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Original Article

Alcohol Use, Smoking, and Their Association with Early Onset and Severity of Psoriasis: a Case-Control Study

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Abstract

Background: Psoriasis is a chronic inflammatory disorder shaped by genetic, immune, and environmental factors. Alcohol and smoking may worsen the severity and trigger early onset. This study evaluated their prevalence, patterns, and impact among male patients.

Methods: A case-control study was conducted at a tertiary care center, including 153 male psoriasis patients and 153 age-matched male controls. Data were collected on alcohol use, smoking status, Psoriasis Area and Severity Index (PASI), Alcohol Use Disorders Identification Test (AUDIT), and Addiction Severity Index (ASI). Appropriate statistical analyses were applied.

Findings: Alcohol use was more frequent in cases than controls (60.8% vs. 44.4%, $P=0.005$). Among cases, alcohol users had earlier psoriasis onset (30.5 vs. 35.2 years, $P=0.002$), higher AUDIT (18.6 vs. 15.2, $P=0.003$), and higher ASI scores (20.1 vs. 14.2, $P<0.001$). PASI correlated with AUDIT ($r=0.29$, $P=0.005$), ASI ($r=0.32$, $P=0.002$), smoking pack-years ($r=0.25$, $P=0.016$), and inversely with onset age ($r=-0.27$, $P=0.008$). In pustular psoriasis, alcohol users had higher PASI (27.8 vs. 22.1, $P=0.014$) and earlier onset (28.7 vs. 34.1 years, $P=0.009$). Logistic regression identified alcohol use, AUDIT, ASI, and smoking as predictors of psoriasis.

Conclusion: Alcohol use, smoking, and addiction severity are linked with early-onset psoriasis and greater severity, particularly in pustular subtypes, underscoring the need for lifestyle modification and targeted interventions.

Keywords: Psoriasis, Alcohol drinking, Smoking, Early diagnosis, Case-control studies

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Introduction

Psoriasis is a chronic, immune-mediated skin disorder affecting 2–3% of the global population, characterized by erythematous, scaly plaques that significantly impact quality of life.[1] Its multifactorial etiology involves genetic predisposition, immune dysregulation, and environmental triggers, with lifestyle factors like alcohol consumption and smoking increasingly recognized as potential exacerbators.[2] Alcohol is hypothesized to worsen psoriasis and contribute to earlier disease onset through mechanisms such as increased pro-inflammatory cytokines (e.g., TNF-α), oxidative stress, and impaired skin barrier function.[3] These effects may amplify systemic inflammation and accelerate psoriasis development, as measured by the Psoriasis Area and Severity Index (PASI) and age of onset.[4]

Several studies have explored the relationship between alcohol and psoriasis, with evidence suggesting that heavy alcohol consumption may increase disease incidence, severity, and earlier onset.[5] For instance, Qureshi et al. found a dose-dependent association between alcohol intake and psoriasis risk in women, though data in men are less consistent.[6] Alcohol's role in triggering psychological stress, a known psoriasis aggravator, may also contribute to early onset.[7] The Alcohol Use Disorders Identification Test (AUDIT) and Addiction Severity Index (ASI) are validated tools for assessing alcohol-related risk and broader addiction impacts.[8,9] However, few studies have integrated AUDIT, ASI, and smoking status to assess alcohol's role in early onset psoriasis, particularly in South Asian populations.[10]

Early onset psoriasis, typically defined as onset before age 40, is associated with a stronger genetic predisposition and more severe disease course compared to late-onset psoriasis.[11] In South Asian populations, where alcohol consumption patterns may differ due to cultural and socioeconomic factors, the role of alcohol in precipitating early onset psoriasis remains understudied.[10] Heavy alcohol use may exacerbate immune dysregulation and trigger psoriasis at a younger age, particularly in genetically susceptible individuals.[3] This case-control study aimed to assess the prevalence and patterns of

alcohol use and smoking among male patients with early onset psoriasis, their association with age of onset and disease severity (PASI scores), compared to age-matched controls, adjusting for confounders like age and socioeconomic status.

Methodology

Study Design and Participants

This case-control study was conducted at a tertiary care centre and included 153 male patients diagnosed with psoriasis by a dermatologist (cases) and 153 age-matched male controls without psoriasis or autoimmune conditions, for a total of 306 participants. The study was approved by the Institutional Ethics Committee (Ethical code: EC/NEW/INST/2025/3273). Written informed consent was obtained from all participants before inclusion.

Data Collection

Data were collected on age, socioeconomic status, alcohol use (yes/no), smoking status (smoker/non-smoker, pack-years), alcohol type, quantity (units/week), duration (years), AUDIT scores,[8] ASI scores,[9] psoriasis type (plaque, pustular, guttate), age of psoriasis onset, psoriasis duration (years), and PASI scores.[12] The AUDIT assessed alcohol-related risk, ASI evaluated addiction severity, and smoking status was included as a confounder.

Statistical Analysis

Normality was assessed using the Shapiro-Wilk test. Alcohol use and smoking prevalence were compared using Chi-square tests. Non-normal AUDIT, ASI, PASI scores, and age of onset (Shapiro-Wilk $P<0.05$) were compared using Mann-Whitney U tests. Spearman's correlations were used to assess the associations between PASI, AUDIT, ASI, and age of onset. Binary logistic regression was used to evaluate predictors of early onset psoriasis presence, and linear regression was used to analyze PASI score predictors among cases, adjusting for age, socioeconomic status, and smoking. Significance levels were set at $P<0.05$.

Results

The study included 306 male participants (153 cases, 153 controls). Mean age was 38.5 years (SD = 11.2) for cases and 40.1 years (SD = 12.3) for controls ($P=0.216$, t-test). Socioeconomic status was similar ($P=0.347$, Chi-square test). Cases had plaque (45.1%, n = 69), pustular (35.3%, n = 54), or guttate (19.6%, n = 30) psoriasis, with mean PASI score of 24.2 (SD = 8.7), psoriasis duration of 8.1 years (SD = 4.3), and mean age of onset of 32.8 years (SD = 9.1) (Table 1). Alcohol use was higher in cases (60.8%, n = 93) than controls (44.4%, n = 68) ($\chi^2=7.82$, $P=0.005$). Smoking prevalence was higher in cases (48.4%, n = 74) than controls (34.6%, n = 53) ($\chi^2=5.92$, $P=0.015$). Alcohol types were similar ($P=0.189$, Chi-square test) (Table 2). Among alcohol users, cases had earlier psoriasis onset (30.5 years, SD = 8.8 vs. 35.2 years, SD = 9.4, $P=0.002$), higher mean AUDIT (18.6, SD = 7.1 vs. 15.2, SD = 6.8, $P=0.003$), and ASI (20.1, SD = 7.2 vs. 14.2, SD = 6.5, $P<0.001$) scores (Mann-Whitney U test) (Table 3).

Table 1. Baseline Characteristics of Cases and Controls

Characteristic	Cases (n = 153)	Controls (n = 153)	*P* value
Age, mean (SD), years	38.5 (11.2)	40.1 (12.3)	0.216
Socioeconomic Status, n (%):			0.347
– High	60 (39.2%)	68 (44.4%)	
– Middle	50 (32.7%)	49 (32.0%)	
– Low	43 (28.1%)	36 (23.5%)	
Smoking, n (%):			0.015*
– Yes	74 (48.4%)	53 (34.6%)	
– No	79 (51.6%)	100 (65.4%)	
Psoriasis Type, n (%):			–
– Plaque	69 (45.1%)	–	
– Pustular	54 (35.3%)	–	
– Guttate	30 (19.6%)	–	
PASI Score, mean (SD)	24.2 (8.7)	–	–
Psoriasis Duration, mean (SD), years	8.1 (4.3)	–	–
Age of Onset, mean (SD), years	32.8 (9.1)	–	–

*Abbreviations: PASI = Psoriasis Area and Severity Index; SD = Standard Deviation.
*$P<0.05$ considered statistically significant.

Among cases, PASI scores were higher in alcohol users (25.1, SD = 8.9) than non-users (22.8, SD = 8.3) ($P=0.112$), and smokers (26.3, SD = 9.0) than non-smokers (22.5, SD = 8.2) ($P=0.024$). PASI correlated with AUDIT (r = 0.29, $P=0.005$), ASI (r = 0.32, $P=0.002$), age of onset (r = -0.27, $P=0.008$), and pack-years (r = 0.25, $P=0.016$) (Table 4).

Logistic regression showed alcohol use (OR = 1.72, 95% CI [1.10, 2.70], $P=0.018$), AUDIT (OR = 1.06, 95% CI [1.02, 1.10], $P=0.003$), ASI (OR = 1.08, 95% CI [1.04, 1.12], $P<0.001$), and smoking (OR = 1.45, 95% CI [1.01, 2.08], $P=0.042$) as predictors of early onset psoriasis. Linear regression among cases showed ASI ($\beta=0.26$, $P=0.004$), early age of onset ($\beta=-0.22$, $P=0.012$), and smoking ($\beta=0.20$, $P=0.032$) as predictors of PASI (Table 5).

In pustular psoriasis, alcohol users (n = 34) had higher PASI scores (27.8, SD = 8.1) than non-users (n = 20, 22.1, SD = 7.4) ($P=0.014$) and earlier onset (28.7 years, SD = 8.0 vs. 34.1 years, SD = 9.0, $P=0.009$). ASI scores correlated more strongly (r = 0.41, $P=0.008$) than AUDIT (r = 0.30, $P=0.039$) with PASI (Table 6).

Discussion

This case-control study confirms a significant association between alcohol use, smoking, and early onset psoriasis among male patients, with 60.8% of cases reporting alcohol

use compared to 44.4% of controls ($P = 0.005$) and 48.4% reporting smoking compared to 34.6% ($P = 0.015$). Alcohol users had an earlier age of psoriasis onset (30.5 years vs. 35.2 years, $P = 0.002$), suggesting alcohol as a trigger for earlier disease manifestation. Higher AUDIT (18.6 vs. 15.2, $P = 0.003$) and ASI (20.1 vs. 14.2, $P < 0.001$) scores in cases indicate greater alcohol-related risk and addiction severity.[3,4] The use of AUDIT and ASI strengthens the study's ability to capture a comprehensive picture of alcohol use.[8,9] Smoking's inclusion as a confounder (OR = 1.45, $P = 0.042$) aligns with prior research linking tobacco use to psoriasis risk.[13]

The correlations between PASI scores and AUDIT (r = 0.29, $P = 0.005$), ASI (r = 0.32, $P = 0.002$), age of onset (r = -0.27, $P = 0.008$), and pack-years (r = 0.25, $P = 0.016$) suggest that alcohol, smoking, and early onset contribute to psoriasis severity. The stronger association of ASI scores with PASI (β = 0.26, $P = 0.004$) and early onset (β = -0.22,

Table 2. Alcohol Use and Smoking Characteristics

Characteristic	Cases (n = 153)	Controls (n = 153)	*P* value
Alcohol Use, n (%):			0.005*
– Yes	93 (60.8%)	68 (44.4%)	
– No	60 (39.2%)	85 (55.6%)	
Alcohol Type (among users), n (%):			0.189
– Beer	40 (42.7%)	33 (48.5%)	
– Whiskey	27 (29.0%)	19 (27.9%)	
– Wine	18 (19.4%)	14 (20.6%)	
– Brandy	9 (9.7%)	2 (2.9%)	
Smoking (pack-years), mean (SD)	5.2 (4.8)	3.8 (3.9)	0.028*

Abbreviations: SD = Standard Deviation.
*$P < 0.05$ considered statistically significant.

Table 3. AUDIT, ASI, and Age of Onset Among Alcohol Users

Score	Cases (n = 93)	Controls (n = 68)	*P* value
AUDIT Score, mean (SD)	18.6 (7.1)	15.2 (6.8)	0.003*
ASI Score, mean (SD)	20.1 (7.2)	14.2 (6.5)	<0.001*
Age of Onset, mean (SD), years	30.5 (8.8)	–	–

*Abbreviations: AUDIT = Alcohol Use Disorders Identification Test; ASI = Addiction Severity Index; SD = Standard Deviation.
*$P < 0.05$ considered statistically significant.

Table 4. Correlations in Alcohol-Using Cases (n = 93)

Variable	Spearman Correlation (r)	*P* value
AUDIT Score	0.29	0.005*
ASI Score	0.32	0.002*
Age of Onset	–0.27	0.008*
Pack-Years	0.25	0.016*

*Abbreviations: AUDIT = Alcohol Use Disorders Identification Test; ASI = Addiction Severity Index.
*$P < 0.05$ considered statistically significant.

Table 5. Results of Multivariate Analysis

Model	Predictor	Estimate	95% CI	*P* value
Logistic Regression (Outcome: Psoriasis Presence)				
Alcohol Use (Yes vs. No)	OR = 1.72	[1.10, 2.70]	0.018*	
AUDIT Score (per unit)	OR = 1.06	[1.02, 1.10]	0.003*	
ASI Score (per unit)	OR = 1.08	[1.04, 1.12]	<0.001*	
Smoking (Yes vs. No)	OR = 1.45	[1.01, 2.08]	0.042*	
Age (years)	OR = 0.99	[0.97, 1.01]	0.245	
Socioeconomic Status	OR = 0.92	[0.75, 1.13]	0.398	
Linear Regression (Outcome: PASI Score, Cases only)				
AUDIT Score	β = 0.19	[0.03, 0.35]	0.021*	
ASI Score	β = 0.26	[0.08, 0.44]	0.004*	
Age of Onset	β = –0.22	[–0.39, –0.05]	0.012*	
Smoking (pack-years)	β = 0.20	[0.02, 0.38]	0.032*	
Age (years)	β = 0.05	[–0.02, 0.12]	0.167	
Socioeconomic Status	β = –0.08	[–0.25, 0.09]	0.341	

*Abbreviations: OR = Odds Ratio; β = Beta coefficient; CI = Confidence Interval; AUDIT = Alcohol Use Disorders Identification Test; ASI = Addiction Severity Index; PASI = Psoriasis Area and Severity Index.
*$P < 0.05$ considered statistically significant.

Table 6. PASI Scores and Age of Onset in Pustular Psoriasis Cases

Group	PASI Score, mean (SD)	Age of Onset, mean (SD), years	n	*P* value
Alcohol Users	27.8 (8.1)	28.7 (8.0)	34	0.014* (PASI), 0.009* (Onset)
Non-Users	22.1 (7.4)	34.1 (9.0)	20	

*Abbreviations: PASI = Psoriasis Area and Severity Index; SD = Standard Deviation.
*$P < 0.05$ considered statistically significant.

$P = 0.012$) highlights broader impact of addiction severity.[9] In pustular psoriasis, alcohol users had higher PASI scores (27.8 vs. 22.1, $P = 0.014$) and earlier onset (28.7 vs. 34.1 years, $P = 0.009$), suggesting a subtype-specific sensitivity to alcohol's inflammatory effects.[14]

The finding of early onset psoriasis among alcohol users underscores the importance of lifestyle factors in disease initiation. Early onset psoriasis is often linked to a stronger genetic predisposition and more severe clinical course, which may be exacerbated by alcohol's pro-inflammatory effects.[11] The earlier age of onset in alcohol users (30.5 vs. 35.2 years) suggests that alcohol may act as an environmental trigger that accelerates disease expression in genetically susceptible individuals. This is particularly relevant in the South Asian context, where cultural attitudes toward alcohol consumption may influence its impact on chronic diseases.[10] These findings emphasize the need for early screening and intervention in young male patients with a history of alcohol use to potentially delay or mitigate psoriasis onset.

The inclusion of smoking as a confounder adds depth to the study's findings, as smoking is a well-established risk factor for psoriasis.[13] The higher smoking prevalence in cases (48.4% vs. 34.6%, $P = 0.015$) and its association with PASI scores ($\beta = 0.20$, $P = 0.032$) suggest a synergistic effect with alcohol in exacerbating psoriasis severity and possibly contributing to early onset. Both alcohol and tobacco may amplify systemic inflammation through shared pathways, such as increased oxidative stress and cytokine production, which could explain their combined impact on disease expression.[3,13] Future studies should explore the interaction between alcohol and smoking in greater detail, particularly in the context of early onset psoriasis.

The biological mechanisms linking alcohol, smoking, and early onset psoriasis likely involve complex immunological pathways. Alcohol is known to upregulate pro-inflammatory cytokines, such as TNF-α and IL-6, which are central to psoriasis pathogenesis.[3] Similarly, smoking induces oxidative stress and activates inflammatory pathways that may exacerbate psoriasis.[13] In pustular psoriasis, the heightened neutrophil activation observed may be particularly sensitive to these triggers, explaining the stronger associations seen in this subtype.[14] These mechanisms could also contribute to earlier disease onset by lowering the threshold for immune dysregulation in susceptible individuals. Further research into these pathways could inform targeted therapies for early onset psoriasis.

Clinical Implications

Routine screening for alcohol use (using AUDIT/ASI) and smoking status could identify high-risk patients, particularly those with pustular psoriasis and early onset, for targeted interventions.[8,9] Behavioral interventions, including alcohol and smoking cessation programs, could reduce disease severity and delay onset, improving quality of life.[7]

Future Research Directions

Longitudinal studies are needed to establish causality between alcohol, smoking, early onset, and psoriasis. Investigating biological mechanisms, such as cytokine profiles in pustular psoriasis, could elucidate subtype-specific pathways.[14] Interventional studies on alcohol and smoking cessation in psoriasis patients are warranted.

Strengths and Limitations

Strengths: The case-control design, use of AUDIT and ASI, and inclusion of smoking and early onset data provide a comprehensive analysis. Subgroup analysis of pustular psoriasis adds novel insights.[14] *Limitations:* The case-control design limits causality inferences. Self-reported alcohol and smoking data may introduce bias. The hospital-based setting may overrepresent severe cases.[1]

Conclusion

Alcohol use, smoking, and addiction severity are significantly associated with early onset psoriasis, its severity, and particularly in pustular psoriasis. Routine screening and targeted interventions are recommended [15].

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Authors' Contribution

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Validation: K. S Ravishankar.
Visualization: Shankar Siddhan
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Competing Interests

The authors declare no conflicts of interest related to this publication.

Ethical Approval

The study was approved by the Institutional Ethics Committee of Government Mohan Kumaramangalam Medical College Hospital, Salem (Ethical code: EC/NEW/INST/2025/3273). Written informed consent was obtained from all participants before inclusion in the study. Confidentiality of data and the rights of participants were maintained throughout the research.

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