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Original Article

Effect of cigarette smoking and alcohol consumption on disease activity and physical functioning in ankylosing spondylitis: a cross-sectional study

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Abstract: The effect of cigarette smoking and alcohol consumption on the disease activity and physical functioning in ankylosing spondylitis (AS) is currently understated. Present study aims to investigate the relationship between them. A total of 425 patients with AS were recruited in the study and their smoking and drinking habit were investigated with a semi-quantitative food frequency questionnaire. Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), Bath Ankylosing Spondylitis Functional Index (BASFI), and Metrology Index (BASMI) were evaluated. Parameters including fingertip-to-floor distance, overall assessment of health, nocturnal pain, total back pain and morning stiffness were analyzed as well. Blood erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were determined. For 118 (27.8%) AS patients with smoking habit, the scorings of BASDAI, BASFI, BASMI and other physical parameters (including fingertip-to-floor, overall assessment of health, nocturnal pain and total back pain) were higher than those in patients without smoking. 101 (23.8%) AS patients with alcohol consumption demonstrated significantly higher scores in BASMI ($P < 0.05$). In hierarchical multiple regression analysis, the cigarette smoking and alcohol consumption variables contributed to the variance in BASDAI scores, adding an additional 1.6% to the overall R-square, resulting in a final R-square of 5.1%. Smoking has a negative effect on disease activity of patients with AS and the patients' physical functioning. Alcohol consumption would aggravate the overall physical functioning of AS patient. The results indicated the potential benefit of quitting smoking and drinking for AS patients.

Keywords: Ankylosing spondylitis, disease activity, inflammation, cigarette smoking, alcohol consumption

Introduction

Ankylosing spondylitis (AS) is a chronic systemic inflammatory disease with various symptoms mainly associated with sacroiliac joint, spine, peripheral joints, and entheses [1]. Tobacco and alcohol consumption are major stimulators of the disease occurrence and contribute to nearly seven million deaths each year worldwide as reported [2].

Smoking is not only involved in the pathogenesis of multiple rheumatic diseases, but also makes the treatment much more difficult in patients comparing with non-smoking patients [3-6]. Some studies documented that smoking could induce rheumatoid arthritis (RA) with increased risks, more severe disease activity, and significant arthrosis damage, which resulted in higher risks of functional disability [7, 8]. It has been highly suggested that there are closely association between cigarette smoking and alcohol consumption. Smokers are of 10 times likelihood to develop alcoholism than their non-smoking counterparts [9]. Alcohol consumption may play a role in preventing of RA. Källberg H et al [10] reported that alcohol consumption could decrease the risk of RA, as well as reduced the risk of smoker carrying HLA-DRB1 SE alleles susceptible to RA. Other studies indicated that moderate alcohol consumption could relieve the disease activity of RA and improve the prognosis of the disease [11]. The cigarette smokers are generally more susceptible in the development of systemic lupus erythematosus (SLE), however such risk could be

modified inversely by alcohol consumption. SLE patients with smoking were vulnerable to the disease deterioration and were often required for more complicated medicines than non-smokers [12-14]. Callahan LF et al. [15] reported that smoking was protective for men with radiographic osteoarthritis (OA) or bilateral radiographic OA (but not for the women). On the other hand, alcohol consumption was protective for women in OA and symptomatic OA (but not for men).

Ward MM et al. [16, 17] studied that BASFI scores were higher among current smokers compared to former/nonsmokers. Cigarette smoking has been associated with radiographic severity (being in the top quartile of Bath AS Radiology Index-Spine/duration of AS) and the accuracy of the prognosis of radiographic severity in AS is improved by knowing the age at disease onset, sex, smoking history. Poddubnyy D et al. [18] concluded that cigarette smoking was associated with spinal radiographic progression in patients with early axial spondyloarthritis (SpA). The epidemiological investigation participated by 75 AS patients in Taiwan also showed that among the physical mobility parameters, modified Schober's index, cervical rotation, later lumbar flexion, chest expansion and occiput-to-wall distances were significantly impaired in smoking AS patients compared with non-smoking ones. Systemic inflammation parameter, ESR was also significantly higher in smoking AS patients. Moreover, the smoking intensity is positively correlated with BASFI significantly [19]. Currently the relationship between alcohol consumption and AS was underreported. Only one study based on nationwide sickness insurance data in Finland in 1989 confirmed that uncontrolled use of alcohol is an important determinant in the surplus of deaths from accidents and violence in Finnish patients with AS. The lifespan of the studies with AS was shorter by 6-8 years compared to Finnish population average [20].

Although it has been widely accepted that smoking has a negative effect on AS patients, the achieved results were somehow limited by small sample size and the effect of cigarette smoking on overall conditions of AS patients is ill defined. Besides, there's no research on the effect of alcohol consumption on disease activity of AS and patients' physical functioning.

The objectives of this study were (1) to determine the effect of cigarette smoking and alcohol consumption on disease activity of AS and patients' physical functioning; and (2) to evaluate the statistical contribution of tobacco and alcohol consumption to BASDAI scores.

Patients and methods

Patients

For long-term follow-up, patients were recruited from rheumatology clinics and departments in the Fuzhou General Hospital. All patients met the 1984 modified New York classification criteria for AS [21]. Patients who had malignancy, fibromyalgia, serious infections or systemic diseases, and other chronic diseases were excluded from the study. The study was approved by the ethics committee of Fuzhou General Hospital, and all participants gave their written informed consent, according to the Declaration of Helsinki. Demographic characteristics, age, gender, height, weight and disease duration were documented for each patient. The patients were subjected to a detailed investigation including hemogram, liver, and kidney function tests, to exclude other diseases that may cause divergence to evaluated parameters.

The disease activity was evaluated with BASDAI, CRP (normal $\leq$ 0.8 mg/dl), ESR (normal $\leq$ 20 mm/h), and duration of morning stiffness; the functional state was assessed by BASFI, metrology measurements by fingertip-to-floor distance, and BASMI. Overall assessment of health score in AS patients was determined from a visual analogue scale (VAS), recording from 0 cm (none) to 10 cm (very severe); the pain score by visual analogue scale (VAS, 0 to 10 cm), including nocturnal pain (last week/spine/at night due to AS) and total back pain (last week/spine/due to AS).

Smoking status

Information regarding smoking status (current, never) was collected at the time of enrollment, and pack-years of smoking served as the measure of cumulative exposure. Never smoking was defined as having smoked fewer than 100 cigarettes in the subject's lifetime. Information specific to 'second-hand', former smokers or other environmental smoking exposures were not collected as part of this study [22].

Table 1. Demographic and disease-related characteristics of patients with ankylosing spondylitis

Variables	Mean ± SD (%)
Age (years)[a]	29.2 ± 8.3
Gender: male n (%)[b]	348 (81.9%)
Disease duration(years)[a]	7.5 ± 5.8
HLA-B27 (+)[b]	335 (78.8%)
smoking[b]	307 (72.2%)
Average daily amount of smoking (cigarettes)[a]	4.3±19.7
Total tobacco consumption (cigarettes)[a]	39286.1 ± 49344.9 (730~273750)
Average daily alcohol consumption (g)[a]	10.7 ± 7.3
Total alcohol consumption (g)[a]	63534.7 ± 151705.1 (182.5~983894.7)
non-drinkers[b]	324 (76.2%)
moderate drinking[b]	63 (14.8%)
heavy drinking[b]	38 (8.9%)
Morning stiffness (minutes)[a]	11.1 ± 20.4
Nocturnal pain (VAS)[a]	4.1 ± 2.9
Total back pain (VAS)[a]	3.9 ± 2.5
Overall assessment of health (VAS)[a]	5.9 ± 3.2
BASDAI[a]	3.8 ± 1.9
BASFI (VAS)[a]	1.6 ± 2.0
BASMI[a]	1.7 ± 2.1
Fingertip-to-floor distance (cm)[a]	11.8 ± 15.5
ESR (mm/h)[a]	14.7 ± 17.0
CRP (mg/dl)[a]	1.38 ± 2.0

[a]Mean ± SD. [b]Percentage. N = 425. VAS, visual analogue scale; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BASFI, Bath Ankylosing Spondylitis Functional Index; BASMI, Bath Ankylosing Spondylitis Metrology Index; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.

Alcohol consumption

Participants were asked to report their average frequency of consumption of specified foods and beverages during the previous 6 months. Despite of the separate items including beer, wine and liquor, participants who drink more than once a week were categorized as "alcohol drinker", and those drink less than once a week or have no alcohol intake for more than 1 year were considered as non-drinking patients. Alcohol consumption was assessed with a semi-quantitative food frequency questionnaire including separate items for beer, wine, and liquor. We specified standard portions as a glass, bottle, or can of beer; a 4-oz glass of wine; and a shot of liquor. For each beverage, participants were asked to estimate their average consumption over the past year. We calculated the average number of glasses of alcoholic beverage per week (1 standard glass = 15 g of alcohol, corresponding to approximately 500 ml of beer, 150 ml of wine, and 50 ml of liquor) combining information about frequency and amount of drinking. Based on the daily alcohol intake, the participants were arranged to be heavy drinking group (daily alcohol intake > 25 g/d), moderate drinking group (daily alcohol intake ≤ 25 g/d) and non-drinkers [23].

Statistical analysis

Descriptive statistics were used for assessing the parameters related to disease. The differences in terms of variables studied in patients who are grouped as experiencing high disease activity and low disease activity, according to the BASDAI, were evaluated with independent sample chi-square test. The relations between smoking, alcohol consumption and the other evaluation parameters were examined with Mann-Whitney u test and analysis of variance. Hierarchic multiple regression analysis was chosen to analyze the contribution of demographic, smoking and alcohol consumption variables to BASDAI. Statistical analysis was

Table 2. Comparison of the Variables of BASDAI ≥ 4 and BASDAI < 4

	BASDAI (≥ 4)	BASDAI (< 4)	$\chi^{2/t}$	*P*
	N = 184 (43.3%)	N = 241 (56.7%)		
Age (years)	30.2 ± 8.5	28.5 ± 8.1	-2.1	0.036
ESR	16.6 ± 16.8	13.3 ± 17.1	-1.99	0.048
CRP	105	88	17.8	< 0.0001
Drinking			7.4	0.024
non-drinkers	133	191		
moderate drinking	37	26		
heavy drinking	14	24		
smoking	62	56	5.69	0.017
Disease duration(years)			7.9	0.048
<1 year	17	24		
1~5 years	41	71		
5~10 years	72	102		
> 10 years	54	44		
HLA-B27 (+)	145	190	0.00001	0.993
BASFI	2.8 ± 2.3	0.7 ± 1.0	-11.8	< 0.0001
BASMI	2.2 ± 2.3	1.4 ± 1.9	-3.8	< 0.0001
Fingertip-to-floor distance (cm)	15.0 ± 15.0	9.4 ± 15.5	-3.7	< 0.0001

N = 425; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BASFI, Bath Ankylosing Spondylitis Functional Index; BASMI, Bath Ankylosing Spondylitis Metrology Index; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.

Table 3. Comparison between smoking and non-smoking patients

	BASDAI	BASFI	BASMI	Overall assessment of health (VAS)	Nocturnal pain (VAS)	Total back pain (VAS)	ESR	Number with CRP > 0.8 mg/dl	Morning stiffness	Fingertip-to-floor distance
smoking	4.2 ± 2.0	2.2 ± 2.2	2.2 ± 2.3	6.6 ± 2.7	4.8 ± 3.1	4.3 ± 2.7	14.6 ± 17.5	62	14.0 ± 26.3	14.4 ± 12.8
non-smoking	3.6 ± 1.9	1.4 ± 1.8	1.5 ± 2.0	5.6 ± 3.3	3.9 ± 2.8	3.7 ± 2.4	14.8 ± 16.9	131	9.9 ± 17.6	10.8 ± 16.3
Z/χ^2	-3.05	-3.54	-3.35	-3.47	-2.69	-2.08	-0.57	3.4	-1.04	-3.70
P	0.00	0.00	0.00	0.00	0.01	0.04	0.57	0.067	0.30	0.00

BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BASFI, Bath Ankylosing Spondylitis Functional Index; BASMI, Bath Ankylosing Spondylitis Metrology Index; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.

performed by using SPSS 17.0, and the level of significance was set at $P < 0.05$.

Results

Patient characteristics

Among the enrolled 425 AS patients, 348 (81.9%) were male. The average age is 29.2 ± 8.3 yrs and average disease duration lasts for 7.5 ± 5.8 yrs. 335 (78.8%) cases were HLA-B27 positive and 118 cases (27.8%) were smokers. The total tobacco consumption counts to 39286.1 ± 49344.9 cigarettes (min = 730 cigarettes, max = 273750 cigarettes). 101 cases (23.8%) were reported with total alcohol consumption of 63534.7 ± 151705.1 g (ranged from 182.5 g to 983894.7 g). 324 cases (76.2%) were categorized as non-drinkers, while for the drinking patients, the moderate and heavy drinking one occupied 63 (14.8%) and 38 (8.9%) respectively. Other general demographic and clinical characteristics related with AS were listed in **Table 1**.

Chi-square test

Patients were allocated to 2 groups based on BASDAI results: 184 AS patients got 4 scores or above and the rest 241 cases (56.7%) were scored less than 4 [24]. Significant difference was observed in age, the case number with CRP ≤ 0.8 mg/dl, case number of smokers, disease duration, and value of BASFI, BASMI or figure-to-floor distance ($P < 0.05$) (see **Table 2**).

Table 4. Comparison among heavy drinking group, moderate drinking group and non-drinkers

	BASDAI	BASFI	BASMI	Overall assessment of health (VAS)	Nocturnal pain (VAS)	Total back pain (VAS)	ESR	Number with CRP > 0.8 mg/dl	Morning stiffness	Fingertip-to-floor distance
heavy drinking	3.4 ± 1.8	1.8 ± 2.5	2.4 ± 2.4	6.1 ± 3.0	3.9 ± 2.9	3.4 ± 2.4	12.5 ± 13.8	20	16.5 ± 26.6	14.8 ± 16.9
moderate drinking	4.2 ± 1.7	1.8 ± 1.9	2.1 ± 2.2	6.5±2.8	5.0±2.6	4.5±2.3	14.2 ± 17.1	26	11.2 ± 16.6	11.9 ± 11.1
non- drinking	3.7 ± 2.0	1.5 ± 2.0	1.6 ± 2.0	5.7 ± 3.3	4.0 ± 2.9	3.8 ± 2.5	15.1 ± 17.4	147	10.4 ± 20.2	11.5 ± 16.0
F/2	2.35	-0.99	4.02	1.96	3.43	3.37	0.43	1.24	1.54	0.762
P	0.097	0.32	0.019	0.142	0.033	0.035	0.652	0.539	0.216	0.467

BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BASFI, Bath Ankylosing Spondylitis Functional Index; BASMI, Bath Ankylosing Spondylitis Metrology Index; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.

Table 5. Hierarchic multiple regression analysis of demographic, smoking, and drinking variables in relation to BASDAI

	Model 1		Model 2		Model 3		Model 4	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
Age	1.02 (1.00~1.05)	0.10	1.02 (0.99~1.05)	0.14	1.02 (0.99~1.05)	0.12	1.02 (0.99~1.05)	0.14
Gender	1.12 (0.59~2.14)	0.72	0.97 (0.50~1.87)	0.92	1.06 (0.55~2.04)	0.85	0.94 (0.48~1.84)	0.86
Height	1.01 (0.98~1.04)	0.53	1.01 (0.98~1.04)	0.58	1.01 (0.98~1.04)	0.52	1.01 (0.98~1.04)	0.55
Weight	0.99 (0.97~1.00)	0.12	0.97 (0.97~1.01)	0.15	0.99 (0.97~1.01)	0.21	0.99 (0.97~1.01)	0.23
Disease duration		0.18		0.24		0.16		0.21
1~5 years	0.76 (0.36~1.60)	0.47	0.77 (0.37~1.61)	0.49	0.70 (0.33~1.47)	0.34	0.71 (0.33~1.49)	0.36
5~10 years	0.92 (0.45~1.88)	0.81	0.89 (0.43~1.84)	0.75	0.81 (0.39~1.69)	0.58	0.81 (0.39~1.67)	0.56
>10 years	1.45 (0.65~3.20)	0.36	1.38 (0.62~3.06)	0.43	1.31 (0.59~2.92)	0.51	1.27 (0.57~2.83)	0.57
HLA-B27	0.96 (0.60~1.56)	0.88	0.92 (0.57~1.50)	0.75	1.00 (0.61~1.62)	0.99	0.96 (0.59~1.56)	0.86
smoking			1.62 (1.03~2.55)	0.04			1.53 (0.96~2.44)	0.07
drinking						0.04*		0.06
moderate drinking					1.86 (1.05~3.28)	0.03*	1.72 (0.97~3.07)	0.07
heavy drinking					0.66 (0.31~1.38)	0.27	0.63 (0.30~1.34)	0.23
R^2* (%)	2.8		3.8		4.4		5.1	
Adjusted R^2	3.8		5.1		5.8		6.8	

BASDAI, Bath Ankylosing Spondylitis Disease Activity Index.

Mann-Whitney u test and analysis of variance

The Mann-Whitney u test was performed between patients who were smoking and those who were not. Compared to non-smoking patients, those with tobacco use scored significantly higher in BASDAI, BASFI, BASMI, and multiple parameters of functional state including fingertip-to-floor distance, overall assessment of health (VAS), nocturnal pain (VAS) and total back pain (VAS) ($P < 0.05$). With regard to age, ESR, case proportion with CRP > 0.8 mg/dl or morning stiffness, no statistically significant differences were found between the two groups ($P > 0.05$) (see **Table 3**).

As shown in **Table 4**, among heavy drinking group, moderate drinking group and non-drinkers, significant difference was detected in BASMI, nocturnal pain (VAS) and total back pain (VAS) ($P < 0.05$), while the results for BASDAI, BASFI, overall assessment of health VAS score, ESR, case proportion with CRP > 0.8 mg/dl, morning stiffness or fingertip-to-floor distance among the three groups were similar ($P > 0.05$). The heavy drinking group scored significantly higher (2.4 ± 2.4) than non-drinking group (1.6 ± 2.0) ($P < 0.05$), and their scores for nocturnal pain (VAS) (3.9 ± 2.9) and total back pain (VAS) (3.4 ± 2.4) were both remarkably lower than moderate drinking group, which were (5.0 ± 2.6) and (4.5 ± 2.3), respectively ($P < 0.05$).

Hierarchical multiple regression analysis

Hierarchical multiple regression analysis was performed with BASDAI was chosen as independent variable and the rest parameters as dependent variable (**Table 5**).

Model 1 (demographic model) tests the contribution of demographic variables (including age, gender, height, weight, duration, HLA-B27) to BASDAI. The disease duration was stratified as less than 1 year, 1 to 5 years, 5 to 10 years, and over 10 years.

Model 2 (smoking model) tests the contributions of cigarette smoking to BASDAI after controlling for the demographic variables.

Model 3 (drinking model) calculates the contribution from alcohol intake to BASDAI after controlling for the demographic variables. Model 1 to 3 are the results of hierarchic regression analysis, which are usually done in research to determine the importance of factor variables once the other predictor variables have been already been entered into the equation [25].

Model 4 is the standard regression analysis in which all of the variables were entered simultaneously into the model to assess the relative contributions of these variables to BASDAI. This model is used for standard regression analysis for interpreting the interrelation between independent and dependent variable (BASDAI). The first model testing the contributions of demographic variables to BASDAI was found to be statistically significant (R^2 = 0.028). In this model, R^2 is the determinant factor reflecting the contribution of the analyzed variable to the target independent variable. Introduction of the smoking variable (model 2) resulted in a significant increase in the R^2 valve (R^2 = 0.038). Further addition of the drinking variable increased the R^2 to 0.044. After inclusion of the model 4, the final full model (standard multiple regression analysis) in which all variables entered simultaneously could explain the 5.1% of the total variance in BASDAI.

Discussion

To our knowledge, this study is for the first time to show the association of cigarette smoking and alcohol consumption in Chinese AS patients. It is general acknowledged that the management of pain and functional disability are two most challengeable focuses in AS patients. All the therapeutic approaches available currently are mainly conducted for relieving the pain and preventing the disability of AS patients.

Our study showed the rates of smoking and drinking in patients were 27.8% and 23.8%, respectively. Compared to the non-smokers, smoking patients had significantly higher BASDAI, BASFI, BASMI, fingertip-to-floor distance, overall assessment of health score, nocturnal pain (VAS) score, and total back pain (VAS) score, reflecting the higher disease activity and worse physical functioning. The results also indicated that AS treatment in these patients may become more difficult, which consistent with previously reports [18, 19]. Research showed that cigarette smoking may increase tumor necrosis factor (TNF)-α in obese patients [26]. Rodigues FM et al. [27] reported that there was a decrease of TNF-α level only in blood serum at 30 days of abstinence compared to current smokers. Similar results have been discovered with male mice smoking exposure experiment, in which the mice exposed to the smoke had increased concentrations of the interleukin (IL)-6 and TNF-α compared with normal control group. Study reported by Højgaard P et al. [28] indicated that for patients with psoriatic arthritis (PsA), smokers had worse baseline patient-reported outcomes, shorter treatment adherence and poorer response to first TNF-α inhibitor therapy compared to non-smokers. This was most pronounced in men and in patients treated with infliximab or etanercept. Although no direct evidence of higher level of TNF-α in AS patients with smoking than those without smoking, smoking may have an effect on TNF-α involved signaling which provides explanation for the exacerbated disease activity for smoking AS population [29, 30]. No significant difference was observed in ESR or CRP between smoking or non-smoking patients group, which differs from previous study reports. We suspected that such divergence can be partly caused by the fact that ESR and CRP both have low sensitivity and specificity for disease activity of AS [31, 32].

From the present study, no significant difference was observed when analyzing the impact of alcohol consumption to AS activity (BASDAI, ESR, CRP and morning stiffness). The correlation between alcohol consumption with BASFI, overall assessment of health (VAS), or fingertip-to-floor distance was not significant as well. It has been supported by multiple reports that alcohol drink can be a protective factor for inflammatory diseases and RA patients with

lower disease activity. Mild to moderate alcohol consumption is associated with lower high-sensitivity-CRP concentration, which may provide the protection of cardiovascular system through anti-inflammatory mechanism [11, 33]. However, alcohol consumption does not exert significant protection for AS patients. Both BASDAI and inflammation indicator, ESR and CRP did not shown significant differences.

The nocturnal pain score (VAS) (3.9 ± 2.9) in heavy drinking was significantly lower than that in moderate drinking group, as well as the total back pain score (VAS) (3.4 ± 2.4). It was reported that pain threshold could be enhanced by alcohol consumption, however, the pain sensitivity would increase at alcohol withdrawal syndrome [34, 35], which indicating large amount of drinking could relieve pain by suppressing pro-inflammatory cytokines. Alcohol can down-regulate production of pro-inflammatory molecules via influence on innate immunity [11]. Several investigations suggested that alcohol influenced inflammation in general and arthritis in particular; alcohol was able to diminish the response to immunogens in animals as well as in humans [36-39].

Heavy drinking AS patients scored remarkably higher in BASMI compared with non-drinkers (2.4 ± 2.4 vs. 1.6 ± 2.0). As it has been accepted that exercise is the foundation of rehabilitation for AS patients, while drinking reduces central nervous system activity and the impaired muscle strengthen prevents the progress of rehabilitation [40]. Moreover, lack of exercise makes one of the alcoholism-related lifestyle. Thus it has been presumed that heavy drinking patients were evaluated higher BASMI scores due to lack of exercise [41]. Our data revealed that heavy drinking AS patients were more likely to show functional disability.

In our hierarchic multiple regression analysis to access the effect of smoking and drinking on BASDAI of AS patients, with the demographic variables controlled, the smoking variable contributed 1% change of BASDAI and 1% increase of R2. While alcohol consumption added an additional 0.6% increase to BASDAI and the overall R2. Combinative model of general characteristics and tobacco/alcohol consumption contributed to 5.1% to BASDAI. In multiple stepwise regression analysis, the relatively low effect of drinking to BASDAI of AS patients compared with smoking or general variable aligned with the results from Mann-Whitney u test that significant difference was detected between smoking and nonsmoking group, but not between drinking and sober groups. It brought us back to the point that drinking may serve as protective role by inhibiting inflammation and relieving pain.

There are several limitations in the current study. The primary limitation of the present study was its cross-sectional study design, which provided only correlational findings, precluding an understanding of directional relations between Cigarette smoking, alcohol consumption and disease status. Other limitations include: the data on smoking and alcohol consumption was retrospective, which may have caused misclassification of some patients due to problems with recall, single center study and lack of a control group.

In summary, smoking exerts negative effect on both disease activity and physical functioning of AS patients. However, alcohol consumption did not show the negative impact on it, but not included physical functioning. Abandoned of cigarette smoking and alcohol consumption favor to the treatment of AS.

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Disclosure of conflict of interest

None.

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