Botulinum Toxin Use as an Adjunctive Modality in a Patient with Multiple Flexor Tendon Ruptures

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Received: 26 October 2007 / Accepted: 22 February 2008 / Published online: 14 March 2008
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Abstract Botulinum toxin A has been described in treatment protocols for several disease processes, from primary axillary hyperhidrosis to esophageal dysfunction. It is best known for its use in plastic and dermatological practices. Botulinum toxin has a straightforward mechanism of action. The toxin inhibits acetylcholine release at the neuromuscular junction causing a chemical denervation, which ceases contractions of the muscle. With its minimal side effect profile, botulinum toxin should be considered when muscle spasm is a detriment. This case involves an injury to the hand of a patient with a history of intermittent diffuse muscle spasms. Subsequently, due to the patient's previous upper extremity muscle spasms, repeated flexor tendon repair ruptures of the index finger occurred until botulinum toxin was administered to the offending muscle. The patient has not required any additional surgical interventions for the repaired tendon and is now actively flexing all digits. This case report demonstrates how botulinum toxin can be used in a clinical scenario when decreased muscle activity is desired to promote tendon healing.

Keywords Flexor tendon rupture · Botulinum toxin A · Botox · Teno fix · Flexor tendon repair

Case Report

A 44-year-old right-hand-dominant male presented to the emergency room with injuries sustained to his left hand

owing to a table saw laceration across his palm. His exam and operative findings noted the following: fractures of the index and small finger proximal phalanges, zone III flexor digitorum superficialis and profundus tendon lacerations to the index, zone III flexor pollicis longus tendon laceration, and zone III flexor tendon injuries to the small finger. Concurrent injuries also included digital nerve lacerations to the thumb, index, and small finger. There were vascular lacerations of the digital artery to the small and index fingers.

His past medical history is significant due to an ongoing neurological dysfunction from a prior traumatic injury. This prior injury has left the patient with intermittent diffuse muscle spasms. His medications for this condition include neurontin 600 mg four times a day, baclofen 20 mg three times a day, morphine 30 mg three times a day, and celebrex 200 mg twice a day. Despite this regiment of medications, he describes ongoing symptoms of muscle spasm including his injured extremity. His past surgical history is also significant for a prior left middle finger amputation through the level of the middle phalanx.

Initial emergent operative management incorporated irrigation and debridement along with repairs of the tendon lacerations to the thumb, index, and small fingers. Tendon repairs were carried out using a four-strand core suture technique augmented with a 6–0 prolene epitendinous suture. The bony fractures were stabilized with 0.045 Kirshner wires. Repairs to the lacerated digital nerves and arteries were also performed. Finally, a protective dorsal block plaster splint was applied.

The patient was examined postoperatively in a routine fashion. An early active range-of-motion exercise program (Indiana protocol) was prescribed with continued use of a dorsal block. This was performed under the care and guidance of a senior distinguished hand therapist. Unfortunately, while at rest in bed, the patient reported an uncontrollable muscle spasm with acute complaints of pain

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and loss of active flexion ability and finger cascade. The poor function and physical examination pointed to a tendon rupture. The patient returned to the operating room, and the intraoperative findings noted rupture of the previously repaired index finger superficialis and profundus tendons. The flexor digitorum profundus tendon was repaired again using the four-strand core technique with a 3–0 ethibond suture. The repair was augmented with a 6–0 prolene epitendinous repair. The flexor digitorum superficialis tendon tissue of the index finger was of poor quality and was not repaired in an attempt to minimize potential adhesions and scar formation. The small finger superficialis and profundus tendons along with the flexor pollicis longus were found to be intact and required no surgical intervention.

Again, this repair failed on postoperative day 5 secondary to further night muscle spasms in the injured upper extremity despite splint usage. His treatment options were discussed, and he elected to proceed with another surgery. He subsequently underwent a revision repair of his flexor digitorum profundus tendon of his index finger. The tendon was repaired with a four-strand core technique using a 4–0 fiberwire and a 3–0 ethibond. The repair was again augmented with a 6–0 prolene epitendinous repair.

The patient reinitiated hand therapy (Indiana protocol) postoperatively but, again, felt a sharp contracture in his forearm on postoperative day 6 with subsequent loss of active flexion in his left index. The patient stated that these spasms were unrelated to his hand therapy and usually were more uncontrollable at night during sleep. Even though he was complying with his dorsal block splint, it was suspected that the nightly muscle spasms were causing the tendon rupture due to the patient's inability to counteract their force while sleeping.

With uncontrolled upper extremity muscle contractions continuing from his underlying neurologic condition, a repeat tendon repair seemed likely to have a similar dismal outcome. Alternatives to repeated tendon repair surgery were discussed. With the assumption that sufficient tendon length would permit, the patient opted for tendon repair. Again, the index finger flexor digitorum profundus tendon rupture was addressed surgically. A TenoFix tendon repair system was employed. Intraoperative observations displayed sufficient length and strength of the tendon repair with passive range of motion of the digit. In addition, botulinum toxin was injected into the muscle belly of the flexor digitorum profundus prior to the awakening of the patient. One hundred units of botulinum toxin were diluted with 2.5 ml of 0.9% sodium chloride solution to obtain a final concentration of 4 U per 0.1 ml. The 2.5-ml volume was equally placed in four separate locations along the radial aspect of the flexor digitorum profundus, in attempt to paralyze the muscle associated with the index finger.

A dorsal block splint was applied intraoperatively and maintained in the postoperative period. Hand therapy

(Duran protocol) was initiated on postoperative day 1 with no rerupture of the tendon. The patient had not reported the forceful muscle spasms that were previously encountered and had been able to actively flex his digits. No additional botulinum injections were required. Over the next 2 to 3 months, the patient recovered full function without rupturing the tendon. The achieved range of motion of the index finger is 80% of total active motion. The patient continues to possess a minimal proximal interphalangeal flexion contracture of 10°.

Discussion and Literature Review

The goal of flexor tendon repair is to obtain a repair with enough strength to allow postoperative therapy to initiate and healing to proceed. This therapy decreases flexor tendon adhesions and allows proper gliding patterns to redevelop. Traditionally, a four-strand repair has provided enough strength to accomplish this goal. However, there is a population of patients in which the repair may be jeopardized. This may be especially evident in children, noncompliant patients, and patients with muscle spasms. Most literature concerning the use of botulinum toxin in the management of muscle spasms has been associated with cerebral palsy and hemifacial spasm [2–4, 7]. However, there appears to be a patient subset in which adjunctive modalities, such as botulinum toxin, are beneficial for the treatment of their upper extremity condition [1, 5, 6].

This case report illustrates a useful application of botulinum toxin injection in the process of restoring function to a patient. The effects from this botulinum injection do not need to be permanent or repeated. The tendon, once healed (average of 6 weeks), will be able to withstand the added load from this patient's muscle spasms without additional injections. Besides one study in young children, little has been described in the literature concerning the use of botulinum for flexor tendon repairs [6]. The use of botulinum toxin A in the proper patient subset can be of extreme value in decreasing undesired tendon strain to promote improved patient healing.

The combination of the Teno Fix device and botulinum toxin A has allowed our patient to progress in his therapy but, more importantly, not to rerupture.

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