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MYOFACIAL PAIN DYSFUNCTION SYNDROME-A REVIEW OF TREATMENT OPTIONS

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ABSTRACT

Myofascial pain dysfunction syndrome (MPDS) is defined as pain and that originates from myofascial trigger points in skeletal muscles of face and neck. It is prevalent in regional musculoskeletal pain syndromes, either alone or in combination with other pain generators. The various treatment modalities are occlusal therapy, physiotherapy, use of pharmacological, non-pharmacological methods, low level lasers, transcutaneous electric nerve stimulation, and botulinum toxin. This article discusses the advantages and disadvantages of various treatment methods with reference to the literatures.

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INTRODUCTION

Myofascial pain dysfunction syndrome (MPDS), also known as chronic myofascial pain, is a syndrome characterized by chronic pain in multiple myofascial trigger points ("knots") and fascial (connective tissue) constrictions. Characteristic features of a myofascial trigger points include: focal point tenderness, reproduction of pain upon trigger point palpation, hardening of the muscle upon trigger point palpation, pseudo-weakness of the involved muscle, referred pain, and limited range of motion following approximately 5 seconds of sustained trigger point pressure[1]

MPDS is a pain disorder, in which unilateral pain is referred from the trigger points in myofascial structures, of the head and neck region. These are localized tender areas within bands of skeletal muscles when stimulated by macro- micro traumatic episodes called TRIGGER POINTS. About 30 % of population suffers from myofascial pain, with Female to male ratio of 3:1 with an Age group of 15-40 years. All Etiological factors leads to micro or macro trauma to musculoskeletal system leading to muscle spasm This hyper tonicity may lead to muscle fatigue and accumulation of metabolic byproducts such as Lactic acid, prostaglandins, bradykinins, histamine. The accumulation of these chemical pain mediators, lowers pain threshold to mechanical and chemical stimuli leading to MPDS.[2,5]

Etiological factors

Major etiological factors such as 1.Muscular hyperfunction 2.physical disorders 3.injury to the tissue 4.para functional habits 5.disuse 6.nutritional problems 7.sleep disturbances and. Some systemic diseases, such as connective tissue disease, can

cause MPDS.[3] Poor posture and emotional disturbance might also instigate or contribute to MPS.[4] direct or indirect trauma, spine pathology, exposure to cumulative and repetitive strain, postural dysfunction, and physical deconditioning [6,7].

Clinical signs and symptoms

Cardinal symptoms of MPDS is Pain or discomfort, Limited motion of the jaw, Joint sounds Clicking, snapping. Tenderness to palpation of the muscles of mastication and Associated symptoms of MPDS are Neurologic which includes Tingling sensation, Numbness Blurred vision, muscle Twitches, Lacrimation, Otologic - Tinnitus, Ear pain, Dizziness, Vertigo, Diminished hearing Gastrointestinal tract Nausea Vomiting Diarrhea Constipation Dry mouth Musculoskeletal Fatigue Tension Tiredness Weakness Joint pain

According to Laskin, the diagnostic criteria are Four cardinal signs

Unilateral pain • Muscles tenderness • Clicking- clicking or popping noise in the TMJ • Limitation of jaw movement
Negative characteristics - No radiographic evidence • No tenderness in TMJ area on palpation via the external auditory meatus

Clinical examination includes

Range of motion

Vertical opening: checked for hypo mobility, Normal range for an adult is 40-50 mm Deviation while opening and closing Protrusive deviation and movement should be checked, the normal range is 10mm

Lateral excursion the Normal range is 10mm Palpation for tenderness should be checked Resistance test is done in

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Opening Protrusion, Left excursion and Right excursion The area on myofacial structures responsive to palpation are called "Trigger points". The muscles are palpated bilaterally and simultaneously with firm but gentle pressure lasting for 1-2 mins Middle finger is used for palpation of larger area adjacent fingers can be used.

Grading of click-Early, immediate or wide open zones of condylar excursion are noted • It should be noted whether the sound is on opening, closing or both.

Review of Treatment options for Myofacial pain dysfunction syndrome

The various treatment modalities are

Occlusal rehabilitation

1.Remove - extract 2.Reshape - grind 3.Reposition - orthodontia 4.Restore - conservative dentistry 5.Replace - prosthesis 6.Reconstruct - TMJ surgery 7.Regulate - control habits and symptoms

Pharmacotherapy includes

Analgesic drugs Analgesic Drugs Nonsteroidal anti-inflammatory drugs (NSAIDs) are the most commonly used drugs for MPS as they are readily available and have a relatively mild side-effect profile. Their use is appealing because of their analgesic and anti-inflammatory properties. Despite their widespread use, there are no randomized, controlled trials (RCTs) specifically evaluating oral NSAIDs in the treatment of MPS. Therefore, there is a lack of strong evidence for the role of an anti-inflammatory in MPS Multiple studies exist that demonstrate strong evidence in support of NSAIDs in treating acute MSK disorders, particularly low-back pain (LBP) [8].

Although the role of NSAIDs in treating MPDS is unclear, there is clear evidence that the analgesic properties of NSAIDs relieve pain in acute MPDS [9]. It would be reasonable to consider NSAIDs as an appropriate initial treatment in both disorders. However, long-term use should be considered with caution due to the gastrointestinal, renal, and antiplatelet side effects. A diclofenac patch was evaluated for myofascial pain of the trapezius muscle. There were statistically significant benefits with the diclofenac patch for pain(10)

Cyclooxygenase-2 (COX-2) inhibitors have an analogous effect to traditional NSAIDs with a relatively more-tolerable side-effect profile. Similar to NSAIDs, there are few supporting evidence for their efficacy in MPDS.(REF)

Tramadol is a centrally acting mu-receptor agonist, an inhibitor of dorsal horn presynaptic norepinephrine/serotonin reuptake, and increases central serotonin release. Studies have shown that tramadol is an effective and well-tolerated agent for use in chronic pain syndromes, and osteoarthritis [11]. With regard to myofascial pain, there are no studies investigating the efficacy of this agent however, it is used often for its multimodal analgesic effects and low abuse potential(2)

Tropisetron is a 5-HT₃ receptor antagonist and alpha-7-nicotinic receptor agonist recently used as an analgesic for fibromyalgia and myofascial pain with a limited commercial availability. In a RCT, local injections of tropisetron at trigger points provided a statistically significant improvement in pain

(P = 0.006)[12]. Its effects began rapidly and lasted longer than those of local anesthetics. Although this may appear a promising treatment for MPDS, most of the available studies were written by the same group of authors, and thus, this requires further investigation. Opioids are not normally indicated in the treatment for MPS. Although some limited studies demonstrate that weak opioids are moderately effective in the treatment of myofascial pain, most studies do not support the use of opioids in MPDS. (13)

The lidocaine patch is a transdermal local anesthetic preparation that alters the ability of nerves to conduct pain impulses. A number of RCT studies, case reports, and observational studies examining the efficacy of lidocaine patches on MPDS have been conducted. (14)

Muscle relaxants

Tizanidine As myofacial pain commonly result of clinical states like increased muscle tension, muscle spasm, spasticity and trigger point formation therefore the role of muscle relaxants in relieving pain is found to be beneficial and repeatedly being investigated. Tizanidine is the one most frequently used centrally acting muscle relaxant having alpha-2-adrenergic agonist properties, which decrease the muscle spasm, and is thought to prevent release of excitatory amino acids by suppressing polysynaptic excitation of spinal cord interneurons. Tizanidine should be administered initially at bed time owing to its sedative effect with gradual dose increment from an initial dose of 2 to 4 mg at bedtime up to the maximum of 8 mg 3 times/day. The bedtime dose can provide an analgesic effect as well as improve quality of sleep. Malanga *et al.* studied the effect of tizanidine in treatment of myofascial pain in 29 patients. Subjects were titrated up to 12 mg of tizanidine over 3 weeks and maintained for 2 weeks. Pain intensity and disability decreased significantly ($P < 0.001$) with improvement in pressure threshold and sleep quality and suggested it to be used as first line drug for myofascial pain. Commonly reported side effects are daytime drowsiness, hypotension, weakness, and dry mouth. Less commonly reported side effects of tizanidine are palpitations, bradycardia, dizziness, headache, nausea, elevated liver enzymes. Liver function enzymes are recommended to be monitored closely during the treatment[15]

Benzodiazepines depress the presynaptic release of serotonin and excite gammaaminobutyric acid (GABA), which causes rapid inhibitory neurotransmission.

Cyclobenzaprine is another muscle relaxant that was recently studied in a Cochrane literature review.

Thiocolchicoside (TCC) is a competitive. Thiocolchicoside is another agent that functions as an anti-inflammatory and analgesic, as well as a muscle relaxant. It exhibits selective affinity for the inhibitory gamma-aminobutyric acid and glycinergic receptors. It has an agonistic action at the spinal-strychnine-sensitive receptors that could mediate its myorelaxant effect. In a single blind phase IV clinical study on the efficacy of topical thiocolchicoside in the treatment of myofascial pain, it was found that ointment form may be a good alternative, particularly in patients who cannot receive injections.[16]

Anticonvulsants

Gabapentin and pregabalin have analgesic, anxiolytic-like, and anticonvulsant activity, which reduces the release of several neurochemicals, including glutamate, noradrenaline, and substance P. MPS may be mediated at the spinal level; therefore, anticonvulsants might be considered in its treatment(17)

Antidepressants

Tricyclic antidepressants (TCAs) are a class of medications that have been indicated for chronic pain, fibromyalgia, and neuropathic pain. Their pain mitigating effects are not clear, but it is postulated that TCAs work on central serotonergic and noradrenergic signals, which affect central pain pathways

Another study investigated amitriptyline use in the treatment of chronic temporomandibular disorder pain and showed a statistically significant reduction in all pain scores after 6 weeks of treatment. Currently, there is no indication for the use of these medications in the treatment of MPDS; however, the growing body of evidence for their efficacy in chronic pain syndromes suggests an increased role in MPDS when conventional treatments fail.

Duloxetine, a serotonin-norepinephrine reuptake inhibitor (SNRI), was recently found to be an evolving and possibly efficacious treatment for myofascial pain.

A clinician must be vigilant of the numerous side effects of SNRIs (i.e., nausea, fatigue, diarrhea, hyperhidrosis, dizziness, constipation, and dry mouth) as well as its interaction with other medications

Sumatriptan is a peripheral 5-HT receptor agonist commonly used for migraines. Data suggest that serotonin plays a role in the pathophysiology of myofascial pain. A RCT examined its efficacy in reducing pain in patients with temporal muscle pain and yielded a statistically significant reduction in pain intensity and increased pain relief(P<0.05). However, when compared to the placebo there was no significant difference (P=0.57). This study demonstrated an effective use of sumatriptan; however, more studies are required before endorsing this as a mainstream treatment for MPS.(18)

Other Treatments

Botulinum type A toxin (BoNT-A) is a potent neurotoxin that prevents muscle contraction. Its use in the treatment of muscle pain has been a topic of investigation recently and shows promise. It may have analgesic properties through decreased production of substance P and glutamate. The literature suggests that BoNT-A injection is a promising therapy to alleviate Myofascial Pain, especially when it persists despite conservative treatment. Another appeal is that the side effects of muscle weakness and paralysis are transient, mostly local, and reversible [19].

Ketamine is a dissociative anesthetic, analgesic, and sedative that works as a noncompetitive N-methyl-D-aspartic acid (NMDA) receptor antagonist. A RCT studied the use of intramuscular ketamine in the treatment of chronic myofascial pain in temporomandibular disorder. The study showed there was no statistically significant benefit to treatment with ketamine

L-tryptophan and memantine are drugs that have been studied for the treatment of pain; however, these are older studies and there has been no recent research indicating their use in the treatment of myofascial pain

Nonpharmacological Methods

Physical Therapy: Mouth opening exercise Hot packs Ultrasound Massage Transcutaneous Electrical nerve stimulation -TENS Transcutaneous electric nerve stimulation involves application of electrodes connected to a small battery-powered unit along the painful muscle and is a form of electroanalgesia. A low level of electrical stimulation is applied to relieve muscle tension and pain. The mechanism of the analgesia produced by TENS is explained by the gate control theory proposed by Melzack and Wall in 1965.[20] Graff-Radford *et al.* 1989[17] studied the effect of TENS on myofascial pain and trigger point sensitivity and observed pain reduction occurs with 100 hz, 250 ms stimulation followed by 100 hz, 50 ms. No pain reduction found in 2 hz, 250 ms. They suggested that high frequency, high intensity TENS is effective in reducing myofascial pain without having any effect on local trigger point sensitivity. Marchand *et al.* 1993 found that TENS was efficient in reducing pain intensity but not pain unpleasantness. TENS also produced a significant additive effect over repetitive treatment sessions.(21)

Low level laser therapy(3) The effects of low-level lasers (LLs) for controlling the pain in mpds and the efficacy of 660 nm and 890 nm wavelengths laser was recommended to reduce the pain in the masticatory muscles. For the laser group, two diode laser probes (660 nm (nanometers), 6.2 J/cm², 6 min, continuous wave, and 890 nm, 1 J/cm² (joules per square centimetre), 10 min, 1,500 Hz (Hertz)) were used on the painful muscles. Treatment was given twice a week for 3 weeks. The amount of patient pain was recorded at four time periods (before and immediately after treatment, 1 week after, and on the day of complete pain relief). According to this study, this type of LLLT was the effective treatment for pain reduction in MPDS patients.(22)

ANESTHESIA •Muscle and fascia (trigger point) •TMJ (Intracapsular and extracapsular) - 0.5 ml of 0.5% Xylocaine in conjugation with injection of hydrocortisone • Refrigerated spray-vapocoolant spray, such as ethyl chloride or fluoromethane is used to reduce muscle spasm Hypnotherapy - here patient cooperation is must and should follow hypnotist suggestions. It provides muscle relaxation Acupuncture-it is a simple, effective and conservative pain control modality. But this therapy is used only to give relief from pain and will not remove basic cause. Surgery - various surgical procedures like eminectomy, zygomectomy, menisectomy, high condylectomy

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