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Temporomandibular disorders in fibromyalgia: a critical view

Disfunção temporomandibular na fibromialgia: uma visão crítica

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Abstract

Temporomandibular disorders (TMD) and fibromyalgia (FM) are musculoskeletal syndromes that appear to be associated to each other. Various studies throughout the literature relate the signs and symptoms of TMD in patients with FM. The objective of this study was to analyze the literature regarding clinical association between FM and TMD. Even though these conditions present different etiologies, the pain modulating mechanisms are similar. Many studies in this field aim to elucidate questions which are still little understood. However, it has been observed that these syndromes are characterized by a reduction in pain threshold and in the attenuation capabilities of descending pain modulating systems. Moreover, there is a certain degree of comorbidities between these two pathologies, which share many clinical characteristics. Numerous patients with FM present various signs and symptoms of TMD, while only a small quantity of individuals with TMD is diagnosed with FM. Therefore, an adequate and precise evaluation of the stomatognathic system in patients with FM, which encompasses TMD diagnosis in therapeutic intervention, is paramount.

Keywords

Temporomandibular disorder; fibromyalgia; facial pain.

Introduction

Temporomandibular disorder (TMD) is a syndrome which primarily involves structures of the stomatognathic system, such as masticatory muscles, temporomandibular joints, cervical muscles, as well as other related elements, like blood vessels and nervous structures [1]. The disorder is of great relevance, since nearly 10% of the adult population presents some sign or symptom of TMD [2]. Among the most common signs and symptoms, one can observe limitation of mandibular motions, difficulty in mastication and mouth opening, TMJ sounds, and, almost always, intense craniocervical pain. Its etiology seems to involve more than just teeth occlusion, and it is now believed that its diagnosis should contemplate predisposition elements, as well as the triggering and perpetuating factors, among

which genetic predisposition [3-5] and psycho-socio-economical conditions [6-9] are included.

Facial pain is one of the most influential reasons for seeking a healthcare provider, since it can lead to important physical, psychological, and social limitations to the patient, regardless of whether the pain is localized or referred [10-11]. However, the diagnosis of facial pain is not simple and involves various confounding factors, such as the situations and syndromes which are characterized by the presence of pain in the maxillomandibular region and annexed structures. Among these conditions are included sinusitis, tension headaches, cranial nerves neuralgia, atypical facial pains, Eagle's syndrome, stylohyoid ligament syndrome, facial reflex sympathetic dystrophy, cervical radiculopathy, neck tension, cervicothoracic interspinous bursitis, supraspinatus syndrome, intercostals neuralgias, thoracic vertebral compression, spinal stenosis, benign and malignant lesions of the maxillomandibular complex, as well as fibromyalgia (FM) [12].

Fibromyalgia is a syndrome of still unknown etiology, characterized by chronic and diffuse pain, muscle rigidness, non-repairing sleep, fatigue, and comorbidity conditions [13]. According to the American College of Rheumatology, patients with fibromyalgia present generalized pain through the body for at least 3 months, as well as positive palpation of at least 11 of the 18 specific tender points [14]. This syndrome is among the most common musculoskeletal rheumatoid diseases affecting the adult population, and epidemiological studies point to a predominance in females. Table 1 illustrates a chronological perspective regarding major studies about the prevalence of FM in the human population.

Table 1 – Chronological and topographical perspective of the major studies regarding the prevalence of fibromyalgia in the human population

AUTHOR	YEAR	LOCATION	N	METHOD	RESULTS
Makela & Heliovaara [15]	1991	Finland	3434	Interview and tender points localization by palpation	1,57%
Prescott *et al.* [16]	1993	Denmark	123	American College of Rheumatology criteria for fibromyalgia	0.66%
Forseth & Gran [17]	1993	Norway	2498 women	Questionnaire regarding the presence of pain, as well as exams according to rheumatic disease criteria	10,5%
Wolfe *et al.* [18]	1995	London – England	391	Questionnaire regarding the presence of no pain, non-diffuse pain, and diffuse pain	3.4% for women; and 0.5% for men. Prevalence increases with age
White *et al*. [19]	1999	Kansas - EUA	311	American College of Rheumatology criteria for fibromyalgia	3.4% for women and 0.5% for men
Lindell *et al*. [20]	2000	Sweden	147	Use of algometer for assessing tender points, pain threshold, prevalence of chronic pain and fibromyalgia	1,3%
Lundberg & Gerdle [21]	2002	Nikoping – Sweden	643 women	Presence of tender points, quality of life, pain provocation index, presence and intensity of pain, quality of vacation, and time away from work due to pain	2.0%.
Schochat & Rasp [22]	2003	Germany	623 women	Subjects were clinically evaluated regarding the presence of trigger points	11,5%
Salaffi *et al*. [23]	2005	Italy	2155	Questionnaire were sent by mail to subjects, which were then evaluated by trained rheumatologists	2,2%
Topbas *et al*. [24]	2005	Turkey	1930 women	Interview regarding items related to the diagnosis of fibromyalgia and examination by a speciallist	3.6%
Haq *et al*. [25]	2005	Bangladesh	5211	Questionnaire conducted door-to-door by trained interviewers, and examination by trained rheumatologists to those who responded positively to the presence of musculoskeletal pain	4,4%
Mas [26]	2008	Spain	2192	Classification and exams conducted by experience rheumatologists, which investigated musculoskeletal dysfunctions by validated instruments found in the literature	2.4%; of which 4.2% in women and 0,2% in men

Assumpção *et al*. [27]	2009	Embú - Brasil	304	Visual analog scale, questionnaire about the impact of fibromyalgia, algometer over the tender points	4,4%
Bannwarth *et al*. [28]	2009	France	1014	Questionnaire over the phone about the epidemiological classification for fibromyalgia, confirmatory exames conducted by trained rheumatologists	2,2,%
Branco *et al*. [29]	2010	France, Germany, Italy, Portugal and Spain	1225	Epidemiological classification questionnaire for fibromyalgia of London, conducted over the phone. Confirmatory exams conducted by 8 rheumatologists according to the American College of Rheumatology	2,9 to 4,7%
Alvarez-Nemegyei *et al*. [30]	2011	Mexico	3915	Transverse crossover study utilizing the Community Orientated Program in the Rheumatic Diseases	0,2%
Chaaya *et al*. [31]	2012	Lebanon	3530 subjects over 15 years of age, 944 clinically examined	Interview by the Community Orientated Program in the Rheumatic Diseases method, clinical evaluation of positive cases with the American College of Rheumatology criteria	6,2%

Both TMD and FM can be considered functional disorders of the musculoskeletal system, which are not necessarily related to traumatic causes. According the World Health Organization [32], these disorders are among the greatest causes of morbidities in the world population, contributing to important socio-economical issues and causing enormous burdens on healthcare systems. Approximately one quarter of Europe's adult population present musculoskeletal alteration capable of limiting their daily activities [33], and this reality could be very similar to that of Brazil. Even though it can appear in all age levels, musculoskeletal disorders tend to become aggravated as age progresses. Considering the increase of the elderly population in the world, and observing changes in life style that contribute to an increase in musculoskeletal alterations, there is a tendency that these diseases will overload healthcare systems in the near future.

Pain Pathogenesis

It is known that the pain found in FM is strongly related to an increase in central sensitization as a source of persistent musculoskeletal pain, which apparently is responsible for aggravation of chronic cranial pain [34, 35], and possibly the maxillofacial complex. Central sensitization is a phenomenon found in practically all chronic nociceptive or neuropathic pain events, where nociceptive stimuli induce a reduction in pain threshold, therefore creating hyperalgesia in primary sensitive areas and allodynia in areas adjacent to inflammation [35]. It is possible to believe that these reactions are closely correlated to spatial and temporal summation phenomena resulting from neuronal excitability [36, 37], which could account for greater prevalence of such cases in women rather than men [38]. Apparently, other regional painful syndromes are correlated, such as irritable bowel syndrome, interstitial cystitis, headaches, chronic low back pain, chronic neck pain, and some chronic manifestations of TMD, which appear to be associated with this abnormal amplification of nociceptive stimuli arising from visceral or musculoskeletal structures [39, 40]. Although these syndromes posses different etiologies, the pain modulating mechanism appears to be the same. Moreover, these disturbances generally occur simultaneously [41], can act as risk factors for the development of another syndrome [42], or can lead to a transition from a localized pain disorder to a generalized pain condition [43]. While the beginning of these disorders result from peripheral pain mechanisms (inflammation and/or irritation of mucosa or neural structures), the persistent nociceptive afferents lead to changes in the central nociceptive system. After induction of central sensitization, painful sensations can occur independently of peripheral nociceptive input [44], which significantly increases the complexity of treatment for such disorders. By broadening our comprehension of the shared pathophysiology of these conditions, it promotes greater collaboration between researches from different fields, in order to improve our ideas regarding the underlying mechanisms and aide in advancing treatment options for these pain processing disturbances.

In reality, the cause of increased sensitivity to pain in FM is still unknown, but the correlations of FM

with sensitive processing alterations of the central nervous system have been identified, as well as the alterations in peripheral tissue [45]. The central abnormalities appear to be related to the blunting of the hypothalamic-hypophyseal axis with regards to response to stressor events [46,47], and to the increase in substance P levels [48,49], excitatory amino acids [50], and neurotrophins in cerebrospinal fluid [51], as well as in activation and differentiation of glial cells in light of an exaggerated excitatory stimulus [45]. Pre-existing pain, psychological factors, congenital elements, sexual hormones, stress, negative emotions, depression and alterations in sleep patterns should be considered due to their importance in stabilizing spatial summation of the nociceptive afferents.

While our understanding of the developmental stages of chronic pain and hyperalgesia is traditionally focused on transmission of pain signals through ascending pathways (from periphery to the spinal and supra-spinal centers), a rising body of research amplifies our notion of the decreasing influence on generation and maintenance of sensitization [52]. The periaqueductal gray-rostral ventromedial medulla (PAG-RVM) is essential for descending modulation of pain. The influence of the descending inhibitory system (anti-nociceptive) has been documented for a long time [53]. The first evidence of its existence observed that the stimulation of PAG produced profound analgesia in rats [54]. The study demonstrated that the stimulation of PAG-RVM related to descending serotonergic, noradrenergic, and opioid pathways results in an analgesic response at the dorsal horn level.

Correlation between TMD and FM

The presence of signs and symptoms of TMD in individuals with FM was observed in 1988, when Ericksson et al. [55] conducted a study in Switzerland and observed a positive response to the presence of TMD signs in four of eight patients with primary FM. These patients were analyzed according to the Helkimo TMD index, but the same authors also suggested that a more detailed assessment of the stomatognathic system might have been necessary in order to elucidate possible etiological factors contributing to the subjects' complaints. Although this study can be taken into consideration due to its historic value, it also presents a series of experimental flaws, such as an extremely reduced sample size, an absence of a control group, a lack of proper examination, and other important methodological factors.

Hedenberg-Magnusson et al. (1997) [56], in another study from Switzerland, compared patients with FM, patients with TMD, and a control group, and observed that pain threshold due to pressure and pain tolerance levels were less for patients with FM than those with TMD. However, both groups were more sensitive to the painful stimuli than the individuals from control group. These authors concluded that patients with FM frequently have symptoms of TMD, primarily in relation to local myalgias.

The association between TMD and FM was defended by Fricton [57] in 2004, in which the author discussed the implications of comorbidities between diagnosis and treatment of TMD and FM. This research suggests that a straight correlation exists between both diseases. However, the author does not refer to important criteria pertinent to this conclusion, since there is no adequate information related to the populations studied, if correlations were found between the affected groups and the control groups, the classification methods utilized, and similar important aspects.

Manfredini et al. (2004) [58], investigated patients with TMD with regards to the presence of FM and vice-versa, and observed that the difference between both groups was not relevant, with the exception of values for active and passive mouth opening, and for the presence of trigger points, which were more common and aggravated in individuals with FM. However, the majority of patients with FM (86.7%) presented signs and symptoms within the stomatognathic system. On the other hand, only a fraction of the patients with TMD (10%) presented signs and symptoms of FM when assessed by rheumatologic examination and evaluation. Plesh et al. (1996) [59] also studied the prevalence of TMD in patients with FM and vice-versa, and discussed about which characteristics better distinguish between the two disorders. These authors verified that a small portion of patients with TMD (18.4%) presented symptoms of FM, but most subjects with FM (75.0%) complied with the criteria for myofascial TMD. Again, it was observed that patients with FM present inferior pain threshold than those with TMD.

Leblebici et al. (2007) [60] sought to determine the coexistence between FM, TMD, and myofascial masticatory pain (MMP). All patients were examined by a dentist and a physiatrist in order to assess the coexistence of FM and TMD. In the FM group, TMD was evidenced in 80% of the patients, and only 19% of them were of arthrogenic origin with MMP. In the TMD group, FM was observed in 52% of the patients,

which was significantly higher in patients with TMD of arthrogenic origin with MMP. The results indicate that the coexistence of TMD and FM with MMP is high. Pain and the sensitivity of masticatory muscles seem to be an important element in FM, which may actually be the chief complaint in some patients.

Fraga et al. (2012) [61] observed the presence of signs and symptoms of TMD in patients with fibromyalgia. Sixty patients diagnosed with FM were evaluated by means of the Research Diagnostic Criteria for Temporomandibular Disorder (RDC-TMD), which provides standardized criteria for the diagnosis of TMD. The most common symptoms related by patients with FM were headaches (97%) and facial pain (81.7%). Myofascial pain without limitation of mouth opening was diagnosed in 47% of patients, which represents a subgroup I diagnosis on the RDC-TMD. Dislocation of the disc with reduction was the most prevalent diagnosis in subgroup II (21.6%), and 36.7% of subjects presented osteoarthritis, a subgroup III diagnosis. Only 6.6% of patients did not present any sign of TMD. There is an elevated prevalence of signs and symptoms of TMD in patients with FM, indicating the need of an integrated diagnosis and treatment approach to these patients, as it is suggested that FM can be a risk factor in the mid- to long-term for the development of TMD.

In light of the exposed, it is possible to notice that there is less evidence of suffering in patients with TMD, and that this condition is much more localized. However, FM is a generalized disorder, where many patients present symptoms of TMD, and these are generally muscular in nature, as opposed to articular. Possibly, muscular TMD, when induced by localized trauma [62,63] and associated to chronic stress [64], could alter the temporomandibular joints and cause sensitive modifications to joint biomechanics, which promotes capsular and discal microtraumas, and leads to a greater perception of pain. This would not necessarily be present in patients with FM, where pain from a centralized origin would be exerting greater influence in sensitivity to pain.

Discussion

There is still controversy when dealing with the pathophysiology of pain in FM and TMD. Various study strands are found in the literature, however there is still little consensus regarding this topic. It is understood that the comprehension of the pathophysiology of neurogenic pain, as well as its influence on pain perception threshold, is limited due to the infeasibility of experimental models in humans, since our behavior in response to pain is not the same as that observed in animals. The scarcity of studies with human subjects, due to ethical questions, urges the improvement of experimental models in animals to standards which are closely related to clinical reality. It is our belief that these translational studies, which establish correlation between experiments with animal models and the clinical, controlled, and randomized trials, are invaluable to comprehend and establish interventions for pain's triggering and maintenance mechanisms, specifically regarding FM and TMD.

In the same manner, some evidence suggests that congenital factors predispose some people to symptoms of chronic pain, both in FM [65,66] and TMD [67-70]. Studies conducted by fellow researches [3-5] also confirm our hypothesis that polymorphic alterations appear to change the expression of signs and symptoms of TMD. Genetic mutations of a certain species are the foundation for its evolution, since it creates new elements (allele) which may or may not become part of the genetic pool for that population. When a genetic variance reaches a populational frequency of 1%, it becomes a polymorphism. To date, many genetic polymorphisms are being related to an increase in occurrence and/or reoccurrence risk of various multifactorial pathologies, and are considered as part of the genetic component which increases the susceptibility of an individual to present certain behavior due to a certain factor, which consists as an augmented risk to this behavior. This is what occurs in pain expression in virtue to greater susceptibility to central neuronal alterations. It is our belief that this fact, as evidenced in the literature, will lead a new frontier of research which will elucidate the reason such alteration in pain modulating systems occur in certain groups of people. This will certainly open a new door of understanding to this intricate topic.

In light of this literature review, we conclude that a comorbidity between FM and TMD probably exist, since there is a high prevalence of TMD in patients with FM. This indicates a need for adequate diagnosis and management of signs and symptoms, suggesting that FM can be a mid- to long-term risk factors for the development of TMD [61]. Individuals with FM presented greater complexity with regards to their clinical TMD condition, therefore the importance of a precise and adequate evaluation of the stomatognathic systems in patients with FM is evident, and require greater care during diagnosis and therapeutic intervention. It is the responsibility of the

dental surgeon to comprehend the complex nature of these diseases and, based on their particularities, propose not only care related to the ongoing pain, but especially identify the initial signs and symptoms related to FM and refer the patient to a rheumatologist. We suggest that, due to the complexities of the stomatognathic system and its interrelations with structures commonly involved in FM, the dental surgeon should be consulted in order to manage the orofacial manifestations of the disease, both in TMD and in the interventions aimed at the oral symptoms generated by FM.

Resumo

A disfunção temporomandibular (DTM) e a fibromialgia (FM) são síndromes musculo-esqueléticas que parecerem estar associadas. Vários estudos na literatura relatam sinais e sintomas da DTM em pacientes com FM. O objetivo deste trabalho foi analisar a literatura quanto à associação clínica entre fibromialgia e as disfunções temporomandibulares. Apesar de estes distúrbios possuírem etiologias diferentes, o mecanismo de modulação de dor é semelhante. Muitas pesquisas desenvolvidas nessa área buscam elucidar esta questão ainda pouco compreendida. Mas sugere-se que estas síndromes são caracterizadas por uma diminuição no limiar de dor e na capacidade de atenuação de sistemas descendente de modulação da dor. Além disso, há certo grau de comorbidade entre estas duas patologias, que compartilham muitas características clínicas. Muitos pacientes com FM apresentam vários sinais e sintomas de DTM, entretanto, uma pequena quantidade de indivíduos com DTM recebem um diagnóstico de FM. Portanto, é importante uma avaliação precisa e adequada do sistema estomatognático em pacientes com fibromialgia englobando o diagnóstico de disfunção temporomandibular na intervenção terapêutica.

Palavras-chave

Disfunção temporomandibular; fibromialgia; dor facial.

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