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Treatment of Fibromyalgia, Myofascial Pain, and Related Disorders

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A 35-year-old woman was rear ended in a motor vehicle accident 1 year ago. She initially complained of diffuse posterior neck pain but no discomfort in her shoulder girdle, midback, or low back, and no symptoms suggesting a cervical radiculopathy. A cervical MRI scan was negative for a disk herniation or compromise of neural elements. The patient was seen by an interventional pain physician who suspected a facet arthropathy; however, diagnostic medial branch blocks to anesthetize the C5-5 and C6-7 facet joints produced no pain relief, and the physician did not believe he had more to offer the patient. Since the time of the accident, the patient's pain has gradually spread such that it now involves essentially the entire spine. On examination, the patient appears to have trigger points involving the upper trapezius and levator scapulae muscles bilaterally. Also, she reports tenderness in 14 of the 18 sites designated by the American College of Rheumatology for the diagnosis of fibromyalgia.

Definition

Fibromyalgia syndrome (FMS) is a disorder defined as chronic widespread pain of at least 6 months' duration with widespread musculoskeletal aching accompanied by multiple widespread tender points. According to the 1990 American College of Rheumatology criteria, a patient must have pain in the axial skeleton, pain above and below the waist, and pain to palpation in at least 11 of 18 paired tender points throughout the body. The majority of patients (80%) are women [1].

Myofascial pain syndrome (MPS) is defined as pain that originates from myofascial trigger points (MTrPs) in skeletal muscle, either alone or in combination with other pain generators. MTrPs are discrete areas of focal

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tenderness within a muscle that are characterized by hypersensitive palpable taut bands of muscle that are painful to palpation. Manual pressure over these points reproduces the patient's pain and refers pain in a characteristic pattern. Some clinicians prefer the term *regional soft tissue pain* as clinically useful, encompassing pain and localized tenderness not only in muscle but also in other contiguous soft tissues, such as ligaments and tendons [2].

Epidemiology

Fibromyalgia is present in 6 to 10 million Americans [3]. Chronic widespread musculoskeletal pain has been subjected to several epidemiologic studies during the last decade. According to these studies, 10% of the general population reports such complaints, clearly indicating chronic widespread musculoskeletal pain as a major health problem in the Western world. The prevalence of fibromyalgia is reported to be 3% to 5% with a significant female predominance [4]. Recent evidence suggests that fibromyalgia and related syndromes may share heritable pathophysiologic features. Serotonin and dopamine-related genes may have a role in the pathogenesis [5].

Myofascial pain has a high prevalence among individuals with regional pain complaints. The prevalence varies from 21% of patients seen in a general orthopedic clinic to 30% of general medical clinic patients with regional pain to as high as 85% to 90% of patients presenting to specialty pain management centers. Women and men are affected evenly [6–8].

Clinical presentation

The symptoms of fibromyalgia consist of musculoskeletal pain and stiffness in a widespread distribution, usually involving the neck, shoulder, and pelvic girdles as well as all of the extremities. Patients may present with pain predominantly in one or two regions (ie, the low back or neck are the most common areas), but direct questioning reveals pain in many other areas. Other common symptoms are general fatigue, poor sleep, and morning fatigue. Paresthesia is present in about one half of cases, usually in the extremities, and may mimic nerve root compression. Associated conditions such as migraine, irritable bowel syndrome, and restless legs syndrome are common [9].

The characteristic symptoms of myofascial pain may begin after a discrete trauma or injury or may be of insidious onset. Patients note localized or regional deep aching sensations, which can vary in intensity from mild to severe [10]. The MTrPs of each muscle have their own characteristic pain pattern; therefore, the distribution of pain can help identify which muscles may contain the responsible MTrP [11]. Frequently, associated autonomic dysfunction may occur, including abnormal sweating, lacrimation, dermal flushing, and vasomotor and temperature changes [12]. Cervical myofascial

pain may be associated with neuro-otologic symptoms including imbalance, dizziness, and tinnitus [13]. Functional complaints include decreased work tolerance, impaired muscle coordination, stiff joints, fatigue, and weakness. Other associated neurologic symptoms include paresthesias, numbness, blurred vision, twitches, and trembling. Later stages can be compounded by sleep disturbance, mood changes, and stress [14–16].

Physical examination of the patient with fibromyalgia reveals pain to palpation over 11 of 18 characteristic tender points (Fig. 1). The pressure applied should just blanch the fingernail bed of the examining physician. Examination of the joints and nervous system is normal despite the symptom of a swollen feeling in the joints and numbness. Range of motion of the cervical and lumbar spines may be slightly restricted because of pain. Diffuse soft tissue tenderness on palpation of the cervical, thoracic, and lumbar spine areas (including ligaments and paraspinal muscles) may be present [17].

Physical examination of the patient with myofascial pain begins with a careful medical, neurologic, and musculoskeletal examination. Posture, biomechanics, and joint function should be analyzed to identify any underlying factors that may have contributed to the development of the local or regional pain. An active MTrP is usually associated with a painful restricted range of motion. The trigger point should be identified by gentle palpation across the direction of the muscle fibers. The examiner should appreciate a "ropelike" nodularity to the taut band of muscle. Palpation of this area is exquisitely painful and reproduces the patient's local and referred pain pattern [18].

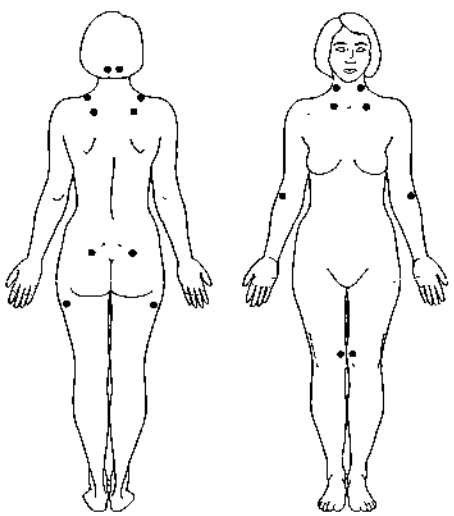

Fig. 1. 18 tender points of fibromyalgia. (*From* Freundlich B, Leventhal L. The fibromyalgia syndrome. In: Schumacher HR Jr, Klippel JH, Koopman WJ, editors. Primer on the rheumatic diseases. 10th edition. Atlanta: Arthritis Foundation; 1993. p. 247–9; with permission).

Laboratory tests

Routine laboratory tests such as a complete blood count, erythrocyte sedimentation rate, and chemistry profile are normal in FMS and MPS. Thyroid function tests are normal but may be helpful to exclude hypo- or hyperthyroidism in patients with muscle pain. Radiologic examinations (radiographs, CT scan, MRI) are normal; however, it is common to find coincidental osteoarthrosis or diskogenic changes. The treating clinician must determine the relevance of these findings to the patient based on the clinical scenario [19].

Well-controlled studies in patients with fibromyalgia and chronic muscle pain demonstrate normal muscle biopsy, electromyography, and nerve conduction studies [20]. Sleep electroencephalogram (EEG) studies may be requested to confirm a clinical suspicion of sleep disorders, such as periodic limb movement disorder, REM-behavior disorder, and sleep apnea. Alpha intrusion into stage 4 delta waves is seen in about 40% of patients, but these studies should be ordered only when there is clinical suspicion of the previously mentioned disorders [21].

Differential diagnosis

In clinical practice, there is often substantial overlap in the presentation of muscle pain disorders. The differences among myofascial pain, regional soft tissue pain, and widespread muscle pain are often indistinct. The differential diagnosis of muscle pain is broad. The following questions may be useful in distinguishing the contributions of different factors and may help the clinician develop an appropriate and specific treatment plan.

Is there regional myofascial pain, with trigger points present?
Is the myofascial pain the primary pain generator, or are there other coexisting or underlying structural diagnoses?
Is there a nutritional, metabolic, psychologic, visceral, or inflammatory disorder that may contribute to or cause the myofascial pain or regional muscle pain?
Is there widespread pain and other associated symptoms?

The differential diagnosis should include (but is not limited to) the following factors:

Joint disorders: zygoapophyseal joint disorder, osteoarthritis, loss of normal joint motion
Inflammatory disorders: polymyositis, polymyalgia rheumatica, rheumatoid arthritis
Neurologic disorders: radiculopathy, entrapment neuropathy, metabolic myopathy
Regional soft tissue disorders: bursitis, epicondylitis, tendonitis, cumulative trauma

Diskogenic disorders: degenerative disk disease, annular tears, protrusion, herniation
Visceral referred pain: gastrointestinal, cardiac, pulmonary, renal
Mechanical stresses: postural dysfunction, scoliosis, leg length discrepancy
Nutritional, metabolic, and endocrine conditions: deficiency in vitamins B_1, B_{12}, or folic acid; alcoholic and toxic myopathy; iron, calcium, magnesium deficiency; hypothyroidism
Psychologic disorders: depression, anxiety, disordered sleep
Infectious diseases: viral illness, chronic hepatitis, bacterial or viral myositis
Fibromyalgia or widespread chronic pain [2]

Pathophysiology of fibromyalgia and myofascial pain syndrome

Chronic muscle pain, regional myofascial pain, and fibromyalgia may be considered as a spectrum of clinical disorders. They share pathophysiologic mechanisms and often coexist in the same patient. The discussion herein is organized by neuroanatomic location; however, one should appreciate that these processes are interrelated and should be considered in an integrated fashion.

Motor end plate

An important finding in the pathophysiology of myofascial pain is a pathologic increase in the release of acetylcholine (Ach) by the nerve terminal of an abnormal motor end plate under resting conditions, an occurrence supported by electrodiagnostic evidence [22,23]. This abnormality is considered the primary dysfunction in the "integrated hypothesis" proposed by Simons and coworkers [11,18], which postulates a positive feedback loop (Fig. 2).

In support of the concept of the abnormal motor end plate, electrodiagnostic studies have demonstrated end plate noise (EPN) significantly more frequently in MTrPs than in the same end plate zone outside of the MTrP [23,24]. Because EPN is characteristic but not diagnostic of MTrPs, the significance of these findings remains disputed. An increase in EPN has been seen in

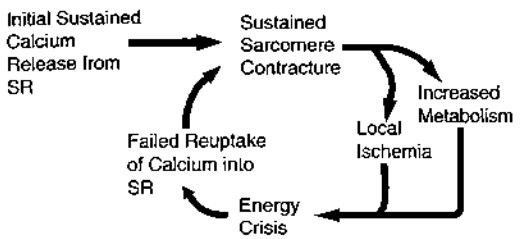

Fig. 2. "Integrated hypothesis," which postulates a positive feedback loop. (*From* Simons DG, Travell JG, Simons LS. Myofascial pain and dysfunction: the trigger point manual. Vol. 1. Upper half of body. 2nd edition. Baltimore: Williams & Wilkins; 1999, p. 71; with permission.)

response to many types of mechanical and chemical stimulation of the end plate structure and does not seem to be specific to myofascial pain [25,26].

Muscle fiber

It is hypothesized that increased Ach release could result in sustained depolarization of the postjunctional membrane of the muscle fiber and produce sustained sarcomere shortening and contracture. This maximally contracted sarcomere in the region of the motor end plate, referred to as a "contraction knot" by Simons [11], is diagrammed in Fig. 3. Compelling histologic support for this phenomenon is found in canine models of MTrPs. Longitudinal sections of dog trigger points demonstrate this sarcomere shortening, and cross-sections of dog and human MTrPs strongly suggest it as well [27,28].

One consequence of a chronically sustained sarcomere shortening may be greatly increased local energy consumption and reduction of local circulation, a combination that produces local ischemia and hypoxia. The unrelieved sustained tension of muscle fibers in the taut band produces an enthesopathy at the myotendinous junction that can be identified as an attachment MTrP. Muscle stretching techniques may be effective by equalizing sarcomere length throughout the affected muscle fibers and by breaking the feedback cycle.

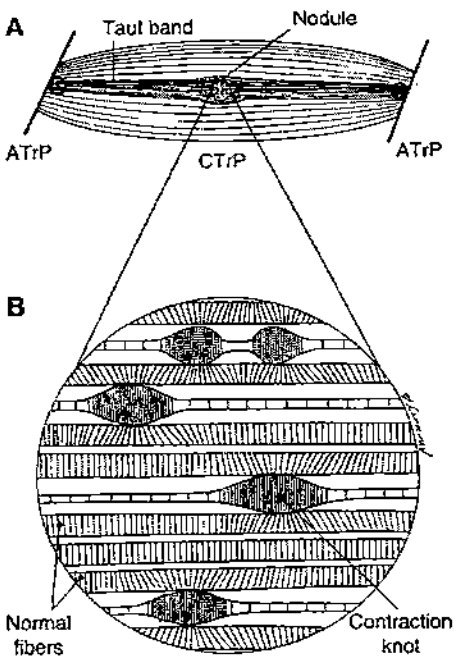

Fig. 3. Trigger point complex. (*From* Simons DG, Travell JG, Simons LS. Myofascial pain and dysfunction: the trigger point manual, Vol. 1. Upper half of body. 2nd edition. Baltimore: Williams & Wilkins; 1999, p. 70; with permission.)

The localized muscle ischemia stimulates the release of neurovasoreactive substances such as prostaglandins, bradykinin, capsaicin, serotonin, and histamine that sensitize afferent nerve fibers in muscle. These sensitized fibers may in turn account for local muscle tenderness [18]. The most recent research by Shah and coworkers [29] demonstrates increased concentrations of protons (H+), bradykinin, calcitonin gene-related peptide, substance P, tumor necrosis factor-alpha, interleukin-1 beta, serotonin, and norepinephrine in the biochemical milieu of human trapezius muscle in patients with neck pain and active MTrPs when compared with controls.

Central mechanisms: spinal and supraspinal

The referred pain resulting from trigger points arises from central convergence and facilitation. It is known from experimental data [18,30] that, under pathologic conditions, convergent connections from deep afferent nociceptors to dorsal horn neurons are facilitated and amplified in the spinal cord. Referral to adjacent myotomes occurs owing to spreading of central sensitization to adjacent spinal segments [31,32]. This pattern results in referred pain and expansion of the region of pain beyond the initial nociceptive region.

At the level of the central nervous system (CNS), spinal neuroplastic changes occur in the second order neuron pool of the dorsal horn owing to persistent pain. These changes produce a long lasting increase in the excitability of nociceptor pathways. Central sensitization results and is characterized by increased excitability of the neurons and expansion of the receptive pool of neurons. Neurotransmitters involved in the process of central sensitization include substance P, *N*-methyl-D-aspartate, glutamate, and nitric oxide [22]. In addition, there may be impairments in supraspinal inhibitory descending pain control pathways releasing inhibitory neurotransmitters such as gaba-aminobutyric acid, serotonin, and norepinephrine [33]. A recent study of cerebrospinal fluid levels of opioid peptides in fibromyalgia demonstrates that opioid dysfunction may contribute to pain [34].

In FMS, significant peripheral pathology is absent. Recent studies suggest central sensitization as the most important CNS aberration. Controlled studies have shown an increase in cerebrospinal fluid substance P (which mediates pain transmission) as well as a decrease in serum serotonin (which mediates pain inhibition) and CSF 5HIAA (a metabolite of serotonin). These findings may explain the amplified pain and decreased pain threshold in FMS [35,36]. Sleep abnormality has been objectively documented by EEG studies. Disturbed stage 4 sleep may explain the reported decrease in serum insulinlike growth factor-1 (which reflects the integrated secretion of growth hormone) [37,38].

FMS is not a psychiatric condition; however, a psychologic disturbance (anxiety, mental stress, depression) is present in 30% to 40% of patients (generally similar to rheumatoid arthritis). Psychologic factors seem to aggravate but not cause the pain. There is no correlation between psychologic

status and other symptoms of FMS besides pain (eg, swollen feeling, paresthesia, number of tender points) [39].

Most recent fMRI studies of the brain demonstrate that fibromyalgia is characterized by cortical or subcortical augmentation of pain processing. These studies provide further evidence for a physiologic explanation for fibromyalgia pain [40,41].

Treatment of fibromyalgia and myofascial pain syndrome

The following discussion refers to management of muscle pain syndromes including FMS and MPS. Much of the research has been done on one or both of these overlapping populations of patients. When clinically relevant, techniques specific to MPS or FMS are noted.

Diagnosis and education

Make a firm diagnosis of FMS or MPS based on its characteristics; avoid unnecessary investigations.

Educate patients regarding FMS and MPS.

Reassure the patient the muscle pain does not cause tissue damage.

Demonstrate an attitude of understanding and empathy; this is crucial for success in management. Never imply that symptoms are "all psychologic."

Elucidate probable mechanisms of pain to the patient in simple language (neuroendocrine dysfunction equals a chemical imbalance). Explain low serotonin and how its deficiency causes pain. Significant psychologic factors, if present, should be explained as aggravating factors.

Recognize and address significant psychologic factors, such as depression, anxiety, mental stress (at home or work), and poor coping skills. A minority of patients will require referral to a psychiatrist for management of more severe psychiatric disease and psychopharmacologic consultation and management.

Inquire about all aggravating factors that vary from patient to patient and individualize management.

Help patients to have restful sleep.

Encourage cardiovascular fitness.

Provide physical therapy for management of regional musculoskeletal disorders.

Promote behavioral modification through education including cognitive behavioral concepts [9].

Pharmacologic management

Given the considerable clinical overlap among myofascial pain, fibromyalgia, regional soft tissue pain, and tension headache, agents beneficial

in one syndrome may be useful in another. In the absence of controlled data specifically examining drug efficacy in each disorder, clinicians often generalize from these associated disorders.

Nonsteroidal anti-inflammatory drugs

Nonsteroidal anti-inflammatory drugs (NSAIDs) have minimal literature evaluating their use in chronic muscle pain. Several studies have found a small benefit of NSAIDs for management of pain in fibromyalgia if used in combination with alprazolam, amitriptyline, or cyclobenzaprine [42–44]. Interestingly, NSAIDs continue to be popular among patients, and in a recent study by Wolfe and coworkers [45], NSAIDs were considered more effective than acetaminophen for pain management by patients with fibromyalgia.

Tramadol

Tramadol is a combination of a weak opioid agonist and an inhibitor of the reuptake of serotonin and norepinephrine in the dorsal horn. There are no published controlled trials of tramadol for the treatment of myofascial pain; however, several studies support its efficacy in fibromyalgia, chronic low back pain, and osteoarthritis, all of which are commonly seen in association [46–50].

Antidepressants

Tricyclic antidepressants such as amitriptyline are effective for chronic tension-type headache, fibromyalgia, and intractable pain syndromes associated with muscle spasm [51–54]. Selective serotonin reuptake inhibitors (SSRIs) have not been specifically studied for myofascial pain, although efficacy has been documented in fibromyalgia for improving pain, sleep, and the global sense of well-being [51,55]. More recent studies support the use of dual reuptake inhibitors (serotonin and norepinephrine) for the treatment of fibromyalgia. Evidence supports the use of venlafaxine, milnacipran, and duloxetine [56–58].

Alpha-2 adrenergic agonists

The two major alpha-2 adrenergic agonists available for clinical use are clonidine and tizanidine. Tizanidine acts centrally at the level of the spinal cord to inhibit spinal polysynaptic pathways and to reduce the release of aspartate, glutamate, and substance P [59,60].

Anticonvulsants

To date, there is one controlled trial of pregabalin in the treatment of fibromyalgia that demonstrates reduction of pain, disturbed sleep, and fatigue when compared with a placebo [61]. One open label study of gabapentin in the treatment of chronic daily headache found possible efficacy [62].

Botulinum toxin

Botulinum toxin type A is emerging as a promising but expensive agent with efficacy in chronic MPS and chronic daily headache [63–65]. Cheshire and coworkers [66] performed a small randomized, double-blind, placebo-controlled trial of botulinum toxin type A and demonstrated a reduction of at least 30% in visual analogue pain scales, verbal pain descriptors, palpable muscle firmness, and pressure pain thresholds in the botulinum toxin group when compared with a placebo (saline) group. Fishman [67,68] has demonstrated improvement in patients with piriformis syndrome with injection of botulinum toxins (type A and type B). Porta [69] compared botulinum toxin A with steroid injection for the treatment of chronic myofascial pain and found greater improvement at 30 and 60 days posttreatment in the botulinum toxin treated group. By comparison, Wheeler and coworkers [70] were unable to demonstrate a statistically significant difference between botulinum toxin and placebo for the treatment of refractory unilateral cervicothoracic myofascial pain.

A peripheral and a central mechanism may explain the apparent efficacy of botulinum toxin in the treatment of chronic muscle pain. First, the blockade of Ach release at the neuromuscular junction reduces muscle hyperactivity, which, in turn, may decrease local ischemia. Second, if, as theorized, trigger points are sustained by excessive Ach release and sarcomere shortening, botulinum toxin may disrupt the abnormal neurophysiology of the trigger point. Evidence has also been found of retrograde uptake of botulinum toxin into the spinal cord and nucleus raphe, structures that modulate expression of neurotransmitters important in pain perception (eg, substance P, enkephalins) [71]. Additionally, botulinum toxin type A inhibits neurotransmitter release from primary sensory neurons in the rat formalin model. Through this mechanism, Botox inhibits peripheral sensitization in these models, which leads to an indirect reduction in central sensitization [72].

Nonpharmacologic treatment of muscle pain

Postural, mechanical, and ergonomic modifications

Although standard clinical practice and conventional wisdom includes efforts to correct postural and ergonomic abnormalities, there are limited direct data to support this approach in treating muscle pain. One study by Komiyama and coworkers [73] combined postural training and behavioral therapy in the treatment of myofascial oral pain and found that the subjects receiving the combination therapy were able to regain free unassisted mouth opening earlier than those treated with behavioral therapy alone; however, the differences in outcome were clinically minor.

The occupational medicine literature provides evidence that injuries are more common when workers are subjected to greater loads and have undesirable postures during their work [74]. Occupational muscle pain syndromes

are theorized to occur as the result of repetitive microtrauma and myofascial shortening. Correction of awkward postures is a standard part of treatment of these disorders, although long-term efficacy studies are lacking [75].

Stress reduction

Stress reduction techniques, including cognitive-behavioral programs, meditation, progressive relaxation training, and biofeedback, are often incorporated into chronic pain rehabilitation programs. Studies specifically addressing the efficacy of these techniques for myofascial pain are few. Crockett and coworkers [76] compared a multifaceted relaxation program with physiotherapy with dental splinting and transcutaneous electric nerve stimulation (TENS) for management of chronic facial and masticatory myofascial pain. Equivalent results and good response were found among all treatment groups. Electromyographic biofeedback and meditation-based stress reduction programs are established as beneficial in fibromyalgia [77,78].

Acupuncture

A growing body of evidence supports the efficacy of acupuncture in myofascial pain and fibromyalgia. The limited amount of high-quality data suggest that real acupuncture is better than sham for relieving pain, improving global ratings, and reducing morning stiffness in fibromyalgia [79]. One exception to this is a recent randomized clinical trial of acupuncture compared with sham acupuncture in fibromyalgia that did not demonstrate any difference between the patients in the sham groups when compared with the treatment group [80]. The 1997 National Institutes of Health consensus statement on acupuncture [81] concluded that "acupuncture may be useful as an adjunct treatment or an acceptable alternative to be included in a comprehensive management program" in the treatment of fibromyalgia, myofascial pain, low back pain, osteoarthritis, and lateral epicondylitis. Birch and Jamison [82] found relevant acupuncture (over points relevant to myofascial neck pain) to be superior to NSAID treatment and irrelevant acupuncture (superficial needling not related to neck pain) in a group of 46 patients with chronic myofascial pain. Interestingly, a remarkably close correspondence has been described between acupuncture points and trigger points [83]. Questions that need to be answered in future randomized controlled trials include the true benefit of acupuncture in chronic muscle pain, the duration of benefit of acupuncture, the optimal acupuncture techniques, and the value of booster treatments for the treatment of muscle pain.

Massage, transcutaneous electrical nerve stimulation, and ultrasound

Studies suggesting the efficacy of massage as part of treatment for muscle pain are scant. In a study by Gam and coworkers [84], massage combined with stretching exercises was better than control treatment in reducing the number and intensity of MTrPs. There was only a mild reduction in neck and shoulder pain. Hernandez-Reif [85] found that massage therapy was

effective in reducing pain, increasing serotonin and dopamine levels, and reducing symptoms associated with chronic low back pain; however, this study did not specify the etiology of the pain.

TENS treatment has shown mixed results in the treatment of myofascial pain. One single-blinded study [86] compared TENS with sham TENS in 10 patients for the treatment of myofascial pain and found no benefit for pain reduction; however, the study used subthreshold TENS parameters. By comparison, Graff-Radford and coworkers [87] compared four different TENS settings with a no-stimulation control in a double-blind study and found that high-frequency, high-intensity TENS reduced myofascial pain.

Ultrasound in combination with massage and exercise has been tested in a randomized controlled trial [84]. In that study, ultrasound, massage, and exercise had no additional benefit over sham ultrasound with massage and acupuncture for the treatment of MTrPs.

Exercise for fibromyalgia and myofascial pain

Exercise is one of the most important aspects of the rehabilitation and management of chronic muscle pain syndromes [88]. Reasons for this include optimization of flexibility, improvement of functional status, improvement of mood, self-efficacy, and reduction of pain.

Stretching exercises form the basis of treatment for myofascial pain. This treatment addresses the muscle tightness and shortening that are closely associated with pain in this disorder and permits gradual restoration of normal activity. Slow sustained stretch throughout the available range of motion is the most effective approach. Once muscle pain is decreased and range of motion restored, exercise to improve muscle strength and endurance should be instituted to maximize functional outcome. Aerobic exercise should also be included as part of an overall musculoskeletal and cardiovascular fitness program to prevent recurrence [89].

Patients should be encouraged to remain active but to perform daily activities in a gently lightly loaded manner. When a movement leads to pain, the patient should stop at that point and slowly and gently explore extending the movement just a little further to help release the muscle tightness. Clinical experience suggests that leaving a muscle in the shortened position aggravates MTrPs [2].

Trigger point injection for fibromyalgia and myofascial pain

In general, trigger points are the hallmark of MPS. Frequently, patients with generalized muscle pain, such as FMS, also have trigger points and painful shortened areas of muscle pain. These local areas may be treated with trigger point injections.

Stretching exercises are the mainstay of myofascial pain management, and injection therapy should be reserved to supplement or augment these exercises [89]. When MTrP injection is used as the primary therapy, patients are at risk for becoming dependent on this treatment for pain relief. Educating patients

about the effectiveness of manual techniques and instruction in the specific techniques empowers patients to self-manage their symptoms. With increasing relief of pain and increasing function, resumption of normal activity helps to further inactivate MTrPs. Optimal results are obtained when injections are preceded and immediately followed by manual MTrP release techniques with patient training on how to perform a continuing home program [90].

When injection proves necessary for initiating therapy or dealing with a recalcitrant area of myofascial pain, a series of injections should be initiated, and the patient should be informed of the limited role of this treatment in the long-term management of myofascial pain. Often, three consecutive visits for injection are recommended in chronic myofascial pain, with reassessment after the third visit to evaluate the efficacy of the injections and to determine whether further injections are necessary.

MTrP injections may employ several medications, including no medication (dry needling), short- or long-acting anesthetics, steroids, and botulinum toxin. Injections may use several different techniques such as slow search [91], fast in–fast out, superficial dry needling, intramuscular stimulation, twitch-obtaining intramuscular stimulation, and needling and infiltration with preinjection blocks. Several theories exist regarding the mechanism of action of injections for myofascial pain.

Dry needling of the MTrP provides as much pain relief as injection lidocaine but causes more postinjection soreness [92]. The effectiveness of needling depends on the needle eliciting local twitch responses [92]. Presumably, the needle mechanically disrupts and terminates the dysfunctional activity of involved motor end plates, with or without injection. Longer-acting anesthetics are more myotoxic without a proven increase in MTrP pain relief. The effectiveness of injection of steroids in MTrPs is controversial and without a clear rationale, because little evidence exists to support an inflammatory pathophysiology for MTrPs. Injection of botulinum toxin is emerging as a good option for treatment of chronic muscle pain [93].

In a recent systematic review article on needling therapies for MTrPs, Cummings and White [94] concluded that, based on current medical evidence, the "nature of the injected substance makes no difference to the outcome and that wet needling is not therapeutically superior to dry needling." This treatment is clearly an area in which more research is needed.

Hong's fast in–fast out technique [92] elicits local twitch responses more quickly than other techniques and presumably reduces needle trauma to muscle fibers by the twitch movement. Baldry and coworkers [95] recommend superficial dry needling, which they speculate may inactivate MTrPs through stimulation of cutaneous A delta fibers. Chu [96], based on Gunn's work [97], has reported a technique in which neurogenically evoked muscle twitches relieve myofascial-type pain. The needling and infiltration technique described by Fischer and Imamura [98], who use a preinjection block, permits more thorough injection of the trigger point and taut band region with reportedly less patient discomfort.

All of these techniques rely on an accurate identification of MTrPs by means of palpation. There is no definitive evidence that one technique is superior to another in long-term outcome. Cummings and White [94] remark, "because no technique is better than any other, we recommend that the method safest and most comfortable for the patient should be used." Very slim acupuncture needles may have the advantage of minimizing tissue trauma, allowing the practitioner to needle four to six trigger points at one session.

Functional outcomes in chronic muscle pain

Outcome studies of myofascial pain and its treatment are few. In one study [99] of pain, disability, and psychologic functioning in chronic low back pain subgroups, patients with low back pain of myofascial origin demonstrated similar or slightly worse outcomes than those with disk herniation as measured by several standardized questionnaires on pain and disability. Patients with myofascial pain have less accurate beliefs regarding their pain symptoms, express more dissatisfaction with physician efforts to treat their pain, and report receiving a dearth of information from their physician [100]. A 1998 study by Heikkila and coworkers [101] investigated the outcome of a multidisciplinary rehabilitation program for patients with whiplash and myofascial pain. After the rehabilitation period, 49% of patients had improved their coping skills. This percentage rose to 63% after 2 years. In addition, 46% of patients had increased their life satisfaction. The myofascial pain group also decreased their sick-leave time [101].

In a national six-center longitudinal study, Wolfe and coworkers [102] determined the intermediate and long-term outcomes of fibromyalgia in patients seen in rheumatology centers that had a special interest in the syndrome. Although functional disability worsened slightly and health satisfaction improved slightly, measures of pain, global severity, fatigue, sleep disturbance, anxiety, depression, and health status were markedly abnormal at study initiation and were essentially unchanged over the study period.

Summary

Chronic muscle pain is a common clinical complaint among patients who seek care for musculoskeletal disorders. There is a spectrum of clinical presentation ranging from focal or regional complaints to widespread pain. Nevertheless, treatment paradigms overlap and are guided by the following major principles (as put forth by this author):

- Be a sympathetic provider.
- Make an accurate diagnosis of myofascial pain or fibromyalgia.
- Identify and treat any "peripheral pain generators" (eg, tendonitis, bursitis, radiculopathy), which decreases the peripheral nociceptive input to the CNS.

- Treat the CNS dysfunction with a balanced contemporary pharmacologic approach.
- Treat (or refer to another provider) the associated symptoms, such as disorders in sleep, mood, headache, and bowel, and fatigue.
- Engage all patients in a comprehensive exercise and functional rehabilitation program.
- Judicially offer injection techniques for local trigger points or other peripheral pain generators to reduce pain and facilitate rehabilitation.
- Educate all patients about the diagnosis and encourage self-management.
- Incorporate mind-body techniques for management of chronic pain.

Case management

In the case presented at the beginning of this article, the treating physician is presented with a patient who probably has the common combination of fibromyalgia and myofascial pain. Although there are many options for management, I might treat the patient as follows:

Begin with patient education about the diagnosis.

Use a CNS active agent at low doses in the evening around bedtime for aid with sleep and pain. For example, 10 mg of cyclobenzaprine or nortriptyline is a reasonable starting option.

Encourage an active aerobic exercise program.

Refer the patient to physical therapy for education in myofascial stretching techniques and a spine stabilization program.

On follow-up, if the nighttime medication is not adequate, consider the addition of a daytime antidepressant, such as the serotonin norepinephrine reuptake inhibitor venlafaxine (37.5 mg) or duloxetine (20 mg). Low doses are started initially with gradual upward titration based on clinical response.

On further follow-up, local trigger point injection might be used in the trapezius and levator scapulae if physical therapy and exercise do not diminish the local pain and dysfunction.

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Low-dose naltrexone for the treatment of fibromyalgia: Findings of a small, randomized, double-blind, placebo-controlled, counterbalanced, crossover trial assessing daily pain levels.

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Abstract

OBJECTIVE: To determine whether low dosages (4.5 mg/day) of naltrexone reduce fibromyalgia severity as compared with the nonspecific effects of placebo. In this replication and extension study of a previous clinical trial, we tested the impact of low-dose naltrexone on daily self-reported pain. Secondary outcomes included general satisfaction with life, positive mood, sleep quality, and fatigue.

METHODS: Thirty-one women with fibromyalgia participated in the randomized, double-blind, placebo-controlled, counterbalanced, crossover study. During the active drug phase, participants received 4.5 mg of oral naltrexone daily. An intensive longitudinal design was used to measure daily levels of pain.

RESULTS: When contrasting the condition end points, we observed a significantly greater reduction of baseline pain in those taking low-dose naltrexone than in those taking placebo (28.8% reduction versus 18.0% reduction; P = 0.016). Low-dose naltrexone was also associated with improved general satisfaction with life (P = 0.045) and with improved mood (P = 0.039), but not improved fatigue or sleep. Thirty-two percent of participants met the criteria for response (defined as a significant reduction in pain plus a significant reduction in either fatigue or sleep problems) during low-dose naltrexone therapy, as contrasted with an 11% response rate during placebo therapy (P = 0.05). Low-dose naltrexone was rated equally tolerable as placebo, and no serious side effects were reported.

CONCLUSION: The preliminary evidence continues to show that low-dose naltrexone has a specific and clinically beneficial impact on fibromyalgia pain. The medication is widely available, inexpensive, safe, and well-tolerated. Parallel-group randomized controlled trials are needed to fully determine the efficacy of the medication.

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