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Review Article

Botox in Myofascial pain syndrome: A comprehensive review

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ABSTRACT:
Myofascial pain syndrome (MPS) is characterized by the presence of trigger points, palpable muscle abnormality (fibrocystic nodule) and referred pain distal to the trigger point. Most of the treatment methods for MPS are aimed to reduce the pain in trigger points and reduce the muscle spasm. Botulinum toxin has been studied in MFS eradication because an activation of toxin might be able to block a release of acetylcholine at the presynaptic terminal resulting in a muscle relaxation. Therefore, the pain is relieved by preventing the release of pain neurotransmitters at the sensory neuron.
Key words: Myofascial pain, Botox

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INTRODUCTION

Myofascial pain syndrome is a common condition characterized by chronic, focal muscle pain, stiffness and fatigue. Its clinical hallmark is the 'trigger point,' defined as a region of focal tenderness located in a palpable taut band of muscle fibers, brisk palpation of which results in a muscle twitch response. Since tender, taut muscle bands, or 'latent' trigger points, can occur in normal individuals, two additional criteria must be present for the trigger point to be termed 'active' or abnormal. Mechanical stimulation of the trigger point produces referred pain, and this reproduces a chronic pain complaint. The pathophysiology of myofascial pain syndrome remains unclear despite its being a major source of physical suffering and disability.[1-3]

ETIOLOGY

Various factors can contribute to MPS. The common risk factors are:

- Traumatic events
- Ergonomic factors (e.g., overuse activities, abnormal posture)
- Structural factors (e.g., spondylosis, scoliosis, osteoarthritis)
- Systemic factors (e.g., hypothyroidism, vitamin D deficiency, iron deficiency)[4-6]

EPIDEMIOLOGY

The exact prevalence of MPS in the general population has rarely been mentioned in existing literature. However, 30% to 85% of patients with musculoskeletal pain suffer from this condition. MPS is usually found in the population aged from 27 to 50 years. The gender difference in MPS incidence remains unclear.[5, 6]

PATHOPHYSIOLOGY

Nowadays, the exact pathophysiology of MPS is still unknown. Many researchers try to find scientific evidence and formulate hypotheses. One of the most accepted theory is energy crisis of muscle fibers. Repetitive or prolonged activity can cause overloading of the muscle fibers which lead to muscle hypoxia and ischemia. In addition, intracellular calcium pumps are dysfunctional due to energy depletion. Intracellular calcium increase induces sustained muscle contraction which results in the development of taut bands. Moreover, inflammatory mediators caused by muscle injury contribute to pain and tenderness of the affected muscles. Other than this

hypothesis, there are many theories such as neurogenic inflammation, sensitization and limbic dysfunction that are proposed to relate to MPS.[7- 10]

BOTULINUM TOXIN

The bacteria, Clostridiu botulinum, produce the most potent human biological toxins known and are responsible for the life-threatening paralytic illness, botulism. Botulinum toxin, a 150 kDa protein, binds irreversibly to presynaptic cholinergic nerve terminals, and once internalized, blocks the exocytosis of the neurotransmitter, acetylcholine, thereby inhibiting muscle contraction. Skeletal muscle, thus chemically denervated, remains paralyzed until the motor nerve supplying it sprouts new axons and forms new synaptic contacts to re-establish the neuromuscular junction. Toxin potency is expressed in mouse units, 1 unit representing the estimated LD,, for 18-20 g female Swiss-Webster mice, which is equal to approximately 0.4 ng of botulinurn toxin type A (Botox). [7- 10]

BOTOX FOR DYSTONIA

The treatment of dystonia recently has been revolutionized by Botox. Dystonia refers to a neurological disorder characterized by involuntary, sustained, inter mittent or repetitive, muscie contractions resulting in abnormal twisting or deviations of posture. When injected into the muscles of patients with focal dystonias such as blepharospasm, spasmodic torticollis, spasmodic dysphonia, or hemifacial spasm, abnormally contracting muscles are made to relax. Many of these patients will experience substantial relief for several months, the time required for new synapses to form. Efficacy and safety of Botox for dystonia are supported by extensive literature. Most patients with dystonia will require repeated injections at intervals of several months. [7- 10]

BOTULINUM TOXIN INJECTIONS ON TRIGGER POINTS FOR MYOFASCIAL PAIN

Dosage and the number of trigger points injected for each patient varies in clinical practice depending on the physician's experience and expertise. It is intuitive that these factors may affect the clinical outcomes. In Ferrante's study, subjects were given either BTX-A (10, 25, or 50 units per trigger point) for doses that ranged up to 250 units, or saline in up to five trigger points. Wheeler et al identified the most tender trigger point, and injected it with 50 units of BTX-A, 100 units of BTX-A, or normal saline, but there was no significant difference among the three groups. The studies that showed no significant improvement over placebo involved injections in no more than five trigger points per patient. In Graboski's trial, up to eight trigger points were injected per patient. Although no significant difference was demonstrated between the BTXA and 0.5 % bupivacaine group, both BTX-A and bupivacaine treatments were effective in reducing pain when compared to baseline. Another randomized, double-blinded, placebocontrolled trial by Benecke et al used a set of standardized trigger-point sites that included ten predetermined, fixed trigger-point injections in the head, neck, and shoulder for myofascial pain. This study found significant improvement at eight weeks after treatment with BTX-A in patients with upper-back myofascial pain syndrome. [10- 15]

A more recent study by Qerama et al. with 30 patients compared motor endplate activity with EMG and pain (spontaneous and referred) thresholds between two groups that received only one injection of either 50 units BTX-A or 0.25 ml isotonic saline. The BTX-A group had decreased motor endplate activity on EMG readings, but had no effect on either referred pain or spontaneous pain compared to placebo group. These trials suggest that the total number of trigger points injected may play a role in determining the efficacy of BTX-A injections. Inadequate coverage of trigger points is a potential reason for poor clinical outcomes in some of the trials. Although all the trials in this review adopt a diagnostic criterion to identify active trigger points, the results produced from this review are still mixed. Another factor that may potentially influence the outcome is that various diagnostic criteria exist for myofascial pain, and the diagnosis of myofascial pain is based mainly on history and physical examination. The essential part of the physical examination is to locate the trigger points by palpation of the taut band in the musculature. Proper identification of trigger points requires precision of the pressure applied during examination and the subjective experience of the examiners. Prior studies have confirmed poor consistency among the examiners in identifying a trigger point.[10- 15]

PITFALLS IN THE STUDY OF BONT EFFICACY IN MYOFASCIAL PAIN SYNDROMES

When studying the effect of BoNT in the treatment of MPS, the onset of action of BoNT-A and the duration of its clinical effect must be kept clearly in mind. BoNT takes about 5 days before its clinical effect becomes evident. The clinical effect may not be evident for 1–2 weeks after injection. The effect of BoNT on the sensory or pain component may be much sooner. The timing of the effect on the taut band and the effect on pain are dissociated, and may not be congruent. The clinical effect of BoNT is variable, but on average is about 12 weeks, with a range of 10–14 commonly observed. The effect may be shorter or longer in some individuals, but the point is that the effect is limited. The maximum effect on the motor unit is reached at about 6 weeks. The timing of the maximum effect on the sensory component of pain is not known in MPS. Harden et al, in their trigger point headache study, noted that the effect was gone at 12 weeks, but it was recaptured in the open label trial.

This is not a failure of BoNT treatment, but an expected time course of the action of BoNT. [15- 18]

If a longer effect is desired, BoNT may be used again in the pertinent muscles. When used in treating MPS, BoNT can reduce or inhibit the presence of the taut band, and, secondarily, reduce pain. It may also have effect on pain directly. The advantage of BoNT is its longer duration of action compared with lidocaine or to deep dry needling. The goal of treatment with BoNT should be to reduce the burden of painful trigger points without producing weakness. The referred pain from the trigger points should be reduced, whether or not the tenderness at the trigger point zone is reduced, although that, too, is a goal. Given this understanding of the time course and action of BoNT, the questions asked must be framed appropriately. Thus, it is pertinent to investigate the primary question as to whether or not there is reduction in pain at all. It is appropriate to ask about overall global sense of pain, and also to investigate the issue of whether or not referred pain is reduced. BoNT may partially or totally inactivate a group of trigger points. It is relevant to ask, then, if there is a decrease in the use of adjunctive medications or treatments, including lidocaine injections. MPS often results from the involvement of multiple trigger points in multiple muscles.[15- 18]

A study should therefore address the inactivation of trigger points in all the muscles involved in producing pain in a given MPS, not just those in one or 2 muscles. BoNT should be injected into the trigger point zone, not just at any predetermined site in the muscle that may be irrelevant to the trigger points in that muscle. Treatment outcomes commonly compare the effect of BoNT to the effect of lidocaine, but the relative number of lidocaine treatments involving time off of work or the number of visits to physical therapists to achieve the same effect should be taken into account. Other points that affect our interpretation of the BoNT effect are discussed immediately following, but it is clear that most studies comparing BoNT and other treatments (usually lidocaine trigger point injections) do not address these issues.[15- 18]

CONCLUSION

The use of botulinum toxin in MPS has a pharmacological and pathophysiological basis. In MTrPs there is excessive Ach release and an increase in the concentration of nociceptive neurotransmitters in the biochemical milieu of the MTrPs. BTA appears to be effective on both targets, reducing ACh release and blocking nociceptive neurotransmission.

REFERENCES

1. Kuan TS, Chang YC, Hong CZ. Distribution of active loci in rat skeletal muscle. Journal of Musculoskeletal Pain. 1999;7(4):45–54.
2. Watkins LR, Hutchinson MR, Ledeboer A, Wieseler-Frank J, Milligan ED, Maier SF. Glia as the "bad guys": implications for improving clinical pain control and the clinical utility of opioids. Brain, Behavior, and Immunity. 2007;21(2):131–146.
3. Sluka KA, Kalra A, Moore SA. Unilateral intramuscular injections of acidic saline produce a bilateral, long-lasting hyperalgesia. Muscle & Nerve. 2001;24(1):37–46.
4. Gerwin RD, Dommerholt J, Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. Current pain and headache reports. 2004;8(6):468–475.
5. Costa J, Espírito-Santo C, Borges A, et al. Botulinum toxin type A therapy for cervical dystonia. Cochrane Database of Systematic Reviews. 2005;(1)CD003633
6. Simons DG, Hong CZ, Simons LS. Endplate potentials are common to midfiber myofacial trigger points. American Journal of Physical Medicine and Rehabilitation. 2002;81(3):212–222.
7. Hong CZ, Simons DG. Pathophysiologic and electrophysiologic mechanisms of myofascial trigger points. Archives of Physical Medicine and Rehabilitation. 1998;79(7):863–872.
8. Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. Journal of Applied Physiology. 2005;99(5):1977–1984.
9. Mense S. Muscle pain: mechanisms and clinical significance. Deutsches Arzteblatt. 2008;105(12):214–219.
10. Ferrante FM, Bearn L, Rothrock R, King L. Evidence against trigger point injection technique for the treatment of cervicothoracic myofascial pain with botulinum toxin type A. Anesthesiology. 2005;103:377–83.
11. Wheeler AH, Goolkasian P, Gretz SS. Botulinum toxin A for the treatment of chronic neck pain. Pain. 2001;94(3):255–60.
12. Graboski CL, Gray DS, Burnham RS. Botulinum toxin A versus bupivacaine trigger point injections for the treatment of myofascial pain syndrome: a randomized double blind crossover study. Pain. 2005;118:170–5.
13. Wheeler AH, Goolkasian P, Gretz SS. A randomized, double-blind, prospective pilot study of botulinum toxin injection for refractory, unilateral, cervicothoracic, paraspinal, myofascial pain syndrome. Spine (Phila Pa 1976). 1998;23(15):1662–6.
14. Qerama E, Fuglsang-Frederiksen A, Kasch H, Bach FW, Jensen TS. A double-blind, controlled study of botulinum toxin A in chronic myofascial pain. Neurology. 2006;67:241–5.
15. Benecke R, Heinze A, Reichel G, Hefter H, Göbel H. Botulinum type A toxin complex for the relief of back myofascial pain syndrome: how do fixed-location injections compare with trigger pointfocused injections? Pain Medicine. 2011;12:1607–14.
16. Harden RN, Cottrill J, Gagnon CM, Smitherman TA, Weinland SR, Tann B, et al. Botulinum toxin a in the treatment of chronic tension-type headache with cervical myofascial trigger points: a randomized, double-blind, placebo-controlled pilot study. Headache. 2009;49:732–43.
17. Venancio RA, Alencar Jr FG, Zamperini C. Botulinum toxin, lidocaine, and dry needling injections in patients with myofascial pain and headaches. Cranio. 2009;27:46–53.
18. Langevin P, Peloso PM, Lowcock J, Nolan M, Weber J, Gross A, et al. Botulinum toxin for subacute/chronic neck pain. Cochrane Database Syst Review. 2011;6:CD008626.