Headache Exacerbates Pain Characteristics in Temporomandibular Disorders

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Aims: To evaluate the impact of headache in adults with masticatory myofascial pain (MMP) on the outcome variables clinical pain (ie, self-reported pain intensity and pressure pain sensitivity), sleep quality, and pain catastrophizing. **Methods:** A total of 97 patients with MMP were diagnosed with co-existing headache (MMPH group, n = 50) or without headache (MMP group, n = 47) according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD). The outcome parameters were the Pittsburgh Sleep Quality Index (PSQI); the Catastrophizing Thoughts subscale of the Pain-Related Self-Statement Scale (PRSS-C); pressure pain thresholds (PPTs) of the masseter and anterior temporalis muscles; and self-reported facial pain intensity measured on a 0- to 10-cm visual analog scale (VAS). Student *t* test for independent samples (α = 1.2%) and factorial analysis of variance (ANOVA) (α = 5%) were used to analyze the data. **Results:** The MMPH group showed significantly impaired sleep quality (mean ± standard deviation [SD] PSQI score 9.1 ± 3.5) compared with the MMP group (7.2 ± 3.4; P = .008). Subscale scores on the PRSS-C were significantly higher in the MMPH (2.1 ± 1.2) than in the MMP group (1.6 ± 1.4, uncorrected P = .048). Also, the PPTs (kgf/cm^2) of the masseter and anterior temporalis muscles were significantly lower in the MMPH group (1.52 ± 0.53; 1.29 ± 0.43, respectively) than in the MMP group (2.09 ± 0.73; 1.70 ± 0.68, respectively; P < .001), with no differences in self-reported facial pain intensity. Factorial analyses further indicated that chronic migraine was associated with poorer sleep quality (P = .003) and that tension-type headache patients had lower PPTs in the anterior temporalis muscle (P = .041) in comparison with non-headache patients. **Conclusion:** Co-existence of headache further exacerbates clinical characteristics in patients with painful TMD, which implies involvement of common mechanisms and pathways of vulnerability in these patients. *J Oral Facial Pain Headache 2017;31:339–345. doi: 10.11607/ofph.1746*

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Studies dealing with the relationship between headache and temporomandibular disorders (TMD) have been mostly related to the diagnostic comorbidity between the two conditions.[1–3] In general, it has been demonstrated that the presence of headache is more pronounced in TMD patients and that TMD are more present in headache patients.[4–6] Additionally, when headache is associated with TMD, self-reported pain severity is worse and masticatory muscle sensitivity increased.[6,7] Accordingly, it is generally accepted that there is a negative impact of headache in patients with TMD in terms of pain outcomes (ie, pain intensity and muscle pain sensitivity). However, data are lacking related to the impact of headache associated with TMD on other important clinical variables, such as sleep quality, life-style, and psychological constructs.

The proposal of the biopsychosocial pain model in the late 1970s was an attempt to explain how somatic processes affect mental states and vice versa.[8] With this paradigm, pain perception could be better understood together with cultural, social, psychological, and behavioral dimensions. In addition, psychological aspects were acknowledged as essential factors in the epidemiology and management of chronic pain.[9–11] For instance, global psychological and

somatic symptoms were regarded as strong predictors of first-onset TMD in a multivariate analysis,[12] and depression is notably a major risk factor for migraine chronification.[13] However, despite this well-established relationship between headache and painful TMD, there are few studies evaluating the impact of TMD-associated headache on psychosocial aspects in general. There is new evidence of a possible triple comorbidity between headache, TMD, and sleep bruxism,[14] and another recent study has shown an association between headache and TMD symptoms, depression, anxiety, and poor sleep.[15] However, the cross-sectional design of both studies and the lack of a standardized clinical diagnosis of TMD in the latter study warrant further investigation.

It is of clinical relevance to clearly assess how headache and TMD intermingle with each other in a comprehensive perspective, which could call attention to the importance of differential diagnosis and multiple management strategies. In this scenario, it is plausible to assume the more pronounced the negative influence of headache (ie, the more clinical variables are affected by the presence of headache), the more closely related these conditions are. Therefore, there is a need for a more multidisciplinary approach.

Based on these considerations, the aim of this study was to evaluate the impact of headache in adults with masticatory myofascial pain (MMP) on the outcome variables clinical pain (self-reported pain intensity and pressure pain sensitivity), sleep quality, and pain catastrophizing. It was hypothesized a priori that the presence of headache would negatively influence pain sensitivity, sleep quality, and pain catastrophizing ratings.

Materials and Methods

Sample and Design

The eligible participants of this case-control study, which was designed in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines,[16] were all adults (≥ 18 years) who sought care for facial pain in two institutions: Bauru School of Dentistry and Federal University of Sergipe, Brazil. The participants (undergraduate students, university staff, and local community) were recruited through advertisements, local newspapers and radio stations, websites, social networks, and referral from primary care centers from 2011 to 2013.

General inclusion criteria were the presence of MMP according to the revised Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD)[17] and at least 3 months of pain duration. The general exclusion criteria were: history of head trauma, vascular and intracranial disorders, or other major causes of secondary headache listed in the International Classification of Headache Disorders, 2nd Edition (ICDH-2)[18]; regular use of medications such as muscle relaxants, anticonvulsants, antidepressants, and anxiolytics; presence of odontogenic pain disorders; presence of other chronic pain conditions besides TMD (such as fibromyalgia, idiopathic facial pain, trigeminal neuralgia, or atypical odontalgia); chief complaint of temporomandibular joint (TMJ) pain; systemic conditions; and any TMD or headache treatment performed in the last 3 months. For the case group (masticatory myofascial pain with headache [MMPH group]), a specific inclusion criterion was the complaint of headache for at least 3 months. The headache assessment was questionnaire based[6,19–21] and made according to the criteria of the ICHD-2.[15] Twenty-six questions related to headache features (eg, intensity, duration, frequency, quality and side of headache pain, headache triggers, and the presence of associated symptoms such as nausea, photophobia, phonophobia, and autonomic symptoms) comprised this headache questionnaire. Considering the ICHD-2 criteria framework for these questions, diagnosis of tension-type headache (TTH) or migraine was made according to headache characteristics, and frequency was determined based on the temporal criteria for TTH, namely infrequent (< 1 day/month on average), frequent (1 < 15 days/month on average), or chronic (≥ 15 days/month on average).[18] Less common primary headaches (eg, trigeminal autonomic headaches) were not found in the sample. The temporal relationship of headache with TMD was estimated during the clinical examination, and all patients reported that the headache began together or became worse with the onset of MMP. On the other hand, the control group (MMP group) had no headache complaints of any kind in the last 3 months, although headache history over this 3-month period was not recorded.

Two examiners (one from each institution) were responsible for the assessment of eligibility, and both performed a comprehensive training in the RDC/TMD assessment procedure prior to data collection (Y.M.C., A.P.L.F.). However, the interrater reliability was not estimated. A detailed medical interview/anamnesis and clinical examination were performed to determine whether a patient fulfilled the inclusion criteria. No further tests (eg, imaging or blood tests) were performed. Accordingly, the presence of headache was the controlled factor in this case-control design. Furthermore, all the procedures were conducted in accordance with the Helsinki Declaration II and had been approved by the regional Human Ethics Committee, and all participants gave their informed consent before they were included in the study protocol.

Clinical Outcome Variables

The following clinical variables were measured in both groups: sleep quality, pain catastrophizing, self-report of facial pain intensity, and pressure pain threshold (PPT).

Sleep Quality. The Pittsburgh Sleep Quality Index (PSQI) was used to assess each patient's quality of sleep.[22] The PSQI is a self-report questionnaire consisting of 19 items and is used to measure the frequency of sleep disturbances and subjective sleep quality in the last 30 days. The items are scored and merged into components that range from 0 (no difficulty) to 3 (severe difficulty). The sum of the components generates a final score that ranges from 0 to 21, where values greater than 5 indicate poor sleep. The original version has shown acceptable psychometric properties for internal consistency (Cronbach's α = 0.83) and validity (Hotelling's T^2 = 2.62, $P < .001$),[22] and the Brazilian Portuguese version has shown acceptable internal consistency (Cronbach's α = 0.82).[23]

Pain Catastrophizing. The Catastrophizing Thoughts subscale of the Pain-Related Self-Statement Scale (PRSS-C) was used to assess pain catastrophizing.[24] The PRSS-C is a self-report questionnaire consisting of nine items and is used to measure the frequency of catastrophic thoughts related to painful experiences. The items are rated on a 5-point scale ranging from 0 (not at all) to 4 (all the time), and the final score is the sum of all individual items divided by the total number of questions. The original version has shown acceptable psychometric properties for internal consistency (Cronbach's α = 0.92) and goodness of fit (χ^2 = 383.18, $P < .001$),[24] as has the Brazilian Portuguese version, with an internal consistency (Cronbach's α = 0.89) and split-half Pearson correlation of 0.74.[25]

Pain Intensity. Self-report of facial pain intensity was measured with the aid of a 0- to 10-cm visual analog scale (VAS) that consisted of a horizontal line with the anchor edges "no pain" and "worst imaginable pain." The participants were asked to draw a vertical mark on the line at the point that best represented the pain intensity during the last month.

Pressure Pain Threshold. Pressure pain threshold (PPT) measurements for the masseter and anterior temporalis muscles were carried out with a digital dynamometer (Kratos) that included a rod with a 1-cm^2 flat circular tip.[26] It was emphasized to the participants that the purpose was to measure the minimal amount of pressure at the first perception of pain. The device had a button controlled by the participant, who was asked to press it at the first sensation of pain (ie, when the pressure was just barely perceived as painful). The application rate was close to 0.5 kgf/cm^2/second. During the examination, the individual's head was firmly supported by the examiner's hand. Two measurements were taken for each side, and the PPT value for each muscle as a measure of pressure pain sensitivity was determined as the arithmetic mean of both sides.

Statistical Analyses

The mean and standard deviation (SD) of each quantitative variable (age, PSQI, PRSS-C, VAS, PPT) was calculated along with a description of gender and headache characteristics. In addition, age, PSQI, PRSS-C, VAS, and PPT values were assessed with the Kolmogorov-Smirnov test and showed Gaussian distribution ($P > .050$). Student *t* test for independent samples was computed to compare age, PSQI, PRSS-C, VAS, and PPT values between the groups, and chi-square test was used to compare sex distribution. The effect size of all significant results was also calculated according to Cohen's coefficient (Cohen's d), which scores the effect as small (d = .2), moderate (d = .5), or large (d ≥ .8).[27] In order to adjust for multiple comparisons, a Bonferroni correction lowered the significance level to 1.2% (P = .012) as the cut-off point for establishing statistical significance. Each group of variables (PSQI, PRSS-C, VAS, and PPT) was considered as a family of comparisons. Therefore, the family-wise error rate was established considering four multiple comparisons, and, according to the Bonferroni formula (.050/k, where k = number of comparisons), an alpha level of P = .012 was set.

A secondary analysis was performed with factorial ANOVA with the factors headache type (three levels) and frequency (three levels) to evaluate the effect of headache presentation on the PSQI, PRSS-C, VAS, and PPT. Such analysis categorized headache type as follows: no headache (1st level); tension-type headache (TTH) (2nd level); or migraine (3rd level); and frequency was categorized as absent/infrequent (< 1 day/month on average [1st level]), frequent (1 < 15 days/month on average [2nd level]), or chronic (≥ 15 days/month on average [3rd level]). When appropriate, post hoc analyses were performed by using Tukey's Honest Significant Difference (HSD) test. The significance level was set at 5% (P = .050).

Results

A total of 345 patients were assessed for eligibility, and 97 patients fulfilled the study criteria. The main reasons for exclusion were the report of medication usage and pain treatment performed in the last 3 months (n = 106) and, for the control group, the report of any headache in the last 3 months (n = 92). Table 1 shows the demographic and clinical pain

Table 1 Demographic and Clinical Pain Characteristics of the Study Sample

	MMPH (n = 50)	MMP (n = 47)	*P* value	Cohen's d
Age, mean (± SD)	29.4 (± 6.6)	27.5 (± 6.7)	.135	–
Sex, n (%)	44 (88) F, 6 (12) M	38 (80) F, 9 (20) M	.132	–
Headache characteristics				
Frequency,* n (%)				
Infrequent	2 (4)	–	n/a	n/a
Frequent	26 (52)	–	n/a	n/a
Chronic	22 (44)	–	n/a	n/a
Phenotype, n (%)		–	n/a	n/a
Migraine-like	23 (46)	–	n/a	n/a
TTH-like	27 (54)	–	n/a	n/a
TMD characteristics				
Pain intensity, mean (± SD)	6.2 (± 1.8)	5.4 (± 2.2)	.080	–
PPT, mean (± SD)				
Anterior temporalis	1.52 (± 0.53)	2.09 (± 0.73)	< .001	.89
Masseter	1.29 (± 0.43)	1.70 (± 0.68)	< .001	.72

Frequency was defined according to the tension-type headache (TTH) criteria, International Classification of Headache Disorders, 2nd Edition.[15]
F = female; M = male; TMD = temporomandibular disorder; PPT = pressure pain threshold; MMPH = masticatory myofascial pain with headache; MMP = masticatory myofascial pain.

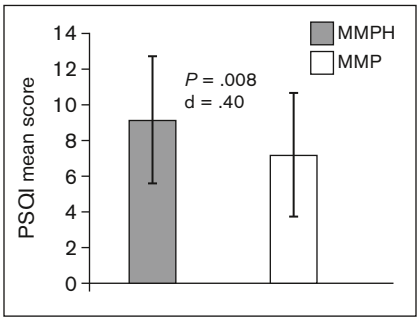

Fig 1 Column chart indicating the means and standard deviations (SDs) of sleep quality scores between TMD pain patients with and without associated headache. PSQI = Pittsburgh Sleep Quality Index; MMPH = masticatory myofascial pain associated with headache; MMP = masticatory myofascial pain; d = effect size.

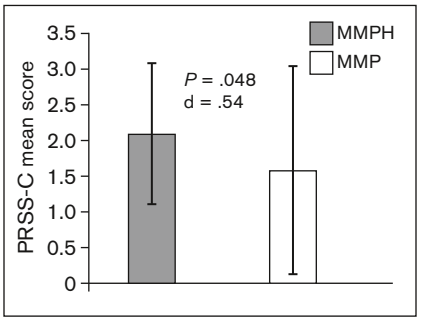

Fig 2 Column chart indicating the means and standard deviations (SDs) of pain catastrophizing scores between TMD pain patients with and without associated headache. PRSS-C = Catastrophizing Thoughts subscale of the Pain Related Self-Statement Scale; MMPH = masticatory myofascial pain associated with headache; MMP = masticatory myofascial pain; d = effect size.

characteristics of all included participants. The age and sex distributions were comparable between the two groups (P > .050). A slight majority of participants (54%) had TTH-like headache, and 46% had migraine-like headache. Headaches were rated as infrequent in 4% of the MMPH group, frequent in 52% (1 < 15 days/month on average), and chronic in 44%. The frequency distributions for those with TTH-like headache (n = 27) were: infrequent (3.7%), frequent (63%), and chronic (33.3%); and for those with migraine-like headache (n = 23) were: infrequent (4.3%), frequent (39.1%), and chronic (56.6%). The majority (58%) of headache patients reported having the symptoms for at least 6 years, 16% for 2 to 5 years, and 26% for less than 2 years.

Figure 1 presents the between-group comparison for sleep quality. The MMPH group reported poorer sleep quality compared to the MMP group (P = .008, d = .40). In addition, Fig 2 shows the significant between-group difference in pain catastrophizing scores, with the MMPH group reporting higher scores than the MMP group (P = .048, d = .54; uncorrected: P = .050).

For clinical pain outcomes, there was no between-group difference for facial pain intensity assessed on the VAS (P = .080). However, there were significant differences in PPT comparisons: The MMPH group presented lower PPTs than the MMP group for the masseter (P < .001, d = .72) and anterior temporalis (P < .001, d = .89) muscles (Table 1).

There were no main effects of headache type or frequency (P > .050) for any of the variables; however, there were two significant interactions between the factors. Participants with the chronic migraine–like phenotype reported poorer sleep quality than the non-headache group (Tukey: P < .003), and participants with the frequent TTH–like phenotype had lower PPT values only for the anterior temporalis muscle in comparison with the non-headache group (Tukey: P < .041).

Discussion

The main findings of this case-control study were that presence of headache is associated with poorer sleep in patients with MMP and headache, deep pain sensitivity of masticatory muscles is more pronounced in patients with MMP and headache, and headache phenotype seems to be distinctly related to the severity of clinical outcomes in patients with MMP.

There is compelling evidence showing the impact of headache on clinical pain outcomes in TMD patients.[7,19,20,28] It has already been established that the comorbidity between headache and TMD

is associated with higher levels of reported pain intensity and muscle pain.[5–7] Similarly, TMD severity as measured by accepted criteria (RDC/TMD) is higher in patients with more frequently occurring headaches.[6] The biopsychosocial paradigm has pointed out that it is as important to focus on psychological aspects as on the common clinical pain outcomes generally assessed in musculoskeletal disorders (ie, pain intensity and muscle tenderness).[11] In this respect, the present results are important, as they shed some light on the multidimensional impact of headaches on painful TMD.

Sleep impairment is a common finding in chronic pain patients,[29,30] and a recent review specifically addressed the mechanisms of orofacial pain and sleep and their interactions.[31] Epidemiologic data with different strengths of evidence have also addressed the relationship between sleep and TMD and between sleep and headache. A recent cross-sectional study showed a significantly higher chance of the presence of sleep disturbance in patients with MMP (odds ratio [OR] of 2.41).[32] In addition, a large cohort study of TMD patients has found that poorer sleep outcomes measured with the PSQI may be risk factors (standardized hazard ratio [HR] = 1.47) for first-onset TMD pain.[33] Pain intensity is also considered a risk factor for sleep impairment.[34] Thus, the evidence suggests bidirectional relationships between sleep quality and painful TMD. Likewise, sleep quality is affected in headache patients, and a reciprocal causal relationship is acknowledged.[35] Not only can poor sleep predict headache chronification, but the presence of headache can trigger sleep disturbances.[35–37] Thus, the comorbidity of head pain and sleep could be due to different conditions sharing similar pathophysiologic mechanisms.[31] Indeed, some brain structures and neurotransmitters (eg, thalamus, hypothalamus, melatonin, and orexin) are essentially interrelated with sleep and headache.[38] An interesting finding of the present study was the interaction between headache type and frequency, implying the relationship between pain and sleep was apparently dependent on severity. This is in line with the so-called continuum model, which postulates primary headache disorders as different points on a line of progression, with chronic migraine as the end point.[39]

The focus on the negative consequences of a painful experience (ie, pain catastrophizing) can also be considered both a risk factor and a consequence of chronic pain disorders.[9,10,12] In particular, a cohort study in TMD pain patients demonstrated that baseline pain catastrophizing level was a significant predictor of the progression of pain and disability.[12] On the other hand, successful TMD management addressing clinical pain outcomes, such as pain reduction and muscle tenderness/pain on palpation, can decrease the level of pain catastrophizing.[10] Similarly, pain catastrophizing has been associated with greater headache pain in the general population.[9] Nevertheless, the present findings do not support the additional negative consequence of pain catastrophizing, even though there was a significant between-group difference based on the conventional *P* value indicative of statistical significance (< .05). In a similar case-control study, chronic headache patients were more likely to catastrophize than TMD patients,[40] though no controlling for multiple comparisons was performed and a different measurement instrument (Coping Strategies Questionnaire [CPS]) was used to assess pain catastrophizing.

The negative impact of headache on masticatory muscle pain has been addressed in previous studies.[5,7,41,42] It is well established that the additional diagnosis of headache in TMD patients is associated with increased deep pain sensitivity,[7,42] which in turn could be considered a consequence of increased central nervous system synaptic excitability associated with painful TMD.[43] The present results are in line with this evidence; nevertheless, they suggest that the gain of somatosensory function in terms of decreased PPT might be dependent on the headache characteristics and specific muscle sites. The subgroup analyses showed hyperalgesia in the anterior temporalis muscle but not in the masseter muscle in patients with frequent TTH compared with non-headache patients. In fact, the anterior temporalis muscle is the most affected site in patients with headache attributed to TMD.[41] It is also noteworthy that this secondary headache resembles TTH in most cases.[44] Future studies aimed at addressing headache and TMD should differentiate between TTH and migraine and primary and secondary headache in order to better understand the pathophysiologic relationships between these disorders.

The strengths of the present study were the large sample size, which allowed the necessary power to discriminate between many variables, and the clinical characterization of the subtypes of headache in terms of the clinical outcome variables. In addition, the comprehensive evaluation of the patients that included psychosocial domains that affect and are affected by painful disorders needs to be highlighted. Some limitations also need noting. The case-control design did not allow inferences to be drawn about causal relationships; the non-differentiation between primary headache and headache secondary to TMD introduces the possibility of recall bias (which can occur with case-control designs and in history taking) and imposed difficulties on accurately determining the chronologic order of appearance of MMP and headache; lack of control for some confounders, such as depression, anxiety, and burden of

disease comorbidity; possible study site variations; the non-inclusion of another control group with only headache; and headache history that covered only the last 3 months in the MMP group. Nevertheless, the use of the criterion of a headache-free interval of 3 months could minimize bias related to previous headache treatment and detailed medication intake assessment, although patients with a history indicative of medication overuse were excluded.[15] In addition, the younger age of the MMPH group compared to what is generally found for primary headache patients could be related to the presence of concomitant TMD, since the mean age of the groups was in line with epidemiologic figures of myofascial TMD[45] and within the onset range for primary headaches.[46] However, a selection bias may not have been totally excluded, since undergraduate students of both institutions were part of the university populations.

Conclusions

There appears to be a broad negative impact of headache when associated with MMP, since co-existence of headache further exacerbates clinical characteristics in patients with painful TMD. Thus, from a management perspective, a multidisciplinary approach is warranted to reduce the burden of both conditions.

Acknowledgments

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