CASE STUDY

A Case Study Looking at the Effectiveness of Deep Dry Needling for the Management of Hypertonia

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ABSTRACT. Backgrounds: The patient is a four-year-old child with spastic tetraparesia.

Findings: A decrease in spasticity was observed in all the muscles being treated with deep dry needling, measured with the Modified Ashworth Scale [MAS]. There was also a gain in passive range of movement in the thumb.

Conclusions: Treatment with deep dry needling decreased resistance to passive movement. It is difficult to determine whether decreased resistance to passive movement measured with the MAS is due to changes in viscoelastic properties or to decreased spasticity. Since we treat trigger points, it is possible that improvement in MAS scores could be more due to changes in the viscoelastic properties than in spasticity. doi:10.1300/J094v15n02_09

KEYWORDS. Muscle spasticity, muscle hypertonia, myofascial pain syndromes, trigger point, dry needling, Modified Ashworth Scale

INTRODUCTION

The resistance felt when moving a limb passively, or the resistance to passive movement [RTPM], is called hypertonia. Hypertonia in patients with upper motor neurone [UMN] lesions results from a combination of spasticity, thixotropy, and changes in the viscoelastic properties of muscle, which may ultimately lead to the development of fixed muscle contractures.

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Spasticity is defined as "a velocity dependent increase in the tonic stretch reflex with exaggerated tendon reflexes, resulting from the hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome" (1). It is now accepted that the exaggerated stretch reflex in a muscle is only partly responsible for hypertonia and that other positive features of the UMN syndrome and biomechanical changes contribute significantly to RTPM.

Pediatric physiotherapists who work with profoundly impaired children often realize that one of the most important difficulties for parents managing their children [dressing, bathing, etc.] is the increase of RTPM.

The current methods of treatment for muscle spasticity include systemic antispascity drugs such as baclofen, dantrolene, tizanidine, diazepam, chlorazepate dipotassium, clonazepam, or clonidine, which are nonselective in their action and may cause functional loss. Paradoxically, in some patients, some of these drugs reduce force in the normal muscles without having an effect on muscle spasticity. Furthermore, the value of the oral antispasticity drugs diminishes with prolonged use. Tolerance develops after a few months of treatment, and incremental increases in dosage are often required to mantain the initial clinical response. The high doses required often increase the incidence and severity of these drugs' adverse effects. An alternative strategy in the management of muscle spasticity is chemical neurolysis with alcohol such as phenol. However, nerve blocks and motor point injections in the upper limbs often cause skin sensory loss and may cause dysesthesic pain or causalgia, which can be persistent. Additionally, their effect often diminishes with repeated treatment. In recent years botulinum toxin type A [BTX-A] has been shown to be an effective antispasticity agent. However it can also have minor adverse effects such as skin rashes, flu-like symptoms, and weakness of the injected muscles.

Focal injection of BTX-A has been demonstrated to be the elective treatment for spasticity although the review of the current evidence suggests the lack of general consensus amongst clinicians about the dose, site of injection, injection technique, etc. The BTX-A inhibits the release of acetylcholine into the synaptic cleft. It also seems to have a remote effect, which could be explained by indirect central effect.

For about 30 years, dry needling has been used as a pain-relieving procedure. It has also proved its efficacy in the treatment of the hemiparetic shoulder pain syndrome (2). In this study, the effectiveness and efficiency of deep dry needling [DDN] of trigger points [TrPs] was investigated based on the widely reported success of DDN on neuropathic pain. Apart from pain, TrPs have been associated with a wide variety of signs and symptoms, such as tingling, weakness, resistance to passive stretching, muscle shortening, and autonomic dysfunction (3).

When comparing BTX-A treatment with DDN, we could state that the effect of BTX-A is produced in the same place as DDN, the motor endplate zone. However, the way they act is different; while BTX-A acts in a chemical way, DDN acts in a mechanical way.

The hypothetic action mechanism of DDN in TrP treatment is the mechanical disruption of dysfunctional motor endplates in which, according to the integrated hypothesis described by David Simons about etiopathogeny of TrPs (3,4), there are contraction knots [active loci] which lead to palpable findings of TrPs and taut bands.

The BTX-A has also proven to be effective in the treatment of TrPs, which has been used to support the integrated hypothesis. According to this hypothesis, TrPs are located in dysfunctional motor endplates in which excessive acetylcoline release occurs [neurotransmision inhibition provoked by BTX-A would solve part of the problem of TrPs as it acts on its initial cause]. From this point of view, DDN and BTX-A would act in the same anatomical structure, although by different mechanism means. Recent pioneering research (5) has proven how twitch obtaining DDN produces a lavage of sensitizing substances whose presence could promote the persistence of motor endplate dysfunction (6).

In the case report that we are presenting, it is examined whether DDN can also have an effect on resistance to passive muscle stretch in a patient with hypertonia.

CASE DESCRIPTION

The patient is a four-year-old child. He was born in the 38th week of pregnancy by caesarean section.

Medical diagnosis was severe hypoxic-ischemic encephalopathy caused by perinatal fetal distress which appears clinically as a spastic tetraparesia with axial hypotonia, with severe impairment of the right upper limb.

The child has central hypovision and severe ocular motricity impairment. He depends on a caregiver for all his activities of daily living. His only means of communication is by smiling or crying to express happiness or pain.

ASSESSMENT

Passive Range of Movement

Passive range of movement [PROM] was assessed in the elbow, wrist, and fingers joints. Although the upper limb is fixed in a position of 30º shoulder abduction, fully flexed elbow, and 70º of wrist flexion with a fisted hand, it is possible to attain the full PROM for all joints except the thumb, if the stretch is performed slowly. For this reason, PROM was only assessed in the thumb. With the hand in the patient's resting position the following positions were used: the thumb fully flexed and opposed, one-quarter open, one-half open, three-quarters open, and fully extended with passive muscle stretch. On initial assessment, the patient's thumb could only be passively stretched to one-half open.

The hand is closed in the resting position and it can be moved to one-half open with passive muscle stretch.

Spasticity and Resistance to Passive Movement

Spasticity was assessed with the Modified Ashworth Scale [MAS] (7) before and after the treatment. The patient was in the supine position, with head in middle line to prevent the tonic-asimetric reflex possibly causing increased spasticity.

Spasticity assessment shows a grade 3 in elbow, wrist, and finger flexors muscles, and in thenar muscles.

INTERVENTION

Objectives of the intervention set by both parents and the physiotherapist were to diminish spasticity or RTPM in order to improve the parents' management of the child.

Muscles treated were the thenar muscles [opponens pollicis], the wrist flexors [flexor carpis radialis, flexores digitorum superficialis, and profundus], and the elbow flexors [biceps brachii and brachialis].

Intervention consisted of nine sessions for the thenar muscles. From the fifth to the ninth session, elbow and wrist flexor muscles were also treated. Intervention was performed twice a week for the first four sessions [thenar muscles] and once a week for the remaining five sessions [all muscles].

For diagnosis of the TrPs the following criteria were used:

Essential Criteria

1. Restriction to passive stretching (3)
2. Taut band palpable (3) in affected muscles
3. Palpable nodule in a taut band

Confirmatory Criteria

1. Visual or tactile identification of local twitch response [LTR]. This finding is probably the most specific single clinical test of a TrP (8).
2. Global increase of a spastic response [GIS] in the axial muscles. This criteria has not been published, but it has been established through clinical experience. The presence of GIS response may or may not be associated with LTR. This response did not correspond with any sign of pain or discomfort in the patient, and the GIS was immediately followed by a substantial decrease in muscle resistance of muscles treated for a few seconds.

Muscles were positioned in a sub-maximal stretch position, where a significant increase of resistance is felt. As the treatment works, the therapist applies a muscle stretch until a 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 or GIS can be felt.
2. Other manipulation of the needle [twisting].

OUTCOMES

The primary outcome measure was the degree of RTPM of the target muscle group which was assessed using the MAS of spasticity. Video recording was used to allow observational analysis of both parameters.

A clinically significant improvement in spasticity for all muscles treated was reported. See Tables 1, 2, and 3.

An improvement in the hand opening in the resting position and with passive muscle stretch was reported. When treatment started, the hand was fisted and the thumb could be passively moved to a half opened position. After nine treatments, the hand was in a one-quarter open position and it could be fully opened with passive muscle stretch.

Although it is a very subjetive measurement, parents reported that they experienced fewer difficulties in handling the child and that they have also observed a decrease in RTPM in the contralateral limb. Nevertheless, this last point cannot be supported by the MAS measurements, which showed no changes in left upper limb RTPM. In this case report, the last treatment was just before the Christmas holidays; a new assessment was performed after this vacation period. This showed that results had been mantained. After the Christmas holidays we did not continue with the treatment because the child started a new medical regime with diazepam that could interfere with the outcome measures.

TABLE 1. Improvements in Spasticity for Opponens Pollicis

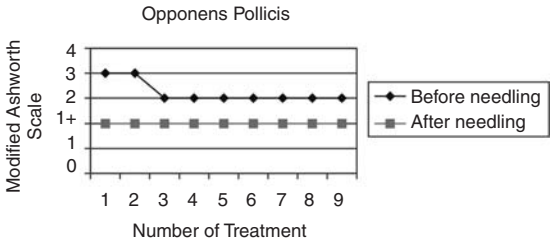


TABLE 2. Improvement in Spasticity for Wrist and Finger Flexor Muscles

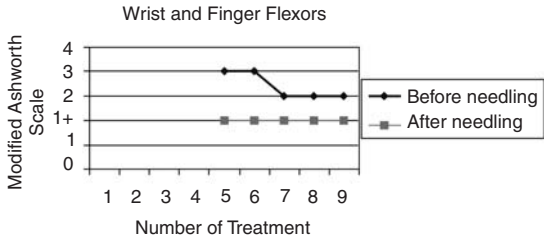
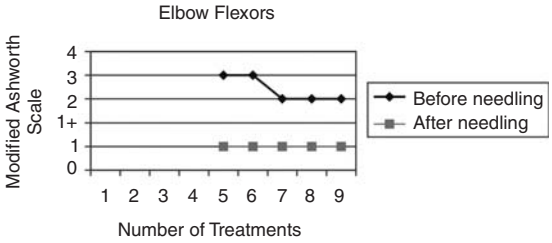


TABLE 3. Improvement in Spasticity for Elbow Flexor Muscles



DISCUSSION

As stated in the introduction, RTPM is a complex measure that will be influenced by many factors, only one of which could be spasticity. The MAS has important limitations and does not reliably distinguish between the different components of hypertonia. Another limitation of the MAS is that the test conditions have not been standardized. For example, while some clinicians assess the muscle tone from the resting state without previous muscle stretch,

others (9) have recommended flexion and extension of the limb a few times immediately before the actual measurement is taken. This lack of standardization may introduce measurement error because the stretch reflex excitability in the resting state may be different from that of the activated muscle (10). Nevertheless the MAS is probably the most widely used test for the measurement of muscle spasticity in research and clinical practice, and it has been demonstrated to be moderately reliable for classifying the RTPM at the elbow and the wrist flexors (11).

Some authors state that the MAS measures resistance to passive muscle stretch [hypertonia] rather than spasticity (12-14).

There are factors that can confound the MAS. Evidence from the literature suggests that the increase in RTPM could have resulted from decreased soft tissue compliance associated with reduced use (13). The RTPM is influenced by the immediate past history of movement. This would suggest that the increase in RTPM observed in the impaired arm might have been predominantly associated with changes in the viscoelastic properties of the soft tissues and not spasticity (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 patients 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 in different children treated in the school [non-published data] and in adult patients with incomplete spinal cord injury (16). In these clinical cases, despite not having scientific evidence, it was observed that DDN had more lasting effects in upper limbs than in lower limbs, possibly caused by the weight bearing factor that could be a perpetuating factor of spasticity. In the treatment of children, results were better with severe spasticity and restriction of PROM than with mild impairments. Apart from these factors, some difficulty was experienced in the treatment of children capable of recognizing the "threatening presence" of a needle. According to all these data obtained from the clinical practice, it was decided that the most suitable patient had to be a child with severe spasticity or restricted PROM, and that he/she should have a cognitive impairment and/or a visual loss that would prevent him/her from realizing that he was going to be treated with needles.

Although this kind of treatment seems to have very restricted effects [mainly for upper limb severe spasticity], it can help many patients with the characteristics previously described.

There is a lack of published knowledge in this field. The effect of TrP injection (17), acupuncture needling (18), and electroacupuncture and moxibustion (19) for treating spasticity have been reported, but not the use of DDN in TrP for decreasing spasticity/resistance to passive muscle stretching. The only clinical evidence of effectiveness of DDN for spasticity treatment was shown in patients with incomplete spinal cord injury (16).

Although the efficacy of DDN has yet to be demonstrated for the treatment of spasticity, an advantage of this technique is that it does not involve medication.

In reference to esential 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.

The second criterion [GIS] has not been documented in the literature, but can be used as a guide for the treatment of spasticity. According to the authors' clinical experience, there is a relaxation period after GIS during which more muscle stretch is allowed. Some may interpret

GIS as the patient expressing pain or discomfort, but in the authors' experience, this is unlikely since the patient's facial expression does not change. The patient has been observed to cry to express pain in response to other stimuli.

CONCLUSIONS

The treatment with DDN decreased RTPM in the treatment session and throughout the sessions in spastic muscles located in our patient's upper limb. It is difficult to determine whether decreased RTPM measured with the MAS is due to changes in viscoelastic properties or to decreased spasticity. Since we treat TrPs, it is possible that improvement in MAS scores could be more due to changes in the viscoelastic properties than in spasticity.

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