Comorbidities associated with temporomandibular joint disorders and the role of central sensitization: literature review

Comorbidades associadas aos transtornos da articulação temporomandibular e o papel da sensibilização central: revisão de literatura

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DOI 10.5935/2595-0118.20220003

ABSTRACT

BACKGROUND AND OBJECTIVES: Temporomandibular joint (TMJ) disorder is a musculoskeletal disorder that causes the exacerbation of painful sensation during function and the presence of sensitivity/pain to palpation of the masticatory muscles and/or TMJ. People with pain complaints in the head, neck, shoulder or lower back may present signs and symptoms of TMJ disorders, sharing sleep disturbances, forgetfulness or difficulty concentrating, abdominal pain and differences in fecal consistency. Studies have also shown that TMJ disorders can be associated with emotional distress and multiple comorbidities related to central sensitization (CS). This, in turn, is responsible for producing hypersensitivity to pain, altering the sensory response. The pathophysiology of CS in TMJ disorders is not yet well understood. Thus, it is the scope of this review to synthesize knowledge about the relationship between CS and Temporomandibular Disorder (TMD) and describe the comorbidities most frequently found in this profile of patients.

CONTENTS: TMJ disorders do not seem to occur in isolation. Comorbidities such as migraine, tension headache, fatigue, dizziness, tinnitus and allergies have been reported. Hyperexcitability in central nociceptive processing is part of the pathophysiology of TMJ disorder, which could explain the greater sensitivity to pain in other areas of the body in these individuals, characterizing a CS process.

CONCLUSION: The most frequently reported comorbid conditions are headache, psychological factors, allergies, irritable bowel syndrome, and sleep disturbances. It is admitted that CS is a neurophysiological phenomenon present in some chronic pain disorders, including TMD.

Keywords: Central nervous system sensitization, Chronic pain, Myofascial pain syndromes, Temporomandibular joint disorders.

RESUMO

JUSTIFICATIVA E OBJETIVOS: O transtorno da articulação temporomandibular (ATM) apresenta-se como uma desordem musculoesquelética que causa a exacerbação da sensação dolorosa durante a função e a presença de hipersensibilidade/dor à palpação da musculatura mastigatória e/ou da ATM. Pessoas com queixas álgicas na cabeça, pescoço, ombro ou lombar podem apresentar sinais e sintomas de transtornos da ATM, compartilhando de distúrbios do sono, dificuldade de concentração ou esquecimento, dor abdominal e diferenças na consistência fecal. Estudos também têm mostrado que os transtornos da ATM podem estar associados a sofrimento emocional e múltiplas comorbidades relacionadas à sensibilização central (SC). Esta, por sua vez, é responsável por produzir hipersensibilidade à dor, alterando a resposta sensorial. A fisiopatologia da SC nos transtornos da ATM ainda não está bem esclarecida. Desse modo, torna-se escopo dessa revisão sintetizar o conhecimento sobre a relação entre SC e Disfunção Temporomandibular (DTM) e descrever as comorbidades mais frequentemente encontradas nesse perfil de pacientes.

CONTEÚDO: Os transtornos da ATM parecem não ocorrer isoladamente. Comorbidades como enxaqueca, cefaleia tensional, fadiga, tontura, zumbido e alergias têm sido relatadas. Uma hiperexcitabilidade no processamento nociceptivo central faz parte da fisiopatologia da desordem da ATM, o que poderia explicar a maior sensibilidade à dor em outras áreas do corpo nesses indivíduos, caracterizando um processo de SC.

CONCLUSÃO: As condições de comorbidades mais frequentemente relatadas são cefaleias, fatores psicológicos, alergias, síndrome do intestino irritável e distúrbios do sono. Admite-se que a SC é um fenômeno neurofisiológico presente em alguns distúrbios de dor crônica, incluindo as disfunções temporomandibulares.

Descritores: Dor crônica, Sensibilização do sistema nervoso central, Síndromes da dor miofascial, Transtornos da articulação temporomandibular.

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Submitted on January 20, 2021.
Accepted for publication on December 21, 2021.
Conflict of interests: none – Sponsoring sources: none.

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INTRODUCTION

Temporomandibular joint (TMJ) disorder is a musculoskeletal disorder that causes an increase in pain sensation during function, such as speaking and chewing, and the presence of tenderness/pain on palpation of the masticatory muscles and/or the TMJ, with possible limitation of range of motion, joint noises and otologic symptoms[1-4]. Its prevalence ranges from 21.1 to 73.3% and is more common in women of reproductive age[3-6].
Individuals with TMJ often report persistent pain in other locations of the body, implying a generalized dysfunction of the nociceptive system, such as hyperexcitability of the central nociceptive neurons[2,5,7]. It has been described that individuals with head, neck, shoulder or low back pain complaints may present signs and symptoms of TMJ[7], sleep disturbances, forgetfulness or difficulty concentrating, abdominal pain, and differences in fecal consistency[5,8]. The origin of these patterns is yet unknown, but neurobiological sensitization processes, genetically determined vulnerability, and psychological factors are commonly reported[9-12].
Studies have shown that TMJ may be associated with emotional distress and multiple comorbidities related to CS. This, in turn, is responsible for producing hypersensitivity to pain, altering the sensory response. The pathophysiology of CS in TMJ is not yet well understood, however, there is a decrease in the pressure pain threshold (PPT) after receiving mechanical stimuli[13].
The manifestations of CS occur in the form of a hypersensitivity to pain called tactile allodynia and secondary hyperalgesia. This sensitization induced in the nociceptor can be adaptive, temporary, or permanent, making this system become hyperexcitable. The exaggerated and prolonged sensation of pain in response to the generating stimuli may be distributed beyond the location of the injury as a result of the enlargement of the receptive field and generalized hyperexcitability in the central nociceptive pathways. Therefore, CS explains how painful sensations exist even in the absence of peripheral diseases or nociceptive stimuli[5,13,14].
In response to inflammatory stimuli, trigeminal ganglion neurons release neuropeptides as well as other molecules that initiate and perpetuate neurogenic inflammation in peripheral tissues, facilitating sensitization of peripheral trigeminal nociceptors. Furthermore, excitation of trigeminal ganglion neurons activates second-order neurons and glial cells, leading to the onset of CS, with consequent hyperalgesia and allodynia[12,13,15]. Thus, the trigeminal system provides a nociceptive link between the peripheral inflammation and the activation of central pain pathways in TMJ. This would be one of the possible explanations for the association between TMJ and CS[13].
Individuals with TMD often present comorbidities and extracephalic pain complaints that constitute a diagnostic confounding factor. Another relevant point is the association between TMD and CS processes, since under these conditions pain may occur even in the absence of structural damage. Thus, the aim of the present study was to synthesize knowledge about the association between CS and TMD and describe comorbidities most frequently found in this patient profile.

CONTENTS

It has been described that TMJ are a spectrum of disorders with different pathophysiology, clinical manifestations and associated comorbid conditions[2], which are present in 82.5% of cases[16]. A study[14] reported nine most prevalent comorbidities in individuals with TMJ migraine, tension headache, depression, degenerative arthritis, chronic fatigue, dizziness, tinnitus, gastrointestinal problems and allergies, and concluded that the disorder does not seem to occur in isolation, as almost two thirds of patients reported three or more of the main comorbid conditions[17]. Corroborating such statements, another study[5] showed the existence of an association of craniofacial sensitivity and psychosocial factors with disability in this profile of patients.
It remains unclear to what extent TMJ may be a symptom or manifestation of another underlying disorder. The high prevalence of allergies preceding the diagnosis of the disorder is noteworthy, suggesting an autoimmune etiology. Other frequently associated conditions are chronic fatigue syndrome, irritable bowel syndrome, chronic pelvic pain, and sleep disorders[14,16]. It is also known that individuals with TMJ are 5.5 times more likely to have pain in other joints[2], and the most reported areas with concomitant pain were the cervical region followed by the low back, shoulders and lower limbs[18,19].
People with TMDs reported more pain during cervical examination in all directions and it was found that the greater the cervical involvement, the greater the degree of temporomandibular involvement[20,21]. Based on these findings, the inclusion of physical examination of the neck during the examination of patients with TMDs is recomended[20]. It has been found that the severity of temporomandibular symptoms accompanies the severity of cervical spine disorders, but the reverse does not seem to be true. This finding suggests that, chronologically, TMD may appear before the cervical disorder. Another aspect that supports this hypothesis is that even individuals with mild cervical disorder have a high incidence of orofacial signs and symptoms[22].
One study[23] describes the journey of a 55-year-old male patient with a history of right-sided orofacial pain. The pain was located in the right side premolars, spreading through the right TMJ and ear. The orofacial pain was initially misdiagnosed as dental pain and trigeminal neuralgia. Upon examination, cervical joint and muscle disorders were identified. The suboccipital and upper paraspinal cervical muscles were found to be in spasm and tender on palpation. The patient was treated three days a week for two weeks by a physical therapist, reporting significant reduction in pain and improvement in health status. These improvements in function were accompanied by increased cervical range of motion[23]. In addition, significant limitation of upper cervical rotatory motion was found among patients suffering from myogenic TMD, supporting the clinical relationship between these disorders[24].
Back pain was associated with jaw pain and dysfunction[25,26]. The results suggest that back pain and musculoskeletal disorders in

the orofacial region may not be two separate entities, which implies a mutual influence or that these conditions are being caused by the same contributing factors[25]. The prevalence of fatigue/rigidity, pain, impaired mandible opening and headaches, as well as the overall prevalence of any TMD symptoms increase according to the frequency and severity of back pain. A significant relationship has been evidenced between spinal pain and all TMD variables, as well as between spinal pain and headaches[9].

Approximately 40% to 60% of TMD cases present characteristics of a more generalized pain profile[26,27]. A study about the relationship between the different TMDs, generalized tenderness to palpation and multiple pain conditions in women with painful TMD, reported that 22% of participants had two or more pain conditions outside the orofacial region[27]. Another study[19] found a 55% frequency of pain reports throughout the body, present for more than 3 months, in patients with TMJ muscle disorders.

Central sensitization and TMD

Its suggested that a general hyperexcitability in the central nociceptive processing is part of the pathophysiology of TMDs, which could explain the increased sensitivity to pain in various areas of the body of these individuals[2,5,13,21]. Factors such as trauma, parafunctional activity or surgical procedures can cause local inflammation and ischemia, increasing nociceptive input at the region of the tissue injury. This can develop to induce a sensitization of higher order neurons, which characterizes a CS process. This, in turn, can be evidenced by increased and prolonged responsiveness to noxious stimuli (hyperalgesia) and the perception of pain after a non-painful stimulus (allodynia).

Individuals with chronic TMD are more likely to experience changes in the processing of external stimuli, resulting in lower sensory thresholds[27], which can be evidenced by changes in PPT measurements and vibrotactile perception. Previous studies have shown an association between painful TMD and cutaneous allodynia in both trigeminal and extratrigeminal areas. Moreover, the lowest PPT in these patients was found in localized pain areas as well as sites distant to the pain. The existence of sensitization mechanisms in local pain syndromes suggests that prolonged peripheral nociceptive input to the central nervous system (CNS) plays a role in the initiation and maintenance of CS[10].

In a study[13] involving women with TMJ pain disorder of joint, muscular or mixed nature, as classified by the RDC/TMD (Research Diagnostic Criteria for Temporomandibular Disorders), compared with women without orofacial pain who were undergoing routine dental procedures, CS was evaluated through vibrotactile stimulation and PPT. The stimuli were applied bilaterally to the lateral pole of the TMJ, middle masseter, anterior temporal, and ventral region of the forearms. Evidence of increased pain and sensitivity was found in women with painful TMD. The increase of pain sensitivity in the trigeminal and extratrigeminal areas in this population points to an increased risk of cutaneous allodynia and hyperalgesia, suggesting the existence of concomitant CS[28].

This state of altered pain modulation can be observed early in children and adolescents, resulting in increased pain sensitivity in adulthood. These results indicate that adolescents with painful TMD may present dysfunction and more generalized sensitivity of the central nociceptive system, with a consequent increased risk of CS. Furthermore, generalized chronic pain is noted as an early sign of fibromyalgia[12].

Headaches and TMD

It has been reported that the trigeminal system is related to the pathophysiology of TMDs as well as migraine. Neurons of the ophthalmic branch of the trigeminal nerve are responsible for migraine pain, while the second and third branches are more associated with the TMJ areas. Peripheral nociceptive information converges to the trigeminal nucleus, and from there, migraine and TMDs share specific central pathways associated with cross-excitation[5,15.] Diseases of the craniofacial area are more frequent and intense in patients suffering simultaneously from headache[29]. The increased pain in the TMD group may be explained by the greater sensitization of the central and peripheral nervous system, which characterizes patients with headache. In addition, considering the hypothesis that different types of pain tend to reinforce each other[25,29], the data showed direct proportionality between pain intensity in TMDs and headache, suggesting that these patients should be treated jointly in both clinical entities.

Frequent headaches were reported by 55% of patients who described TMJ pain compared to 8% in the control group[25]. A study[15] observed the association of migraine and TMDs in adolescents and the findings suggest that TMJ pain disorder was strongly associated with higher frequency of primary headache and migraine was significantly associated with a greater number of pain areas on palpation (muscle and joint) in the trigeminal region, concluding that these individuals develop greater sensitivity to pain in other areas of the body. The assumption is that, after the onset of a migraine episode, CS involving the trigeminal caudal nucleus may develop and decrease the pain threshold in areas such as TMJ and masticatory muscless[5,15,30].

The coexistence of TMD and migraine leads to increased thermal and mechanical hyperalgesia, as well as cutaneous allodynia, suggesting the intensification of CS. Studies report pain sensitivity in migraine and TMD, even in neutral body areas unrelated to the main complaint. A slightly reduced heat pain tolerance was observed in individuals who reported multiple comorbid pain conditions[27]. Paradoxically, it has also been observed that patients with isolated cases of migraine or TMD present hyperalgesia to cold in the extracephalic region and, furthermore, in this group of individuals, hyperalgesia to cold seems to be more common than to heat[10,30]. Therefore, it's possible to assume that the TMJ myofascial disorder features sensitization processes restricted not only to second order trigeminal neurons, but also to extratrigeminal nociceptive neurons[10].

Psychological disorders and TMD

In addition to the increased risk of CS, individuals with painful TMD, as in other chronic pain scenarios, are also at increased risk for psychological disorders. Depression appears to interfere with the central modulation of the pain response, and when

deficits occur in these areas, the modulation of signals disrupts the body, leading to a more intense pain experience. Furthermore, depression induces stress and increases the production of pro-inflammatory cytokines, which can increase experience of pain[5,13].

It is noteworthy that psychological factors may be responsible for increasing the severity and intensity of headaches[8,17,27,28,31]. In a study[7], the comorbid conditions which were found to be most likely associated with TMD included migraine, depression[26], post-traumatic stress disorders, and anxiety disorders. Also, it was found that women with myofascial pain and a history of concomitant generalized pain were more likely to have depression than women with pain in the orofacial region alone[19].

A study analyzed the correlation of somatization, depression, and chronic pain with clinical findings of TMD in asymptomatic women[32]. All participants with myofascial pain showed the presence of moderate or severe depression. Still, it's not possible to determine whether psychological differences are the cause or consequence of the pain and dysfunction present in the subtypes of TMDs, nevertheless, the literature agrees that these psychological difficulties may exacerbate the pain condition[7,17,27].

Emotional stress has a positive relation with physical symptoms, including chronic headaches, low back pain, chemical sensitivities, pelvic pain, as well as TMDs. The overlap of comorbidity symptoms in CS and pain-related disability are more specific to individuals with myofascial disorder and this association may be partially explained by emotional distress. It is suggested that prolonged exposure to stress plays a more direct role in the pathogenesis of TMD, causing sustained increases in hormones that promote tonic muscle contraction and consequent orofacial pain. However, it's possible that emotional stress is a result of pain, although a scenario involving dynamic interactions between these factors is plausible[33].

Sleep disorders and TMDs

Sleep disorders are more frequent in patients with chronic pain, including TMJ pain disorders, and are directly associated with greater pain intensity. Individuals with TMDs have much worse levels of sleep disorders, including insomnia, non-restorative sleep, daytime sleepiness and sleep apnea, when compared to asymptomatic controls[34]. People with poor sleep quality have also been described to more likely develop TMJ degenerative changes[35] and those with excessive daytime sleepiness have significantly greater severity of TMDs, both in pain and dysfunction[36].

Study[17] suggests that individuals with sleep bruxism and TMD with generalized pain have lower sleep efficiency. The presence of concomitant body pain outside the orofacial area may be due to an older age and, consequently, poor sleep quality may also be age-related. This finding may suggest that young individuals have localized pain, and that with aging, these patients tend to develop diffuse pain, indicating that the patient's age is another important issue to consider in the diagnostic approach. It's also noteworthy that persistent pain conditions are associated with prolonged functional changes in the CNS.

CONCLUSION

Painful TMDs should not be considered in isolation. The most frequently reported comorbid conditions are headaches, psychological factors such as stress and depression, allergies, chronic fatigue syndrome, irritable bowel syndrome, and sleep disorders. CS is a neurophysiological phenomenon present in some chronic pain disorders, including TMDs. After a first painful episode, the process of peripheral sensitization (PS) begins, increasing nociceptive input at the location of the injury. If the aggravation persists over time, it may culminate in CS. These phenomena may explain the presence of sensitivity and pain in another body area observed in patients presenting TMDs.

AUTHORS' CONTRIBUTIONS

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Data Collection, Conceptualization, Project Management, Research, Methodology, Writing - Preparation of the original, Writing - Review and Editing
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