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Surgical Intervention for Masticatory Muscle Tendon-Aponeurosis Hyperplasia Based on the Diagnosis Using the Four-Dimensional Muscle Model

Kazutoshi Nakaoka, DDS, PhD, Yoshiki Hamada, DDS, PhD,* Hayaki Nakatani, DMD,† Yuko Shigeta, DMD, PhD,‡ Shinya Hirai, DMD, PhD,‡ Tomoko Ikawa, DMD, PhD,‡ Akira Mishima, RT,§ and Takumi Ogawa, DMD, PhD‡*

Objectives: The surgical target of Masticatory muscle tendon-aponeurosis hyperplasia (MMTAH) is the masseter or temporal muscle. In our clinic, the 4-dimentional muscle model (4DMM) has been used to decide if we should approach to the masseter or temporal muscle. The aim of this study is validate the clinical usefulness of 4DMM on the basis of the surgical results.

Methods: The 4DMM was constructed from the digital data of 3D-CT and 4-dimentional mandibular movements of the patients. It made us to able to visually observe the expansion rate of masticatory muscles at maximum mouth opening comparing to their length at closed mouth position. Fifteen patients were applied the 4DMM before the surgical treatment and 2 healthy volunteers were enrolled as control group.

Results: The expansion rate of temporal muscle at the maximum mouth opening in the patient group was significantly less than that in the control group ($P < 0.05$). On the other hand, the masseter muscles of all patients were expanded as same as the control group. Therefore the main cause of limitation of mouth-opening was suggested to be a contracture of the temporal muscle. Consequently, we performed successful bilateral coronoidectomy with no surgical intervention to the masseter muscles in all patients.

Conclusion: The present 4DMM would be valuable modality to decide the target muscle of surgical treatment for patients with MMTAH. In this pathology, contracture of the temporal muscle seems to be main cause of limited mouth opening.

From the *Department of Oral and Maxillofacial Surgery, Tsurumi University School of Dental Medicine; †Department of Oral and Maxillofacial Surgery, Yokohama Rosai Hospital; ‡Department of Fixed Prosthodontics, Tsurumi University School of Dental Medicine; and §Department of Oral and Maxillofacial Radiology and Diagnosis, Tsurumi University School of Dental Medicine, Yokohama, Japan.

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Address correspondence and reprint requests to Kazutoshi Nakaoka, Department of Oral and Maxillofacial Surgery, School of Dental Medicine, Tsurumi University, 2-1-3 Tsurumi Tsurumi-ku, Yokohama 230–8501, Japan; E-mail: nakaoka-k@tsurumi-u.ac.jp

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Masticatory muscle tendon-aponeurosis hyperplasia (MMTAH) is characterized by painless and severe limited mouth opening due to contracture of the masticatory muscles with hyperplasia of tendons and aponeurosis. Many patients reveal a characteristic square mandible with hyperplasia of coronoid process and mandibular angle.[1–5] Diagnostic images usually depict no specific findings of the temporomandibular joint (TMJ). Also no interferences between the coronoid process and zygomatic bone are observed even at maximal mouth opening.

Etiology of MMTAH has not been clarified. Any conservative therapies including pharmacotherapy, physical therapy, and bite splint therapy are not effective. Only surgical intervention to the masseter muscle or temporal muscle with coronoid process contributes to the improvement of mouth opening. However, we have not known how to surely diagnose which muscle was main cause of limited mouth opening. Therefore, we clinically applied the four-dimensional (4-D) muscle model[4] to explore the real target muscle.

In this report, we described the clinical course of 15 cases underwent surgical treatment based on the diagnosis by using 4-D muscle model, and the usefulness of 4-D muscle model to decide the surgical target of MMTAH.

MATERIALS AND METHODS

Patients and Clinical Characteristics

Fifteen patients (13 women, 2 men) with MMTAH were enrolled in this study. They were referred to the Department of Oral and Maxillofacial Surgery, Tsurumi University Dental Hospital with chief complaint of a severe mandibular hypomobility with no pain. They had no significant medical histories including trauma or infection in the head and maxillofacial area. In all patients, painless mandibular hypomobility had gradually progressed for many years. Limited mouth opening was first recognized at 26.1 years old (mean; range 10–62-years old), and it had been identified by the patient's parents or home dentists. At the first visit, their mean of age was 38.8 years (range 23–68 years), and that of maximal mouth opening was 21.3 mm (range 16–27 mm). Thirteen of the 15 patients had consciousness of bruxism, such as grinding or clenching. Thirteen of the 15 patients had a characteristic facial shape of square mandible concomitant with prominent mandibular angle (Table 1). In these typical patients, panoramic radiograph showed a hyperplasia of the mandibular angle and anteroposterior enlargement of the coronoid process. Moreover, cephalometric analysis

TABLE 1. Clinical Characteristics of Patients

Patient No.	Gender	Age at the First Visit, years	Age at Which Limited Mouth Opening Was First Identified, years	Mouth Opening, mm	Consciousness of Bruxism	Square Mandible
1	M	23	13	20	Yes	Yes
2	F	24	18	24	Yes	Yes
3	F	27	12	19	Yes	Yes
4	F	39	37	20	Yes	No
5	F	35	32	19	No	Yes
6	F	43	37	24	No	Yes
7	F	41	20	23	Yes	Yes
8	F	46	16	21	Yes	Yes
9	F	68	62	20	Yes	Yes
10	F	43	10	24	Yes	Yes
11	F	46	43	16	Yes	Yes
12	F	42	40	20	Yes	No
13	M	47	20	27	Yes	Yes
14	F	35	17	22	Yes	Yes
15	F	23	15	20	Yes	Yes
mean		38.80	26.13	21.27		

showed low mandibular plane and gonial angles with increase of the ramus inclination (Fig. 1). On the magnetic resonance imaging (MRI), no pathologic findings were depicted in the TMJ. On the other hand, thick aponeurosis was depicted on the anterior surface and inside of the masseter muscles in all patients. These aponuerosis extended from one half to three-fourths of the length of masseter muscles (Fig. 2). Also, on the MRI and computed tomography (CT), a relatively hypertrophic temporal muscle tendon and aponerosis was noted in all patients (Fig. 3). Unsuccessful nonsurgical treatments including pharmacotherapy, physical therapy, and bite splint therapy were applied to all patients for more than 3 months.

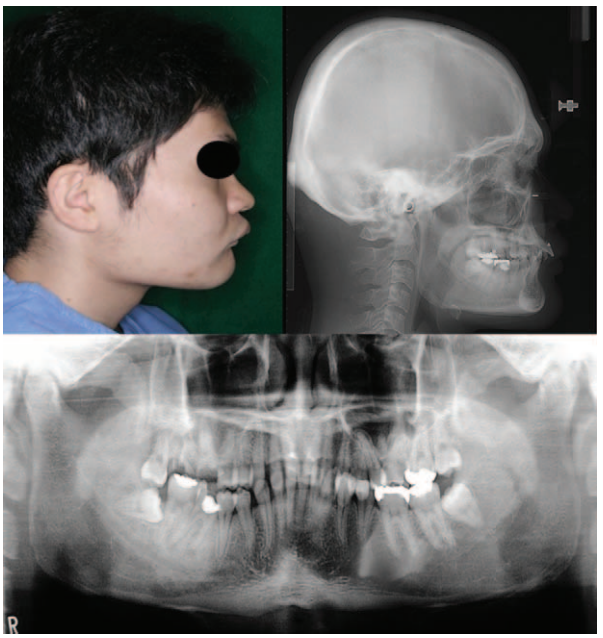

FIGURE 1. Facial profile and panoramamic radiograph of a patient (No. 1) show typical square mandible and anteroposterior enlargement of the coronoid process.

Analyses to Determine the Main Causal Muscle of Painless Limited Mouth Opening by the 4-D Muscle Model

Recently, we have been able to observe the virtual simulation of actual mandibular movements in the 4-D virtual model of the skull (4-D analysis system)[4] reconstructed by the data of mandibular movements and three-dimensional (3-D) CT. The 4-D muscle model is constructed by inputting the information of mastication muscles to the 4-D analysis system.[5] The origin and insertion of each masticatory muscle were positioned on the surface of the 3-D skull model in the 4-D analysis system, and connected together with a virtual string, respectively. In this system, the string was designed to be passively changed in accordance with mandibular movements, and we can measure the change of masticatory muscle virtual string length. Therefore, we can investigate the expanded rate of masticatory muscle at the maximal mouth opening against to the mouth closing (Fig. 4).

In this study, we could not set the origin of temporal muscle because of the lack of 3D-CT data that resulted by avoiding unnecessary radiation exposure to the brain. Therefore, we set a certain point in the 3-D space in accordance with the normal adult skull. And, we selected the bilateral masseter muscles and temporal muscles as the subjects for investigation. Moreover, the masseter muscles were classified into superficial part and deep part, and the temporal muscles were classified into anterior part, middle part, and posterior part (Fig. 5).

By using this system, we also observed the relationship of the condyle to the eminence-fossa during mandibular movements, and whether there was interference between the coronoid process and zygomatic bone at the maximal mouth opening. The expanded rate of bilateral masseter muscles (superficial part and deep part) and bilateral temporal muscles (anterior part, middle part, and posterior part) were compared between the patients group and the control group. The control group was composed of 2 healthy volunteers (a 20-year-old man and a 20-year-old woman) with no missing teeth and no clinically morbid findings. Informed consents on the study were obtained from all subjective patients and healthy volunteers.

Statistical Analysis

Mann-Whitney test was used for statistical analyses, and a probability (P) value less than 0.05 was considered to be significant. The SPSS Version 12 (SPSS, Tokyo Japan) was used for all analyses.

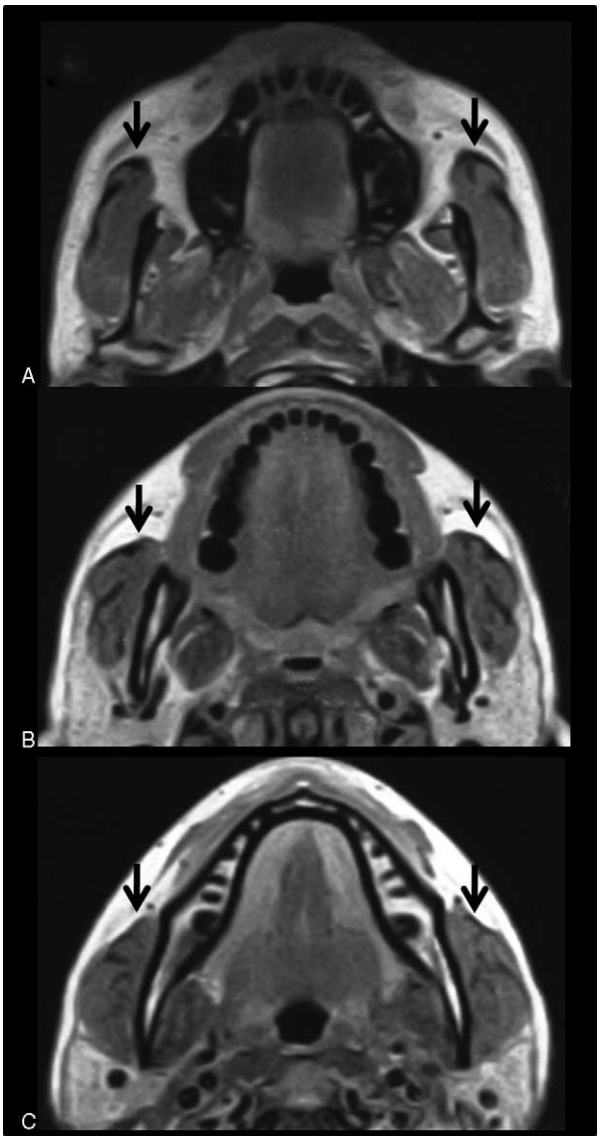

FIGURE 2. Axial MR images show elongated and thickened aponeurosis on the anterior surface (arrows) and inside of both masseter muscles. (A) A section at one-third from the inferior border of zygomatic arch. (B) A section about one half from the lower border of zygomatic arch. (C) A section about three-fourths from the lower border of zygomatic arch.

Ethical Approval

Tsurumi University Ethics Committee, approval number: 708

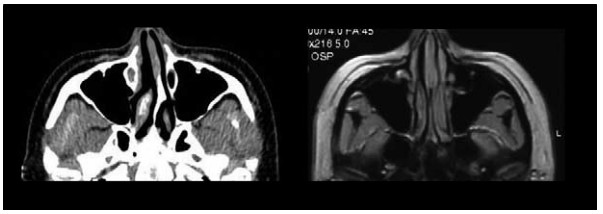

FIGURE 3. Axial MR image and CT show hypertrophic temporal muscle tendon and aponeurosis. These images are same slice around the top of coronoid process.

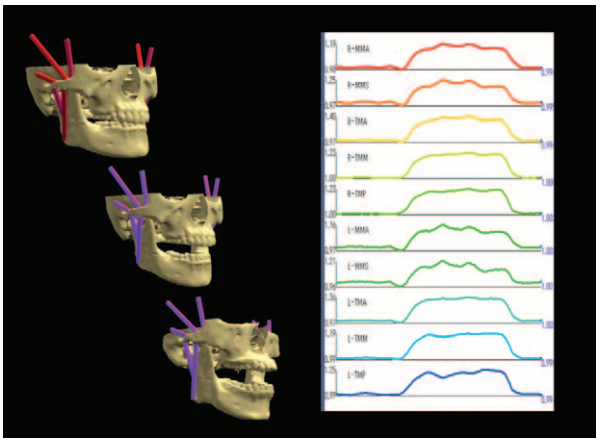

FIGURE 4. Four-dimensional muscle model. The strings are designed to be passively changed in accordance with mandibular movements, and we can measure the change of masticatory muscle length and investigate the expanded rate of masticatory muscle at the maximum mouth opening against at the mouth closing.

RESULTS

Analytical Results by 4-D Muscle Model

The 4-D muscle model clarified that there was no contact of the enlarged coronoid process to the zygomatic bone, and the condyle was not beyond the articular eminence at the maximal mouth opening in all patients.

The expanded rates of temporal muscle at the maximal mouth opening in the patients group were significantly less than those in the control group (anterior part: $P = 0.003$, middle part: $P = 0.002$, and posterior part: $P = 0.001$) (Table 2). On the other hand, the expanded rates of masseter muscle in the patients were similar or larger than those of the control group (Table 2). Consequently, in all patients, the main cause of limited mouth opening was suggested to be a contracture of the temporal muscles.

Surgical Intervention and Its Clinical Results

On the basis of the mentioned analytical results by 4-D muscle model, we performed a successful bilateral coronoidectomy combined

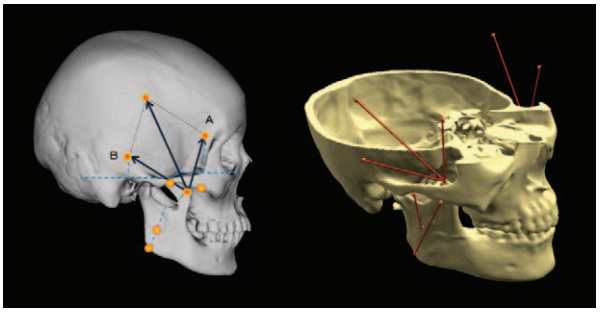

FIGURE 5. The origin and insertion of each masticatory muscle are positioned on the surface of the three-dimensional bone model, and connected together with a string respectively. We set a certain point in the three-dimensional space in accordance with the normal adult skull. Temporal musucle: anterior part (origin: point A is on the temporal fossa of 40 mm upper from superior margin of the temporal process base of zygomatic bone. Insertion: coronoid process). Middle part (origin: the summation of the vector of A and B. Insertion: voronoid process). Posterior part (origin: point B is on the temporal fossa of 20 mm upper from superior margin of the external acoustic meatus. Insertion: coronoid process). Masseter muscle: superficial part (origin: the middle point of the zygomaticomaxillary suture and the zygomaticotemporal suture. Insertion: mandibular angle). Deep part (origin: the middle point of the zygomaticotemporal suture and articular tubercle. Insertion: the middle point of the mandibular angle and mandibular notch).

TABLE 2. Results of the Four-Dimensional Muscle Model Analysis

			Expanded Rate of Muscle at the Maximal Mouth Opening				
			Musseter Muscle		Temporal Muscle		
Patient	Side	Maximal Mouth Opening, mm	Superficial Part	Deep Part	Anterior Part	Middle Part	Posterior Part
1	Rt	20	1.16	1.15	1.25	1.10	1.08
	Lt		1.12	1.09	1.25	1.16	1.13
2	Rt	24	1.15	1.13	1.29	1.19	1.20
	Lt		1.10	1.12	1.31	1.19	1.22
3	Rt	19	1.24	1.71	1.39	1.23	1.21
	Lt		1.16	1.01	1.32	1.20	1.20
4	Rt	20	1.21	1.25	1.22	1.12	1.11
	Lt		1.26	1.37	1.33	1.20	1.25
5	Rt	19	1.04	1.01	1.05	1.06	1.07
	Lt		1.14	1.17	1.15	1.07	1.04
6	Rt	24	1.12	1.29	1.29	1.19	1.23
	Lt		1.18	1.32	1.32	1.17	1.16
7	Rt	23	1.20	1.23	1.25	1.13	1.12
	Lt		1.21	1.26	1.27	1.15	1.14
8	Rt	21	1.24	1.33	1.31	1.18	1.17
	Lt		1.25	1.32	1.33	1.18	1.16
9	Rt	20	1.05	1.11	1.17	1.13	1.20
	Lt		1.04	1.08	1.14	1.12	1.21
10	Rt	24	1.06	1.15	1.29	1.22	1.30
	Lt		1.21	1.32	1.38	1.20	1.28
11	Rt	16	1.17	1.24	1.23	1.13	1.15
	Lt		1.24	1.29	1.17	1.07	1.01
12	Rt	20	1.16	1.18	1.21	1.11	1.11
	Lt		1.29	1.32	1.28	1.12	1.10
13	Rt	27	1.11	1.19	1.31	1.19	1.24
	Lt		1.08	1.10	1.24	1.16	1.19
14	Rt	22	1.13	1.13	1.18	1.11	1.11
	Lt		1.20	1.26	1.28	1.14	1.17
15	Rt	20	1.12	1.13	1.20	1.15	1.22
	Lt		1.09	1.11	1.23	1.18	1.27
mean		21.27	1.16	1.22	1.26	1.16	1.17
Control							
1	Rt	43	1.12	1.14	1.41	1.23	1.32
	Lt		1.14	1.13	1.36	1.21	1.31
2	Rt	42	1.13	1.21	1.40	1.26	1.37
	Lt		1.15	1.25	1.36	1.24	1.32
mean		42.5	1.14	1.18	1.38	1.24	1.33
*P-value (patients versus control group)			0.486	0.789	0.003	0.002	0.001

*Mann-Whitney test.

with surgical stripping of masseter muscle for all patients. Intraoperatively, when bilateral surgical masseter muscle stripping was performed as a preparation for the coronoidectomy, limited mouth opening hardly improved. However, immediately after the bilateral coronoidectomy, their mean maximal mouth opening increased up to 57.4 (range 48–60 mm) with a jaw-opening device (Table 3).

A serial doctor-oriented physical therapy was started 3 days after the surgery. The passive mouth opening using a jaw-opening device with 5-minute interval was performed 4 times per day under the hospitalization. After discharge from the hospital, the patients carried out the same regimen of mouth-opening exercise by themselves for 3 months postoperatively, with every weekly regular follow-up by us. After then, they were instructed to open the mouth maximally several times a day, with every monthly regular follow-up. As a result, in all the patients, the maximal mouth opening had evidently increased up (mean = 45.0 mm: range 32–58 mm) at 1-year follow-up after the surgery (Table 3).

DISCUSSION

The pathogenesis of MMTAH is not sufficiently clarified, and diagnostic criteria or classification is still controversial. The similar cases comparing to MMTAH were reported as ''Square Mandible,''[1,4,5] hyperplasia of the coronoid process without interference to the zygomatic bone,[6] or masseter muscle contracture without history of trauma or infection.[7] Because there are strong similarities in the clinical findings among these cases, we think that they should be classified into the same category of MMTAH. Their sever limited mouth opening may have also been caused by contracture

TABLE 3. Results of Operation

Patient No.	Operation	Cours of Mouth Opening, mm		
		Preoperative	Immediate Postoperative	Follow-Up (1 year)
1	Co + Ms	20	55	46
2	Co + Ms	24	56	47
3	Co + Ms	19	48	32
4	Co + Ms	20	59	49
5	Co + Ms	19	54	46
6	Co + Ms	24	60	49
7	Co + Ms	23	60	47
8	Co + Ms	21	60	50
9	Co + Ms	20	55	46
10	Co + Ms	24	54	40
11	Co + Ms	16	60	40
12	Co + Ms	20	60	39
13	Co + Ms	27	60	43
14	Co + Ms	22	60	58
15	Co + Ms	20	60	43
mean		21.27	57.40	45.00

Co, coronoidectomy; Ms, masseter muscle stripping.

of the masticatory muscles with hyperplasia of tendons and aponeurosis.

Regarding the male/female ratio of MMTAH, only 2 of the consecutive 15 patients was male in this study. Although, there is no worldwide report on the gender gap of MMTAH, it is reported that the male/female ratio is approximately 2/5 in the Japanese epidemiological survey.[8]

Inoue et al[2] advocated that masseter muscle aponeuroectomy sufficiently contributes to clinical improvements of MMTAH. On the other hand, Murakami et al[1] and Yoda et al[3] reported that clinical state of the patients with mandibular hypomobility could be improved by surgical intervention to not only masseter muscles but also temporal muscles (coronoidectomy). The present "4-D muscle model" demonstrated that the temporal muscle, especially in posterior part, in the patients could not be expanded enough as much as control group, and the condyle was not beyond the articular eminence even at the maximal mouth opening. Therefore, we thought that the main causal muscle of limited mouth opening in this pathology would be the contracture of the temporal muscles. Then, bilateral coronoidectomy with only masseter muscle stripping has been successfully applied to our patients. The surgical results and analytical results by 4-D muscle model suggested that the hyperplasia of temporal muscle tendon-aponeurosis more strongly restricts the mouth opening than the hyperplasia of masseter muscle tendon-aponeurosis.

Minowa et al[9] reported the usefulness of MRI as a diagnostic modality of masseter muscle aponeurosis, and that no aponuerosis was seen below the lower half of the anterior margin of the masseter muscles in almost normal volunteers. Inoue et al[2] and Yoda et al[3] observed that aponeurosis extended three-fourths or more than two-thirds of the length of the masseter muscle down to the inferior border of the mandible on the MRI in patients with MMTAH. Additionally, on the CT images in the patients with coronoid process hyperplasia without interference between the coronoid process and the zygomatic bone, it was found that the thickness of temporal muscle tendon was significantly bulky.[6] Also, in our all patients, MRI showed the elongated and thick aponeurosis on the anterior surface and inside the masseter muscles, and CT showed the bulky tendon of the temporal muscle. Although the hyperplasia of tendon-aponeurosis in the masseter muscles and temporal muscles was observed in all patients, it was not identified through our surgical results if tendon-aponeurosis hyperplasia of the masseter muscle significantly contributes the limited mouth opening.

Almost our patients had a conscious of bruxism, and it might be contributing factors to the hyperplasia of tendons and aponeurosis. Long-term mandibular hypomobilty might induce the fibrous change and atrophy of the masticatory muscles, which results in significant muscle contracture.[10] Even a minor strain may cause myositis, and the resultant inflammation may lead to fibrous scarring ending in muscle contracture if myositis persists.[11] It seems like that prolonged and significant bruxism and increased traction force by the mouth-closing muscles induced the contracture of masseter muscle or temporal muscle, and consecutive bone overgrowth on the angle of mandible and the coronoid process. However, we could not speculate the mechanism of the relationship of hyperplastic tendon and aponeurosis with the fibrous change of the masticatory muscles. In recent years, however, Nakamoto et al[12] identified that myosin light chain 4 was downregulated and fibrinogen was upregulated in temporal tendon tissue of patients with MMTAH by the proteomics analysis, and suggested that the distinctive expression of these proteins is associated with the pathology of MMTAH. A further molecular analysis in this disorder is expected in future.

In conclusions, in this pathology, contracture of the temporal muscle seems to be main cause of limited mouth opening. Clinically, in any case, coronoidectomy that can release the contracture of the temporal muscle is the best reasonable surgical modality.

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