Nocturnal Bruxism and Botulinum Toxin Effect on the Subjects with Masseteric Hypertrophy

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This study aimed to evaluate a relation of bruxism with clinical effects of botulinum toxin type A(BTX-A) injection. 5 bruxers and 5 nonbruxers with bilateral masseter hypertrophy were participated in this study. After injecting 25 unit of BTX-A(Allergen Inc, Botox®) into each masseter muscle, the thickness of masseter(Mm) and anterior temporalis(Ta) muscles was measured by ultrasonography and the maximum bite force was evaluated during a 9-month period. Self-estimation on the recovery of occlusal force during mastication was done as well.

Regardless of presence of bruxsim, all subjects showed significantly reduced Ms thickness($p<0.001$) and maximum bite force at 1st molars($p=0.027$) with their peak at 3 months after injection, which then started to return. No significant difference was observed in Ta thickness and the bite force at the central incisors. While self-estimated occlusal force was the least at 2 weeks after injection and then rapidly returned to the baseline level with full recovery at the time of 6 to 9 months after injection, the maximum bite force measured by bite force recorder did not recover the original value, particularly in the nonbruxer group.

It is assumed that nocturnal bruxism can influence recovery of atrophic masseter and decreased occlusal force due to BTX-A injection. These findings suggest a need of occlusal appliance to control bruxism or clenching habit for longer clinical effect of BTX-A injection.

Key words : Bruxism, Masseteric hypertrophy, Botulinum toxin, Ultrasonography, Occlusal (bite) force

I. INTRODUCTION

Masseteric hypertrophy, first described by Legg in 1880,[1)] is defined as a painless swelling of the masseter muscles and can be unilateral or bilateral with having its highest incidence in the second and third decades of life and no sex predilection.[2)] Although the etiology still remains unclear, it is thought to be commonly associated with parafunctional habits such as bruxism, possibly leading to 'work hypertrophy' of the masseter.[3,4)]

Although it is known that this benign condition is rarely painful and does not compromise function, esthetic disfigurement requires its treatment to decrease the muscle volume. There has been tried several treatments which were largely categorized into conservative treatment including systemic medication of muscle relaxants and surgical treatment such as partial resection of the masseter

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muscles through skin incision or intraoral route.[4-7] Ostectomy of bony prominence has been also performed frequently with or without the muscle resection.[2,5] The resection of the masseter muscle is likely to cause a variety of undesirable side effects such as bleeding, hematoma, facial nerve injury, and asymmetric resection, although the operation itself is relatively simple.[2,3,5] In addition, difficulty to judge the correct amount of muscle to be resected is pointed out.[2]

As it has been reported that the masseter muscle can be reduced using botulinum toxin type A(BTX-A), BTX-A injection has been a popular, successful alternative for treatment of masseteric hypertrophy because of its minimal invasiveness and no worrisome side-effect.[2,8,9] BTX-A is well-known potent bacterial substance naturally produced by *Clostrium botulinum* and binds to the presynaptic cholinergic nerve terminals and inhibiting the release of acetylcholine, causing paralysis and subsequent functional denervated muscle atrophy. Although some studies[3,9] showed that the clinical effect of BTX-A injection lasted until a year or even 25 months after injection, its effect is generally believed to be temporary because new nerve terminal axon sprouts, restoring neuromuscular transmission over 2 to 4 month.[10] Due to its pharmacologic mechanism, seeking a re-injection of BTX-A is not uncommon. Therefore, every endeavor should be made in order to maintain the effect as long as possible and, therefore, to lessen repeated injection which may result in tolerance with the development of anti-toxin antibodies.[11]

This study aimed to investigate whether or not the presence of bruxism influences the treatment effect of BTX-A injection in the subjects with the masseteric muscle hypertrophy.

Ⅱ. MATERIALS AND METHODS

1. Subjects

10 healthy volunteers with bilateral masseter hypertrophy were selected for this study. 5 of them, proven to have nocturnal bruxism through self-reported questionnaires and clinical examination including assessment of occlusal wear, were categorized as the bruxer group(aged 26.4±2.6 years) and the other 5 subjects without bruxism as the nonbruxer group(aged 27.0±2.2 years). Questionnaires to identify bruxers consisted of 4 questions about self-awareness of bruxism during sleep, report from bed partner, headache and/or stiffened jaws on awakening. None of them had serious medical problem including pregnancy and history of drug allergy and dental problems (i.e., missing tooth except 3rd molar). Prior to the study, informed consent was obtained from all the subjects.

2. Injection of botulinum toxin type A

BTX-A used was Botox® (Allergan Inc.), which contained 100 unit BTX-A powder per one vial. BTX-A was diluted with saline without conservative to 100 unit/2ml and 25 unit was injected into the lower posterior portion of each masseter muscle in order to avoid potential paralysis of the risorius and zygomaticus muscle and injury to the parotid duct. A 1ml syringe with a 26-gauge needle was employed for the injection. The effect of BTX-A injection was evaluated by two parameters of muscle thickness and occlusal force. Follow-ups for 9 months included taking photographs of all the subjects to identify change of facial appearance as well.

3. Measurement of muscle thickness using ultrasonography

The changes in the muscle thickness of superficial masseter(Ms) and anterior temporalis (Ta) due to BTX-A injection was determined by ultrasonography. Ultrasonographic examinations were performed with a linear(B-scan) 7.5MHz small-part transducer(SA 6000II, MEDISON Co., Seoul, Korea) to register the LCSDs(local cross sectional dimensions) of each muscle. Each subject

was asked to sit in an upright position with the head in a natural position.

The origin, insertion and anterior border of Ms were identified by manual palpation while each subject was instructed to relax and clench alternatively. Scan of the muscle was made bilaterally on the midst level of the 4 equal parts determined by dividing the distance between zygomatic arch and mandibular angle. Ta was also identified in the same way and then the transducer was placed perpendicular to posterior border of zygomatic process of frontal bone.

The transducer was held against the skin with a light pressure and oriented perpendicular to direction of the muscle fibers and underlying bone. Scans were made under relaxed(by asking the subject to maintain slight interocclusal contact) and maximum clenching conditions before and 2 weeks, 4 weeks, 8 weeks, 3 months, 6 months and 9 months after BTX-A injections.

The muscle thickness was measured directly on the screen. Ms thickness was determined as the mean value of 3 measurements with the distance of 5 mm from the anterior border of mandibular ramus and Ta thickness as the mean value of the 3 measurements with the distance of 5 mm anteroposterior to its thickest part(Fig. 1). No information about the groups was given to the examiner who took and analyzed the ultrasonographic images through the all experimental period.

4. Measurement of occlusal force

To investigate the changes in occlusal force of the subjects, the maximum bite force at each follow-up was measured at anterior and posterior teeth,

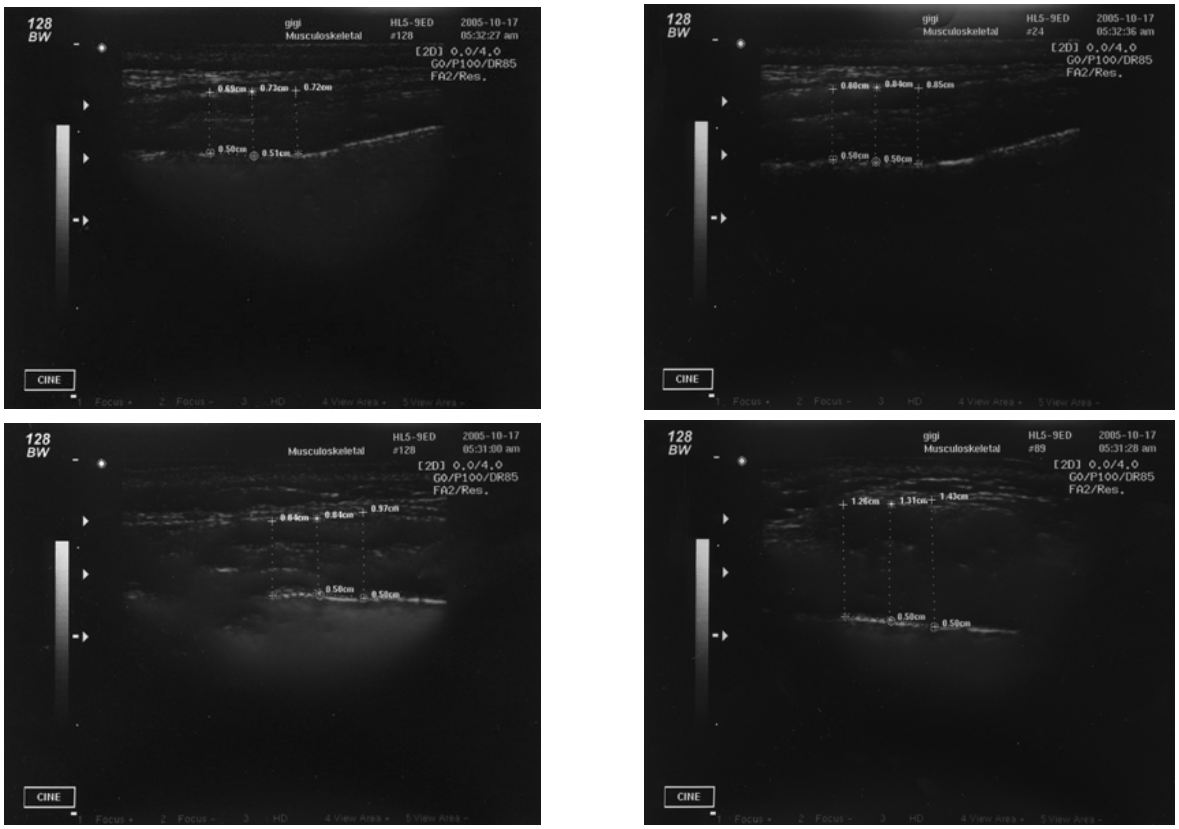

Fig. 1. Ultrasonographic images.(*upper left,* anterior temporalis (Ta) in a relaxed condition; *upper right,* Ta in a clenching condition; *lower left* masseter (Ms) at relax; and *lower right* Ms at clenching)

Table 1. Baseline data of the subjects in this study.

Variables		Group		*Unpaired t-test*
		Bruxers	Non-bruxers	
Age (years)		26.4±2.4	27.0±2.2	
Gender (M:F)		1:4	1:4	
Ultrasonographic muscle thickness (cm)	*Ta (at rest)*	0.686±0.161	0.556±0.088	*p=0.008*
	Ta (at clenching)	0.765±0.201	0.588±0.117	*p=0.003*
	Ms (at rest)	1.157±0.195	0.944±0.115	*p=0.034*
	Ms (at clenching)	1.457±0.256	1.244±0.137	*p=0.169*
Maximum bite force (kg force)	*anterior*	83.84±4.50	81.30±3.56	*p=0.624*
	posterior	100.90±8.54	104.58±6.08	*p=0.253*

Ta: anterior temporalis muscle, Ms : Masseter muscle.

separately by the bite force recorder. After placing the recorder between antagonizing 1st molars on the right side (posterior bite force) and being asked to bite lightly for several times in order to be familiar with the recorder, the subject was instructed to clench and increase the force successively to the maximal strength for five seconds. Rest period of 3 minutes was given to avoid muscle fatigue and then the same procedures were repeated on the left side. After an interval of 3 minutes, the bite force recorder was also placed between the central incisors(anterior bite force), followed by a short accommodation-period and forceful biting for 5 seconds. Measurements were done by a single examiner who had no information about the group which each subject belonged to.

In addition to measurement with the bite force recorder, the subjects were instructed to self-estimate the changes of bite force during mastication following BTX-A injection using percentage value. 100% was defined as baseline level before BTX-A treatment.

5. Statistical analysis

To compare baseline data between the bruxer and nonbruxer groups, unpaired t-test was used. Repeated measures two-way ANOVA was performed to investigate time-related changes of the muscle thickness and occlusal force after BTX-A injection. Significance level was determined at $p<0.05$.

Ⅲ. RESULTS

Based on the presence of nocturnal bruxism, the subjects with bilateral masseteric hypertrophy were categorized into two groups; the bruxer and nonbruxer groups and their baseline data was compared by unpaired t-test(Table 1). It is noticeable that muscle thickness of Ta and Ms measured by ultrasonography was significantly greater in the bruxer group compared with the subjects without bruxing habit. The mean thicknesses at the relaxed condition were 0.686±0.161 cm for Ta and 1.157±0.195 cm for Ms in the bruxer group and 0.556±0.088 cm for Ta and 0.944±0.115 cm for Ms in the nonbruxer group($p<0.005$). On the contrary to the difference of the muscle thickness, the maximum bite forces obtained at the anterior and posterior teeth regions did not show any significant difference between the two groups.

After BTX-A injection into the both MS, it was found that the muscle thickness of Ms exhibited significant difference not only between the bruxer

Table 2. The muscle thickness of masseter measured by ultrasonography after BTX-A injection into both masseter muscles.

Condition	Group	Baseline	2W	4W	8W	3M	6M	9M	ANOVA
Relaxed	Bruxer	1.157 ±0.195	1.001 ±0.095	1.021 ±0.205	1.078 ±0.164	0.924 ±0.134	0.946 ±0.174	1.002 ±0.186	p=0.000
	Non-bruxer	0.944 ±0.115	0.894 ±0.139	0.814 ±0.100	0.808 ±0.097	0.796 ±0.112	0.804 ±0.098	0.797 ±0.084	
	ANOVA	p=0.001							
Clenching	Bruxer	1.457 ±0.256	1.270 ±0.231	1.203 ±0.263	1.178 ±0.210	1.176 ±0.171	1.269 ±0.236	1.298 ±0.274	p=0.000
	Non-bruxer	1.244 ±0.137	1.048 ±0.171	0.970 ±0.113	1.001 ±0.148	1.030 ±0.113	1.084 ±0.127	1.111 ±0.145	
	ANOVA	p=0.000							

(unit:cm)

and nonbruxer groups but also among the time groups($p<0.001$)(Table 2). Atrophic changes of Ms in both groups started since 2 weeks after treatment, having with their peak at 3 months and then began to return. Recovery of the masseter thickness in the bruxer group was noticeable compared with the nonbruxer group, but significant difference still existed between before and 9 months after injection, which indicated that Ms thickness had not recovered their baseline values at 9 months after injection. When it comes to Ta, there was no significant difference among the time groups but significant difference still existed between the bruxer and non-bruxer groups(Table 3).

While the maximum bite force at the anterior teeth did not show any significant change among the time groups, posterior bite forces exhibited significant difference in relation with time($p= 0.027$)

Table 3. The muscle thickness of anterior temporalis measured by ultrasonography after BTX-A injection into both masseter.

Condition	Group	Baseline	2W	4W	8W	3M	6M	9M	ANOVA
Relaxed	Bruxer	0.686 ±0.161	0.762 ±0.068	0.752 ±0.071	0.703 ±0.125	0.699 ±0.086	0.653 ±0.148	0.702 ±0.109	p=0.000
	Non-bruxer	0.556 ±0.088	0.602 ±0.126	0.578 ±0.114	0.529 ±0.118	0.548 ±0.127	0.553 ±0.171	0.544 ±0.099	
	ANOVA	NS							
Clenching	Bruxer	0.765 ±0.201	0.841 ±0.094	0.820 ±0.099	0.821 ±0.163	0.838 ±0.155	0.747 ±0.166	0.772 ±0.158	p=0.000
	Non-bruxer	0.588 ±0.117	0.680 ±0.150	0.590 ±0.103	0.586 ±0.158	0.614 ±0.154	0.606 ±0.179	0.584 ±0.117	
	ANOVA	NS							

NS: not significant

(unit:cm)

Table 4. The maximum bite force after BTX-A injection into both masseter muscles.

Region	Group	Baseline	2W	4W	8W	3M	6M	9M	ANOVA
Anterior	Bruxer	83.84 ±4.50	81.02 ±4.08	80.52 ±4.29	81.18 ±5.25	82.48 ±3.83	83.04 ±6.03	81.54 ±5.75	NS
	Non-bruxer	81.30 ±3.56	77.60 ±2.67	78.64 ±4.38	80.90 ±4.57	81.98 ±3.66	81.96 ±3.20	81.36 ±2.21	
	ANOVA	NS							
Posterior	Bruxer	100.90 ±8.54	94.81 ±6.31	98.19 ±10.48	99.11 ±9.71	98.46 ±9.57	101.53 ±11.81	98.77 ±11.27	NS
	Non-bruxer	104.58 ±6.08	91.78 ±5.45	92.27 ±6.64	97.53 ±10.17	96.94 ±5.56	96.21 ±6.90	96.66 ±4.95	
	ANOVA	p=0.027							

NS: not significant (unit: kg force)

(Table 4). No significant difference was found between the bruxer and nonbruxer groups. However, comparison of relative posterior bite force (relative value of bite force(%) when baseline bite force was considered as 100%) showed significant differences between the bruxers and nonbruxers (p=0.000) and among time groups (p=0.000)(Fig 2). The maximum posterior bite forces in the nonbruxer group decreased more rapidly after BTX-A injection and their recovery was slower than the bruxer group.

While self-estimated bite force during mastication was compromised the most at the time of 2 weeks after injection and then rapidly return to the baseline

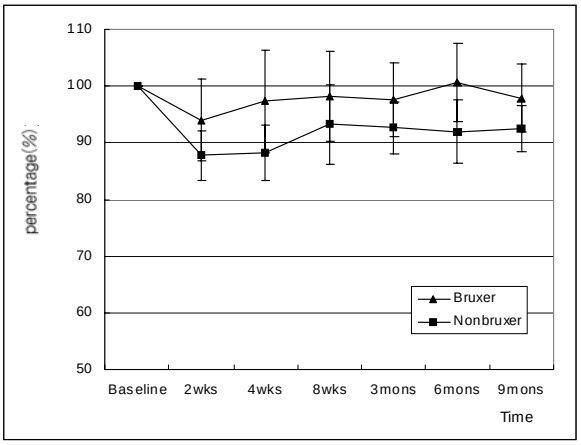

Fig. 2. Relative changes of the maximum bite force at the posterior teeth after BTX-A injection(p=0.000 between the bruxers and nonbruxers, p=0.000 among the time groups).

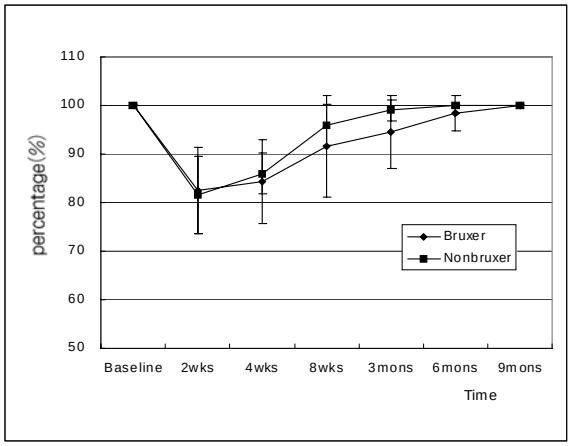

Fig. 3. Changes of self-estimated bite force during mastication related to elapsed time after BTX-A injection into both masseter muscles(p=0.235 between the bruxers and nonbruxers, p=0.000 among the time groups).

value, having with full recovery at the time of 6 to 9 months after injection(p=0.000 among time groups)(Fig 3), relative changes of the maximum bite force on the posterior teeth measured by bite force recorder demonstrates that it did not recover the original value(Fig. 2).

Ⅳ. DISCUSSION

Although masseteric hypertrophy is a clinical entity of unknown etiology, it has been attributed to unilateral chewing due to loss of teeth or pain from caries or sepsis, to congenital arterovenous fistula, temporomandibular disorders and to focal dystonia. Non-organic causes include work hypertrophy from chewing habits including gum chewing, grating of teeth, changes in proprioceptive influences in emotionally disturbed patients and cradling the jaw when reading.[7,8,12] Purposeless activity, represented by clenching and bruxism is more likely to lead to increased muscle bulk than purposeful gum chewing or mastication.[13,14]

Masticatory muscle hyperactivity or parafunction and dysfunction in the stomatognathic system cannot be verified in all instances of hypertrophy,[9] but it is difficult to deny that abnormal habit such as bruxism is commonly associated with masseteric hypertrophy. Occasionally other muscles of mastication such as temporalis may also be hypertrophied.[2] In regards with the baseline data of the subjects in this study, 5 bruxers had significantly greater muscle thickness of the Ta and Ms as compared to other 5 subjects without bruxism(Table 1).

While a lot of researchers concerned about treatment outcome, based on esthetic aspect, of BTX-A for masseteric hypertory and a few studies[15,16] reported treatment of bruxism with BTX-A, this study focused a relation of bruxism with clinical effect of BTX-A treatment for the subjects with masseter hypertrophy. The main difficulty in this study was to identify and quantify of nocturnal bruxism.

Seligman *et al.*[17] stated that the three most common methods to evaluate bruxism are self-report questionnaires, clinical oral examination and sleep laboratory assessment. Although sleep laboratory electromyography(EMG)-based assessments are thought to be reliable,[18-20] limited diagnostic utility in the clinical setting makes its clinical application hesitated.[21] In addition, clinical oral examination on bruxism primarily includes tooth wear, but it is thought that bruxsim and tooth wear are not synonymous. Because consensus on the definition of bruxsim is lacking and validated its severity scale is not available,[15] the questionnaires including self-awareness and report from bed-partner and clinical examination were employed for diagnosis of bruxism in this study.

The techniques available for measuring muscle's cross-section *in vivo* are computerized tomography (CT), magnetic resonance imaging(MRI) and ultrasonographic imaging.[22] Ultrasonography offers several advantages over MRI and CT for clinical examinations because it is a rapid, inexpensive technique and the equipment can be easily handled and transported. In addition, it has no known cumulative biological effect as compared to CT. In a study investigating muscle thickness of the temporalis and masseter, ultrasonography was found to be a highly reliable and accurate method for imaging and measuring masticatory muscle thickness.[23] Raadsheer *et al.*[22] compared masseter thickness from ultrasonoraphic images with that of MRI and concluded that ultrasonography was an accurate and reproducible method. The registration level with highest reproducibility was halfway between the origin and insertion of the masseter muscle and measurements in a contracted, clenching condition were more reproducible than those in a relaxed condition.

Choe *et al.*[24] investigated the optimal dose in the treatment of masseter hypertrophy by comparison of 10 to 30 units of BTX-A and came to the conclusion

that above 20 unit should be used. They also revealed that atrophic effect due to BTX-A was maintained for at least 9 months after injection. Another study[25] comparing 25 and 35 unit demonstrated that there was no significant difference between them. 25 unit of BTX-A per each masseter was injected in this study and atrophic appearance of the masseter still remained at the follow-ups of 9 months after treatment in both the bruxer and nonbruxer groups(p=0.001 and 0.019 for relaxed and clenching condition, respectively) (Table 2).

Recovery to baseline values of both Ms thickness and the maximum bite force at the posterior teeth region was more noticeable in the bruxer group compared with the nonbruxer group. Jang *et al.*[25] in the aforementioned study exhibited that the atrophic effect of the masseter due to BTX-A was considerable in case of combination with occlusal splint therapy, which needs to consider that 30% of the subjects had parafunctional habit such as bruxsim.

Although significant difference of Ta thickness between the subject groups existed in both relaxed and clenching conditions after injection(p=0.000 and 0.000, respectively), it is likely thought to be a reflection of difference in thickness before treatment(p=0.008 and 0.003 for relaxed and clenching condition, respectively)(Table 1 and 3). There was no significant difference in Ta thickness in the time groups, possibly indicating no compensatory mechanism due to the atrophic Ms. In a study on the change of EMG activity due to BTX-A injection during 6 months, Kim *et al.*[26] also reported that there was no change in the EMG activity of temporal muscle after injection while that of the masseter was reduced. To explain the time gaps between duration of decreased chewing ability(occasionally for 1 to 2 months) and duration of atrophic effect of the masseter(generally for 6 to 9 months) following BTX-A injection, Kim et al[5] suggested that there are compensations from other muscles including the pterygoid and temporal muscles. It is likely difficult to accept their assumption, at least in case of the temporalis.

It has been reported several complications due to BTX-A which include pain, swelling, dysphagia, decrease of bite force during chewing and mouth dryness and most of them can be considered to be temporary and minor.[5,15,25] Reduced bite force was the only side-effect reported from our subjects. Self-estimated chewing ability was compromised the most at the time of 2 weeks after injection and then rapidly returned to the baseline level with full recovery at the time of 6 to 9 months after injection. On the while, relative change of the maximum bite force on the posterior teeth measured by bite force recorder demonstrates that it did not recover the original value(p=0.010 between before and 9 months after injection). Decrease of the relative maximum bite force was more considerable in the nonbruxer group(p=0.000).

According to the results of this study, it is indicated that bruxism can influence the recovery of atrophic masseter and reduced occlusal force due to BTX-A injection, suggesting a need of occlusal appliance to control bruxism for longer clinical effect of BTX-A injection.

A further study in a larger sample is required to demonstrate more definitely the effect of parafunctional habits on treatment outcome of BTX-A injection and quantitative measures for severity of bruxism is also needed.

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국문요약

야간 이갈이와 교근비대 환자의 보툴리눔 독소 주사 효과

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본 연구는 교근비대의 중요한 기여요인으로 간주되는 이갈이의 영향을 평가하기 위해 이갈이 습관의 유무에 따른 BTX-A 주사후의 임상적 변화를 조사하였다.

실험을 위해 양측성 교근비대를 가진 지원자 중, 야간 이갈이 습관을 가지고 있는 지원자 5명과 이갈이 습관이 없는 지원자 5명을 선택하여 BTX-A(Allergen Inc., Botox®)를 25 unit씩을 양측 교근에 각각 주사하였다. BTX-A의 교근주사 후에 나타나는 변화를 평가하기 위하여 주사 전과 주사 후 2주, 4주, 8주, 3개월, 6개월 9개월에 각각 검사를 시행하여 초음파를 이용한 전측두근과 교근의 두께 측정, 전치부와 구치부의 최대교합력 측정, 교합력의 변화에 대한 주관적 평가를 비교하였다.

교근에 BTX-A를 주사한 후에 이갈이군과 비이갈이군 모두 초음파검사에서 교근의 두께가 감소되어 3개월 정도에 가장 현저한 위축 소견을 보였으며 이후 점차 회복되어가는 양상을 보였다($p<0.001$). 비이갈이군과 비교하였을 때 이갈이군에서 교근두께의 회복이 더 현저하였으나, 주사 후 9개월에도 치료전과 비교했을 때는 여전히 근위축이 관찰되었다. 구치부 최대교합력도 교근두께 변화와 유사한 양상을 보였다. 전측두근과 전치부 최대교합력은 주사 후 시간경과에 따른 변화를 보여 주지 않았다($p>0.05$). 피검자가 스스로 느끼는 교합력은 주사 2주 후에 가장 저하되었다가 점차 빠르게 회복되어 6개월에서 9개월 사이 이전의 상태로 회복한 반면 교합력측정기로 측정한 구치부 최대교합력의 상대적 변화는 최대교합력이 원래의 상태로 회복되지 못했음을 보여주었다. BTX-A 주사로 인한 상대적인 구치부 최대교합력의 저하는 비이갈이군에서 더욱 현저하게 관찰되었다

이 실험의 결과는 이갈이는 BTX-A 주사 후에 발생한 교근 위축과 교합력 감소가 원상태로 회복되는 과정에 영향을 미칠 수 있음을 보여준다. 그러므로 이갈이 등의 이상기능 습관을 가진 교근비대 환자의 BTX-A 주사 효과를 보다 오래 유지하기 위해서 주사와 함께 습관조절을 위한 교합장치의 사용을 고려할 필요가 있을 것으로 생각된다.

주제어 : 이갈이, 교근비대, 보툴리눔독소, 초음파검사, 교합력