SCIENTIFIC ARTICLE

Multiple Pulley Rupture Following Corticosteroid Injection for Trigger Digit: Case Report

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We report a case of pulley rupture following repeated local corticosteroid injections for trigger digit. The treatment involved exploration, tenolysis, and reconstruction using the palmaris longus tendon. *(J Hand Surg 2009;34A:1444–1448. © 2009 Published by Elsevier Inc. on behalf of the American Society for Surgery of the Hand.)*

Key words Corticosteroid, pulley rupture, trigger digit.

Trigger digit, or *stenosing tenosynovitis*, is a condition characterized by painful locking or snapping of a digit caused by mechanical impingement of the flexor tendon passing through a hypertrophic A1 pulley. Initial conservative management of trigger digits with various corticosteroid preparations is well described.[1–4] Side effects, including subcutaneous fat atrophy, pain, depigmentation of the skin, and transient elevation of urine and blood glucose levels in patients with diabetes, are generally mild and self-limiting.[5,6] We are aware of 2 previously reported cases of delayed flexor tendon rupture following corticosteroid injections for trigger digit[7,8] thought to be the result of intratendinous injection. One case of closed pulley rupture in the thumb following corticosteroid injection has been previously reported.[9] We report a case of multiple pulley rupture following corticosteroid injection for stenosing tenosynovitis of the middle digit.

CASE REPORT

A 42-year-old right-handed woman, a primary care physician, presented with pain and proximal interphalangeal (PIP) joint flexion contracture of the right middle digit. Twenty-one months earlier, the patient was diagnosed with stenosing tenosynovitis and received a local corticosteroid injection through a lateral approach at the proximal phalanx[10] (0.5 mL local anesthetic and 0.5 mL triamcinolone acetonide 40 mg/mL [Kenalog-40, Bristol-Meyers Squibb Co, Princeton, NJ]). Her symptoms of pain temporarily improved, but 4 months later, the patient returned with recurrent pain and a second local corticosteroid injection was administered, again using the lateral approach. During the ensuing 8 months, the patient had persistent pain and tenderness over the A2 pulley. The patient also developed pain at the A1 pulley, and a third injection (0.5 mL local anesthetic and 0.5 mL triamcinolone acetonide 40 mg/mL) was given into the palmar surface overlying the A1 pulley. Five months after the third injection, the patient presented with worsening pain and a PIP joint flexion contracture. Given the failure of conservative management of her trigger digit, the patient was taken to the operating room for trigger release. During surgery, the A1 pulley was released, and local tenolysis was performed. Further examination through the palmar incision revealed absence of the proximal edge of the A2 pulley; thus, an attempt at reconstruction of the A1 pulley was performed with the previously incised leaflets of the pulley. The distal A2 and A3 pulley were not exposed.

After several months of therapy, pain and range of motion failed to improve. At the time of presentation to our institution 7 months after surgery, the patient had persistent pain, bowstringing, and a severe PIP joint flexion contracture. The patient was in good health and had no history of inflammatory arthritis, diabetes, collagen vascular disease, or systemic steroid use. Physical examination of the middle digit revealed a 55° flexion contracture of the PIP joint (Fig. 1A). The distal inter-

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Received for publication December 5, 2008; accepted in revised form April 24, 2009.

No benefits in any form have been received or will be received related directly or indirectly to the subject of this article.

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0363-5023/09/34A08-0010$36.00/0
doi:10.1016/j.jhsa.2009.04.037

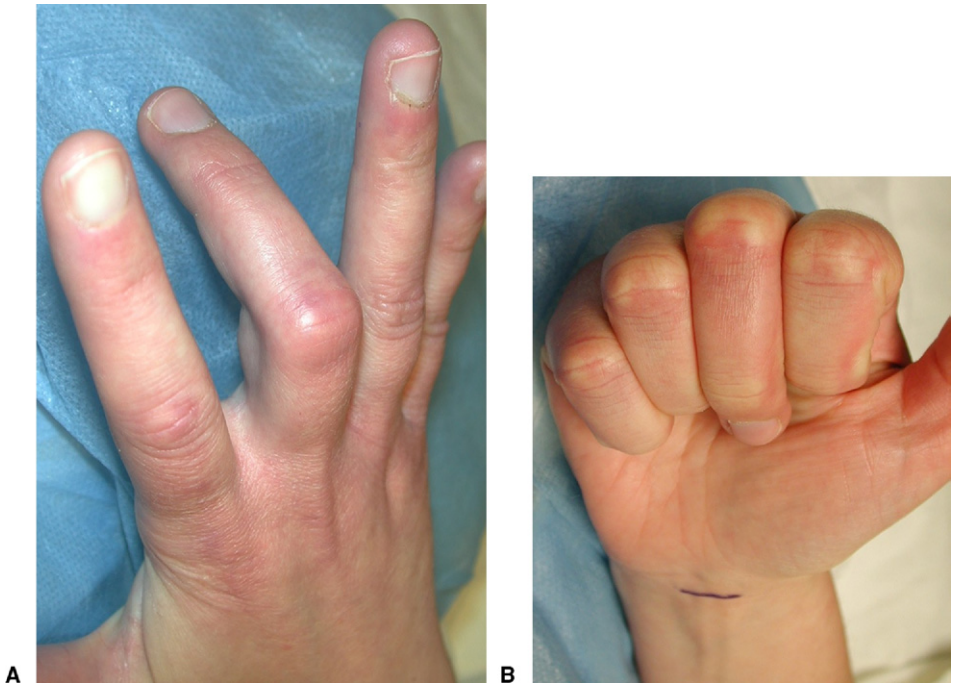

FIGURE 1: Clinical photographs of the patient's affected hand at time of presentation to our institution, showing **A** the 55° flexion contracture and **B** the limitation of active composite flexion.

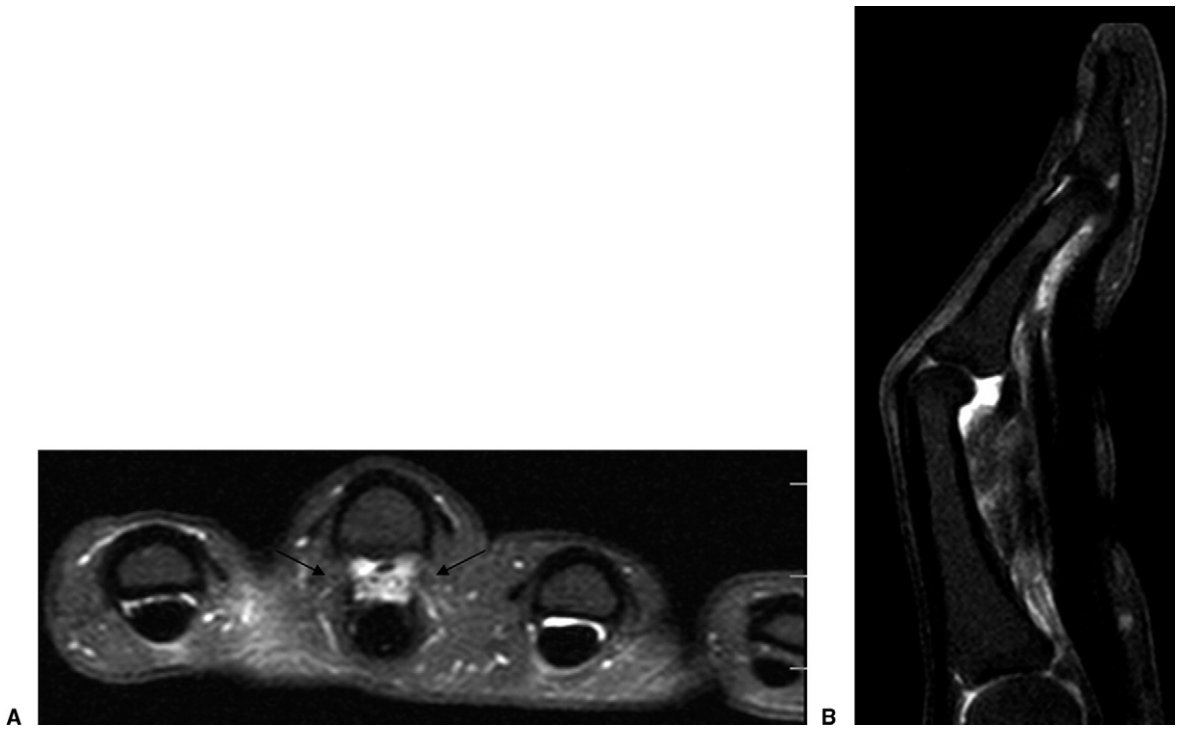

FIGURE 2: Axial image at the level of the A2 pulley from magnetic resonance imaging of the middle digit. There is complete disruption of the A2 pulley (arrows), with palmar displacement of the flexor tendon. **A** The intact pulley is present in adjacent digits. **B** Sagittal image of the middle digit demonstrating palmar displacement of the flexor tendon.

phalangeal (DIP) joint was supple, and the patient had full passive composite flexion. Good pull-through of both the profundus and superficialis tendons was evidenced by independent PIP and DIP motion. Flexor tendon excursion was limited, as evidenced by active composite flexion to within 2 cm from the distal palmar

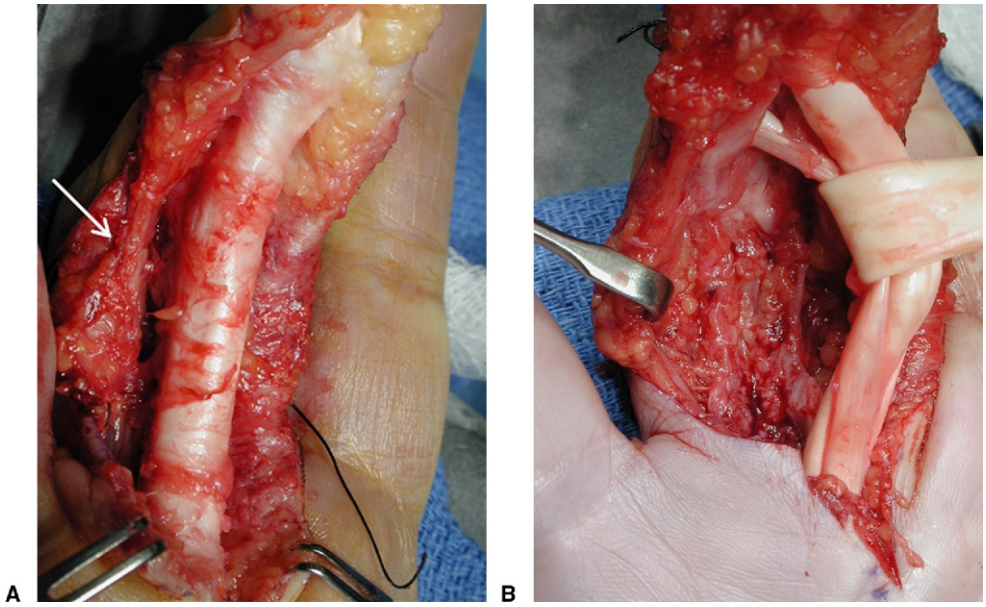

FIGURE 3: The flexor tendon sheath was gray, with evidence of chronic inflammation. The pulley system between A1 and A4 was completely disrupted. **A** The flexor tendon sheath lies volar to the neurovascular bundle (white arrow), consistent with bowstringing of the tendon. **B** The incompetent sheath and pulley remnants were excised, leaving the "ever present rim" at the base of the flexor tendon sheath intact.

crease (Fig. 1B). Magnetic resonance imaging revealed complete disruption of the A1, A2, C1, and A3 pulleys (Figs. 2A, 2B). Surgical treatment with pulley reconstruction was recommended.

During surgery, the flexor apparatus from the DIP joint to the metacarpophalangeal (MCP) joint was exposed through a Bruner incision. The flexor tendon sheath was gray, with evidence of chronic inflammation (Fig. 3A), and the tendons were elevated from the proximal and middle phalanges. Complete disruption of the A1 through a portion of A4 pulleys was confirmed. The incompetent sheath and pulley remnants were excised, leaving the "ever present rim"[11] at the base of the flexor sheath intact (Fig. 3B). Lysis of adhesions along the full length of the flexor tendon and gentle manipulation of the digit permitted full passive extension of the digit without capsulotomy or collateral ligament release. The palmaris longus tendon was harvested with a tendon stripper through a transverse incision at the wrist crease, and split longitudinally in preparation for pulley reconstruction. Using the Weilby technique for pulley reconstruction,[11] the tendon was woven in a shoelace pattern over 5 cm of the flexor tendon, through the intact fibrosseous rim of the previous pulley system, and secured using 3-0 braided polyester suture (Fig. 4). Given the long-standing flexion contracture, the PIP joint was temporarily pinned in extension using a single 1.14-mm (0.045 in) K-wire to relieve tension on the pulley repair.

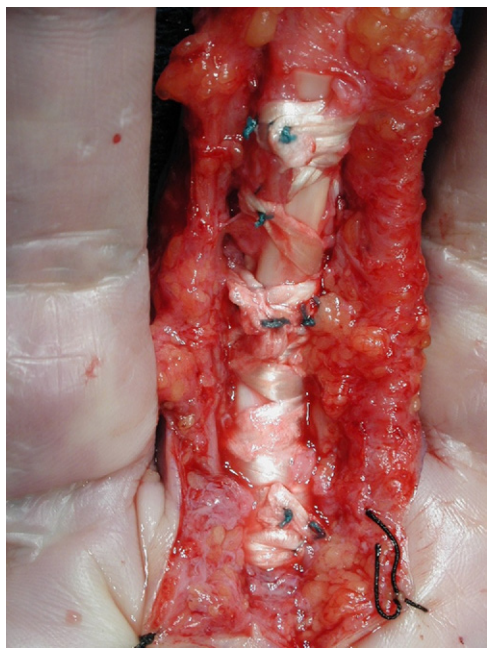

FIGURE 4: Reconstructed pulley, using the palmaris tendon graft woven through the intact pulley rim in a shoelace pattern over the flexor tendon.

Immediate motion of the DIP was begun; MCP joint motion was begun at 3 days after surgery. The pin was removed at 3 weeks, and a protected tenodesis flexor tendon protocol with a pulley ring splint was begun, including place and hold exercises. At 7 weeks after surgery, the patient's progress had plateaued, demon-

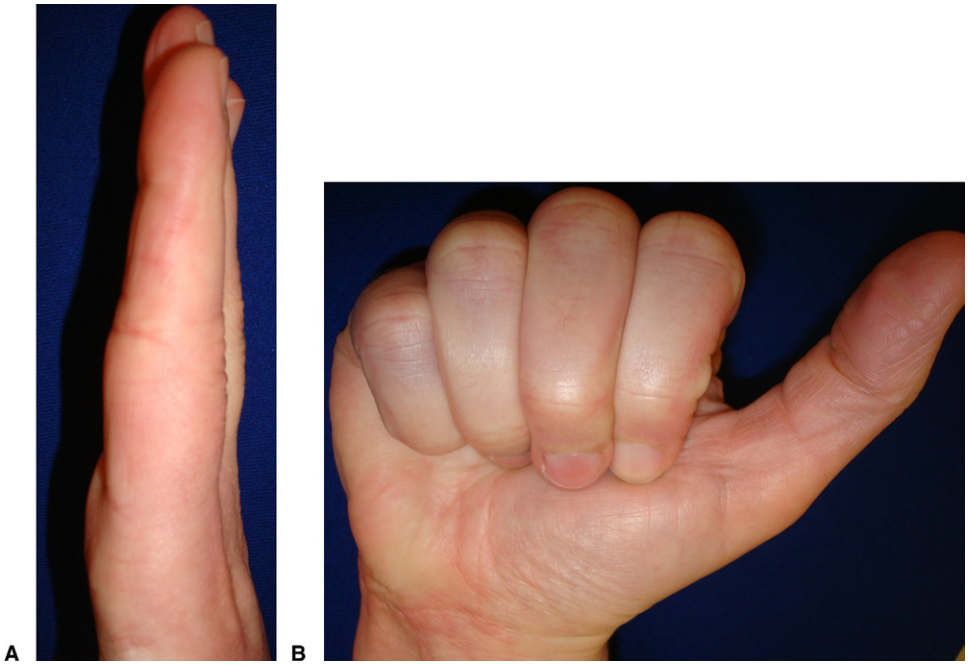

FIGURE 5: At 15 months after surgery, the patient has **A** only 5° flexion contracture at the DIP and PIP joints and **B** near full composite flexion.

strating PIP active motion of 0° to 35° and DIP motion of 5° to 10°; consequently, a tenolysis was recommended. At the second surgery under local anesthesia, using tenolysis blades (Meals tenolysis blades, George Tiemann & Co, Hauppauge, NY) through limited windows at the MCP and PIP joint lines, full active tendon excursion was restored in the operating room through the intact pulley reconstruction. At 15 months after tenolysis, the patient had 5° to 88° of motion at the PIP joint, 5° to 65° at the DIP joint, and 0° to 90° at the MCP joint (Figs. 5A, 5B) Strength measurements on the affected side (vs the unaffected side in parentheses) were grip strength, 27 kgf (30); lateral pinch, 8.2 kgf (6.8); 3-jaw pinch, 6.4 kgf (5.5); and tip pinch, 2.3 kgf (2.3). There was no evidence of bowstringing. The patient found minor difficulty with writing and had fatigue and cramping pain with prolonged manual activity, but she was able to return to her work as a general practitioner and all daily activities.

DISCUSSION

Corticosteroid injection is a mainstay of nonsurgical management of trigger digit. The success rate of injection ranges from 60% to 92% using 1 to 3 injections,[1,12] and the procedure enjoys a low complication rate. Two previous case reports document flexor tendon ruptures following suspected intratendinous injection.[7,8] In 1 case, the flexor pollicis longus tendon ruptured in a 62-year-old patient who was opening a drawer 4 years after 2 injections for trigger thumb.[7] The particular medication used in this case was not cited. In the second case, flexor digitorum superficialis and profundus tendon ruptures were diagnosed in a 77-year-old patient 13 months after 2 corticosteroid injections (1 mL local anesthetic and 0.5 mL triamcinolone acetonide 40 mg/mL).[8] Although the direct link of corticosteroid injection to tendon rupture is not possible, both case reports conclude that intratendinous injection should be avoided, given the attritional effects of corticosteroids on tendons. One case in the literature describes closed thumb pulley rupture in a 38-year-old patient after 2 corticosteroid injections for tenosynovitis.[9] The medication, dose, site of injection, and extent of bowstringing are not noted in the case report.

Numerous *in vitro* and animal studies have been performed in attempts to investigate the relationship between corticosteroid injection and tendon ruptures. The varying doses and different steroid preparations make direct causal conclusions difficult. Intratendinous steroid injection has been shown to cause collagen necrosis and subsequent tendon weakness.[13] Triamcinolone and dexamethasone have been shown to suppress human tenocyte cellular activity and reduce collagen production.[14,15] Fragmentation of collagen bundles and inflammatory cell infiltration have been observed with tendon injection of methylprednisolone and betamethasone.[16] Although a precise etiology for pulley rupture cannot be determined, the finding of an amorphous flexor sheath without discernable pulleys between A1 and A4 suggests collagenous necrosis related to corti-

costeroid injection. This is further supported by the lateral approach, which delivers the corticosteroid in direct proximity to the pulley system. Whether a more water-soluble preparation would have had the same effect cannot be ascertained. The effect of various corticosteroid preparations on collagen production and function would be of interest in determining whether future complications could be avoided.

Given the potential for tendon rupture, as documented in previous case reports, and our finding of multiple pulley rupture in this patient, we advise no more than 2 injections of a relatively water-soluble corticosteroid preparation, placed in close proximity to the A1 pulley, and advise against injection in or near the critical A2 pulley.

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