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Association between tobacco use and central sensitization: A cross-sectional study

Tütün ürünü kullanımı ile santral duyarlılaşma arasındaki ilişki: Kesitsel bir çalışma

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ABSTRACT

Background: This study aims to investigate the association between tobacco use and central sensitization (CS), as measured by the Central Sensitization Inventory (CSI), and to identify demographic and clinical factors associated with CS.

Patients and Methods: In this cross-sectional study, a total of 380 adults were included using a convenience sampling method between December 2024 and January 2025. Data collection was performed through an online survey platform to ensure accessibility and broad participation. The survey was distributed via digital platforms, social media networks, and email lists to reach a diverse adult population across Türkiye. Demographic characteristics, clinical variables, and tobacco product use status were recorded. Central sensitization was assessed using the CSI. Multiple linear and logistic regression analyses were performed to identify predictors of CSI scores.

Results: Of a total of 380 participants, 131 were male and 249 were female with a mean age of 41.21 ± 11.40 (range, 18 to 65) years. Tobacco users had significantly higher CSI total scores compared to non-users (p = 0.029). A higher proportion of individuals with a CSI score ≥ 40 was observed among tobacco users (p = 0.003). In the multiple linear regression model, age (β = −0.295), female sex (β = 6.255), chronic disease status (β = 4.524), chronic pain (β = 12.093), and tobacco use (β = 6.538) were independent predictors (p < 0.001). Logistic regression analysis indicated a significant association between tobacco use and the likelihood of having a CSI score ≥ 40 (odds ratio [OR] = 0.488, 95% confidence interval [CI]: 0.302-0.789).

Conclusion: Tobacco use is positively associated with higher CS symptom severity. Given the cross-sectional design, these findings reflect a correlation rather than direct causality. Nevertheless, screening for smoking habits may provide valuable context during the clinical evaluation of chronic pain.

Keywords: Central sensitization, chronic pain, smoking, tobacco use.

ÖZ

Amaç: Bu çalışmada, Santral Duyarlılaşma Envanteri (CSI) ile değerlendirilen santral duyarlılaşma (SD) ile tütün kullanımı arasındaki ilişki araştırıldı ve SD ile ilişkili demografik ve klinik faktörler belirlendi.

Hastalar ve Yöntemler: Bu kesitsel çalışmaya, Aralık 2024 ile Ocak 2025 tarihleri arasında kolayda örnekleme yöntemi ile toplam 380 erişkin dahil edildi. Veri toplama, erişilebilirliği artırmak ve geniş katılım sağlamak amacıyla çevrim içi bir anket platformu aracılığıyla gerçekleştirildi. Anket, Türkiye genelinde farklı yetişkin gruplarına ulaşmak amacıyla dijital platformlar, sosyal medya ağları ve e-posta listeleri üzerinden dağıtıldı. Katılımcıların demografik özellikleri, klinik değişkenleri ve tütün ürünü kullanım durumları kaydedildi. Santral duyarlılaşma, CSI kullanılarak değerlendirildi. Santral Duyarlılaşma Envanteri puanlarının belirleyicilerini ortaya koymak amacıyla çoklu doğrusal regresyon ve lojistik regresyon analizleri yapıldı.

Bulgular: Toplam 380 katılımcının 131'i erkek, 249'u kadın olup, yaş ortalaması 41.21 ± 11.40 (aralık: 18-65) yıl idi. Tütün ürünü kullanan katılımcıların toplam CSI puanları, kullanmayanlara göre anlamlı olarak daha yüksekti (p = 0.029). Ayrıca, CSI puanı ≥ 40 olan bireylerin oranı tütün ürünü kullananlarda anlamlı düzeyde daha yüksekti (p = 0.003). Çoklu doğrusal regresyon analizinde yaş (β = −0.295), kadın cinsiyeti (β = 6.255), kronik hastalık varlığı (β = 4.524), kronik ağrı varlığı (β = 12.093) ve tütün ürünü kullanımı (β = 6.538), CSI puanının bağımsız belirleyicileri olarak saptandı (p < 0.001). Lojistik regresyon analizinde ise tütün ürünü kullanımı ile CSI puanının ≥ 40 olma olasılığı arasında anlamlı bir ilişki bulundu (olasılık oranı [OR] = 0.488, %95 güven aralığı [CI]: 0.302-0.789).

Sonuç: Tütün ürünü kullanımı, SD semptom şiddetinin daha yüksek olmasıyla ilişkilidir. Çalışmanın kesitsel tasarımı nedeniyle bu bulgular nedensel bir ilişkiyi değil, yalnızca bir ilişkiyi göstermektedir. Bununla birlikte, kronik ağrılı hastaların klinik değerlendirilmesi sırasında sigara kullanım alışkanlığının sorgulanması, hastaların değerlendirilmesine değerli katkılar sağlayabilir.

Anahtar sözcükler: Santral duyarlılaşma; kronik ağrı; sigara kullanımı, tütün ürünü kullanımı.

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Tobacco smoking is a globally prevalent and preventable health hazard, associated not only with well-established risks such as cardiovascular and respiratory diseases, but also increasingly recognized for its potential influence on chronic pain sensitivity and central nervous system (CNS) mechanisms of pain modulation.[1] Experimental and clinical studies have shown that cigarette smoking may exacerbate pain through neuroinflammatory pathways, altered neurotransmitter balance, and oxidative stress.[2,3]

Central sensitization (CS), defined as the amplification of neural signaling within the CNS leading to hypersensitivity to painful and non-painful stimuli, is a key mechanism in the pathophysiology of various chronic pain syndromes. The Central Sensitization Inventory (CSI) has been widely validated and utilized to quantify symptoms of CS in both research and clinical settings.[4-6]

Until recently, empirical evidence directly linking smoking behavior to CS has been scarce. In a pivotal web-based cross-sectional study of Japanese adults with pain, Chiba et al.[7] demonstrated that current smoking was significantly associated with increased CSI scores (β = 0.07) and that a higher Brinkman index (a metric of pack-years) also correlated with elevated CSI values (β = 0.06). Moreover, smokers had a 29% higher risk (relative risk [RR]: 1.29; 95% confidence interval [CI]: 1.04-1.60) of exceeding the CSI threshold suggestive of CS, independent of confounders such as age, sex, body mass index (BMI), comorbidities, and pain catastrophizing.

Furthermore, a clinical study examining sex-specific associations found that smoking was significantly associated with heightened CS severity among women (adjusted odds ratio [OR]: 3.21; 95% CI: 1.29-7.99; $p < 0.01$), whereas no conclusive link was identified in men.[8] These findings highlight the potential sex differences in the smoking-CS relationship, warranting sex-stratified analyses in future research.

Despite these advancements, the body of literature remains limited, particularly in diverse populations and clinical contexts. To the best of our knowledge, no study to date has specifically investigated the relationship between cigarette smoking and CS in Turkish adults, measured via the CSI. Addressing this gap, in the present study, we hypothesized that smokers would yield higher CSI scores, reflecting increased CS risk. We, therefore, aimed to investigate the association between smoking characteristics, such as status and pack-year exposure, and CS severity in a Turkish population.

PATIENTS AND METHODS

This cross-sectional observational study was conducted at Atılım University, Department of Physiotherapy and Rehabilitation between December 2024 and January 2025. A total of 380 individuals were enrolled in the study using a convenience sampling method. Adults aged between 18 and 65 years who were able to complete questionnaires independently were included. Those having a diagnosis of schizophrenia, delusional disorders, other severe psychiatric or neurological conditions, major physical disabilities, or those having any chronic/acute pain condition currently under treatment (e.g., analgesic medication, physiotherapy) were excluded. Data collection was performed through an online survey platform to ensure accessibility and broad participation. The survey was distributed via digital platforms, social media networks, and email lists to reach a diverse adult population across Türkiye. A written informed consent was obtained from each participant. The study protocol was approved by the Atılım University Ethics Committee (Date: 27.11.2024, No. E-59394181-604.01-100553). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Outcome measures

Central Sensitization Inventory: Central sensitization symptoms were evaluated using the Turkish validated version of the CSI.[9] The CSI is a 25-item self-report questionnaire designed to identify symptoms associated with CS syndromes, including fibromyalgia, chronic fatigue syndrome, irritable bowel syndrome, and tension-type headache. Items are rated on a five-point Likert scale ranging from 0 ("Never") to 4 ("Always"), resulting in a total score between 0 and 100, with higher scores indicating greater symptom severity.[10,11]

Previous validation studies have reported acceptable reliability for the CSI, with evidence of internal consistency and stability over time.[5] It has also shown validity in distinguishing individuals with chronic pain conditions from healthy controls. Severity levels are commonly classified as subclinical (0-29), mild (30-39), moderate (40-49), severe (50-59), and extreme (≥ 60). A score of ≥ 40 is widely accepted as clinically relevant for the presence of CS symptoms.[10]

Smoking variables: Smoking-related data were collected via a structured, self-report form, and individuals were subsequently grouped according to their smoking status as current, former, or never smokers. For those who reported smoking, the form

collected information on the type of tobacco product used (such as conventional cigarettes, electronic cigarettes, or hookah), the age at smoking initiation, the duration of smoking categorized by years, and the daily amount of smoking expressed in approximate packs per day.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 29.0 software (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was evaluated using the Shapiro-Wilk test. Continuous data were presented in mean ± standard deviation (SD) or median (min-max), while categorical variables were presented in number and frequency. Differences in CSI scores between smokers and non-smokers were examined using the independent-samples t-test or the Mann-Whitney U test, according to data distribution. Multiple linear regression analysis was performed to evaluate whether smoking status and cumulative smoking exposure independently predicted CSI scores after adjustment for age, sex, BMI, and the presence of chronic disease. In the linear regression analysis, the CSI total score was determined as the dependent variable; age, sex, BMI, chronic disease, chronic pain, and tobacco use were determined as independent variables. A p value of < 0.05 was considered statistically significant.

RESULTS

Of a total of 380 participants, 131 were male and 249 were female with a mean age of 41.21 ± 11.40 (range, 18 to 65) years. Demographic characteristics are shown in Table 1.

Table 2 shows the comparison of demographic and clinical characteristics between smokers and non-smokers. There were no significant differences between the groups in terms of age and BMI (p > 0.05). However, significant differences were observed in sex distribution, chronic disease and chronic pain (p < 0.05). The mean CSI total score was significantly higher in smokers compared to non-smokers (p = 0.029). Additionally, smokers had a significantly higher proportion of CSI scores ≥ 40 than non-smokers (p = 0.003).

Factors associated with the CSI total score were examined using multiple linear regression analysis (Table 3). The model demonstrated statistical significance (p <0.001) and accounted for 18.5% of the variability in CSI total scores (R^2 = 0.185; adjusted R^2= 0.172).

Table 1. Demographic and clinical data of participants (n = 380)

	n	%	Mean ± SD
Age (year)			41.21 ± 11.40
Body mass index (kg/m²)			25.07 ± 4.18
Sex			
Female	249	65.5	
Male	131	34.5	
Education			
Primary School	3	0.8	
Secondary School	4	1.1	
High School	51	13.4	
Bachelor's degree	238	62.6	
Master's/PhD	84	22.1	
Marital status			
Single	88	23.2	
Married	256	67.4	
Divorced	32	8.4	
Widowed	4	1.1	
Chronic disease			
Yes	81	21.3	
No	299	78.7	
Chronic pain			
Yes	93	24.5	
No	287	75.5	
Pain medication use			
Yes	56	14.7	
No	324	85.3	
Tobacco use status			
Yes	162	42.6	
No	218	57.4	
Tobacco product type			
Cigarette	155	40.8	
E-cigarettes	4	1.1	
Waterpipe (hookah/shisha)	1	0.3	
Others	2	0.5	
Smoking duration (year)			
1	4	1.0	
1-5	20	5.2	
5-10	15	3.9	
10-15	25	6.5	
> 15	98	25.6	
Packs per day			
1 packs	136	35.5	
2 packs	20	5.2	
3 packs	3	0.8	
CSI score			27.83 ± 18.63
< 40	291	76.6	
≥ 40	89	23.4	

SD, standard deviation; CSI, central sensitization inventory.

Age, sex, presence of chronic disease, presence of chronic pain, and smoking status were significant predictors of CSI total score (p < 0.05). Specifically,

increasing age was associated with lower CSI total scores, whereas female sex, presence of chronic disease, presence of chronic pain, and smoking were associated with higher CSI total scores (Table 3).

The association between smoking status and a CSI score of ≥ 40 was evaluated using logistic regression analysis (Table 4). The logistic regression model was statistically significant (p = 0.003),

Table 2. Comparison of demographic and clinical characteristics according to smoking status

	Current tobacco users		Non-tobacco users					
	n	Mean ± SD	n	Mean ± SD	Z (Mann-Whitney U)	Chi-square	Eta	*p*
Age (year)		41.64 ± 10.40		40.89 ± 12.09	–0.898			0.369
Body mass index (kg/m²)		25.25 ± 3.92		25.00 ± 4.38	–0.156			0.876
Sex					**< 0.001**			0.181
Female	90		159					
Male	72		59					
Education					0.048			0.061
Primary School	0		3					
Secondary School	4		0					
High School	23		28					
Bachelor's degree	105		133					
Master's/PhD	30		54					
Marital status					0.010			0.086
Single	32		56					
Married	109		147					
Divorced	21		11					
Widowed	0		4					
Chronic disease					0.698			**0.020**
Yes	33		48					
No	129		170					
Chronic pain					0.419			**0.041**
Yes	43		50					
No	119		168					
CSI		31.06 ± 20.78		25.43 ± 16.50	–2.186			**0.029**
< 40	112		179			**0.003**	0.152	
≥ 40	50		39					

SD, standard deviation; CSI, central sensitization inventory.

Table 3. Multiple linear regression analysis for CSI total score

	B	%95 CI for *B*		SE *B*	β	R2	Δ R2	t	*p*
		Lower bound	Upper bound						
Model						0.185	0.172		
Constant	23.447	10.835	36.059	6.414				3.656	< 0.001
Age	–0.295	–0.453	–0.137	0.080	–0.180			–3.678	**< 0.001**
Body mass index	0.229	–0.206	0.663	0.221	0.051			1.035	0.301
Sex	6.255	2.434	10.076	1.943	0.160			3.219	**0.001**
Chronic disease	4.524	0.058	8.991	2.271	0.100			1.992	**0.047**
Chronic pain	12.093	7.909	16.278	2.128	0.279			5.682	**< 0.001**
Tobacco usage	6.538	3.010	10.065	1.794	0.174			3.644	**< 0.001**

CSI, central sensitization inventory; CI, confidence interval; SE, standard error; Durbin-Watson = 2.016 F = 14.125, p < 0 .001 R = 0.430, R2 = 0.185, Adjusted R2= 0.172* = p < 0.05.

Table 4. Logistic regression analysis of smoking status for CSI scores ≥ 40

	B	SE	Wald	df	Sig.	Exp (B) OR	95% CI for Exp (B)	
							Lower	Upper
Tobacco (R = Non-tobacco users)								
Current tobacco users	−0.717	0.245	8.554	1	**0.003**	0.488	0.302	0.789

CSI, Central Sensitization Inventory; OR, odss ratio; CI, confidence interval; SE, standard error; Omnibus Chi-square: 8.646, df = 1, $p < 0.05$; Nagelkerke $R^2 = 0.034$; $p < 0.05$.

explaining 3.4% of the variance in CSI ≥ 40 status ($R^2 = 0.034$).

Smoking status was significantly associated with having a CSI score ≥ 40. Compared to non-smokers, smokers had lower odds of having a CSI score < 40 (OR = 0.488, 95% CI: 0.302-0.789, $p = 0.003$), indicating a higher likelihood of CSI scores ≥ 40 among smokers.

DISCUSSION

In the current study, we examined the association between tobacco use status and the severity of CSS, as evaluated by the CSI total score, in a substantial cross-sectional sample. The main findings indicated that tobacco use was significantly associated with higher CSI total scores and an increased likelihood of having CS. Furthermore, age, sex, the presence of chronic disease, and the presence of chronic pain were identified as independent factors associated with CS. These findings suggest that tobacco use may represent an important clinical correlate of CS symptom severity. However, prospective longitudinal studies are needed to determine whether this association reflects a causal relationship or is driven by shared biological and psychosocial factors.

The findings of this study are consistent with a growing body of literature that identifies smoking as a significant, independent risk factor for CSS. A multitude of web-based cross-sectional studies have reported a positive correlation between current smoking and an increase in CSI values (β = 0.07) in individuals experiencing pain.[7] This correlation has been shown to increase the relative risk of exceeding the clinical cut-off score for CSS (RR ≈ 1.29). This convergence of findings lends further credence to the hypothesis that tobacco use may play a role in the development or exacerbation of CS.

Previous research suggests that cumulative cigarette exposure is related to greater pain severity and heightened experimental pain responses.[12] Individuals suffering from pain who also smoke tend to report higher levels of pain intensity and impairment in comparison to non-smokers.[13] It has been demonstrated that smoking has the potential to induce dysregulated pain processing and suboptimal outcomes through a variety of mechanisms. These include specific effects related to nicotine and tobacco, such as tissue degeneration and impaired healing, as well as general neurobiological effects, including the accumulation of stress and reward neurocircuitry.[1,14]

In a study, Hawkins et al.[15] showed that long-term systemic nicotine administration caused cellular and behavioral alterations in the expression of important proteins in the trigeminal ganglion and spinal trigeminal nucleus involved in the onset and maintenance of peripheral and CS. It has been demonstrated that exposure to cigarette smoke instigates a state of chronic, low-grade systemic inflammation within the body. Pro-inflammatory cytokines, which are caused by nicotine and other chemicals, have the capacity to traverse the blood-brain barrier, thereby exerting an effect on the functioning of the CNS. This neuroinflammation can lead to hypersensitivity to pain signals, forming the basis of CS by increasing the sensitivity of nociceptive neurons.[16]

A study using pack-years to quantify smoking exposure found that increased tobacco consumption corresponded with higher pain severity, pain frequency, and enhanced experimental and mechanical pain sensitivity.[12] More importantly, the authors suggested that CS might represent a key mechanism underlying the association between chronic tobacco smoking and increased risk of persistent pain. Consistent with this perspective, the present study demonstrated that tobacco product use was associated with higher CSI total scores and a greater likelihood of clinically relevant CS. Although direct comparisons are limited by differences in outcome measures, both findings support the notion that prolonged tobacco exposure may contribute to altered central pain processing and heightened pain sensitivity.

In the present study, female sex and the presence of chronic pain were independently associated with higher CSI total scores, consistent with literature indicating sex differences in CS and pain sensitivity. In a study, healthy women were shown to exhibit greater experimentally induced CS compared to men, suggesting inherent sex differences in central pain processing mechanisms that may contribute to women's increased vulnerability to chronic pain conditions.[17] Moreover, women with migraine demonstrate more prominent CS symptoms than men, further supporting female predisposition to CS phenomena.[18] These sex-based differences may be influenced by biological factors such as hormonal modulation and immune mechanisms, as highlighted in recent reviews.[19]

Similarly, the presence of chronic pain conditions has been repeatedly linked to elevated CS symptoms. To illustrate, individuals with chronic low back pain and fibromyalgia demonstrate significantly higher CSI scores and enhanced pain facilitation compared to pain-free controls, underscoring the role of persistent pain in maintaining CNS hyperexcitability.[20,21]

In addition, the observed association between tobacco exposure and CSI aligns with evidence that cumulative smoking history (e.g., pack-years) is positively related to multiple measures of pain severity and mechanical hyperalgesia, with CS proposed as an underlying mechanism.[7] The correlation between cumulative exposure indices and CSI in the present sample suggests a potential dose-response relationship, which future longitudinal and mechanistic studies should investigate further. Together, these findings highlight the multifactorial nature of CS, with behavioral (tobacco use), demographic (sex), and clinical (chronic pain) factors jointly contributing to CS severity.

Previous studies have reported inconsistent findings regarding the association between age and CS. Some research based on experimental pain sensitivity measures has suggested that aging may be associated with increased pain facilitation and reduced pain inhibition.[22] However, these findings were generally derived from relatively small samples and did not directly assess self-reported CS. In contrast, several epidemiological studies using the CSI have demonstrated lower CS scores with increasing age.[7,23] Consistent with these findings, the present study showed that increasing age was independently associated with lower CSI total scores. More importantly, the association between tobacco use and CSI remained statistically significant even after adjustment for age, suggesting that tobacco use may be related to CS beyond age-related effects. Nevertheless, given the cross-sectional design of the study, further research, particularly longitudinal studies, is needed to clarify the temporal and mechanistic relationships between tobacco use, aging, and CS.

Nonetheless, there are some limitations to this study that should be acknowledged. The cross-sectional nature of the study limits conclusions regarding causality between tobacco use and CS. Consequently, it cannot be determined whether tobacco use precedes the development of CS or whether individuals with higher levels of CS are more inclined to use tobacco. Furthermore, the use of an online survey for data collection introduces potential selection bias, as it relies on participants with internet access and digital literacy, which may not fully represent the general population. Tobacco exposure was derived from participant-reported data and may therefore be affected by imperfect recall. Additionally, potential confounding factors such as psychosocial stress, sleep disturbances, and physical activity levels were not directly assessed and may partially influence the observed associations. Nevertheless, the finding that tobacco use remained independently associated with higher CSI scores after adjustment for age, sex, chronic disease, and chronic pain highlights the potential clinical relevance of tobacco exposure in central pain processing. From a clinical perspective, assessment of tobacco product use may provide valuable context when evaluating patients with chronic pain and suspected CS, and may inform more comprehensive, biopsychosocially oriented management strategies. Future longitudinal and mechanistic studies are warranted to clarify temporal relationships and underlying biological pathways, and to determine whether tobacco cessation may represent a modifiable target for reducing CS–related symptom burden.

In conclusion, our study findings showed that tobacco product use was independently associated with higher CS severity, as indicated by increased CSI total scores, even after adjustment for relevant demographic and clinical factors. Female sex and the presence of chronic pain were also associated with greater CS, whereas increasing age was associated with lower CSI scores. Given the cross-sectional design of the study, causality cannot be inferred from these findings. Taken together, tobacco use may be an important factor to consider in the

clinical assessment of individuals with chronic pain. Further longitudinal studies are needed to clarify the nature and direction of the relationship between tobacco use and CS.

Author Contributions

Z.C.K., P.M.: Idea/concept, design, writing the article; Z.C.K.: Control/supervision, references and funding; Z.C.K., P.M., A.R.T., H.B.Ş., M.R., S.P., Z.N.F., N.U., N.Ş.: Data collection and/ or processing, literature review, materials; N.Ş.: Analysis and/or interpretation; Z.C.K., N.Ş.: Critical review.

Conflict of Interest

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Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

AI Disclosure

The authors declare that artificial intelligence (AI) tools were not used, or were used solely for language editing, and had no role in data analysis, interpretation, or the formulation of conclusions. All scientific content, data interpretation, and conclusions are the sole responsibility of the authors. The authors further confirm that AI tools were not used to generate, fabricate, or 'hallucinate' references, and that all references have been carefully verified for accuracy.

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