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Painful temporomandibular disorders and central sensitization: implications for management—a pilot study

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Abstract. The objective was to investigate the presence of cutaneous allodynia and hyperalgesia in the trigeminal and extra-trigeminal areas, as a surrogate for central sensitization (CS), in women with a painful temporomandibular disorder (TMD) and without other painful conditions. Painful TMDs, depression, and non-specific physical symptoms (NSPS) were classified according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD). The amount of pain in the trigeminal and extra-trigeminal areas was determined using a visual analogue scale (0–100 mm) after the application of a vibrotactile stimulus and assessment of the pressure pain threshold (PPT). Statistical tests (Fisher's, χ^2, and Mann–Whitney) were performed, with a significance level of 5%. The sample comprised 45 women (mean age 37.5 years; 16 with a painful TMD) who were free of any headache, fibromyalgia, or other painful condition. Painful TMD was associated with higher pain sensitivity and lower PPT values in the trigeminal ($P < 0.01$) and extra-trigeminal regions ($P < 0.01$). The presence of depression contributed significantly to increased pain sensitivity. The presence of hyperalgesia and allodynia in both the trigeminal and extra-trigeminal regions among women with a painful TMD indicated the presence of CS. Changes involving the central nervous system should be considered during the evaluation and management of patients with a painful TMD.

Key words: temporomandibular joint disorders; central nervous system sensitization; hyperalgesia.

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Temporomandibular disorders (TMD) represent a cluster of conditions involving the structures of the masticatory system. The most frequent symptoms include pain in the masticatory muscles and/or the temporomandibular joint (TMJ), TMJ sounds, and limited or asymmetric mandibular movements.[1] The prevalence of TMD ranges from 21.5% to 51.8% in different studies,[2,3] being approximately twice as common in women as in men.[3]

The presence of pain and the reduced pressure pain threshold (PPT) in structures related to the TMD can be explained by peripheral sensitization (PS),[4] characterized by a reduction in threshold and amplification in responsiveness of nociceptors.[5] Local factors such as trauma, parafunctional activity, and surgical procedures can cause local inflammation and ischemia, increasing the nociceptive input restricted to the site of tissue injury. This can then evolve, inducing a sensitization of higher-order neurons, which characterizes a central sensitization (CS) process.[6]

CS is an important aspect in the pathophysiology of various types of chronic musculoskeletal pain, including TMDs.[7] It is characterized by hyperexcitability and an expansion of the nociceptive second-order neuron receptive fields, reduction of the activation threshold, and prolonged neuronal discharge.[8] Clinically, CS can be evidenced by an increased and prolonged responsiveness to noxious stimuli (hyperalgesia) and the perception of pain following a non-painful stimulus (allodynia).[8] These phenomena may explain the presence of sensitivity and pain in another area of the body observed in patients presenting a painful TMD,[4] featuring centrally mediated pain.[9]

Previous studies have demonstrated the presence of cutaneous allodynia, hyperalgesia, and therefore CS in patients with a painful TMD.[10] Nevertheless, TMD patients frequently present painful comorbidities such as primary headaches[11] and fibromyalgia,[12] conditions that may involve the presence of CS. Only a few studies have investigated the presence of cutaneous allodynia and hyperalgesia in TMD patients with no other persistent painful conditions.[13] Furthermore, the presence of fibromyalgia, primary headaches, and emotional disorders are potential confounders in the association between allodynia, hyperalgesia, and painful TMDs.[14]

The identification of the presence of CS is of high clinical relevance to choosing treatments not limited to peripheral approaches and capable of producing analgesia by normalizing the hyperexcitable central neural activity.[12] It is also important to predict the development of severe postoperative and persistent pain.[15] Osteoarthritis patients with high levels of comorbid centrally-mediated symptoms, for example, showed severe pain and increased analgesic requirements after total knee arthroplasty in the early postoperative period. Moreover, these patients seemed to be at higher risk of persistent pain and showed low satisfaction regarding pain relief after surgery.[16]

Therefore, the aim of this study was to verify the presence of CS, manifested as trigeminal and extra-trigeminal cutaneous allodynia and hyperalgesia, in a controlled sample of women presenting with a painful TMD who were free of headaches, fibromyalgia, and other chronic painful conditions.

Patients and methods

A cross-sectional study was conducted involving a sample of women presenting with a painful TMD, identified among individuals seeking treatment for orofacial pain. To be included in the painful TMD study group, individuals had to present a joint, muscle, or mixed painful TMD, according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) Axis I.[17–19] The control group comprised women who were free of any orofacial pain seeking routine dental care, with neither a current nor a past history of painful TMD or other forms of chronic orofacial pain. Consecutive individuals aged between 20 and 65 years were enrolled, considering the following exclusion criteria: (1) total absence of natural teeth (even using conventional complete dentures); (2) partial absence of teeth with no use of a fixed or removable prosthesis; (3) abnormal cognitive function and communication skills; (4) current daily use of pain medication; (5) presence of any headache, fibromyalgia, or other chronic painful condition.

The local research ethics committee approved this study, and informed consent was obtained from each participant (patients and controls).

Study protocol

Two trained researchers (R1 and R2) conducted the evaluations. R1 conducted the interview and clinical examination, and applied the RDC/TMD Axis I and II criteria.[17–19] R2, who was blinded to the individual's pain status, applied the psychophysical and algometry tests. The sequences of the assessments and the areas of evaluation were determined randomly.

The socio-demographic data, main complaint, pain characteristics, dental examination, and medical history were assessed through an interview and clinical examination. The TMD diagnoses and differential diagnosis with other orofacial pain conditions were made according to the American Academy of Orofacial Pain (AAOP) diagnostic criteria.[1]

RDC/TMD Axis I criteria were used to classify the TMD into group I (myofascial TMD), group II (TMJ disc displacement), or group III ((a) arthralgia, (b) osteoarthritis, or (c) osteoarthrosis). Women classified as presenting group I and/or group IIIa or IIIb were included in the painful TMD group. Individuals who fulfilled the criteria for group II or no TMD were classified as controls.

The RDC/TMD Axis II criteria were applied to assess the grade of depression and somatization (non-specific physical symptoms (NSPS)). Depression and NSPS were individually classified as normal, moderate, or severe. For the analyses, the moderate and severe categories were grouped together, and both depression and NSPS were treated as dichotomous variables (no depression/depression; no NSPS/NSPS).

Psychophysical test—vibrotactile stimulation

The vibrotactile stimulus was applied using an electric toothbrush, a validated method for the assessment of pain sensitivity and CS for screening purposes.[7] Following the previously validated method,[7] an electric toothbrush with a brush head of 1 cm in diameter, with 22 tufts of bristles and approximately 50 polished bristles per tuft was used (Braun – Oral-B). The brush head moves in a rotational direction at a frequency of 5 Hz. The bristles were positioned perpendicular to the skin with 1 lb of pressure for 30 s. Researcher R2 calibrated the pressure applied immediately before and then after the application of the stimulus, using an electronic scale.[7]

The stimuli were applied bilaterally at the lateral pole of the TMJ, mid-masseter, and anterior temporal muscles, and also in the ventral region of the forearms, following the same protocol. The pain or unpleasantness (if any) evoked by the vibrotactile stimuli was assessed using a 100-mm visual analogue scale (VAS). At each point the participant was required to estimate the pain at the initial moment of vibrotactile stimulus application (0 s), and after 15 s, 30 s (when the stimulus was interrupted), and 60 s (30 s after cessation of the stimulus).[7]

The resultant right and left pain figures were added for each point stimulated in the trigeminal area (lateral pole of the TMJ, mid-masseter, and anterior temporal muscles) to obtain the total trigeminal region pain. The same was done for the extra-trigeminal area: the resultant right and left pain figures for the ventral area of