Evaluation of the Spasticity after Botulinum Toxin Injection Using Paired-Pulse Transcranial Magnetic Stimulation

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To the Editor: Spasticity is a common movement disorder after stroke. In 1 to 6 weeks after the stroke, approximately 40% of the stroke patients develop the spasticity on the affected side. Here, we report a stroke patient with spasticity using paired-pulse transcranial magnetic stimulation to explore the central mechanism of the release of the spasticity after botulinum toxin injection.

A 44-year-old male patient who was diagnosed with hypertensive hemorrhage of basal ganglia accompanying poor activity of the left limbs for 2 years came for seeking management of spasticity in his left upper limb. After the evacuation of intracranial hematoma, he received regular rehabilitation therapy. We found that he had a better recovery in his proximal limb. However, his wrist and hand joint manifested poor recovery and developed spasticity. Botulinum toxin type A (BTA, Botox, Allergan) was injected into the flexor muscles of the wrist and fingers including the flexor carpi radialis 50 U, flexor digitorum profundus 50 U, flexor digitorum superficialis 70 U, and flexor pollicis longus muscle 20 U (190 units in total) under the double guidance of electromyography and electrical stimulation. Every target muscle distributed the spastic potential when localization. After the injections, stretching and extensive physical and occupational therapy were performed persistently. Two weeks after the injection, the spasticity in his left limb reduced from modified Ashworth scale (MAS) Grade 1+ to 1 in his elbow and wrist and Grade 3 to 1 in his fingers. The condition of the modified Ashworth scale was persisted for 12 weeks [Figure 1, we use 1.5 to displace MAS Grade 1+ to easily demonstrate on the line graph].

Transcranial magnetic stimulation (TMS) studied before and after the treatment was conducted with OSF-priming TMS. Neither before nor after the injection could we induce the motor-evoked potential (MEP) from the patient's right hemisphere (affected hemisphere, AH). After the Botox injection, we found that the rest motor threshold of the unaffected hemisphere (UH) increased. The progress was 42% maximal stimulator output (MSO) for before, 48% MSO for 1 week after, 52% MSO for 2 weeks after, and 51% MSO for 12 weeks after that suggesting the decline of the excitability of the UH.[1] The short intracortical inhibition of the UH was drastically increased 2 weeks after the injection. The rate of rising became slower 2 weeks after the treatment in which 118.57% MEP for before, 88.19% MEP for 1 week after, 73.08% MEP for 2 weeks after, and 68.18% MEP for 12 weeks after indicating the inhibition mediated by the gamma-aminobutyric acid (GABA)-α receptors increase [Figure 1].

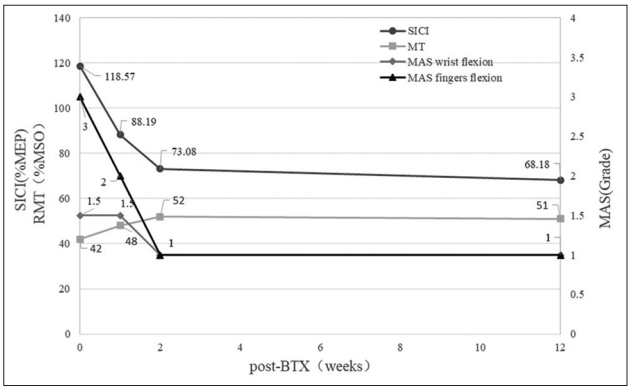

Figure 1: Changes in spasticity as measured using the modified Ashworth scale, rest motor threshold, and short-interval intracortical inhibition over the unaffected hemisphere at multiple time points after the injection of botulinum toxin.

The intramuscular Botox injection not only causes the modification of the neuromuscular junctions at the injection site but also affects the central through entering the central nervous system by retrograde transportation. Basaran *et al*.[2] used somatosensory-evoked potential to evaluate the cortical effect of the Botox injection in the stroke patients with the spastic upper limb. They founded that the N20 latency decreased significantly and the amplitude of N20-P25 increased significantly, suggesting the increasing input of the proprioception and the remodeling of the cortical. The functional magnetic resonance imaging studies

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also provide the evidence that central cortical patterns can be modified using Botox treatment. Our previous research found that, with the improvement of the spasticity after Botox injection, the excitability of the primary motor cortex (M1) in AH and the cortex around the lesion was increased while the excitability of the corresponding area in UH was decreased means that the lateralization improvement appeared.[3]

It is a compensation mechanism which inhibition of the UH decreases after stroke. When the AH of the stroke patients is severely injured, the excitability of the UH will increase. Furthermore, some of them will present ipsilateral dominant. The change of the cortical excitability is the central mechanism of the spasticity.[4] Using repetitive TMS for upregulating the inhibition effect of the UH in the patients with spasticity showed the release of the spasticity. In this case study, the TMS results indicate that the inhibition mediated by GABA-α receptors increased continually after the Botox injection and were more drastic in the first 2 weeks, coinciding with the previous studies.[5] The Botox enters the AH by retrograde transportation, and the UH will correspondingly present the increasing of the inhibition which is the result of upregulating of the GABA-α receptors.

In conclusion, the change of the neural excitability after stroke may cause the spasticity which can be released through modulating the activity of the GABA-α receptors in the cerebral cortex by the peripheral injection of the Botox.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initial will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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