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Myofascial Pain Syndrome: An Update on Clinical Characteristics, Etiopathogenesis, Diagnosis, and Treatment

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ABSTRACT

Myofascial pain syndrome (MPS) is a chronic regional pain condition characterized by trigger points—hyperirritable spots within taut bands of muscle fibers that cause both localized and referred pain. The pathogenesis, diagnostic criteria, and classification of MPS are still under investigation, which complicates the development of standardized treatment protocols. Although diagnostic tools have improved, MPS often remains underrecognized due to symptom overlap with other pain disorders, such as fibromyalgia, neuropathic pain, and joint disorders. Factors contributing to its onset and persistence include muscle overuse, postural imbalance, systemic conditions, and psychological and behavioral influences. This narrative review explores the primary risk factors, current hypotheses on pathogenesis, diagnosis and differential diagnosis, and both conventional and emerging treatments. Sufficient evidence supports the use of local anesthetic injections for MPS. Some evidence suggests that dry needling, acupuncture, magnetic stimulation, ultrasound therapy, laser therapy, extracorporeal shock wave therapy, and manual therapy may be effective, particularly compared to sham or placebo. However, non-steroidal anti-inflammatory drugs, diclofenac, botulinum toxin, and transcutaneous electrical nerve stimulation show insufficient evidence, while the effectiveness of muscle relaxants, antidepressants, gabapentin, opioids, topical lidocaine, capsaicin, EMLA cream, and kinesio taping remains inconclusive. Effective management of MPS requires a patient-centered approach that integrates empirically supported and evidence-based treatments tailored to individual needs. This review synthesizes the current understanding of MPS and highlights the need for high-quality research to improve clinical decision-making in managing this complex condition.

Abbreviations: ACh, acetylcholine; AL-TENS, acupuncture-like transcutaneous electrical nerve stimulation; BTX-A, botulinum toxin type A; COX, cyclooxygenase; DN, dry needling; ESWT, extracorporeal shock wave therapy; FREMS, frequency-modulated neural stimulation; GABA, gamma-aminobutyric acid; IMMPACT, Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials; KOA, knee osteoarthritis; KT, kinesio taping; LA, local anesthetics; LLLT, low-level laser therapy; MPPS, myofascial pelvic pain syndrome; MPS, myofascial pain syndrome; MS, magnetic stimulation; MTrPs, myofascial trigger points; NGF, nerve growth factor; NSAID, non-steroidal anti-inflammatory drugs; PPT, pain pressure threshold; RCT, randomized control trial; rMS, repetitive magnetic stimulation; ROM, range of motion; SRMA, systematic review and meta-analysis; SSRI, selective serotonin reuptake inhibitor; TCAs, tricyclic antidepressants; TENS, transcutaneous electrical nerve stimulation; TMJD, temporomandibular joint disorder; TPI, trigger point injection; US, ultrasound.

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The objectives of this activity are to: (1) understand the risk factors for myofascial pain syndrome and consider these when taking a history and performing an examination; (2) be familiar with the differential diagnosis and with current diagnostic techniques to guide your evaluation of the patient presenting with possible myofascial pain syndrome; (3) be able to make choices among, and properly implement, pharmacological, needling, and nonpharmacological therapies.

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1 | Introduction

The term *myofascial* refers to the combination of muscle (*myo*) and the surrounding connective tissue. Myofascial pain syndrome (MPS) is a recurrent or chronic musculoskeletal condition characterized by localized pain and tenderness in specific regions of the muscle and fascia, often associated with the presence of hyperirritable nodules known as myofascial trigger points (MTrPs) [1]. These MTrPs are localized, hypersensitive nodules within muscle tissue and are classified as either active or latent [2]. Active MTrPs produce spontaneous pain, whereas latent MTrPs are clinically silent and become painful only upon appropriate stimulation, such as direct palpation or needling [3]. Regardless of their classification, MTrPs can cause restricted range of motion (ROM) and muscle weakness and may present with characteristic local and referred pain patterns, motor dysfunction, and autonomic symptoms [4].

MPS is often underdiagnosed or misdiagnosed due to an incomplete understanding of its nature, the lack of internationally validated diagnostic criteria, and the frequent overlap of its symptoms with those of other musculoskeletal pain disorders [5]. Consequently, the treatment of MPS is often inappropriate or delayed, leading to significant suffering and disability in affected patients [6]. In this review, we provide a comprehensive overview of the prevalence, risk factors, pathogenesis, diagnosis, and treatment of MPS, incorporating the latest research, guidelines, and clinical expertise.

2 | Prevalence

Estimates of the prevalence of MPS vary widely due to differences in diagnostic criteria, study methodologies, and the populations examined. Nevertheless, the condition is recognized as a major cause of musculoskeletal pain across various populations. Among middle-aged adults (30 to 60 years), the mean prevalence is reported to be 37% in men and 65% in women [7], with estimates reaching up to 85% among adults over 65 years [8]. In occupational settings, a study involving 243 female sewing machine operators (mean age: 38 years) found a prevalence of MPS of 15.2% in neck and shoulder muscles, compared to 9.0% in 357 control subjects [9].

In clinical settings, the prevalence of MPS appears notably high. In general internal medicine practice, one study found that among patients presenting with pain, 29.6% had MPS, making it the most common cause of pain [10]. Studies conducted in chronic pain clinics have reported even higher prevalence rates. At one pain rehabilitation referral center, 85% of patients were diagnosed with MPS [11]. A pain treatment referral program known for its focus on MPS reported that 93% of individuals with musculoskeletal pain had MTrPs, and in 74% of cases, MTrPs were considered the primary cause of pain [12].

Beyond these settings, several studies have investigated the prevalence of MPS in specific populations. One retrospective prevalence study showed that MPS was detected in 61% of individuals with complex regional pain syndrome [13]. One recent prospective cross-sectional study examined 224 patients previously diagnosed with non-specific neck pain and found that 100% of them also had MTrPs [14].

Among oncology populations, one study reported that 45% of breast cancer survivors developed MPS 1 year after surgery, with active MTrPs predominantly occurring in the shoulder girdle muscles [15]. Another study found that 11.9% of head and neck cancer patients developed MPS, particularly following surgical interventions or radiation treatments [16].

3 | Risk Factors

MPS is a multifactorial condition influenced by a range of intrinsic and extrinsic factors, which complicate both its diagnosis and management [17]. These risk factors can be broadly categorized into physical, systemic, psychological, and lifestyle-related factors, many of which interact synergistically, further contributing to the onset and persistence of MPS.

3.1 | Physical and Mechanical Factors

Muscle overuse is one of the most well-documented risk factors for MPS, particularly in individuals whose occupations or lifestyles require repetitive or sustained muscle contractions [18]. These overuse patterns can lead to microtrauma in muscle fibers, resulting in the formation of MTrPs [19]. This repetitive strain can occur in various settings, including manual labor, sports, and prolonged periods of poor posture, such as during desk work or driving. Additionally, acute muscle trauma, such as strains or tears, can initiate MTrP formation, setting off a cascade of pain and dysfunction that may persist long after the initial injury has healed [20].

Another significant contributor to MPS is postural imbalance, particularly when improper posture is sustained over long periods [21]. For instance, forward head posture, commonly associated with prolonged computer use, can lead to increased stress on the cervical and shoulder muscles, promoting the development of MTrPs. Postural dysfunctions can also arise from musculoskeletal abnormalities such as scoliosis, where uneven muscle tension places certain muscle groups under constant strain, increasing their susceptibility to MTrP formation [22, 23]. Similarly, conditions such as osteoarthritis, particularly in the spine and major joints, can alter muscle recruitment patterns, causing compensatory overuse in adjacent muscle groups, which may contribute to chronic pain [17, 24].

3.2 | Systemic and Metabolic Conditions

Systemic factors, including vitamin deficiencies and metabolic disorders, have also been implicated in the development of MPS [25]. For example, vitamin D deficiency has emerged as a notable risk factor. A recent cross-sectional study found that approximately one-third of patients with MPS had vitamin D deficiency [26]. This deficiency can impair muscle function and contribute to the persistence of MTrPs [27]. Similarly, hypothyroidism is associated with generalized muscle weakness

and fatigue, which predispose individuals to the development of MTrPs by reducing the muscle's ability to recover from everyday stress [28].

Iron deficiency, particularly in premenopausal women, has been linked to increased muscle fatigue and reduced muscle oxygenation, both of which can contribute to MTrP formation [29]. Muscle tissue requires adequate oxygen and nutrients for normal function, and deficiencies in essential elements like iron, magnesium, and calcium can lead to disturbances in muscle contraction and relaxation, making MTrPs more likely to develop [2]. These systemic risk factors highlight the importance of maintaining nutritional and endocrine health in the prevention and management of MPS.

3.3 | Psychological and Emotional Factors

Psychological stress plays a central role in the development and exacerbation of MPS. Chronic stress, anxiety, and tension can lead to prolonged muscle contraction, particularly in areas like the neck, shoulders, and jaw [30, 31]. This sustained muscle activation can create a cycle of pain and dysfunction, as MTrPs are more likely to form in muscles that are constantly under tension. Stress also increases the body's sensitivity to pain, which can worsen the symptoms of MPS [32].

Depression has been linked to a higher prevalence of MPS, with chronic pain patients often exhibiting depressive symptoms that can exacerbate their condition [33]. The relationship between depression and MPS may be bidirectional; individuals suffering from chronic pain are more likely to develop depressive symptoms, while depression can heighten the perception of pain [34, 35].

3.4 | Lifestyle and Behavioral Factors

A sedentary lifestyle significantly increases the risk of developing MPS, as prolonged inactivity contributes to muscle stiffness and reduced flexibility [36]. Individuals who do not engage in regular physical activity are more prone to muscle deconditioning, which can exacerbate MTrP formation and prolong the duration of symptoms. Regular exercise, on the other hand, helps maintain muscle health by improving flexibility, strength, and endurance, thereby reducing the likelihood of MPS [37, 38].

Oral parafunctional behaviors, such as teeth grinding (bruxism) and jaw clenching, are particularly common in patients with temporomandibular joint disorders (TMJD) and are known to contribute to MPS, especially in the orofacial and neck regions [31, 39]. Bruxism creates chronic tension in the masticatory muscles, leading to the activation of MTrPs in the jaw and face. This can result in referred pain patterns that exacerbate temporomandibular joint-related symptoms and complicate diagnosis and treatment [40].

Sleep disturbances, particularly insomnia, are another critical risk factor for MPS. Poor sleep quality has been linked to heightened pain sensitivity and reduced muscle recovery, both of which contribute to the persistence of MTrPs [41]. A longitudinal population-based study demonstrated that individuals with insomnia had a significantly higher risk of developing MPS than those with healthy sleep patterns, underscoring the importance of addressing sleep hygiene in the management of chronic pain [42].

4 | Hypothetical Pathogenic Mechanisms

The pathophysiology of MPS is complex and not yet fully understood. However, several hypotheses, theories, and mechanisms have been proposed to explain the development of MTrPs. These proposed mechanisms describe the peripheral and central effects observed in MPS. The concept of pain originating from muscle and/or fascia, felt locally and at a distant site ("referred pain"), was first introduced by Kellgren [43] and later expanded upon by Hockaday and Whitty [44] and Mense et al. [45–47]. Kellgren investigated pain patterns induced by stimulating deep muscle, fascia, and tendon through injections of small quantities of hypertonic saline. He demonstrated that this stimulation produced referred pain following a spinal segmental pattern and found that referred pain from muscle was associated with referred tenderness in deep structures [43]. Based on these observations, Kellgren postulated that the mechanism of pain referral could involve a common pathway shared by muscular and deep somatic structures. Hockaday and Whitty reproduced Kellgren's findings, demonstrating that hypertonic saline injections into interspinous ligaments commonly induced referred pain [44]. Mense et al. used a rat model to show that local increases in acetylcholine could create abnormally contracted muscle fibers [47]. These observational studies were the starting point for the development of the hypotheses described in this section.

4.1 | Cinderella Hypothesis

The Cinderella hypothesis describes how musculoskeletal disorder symptoms may arise from muscle recruitment patterns during sub-maximal exertions involving moderate or low physical loads [48, 49]. According to Henneman's "size principle," smaller type I muscle fibers are activated before larger ones and are deactivated after them [49]. Consequently, these smaller muscle fibers are continuously engaged during prolonged motor tasks. In sub-maximal exertions, such as sustained muscle contractions, only a fraction of the available motor units are used, without the typical substitution that occurs during higher-force contractions [50, 51]. This leads to metabolic overload in these smaller motor units, making them susceptible to a loss of cellular calcium (Ca^{2+}) homeostasis, activation of autogenic destructive processes, and the onset of muscle pain. The hypothesis is therefore named the Cinderella hypothesis because these smaller muscle fibers are continuously activated during prolonged tasks ("always working") without adequate rest, much like the fairy tale character Cinderella [52].

Growing evidence supports the idea that low-level static muscle exertions can cause degeneration of muscle fibers [53]. Studies have shown that such exertions increase Ca^{2+} release in skeletal muscle cells, cause damage to muscle membranes due to leakage of intracellular enzymes like lactate dehydrogenase, lead to structural damage and energy depletion, and result in myalgia

[54, 55]. Additionally, low-level muscle stimulation has been found to trigger the release of interleukin-6 and other cytokines [56, 57].

Several studies have applied the Cinderella hypothesis to the development of MTrPs. Two studies demonstrated that sustained low-level muscle contractions, such as continuous typing or playing the piano, frequently result in the formation of MTrPs [58, 59]. Other research has shown that MTrPs are common among occupational groups exposed to prolonged low-level muscle exertions, including office workers, dentists, and musicians [60].

4.2 | Neuromuscular Junction Dysfunction

MTrPs are hypothesized to result from physiological dysfunctions at the neuromuscular junction and within the surrounding connective tissue. Evidence suggests that motor endplates innervating muscle fibers within MTrPs exhibit abnormal activity. Electromyographic studies have demonstrated spontaneous electrical activity in the form of endplate noise and spikes at MTrP sites, which is absent in adjacent tissue [61–63]. Initially, this excessive electrical activity was attributed to dysfunctional muscle spindles; however, it was later identified as increased miniature endplate potentials resulting from excessive release of acetylcholine (ACh) [64, 65]. The presence of dysfunctional motor endplates in MTrP tissue may explain the taut band phenomenon characteristic of these trigger points [32].

4.3 | Integrated Trigger Point Hypothesis

Simons' Integrated Trigger Point Hypothesis proposes a similar explanation for the pathophysiology underlying MTrPs [66]. According to this hypothesis, repetitive microtrauma to muscle fibers leads to an excessive release of ACh, resulting in increased motor endplate activity (noise and spikes), which mediates the manifestation of a localized, palpable, hyperirritable locus within the peripheral muscle [32]. This persistent contraction leads to a cascade of biochemical responses, including increased concentrations of protons (H+) and the release of vasoactive components and inflammatory mediators such as histamines and bradykinin [67, 68]. The lowered pH and accumulation of these biochemicals in active MTrPs contribute to the expression of localized muscle pain [69].

4.4 | Central Sensitization

Central sensitization refers to the heightened responsiveness of neurons in the central nervous system, particularly in the dorsal horn of the spinal cord, to nociceptive input from the periphery [70]. This phenomenon amplifies pain signals, leading to exaggerated responses to both painful and non-painful stimuli. Central sensitization develops due to persistent peripheral nociceptive input from MTrPs and contributes to the persistence and spread of pain beyond the original site of injury [32]. Research has shown that repeated activation of peripheral nociceptors by MTrPs releases substance P into the dorsal horn, which induces neuroplastic changes (increased excitability) within the central nervous system [71, 72]. As a result, these neurons respond more intensely to nociceptive input, further sensitizing the patient to pain and contributing to its chronic nature. This mechanism may explain why patients with MPS often experience widespread or referred pain extending beyond their local MTrPs.

4.5 | Neurogenic Inflammation

Neurogenic inflammation has been proposed as a significant contributor to the pathophysiology of MPS, particularly by promoting the sensitization of spinal circuits [73] and motor neurons following central sensitization [74]. It occurs when nociceptive fibers—responsible for transmitting pain signals—release inflammatory mediators such as histamines, substance P, and calcitonin gene-related peptide [75]. These mediators cause vasodilation, increase vascular permeability, and promote the further release of pro-inflammatory cytokines in the surrounding muscle tissue [32]. This inflammatory response amplifies the sensitivity of nociceptors, making the affected area more susceptible to pain from stimuli that are not typically painful—a condition known as allodynia [64]. Neurogenic inflammation also contributes to peripheral sensitization by lowering the threshold for nociceptor activation, leading to exaggerated pain responses [64]. This process is also thought to be one of the mechanisms that allow MTrPs to persist and remain hyperalgesic over time [69, 76]. The continuous release of inflammatory mediators and their impact on local muscle tissues may further explain why MPS often becomes a chronic condition. Studies have shown that neurogenic inflammation, following central sensitization, may initiate and facilitate the formation of localized MTrPs even in the absence of local peripheral muscle injury [77].

4.6 | Densification of Fascia

Another proposed mechanism in the pathophysiology of MPS involves pathological changes in the muscular fascia—the connective tissue that surrounds muscles. Stecco et al. suggested that under conditions of overload and damage, the fascia can undergo densification, leading to alterations in the biomechanical properties of muscle tissue [78]. This densification results in reduced muscle contraction force and decreased flexibility [79]. Furthermore, the inflammatory processes previously discussed may exacerbate these pathological changes, intensifying pain [80, 81]. These alterations in the muscular fascia are thought to be associated with abnormal changes in myofibrils, fibroblasts, and the extracellular matrix [78, 81]. Refer to Table 1 for a summary of these mechanisms and Figure 1 for an illustration of the interplay between central and peripheral contributions to MPS.

5 | Current Method of Diagnosis

Diagnosing MPS remains challenging due to the absence of widely accepted, objective, and standardized criteria. The diagnosis is typically based on subjective clinical criteria, including patient-reported pain, the exclusion of other pathologies, and the clinical palpation of MTrPs [82]. However, these criteria are not standardized, leading to variability in the diagnosis of MPS among clinicians.

TABLE 1 | Summary of hypothetical pathogenic mechanisms for myofascial pain syndrome.

Mechanistic hypothesis	Central or peripheral mechanism	Key features	Proposed pathophysiological process
Cinderella hypothesis [48–60]	Peripheral	– Sustained low-level muscle contractions – Continuous activation of small type I muscle fibers	– Metabolic overload in smaller motor units – Disruption of cellular calcium homeostasis – Release of cytokines (e.g., interleukin-6) – Onset of muscle pain
Neuromuscular junction dysfunction [32, 61–65]	Peripheral	– Abnormal activity at motor endplates – Spontaneous electrical activity at MTrP sites	– Excessive ACh release – Increased miniature endplate potentials – Taut band phenomenon characteristic of MTrPs
Integrated trigger point hypothesis [32, 66–69]	Peripheral	– Repetitive microtrauma – Excessive ACh release and pH reduction	– Persistent muscular contraction – Release of inflammatory mediators (e.g., histamines, bradykinin) – Biochemical cascade contributing to localized pain
Central sensitization [32, 70–72]	Central	– Increased responsiveness of CNS neurons – Amplified pain signaling	– Persistent nociceptive input from MTrPs – Substance P release inducing neuroplastic changes – Pain spreading beyond the original injury site
Neurogenic inflammation [32, 64, 69, 73–77]	Central and peripheral	– Release of inflammatory mediators (e.g., substance P, histamines) – Vasodilation and increased vascular permeability	– Amplified nociceptor sensitivity – Peripheral and central sensitization – Persistent hyperalgesia and allodynia
Densification of fascia [78–81]	Peripheral	– Pathological changes in the muscular fascia – Altered biomechanics	– Fascia densification reducing contractile force and flexibility – Increased inflammation exacerbating pain – Extracellular matrix and fibroblast alterations

Abbreviations: ACh, acetylcholine; CNS, central nervous system; MTrP, myofascial trigger point.

The most commonly used diagnostic criteria for MPS were proposed by David Simons in 1999, requiring the presence of five major and at least one minor criterion [17]. Major criteria include localized and spontaneous pain, referred pain patterns, a taut palpable band within the muscle, tenderness at the MTrP, and reduced range of movement. Minor criteria involve the reproduction of pain upon pressure at the MTrP, the elicitation of a local twitch response, and pain relief following muscle stretching or MTrP injection [83]. While these criteria have been important in clinical practice, physical examination has been shown to vary between practitioners, leading to inconsistencies in diagnosis [84–86].

Since the subjective nature of these criteria poses challenges to the reliability and consistency of MPS diagnosis, recent efforts have focused on developing more objective measures for diagnosing MPS [87]. Techniques such as quantitative sensory testing, algometry, and conditioned pain modulation assess pain sensitivity and central processing through controlled stimuli and pain responses, offering insights into sensory abnormalities associated with MPS [88–98]. Imaging techniques, including magnetic resonance imaging, magnetic resonance elastography, ultrasound (US), and infrared thermal imaging, are being explored to visualize structural and functional changes in muscle tissue and assess blood flow and stiffness differences associated with MPS [99–108]. Vibration elastography provides a non-invasive method to measure stiffness differences in taut bands, helping to distinguish them from surrounding healthy tissue [109]. Electromyography identifies spontaneous electrical activity at trigger points, potentially reflecting abnormal ACh release

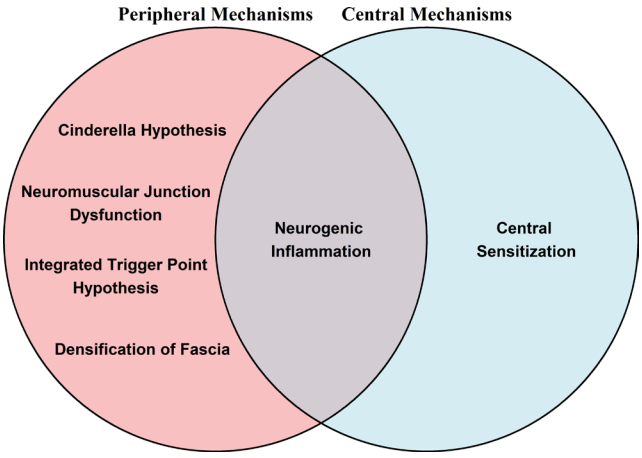

FIGURE 1 | Venn diagram illustrating the interplay between central and peripheral mechanisms contributing to myofascial pain syndrome.

at the neuromuscular junction in MPS patients [32, 110]. Despite their potential, these diagnostic aids are still in the early stages of development and have yet to be widely adopted in clinical practice. Refer to Table 2 for a summary of diagnostic modalities along with their primary advantages and limitations.

6 | Differential Diagnosis

The diagnosis of MPS is also challenging because its clinical presentation may overlap with several other chronic pain conditions, including fibromyalgia, neuropathic pain conditions, and joint disorders. Refer to Figure 2 for a flow diagram illustrating our proposed clinical diagnostic evaluation for patients with suspected MPS.

6.1 | MPS Versus Fibromyalgia

MPS and fibromyalgia are two common pain syndromes that often present with overlapping symptoms, making differential diagnosis challenging due to the absence of validated gold-standard criteria [3, 82, 111]. The clinical distinction between them currently relies on a thorough patient history and physical examination [112].

A key determinant in differentiating MPS from fibromyalgia is the nature of the tender regions and the pattern of pain presentation [112]. MPS is typically characterized by regionally distributed pain associated with discrete MTrPs [111]. These MTrPs are located within taut bands of muscle fibers and, when compressed, produce stereotypical referred pain patterns. They may elicit a local twitch response upon snapping palpation (i.e., rapid compression across muscle fibers) and are often responsive to local treatments [113].

In contrast, fibromyalgia patients present with widespread pain lasting longer than 3 months, without clear localization to specific muscles, and with symmetrically distributed tender regions within soft tissues [114–116]. These tender regions are sites of exquisite tenderness but differ from MTrPs in several ways: they are not located within taut bands of muscle fibers, do not elicit a local twitch response upon snapping palpation, do not give rise to pain at distant sites upon local stimulation, and are generally unresponsive to local treatments targeting specific points [112, 117]. This suggests that maladaptive central processing may be an important underlying mechanism driving the clinical features of fibromyalgia.

The underlying pathophysiology further distinguishes the two conditions. MPS involves localized peripheral tissue changes, including elevated levels of inflammatory mediators in MTrPs [69]. Conversely, tender regions in fibromyalgia do not typically exhibit these inflammatory changes, supporting the notion of central sensitization rather than peripheral tissue pathology [118].

Differentiating MPS from fibromyalgia is complicated by several factors. The nonspecific nature of localized tender regions and associated pain is common to various clinical conditions [112]. Moreover, MPS, although primarily a regional pain syndrome, can become widespread and persist for more than 3 months, mirroring the chronicity of fibromyalgia [119]. Consequently, patients may exhibit characteristics and symptoms that satisfy criteria for both syndromes. Clinically, we recommend considering the following classification: regional pain without central sensitization, regional pain with central sensitization, and widespread pain (which has central sensitization) [115, 116, 120].

6.2 | MPS Versus Neuropathic Pain Conditions

Neuropathic pain conditions, including radiculopathies and peripheral neuropathies, often mimic MPS due to overlapping symptoms, such as radiating or referred pain patterns [121, 122]. Radiculopathies involve compression or irritation of the nerve roots, resulting in pain, numbness, and weakness along the distribution of the affected nerve root [123]. Similarly, peripheral neuropathies arise from damage to peripheral nerves and typically present with burning, tingling, or electric shock-like sensations alongside pain [124].

Despite the similarities, there are distinct differences. Neuropathic pain is characterized by specific neurological signs, including sensory deficits and, when motor nerve fibers are involved, muscle weakness and altered reflexes—features that are absent in MPS [125]. Additionally, while neuropathic pain patterns align with dermatomal distributions corresponding to affected nerve pathways [125], the referred pain patterns in MPS originate from trigger points in specific muscles and do not follow a dermatomal pattern [30]. A thorough neurological examination is crucial, as it can help identify sensory abnormalities, muscle weakness, and reflex changes that are hallmarks of neuropathic conditions.

6.3 | MPS Versus Joint Disorders

MPS often overlaps with joint-related disorders, such as osteoarthritis, bursitis, tendinopathies, and TMJD, as these conditions present with localized musculoskeletal pain [126, 127]. Both MPS and joint disorders may present with pain during movement, making it challenging to differentiate them based

TABLE 2 | Common diagnostic aids for myofascial pain syndrome.

Modality	Diagnostic strategy	Main advantages	Main disadvantages
Quantitative sensory testing [88, 91, 96, 97]	Applies repeated, controlled stimuli (e.g., pressure, heat, or vibration) and assess altered sensory experiences	Clinical measure of central processing	Methodological differences across studies; relies on patient self-report; lacks a detailed description of MTrPs
Algometry [90, 93, 94]	Applies increasing pressure using an algometer and correlates pain response with the reading	Quantifies pain relative to applied pressure	Methodological differences across studies; relies on patient self-report; algometry can be more painful than palpation and is less specific; lacks a detailed description of MTrPs
Ultrasound [103–105]	Detects hypoechoic regions, taut bands, and reduced vibration amplitude in muscle tissue	Identifies non-palpable trigger points; visualizes structural changes in muscle tissue	Diagnostic accuracy not fully validated; requires operator training
Vibration elastography [109]	Measures stiffness differences between taut bands and surrounding tissue	Non-invasive, quantitative stiffness assessment	Limited clinical availability; requires specialized training and equipment
Magnetic resonance imaging [106, 107]	Visualizes T2 signal alterations, increased stiffness, and changes in brain-related pain modulation	High-resolution imaging; identifies structural and functional alterations	High cost; limited accessibility; not routinely used in clinical settings
Magnetic resonance elastography [99]	Measures vibratory displacements to assess taut band stiffness	Non-invasive; quantitative assessment of stiffness	High cost; limited accessibility; does not directly target MTrPs
Infrared thermal imaging [100–102, 108]	Detects temperature changes overlying MTrPs, indicating altered microcirculation	Non-invasive; visualizes regional temperature patterns	Lacks specificity; influenced by external factors; lacks strong clinical validation
Electromyography [32, 110]	Detects spontaneous electrical activity (endplate noise) at MTrPs	Provides direct evidence of electrical abnormalities in MTrPs	Requires expertise; findings vary across studies; invasive
Conditioned pain modulation [89, 92, 95, 98]	Tests descending pain inhibitory system by applying a noxious stimulus in one area and assessing pain response in another	Differentiates central sensitization; identifies responders and non-responders	Requires specialized equipment; limited clinical use; influenced by variability among individuals

Abbreviation: MTrPs, Myofascial trigger points.

solely on pain location [128]. However, MPS is primarily characterized by the presence of discrete MTrPs, which produce referred pain that may not correspond directly to joint involvement [2, 129]. For example, shoulder pain in MPS may originate from MTrPs in the trapezius or infraspinatus muscles rather than from the glenohumeral joint itself. Similarly,

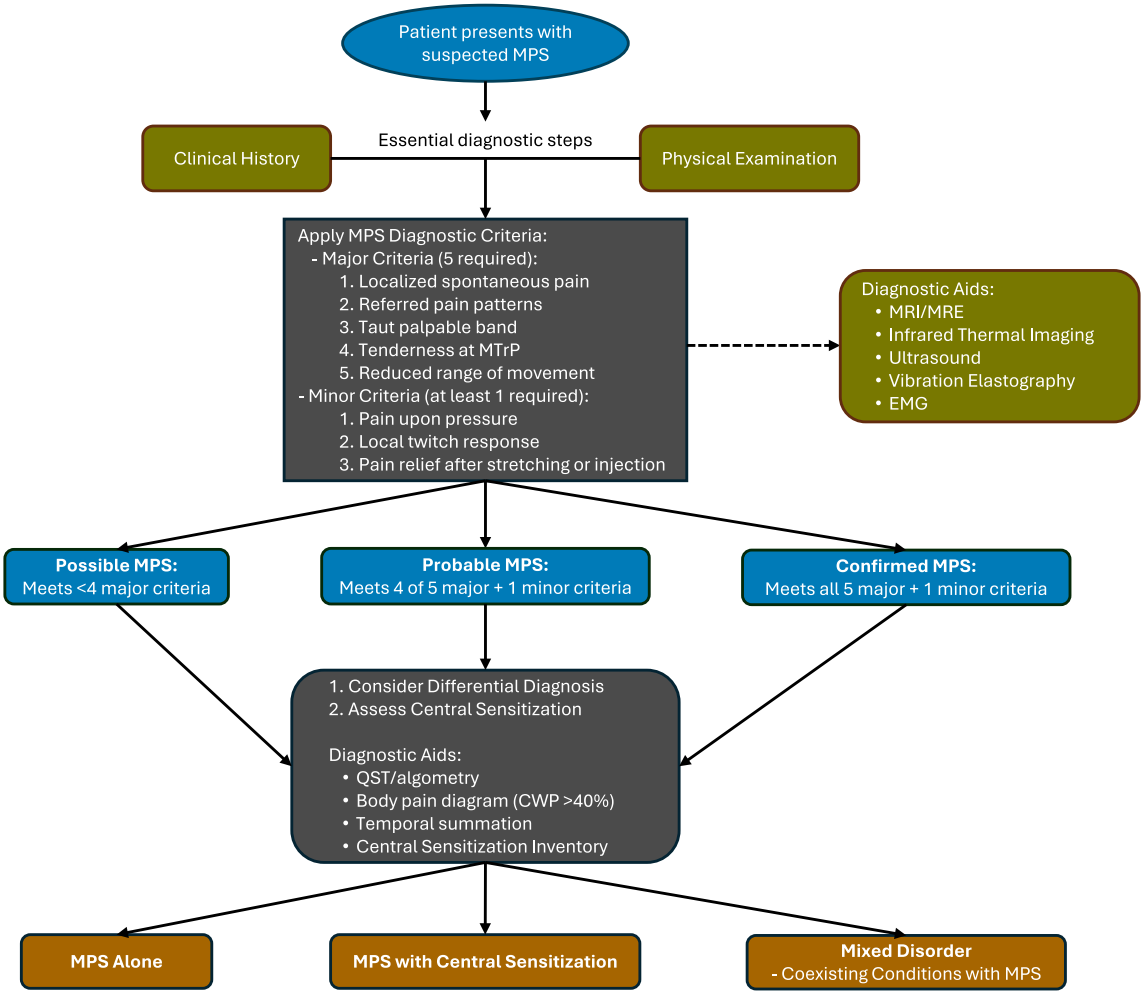

FIGURE 2 | Flowchart depicting the proposed diagnostic evaluation pathway for patients with suspected myofascial pain syndrome. CWP, chronic widespread pain; EMG, electromyography; MPS, myofascial pain syndrome; MRE, magnetic resonance elastography; MRI, magnetic resonance imaging; MTrP, myofascial trigger point; QST, quantitative sensory testing.

TMJD and MPS can both cause pain in the jaw, neck, and facial muscles [130]. However, TMJD pain is typically localized to the temporomandibular joint and is associated with joint-specific dysfunctions, such as clicking, locking, or reduced jaw movement [131]. In contrast, MPS-related pain radiating to the jaw and face is often traced back to MTrPs in muscles like the masseter, temporalis, or sternocleidomastoid [2]. Within the TMJD literature, a model for studying chronic pain has been developed that uses repeated intra-muscular injections of nerve growth factor (NGF) to induce progressively worsening muscle pain, mechanical hyperalgesia, and functional limitations. NGF contributes to these effects by sensitizing peripheral nociceptive terminals, altering nociceptor transcription, and promoting nociceptor sprouting [132, 133]. With future research, the NGF pain model may improve our understanding of the pathophysiology of MPS.

A detailed clinical examination is essential to differentiate MPS from joint disorders, including TMJD [126]. In MPS, palpation of MTrPs reproduces the patient's characteristic pain patterns [2], while in joint disorders, pain is more closely linked to joint movement and loading [134]. Additionally, radiographic evidence of joint degeneration or inflammation is more commonly seen in joint disorders [135], whereas MPS is associated with muscle activation and the presence of taut bands [2].

In cases where both MPS and joint pathology coexist, effective pain management requires addressing both the joint dysfunction and myofascial components [136]. For example, TMJD may coexist with MTrPs in the masticatory muscles, requiring targeted treatments for both joint dysfunction and myofascial pain to achieve optimal results. Please refer to Table 3 for a summary of the key characteristics and distinctions used in the differential diagnosis of MPS.

7 | Treatment

7.1 | Pharmacological Treatment

7.1.1 | Muscle Relaxants

Several muscle relaxants have been investigated for their effectiveness in treating MPS, including cyclobenzaprine, tizanidine, baclofen, and thiocolchicoside. These medications act on the central nervous system to reduce muscle tone and disrupt

TABLE 3 | Differential diagnosis of myofascial pain syndrome: key characteristics and distinctions.

Characteristics	MPS [1–4, 30, 69, 111–113, 119, 129]	Fibromyalgia [112, 114–118]	Neuropathic pain [123–125]	Osteoarthritis (representative joint disorder) [130–135]
Pain distribution	Regionally distributed pain associated with discrete MTrPs within taut bands	Widespread pain lasting longer than 3 months; symmetrically distributed tender regions	Radiating pain along dermatomal distributions	Localized pain linked to joint movement and dysfunction; MTrPs can cause referred pain
Trigger points/ tenderness	Trigger points elicit a local twitch response, referred pain, and reproduce characteristic pain patterns upon palpation	Tender regions are not located in taut bands, do not elicit local twitch responses, or produce referred pain	Pain follows nerve root pathways, often accompanied by burning or tingling sensations	Discrete MTrPs may be present, pain originates from joint dysfunction
Pathophysiology	Localized inflammatory mediators in MTrPs; peripheral tissue changes; may also have central sensitization	Central sensitization is the main mechanism; absence of peripheral inflammatory changes	Nerve root or peripheral nerve irritation or damage; altered sensory and reflex functions may be present	Joint-specific pathology such as degeneration or inflammation; nociceptive pain is the primary mechanism
Response to palpation	MTrP palpation reproduces characteristic pain patterns and may trigger local twitch responses	Tender regions (not trigger points) are sore but do not produce referred pain or elicit a twitch response; MTrPs may be present and indicate co-existing MPS	Neurological signs (e.g., sensory deficits, altered reflexes) are present and localized palpation does not reproduce pain	Palpation of joint may not reproduce pain; MTrP palpation reproduces characteristic pain patterns if MPS is present; palpation of the joint may elicit pain, resulting in both joint dysfunction and MPS contributing to overall pain
Response to treatment	Local treatments like needling or manual therapy often effective	Generally unresponsive to local treatments; systemic therapies targeting central mechanisms are more effective	Requires treatment targeting the underlying nerve condition	Joint dysfunction requires targeted interventions; MPS-related pain requires treatment addressing both myofascial and joint components

(Continues)

TABLE 3 | (Continued)

Characteristics	MPS [1–4, 30, 69, 111–113, 119, 129]	Fibromyalgia [112, 114–118]	Neuropathic pain [123–125]	Osteoarthritis (representative joint disorder) [130–135]
Associated symptoms	Reduced ROM, muscle stiffness, referred pain, local twitch response	Fatigue, cognitive dysfunction, depression, and systemic symptoms such as numbness, diaphoresis, and temperature changes	Burning, tingling, or electric shock-like sensations	Joint clicking, locking, or reduced movement; if MPS coexists, referred pain and local twitch response may also be present
Key differentiators	Pain is localized to specific MTrPs; referred pain does not follow a known dermatomal pattern and is associated with localized peripheral tissue pathology	Pain is widespread, not localized, and involves central sensitization mechanisms	Neurological signs like sensory deficits and pain distribution within dermatome, nerve root or nerve(s)	Radiographic evidence of joint degeneration or inflammation; MTrPs in associated muscles may coexist

Abbreviations: MPS, myofascial pain syndrome; MTrP, myofascial trigger point; ROM, range of motion.

reflexes that contribute to muscle contraction, thereby providing pain relief and improving functional mobility [137]. While muscle relaxants are well-studied in populations with general musculoskeletal pain [138], evidence specific to their use in MPS patients is limited.

7.1.1.1 | Cyclobenzaprine. Cyclobenzaprine is a centrally acting muscle relaxant structurally related to tricyclic antidepressants and is used to relieve skeletal muscle spasms without impairing muscle function [139]. One Cochrane review found insufficient evidence to support the use of cyclobenzaprine for the treatment of MPS [140]. One randomized controlled trial (RCT) on orofacial MPS included in this review reported that cyclobenzaprine significantly improved short-term pain compared to clonazepam and placebo, but not sleep quality [141]. Conversely, a more recent RCT on orofacial MPS found no significant differences between cyclobenzaprine and placebo for both pain and sleep quality [142]. Cyclobenzaprine may provide potential benefits for short-term MPS-related pain; however, the evidence is insufficient compared to placebo and alternative therapies.

7.1.1.2 | Tizanidine. Tizanidine is a centrally acting alpha-2 adrenergic receptor agonist with both antispastic and antispasmodic properties [143]. One RCT on orofacial MPS reported no significant differences in pain or sleep quality between tizanidine and placebo [142]. In contrast, a cohort study involving females with MPS found that 5 weeks of tizanidine treatment improved pain intensity, disability, pain pressure threshold (PPT), and sleep quality [144]. Another cohort study on patients with myofascial pain of the masticatory muscles reported only slight improvements in pain after 2 weeks of tizanidine treatment, with many painful sites persisting [145]. While tizanidine may offer potential benefits for MPS-related pain, disability, and sleep quality, the evidence remains insufficient to determine its effectiveness compared to placebo, as conclusions are drawn from only one RCT and mixed findings from observational studies.

7.1.1.3 | Baclofen. Baclofen is a gamma-aminobutyric acid (GABA)-B receptor agonist that inhibits excitatory neurotransmitter release, leading to reduced spasticity [146]. One RCT involving MPS patients reported slight improvements in pain and function over a 3-week follow-up period; however, these outcomes were not significantly different from those observed in patients taking other muscle relaxants (chlorzoxazone, phenprobamate, mephenoxalone) or in the control group [147]. While baclofen may offer potential benefits for MPS-related pain and function, the evidence is insufficient to determine its effectiveness compared to placebo or alternative therapies, based on findings from only one RCT.

7.1.1.4 | Thiocolchicoside. Thiocolchicoside is a competitive GABA-A receptor antagonist that provides anti-inflammatory, analgesic, and muscle-relaxing effects [148]. One RCT evaluated the efficacy of a fixed-dose combination of thiocolchicoside and aceclofenac versus chlorzoxazone, aceclofenac, and paracetamol in MPS patients [149]. The group receiving thiocolchicoside and aceclofenac demonstrated significantly greater pain improvement after 1 week compared to the other group [149]. Another RCT found that US therapy

combined with a gel containing diclofenac and thiocolchicoside improved pain and mouth opening more than US therapy alone in patients with masticatory MPS [150]. A case–control study reported significant improvements in pain and quality of life in MPS patients when combining mesotherapy with thiocolchicoside, though these improvements were less pronounced than those achieved with extracorporeal shock wave therapy (ESWT) [151]. While thiocolchicoside may provide benefits for pain, quality of life, and ROM in MPS when used alongside other therapies, the evidence remains insufficient to determine its effectiveness. Current findings are limited to two RCTs and one case–control study, none of which included direct comparisons to placebo.

7.1.2 | Antidepressants

7.1.2.1 | Tricyclic Antidepressants (TCAs). Amitriptyline and nortriptyline are tricyclic antidepressants (TCAs) commonly used in chronic pain management for their analgesic effects, achieved by inhibiting the reuptake of norepinephrine and serotonin, which enhances descending pain modulation pathways [152, 153]. One RCT reported significant improvements in myofascial tenderness and headache intensity with amitriptyline compared to placebo in patients with chronic tension-type headaches [154]. Another clinical trial on MPS and TMJD found significant pain reductions at 6 weeks and 1 year with amitriptyline use, although the effect diminished over time, and no improvement was observed in depression scores [155]. An observational study reported clinically meaningful pain reductions for both amitriptyline and nortriptyline in patients with masticatory myofascial pain, with nortriptyline showing slightly greater efficacy [156]. While amitriptyline and nortriptyline have shown short- to medium-term benefits in reducing pain for MPS patients, there is insufficient evidence to determine their long-term effectiveness.

7.1.2.2 | Serotonergic Modulators. Citalopram, a selective serotonin reuptake inhibitor (SSRI), increases serotonin availability by blocking its reuptake, potentially modulating pain perception through its effects on neurotransmission in the central nervous system [157, 158]. However, an RCT in patients with chronic tension-type headaches found no significant differences between citalopram and placebo in reducing myofascial tenderness, headache intensity, or PPT [154].

Sumatriptan, a 5-HT1B/1D receptor agonist used for its vasoconstrictive and pain-modulating effects [159], showed limited effectiveness in a crossover pilot study on temporalis muscle MPS, with no significant differences observed compared to placebo [160].

Tropisetron, traditionally used to manage chemotherapy-induced nausea, has demonstrated potential analgesic effects in musculoskeletal pain by inhibiting the release of substance P and neuropeptides from nociceptors [161]. An RCT found that local trigger-point injections of tropisetron significantly reduced pain intensity compared to baseline at 7 days post-injection [161].

There is insufficient evidence to determine the effectiveness of citalopram, sumatriptan, or tropisetron for managing MPS, as limited RCTs show no significant improvements in outcomes compared to placebo.

7.1.3 | Anticonvulsants

7.1.3.1 | Gabapentin. Gabapentin, commonly prescribed for neuropathic pain, reduces neuronal excitability by binding to the α2-δ1 subunit of voltage-gated calcium channels [162]. While no RCTs have specifically evaluated gabapentin for MPS, observational data from open-label trials suggest some within-group pain improvements [163]. However, when compared to TCAs, gabapentin was less effective, with fewer patients experiencing significant pain relief [164]. Gabapentin may offer potential benefits for MPS-related pain, but there is insufficient evidence to determine its effectiveness compared to alternative therapies, as findings are based on limited observational data.

7.1.4 | Analgesics

7.1.4.1 | Nonsteroidal Anti-Inflammatory Drugs (NSAIDs). Flurbiprofen, a non-selective cyclooxygenase (COX) inhibitor, exerts anti-inflammatory and analgesic effects [165]. One RCT in MPS patients found that oral flurbiprofen significantly improved pain severity, tenderness, ROM, and quality of life within the treatment group; however, these outcomes were not superior to those achieved with dry needling (DN) or lidocaine injections [166].

Ibuprofen, another non-selective COX inhibitor, also provides anti-inflammatory and analgesic benefits [167]. In an RCT on masticatory MPS, patients receiving ibuprofen (400 mg every 12 h for 3 weeks) showed no significant improvements in pain or mouth opening at any time point, and ibuprofen was inferior to laser treatment in both outcomes [168]. Another RCT on myogenous fascial pain reported significant pain improvements with diazepam and a combination of diazepam and ibuprofen, but not with ibuprofen alone [169].

Nimesulide is a selective COX-2 inhibitor that has anti-inflammatory and analgesic properties [170]. An RCT on patients with MPS and unilateral temporomandibular joint pain found that combining an occlusal appliance with nimesulide significantly improved orofascial pain and sleep quality compared to using the appliance alone or in combination with DN [171].

Evidence is insufficient to support the effectiveness of NSAIDs for MPS, as studies have not demonstrated significant benefits compared to alternative therapies.

7.1.4.2 | Diclofenac. Diclofenac, a non-specific COX inhibitor, possesses anti-inflammatory and analgesic properties.

Topical diclofenac, delivered via a patch for localized pain relief, showed moderate effectiveness in an RCT for MPS in the upper trapezius, demonstrating significant improvements in pain intensity, disability, and cervical ROM compared to placebo, though it did not improve PPT [172]. Additional RCTs found that traditional Thai massage and hot herbal compresses were more effective than topical diclofenac gel in improving pain and

quality of life [173, 174]. Another RCT involving patients with knee osteoarthritis (KOA) and knee-associated MTrPs reported that oral diclofenac combined with stretching improved pain intensity, function, and knee ROM from pre- to post-intervention [175]. However, DN combined with stretching showed significantly greater and longer-lasting benefits [175]. The evidence is insufficient to support the effectiveness of diclofenac for MPS compared to placebo and alternative therapies.

7.1.4.3 | Lidocaine (Topical). Topical lidocaine, delivered via patches to block pain signal conduction, demonstrated significant improvements in pain intensity, pain interference, and neck disability in RCTs involving patients with MTrPs [176, 177]. However, its effects on PPT and ROM were inconsistent across studies, with some reporting no significant benefits [176–178]. For example, one RCT found no significant difference in pain intensity between lidocaine patches and bupivacaine injections, although bupivacaine demonstrated greater improvements in PPT [176]. Open-label studies suggest that heated lidocaine/tetracaine patches may provide clinically meaningful pain relief for some patients [179]. Topical lidocaine may provide benefits for pain, disability, and ROM. However, there is insufficient evidence to determine its effectiveness compared to alternative therapies, with findings from one RCT.

7.1.4.4 | Other Topical Analgesics. Other topical analgesics, including capsaicin and EMLA Cream, have shown varied effects in managing MPS. Capsaicin cream, which defunctionalizes nociceptor fibers [180], demonstrated significant pain improvements in an RCT, though temporary hyperemia and burning sensations were common adverse effects [181]. Adding capsaicin to an NSAID patch did not provide further improvements in pain, disability, or ROM compared to the NSAID patch alone [182]. EMLA Cream, containing lidocaine and prilocaine, was found to be more effective than ultrasound therapy in improving pain, cervical ROM, and reducing the number of MTrPs in MPS patients [183]. While capsaicin and EMLA cream may provide benefits for pain and ROM, the evidence remains insufficient to determine their effectiveness compared to alternative therapies, with findings based on only one RCT for each.

7.1.4.5 | Opioids. Opioids provide pain relief by binding to mu, delta, and kappa opioid receptors [184]. A cross-sectional study suggested moderate effectiveness of weak opioids [185], and a longitudinal audit in patients with abdominal MPS reported significant pain improvements [186]. However, the evidence is insufficient to determine their effectiveness in managing MPS due to lack of RCT data.

7.1.5 | Needling Therapies: Trigger Point Injections (TPIs)

7.1.5.1 | Local Anesthetics (LA). LA injections alleviate pain by binding to sodium channels on the membrane of nerves, inhibiting pain signal conduction. LA injections have consistently provided significant short-term pain reduction compared to DN in systematic reviews and meta-analyses on neck, head, and shoulder MPS [187, 188]. However, these benefits were less pronounced in double-blinded studies and did not extend to functional outcomes such as cervical mobility or psychological factors [187, 188]. A network meta-analysis on masticatory MPS found that LA showed significant improvements only in maximum mouth opening compared to placebo [189]. Another systematic review and meta-analysis showed that LA injections were significantly more effective than Botulinum Toxic Type A (BTX-A) for pain relief in MPS, with LA providing consistent benefits up to 16 weeks post-treatment [190]. Repeated sessions of LA also yielded more sustained pain reduction than a single injection [190]. LA injections provide consistent short-term pain relief and improvements in maximum mouth opening (for masticatory MPS), with evidence supporting their effectiveness compared to placebo, DN, and BTX-A.

7.1.5.2 | Botulinum Toxin Type A (BTX-A). BTX-A relieves pain by blocking ACh at neuromuscular junctions, promoting muscle relaxation and reducing the release of pain-sensitizing neurotransmitters such as substance P and calcitonin gene-related peptide [191]. A systematic review and meta-analysis on pelvic floor MPS reported significant pain relief lasting beyond 12 weeks post-injection [192]. The review also found significant improvements in dyspareunia and emotional functioning (SF-12 mental) at 4 to 11 weeks, with dyspareunia improvements persisting beyond 12 weeks [192]. Comparatively, two systematic reviews on BTX-A and LA found that LA provided more consistent pain relief, with greater effects observed across multiple follow-up periods [190, 193]. While BTX-A achieved significant pain reduction compared to placebo at 2 to 6 months, its effects were less consistent at shorter intervals, such as 4 to 6 weeks [190, 193]. A Cochrane review assessing the effectiveness and safety of BTX-A for MPS found mixed results, with only 1 of 4 RCTs showing statistically significant pain improvement over placebo [194]. BTX-A may offer potential benefits for pain, dyspareunia, and emotional functioning. However, evidence of its effectiveness compared to placebo or alternative therapies remains insufficient.

7.1.6 | Needling Therapies: Non-Pharmacological Needling Techniques

7.1.6.1 | Dry Needling (DN). DN is a minimally invasive technique that involves inserting a thin filiform needle into MTrPs without using solutions or pharmacological agents [195]. The primary objective of DN is to mechanically stimulate the MTrP, eliciting local tissue responses that may result in pain relief and improved muscle function [196]. This is often accompanied by a "twitch response," an involuntary muscle contraction at the MTrP site, which is thought to indicate effective treatment and deactivation of the MTrP [196].

Several systematic reviews and meta-analyses have assessed the effectiveness of DN compared to sham or placebo in patients with MPS. Most studies suggest that DN provides significant short-term pain relief over sham [197–201]. A systematic review on upper-quarter MPS found that DN significantly reduced pain immediately after treatment and at 4 weeks post-intervention compared to sham or placebo [197]. Another review reported that DN reduced pain intensity in the short term (1 to 12 weeks after intervention) compared to sham/placebo/no intervention, supported by low- to moderate-quality evidence [199]. Similarly,

a systematic review focusing on neck and shoulder MPS demonstrated that DN was effective in reducing pain in the short term (immediately to 3 days) and medium term (9 to 28 days) compared to control or sham [198]. These findings support the use of DN as an evidence-based option for immediate pain relief in MPS patients.

While DN provides short-term pain relief, evidence suggests that trigger point injections (TPIs) and alternative therapies, such as physiotherapy, may yield superior outcomes for pain reduction and functional improvement. Several systematic reviews and meta-analyses have shown that TPIs provide greater pain relief compared to DN [198, 200, 201]. For example, one review found that lidocaine TPIs were more effective than DN in reducing pain in individuals with MTrPs in facial muscles [200]. Another review demonstrated that TPIs outperformed DN in relieving myofascial pain in the medium term (9 to 28 days), and other therapies, including physiotherapy, also surpassed DN during this timeframe [198]. Furthermore, studies indicate that while DN can improve ROM compared to placebo, other treatments achieve better results in increasing ROM and reducing pain after 3 to 4 weeks [201].

These findings indicate that there is some evidence supporting the use of DN for short-term pain relief in MPS. However, clinicians should consider alternative treatments, such as LAs or physiotherapy, which may provide more sustained pain reduction and functional improvement.

7.1.6.2 | Acupuncture. Acupuncture, rooted in traditional Chinese medicine, involves inserting and manipulating needles at specific points on the body, with the needles typically left in place for a set duration [202]. A systematic review and meta-analysis on lumbar MPS found that acupuncture was more effective than NSAIDs, lidocaine, and low-frequency electrical stimulation for pain relief [203]. In KOA-related MPS, acupuncture demonstrated significant improvements in pain and functional indices (WOMAC and Lysholm scores) compared to controls [204]. Similarly, two systematic reviews [205, 206] on head, neck, or back MPS reported significant improvements in pain, with one review [206] noting that benefits were observed only when acupuncture was applied directly to MTrPs rather than traditional points. Another systematic review on TMJD-related MPS found that acupuncture provided similar pain relief to occlusal splints and was superior to placebo [207]. Acupuncture may offer potential benefits for improving pain and functionality, with some evidence supporting its effectiveness compared to placebo and alternative therapies. However, these findings should be interpreted with caution due to variability in acupuncture techniques, including traditional, trigger point, and laser acupuncture. Refer to Figure 3 for a summary of the mechanisms of action underlying needling therapies in MPS.

7.2 | Non-Pharmacological Treatment

7.2.1 | Therapeutic Physical Modalities

7.2.1.1 | Transcutaneous Electrical Nerve Stimulation (TENS). TENS is widely used for myofascial pain relief by targeting large non-noxious afferents to decrease nociceptor activity in the central nervous system [208]. A systematic review on TENS for MPS affecting the trapezius muscle reported significant improvements in pain, neck ROM, and PPT [209]. High-intensity, low-frequency TENS was identified as the most effective for pain sensitivity and ROM, while both acupuncture-like TENS (AL-TENS) and conventional TENS improved functional outcomes, with AL-TENS specifically improving neck flexion [209]. Burst TENS outperformed amplitude-modulated frequency for PPT and ROM, and TENS showed improvements in pain and functionality compared to interferential therapy; however, no significant differences were observed when compared with kinesio taping (KT) [209]. Another meta-analysis found that TENS was significantly more effective in improving pain compared to exercise but not compared to sham [210]. PPT improvements did not differ significantly between TENS, exercise, or sham, suggesting limited incremental benefit in this measure [210].

Individual RCTs provide additional insights into TENS comparisons with other modalities. One trial found that frequency-modulated neural stimulation (FREMS) demonstrated superior improvements in pain, cervical ROM, and MTrP characteristics at a 3-month follow-up compared to TENS [211]. Other trials found no significant advantage of TENS over lidocaine or BTX-A

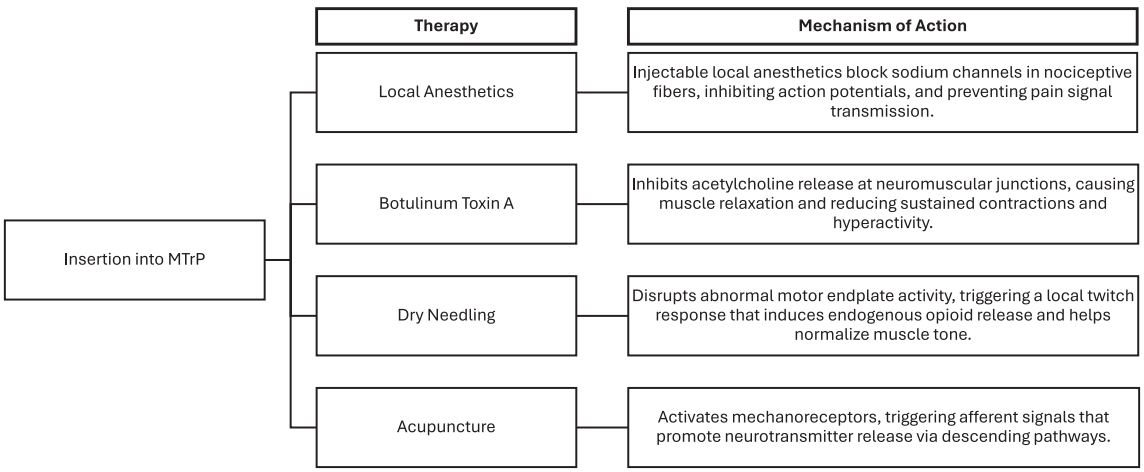

FIGURE 3 | Mechanisms of action underlying needling therapies for myofascial trigger points. MTrP, myofascial trigger point.

injections for MPS [212], and one study reported that TENS, biofeedback-enhanced relaxation, and dental physiotherapy yielded similar outcomes in managing masticatory MPS [213]. TENS may offer potential benefits for pain and ROM. However, there is insufficient evidence to support its effectiveness compared to placebo or alternative therapies.

7.2.1.2 | Magnetic Stimulation (MS). Magnetic stimulation (MS) is a therapeutic modality used to relieve myofascial pain by targeting muscles and reducing trigger point sensitivity [214]. Two RCTs involving patients with upper trapezius MPS reported significant improvements in pain and cervical ROM with repetitive MS (rMS) compared to placebo, with benefits sustained for at least 1 month in one study [215] and at least 3 months [216] in the other. An RCT on myofascial pelvic pain syndrome (MPPS) found significant improvements in pain and pelvic function, with combined pelvic floor and sacral nerve root MS demonstrating the most pronounced effects [217]. Additional studies on MPPS showed comparable pain relief between MS and myofascial release therapy or combined treatment approaches [218]. There is some evidence supporting the effectiveness of MS for improving pain and ROM compared to placebo. However, it is not consistently more effective than alternative therapies, such as myofascial release or combined treatments.

7.2.1.3 | Ultrasound (US) Therapy. US therapy treats MPS by converting electrical energy into deep-penetrating sound waves to reduce pain and inflammation [219]. Three RCTs on low-intensity US demonstrated significant improvements in pain and PPT compared to sham, with continuous US showing particularly strong effects in reducing resting pain compared to both sham and pulsed US [220–222]. Another RCT on abdominal MPS found that US and LAs similarly improved pain and quality of life [223]. In TMJD-related MPS, an RCT comparing US, stabilization splints, and masticatory exercises reported that US provided the fastest pain relief and functional gains over a 5-month period [224]. Low-intensity US may improve pain, quality of life, and functionality, with some evidence supporting its effectiveness compared to sham and alternative therapies. However, further high-quality studies are needed to clarify its comparative effectiveness against alternative interventions.

7.2.1.4 | Laser Therapy. Laser therapy uses concentrated light to stimulate cellular repair and promote recovery, showing potential benefits in managing MPS [225]. A systematic review highlighted low-level laser therapy (LLLT) as superior to sham treatments in improving pain and ROM, with high-intensity applications also improving the Neck Disability Index and SF-36 scores [209]. Another systematic review and meta-analysis of LLLT for neck MPS found significant improvements in pain and PPT but no effect on disability [226]. Several other systematic reviews on LLLT for temporomandibular MPS [227] and general MPS [228, 229] reported significant improvements in pain, PPT, disability, and physical functioning compared to placebo. LLLT may improve pain, ROM, disability, and physical functioning, with some evidence supporting its effectiveness compared to sham/placebo. However, variability in treatment frequency and duration, along with inconsistencies in the sustainability of benefits, highlights the need for more rigorous trials to establish definitive recommendations.

7.2.1.5 | Extracorporeal Shock Wave Therapy (ESWT). ESWT may relieve pain and improve physical function in MPS by stimulating tissue regeneration, wound healing, angiogenesis, and bone remodeling, as well as reducing inflammation through mechanotransduction [230]. A systematic review and meta-analysis on MPS patients found that ESWT significantly improved pain, PPT, and functional outcomes compared to sham; however, it did not show significant advantages over other treatments, such as DN, exercise, TPIs, or laser therapy [231]. Two systematic reviews on neck, shoulder, and trapezius MPS reported that ESWT was more effective in improving short-term pain intensity compared to other therapies [232, 233]. One review also found ESWT to be more effective in improving PPT but not neck disability [232]. Another systematic review on trapezius MPS concluded that ESWT was superior to sham and US for improving pain intensity but showed no significant differences in pain intensity or neck disability compared to DN, laser therapy, TPIs, or TENS [234]. ESWT may provide benefits for pain and functionality, with some evidence supporting its effectiveness compared to sham and as an adjunct therapy. However, its comparative effectiveness against alternative therapies remains unclear, emphasizing the need for further research to determine its clinical role in MPS management.

7.2.1.6 | Manual Therapy. Manual therapy is a hands-on approach designed to deactivate MTrPs, address soft tissue dysfunction, and improve movement limitations [235]. A network meta-analysis comparing various manual therapy techniques for MPS found that combined interventions achieved the largest effect size for reducing pain intensity, while afferent reduction techniques had the greatest effect on PPT [236]. Additionally, a systematic review and meta-analysis reported significant improvements in ROM with manual therapy interventions [237], and another review identified immediate and short-term benefits in both pain and ROM specifically using ischemic compression for shoulder MPS [238].

The effectiveness of manual therapy compared to alternative therapies varies by MPS region. For neck and upper back MPS, a systematic review found no significant differences in pain, PPT, or disability outcomes between DN and manual trigger point therapy, though both demonstrated similar efficacy in improving pain and function in the short to medium term [239]. For MPS associated with TMJD, manual therapy provided effective pain relief but was not superior to counseling or BTX-A [240]. In orofacial MPS, a systematic review reported improvements in pain intensity and PPT compared to no treatment; however, manual therapy was not more effective than KT or stretching techniques [235].

There is some evidence supporting the effectiveness of manual therapy for improving pain and PPT compared to no treatment. However, studies suggest that manual therapy does not provide additional benefits in pain, PPT, or disability compared to alternative therapies.

7.2.1.7 | Kinesio Taping (KT). KT is increasingly used by clinicians as a supportive treatment for MPS. A systematic review and meta-analysis on individuals with MPS found that KT was more effective than other treatments in improving pain intensity and ROM immediately after treatment; however, these

benefits did not persist at follow-up [241]. Additionally, no significant improvements were observed in PPT, muscle strength, or functional disability [241]. Another systematic review focusing on upper trapezius MPS reported that KT significantly improved pain compared to control groups, including sham, myofascial pressure release, or exercise [242]. While KT may provide short-term improvements in pain intensity and ROM for MPS patients, its effectiveness beyond the immediate post-treatment period remains uncertain. Refer to Table S1 for a summary of MPS therapies, including supporting evidence, key findings, and common adverse effects. The classification of evidence levels used in Table S1 is based on standardized effectiveness statements, as outlined in Table S2.

8 | Conclusions

MPS is a complex biopsychosocial condition characterized by MTrPs that cause pain, muscle dysfunction, and restricted mobility. Diagnosis remains challenging due to the lack of standardized criteria and symptom overlap with other chronic pain conditions. Identifying MTrPs is currently the primary diagnostic method, though advancements in imaging technology hold promise for more objective approaches. A deeper understanding of MPS neurophysiology can enhance diagnostic accuracy, guide management, and clarify differential diagnoses.

Effective treatment of MPS generally requires a multimodal approach that integrates pharmacological and non-pharmacological therapies. Evidence indicates sufficient support for the use of local anesthetic injections, which consistently demonstrate significant improvements in pain and functionality compared to placebo and alternative therapies. Some evidence supports the use of dry needling, acupuncture, magnetic stimulation, ultrasound therapy, laser therapy, and manual therapy for improving pain and/or ROM, particularly when compared to sham or placebo; however, their effectiveness relative to alternative therapies remains inconsistent. Extracorporeal shock wave therapy has some evidence supporting its use as an adjunct therapy. Conversely, therapies such as cyclobenzaprine, diclofenac, thiocolchicoside, capsaicin, EMLA cream, and TENS show insufficient evidence for their effectiveness in MPS, and further high-quality research is needed. Additionally, the effectiveness of baclofen, gabapentin, tizanidine, and triptans remains insufficient to determine, as the current evidence is limited or inconclusive.

A patient-centered approach that combines evidence-based treatments with shared goals between the patient and clinician is essential for managing MPS effectively. Future research should aim to refine diagnostic methods and evaluate the long-term outcomes of integrated treatment strategies to improve patient quality of life and reduce the healthcare burden of MPS.

Author Contributions

Jeremy P. Steen: conceptualization, methodology, investigation, writing – review and editing, writing – original draft, formal analysis, project administration, data curation. **Kishore S. Jaiswal:** investigation, writing – original draft, methodology, writing – review and editing, formal analysis, data curation. **Dinesh Kumbhare:** conceptualization, investigation, methodology, supervision, writing – review and editing, project administration.

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Ethics Statement

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.