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Relationship Between Psychological Factors and Pain Intensity in Temporomandibular Disorders with or without Central Sensitization: A Cross-Sectional Observational Study Using Multiple Regression Analysis

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Purpose: To quantify the relationship between perceived pain intensity and psychological variables in a sample of participants with temporomandibular disorder, with or without central sensitization (CS).

Methods: A cross-sectional study with nonprobability convenience sampling was conducted from January 1, 2022, to June 30, 2023. Pain intensity (Numeric Pain Rating Scale), anxiety (State-Trait Anxiety Questionnaire, STAI), catastrophizing (Pain Catastrophizing Scale, PCS), perceived stress (Perceived Stress Scale, PSS), and sleep quality (Pittsburgh Sleep Quality Index, PSQI) were assessed. Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 20.0 (IBM Co.), which included descriptive and normality analyses and the calculation of strength of multiple correlational regression.

Results: A total of 52 (n=34 female 65.4%; n=18 male 34.6%) subjects with diagnosis of temporomandibular disorders (TMD) were finally included. A total of 26 participants (n=26, 50.0%) were cases suffered from CS (TMD-CS mean=46.62±11.24) while the remaining participants (n=26, 50.0%) were the controls (TMD-nCS mean=26.77, standard deviation [SD]=8.42). The pain intensity was moderate in both groups TMD-CS (mean=7.62, SD=0.83) and TMD-nCS (mean=7.05, SD=0.86), anxiety (TMD-CS STAI mean=53.27, SD=11.54; TMD-nCS STAI mean=49, SD=11.55), catastrophizing (TMD-CS PCS mean=46.27, SD=9.75; TMD-nCS PCS mean=26.69, SD=4.97), perceived stress (TMD-CS PSS mean=30.35, SD=4.91; TMD-nCS PSS mean=26.12, SD=6.60) and sleep quality (TMD-CS PSQI mean=15.81, SD=3.65; TMD-nCS PSQI mean=12.77, SD=2.76) levels were measured in both groups. In TMD-CS and TMD-nCS, higher anxiety levels were moderately and significantly associated with greater pain intensity β=0.4467 (t=2.477, p=0.021) and β=0.5087 (t=2.672, p=0.014). Nevertheless, catastrophizing, perceived stress and sleep quality were not associated to pain intensity in neither of group.

Conclusions: In both TMD-CS and TMD-nCS patients, elevated anxiety levels were moderately and significantly associated with increased pain intensity. However, heightened levels of pain catastrophizing, perceived stress, and poor sleep quality were not significantly associated with increased pain intensity in either of the two analyzed groups.

Keywords: Anxiety; Catastrophization; Central nervous system sensitization; Pain; Stress, psychological; Temporomandibular joint disorders

INTRODUCTION

Temporomandibular disorders (TMD) are a group of conditions that affect the function of the muscles and joints of the masticatory system [1]. Its etiology is multifactorial and can be explained by the interaction of different arthritic components (e.g., internal or degenerative joint diseases), myogenic factors, and, in exceptional cases, neurogenic components [2,3].

The clinical manifestations of TMD vary from individual to individual, but the most common symptom is pain in the temporal region and the cheeks, which worsens with chewing, yawning, and talking. This pain is usually intermittent and persistent, ranging from moderate to severe [4,5]. In addition, the range of motion of the joints may be reduced, or joint noises may occur, which manifest as clicking or crackling [6]. Other symptoms, such as ear pain, stiffness, fatigue, headache, or neck pain, may also occur during chewing function or at rest [7].

TMD can be influenced by the existence of risk factors that can precipitate or aggravate orofacial pain [8]. Biomechanical risk factors related to TMD are frequently linked to oral parafunctional habits or malocclusive disorders [9-13], such as bruxism [14]. In addition, psychological factors such as stress, anxiety, depression, and other medical conditions such as sleep disturbances can increase the severity of symptoms and the risk of prolonging the duration of pain [15-18].

The combination of both types of factors can lead to maladaptive pain mechanisms characterized by the persistence of a peripheral neuroinflammation stimulus-related to tissue injury and the emergence of a secondary sensitization of the central nervous system (CNS) [19]. This process called central sensitization (CS) is a consequence of the amplification of neural signals within the CNS associated with alterations in the descending opioid inhibitory pathways that facilitate the transmission of nociceptive signals across the peripheral nerve system to central processing centers [20]. At the clinical level, CS manifests in patients as spontaneous and disproportionate hyperalgesia, often with an illogical pattern of anatomical distribution barely related to the original injury. Moreover, somatosensory disturbances include hypersensitivity to mechanical stimuli (e.g., allodynia) or heightened painful responses to non-noxious thermal, vibrational, or electrical stimuli [21]. In combination with maladaptive psychosocial factors and autonomic nervous system dysfunction, CS can severely impact functional and cognitive performance [22] and overall quality of life [23-25].

The existence of the phenomenon of CS in TMD has recently been revealed to involve an alteration in the descending inhibitory pathway that triggers a significant reduction in influence from key structures within the central opioid pathway. These structures include the rostral ventromedial medulla, dorsal periaqueductal gray matter, thalamus or prefrontal cortex [26]. CS in TMD is characterized by the presence of an aberrant pattern of hyperalgesia and hypersensivity that are not related to real damage to orofacial tissues [27,28].

Although information is available on the existence of TMD in several chronic pain syndromes (e.g., myofascial pain syndrome, fibromyalgia, chronic fatigue syndrome, tension-type headache, and migraine), limited knowledge exists on the influence of psychological factors on the somatosensory system in patients with TMD. Moreover, there is a scarcity of literature that quantifies the association between these psychological variables and the intensity of TMD-related pain in symptomatic participants with and without CS [29-31]. The detection of CS phenomenon among these patients early can help the professional, and can aid healthcare professionals in establishing a prognosis based on affective profiles and improve the prediction of pain responses in TMD in presence of psychic disturbances. Due to the lack of previous research on this topic, our goal was to quantify the relationship between perceived pain intensity and psychological variables in a sample of subjects with TMD, with or without CS.

MATERIALS AND METHODS

1. Study Design

We conducted a cross-sectional observational study following the Statement for Reporting Observational Studies (STROBE) guidelines, and employed nonprobability convenience sampling to quantify the association between pain intensity and psychological factors associated with CS in a

sample of participants with TMD [32]. The study was conducted between January 1, 2022, and June 30, 2023, in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee. Written informed consent was obtained from each participant and registered in an anonymous database prior to inclusion in the study.

2. Participants

After signing the informed consent, LMPC conducted clinical interviews to evaluate compliance with the previously established inclusion and exclusion criteria, which required participants to be (1) male or female patients over 18 years of age, (2) diagnosed with symptomatic TMD, and (3) medically validated for the presence or absence of CS-related pain using the Spanish version of the Central Sensitization Inventory (CSI) (>40) [33]. Exclusion criteria encompassed individuals who (1) did not have a psychiatric illness, (2) have hearing limitations or Spanish comprehension issues, and (3) express their willingness to participate by signing an informed consent.

3. Collection of Information and Measuring Instruments

After evaluating the eligibility criteria and signing the informed consent to participate in the study, JADC carried out the collection of information in the consultation of the Simulated Hospital at European University of the Canary Islands (from March 2, 2022, to April 14, 2023). During this phase, the participants were provided with questionnaires, and assessed with scales and instruments relevant to the primary (*pain intensity*) and secondary variables associated with pain-related psychological factors (*anxiety, pain catastrophizing, perceived stress, and sleep quality*).

4. Study Variables

1) Primary variable

(1) Pain intensity: The primary variable of the study was pain intensity, measured using the Numeric Pain Rating Scale (NPRS) [34]. The NPRS is a simple, common, and validated subjective measurement scale for assessing variations (acute and chronic) in pain intensity. It consists of a numbered line of 10 cm on which the patient indicates the level their pain, with 0 representing no pain and 10 indicating the worst pain ever experienced.

2) Secondary variables

(1) Anxiety: Anxiety levels were measured through the State-Trait Anxiety Questionnaire (STAI) [35]. This self-report inventory measures the presence and severity of anxiety symptoms and the overall propensity for anxiety. The STAI consists of 40 items, with 20 assigned to each of its subscales, which evaluate two facets of anxiety: the State Anxiety Scale and the Trait Anxiety Scale.

(2) Catastrophizing: The variable of catastrophizing of pain, was measured using the Pain Catastrophizing Scale (PCS) [36]. This scale measures negative thoughts preceding pain experiences, using patients' painful past experiences to gauge the degree to which they experience certain thoughts or feelings. The scale consists of 13 items and evaluates three dimensions: *rumination* (constant worry and the inability to inhibit pain-related thoughts), *magnification* (exaggeration of the unpleasantness of painful situations and expectations of negative consequences), and *despair* (inability to cope with painful situations). The final score was between 13 and 62, with the highest scores, which refer to a high catastrophizing.

(3) Perceived stress: The variable of perceived stress was measured using the Perceived Stress Scale (PSS) [37], whose purpose, validity, and sensitivity have been demonstrated by previous studies. This scale was designed to measure the degree to which individuals rate situations in their lives as stressful. The scale comprises 14 questions intended to assess the current level of stress experienced by the participants. The items incorporated into this instrument assess the degree to which people find life unpredictable, uncontrollable, or overwhelming. These three aspects have consistently been confirmed as central components of the experience of stress.

(4) Sleep quality: The sleep quality variable was measured using the Pittsburgh Sleep Quality Index (PSQI) [38]. It is a self-administered questionnaire that provides an overall rating of sleep quality by assessing seven hypothetical components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disorders, sleep medication usage, and daytime dysfunction. The PSQI has gained widespread acceptance in both clinical and research settings and

has been translated into several languages.

5. Statistical Analysis

Statistical analysis was carried out using IBM SPSS Statistics for Windows, Version 20.0 (IBM Co.) for data analysis and representation. First, LMPC recorded the results of the assessment instruments in an electronic database, while JADC verified the accuracy of the data by completing a double data entry. Afterward, SMP conducted statistical calculations to characterize the sample and numerical variables from the parameters of centralization (mean and median), dispersion (standard deviation [SD]), and position (first quartile and third quartile). Thereafter, for categorical variables, we describe as frequencies and percentages. Differences between the sociodemographic groups were assessed using either the Mann–Whitney U or Chi-Square (χ^2) tests. Furthermore, the assumption of normality of each variable was tested using the Shapiro–Wilk test. Ramsay test was carried out while homoscedasticity and independence were checked using the Breusch–Pagan–Godfrey and Durbin–Watson tests, respectively. Statistical significance was set at $p<0.05$. If the criteria for bivariate correlations were met, Pearson's correlation coefficient was used to determine the relationship between the primary and secondary variables as well as the coefficient of determination and alienation of each association.

In cases where these criteria were not met, Spearman's rho correlation coefficient was employed to calculate the strength of the association between variables. Significantly associated variables were entered into multiple linear regression models (using each outcome in an independent model). Results are expressed as beta coefficients (β) and their respective 95% confidence intervals.

For interpretation purposes, correlation coefficients from 0.26 to 0.49 were considered weak, from 0.50 to 0.69 were considered moderate, from 0.70 to 0.89 were considered strong, and from 0.90 to 1.00 were considered very strong. No additional selection criteria were applied to include independent variables in the multiple regression model. Statistical significance was set at $p<0.05$.

RESULTS

1. Description of the Sample

A total of 52 participants with diagnosis of TMD were finally included in the study, comprising 34 female (65.4%)

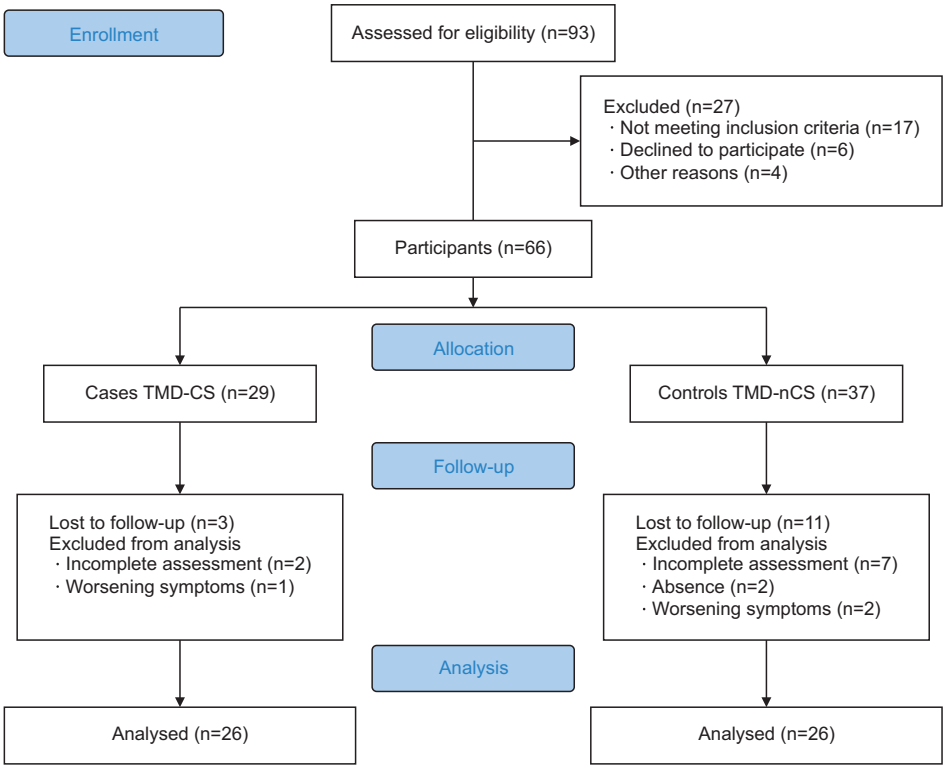

Fig. 1. Flow diagram for participant selection. This figure represents the phases on which participant recruitment was based. After the process, 52 participants completed all the analysis phases. TMD-CS, temporomandibular disorder with central sensitization; TMD-nCS, temporomandibular disorder without central sensitization.

and 18 male (34.6%) aged 28-67 years old. Fig. 1 shows a flow diagram for participant selection. According to the CSI cutoff reference values (>40), 26 participants (50.0%, mean age=46.52 years, SD=11.24) suffered from CS (TMD-CS mean=46.62±11.24), while the remaining participants (n=26, 50.0%, mean age=38.9 years, SD=10.25) served as controls (TMD-nCS mean=26.77, SD=8.42). The duration of symptoms varied from 4 to 28 months, with an average duration of 12.19 months (SD=5.54) for the TMD-CS cases and 11.0 months (SD=4.38) for the TMD-nCS controls. This variation indicates that all subjects experienced at least one subacute and chronic TMD pain episode.

2. Description of Study Variables

Pain intensity was moderate in the TMD-CS (mean=7.62, SD=0.83) and TMD-nCS (mean=7.05, SD=0.86) groups. Additionally, secondary variables related to psychological factors were measured, including anxiety (TMD-CS STAI mean=53.27, SD=11.54; TMD-nCS STAI mean=49, SD=11.55), catastrophizing (TMD-CS PCS mean=46.27, SD=9.75; TMD-nCS PCS mean=26.69, SD=4.97), perceived stress (TMD-CS PSS mean=30.35, SD=4.91; TMD-nCS PSS mean=26.12, SD=6.60), and sleep quality (TMD-CS PSQI mean=15.81, SD=3.65; TMD-nCS PSQI mean=12.77, SD=2.76). Table 1 shows the sample characteristics.

3. Multiple Correlation Analysis

1) Temporomandibular disorder with central sensitization

In TMD-CS patients, elevated anxiety levels were moderately and significantly associated with increased pain intensity (β=0.468, t=3.778, p<0.001). However, heightened levels of pain catastrophizing (β=0.028, t=0.187, p=0.853) and perceived stress (β=0.084, t=0.613, p=0.543) were not significantly associated with increased pain intensity. Furthermore, the inverse relationship between sleep quality and pain intensity was insignificant (β=−0.047, t=−0.355, p=0.724). The predictive value of the regression model was moderate for pain intensity (R=0.588, adjusted R^2=0.290, F=4.04, p=0.001), and the model exhibited a good fit (variance inflation factor [VIF]<1.2 and tolerance >0.85). The statistical power of the analysis was also good (Power [1-β]=0.98) (Table 2).

2) Temporomandibular disorder without central sensitization

In TMD-nCS patients, heightened anxiety levels were moderately and significantly associated with increased pain intensity (β=0.5087, t=2.672, p=0.014). However, elevated levels of pain catastrophizing (β=−0.086, t=−0.452, p=0.655) and perceived stress (β=−0.0187, t=−0.095, p=0.925) were not significantly associated with increased pain intensity. Moreover, the inverse relationship between

Table 1. Sample characteristics (n=52)

Variable	CSI	Number	Mean	SD	W	p-value	25th	75th
Pain intensity (NPRS 0-10)	TMD-CS	26	7.62	0.830	0.960	0.396	6.93	8.07
	TMD-nCS	26	7.05	0.860	0.956	0.326	6.50	7.80
Anxiety (STAI 0-80)	TMD-CS	26	53.27	11.540	0.954	0.290	44.25	60.00
	TMD-nCS	26	49.00	11.559	0.949	0.225	40.25	58.25
Catastrophizing (PCS 13-62)	TMD-CS	26	46.27	9.751	0.958	0.362	40.75	54.00
	TMD-nCS	26	26.69	4.970	0.933	0.090	22.25	30.75
Perceived Stress (PSS 0-40)	TMD-CS	26	30.35	4.915	0.936	0.108	28.25	34.75
	TMD-nCS	26	26.12	6.605	0.965	0.509	21.25	31.00
Sleep quality (PSQI 0-21)	TMD-CS	26	15.81	3.655	0.945	0.173	12.25	19.00
	TMD-nCS	26	12.77	0.830	0.860	0.002*	11.00	13.75

CSI, Central Sensitization Inventory; SD, standard deviation; W, Shapiro-Wilk goodness-of-fit test; 25th, Percentile 25; 75th, Percentile 75; NPRS, Numeric Pain Rating Scale; STAI, State-Trait Anxiety Questionnaire; PCS, Pain Catastrophizing Scale; PSS, Perceived Stress Scale; PSQI, Pittsburgh Sleep Quality Index; TMD, temporomandibular disorders; CS, central sensitization.
*p≤0.01.

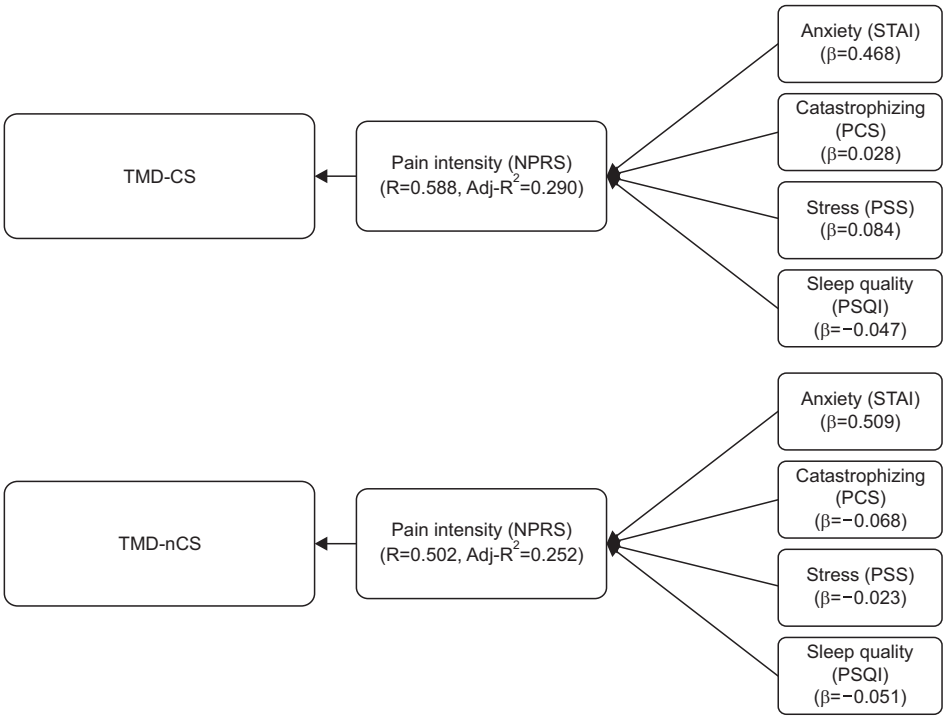

Fig. 2. Multiple linear associations between pain intensity and psychological factors in the TMD-CS and TMD-nCS groups. The direction of the arrows represents the standardized coefficients (β), moving from independent (psychological factors related to pain) to dependent (pain intensity) variables. (A) In the TMD-CS group, pain intensity was most affected by anxiety, followed by perceived stress. (B) In the TMD-nCS group, pain intensity was affected only by state anxiety. TMD-CS, temporomandibular disorder with central sensitization;TMD-nCS, temporomandibular disorder without central sensitization; NPRS, Numeric Pain Rating Scale; STAI, State-Trait Anxiety Questionnaire; PCS, Pain Catastrophizing Scale; PSS, Perceived Stress Scale; PSQI, Pittsburgh Sleep Quality Index.

Table 2. Multiple correlation analysis of patients with TMD-CS

Model	Dependent variable	Independent variable	R	Adj-R^2	RMSE	F-value	p-value	β	T-value	p-value
1	Pain intensity		0.588	0.29	0.710	4.04	0.001			
		Anxiety						0.468	3.778	<0.001
		Catastrophizing						0.028	0.187	0.853
		Perceived stress						0.084	0.613	0.543
		Sleep quality						−0.047	−0.355	0.724

TMD, temporomandibular disorders; CS, central sensitization; Adj-R^2, adjusted R-square; RMSE, root mean square error; β, beta standardized coefficient.

Table 3. Multiple correlation analysis of patients with TMD-nCS

Model	Dependent variable	Independent variable	R	Adj-R^2	RMSE	F-value	p-value	β	T-value	p-value
2	Pain intensity		0.502	0.252	0.690	0.088	2.47			
		Anxiety						0.509	2.619	0.016
		Catastrophizing						−0.068	−0.324	0.749
		Perceived Stress						−0.023	−0.118	0.907
		Sleep Quality						−0.051	−0.253	0.803

TMD, temporomandibular disorders; CS, central sensitization; Adj-R^2, adjusted R-square; RMSE, root mean square error; β, beta standardized coefficient.

sleep quality and pain intensity was insignificant (β=−0.051, t=−0.253, p=0.803). The predictive value of the regression model was moderate for pain intensity (R=0.502, adjusted R^2=0.252, F=2.47, p=0.088), and the model exhibited a good fit (VIF<1.2 and tolerance >0.85). The statistical power of the analysis was also good (Power [1-β]=0.99) (Fig. 2, Table 3).

DISCUSSION

The aim of the study was to determine the association between pain intensity and psychological factors in a sample of individuals with TMD, both with and without CS. Multiple linear regression analyses showed that anxiety was

associated with increased perceived pain in both TMD-CS patients (β=0.468, t=3.778, p<0.001) and those with predominantly nociceptive pain (β=0.5087, t=2.672, p=0.014). To the best of our knowledge, pain centralization can mediate the experience of pain, which could explain why patients with this pain phenotype have increased pain perception. From our results it is clear that the presence of CS in TMD is not mandatory for developing a phenomenon of enhanced pain response. We believe this could be due to the important role anxiety plays in amplifying pain in TMD, as it is not only significantly associated with a high prevalence of TMD symptoms but also correlated with pain severity. From a neurophysiological perspective, persistent pain, as observed in our sample, induces persistent anxiety and stress, resulting in long-term neuroplastic changes in the hypothalamic–pituitary–adrenal axis [39]. According to recent studies, this would result in increased facilitatory activity with a hypercortisolemic state, with a consequent decrease in inhibitory feedback through an imbalance of glucocorticoid propensity between hormone and mineralocorticoid receptors mineralocorticoid receptors [40].

However, no association was found between perceived pain intensity and other psychological factors, such as catastrophizing [41,42], perceived stress [43], and sleep quality [44,45]. These results are inconsistent with those published in the scientific literature, which have traditionally identified these factors as modulators of orofacial pain responses.

First, catastrophizing was not particularly high in the sample (TMD-CS PCS mean=46.27, SD=9.75; TMD-nCS PCS mean=26.69, SD=4.97), which could explain the weak correlation. Catastrophizing is a multidimensional phenomenon dependent on various factors related to an individual's personality, as previously described in the literature [46,47]. For this reason, we recommend studies based on personality aspects related to personality, as it has been identified as a predictor of pain aversion behavior. Moreover, the complexity of its assessment requires the use of psychometric tools, such as the one employed in this study, which defines catastrophizing as the integration of three dimensions of analysis: rumination, magnification, and despair. Therefore, utilizing a linear analysis based on the overall score of the instrument, rather than considering each dimension separately, could weaken the association strength in the proposed analytical model (TMD-CS β=0.028, t=0.187, p=0.853; TMD-nCS β=−0.086, t=−0.452, p=0.655).

Second, one possible hypotheses for the lack of correlation between perceived stress and pain intensity is that both groups had a clinically significant perception of stress but with magnitudes that ranged from mild to moderate (TMD-CS PSS mean=30.35, SD=4.91; TMD-nCS PSS mean=26.12, SD=6.60). Furthermore, the instrument used to evaluate perceived stress scores the feelings and thoughts experienced by the patient over the last month. This is crucial because the mean duration of symptoms was 12.19 months (SD=5.54) for the TMD-CS group and 11.0 months (SD=4.38) for the TMD-nCS group. Variations in stress levels over time may change the overall assessment of this dimension, potentially explaining the weak correlation between this and the pain intensity in TMD-CS patients (β=0.084, t=0.613, p=0.543) and TMD-nCS patients (β=−0.0187, t=−0.095, p=0.925).

Finally, sleep quality was poor (TMD-CS PSQI mean=15.81, SD=3.65; TMD-nCS PSQI mean=12.77, SD=2.76), but it was not correlated with pain intensity in either of the two regression analysis models proposed in this study (TMD-CS β=−0.047, t=−0.355, p=0.724; TMD-nCS β=−0.051, t=−0.253, p=0.803). Similar to catastrophizing, the relationship between pain intensity and sleep quality may depend on several factors that have been analyzed. However, its impact on patients with CS does not appear to be well defined based on the results obtained.

1. Limitations

In this study, we identified some limitations that may affect the external validity of the results. As far as samples are concerned, recruitment was performed using non-probability sampling techniques, far from the expected sample size, which may affect the generation of biased samples. Furthermore, we believe that using only two reference centers for sample recruiting may have biased the results. However, considering the possible impact of this participant selection technique, we believe that, from an organizational standpoint, this is the most straightforward recruitment system to implement in the program, as it allows us to obtain representative sample sizes.

If we focus on follow-up, the chosen study design, a

cross-sectional correlation study, does not allow us to understand how psychological factors fluctuate over time, or how the relationships between variables change over the course of possible final symptoms. Focusing on follow-up, the chosen study design, correlational cross-sectional studies, does not give us an idea of how psychological factors fluctuate over time, nor how the relationships between variables change throughout the final symptoms that may arise. during long-term follow-up. This question forces us to consider the need for observational studies with long-term cohorts, which allow us to make long-term assessments of outcome variables.

In both TMD-CS and TMD-nCS patients, elevated anxiety levels were moderately and significantly associated with increased pain intensity. However, heightened levels of pain catastrophizing, perceived stress, and poor sleep quality were not significantly associated with increased pain intensity in either of the two analyzed groups.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

DATA AVAILABILITY STATEMENT

Data will be provided anonymously to protect study participants' privacy.

FUNDING

None.

AUTHOR CONTRIBUTIONS

Conceptualization: IMMP. Data curation: SEMP, JADC, LMPC. Formal analysis: SEMP, JADC, LMPC. Funding acquisition: IMMP. Methodology: SEMP, IMMP. Project administration: SEMP. Visualization: JADC, LMPC. Writing original draft: JADC, LMPC. Writing review & editing: SEMP, IMMP.

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