Current Studies on Myofascial Pain Syndrome

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Recent studies have clarified the nature of myofascial trigger points (MTrPs). In an MTrP region, multiple hyperirritable loci can be found. The sensory components of the MTrP locus are sensitized nociceptors that are responsible for pain, referred pain, and local twitch responses. The motor components are dysfunctional endplates that are responsible for taut band formation as a result of excessive acetylcholine (ACh) leakage. The concentrations of pain- and inflammation-related substances are increased in the MTrP region. It has been hypothesized that excessive ACh release, sarcomere shortening, and release of sensitizing substances are three essential features that relate to one another in a positive feedback cycle. This MTrP circuit is the connection among spinal sensory (dorsal horn) neurons responsible for the MTrP phenomena. Recent studies suggest that measurement of biochemicals associated with pain and inflammation in the MTrP region, the sonographic study of MTrPs, and the magnetic resonance elastography for taut band image are potential tools for the diagnosis of MTrPs. Many methods have been used to treat myofascial pain, including laser therapy, shockwave therapy, and botulinum toxin type A injection.

Introduction

Myofascial pain syndrome (MPS) has been widely accepted as a clinical entity based upon recent studies on myofascial trigger points (MTrPs) [1–3]. MPS is defined as a regional pain syndrome characterized by muscle pain caused by MTrPs [1,3]. MPS may include a regional muscle pain syndrome of any soft tissue origin that is associated with muscle tender points or trigger points [4]. In skeletal muscle, a tender point can be an MTrP if it locates in the endplate zone with all characteristics of an MTrP, such as taut band, referred pain, and local twitch response (LTR) [5]. Because MPS is caused by MTrPs, this article concentrates on the research on MTrPs.

Studies on the MTrP Region

The concept of multiple sensitive loci in an MTrP region suggested by Hong and Simons [1] has been well supported by human and animal studies (Fig. 1). An MTrP locus contains a sensory component and a motor component. At the sensory locus, pain, referred pain, and LTR can be elicited when this locus is mechanically stimulated with adequate pressure. This locus has been defined as a sensitive locus or LTR locus. At the motor locus, spontaneous electrical activity ([SEA], including endplate noise [EPN] and endplate spikes) can be found in electromyographic (EMG) recordings. This motor locus has been defined as an active locus, SEA locus, or EPN locus. An LTR locus is a sensitized nociceptor (free nerve ending) [6], and an SEA locus is a dysfunctional endplate [2,7–9]. An SEA locus is in the closed vicinity of an LTR locus. They interact mutually for the formation of a taut bend. Stimulation of an LTR locus can elicit pain, referred pain (due to central sensitization), and LTR (via spinal cord reflex).

Hong et al. [6] reported that a nerve ending (nociceptor) could be frequently found at the site where LTR can be elicited. Kuan et al. [10•] has further confirmed that by injecting horseradish peroxidase into the nociceptors, subsequent spreading to the dorsal ganglia and spinal cord dorsal horn region occurred.

SEA recorded from an MTrP region is actually abnormal endplate potentials [2,8,9]. Simons [2] suggested that the occurrence of EPN indicates excessive leakage of acetylcholine (ACh) in the endplate region based on extensive reviews of old physiological literature. This was further supported by an animal study showing that EPNs recorded in the MTrP region were suppressed after the injection of botulinum toxin type A (BTX-A) [11]. The leakage of ACh molecules can cause focal contracture of sarcomeres to form a contraction knot [3]. In several studies, Simons [3,4,12] discussed an energy crisis hypothesis to explain the formation of a taut band. The author postulated that excessive ACh release, sarcomere shortening, and release of sensitizing substances are three essential

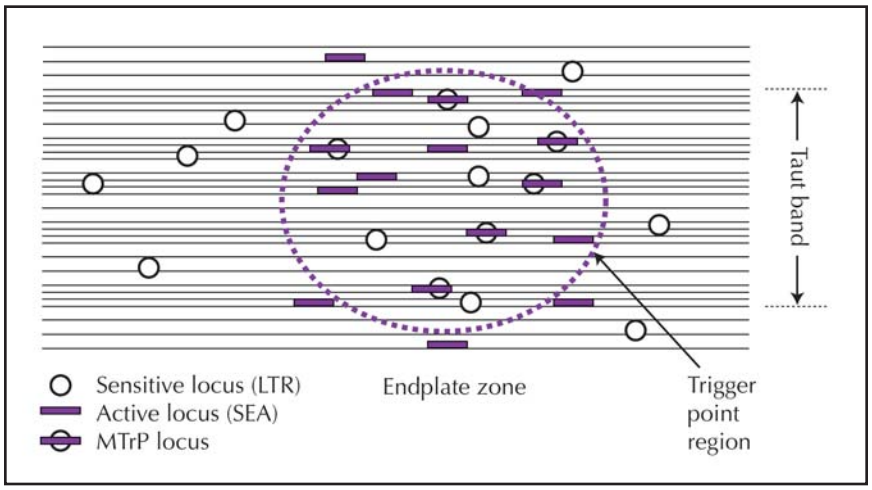

Figure 1. Multiple loci in an MTrP. LTR—local twitch response; MTrP—myofascial trigger point; SEA—spontaneous electrical activity.

features that relate to one another in a positive feedback cycle. An increased ACh release in the neuromuscular junction (motor endplate) can cause an increase of the muscle fiber tension (taut band) that contains an MTrP, and subsequently can cause energy crisis due to increased metabolism and local ischemia with hypoxia. In this situation, secretion of sensitizing substances can be increased to cause pain. The sensitizing substances can further cause abnormal ACh release to create a vicious cycle [3].

An animal study demonstrated that SEA persisted after transection of peripheral nerve and a high-level spinal cord [13]. Kuan et al. [14] also found no increase in neuromuscular jitter in the myofascial trigger spot region based on a single-fiber EMG study. It appears that the excessive leakage of ACh in the endplate is not immediately controlled by nervous system. These two findings support the hypothesis that energy crisis in the MTrP region is a focal reaction and not related to neural controls. However, in another recent single-fiber EMG study, Chang et al. [15••] found the evidence of degeneration in motor nerve endings in the MTrP region. Further study is required to clarify if any motor nerve lesion is involved in the pathogenesis of an MTrP.

In recent studies by Shah's group [16,17••] using a microanalytic technique to measure biochemicals (associated with pain and inflammation) at the MTrP region of upper trapezius muscle in subjects identified as active (neck pain and MTrP), latent (no neck pain but with MTrP), or normal (no neck pain, no MTrP) have demonstrated increases in concentrations of all analytes in active subjects compared with the latent or normal ones. These findings strongly support Simons' integrated hypothesis of energy crisis [12,18]. They also found remarkable elevation of biochemicals during the LTR. However, substance P and calcitonin gene–related peptide (CGRP) were the only two analytes for which concentrations during the recovery period after the LTR were significantly below the baseline concentrations [16]. This interesting finding could explain the immediate relief of pain (reduced substance P and CGRP) after eliciting LTRs during MTrP injection. However, the mechanism is unclear.

Recent studies have demonstrated that the irritability of an MTrP is proportionate to the prevalence [19••] and the amplitude [20] of EPN recorded from that MTrP region. The assessment of SEA (including EPN) in an MTrP region has been used for the evaluation of the effectiveness of a certain therapeutic method [11,21–24].

Studies on the Spinal Cord Mechanism

Referred pain and LTR are two important characteristics of MTrPs. Both are mediated via the spinal cord mechanism, based on recent human and animal research studies [1,4].

In animal studies on the referred pain from a muscle to other distant ones, Mense and Simons [4] demonstrated that the elicited referred pain is secondary to the unmasking of formerly ineffective synaptic connections among neurons corresponding to different receptive fields. A strong noxious stimulus can send the impulse to the corresponding dorsal horn neuron and induce it to release substance P and CGRP, which diffuse to other dorsal horn neurons and increase the efficacy of silent synaptic connections as a consequence of central sensitization in the spinal cord level [4]. In a human study, referred tenderness could be elicited not only from the active MTrP, but also from a latent MTrP region or even normal muscle tissues [25]. However, a higher pressure was required to elicit referred pain from a latent MTrP or normal muscle tissues than from an active MTrP. The pressure required to elicit referred pain from a compressed site is proportionate to the degree of irritability [5].

LTRs can be recorded electromyographically from a taut band that contains an MTrP when this MTrP is mechanically stimulated with a high pressure [26,27]. In an animal study, LTRs could also be elicited when the MTrP was mechanically stimulated, and could only be perfectly recorded in the taut band but not other sites [28]. It was further demonstrated that the LTRs could be recorded from a muscle only if the innervated nerve was intact with a complete connection with the spinal cord [28]. However, LTRs recorded from a muscle subsided transiently after a complete transection

of the spinal cord at a level higher than that providing innervation to that muscle, but recovered to nearly the original level after the spinal shock period [29]. These findings strongly suggested that LTR is mediated via a spinal cord reflex [1,29].

Based on these findings, the neural network with connections among dorsal horn neurons related to an MTrP was defined as an *MTrP circuit* [30,31]. An MTrP circuit corresponding for a certain MTrP can also send nerve branches to connect with the other MTrP circuit corresponding to other MTrPs. A latent MTrP can become active if stimuli from peripheral sites are strong enough to trigger the MTrP circuit of this latent MTrP. Most adults have latent MTrPs in most skeletal muscles that can become active in response to any related lesion in another site. A recent study found no latent MTrPs in children under the age of 1 year [32••]. It appears that MTrP circuits in the spinal cord develop in later life when the child is growing up.

Diagnosis of Myofascial Pain

The diagnosis of MTrPs depends on manual palpation and clinical judgement. However, manual palpation has been considered to be an unreliable technique [33]. Special training is usually required to obtain a common agreement in the judgement of palpation criteria [24]. It has been suggested that spot tenderness, taut band, and pain recognition are the three important criteria for the diagnosis of MTrPs, and referred pain and LTRs are the confirmatory signs for MTrP diagnosis [34].

Measurement of biochemicals associated with pain and inflammation in the MTrP region [16], the sonographic study of MTrPs [35], and the magnetic resonance elastography for taut band image [36••] are potential tools for the diagnosis of MTrPs. However, some of these tools are relatively expensive at this time.

Treatment of MTrPs

Hong [1,30] defined MPS as any pain phenomenon due to activation of latent MTrPs as a consequence of certain pathological conditions, including chronic repetitive minor muscle strain, poor posture, systemic diseases, or neuromusculoskeletal lesions (eg, strain, sprain, enthesopathy, bursitis, arthritis, vertebra disc lesion). The underlying pathological lesions associated with activation of MTrPs are usually found in other regions remote to the activated MTrP due to central sensitization, but can also be due to overactivity of a muscle because of peripheral sensitization [37,38]. However, MTrPs due to muscle overactivity can be easily inactivated after avoidance of overuse or inappropriate use. Persistent or recurrent MTrPs are usually related to remote lesions. It has been strongly suggested that the most important strategy to treat MPS is to identify and treat the underlying etiological lesions appropriately [30,31,39].

In certain situations, treatment (inactivation) of active MTrPs is necessary. Inactivation of MTrPs may be important in cases of intolerable pain, pain or discomfort that interferes with functional activities, persistent pain after elimination of the underlying etiological lesion, and failure in identifying or treating the underlying pathology. Release of muscle tightness due to taut band may improve the local circulation to facilitate the healing process of the underlying etiological lesion. By either treating active MTrPs or their underlying pathology, conservative treatment should be performed before more aggressive therapy [30,31]. Any perpetuating factor that may cause persistent existence or recurrence of active MTrPs should also be eliminated, and adequate education and home programs should be provided to patients so that recurrent or chronic pain can be avoided [3,40].

The management of myofascial pain due to MTrPs has been extensively described [3,4,30,31,40]. The commonly applied MTrP therapies include intermittent cold and stretch (spray and stretch), deep pressure soft tissue massage, trigger point pressure release, postisometric relaxation, manipulation, thermotherapy (usually combined with others), ultrasound therapy [41], electrotherapy, and needling (MTrP injection, dry needling, or acupuncture) [3,30,42]. Laser therapy has also been used to treat MTrPs. Snyder-Mackler et al. [43] noted significant pain reduction and an increase in skin resistance after laser therapy; therefore, they suggested that the effectiveness was sympathetically mediated. The pain-relieving effect of laser treatment has been proposed by one or a combination of the following mechanisms: circulation enhancement, collagen proliferation, peripheral nerve stimulation, an anti-inflammatory effect, and direct analgesic effect [21]. A recent animal study demonstrated a decrease of EPN prevalence (related to MTrP irritability) after laser treatment [21]. Shockwave therapy is a newly developed device for treating MTrPs [44]. However, the mechanism in treating MTrPs is also still unclear. Combination of various methods is frequently applied in treating myofascial pain.

MTrP injection with BTX-A has been recommended to treat MTrPs [45,46]. However, some recent studies found no significant benefit from BTX-A injection compared with dry needling [47,48] or bupivacaine injection [49]. Considering the cost of BTX-A, it may not be used routinely.

The most likely mechanism of pain relief by needle stimulation is hyperstimulation analgesia [50]. The strong pressure stimulation to the MTrP loci (nociceptors) can provide very strong neural impulses to the dorsal horn cells in the spinal cord, which may then break the vicious cycle of the MTrP circuit [30,31].

Conclusions

Recent basic and clinical studies on both animal and human subjects have made the pathophysiology of MTrPs much better understood. Each MTrP contains many basic units of an MTrP, the MTrP locus. Each

MTrP locus consists of a sensory component (a sensitive locus; an LTR locus) and a motor component (an active locus; a SEA locus). The pathogenesis of MTrPs is probably related to an integrative mechanism in the spinal cord in response to sensitized nociceptors (sensory loci) associated with dysfunctional endplates (active loci). With current knowledge of the pathogenesis of MTrPs, we can keep making progresses in developing new and effective therapies for the management of MPS.

Disclosure

No potential conflict of interest relevant to this article was reported.

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