Botox and Thumb MCP Radial Collateral Ligament Reconstruction

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Abstract

Keywords

- radial collateral ligament
- reconstruction
- botulinum toxin
- thumb
- adductor pollicis

Background We report the use of botulinum toxin to aid in the treatment of chronic radial collateral ligament insufficiency of the thumb.

Case Description Treatment included autograft tendon reconstruction and cast immobilization. Six weeks postoperatively, prior to hand therapy, the patient underwent an ultrasound-guided botulinum neurotoxin A injection into the adductor pollicis muscle to negate its deforming forces on the reconstruction. The patient made an excellent recovery and 1 year postoperatively was pain free and had a stable radial collateral ligament (RCL) upon examination.

Literature Review The outcomes of RCL repair and reconstruction are unpredictable, in part, due to the strong opposing forces of the adductor pollicis.

Clinical Relevance The use of Botox injection may enhance the outcomes of RCL repair or reconstruction by neutralizing the deforming forces of the adductor muscle.

Radial collateral ligament (RCL) injuries of the thumb are, on an average, 50% less common compared with those of the ulnar collateral ligament (UCL).^{1–3} Partial tears (grades I and II) are typically treated nonoperatively, whereas the management for complete tears (grade III),⁴ particularly those that are chronic, remains controversial.¹ Several surgical reconstructive techniques have been described, including direct RCL repair, RCL soft tissue advancement, RCL soft tissue overlap imbrication, RCL advancement with abductor pollicis brevis advancement, and RCL tendon graft reconstruction.^{1–3,5–7} Outcomes are varied and tend to result in decreased range of motion and instability compared with those of the adjacent UCL. These suboptimal outcomes may be, in part, due to the strong deviating pull of the adductor pollicis (ADP) on the ulnar side of the metacarpophalangeal (MCP) joint.⁵ Botulinum neurotoxin A (BoNT-A) inhibits the release of acetylcholine from the presynaptic nerve terminal, rendering a temporary paralytic effect on skeletal muscle. Ma and colleagues⁸ injected BoNT-A into the gastrocnemius muscle of Sprague Dawley rats to “protect” their Achilles tendon repair by reducing the deforming force. Similarly,

BoNT-A injections have been used to protect flexor tendons of the hand⁹ and extensor tendons of the wrist. To our knowledge, no such use has been described after RCL reconstruction of the thumb. We present a case report of injecting BoNT-A into the ADP postoperatively as an adjunctive treatment to help protect the reconstruction and optimize functional outcome after RCL reconstruction of the thumb.

Case Report

A 45-year-old, right hand dominant male patient presented for the evaluation of symptomatic right thumb chronic RCL insufficiency (pain and instability) that was refractory to nonoperative treatment. On examination, there was focal tenderness over the radial side of the thumb MCP joint with grade III instability (laxity without an end point). Thumb MCP joint range of motion was 25 to 50 degrees. Interphalangeal (IP) joint motion was – 5 to 45 degrees. His UCL was stable. Plain radiographs demonstrated maintenance of the joint space with some deformity of the metacarpal head (► **Fig. 1**). The patient

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Fig. 1 Preoperative anteroposterior and lateral radiographs of the right first metacarpophalangeal joint.

was keen to maintain motion and did not want an arthrodesis. Given this, a decision was made to proceed with RCL reconstruction using palmaris longus (PL) autograft.

Surgical Procedure

Under regional anesthesia, a chevron incision was fashioned overlying the radial aspect of the thumb MCP joint, protecting the dorsal sensory branch of the radial nerve. The extensor

Fig. 3 Postoperative anteroposterior and lateral radiographs of the right first metacarpophalangeal joint with some asymptomatic, degenerative changes.

Fig. 2 Intraoperative photograph demonstrating the radial collateral ligament reconstruction with palmaris longus autograft tendon.

pollicis brevis (EPB) and extensor pollicis longus (EPL) tendons were found to have dislocated in an ulnar direction. The ruptured RCL was fibrotic and irreparable, and there was some deformity of the metacarpal head with fibrillation of the articular cartilage. Given the fibrotic nature of the RCL, it was reconstructed using a PL autograft and biotenodesis screw stabilization (►Fig. 2). The MCP joint was temporarily pinned in 20 degrees of flexion using a 0.045-in Kirschner wire. After capsular closure, the EPB and EPL tendons were realigned over the central aspect of the MCP joint. The patient was placed in a thumb spica postoperative splint for 2 weeks and then into a thumb spica cast for an additional 4 weeks after which the Kirschner wire was removed.

Before starting physical therapy, the patient received a BoNT-A injection to protect the RCL reconstruction from the deforming force of the ADP on the MCP joint. After informed consent had been obtained, 25 units of botulinum toxin type A (BOTOX, Allergan, Irvine, CA) were injected into the ADP muscle under ultrasound guidance using a 27-gauge, 1–1.25-in needle.¹⁰ A removable thumb spica splint was fabricated and worn for an additional 6 weeks during which hand therapy was initiated emphasizing range of motion of the MCP and IP joints as well as strengthening of the abductor pollicis muscle.

The patient made an excellent recovery and 1 year postoperatively was pain free and had a stable RCL upon examination (►Fig. 3). His MCP and IP joint range of motions were 10 to 60 and 0 to 45 degrees, respectively. Compared with the contralateral side, the patient's grip strength, apposition pinch, and opposition pinch were 96, 93, and 100%, respectively. His grip strength, apposition pinch, and opposition pinch improved by 54, 108, and 75%, respectively, compared with his preoperative values. The Kapandji score was 10/10 bilaterally.¹¹

Discussion

RCL injuries of the thumb occur much less frequently than those involving the UCL.^{1–3} While grade I and II tears are often treated nonoperatively, grade III tears may be treated with surgery and include a myriad of surgical reconstructive techniques.^{1–3,5,6} Their outcomes are varied and difficult to compare due to a lack

of standardization between the studies;¹ however, most series have noted a decreased range of motion and instability to be relatively common after RCL reconstruction, especially when compared with that of the adjacent UCL.^{2,3,5,6}

Catalano and colleagues³ reviewed 10 patients with grade III chronic RCL tears who underwent PL autograft reconstruction at a mean of 6.8 months from the time of injury. Median MCP joint motion at the time of follow-up was 59% of the uninvolved side. Stress testing of the MCP joint showed 8 degrees of laxity in extension and 13 degrees of laxity in 30 degrees of flexion.

Coyle² reported on 38 patients with grade III RCL injuries, 34 of which were chronic. Reconstruction consisted of soft tissue advancement with bony reattachment. At follow-up, 70% had MCP joint opening within 5 degrees of the contralateral side with stressing. Those patients with chronic tears greater than 8 months post injury had increased MCP joint opening (mean: 25 degrees). Cooney et al⁶ suggested that surgical reconstruction of chronic RCL injuries led to “less than ideal results.”

The ADP is an intrinsic muscle that originates from the base of the second and third metacarpals and capitate. Its oblique and transverse heads insert on the ulnar sesamoid, extensor aponeurosis, and volar aspect of the proximal phalanx.¹² When the muscle contracts, a combined adduction-pronation-type force is exerted on the proximal phalanx that contributes to flexion of the MCP joint, tip pinch, and opposition of the thumb.^{1,5,12} The intact RCL exhibits a tension-band-like effect on the MCP joint, converting translational forces to compressive forces. Given the pull of the ADP, after a RCL injury, there is radial diastasis of the MCP joint and volar and ulnar subluxation of the proximal phalanx with simultaneous pronation.^{1,5,12} This loss of stability and translation of the proximal phalanx may lead to early articular deterioration and pain; therefore, repair or reconstruction is often recommended.

Unfortunately, the deforming force of the ADP may impair RCL healing after its reconstruction. To prevent such loss of stability, immobilization for 6 weeks or greater is often utilized.⁵ While this helps protect the reconstruction, it does not allow for range of motion, and there is certainly no additional protection once the splint/cast is removed.

BoNT-A, one of the seven neurotoxin subtypes produced by the bacterium, *Clostridium botulinum*, inhibits the release of acetylcholine from the presynaptic nerve terminal and prevents its activation. First recognized as a paralytic in 1817 by the German physician and poet, Justinus Kerner, it was not until 1989 when its use was approved for therapeutic application by the United States Food and Drug Administration.¹³ Given this, BoNT-A has been used to treat a variety of conditions, including strabismus, blepharospasm, spasmodic dysphonia, hemifacial spasm, vascular occlusion, pain, and spasmodic torticollis, and its applications and indications continue to evolve. Several clinical trials evaluating the effectiveness of BoNT-A in movement disorders and spasticity have suggested that it is both safe and effective.^{8,9,14} Nevertheless, some concerns remain including the potential for local weakness, lack of standardization of biological activity in different preparations, variations in injection methods, and cost-effec-

tiveness¹³ (BoNT-A has been used in children for spasticity in the hand, in the fingers for flexor tendon repair, after extensor tendon repair, and for Raynaud's phenomenon).^{9,15} It is reversible, temporary, and paralytic when injected into muscle, leading to peak effects by 2 weeks, clinically detectable weakness over the next 2 to 4 months, and nearly full return of muscle mass, electrophysiologic variables, and muscle force generation by 6 months.^{8,9}

In 2007, Ma and colleagues⁸ described a form of “bioprotection” for an Achilles tendon repair in Sprague Dawley rats by injecting BoNT-A into the gastrocnemius muscle. The tetanic force reduction was 5- to 10-fold lower than the force required for Achilles tendon repair rupture and lasted on average for 4 weeks. Overall, the authors noted that tendon rupture rates were three times lower in the BoNT-A group compared with the control group.

In the surgical case presented, we hypothesized that the pull of the ADP may impair the outcome by increasing the strain on the reconstruction. To mitigate this, we sought to inject the ADP with BoNT-A, thus allowing earlier “protected” motion and enhanced physical therapy. Six weeks postoperatively, we injected 25 units of BoNT-A into the ADP under ultrasound guidance. The outcomes at 1 year were excellent with a painless, stable thumb. The increase in stability, compared with previous studies, may, in part, be attributed to the lack of deforming force by the ADP muscle.

We agree with previous authors^{8,9} that bioprotection using a BoNT-A injection, as an adjunctive treatment, may provide benefits by protecting the reconstruction during cast change/removal and during unintended/excessive isometric muscle contractions of the ADP. Indeed, it may also decrease the period of immobilization, as active adduction of the MCP may be negated by the pull of the ADP, but one would need to guard against the adduction force generated by the extensor pollicis longus. Nevertheless, the protective benefits of BoNT-A may lead to a more stable reconstruction, thereby improving the overall success of the RCL reconstruction.

While this adjunctive treatment may be applicable to other surgeries in the hand, further research into the safety and effectiveness is needed.

Conflict of Interest

None.

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