outcomes I

Background/Purpose: We assessed whether alcohol consumption (AC) is associated with spinal radiographic progression in axSpA as measured by the modified Stoke Ankylosing Spondylitis Spinal Score (mSASSS). Progression was defined as an increase of ≥2 mSASSS units in 2 years. In addition, we evaluated to what extent this association is mediated through Ankylosing Spondylitis Disease Activity Score (ASDAS).

Methods: We used the data from the University Health Network (UHN)- Spondyloarthritis cohort, which followed axSpA patients over 6 years. We included axSpA patients with available data on alcohol consumption and spinal radiographs every 2 years. Generalized estimating equation (GEE) analyses were conducted to determine the association between AC and spinal progression, utilizing repeated measures of alcohol over time. Sensitivity analyses were performed to assess the impact of sex and disease subgroup. Mediation analysis were performed to test whether the effect of alcohol consumption on spinal progression in axSpA patients operates through ASDAS. Linear imputation methods were utilized to address missingness.

Results: GEE analyses were performed in 769 records of radiographic intervals from 387 patients (70% male, 84% radiographic-axSpA (r-axSpA), mean (\pm SD) age 38.6 \pm 13.0 years). Patients reported consuming alcohol (> 0 units/week) in 47% of radiographic interval records analyzed. The multivariable GEE model revealed increased odds of spinal progression in consumers, adjusted for important confounders (Table 1). In the adjusted analysis, length of follow-up, ASDAS,

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syndesmophytes at baseline, male sex, and age were also associated with increased odds of spinal progression. Subgroup analysis showed a significant association between alcohol and spinal progression only in males and patients with r-axSpA (OR 2.28, 95%CI 1.31 to 3.96 and OR 1.90, 95%CI 1.15 to 3.15, respectively). Using mediation analysis, we demonstrated a significant direct effect of alcohol on spinal progression, suggesting heightened spinal damage progression in consumers (Table 2). The indirect effect of alcohol via ASDAS was negatively associated with spinal progression, as consumers had lower ASDAS and, thereby, lower odds of spinal progression. The total effect showed only a trend toward increased odds of progression.

Conclusion: These findings highlight the detrimental effect of alcohol on spinal progression in axSpA, especially in males and r-axSpA patients. The exact mechanism underlying alcohol's effect on spinal progression remains unclear. Our mediation analysis failed to show that inflammation and symptoms driven by inflammation play a role in this relationship. Future studies assessing alcohol's effect on spinal structural damage in axSpA should explore other biological mechanisms beyond inflammation.

Table 1. Univariable and Multivariable GEE Models Assessing the Impact of Alcohol Consumption on Spinal Radiographic Progression

Variables	Univariable analysis	Multivariable analysis
	Crude OR (95%CI)	Adjusted OR (95%CI)
Length of follow-up, years	1.13 (1.02, 1.25)	1.17 (1.02, 1.35)
Age, years	1.06 (1.05, 1.08)	1.03 (1.01, 1.06)
Male sex	4.86 (2.35, 10.06)	3.21 (1.45, 7.11)
Alcohol consumption Non-consumer Consumer	Ref 1.89 (1.27, 2.80)	Ref 1.89 (1.15, 3.09)
Disease duration, years	1.04 (1.02, 1.06)	0.99 (0.96, 1.01)
Ever-smoking	1.85 (1.18, 2.91)	0.75 (0.43, 1.31)
ASDAS	1.36 (1.14, 1.62)	1.43 (1.14, 1.79)
Presence of syndesmophytes at baseline	13.32 (7.56, 23.60)	8.12 (3.72, 17.72)
HLA-B27 positive	0.99 (0.57, 1.69)	0.91 (0.47, 1.75)
Biologic DMARDs use	0.94 (0.55, 1.60)	0.97 (0.54, 1.73)

Statistically significant results are presented in bold. Results from the GEE model after linear imputations and excluding patients with missing covariate data. Spinal radiographic progression was defined as an increase of ≥2 mSASSS units in 2 years. The GEE model included only patients with complete observations. Analyses were performed in 769 radiographic intervals from 387 axSpA patients (148 progression events). The follow-up length

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signifies the years from baseline to the last recorded set of X-rays. Biologic DMARDs use was defined as patients treated with biologic from baseline to the last recorded set of X-rays.

ASDAS: Ankylosing Spondylitis Disease Activity Score; bDMARD: biologic disease-modifying antirheumatic drug; aOR: adjusted Odds Ratio; Ref: reference.

Table 2. Mediation Analysis of the Impact of Alcohol Consumption on Spinal Progression with ASDAS as a Mediator

	aOR (95%CI)	P-value
Direct effect of alcohol	1.92 (1.04, 2.81)	0.036
Indirect effect of alcohol (transmitted through ASDAS)	0.94 (0.85, 0.98)	0.012
Total effect of alcohol	1.81 (0.97, 2.53)	0.07

Statistically significant results are presented in bold. Results from the mediation analysis included 387 patients. 1000 bootstrap samples were used to calculate confidence intervals and p-values. Spinal radiographic progression was defined as an increase of ≥2 mSASSS units in 2 years. Analysis adjusted for length of follow-up, age, sex, ever-smoking, presence of syndesmophytes at baseline, disease duration, HLA-B27, and bDMARDs use. The direct effect signifies the direct effect of alcohol on spinal progression. The indirect effect means the effect of alcohol transmitted through ASDAS on spinal progression. The total effect includes both the direct and the indirect effect of alcohol on spinal progression. ASDAS: Ankylosing Spondylitis Disease Activity Score; aOR: adjusted Odds Ratio.

Disclosures: E. Gendron: None; **S. Maguire**: None; **Y. Deng**: None; **N. Haroon**: AbbVie, 2, Novartis, 2, UCB Pharma, 2; **R. Inman**: Abbvie, 2, Janssen, 2, novartis, 2, 5, UCB, 2.

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