Myofascial Trigger Point Syndrome Effect On Chronic Tension-Type Headache Disorder

A Literature Review

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Fall, 2008

ABSTRACT

Objective: The purpose of the present literature review is to investigate the degree of involvement of Myofascial Pain Syndrome (MPS) in the development of chronic tension type headaches (CTTH). Emphasis is given to the involvement of trigger points (TrPs), central sensitization, and chronification of the headache disorder. This paper describes the presence of referred pain from TrPs in the suboccipital muscles, upper trapezius, sternocleidomastoids, and temporalis muscles.

Data Collection: A computer search using PubMed and MANTIS generated articles relevant to tension-type headaches (TTH), myofascial trigger points, chronic tension-type headache, central sensitization, and myofascial pain syndromes.

Data Synthesis: Chronic pain is a complex process which involves trigger points inducing referred pain pattern in tension headache patients.

Conclusions: A consensus of over two thousand articles consisting of selective reviews and controlled studies, in addition to several noteworthy text books, supports the concept that myofascial pain syndromes contribute to, or cause, recurrent and chronic headaches. Referred myofascial pain must be considered when evaluating patients with a number of different headache problems including cervicogenic headaches, occipital neuralgia, chronic tension-type headaches with pericranial tenderness, head pain associated with temporomandibular disorders, and migraine.

Keywords: Tension-type headache; Chronic tension-type headache; Myofascial trigger points; Suboccipital muscles; Referred pain; Headache; Diffuse noxious inhibitory controls; Myofascial pain syndrome; Central sensitization; Pressure-pain thresholds.

INTRODUCTION

Headaches are classified into two categories: primary and secondary. Primary headaches have no apparent underlying disease process and account for about 90% of all headaches, they include: migraine, tension-type, and cluster headaches. Secondary headaches, on the other hand, are caused by an underlying organic disease such as: cerebrovascular disease, head trauma, infection, tumor, and several other factors (1).

Tension-type headache (TTH) is one of the most prevalent primary headaches in adults. Population based studies suggest 1-year prevalence rates of 38.3% for episodic (TTH) and 2.2% for chronic TTH (2). It has been reported that increased excitability of the central nervous system generated by repetitive and sustained pericranial myofascial nociception may be responsible for transformation of episodic tension-type headache into chronic form (3). Despite some advances, the pathogenesis of TTH is still not clearly understood. However, since the publication of the International Headache Classification (4), an increasing number of well-performed studies have been published. Myofascial tissues have been extensively studied, and it has been demonstrated that increased tenderness to palpation of pericranial myofascial tissues is the most apparent abnormality in patients with tension-type headache (5-7). Research suggests several possible mechanisms leading to myofascial pain and tenderness, these include: (i) sensitization of peripheral myofascial nociceptors, (ii) sensitization of second order neurons at the level of the spinal dorsal horn/trigeminal nucleus, (iii) sensitization of supraspinal neurons, and (iv) decreased anti-nociceptive activity from supraspinal structures (7-10). This increased knowledge of central sensitization, i.e. increased excitability of neurons in the central nervous system, has been a major breakthrough in the understanding of chronic pain (11).

Myofascial pain syndromes are common conditions that, by definition, result from small, hyperalgesic trigger points (TrPs) which refer pain to distant sites. These TrPs cause persistent pain that can be intense and often disabling (12, 13). Patients with myofascial problems caused by TrPs typically complain of protracted, dull, deep, diffuse aching pain or soreness in a specific muscle or muscle group (14). TrPs can be divided into active and latent. Active TrPs are responsible for spontaneous pain. Latent TrPs do not cause a clinical complaint of pain, but are diagnosed when manual palpation demonstrates a hypersensitive muscle area from which pain is referred elsewhere in response to pressure (12, 14). Fibromyalgia is the closest disease to resemble myofascial pain syndrome in terms of chronic muscle pain conditions. However, TrPs play an important role in differentiating between the two disorders. Patients with fibromyalgia tend to develop tender points that result in local, but not referred pain. In myofascial pain syndrome, pain is referred from irritable TrPs in muscle or its associated fascia to other locations where it is perceived (12).

The aim of the present review was to investigate the pathophysiology of chronic tension-type headache (CTTH) with special reference to central mechanisms. It is believed that the main problem in CTTH is central sensitization at the level of the spinal dorsal horn/trigeminal nucleus due to prolonged nociceptive inputs from pericranial myofascial tissues. The central neuroplastic changes may affect the regulation of peripheral mechanisms and thereby lead to, for example, increased pericranial muscle activity or release of neurotransmitters in the myofascial tissues. By such mechanisms, the central sensitization may be maintained even after the initial eliciting factors have been normalized, resulting in the conversion of episodic into chronic TTH (7, 15).

DISCUSSION

Tension-type headache (TTH) is a prototypical headache in which pericranial myofascial tissues can play an important role. Some authors have claimed that pain from pericranial head, neck, and/or shoulder muscles are referred to the head, and are experienced as headache (16). The International Headache Society (IHS) defines TTH more precisely and differentiates between the episodic and the chronic types. The following is a modified outline of the IHS diagnostic criteria:

Episodic tension-type headache (ETTH): At least 10 previous headaches in less than 15 days per month. Headaches last from 30 minutes to 7 days and have at least 2 of the following pain characteristics: 1) Pressing/tightening (non-pulsating) quality; 2) Mild or moderate intensity (may inhibit activities); 3) Bilateral location; 4) No aggravation from climbing stairs or similar physical activity. Nausea or vomiting should not be present(17).

Chronic tension-type headache (CTTH): Average headache frequency of more than 15 days per month for more than 6 months fulfilling the following criteria: Bilateral tenderness most marked in the upper trapezius, the posterior cervical, suboccipital, and sternocleidomastoid muscles (17). The degree of tenderness is highly correlated with the frequency of tension-type headaches, although less so with the severity of headache (18). At least 2 of the pain characteristics mentioned in ETTH should be present as well (17).

Headaches of cervical origin, chronic tension-type headaches with pericranial tenderness, head pain associated with temporomandibular disorders, migraine, and occipital neuralgia are among the headache problems putatively linked to trigger points (TrPs). There is no reliable one-to-one correspondence between the presence of TrPs and

any of these entities; instead, TrPs may be involved as an element contributing to the headache, or as a precipitating process. Because central changes in the processing and modulation of nociceptive stimuli from muscle may often interact with peripheral myofascial nociceptive mechanisms (19, 20), TrPs should be considered as possible factors in producing or triggering pain in any of the above-mentioned headaches (12).

Trigger points (TrPs) can be divided into active and latent. Active TrPs are responsible for spontaneous pain. Latent TrPs do not cause a clinical complaint of pain, but are diagnosed when manual palpation demonstrates a hypersensitive muscle area from which pain is referred elsewhere in response to pressure (21). Active trigger points are localized, 2-5 mm, hypersensitive areas in the skeletal muscle. They can occur in any muscle in the body, but are most often in the head, neck, shoulders, and lower back. Typically, TrPs are found in regions of muscle that are firm to palpation (22). Muscles containing TrPs may be stiff and have a limited range of movement (13). Because referred myofascial pain is often exacerbated after active contraction, patients attempt to diminish pain by modifying their posture. Consequently, affected muscles may shorten. Pressure sufficient to stimulate a TrP intensifies or mimics the pain complaints of the patient, eliciting patterns of pain referral that are not only reproducible, but are uniform among patients who have TrPs in corresponding locations (14, 23). The referred pain may be perceived immediately after TrPs palpation, or perception maybe delayed for seconds. Most TrPs refer pain in a distribution that neither follows, nor is limited to a segmental or peripheral nerve distribution. Nor do the patterns of pain referral usually follow dermatomal, myotomal, or sclerotomal patterns (24). Autonomic phenomena occasionally occur upon TrP stimulation. In particular, the pain referred from the

sternocleidomastoids may be accompanied by ipsilateral lacrimation and reddening of the conjunctiva (14). Trigger points are not just noteworthy for their capacity to refer pain to distant sites. They are impressively tender. Almost all patients with TrPs demonstrate a jump sign, which is a behavioral response to pressure on a trigger point (12).

Tender points can be distinguished from trigger points. Tender points, common in fibromyalgia, are discrete areas of tenderness over muscle, bone, tendon, and fat that cause local pain and are tender to palpation. Patients do not jump when tender points are palpated, also tender points do not refer pain to nearby or distant locations (12-14).

Headaches caused by pain referred from trigger points: It has been argued that tender areas in neck and shoulder muscles may not be causal, but result from the headache (25). Many of these tender areas, however, may in fact be trigger points (TrPs). Both between and during attacks, active TrPs can frequently be found in the cervical and shoulder musculature of patients whose putative diagnosis is headache caused by cervical pathology (26, 27). This causal relationship is supported by findings that between attacks, palpation of TrPs in the cervical and shoulder muscles can transiently reproduce or precipitate headache (12, 28, 29). Stimulation of TrPs during an attack exacerbates or intensifies the headache, and inactivation of TrPs can eliminate such headaches (30). Noxious stimulation of muscle nerve endings causes pain characteristically referred to distant, deep somatic structures such as muscles, fascia, tendons, joints, and ligaments. In an experiment, hypertonic saline injections into skeletal muscle and interspinous ligaments of human subjects induced firing in a large proportion of A-delta and C-fibers and caused distinctive, deep, aching, referred pain comparable to that perceived by patients during activation of trigger points (12, 44).

Myofascial pain from trigger points in muscles receiving their sensory innervations from C1 to C3 (e.g., the small cervico-occipital muscles, the semispinalis capitis, the splenius capitus, the splenius cervicis, the trapezius, and the sternocleidomastoid muscles) can be referred to various regions of the head (14, 31). Trigger points in the trapezius muscle refer head pain to the temple, the neck, and the orbital and periorbital regions. In the sternocleidomastoid muscle, they can cause pain in the fronto-temporal region, the occiput, the vertex, the forehead, and the orbit. From the splenius capitis and splenius cervicis, pain is often referred to the vertex of the head on the same side, behind the eye, and the occiput. Cervico-occipital muscles project pain to the occiput, the eye, and the forehead. Experimental stimulation of muscles innervated by C1 to C3 induces headaches in normal individuals (32). In contrast, TrPs in muscles innervated by lower cervical nerves do not directly cause headaches (12).

Head pain associated with temporomandibular disorders: Headache has been reported as the most common symptom in patients with temporomandibular joint (TMJ) problems (33), usually tension-type, but occasionally a combination of migraine and tension-type headaches. They are thought to originate in the masticatory muscles for two reasons: (I) trigger points (TrPs) are frequently found in masticatory muscles, and (II) patients' headache symptoms lessen when treatment is focused on the musculature (e.g., injection of TrPs in masticatory muscles with local anesthetics can produce reduction of headache) (12, 33, 34). TrPs in masticatory muscles are usually caused by malocclusion, misalignment of the jaws, habitual parafunction of the jaws (clenching, bruxism, jaw posturing), abnormal head and neck postures, or trauma (35). A study by Fernandez-de-las-penas et al. (35) provides for the first time the evidence that the local and referred

pain and pain characteristics elicited by manual examination of TrPs in the temporalis muscle share similar patterns as habitual headache pain in CTTH patients. They also found that headache duration and intensity were greater in CTTH patients who had active TrPs compared to those with latent TrPs in the temporalis muscle. Arendt-Nielsen et al. (63) add that peripheral and central sensitization and decreased descending inhibition induced by longterm nociceptive stimuli from TrPs may also involve in referred pain to trigeminal region from active TrPs in the temporalis muscle.

Muscle Atrophy in the Rectus Capitis Posterior Minor and its relation to CTTH: A recent pilot study conducted by Fernandez-de-las-penas et al. investigated whether cross-sectional area (CSA) of the suboccipital muscles was associated with active trigger points (TrPs) in Chronic tension-type headache (CTTH). They used magnetic resonance imaging (MRI) on the cervical spine of 11 females with CTTH aged from 26 to 50 years old. CSA for both rectus capitis posterior minor (RCPmin) and rectus capitis posterior major (RCPmaj) muscles were measured from axial T1-weighted images, using axial MRI slices aligned parallel to C2/3 intervertebral disc. Trigger points in the suboccipital muscles were identified by eliciting referred pain to palpation, and increased referred pain with muscle contraction. TrPs were considered active if the elicited referred pain reproduced the head pain pattern and features of the pattern seen during spontaneous headache attacks. Their conclusion was that muscle atrophy in the RCPmin, but not in the RCPmaj, was associated with suboccipital active TrPs in CTTH patients (59). It is known that nociceptive inputs in active TrPs could lead to greater atrophy of the involved muscles. In that way, the nociceptive signals generated by active TrPs in the suboccipital muscles may reduce muscle function, and this can contribute to disuse muscle atrophy,

perpetuating the pain cycle (59). Muscle atrophy could act as a perpetuating factor for chronic pain, because it could account for a reduction of proprioceptive output of the affected muscles, which may conceivably facilitate the transmission of impulses from a wide dynamic range nociceptors. Several studies in the past investigated muscle atrophy in patients with low back pain and found that these patients have atrophy of specific muscles, particularly the multifidus muscles (60). This finding suggested that specific muscle atrophy could be related to protective mechanisms, where patients tend to restrict movement of those muscles from areas involved in pain. Since the suboccipital region where rectus capitis posterior minor (RCPmin) is located, is one of the painful areas during headaches, atrophy of the RCPmin could be caused by avoidance behavior of the CTTH patients (59). On the other hand, it has been found that patients with CTTH had a greater head forward posture than did healthy individuals (61). Forward head posture can result in shortening of the posterior cervical muscles that extend the neck, particularly the RCPmin muscle. The limitation of muscle function in this biomechanical situation could be contributing to muscle atrophy of the RCPmin (59). Hack et al. (62) found that RCPmin has a greater muscle spindle concentration (36 spindles/gram) than does RCPmaj (30.5 spindles/gram). According to Fernandez et al. (59), It is possible that muscles with greater concentration of muscle spindles might be more sensitive to muscle atrophy in painful conditions such as CTTH.

Central sensitization in chronic tension-type headache: It has been postulated that increased pericranial tenderness and decreased pressure pain threshold levels in chronic tension-type headache (CTTH) patients may be due to an increased sensitivity or hyperexcitability in the central nervous system or in the periphery (12). Bendtsen et al.

(7) established a pain model in which the main problem in CTTH is the sensitization of central pathways due to prolonged nociceptive inputs, possibly provoked by the liberation of algogenic substances (e.g., bradykinin, serotonin, substance P) at the periphery, from pericranial myofascial tender tissues. The presence of prolonged peripheral inputs can be a mechanism of major importance for the conversion of episodic into chronic tension-type headache (7, 15). The spinal dorsal horn neurons that receive inputs from myofascial tissues can be classified as high-threshold mechano-sensitive (HTM) neurons and as low-threshold mechano-sensitive (LTM) neurons. The HTM neurons require noxious intensities of stimulation for activation, whereas, the LTM are activated by innocuous stimuli (15, 36). It is known that chemical mediators may sensitize the nociceptive nerve endings. Particularly effective stimulants for skeletal muscle nociceptors are endogenous substances such as bradykinin or serotonin (36). Several studies have found that sensitization of nociceptive nerve endings is greater with the combination of both substances rather than with each substance alone (37, 38). Therefore, muscle pain is produced mainly by noxious stimuli that lead to increased synthesis and release of endogenous algogenic substances such as serotonin, bradykinin, histamine or prostaglandins. Such stimuli may cause the release of neuropeptides from the nerve endings of C fibers which contain neuropeptides such as calcitonin gene-related peptide, substance P or neurokinin A. The liberation of algogenic substances would lower tissue pH and then activate the arachidonic acid cascade that produces a number of unsaturated lipid products (39, 40). Previous studies have confirmed the presence of sensitization of peripheral muscle nociceptors in both chronic and episodic tension-type headache (7, 41). Finally, other inflammatory chemicals believed to be involved include bradykinin from

plasma, serotonin (5HT) from platelets and glutamate, which are known to affect the membranes of polymodal nociceptors to produce sensitization (42).

Sensitization of second-order neurons in the dorsal horn and in the trigeminal nucleus. Dorsal horn neurons that receive afferents from muscle, frequently receive input from receptive fields in more than one muscle, from other deep structures, and from skin (43). This extensive convergent input to dorsal horn neurons may account for the often diffuse and poorly localized nature of deep pain sensation in humans, particularly when pain is intense (44). The intense afferent nociceptive input from muscle may alter the dorsal horn circuitry by unmasking, or activating, previously ineffective synapses to form novel synaptic contacts between low threshold afferents and high threshold mechanosensitive dorsal horn neurons. This process increases the excitability of dorsal horn neurons that amplify all sensory inputs, including those from low threshold afferents that normally convey information about innocuous mechanical stimuli. Low threshold afferents can thus mediate prolonged nociception under certain conditions (45, 46). Moreover, myofascial afferents from several different areas converge into the same second-order nociceptive relay neurons in the spinal cord and the trigeminal nucleus caudalis. Pain referred to the head from muscle and other deep structures innervated by either the C1-C3 roots or the trigeminal nerve results from this convergence (47).

Impairment of pain inhibition in chronic tension-type headache. Evidence has been accumulated suggesting that a dysfunction in pain inhibitory systems such as in 'diffuse noxious inhibitory controls' (DNIC)-like mechanisms, might be, amongst other factors, responsible for the development of chronic tension-type headache (48). The DNIC allow that the activity of pain-signaling neurons in the spinal dorsal horn and in trigeminal

nuclei can be inhibited by noxious stimuli applied to body areas far remote from the excitatory fields of these neurons (49, 50). Functionally seen, DNIC-like mechanisms act like a barrier against the uncontrolled spread of pain and keep pain regional and bearable. In a few studies, dysfunctions of DNIC-like mechanisms have been observed in fibromyalgia patients. Pielsticker et al. (48), conducted a study on 29 patients suffering from chronic tension-type headache (CTTH) and 25 healthy control subjects to see whether patients with CTTH suffer from deficient DNIC-like pain inhibitory mechanisms in a similar manner, as do patients with anatomically generalized chronic pain like fibromyalgia. In accordance with their hypothesis on DNIC deficiencies in chronic pain, patients with CTTH showed significantly less pain inhibition both at cranial and at extra-cranial sites tested than healthy control subjects. This result suggested that the DNIC-like pain inhibitory mechanism is deficient when suffering from CTTH (48). Usually, in a normal, healthy subject, transmission of nociceptive information by trigeminal and spinal neurons to supraspinal structures is modulated and controlled by central nervous system circuits which form endogenous antinociceptive, or pain control, systems. These include systems in the periaqueductal gray matter, in the raphe nuclei, and in locus coeruleus (51). When activated by electrical stimulation, these structures can modify pain sensation and inhibit behavioral reactions evoked by noxious stimuli.

TREATMENT

Since trigger points within myofascial structures were found to refer the majority of pain to the head, many treatments aimed at eliminating those trigger points: Ischemic compression (52) maintained long enough to inactivate the muscle spasm. Depending on the trigger points sensitivity, pressure is increased until tension of the tissue containing

the trigger points eases; Spray and stretch (52); Strain and counterstrain (53); Trigger point pressure release (54); Ultrasound deep heat therapy (55); Thermotherapy (56); and needling therapies (57). All of these treatments are designed to result in myofascial release which mobilizes distorted, tight fascia, increases mobility of soft tissue structures, and restores normal biomechanical function to the muscular system. It utilizes feedback from the tissue of the patient to the practitioners hand as a therapeutic guide. Results of a study conducted by Cesar Fernandez-de-las-Penas et al. (58) suggested that ischemic compressive technique and transverse friction massage are comparably effective in reducing tenderness of myofascial trigger points. A 1995 study in the Journal of Manipulative and Physiological Therapeutics found that spinal manipulative therapy is an effective treatment for tension headaches and that those who ceased chiropractic treatment after four weeks experienced a sustained therapeutic benefit in contrast with those patients who received a commonly prescribed medication (54). On the other hand, Conventional medicine relies heavily on Amitriptyline tricyclic anti-depressant in the treatment of chronic tension-type headache and fibromyalgia. Bendtsen et al. (64) designed a study to investigate whether the analgesic effect is caused by a reduction of muscle pain or by a general reduction of pain sensitivity. Their conclusion was that amitriptyline reduces myofascial tenderness and headache in patients with CTTH by reducing the transmission of painful stimuli from the myofascial tissues rather than by reducing overall pain sensitivity. On the basis of previous and present results, they suggested that the reduction of myofascial pain exerted by amitriptyline may be caused by reduction of segmental central sensitization in combination with a peripheral anti-nociceptive action (64).

CONCLUSION

Myofascial pain syndromes are common muscular pain complaints. These syndromes contribute to, or cause, recurrent and chronic headaches because small, hyperalgesic trigger points (TrPs) located within myofascial structures distant from the painful region refer pain to the head or trigger headaches. Such TrPs are produced and maintained by processes that damage and stress muscles, resulting in sensitization of muscle proprioceptors and of the dorsal horn sensory apparatus. Excessive muscle stretch by acute trauma or injury, and chronic excessive muscle contraction as a consequence of improper or awkward postures, ergonomic factors, or repetitive muscular activity are the most frequent causes. According to a number of studies, several musculoskeletal disorders from the cranio-cervical region (temporalis muscle, suboccipital muscles, upper trapezius muscle, misalignment of upper cervical vertebrae, or superior oblique muscle) are contributing at the same time to headache pain perception in tension-type headache. When referred myofascial pain causes or contributes to headache, relief is unattainable without addressing the mechanical causes that maintain the offending trigger points. Overall, the idea is that headache pain is most likely referred from trigger points in the posterior cervical, neck, and shoulder muscles, mediated through the spinal cord and the brainstem trigeminal nucleus caudalis, rather than direct tenderness of the pericranial muscles themselves. The present review emphasizes that tension-type headache is a multifactorial disorder with several concurrent pathophysiological mechanisms. It supplements the understanding of the several different components in tension-type headache, and thereby, hopefully, leads us to a better prevention and treatment of the most prevalent type of headache.

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