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A Comprehensive Review of the Treatment and Management of Myofascial Pain Syndrome

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Abstract

Purpose of Review Myofascial pain syndrome (MPS) is a musculoskeletal pain condition that stems from localized, taut regions of skeletal muscle and fascia, termed trigger points. The purpose of this comprehensive review is to provide updated information on prevalence, pathophysiology, and treatment modalities with a focus on interventional modalities in managing MPS.

Recent Findings Though MPS can present acutely, it frequently presents as a chronic condition, affecting up to 85% of adults during their lifetime. MPS is an often-overlooked component of pain with overarching effects on society, including patient quality of life, physical and social functioning, emotional well-being, energy, and costs on health care. The prevalence of MPS is generally increased among patients with other chronic pain disorders and has been associated with various other conditions such as bladder pain syndrome, endometriosis, and anxiety.

Summary MPS is poorly understood and remains a challenging condition to treat. Non-pharmacologic treatment modalities such as acupuncture, massage, transcutaneous electrical stimulation, and interferential current therapy may offer relief to some patients with MPS. Additional studies are warranted to get a better understanding of managing myofascial pain.

Keywords Myofascial pain syndrome · Trigger points · Chronic pain · Musculoskeletal pain

Introduction

Chronic musculoskeletal pain is a common problem across the USA and the globe contributing to disability, along with other comorbidities including anxiety and depression and leading to numerous health care interventions. In fact, in a publication describing US spending on personal health care and public health from 1996 to 2013, Dieleman et al. [1] showed an estimated spending of $183.5 billion in managing musculoskeletal disorders including chronic musculoskeletal and spinal pain. Numerous modalities of treatment have been employed in managing chronic musculoskeletal pain ranging from simple over-the-counter medications to complex surgical fusions, including multiple types of interventional techniques

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[1–10]. Two common conditions included in musculoskeletal pain are related to myofascial pain syndrome (MPS) and fibromyalgia syndrome. In addition to various modalities, opioids have commonly been used in managing chronic musculoskeletal pain leading to overuse, abuse, dependency, addiction, and other adverse effects, such as overdose deaths [11–14]. Consequently, multiple other techniques with interventions have been recommended in managing musculoskeletal pain [11–14].

Myofascial pain syndrome is a musculoskeletal condition that is thought to stem from localized, taut regions comprised of skeletal muscle and fascia, termed trigger points [15•]. Trigger points create foci of pain frequently characterized as dull, aching, boring, and burning. MPS also presents with a neuropathic component, evidenced by its referred pain patterns. While myofascial pain is unique in its combination of neurocutaneous and neuropathic components, the nature of symptoms, such as the quality of pain experienced, is highly dependent on the patient's perception. It has been postulated that chronic MPS leads to fibromyalgia, with multiple treatments overlapping fibromyalgia and MPS.

Prevalence

MPS can present acutely, though it is most often described as a chronic condition. Among the general population, the lifetime prevalence of MPS is up to 85%, with variable rates between males and females [16]. Reported overall rates, however, vary considerably with different patient populations. Rates of 21% have been reported in the general orthopedic population while rates as high as 85% to 93% have been reported in specialty pain clinics and more commonly in women. MPS is the leading cause of chronic and persistent regional pain, including shoulder pain, chronic back pain, tension type headaches, and facial pain [10].

Etiology

A clear mechanism for the development of trigger points for myofascial pain does not exist; however, trigger points are believed to emerge from several categories including muscle overuse, muscle trauma, psychological stress, or ergonomic, structural, or systemic factors. Some examples of ergonomic factors that include muscle overuse include activities of daily living, lifting heavy objects repeatedly, or ongoing repetitive activities. In these instances, improper or abnormal posture, deconditioned muscle, poor ergonomics, and fatigue all contribute to the development of myofascial trigger points that contribute to myofascial pain. Sleep deprivation can also exacerbate these symptoms [17•]. Abnormal structural factors that can contribute to the development of these trigger points include scoliosis, spondylosis, osteoarthritis, chronic disease states, and spinal degenerative conditions [18, 19•]. Finally, there are also systemic factors that can contribute to the development of myofascial pain including hypothyroidism, vitamin D deficiency, and iron deficiency [19•].

Pathophysiology

Multiple hypotheses have been put forth in describing the pathophysiology of MPS and fibromyalgia ranging from pain beliefs to trauma to the musculature. In essence, chronic musculoskeletal pain has been considered a biopsychosocial condition in which contextual, cognitive, and emotional factors as well as biological factors significantly affect pain perception [20]. There has been significant debate in reference to development of chronic musculoskeletal pain and its progression from acute and subacute phases [21]. Due to heterogeneity of pain mechanisms, the transition from acute musculoskeletal pain to chronic pain continues to be difficult to predict [21]. However, in a significant proportion of chronic musculoskeletal pain populations, central sensitization of the multiple pathways has been proposed with altered central pain modulation [21]. Altered central pain modulation manifests as a predominantly non-nociceptive, non-neuropathic pain mechanism defined as dysregulation of the central nervous system (CNS) causing neuronal hyperexcitability, characterized by generalized hypersensitivity of the somatosensory system to both noxious and non-noxious stimuli. Altered central pain modulation involves impaired modulatory mechanisms within the CNS whereby nociceptive pathways are less inhibited and nociceptive facilitatory pathways enhanced, resulting in augmentation of nociceptive transmission [21]. Further, multiple studies have demonstrated that there is correlation between the functional connectivity and pain symptoms in pain conditions including fibromyalgia, complex regional pain syndrome, and neuropathy [22]. A systematic review by Orhan et al. [20] assessed the effect of pain beliefs, cognitions, and behaviors influenced by race, ethnicity, and culture in patients with chronic musculoskeletal pain. They identified differences in coping strategies among various races and cultures, along with illness perceptions, self-efficacy, fear avoidance, beliefs, locus of control, and pain attitudes. Further, it was also shown that there was evidence for decreased regional gray matter volume in women with chronic whiplash-associated disorders related with processing cognition and pain [23]. Hypovitaminosis D has also been identified in patients with musculoskeletal pain [24]. To support various considerations with the CNS, it has been shown that conservative treatment changes the brain in patients with chronic musculoskeletal pain with induction of functional and structural brain changes in prefrontal regions [25••]. Further, increased risk of inflammatory bowel disease has been reported in patients with fibromyalgia among males [26]. It is also important to consider that patient perceptions of chronic pain during economic crisis lead to worsening of their symptoms and deterioration of quality of life [27]. Thus, the

pathogenesis of MPS is complex and occurs as a result of multiple interacting mechanisms. MPS is postulated to be caused by an abnormal increase in acetylcholine release at the motor endplate nerve terminal, which causes sustained muscle fiber contractions. These sustained contractions in turn cause local muscle ischemia and pain presenting as taut bands.

Myofascial trigger points (MTrPs) can produce symptoms of pain upon palpation and pressure. A snapping palpation or needle insertion can trigger a local twitch response (LTR), which can reproduce symptoms of pain upon palpation.

Various mechanical and electrophysiological studies have elucidated the mechanisms behind MTrP pain production in MPS [28]. The MTrP locus is hypothesized to be composed of an active locus (motor) and a sensory locus (sensory). Propagation of potentials along extrafusal fibers and attenuation of pain through botulism treatment imply an endplate zone localization [28]. Constant nociceptor stimulation from the primary muscle site contributes to localized and referred pain. Various studies have identified spontaneous electrical activity (SEA) from MTrPs, but have not identified them in non-MTrP sites [29]. Moreover, electromyography (EMG) activity can be seen in electrodes inserted in taut bands that elicit LTRs [30].

Diagnosis

Current diagnostic criteria require a detailed history and physical exam. While guidelines for palpation points of different body systems have been developed, unifying criteria are lacking. The presence of the treatable tender MTrPs and a referred pain pattern is pathognomonic for MPS. Palpation or needle insertion can trigger a LTR, defined by rapid contractions of muscle fibers which can ultimately lead to symptoms of pain. Primary MTrP refers to the primary muscles affected, which can sometimes be felt as taut bands. Secondary MTrP refers to both synergistic and antagonist muscles. An active MTrP spontaneously induces pain along with an associated pattern of referred pain, while a latent MTrP does not trigger a localized pain until it is palpated. Taut MTrPs are more commonly found in the trunk, including the trapezius, rhomboid, and neck, as opposed to the extremities. Muscle weakness and decreased range of motion are commonly associated with these tight knots.

It is important to rule out other causes such as trauma, fibromyalgia, sedentary lifestyle, and other forms of neuropathic pain. Multiple assessments with imaging and laboratory tests are performed to rule out other causes of pain. In a systematic review, Thibaut et al. [31] emphasized the relationship between increased intracortical disinhibition (measured by transcranial magnetic stimulation (TMS)) and MPS. This study was centered on the central sensitization theory, which asserts that chronic pain can cause the CNS neurons to be less inhibited and thus hyperexcitable.

To objectively quantify the physical properties of trigger points, Chen et al. [32] used magnetic resonance elastography (MRE) to determine that band stiffness parameters correlated well with predictions by gel replica experiments. In contrast to the subjective touch of a health practitioner's fingers, MRE demonstrates that MTrPs can be consistently characterized quantitatively.

Relationship to Other Pain Disorders

MPS has been associated with other types of pain disorders [33]. Myofascial pelvic pain (MFPP) is characterized similarly to MPS, but with trigger points localized to the pelvic floor muscles [33]. MFPP has been demonstrated in various conditions such as bladder pain syndrome, endometriosis, and anxiety. Multiple studies have recorded the increased prevalence of MTrPs in patients with migraines or tension-type headaches (TTH) [34]. Palacios-Cena et al. provided supporting evidence to the central sensitization theory by observing hypersensitivity of MTrPs [35]. This hypersensitivity may be a contributing factor to lowered pressure pain thresholds in the pericranial muscles of patients with TTH. Osteoarthritis (OA) has been hypothesized to be associated with MTrPs, given the occurrence of patient complaints of pain without obvious radiographical findings. Observational studies, such as those of Bajaj et al. [36] and Henry et al. [37] have reported a positive correlation between knee OA and MTrPs in the lower extremity muscles (e.g., vastus lateralis, rectus femoris, and gracilis). MPS of the temporomandibular (TMJ) region is common, especially in patients with a history of TMJ trauma, and can be mistakenly diagnosed as a TMJ disorder refractory to treatment [38]. In a systematic review and meta-analysis by Chiarotto et al. [39], MPS was found to be commonly associated with whiplash-associated disorder, lumbar disc herniation, idiopathic neck pain, and cervical radiculopathy.

Pharmacologic Treatment Modalities

Nonsteroidal Anti-inflammatory Drugs (NSAID)

Nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used for pain relief; however, their use in chronic pain disorders is limited related to adverse gastrointestinal (GI) and renal effects. These include but are not limited to dyspepsia, GI ulceration and bleeding, peripheral edema, and organ failure [40]. NSAIDs help relieve pain via inhibition of cyclooxygenase (COX) enzyme and thus inhibition of prostaglandin synthesis, which allows for reduced sensitization and excitation of peripheral nociceptors. There is limited evidence to support the use of oral NSAIDs for the treatment of MPS. In this regard, topically administered NSAIDs have been shown to be effective [41]. In a prospective randomized control trial

(RCT) of 153 patients, Hsieh et al. [41] demonstrated that topical diclofenac sodium, administered as a patch, provided pain relief and improved function in patients with MPS of the upper trapezius, when compared with patients who were given a menthol patch.

Tri-cyclic Antidepressants

Tri-cyclic antidepressants (TCA) have a wide variety of uses in the management of depression and various pain syndromes. Typical doses of amitriptyline range from 20 to 100 mg daily for the treatment of MPS. TCAs may provide analgesia through inhibition of serotonin and norepinephrine (NE) reuptake along the descending spinal pain pathways. TCAs also exert effects on sodium channels and histamine receptors [42]. Studies investigating the efficacy of TCAs in MPS have demonstrated significant benefit [43, 44]. Haviv et al. [45] demonstrated favorable effects of tri-cyclic antidepressants in patients with persistent facial pain and tenderness of regional muscles [45]. Amitriptyline has been found effective in patients with chronic tension-type headaches and chronic pain associated with temporomandibular disorders [46].

Muscle Relaxants

The efficacy of various muscle relaxants in the treatment of MPS has been investigated. These include cyclobenzaprine, baclofen, tizanidine, clonazepam and other benzodiazepines, and orphenadrine [47]. Cyclobenzaprine provides analgesia by the inhibition of NE reuptake in the locus coeruleus and the inhibition of descending serotonergic pathways in the spinal cord [47]. Clonazepam and other benzodiazepines act on chloride channels to enhance GABA-A receptors, resulting in inhibition at presynaptic and postsynaptic sites on the spinal cord [48]. Muscle relaxants function by decreasing skeletal muscle tone, thus alleviating the increased muscle activity seen in MPS. Muscle relaxants may cause adverse effects, which can include sedation, dizziness, depression, anticholinergic effects, and ataxia. Based on the present literature, there is no significant evidence for muscle relaxants in the use of any musculoskeletal pain. Further benzodiazepines are contraindicated due to their abuse potential and enhancing opioid-related deaths.

Another muscle relaxant, tizanidine, an alpha-2 adrenergic agonist, has also been recommended [49]; however, it is associated with additional side effects including hypotension, bradycardia, urinary frequency, and blurred vision, along with lack of proven effectiveness.

Local Anesthetics

Lidocaine functions as a nonspecific sodium channel blocker, stabilizing neuronal cell membranes and inhibiting nerve impulse initiation and conduction [47]. Possible side effects of lidocaine injections include anaphylaxis, CNS depression, seizures, and arrhythmias. Xie et al. [50] investigated the efficacy of lidocaine injections in the trapezius muscle for chronic neck pain associated with MTrP in a prospective observational study of 120 patients. They determined that lidocaine injection therapy significantly reduced the degree and frequency of neck pain in patients after 6 months of treatment. Firmani et al. [51] examined the use of lidocaine patches instead of injections for neck pain due to MTrP and found that 5% lidocaine patches provided pain relief when compared with the placebo group. Affaitati et al. [52] compared the effectiveness of lidocaine injection with lidocaine patch and concluded that both were equally effective in relieving myofascial pain, but that discomfort from therapy was lower with the lidocaine patch. More recently, Affaitati et al. [53] explored the use of lidocaine injection versus topical nimesulide (an NSAID) gel in treating cervical MTrP. They found no difference in the efficacy between the two treatment groups, but less discomfort in therapy with the nimesulide gel. These findings encourage the use of lidocaine patch or topical NSAID gel for MPS rather than lidocaine injection.

Historically, local anesthetic injections have been used in managing pain since 1901 in the form of epidural injections [54–57]. The data related to the effectiveness of local anesthetic with or without steroids extends not only to epidural injections but also to multiple other types of injections including facet joint interventions, trigger point injections, and local anesthetic infusions [54–69]. In addition, multiple studies have shown the long-term effect of local anesthetic with epidural administration and nerve blocks by multiple mechanisms which include neural blockade altering nociceptive input, the reflex mechanism of afferent fibers, self-sustaining activity of neurons, and the pattern of central neuronal activities [70–78]. Additionally, studies have also shown that corticosteroids failed to provide any significant additional benefit in nerve infiltration for lumbar disc herniation [77, 78]. Multiple issues related to conflict or confluence of interest and lack of understanding of clinical utility in evidence synthesis have also been raised [79–87]. Despite extensive literature or effectiveness of interventional techniques with clinical and cost utility analysis, the usage has been declining and they have been criticized. There continues to be significant discussions in relation to their medical necessity and indications.

Botulinum Toxin

Botulinum toxin (Botox) functions by preventing acetylcholine release at the neuromuscular junction in order to prevent muscle hyperactivity and spasm. It also prevents release of pain neurotransmitters at primary sensory neurons [47, 88]. There are many possible side effects of Botox, which include

excessive or adjacent unwanted muscle weakness, hypersensitivity reaction, anaphylaxis, autonomic dysreflexia, respiratory compromise, urinary retention, myasthenia gravis, and facial paralysis [15, 47]. Multiple investigations of the effectiveness of Botox in the management of temporomandibular dysfunction showed conflicting data, with several studies showing significant effectiveness of Botox, while some studies showing equal effectiveness compared with facial manipulation, whereas others find no benefit [47, 48, 88]. Table 1 summarizes pharmacological treatment options [41, 44, 49–51, 88].

Minimally Invasive Non-pharmacologic Treatment Options

Myofascial Release

A number of non-pharmacological methods have been employed to help alleviate the chronic pain in MPS. Myofascial release is one of the non-pharmacological methods that can be applied in 2 different ways utilizing direct myofascial release involving slow, sustained, pressure applied to areas of muscular tension with the goal of applying enough force to stretch the fascia [89]. Indirect myofascial release involves using the hands to hold the fascia in a gentle stretch, applying just small force along the direction of the fascial restrictions, allowing the restricted fascia to unravel itself [90]. However, the purpose of both techniques is to break pathologic fascial adhesions in turn reducing the muscle stiffness. While the data showing the effectiveness of myofascial release (MFR) is scant, a systematic review by Kalichman and Ben David [91] of 8 RCTs ($n = 457$) reported that while all 8 of the RCTs found reduced pain and improved function as a result of MFR, only 3 of them had enough effect to be considered clinically significant at short-term follow-up (up to 2 months).

Dry Needling

Dry needling is a non-pharmacological approach to the management of pain associated with MPS. Dry needling is a minimally invasive therapeutic procedure that involves the insertion of a thin filiform no-bore needle into MTrP without the addition of solutions or local pharmacological agents [92–94]. The needle is inserted until it causes a LTR and is then removed. Though the mechanism of action of dry needling is debated, it is thought that dry needling offers relief through the gate control theory of pain. In a systematic review, Liu et al. [95] examined 11 RCTs that compared dry needling with other treatment modalities (e.g., sham dry needling and acupuncture); the results showed dry needling with significant postintervention reduction in pain intensity and functional improvement when compared with alternate treatment. However, a review by Rodriguez-Mansilla et al. [93] was more skeptical, citing inconsistencies in results and superior efficacy of alternative treatments, such as lidocaine and corticosteroid injections. Gerber et al. [96] attempted to clarify some of the ambiguity by labeling participants as "responders" or "non-responders." They found that as a result of dry needling treatment, many patients' MTrP transitioned from being active (spontaneously painful) to latent (painful only on palpation). They labeled these participants as responders and found that this group experienced significantly greater analgesia than non-responders. The implications of this are that if patients achieve analgesia after the first treatment, they are more likely to receive a sustained benefit; thus, these patients may be better candidates for dry needling.

The effectiveness of dry needling has been compared with various alternative therapeutic modalities including manual therapy, transcutaneous electronic nerve stimulation, Botox, and lidocaine injections.

Acupuncture

Acupuncture (AcP) is a non-pharmacological approach to the management of MPS. AcP involves the insertion and subsequent manipulation of needles in specific points around the body. The needles are typically left inserted for a short period of time and may be gently shifted or twirled [97]. The traditional Chinese belief held that this practice helps to balance one's energy, but modern Western medicine suggests a mechanism of action similar to the gate control theory of dry needling, and proposed that dry needling and AcP are essentially the same [98]. However, a distinction has been made between the 2 with dry needling targeting MTrP, while the needling in AcP is directed at specific patterns or meridians along the human body. A systematic review by Wang et al. [97] examined 16 studies ($n = 477$) on the efficacy of AcP in MPS and found significant improvements in pain and reduced irritability after just one session. The authors noted that these analgesic effects were significant only when AcP was used to target trigger points, but no analgesic effects were noted with use of traditional AcP points.

Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) involves the application of adhesive electrodes to the skin with subsequent electrical stimulation of painful areas [99]. Different combinations of intensity and frequency of electrical stimulation may be applied, but high frequency is usually paired with low intensity, and vice versa. While there are no concrete guidelines describing what combination of these variables produces the greatest efficacy, it is believed that intensity is the most important factor and that the treatment should

Table 1 Summary of pharmacological treatment options for myofascial pain syndrome

Drug class	Commonly used drugs	Maintenance dose	Adverse effects	Mechanism of action	Demonstrated efficacy
NSAIDs	- Ibuprofen - Naproxen - Ketorolac - Indomethacin - Diclofenac	- 400-800 mg q6–8 h - 500-750 mg bid - 10 mg q6 h (IM/IV) for < 5 days - 50 mg tid/qid - 25 mg tid to 50 mg tid/qid	- GI symptoms - Renal injury - Nausea and vomiting - Dizziness	- COX inhibition	- RCT of 153 patients demonstrated that a topical diclofenac sodium patch provided pain relief and improved function in patients with MPS of the upper trapezius when compared with control of menthol patch [41].
TCA	- Doxepin - Nortriptyline - Amitriptyline - Imipramine - Desipramine	- Effective ranges are between 20 and 100 mg daily. Up to 300 mg daily. Nortriptyline has a maximum dose of 150 mg	Anticholinergic effects - Sedation - Dizziness - Confusion - Tachycardia - Weight gain	- NE and 5HT reuptake inhibition - Muscarinic acetylcholine receptor antagonism - Sodium channel blockade [47]	- RCT of 50 patients determined that amitriptyline and nortriptyline are both effective in reducing pain in patients with MPS [44].
Muscle relaxant	- Clonazepam - Orphenadrine - Cyclobenzaprine - Baclofen	- 0.25 qhs to 1 mg qid - 100 mg qhs/bid - 10 mg qhs to 20 mg tid - 5 mg tid to 20 mg qid	- Sedation - Cognitive dysfunction - Dizziness - Depression - Ataxia - Anticholinergic effects	- Benzodiazepine: acts on chloride channels ➔ enhances GABA-A receptors ➔ inhibition at presynaptic and postsynaptic sites on the spinal cord [48] - Cyclobenzaprine: inhibits reuptake of NE in locus coeruleus and inhibits descending serotonergic pathways in the spinal cord [47] - Baclofen: acts on potassium channels and acts presynaptically at GABA-B receptors in the spinal cord to reduce transmitter and releases potassium	- Meta-analysis encompassing 45 articles determined that both cyclobenzaprine and clonazepam were effective in reducing pain intensity in patients with temporomandibular disorders [89]. - Meta-analysis of 10 trials comparing use of tizanidine to baclofen and diazepam for the treatment of spasticity concluded that all three treatment options were equally effective [49].
Alpha-2-adrenergic agonist	- Tizanidine	- 2 mg qhs to start and increase by 2-4 mg every 1–4 days at breakfast, midday and bedtime. Max dose is 36 mg daily	- Sedation - Dizziness - Confusion - Dry mouth, asthenia, hypotension, constipation, bradycardia, urinary frequency, dyskinesia, xerostomia, and blurred vision	- Acts presynaptically at alpha-2 adrenergic receptors - Inhibits spinal motor neurons	- Tizanidine in treatment of MPS was found to be effective in relieving pain in 89% of patients.
Local anesthetics	- Lidocaine - Bupivacaine - Mepivacaine	- 0.5–2%; max dose 500 mg - 0.25–0.5%; max dose 200 mg - 1.0–1.5%; max dose 500 mg	- Anaphylaxis, CNS depression, seizures, arrhythmias Bupivacaine alters myocardial conduction	- Non-specific sodium channel blocker ➔ stabilizing neuronal cell membranes and inhibiting nerve impulse initiation and conduction	- Lidocaine injection therapy reduced the degree and frequency of neck pain in patients after 6 months of treatment [50]. −5% lidocaine patches provided pain relief when compared with the placebo group [51].
Botox	- Botulinum toxin	- Per guidelines of number of units per muscle	- Muscle weakness - Hypersensitivity reaction - Anaphylaxis - Autonomic dysreflexia - Respiratory compromise - Urinary retention - Myasthenia gravis	- Prevents ACh release to prevent muscle spasm - Prevents release of pain neurotransmitters at primary sensory neurons	- A meta-analysis of 13 studies concluded that pain was significantly reduced in groups that received botulinum toxin compared with placebo [15, 47, 48, 88].

produce a strong, but non-painful sensation for maximum efficacy. The mechanism of action of TENS may be multifactorial, but it is believed that the muscle contractions induced by TENS may normalize acetylcholine concentrations in the motor endplate, which may help relax taut bands of muscle [100••]. Although not thoroughly researched in the setting of MPS, what data there is for the efficacy of TENS in MPS appears to be modestly favorable.

Interferential Current Therapy

Interferential current (IFC) therapy involves the application of medium-frequency alternating currents, which is thought to increase blood flow and reduce pain [101, 102••]. It is said to be advantageous over TENS as it generates an amplitude-modulated frequency (AMF), allowing it to penetrate more deeply than TENS. Despite the efficacy, IFC has been demonstrated to be similar to that of TENS [101, 102••]. A few small studies have shown that IFC may be beneficial in the treatment of MPS, but more research is needed to determine its efficacy.

Biofeedback

Biofeedback is another non-pharmacological approach to managing myofascial pain. In this method, participants are able to receive real-time feedback on biological information like heart rate and muscle tone, which can then be interpreted by the participant and used to alter their behavior [103]. Information is usually displayed to the patient by visual display, sound, or vibration. For musculoskeletal pain syndromes like MPS, electromyogram biofeedback may provide information on the contraction of muscles to ensure that activities like stretching and exercise are being done appropriately. Biofeedback requires active participation by the patient and is also targeted at improving coping skills and psychological response to pain. Its use in MPS specifically has only been scarcely studied, so little is known about its efficacy.

Trigger Point Injections

Trigger point injections are the most common modality of treatment in managing resistant MPS [5, 104, 105]. Trigger point injections have been performed for years to treat musculoskeletal pain. Trigger points continue to be the hallmark physical examination sign of myofascial pain with a tender point in a taut band and a recognized or predicted pain referral [106]. While trigger point injections are performed for any type of pain, they are most commonly performed for head and neck pain. The upper trapezius, sternocleidomastoid, and temporalis muscles are commonly treated with trigger point injections for chronic neck pain and tension-type headache. One study showed that trigger points were present in 94% of the patients with migraine compared with 29% of controls [107]. While trigger point injections have been performed based on physical examination, ultrasound-guided MTrPs have been proposed [104]. A comprehensive review by Kumbhare et al. [104] described in detail the sonoanatomy of trigger points and correlation of physical characteristics with ultrasound examination. Their evidence synthesis identified 31 references of which only 2 studies used ultrasound to localize MTrP. The remaining studies most likely used a "blind technique." The review of these studies indicated that the blind technique provided variable improvements in visual analog scale pain ratings; however, the 2 studies using ultrasound demonstrated significant improvements in pain ratings, increased the LTR, and significantly reduced the number of MTrP needled, as well as the number of treatment sessions [108, 109]. Among the 2 studies, the larger one involving 133 patients by Bubnov and Wang [109] performed ultrasound-guided pterygopalatine muscle trigger point injections showing significant improvement in a large proportion of patients. They also studied 44 patients [108] with shoulder muscles pain, again showing significant improvements with ultrasound-guided injections.

Expert consensus methodology of trigger point injections for headache disorders described multiple randomized and observational studies showing improvement; however, myofascial trigger point injections may be associated with potential risks, even though they are minimally invasive techniques. Adverse effects have been described as fibrous and contractures, nerve injury, abscesses, gangrene, pneumothorax, and local and systematic reactions [104, 105, 110]. It has also been described that utilization of ultrasound may reduce the complication rates significantly with improvement in outcomes.

Additional Strategies

Given that MPS is typically chronic and challenging to treat, adjunctive strategies, not completely supported in current literature, are commonly employed, often with beneficial effects. Some additional alternative therapies include massage therapy, especially deep tissue massage, which has been utilized for centuries. Others include meditation and relaxation techniques, as well as tai chi. Finally, nutrition alterations, cannabis, and probiotics are described as potential productive therapies.

Conclusion

MPS is characterized by localized, taut regions comprised of skeletal muscle and fascia [15]. This review of current interventions used in the treatment of MPS demonstrates the need for additional evidence to support the use of both

pharmacologic and non-pharmacologic options. Presently, pharmacologic interventions with evidence to support their use in MPS include muscle relaxants such as benzodiazepines, tizanidine, and cyclobenzaprine; TCAs, and topical agents such as diclofenac gel and lidocaine patches, as well as injection therapy of Botox or lidocaine. Other modalities with some evidence to support their use include AcP, dry needling, and to a certain extent TENS therapy. Further large studies are needed to better ascertain the safety and efficacy of available treatment modalities for the management of MPS pain symptoms.

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Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

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