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

Kazutoshi Nakaoka, DDS, PhD,* Yoshiaki 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-dimensional 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-dimensional 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.

Key Words: Four-dimensional masticatory muscle model, limited mouth opening, masticatory muscle tendon and aponeurosis hyperplasia, square mandible

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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.1-3 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 model1 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
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 aponeurosis 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 aponeurosis 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.

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) 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. 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.
Ethical Approval
Tsurumi University Ethics Committee, approval number: 708

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...
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 ¼ 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,” hyperplasia of the coronoid process without interference to the zygomatic bone, or masseter muscle contracture without history of trauma or infection. 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 severe limited mouth opening may have also been caused by contracture.

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

| Patient | Side | Maximal Mouth Opening, mm | Musseter Muscle Expanded Rate of Muscle at the Maximal Mouth Opening | Temporal Muscle Expanded Rate of Muscle at the Maximal Mouth Opening |
|---------|------|---------------------------|-------------------------------------------------|-------------------------------------------------|
|         |      |                           | Superficial Part | Deep Part | Anterior Part | Middle Part | Posterior Part | 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 |     |                           | 43               |           | 1.12          | 1.14        | 1.41          | 1.23        | 1.32          |
|         |     |                           | 1.14             |           | 1.13          | 1.36        | 1.21          | 1.31        |
| mean    |     |                           | 42.5             |           | 1.14          | 1.18        | 1.38          | 1.24        | 1.33          |

*C*P-value (patients versus control group) 0.486 0.789 0.003 0.002 0.001

Mann-Whitney test.
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 al9 advocated that masseter muscle aponeuroctomy sufficiently contributes to clinical improvements of MMTAH. On the other hand, Murakami et al10 and Yoda et al11 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 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 al10 reported the usefulness of MRI as a diagnostic modality of masseter muscle aponeurosis, and that no aponeurosis was seen below the lower half of the anterior margin of the masseter muscles in almost normal volunteers. Inoue et al9 and Yoda et al11 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.9 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 hypomobility 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 al12 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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