치의과학박사 학위논문

Effects of Occlusal Splint Therapy on Dentofacial Skeletal Pattern in Juvenile Patients with Temporomandibular Joint Osteoarthritis

사춘기 턱관절 골관절염 환자에서 교합안정장치 치료가 안모의 골격변화에 미치는 영향에 관한 연구

2016년 8월

서울대학교 대학원

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지도교수 정 진 우

이 논문을 치의과학박사 학위논문으로 제출함
2016 년 4 월

서울대학교 대학원

치의과학과 구강내과진단학 전공

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김형준의 치의과학박사 학위논문을 인준함
2016 년 6 월

위 원 장 (인)

부위원장 (인)

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ABSTRACT

Effects of Occlusal Splint Therapy on Dentofacial Skeletal Pattern in Juvenile Patients with Temporomandibular Joint Osteoarthritis

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Temporomandibular joint osteoarthritis (TMJ OA) has been generally known to be a degenerative condition of TMJ led by a pathological tissue response of the joint under a mechanical overload.

The aim of this study was to evaluate the effects of occlusal splint therapy on the dentofacial skeletal pattern and symptoms of juvenile patients with TMJ OA based on the cephalometric radiograph analysis and Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD).

A total of 34 juvenile patients under 16 years old diagnosed with TMJ OA based on computed tomography who visited Seoul National University Dental Hospital were studied. The patients were classified into 28 splint therapy group treated by stabilization splint for at least 6 months and 6 non-splint therapy group(control group) treated by physical therapy, medication, and behavioral control only. Pre-treatment and post-treatment clinical symptoms including subjective pain intensity, maximum mouth opening (MMO), comfortable mouth opening (CMO), number of muscle tenderness, and cortical bone erosion of mandibular condyle based on computed tomography.

Cephalometric analysis of 9 angular measurements including articular angle, gonial angle, Y-axis to SN, SN to mandibular plane angle, SNA, SNB, ANB, gonial upper angle,

and gonial lower angle were analyzed on each patient. Mean durations of the evaluation were 19.9 ± 9.7 months for the splint therapy group and 20.2 ± 11.4 months for the control group. Obtained results were as follows:

1. Splint therapy group showed more significant increases in MMO and CMO after treatment than control group.
2. Condylar erosion severity did not show significant decrease in both groups.
3. Pain intensities were significantly decreased after treatment in both groups, but there were no significant differences between both groups.
4. Number of positive masticatory muscle palpation was significantly decreased after treatment in both groups, but there were no significant differences between both groups.
5. Articular angle, Y-axis to SN, SN to mandibular plane angle, gonial upper, and lower angles were significantly changed in splint therapy group and Y-axis to SN, SN to mandibular plane angle, SNB, ANB, and gonial lower angle were significantly changed in control group. SNB was less significantly decreased after treatment in splint therapy group than control group.

In conclusion, there were no significant differences in condylar erosion severity and most of cephalometric parameters of juvenile TMJ OA patients between splint therapy and control groups within 20 months. Clockwise rotations of mandibles were shown in both groups, however modestly less decrease of SNB was shown in splint therapy group than control group. Further longitudinal study based on a larger population should be considered for establishing the effective treatment planning of juveniles TMJ OA patients.

Key words: Temporomandibular joint, Osteoarthritis, Stabilization splint, Juvenile, Cephalography

Student Number: 2013-30634

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Ⅰ. INTRODUCTION

Temporomandibular joint osteoarthritis (TMJ OA) has been generally known to be a degenerative condition of TMJ disease led by a pathological tissue response of the joint under excessive mechanical stress, inflammation, abnormal remodeling of subchondral bone, chondrocyte apoptosis, genetic, and hormonal factors.[1,2)]

Symptoms of patients with TMJ OA can progress as a consequence of sustained mechanical load which exceeds the adaptive capacity of the joint and the tissue responses of the joint can be changed to degeneration, causing further degradation of hard tissue, and secondary inflammation changes.[2)]

A number of studies have suggested conservative treatments including pharmacological therapy such as administration of NSAIDs, physical therapy, behavior control to relax and avoid parafunction, and occlusal splint therapy for successful treatment of TMJ OA.[3-7)] However, most of these studies mainly focused on improving symptoms, clinical signs, and cortical bone of articular joint than improving or preventing skeletal pattern.

There are numerous studies on the effect of TMJ OA on dentofacial skeletal structures in juveniles.[8-14)] Main growth area of the lower jaw placed in the condyle is close to the synovial membrane which could be easily affected by inflammation owing to their proliferate area in the articular cartilage.[8)] Therefore, it is susceptible to growth disturbances, leading to ramus height shortening, clockwise rotation of mandible, anterior open-bite, mandibular plane angle increase, and facial asymmetries.[9-14)] So, it is important to detect the osteoarthritis of mandibular condyle and provide early treatment by careful medication, physical therapy, and occlusal splint treatment in order to provide the possibility for restoration of influenced mandibular condyle and to avoid facial deformity. But, there have been few clinical information concerning the prognosis of skeletal pattern change related to TMJ OA in juvenile patients.

Generally, it has been reported that clinical symptoms of TMJ OA are not different from other types of temporomandibular disorders including joint noise, articular pain, and masticatory dysfunctions. However, other study reported that the clinical signs and

symptoms might not associated with prognosis of osteoarthritic bony changes.[15] Currently, according to Diagnostic Criteria of Temporomandibular Disorders (DC/TMD), TMJ OA is diagnosed by not only clinical signs and symptoms but bony change based on computed tomograpy (CT). Moreover, the diagnosis for juvenile arthritis has been considered difficult because presentation of subjective symptoms in the children is insufficient or delayed, and varied signs may be unclear to diagnose.[16-18] Therefore, most juvenile patients with TMJ OA are diagnosed by bony change based on CT rather than clinical signs and symptoms.

For these reasons, there are few longitudinal studies about treatment effects and changes on the skeletal pattern of juvenile patients with TMJ OA. Furthermore there are very few reports whether the occlusal splint prevent or accelerate the clockwise rotation of mandible, and affect the growth of jaw bones of juvenile TMJ OA patients.

The aim of this study was to evaluate the effects of occlusal splint therapy on the dentofacial skeletal pattern and symptoms of juvenile patients with TMJ OA based on the cephalometric radiograph analysis and Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD).

Ⅱ. REVIEW OF LITERATURE

1. Epidemiology of TMJ OA

(1) TMJ OA

The temporomandibular joint (TMJ) is defined as a synovial joint carrying out the most complex movements among human joints. Osteoarthritis (OA) is a degenerative disease characterized by cartilage destruction, subchondral bone remodeling, inflammation, and chronic pain.[19]

Generally, it has been reported that clinical symptoms of TMJ OA are not different from other types of temporomandibular disorders including joint noise, articular pain, and masticatory dysfunctions. According to the clinical diagnostic criteria of the RDC/TMD Axis I diagnosis, TMJ OA is defined as an Inflammatory condition within the joint that

results from a degenerative condition of the joint structures.[20] Meanwhile, according to DC/TMD, suggested by international RDC/TMD consortium and orofacial pain special interest group to provide more clear and simple criteria in clinical treatment and research, TMJ OA is classified under degenerative joint disease (DJD), a degenerative disorder involving the joint characterized by deterioration of articular tissue with concomitant osseous changes in the condyle and/or articular eminence.[21] Currently, cone-beam computed tomography (CBCT) has led to more comprehensive understanding of TMJ bone than conventional radiographic methods, indicating a superior benefit in TMJ OA diagnosis.[22] So, the diagnosis of TMJ OA by DC/TMD is confirmed by CT of the articular eminence and condyle including subchondral cyst, erosion, generalized sclerosis or osteophyte.

TMJ OA patients generally suffer from dysfunction of the TMJ and consequent pain and low quality of life. TMJ OA etiology is complicated, multifactorial and/or obscure. TMJ OA is also a significant subtype of temporomandibular disorders (TMDs).[23]

Mechanical overload on normal articular cartilage or ordinary mechanical load on damaged articular cartilage is commonly considered to originate degradation of cartilage matrix homeostasis, causing OA.[2] TMJ OA may be different from OA in hip or knees, associated with aging, obesity, and overloading.[24] The causes of condylar cartilage degradation in the TMJ is still ambiguous. Increasing attention has placed emphasis on synovitis and subchondral bone remodeling, but the pathophysiology of TMJ OA is still contentious and unclear.

(2) Epidemiology

It has been suggested in literatures that prevalence of TMD is extensively variable. Fifty-six percent of the population showed at least one clinical sign, 5% to 33% reported subjective signs and symptoms of TMD, and the occurrence of TMD pain was about 7-15% of the adult population in United States.[25-28]

TMJ OA prevalence has been known about 8-16% of general population, relatively lower than that of TMD, and articular joint involvement may be unilateral or bilateral.[28-30] Before adolescence, the percentage of prevalence of TMJ OA was reported 2% to 3.5% lower than general population.[16,31]

Numerous researches revealed that females showed a strong predominance of symptoms and signs of TMD. In various epidemiologic researches, the prevalence of TMD pain demonstrated 1.5 - 2 times higher in females than males of the community and 80% of treated cases were of females.[29] In accordance with age, for adults, the prevalence of TMD was higher in females than in males; particularly, females in reproductive age displayed higher prevalence of TMD than those of in postmenopausal years.[28,32] Some researchers showed that females more often had more severe degrees of psychological and physical signs and symptoms than males.[30]

(3) Juvenile TMJ OA

According to a review of 34 epidemiological researches of juvenile arthritis (JA) began since 1966, in general, occurrence of JA is subtle, which may bring about little pain in children and result in delayed or completely missed diagnosis.[16] Signs are varied, and sometimes the clinical condition may be unclear and hard to diagnose.[17,18] So, researches relying on surveys of children, their parents, or of hospital or practitioners' notes may bring about certain prejudice like the capability of parents to answer surveys, language obstacles, and insufficient presentation of symptoms in the children.[16]

Since many children first show obvious symptoms of JA between 12 and 16 years of age, prevalence will be undervalued where the upper limit is 12 years. Current definition of JA starts before the 16th birthday.[16]

In juvenile TMD patients, joint sounds were reported 11%, and mandibular movement restriction and stiffness in 3%.[33] There was also a study reporting that TMJ sounds were observed in 7% of juvenile patients, orofacial pain in 7%, joint pain in 16%, and muscle tenderness in 25% while adult patients reported joint sounds in 38% of the subjects and muscle tenderness in 12%.[34] these previous studies reported that it might be difficult to diagnose TMJ OA in juvenile patients.

It is important to detect TMJ arthritis and to provide early treatment by careful medication, physical therapy, behavioral control to relax and avoid parafunctional habits like bruxism and clenching, and occlusal splint therapy in order to stabilize the TMJ condition and to provide the possibility for restoration of the influenced condyle, and to avoid deformity.[35,36]

2. Pathophysiology of TMJ OA

(1) Inflammation

In comparison with rheumatoid arthritis, there was a report that TMJ OA was categorized as an "arthritic condition in low inflammation," contrary to rheumatoid arthritis, which was categorized as a high-inflammatory condition.[37] However, Inflammation that occurs during progression of TMJ OA has been considered important. It was reported that inflammatory cytokines such as Interleukin (IL)-12, tumor necrosis factor (TNF)-α, and IL-6 were increased in the articular joint fluid of TMJ OA patients,[38,39] meaning that chronic inflammation in TMJ might lead to the degradation of hard and soft tissues of articular joint.

Whether markers indicating cartilage destruction go higher in the synovial fluid of TMJ OA patients or not remains to be studied. So far, the diagnostic markers or potential prognostic indicators for TMJ OA has not been found.

(2) Mechanical load

The articular surface of TMJ, which consists of fibrocartilage and underlying subchondral bone, is sensitive to mechanical stress and tends to remodeling extensively. Excessive mechanical stress has been considered a main cause of cartilage destruction in the TMJ.[2]

In spite of host-adaptive capacity, extreme or unbalanced mechanical load on the TMJ can bring about overloading on articular soft and hard tissues, which can cause progression of TMJ OA. Moreover, excessive or unbalanced loading on TMJ may result in internal derangement (ID) of the TMJ. Trauma, parafunction such as bruxism, malocclusion, functional overload, and increased joint friction also affect TMJ OA and ID of the TMJ.[40-43]

Macrotrauma, which is in the condylar area, can result in deterioration of articular cartilage and elaboration of inflammatory and pain mediators. It has been reported that trauma changes the mechanical characteristics of the disc.[44] and gives rise to mechanical fatigue of the disc.[45,46] TMJ changes happened over time after the macrotrauma, resulting in progression of condylar bone resorption and deformation.[47] Yet, only about one-third

of the TMJ individuals with degenerative alteration apparently had previous trauma to the neck and head.[48] The mechanism of condylar resorption and deformation within secondary macrotrauma is not yet fully understood.

Parafunction such as sleep bruxism and clenching may also result in excessive compression and shear forces, which can initiate disc displacement and TMJ degenerative bone changes.[49] The lateral pterygoid muscle that is related to parafunctional hyperactivity has been considered to cause masticatory muscle pain.[50,51] Because the lateral pterygoid muscle's superior head attaches partly to the TMJ articular capsule and its articular disc,[51] Dysfunction of this muscle has been speculated to bring about TMJ OA and ID.[50]

Meanwhile, Milam et al. suggested the reperfusion/hypoxia damage model due to direct mechanical damage, proposing that oxidative stress gives rise to accumulation of free radicals damaging the TMJ articular tissues.[52] Several studies reported that reactive oxidative radical species was detected in synovial joint fluid of TMD.[53,54]

Recent studies have reported on the molecular pathway of the TMJ cartilage destruction. The effect of mechanical load on the TMJ chondrocytes has been assessed in vitro and in vivo. Excessive mechanical stress activates the plasminogen activator system, resulting in proteolysis of extracellular matrix components.[55] There is growing evidence that aberrant biomechanical stimulation has an important role in the onset and progression of TMJ OA. However, mechanical stress is also needed to develop the condyle of mandible. So, there are needs for further research to uncover the mechanism.

(3) Subchondral bone remodeling

Radiographic characteristics of the subchondral bone has been known to be substantially important for TMJ OA diagnosis.[56] Numerous studies have currently paid attention to the role of subchondral bone on TMJ OA pathophysiology.[57-59] These studies propose that active change of the subchondral bone has some relation to the initial stage of TMJ OA.

In the TMJ OA model, decreased bone mineral density and subchondral bone loss showed subsequent destruction of the cartilage. The chondrocytes in the impaired cartilage may control osteoclastogenesis.[58] In the early stages on TMJ OA, TMJ

subchondral bone remodeling is often detected. However, the etiological function of subchondral bone turnover in TMJ OA still remains to be further studied.

(4) Biochemical factors

Chondrocyte death which is caused either by necrosis or by apoptosis is speculated to be a significant characteristic in the TMJ cartilage degradation clinically or experimentally.[60] The catabolic enzymes up-regulation in the cartilage matrix, like MMP, disintegrin, and metalloproteinase with thrombospondin motifs (ADAMTS), is concerned with the condylar cartilage degradation in the early stage of TMJ OA.[61] Several studies have investigated the molecular mechanisms of cartilage destruction. However, it needs to be further studied.

TMJ OA has a female predominance, proposing a possible function of female hormones in the progression of the disease. Accordingly, the estrogen effects on subchondral bone and condylar cartilage have been assessed. It is reported that estrone/17β-estradiol can be conversed to pro-inflammatory metabolites in the OA synovial cells of the knee joint, indicating that pro-inflammatory metabolites in joint membrane is related to the effects of estradiol in the TMJ inflammation.[62] Wang et al. also reported that estrogen impaired cartilage and subchondral bone by upregulating Fas and caspase 3–related proapoptotic genes in an iodoacetate-induced rat model of TMJ OA.[63] These findings propose that estrogen may have an impact on the TMJ OA. In contrast, it is reported that estrogen has a protective impact on the TMJ chondrocyte by preventing the expression of nitric oxide.[64] Therefore, the function of estrogen in TMJ OA pathogenesis remains still questionable. Furthermore, the effects of other female hormones such as relaxin and progestin on the progress of cartilage destruction in the TMJ should be studied further.

Several genes have been known to be related to TMD.[65] However, no single-nucleotide polymorphism has been found in the TMJ OA genome study.[66] Suggestion of a genetic vulnerability or predisposition towards TMJ OA should be investigated further.

(5) Juvenile TMJ OA pathogenesis

It has been reported that main growth area of the lower jaw placed in the condyles is close to the synovial membrane, which could be easily affected by inflammation owing to their proliferate area in the articular cartilage; therefore, condylar destruction of TMJ OA can lead to growth disturbances including ramus and mandibular body length decrease.[67)] Consequently, this can bring about mandibular development of a posterior growth rotation, increase in mandibular plane angle, and lower facial height and antigonial notching.[67-69)] This negative growth pattern may result in a Class II division 1 abnormal occlusion with anterior open-bite.[69-72)]

The maxilla is frequently horizontally well located even in the presence of serious interruption of mandibular growth.[67)] Meanwhile, vertically, posterior maxillary development tends to be retarded because of ramus height decrease. Clinically, this is shown as an inclined occlusal plane which can have a negative impact on facial morphology and mandibular function.[68,69,73)]

It is reported that condylar shape and volume asymmetry was found in juvenile patients with TMJ OA (20 subjects: 14 women, 6 men) diagnosed by CBCT. Still, a limitation of a cross-sectional study existed.[74,75)] There was also a retrospective study suggesting that unilateral TMJ OA is related to mandibular asymmetry and increased activity of the masseter muscle on the OA side.[76)] However, the relationship among TMJ OA, dentofacial morphology, and muscle overuse remains to be unclear.

Arvidsson et al. reported that according to a 30-year prospective longitudinal study, 70% of adult subjects who had been diagnosed as juvenile idiopathic arthritis(JIA) presented frequent bilateral TMJ condyle deformities, or osteoarthritis.[76)]

Consequently, TMJ OA can lead to dentofacial skeletal change including ramus height shortening, mandible's clockwise rotation, mandibular plane angle increase, bird face, micrognathia, abnormal occlusions, and facial asymmetries.[9-14)]

(6) Disc displacement vs clockwise rotation

Disk displacement (DD) is commonly found in TMD, which may result in TMJ sounds such as clicking and crepitus, arthrogenous pain, masticatory muscle pain, and mouth opening limitation.[77,78)] If TMD symptoms are aggravated, DD with reduction state usually progresses to DD without reduction state.[79)]

DD also can affect facial morphology, since it can change the condylar and mandibular shape.[80,81] Ahn et al. reported that TMJ DD patients have skeletal features of mandibular backward rotation, ramus height shortness, mandibular body length decrease, and mandibular asymmetry.[82,83] Notably, mandibular backward rotation and body length decrease are considered as cephalometric characteristics of TMJ DD.[82,83] These skeletal features come to be more serious as DD develops.[82,83] This was consistent to a previous study based on 30-year follow-up data, where radiograph of OA changes were extensively observed in the most of permanent disk displacements.[84]

Therefore, if CMO or MMO increase is significantly achieved by treatment such as occlusal splint therapy, stabilization splint therapy may be effective in improving mandibular range of motion leading to prevent clockwise rotation of mandible. However, there is also a possibility that other common causes of TMJ OA may bring about condylar resorption resulting in negative skeletal changes. Thus, the effect of DD on dentofacial skeletal changes needs to be studied further.

3. Treatment of TMJ OA

Treatment of TMD may be classified into non-invasive and invasive treatments. The purposes of treatment in TMD should be: (1) to reduce TMJ pain, inflammation, and muscle spasm, (2) to recover joint function, (3) to inhibit further joint impairment, and (4) to inhibit morbidity of disease.[2]

(1) Non-invasive treatments

The non-invasive treatments involve medications, biochemical therapy, physical therapy, behavioral control to relax and avoid parafunction habits like bruxism and clenching, and occlusal splint therapy.

Medications such as NSAIDs, like ibuprofen, should be prescribed on a time-contingent basis in order to utilize the effects of their pharmacokinetics. Muscle relaxants may be supportive in regulating the masticatory muscle spams.[85]

Biochemical therapy such as platelet-rich plasma (PRP) contains considerable growth factors that are released from greatly concentrated activated platelets. PRP can be helpful in improving formation of bone in severe surgically-induced degenerative TMJ alteration

in rabbits. However, there was no significant influence on repair of cartilage destruction.[86] Mesenchymal stem cells (MSCs) can be a biochemical means of regenerative treatment for TMJ OA since they can be easily collected and differentiated into cartilage and bone.[87] The stem cells injection for restoration of articular tissues has been suggested.[88] However, such researches are mainly related to OA in the knee. Mechanisms based on osteogenic effects of MSCs for preventing TMJ OA progression are still unclear.

Physical therapy has been known to play a role in decreasing pain and inflammation. Moist hot pack or localized cold pack may adequately reduce pain and allow exercise. Remedial exercises have goals to enhance muscle strength, decrease joint contractures, and preserve a functional range of motion. Electro-stimulation, ultrasound, and massage therapy can help to reduce inflammation and pain.[89] There was also a report that passive movements of jaw, relaxation, and manual therapy were applied for treatment of 20 consecutive individuals with TMJ OA, resulting in significant TMJ pain decrease by 80% without functional damage.[90]

(2) Invasive treatments

Hyaluronic acid (HA) injection applied in other body joints has been revealed to offer significant superior results than saline in double-blind studies on other joints after 2 months. However, there were no significant differences found in radiographic progression on the disease.[91] There was also an in vivo animal study, reporting that HA injected rabbit joints showed less fibrillation and limited cartilage change, whereas the prednisolone-treated joint showed diminishing cartilage damage.[92] So far, however, hyaluronic acid has not been accepted by the USA FDA as a secure and effective medicine in the treatment of arthritic disease of the TMJ.

Corticosteroids injections have limits to usage on other body joints.[93] Core limitations of repetitive steroid injections are risks of infection and articular cartilage damage. Repetitive corticosteroid injections can lead to TMJ "chemical condylectomy."[94] Steroid injection must be considered only for an acute high inflammation in the joint. Multiple steroid injections should not be used.

Arthrocentesis has been known to have an efficacy in restoration of functional ability to the osteoarthrosis joints. But there was a 32% failure rate because of painful limited jaw function resulting from fibrous adhesion.[95] Meanwhile, It has been reported that TMJ arthroscopy is important since it can be used to for early diagnosis and help management of TMJ OA particularly in early-stage.[96] However, late-stage characterized by fibrosis or ankylosis can make arthroscopy impractical or useless.

Arthroplasty means altering articular surfaces in order to remove osteophytes, erosions, and irregularities detected in OA that shows no reaction to other treatments was suggested by Dingman and Grabb.[97] But there can be a failure of mandibular dysfunction, facial asymmetry, abnormal occlusion, articular bone deterioration, disc loss or disorder, and ankylosis. The requirement for TMJ disc replacement in such cases is controversial.[98]

Orthognathic surgery like osteotomy for patients with active TMJ OA has been reported to frequently show low outcomes and noteworthy relapse. If patients have active TMJ pathology, orthognathic surgery can have negative results, including condylar resorption, osteochondroma, and congenital deformities.[99]

Joint Replacement Procedures such as costochondral graft has been suggested for TMJ reconstruction because of its good adjustment to the recipient site, low morbidity rate at the donor site and its growth potential in juveniles.[100] Meanwhile, orthopedists recommend alloplastic restoration when total joint replacement is needed for patients who stop growing.[101] But alloplastic restoration in the TMJ has been argued. Most studies have suggested that only when the mandibular condyle is widely impaired, deteriorated, or lost, as in conditions of arthritis, either alloplastic or autogenous graft could be an suitable treatment.[102,103] But long-term prognosis is uncertain. Hence, it seems that entire alloplastic TMJ reconstruction should be accepted for advanced-stage TMJ OA.

(3) Splint therapy and its therapeutic effects

Occlusal splint are generally used to treat functional disorder of the masticatory system.[104] When appropriately adapted, it can be a good treatment tool to stabilize articular tissues, decreasing abnormal neuromuscular activity and achieving stable occlusal interaction. Conservative treatment including occlusal splint has been reported to succeed clinically in the treatment of TMD including TMJ OA.[105-109]

The efficacy of occlusal splints has been controversial.[110] However, there has been sufficient evidences to show that using a occlusal splint reduce hyperactivity of the elevator muscles and to reduce muscle tension and muscle spasms.[111-113]

Patients with sleep bruxism who use occlusal splint have shown a remarked decrease in nocturnal activity in the masseter muscle.[114] But electromyographic (EMG) values were back to pretreatment levels when the occlusal splint was eliminated after two weeks testing.

Mandibular perception was also enhanced in TMD patients with use of occlusal splint.[115,116] However, it was reported that TMD patients who had used occlusal splint for one month did not show reproducibility of pantographic recordings, though it provided relief of clinical symptoms and decreased EMG muscle rest activity.[117]

Carraro et al. suggested three goals of occlusal splint including : 1. TMJ Pain and dysfunction will take advantage of occlusal splint treatment. 2. Patients treated with an occlusal splint will obtain better result in pain response than in dysfunction response. 3. Full-coverage occlusal splint had about 80% success rate for TMD patients.[118]

It has been also reported that occlusal splints help prevent the TMJ from involuntary overload and to decrease muscle hyperactivity and articular tension owing to bruxism. In a controlled research on how occlusal splint treatments work in severe TMJ OA patients, splint therapy improved the clinical symptoms of TMJ arthralgia.[119] On the contrary, some randomized controlled trials (RCTs) research on 80 TMD patients showed that splint therapy was not advantageous in relief of pain compared to counseling and TMJ related muscle exercises (duration of one month).[120] Splint therapy effect on OA has not been fully investigated, despite its increasing clinical use. However, a current retrospective clinical study indicates that a stabilization splint has a positive impact on reconstruction of condylar bone for patients with TMJ OA.[121]

Ⅲ. METHODS

1. Subjects

A total of 34 juvenile patients under 16 years old diagnosed with TMJ OA based on computed tomography who visited the Orofacial Pain Clinic of Seoul National University Dental Hospital from February 2005 to February 2013 were studied. The patients were classified into 28 splint therapy group treated by stabilization splint (Figure 1) for at least 6 months and 6 non-splint therapy group(control group) treated by physical therapy, medication, and behavioral control only. The stabilization splint was adjusted to the centric occlusion (CO) position.

Patients with rheumatoid arthritis, history of facial and jaw bone fracture, and orthognathic surgery were excluded in the study.

The research protocol was approved by the Institutional Review Board of the Seoul National University Dental Hospital (CRI 16006).

2. Clinical assessment

Clinical examinations were performed on each patient based on the RDC/TMD axis I guidelines including pain duration, pain intensity, pain on mouth opening, comfortable mouth opening (CMO), maximum mouth opening (MMO), and number of positive palpation on masseter (anterior, deep, inferior) and temporalis (anterior, middle, posterior) muscles.

Pain intensities were evaluated by both numeric rating scale (NRS) and a five-point rating scale. A five-point rating scale was used to supplement the missing case of NRS recording and classified patient's expression of pain and NRS into five pain level (Table 1).

3. Radiological assessment

TMJ osteoarthritis was defined when degenerative symptoms including erosion, generalized sclerosis, or osteophyte on the mandibular condyle were observed on radiograph, digitally generated by computed tomography (Somatom Sensation : Simens, Erlangen, Germany). All the radiographic evaluations were performed on the computed tomography and diagnosed by oral and maxillofacial radiologist.

Severity of TMJ condyle's erosion was classified using a four-point rating scale.[122-125)] Classification criteria are as follows: 0: absence of erosion, 1: slight erosion, when

decreased density is observed only in the cortical bone, 2: moderate erosion, when decreased density is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone, and 3: extensive erosion, when decreased density is observed in the cortical bone and extends below the upper layers of adjacent subcortical bone (Figure 2).

Pretreatment and posttreatment lateral cephalograms were analyzed on all the patients. All cephalograms were double-blind traced by a single investigator. Nine variables of angular measurements were calculated from fourteen landmarks digitized on each radiograph. All the landmarks are shown in Figure 3 and their angular measurements are shown in Figure 4. Nine angular measurements is as follows: articular angle, gonial angle, Y-axis to SN, SN to mandibular plane angle, SNA, SNB, ANB, gonial upper angle, and gonial lower angle.

Mean durations of the evaluation were 19.9 ± 9.7 months for the splint therapy group and 20.2 ± 11.4 months for the control group.

4. Statistical assessment

Differences in demographic and clinical features between two groups were analyzed by independent t-test and chi-square test.

Differences in clinical symptoms, condylar erosion severity, and cephalometric parameters before and after treatments in each group were analyzed by paired t-test.

The comparison of changes in clinical symptoms, condylar erosion severity, and cephalometric parameters skeletal patterns after treatment between splint therapy and control groups were assessed by multivariate repeated-measures ANCOVA. Because the age between the two groups were significantly different at the baseline, the age was calculate as a covariate.

Ⅳ. RESULTS

1. Demographic features

Table 2 shows the demographic findings of the subjects. There was a significant difference in age between splint therapy and control groups. There were no significant differences between gender ratio and duration of evaluation between groups.

2. Comparisons of clinical symptoms and condylar erosion severity between splint therapy and control groups

Comparisons of clinical symptoms and condylar erosion severity at the baseline between splint therapy and control groups are shown in Table 3.

Onset duration of splint therapy group was significantly lower than control group ($p < 0.05$). CMO and MMO at the baseline were significantly lower in the splint therapy group than control group ($p < 0.05$). There was no significant difference in the existence of pain during mouth opening between two groups

The mean value of erosion severity was 2.1 for the splint therapy group and 1.9 for the control group. There was no significant difference in the condylar erosion severity at the baseline between the two groups. There was no significant difference in the NRS of pain intensity between the groups, but five-point scale of pain level of splint therapy group was significantly lower than control group ($p < 0.05$) .

3. Changes in clinical symptoms and condylar erosion severity

Changes in clinical symptoms and condylar erosion severity after treatment in both groups are shown in Table 4.

For the range of mandibular movement, the amounts of CMO and MMO were significantly increased after treatment in splint therapy group, but there were no significant differences in amounts of both CMO and MMO after treatment in control group. For multivariate repeated-measures ANCOVA analysis, the interaction between time (before and after treatment) and group (splint therapy and control groups) was significant in the amount of both CMO and MMO ($P<0.001$), i.e., the amounts of both CMO and MMO were increased significantly in splint therapy group than control group.

Condylar erosion severity did not show significant decrease in both groups. Pain intensities of both NRS and five-point scale pain level were significantly decreased after

treatment in both groups, but there were no significant interactions between time and group.

Number of positive masticatory muscle palpation was significantly decreased after treatment in both groups, but there were no significant differences between both groups.

4. Changes in cephalometric parameters after treatment.

Table 5 shows the changes in cephalometric parameters after treatment in both groups.

Articular angle, Y-axis to SN, SN to mandibular plane angle, and gonial lower angle were significantly increased and gonial upper angle significantly decreased in splint therapy group. Y-axis to SN, SN to mandibular plane angle, ANB, and gonial lower angle were significantly increased and SNB significantly decreased in control group.

For multivariate repeated-measures ANCOVA analysis, the interaction between time (before and after treatment) and group (splint therapy and control groups) was marginally significant in SNB (P=0.051), i.e., SNB was decreased significantly in control than splint therapy groups. There were no significant interactions between time and group in other cephalometric parameters.

Ⅴ. DISCUSSION

This study was performed to provide clinical information on occlusal splint therapy, generally used in orofacial dental clinic, effects on the longitudinal dentofacial skeletal change of juvenile patients who was diagnosed as TMJ OA based on computed tomography. TMJ OA is a common and severe condition of TMD which is a degenerative condition of joint structures characterized by gradual degradation of cartilage and remodeling of subchondral bone. Especially, OA in juveniles have been known to lead to dentofacial growth disturbance including decrease of mandibular body and clockwise rotation of lower jaw. Therefore, it is suggested that proper and early treatment for juvenile patients based on systematic diagnosis such as careful radiography examination including CT and clinical assessment using RDC/TMD guideline. But, sometimes clinical

signs such as masticatory pain and restriction of mouth opening may not be clearly shown in juvenile patients with TMJ OA. So, studies on the long-term effect on dentofacial skeletal change in juvenile patients with TMJ OA who had been treated properly using occlusal splint and other treatments are few. Our study investigated differences of dentofacial skeletal pattern between splint therapy and control groups.

Our data showed the significant increases of articular angle, Y-axis to SN, SN to mandibular plane angle, and gonial lower angle, and the significant decrease of gonial upper angle in splint therapy group, the significant increases of Y-axis to SN, SN to mandibular plane angle, ANB, and gonial lower angle and the significant decrease of SNB in control group. These results indicated that mandibular condyle erosion of TMJ OA led to skeletal pattern change, which was a clockwise rotation of the mandible. It was consistent with the previous study. [67-69)] However, there were no significant differences in most of cephalometric parameters of juvenile TMJ OA patients between splint therapy and control groups within 20 months. This result suggests that splint therapy did not significantly prevent mandible clockwise rotation in 20 months, which might be considered relatively short-period time, compared to non-splint therapy including physical therapy, medication, and behavioral control. SNA was not significantly changed after treatment in both splint therapy and control groups. Considering this result, we can postulate that upper jaw did not change in TMJ OA patients. This result is consistent with the previous study that maxilla is frequently horizontally well located even in the presence of serious interruption of mandibular growth.[67)]

However, SNB was modestly less decreased after treatment in splint therapy group than control group. Decrease in SNB means clock-wise rotation of mandible. Our result implies that occlusal stabilization splint therapy might prevent the clockwise rotation of mandible in TMJ OA juvenile patients rather than non-splint therapy. We can suggest two possibilities related to the smaller SNB decrease after treatment in splint therapy than control groups. Firstly, progress of condylar erosion might be less in splint therapy group than control group although our results showed that there were no significant changes in condylar erosion severity in both groups. Secondly, occlusal splint used in our study was adjusted to the maximum intercuspal position. The condylar position of patient with TMJ OA is unstable and changed as condylar erosion progresses, thus the

occlusal splint adjusted to the centric relation (CR) position can move the mandible backward which might lead to clockwise rotation of mandible.[126] Therefore, clockwise rotation of the mandible might be less shown in CO adjusted splint therapy group in our study. Further longitudinal study based on a larger population should be needed to support this implication.

Interestingly, the increases of both CMO and MMO in splint therapy group were significantly higher than control group. This result implies that stabilization splint therapy may be effective in improving mandibular range of motion leading to prevent clockwise rotation of mandible of juvenile patients with TMJ OA rather than non-splint therapy.

There was a study which is performed based on TMJ OA patients who had been followed-up 1 year with conservative treatments including physical therapy, splint therapy, and intra-articular injections of hyaluronic acid, suggesting that osteoarthritic bony changes might be more severe when TMJ OA patients who had limited mouth opening range.[15] Regarding disk displacements without reduction is the most common cause of mouth opening restriction, this is consistent to a previous study on 30-year follow-up data, where radiograph of OA changes were extensively observed in the most of permanent disk displacements.[84] However, limited joint movements are not always associated with disk displacement. Disk displacement is not always associated with OA changes.[127] It should be considered that various causative factors of TMD could affect TMJ movement restriction, leading to TMJ OA.

CMO and MMO at the baseline were significantly lower in the splint therapy group than control group. This indicated that the control group displayed within normal range of mouth opening on the baseline. Thus, non-splint therapy such as medication, physical therapy, and behavioral control to avoid contribution factors like parafunction habits might be used to the control group rather than relatively expensive splint therapy in our subjects. However, there was no significant difference in condylar erosion severity between two groups.

Previous study suggested that prognosis of dentofacial skeletal changes are independent of clinical signs and symptoms.[15] Moreover, numerous studies suggested that it might be difficult to detect the signs and symptoms of TMD in juveniles.[16-18] So, it is important to detect the osteoarthritis of mandibular condyle based on computed

tomography and provide early treatment in order to provide the possibility for restoration of influenced mandibular condyle and to avoid facial deformity in juvenile patients during growth period.[35,36] Treatment options for juvenile patients with TMJ OA also should be carefully considered.

Our study evaluated the longitudinal effect of the occlusal splint therapy on juvenile dentofacial skeletal pattern, which has been rarely investigated by other studies. However, our study has several limitations. Firstly, the number of samples was relatively small. Secondly, the data after completion of skeletal growth was not analyzed. Thus, more longitudinal studies based on larger numbers of population should be considered in the future research for evaluating the dentofacial skeletal patterns after completion of skeletal growth in orofacial region. This study would be likely to present a better understanding of splint therapy effects on the dentofacial skeletal change in juvenile patients with TMJ OA.

Ⅵ. CONCLUSIONS

In conclusion our research showed that there were no significant differences in condylar erosion severity and most of cephalometric parameters including articular angle, gonial angle, Y axis to SN, SN to mandibular plane angle, SNA, ANB, gonial upper angle, and gonial lower angle after treatment between splint therapy and control groups. However, there was a modest smaller decrease of SNB in splint therapy group than control. Our result implies that occlusal stabilization splint therapy might prevent the clockwise rotation of mandible in TMJ OA juvenile patients rather than non-splint therapy. Further longitudinal study based on a larger population should be needed to support this implication and to establish the effective treatment outcomes of juvenile TMJ OA patients.

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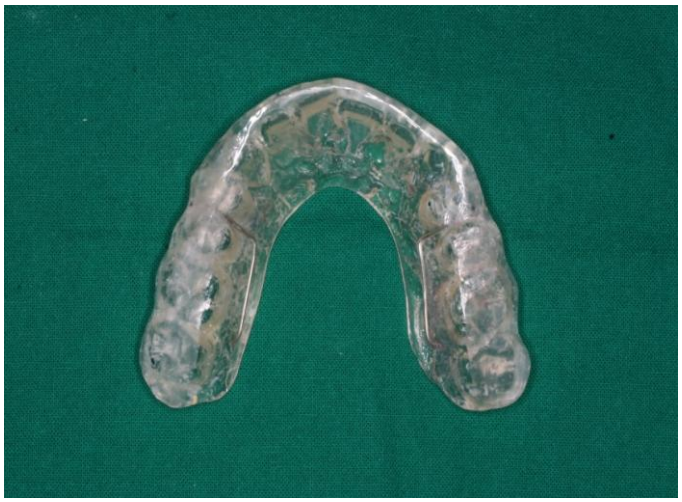
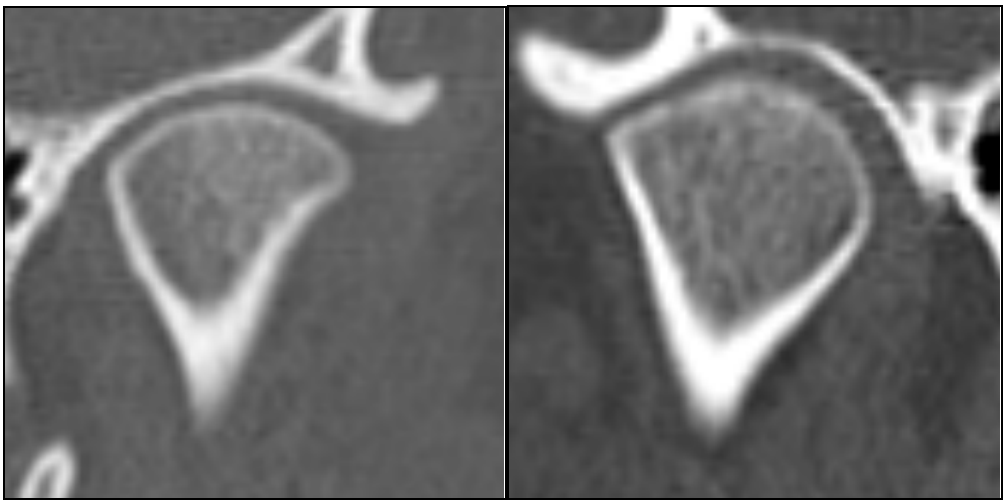
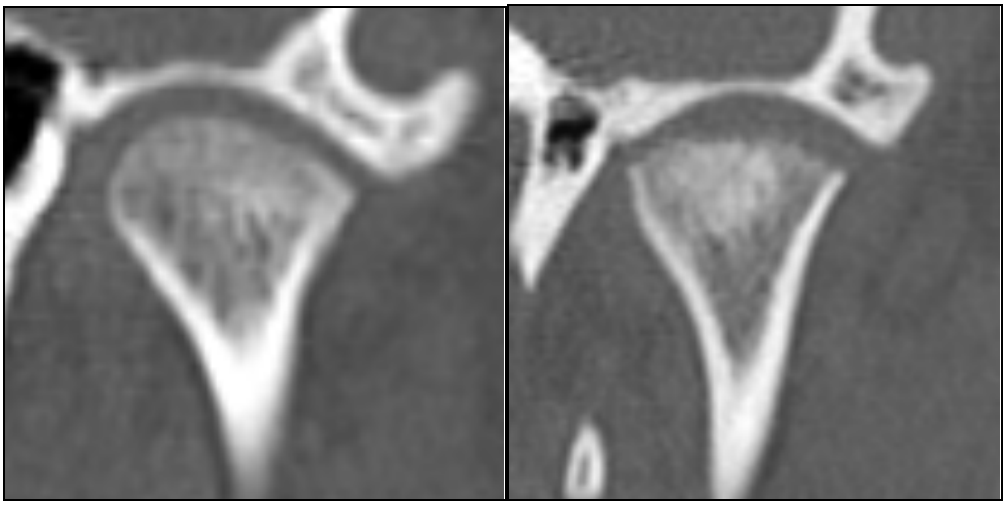


Figure 1. Stabilization splint used in our study.



(A) (B)



(C) (D)

Figure 2. Severity of condylar erosion. (A) scale 0: absence of erosion, (B) scale 1: slight erosion, when decreased density is observed only in the cortical bone, (C) scale 2: moderate erosion, when decreased density is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone, and (D) scale 3: extensive erosion, when decreased density is observed in the cortical bone and extends below the upper layers of the adjacent subcortical bone.

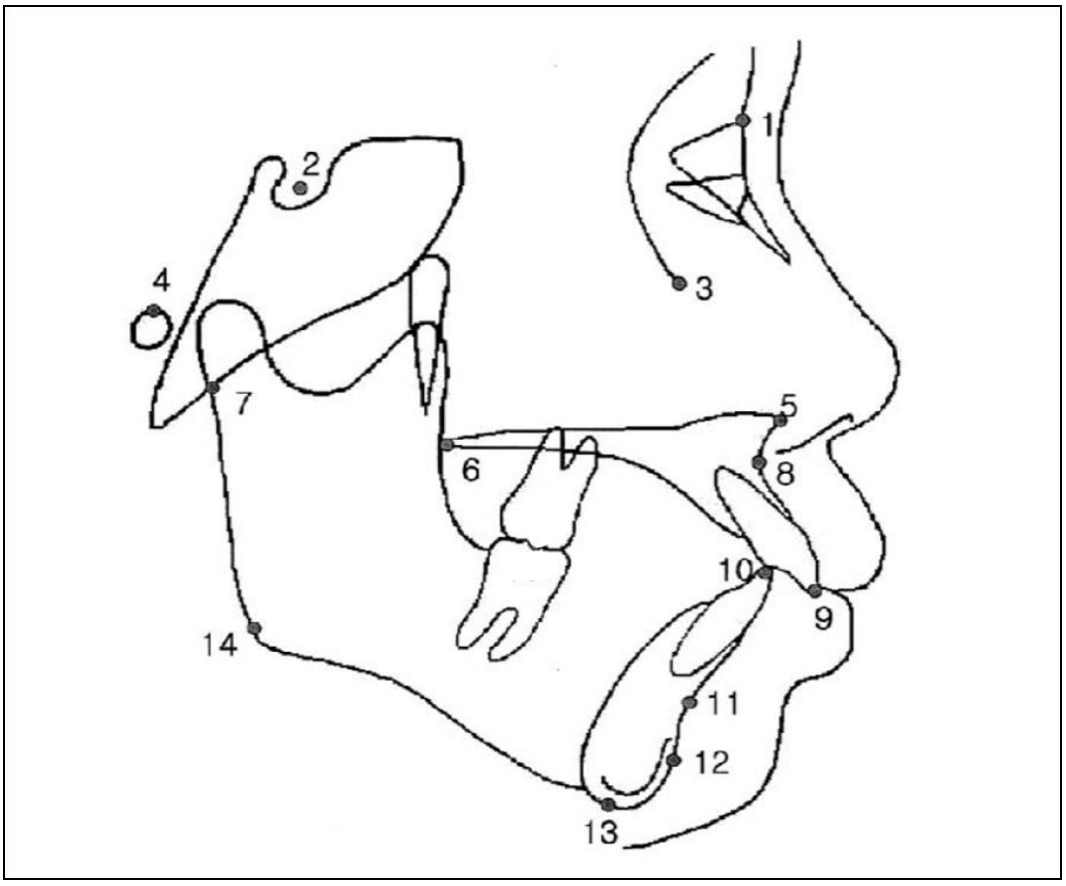


Figure 3. The landmarks used in this study. (1) nasion, (2) sella, (3) orbitalle, (4) porion, (5) anterior nasal spine, (6) posterior nasal spine, (7) articulare, (8) point A, (9) incisal end of upper incisor, (10) incisal end of lower incisor, (11) point B, (12) pogonion, (13) menton, and (14) gonion.

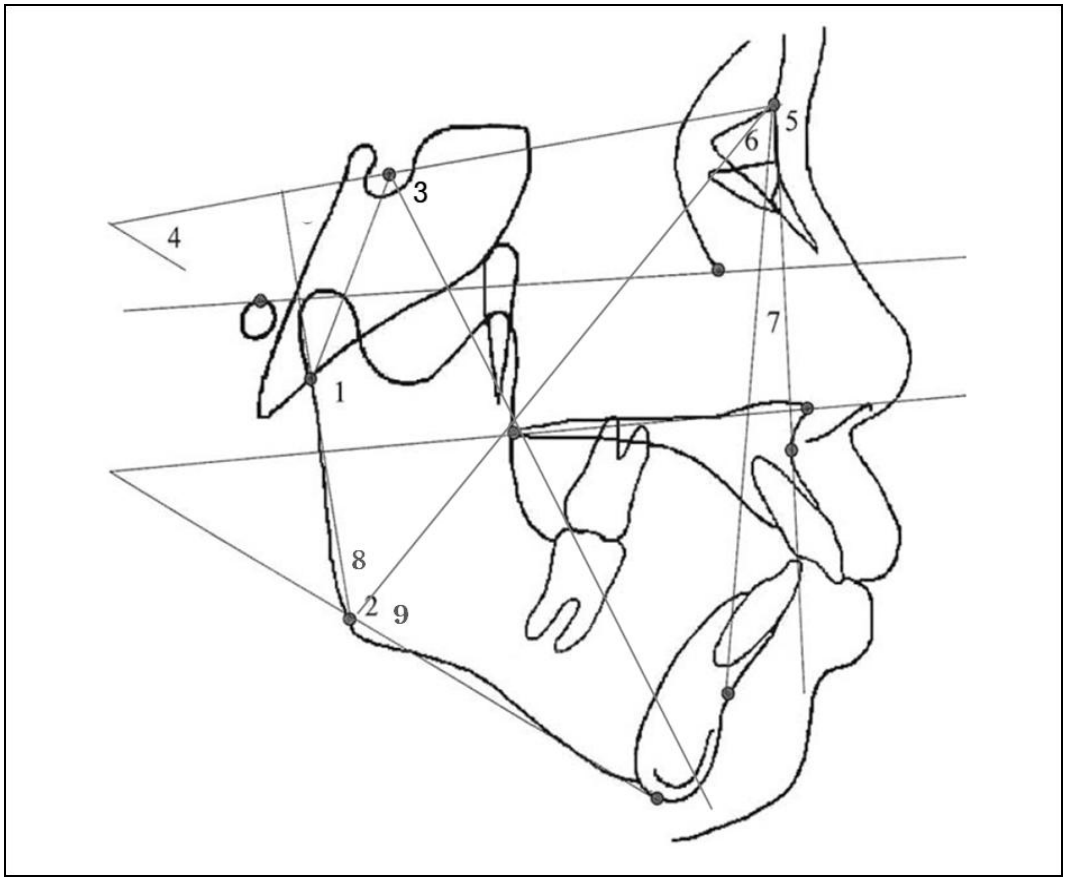
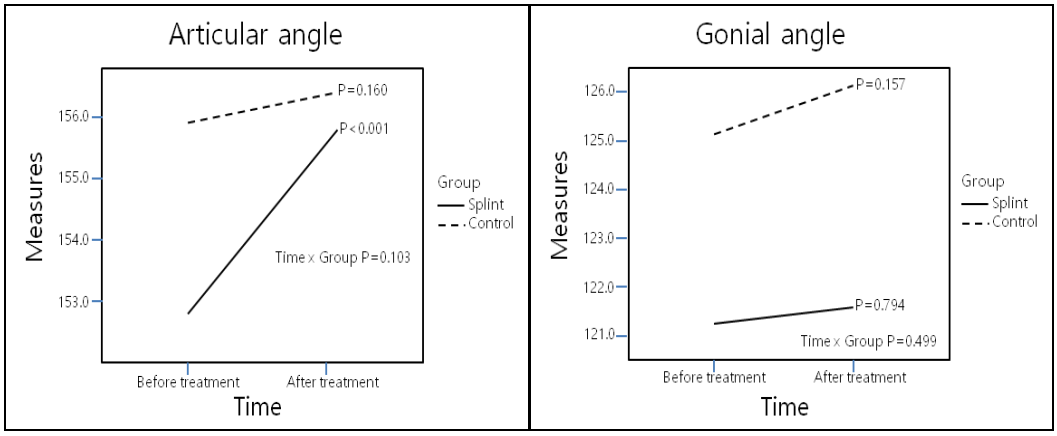


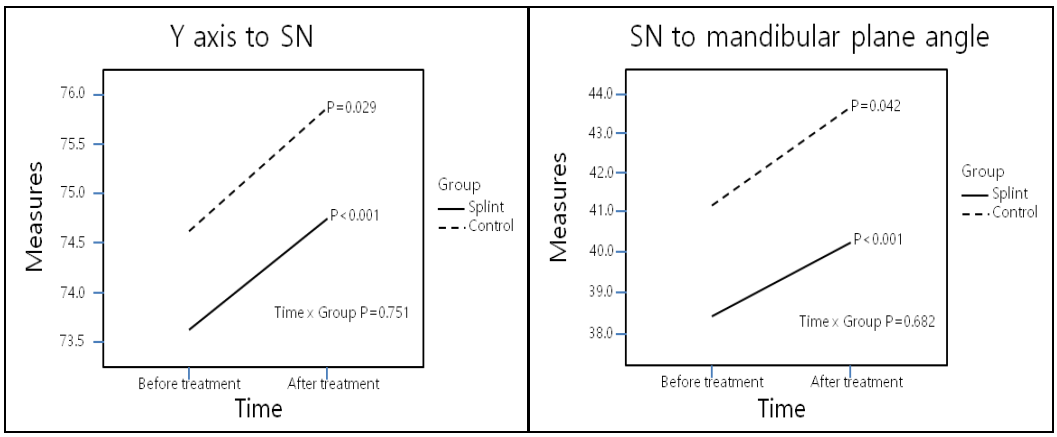
Figure 4. The angular measurements used in this study. (1) articular angle (S-Ar-Go), (2) gonial angle (Ar-Go-Me), (3) Y axis to SN, (4) SN to mandibular plane angle (Go-Me), (5) SNA, (6) SNB, (7) ANB (8) gonial upper angle (Ar-Go-N), and (9) gonial lower angle (N-Go-Me).


Articular angle
Measures
156.0
155.0
154.0
153.0
P=0.160
P<0.001
Time x Group P=0.103
Group
Splint
Control
Before treatment
After treatment
Time

(A)

Gonial angle
Measures
126.0
125.0
124.0
123.0
122.0
121.0
P=0.157
P=0.794
Time x Group P=0.499
Group
Splint
Control
Before treatment
After treatment
Time

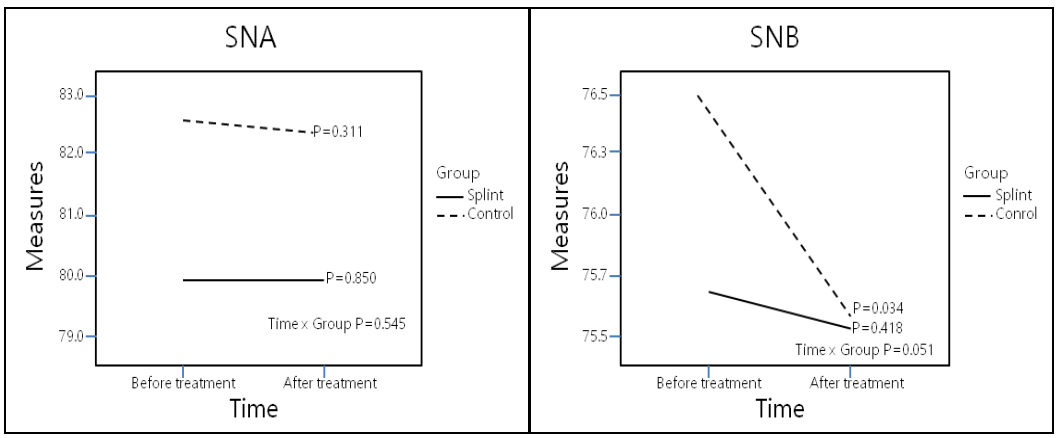
(B)


Y axis to SN
Measures
76.0
75.5
75.0
74.5
74.0
73.5
P=0.029
P<0.001
Time x Group P=0.751
Group
Splint
Control
Before treatment
After treatment
Time

(C)

SN to mandibular plane angle
Measures
44.0
43.0
42.0
41.0
40.0
39.0
38.0
P=0.042
P<0.001
Time x Group P=0.682
Group
Splint
Control
Before treatment
After treatment
Time

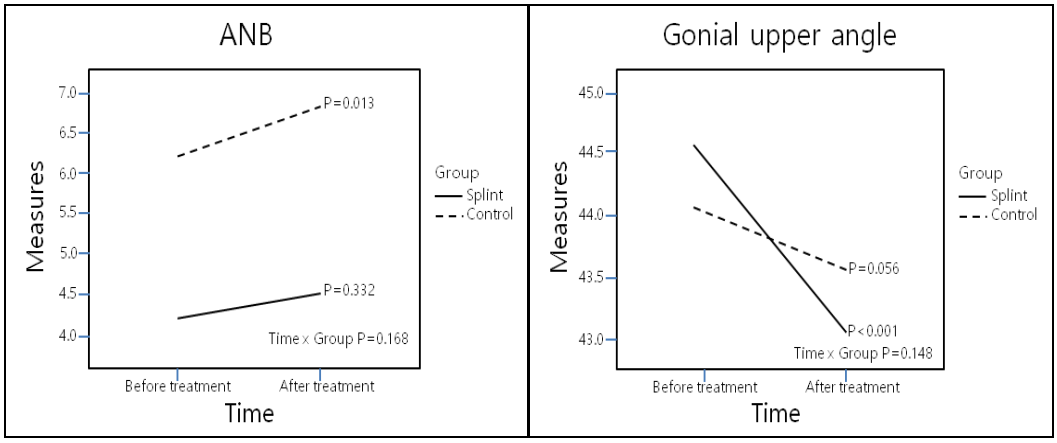
(D)


SNA
Measures
83.0
82.0
81.0
80.0
79.0
P=0.311
P=0.850
Time x Group P=0.545
Group
Splint
Control
Before treatment
After treatment
Time

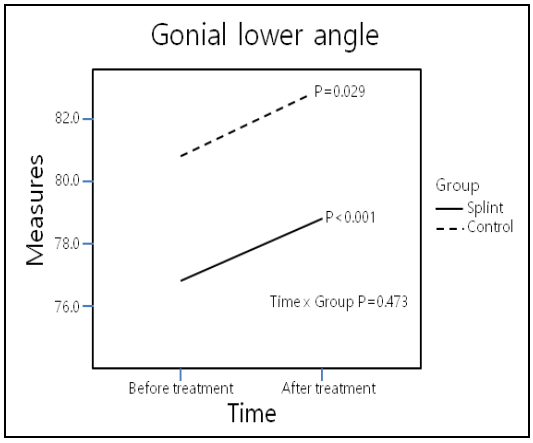
(E)

SNB
Measures
76.5
76.3
76.0
75.7
75.5
P=0.034
P=0.418
Time x Group P=0.051
Group
Splint
Conrol
Before treatment
After treatment
Time

(F)

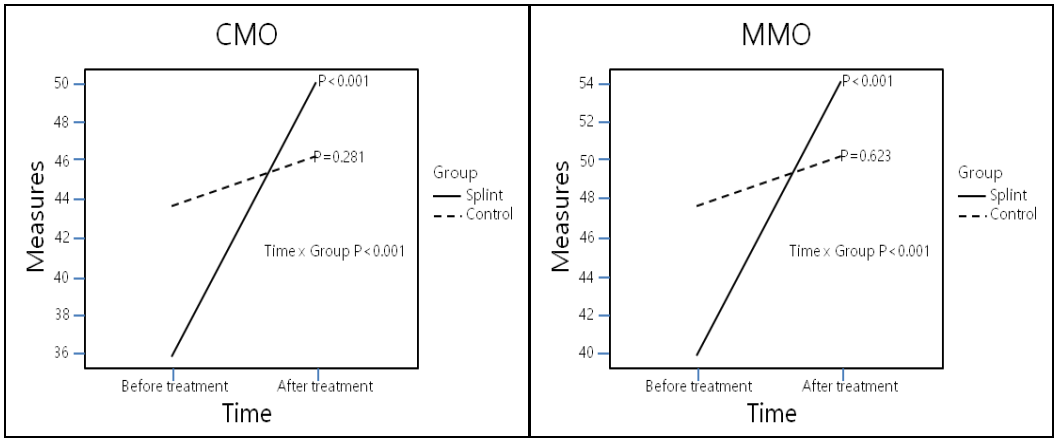


(G) (H)



(I)

Figure 5. Comparisons of changes in cephalometric parameters after treatment between splint therapy and control groups. (A) articular angle, (B) gonial angle, (C) Y axis to SN, (D) SN to mandibular plane angle, (E) SNA, (F) SNB, (G) ANB (H) gonial upper angle, and (I) gonial lower angle.



(A) (B)

Figure 6. Comparisons of changes in mandibular range of motions after treatment between splint therapy and control groups. (A) CMO, (B) MMO.

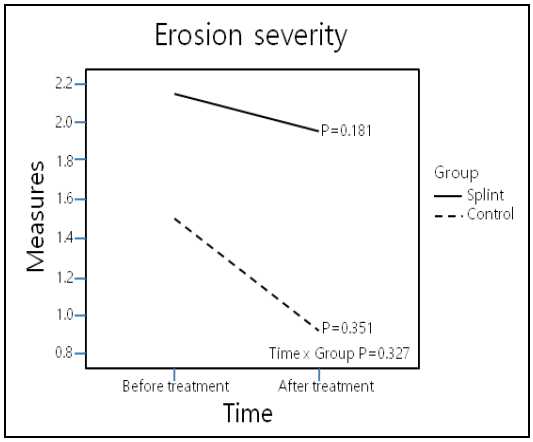
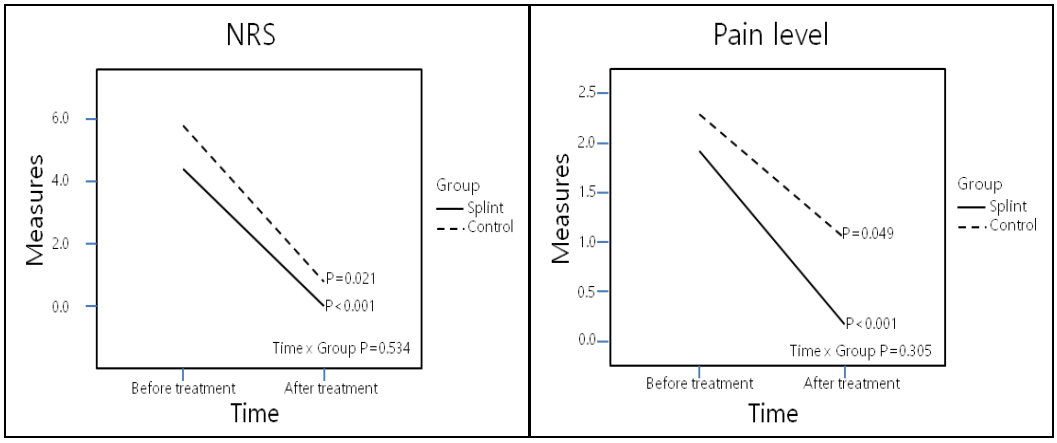


Figure 7. Comparisons of changes in condylar erosion severity after treatment between splint therapy and control groups.



(A) (B)

Figure 8. Comparisons of changes in pain intensities after treatment between splint therapy and control groups. (A) NRS : numeric rating scale, (B) pain level.

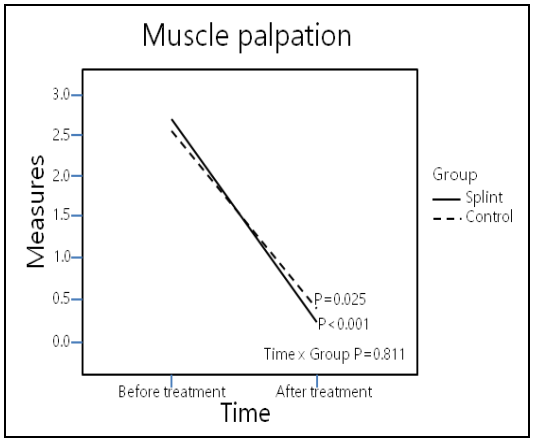


Figure 9. Comparison of changes of number of positive palpation in masseter and temporal muscles after treatment between splint therapy and control groups.

Table 1. Evaluation of pain intensity.

Pain level	NRS	Expression
0	0	None
1	1,2	Slight painful
2	3,4,5	Painful
3	6,7	Severe painful
4	8,9,10	Disable

NRS : numeric rating scale.

Table 2. Demographic data.

Variables	Splint therapy	Control	P-value
Gender (% of female)	82.1	83.3	0.922[a]
Age (years)	14.8 ± 1.8	12.7 ±2.1	0.001[b]
Cephalometric duration (months)	19.9 ± 9.7	20.2 ±11.4	0.932[b]

[a] P-values were obtained from Chi-square test.

[b] P-values were obtained from independent T-test.

Table 3. Comparison of clinical symptoms and condylar erosion severity between splint therapy and control groups.

Variables	Splint therapy	Control	P-value[a]
Onset duration(months)	14.6±16.4	27.0±5.6	<0.001
CMO	35.7 ± 8.9	43.8 ± 7.6	0.004
MMO	41.6 ± 7.4	46.8 ± 5.8	0.024
Erosion severity	2.1±1.0	1.9±1.3	0.649
NRS	4.3 ± 2.3	3.7 ± 2.7	0.544
Pain level	2.0 ± 0.9	2.3 ± 0.9	<0.001
Pain on MO	0.4 ± 0.5	0.3 ± 0.5	0.198

[a] P-values were obtained from independent T-test.

CMO: comfortable mouth opening, MMO: maximum mouth opening, MO: mouth opening.

Table 4. Changes in clinical symptoms and condylar erosion severity after treatment in each group.

Variables	Group	Before	After	P-value[a]	Time x Group[b]
CMO	Splint	35.7 ± 8.9	49.4 ± 7.5	<0.001	<0.001
	Control	43.8 ± 7.6	46.1 ± 8.4	0.281	
MMO	Splint	41.6 ± 7.4	50.3 ± 6.9	<0.001	<0.001
	Control	46.8 ± 5.8	46.1 ± 8.4	0.623	
Erosion severity	Splint	2.0 ± 1.0	1.8 ± 1.0	0.181	0.327
	Control	1.8 ± 1.3	1.4 ± 1.3	0.351	
NRS	Splint	4.3 ± 2.4	0.0 ± 0.0	<0.001	0.534
	Control	5.0 ± 2.3	0.8 ± 1.5	0.021	
Pain level	Splint	2.0 ± 0.8	0.1 ± 0.4	<0.001	0.305
	Control	2.3 ± 0.9	1.0 ± 0.9	0.049	
Muscle palpation (numbers)	Splint	2.7 ± 2.1	0.3 ± 0.9	<0.001	0.811
	Control	2.5 ± 2.7	0.3 ± 0.8	0.025	

[a] P-values were obtained from paired T-test.

[b] P-values were obtained from multivariate repeated-measures ANCOVA.

Table 5. Cephalometric measurements before and after treatment in each group.

Variables	Group	Before	After	P-value[a]	Time x Group[b]
Articular angle	Splint therapy	153.3 ± 7.4	155.6 ± 6.7	<0.001	0.103
	Control	154.0 ± 10.0	156.1 ± 9.9	0.160	
Gonial angle	Splint therapy	120.9 ± 6.3	121.0 ± 5.1	0.794	0.499
	Control	126.6 ± 8.9	127.6 ± 11.0	0.157	
Gonial upper angle	Splint therapy	44.3 ± 3.6	43.0 ± 3.3	<0.001	0.148
	Control	45.3 ± 6.5	43.9 ± 5.8	0.056	
Gonial lower angle	Splint therapy	76.7 ± 4.8	78.0 ± 4.4	<0.001	0.473
	Control	81.3 ± 4.7	83.7 ± 7.2	0.029	
Y axis to SN	Splint therapy	73.5 ± 3.0	74.4 ± 3.3	<0.001	0.751
	Control	75.3 ± 4.1	77.3 ± 5.3	0.029	
SN to mandibular plane angle	Splint therapy	38.2 ± 5.3	39.6 ± 5.6	<0.001	0.682
	Control	42.3 ± 5.6	45.6 ± 8.7	0.042	
SNA	Splint therapy	80.0 ± 3.5	80.0 ± 3.1	0.850	0.545
	Control	81.8 ± 2.7	81.4 ± 3.4	0.311	
SNB	Splint therapy	75.8 ± 3.2	75.7 ± 3.4	0.418	0.051
	Control	75.6 ± 3.0	74.1 ± 4.2	0.034	
ANB	Splint therapy	4.1 ± 2.7	4.3 ± 2.5	0.332	0.168
	Control	6.2 ± 1.8	7.2 ± 1.5	0.013	

[a] P-values were obtained from paired T-test.

[b] P-values were obtained from multivariate repeated-measures ANCOVA.

국문초록

사춘기 턱관절 골관절염 환자에서 교합안정장치 치료가 안모의 골격변화에 미치는 영향에 관한 연구

서울대학교 대학원 치의과학과 구강내과진단학 전공
(지도교수 **정 진 우**)

김 형 준

본 연구의 목적은 턱관절의 골관절염에 이환된 사춘기 환자의 교합안정장치의 치료 효과 및 안모의 골격 변화에 미치는 영향을 평가하는데 있다.

서울대학교치과병원 구강내과에 턱관절장애 증상을 주소로 내원하여 컴퓨터단층촬영 영상에서 턱관절 부위 골관절염으로 진단되어진 16세 이하 환자들 중 교합안정장치 치료를 받은 28명의 교합안정장치 치료군과 교합안정장치 외의 치료를 받은 6명의 대조군을 대상으로 치료 전후의 임상 증상, 하악과두의 피질골 침식 정도, 측모두부방사선 사진상 계측치를 비교 분석하였다. 주관적 통증 정도, 최대 개구량, 무통성 개구량, 근육 촉진 시 통증 근육의 수 등을 평가하였으며, 컴퓨터단층촬영 영상에서 하악과두의 피질골 침식 정도, 측모두부방사선 사진상 articular angle, gonial angle, Y axis to SN, SN to mandibular plane angle, SNA, SNB, ANB, gonial upper angle, gonial lower angle 등의 계측치를 측정하였다. 전후 계측치 평가의 평균기간은 교합안정장치 치료군은 19.9±9.7개월, 대조군은 20.2±11.4개월이었다.

교합안정장치 치료군은 대조군에 비하여 치료 후 최대개구량, 무통성

개구량의 개선이 유의하게 많게 나타났다. 하악과두의 피질골 침식 정도는 두 군 모두에서 유의하게 감소하지 않았다. 통증의 강도는 두군 모두 치료 후 유의하게 감소하였으나 두군 간에 유의한 차이를 보이지는 않았다. 근육 촉진 시 통증 근육의 수에서도 두 군 모두 치료 후 유의하게 감소하였으나 두군 간에 유의한 차이를 보이지는 않았다.

치료 전후 측모두부방사선사진 계측치 변화에서 교합안정장치 치료군에서는 articular angle, Y-axis to SN, SN to mandibular plane angle, gonial upper angle, gonial lower angle 수치가 치료 후 유의한 변화를 보였으며, 대조군에서는 Y-axis to SN, SN to mandibular plane angle, SNB, ANB, gonial lower angle 수치가 치료 후 유의한 변화를 보였다. 두군 모두에서 하악이 시계방향으로 회전되었으나 교합안정장치 치료군은 대조군에 비하여 SNB의 감소가 적게 나타났다.

주제어 : 턱관절, 골 관절염, 교합안정장치, 사춘기, 측모두부방사선사진

학 번 : 2013-30634