Myofascial Pain Dysfunction Syndrome : An Overview

(Part I : Epidemiology, Etiopathogeny, Clinical Characteristics & Diagnosis)

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Abstract

Myofascial Pain Dysfunction Syndrome (MPDS) associated to trigger points is a non inflammatory disorder of musculoskeletal origin, associated with local pain and muscle stiffness. It is characterized by the development of trigger zones, which are a palpable focus of hyperirritability in a tissue that when compressed, is locally tender and, if sufficiently hypersensitive, gives rise to referred pain and tenderness, which can be attributed to multiple etiological factors like occlusal disharmony, psychogenic influence like stress, strain, trauma, habits like bruxism. However, various aspects of its physio-pathology, clinical manifestation and treatment remain unclear. This review strives to compile the epidemiology, etiologic factors, clinical features and the diagnosis of this multi factorial problem. The available treatment modalities will be dealt with in the second part of this review article.

Key words: Myofascial pain dysfunction syndrome; Trigger points; Referred pain; Myofascial pain; Diagnosis; Etiology.

Introduction

Myofascial pain dysfunction syndrome (MPDS) is a disorder characterized by pain and dysfunction of the masticatory and associated muscles.[1] It is one of the most common causes of chronic musculoskeletal pain.[2-3] In 1934, Costen described an abnormality associated with ear and maxillary sinus which was found to be associated with Temporomandibular joint (TMJ) and referred to it as "Costen Syndrome".[4] Painful areas within muscles called "trigger areas", were described by Travell and Rinzler (1952), who pointed out the existence of syndromes associated with trigger areas within the muscles coupled with pain, spasm, tenderness and dysfunction. In 1959, Shore introduced the term "Temporomandibular Joint Dysfunction Syndrome".

In 1969, Laskin proposed a revised theory of Schwartz's TMJ pain dysfunction syndrome. He was of the view that although mechanical factors related to occlusion may sometimes be the cause of this condition by producing muscular overextension or overcontraction, the role of muscle fatigue was the most frequently encountered cause of such spasm.[5] To stress the role played by muscles, Laskin suggested that the term "Myofascial pain dysfunction syndrome" as more accurate to describe the condition than "Temporomandibular joint pain dysfunction syndrome".[4] It was defined clinically by Janet Travell, and later by David Simons.[6] According to International Association for the Study of Pain, Myofascial Pain Dysfunction Syndrome is a disorder characterised by pain and dysfunction of the masticatory and associated muscles. American Academy of Craniomandibular disorders has classified myofascial pain-dysfunction (MPDS) syndrome under craniomandibular disorders of non organic (functional) origin.

Epidemiology

MPDS has a lifetime prevalence of 85% in general population.[7] According to some studies it is more common in females.[8] It is most frequently seen in young unmarried females (married to unmarried ratio of 1:2) and female to male ratio of 3:1. It occurs commonly between 15-35 years of age. In explaining the predominance in women, the authors agree with the concept that women are more health conscious and seek medical and dental attention more readily than men. It has also been suggested that women have a high degree of psychosomatic disease that women are less tolerant to pain, and that women consider life events more stressful than men. MPDS is more common also in speech therapists or professional singers. Some workers have reported that MPDS is related to higher social classes. It increases with age.[9]

Etiology

MPDS has a multifactorial etiology. Several authors maintain that inadequate dentitions and unsatisfactory occlusion are the most frequent causes of MPDS.[10] Other investigators noted that hyper function may provoke myofascial pain and assert that TMJ disturbances are usually related to dysfunction of the masticatory muscles and/or emotional disorders.[8] The etiological factors of craniomandibular disorders that can give rise to MPDS include genetic, developmental, physiologic, traumatic, pathologic, environmental and behavioural factors.[5] Etiology of MPDS can be divided into- (1) Predisposing Factors, (2) Precipitating Factors, (3) Perpetuating Factors.

1. Predisposing Factors : Which include structural (size and/or shape) discrepancies with any of the tissues of the masticatory system. In addition, physiologic disorders such as neurologic, vascular, nutritional or metabolic disorders can predispose the patient to craniomandibular problems. Pathologic factors include systemic diseases and infections, neoplasias and orthopaedic imbalances. Behavioural factors relate to the personality profile of the patient and how that patient responds to stress, which can be expressed as noxious habits such as bruxism and tooth clenching.[10]

2. Precipitating (Triggering) : Factors include trauma not only to the masticatory system itself but also to the entire head and neck of the patient, an adverse stress response, iatrogenic problems, infection and idiopathic factors.[8-9]

3. Perpetuating (Sustaining) : Factors are manifested primarily by the myospasm-pain spasm cycle and can be related to any one or a combination of the above predisposing or precipitating factors.

The majority of current muscular pain syndrome models assume the existence of some form of event as the cause of muscular pain symptoms either local (e.g. dental fractures, muscle fatigue caused by oral parafunctional habits and micro or macro muscular trauma, orthopedic disorders, such as disc or class II skeletal discrepancies associated with craniomandibular problems, certain antihypertensive medicines such as calcium channel blockers) or systemic factors (e.g. increased emotional tension, endocrine problems, sleep disorders, nutritional deficiencies and viral infections), although in cases of severe painful conditions, the importance of these causes in the genesis of the pain is not clear.[11-13] Local or systemic factors increase the predisposition of an individual to develop myofascial pain syndrome and, if not detected or treated appropriately, become perpetuating factors. In some cases, the elimination of the perpetuating factors can produce the inactivation of the trigger points (TrPs) associated with myofascial pain. In patients with chronic myofascial pain, the proper identification and treatment of the perpetuating factors can mean the difference between success and failure of the treatment.[12]

Pathophysiology of Pain in MPDS

The muscular derangement and the formation of myofascial trigger zones with pain referral following predictable pain-referral patterns have been called the "Pain-spasm-pain Cycle". It has been described as muscular contraction turning to muscular fatigue, yielding muscular spasm. Muscle is an energy machine, the machine being the contractile proteins, the regulatory proteins, the sarcoplastic reticulum and calcium. The engine is run by ATP, which is required for contraction of actin and myosin. Prolonged or sustained muscular contraction will cause the disruption of the delicate sarcoplastic reticulum. This in turn releases free calcium ions that are stored within. These free calcium ions lead to actin-myosin complexing and muscular shortening (calcium excess shortening). This shortened muscle experience causes a depletion of ATP (muscular fatigue).[14] This depletion of ATP intensifies the actin-myosin binding which in turn causes a mechanical interruption of

blood flow through this area of bio-chemical derangement. This decrease in blood flow causes a decrease in oxygen coursing through the muscular fibres that are affected; necessitating the shift to anaerobic metabolism. These result in decreased pH and accumulation of nocigenic and spasmogenic by-products called the "biogenic amines" (histamin, bradykinin and serotinin). These noxious cellular metabolites cause the activation of group 3 and group 4 muscle nociceptive fibres which in turn cause pain. A three stage vicious biochemical cycle develops i.e. calcium pump inactivation, ATP depletion and increased in free calcium ions from sarcoplastic reticulum.[15] This cycle results in the production of more spasmogenic agents while decreasing the efficiency of the cells to produce ATP that can facilitate muscular relaxation. When muscles are pathologically shortened and painful, overt disease occurs. This can and does create postural disorders which force the patient to hold the jaw in abnormal positions in order not to be painful. This in turn creates a framework for malocclusion, chronic mandibular excursive disorders, possible internal derangements and restricted opening of the mouth.

Trigger Zones And Referred Pain

MPDS is characterised by the development of trigger zones.[7,9,16,17] A trigger zone is a localized area of spastic muscular fibres. It is a hypersensitive area from which impulses bombard the central nervous system and give rise to referred pain.[8,18] A trigger zone or sensitive painful area in the muscle or the junction of the muscle and fascia (hence, myofascial pain) develops due to number of causes. This trigger zone is locally tender and when active, refers pain through specific patterns to distant areas.[19] Increased energy consumption observed in an active TrP site is caused by an abnormal rise in the production and release of acetylcholine in the motor endplate in the resting state.[8,20] This rise in activity of the motor endplate produces a sustained depolarization of the muscle fibre, causing incorrect release and reuptake of calcium ions by the local sarcoplasmic reticulum.[9,15-16] The increase in free calcium ions causes a sustained muscle contraction, which raises energy demand. The supply of nutrients and oxygen is also compromised by the compression of nearby blood vessels.

This 'energy crisis' impedes the calcium pump which is responsible for returning the free calcium to the sarcomere (segmental reduction) and could also initiate the release of algogenic substances, producing sensitization of the autonomic and sensory nerve endings.[8] This release of neuroactive substances helps to further increase the production of acetylcholine and/or creating a vicious cycle of events. Both the sustained muscle contraction, produced by the continual release of acetylcholine, and the sensitization of local nociceptors by the generation of algogenic substances would explain clinical findings such as the presence of palpable nodules and/or pain arising from palpation of TrPs.[12,21]

Factors which cause trigger zones are as follows-

1. Sudden trauma to muscular skeletal tissues (muscle, ligaments, tendons, bursae).
2. Excessive exercise.
3. Chilling of areas of the body.
4. Injury to intervertebral discs.
5. Systemic conditions (heart attack, appendicitis)
6. Lack of activity (e.g., broken arm in a sling)
7. Muscle strain due to over activity
8. Generalized fatigue (chronic fatigue syndrome may produce trigger point).
9. Nutritional deficiencies
10. Hormonal changes (e.g. trigger point development during PMS times).
11. Nervous tension or stress
12. Obesity
13. Depression
14. Anxiety
15. Poor posture
16. Repetitive motion
17. Hypoglycaemia
18. Menopause

Clinical Features

MPDS is characterized by the development of trigger zones.[22] A trigger zone is a palpable focus of hyperirritability in a tissue that when compressed is locally tender and, if sufficiently hypersensitive, gives rise to referred pain and tenderness, which can be attributed to multiple etiological factors viz: occlusal disharmony, psychogenic influence like stress, strain, trauma, habits like bruxism.[9,23] Diagnosing these is of key importance.[24,]

The signs and symptoms arising from dysfunction of the TMJ are varied. There are four cardinal signs and symptoms of the syndrome viz. pain, muscle tenderness, clicking or crepitus noise in the TMJ and unilaterally or bilaterally limitation of jaw movement with deviation on opening.[1,10,25]

Pain: It can be either localised to the joint or referred to the head, neck or shoulders. It is usually unilateral and described as a dull ache in the ear or pre-auricular area which radiate to angle of the mandible, temporal area or lateral cervical area. Usually persistent in nature, often the pain is worse in the morning, in nocturnal bruxism, or in the late afternoon if parafunctional clenching or bruxing habits are correlated with work stress.

Tenderness: It is present over the affected TMJ during normal opening and closing motions. It is best elicited by placing the examining fingers at the posterosuperior aspect of both the condyles and expressing pressure anteriorly during their excursion. It is more common over the neck of the condyle, above the maxillary tuberosity, at the angle of the mandible and the temporal crest.

Clicking or Popping Noises in the TMJ: They are common and described as clicking, popping or crepitus. The nature of the click is still uncertain. It is usually bilateral. It can occur at any point of jaw movement and there may be multiple clicks. It may be audible, palpable or both and usually noted on simple palpation directly over the condylar head during the opening movement. Crepitus has been associated with perforations in the disk, which is usually followed by osteoarthritic change on the condylar surface followed by similar bony alterations on the opposing surface of the fossa.

Deviation of the Jaw to the Affected Side During the Normal Opening Motion: It is a common finding, since muscle spasm frequently accompanies joint dysfunction and as such contributes to the pain. [25] This restricts the motion of the condyle, impairing or completely eliminating the forward gliding motion so that all that remains is a simple hinge action, with the condyle remaining in the fossa.

Inability to close the posterior teeth completely into occlusion on the affected side.

Discrepancy in Occlusion which may be due to-

a) **Acquired Malocclusion:** The loss of a tooth without replacement will disrupt normal occlusal function by producing cuspal interference and premature contacts; resulting in alteration in joint function and the subsequent development of pain.
b) **Inherent Malocclusion:** Cuspal interference may be considerable in a dentition in which no teeth have been lost, despite the fact that the teeth may be acceptable cosmetically.
c) **Improper Dental Restoration:** May result in development of a painful TMJ. An important contributing factor is nervous tension with subsequent clenching, clamping or grinding of the teeth.
d) **Nervous Tension**: It is an active factor in the production of joint pain. The clenching, clamping and grinding of the teeth are direct results of tension and produce a state of muscle fatigue that in itself may be productive of pain even though the joint may not be involved. Pain and limitation of temporo-mandibular joint movement results from stress-induced muscle contraction.

Diagnosis of MPDS

The following baseline records should normally be made for patients suspected of having MPDS-

Chief Complaint: Related to Pain: date of onset, location, unilateral or bilateral frequency, duration, quality, trigger devices, and factors alleviating pain. Non-pain: date of onset, location, unilateral or bilateral, frequency; duration and quality.

Medical History: Ruling out etiologic factors (cardiovascular, neurologic, arthritis or systemic disease, hormonal imbalance, drug side-effects, local ear, nose, throat, sinus, cervical spine pathology) and evaluating medical consultation data (otolaryngologic, ophthalmic, neurologic, orthopaedic, psychiatric, and endo-crinologic).

Dental History: Evaluation of previous dental experiences, previous TMJ treatment, pain in specific teeth, oral symptoms other than pain: bruxism, muscle fatigue, gingival

bleeding and facial swelling.

Personal History and Psychological Evaluation: Marital status (past and present), children, in-laws, parents, sickness in the family, working habits and environment, commuting to and from work, sleeping habits and gastro- intestinal history- duodenal ulcers, colitis, nervous stomach, etc.

Radiographic Examination: Intra oral: minimum of 14 periapical radiographs and four bite-wings, Extra oral, Right and left lateral temporomandibular radiographs: closed, rest position and wide open, Right and left anteroposterior temporomandibular radiographs, if required, Cephalometric radiographs, if required, Tomograms, if required.

Clinical Examination: Facial symmetry, Relation of midlines: Open and closed showing mandibular deviation (generally open to affected side), Intraoral examination: Charting of mouth for caries, various types of prosthesis and restorations, missing teeth, extruded teeth, lingually and buccally displaced teeth, Deviate swallowing habits, Three-dimensional analysis of diagnostic casts, Intraoral functional occlusal analysis: Centric occlusion, centric relation, vertical dimension, working and balancing side 'interference, protrusive interferences. Palpation is the basic method of diagnosis.[9]

Palpation: Painful joints, coronoid processes, tenderness in muscles of mastication, head, neck, shoulders, back, anterior wall tenderness (by placing small fingers into external auditory meatus and pressing forward), Auscultation: Crepitation, rubbing, sagittal opening click, sagittal closing click, Pulp test for vitality or percussion testing of various teeth, Applied kinesiology (testing procedures), Nutritional work up, Urine analysis, Blood analysis.

Muscle Examination

A healthy muscle does not elicit sensations of tenderness or pain when palpated. If a patient reports discomfort during palpation of a specific muscle, it can be deduced that the muscle tissue has been compromised by either trauma or fatigue. Palpation of the muscle is accomplished mainly by the palmer surface of the middle finger, with the index finger and forefinger testing the adjacent areas. Soft but firm pressure is applied to the designated muscles, the fingers compressing the adjacent tissues in a small circular motion. A single firm thrust of 1 or 2 seconds duration is usually better than several light thrusts. During palpation, the patient is asked whether it hurts or is just uncomfortable.

The degree of discomfort is ascertained and recorded in one of four categories (Jeffrey P. Okeson).[26]

0 : No pain or tenderness reported by the patient.

1 : Patient responds that the palpation is uncomfortable (tenderness or soreness)

2 : Patient experiences definite discomfort or pain.

3 : Patient shows evasive action or eye tearing or verbalizes a desire not to have the area palpated again.

Temporalis

The temporalis muscle is divided into three functional areas; therefore, each area is independently palpated. The anterior region is palpated above the zygomatic arch and anterior to the TMJ and superior to the Fibres of this region run essentially in a vertical direction.

The middle region is palpated directly above the TMJ and superior to the zygomatic arch. Fibres in this region run in an oblique direction across- the lateral aspect of the skull.

The posterior region is palpated above and behind the ear. These fibres run in a horizontal direction. If uncertainty arises regarding the proper finger placement, the patient is asked to clench the teeth together. The temporalis will contract and the fibres should be felt beneath the finger tips. During palpation of each area, the patient is asked whether it hurts or is just comfortable and the response is classified as 0, 1, 2, & 3 according to the previously described criteria.

When evaluating the temporalis muscle, it is important to palpate its tendon. It is palpated by placing the finger of one hand intraorally on the anterior border of the ramus and the finger of the other hand extraorally on the same area. The intraoral finger is moved up the anterior border of the ramus until the coronoid process and the tendon are palpated. The patient is asked to report any discomfort or pain. The reference zone of the temporalis muscle includes all the maxillary teeth and upper portion of the face. Headache and toothache are the common complaints.

Masseter

From the anterior border and upper part of muscle, pain is referred to the upper molar teeth, whereas from the lower part it is referred in the lower molars. Trigger areas in the deep layer refer pain mainly to the TMJ and deep into the ear. Moderate restriction of opening associated with ipsilateral deflection of the midline incisal path may be observed.

Sternocleidomastoid

The palpation of sternocleidomastoid muscle is done bilaterally near its insertion on the outer surface of the mastoid fossa behind the ear. The entire length of the muscle is palpated down to its origin near the clavicle. Earache, temporomandibular pain and frontal headache are common complaints. This muscle is a frequent source of so called tension- type headache.

Posterior Cervical Muscle

Posterior cervical muscles (trapezius, longissimus capitis and cervicis, splenius capitis and cervicis and levator scapulae) are the major muscle responsible for cervical function. In palpating these muscles, the examiner's fingers slip behind the patient's head. Those of the right hand palpate the right occipital area and those of the left hand the left, at the origins of the muscles. Spasm of trapezius causes “stiff neck”, with limitation of motion on looking to the contralateral side and negligible restriction of mandibular movement.

Pterygoid Muscles

These muscles are basic to jaw movement but impossible to palpate. They are best evaluated with functional manipulation which means that a painful muscle will not only be tender to palpation but also be painful to contraction and/or stretching of the muscle.

Inferior Lateral Pterygoid Muscle

The lateral pterygoid muscle has been incriminated as the cause of numerous temporomandibular complaints. It protracts the mandible and therefore can act to create acute occlusal disharmony when it is shortened. Since this muscle is inaccessible for palpation, functional manipulation is required to help identify the presence of myofascial pain: a pain source within the muscle will be accentuated by maximum intercuspation (stretching the muscle) and by protruding the jaw against resistance (contracting the muscle).

Superior Lateral Pterygoid Muscle

Trigger zones in the superior lateral pterygoid muscle refer to the zygomatic area. No dysfunction is observed unless there is extensive deterioration in the temporo-mandibular joint sufficient to permit anterior displacement of the articular disc.

Medial Pterygoid Muscle

The reference zone for the medial pterygoid muscle includes the posterior part of the mouth and throat as well as the temporomandibular and infra-auricular areas. Throat and post mandibular pain is the common complaint. Moderate restriction of mouth opening associated with contralateral deflection of the midline incisal path may be observed.

Anterior Digastric Muscle

Trigger zones in the anterior belly of the digastric muscle, which is the trigeminal-innervated part, refer pain to the mandibular incisor area. No dysfunction is observed.

Conclusion

Musculoskeletal pain is a major cause of morbidity. It's prevalence increases with age. A growing number of individuals in our ageing population have musculoskeletal pain that affects their daily activities and function. It has a significant impact on their quality of life. This is creating a growing financial burden on our healthcare system. Early diagnosis and management may help reduce psychosocial complications and financial burden of chronic pain syndrome. However, standard diagnostic procedures to identify Myofascial pain are not available.

Therefore we conclude that as multiple diagnostic approaches may lead to therapeutic confusion, there is an urgent need for clinical research to establish evidence based guidelines for the diagnosis and treatment of MPDS.

References

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