ORIGINAL RESEARCH PAPER

Dental Science

UNDERSTANDING MYOFASCIAL PAIN DYSFUNCTION SYNDROME

KEY WORDS:

Dr Mahender Singh — Medical Officer, MDS Oral & Maxillo-facial Surgery, Department of Health and Family Welfare, Himachal Pradesh India.

Dr Shweta Verma* — MDS Conservative Dentistry & Endodontics, Professor, Department of Conservative Dentistry & Endodontics, Himachal Dental College, Sundernagar, Himachal Pradesh, India. *Corresponding Author

Dr Munish Goel — MDS Conservative Dentistry & Endodontics, Professor, Department of Conservative Dentistry & Endodontics, Himachal Dental College, Sundernagar, Himachal Pradesh, India.

ABSTRACT

MPDS is a psychological disorder which involves the masticatory muscles and results in pain, limitation in jaw movement, joint noise, jaw deviation in closing and opening the mouth and sensitivity in touching one or more masticatory muscles or their tendons. The present article reviews the incidence, etiology, pathophysiology, clinical features, diagnosis and treatment aspects of MPDS. Understanding the multifactorial etiology and complex clinical presentation of MPDS need to be fully understood for successful execution of various treatment modalities. Laser (Helium Neon) as monotherapy or in combination with exercise had shown promising results and can be used as an effective treatment modality for the treatment of MPDS.

INTRODUCTION:

MPDS (Myofascial Pain Dysfunction Syndrome) is the most common form of temporomandibular disorders and the most common cause of oro-facial chronic pains[1,2]. In fact, MPDS is a psychological disorder which involves the masticatory muscles and results in pain, limitation in jaw movement, joint noise, jaw deviation in closing and opening the mouth and sensitivity in touching one or more masticatory muscles or their tendons[3]. The conventional definition of myofascial pain syndrome (MPS) is characterized by regional pain originating from hyperirritable spots located within taut bands of skeletal muscle, known as myofascial trigger points (MTrPs).

Painful areas referred to as trigger zones 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. The presence of MPDS in the maxillofacial area mainly concerns the masseter, temporalis, lateral and medial pterygoid muscles. TrPs produce pain to any activating stimulus (direct or indirect trauma).

The present article reviews the incidence, etiology, pathophysiology, clinical features, diagnosis and treatment aspects of MPDS.

INCIDENCE:

MPDS has a lifetime prevalence of 85% in general popuation[4]. Several studies show that it is more common in females. It is most frequently seen in young unmarried females (married to unmarried ratio 1:2) and female to male ratio is 3:1. It occurs commonly between 15-35 years of age, begins in teenagers and progresses into middle age. The incidence of MPDS can be as high as 54% in women and 45% in men, although the prevalence of TrPs doesn't exceed 25%[5].

ETIOLOGY:

MPDS has a multifactorial etiology. The causes include;

- inadequate dentitions and unsatisfactory occlusion.
- hyper function may provoke myofascial pain and assert that TMJ disturbances are usually related to dysfunction of the masticatory muscles and/or emotional disturbances.[6]
- Muscle overload is thought to be the result of sustained or repetitive low-level muscle contractions, eccentric muscle contractions, and maximal or submaximal concentric muscle contractions.[7]
- Intracapsular disorders
- Emotional turmoil: The psychologic factors lead to an overall increase in masticatory muscle activity, while other factors such as occlusal and anatomic lead to selective increase in muscle activity.

Factors which cause trigger zones are as follows[3,7,8]:

1. Sudden trauma to muscular skeletal perforations in the disk, which is usually the muscular fibres that are affected; tissues (muscle, ligaments, tendons, followed by osteoarthritic change on the necessitating the shift to anaerobic 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 or completely eliminating the forward gliding depletion.
9. Nutritional deficiencies, Hormonal changes, Hypoglycaemia.
10. Nervous tension or stress.

PATHOPHSIOLOGY:

The steps involved in pathophysiology of MPDS are as follows:

1. Injury to muscle fibre type I.
2. Metabolic distress at the motor end plates.
3. Activation of muscle nociceptors.
4. Transmission of pain to the CNS.

Although muscle damage is not required for the development of TrP, there may be a disruption of the cell membrane, damage to the sarcoplasmic reticulum with a subsequent release of high amounts of calcium-ions, and disruption of cytoskeletal proteins, such as desmin, titin, and dystrophin.[7] The presynaptic, synaptic and postsynaptic mechanisms of abnormal depolarization (i.e. excessive release of acetylcholine (Ach), defects of acetylcholinesterase and upregulation of nicotine Ach-receptor activity, respectively) have been proposed as the possible etiological mechanisms.

CLINICAL PICTURE:

MPDS are characterized by the presence of hypersensitive spots called trigger points (TrPs). trauma) and can provoke

referred pain, referred tenderness, motor dysfunction, autonomic phenomena and hyperexcitability of the central nervous system. Myofascial pain is perceived as a dull, non-pulsating pain, which can vary from mild discomfort to incapacitating pain, both at rest and during activity; it is rarely symmetric and adopts a segmented distribution (non-dermatomal spinal segmentation pattern) [5,8,9,10].

Clinically MPDS is usually associated with unilateral pain [Table 1]. In some cases, pain can be bilateral but it need not be symmetrical. The quality or character of the pain reported by the patient most often will fall into three gross categories:

Category I: A dull-aching pain,
Category II: A sharp-shooting pain (burning), and
Category III: A tight-drawing sensation.

Laskin has proposed 4 cardinal signs and negative characteristics[1]

• Unilateral pain
• Muscle tenderness
• Clicking or propping noise in the TMJ
• Limitation of jaw movement
• No tenderness in TMJ area on palpation via the external auditory meatus • No radiographic evidence.

DIFFERNTIAL DIAGNOSIS:

It is important to distinguish between myofascial pain and neuropathic pain[11]. While myofascial pain originates at the muscle, neuropathic pain results from an injury to or malfunction of the peripheral or central nervous system.[6]

1. Migraine headache[11]: Migraine headache is a result of specific changes within the brain. It causes severe head pain that is often accompanied by sensitivity to light, sound, or smells.
2. Trigeminal neuralgia: Trigeminal neuralgia is a chronic pain condition that affects the trigeminal nerve, which carries sensation from your face to your brain.
3. Cluster headache: Cluster headaches are a series of relatively short but extremely painful headache every day for weeks or months at a time.
4. Post herpetic neuralgia: Postherpetic neuralgia (PHN) is nerve pain which occurs due to damage to a peripheral nerve caused by the reactivation of the varicella zoster virus (herpes zoster, also known as shingles).
5. Middle ear infection: An ear infection (acute otitis media) is most often a bacterial or viral infection that affects the middle ear, the air-filled space behind the eardrum that contains the tiny vibrating bones of the ear.

INVESTIGATIONS:

Clinical features are the commonest way to diagnose MPDS. Other investigations include radiological imaging. There is a great deal of emphasis on the "joint space", however concentricity of the condyle in its fossa is quiet variable. When it appears displaced on radiographic examination such as tomography, arthrography, magnetic resonance imaging (MRI) the diagnosis is usually related to a disc displacement problem, and the clinical features are more consistent with that diagnosis than with MPDS. Muscle activity can be investigated by electromyography.

MANAGEMENT:

Management of MPDS is based on certain principles that include the recognition of symptoms leading to an accurate diagnosis followed by appropriate treatment. Based on the multifactorial etiology of such problems, the treatment usually involves more than one modality in order to obtain complementary effects and that includes counselling, drug therapy, and physical therapy[12]. For long-term effect, treatments such as exercise therapy, anti-inflammatory drugs and local anaesthetics injections, stretching therapy, occlusal splint, psychotherapy, ultrasound, biofeedback, and Transcutaneous Electrical Nerve Stimulation (TENS) are used, but every treatment modality has its own pros and cons. A plastic splint or mouth guard from a dentist can keep teeth from contacting each other and prevent the damages caused by bruxism. Comfortable, heat-mouldable splints are available from many sporting goods stores or drugstores; however, these types of splints should be used briefly and only as short-term diagnostic tools. Because teeth may move, mouth guards that are properly made and fitted by a dentist are recommended.[13]

Low doses of a benzodiazepine at bedtime are often effective for acute exacerbations and temporary relief of symptoms; however, in patients with associated sleep disorders, such as sleep apnea, anxiolytics and muscle relaxants should be used with caution because they can aggravate these conditions.[14] Mild analgesics, such as NSAIDs or acetaminophen, individually or in combination are indicated.

Cyclobenzaprine may help muscle relaxation in some people. Because the condition is chronic, opioids should not be used, except perhaps briefly for acute exacerbations. In some cases of chronic pain, depression can follow. In such cases, antidepressant medication is useful under medical supervision.[13]

The patient must learn to stop clenching the jaw and grinding the teeth when awake. Hard-to-chew foods and chewing gum should be avoided. Physical therapy, biofeedback to encourage relaxation, and counselling help some patients[15]. Physical modalities include transcutaneous electric nerve stimulation (TENS) and "spray and stretch," in which the jaw is stretched open after the skin over the painful area has been chilled with ice or sprayed with a skin refrigerant, such as ethyl chloride [16,17]. Botulinum toxin has recently been used successfully to relieve muscle spasm in myofascial pain syndrome. Most patients, even if untreated, stop having significant symptoms within 2 to 3 years.

Laser is one of the most recent treatment modalities in the field of physiotherapy. On the basis of energy level, it is divided as soft tissue (wavelength of 500-900 nm) and hard tissue (wavelength of 1000-2800 nm) laser. Modern dentistry utilizes low-level Lasers (soft laser) in tissue healing, pain alleviation, reducing inflammation in the orofacial region. Low-level laser therapy (LLLT) is non-invasive modality and has been safely used in the treatment of myofascial pain due to its analgesic, myorelaxant, tissue healing, and biostimulation effects through direct irradiation without causing thermal response.[18] Medications used for MPDS include:-Aspirin: 2 tabs 0.3 to 0.6 mg/ 4 hourly, Piroxicam: 10-20 mg/ 3 to 4 times a day, Ibuprofen: 200-600mg/ 3 times a day, Pentazocine: 50 mg/ 2 to 3 times a day, Valium/ Librium: 5 to 1omg/ 2 to 3 times a day, methocarbomol 500mg/ 2 to 3 times a day[12]. There are many types of splints in dentistry such as custom splints, prefabricated splints, posterior splints, anterior jig, hard splints, soft splints, etc. It should be realised that all the splints should be provided by the skilled clinicians and all the masticatory activities can be changed by changing the occlusion.[13] Splint therapy changes the tooth-to-tooth occlusal relationships, which then changes the neuromuscular activity.

CONCLUSIONS:

Understanding the multifactorial etiology and complex clinical presentation of MPDS need to be fully understood for successful execution of various treatment modalities. Laser (Helium Neon) as monotherapy or in combination with exercise had shown promising results and can be used as an effective treatment modality for the treatment of MPDS.

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