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Review Paper

Pathophysiology of myofascial trigger points: a review of literature

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Abstract

The most common source of musculoskeletal algia is the myofascial pain syndrome (MPS), its occurrence is related to excessive involuntary muscle contraction and is characterized by hardened regions. Myofascial trigger points (MTP) are hypersensitive nodules that refer pain spontaneously or under mechanical stimuli, present in 37 % of men and 65 % women, and may be classified as: active, latent, satellite, central junction. Assuming this pathological situation affects high number individuals may cause functional impairment and compromised quality life realized the need enlarge and update searches about MTP physiology. This literature review was conducted in English, Portuguese, and Spanish articles, published since 2009 (trigger points, musculoskeletal pain, MPS) in electronic databases: Bireme, PEDro and Scielo, and a book 2007. There are four important assumptions about the pathogenesis and pathophysiology of MTP: muscle spindle, the neuropathic process, scar tissue and terminal buttons and dysfunctional energy crisis. Most studies have only two classifications for MTP: active and latent, and few describe in great detail about the possible hypotheses of the pathogenesis of these. Recent literature is sparse regarding the physiology and aetiology, however, recent studies have sought to understand the pathophysiology ensure that the hypothesis with the highest validity.

Keywords: *Musculoskeletal Pain; Musculoskeletal Physiological; Trigger Points.*

1. Introduction

The most common source algia skeletal muscle (muscle, connective tissue and fascia) is myofascial pain syndrome (MPS), which is responsible for generating disability in patients who have pain this genre box, but many health professionals have a recognition flawed on this issue [1], [2], [3].
The occurrence of pain is related to excessive involuntary muscle contraction and is characterized by stiff regions, the presence of bands of palpable tension, internal to the muscle tissue, and / or associated with fascia and small, hypersensitive [4] points, regions hardened [5], [6] and well defined, called myofascial trigger points (MTP). As such, these spontaneously or under mechanical stimulation of the skeletal muscle (muscle, tendon, or fascia) in the presence or absence of a adjacent local or pain [7], [8], [9].
Its existence is related to degenerative, inflammatory, infectious, neoplastic, metabolic, macro or micro trauma of various structures processes [10], [11] having as injury mechanisms postures, over weight, and physical and emotional stress, causing the tensioning on specific muscle groups and / or associates. Affecting mainly the cervical, shoulder girdle, lumbar and temporomandibular joint of individuals in the ost active age groups between 31 and 50 years of age [1], [8]. Estimates from a study [12] conducted by Danes found that in 37% of men and 65% women, aged 30-60 years have MTP. In the United States (U.S.), it is believed that the MTP affects 23 million Americans and is therefore considered a common disorder / neuromuscular dysfunction of motor and sensory origin [10]. Other studies claim that this disorder is prevalent in 20% to 50% of the population of many countries [13].

There is a great variability among individuals affected muscles, the trapezius and the hardest hit by MTP may, these, being present in six different locations and each with its pattern of referred pain, lying bilaterally or unilaterally in higher fiber, medium and / or lower [14] 8).
One study found, in 200 asymptomatic young adults, a prevalence of MTP in 54% of women and 45% men, stating that the most affected is the trapezius muscle in both men and women, however, reported common occurrence the lower back, gluteal muscles, and the lumbar region square [15].
Symptoms associated with MTP are algia (burning, aching, stabbing or weight), decreased muscle strength, restricted range of motion, location or surrounding areas muscle fatigue, muscle stiffness, soreness, proprioceptive disorders (distortion of the weight of objects, tinnitus, dizziness and changes in perception) and autonomic phenomena (pilomotora activity, sweating, localized vasoconstriction, lacrimation and rhinorrhea) [1], [10], [14].
The MTP can be divided, according to their degree of sensitivity in latent, active, satellite and central junction. Latent produces continuous pain, but can be referred to mechanical stimulation or muscle contraction. The same can remain for years "silent" until the appearance of an injury due to a small business or by excessive stretching of the affected muscles. While the assets are causes of clinical symptoms such as constant pain with or without movement responsible for disabling the affected and the patient complaints muscle. The active MTP can be regressed naturally to the latent, if the rest is correct and the absence of evidence favoring the emergence of active MTP. The MTP satellite can develop in the same muscle of the principal MTP in other muscles in referred pain pattern of the primary MTP or synergistic muscles. The central MTP has a close relationship with the terminals dysfunctional buttons, and is near the center of the muscle fibers. The junction is a MTP in the musculotendinous junction and / or insertion of bone muscle enthesopathy identifying characteristic caused by the voltage produced by a taut band central MTP [10], [16], [17].
Research reports that a MPT can imaging be considered a preclinical MTP active, phase and states that it is unknown to the natural history of latent MTP, but says as the importance of knowing timely identify them in order to prevent progression to active also telling queos MTP can be divided into primary and secondary. The primary would form from acute muscle injuries, such as injuries and chronic injuries caused by repetitive use and overloading. The secondary would be originated by nociceptive stimuli of various disorders, such as tension headaches, radiculopathy, shoulder impingement syndrome, acute and chronic back pain, other chronic pain syndromes, temporomandibular disorders. However, the same study suggests that the pathophysiology of MTP motives remains controversial, although it has increased scientific knowledge on the subject in recent years. It is known that dysfunction of motor core plate, changes in muscle fiber and peripheral nociceptive pathways and are associated with this condition [18].
The MTP are commonly assessed by physical examination and palpation, and for higher reliability is required accuracy, consistency, stability, consistency and manual skill of the examiner, who must observe the presence of nodules and hypersensitive palpable tension zone a musculoskeletal area, visible or palpable location compression contraction [2], [4], [14]. Assuming that this pathological situation results in a large number of individuals with decreased functional capacity and impaired quality of life. Realizes the need to enhance the kinetic-functional evaluation to better physical therapy intervention promoting well-being for these patients, so that the present study was conducted with the objective of expanding and upgrading research on the physiology of MTP.

2. Methodology

A survey was conducted in Portuguese, Spanish and English, addressing the following keywords: trigger points, musculoskeletal pain, myofascial pain syndromes, in electronic databases: Bireme, PEDro and SciELO.
Articles published from 2009, full texts were freely available, that contained in its title or abstract some reference to etiology and / or physiology of activation MTP, and they were not related to any specific comorbidity were included as fibromyalgia, temporomandibular dysfunction, tinnitus. Exceptionally, some can study after examining the full article, contained some important information about what this article aims have been included.
In Bireme portal search revealed 91 articles, but only six of these articles met the inclusion criteria of the study. In PEDro portal 151 articles were found, nine were selected. The SciELO portal revealed publication of 14 articles on the subject, only two of these were selected. The texts independently by three authors, who subsequently synthesized in reflectively in order to obtain consistent information were analyzed.
Only one book of 2007 was used, since this was the only material found with detailed explanation about the pathogenesis and pathophysiology of MTP.

3. Results

Of the 25 articles initially selected, only six articles were included in the literature analysis, as were related to the theme. Systematic reviews and clinical trials, whose characteristics are systematized in Table I were included. It is worth noting the difficulty of extracting information and conclusions about the subject because few articles depict the physiology of MTP.

Table 1: Characteristics of Studies

Type of Study	Reference	Sample	Type of Intervention	Results
Literature Review	Kostopoulos and Rizopoulos, 2007 [17]	-	-	Reports four main theories on the pathogenesis and pathophysiology of MTP: the hypothesis of muscle spindle; the hypothesis of neuropathic process; the hypothesis of scar tissue, the hypothesis of dysfunctional terminals and power buttons crisis.
Literature Review	Bron and Dommerholt, 2012 [20]	-	-	Reports that it is important to know the cause of MTP to prevent its development and recurrence, since muscle overuse or direct trauma, and low sustained muscle contractions or repetitive, eccentric or concentric, maximal or submaximal level, developed during recreational activities , or sports when the muscle is used more than muscle and its recovery capacity is changed, leading to the emergence of MTP.
Literature Review	Carneiro, 2009 [6]	14 articles published in the last five years	-	The chronic tension-type headache was the most studied and is significantly related to the presence of MTP, noting that nociceptive signal coming from myofascial tissues - MTP in several pericraniais muscles - resulting in sensitization of the SS.
Clinical Trial	Moraska, Hickner, Kohrt and Brewer, 2013 [7]	2 individuals	Ischemic compression MTP upper trapezius	Observed changes in cell metabolism and blood flow in regions compared with MTP interstitial fluid content before, during and after ischemic compression thereof.
Clinical Trial	Bigongiari, Franciulli, Souza, Mochizuki and Araújo, 2008 [14]	56 individuals with or without MTP.	Conducting surface EMG and change detection of neuromuscular activity in MTP in situations of rest and maximal voluntary isometric contraction.	The EMG signal of the muscular portion with MTP was significantly higher compared with healthy muscle portion of the PG, and HG during rest. Individuals with active MTP had higher EMG signal than those latent MTP. In addition, individuals who had autonomic symptoms had higher EMG signal than those who did not have them at rest.
Clinical Study	Srbely, Dickey, Lee and Lowerison, 2010 [19]	40 individuals	Dry needle stimulation on muscles SS, IP and GM.	Through the stimulation needle dry it was observed that the MTP has anti-nociceptive effect through the perforation SS, IP and GM. The anti-nociceptive effect is related to the fact that IP and SS are neurologically linked to the root of C5, while SS and GM are independent segments. Suggested that the trigger point stimulation to elicit anti-nociceptive effect associated with modulation of segmental mechanisms.

MTP = Myofascial Trigger Points; EMG = Electromyography; PG = Pain Group; HG = Healthy Group; SS = Supraspinatus; IP = Infrastructure Prickly; GM = Gluteus Medium.

4. Discussion

There are several theories on the pathogenesis and pathophysiology of MTP, and only four are considered most important:. Muscle spindle, the neuropathic process, scar tissue and terminal buttons and dysfunctional energy crisis [17].

According to the theory of muscle spindle when they are abnormal produce abnormal electrophysiological signals, playing an important role in the pathogenesis of MTP. As spontaneous electrical activity and peaks identified in its vicinity and the endplate region. Spindles favor for the existence of MTP, giving tonics disorders and muscle spasms, as well as contribute to its onset in adjacent muscles without an obvious MTP due to muscle imbalance found in a region where one or more muscles myofascial present amendment. This imbalance causes compensatory movements, often of

abnormal electrical discharges and contractions by interfering with muscle spindle fibers and preventing their normal function, leading to a higher level of sensitivity [17].

In the event of a neuropathic process, MTP sea hypersensitivity are caused by a damaged nerve that innervate the affected muscle. Study adds to the pathophysiology of MTP is related to a discrete peripheral neurogenic secondary manifestation of central sensitization governed in part by segmental spinal mechanisms. The dry needle stimulation of different muscles within the same segment of the spinal cord provide an anti-nociceptive effect, this is an important consideration for understanding myofascial pain [19] would occur an excessive release of acetylcholine caused by stimulating the autonomic modulation release of chemicals that alter muscle tissue and your tone by changing the stimulation of the sympathetic nervous system [14].

As the hypothesis of scar tissue, the MTP may cause the production of scar tissue with histochemical findings characterized by high concentrations of protons, bradykinin, calcitonin peptide linked to the gene, substance P, tumor necrosis factor, interleukin 1-β, serotonin and norepinephrine [2], [7].

The hypothesis of dysfunctional terminals and power buttons crisis is considered the most current and debated about the pathophysiology of MTP. Claiming that they are originated by microtrauma, leading to destruction of the sarcoplasmic reticulum and are caused by very rapid repetitive movements (sudden falls, sports injuries, auto accidents), or asymmetrical postures and stress positions for long período [20]. After the destruction of the sarcolemma happen to Ca2+ release from concentrating near the lesion. If sufficient blood supply to the injured area the removal of Ca2+ occurs and the muscle returns to its normal condition, but if there is a good blood circulation, there will be an excessive release of acetylcholine in the synaptic cleft, depolarizing the postsynaptic membrane. However, the presence of acetylcholinesterase in the synaptic cleft is not sufficient to hydrolyze acetylcholine is released constantly. This disorder increases the opening of Ca2+ channels, which together with the large amount of free Ca2+ crosses the presynaptic membrane contributing to the connection with the seminal vesicles and the diffusion of acetylcholine in the synaptic cleft. Favoring for maximum and sustained contractile activity of the sarcomeres, increased metabolic needs. This prolonged muscle contraction of 30% to 50% of maximum effort provoke ischemia, hypoxia and accumulation of metabolic waste in the injury, as blood flow is compromised gathering Ca2+ is flawed, leading to lack of ATP and causing the crisis energy. The intense local hypoxia along with the energy crisis leads to release of nociceptive substances (bradykinin, prostaglandins and histamine), which sensitize the injured area causing pain. Thus, characterizing the activation and formation of MTP to combat local pain, muscle creates a defense mechanism in which there is loss of flexibility for changing physiological mechanical joint, leaving the structures easily exposed to other injuries [17].

Study [19] suggests that repetitive muscle contractions of low intensity would cause an injury by disrupting the cell membrane, damage to sarcoplasmic reticulum Ca2+ release, disruption of cytoskeletal proteins, such as desmin, titin and dystrophin. Suggesting an increase in cytochrome c oxidase (COX) and impaired oxidative metabolism in patients with myalgia. The same adds that the energy crisis resulting from circulatory alteration causes the mechanism of anaerobic muscle producing pyruvic acid converted during glycolysis, increasing the concentration of lactic acid, decreasing intramuscular pH. Researchers at the National Institutes of Health in the United States found that the pH in the region of MTP asset is the low five, stimulating excitement of nociceptors. It was suggested that small increases in the concentration of H+ íons contribute to hyperalgesia and central sensitization. The change in muscle metabolism weakens the binding between actin and myosin decreasing the effectiveness of muscle contraction, moreover studies have demonstrated changes in the waveform of blood flow when compared MTP active, latent MTP and normal sites confirming the hypothesis of terminals and knobs dysfunctional energy crisis [17].

Search [7] recently demonstrated changes in cell metabolism and blood flow before, during and after manual therapeutic interventions inhibition of MTP by moderate compression with the fingertips causes local ischemia and changes in interstitial fluid with consequent reduction contracture and pain.

5. Conclusions

There is a recent sparse literature on the pathophysiology and etiology of MTP. Most studies have only two calssificações for these: active and latent, and few describe in great detail about the possible hypotheses of the pathogenesis of these hypersensitive painful nodules on palpation. Recent studies have sought to understand the pathophysiology of the interstitial MTP watching your content, as well as through tests like EMG in an attempt to ensure that the most accepted hypothesis among the most important consideration.

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