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FACTORS INVOLVED IN THE ETIOLOGY OF TEMPOROMANDIBULAR DISORDERS - A LITERATURE REVIEW

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Abstract

Background and aim. This review aims at presenting a current view on the most frequent factors involved in the mechanisms causing temporomandibular disorders (TMD).

Method. We conducted a critical review of the literature for the period January 2000 to December 2014 to identify factors related to TMD development and persistence.

Results. The etiology of TMD is multidimensional: biomechanical, neuromuscular, bio-psychosocial and biological factors may contribute to the disorder. Occlusal overloading and parafunctions (bruxism) are frequently involved as biomechanical factors; increased levels of estrogen hormones are considered biological factors affecting the temporo-mandibular-joint. Among bio-psychosocial factors, stress, anxiety or depression, were frequently encountered.

Conclusions. The etiopathogenesis of this condition is poorly understood, therefore TMDs are difficult to diagnose and manage. Early and correct identification of the possible etiologic factors will enable the appropriate treatment scheme application in order to reduce or eliminate TMDs debilitating signs and symptoms.

Keywords: temporomandibular joint disorder, etiology, factors, pathogenesis

Background and aim

Temporomandibular disorders include alterations of the temporomandibular joint (TMJ) and associated structures, including facial and neck muscles [1].

About 60-70% of the general population has at least one sign of temporomandibular joint dysfunction (TMD), but only one out of four individuals is aware of these symptoms and reports them to a specialist [2].

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The most important symptom is pain, followed by restricted mandibular movements, which can cause difficulty in eating or speaking; noises from the temporomandibular joints during jaw movement are also recorded. The etiology and pathogenesis of this condition is poorly understood, therefore treatment of temporomandibular joint diseases is sometimes difficult. Understanding the etiology of temporomandibular joint disorders is extremely important in identifying and avoiding potential pathologic factors

This review aims at presenting a current view on the most frequent factors involved in the mechanisms associated with TMD development.

Material and method

A literature search was carried out in the Cochrane, PubMed, Scopus and Web of Science databases covering the period January 1980 through December 2014. Various combinations of keywords related to TMJ dysfunction and aspects of etiology were used (etiologic factors, etiology, temporomandibular joint dysfunction, disorder, disc displacement, dental occlusion, estrogen hormones, emotional stress, anxiety, depression). Databases were searched for papers published in English. Of the initial 1215 abstracts found 1025 were excluded. Excluded abstracts were those of repeated studies and studies with unrelated scopes. Another 136 studies were also excluded because they were not clearly related to the review topics.

Results

A total of 54 articles being considered most relevant were selected for this review.

There are numerous factors that can contribute to this disorder, which are grouped into three categories. Predisposing factors increase the risk of developing TMD, initiating factors cause the onset of the disease and perpetuating factors interfere with the healing process or enhance the progression of TMD. In some instances, a single factor may serve one or all of these roles. The successful management of TMD is dependent on identifying and controlling the contributing factors.

Etiological factors include occlusal abnormalities, orthodontic treatment, bruxism and orthopedic instability, macrotrauma and microtrauma, joint laxity and exogenous estrogen [3]. Psychological factors such as stress, mental tension, anxiety or depression can cause TMD.

Initiating factors lead to the onset of the symptoms and are primarily related to trauma or adverse loading of the masticatory system. Perpetuating factors may include the following:

- Behavioral factors (grinding, clenching and abnormal head posture)
- Social factors (affect perception and influence of learned response to pain)
- Emotional factors (depression and anxiety)
- Cognitive factors

Predisposing factors are pathophysiological, psychological or structural processes that alter the masticatory system and lead to an increase in the risk of development of TMD.

1. Occlusal factors

Occlusion is the first and probably the most controversial etiologic factor of TMD. Costen was the one who first established with certainty the involvement of occlusion in the development of TMD. Nowadays, most researchers include occlusion among all the factors related to TMD, having a possible role in both susceptibility and onset or perpetuation of TMD (Table I) [4,5,6].

Pullinger and Seligman applied multiple factor analysis, which indicated the low correlation of occlusion to temporomandibular disorders. In this research, they compared occlusal characteristics in patients with symptoms of TMD with a group of patients without symptoms of TMD. The authors concluded that malocclusion could act as a co-factor in the etiology of TMD and some occlusal features might be consequences of the disease rather than initiating factors. They estimated that occlusal factors contributed about 10-20% to the total spectrum of etiological factors in TMD [7]. On the other hand, Rammelsberg offered a review of the etiopathogenic model of TMD development wherein high abrasion and insufficient restorative procedure on posterior teeth are risk factors causing occlusal instability [8].

According to the literature, the existing data cannot determine the exact role of occlusal factors in temporomandibular joint disorders. Conversely, in the meta-analysis conducted by Koh et al. [9], the authors concluded that there was insufficient data on the treatment or prophylaxis of TMD by occlusal rebalancing.

With regard to the distribution of occlusal contacts, the symmetry of their intensity rather than the symmetry of their number in the posterior occlusion is more important for temporomandibular function [10].

Improper occlusions due to dental malpositions, untreated or improperly treated edentulism are pathological states of temporomandibular complex, but they are not considered the main etiological factors of TMD [11]. Badel et al. [12] have identified a low incidence of certain variables of malocclusion (unilateral open bite, negative overjet, unilateral cross bite in men, and edge-to-edge bite in women) with signs or symptoms of TMD. In the

Table I. Occlusal factors in TMD.

The following occlusal factors showed a possible correlation with temporomandibular disorders:

Posterior cross-bite

Overiet/overbite greater than 5 mm

Centric Relation/ Maximum Intercuspal sliding greater than 2 mm

Edge-to-edge bite

Sagittal relation Class III

Anterior open bite

Five or more missing teeth

same study, they found a significantly higher prevalence of hyperbalance and interference contacts in asymptomatic patients compared to TMD patients. No difference was found between Angle's classes in patients with TMD and asymptomatic individuals.

The importance of occlusal interferences is perceived differently based on the etiopathogenesis of TMD. Le Bell et al. [13] found that artificial interferences do not stimulate the development of dysfunctional symptoms in healthy subjects, who adapt successfully to them. In patients with medical histories of TMD, artificial interferences enhance clinical symptoms.

Posterior cross-bite is a cause of asymmetric muscle functioning, but no certain correlation with TMD has been determined as yet [14,15].

The presence of mediotrusive interferences is considered by some authors to be a predisposing factor for disk displacement [16,17], whereas others suggest that they can exert a protective action [18]. The presence of an anterior open-bite can be considered a consequence of articular remodeling [19] rather than the cause [20].

Condylar position may also play a significant role in the etiopathogenesis of TMJ disorders [21,22]. A recent study conducted by Padala et al. [23] evaluated the relationship between condylar position and centric occlusion-centric relation discrepancy and presence of signs and symptoms of TMD. The findings in this study indicate that recording and evaluating the centric relation-centric occlusion discrepancy in individuals with TMD may reveal significant dental inter-arch discrepancies and condylar displacements of significant magnitude. Weffort et al. [24] obtained similar results indicating that statistically significant differences between centric relation and maximum intercuspation were quantifiable at the condylar level in symptomatic and asymptomatic individuals.

Debates over occlusal characteristics have influenced and limited therapeutic options for TMD, but recent research works have highlighted the multifactorial etiology of the disease and reduced the importance of occlusion as a major etiological factor of TMD [25].

2. Psychological factors

The role of stress and personality in the etiology of the temporomandibular pain dysfunction syndrome has undergone extensive scrutiny. Psychological studies have shown that patients with TMD have similar psychological profiles and psychological dysfunction as other chronic musculoskeletal pain disorders, such as tension type headache and back or arthritic pain [26,27]. There is considerable evidence that psychological and psychosocial factors are of great importance in the understanding of TMD, but there is less evidence that these factors are etiologic [28].

Nowadays the association between depression and stress and different physical symptoms of TMD is widely acknowledged [29,30,31].TMD symptoms, especially pain,

are also discussed as being a causative or intensifying factor in the development of depression and psychic diseases.

Stress, anxiety and other psychological factors induce muscle hyperactivity and muscle fatigue with the appearance of muscle spasms and the following consequences: contracture, occlusal disharmony, internal disturbances and degenerative arthritis. These factors can alter the occlusal scheme of the masticatory cycle, so that these alterations are more a result of TMD and not a triggering factor. Various studies have confirmed that patients with myofascial pain or myofascial pain associated with arthralgia, arthritis or osteoarthritis present more advanced stages of depression and somatization than those diagnosed with disc displacement [32].

3. Hormonal factors

Signs and symptoms of TMD are four times more common among women, who seek specialized treatment for this disease three times more frequently than men. Despite the fact that the low prevalence of TMD in men has not been completely elucidated yet, the presence of higher testosterone levels may be a plausible explanation [33].

There is the hypothesis that the presence of estrogen receptors in women's TMJ changes metabolic functions increasing ligament laxity. Estrogen also increases susceptibility to painful stimuli by modulating the limbic system. Although researchers do not share the same opinion, studies in humans have shown that painful symptoms increase by 30% among patients on menopause treatment with estrogen replacement therapy and by 20% in women using oral contraceptives [34].

Polymorphism in the estrogen receptor has been shown to be correlated to the intensity of pain, facial axis angle and mandibular body length in patients who suffer from TMJ osteoarthritis. However, despite these studies, until recently no direct evidence has been found that links female reproductive hormones to TMJ disease or that defines the mechanisms by which these hormones may cause TMJ disease. A recent study has shown that estrogens and relaxin could contribute to the degeneration of cartilage homeostasis by disrupting TMJ and inducing activation of metalloproteinases (MMP) that degrade cartilage matrix macromolecules (collagen and proteoglycans) [35].

4. Macrotrauma

Macrotrauma is a predisposing and initiating factor for TMD. Whiplash-type injuries to the head or neck are commonly considered significant risk factors in the development of this pathology [36,37]. Sale [38] suggested that one in three people exposed to whiplash trauma is at risk of developing delayed TMJ symptoms.

A study including 400 patients with TMD revealed that in 24.5% of them, the presence of TMJ pain was directly correlated with a history of trauma [39].

Probert et al. [40] conducted an extensive study in Australia, including 20,673 patients who were victims of car accidents. TMD was diagnosed in only 28 patients,

and only one of the 237 patients who suffered fractures of the jaw required further treatment for TMD. It was thus concluded that the incidence of TMD after whiplash injury was very low and individual trauma couldn't be considered a triggering factor for the onset of TMD.

Endotracheal intubation has also been proposed as a risk factor for TMJ dysfunction in case reports and systematic studies. Any association between endotracheal intubation and the development of short-term TMD symptoms is likely to be found in patients with a history of such dysfunctional symptoms [41].

5. Parafunctions

Parafunctions are defined as impaired or altered functions of TMJ. Of these, excessive gum chewing, teeth clenching and bruxism have been extensively studied as possible risk factors for TMD.

In a study including 3,557 students, Miyake et al. [42] identified bruxism and chewing gum on one side as risk factors for TMD. In individuals who frequently chew gum, more than four hours a day, auricular pain is more frequent at rest and during movements and there is a higher frequency of joint noise [43]. Lateral movement of the jaw or protrusion without tooth contact are often significantly associated with joint pain, joint noise and joint blockage.

Studies found bruxism in 87.5% of patients with disc displacement and joint pain. The association between bruxism and TMD symptoms is based on the theory according to which the repeated overuse of TMJ determines functional abnormalities [44]. Bruxism is more commonly associated with muscle dysfunction and less associated with joint dysfunction, such as disc displacement. This parafunction may result in condylar bone remodeling and articular cartilage degradation and may contribute to the development of osteoarthritis of the TMJ [45,46].

The prevalence of bruxism is reported to be 20% among the adult population, similar to the frequency of the disease among children. In a study conducted in Boston and based on parent's reports, Cheifetz et al. [47] reported a 38% frequency of bruxism in children. Only 5% of them showed signs of TMD. The highest incidence of bruxism is in the 20-50 years age range and then it gradually declines. Magnusson et al. [48] conducted a longitudinal study on a group of 420 individuals over a period of 20 years, reporting a significant correlation between bruxism and TMD.

Huang et al. [49] assessed a group of patients diagnosed with myofascial pain (n=97), arthralgia (n=20) and combined myofascial pain and arthralgia (n=157), identifying a strong correlation between tooth clenching and the presence of myofascial pain (OR=4.8).

6. Joint hyperlaxity and joint hypermobility

The relationship between hypermobility and TMD has also been studied. Some authors have reported no association between TMD and systemic hyperlaxity or between TMJ mobility and systemic hypermobility, while others found a positive relationship between generalized

joint hypermobility and TMD [50]. Kavuncu et al. assessed the risk for TMD in patients with systemic hypermobility and TMJ hypermobility. They found that both local and general hypermobility were more frequently detected in patients with TMD than in the controls, and that the risk of TMJ dysfunction was greater if the patient presented both alterations simultaneously [51].

These data are consistent with the results of the study conducted by Coster et al. [52], who examined 31 subjects with Ehler-Danlos syndrome, all of them showing signs and symptoms of TMD with recurrent condylar subluxations. These results are in contrast to those presented by Conti et al. [53] comparing a group of 60 patients with symptoms of TMD with a group of 60 asymptomatic patients. There was no association between TMD and systemic hyperlaxity and between TMD hyperlaxity and systemic hyperlaxity.

7. Hereditary factors

Michalowicz et al. [54] evaluated the hypothesis that signs and symptoms of TMD may be hereditary. They collected information through questionnaires from 494 monozygotic and dizygotic twins. Monozygotic twins showed no significant similarities with dizygotic twins, and monozygotic twins who grew up together showed similar characteristics when compared to each other. The authors conclude that genetic factors and the family environment exert no relevant effect upon the presence of symptoms and signs of the TMJ.

Conclusion

The etiology of TMD is multifactorial, as evidenced by the combination of psychological, physiological, structural, postural and genetic factors, altering the functional balance between the fundamental elements of the stomatognathic system: dental occlusion, jaw muscles and TMJ.

In time TMD symptoms (pain, psychological discomfort, physical disability and limitation of mandibular movements) can become chronic and affect quality of life. Treatment options are limited and sometimes fail to meet the long-term demands of the relatively young patient population. That is why it is particularly important to quickly identify possible etiologic factors and their degree of involvement, enabling the best treatment to improve and eliminate the debilitating symptoms of TMD.

References

- 1. Dimitroulis G. Temporomandibular disorders: a clinical update. BMJ. 1998;317(7152):190-194.
- 2. List T, Stenstrom B, Lundstrom I, Dworkin SF. TMD in patients with primary Sjogren syndrome: a comparison with temporomandibular clinic cases and controls. J Orofac Pain. 1999;13:21-28.
- 3. Gage JP. Collagen biosynthesis related to temporomandibular joint clicking in childhood. J Prosthet Dent. 1985;53:714–717.
- 4. McNeill C. Craniomandibular Disorders: Guidelines for Evaluation, Diagnosis and Management. Chicago: Quintessence;

- 1990, pp. 25-39
- 5. Almăşan OC, Băciuţ M, Almăşan HA, Bran S, Lascu L, Iancu M, et al. Skeletal pattern in subjects with temporomandibular joint disorders. Arch Med Sci. 2013;9(1):118-126.
- 6. Almăşan OC, Băciuţ M, Băciuţ G. Influenţa disfuncției temporomandibulare asupra tiparului scheletic la subiecţi cu anomalie de clasa a III-a scheletică [The influence of temporomandibular dysfunction on the skeletal pattern in patients with class 3 skeletal abnormality]. Clujul Medical. 2012; 85(S1):47-50.
- 7. Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. J Prosthet Dent. 2000;83:66-75.
- 8. Rammelsberg P. Untersuchungen über Ätiologie, diagnose und Therapie von Diskopathien des Kiefergelenkes. Berlin: Quintessenz; 1998.
- 9. Koh H, Robinson PG. Occlusal adjustment for treating and preventing temporomandibular joint disorders. Cochrane Database Syst Rev. 2003;(1):CD003812.
- 10. Gianniri AI, Melsen B, Nielsen L, Athanasiou AE. Occlusal contacts in maximum intercuspation and craniomandibular dysfunction in 16- to 17-year-old adolescents. J Oral Rehabil. 1991;18:49–59.
- 11. Carlsson GE. Some dogmas related to prosthodontics, temporomandibular disorders and occlusion. Acta Odontol Scand. 2010:68:313-322.
- 12. Badel T, Marotti M, Krolo I, Kern J, Keros J. Occlusion in patients with temporomandibular joint anterior disk displacement. Acta Clin Croat. 2008;47:129-136.
- 13. Le Bell Y, Jämsä T, Korri S, Niemi PM, Alanen P. Effect of artificial occlusal interferences depends on previous experience of temporomandibular disorders. Acta Odontol Scand. 2002;60:219-222.
- 14. Landi N, Manfredini D, Tognini F, Romagnoli M, Bosco M. Quantification of the relative risk of multiple occlusal variables for muscle disorders of the stomatognathic system. J Prosthet Dent. 2004;92:190-195.
- 15. McNamara JA Jr, Seligman DA, Okeson JP. Occlusion, Orthodontic treatment, and temporomandibular disorders: a review. J Orofac Pain. 1995;9:73-90.
- 16. Kirveskari P, Alanen P, Jämsä T. Association between craniomandibular disorders and occlusal interferences in children. J Prosthet Dent. 1992;67:692-696.
- 17. Shiau YY, Chang C. An epidemiological study of temporomandibular disorders in university students of Taiwan. Community Dent Oral Epidemiol.1992;20:43-47.
- 18. Minagi S, Ohtsuki H, Sato T, Ishii A. Effect of balancing-side occlusion on the ipsilateral TMJ dynamics under clenching. J Oral Rehabil. 1997;24:57-62.
- 19. John MT, Frank H, Lobbezoo F, Drangsholt M, Dette KE. No association between incisal tooth wear and temporomandibular disorders. J Prosthet Dent. 2002;87:197-203.
- 20. Schmitter M, Balke Z, Hassel A, Ohlmann B, Rammelsberg P. The prevalence of myofascial pain and its association with occlusal factors in a threshold country non-patient population. Clin Oral Investig. 2007;11:277-281.
- 21. Almăşan OC, Hedesiu M, Baciut G, Baciut M, Bran S, Jacobs R. Nontraumatic bilateral bifid condyle and intermittent joint lock: a case report and literature review. J Oral Maxillofac Surg. 2011;69(8):e297-e303.

- 22. Almăşan OC, Hedeşiu M, Băciuţ G, Leucuţa DC, Băciuţ M. Disk and joint morphology variations on coronal and sagittal MRI in temporomandibular joint disorders. Clin Oral Investig. 2013;17(4):1243-1250.
- 23. Padala S, Padmanabhan S, Chithranjan AB. Comparative evaluation of condylar position in symptomatic (TMJ dysfunction) and asymptomatic individuals. Indian J Dent Res. 2012;23(1):122. 24. Weffort SY, de Fantini SM. Condylar displacement between centric relation and maximum intercuspation in symptomatic and asymptomatic individuals. Angle Orthod. 2010;80:835-842.
- 25. Gesch D, Bernhardt O, Mack F, John U, Kocher T, Alte D. Association of malocclusion and functional occlusion with subjective symptoms of TMD in adults: results of the Study of Health in Pomerania (SHIP). Angle Orthod. 2005;75:183-190.
- 26. Suvinen TI, Reade PC. Temporomandibular disorders: a critical review of the nature of pain and its assessment. J Orofac Pain. 1995;9:317-339.
- 27. Dworkin SF, Massoth DL. Temporomandibular disorders and chronic pain: disease or illness? J Prosthet Dent. 1994;72:29-38.
- 28. Lupton DE. Psychological aspects of temporomandibular joint dysfunction. J Am Dent Assoc. 1969;79:131-136.
- 29. Yap AU, Dworkin SF, Chua EK, List T, Tan KB, Tan HH. Prevalence of temporomandibular disorder subtypes, psychologic distress, and psychosocial dysfunction in Asian patients. J Orofac Pain. 2003;17:21-28.
- 30. Yap AU, Tan KB, Chua EK, Tan HH. Depression and somatization in patients with temporomandibular disorders. J Prosthet Dent. 2002;88:479-484.
- 31. Steed PA, Wexler GB. Temporomandibular disorders—traumatic etiology vs. nontraumatic etiology: a clinical and methodological inquiry into symptomatology and treatment outcomes. Cranio. 2001;19:188-194.
- 32. Ferrando M, Andreu Y, Galdon MJ, Dura E, Poveda R, Bagan JV. Psychological variables and temporomandibular disorders: distress, coping, and personality. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004;98:153-60.
- 33. Fischer L, Clemente JT, Tambeli CH. The protective role of testosterone in the development of temporomandibular joint pain. J Pain. 2007;8:437-442.
- 34. LeResche L, Saunders K, Von Korff MR, Barlow W, Dworkin SF. Use of exogenous hormones and risk of temporomandibular disorder pain. Pain. 1997;69:153-160.
- 35. Wang W, Hayami T, Kapila S. Estrogen and relaxin induce while progesterone represses MMP expression in TMJ fibrochondrocytes. J Dent Res. 2007;86:1279.
- 36. Fischer DJ, Mueller BA, Critchlow CW, LeResche L. The association of temporomandibular disorder pain with history of head and neck injury in adolescents. J Orofac Pain. 2006;20:191-198.
- 37. Klobas L, Tegelberg A, Axelsson S. Symptoms and signs of temporomandibular disorders in individuals with chronic whiplash-associated disorders. Swed Dent J. 2004;28:29-36.
- 38. Packard RC. The relationship of neck injury and post-traumatic headache. Curr Pain Headache Rep. 2002;6:301-307.
- 39. De Boever JA, Keersmaekers K. Trauma in patients with temporomandibular disorders: frequency and treatment outcome. J Oral Rehabil. 1996;23:91–96.
- 40. Probert TC, Wiesenfeld D, Reade PC. Temporomandibular pain dysfunction disorder resulting from road traffic accidents-an Australian study. Int J Oral Maxillofac Surg. 1994;23(6 Pt 1):338-341.

- 41. Martin MD, Wilson KJ, Ross BK, Souter K. Intubation risk factors for temporomandibular joint/facial pain. Anesth Prog. 2007;54:109-114.
- 42. Miyake R, Ohkubo R, Takehara J, Morita M. Oral parafunctions and association with symptoms of temporomandibular disorders in Japanese university students. J Oral Rehabil. 2004;31:518-523.
- 43. Winocur E, Gavish A, Finkelshtein T, Halachmi M, Gazit E. Oral habits among adolescent girls and their association with symptoms of temporomandibular disorders. J Oral Rehabil. 2001;28:624-629.
- 44. Schierz O, John MT, Schroeder E, Lobbezoo F. Association between anterior tooth wear and temporomandibular disorder pain in a German population. J Prosthet Dent. 2007;97:305-309.
- 45. Güler N, Yatmaz PI, Ataoglu H, Emlik D, Uckan S. Temporomandibular internal derangement: correlation of MRI findings with clinical symptoms of pain and joint sounds in patients with bruxing behaviour. Dentomaxillofac Radiol. 2003;32:304-10.
- 46. Israel HA, Scrivani SJ. The interdisciplinary approach to oral, facial and head pain. J Am Dent Assoc. 2000;131:919-926.
- 47. Cheifetz AT, Osganian SK, Allred EN, Needleman HL. Prevalence of bruxism and associated correlates in children as reported by parents. J Dent Child (Chic). 2005;72:67-73.
- 48. Magnusson T, Egermarki I, Carlsson GE. A prospective

- investigation over two decades on signs and symptoms of temporomandibular disorders and associated variables. A final summary, Acta Odontol Scand. 2005;63:99-109.
- 49. Huang GJ, LeResche L, Critchlow CW, Martin MD, Drangsholt MT. Risk factors for diagnostic subgroups of painful temporomandibular disorders (TMD). J Dent Res. 2002;81:284-288.
- 50. De Coster PJ, Van den Berghe LI, Martens LC. Generalized joint hypermobility and temporomandibular disorders: inherited connective tissue disease as a model with maximum expression. J Orofac Pain. 2005;19:47-57.
- 51. Kavuncu V, Sahin S, Kamanli A, Karan A, Aksoy C. The role of systemic hypermobility and condylar hypermobility in temporomandibular joint dysfunction syndrome. Rheumatol Int. 2006;26:257-260.
- 52. De Coster PJ, Martens LC, De Paepe A. Oral health in prevalent types of Ehlers-Danlos syndromes. J Oral Pathol Med. 2005;34:298-307.
- 53. Conti PC, Miranda JE, Araujo CR. Relationship between systemic joint laxity, TMJ hypertranslation, and intra-articular disorders. Cranio. 2000;18:192-197.
- 54. Michalowicz BS, Pihlstrom BL, Hodges JS, Bouchard TJ Jr. No heritability of temporomandibular joint signs and symptoms. J Dent Res. 2000;79:1573-1578.