Temporomandibular disorders and Headaches: associative risk factors

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Abstract

Temporomandibular disorders (TMDs) and headaches are closely linked, and this has been proven by several studies in the literature. Although it was clarified the association between the two diseases, little is known about the etiology that determine the onset of both disorders.

The purpose of this work is to investigate the risk factors present in both disorders, such as adulthood, female sex, morphological and genetic factors, inflammatory mediators, neurotransmitters, parafunctions and mood disorders, with the aim of review, analyze and evaluate the etiological hypotheses proposed in the literature.

Introduction

Temporomandibular disorders (TMDs) are conditions of musculoskeletal pain characterized by pain in the temporomandibular joint and/or at the level of the masticatory muscles.

TMD pain is a very common problem, affecting approximately 7-15% of the population \(^{(1)}\). TMD pain is 1.5-2 times more common in women than in men and 80% of the treated cases are women \(^{(2)}\).

Headache is a painful disease in neuro-cranial localization and distribution of pain does not coincide with the territory of innervation. This definition is applicable for headaches but must be distinguished from the cranial neuralgia. Headache involves the activation of the fibers that innervate structures painful intra and extra cranial \(^{(3)}\).

Headache is perhaps the most widespread disease; few people are spared during their life by at least one episode of headache. It is estimated that about 90% of the total population, in a year, has had at least one episode of headache, and that in 40% of cases the attack is disabling \(^{(4)}\). Headache prefer adulthood (20-50 years) and economically productive life, but does not spare even children, adolescents and the elderly and is prevalent in developed countries than in developing \(^{(5)}\).

The worldwide used classification for the diagnosis of headache is the classification of the International Headache Society (IHS), currently accepted throughout the world, which defined the diagnostic criteria to frame the various forms of headache. This classification reached its third edition in 2013 \(^{(6)}\).

The classification most internationally used for the diagnosis of the TMD is, the System Diagnostic Criteria (CC / TMD) updated in 2014, which represents a very useful tool in the clinical practice, for the high level reliability \(^{(7)}\).

Patients diagnosed with temporomandibular disorders associated with pain often report a concomitant headache \(^{(8)}\).

The pain reported by TMD patients is localized usually in the masticatory muscles, preauricular area, or at the level of the temporomandibular joint (TMJ) \(^{(9)}\) frequently associated with headache and other facial pains.

The symptoms of headache described by TMD patients are similar to those reported by patients diagnosed with tension-type or migraine, according to nosology developed by the International Headache Society \(^{(10)}\). Several clinical and epidemiological studies have demonstrated the association between primary headache and Temporomandibular Disorders, indicating that individuals with headache and TMD have a number of signs and symptoms in common \(^{(11-14)}\).

The multifactorial origin of headache and TMD and the several cases in which subjects have similar symptoms and signs and/or overlapping make the process of diagnosis and treatment planning extremely difficult.

Despite this, the exact ratio and etiology association between these two entities is still largely unknown. The purpose of this article is to elucidate the status of the literature on the subject, trying to clarify the current assumptions about.

Associative risk factors

Morphological and genetic:

Some researchers have suggested the existence of a genetic risk shared between the two conditions.

Patients with TMD and migraine showed less nociceptive levels in most areas of the body, this
causes a dysfunction and a generalized upregulation of nociceptive system, predisposing the individual to pain in other districts (15, 16).

A recent study describes the changes in the neural mechanism in TMD patients involving an altered functional connectivity of the insula and cingulate cortex (17).

It has been shown that there is a reduction of gray matter volume in the anterior cingulate gyrus and left at the front right of the insular cortex (18). A recent review of Bashir and others, found similar morphological changes of brain structures in people with headache (19).

**Adult population:**

As regards the age, according to the literature, temporomandibular disorders show a maximum peak prevalence between 30 and 45 years, and then a subsequent tendency to decrease.

Headache mainly hit adulthood and individuals who lead an economically active and productive life. In particular, the peak prevalence of migraine and tension headache is reached between the third and fourth decade of life, and tends to decrease with age (19-22). It is clear that the trends of the two diseases are substantially overlapping.

**Female sex:**

In both of these diseases, the prevalence in the female gender can be justified by a lower pain threshold, which would induce a more timely request of treatment and, not least, the role that estrogen, whose receptors are richly represented in the joints, play in the etiopathogenesis of the dysfunctions (23).

A number of aspects of the model prevalence of TMD suggests that reproductive hormones may play a role in TMD and headache. The prevalence of temporomandibular pain in early adolescence is low (2-4%), and does not seem to differ for boys and girls (24), however, the prevalence rates are higher in adult women than adult men, and lower for women in post-menopausal to people of reproductive age.

The pattern of prevalence of TMD pain is similar to that of migraine, where hormonal factors are thought to account for at least some of the differences between the prevalence rates.

Hormonal factors, both in migraine both TMD, appear to play an important role etiopathogenetic, just think that the intensity of musculoskeletal pain associated with TMD varies with the menstrual cycle. The highest level of pain occurs in periods of low estrogen levels (during menstruation) and in case of rapid changes in estrogen levels (late luteal phase and the middle of the cycle).

**Inflammation and altered release of neurotransmitters:**

According to other research, dysfunctions associated with temporomandibular disorders induce the release of serotonin and norepinephrine in the dorsal raphe and locus coeruleus, causing a cascade of events that can worsen headaches and contribute to existing state of general muscle tension (25).

High levels of prostaglandin E2, cytokines IL-1, IL-6, TNF-α have been found in the synovial fluid of inflamed joints, and demonstrate a strong correlation with the pain, in particular for the calcitonin gene-related peptide known as potent vasodilator and one of the main contributors of neurogenic inflammation and nociception (26). These pro-inflammatory factors present in peripheral joints and inflamed masticatory muscles could cause an activation of the trigeminal system.

The integration of nociceptive input from extra and intracranial peripheral tissues occurs at the level of the caudal trigeminal nucleus, hence nociceptive input facilitators and inhibitors before they reach the thalamus and then the cortex (27). The effect of these inputs would be a peripheral central sensitization to pain.

The symptoms of TMD could lead to greater central sensitization especially in migraine sufferers who have severe and frequent migraine attacks (28, 29). The activation of the trigeminal nerve in response to inflammatory processes has been shown to increase the neural communication through cell junctions at the level of the trigeminal ganglion (30). Additional evidence suggests that the excitation of neurons in the trigeminal ganglion from any branch of the trigeminal nerve, promotes central sensitization, hyperalgesia, allodynia, headache and facial pain (31).

More than the central and peripheral sensitization that occurs in patients with proven temporomandibular disorders is also significant the association between painful sites on palpation in trigeminal areas and the frequency of occurrence of headache in dysfunctional patients (32).

**Parafunctional habits**

Several claimed that parafunction is closely related to the presence of headache (33).

Jensen et al. reported that about 30 minutes of voluntary clenching activity leading to headache in 2/3 of the patients tested (34).

Several studies (35-37) reported that prolonged muscle stimulation can lower the threshold of perceived pain...
and this could favor the onset of headache, it is stated by other studies. In patients with headache the contact between the teeth is more frequent and intense, as well as muscle tension and stress, compared to subjects not affected, or to the moments when patients did not report the disease [38]. The same study have shown that the reduction of dental contact is associated with a reduction of the self-reported pain [38].

On the other hand, the local muscle contracture is often referred to as the first symptom of migraine attacks and treatment of muscle pain during migraine induces a marked improvement of symptoms.

In the literature it is also reported that the contraction of the muscles of the jaw occurs 14 times more often during sleep in patients with tension-type headache, compared to the control group, with no headaches [39, 40]. Fernandez et al [41] found a significant correlation between sleep bruxism (SB) and chronic migraine, also a further study revealed that 83.3% of patients with SB had a diagnosis of headache. Patients with TMD and SB are also very inclined to have headaches [42].

The pathogenetic mechanism that connects the start of the parafunction of headache is not entirely known. According to some authors [43], during the night the mechanism parafunctional, creating a burden of disease at the level of the structures of the TMJ, generates the release of pro-inflammatory factors (prostaglandine E2, interleukin-1 and 6, tumor necrosis factor, nitric oxide, etc.) that would act as a trigger for headaches. These substances do not act directly, but rather the headache appears to be an indirect effect of their presence. Based on the evidence, the exogenous administration of these triggers can push a highly regulated system out of balance, triggering a cascade of neurochemical changes that eventually leads to a migraine attack.

**Depression and mood disorders**

Many studies suggest that psychosocial factors such as depression, stress, anxiety, alexithymia (difficulty describing or recognize emotions, a constriction general affective and fantasy life), and catastrophizing (waiting or worrying about major negative consequences a situation, even one of lesser importance) play a role in the preparation, initiation and perpetuation of TMD and in the response of patients to treatment TMD [44, 45].

Psychiatric diagnosis is associated with the conversion of episodic migraine to chronic migraine [46]. Patients with headaches are more likely to develop depression and patients with depression have a higher chance of developing migraines [47].

In the study of Glaros et al [38] patients with headache dysfunctional reported high levels of depression and somatization in population control, no headaches.

The pain can be non-specific symptoms of psychosocial dysfunction, depression and somatization [48].

Comorbidity of psychiatric disorders in headache and TMD is in fact a well-known clinical phenomenon [49-51], so the presence of psychiatric disorders could be considered a risk factor for the development of both the DTM that headache.

Among the possible associative etiopathogenesis between TMD and Headache, the literature stated that [52]:

- TMD can function as an independent source of pain
- TMD can worsen existing primary headaches
- TMD can be a risk factor for chronic headache through a mechanism of central sensitization
- Headache can lead to activation of the trigeminal system by increasing the propensity to TMJ pain in susceptible individuals.

**Conclusions**

Headaches and TMD diseases are very common and with overlapping signs and symptoms, they share many risk factors: genetics and morphological alterations, female gender, adulthood, mood and functional habits.

These risk factors, if present, may contribute to a deterioration in the quality of life of the patients.

In clinical settings becomes very important to intercept these risk factors in dysfunctional patients and/or with headache, to set treatment protocols aimed at improvement of the clinical conditions both in acute than in chronic.

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