Original

Change in muscle hardness after trigger point injection and physiotherapy for myofascial pain syndrome

Akiko Okada-Ogawa^{1,2,3)}, Naohiko Sekine¹⁾, Kosuke Watanabe¹⁾, Ryutaro Kohashi¹⁾, Sayaka Asano¹⁾, Koich Iwata^{4,5)}, and Yoshiki Imamura^{1,2,3)}

¹⁾Department of Oral Diagnostic Sciences, Nihon University School of Dentistry, Tokyo, Japan

²⁾Division of Orofacial Pain Clinic, Nihon University Dental Hospital, Tokyo, Japan

³⁾Division of Clinical Research, Dental Research Center, Nihon University School of Dentistry, Tokyo, Japan

⁴⁾Department of Physiology, Nihon University School of Dentistry, Tokyo, Japan

⁵⁾Division of Functional Morphology, Dental Research Center, Nihon University School of Dentistry, Tokyo, Japan

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Abstract: Assessment and treatment of masticatory myofascial pain syndrome (MPS) are not standardized and remain controversial. We examined whether muscle hardness was useful for evaluating masticatory MPS and analyzed the effectiveness of treatments such as stretching and massage (SM) and trigger point injection (TPI). Twenty healthy volunteers and 20 MPS patients were enrolled. MPS patients were divided into TPI and SM treatment groups. Hardness of masticatory muscle with a taut band (TB) and change in hardness were evaluated after SM and TPI treatments. Hardness values were significantly higher in muscle including a TB (TB point) than in the muscle of healthy controls. Visual analogue scale scores were significantly lower after SM and TPI treatments, and hardness of the TB point was significantly lower after SM but not after TPI. These results suggest that measurement of muscle hardness, including the TB, is useful for evaluating masticatory MPS. However, TPI analgesia might not be caused by change in muscle hardness. The mechanisms underlying the effects of SM and TPI on reducing pain in MPS may differ and thus warrant further research.

Keywords: masticatory myofascial pain syndrome; taut band; muscle hardness; referred pain; trigger point.

Introduction

Masticatory myofascial pain syndrome (MPS) is characterized by the presence of a myofascial trigger point (MTrP), which induces referred pain in teeth or the temporomandibular joint (TMJ) area. MPS is easily misdiagnosed, which can lead to mistreatment (1-4). Although various methods have been used to assess masticatory MPS and its treatment—such as visual analog scale (VAS) score, pressure pain threshold, maximum mouth opening, tooth contact ratio, magnetic resonance elastography, ultrasound imaging, and Doppler flow waveform—evaluation is not standardized and remains controversial (5-7).

Muscle hardness might be useful for assessing masticatory MPS (8,9) and tension-type headache (10,11). Noninvasive methods of measuring muscle hardness include the pressure method, which measures muscle hardness with a portable muscle hardness meter (8,10-12). Prior studies using a hand-held pressure algometer showed significantly greater muscle hardness in patients with masticatory MPS and tension-type headache than in controls (9,10). To our knowledge, only one study has compared change in masticatory muscle hardness in MPS after massage treatment (9). Masseter muscle hardness was significantly greater in MPS than in healthy

Correspondence to Dr. Akiko Okada-Ogawa, Department of Oral Diagnostic Sciences, Nihon University School of Dentistry, 1-8-13 Kanda-Surugadai, Chiyoda-ku, 101-8310 Tokyo, Japan
Fax: +81-3-3219-8346 E-mail: okada.akiko1@nihon-u.ac.jp

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Table 1 Demographic and baseline characteristics of participants.

	MPS (<i>n</i> = 20)		Controls (<i>n</i> = 20)
	TPI group (<i>n</i> = 10)	SM group (<i>n</i> = 10)	
Age, mean SD (range)	39.44 ± 5.68	40.88 ± 5.00	38.17 ± 5.41
Sex			
female	6	5	12
male	4	5	8
Weight (kg)	58.0 ± 5.58	60.63 ± 4.32	59.61 ± 4.29
Height (cm)	160.78 ± 2.75	163.25 ± 3.82	159.89 ± 1.94
Pain duration (months)	28.33 ± 6.99	32.38 ± 11.67	
Medications			
hypnotics	1	1	
vitamins	1	1	
anti-allergics	1	0	

controls and decreased after massage treatment. Nevertheless, assessment of muscle hardness in MPS is not well understood.

MTrPs are present in palpable nodules of muscle fibers, i.e., a taut band (TB). The TB is a contracted or shortened muscle fiber band that has increased muscle tone (13) and develops a hard consistency during palpation (14). Muscle with a TB is likely harder than muscle without a TB; however, to our knowledge, no study has quantitatively assessed muscle, including TB hardness, in patients with masticatory MPS.

Treatments proposed for MPS include drugs, sprays, TPI, physiotherapy, exercise, thermotherapy, electrotherapy, oral splints, and acupuncture. Physiotherapy, such as stretching and massage treatment, is the most popular treatment for MPS (5,15), and massage treatment for muscles of MPS patients is considered one of the most effective methods for reducing pain and muscle hardness (9). In addition, the use of the local anesthetic lidocaine in trigger point injection (TPI) of MTrPs reduces localized and referred pain and is a mainstay of short-term MPS management (16-18). However, the outcomes of these MPS treatments have not been carefully evaluated.

This study assessed muscle hardness, including TB-referred pain, in the orofacial area of MPS patients and compared the findings with those of healthy controls. The secondary aim was to evaluate the effect of self-stretching and massage (SM) and TPI treatments on referred pain and muscle hardness in patients with masticatory MPS.

Materials and Methods

Participants

Twenty patients were randomly recruited from the outpatient pain clinic at Nihon University Dental Hospital. The

inclusion criteria were chronic unilateral pain of longer than 3 months' duration that was diffusely distributed in the face, mouth, or TMJ and was referred from MTrPs in the masseter or trapezius muscles (MPS group). These inclusion criteria for MPS are consistent with the guidelines of the American Academy of Orofacial Pain and the Research Diagnostic Criteria for TMD (RDC/TMD) (19,20). MPS patients were randomly assigned to TPI treatment (TPI group, *n* = 10) or self-stretching and massage (SM group, *n* = 10). Intra/extraoral examinations and routine X-ray screening showed no dental or maxillofacial lesions that might be the source of pain. Magnetic resonance imaging and joint X-rays for the MPS patients did not reveal disc displacement, effusion, disc deformity, or bone changes.

Twenty healthy controls were also enrolled (control group). The exclusion criteria were any pain or discomfort in the orofacial area, including the TMJ, or a history of facial or head/neck pain. The controls could not be evaluated with magnetic resonance imaging or joint X-rays for ethical reasons; however, extraoral examination showed no tenderness in the TMJ or masticatory muscles and no disorder of jaw movement.

In addition, MPS patients and healthy controls were excluded if they (a) presented with disc displacement, arthrosis, or arthritis of the TMJ, according to categories II and III of the RDC/TMD (19); (b) had received a diagnosis of muscle disease, such as a fibromyalgia syndrome or myopathy; (c) had any other medical or psychological condition, including major depression, schizophrenia, cancer, or endocrine diseases; or (d) were taking muscle relaxants, nonsteroidal anti-inflammatory drugs, or any other analgesic medicine. Details of the MPS and control groups are shown in Table 1.

Table 2 Number of active and latent trigger points in taut bands and symptomatic areas of patients receiving TPI and SM.

	Masseter m.		Trapezius m.		Temporal m.		SCM		Splenius m.	
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
<i>TPI group</i>										
Active MTrPs (n)	2	1	1	2	1	1	0	0	0	0
Latent MTrPs (n)	3	4	1	3	4	3	2	3	1	0
<i>SM group</i>										
Active MTrPs (n)	1	1	0	2	1	1	0	0	0	0
Latent MTrPs (n)	2	5	1	2	3	3	3	4	1	1

<i>Symptomatic area</i>	TMJ		Upper Molar		Lower Molar		Head		Cheek		Jaw	
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
<i>TPI group (n)</i>	2	3	1	3	2	3	0	1	0	0	1	0
<i>SM group (n)</i>	1	2	1	3	2	4	0	0	1	1	1	0

TPI; trigger point injection. SM; stretching and massage. SCM; sternocleidomastoid muscle. MTrPs; myofascial trigger points.

Study design and procedure

This study was conducted in accordance with the Helsinki Declaration and was approved by the Nihon University School of Dentistry Bioethics Committee (EP2007-13). The examiners were blinded to data on muscle hardness during comparison of the MPS and control groups.

The details of the study were explained to the 40 study participants, who were later informed of the measurements. All participants provided written informed consent before enrollment. Once the consent form was signed, demographic data were collected. In the MPS group, primary pain complaints at rest and during mastication were measured at baseline by using a VAS score ranging from 0 (no pain at all) to 100 (worst pain imaginable). Active MTrPs cause spontaneous local and referred pain, whereas latent MTrPs cause only local and referred pain during direct muscle palpation (21). Therefore, well-trained examiners used manual palpation to carefully explore MTrPs in the bilateral masseter, temporal, sternocleidomastoid, and trapezius muscles of all participants. Active and latent MTrPs where severe pain was referred to an area are shown in Table 2.

The TPI group received a weekly treatment for 2 weeks, and the SM group completed the assigned exercises for 2 weeks, in accordance with the procedure described below. VAS scores were collected at 0 weeks (baseline), 2 weeks before TPI treatment, and immediately after TPI treatment in the TPI group, and at 0 and 2 weeks in the SM group. Muscle hardness of the masseter and/or trapezius muscles was measured at 0 weeks (baseline) and at 2 weeks in both groups. Figure 1 shows the study timeline for the TPI and SM groups. The hardness of the masseter and trapezius muscles was also measured in healthy controls at the initial visit.

Fig. 1 Study timeline for the trigger point injection (TPI) and stretching and massage (SM) groups.

Measurement of muscle hardness

In the MPS group, a hardness meter operated with an electronic solenoid (DPS-260, DIA medical system, Tokyo, Japan) was used to measure the hardness of the masseter and/or trapezius muscles, including the TB (TB point), which also included active or latent MTrP. The presser bar was placed at the most tender point on palpation of the TB, which was referred to as the symptomatic orofacial area. In contrast, no TB or tender points were noted on the asymptomatic side of patients in the

Fig. 2 Schematic diagram of system for measuring muscle hardness and curve of the displacement-force relationship. (a) The vibrator system for measuring muscle hardness and head fixation device were secured to a fixed pole. (a') The upper, middle, and lower parts represent tissue displacement, force response, and calculated hardness, respectively. (b) The dotted line is a representative curve for the displacement-force relationship at the masseter muscle control point. The regression line of the curve is shown as a solid line.

MPS group. The bar was located at a contralateral site corresponding to the TB point, which was designated the non-TB point. In the control group, the bar was located close to the thickest part of the masseter muscle belly, near the midpoint of the trapezius muscle belly, which served as the control point.

The hardness of TB, non-TB, and control points was measured with a fixed hardness meter, as described previously (12). Briefly, participants were seated in a relaxed position in a dental chair, and their heads were immobilized with a brief stereotaxic apparatus. The hardness meter includes a presser made up of a solenoid and an electric stimulator (Fig. 2a). The presser and stereotaxic apparatus were mounted on a fixed pole, and the height of the presser and stereotaxic apparatus were adjusted to the height of the participant. The axle angle of the presser was also adjusted to the position required to stimulate the muscle perpendicularly. Displacement and force response were recorded by using a data recorder with a data acquisition system connected to the hardness

meter (Fig. 2a'). The plastic tip of the presser was placed perpendicularly to the body surface. The smooth contact surface (1 cm²) of the tip touched the skin gently at the beginning of each measurement, and the sensor increased pressure at a constant rate (2.5 mm/s) while simultaneously detecting tissue deformity. The relationship between tissue displacement and force response is shown in Fig. 2b; the regression line was calculated with the least-squares method (12). The first component (curved line) corresponds to subcutaneous tissue, whereas the second component (straight line) corresponds to the muscular portion. Therefore, 50 kPa was selected as the appropriate pressure to obtain muscle hardness in the present study. To avoid observer bias, muscle hardness was calculated automatically with a software program (UAS-108S, Unique Medical, Tokyo, Japan) using the following equation:

$$E = Id(1-\mu^2)(yx^{-1})$$

where E is muscle hardness, I is the influence coefficient (0.85), d is the diameter of the probe, μ is Poisson's ratio (0.5), x is the amount of displacement, and y is force. Measurements were repeated three times at a constant interval of 15 s, and the average of the three measurements was analyzed as the hardness score.

In the control group, measurements of hardness were also repeated three times for both sides of the masseter and trapezius muscles. The average of values from both sides was used for hardness score, because the averages of the right and left sides did not significantly differ.

TPI and SM treatment

In the TPI group, patients were treated with a standard TPI procedure (22,23). Trigger points were identified by manual palpation of the masseter, temporal, sternocleidomastoid, and trapezius muscles, and the skin over the trigger points was prepared by cleaning with alcohol. A 0.5-mL injection to the trigger point was made with a 27-gauge, 1.5-inch needle connected to a 5-mL syringe filled with 1% lidocaine. One to six of the most painful trigger points in each muscle were injected per participant. After injection, the injection area was compressed firmly to achieve hemostasis. The diameter of MTrPs is a few millimeters, and a previous study reported that injections outside the trigger point did not alleviate pain (24). Therefore, highly skilled practitioners performed the TPI, and a radiating sensation was confirmed after each injection. Patients were treated with TPI weekly for 2 weeks—at the initial, second, and third visits—as shown in Fig. 1a.

In the SM group, patients were instructed to follow

Fig. 3 Muscle hardness at the control, TB, and non-TB points in masseter and trapezius muscle. * $P < 0.05$.

a home exercise program that included stretching and massaging of their masseter, temporal, sternocleidomastoid, and trapezius muscles, according to the procedure detailed at the first visit (7). Briefly, they were instructed to use their bilateral hypothenars to massage the symptomatic temporal and/or masseter muscles at the tender point for 30 s each. They were also instructed to lift their bilateral temporal muscles, pull down their mandible, move both shoulders in circles, and incline their head to both sides to stretch the sternocleidomastoids 10 times for 5 s. They were encouraged to massage and stretch five times a day (in the morning, after each meal, and just before sleep) for 2 weeks. They were able to follow the instructions and complete the SM program and were asked, 1 week after instruction, to confirm that they had completed the program correctly (Fig. 1b).

Statistical analysis

After using the Levene test for homogeneity of variances to check for significant differences in variance, we used repeated-measures two-way ANOVA to assess post-treatment change in VAS and muscle hardness. If significance was reached on ANOVA, *post hoc* comparisons were performed with the Tukey-Kramer test. The Student *t* test or Mann-Whitney *U* test was used to compare values in the MPS and control groups. Differences were considered significant when P was < 0.05 . The results are presented as mean $\pm$ SEM.

Results

Participants

Of the 25 MPS patients originally enrolled, five declined further participation and 20 underwent initial examination. Demographic data for the three groups are presented in Table 1. Among the 20 MPS patients, the TPI group comprised 6/10 (60.0%) women and 4/10 (40.0%) men,

and the average age was 39.44 ± 5.68 years (range, 24-69 years). The SM group consisted of 5/9 (55.6%) women and 4/9 (44.4%) men, and the average age was 40.88 ± 5.00 years (range, 27-66 years). The control group consisted of 12/20 (60.0%) women and 8/20 (40.0%) men, and the average age was 38.17 ± 5.41 years (range, 25-60 years). There was no difference between groups at baseline with respect to age, sex, weight, height, or pain duration.

Table 2 shows the general locations of active and latent MTrPs and the chief complaints in the TPI and SM groups. They had MTrP not only in the masseter and trapezius muscles but also in the temporal, sternocleidomastoid, and splenius muscles. The mean numbers of active and latent MTrPs were 1.6 and 4.8, respectively, in the TPI group and 1.2 and 5.0, respectively, in the SM group. There was no significant difference between groups with respect to active or latent MTrPs numbers. The TPI and SM groups had chronic unilateral pain (without sideshift) in the upper or lower molars, TMJ, head, cheek, face, or jaw that had not been relieved by previous dental treatments (TPI group: 28.33 ± 6.99 months; SM group: 32.38 ± 11.67 months). For MTrPs, all symptomatic areas were ipsilateral, and referral patterns were consistent with previous descriptions (25).

Comparison of the MPS and control groups

TB point hardness was significantly greater at the masseter muscle than at the control point ($F_{1,38} = 6.776$, $P < 0.05$). Although non-TB point hardness was greater than that of the control point, the difference was not significant ($F_{1,38} = 2.872$, $P < 0.1$; Fig. 3). In trapezius muscles, TB point hardness was slightly higher than the values for the non-TB and control points, but the differences were not significant (non-TB point: $F_{1,38} = 2.284$, $P < 0.2$; control point: $F_{1,38} = 3.735$, $P < 0.07$; Fig. 3).

Comparison of the TPI and SM groups

In the TPI group, as compared with baseline at 0 weeks, mean VAS scores for pain intensity at rest in the symptomatic area were significantly lower immediately after injection, but not before injection, at 2 weeks ($F_{1,18} = 8.134$, $P < 0.05$). VAS during mastication was significantly attenuated in the symptomatic area immediately after injection at 2 weeks, as compared with baseline at 0 weeks, and before injection at 2 weeks (0 weeks: $F_{1,18} = 8.435$, $P < 0.05$; before injection at 2 weeks: $F_{1,18} = 6.162$, $P < 0.05$; Fig. 4a).

VAS scores in the SM group at rest and during mastication were significantly lower after SM treatment at 2 weeks than at baseline (at rest: $F_{1,18} = 9.308$, $P < 0.01$;

Fig. 4 VAS scores for the TPI group at 0 weeks, immediately after injection (pre-inj.), and 2 weeks after injection (post-inj.) (a) and for the SM group at 0 and 2 weeks post-treatment (b). VAS scores are for the primary complaint of oral or perioral pain at rest and during mastication. * $P < 0.05$, ** $P < 0.01$.

Fig. 5 TB and non-TB masseter muscle hardness in the TPI group (a) and SM group (b) at 0 and 2 weeks post-treatment. * $P < 0.05$.

during mastication: $F_{1,18} = 22.789$, $P < 0.01$; Fig. 4b).

As shown in Fig. 5a, TPI group masseter muscle hardness at the TB and non-TB points decreased after treatment; however, the difference was not significant (TB: $F_{1,18} = 3.852$, $P < 0.07$; non-TB: $F_{1,18} = 3.122$, $P < 0.1$). In contrast, in the SM group, masseter muscle hardness at the TB point was significantly lower after SM treatment ($F_{1,18} = 5.437$, $P < 0.05$). Non-TB hardness was not significantly different after treatment ($F_{1,18} = 0.022$, $P < 1.00$; Fig. 5b).

Discussion

We assessed muscle hardness, including the TB, in masticatory MPS patients and evaluated changes in muscle hardness and referred pain after TPI and SM treatments. Masseter muscle hardness at TB points was significantly higher in MPS patients than in healthy controls. In contrast, previous studies reported no significant differences in muscle hardness between MPS patients and healthy controls (8), perhaps because these studies did not include the TB when measuring muscle hardness in MPS. Moreover, prior studies used a hand-held pressure algometer that could not incorporate the hardness of subcutaneous fat over muscle, which has a substantial

effect on muscle hardness assessment. In the present study, we measured muscle hardness with a two-layer spring model that can measure muscle hardness without subcutaneous fat (12,26). Moreover, fixed hardness meters are likely to yield reproducible results.

Hiraiwa et al reported a significant difference between the right and left sides in the masseter hardness of MPS patients, which suggests an imbalance in myofascial pain in the right and left masseter muscles (9). In the present study, although TB hardness was slightly higher than non-TB hardness on the contralateral side, the values did not significantly differ in MPS patients. A possible cause of MPS is involuntary low-level contraction of masticatory muscles; thus, the contralateral muscles might also be somewhat contracted. Therefore, symptoms related to the hardness of the contralateral masseter muscle might also increase over a long period. The pain duration of MPS in the present study was relatively long, 2-3 years; therefore, contralateral, non-TB, hardness might have increased.

Techniques such as magnetic resonance elastography and ultrasound elastography have recently been developed to quantify muscle elasticity (6,27,28), and some studies have assessed muscle hardness in MPS patients.

However, prior studies included a novel elasticity index that measures absolute muscle hardness. For example, a reference of known hardness was placed over tissue and the strain ratio was calculated (27), or the color of the compressed area was compared to that of the surrounding area (29). These techniques may not be truly quantitative, since the stress distribution within muscle tissue is neither known nor uniform (28). Akagi and Kusama measured the hardness of neck and shoulder muscles with ultrasound elastography and a portable hardness meter and compared the findings (30). Ultrasound elastography findings were not correlated with hardness meter measurements of muscle stiffness. The authors concluded that the precision of ultrasound elastography was not adequate for measuring muscle stiffness. The present findings suggest that direct measurement of muscle hardness with a hardness meter that is not affected by the presence of subcutaneous fat improves clinical diagnosis and treatment.

Long-term low-level masseter muscle contraction may be related to edematous muscle thickening, which can induce muscle hardness (31-33). In subcutaneous edema with high water content, the area usually becomes soft. In contrast, muscle edema usually becomes hard because of the thick fascia covering the muscles (9). Ariji et al reported changes in ultrasound elastography findings after low-level static contraction of masseter muscles (6). The intramuscular soft and hard area increased, and the area of middle hardness decreased, thus increasing total muscle hardness after muscle contraction. Therefore, edematous changes and production of TB may increase overall muscle hardness in MPS patients.

Analysis of the effect of SM and TPI treatments on referred pain and muscle hardness in masticatory MPS patients showed that referred pain at rest and during mastication was relieved after TPI and SM treatment, which is consistent with previous results (34). MPS patients report referred pain at rest, and this pain is sometimes aggravated during mastication (20). Therefore, we investigated the effect of TPI and SM treatment on referred pain both at rest and during mastication in the present study.

Muscle hardness in TB points was significantly lower after SM but after not TPI treatment. Although previous studies developed an index for evaluating the effect of massage treatment for muscles (9,35,36), no study used muscle hardness to examine the effect of TPI treatment. We suspected that a decrease in muscle hardness would correspond with a TB-induced decrease in pain. However, the present results showed that the effect of TPI on pain release was not associated with decreased

muscle hardness.

Physical therapy, such as stretching or massage, can reduce edema, muscle tension, and naturally occurring discomfort, and markedly suppress pain, perhaps through the release of endogenous opiates (37-39). The present decrease in muscle hardness and referred pain after SM suggests that it improved muscle tension and edema at the TB point, perhaps by inducing an analgesic effect. However, the mechanism by which TPI reduces referred pain might not be associated with edematous change and muscle tension. Injection of local anesthetics to MTrPs is most effective in inactive MTrPs (17,40,41). However, the analgesic effect of TPI seems to depend on the needling effect, as previous studies found no significant difference between the effect of anesthetic injection and that of dry needling of MTrPs (42,43). Several prior investigations reported that repeated dry needling possibly decreased neuropeptides such as substance P and calcitonin gene-related peptide concentrations in active MTrPs of MPS (44-46). Some evidence indicates that the descending pain inhibitory system is the most likely mechanism for this analgesic effect (47,48). Nevertheless, the mechanisms underlying the analgesic effects on MPS are unclear and should be investigated in future studies of the pathogenesis of MPS and the effect of TPI.

This study has several limitations. Because repeated intramuscular injection of local anesthetics results in myotoxicity (49,50), this study used a short-term intervention, 2 weeks of treatment, to compare the effects of SM and TPI treatment. Although prior investigations reported that weekly TPI for 1-2 weeks immediately improved myofascial pain (16-18,40,51), some studies noted significant decreases in pain scores over 4 weeks after repeated TPI for MTrPs in patients with myofascial pain (43,52,53). Ideally, future studies should assess the effects of repeated TPI and dry needling for longer than 4 weeks on muscle hardness, including the TB.

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Conflict of interest

The authors report no conflict of interest in relation to this study.

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