Laser phototherapy in acute posttraumatic trismus – Case-series study

Emilia Rasca, Amaury Namour, Aude Fauchon-Giumelli, Samir Nammour

Department of Dental Sciences, Faculty of Medicine, University of Liège, Belgium
Institut de dentisterie – Polycliniques Brull, Liège, Belgium

Backgroud and aims: There are very few studies on laser phototherapy (LPT) in acute temporomandibular disorders (TMDs). Our objective is to assess the effectiveness of laser phototherapy (LPT) on the limitation of the mouth opening due to an acute mandibular trauma.
Subjects and methods: Fourteen women of 41 ± 3 years and 24 men of 38 ± 3 years, with no history of TMD and having sustained a mandibular trauma within the prior 20 hours, were treated exclusively by using an 810-nm laser beam in a continuous wave mode, with an output power of 1 W. At a speed of 2 cm/s, it scanned twice, for 60 seconds, with a pause in between of 2 minutes, a large cutaneous area (25 cm^2), covering the temporomandibular joint (TMJ), the masseter muscle and a part of the temporalis fossa; also, it scanned just once, for 7 seconds, a small mucous area (3 cm^2), covering the internal pterygoid muscle. The clinical outcomes were evaluated by comparing the maximum unassisted opening (MUO), measured at the baseline and immediately after the end of the LPT procedure.
Results: The MUO improvement of 24.6 ± 4.4 mm represented a highly significant difference ($p < .0001$) between the measurements, in all the patients, regardless of gender.
Conclusions: By scanning with an 810-nm laser beam, within less than 20 hours after the trauma, large areas of all the involved tissues and not just a few points, as described until now, the limited mouth opening in acute posttraumatic trismus was immediately and greatly resolved.

Key words: laser phototherapy • low-level laser therapy • temporomandibular joint disorders • mandibular trauma • masticatory muscles • temporomandibular joint • acute trismus

Introduction

Derived from the Greek word 'trismos', which means 'gnashing', the term trismus defines any restricted mouth opening, regardless of etiology, which can be traumatic, inflammatory, neoplastic, metabolic or neurogenic [1]. Commonly, it refers to a prolonged, tetanic spasm of the jaw muscles. Acute trismus is most often of inflammatory and/or traumatic origin. Orofacial traumas generate nociceptive inputs processed in the spinal trigeminal nuclear complex (STNC), which induce sustained bilateral increase in electromyographic activity of both elevators and depressors of the jaw [2, 3]. This results in mouth opening limitation, occurring within minutes after trauma and involving two pathophysiological mechanisms. The first one is a purely reflex trismus, also called protective co-contraction [4], which is not always associated with myalgia [5]. When prolonged, it may be followed by local muscle soreness [6], characterized by the release of algogenic substances, such as bradykinin, substance P and histamine, which activate and sensitize the muscle nociceptors and thus reinforce the STNC activation. This primary, non-inflammatory, myalgia can also be induced by an excessive use or a local trauma of the muscle. So, at the beginning, when the patient is asked to open slowly, full opening is achieved; after a while, he/she becomes unable to reach the maximal opening of the mouth. Consequently, acute posttraumatic trismus can seriously impair all the mandibular functions, as well as the oral hygiene.

Conventional primary therapy of acute musculoskeletal traumas, including masticatory system, is largely based on an empirical pharmacotherapy [7], including

Addressee for Correspondence:
Dr Emilia Rasca, MD, DDS
Immeuble Le Rond Point 8, route de la Sablière
13011 Marseille, France
Phone: +33-980-682-867 Fax: +33-985-682-867
e-mail: emilia.rasca@orange.fr

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muscle relaxants, nonsteroidal anti-inflammatory drugs (NSAIDs), and/or analgesics. Laser phototherapy (LPT) has been used for more than three decades for the treatment of musculoskeletal disorders [8], including those of the masticatory system [9]. The LPT effectiveness in the treatment of chronic temporomandibular disorders (TMDs) is supported by many studies [10-15], although unsuccessful results have also been reported [16-19].

So far, very few studies [20, 21, 14] addressed the LPT efficacy in acute (< 30 days) or recent (< 6 months) TMDs. Besides, their conclusions were somewhat confusing: one of these studies reported better outcomes in acute than in chronic TMDs [14], while another one reported that the same LPT protocol was successful in chronic cases and totally unsuccessful in recent cases of both muscular and articular TMDs [20]. Our previous study [22] has demonstrated that LPT alone can resolve in 15 minutes an acute posttraumatic painful trismus with a history of less than 48 hours, by using a combined red-infrared laser beam. The aim of this study is to assess the effectiveness of laser phototherapy (LPT) on the limitation of the mouth opening due to an acute mandibular trauma.

Subjects and methods

Study population

Fifty consecutive patients presenting acute posttraumatic painful trismus, following either a mandibular blow (occurred as a consequence of car or bicycle accident, falling-down, sports, violent attack) or iatrogenic procedures (jaw overextension), were recruited for this study, which was conducted with respect to the recommendations of our university ethic committee. Also, this human study was approved by the relevant Institutional Review Board.

The including criteria were an acute posttraumatic trismus with an onset within the prior 20 hours and no treatment preceding the LPT. The exclusion criteria were: previous TMD history, any radiological sign of mandibular fracture or TMJ abnormality, and any therapy prior to the baseline examination, as well as systemic diseases of any etiology, and psychiatric disorders. Finally, 38 patients have been enrolled, 14 women of ages between 22 and 54 (41 ± 3 years) and 24 men aged between 18 and 64 (38 ± 3 years). All of them gave written, informed consent. The time elapsed between the symptoms onset and the clinical examination was between 4 and 19 hours (9.6 ± 3.7 hours).

Clinical examinations

The clinical examination of all the patients was performed according to the examination protocol of diagnostic criteria of TMD (DC/TMD) [23]. Because of the intense pain experienced by most of the participants the maximum assisted opening could not always be measured, so the maximum unassisted opening (MUO), including incisor overbite, was chosen as the representative measurement of the jaw mobility. Each recorded value was a mean of three measurements, read to the nearest millimeter on an interincisally-placed ruler. A panoramic radiograph was taken for each patient and it ruled out any fracture or visible TMJ abnormality. The MUO improvement was assessed immediately after the LPT procedure.

LPT protocol

An 810-nm laser beam (Digilase PDT 5W250 laser, Biophoton, Saint Alban, France, 2014) with an out power of 1 W, delivered in a continuous wave mode by an optic fibre of 600 μm kept perpendicular to the epithelium at a

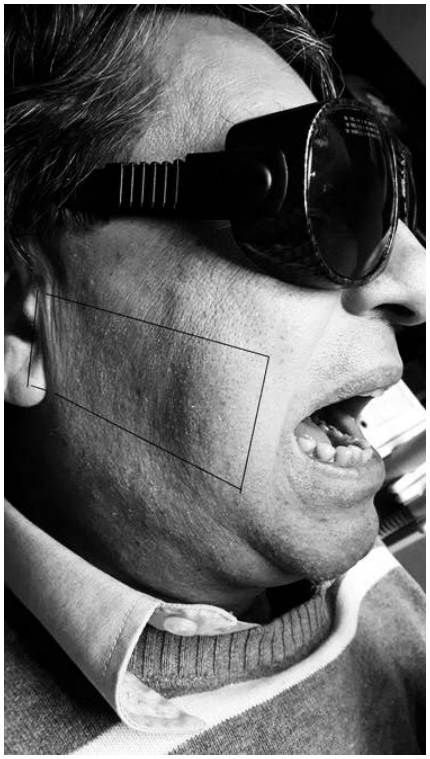

Figure 1: The laser scanned a surface of about 25 cm², including: 1 – the TMJ lateral aspect and the auriculotemporal nerve, running between the condyle neck and the external auditory canal, at minimum 8 mm in front of the posterior aspect of the tragus; 2 – the whole surface of the masseter muscle, underneath the zygomatic arch, including the mandibular notch, from which the masseteric nerve emerges; 3 – the lower part of the temporalis muscle, about 1.5 cm above the zygomatic arch, between the auricle and the orbital ridge, by avoiding the haired skin. The deep temporalis nerves travel upwards at the temporalis bone contact, just above the superior ridge of the zygomatic arch.

distance of 2 to 4 mm, scanned a cutaneous and a mucous area, at a speed of 2 cm/s. The cutaneous area covered the TMJ lateral aspect and part of the auriculotemporal nerve, the masseter muscle and its innervation, as well as the lower part of the temporalis fossa, including the muscle and its innervation **(Figure 1)**. It was 25 cm² (6.5 x 4 cm) and was irradiated twice during 60 seconds, with a pause in between of 2 minutes. As soon as the mouth opening allow it, a systematic LPT of the internal pterygoid muscle was performed, by scanning just a single time 3 cm² (2 x 1.5 cm) of the inner cheek mucous surface overlaying the muscle, during 7 seconds. Hence, LPT was administered in a unique session, bilaterally in case of bilateral myalgia, with the same irradiance at target between 796.2 and 3184.7 W/cm², on both cutaneous and mucous areas **(Table 1)**.

Statistical analyses

The statistics and their graphs were performed by Prism® software version 6 (GraphPad Software, Inc., San Diego, USA). The threshold of significance was set at $p < 0.05$ for all the tests. In the overall group and the gender subgroups, D'Agostino-Pearson omnibus test was used to check the Gaussian distribution of all the parameters: age, time elapsed between the symptoms onset and the clinical examination, MUO before LPT, MUO after LPT and MUO improvement. The variances' homogeneity was checked by the F test.

The gender groups were compared as regards with ages, MUO before and after LPT and MUO improvement by unpaired parametric t test; for the elapsed time from the onset of symptoms, whose distribution was not Gaussian in the women's group, we used Mann-Whitney test. Two-way analysis of variance with repeated measures (ANOVA-RM) in conjunction with Tukey's multiple comparisons test served to compare the MUO before and after treatment in the overall group. Since we have noted, for each patient, the number of hours elapsed between

Table 1: Irradiation parameters and dosimetry of the laser beam. The radiant exposure was about 2.3 J/cm² for both the cutaneous and the mucous areas.

PARAMETER	VALUE
Central wavelength (nm)	810
Spectral bandwidth (FWHM), nm	1.3
Emitter Type	GaAlAs
Operating mode	continuous wave
Radiant power (mW)	1000
Aperture diameter (cm)	$6 \cdot 10^{-2}$
Beam divergence (radians)	0.1
Beam spot size at target (cm²)	$3.14 \cdot 10^{-4}$ to $12.56 \cdot 10^{-4}$
Irradiance at target (W/cm²)	796.2 to 3184.7
Exposure duration (s)	Cutaneous: 60 x 2 (pause 120) Mucous: 7 x 1
Radiant exposure (J/cm²)	Cutaneous: 2.4 Mucous: 2.3
Radiant energy (J)	Cutaneous: 60 x 2 Mucous: 7 x 1
Irradiated area (cm²)	Cutaneous: 25 Mucous: 3
Polarization	no
Beam shape	circular
Beam profile	top hat
Beam Delivery System	silica optical fibre
Application technique	Scanning at a distance of 2 to 4 mm
No. of treatment sessions	1

trauma and LPT, Spearman nonparametric correlation and linear regression allowed us to search for a relationship between the MUO improvement and the duration of symptoms.

Results

Established according to DC/TMD, the pain-related TMD diagnoses **(Table 2 and 3)** always associated acute arthralgia to myalgia. Most often, the masseter and temporalis muscles were both affected, either unilaterally (on the side of the arthralgia or on the opposite side) or bilaterally. Headache associated to TMD was present in 5 patients (13 %), all of them suffering of bilateral myalgia, in both masseter and temporalis muscles. Most often, myalgia was spread in the whole muscle (myofascial pain). Globally, the masseters were more affected (49 locations from 76 muscles, in 38 patients) than the temporalis muscles (43 locations). When present, referral masseteric pain was always observed in the TMJ area.

As soon as the LPT procedure was completed we observed a MUO improvement of 24.6 ± 4.4 mm. Unsurprisingly, a highly significant difference ($p < .0001$) was revealed between the baseline and the final values of the MUO **(Table 3 and Figures 2 and 3)**. None of the studied parameters revealed statistical difference between

Table 2: The pain-related TMD diagnoses in patients with posttraumatic acute trismus evolving for 4 to 19 hours. All the patients suffered of arthralgia, at least on one side. Myalgia and/or headache was present either unilaterally (on the side of arthralgia or on the opposite side) or bilaterally. For each diagnose, the number of cases was reported to the whole group of 38 patients (percentage).

Acute Pain-Related TMD Diagnoses		Same side	Opposite side	Both sides
Arthralgia	without myalgia	0	0	0
	associated to myalgia	19 (50 %)	7 (18 %)	12 (32 %)
Myalgia	temporalis & masseter muscles	20 (53 %)	5 (13 %)	9 (24 %)
	masseter muscle only	2 (5 %)	0	2 (5 %)
	temporalis muscle only	0	0	0
Headache attributed to TMD		3 (8 %)	0	2 (5 %)

Table 3: For each painful muscle, the myalgia type was noted regardless of its association to muscle pain(s) in other location(s). For each myalgia type, the number of cases was reported to the number of 49 masseters, respectively 43 temporalis muscles, affected in all of the 38 patients (percentage).

Myalgia type	Masseter	Temporalis
Local myalgia	4 (8 %)	0
Myofascial pain	41 (84 %)	40 (93 %)
Myofascial pain with referral	4 (8 %)	3 (7 %)

Table 4: Mean, standard error of mean (SEM) and standard deviation (SD) of maximal unassisted opening (MUO) before and immediately after LPT, as well as the MUO gain, have been calculated for the overall group and for each gender.

MUO (mm)		Overall (n = 38)	Females (n = 14)	Males (n = 24)
before LPT	Mean	13.6	14.1	13.3
	SEM	0.6	1.0	0.8
	SD	3.8	3.6	3.9
after LPT	Mean	38.2	37.1	38.9
	SEM	0.4	0.7	0.5
	SD	2.7	2.7	2.6
gain	Mean	24.6	23.1	25.5
	SEM	0.7	1.0	0.9
	SD	4.4	3.8	4.6

genders. Spearman nonparametric correlation (r = 0.04) and linear regression **(Figure 4)** showed that the number of hours elapsed between trauma and LPT does not influence the MUO improvement.

Discussion

Any blow to the jaw induces a trauma in the TMJ, always responsible – at an extent depending on the individual functional reserves – of discal ligaments elongation, overloading of the articular surfaces, capsular abusive strain and/or acute retrodiscitis [24, 25]. However, the chief complaint is not the arthralgia, but a painful trismus, which occurs within hours after the facial trauma. Moreover, the MUO poorest values were associated to the longer durations of symptoms, in patients which received, for 48 hours or more, pharmacological therapy only, as the conventional primary care of acute posttraumatic trismus [22].

Beyond the poor effectiveness of the current conventional therapy, responsible of longer durations of symptoms, the greatest concern should be the higher prevalence, in these patients, of neuroplasticity manifestations, such as heterotopic pain, including headache attributed to TMD, and myalgia of myofascial type (with/without referral), located on the opposite side of the arthralgia or bilaterally [22]. The reasons of this great concern are the observations about chronic posttraumatic TMDs: in these patients, compared to those without trauma history, the maximal mouth opening is significantly more reduced [26] and their response to therapies is much poorer [26-28]. It should be mentioned that acute trauma has been incriminated as the precipitating event in 43 % of chronic TMDs [29]. Unlikely the acute posttraumatic TMDs, where the painful dysfunction has a double origin, articular and muscular, in chronic posttraumatic TMDs the masticatory musculature is the main source of pain and dysfunction [27, 28]. This underlines the strong involvement of central and peripheral sensitization mechanisms, which should be a therapeutic target in itself, from the very beginning of the symptoms, within hours after the facial trauma.

The rational of the necessity to stop theses mechanisms as soon as possible is provided by the animal ex-

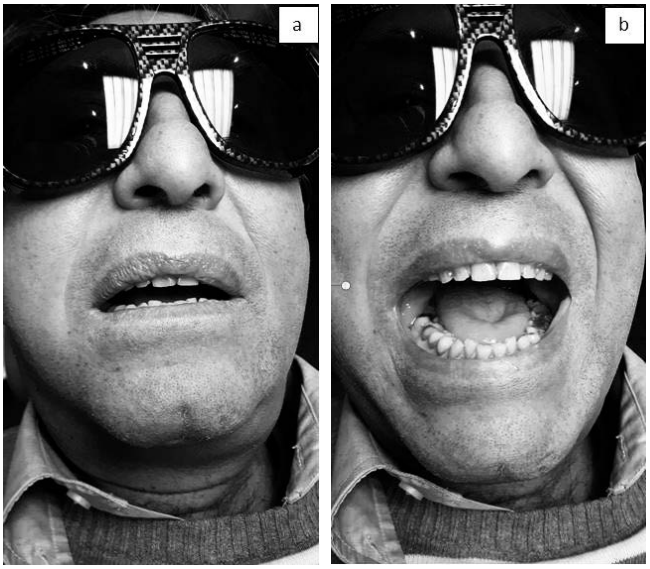

Figure 2: Maximum unassisted opening (MUO) before LPT **(a)** and immediately after LPT **(b)**.

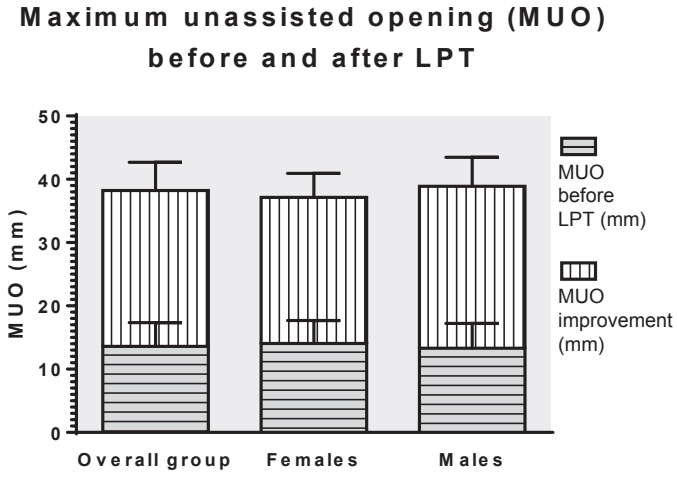

Figure 3: ANOVA-RM in conjunction with Tukey's multiple comparisons test have revealed a MUO mean significantly higher after LPT in all the patients. No between-groups significant differences have been observed in the MUO before LPT, after LPT or in the MUO gain.

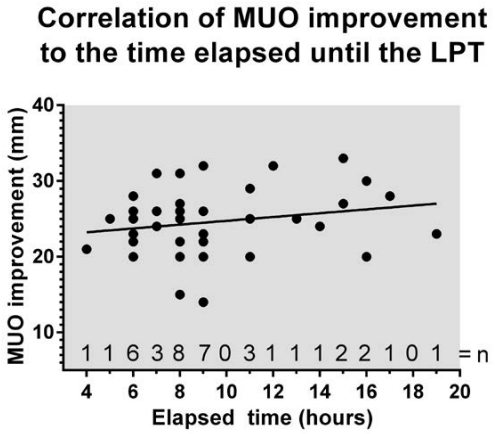

Figure 4: The MUO improvement is not correlated to the number of hours (4 to 19) elapsed between trauma and LPT. The number of cases (n) is indicated for each elapsed hour in this interval.

periments. They have shown that a stronger and more widespread central neuronal activation in the STNC [30-32], as well as a greater activation of the masticatory muscles [33] are induced by the inflammation of orofacial deep tissues than by cutaneous inflammation. Moreover, neuronal activation, as indicated by Fos protein expression, is induced bilaterally by the masseter inflammation, while it is ipsilateral following a skin-cut over the masseter muscle [34, 35]. The inflammation of the orofacial deep tissues, provoked by inflammatory substances injected into masticatory muscles [36] or TMJs [37] increase the excitability and expand the receptive fields of trigeminal nociceptive neurons. As early as at 30 minutes afterwards, reactive astrocytes were seen in the STNC and lasts for about one week after the disappearance of the inflammation [38]. Glial hyperactivity induced by masseter inflammation was correlated to the hyperalgesia onset [39, 40]. Noxious stimulation emanating from injured craniofacial tissues triggers a cascade of cellular events in the central nervous system, including the activation of neurotransmitter receptors and neuron-glia-cytokine interactions, which leads to long-term increases in excitability and plasticity, referred to as central sensitization [41] and underlies the mechanisms of persistent pain [42, 43]. On the other hand, studies on acute posttraumatic muscular dysfunctions in rats revealed an impairment of the mitochondrial function, as indicated by a linear decline of the cytochrome-c oxidase activity of 32 % during the first 5 hours after the injury [44].

Put together, these clinical and experimental data suggest that is extremely important to block the noxious stimuli generated by trauma in all the involved deep orofacial tissues, and to reduce very quickly and as much as possible the muscular dysfunctions at both cellular and tissue levels. Now LPT already demonstrated its ability to optimize the muscular function in hypoxic conditions, such as mechanical stress, fatigue and neurogenic inflammation, responsible of electrolytic and metabolic changes, amongst which ATP and glycogen depletion, oxidative stress, tissue hypoxia and acidification [45, 46]. These LPT effects are considered to be mainly due to the mitochondrial activation, resulting in an increase of electron transport, cell respiration, oxygen consumption and ATP production. Meanwhile, via reactive oxygen species, nitric oxide, and cyclic AMP, an enhanced mitochondrial activity initiates signaling pathways leading to the activation of several transcription factors; by regulating the expression of genes, they modulate the levels of cytokines, growth factors and inflammatory mediators [47]. Beside these anti-inflammatory mechanisms, direct effects on somatosensory and/or motor nerves could participate to LPT-induced muscle relaxation and analgesia by the neural blockade of nociceptors and motor nerves inhibition [48, 49].

It has been stated that one of the major factors responsible for LPT negative outcomes in the treatment of musculoskeletal disorders is an inappropriate irradiation dose [48]. Indeed, to generate favorable clinical outcomes, the LPT has to induce biomodulation in an appropriate volume of the target tissue. Fortunately, most of the masticatory muscles' surface is accessible to LPT. Due to its tissue penetration depth of several centimeters, a laser beam with a wavelength of 810 nm appears to be appropriate for the LPT in acute posttraumatic trismus. So, the present study proposes a LPT protocol using a unique wavelength. However, the major point of this protocol is the irradiation area, which has to cover at the same time all the pathologically-involved tissues, namely TMJs, masticatory muscles (often bilaterally involved), and their afferent/efferent innervation.

To our knowledge, no other study on LPT in TMDs described such large irradiated areas. So far, it is generally considered enough to irradiate just a few points. Together with the treatment moment (very early after the trauma), the large irradiated areas, both cutaneous and mucous, could make a major difference in the clinical outcomes. Our opinion is supported by studies on muscular pre-conditioning with LPT, in which the distribution of the energy applied on muscles, so as to cover the largest area, appears to be an important parameter [50]. On the other hand, the best LPT effectiveness on nerves appears to be due to an additive effect caused by the irradiation at several points rather than to a single point [51]. Such effects are most likely obtained by scanning a large surface, if not the whole volume of a muscle. However, in order to avoid the unpleasant feeling of skin/mucosa overheating that the patient could experience, the defocused mode (2 to 4 mm) and the scanning speed (2 cm/s) need to be thoroughly respected.

To confirm these very encouraging outcomes and their stability, as well as the effectiveness of this LPT protocol to prevent the developing of chronic posttraumatic TMDs, further clinical studies are needed.

Conclusion

Our preliminary results demonstrated the perfect ability of LPT to resolve the mouth opening limitation in acute posttraumatic trismus with a history of less than 20 hours. Consequently, we propose a new protocol, to be applied as a primary care alone: an 810-nm laser beam of 1 W has to scan twice in one session, as soon as possible after the trauma, in a defocused mode at a speed of 2 cm/s, large cutaneous and mucous areas, in order to irradiate as much as possible of the involved structures, i.e. TMJ, masticatory muscles and their respective innervation (bilaterally if necessary).

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