

Advances in imaging studies on the effects of sleep bruxism with temporomandibular joint disorder on joint bone structure: A review

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Abstract: The temporomandibular joint (TMJ) is the only bilateral joint of the human body, which bears important physiological functions such as opening and closing mouth. Temporomandibular joint disorders is the most common oral disease with a high incidence of 30-40 %. Sleep bruxism (SB) is a kind of parafunctional activity, characterized by the contraction of the mandibular muscles and clenching or molars. Studies have shown that bruxism is considered to play a key role in the occurrence and development of temporomandibular disorders (TMD) and is positively correlated with the incidence of TMD. However, the similarities and differences in the occurrence and mechanism of condylar and articular tubercle bone changes between temporomandibular joint disc displacement and bruxism have not been studied. Based on this, CBCT imaging was used to study the bone mineral density (condyle, joint nodules) of joints and the morphology of joint bone structure in patients with bruxism and temporomandibular joint disc displacement, so as to understand whether there are differences in the effects of reversible and irreversible displacement (disk displacement with or without reduction) displacement without reduction and bruxism on joint bone structure.

We compare the changes of joint space and condylar bone structure between patients with bruxism and asymptomatic non-molars, to explore the influence of bruxism on temporomandibular joint, and to provide theoretical basis for clinical diagnosis and treatment of patients with bruxism-temporomandibular joint disorder.

1. Temporomandibular joint disc displacement

The temporomandibular joint, also known as the mandibular joint, forms a bicondylar joint [1]. It is a rotating and sliding joint composed of the articular surface of the temporal bone, the mandibular condyle, the articular disc, and the joint capsule and the ligaments inside and outside the capsule. It is a complex structure in anatomy and biomechanics [2]. Among them, the nutritional environment of the articular disc profoundly affects cell energy metabolism, proliferation and biosynthesis [3].

TMD is a degenerative musculoskeletal disease associated with morphological and functional malformations, affecting up to 25 % of the population. However, little is known about its etiology and progression [4], and its pathogenesis has not yet been elucidated. The symptoms are soreness and pain of joints and corresponding muscles, difficulty in opening mouth or abnormal mandibular movement, accompanied by joint pain, headache, ear pain, dizziness and even tinnitus [5] Mainly through medical history and clinical examination, according to the international general RDC / TMD biaxial diagnostic criteria . Temporomandibular joint disc displacement is a high incidence of clinical lesions, which can be divided into reversible anterior disc displacement and irreversible anterior disc displacement (anterior disk displacement with or without reduction)[6]. Reducible disc displacement refers to the anterior displacement of the articular disc in the closed position, and the normal disc-condylar relationship can be restored in the open position. The clinical manifestations are mostly joint snapping, pain and abnormal mouth opening. The mechanism of irreversible disc displacement is the same as that of reversible disc displacement. The difference is that when the opening moves, the articular disc deformed by the condylar extrusion cannot be reset, and the normal condylar-articular disc relationship cannot be restored. Clinically, there is a typical history of joint snapping, followed by a history of intermittent joint strangulation, and then the snapping disappears, the opening is limited, the jaw is biased toward the affected side when opening, and the joint area is painful. When measuring the passive opening, the opening cannot be increased. The diagnostic criteria for the normal position of the articular disc in the imaging MRI examination is that the posterior band of the articular disc is located at 12 o'clock [7]. When its position on the condyle changes, it is called articular disc displacement. If it is reversible anterior displacement, the middle zone of the articular disc is located between the condyle and the articular tubercle at the maximum opening position; if the anterior disc displacement is irreversible, the posterior band of the articular disc is still located in front of the condyle at the maximum opening position.

The development and rehabilitation of the disease seems to be closely related to the adaptability of the tissues of the joint organs. Studies have shown that inflammatory factors and bone resorption-related factors play an important regulatory role in the pathogenesis of temporomandibular disorders. Among the inflammatory factors, Interleukin-1 and Tumor Necrosis Factor- α (TNF- α) are involved in the regulation; the related factor RANKL is an important index to evaluate the damage degree of inflammatory joint disease. In addition, the destruction of the environmental or genetic clock may also make cartilage susceptible to diseases such as osteoarthritis [8]. The proteins expressed by circadian clock genes in chondrocytes can regulate the homeostasis of cartilage anabolism and catabolism. The expression of circadian clock genes is affected by circadian rhythms. When circadian rhythms are disordered, symptoms such as osteoarthritis will occur. Inflammation is a key driver of the pathogenesis of osteoarthropathy and has been shown to disrupt circadian rhythms in cartilage.

In the study of histological changes of temporomandibular joint disc, Feng Jianying et al. [9] found in animal experiments that active apoptosis and proliferation of rabbit condylar chondrocytes occurred after disc displacement, and finally formed adaptive changes. The changes of condylar surface can be observed by imaging methods. Animal experiments have also shown that under the stimulation of excessive pressure, the cartilage stem cells of the condylar fibrocartilage layer will also undergo significant apoptosis, which is reflected in the macroscopic reduction of the fibrocartilage layer and the reduction of condylar bone formation. Studies have shown that early degenerative lesions can occur in the articular disc and condyle in cases of delayed disc displacement. Anterior disc displacement can activate the apoptosis mechanism of chondrocytes and initiate the adaptive remodeling of articular cartilage.

2. Sleep bruxism

According to its circadian rhythm phenotype, bruxism is designated as sleep bruxism or awake bruxism [10]. Sleep bruxism (SB) is a parafunctional activity characterized by the contraction of the mandibular muscles and the clenching or grinding of teeth. It can lead to abnormal changes in the oral and maxillofacial system, such as tooth wear, headache, temporomandibular joint and muscle pain and other symptoms [11]. The etiology is complex and not completely clear. It can be caused by four groups of factors : group 1-biological factors and genetic factors ; the second group-exogenous factors ; group 3 - psychological factors, stress, some special personality, sleep-wake response, a variety of neurotransmitters, group 4 - sleep disorders (such as obstructive sleep apnea), etc.

It has been reviewed that the abnormality of neurotransmitters can cause abnormal coordinated movement of masticatory muscles to cause bruxism [12], which may be related to the destruction of the balance of basal ganglia output channels [13]. In addition, bruxism is closely related to sleep micro-awakening [14]. The basic form of its attack is rhythmic movement of the masticatory muscles [15]. The damage to the oral and maxillofacial system is first manifested on the head and face muscles, and the temporomandibular joint symptoms appear later. [16] During the attack, the contraction activity of the masticatory muscle increases, and the bite force increases significantly. The load generated can lead to degenerative changes in the temporomandibular joint.

Recurrent bruxism is one of the main causes of excessive load on the temporomandibular joint [17] Frequent overload usually accelerates apoptosis and cartilage degradation, which can cause increased free radicals in the temporomandibular joint to degrade hyaluronic acid to accelerate cartilage degradation and bone resorption. The shear stress generated during molars can increase the release of nitric oxide and the increase of chondrocyte apoptosis, as well as proteoglycan degradation, synovial changes, inflammation and synovial fluid changes, resulting in impaired chondrocyte lubrication and nutrition, and ultimately cartilage degradation. Arnett et al. proposed that the pressure generated by nocturnal molars can compress the temporomandibular joint, causing condylar absorption or enhancing absorption caused by other factors. Israel et al. proved under arthroscopy that the load generated by the side function is an important cause of temporomandibular arthritis.

The normal shape of the articular disc, when it is subjected to abnormal high shear force, such as sleep bruxism activity, can cause damage and degradation. The form and function are compatible, and when it cannot bear these abnormal loads, the form will change. Sleep bruxism is a common source of microtrauma. Repeated and persistent inflammation within the temporomandibular joint can affect the collagen structure of the articular disc, resulting in deterioration of its ultrastructure and nanomechanical properties, which may be a key factor in the displacement of the articular disc. The articular surface is compressed and releases synovial fluid, and the tissue behind the disc produces free radicals and nitric oxide.

3. Others

Most scholars believe that there is a link between self-reported bruxism and TMD, and that bruxism plays an important role in TMD. A longitudinal study has shown that bruxism is an important predictor of TMD. Three-dimensional finite element analysis shows that the shear stress generated by clenching and grinding activities can damage the articular disc and articular cartilage, and form TMD when it exceeds the physiological tolerance limit. Although related cross-sectional studies do not indicate a causal relationship between abnormal molar activity and disc displacement, they can also indicate that abnormal molar activity is associated with disc displacement.

4. Imageology study

In recent years, the imaging methods of temporomandibular joint include panoramic radiography, computed tomography (CT), magnetic resonance imaging (MRI) and CBCT. CBCT has become a common examination method for imaging diagnosis of oral and maxillofacial diseases. Its three-dimensional reconstruction image can also be rotated at any angle to observe and select any reconstruction range, which is of great significance for disease diagnosis, curative effect observation and scientific research. In addition, the operation of CBCT is simple and easy to use. However, for the diagnosis of temporomandibular joint disease, magnetic resonance imaging is the gold standard. Although MRI can be used to evaluate bone changes, CBCT is often used to detect related bone lesions^[18]. CBCT gray value has a strong correlation with bone mineral density, and it is feasible to evaluate bone mineral density with CBCT. CBCT 3D imaging improves the linear measurement accuracy (width, length, and height) of the mandible. It has been reported that CBCT images can accurately evaluate the linear measurement results of the condyle.^[19] CBCT can accurately reflect the changes of bone structure and space of temporomandibular joint. CBCT was used to observe the changes of temporomandibular joint space to reflect the changes of temporomandibular joint position and articular disc position. By observing the changes in the density of hard tissue in the temporomandibular joint area, the effect of TMD on bone was explained. By observing the changes in the morphology of the temporomandibular joint area, it is suggested whether there is a degenerative change^[20]. CBCT shows a high accuracy of the image, which can observe the influence of the three-dimensional data of the scanned area. As far as the temporomandibular joint is concerned, CBCT can observe the tissue structure of the temporomandibular joint from various angles, levels and sections, to better prompt and guide the clinic.

In summary: The development of MRI and CBCT is of great significance for us to explore the influence of bruxism and temporomandibular joint disc displacement on temporomandibular joint. We found that bruxism and temporomandibular joint disorder have a certain effect on the temporomandibular joint, whether it is soft tissue or hard tissue. The temporomandibular joint can undergo adaptive or degenerative changes under stimulation.

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