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Case Presentation

Managing Upper Extremity Clonus With Intramuscular Botulinum Toxin—A Injections in a Patient Poststroke

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Abstract

Spasticity and clonus are common clinical signs of upper motor neuron lesions poststroke. Intramuscular botulinum toxin—A injections have been shown to reduce spasticity and to improve quality of life. Upper extremity clonus can have a significant impact upon activities of daily living and can pose challenges to comfort, self-care, hygiene, and appearance. Although less common than in the ankle, it is important to understand how upper extremity clonus management may be beneficial to patients presenting with this finding. The majority of previous reports have focused on management of clonus in the lower extremity, but very few have addressed the management of upper extremity clonus. We present a case of poststroke upper extremity clonus with marked improvements following intramuscular botulinum toxin—A injections.

Introduction

Spasticity and clonus both represent positive signs of the upper motor neuron syndrome poststroke. Stretch reflex hyperexcitability is considered to be 1 of the mechanisms underlying spasticity and clonus. Both spasticity and clonus can result in movement difficulties and can impair function [1], ranging from gait dysfunction due to ankle clonus, to poor arm function due to upper extremity clonus. There are several treatment options available including medical management using baclofen [2], as well as chemodenervation using botulinum toxin—A (BoNTA) intramuscular injections to manage spasticity and clonus.

Spasticity management with BoNTA has been demonstrated as an important treatment modality to reduce muscle tone and to improve quality of life poststroke [3,4]. The use of BoNTA injections for management of clonus has also been reported to be an effective treatment strategy [3-5]. Previous studies show the effectiveness of BoNTA injections for treatment of ankle clonus [5,6] or finger clonus in the upper extremity (UE) [7]. Clonus is more readily observable in the ankle, but UE clonus is not as readily observed by clinicians [2.8]. Clinicians may observe UE clonus in practice, but very few reports were found in the literature that discussed UE clonus management. Although UE clonus may be less common than ankle clonus, it is important for clinicians to be aware of the manifestation of clonus in the upper extremities and to understand the various treatment options available.

There has recently been a reported case of wrist flexor clonus management using baclofen in a patient poststroke [2]. However, a thorough review of the literature did not reveal any reports of UE clonus affecting the elbow flexors and forearm pronators. Limited information exists in the literature regarding management of clonus in multiple UE muscles. As a result, there is little evidence about beneficial therapeutic approaches to manage UE clonus. Here, we present a unique case of UE spasticity and clonus management in multiple UE muscles in a patient who exhibited marked improvements after treatment with intramuscular BoNTA injections.

Case Presentation

This case presentation describes a 41-year-old, right-hand—dominant man who sustained a right cerebral infarct immediately after a surgical procedure to repair

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a right inguinal hernia. It was determined that the patient experienced a right-sided carotid dissection and ischemia of the right middle cerebral artery (MCA) vascular territory, resulting in left-sided hemiparesis. The patient presented to our spasticity management clinic 3 years after the stroke for management of spasticity. The patient had received 1 set of BoNTA injections in a different facility 1 year before visiting our clinic and reported no improvement. Details of other systemic medications or BoNTA dosage were not available.

The patient reported greater dysfunction in the left UE compared to the lower extremity and muscle stiffness in all muscle groups. Stiffness was also reported to be worse in the morning, upon waking, and was also exacerbated by cold weather. Other than difficulty with dressing and undressing, the patient was independent in the rest of the self-care activities. Clinical examination revealed spasticity in the left UE graded using the Modified Ashworth Scale (MAS; tested in supine position) and limited passive range of motion in the wrist joint (extension of 45° past neutral), but no contractures or range of motion limitations anywhere else in the upper extremity. MAS scores and clonus frequency are reported in Table 1. The patient had some active movement in the left UE, but experienced severe clonus in his left pronators (triggered by active supination) when he actively tried to bring his hand to his mouth. However, clonus in other muscles was not triggered during this same motion.

Upon evaluation, the patient was determined to be a good candidate for focal BoNTA injections for management of spasticity and clonus, and received a total of 350 units of BoNTA (onabotulinumtoxinA), using electrical stimulation and electromyography guidance, in the affected muscles (Table 2). The patient subsequently was provided instructions on splinting (wrist-hand orthosis) by the occupational therapist and was instructed on a self-directed daily home exercise program involving stretching exercise of involved muscles.

Table 1
Modified Ashworth Scale (MAS) and clonus frequency pre- and post-BoNTA injections

Muscle Group	Pre-BoNTA	Post-BoNTA
MAS (0-4)		
Finger flexors	3	1^+
Wrist flexors	3	1^+
Elbow flexors	2-3	1^+
Elbow extensors	1^+-2	1^+
Pronators	3	1^+
Lumbricals	0	0
Pectoralis major	0	0
Clonus frequency (Hz)		
Finger flexors	7	0
Pronators	6	0
Wrist flexors	6-7	4-5

Table 2
BoNTA units administered*

Muscle Group	No. of Sites	Total Units Administered
Brachioradialis	2	50
Brachialis	2	50
Pronator teres	2	50
Flexor carpi ulnaris	2	50
Flexor carpi radialis	2	50
Flexor digitorum profundus	4	40
Flexor digitorum superficialis	4	60

* BoNTA administered as a 2:1 dilution (2 cc of preservative-free saline solution:100 units of onabotulinumtoxinA).

BoNTA significantly improved range of motion when measured at 3 weeks postinjection (Figure 1). From top to bottom, Figure 1 shows the end position of the maximum possible passive elbow extension, supination, and wrist and finger extension pre- and post-BoNTA. The frequency and duration of the wrist and finger flexor clonus (assessed by slowing down the video and using the time stamp to calculate the duration and frequency of clonus) decreased significantly after BoNTA (see accompanying video for the level of clonus elicited before and 3-weeks after BoNTA injections). The spasticity score decreased to grade 1^+ on MAS in the majority of the muscles (Table 1), and wrist extension improved to 70° past the neutral. During his 3-week follow-up, the patient also self-reported improvements in his symptoms.

Discussion

Clonus is typically elicited by a clinician, with a sustained muscle stretch delivered using a constant stretch force, resulting in repetitive stretch reflex response cycles. Muscle stretch triggers both the static and dynamic fibers in the muscle spindle. Clonus is thought to occur as a result of the increased sensitivity and facilitation (and lack of inhibition) of the "dynamic" component of the stretch reflex [9]. The dynamic component of the stretch reflex is very short, lasting less than 1 second before it dies out [9]. As a result, the sustained muscle stretch while testing for clonus can repeatedly trigger new cycles of reflex muscle contraction-relaxation (eg, in finger, wrist, and elbow flexors) for an extended duration.

Previous studies have shown improvement in finger clonus using BoNTA treatment and improvement in wrist clonus using baclofen [2]. In this case presentation, we report improvement in multiple UE muscle clonus using BoNTA injections. BoNTA induces chemodenervation by inhibiting acetylcholine release at the neuromuscular junction in extrafusal fibers in the muscles as well as the intrafusal muscle fibers in the muscle spindles [8]. Physiologically, this means that BoNTA injections have the effect of decreasing the muscle activity by