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Myofascial Pain - Dysfunction Syndrome: A Dilemma for the Clinicians

Abstract

The MPD syndrome is a psychophysiologically altered condition involving the muscles of mastication and cervical group of muscles. The condition characterized by dull aching, radiating pain often results in muscle spasm and restricted movements. Frequently, myofascial pain is overlooked as a common cause of chronic pain because of frequent association with joint dysfunction and other pain disorders. Thus, the therapy should be directed towards reducing stress, rehabilitating the occlusion and relaxing the muscles to alleviate the condition.

Key Words

Myofascial; pain; temporomandibular joint

Ravi Patel[1], Himanshu Arora[2], Sarfaraz Memon[3], Usha Sharma[4], Ramjee Lal Raigar[5], Anant Raghav Sharma[6]

[1]General Practitioner, Dr Ravi's Dental Clinic, Surat, Gujarat, India
[2]Senior Lecturer, Department of Prosthodontics, Crown and Bridge Implantology, Manubhai Patel Dental College, Vadodara, Gujarat, India
[3]Senior Lecturer, Department of Prosthodontics, Crown and Bridge Implantology Manubhai Patel Dental College, Vadodara, Gujarat, India
[4]Senior Lecturer, Dept. of Oral Pathology, KM Shah Dental College & Hospital, Vadodara, Gujarat, India
[3]Post- Graduate Student, Department of Prosthodontics, Crown and Bridge Implantology, RUHS College of Dental Science, Jaipur, Rajasthan, India
[4]Senior Lecturer, Department of Periodontics, Pacific Dental College, Udaipur, Rajasthan, India

INTRODUCTION

Facial pain and its diagnosis have always posed a dilemma for the clinicians. The complex anatomy of the region compounded with the variability of symptoms involved, present a challenging situation during the management of orofacial pain. The study of temporomandibular joint appears to hold an uncommon fascination for clinicians of many disciplines. The dysfunction of these joint results in a large proportion of complaints presented to the dentists. With ever increasing refinements in diagnostic and treatment modalities, dramatic advances have been made in understanding the causes of facial pain related to joint and surrounding musculature. Still the mystery exists regarding the precise diagnosis and treatment of facial pain.

Differentiation between temporomandibular joint pain-dysfunction syndrome and myofascial pain-dysfunction syndrome

Temporomandibular joint pain dysfunction syndrome is a term covering a variety of problems which include the entire scope of temporomandibular joint disorders originating either intra articular or extra articular.[1] Myofascial pain-dysfunction syndrome is a psychophysiologic disease that primarily involves the muscles of mastication. The condition is characterized by dull, aching, radiating pain that may become acute during use of the jaw, and mandibular dysfunction that generally involves a limitation of opening.[2] Frequently, myofascial pain is overlooked as a common cause of chronic pain because of frequent association with joint dysfunction and other pain disorders.

ETIOLOGY

The myofascial pain-dysfunction syndrome has a multicausal etiology and the knowledge about the probable etiological factors seem to have improved over a period of time. *Goodfriend* (1933),[1] *Costen*

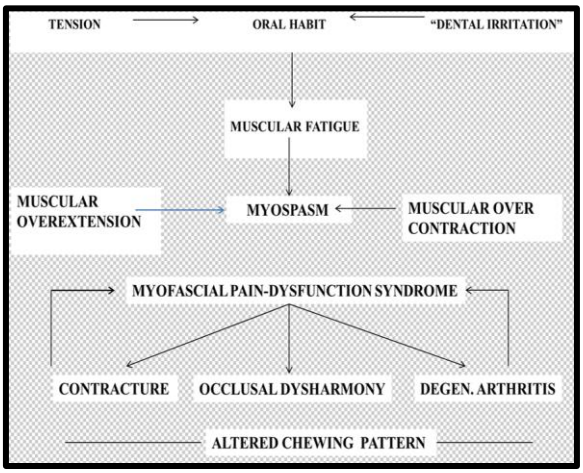

Fig. 1: Etiology of the myofascial pain-dysfunction syndrome

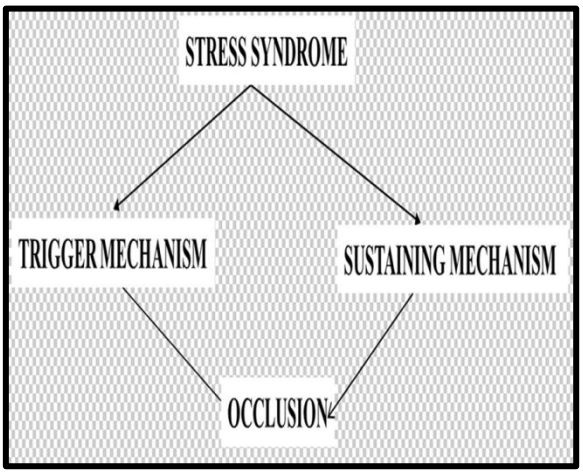

Fig. 2: The concept of "etiologic circle" is useful to understand the mechanism of TMJ dysfunction

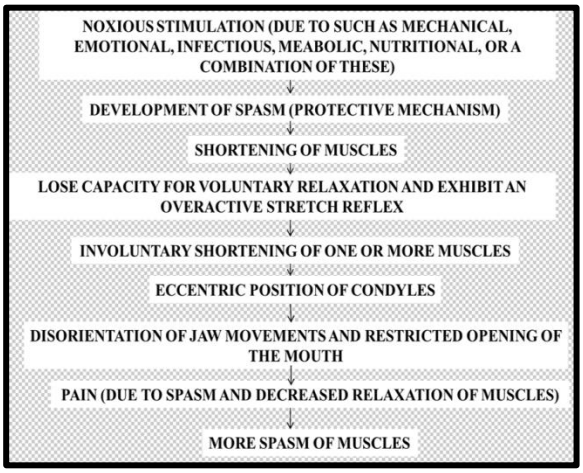

Fig. 3: Pathophysiology of this stress disorder of skeletal muscles

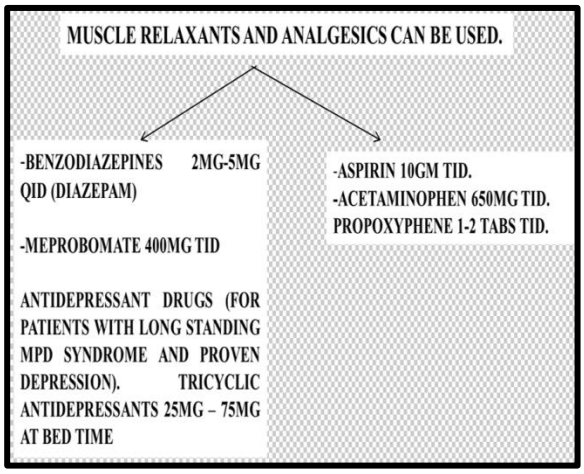

Fig. 4: Medication Thearpy

(1934)[1] and many others initially advocated the probable etiologies for the dysfunction syndrome. Pathologies of temporomandibular joint, trauma, occlusal disharmony and many other factors have been proposed by different authors. Lot of confusion existed until, *Travell* and *Rinzler*[1] first suggested that skeletal muscles in spasm could be the source of pain. They described about the painful areas within the muscles and called them as "*Trigger areas*" which were associated with pain, spasm, tenderness and dysfunction. *Schwartz* (1955)[1] adapted Travell's work and postulated the term temporomandibular joint pain-dysfunction syndrome. He reported that majority of patients with pain in the region of temporomandibular joint were suffering from functional disorders involving painful spasm of masticatory musles. He hypothesized that stress was a significant cause of clenching and grinding habits which resulted in muscle spasm. Occlusal abnormalities were found to play a secondary role. The next significant development towards understanding this aspect of facial pain occurred when *Laskin* (1969)[1] presented a comprehensive explanation of the problem and proposed his psychophysiological theory. He suggested that though mechanical factors related to occlusion may cause this condition by producing muscular overextension or overcontraction leading to muscle fatigue but psychophysiologically motivated oral habits is the frequent cause of painful spasm. To stress the role played by muscles, it was suggested that the term myofascial pain dysfunction syndrome is a more accurate term to describe the condition than temporomandibular joint pain-dysfunction syndrome. Laskin pointed out the key elements of the theory of MPD dysfunction.[3] Fig. 1 shows Etiology of the myofascial pain-dysfunction syndrome. Although there are three means of entry into the syndrome. The darker arrows indicate that most common pathway. The explanation of this mechanism is termed the psychophysiologic theory.

Laskin's theory is an outgrowth of work of Schwartz and is based on premise that MPD

syndrome is primarily a result of emotional rather than occlusal or mechanical factors. The masticatory muscle spasm is the primary factor responsible for signs and symptoms of pain-dysfunction syndrome. Spasm can be initiated in one of the three ways: 1) muscular over extension, 2) muscular overcontraction, or 3) muscle fatigue. The myofascial pain syndrome so produced not only causes pain and limitation of movement but also produces changes in jaw position so that teeth don't occlude properly (occlusal disharmony). In addition it may also cause organic changes such as degenerative changes in the TMJ and muscle contraction which is a manifestation of long-term spasm. These organic changes result in an altered chewing pattern with attendant reinforcement of the original spasm and pain. The changes in neuromuscular control of mandible produced due to occlusal disharmony has been supported by many researchers. Irregularities in occlusion appear to be the precipitating factor in pathogenesis of MPD syndrome. Occlusal interferences, posterior bite collapse, deep overbite/overjet and many other factors tend to restrict movement and predispose the patients to increased parafunctional activity resulting in overuse and thus fatigue of muscles. Moreover *Bruno* (1971)[1] found that the resulting pain in muscles will be concentrated in areas of fascia which upon palpation demonstrated tenderness and pain and these areas were referred to as trigger areas. According to *Weinberg* (1974),[1] every patient has got adaptation to a situation which is determined by his psychological make up. In a given patient, an occlusal interference may trigger the patient's acute symptoms while another factor, such as emotional stress may sustain them.[2,3] Evidence that nocturnal parafunction may be involved in MPD syndrome stemmed from studies by *Trenouth* (1978)[4] who observed that jaw pain and limitation of movement were often noted to be worse on awakening. *Christensen* (1981)[4] and *Yemm* (1979)[4] demonstrated that in chronic cases of MPD syndrome, an inflammatory stage occurs in affected muscles of mastication following the classic spasm. This myositis perpetuates the symptoms of pain and dysfunction. So, it can be seen that one school of thought supported occlusal disharmony as the major etiological factor of the development of MPD syndrome, but it could not explain why pain and dysfunction are uncommon in patients with severe malocclusion. It is now recognized that hyperactivity of muscles leading to myospasm is triggered by emotional disturbances and may be due to a combination of psychologic stress and muscle incordination secondary to malocclusion. To complete the understanding for etiology of MPD syndrome, it is essential to eliminate the possibility of medically linked factors, recent major surgical operations or trauma to head and neck which can give signs and symptoms of MPD syndrome.

PATHOPHYSIOLOGY OF MPD SYNDROME

As explained by Travell (1960),[5] the pathophysiology of this stress disorder of skeletal muscles is outlined on Fig. 3. It has been observed that whenever the pain associated with skeletal muscle spasm is very severe, it is referred to a site from the muscle that is its source. The pain is referred from a small area of hypersensitivity located within the muscle or the fascia. These areas are termed as Trigger areas. The response of muscles against the injury tends to gain momentum and results in a self representating cycle of spasm-pain-spasm which limits the movements and result in fibrosis of tissues.

Clinical Features

Incidence

Women are affected by MPD syndrome more frequently than men, with the ratio ranging from 3:1 to 5:1[2]. The greatest incidence appears to be in the 20 to 40 years age group.[2] The patients suffering from MPD syndrome usually present with complaint of:

- Pain in a zone of reference (most important problem that causes patients to seek treatment).
- Trigger points in muscles which cause pain on stimulation.
- Taut muscle band.
- Limited jaw opening.
- Associated symptoms.
- Presence of contributing factors for onset of pain.
- No tenderness in temporomandibular joint.

Mode of Onset

The patients suffering from MPD syndrome may complain of:

i. Sudden onset of pain and trismus, characterized by forcible contraction of muscle during biting on a hard object, overstretching of jaws, difficult tooth extraction etc.
ii. Gradual onset characterized by appearance of abnormal sounds in the joint followed by pain and limited jaw motion. Major precipitating

Table 1

Complete blood cell count	If infection is suspected.
Serum calcium, phosphorous and alkaline phosphatase measurement.	For bone diseases.
Serum uric acid determination.	For gout.
Serum ceratinine and creatine phosphokinase levels.	Indicators of muscle disease.
ESR, Rheumatoid factor, Latex fixation test.	For rheumatoid arthritis.
EMG	For muscle function evaluation.
Psychologic evaluation and psychometric testing.	For behavioural responses.

factor may be strain of muscles due to occlusal imbalance or asymmetry of face.

iii. May be associated with oral foci of infection, respiratory infection, or acute emotional stress.

Trigger points / Trigger zones / Trigger areas

- Myofascial pain is characterized by pain referred from few hypersensitive areas termed as trigger areas / zones. A trigger point is defined as a localized tender area in taut band of skeletal muscle, tendon or ligaments.
- Points occur frequently in head, neck, shoulder, lower back.
- Any pressure on these areas may initiate pain referred to distant areas (called as zone of reference).
- Trigger areas develop due to direct / indirect trauma (parafunctional habits) to muscles, due to weakening of muscles (nutritional disturbances, lack of exercise, structural disharmony etc.).
- Trigger points range from 2.5mm in diameter and may be active or latent.
- Palpating trigger points with deep finger pressure, elicits alteration in pain, in the zone of reference or causes radiation of pain towards the zone of reference.
- Patients behavioural reaction to firm palpation of trigger points is a distinguishing characteristic of myofascial pain and is termed a positive 'jump sign'. This reaction may include:
  a. Withdrawal of head.
  b. Wrinkling of face or forehead and desensation of skin.

In locating an active trigger point, jump sign should be elicited.

Signs and symptoms of myofascial pains are often accompanied by other pathological conditions and other problems such as:

a) **Neurologic:** Tingling, numbness, blurred vision and excess lacrimation.
b) **Gastrointestinal:** Nausea, constipation and indigestion.
c) **Musculoskeletal:** Fatigue, tension, stiff joints and muscle twitching.
d) **Otologic symptoms:** Tinnitus, ear pain and diminished hearing.
e) **Other symptoms:** Scratchy sensation, teeth sensitivity, increased salivation, increased sweating and skin flushing.

Management of MPD Syndrome

I Diagnosis of the condition

The cardinal signs and symptoms of MPD syndrome are similar to those produced by many organic problems involving the temporomandibular joint and other non-articular conditions. Therefore, a careful history and thorough examination may be helpful in diagnosing the condition. Radiographs may be helpful in diagnosing the condition if it has affected the bony structure also. X rays include: Transcranial, transpharyngeal, panoramic views, CT scans and MRI with arthroscopy can provide reliable diagnosis of the condition. Arthrography can be useful in determining the position of meniscus (when internal derangement of temporomandibular joint is being considered).

1. Certain lab tests are helpful

Shown on Table 1.

Differential diagnosis of MPD syndrome

a) **Non articular conditions that mimic MPD syndrome:** Pulpitis, Pericoronitis, Otitis media, Parotitis, Sinusitis, Trigeminal neuralgia, Atypical (vascular) neuralgia, Temporal arthritis, Trotters syndrome, Eagle's syndrome.
b) **Non articular conditions producing limitation of mandibular movement:** Odontogenic infection, Non-odontogenic infection, Myositis, Myositis ossificans, Neoplasia, Scleroderma, Hysteria, Tetanus, Extra pyramidal reaction, Depressed zygomatic arch, Osteochondroma.
c) **Differential diagnosis of temporomandibular joint disease:** Agenesis, Condylar hypoplasia, Condylar hyperplasia, Neoplasia, Infectious arthritis, Rheumatoid arthritis, Traumatic arthritis, Degenerative arthritis, Ankylosis, Internal disk derangement.

It is not possible to discuss signs and symptoms of these conditions at this juncture but a careful evaluation should rule out these conditions.

II Treatment of MPD syndrome

The treatment of MPD syndrome should be geared towards complete management rather than symptomatic cure. Several treatment modalities have been recommended for MPD syndrome.

1. Initial explanation of the problem

The patients should be explained about the problem and its probable etiology. The psychophysiologic factor shouldn't be stressed while explaining the problem because patient may not accept it. Initial discussion should deal with muscle fatigue, spasm and explaination about the condition.

2. Therapeutic modalities of treatment

a. Therapy at home

- Intake of soft diet with small cut pieces. Jaw motion should be limited and wide opening should be avoided. Parafunctional habits such as clenching, grinding should be avoided (although patients are unaware of these habits, they should be instructed to check for clenching). Other habits such as fingernail biting, lip biting etc. should be avoided.
- Intermediate moist heat application for half an hour twice daily.
- Massage of the affected area using moderate kneading motion. This helps in return of venous blood, lymph and catabolites and reduces muscle pain and spasm.

b. Short term medication

- Shown on Fig. 4.

c. Splint therapy

- If previously described forms of therapy are not successful or there is h/o tooth clenching etc., splint therapy should be considered.
- According to *Kawazoe*,[6] 4 types of splints are used:
  i. Stabilization splint
  ii. Relaxation splint
  iii. Resilient splint
  iv. Pivoting splint
- Hawley's type upper anterior splint is most effective because it prevents occlusion of posterior teeth and thereby prevents parafunctional activity. It is worn at night and 5-6 hours of day. Shouldn't be worn continuously as it results in supraeruption of posterior teeth. Platform of the splint should be flat.

d. Physical therapy

i) Ultrasound produces vibrations within the tissue that cause particle collision and release of energy. This results in production of heat and vibration which reduces the muscle tension and increase in tissue elasticity. Moreover there is mild analgesic and anti-inflammatory action. Lasts for 10-15 minutes, given twice daily for 1-2 weeks.

ii) High voltage electrogalvanic stimulation involves the use of monophasic, pulsed direct current applied through an electrode placed on skin over the involved muscle. Activated at frequencies from 4-80 pulse per second, for 10-15 minutes, 2-3 times a week. Stimulation of muscle increases circulation, reduces pain and spasm and increases resistance to fatigue.

iii) Cryotherapy (cold therapy)

- Reduces tissue temperature, causes local analgesia and has anti-inflammatory effects and diminishes muscle spasm.
- Cooling effect also creates vasoconstriction, reduces myoneural transmission and neuromuscular activity.
- Vapour coolant spray (such as ethyl chloride or fluorimethane) is applied over the involved muscle by spraying for 10 seconds. Repeated for two more times with 10 seconds interval. Mandible is mobilized by gently stretching to maximum opening (also termed as spray and stretch activity).
- Ice packs can be useful in acute phases of MPD syndrome. Cold application is used for 10-15 minutes; removed and reapplied after 5 to 10 minutes, 3-4 times daily.

e. Relaxation therapy

Because MPD syndrome is basically a problem related to increased muscle tension and spasm, any technique designed to induce muscle relaxation should be helpful. Among the modalities that have proven to be effective are biofeedback, conditioned relaxation and hypnosis.

i. **Biofeedback**

- EMG biofeedback involves supplying the patient with visual or auditory information about the moment to moment contractile status of muscle being monitored. The patient then concentrates on relaxing the muscle and is reflected by reduction in level of graphic representation or audible sound.
- The biofeedback is used for two 30-minutes sessions each week for 6 weeks.

Clarke and *Kardachi* (1977)[8] used biofeedback method in 7 patients suffering from MPD syndrome and achieved success by controlling parafunctional habits. *Dohrmann* and *Laskin* (1978)[7] noted

significant reduction in massetric EMG levels of patients treated with biofeedback.

ii. **Conditioned relaxation**

Similar to biofeedback in its end results but differs in that the patients do not have the benefit of a feedback indicator.

iii) Other methods are hypnosis, transcendental mediation (TM) and yoga can also be useful in the treatment of MPD syndrome.

f. Anesthetic injections

- Useful in extremely painful conditions. The injection of LA into tender and painful areas in muscle has been used for diagnostic and therapeutic purposes in patients with MPD syndrome.
- 0.5cc of LA that does not contain epinephrine or other vasoconstrictors are used.

g. Transcutaneous Electrical Nerve Stimulation TENS

- The use of TENS is based on the concept that stimulation of cutaneous branches of fifth nerve (trigeminal) creates an inhibitory effect on the trigeminal spinal tract nucleus, thus reducing the awareness of pain and relaxing the muscles.
- Therapy lasts 30 minutes and should be repeated daily.

III. Final explanation of problem.

- When patients with MPD syndrome begin to show improvements of their symptoms and have gained confidence in doctors ability to deal with their problem, the relationship between stress and MPD syndrome should be discussed and explained.

IV. Psychologic Counseling

- A group of patients are sometimes not able to identify and understand relationship between stress/strain and MPD syndrome and are unable to cope with stressful conditions. Such patients should be referred to psychologists or psychiatrist for counseling. Psychological interventions are aimed at reducing high level of muscle tension or modifying the environment.
- Treatment of contributing factors should be carried out.

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