

Myofascial pain syndrome and sensitization

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Abstract

Myofascial pain syndrome (MPS) is a major musculoskeletal pain that occur in every age group, and has been associated with numerous pain conditions including radiculopathies, osteoarthritis, disc syndrome, tendonitis, migraines, tension type headaches, computer-related disorders, spinal dysfunction, and pelvic pain. Myofascial pain is identified by palpating skeletal muscle for myofascial trigger points (MTrPs). A MTrP is classically defined by Professor Janet G Travell and Professor David G Simons as “a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. Although the specific pathophysiological basis of MTrPs development and symptomatology is unknown, there are evidences of histological, neurophysiological, biochemical, and somatosensory abnormalities. These emerging findings suggest that myofascial pain is a complex form of neuromuscular dysfunction consisting of motor and sensory abnormalities involving both the peripheral and central nervous systems.

Sensitization in corresponding spinal segments plays a major role in the formation of continuous pain in a given part of the body. The term called by Professor Andrew A. Fischer for this phenomenon is “spinal segmental sensitization” (SSS). SSS is a hyperactive state of the spinal cord caused by irritative foci sending nociceptive impulses from a sensitized damaged tissue to dorsal horn neurons. The clinical manifestation of dorsal horn sensitization includes hyperalgesia of the dermatome, pressure pain sensitivity of the sclerotome and myofascial trigger points within the myotomes, which are supplied by the sensitized spinal segment. There are significant elevated levels of substance P, calcitonin gene-related peptide (CGRP), bradykinin, tumor necrosis factor- α (TNF- α) and interleukin-1 β (IL-1 β), serotonin, and norepinephrine in the vicinity of the active myofascial trigger point. Overall, pH was significant lower in the active trigger point. The mechanism consists of the nociceptive stimuli generated in the sensitized areas bombarding the dorsal horn of the spinal cord. This causes central nervous system sensitization with resultant hyperalgesia of the dermatome and sclerotome and spreads from the sensory component of the spinal segment to the anterior horn cells, which control the myotome within the territory of the SSS. The development or amplified activity of MTrPs is one of the clinical manifestations of SSS. The Segmental Desensitization treatment consists of injection of local anesthetic agents in the involved dermatome to block the posterior branch of the dorsal spinal nerve along the involved paraspinous muscles. In addition, local anesthetic injection is applied peripherally near the foci of irritation in local soft tissue, directly into taut bands and trigger points, using a needling and infiltration technique. Stretching exercises, local heat application and additional transcutaneous electrical nerve stimulation (TENS) treatment complete the muscular relaxation after the injections. Extracorporeal shockwave therapy (ESWT) and High Intensity Laser (HTL) also play a role as desensitization. Prevention of recurrence should focus on appropriate ergonomics, posture correction, and strengthening of the injured muscles. In conclusion, MPS, a common pain syndrome consists of local pain for irradiation of trigger point and desensitization of the whole related spinal segment.

Introduction

Myofascial pain syndrome (MPS) is a major musculoskeletal pain that occur in every age group, and always has been associated with primary pain conditions including osteoarthritis, spondylosis, disc syndrome, tendinitis, migraines, tension type headaches, computer-related disorders or office syndrome, spinal dysfunction, and pelvic pain. Myofascial pain is identified by palpating skeletal muscle for myofascial trigger points (MTrPs). A MTrP is classically defined by Professor Janet G Travell and Professor David G Simons as “a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band” [1]. Although the specific pathophysiological basis of MTrPs development and symptomatology is unknown, there are evidences of histological, neurophysiological, biochemical, and somatosensory abnormalities. These emerging findings suggest that myofascial pain is a complex form of neuromuscular dysfunction consisting of motor and sensory abnormalities involving both the peripheral and central nervous systems.

Sensitization in corresponding spinal segments plays a major role in the formation of continuous pain in a given part of the body. The term called by Professor Andrew A. Fischer for this phenomenon is “spinal segmental sensitization” (SSS) [2,3]. SSS is a hyperactive state of the spinal cord caused by irritative foci sending nociceptive impulses

from a sensitized damaged tissue to dorsal horn neurons.

The nociceptive afferents either specific or nonspecific causes posterior dorsal horn sensitization and hyperalgesia and allodynia. This phenomenon gives anterior horn dysfunction, muscular spasm, trigger points and tender spots [2-4].

Clinical diagnostic criteria of myofascial pain

One interesting clinical characteristic of the MTrP is a local twitch response (LTR). It is the involuntary, transient and rapid localized contraction of muscle fibers which can be elicited by manual palpation. Gerwin *et al.* [5] pointed out the essential findings of an MTrP as the follows: 1) an exquisitely tender spot found in a taut band of muscle, 2) an LTR and/or referred pain to distant sites upon manual palpation

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or needling of the tender spot, 3) restricted range of motion, 4) reproduction of the patient's pain complaint through pressure on the MTrP, this criterion is only applicable for active MTrPs since latent MTrPs do not cause spontaneous pain. 5) regional muscle weakness and 6) autonomic symptoms. However, the accurate diagnosis of myofascial pain still depends exclusively upon the palpation skills and anatomical knowledge with specific referred pain patterns of the MTrPs by examiners.

Distinct neurobiology of muscle pain

It is important to identify the posterior horn sensitization which causes pain and the anterior horn sensitization which causes rigidity or muscle spasm and myofascial trigger points (MTrPs). The clinical manifestation of posterior horn sensitization includes hyperalgesia of the dermatome, pressure pain sensitivity of the sclerotome and the clinical manifestation of anterior horn sensitization includes muscle spasm and myofascial trigger points within the myotomes, which are supplied by the same sensitized spinal segment.

Actually muscle pain is described as aching, cramping, deep and difficulty in localization. Muscle pain involves nociceptive-specific neurons in the brainstem and spinal cord [6,7] and stimulates specific cortical areas that are related with affective or emotional components of pain [8]. Although muscle nociception is inhibited more intensely by descending pain-modulating pathways [9,10] persistent muscle nociception, compared to cutaneous nociception, is more effective at inducing maladaptive neuroplastic changes within the dorsal horn [11]. This distinct neurobiology changes explain the clinical characteristic of the difficulty in elicit muscle pain.

The clinical manifestation of dorsal horn sensitization includes hyperalgesia of the dermatome, pressure pain sensitivity of the sclerotome and myofascial trigger points within the myotomes, which are supplied by the same sensitized spinal segment. There are significant elevated levels of substance P, calcitonin gene-related peptide (CGRP), bradykinin, tumor necrosis factor- α (TNF- α) and interleukin-1 β (IL-1 β), serotonin, and norepinephrine in the territory of the active MTrP. Overall, pH was significant lower in the active MTrP. The mechanism consists of the nociceptive stimuli generated in the sensitized areas bombarding the dorsal horn of the spinal cord. Repetitive stimulation of A δ and C fibers causes WDR sensitization in the same segment and produces bombarding. This gives reflexes to segmental α and γ motorneuron, causing muscles spasm and MTrPs. The bombarding causes central nervous system sensitization with resultant hyperalgesia of the dermatome and sclerotome and spreads from the sensory component of the spinal segment to the anterior horn cells, which control the myotome within the territory of the SSS in the same segment. The development or amplified activity of MTrPs is one of the clinical manifestations of SSS [2-11].

Embryological concepts of spinal segmental sensitization [2-4,12-16]

Mesoderm immigrates via primitive streak and transforms itself several times. The epiblast-epithelial (ectoderm cells) are transformed into segmented mesoderm cells. A renewed transformation of the somites into mesenchymal tissue, cells are given off into the gravity from the medial and anterior walls of somites. The central cells as well as the cells of the anteromedial wall from the sclerotome that as a result forms an important component of the axial skeleton. The posterolateral portions of the somite wall is called a dermatomyotome, consists of two portions; one is myotome, which lie more medially and form the origin

of the skeletal musculature; another one is posterolateral dermatome which the subcutaneous tissue proceeds. The myotome form into a posteromedial portion, which represents the origin of the spinal extensor muscles. These muscles retain their segmental ordering. A second anterolateral portion forms the spinal flexor muscles, the shoulder muscles and those of the hip region, as well as the trunk walls and musculature of the extremities.

The development of the nervous system is follow the segment. It is important to understand that the paraspinal muscles and the peripheral muscles have the same innervation since the embryological concept. For this reason, Professor Fisher had better result injecting paraspinal muscles for treating peripheral trigger points.

When muscles hurts, the priosteum, the skin and subcutaneous tissue of the same segments hurts because have the same innervation.

Sensitization of myofascial pain syndrome

Myofascial pain syndrome induced profound altering neuronal excitability and architecture in structures of the pain matrix structures as the spinal cord, thalamic nuclei, cortical areas, amygdala and periaqueductal gray area. This dynamic process can alter pain threshold, pain intensity and emotional affect [10]. Signaling in the pain matrix structures may begin with activation of polymodal nociceptors, structures which can be sensitized by substances released from damaged tissue and the nociceptor terminals themselves. Prolonged noxious input may lead to long-term changes in gene expression, somatosensory processing and synaptic structure. A continuous noxious input into the dorsal horn called "afferent bombardment" results in the co-release of L-glutamate and substance P (SP). The releasing of these two substances can lower thresholds for synaptic activation and open previously ineffective synaptic connections in wide dynamic range (WDR) neurons, leading to central sensitization [2-4,17,18].

Sensitization up-regulates ion channel and receptor expression and increases the number of these membrane proteins on nociceptors and dorsal horn neurons. Under normal circumstances, a dynamic balance exists between pain's role in facilitating and inhibiting function. Neurons conveying nociceptive information are controlled by a variety of inhibitory interneurons, structures critically involved in preventing the transition from acute to chronic pain [2-4,19].

Clinical evaluation of myofascial pain syndrome and sensitization

Spinal segmental sensitization (SSS) is a hyperactive state of the dorsal horn caused by bombardment of nociceptive impulses from sensitized tissues (such as active MTrPs or visceral structures such as the renal calculi). Clinical manifestations of the sensitized spinal segment include MTrPs, dermatomal allodynia (pain by non-painful stimulus) and hyperalgesia (increased pain by painful stimulus) in addition to sclerotome tenderness within the involved myotomes. Then, segmental sensitization occurs throughout neuron hypertrophy as well as upregulation of excitatory neurons, prohyperalgesic peptides, and neurotransmitters at the dorsal horn [2-4,12,20-22].

Segmental dermatomal levels are evaluated by the follows (1) Scratching the skin with the sharp edge of a paper clip or Wartenberg pinwheel – this noxious stimulus is applied across dermatomal borders and the patient is instructed to simultaneously report any sharpening or dulling in the sensation of pain during the procedure. An increased painful response is indicative of hyperalgesia. (2) Picking up the skin

between the thumb and forefinger and rolling the tissue underneath, also known as a —pinch and roll test - this non-noxious stimulus is applied across dermatomal borders and the patient is instructed to simultaneously report any sensation of pain. The sensation of pain is indicative of allodynia, a finding that is the most sensitive indicator for the diagnosis of sensitization [2-4,12,20-22].

Segmental myotome levels are examined by the follows (1) Palpating segmentally related musculature for tender spots, taut bands and MTrPs. (2) Applying a pressure algometer to measure the local tenderness or pain pressure threshold (PPT) along the myotome [2-4,12,20-22].

Segmental sclerotome levels are examined by the follows (1) Palpating segmentally related tendons (tendonitis), entheses (enthesitis), bursae (bursitis) and ligaments (ligament sprain). (2) Applying a pressure algometer to measure the local tenderness (PPT is the minimum pressure that elicits pain and is considered abnormal if it is at least 2 kg/cm² lower than a normosensitive control point) along these sclerotome structures [2-4,12,20-22].

These examination techniques are used to determine the segmental sensitization manifestations of an individual's pain syndrome. Dermatome, myotome and sclerotome segmental findings often overlap, making diagnosis and treatment of the sensitized segment difficult.

Segmental desensitization

Segmental desensitization consists of injection of local anesthetic agents or dry needling in the involved dermatome to block the posterior branch of the dorsal spinal nerve along the involved paraspinal muscles. In addition, local anesthetic injection or even dry needling is applied peripherally near the foci of irritation in local soft tissue, directly into taut bands and trigger points, using a needling and infiltration technique. Elicitation of one or more local twitch responses (LTRs) is a goal and often benefits those with pain secondary to MTrPs. Stretching exercises, local heat application and additional transcutaneous electrical nerve stimulation (TENS) treatment complete the muscular relaxation after the injections. Extracorporeal shockwave therapy (ESWT) and High Intensity Laser (HTL) also play a role as desensitization. Prevention of recurrence should focus on appropriate ergonomic changes common in patients' day-to-day activities to avoid repetitive stress to the injured muscles. In conclusion, MPS, a common pain syndrome consists of local pathology and SSS. Hence therapeutic approaches require varieties of techniques for eradication of trigger point and desensitization of the whole related spinal segment [2-4,12,20-22].

Modalities and manual therapies are often clinically effective for eradication of active MTrPs and desensitization sensitized spinal segments. For example, various forms of electrical stimulation including micro current, transcutaneous electrical nerve stimulation (TENS), manual therapies and spray and stretch are commonly used to treat myofascial pain and SSS. However, if pain relief is only partial or pain persists despite several treatments using such various modalities, then needling and injection techniques should be considered, particularly in chronic cases in which the physical examination reveals severe and persistent allodynia and hyperalgesia, suggesting dense dermatome, myotome and sclerotome manifestations of SSS [2-4,12,20-22].

Fischer *et al.* [2,3,21] developed a technique utilizing injection of 1% lidocaine into the paraspinal muscles adjacent to the spinous processes. A 25-gauge needle, of sufficient length to reach the deep layers up to

the vertebral lamina, is inserted in the sagittal plane. Injection is performed between the levels of the spinous processes corresponding to the affected segmental levels of sensitization as identified on physical examination. The needle is inserted through the paraspinal muscle to a maximal depth but before contacting the vertebral lamina. The needle is aspirated (in order to avoid blood vessels) and then approximately 0.1 mL of anesthetic is injected; the needle is then withdrawn to a subcutaneous level and redirected in the caudal direction, ending about 5mm from the previous deposit of anesthetic solution. One continues this procedure, going as far as the needle reaches. The same procedures are then repeated going in the cephalad direction. The result of this technique is to effectively block the medial branch of the posterior primary rami at affected segmental levels.

Conclusion

MPS, a common pain syndrome consists of local pathology and SSS. Hence therapeutic approaches require varieties of techniques for eradication of trigger point and desensitization of the whole related spinal segment.

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