MPDS- Multifactorial Etiology and Varied Pathophysiology: A Review

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ABSTRACT

Temporomandibular joint (TMJ) diseases and disorders refer to a complex and poorly understood set of conditions, manifested by pain in the area of the jaw and associated muscles. Myofascial pain syndrome is a chronic pain disorder which produces the pain by pressure on sensitive points in muscles (trigger points) and causes pain in seemingly unrelated parts of body.

This article highlights the possible etiology, clinical features, investigations and recent treatment of myofascial pain dysfunction syndrome.

Keywords: Myofascial pain, temporomandibular joints, trigger points

INTRODUCTION

Myofascial pain is a regional myogenous pain condition characterized by local areas of firm, hypersensitive bands of muscle tissue known as trigger points. This condition is also referred as myofascial trigger point pain. Myofascial trigger point pain was first described by Travell and Rinzler in 1952.[1]

A myofascial trigger point (MTrP) is clinically defined as a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is painful on compression and can give rise to characteristic referred pain, referred tenderness, motor dysfunction, and autonomic phenomena.[2]

In 1969 Laskin published a paper and stated that there are many patients with muscle pain complaints in which the cause is not the occlusal condition and described the importance of emotional stress and other factors. After this article dental professionals began using the term myofascial pain dysfunction syndrome.[1]

Historical Aspect of Myofascial Pain Syndrome

Myofascial trigger points (MTrPs) are the principal characteristic of MPS. During the past nearly 200 years, numerous authors have described MTrPs in the English, German, Dutch, and French medical literature, illustrating that musculoskeletal pain due to MTrPs is very common.[3,4] Already in 1816, British physician Balfour described MTrPs as "nodular tumours and thickenings which were painful to the touch, and from which pains shot to neighbouring parts."[5] In 1938, British rheumatologist Kellgren published a seminal paper describing specific referred pain patterns of many muscles and spinal ligaments following injections of hypertonic saline. In 1952, Travell wrote the first of many articles introducing the myofascial genesis of pain illustrated by specific referred pain patterns of over 30 muscles.[6] Travell (1901 - 1997) has been referred to as the pioneer in the treatment of musculoskeletal pain through the recognition of MTrPs. She coined the term "myofascial pain syndrome" to describe pain as a result of trigger points in muscle, tendon, skin, fascia, and ligaments (the term "trigger point" was introduced by Steindler in 1940).[7]

CAUSE

Central nervous system plays significant role. Combination of both central and peripheral factors makes the condition difficult to manage. The following conditions are clinically related to myofascial pain:

Protracted local muscle soreness

Muscles that experience continued muscle soreness may lead to development of myofascial trigger points and myofascial pain.

Local factors

Certain local factors that influence muscle activity such as habits, poor posture and chilling seem to affect myofascial pain.

Systemic factors

Certain systemic factors may precipitate to myofascial pain e.g. fatigue, poor physical condition, hypovitaminosis and viral infections

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Increased emotional stress

Myofascial pain may be exacerbated by increased emotional stress. When individual is experiencing higher levels of emotional states, such as fear, anxiety frustration or anger the following major modifications of muscle activity can occur:

- An increased emotional stress excites the limbic structure and hypothalamus activating the gamma efferent system. Increased gamma efferent activity comes contraction of the intrafusal fibres, resulting in partial stretching of muscle spindle. When spindles are partially stretched, less stretching of the overall muscle is needed to elicit a reflex action. This affects the myotactic reflex and ultimately results in increase in muscle tonus. Muscles also become more sensitive to external stimuli which leads to further increases in muscle tonicity. These conditions lead to an increase in intra-articular pressure of TMJ.
- The increased gamma efferent activity may also increase the amount of irrelevant muscle activity. The reticular formation, with influence from the limbic system and hypothalamus can create additional muscle activity unrelated to the accomplishment of specific task. Often these activities assume the role of nervous habits such as biting on the finger nails or on pencil, clenching the teeth together or bruxism.

Constant deep pain

Constant deep pain input can create central excitatory effects in remote sites. If central excitatory effect involves an efferent (motor) neuron, two types of muscle effects can observed:

- Protective co-contraction.
- Development of trigger point.

When trigger point develops it becomes a source of deep pain and can produce additional central excitatory effects. These secondary trigger points are called satellite trigger point.[1]

CLINICAL FEATURES

1. Females more affected than males with ratio of 4:1.
2. Affects primarily young women (age 20 to 40 years).
3. Presence of trigger points which present as local areas of firm, hypersensitive, bands of muscle tissue.
4. There are four cardinal sign and symptoms:

- Pain
- Muscle tenderness.
- Clicking or popping noise in TMJ.
- Limitation of jaw movement.[8,9]

PAIN

Most common sites of pain in the masticatory system include jaw pain, preauricular pain, ear ache, neck pain, facial pain, and temple, frontal or occipital headaches.

- Pain has following characteristic:
- Pain is usually of dull and deep quality.
- The pain diffuse in nature
- The incidence and severity of pain vary with the stimulus.
- Associated restriction in movement may be present.

Although pain is increased with function of involved muscle, the amount of pain reported usually less than with local muscle soreness.[10,11]

MUSCLE TENDERNESS

In myofascial pain, the tenderness termed trigger points is deep localized and about 2-5 mm in diameter. In many instances, the patient may be aware only of referred pain and not even acknowledge the trigger point. Referred pain is wholly dependent on its original source, palpation of an active trigger point increase such pain.[9]

Trigger point may present in an active or latent state. In active state it produces central excitatory effects. Therefore when trigger point is active a tension type headache is commonly felt. In latent state patient does not report headache complaint. In this state trigger point is no longer sensitive to palpation and therefore does not produce referred pain. These types of trigger points are difficult to find by palpation.[1]

CLICKING NOISE

There will be recurrent clicking in temporomandibular joint at any point of jaw movement and there may be crepitus especially with lateral movements.[12]

LIMITATION OF JAW MOVEMENT

There may be limitations of jaw movement with variable jaw deviation or locking but rarely severe trismus is seen. Patient who clench or grind their teeth during working hours, the symptoms tend to worsen toward evening and some times have psychogenic basis. People with night time habit of clenching or grinding the teeth may awake with joint pain that abates during the day.[12]

Sometimes myofascial pain of head and neck is misdiagnosed due to additional signs and symptoms which are occasionally reported with more severe cases and coincidental pathologic conditions and are often associated with myofascial trigger point. Additional symptoms are increased fatigability, stiffness, subjective weakness, numbness, hyperesthesia, teeth sensitivity, excess lacrimation, increase salivation, nausea and vomiting. Numerous otologic symptoms such as ear pain, tinnitus, diminished hearing, dizziness, vertigo and fullness in ear.[13]

INVESTIGATIONS

Diagnosis is made on the basis of clinical findings.

Muscle spasm	Trigeminal neuralgia	Atypical odontalgia	Pulpal pain
• Characterised by acute onset of pain in jaw, face, ear or temples. • In muscle spasm there is generalised tenderness of muscle	• In TGN there is paroxysmal, unilateral sharp pain, sudden electrical lancinating pain confined to distribution of one or more branches of trigeminal nerve. • TGN occur after four decade and peak in 5th and 6th decades. • Trigger zones are present and stimulated by touch, in MPDS trigger points are deep, localised and 2-5 mm in diameter and produce pain on palpation. • Trismus absent in TGN	• There is pain in tooth or tooth side. • Mandibular function does not affect. • Patient will not have trigger points.	• Sharp oscillating, throbbing tends to coarsen or improve in time and local provocation of tooth exacerbates pain. • Trigger point are absent. • Clinical or radiographic science of pathology present in tooth with pulpal pain and absent in MPDS.

Table-1: Differential diagnosis

Radiographic changes are not commonly present. Arthrography and MRI is seldom indicated.[12]

PRESENCE OF TRIGGER POINTS

Trigger points are localized, firm, hyperirritable nodules that are tender to palpation patients often describe as knots within their muscles if sufficiently sensitized can be the referred pain source. Trigger points are small in head and neck region i.e. about to 2 to 10 mm and larger in shoulder region 10 to 20 mm. Trigger points become aggravated from muscle use, poor sleep, psychological tension and emotional stress and their severity can fluctuate as the contributing factor change. Manual palpation is a common method for identification of trigger zones. Rolling the finger over the muscle and feeling for firm, hyperirritable nodules within the muscle often identifies the trigger zone. It has been demonstrated to apply pressure directly to the trigger point to generate the referred pain.[14]

EXAMINATION OF MUSCLE OF MASTICATION

Muscles should be examined for tenderness using digital palpation. Muscles that should be included in examination are medial and lateral pterygoid, masseter, temporalis, sternocleidomastoid, and trapezius. Medial pterygoid muscles are checked by running a finger in an anterioposterior direction along the medial aspect of the mandible in the floor of the mouth.

Masseter muscle is examined by simultaneous pressing from inside and outside the mouth in the process of bimanual palpation. Lateral pterygoid examined by inserting a finger behind tuberosity region. In temporalis each of three areas (anterior, middle, posterior) should be examined. The anterior region is palpated above zygomatic arch and anterior to TMJ. The middle region is palpated directly above the TMJ and superior to zygomatic arch. Posterior region is palpated above and behind the ear. In sternocleidomastoid palpation is done bilaterally near its insertion on the outer surface of mastoid fossa behind the ear. The entire length of muscle is palpated down to its origin near the clavicle.[1,15]

MEASUREMENT OF STRESS

A useful tool is Symptom check list 90(SCL-90). This evaluation provides an assessment of nine psychologic states:

1. Somatisation
2. Obsessive compulsive behaviour
3. Interpersonal sensitivity.
4. Depression.
5. Anxiety.
6. Hostility
7. Phobic anxiety.
8. Paranoid ideation.
9. Psychotocism.

Assessment of these factors are necessary when evaluating chronic pain.[16]

TREATMENT

Treatment of MPDS is divided in to three categories by Weinberg. These categories are:

1. Palliative therapy.
2. Causative therapy.
3. Adjunctive therapy.

   One more category has been suggested by authors known as:

4. Definitive therapy.

PALLIATIVE THERAPY

This therapy includes procedures such as occlusal splint,

medications, home remedies (ice, moist heat application, exercises and soft diet).

MOIST HEAT AND ICE

Moist heat opens the capillary bed to promote increased blood flow; it also acts as muscle conditioner prior to exercise and physical therapy. Contraindications of heat therapy include circulatory insufficiency, sensory or cognitive impairment, malignancy and inflammation. Application of ice is quite effective for reducing muscle swelling and pain especially in acute condition.[17]

OCCLUSAL SPLINTS

Dental occlusal splinting and permanent occlusal adjustment have been the mainstays of TMJ disorder treatment. Occlusal splint therapy may be defined as "the art and science of establishing neuromuscular harmony in the masticatory system by creating a mechanical disadvantage for parafunctional forces
with removable appliances." Occlusal splint is a diagnostic, relaxing, repositioning, and reversible device. According to the glossary of prosthodontic terms [8th ed.], "occlusal splint is defined as any removable artificial occlusal surface used for diagnosis or therapy affecting the relationship of the mandible to the maxilla. It may be used for occlusal stabilization, for treatment of temporomandibular disorders, or to prevent wear of the dentition." A common goal of occlusal splint treatment is to protect the TMJ discs from dysfunctional forces that may lead to perforations or permanent displacements. Other goals of treatment are to improve jaw-muscle function and to relieve associated pain by creating a stable balanced occlusion. Splint therapy is considered an adjunct to pharmacologic therapy and most appropriate when nocturnal parafunctional activities can be identified. Typically, a flat-plane maxillary occlusal splint designed for bilateral contact of all teeth is fabricated. Such splints are thought to unload the joint by disarticulating the dentition and increasing the vertical dimension of occlusion. By unloading the joint, there will be a reduction in both synovitis and masticatory muscle activity. Therefore, the result is a reduction in symptoms. These appliances may also change condylar position and the existing occlusal relationship, thereby reducing abnormal muscle activity and spasm. Most occlusal splints have one primary function that is to alter an occlusion so they do not interfere with complete seating of the condyles in centric relation.[18]

PHARMACOTHERAPY

Drug therapy should be used on fixed dose schedule rather than as needed for pain. Following drugs can be used for treatment of M.P.D.S.

MUSCLE RELAXANTS

Most common muscle relaxants are metaxalone 400 to 800 mg every six hours or chlorzoxazone 500 mg every six hours. Other muscle relaxants are casrisoprodol, methocarbamol, orphenadrine and cyclobenzaprine.

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

NSAIDS are commonly used for the pain control in cases of MPDS. Ibuprofen should be used in doses of 400m.g four times daily. Chronic long term use is cautioned against because of their systemic and gastrointestinal side effects. The cyclo-oxygenase inhibitors rofecoxib (25-50 mg/day) and celecoxib (100-200mg/day) has same analgesic effects with reduced risk of gastrointestinal injury.

BENZODIAZEPENES

Diazepam (5-10 mg/day) and clonazepam can be used in the patient with muscle pain accompanied by stress and sleep disturbances.

TRICYCLIC ANTIDEPRESSANT

Drug like amitryptylline is effective in management of chronic pain in cases of MPDS. It has analgesic action in low doses, sedative effects and promotes restful sleep. The analgesic effect of TCAs is due to the serotonin and noradrenaline reuptake inhibition at synaptic level in the central nervous system (CNS). The blocking of these two amines increases their concentration and availability in the synaptic space of the nerve endings in the posterior horn of the spinal cord (involved in the transmission of pain) favoring or prolonging the inhibitory action in the transmission of pain. It can be started with dose as low as 10mg at night and dosage can be increased to 75 to 100mg depending upon patient tolerance.[9,10]

CAPSAICIN

Capsaicin cream (0.025% or 0.075%) can be use for pain relief. It releases substance –P and pain related neuropeptides to reduce pain perception and inflammation and must be applied multiple times per day for at least 2 weeks. Side effects of the drug are local burning, warming and reddening of the skin, these side effects diminish with time and eventually disappear.[17]

EXERCISE

Passive stretching i.e. keeping the muscle fibers relaxed while slowly stretching the muscle, preventing it from tightening via the stretch reflex in conjunction with moist

heat(followed by application of ice) is beneficial for decreasing muscle and joint pain and for improving ranges of movement.[19]

COUNTER STIMULATION OF MUSCLE

There are two methods for reducing muscular pain:

1. Repetitive action on trigger point with a mode of counter stimulation.
2. Muscle rehabilitation through active and passive stretching and postural exercises to restore the muscle to normal length, posture and range of motion.

There are several methods for counter stimulation of muscle to reduce trigger points. Common methods are spray and stretch, trigger point injection and acupuncture. Other methods like ultrasound, direct electrical stimulation are also useful for muscle contracture.

SPRAY AND STRETCH

Non-invasive technique for counter stimulation. It involves cooling the skin with fluoromethane, ethyl chloride, spray and then gently stretching the involve muscle to perform spray and stretch therapy. The cooling is done to allow the stretching to take place without the pain leading to reactive contraction or strain. The vapocoolant spray provides abrupt cutaneous stimulation that temporarily reduces pain perception in the area. It must be applied from distance of 18 inch. It is applied in one direction from trigger points towards reference zone in slow, even sweeps over adjacent parallel areas at rate of about 10cm/second.[13,16]

PRESSURE AND MASSAGE

Increased pressure is applied to trigger point can also relieve pain. Pressure is increased to about 20 pounds and is maintained to 30 to 60 seconds. If this technique produce pain it must be stopped since the pain can reinforce cyclic muscle pain.[16]

TRIGGER POINT INJECTIONS

Intramuscular trigger point injection can be performed by injecting local anaesthetic solution,saline or sterile water or by dry needling without depositing a drug or solution.

Procaine diluted to 0.5% with saline has been recommended because of its low toxicity to the muscle, but lidocaine (2% without vasoconstrictor) is also used with standard dental syringe. Injections are often given to muscle group in series of weekly treatments for 3 to 5 weeks; this may be continued with modification of the intervals between injections, depending upon the response.

Trigger point injections with LA are generally more comfortable than dry needling or injecting other substances, although acupuncture may be helpful for patient with multiple chronic muscle trigger points. The LA must be used in concentration less than that required for nerve block. This can remarkably lengthen the refractory period of peripheral nerves and limit the maximum frequency of impulse conduction.[9,13]

ADJUNCTIVE THERAPY

Consist of treatment modalities that augment and assist definitive or causative type of treatment for TMD.

PHYSIOTHERAPY

It is combination of physical therapy, massage therapy and electro modalities. Both passive and active treatments are commonly included as part of therapy. Posture therapy is also useful to avoid forward head positions that are thought to adversely affect mandibular posture and masticatory muscle.[9,17]

ELECTROTHERAPY

Is a part of adjunctive therapy; modalities includes electrogalvanic stimulation, ultrasound, low level laser and infra red.

ELECTROGALVANIC STIMULATION

It utilizes negative polarity over a painful, swollen area. The negative charge produces alkaline effect within the tissues, denaturing proteins and produced vasodilatation of the capillaries; this in turn permits the outward flow of metabolites and tissue fluids. High voltage electrogalvanic stimulation rhythmically pulsates the muscles to the level of fatigue causing muscle relaxation.[17]

TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION (TENS)

Mode of action of transcutaneous electrical nerve stimulation (TENS) has been attributed to neurologic, physiologic, pharmacologic and psychological effects. TENS supposedly blocks pain signals being carried over the small, unmyelinated C fibers by forcing the large myelinated A fibers to carry a light touch sensation. It may provide pain relief by physiologic effects of rhythmic muscle movement. The fasciculation of muscle may result in an increase in circulation, a decrease in edema and a decrease in resting muscle activity. The pharmacologic action of TENS may involve the stimulated release of endorphins which are endogenous morphine like substances. The probable placebo effect of TENS in relieving pain should also be considered. It is thought to increase the action of the modulation that occurs in pain processing at the dorsal horn of the spinal cord and trigeminal nucleus of brainstem.[15]

ULTRASOUND

It is a method of producing deep heat more effectively that the patient could achieve by using surface warming. These mechanical vibrations produce heat and vasodilatation by increasing the tissue temperature. Thus increasing metabolic activity. Vibrations also decrease pain by activating large myelinated peripheral neurons that attenuate pain or nociception stimulation at spinal cord or trigeminal (pons) levels.[9,17]

IONTOPHORESIS

Is a process in which ions in solutions are driven through intact skin by using a direct current between two electrodes. It uses ultrasonic energy to drive a medication deep into the tissue.

Low level laser

Laser therapy includes nitric oxide synthesis, which causes the endothelial linings of capillaries to dilate, improving circulation in the area. Laser therapy also may return injured tissues to a more optimum energy level, improving circulation, decreasing pain and swelling.

Infra red radiation

It produces vasodilatation of capillary bed by initiating the synthesis of nitric oxide, improving circulation and decreasing swelling. This small neurotransmitter improves circulation by opening the endothelial linings of capillaries.[13,17]

Acupuncture (dry needling) and Percutaneous electrical nerve stimulation (electro-acupuncture or PENS)

The effect of electro acupuncture is achieved by stimulation and release of endogenous analgesic peptides. The use of electro acupuncture or PENS with needling of trigger points in these cases will not only implement the local needle effect to reduce pain but also restore normal muscle analgesic system to provide more generalised effect of analgesia.

Local point therapy includes needling points to inactivate them. Distal point therapy includes needling points on opposite end of involved meridians with low frequency (1 to5 hertz) electrical stimulation to stimulate endogenous opiate system.[13]

MANAGEMENT OF STRESS

Integerating behavioural therapy and relaxation techniques in chronic pain management in MPDS are effective. Relaxation techniques decrease sympathetic activity and possibly arousal. Deep methods include autogenic training, meditation, and progressive muscle relaxation. Brief methods of relaxation include self – control relaxation, paced breathing and deep breathing. Hypnosis produces a state of selective or diffuse focus in order to induce relaxation.

Biofeedback is a treatment method that provides continuous feedback, usually by monitoring the electrical activity of the muscle with surface electrodes or by monitoring peripheral temperature. The monitoring instruments provide patients with physiological information that allows them to reliably change physiological functions to produce response similar to that produced by relaxation therapies.[9]

CONCLUSION

Myofascial pain syndrome is a type of muscle pain syndrome that has a defined pathophysiology that leads to the development of characteristic taut or hard band in muscle that is tender and that refers pain to distant sites. If it becomes chronic, it tends to generalize. Myofascial pain syndrome can resolve with ideal treatment regimens. However, many patients with myofascial pain syndrome have symptoms for years. Outcomes are best when a multifaceted treatment approach is done.

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