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Myofascial pain syndrome associated with trigger points: A literature review. (I): Epidemiology, clinical treatment and etiopathogeny

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Abstract

Over the last few decades, advances have been made in the understanding of myofascial pain syndrome epidemiology, clinical characteristics and aetiopathogenesis, but many unknowns remain. An integrated hypothesis has provided a greater understanding of the physiopathology of trigger points, which may allow the development of new diagnostic, and above all, therapeutic methods, as well as the establishment of prevention policies and protocols by the health profession. Nevertheless, randomized studies are needed to provide a better understanding and detection of the different factors involved in the origin of trigger points.

Key words: *Myofascial pain, trigger point, craniocervical disorders.*

Introduction

Myofascial pain associated with trigger points has been studied by the medical and dental profession for more than a century (1,2). However, various aspects of its physiopathology, clinical manifestation and treatment remain unclear.

A great diversity of criteria exists for defining myofascial pain. Some authors consider this syndrome to be a specific entity, whilst others use a more generic definition which incorporates a wide range of musculoskeletal disorders (2).

Okeson, in 1985, (2) classified muscular pain of masticatory origin into five categories, one of these being myofascial pain associated with trigger points. In the present review, Simons and Travell's (3) definition of myofascial pain is used. The aim is to describe myofascial pain syndrome of craniocervical origin, based on the principal characteristics described in the literature with respect to its epidemiology, clinical characteristics and etiopathogeny.

Epidemiology and clinical characteristics

Myofascial pain syndrome associated to trigger points (TrPs), is a noninflammatory disorder of musculoskeletal origin, associated with local pain and muscle stiffness, characterized by the presence of hyperirritable palpable nodules in the skeletal muscle fibers, which are termed TrPs (trigger points) (1,3,4). TrPs produce pain to any activating stimulus (direct or indirect trauma) and can provoke referred pain, referred tenderness, motor dysfunction, autonomic phenomena and hyperexcitability of the central nervous system (Table 1) (2,5-7).

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).

In most cases referred pain is the main nociceptive source perceived by the patient (2). Trigger points are detectable only if superficially located in the muscle, or if associated with areas of localized spasm (3), their average size varying between 2 and 10mm (5, 7).

Myofascial pain syndrome is very common in the general population and its incidence can be as high as 54% in women and 45% in men, although the prevalence of patients with TrPs in the masticatory muscles does not exceed 25% (8). The most common age at presentation is between 27.5 and 50 years, with preference in sedentary individuals (5-7). The majority of publications do not report significant differences between the two sexes (2,3), although a greater prevalence in females has been described (1).

Myofascial TrPs are classified as either active or latent (5). In its active form the pain is continual, with reduced muscular elasticity, muscle weakness and referred pain in response to direct pressure (3, 9). The intensity and extension of the pain depend mainly on the degree of irritability of the TrP. Latent trigger points exhibit the same clinical characteristics as active TrPs, although they tend to be less severe. Moreover, in the latent forms, the pain is induced rather than constant, both in the zone of origin of the pain and in that of the referred pain (2,3,10). Some authors have even considered that the presence of latent TrPs may be connected with the genesis of muscle cramps (10).

TrPs may also be classified as primary or secondary (2,3), the primary TrPs being those that develop from either acute or chronic overloading of the muscle concerned, and where its activation is not due to the action of another muscle. Secondary, or satellite, TrPs are the result of mechanical stress and/or neurogenic inflammation due to the activity of a primary TrP (3).

Patients with myofascial pain syndrome usually exhibit protective habits or reflexes to avoid activating the pain (2,3).

TrPs can also produce changes in the function of the autonomic nervous system (ANS), which may cause localized hypothermia, referred cutaneous hypothermia and persistent lacrimation. Some authors also make reference to proprioceptive changes, such as balance problems or tinnitus in patients with myofascial TrPs (3,10,11). In some cases local and referred pain from active TrPs may be considered a factor related to the pain profile of tension-type headaches (12,13). Entrapment of the nerve branches that cross the TrP may produce sensory and/or motor disturbances due to damage in the affected nerve (3).

Other non-painful accompanying symptoms described

Table 1. Symptomatology of trigger points and their clinical significance (2,5-7).

SYMPTOM	CLINICAL SIGNIFICANCE
Taut bands	Minor
Local pain which heightens with use	Major
Local pain on palpation	Major
Referred pain	Major
Reproducible pain pattern	Major
Local twitch response	Minor
Reduced extension	Minor
50% pain reduction after treatment	Major
Non-clinically proven acute malocclusion	Minor
Muscle tenderness	Minor

in relation to, or as a consequence of, myofascial pain are psychological disorders (Axis II), such as depression and anxiety (3,14). Some recent studies indicate that patients with TrPs have a higher and more frequent consumption of psychotropics than the general population (15). Numerous authors maintain that depressive disorders may be brought on by high levels of chronic pain, such that, the more intense and prolonged the pain, the deeper the depression (6,16). Other studies have found that those patients with depression experience pain more acutely, which increases the symptoms produced by the disease (17), although other authors have questioned whether chronic pain could be the consequence of a psychiatric problem (18).

Anxiety disorders are also common in patients with chronic pain and are manifested in a high number of cases as muscular tension which leads to overload and fatigue in the masticatory muscles, causing the development of TrPs (19). The symptoms of chronic myofascial pain can also increase patient anxiety, thereby establishing a vicious circle (2,3).

Anamnesis, clinical examination and complementary tests

The diagnosis of myofascial pain is based mainly on anamnesis and clinical examination (1-3). Using anamnesis information is gathered regarding the type, intensity, duration, frequency and location of the pain, as well as alleviating or accentuating factors (Table 2) (1,8.15). The essential part of the clinical examination is to locate the TrPs by manual palpation of the cervical and facial musculature (3).

Localization of TrPs is based on three basic maneuvers: a) direct finger pressure, b) flat palpation, c) pincer palpation. Using the first two techniques, an assessment is made of the surface musculature, whilst the third is used to evaluate the deeper layers (3). It is necessary to wait between 2 and 5 seconds (3,20) after applying an appropriate pressure of about 2 kg/cm² (6), in order to reproduce the referred pain. The precision and force applied during an examination will influence the induction of this referred pain and, therefore, the diagnosis.

The literature reports poor concurrence between examiners in the location of TrPs using manual palpation (3,5,7,20). Cummings et al. (5) after an analysis of studies available in the literature, concluded that there was a 41 - 50% concordance between examiners when diagnosing the presence of a TrP. However, when ruling out their presence, reliability was 85 - 90%. Results published by Hsieh et al. (21) show that the stimulation of local or referred pain by manual palpation is the most reliable diagnostic method for detecting myofascial TrPs, whilst the identification of palpable nodules or the provocation of a local twitch response present poor reliability between examiners in the majority of available studies.

Complementary testing is the third link in a clinical history, although there is much controversy as to their utility in the diagnosis of myofascial pain syndrome since they lack the necessary precision, sensitivity and specificity (22).

The majority of authors agree that complementary tests would be of use in the diagnosis of myofascial pain, as long as they were preceded by a proper anamnesis and clinical examination (3,6,23). Ultrasound, electromyography, algometry and thermography are some of the complementary tests quoted in the literature for diagnosis of TrPs. Even so, a detailed analysis of available published material reveals that none of these diagnostic tests has a scientifically-proven usefulness in the diagnosis of myofascial pain disorder, thus better quality scientific studies would be required to determine the real benefit of their application in detecting TrPs (3,5). Within these complementary tests, Ge et al. (24) would include dry needling and topographical mapping as techniques capable of detecting active TrPs.

Table 2. Factors which increase or decrease painful symptoms of trigger points (1,8,15).

AGGRAVATING FACTORS	MODERATING FACTORS
Overuse of musculature	Rest
Active stretching	Passive stretching
Pressure on trigger point	Specific myofascial therapy
Prolonged muscle contraction	Non-isometric contraction activity
Cold, damp, viral infections, tension	Local warming of trigger point

Etiopathogeny

Despite the diverse theories, the exact nature of TrPs is unknown (2,6), although the combination of the two lines of investigation most accepted by the scientific community, electrophysiological and histologic, allows the proposal of an integrated hypothesis regarding the origin of TrPs (25).

This hypothesis postulates that the 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. 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. 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. 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 (3,26). Chang et al. (27) demonstrate that over time there is neuroaxonal degeneration and neuromuscular transmission disorders in muscles with TrPs, and that this mechanism is possibly implicated in the degeneration of motor neurones. A recent study by Shah et al. (25) provides a solid histochemical basis for this theory, as it shows that active TrPs exhibit a biochemical environment of inflammatory mediators, neuropeptides, cytokines and catecholamines different from latent TrPs or normal muscle areas.

None of the theories regarding the origin of TrPs provide a convincing explanation of the phenomenon of referred pain of myofascial origin. However, four theories exist regarding the transmission mechanisms of referred pain: the theory of pain transmission via specific nerve pathways, the theory of transmission via collateral branches in the nerves responsible for the innervation of the TrPs, the convergence-projection theory and the convergence-facilitation theory, the last two of which are linked with the CNS (3). Niddam et al. (28) point out that the central response to stimulus of TrPs indicates a relationship between somatosensory activity, the limbic system and the suppressing right dorsal hippocampal activity such that the hyperalgesia, typical of this pain syndrome, would be associated with abnormal activity in areas which process stimulus intensity and the negative affect.

The majority of current muscular pain syndrome models assume the existence of some form of event as the cause of muscular pain symptoms (2) either local (e.g. dental fractures, muscle fatigue caused by oral parafunctional habits and micro or macro muscular trauma, orthopaedic disorders, such as disc or class II skeletal displacements associated with craneomandibular 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) (2,3), although in cases of severe painful conditions, the importance of these causes in the genesis of the pain is not clear(8).

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 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 (3).

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