An Analysis of a Case Study Examining the Efficiency of Deep Dry Needling in the Treatment of Hypertonia

Gulwish Sadique, Manjit Kumar, Minhaj Tahir
Faculty of Paramedical Sciences, Rama University, Mandhana, Kanpur, U.P., India

Abstract.

Backgrounds: The patient has spastic tetraparesia and is four years old.
Results: Using the Modified Ashworth Scale, a reduction in spasticity was seen in every muscle receiving deep dry needling treatment [MAS]. Also, there was an increase in passive range of the thumb's motion.
Conclusions: Deep dry needling treatment reduced resistance to passive motion. It is challenging to distinguish between decreased spasticity and altered viscoelastic characteristics as the cause of the decreased resistance to passive movement as evaluated by the MAS. Since we address trigger points, it's probable that modifications in the viscoelastic characteristics—rather than spasticity—are more responsible for the improvement in MAS scores.

Keywords words: dry needling, Trigger points, myofascial pain disorders, muscle hypertonia, and spasticit. Trigger points, dry needling, myofascial pain syndromes, muscle hypertonia, and spasticity Adapted Adapted Ashworth Scale.
DOINumber: 10.48047/nq.2022.20.7.NQ33559 **NeuroQuantology 2022;20(7):4660-4665**

INTRODUCTION

.Hypertonia is the resistance experienced when passively moving a limb, or the resistance to pas sive movement [RTPM].

Overindulgence inPatients with upper motor ne urone [UMN] lesions experience spasticity, thick ening of the muscle, and modifications to the m uscle's viscoelastic characteristics, which can ev entually result in the formation of fixed muscle contract res. As one aspect of upper motor neurone syndrome, spasticity is described as "a velocity dependent increase in the tonic stretch reflex with exaggerated tendon reflexes, resulting from the hyperexcitability of the stretch reflex" (1). It is now acknowledged that hypertonia is caused by more than just a muscle's hypertrophic stretch reflex; other positive RTPM is largely influenced by biomechanical alterations and characteristics of the UMN syndrome. Working with children who are profoundly impaired, paediatric physiotherapists frequently observe that one of the biggest challenges parents face in raising their kids is the rise in RTPM. Currently, nonselective systemic antispascity medications like baclofen, dantrolene, tizanidine, diazepam, chlorazepate dipotassium, clonazepam, or clonidine are used to treat muscle spasticity. which may result in functional loss and have a nonselective mode of action. Ironically, in certain individuals, certain medications lessen the force in the healthy muscles without affecting the spasticity of the muscles. Moreover, the effectiveness of oral antispasticity medications decreases with continued use. Acceptance appears after a few months of treatment, and to maintain the initial clinical response, dosage increases must frequently be made gradually. The frequency and intensity of these medications' side effects are frequently increased by the high dosages needed. A different approach to treating muscle spasticity is chemical neurolysis using alcohol-based compounds like phenol. Nevertheless, skin sensory loss is frequently the result of nerve blocks and motor point injections in the upper limbs, and these procedures may also result in persistent dysesthesia or causalgia. Additionally, with continued use, their effects frequently fade. Botulinum toxin type A, or BTT-A, has demonstrated efficacy as an antispasticity agent in recent years. But there may also be minor negative effects. side effects include rashes on the skin, flu-like symptoms, and muscle weakness in the injected area. The selective treatment for spasticity has been shown to be focal injection of BTX-A; however, a review of the available data indicates that clinicians are not in general agreement regarding the dosage, injection site, injection technique, etc. Acetylcholine is prevented from entering the synaptic cleft by BTX-A. It appears to have a remote effect as well, which an indirect central effect could account for.About thirty years ago, dry needling was employed as a method of pain relief. Additionally, studies have demonstrated its effectiveness in treating hemiparetic shoulder pain syndrome (2). Based on the extensively documented efficacy of deep dry needling (DDN) on neuropathic pain, the effectiveness and efficiency of DDN on trigger points (TrPs) were examined in this study. In addition to pain, TrPs have been linked to a broad range of indications and symptoms, including autonomic dysfunction, tingling, weakness, and resistance to passive stretching and muscle shortening (3). When contrasting DDN and BTX-A treatment, we could say that the effect of BTX-A is generated in the motor endplate zone.Nevertheless, their modes of action differ; DDN operates mechanically, whereas BTX-A acts chemically. The proposed mechanism of action for DDN in TrP treatment involves mechanically disrupting dysfunctional motor endplates. This is because, in accordance with David Simons' integrated hypothesis regarding the etiopathogeny of TrPs (3,4), there are contraction knots, or active loci, in these endplates that result in palpable findings of taut bands and TrPs.

The integrated hypothesis has been supported by the fact that the BTX-A has also been shown to be effective in the treatment of TrPs. As stated According to this hypothesis, neurotransmission inhibition triggered by BTX-A would partially resolve the problem of TrPs as it acts on its initial cause. TrPs are found in dysfunctional motor endplates in which excessive acetylcoline release occurs. According to this perspective, DDN and BTK-A would function in the same anatomical structure, albeit through distinct mechanisms. It has been demonstrated by recent groundbreaking research (5) that twitch obtaining DDN results in a lavage of sensitising substances whose presence may encourage the persistence of motor endplate dysfunction (6). It is investigated in the case study we are presenting whether DDN can also have an impact. on a hypertonia patient's resistance to passive muscle stretch.

DESCRIPTION OF THE CASE

The child who is ill is four years old. He was delivered via caesarean section during the 38th week of pregnancy. Severe hypoxicischemic encephalopathy, which manifests clinically as spastic tetraparesia with axial hypotonia and severe impairment of the right upper limb, was the medical diagnosis. This condition was brought on by perinatal foetal distress.The child suffers from severe ocular motility impairment and central hypovision. He is completely dependent on a carer for all of his everyday tasks. His only way to communicate is to cry or smile to show joy or sorrow.

EVALUATION

Range of Motion in Passive elbow, wrist, and finger joints were evaluated for passive range of motion (PROM). With a fisted hand, the upper limbis fixed in a position of 30º shoulder abduction, fully flexed elbow, and 70º wrist flexion, but if the stretch is done slowly, all joints—aside from the thumb—can achieve the full PROM. PROM was therefore only evaluated in the thumb. The thumb was placed in the following positions with the hand in the patient's resting position: fully flexed and opposed, one-quarter, one-half, and three-quarters open, as well as fully extended with passive muscle stretch. The patient's thumb could only be passively stretched to a half-open position during the initial assessment. When the hand is at rest, it is closed and it can Resistance to Passive Movement and SpasticityThe Modified Spasticity Scale was used to measure Ashworth Scale [MAS] (7) both prior to and following the intervention. In order to minimise the possibility of increased spasticity resulting from the tonic-asimetric reflex, the patient was positioned supine with their head in the middle line. It appears to have a remote effect as well, which an indirect central effect could account for.About thirty years ago, dry needling was employed as a method of pain relief. Additionally, studies have demonstrated its effectiveness in treating hemiparetic shoulder pain syndrome (2). Based on the extensively documented efficacy of deep dry needling (DDN) on neuropathic pain, the effectiveness and efficiency of DDN on trigger points (TrPs) were examined in this study. In addition to pain, TrPs have been linked to a broad range of indications and symptoms, including autonomic dysfunction, tingling, weakness, and resistance to passive stretching and muscle shortening (3). It also seems to have a remote effect, which could be explained by an indirect central effect. Three decades ago or so, dry needling was utilised as a means of reducing pain. Furthermore, research has indicated that it is efficacious in managing hemiparetic shoulder pain syndrome (2). This study looked at the effectiveness and efficiency of deep dry needling (DDN) on trigger points (TrPs), based on the well-established efficacy of DDN on neuropathic pain. TrPs have been connected to a wide range of indications and symptoms, such as resistance to passive stretching and muscle shortening, tingling, weakness, and autonomic dysfunction, in addition to pain (3). The integrated hypothesis has been supported by the fact that the BTX-A has also been shown to be effective in the treatment of TrPs. As stated According to this hypothesis, neurotransmission inhibition triggered by BTX-A would partially resolve the problem of TrPs as it acts on its initial cause. TrPs are found in dysfunctional motor endplates in which excessive acetylcoline release occurs. According to this perspective, DDN and BTK-A would function in the same anatomical structure, albeit through distinct mechanisms. The persistence of motor endplate may be aided by the lavage of sensitising substances produced by twitch obtaining DDN, as demonstrated by recent groundbreaking research (5). The parents and physiotherapist established the intervention's goals, which included reducing spasticity or RTPM to enhance the the way parents raise their children.

The elbow flexors (biceps brachii and brachialis), wrist flexors (flexor carpis radialis, flexores digitorum superficialis, and profundus), and thenar muscles (opponens pollicis) were the muscles that were treated. Nine sessions made up the intervention for the thenar muscles. The flexor muscles of the wrist and elbow were also worked on from the fifth to the ninth session. For the first four sessions (theenar muscles), the intervention was done twice a week; for the final five sessions (all muscles), it was done once a week.

The following standards were applied in order to diagnose the TrPs:

ESSENTIAL CRITERIA

1. Limitation to passive elongation (3)
2. Palpable taut bands (3) in the impacted muscles
3. A palpable bump in a tense area

Verifying Standards

1. Identification of the local twitch response [LTR] by touch or vision. This discovery likely represents the most precise individual clinical test of a TrP (8).
2. An increase in the axial muscles' global inflammatory response (GIS). Although this

criteria has not been published, clinical experience has led to its establishment.
The GIS response's presence may or may not be related to LTR. The patient showed no signs of pain or discomfort in response to this response, and a significant reduction in muscle resistance of the muscles treated for a brief period of time followed the GIS. Muscles were positioned in a sub-maximal stretch position, where a significant increase of resistance is felt. As the treatment works, the therapistappliesa musclestretchuntila new increase of resistance is felt. Once the needle has been introduced in the TrP, two DDN techniques have been used:
1. Hong's [fast-in, fast-out] technique until a LTR

RESULTS

Using the MAS of spasticity, the target muscle group's degree of RTPM was measured as the main outcome. The two parameters could be observed and analysed through the use of video recording.
For every treated muscle, there was a reported improvement in spasticity that was clinically significant. Refer to Tables 1 through 3. It was noted that there was an improvement in the hand opening while at rest and when passive muscles were stretched. When the therapy began, the thumb could be passively moved to a partially opened position and the hand was fisted. or GIS can be felt. 2. Other manipulation of the needle [twisting]. The hand was in a quarter-open position and could be fully opened with a passive muscle stretch after nine treatments.
Despite the fact that this is a highly subjective measurement, parents stated that they had less trouble managing the child and that they had also noticed a drop in the contralateral limb's RTPM. However, the MAS measure does not support this final point.which revealed the left upper limb RTPM to be unchanged. The last treatment in this case report was completed shortly before the Christmas break, and following this time off, a new assessment was carried out. This demonstrated that outcomes had been maintained. We stopped the treatment after the holidays because the child started a new medication regimen that included diazepam and might have an impact on the outcome measurements.

DISCUSSION

As the introduction noted, RTPM is a complicated metric that will be impacted by numerous variables, of which only variability may be one. The MAS has significant drawbacks and is unreliable in differentiating between the various hypertonia components. The lack of standardisation in the test conditions is another drawback of the MAS.For instance, some medical professionals evaluate muscle tone during rest without first stretching the muscles. Others (9) advise moving the limb in both directions several times right before the measurement is made. Measurement error may result from this lack of standardisation because the stretch reflex excitability in It's possible that the activated muscle's state differs from the resting state (10). However, it has been shown to be somewhat reliable for classifying the RTM at the elbow and wrist flexors, making the MAS the most frequently used test for measuring muscle spasticity in clinical practice and research (11). 4663
According to some authors, the MAS measures hypotonia, or resistance to passive muscle stretch, as opposed to spasticity (12–14).There are elements that may complicate the MAS. Research from the literature indicates that a decrease in soft tissue compliance brought on by less use may have contributed to the increase in RTPM (13). The recent history of movement has an impact on the RTPM. This would imply that, rather than spasticity, the increase in RTPM seen in the impaired arm may have been primarily related to modifications in the viscoelastic characteristics of the soft tissues (13, 14).
It was also observed that prior to treatment, there was a high velocity-dependent RTPM that diminished after the treatment. This can also be attributed to viscoelastic properties of muscles, which are velocity dependent, but these changes can also be due to changes in spasticity. We have found, as a limitation of the

study, the possibility that the improvement could also be achieved performing DDN in zones other than the endplate zone, even in other parts of the body. Although treatment in neurologic patient must be assessed on the basis of motor and functional improvent, for this severly impaired patient, reducing muscle tone, as assessed by MAS and PROM, is the real goal. Deep dry needling has been tested on adult patients with incomplete spinal cord injury (16) as well as on various school-treated children (non-published data). Although there isn't any scientific proof, it was found in these clinical cases that DDN affected the upper limbs more permanently than the lower limbs, perhaps as a result of the weight bearing component that may serve as a contributing cause to spasticity When treating children with severe spasticity and restriction of PROM, the outcomes were superior compared to those with mild impairments. Other than these factors, it was occasionally difficult to treat kids who could recognise the "threatening presence" of a needle. Based on all of the information gathered from the clinical practice, it was determined that the ideal patient would be a young person with severe spasticity or restricted PROM who also had cognitive impairment or visual loss that would make it impossible for them to understand that they would be receiving needle treatment. Despite appearing to have very specific effects, primarily for severe spasticity in the upper limbs, this type of treatment can benefit a large number of patients who exhibit the previously mentioned symptoms. volve medication. In reference to essential diagnostic criteria: The criteria “restriction to passive stretching” has been obtained in comparison with the criteria “painful limit to full stretch range of motion” because PROM restriction may be caused by TrPs, perpetuated by spasticity. The criteria “taut band palpable” can only be used for superficial muscles, which is, in fact, a limitation. In reference to confirmatory diagnostic criteria: The first criterion [LTR] is explored only with the needle because snapping palpation may increase spasticity which obscures the observation of LTR. Although it hasn't been mentioned in any literature, the second criterion, or GIS, can serve as a treatment guide for spasticity. The authors' clinical experience indicates that following GIS, there is a period of relaxation during which more Stretching your muscles is OK. The authors believe it is unlikely that the patient in Case Study 59 GIS is expressing pain or discomfort because their observation is that the patient's facial expression remains unchanged. The patient exhibits a cryoexpression of pain in response to resting tooth stimuli.

CONCLUSION

Treatment with DDN reduced the patient's spastic potential (RTPM) during each session. upper body. It is challenging to distinguish between decreased spasticity and altered viscoelastic properties as the cause of decreased RTPM as measured by the MAS. Given that TrPs are treated, it's probable that improvements in MAS scores result more from modifications in the viscoelastic characteristics than from increases in spasticity.

REFERENCES

1. Lance JW. Pathophysiology of spasticity and clinical experience with baclofen. In: Feldman RG, Young RR, Koella WP, eds. Spasticity: disordered motor control. London: Year Book Medical Publishers 1980: 185- 204.

2. DiLorenzo L, Traballesi M, Morelli D, Pompa A, Brunelli S, Buzzi MG, Formisano R: Hemiparetic Shoulder Pain Syndrome treated with deep dry needling during early rehabilitation: a prospective, open-label, randomized investigation. J Musculoske Pain 12(2): 25- 34, 2004.

3. Travell JG, Simons DG: Travell & Simons’ Myofascial Pain & Dysfunction: The Trigger Point Manual 2nd Ed Vol. 1: Upper half of body. Lippincot Williams & Wilkins, 1999.

4. Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. J Electromyogr Kinesiol 2004;14(1):95-107

. 5. Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. J Appl Physiol 99: 1977-1984, 2005.

6. Gerwin RD, Dommerholt J, Shah JP. Current Pain and Headache Reports 2004, 8: 468-475.

7. Bohannon RW, Smith MB: Interrater reliability of modified Ashworth scale of muscle spasticity. Phys Ther 67: 206-207, 1987.

8. Hong CZ: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. Am J Phys Med Rehabil 73: 256-263, 1994.

9. Hufschmidt A, Mauritz K: Chronic transformation of muscle in spasticity: a peripheral contribution to increased tone. J Neurol Neurosurg Psychiatry 48: 676- 685, 1985.

10. Ibrahim IK, Berger W, Trippel M, Dietz V: Stretch-induced electromyographic activity and torque in spastic elbow muscles. Differential modulation of reflex activity in passive and active motor tasks. Brain 116: 971-989, 1993.

11. Pandyan AD, Johnson GR, Price CI, Curless RH, Barnes MP, Rodgers H: A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measures of spasticity. Clin Rehabil 13(5): 373-383, 1993.

12. Bakheit AMO, Maynard VA, Curnow J, Hudson N, Kodopala S: The relation between Ashworth scale scores and the excitability of the α motor neurones in patients with post-stroke muscle spasticity. J Neurol Neurosurg Psychiatry 74(5): 646-648, 2003.

13. Pandyan AD, Price CI, Barnes MP, Johnson GR: A biomechanical investigation into the validity of the modified Ashworth Scale as a measure of elbow spasticity. Clin Rehabil 17(3): 290-293, 2003.

14. Pandyan AD, Price CI, Rodgers H, Barnes MP, Johnson GR: Biomechanical examination of a commonly used measure of spasticity. Clin Biomech 16(10): 859-865, 2001.

15. Goldspink G, Williams S: Muscle fibre and connective tissue changes associated with use and disuse. Key Issues in Neurological Physiotherapy. Edited by L Ada, C Canning. Butterworth & Heinemann, Oxford, 1990, pp. 12-78.

16. Fresno MJ, Mediavilla P, Mayoral O: Dry needling of miofascial trigger points for hypertonia spastica in incomplete spinal cord injuries. J Musculoske Pain 12(Suppl 9), 2004.

17. Hong CZ: New trends in myofascial pain syndrome. Zhonghua Yi Xue Za Zhi 65(11): 501-512, 2002. 18. Fink M, Rollnik JD, Bijak M, Borstadt C, Dauper J, Guergueltcheva V, Dengler R, Karst M: Needle acupuncture in chronic poststroke leg spasticity. Arch Phys Med Rehabil 85(4): 667-672, 2004.

19. Moon SK, Whang YK, Park SU, Ko CN, Kim YS, Bae HS, Cho KH: Antispastic effect of electroacupuncture and moxibustion in stroke patients. Am J