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Myofascial Trigger Points in Patients with Whiplash-Associated Disorders and Mechanical Neck Pain

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Abstract

Objective. The aim of this study was to investigate pain patterns and the distribution of myofascial trigger points (MTPs) in whiplash-associated disorders (WADs II and III) as compared with mechanical neck pain (MNP).

Methods. Manual examination of suboccipital, upper trapezius, elevator scapula, temporalis, supraspinatus, infraspinatus, deltoid, and sternocleidomastoid muscles, was done to search for the presence of both active or latent MTPs in 49 WAD patients and 56 MNP patients. Local pain and referred pain from each active MTP was recorded on an anatomical map.

Results. The mean number of active MTPs was significantly greater in the WAD group (6.71 ± 0.79) than in the MNP group (3.26 ± 0.33) ($P < 0.001$), but this was not found for the latent MTPs (3.95 ± 0.57 vs 2.82 ± 0.34; $P > 0.05$). In the WAD group, the current pain intensity (visual analogue scale) of the patients was significantly correlated with the number of active MTPs ($r_s = 0.03$, $P = 0.03$) and the spontaneous pain area ($r_s = 0.25$, $P = 0.07$), and the number of active MTPs was significantly correlated with the spontaneous pain area ($r_s = 0.3$, $P = 0.03$). In the MNP group, significant correlation was found only between pain duration and spontaneous pain area ($r_s = 0.29$, $P = 0.02$).

Conclusions. Active MTPs are more prominent in WAD than MNP and related to current pain intensity and size of the spontaneous pain distribution in whiplash patients. This may underlie a lower degree of sensitization in MNP than in WAD.

Key Words. Myofascial Trigger Points; Whiplash; Mechanical Neck Pain; Pain

Introduction

Whiplash-associated disorders (WADs) are a common injury associated with motor vehicle accidents, affecting up to 83% of the individuals involved in rear collisions [1]. It is a significant public health problem and an important cause of disability, considering that its incidence is estimated at four per 1,000 persons in some countries, and its overall economic burden has been evaluated at $3.9bn annually in the United States [2] and £3.64bn per year in the United Kingdom [3], and up to 40% of those experiencing whiplash injury develop persistent pain [4,5]. Approximately 50% of the patients report other whiplash-related symptoms (e.g., headache and dizziness) up to 1 year after the motor vehicle accident [6]. The development seems to be predicted by the sensory dysfunctions (pressure pain threshold, thermal pressure threshold, brachial plexus provocation test, sympathetic vasoconstrictor reflex) which can be assessed after the accident [7].

The exact mechanisms of chronic pain following whiplash are not fully defined, but Sterling et al. found [7] that chronic pain in WAD is associated with central and peripheral sensitization which may develop as early as 1 month after injury. Sensory changes include hypersensitivity to a variety of mechanical and electrical stimulations and algesic substances [8–10]. Lowered pain thresholds in uninjured tissues have been reported in WAD subjects, and these findings are explained as the expression of an abnormal processing of nociceptive information in the brain and spinal cord [7,11,12].

In WAD, chronic pain cannot be solely explained as the consequence of an obvious anatomical defect or tissue damage [13], although the lack of macroscopically identifiable tissue damage does not rule out the presence of painful lesions [14]. Recent evidence shows that central sensitization may be influenced by peripheral sources of nociception, especially nociception from deep tissue such as zygopophyseal joint capsule, which may be damaged in whiplash and become a source of pain [14]. This is further supported by several studies with medial branches of the cervical dorsal rami blocks [15,16]. Strains of joint capsule (not tear) can nevertheless produce persistent nociceptive activity from the affected joint and induce persistent secondary changes in the activity of various cytokines and transmitters in the dorsal root ganglia and spinal cord [17].

Another important source of deep tissue nociception is active myofascial trigger points (MTPs) in the neck and shoulder muscles, which are considered to be a primary source of pain following whiplash injury [18,19] as has been shown for other widespread pain syndromes such as fibromyalgia [20]. MTPs may perpetuate lowered pain thresholds in uninjured tissues far away from their localization [21] and are one of the most important peripheral pain generators and initiators for central sensitization [22]. Consequently, by treating MTPs, the dysfunctional process of the nervous system may be mitigated leading to clinical improvement [23]..

MTPs can be classified as active (responsible for spontaneous pain of patients; local and referred) or latent (responsible for a pain with which the patient is not familiar).

Latent MTPs are commonly found in healthy persons [24] and can quickly be transformed into active MTPs under the influence of perpetuating factors [25].

However, both active and latent MTPs may provoke muscle imbalance, abnormal motor recruitment, and weakness [26].

The existence of MTPs may predispose the muscle to further damage and cause an accelerated development of muscle fatigue [27].

Several studies have demonstrated that active MTPs are contributing significantly to different pain syndromes, such as chronic tension-type headache [28], episodic tension-type headache [29,30], lateral epicondylalgia [31], migraine [32,33], shoulder pain [34,35], and fibromyalgia [20,36].

Active MTPs are claimed to play an important role in the genesis of mechanical neck pain (MNP) [37]. Most of the symptoms in whiplash (neck pain and stiffness, headache, shoulder pain, arm pain or numbness, paresthesia, weakness, dysphagia, dizziness, and concentration difficulties) can also be found in patients with MNP [38].

Ettlin et al. found that WAD patients have a significantly higher prevalence of the distribution of MTPs in the semispinalis capitis than MNP patients [39].

It has already been found that acute WAD patients present a correlation between the number of MTPs and intensity of pain [40].

It has been found that WAD patients present with more active MTPs in the neck–shoulder muscles than healthy people, but the number of latent MTPs is the same [41].

However, it is still unknown whether active MTPs could be at least in part responsible for pain-related symptoms (e.g., headache and dizziness) in WAD patients, though active MTPs have been found in these patients and contribute to WAD pain.

To our knowledge, there are no other studies in the literature analyzing the distribution of MTPs in different muscles between MNP and WAD patients.

Thus, the aims of this study are to 1) compare the MTP distribution and the pain pattern between WAD and MNP; 2) investigate the relationship between MTPs and current pain intensity (visual analogue scale [VAS]) in both groups; and 3) investigate the relationship between MTPs and spontaneous pain area in both groups.

Material and Methods

Subjects

Forty-nine WAD patients (mean age = 41.6 ± 1, 72 years, 21 men, 28 women) and 56 subjects (mean age = 45.2 ± 1, 81 years, 23 men, 33 women) with MNP (never had a trauma to the neck, just complaining of symptoms in the neck–head area) participated in this study; all patients were recruited at Poliambulatorio Dalla Rosa Prati, Parma (Italy).

WAD patients were included in the study if they were first screened by a physician and if they met the Quebec Task Force Classification of WAD II or WAD III [42].

MNP patients were included in the study if they presented with neck pain for at least 3 months without any trauma.

For the purpose of this study, MNP was defined as generalized neck pain and/or shoulder pain with mechanical characteristics including symptoms provoked by maintained neck posture or by movement, or by palpation of cervical muscles [37]. Patients from both groups were excluded from the study if they, assessed by a physician, exhibited one of the following: 1) previous history of neck surgery; 2) any therapeutic intervention for myofascial pain in the last 3 months; 3) any "red flags" (e.g., infections, malignancy, fracture, rheumatoid arthritis, osteoporosis); and 4) diagnosis of fibromyalgia syndrome according to the American College of Rheumatology [43].

Informed consent was obtained from all participants according to the Declaration of Helsinki, and the study was performed in Poliambulatorio Dalla Rosa Prati, Parma, Italy. The protocol was approved by the local Ethical Committee.

Procedure

Patient demographic characteristics (age, gender, weight, height, pain duration, history of whiplash) were collected. Then, each patient was given a body chart in order to establish their spontaneous pain area. A VAS was used to record the current pain intensity on a scale ranging from 0 ("no pain") to 10 ("worst pain imaginable").

Then each subject was seated, and MTPs (both active and latent) were explored in the suboccipital, upper trapezius, elevator scapula, temporalis, supraspinatus, infraspinatus, and sternocleidomastoid muscles bilaterally. MTPs were identified by an assessor who had more than 3 years of experience with MTPs and who was blinded to the condition of the subject (whiplash or MNP).

It was decided to explore these muscles because their pain pattern is similar to the pain that many MNP and WAD patients present. Furthermore, they were easy to access with palpation; no further muscles were included because of the lack of time for the evaluation.

Semispinalis capitis has already been identified by Ettlin et al. [39] as a muscle that presents more active MTPs in WAD patients compared with nontraumatic chronic neck pain.

The MTP diagnosis was made according to the criteria described by Simons et al. [44] and Gerwin et al. [45]: presence of a palpable taut band in a skeletal muscle, presence of a hypersensitive tender spot within the taut band, local twitch response elicited by the snapping palpation of the taut band, and reproduction of typical referred pain pattern of the MTPs in response to compression. These four criteria, when applied by an experienced assessor, have obtained a good interexaminer reliability [45].

When a tender spot in a taut band was found with manual examination, a constant pressure of approximately 4 kg/cm^2 was maintained for 10 seconds [46]. After releasing the pressure, the patients were asked if this pressure-evoked pain was recognized as familiar or not. If the evoked pain was familiar to the patient, the MTP was considered to be active; otherwise, it was considered to be latent.

Before searching for another MTP, a break was taken to wait for the previously evoked pain to disappear.

Statistical Analysis

The data were analyzed with the software SIGMASTAT 3.5 (Systat, San Jose, CA, USA), and the results were expressed as mean ± standard error of the mean (mean ± SEM). The Shapiro–Wilks test was used to analyze the normal distribution of the data ($P > 0.05$). Quantitative data without a normal distribution were analyzed with nonparametric tests, whereas data with normal distribution were analyzed with parametric tests.

VISTAMETRIX software (SkillCrest, LLC, Tucson, AZ, USA) was used to have a numeric value of the pain area pictured on the map by the patients. Differences in the number of either active or latent MTPs between both study groups were assessed with the Mann–Whitney Rank Sum Test (data without a normal distribution). The chi-squared test was used to assess the differences in the distribution of either active or latent MTPs within each muscle between both study groups. Spearman's correlation (data without a normal distribution) test was used to calculate the correlation among VAS, pain duration, the number of active MTPs, spontaneous pain area, and age. The values in the text and tables are expressed as the mean ± SE. The statistical analysis was conducted at a 95% confidence level; a P value less than 0.05 was considered to be statistically significant.

Results

Demographic Characteristics

Demographic data of each group are given in Table 1. Values are expressed in mean ± SE.

No statistically significant difference was found between the two groups in the distribution of age ($P = 0.16$), height ($P = 0.43$), weight ($P = 0.83$), pain duration ($P = 0.16$), pain area ($P = 0.05$), and VAS ($P = 0.13$) (Table 1).

Although pain duration difference was not statistically significant, MNP patients were mainly chronic as the mean duration of their symptoms was 109.37 months.

The Anatomical Distribution and Pain Pattern of MTPs in WAD

The WAD group showed 6.71 ± 0.79 active MTPs and 3.95 ± 0.57 latent MTPs, and the MNP group showed 3.26 ± 0.33 active MTPs and 2.82 ± 0.34 latent MTPs. The distribution of active MTPs between both groups showed a statistically significant difference ($P < 0.001$),

Table 1 Demographic characteristics of patients

	WAD (N = 49)	MNP (N = 56)	*P* value
Gender (female/male)	28/21	33/23	—
Age (years)	41.61 ± 1.73	45.23 ± 1.81	0.16
Height (cm)	168.5 ± 1.24	170.5 ± 1.45	0.43
Weight (kg)	66.8 ± 2.2	66.9 ± 1.40	0.83
Pain duration (months)	57.12 ± 14.11	109.37 ± 20.89	0.16
Pain area	4521.77 ± 326.72	3,506.71 ± 341.84	0.05
VAS	5.59 ± 0.42	4.69 ± 0.37	0.13

MNP = mechanical neck pain; WAD = whiplash-associated disorder; VAS, visual analogue scale.

but this was not found for latent MTPs ($P = 0.16$). Furthermore, there was a statistically significant difference ($P < 0.001$) in the distribution of total MTPs between the two groups (Table 2). Differences in the distribution of active MTPs in each muscle between both study groups were significant for all muscles except for the left upper trapezius, the left elevator scapula, the left temporalis, and the right deltoid muscles. The difference in the distribution of latent MTPs in each muscle between both study groups was not significant except for the left sternocleidomastoid muscle (Table 3).

Table 2 Distribution of MTPs between both groups

	WAD (N = 49)	MNP (N = 56)	*P* value
Active MTPs	329 (6.71)	183 (3.26)	<0.001
Latent MTPs	194 (3.95)	158 (2.82)	0.16
Total MTPs	523 (5.33)	341 (3.04)	<0.001

MNP = mechanical neck pain; MTP = myofascial trigger point; WAD = whiplash-associated disorder.
Numbers in parentheses are MTP/patient.

The WAD group showed statistically significant correlations between VAS and the number of active MTPs ($r_s = 0.3$, $P = 0.03$), and between the spontaneous pain area and the number of active MTPs ($r_s = 0.3$, $P = 0.03$) (Table 4).

In the WAD group, the pain duration was not significantly correlated with the VAS ($r_s = 0.005$, $P = 0.97$), with the number of active MTPs ($r_s = 0.03$, $P = 0.83$), or with the spontaneous pain area ($r_s = 0.1$, $P = 0.46$). Furthermore,

Table 3 Distribution of MTPs in each examined muscle

	WAD Group		MNP Group		*P* Value	
	Active	Latent	Active	Latent	Active	Latent
Suboccipital left	28	16	20	13	**0.03**	0.28
Suboccipital right	38	14	21	12	**0.01**	0.4
Upper trapezius left	20	26	15	22	0.13	0.16
Upper trapezius right	37	17	20	21	**0.01**	0.77
Sternocleidomastoid left	21	13	13	6	**0.03**	**0.04**
Sternocleidomastoid right	22	15	13	9	**0.02**	0.07
Elevator scapula left	15	12	10	6	0.13	0.06
Elevator scapula right	15	11	8	7	**0.04**	0.18
Temporalis left	16	9	13	11	0.28	0.87
Temporalis right	24	8	15	12	**0.02**	0.51
Sovraspinatus left	14	6	4	8	**0.01**	0.76
Sovraspinatus right	15	11	7	9	**0.02**	0.41
Infraspinatus left	17	7	6	5	**0.01**	0.39
Infraspinatus right	18	11	6	6	**0.01**	0.1
Deltoid left	15	8	4	6	**0.01**	0.4
Deltoid right	14	10	8	5	0.07	0.09
Total	329	194	183	158		
Total (active + latent)	523		341			

MNP = mechanical neck pain; MTP = myofascial trigger point; WAD = whiplash-associated disorder.
The numbers in bold indicate significant differences between groups (P<0.05).

Table 4 Correlations

	WAD (N = 49) *P* Value	MNP (N = 56) *P* Value
VAS/pain duration	0.97	0.83
VAS/number of active MTPs	**0.03**	0.33
VAS/pain area	0.07	0.11
Age/number of active MTPs	0.18	0.57
Pain area/number of active MTPs	**0.03**	0.32
Pain duration/number of active MTPs	0.83	0.84
Pain duration/pain area	0.46	**0.02**

MNP = mechanical neck pain; MTP = myofascial trigger point; WAD = whiplash-associated disorder; VAS, visual analogue scale.
The numbers in bold indicate significant differences between groups (P<0.05).

no significant correlation was found between age and the number of active MTPs ($r_s = –0.19$, $P = 0.18$) or between VAS and spontaneous pain area ($r_s = 0.25$, $P = 0.07$).

In the MNP group, VAS was not significantly correlated with pain duration ($r_s = 0.02$, $P = 0.83$), the number of active MTPs ($r_s = 0.13$, $P = 0.33$), or spontaneous pain area ($r_s = 0.21$, $P = 0.11$). Furthermore, the number of active MTPs was not significantly correlated with age ($r_s = –0.07$, $P = 0.57$), pain area ($r_s = 0.13$, $P = 0.32$), or pain duration ($r_s = 0.02$, $P = 0.84$).

The significant correlation was between pain duration and pain area ($r_s = 0.29$, $P = 0.02$) in the MNP group.

Discussion

The results of this study show that WAD patients have more active MTPs in the neck–shoulder muscles than MNP patients, and these findings are generalized to the majority of the examined muscles.

In WAD, the number of active MTPs is correlated to the current level of pain intensity (VAS) and to the spontaneous pain area, whereas this was not found for MNP patients. These findings suggest that active MTPs may contribute to the pain symptoms in WAD, which is in accordance with the results from other studies [18,19].

Spatial summation of MTP pain reproducing spontaneous pain pattern in WAD is also found in other musculoskeletal disorders like chronic tension-type headache and shoulder impingement [28,35].

In addition, a mean number of 3.2 active MTPs in MNP patients was found in the current study, which is similar to the results in a previous study [37] in which MNP patients exhibited a mean of 1.8 active MTPs (however, the previous study examined four muscles, whereas the present study examined eight muscles). Although active MTPs are more present in MNP patients than in healthy subjects, active MTPs are less prevalent in MNP than in WAD patients.

In contrast to the differences in the number of active MTPs, the number of latent MTPs was found to be similar in the two groups. This indicates that the active, but not the latent, MTPs may contribute to the pain induction in WAD.

Other studies found that the distribution of latent MTPs in neck–shoulder muscles is similar between healthy subjects and WAD patients [41], and between healthy subjects and MNP patients [47], confirming that they do not contribute to spontaneous pain of patients [24].

Central sensitization is a well-described phenomenon in WAD [48]. Several studies have proposed active MTPs to be important peripheral pain generators and initiators for central sensitization acting as one peripheral source of nociception [21,22]. The phenomenon of central sensitization affects how the spinal neural circuits respond to different stimuli, leading to lowered pain thresholds in uninjured and injured parts of the body [22]. It has been reported that central pain mechanisms are more evident in WAD as compared with MNP [49]. Hypersensitivity exists in both injured and uninjured parts of the body in chronic WAD, whereas hypersensitivity is found only in injured parts (the neck) in MNP subjects when compared with healthy controls [49,50].

The smaller number of active MTPs in MNP patients found in the current study, as compared with WAD, may underlie a lower degree of central sensitization in MNP than in WAD.

The persistence of pain in WAD patients may be at least in part related to the dysfunctions induced by the trauma (e.g., deep neck flexor weakness and hyperactivity of superficial muscles, forward head posture); it has been studied that they do not resolve spontaneously [51] even when the acute pain following whiplash resolve inducing a vicious circle of chronic pain that cannot be eliminated until the original dysfunction is corrected.

MTPs may develop easily when this dysfunction persists, inducing overuse of certain muscles (e.g., sternocleidomastoid, upper trapezius, suboccipital muscles) that can now easily develop MTPs.

Furthermore, WAD patients present a high number of MTPs resulting in constant sources of nociception in deep tissues allowing spatial summation and temporal summation phenomenon, which may explain the correlation found between VAS and the number of active MTPs and between the pain area and the number of active MTPs.

Pain duration is not related to the number of active MTPs. Therefore, the persistence of pain in WAD patients can be rationally explained by the dysfunctions usually presented in these patients, but these dysfunctions were not assessed so this could just be an idea of explaining this finding arising from the literature.

The high number of active MTPs in WAD patients could also at least in part explain why these patients often present with symptoms like headache, dizziness, and tinnitus, which may all be related to active MTPs in neck muscles (specially sternocleidomastoid, suboccipital, upper trapezius) that may produce symptoms to the head.

Supplementary studies may further investigate the effects of elimination of active MTPs on sensitization mechanisms in subjects with MNP as compared with WAD.

The association between active MTPs and central sensitization has been found also in a recent study on whiplash [3] and in other local pain syndromes, such as low back pain [13], migraine [32,33], lateral epicondylalgia [31], tension-type headache [29,30], knee osteoarthritis [52], and carpal tunnel syndrome [53]. The results of those studies and of the current study are clinically relevant in that an early inactivation of MTPs in WAD might in part avoid and/or delay the development of chronic pain and central sensitization.

There could be a number of limitations to this study. First of all, it is impossible to state a cause–effect relationship between MTPs and WAD because no treatment was applied to check if the pain area and the pain intensity were reduced with the inactivation of the MTPs.

In fact, if MTPs are considered at least partially to contribute to the symptoms of WAD patients, by removing them, symptoms would be expected to decrease.

Further research is needed to investigate the effect of MTP inactivation on WAD and MNP symptoms. Both situations often become chronic and still present many MTPs. Therefore, removing the deep and constant source of nociception (MTPs) could be an important stage in the managing of these pain situations.

Analyzing the reactions to manual therapy treatment directed to MTPs, inactivation, and normalizing neck dysfunctions would be interesting to understand which one of the two patient group reacts better to this treatment in order to have a real evaluation of the role of the MTPs in the symptoms presented by these patients and this is the current study.

Patients were asked to state their “current pain intensity,” and this could be confusing (many patients reported that they had a different VAS the day before); asking them about the “average pain over the past week” would have given a more clear idea about their pain condition.

Another limitation could be that there was no direct access to all WAD and MNP patients presenting to the clinic because some of them had first been sent to other types of therapies and therefore had to be excluded.

Finally, all patients were recruited from the same clinic where the researchers had access to patients searching for a visit because of their neck problems in this clinic.

Conclusions

WAD patients have more active MTPs, greater pain intensity (VAS), and a larger spontaneous pain area than MNP patients, suggesting a lower degree of sensitization in MNP as compared with WAD.

These findings suggest that managing MTPs in WAD may be a way to reduce pain and sensitization.

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