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Effect of Treatment on Trigger Points

Javid Majlesi · Halil Unalan

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Abstract Patients with muscle pain complaints commonly are seen by clinicians treating pain, especially pain of musculoskeletal origin. Myofascial trigger points merit special attention because its diagnosis requires examinations skills and its treatment requires specific techniques. If undiagnosed, the patients tend to be overinvestigated and undertreated, leading to chronic pain syndrome. Patients with myofascial pain syndrome present primarily with painful muscle(s) and restricted range of motion of the relevant joint. Palpable painful taut bands are named trigger points and are the main and pathognomonic finding on physical examination. Eliciting local twitch response and referred pain requires experience and examination skills. It may be useful to classify the patient as having acute or chronic, and as having primary or secondary, myofascial pain so the decision on the details of treatment can be curtailed to the needs of each patient. Effective treatment modalities are local heat and cold, stretching exercises, spray-and-stretch, needling, local injection, and high-power pain threshold ultrasound.

Keywords Myofascial pain syndrome · Trigger point · Stretching · Injection · High-power pain threshold ultrasound

J. Majlesi (✉) · H. Unalan
Department of Physical Medicine and Rehabilitation,
Cerrahpaşa Faculty of Medicine, Istanbul University,
Çatalçeşme Sk, Şükür Apt,
Bostancı, Istanbul, Turkey
e-mail: javidmajlesi@yahoo.co.uk

Introduction

By mass, weight, and amount of blood circulation needed, skeletal muscle is the largest human organ system, yet its painful conditions are prone to be ignored by clinicians until the patient becomes debilitatingly painful [1]. Modern medicine defines pathologies on the basis of tissue damage. However, this conceptualization often may exclude muscle pain syndromes. Muscle sprains seen as a result of sports injuries or direct blows result from serious tissue damage and are well-defined conditions in the classical textbooks. Conversely, muscle pain syndromes that cannot be measured or visualized sometimes are not classified as a disease. From the standpoint of classical pathology, which stresses the damage and healing cycle, and with nothing objective at hand to explain muscle pain, the etiopathogenesis of muscle pain syndromes has not been defined clearly. Clinicians managing patients with musculoskeletal pain often tend to classify muscle pain as adjacent and secondary conditions that would resolve or go away in the course of tissue healing.

The confusing nomenclature and terms used interchangeably complicate matters further. Fibromyalgia syndrome and myofascial pain syndrome (MPS) sometimes are perceived as identical entities despite strikingly different symptomatologies and pathophysiologies. Referring to myofascial trigger points (MTrPs), many authors and practitioners use terms such as muscle pain, muscle spasm, tenderness, tender points, fibrositis, and muscle tension. Some clinicians tend to label all painful muscles as MPS, while some diagnose MPS sparingly only after definite observation of MTrPs; still, other clinicians ignore the MPS. This state of confusion possibly has led to difficulties in the diagnosis of muscle pain syndromes and generalization of various research findings as well. Due to complexity

of the syndrome, its pathophysiology is understood better in the light of more recent research findings [2•, 3••]. Failure to diagnose MPS may lead to overuse of laboratory and imaging modalities, unnecessary interventions, iatrogenic harm, heavy economical burden, and reduced flexibility and range of motion leading to muscular imbalance [4]. The end result will be a chronic pain syndrome.

MPS originally was defined by Drs. Janet Travell and David Simons [1]. It is a relatively common [5] problem, and its understanding and clinical management often are overlooked. The way MPS is perceived by modern medicine is best described by Simons: "The cause of MPS is often enigmatic to the modern medicine; the cause of MTrPs has been elusive for the past century, and very costly despite an extensive literature that is confusing because of restricted regional approaches and a seemingly endless variety of names. Health care professionals do not receive enough training and they do not appreciate the clinical importance of MPS" [6]. This may be a major problem for clinicians managing pain because some pain clinics report incidence of trigger points (TrPs) to vary between 30% and 85% of presenting people, being more common in women [7]. Overall, these factors have led to underdiagnosis and poor management of MPS over the years [8].

Clinical Characteristics

Etiology of MPS and characteristics of TrPs have not been explored sufficiently by investigators [6]. In general terms, muscles can be painful after overload, excessive stretching, and direct trauma or after repetitive overload, overuse, disuse, biomechanical discrepancies, imbalances, and asymmetries that can lead to overload [9]. It is believed that these factors can lead to development of MTrPs. During history taking, age, level of activity, occupation, hobbies, and sports activities should be noted to reveal hidden precipitating factors. Precipitating factors generally may be embedded in details of lifestyle. Questioning the occupation and its demands, such as prolonged sitting or standing, repetitive movements, lifting and carrying patterns, and discrepancies in loading of the lower extremities, would shed light on these factors. During physical examination, the clinician should look for acquired and congenital abnormalities and skeletal characteristics such as scoliosis and limb-length discrepancies that would be perpetuating factors and lead to asymmetrical and disproportionate loading. Examination of the muscles should detect taut bands (TB), TrPs, and tender spots, and sensitized spinal segments have to be located accurately and identified correctly for effective needling treatment [10•]. MPS can be confined to one or more muscles, be a primary condition, or arise secondary to a disorder [11].

Additional signs and symptoms would be restricted range of motion of the joint, complaints of tingling, burning, ache, and tightness and difficulty during the activities of daily living [9]. MPS often is accompanied by signs and symptoms in addition to pain, coincidental pathologic conditions, and behavioral and psychological problems [5]. Examples of coincidental pathologies would be radiculopathies, nerve entrapments, bone or joint in the stage of healing, and congenital musculoskeletal abnormalities. Psychological problems frequently would be those seen in patients complaining of acute or chronic pain such as fear, anxiety, depression, and verbalization of pain [7]. Patients with MPS have complaints of pain that is more prevalent in the head, neck, shoulders, hips, and low back [7]. The muscles of these regions consistently work against gravity or repetitively during activities of daily living. Pain is dull or achy and associated with autonomic changes such as sweating, lacrimation, flushing, and vasomotor and temperature changes [8, 12]. Local and referred pain are the characteristic complaints that may mimic radiculopathy and nerve entrapment syndromes [13].

MPS is a musculoskeletal pain that can be acute or chronic. Primarily, psychosocial factors can contribute to chronicity along with metabolic disorders, nutritional imbalances, and regional biomechanical imbalances [14•]. Patients with MPS had been found to have higher scores for anxiety and depression [15]. Referred pain from MTrPs can mimic visceral pain syndromes, and visceral pain syndromes can induce TrP development; MPS and the referred pain syndrome can outlast the initial event, making diagnosis difficult [16].

None of the findings on physical examination is a gold standard diagnosis criterion and no routinely available laboratory or imaging test is available [6]. Painful TB(s) presenting with complaint of pain may be the minimum requirement for clinicians to consider myofascial pain as a possibility. These palpable painful TBs named TrPs, which are sensitive areas in the muscle, are painful to palpation and reproduce characteristic referred pain and patient's symptoms [17]. The basic finding in the patients with myofascial pain is the TB painful on palpation. MTrPs may be active (spontaneously painful and symptomatic) or latent (nonspontaneously painful) [3••, 5]. The local twitch response (LTR), activated by snapping palpation, compression, or needle insertion at the TrP, is a characteristic finding of the condition [5, 18].

Studies that have been done on the reliability of the findings on physical examination have found the TB as the single reliable and agreed upon finding [19–22]. Studies also show that agreement between clinicians on the presence of TrPs is low [20–24]. This interrater reliability is higher among the trained examiners [11, 24, 25]. The study findings show that more experienced clinicians tend to distinguish a TB more easily and be able to elicit an

LTR. If the MTrP is not irritable enough, it may require higher pressure to elicit referred pain. This characteristic may be one of the factors that underlie the low reliability in making a diagnosis of TrP because normal muscles also can be painful on enough compression. As a result, this also may be the factor responsible for underdiagnosis or overdiagnosis by clinicians. In the cases of normal muscles or latent TrPs, palpation could not be painful and could not elicit a referred pain or an LTR. The more irritable the TB is, the more readily a referred pain or an LTR could be seen. Similarly, electromyographic, thermographic, and pressure algometric abnormalities show inconsistencies due to the facts that electromyography (EMG) needle insertion can bypass the TrP, that trained and experienced clinicians tend to make a more accurate diagnosis, and that the amount of manual or algometer compression varies among clinicians [7]. These may be the factors behind the faulty methodologies or findings in some studies. Hong et al. [26] found that using an algometer would not guarantee a higher rate of referred pain or more painful TBs. The exact amount of pressure required to elicit referred pain or LTR may be a characteristic of every TrP. The authors found that referred pain could be found upon compression of normal muscles as well, and it was more prevalent in active TrPs.

Despite all the aforementioned findings, there are some minimum clinical findings upon which most clinicians considering a diagnosis of myofascial pain syndrome would agree. These are 1) presence of a trigger point; 2) referred pain upon compression of the trigger point; 3) LTR immediately after snapping palpation or insertion of a needle; and 4) restricted range of motion of the joint**.** Originally defined by Simons et al. [17], additional findings are weakness and associated localized autonomic phenomena including but not limited to vasoconstriction, pilomotor response, ptosis, and hypersecretion. Tough et al. [27•], in an extensive review of 93 out of 607 studies that met their inclusion criteria, found that the four most commonly used diagnostic criteria would be 1) tender spot in a TB; 2) predicted pain referral pattern; 3) patient pain recognition; and 4) LTR. The first two criteria were used by over half of the studies reviewed. However, more than half of the studies failed to use the diagnostic criteria defined by Simons et al. [17] to which they referred. It can be inferred that most clinicians agree upon these original diagnostic criteria, yet do not necessarily seek all of them during physical examination. This would be another factor that would lead to low reliability of the physical examination findings.

Pathophysiology

The integrated hypothesis proposed by Simons et al. [17] and Mense et al. [28] still is considered to be the basic pathophysiological mechanism behind the TrP formation. Chronically shortened sarcomere may increase local energy consumption, and reduction of local circulation leads to local ischemia and hypoxia. This ischemia stimulates the release of neuroactive and vasoactive substances that lead to nociception and perception of pain, such as prostaglandin, bradykinin, serotonin, and histamine.

The findings seen at the TrP can be explained by two hypotheses. First, because TrPs are found at the muscle spindle, the α-adrenergic antagonists may be effective at the TrPs. However, this hypothesis does not fully explain the EMG findings recorded at the TrP. Second, because the EMG findings recorded at TrP resemble the findings described at the end-plate region, TrPs represent hyperactive end-plate regions [18]. Needle EMG examination recordings from TrPs show low-voltage spontaneous activity and activity resembling end-plate spikes. High frequency (10–12 per second) repetitive spikes with an amplitude of about 1000 μV and a duration of 1 to 3 ms have been recorded from MTrP [29]. This EMG activity is called spontaneous electrical activity (SEA) [30–33]. The same SEA can be recorded in animal TrPs [34]. This EMG activity is reduced with infusion of phentolamine and local infusion of phenoxybenzamine [18], after lidocaine block [35], denervating the muscle, or after high-power pain threshold ultrasound (HPPTUS) treatment [34].

Simons et al. [31] hypothesizes that SEA originates from active loci, which are motor structures. There also are sensitive loci, which are sensory structures. A sensitive locus together with an active locus form a TrP. Active loci are widespread in the muscle because referred pain can be elicited with enough pressure in normal muscle. It can be theorized that this electrical activity would stem from excessive acetyl choline (ACh) release and resultant end-plate dysfunction. MTrP is more likely to be located at the end plate [31–35]. The effectiveness of botulinum toxin A (BTA) may arise from this characteristic [36–38].

In light of recent findings and explanations in central and peripheral mechanisms of pain, the pathogenesis of MPS may be thought of as having a central mechanism with peripheral clinical manifestations [8]. Therefore, the components of the MTrP could be classified as sensory (sensitized nociceptors that are responsible for pain and referred pain) and motor (dysfunctional end plates that are responsible for TB formation and LTR). Resultant cellular findings relating to one another in a cycle would be excessive ACh release, sarcomere shortening, and release of sensitizing substances [39•]. Sections of animal TrPs show this sarcomere shortening, and cross-sections of human MTrPs confirm the findings [40, 41]. Animal experiments show that LTR can be abolished by transection of the motor nerve or infusing it with lidocaine [18].

The central mechanism of myofascial pain and dysfunction with characteristic TrPs and TBs can be classified as a spinal reflex disorder caused by a circuit of sustained neural activity in a specific spinal segment [18]. Some authors have tried to explain the referred pain from MTrPs as secondary hyperalgesia of peripheral neural origin, believing that the pain of MPS also is similar to nerve trunk pain, which is an example of somatic referred pain [42]. However, the best possible mechanism that may explain the referred pain is convergence and facilitation. Convergence, or wind-up, is a phenomenon that results from amplification and wind-up of dorsal horn neurons leading to spreading to adjacent segments [43, 44]. This leads to persistent pain due to sensitization in the dorsal horn and increased sensitivity and excitability of neurons [45–49]. Neurotransmitters responsible in these sensitization processes are substance P, *N*-methyl-D-aspartate, glutamate, and nitric oxide [50]. All these changes result in more ACh release and augmentation of the cycle of pain. The calcium in the muscle fibers increases as a result and leads to shortening and increased metabolism. Analysis of TrPs by microdialysis has demonstrated elevated levels of these proinflammatory substances at these sites [51•].

Treatment

In general, the clinician must be aware that treatment of MPS requires different approaches for acute and chronic cases. Muscle-specific modalities and techniques are required for prompt relief when acute, and additional attention to perpetuating factors are required when chronic [6]. MPS treatment should focus on maintenance of independence and restoration of physical activity [52••]. Prevention of recurrences can be achieved after correction of perpetuating factors and if long-term compliance is maintained by the patient. Management programs involving restoring the normal function of the affected muscles and controlling the contributing factors are effective if a long-term approach is attained by the clinician–patient team [53]. The difficulty in managing MPS sometimes can be explained by the clinical need to match the level of complexity of the management program with the complex characteristics of the patient [39•]. There also is a high recurrence rate unless appropriate exercises to restore flexibility and balance to the muscles are prescribed, with active participation from the patient [54]. Some authors define different approaches, such as enhancing central inhibition through pharmacology and behavioral techniques as well as simultaneously reducing peripheral inputs through physical therapies including exercises and TrP-specific therapies [8, 55]. Different approaches may stem from unique symptoms and lifestyle characteristics of each patient. However, clinicians agree that chronic myofascial pain usually may be a product of both physical and psychological factors that complicate convalescence [1]. In the cases of secondary MPS, treating the underlying condition leading to activation of TrPs should be the most important strategy. This approach also would prevent the recurrence of TrPs [56••].

Various modalities, such as the spray-and-stretch technique, ultrasound, manipulative therapy, and injection, are used to inactivate TrPs. Considering the low level of interrater reliability and agreement on the presence of MTrPs [20–24] and the lack of uniformity in using the diagnostic criteria [27], the results of different studies and reviews show various modalities and techniques as effective or ineffective. Noninvasive methods of treatment are vapocoolant spray and stretch, manual stretching by TrP pressure, contract–release method, transcutaneous electrical stimulation, traditional physical therapy, and massage; invasive methods are dry needling and TrP injection [58–60]. Vernon and Schneider [61] reviewed a total of 112 articles and concluded that there is strong evidence for laser therapy; moderate evidence for transcutaneous electrical nerve stimulation, acupuncture, and magnet therapy; and weak evidence for electrical muscle stimulation, high-voltage galvanic stimulation, interferential current, frequency-modulated neural stimulation, and ultrasound therapy. Some authors believe that acute MPS responds well to manual and injection therapies, but requires attention to postural, ergonomic, and structural factors, as well as metabolic factors that impair muscle function [11, 62]. Peloso et al. [63•] reviewed 36 trials from a search of the Cochrane database regarding the effects of NSAIDs, psychotropic agents, steroid injections, and anesthetic agents. The authors found lack of replication of findings and of sufficiently large trials to be the major limitations of the studies. NSAIDs, muscle relaxants, and analgesics were found to be of limited evidence and of unclear benefit. Manual therapy of the TrP also has been found to be effective for prompt relief of symptoms [51•].

Stretching

Stretching exercises originally were defined by Simons et al. [17] as the basic treatment for MPS and have been found to be effective techniques [57]. Most clinicians believe that appropriate treatments should be directed at the TrP to restore normal muscle length and proper biomechanical orientation of myofascial elements, followed by treatments including strengthening and stretching of the affected muscle. As a result, treatment basically should encompass inactivating the TrP, correcting underlying perpetuating factors, and restoring the normal relationships between the muscles of the affected functional units [13]. This basic

strategy renders stretching exercises for the affected muscles the cornerstone for MPS treatment.

Needling and Injection

Borg-Stein and Simons [64] believe that TrP injection should augment the stretching exercises, not be the sole technique, and should be preceded and followed by manual techniques. The main therapy applicable by the clinician for the MPS has been considered to be TrP injection [65]. TrP injection is superior to stretching alone, and has been shown to be one of the most effective treatment modalities to inactivate TrPs and provide prompt relief of symptoms [9]. Some clinicians use superficial (into a depth of 5–10 mm) dry needling. In this technique, the needle is kept in place until the feeling of pain is abolished. This may take 30 seconds [66]. It is believed that the needle mechanically terminates the dysfunctional activity of the motor end plates.

Scott et al. [67•] reviewed the published reviews and randomized controlled trials (RCTs) detailing the use of TrP injection. The criteria were met in 15 RCTs. The authors concluded that TrP injection is a safe procedure when used by clinicians with appropriate expertise and training. The authors also comment that the addition of TrP injection to stretching exercises augments treatment outcome; this also was true of other therapies such as ultrasound and laser.

Peloso and associates [63•] and Ho and Tan [68] carried out systematic reviews and concluded that there is no supporting or moderate evidence for the use of BTA injection in TrP treatment. However, other studies have reported effectiveness of BTA injection [68, 69]. Acupuncture also has been found to be an effective treatment for the MPS [70]. The above mentioned studies and reviews show that overall, dry needling, short- or long-acting anesthetic injection, steroids, and BTA all are effective techniques. Different techniques of needling including slow search, fast in–fast out, superficial dry needling, intramuscular stimulation, twitch-obtaining intramuscular stimulation, and needling and infiltration with preinjection blocks all have been found to be effective [63•, 68–70].

If LTRs were elicited during injection, especially if the fast in–fast-out technique was used, one could expect more immediate relief of pain [18, 52••, 58, 65, 71]. This approach creates hyperstimulation or counterirritation during needling that, as a result, creates analgesia by disruption of reverberatory neural circuits in the central nervous system [72]. LTR elicitation also is believed by acupuncturists to be essential to pain relief in needling of MTrPs [73].

Ultrasound

Studies aimed to investigate the effectiveness of classic techniques of ultrasound have yielded different and inconsistent results. Esenyel and associates [74] compared ultrasound and stretching to TrP injection and stretching and found both methods equally effective. Aguillera et al. [75••] compared ischemic compression with ultrasound and found that both treatments decreased basal electrical activity of the trapezius muscle measured with surface EMG. Active range of motion improved more after ischemic compression. Gam et al. [76] studied the effectiveness of ultrasound in MTrPs. They found ultrasound and sham ultrasound equally effective on the sensitivity of the TrPs. The study groups and the control group did not show any significantly different scores in the visual analogue scale and analgesic usage. We believe all these studies may leave the reader confused because decisions made on the characteristics of ultrasound intensities and durations may determine the effectiveness of ultrasound therapy.

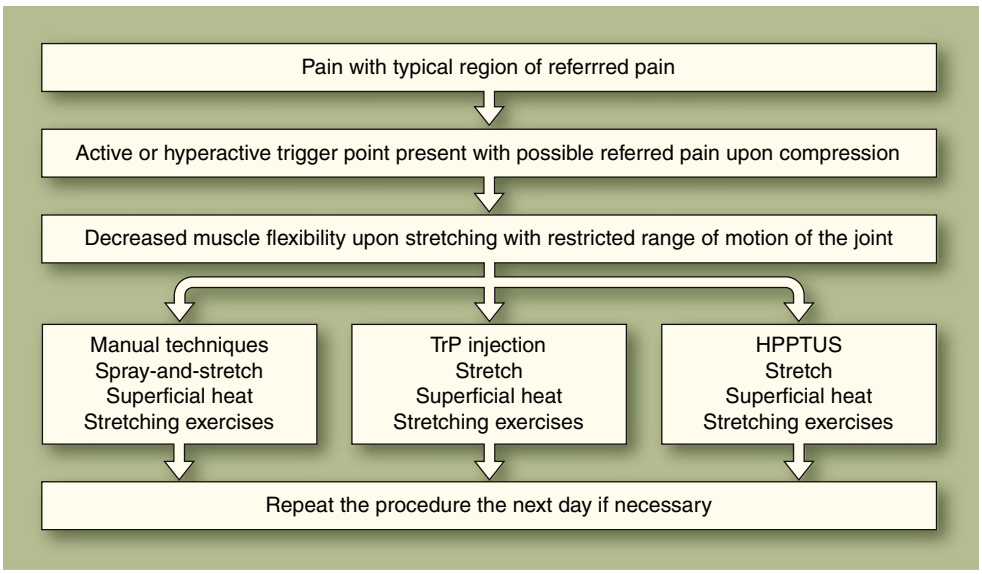

Fig. 1 Suggested algorithm for the treatment of myofascial pain syndrome. HPPTUS—high-power pain threshold ultrasound; TrP—trigger point

The novel approach to MTrP treatment during recent years is ultrasound application to reduce the sensitivity of TrP. Srbely and colleagues [77••, 78••] compared therapeutic intensity of ultrasound with low-intensity application and found pain pressure threshold increase after application at therapeutic intensity. This shows that therapeutic intensity of ultrasound can immediately reduce TrP sensitivity. These authors' approach is a shift from the classic technique of ultrasound applied on an area that includes TrP to application of therapeutic intensities on the TrP.

Another ultrasound technique that delivers sound waves directly to the TrP and results in immediate pain relief and gains in active range of motion is high-power pain threshold ultrasound (HPPTUS). HPPTUS has been cited in the classical textbook by Simons et al. [17] emerging from a personal communication. It is applied in therapeutic to high intensities (1.0–2.5 W/cm²) and by holding the ultrasound probe stationary on the TrP for 1 to 3 s, thereafter reducing the intensity in half for 15 seconds and for a total of two to four cycles. This technique elicits pain during the application. The first study on HPPTUS was done by Majlesi and Unalan [79]. In this study, we compared HPPTUS with sham ultrasound in an RCT. HPPTUS was found to be superior with immediate and short- and long-term improvements in active range of motion of the cervical spine and pain on visual analogue scale. The patients did not report any side effects. Unalan and associates compared HPPTUS with TrP injection in a recent study that is in the review process for publication, and found both treatments equally effective on the level of pain and range of motion of the cervical spine (Unalan, unpublished data). Bahadir and associates [80••] studied effects of HPPTUS to local injection on the EMG activity of trigger points. Both treatments were found to be equally effective in lowering the SEA of the TrP on EMG. To investigate the presence of any side effects after application of HPPTUS in different intensities and durations, Unalan and associates applied the technique on nerve, muscle, and bone tissues of animals. The investigators found that when applied as the recommended technique and cycles, HPPTUS is free of side effects (Unalan, unpublished data). This study has been approved for publication by the *Journal of Musculoskeletal Pain*. Applied directly on the TrP, ultrasound is a promising treatment modality.

Conclusions

Treatment of MPS requires diagnosis of TrPs and determination of precipitating factors. The treatment approach selected should be based upon these findings. Acute MPS can be treated choosing techniques such as manual techniques, spray-and-stretch, TrP injection, and ultrasound (Fig. 1). Chronic MPS may require lifestyle and work modifications, psychological management, and a long-term exercise program.

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