A1 pulley stretching treats trigger finger: A1 pulley luminal region under digital flexor tendon traction (A1 pulley ストレッチによるばね指の治療 -深指屈筋腱牽引に伴う A1 pulley 内腔変化-)

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ABSTRACT

Background: A1 pulley stretching is recognized as a clinically beneficial treatment for trigger finger. It is thought to lead to an increase in the cross-sectional area of the A1 pulley luminal region, thus improving trigger finger symptoms. The purpose of the present study was thus to evaluate the resultant forces during stretching that increase the CSA of the A1 pulley luminal region using fresh-frozen cadavers.

Methods: Using seven fingers from three fresh-frozen cadavers to replicate A1 pulley stretching, we investigated the resultant forces during stretching that increase the cross-sectional area of the A1 pulley luminal region. The traction forces of the flexor digitorum profundus tendons were increased in steps to 150 N, and the cross-sectional area and height of the A1 pulley luminal region were measured using ultrasonography.

Findings: The cross-sectional area of the A1 pulley luminal region increased with step-wise increases in the flexor digitorum profundus traction. On average, the cross-sectional area and height of the A1 pulley luminal region showed increases of 31.4% and 43.6%, respectively, compared to the unloaded condition.

Interpretation: These results confirmed that A1 pulley stretching increases the cross-sectional area of the A1 pulley luminal region. A1 pulley stretching has the potential to reduce the severity of trigger finger in patients facing surgery.

Key words: trigger finger, conservative therapy, A1 pulley stretching, cadaveric anatomical study

INTRODUCTION

Trigger finger is one of the most common hand disorders treated by hand surgeons and the lifetime risk is estimated at 2% to 3% in the general population^{1, 2}. Conservative management approaches for trigger finger include activity modification, splinting, corticosteroid injection, and other adjuvant modalities^{3, 4, 5}. By contrast, physiotherapy is partially accepted as a treatment for trigger finger⁶. It is thought that joint mobilization can increase joint and soft tissue mobility via slow, passive therapeutic traction and translational gliding. Physical exercise for trigger digits was rarely reported until the effect of A1 pulley stretching⁷ was presented in 2015. However, some clinicians remain skeptical about its success.

A1 pulley stretching is a clinically beneficial physical exercise that involves resisted proximal interphalangeal and metacarpophalangeal (MP) joint flexion achieved by fully grasping a block (Fig. 1). A1 pulley stretching should be performed at least 10 times a day for 30 seconds at a time. Chiba et al. reported that A1 pulley stretching generates both an active flexion force and a counteracting flexion tendon force, which might lead to an increase in the cross-sectional area (CSA) of the A1 pulley luminal region and thus improve trigger finger symptoms (Fig. 2). Chiba et al. further indicated that pain score and signs of snapping and locking improve with physiotherapy techniques, including A1 pulley stretching. Iwakura et al. further confirmed the effectiveness of A1 pulley stretching by demonstrating that steroid injection with stretching-including A1 pulley stretching—extended time to recurrence better than steroid injection only⁸. However, the mechanism of A1 pulley stretching has not yet been proven. We support the hypothesis that A1 pulley stretching generates an active flexion force and a counteracting flexion tendon force that together lead to an increase in the CSA of the A1 pulley luminal region. In a preliminary study, we examined the A1 pulley among patients with trigger finger using an ultrasonographic system. However, the CSA of the A1 pulley luminal region shown by the ultrasonographic system was easily changed when patients put their strength into the flexor muscle because the wrist and fingers could not be firmly fixed. Based on those results, we determined that it was not possible to measure the identical CSA of the A1 pulley luminal region quantitatively. However, in a cadaveric study, fingers can be securely fixed in the same position, enabling us to evaluate the identical CSA of the A1 pulley luminal region quantitatively. The purpose of the present study was thus to evaluate the resultant forces during stretching that increase the CSA of the A1 pulley luminal region using fresh-frozen cadavers. We aim to confirm the efficacy of A1 pulley stretching and identify the mechanism underlying its benefits using cadavers.

METHODS

Type of study and level of evidence: Laboratory science study

Study design

The study included seven fingers (three index fingers, three middle fingers, and one ring finger) from three cadavers (85-year-old male, 94-year-old female and 84-year-old female).

kept at -20 °C and not repeatedly frozen. The average hand size was 6.4 inches (5.9, 6.6 and 6.7 inches respectively) and the average ratio of length and width was 2.3(2.3, 2.2 and 2.3 respectively). All specimens were free from signs of trauma, deformity or prior surgery. This study was approved by the appropriate ethics committee of the Graduate School of Medicine, Chiba University.

Replication of A1 pulley stretching

Just before the experiment, the cadavers were thawed and cut in the middle of the upper arm. We made an incision in the skin of the palmar forearm and identified the flexor digitorum profundus (FDP) tendons. The FDP tendons were cut at the junction of the muscles and tendons, and a wire was sutured at the distal end of the FDP tendons. The other end of the wire was loaded into a universal testing machine (Autograph AG-20kN Xplus; Shimadzu, Kyoto, Japan). We fixed the forearm in supination to the jig, the wrist in the neutral position, and the MP joints in 90° flexion using 0.30-cm diameter Kirschner wires. To replicate A1 pulley stretching, an ultrasonographic system (SSA-640A ultrasound machine) fitted with a PLT-1204BT 12-MHz compact linear transducer (Toshiba, Tokyo, Japan) was used. The probe was placed perpendicular against the palmar surface at the A1 pulley level of the examined digit to lock the examined finger in a flexed position and to focus the ultrasonographic analysis on the A1 pulley, flexor tendon, volar plate and head of the metacarpal bone (Fig. 3). We positioned the probe with minimal pressure on the tissue in the same manner described by Sato et al.⁹ and fixed it to the jig to avoid changing the ultrasonographic images. The FDP tendons were pulled proximally at a speed of 1.0 mm/s through the stiff string until 150 N of traction force was recorded at 100 Hz. Still ultrasonographic images were captured at each 5 N increase in FDP traction force up to 150 N. After reaching 150 N of traction force, we released the FDP tendons. At 1 and 15 min after traction release, still ultrasonographic images were obtained. At each step, the CSA and anteriorposterior height of the A1 pulley luminal region were measured using ImageJ 1.46r software (National Institutes of Health, Bethesda, MD, USA) (Fig. 4). The CSA and height of the A1 pulley luminal region under traction divided by the values unloaded were calculated as the rate of change. The roof of the A1 pulley, indicated as a low area is drawn near the body surface, and the outline of the round low area visible in the deep

layer, which is defined as CSA, is shown by ImageJ. The distance between the lowermost edge and the uppermost edge in this CSA was defined as the height. The average value was calculated from three different measures.

The CSAs of the A1 pulley luminal region under traction using 150 N of force and after traction were compared with the CSAs before traction. The Wilcoxon signed-rank test was used to assess differences and p-values < 0.05 were considered statistically significant.

RESULTS

A1 pulley stretching was replicated in all fingers. The CSA and height of the A1 pulley luminal region increased with a step-wise increase in the FDP traction (Fig. 5). When the FDPs were drawn proximally using 150 N of force, the CSA and height of the A1 pulley luminal region showed average increases of 31.4% and 43.6%, respectively, compared to the values in the unloaded condition (Fig. 6). The difference in CSA between no traction force and 150 N traction force was statistically significant (p = 0.05). Additionally, the difference in height between no traction force and 150 N traction force was statistically significant (p < 0.05). One minute after releasing the FDP tendons, the CSA and height of the A1 pulley luminal region showed average increases of 4.8% and 11.3%, respectively, compared to the values in the unloaded condition. Moreover, 15 min after releasing the FDP tendons, the CSA and height of the A1 pulley luminal region showed average increases of 3.0% and 6.7%, respectively, compared to the values in the unloaded condition.

DISCUSSION

The effect of A1 pulley stretching⁷ for trigger finger was first reported in 2015. Iwakura et al. supported clinical use of A1 pulley stretching in combination with corticosteroid injection⁸. However, the extensive mechanism of A1 pulley stretching has not yet been proven. In this study, resisted finger flexion by A1 pulley stretching was replicated using fresh-frozen cadavers. Here, we quantitatively evaluated CSA changes in the A1 pulley luminal region associated with the resultant active flexion force and counteracting flexion tendon force by holding the fingers and using an ultrasonographic probe in a consistent position. The results showed that A1 pulley stretching caused the CSA and height of the A1 pulley luminal region to increase. After terminating traction of the FDPs, the CSA, and height of the A1 pulley luminal region remained higher. When pulling the flexor tendon while fixing the position of the finger, an active flexion force and a counteracting flexion tendon force are applied to the flexor tendon in contact with the A1 pulley. In this study, the isometric finger flexion movement in the A1 pulley stretch was reproduced. The A1 pulley exists to prevent flexor tendon bowstring at the

metacarpophalangeal joint (MP joint) and the structure that is in direct contact with the A1 pulley is considered to be the finger flexor tendon only. As the fingers are fixed, A counteracting force that resists traction force is generated. At this time, if the MP joint is flexed, the direction of the resultant force vector will be in the palm direction. The A1 pulley exists in only direct contact with the palmar side of the flexor tendon at the MP joint level and the resultant force is generated in the direction in which the A1 pulley is expanded to the palm side. In addition, this resultant force is the only force applied to the A1 pulley. It can be considered that the resultant force of the active flexion force and the counteracting flexion tendon force expands the CSA and height of the luminal region of the A1 pulley.

In this study, a force up to 150 N was applied to pull the flexor tendons. From previous research^{10,11}, it is argued that the fingertip force is about three times the tendon force. If the force applied to one tendon is 150 N, assuming that 150 N of traction force is applied to all eight flexor tendons acting on the index, middle, ring, and little finger, the total traction force of all eight fingers will be 1200 N. If the relationship between the above fingertip force and tendon force is used, the actual grip force is expected to be 400 N (40.8 Kgf), so the 150 N tractive force used in this study is considered appropriate. A study of the material characteristics of human pulleys¹² reveals that some

elongation occurs when force is applied to the A1 pulley. Therefore, it is considered that the A1 pulley is extended by the resultant force of the active flexion force and the counteracting flexion tendon force. As a result, the CSA and height of the A1 pulley luminal region are expanded. In addition, since the expansion of the CSA and height of the A1 pulley luminal region remained even after the traction was released, the possibility that the CSA and height of the A1 pulley luminal region could be enlarged by applying this force intermittently cannot be denied.

If the expansion of the CSA of the A1 pulley luminal region can be maintained by A1 pulley stretching, a decrease in friction between the flexor tendon and the A1 pulley can be expected. Trigger finger is characterized by impingement of the flexor tendons under the A1 pulley¹³, and for surgical treatment, the A1 pulley is separated and the symptom is improved structurally by eliminating this stenosis. If structural stenosis can be improved by A1 pulley stretching as a conservative method, improvement of trigger finger symptoms can be expected. In some cases, it may be possible to avoid surgical treatment with this stretch.

As A1 pulley stretching can be easily performed by patients at home, the use of A1 pulley stretching will have economic benefits.

There are several limitations associated with our study design. First, we assessed cadaveric fingers that were not affected with trigger finger. In addition, we did not consider hand dominance, age, job, or underlying diseases. As the cadaveric fingers did not have fibrocartilaginous metaplasia or hypertrophy of the A1 pulley, differences in the form of the A1 pulley may have affected the increase in the CSA and height of the A1 pulley luminal region. However, we decided that it would be difficult to confirm the same cross-section of the A1 pulley by using a living body for preliminary experiments. A cadaver was used in order to reproduce the movement of the A1 pulley and to prioritize quantitative evaluation by drawing the same cross-section.

Here, we completely replicated not only the finger motion but also the finger position at A1 pulley stretching using cadaveric fingers. Our findings show that the CSA of the A1 pulley luminal region of patients with trigger finger cause similar changes as that of cadavers. Second, the results of this study cannot explain the persistent increase in CSA after A1 pulley stretching. Based on the difference in soft tissue properties between cadavers and humans, further clinical research using a living body is required to determine whether this stretch reduces the severity. Third, the same force magnitude was applied to the index, middle, and ring fingers in the tests. Although this may be different from the actual force applied to each tendon, the tendon forces of each finger

in different age and gender groups varies greatly; thus, we considered it difficult to estimate the tendon forces of each finger. Therefore, by changing the traction force for each finger in the same way, we measured the CSA of the A1 pulley for each traction force and considered it to be comprehensive fundamental data.

Finally, the extensors have not been reproduced. We emphasized the following reports: Finger extensors have approximately one-third the work capacity of the finger flexors¹⁴. As long as the finger flexors are active, extensors will not overpower grip and cause release even if the electrical activity is associated with maximal extensor digitorum communis (EDC) contraction¹⁵. In addition, the EDC may also help stabilize the wrist during grip and may help stabilize the metacarpophalangeal joints in flexion during grip by pulling the metacarpals together via the juncturae tendinae¹⁴. In this study, the wrist joint and the MP joint were fixed and only the flexor tendon was pulled since the only tendon in direct contact with the A1 pulley was the flexor tendon. As described above, A1 pulley stretching can be reproduced sufficiently.

In sum, A1 pulley stretching caused the CSA and height of the A1 pulley luminal region to increase. Whether this stretch directly reduces the severity of trigger fingers requires further research. This study basically supports the possibility that A1 pulley stretching is structurally useful as a preservative treatment for trigger fingers.

CONCLUSION

We completely replicated not only the finger motion but also the finger position at A1 pulley stretching using cadaveric fingers. The results confirmed that A1 pulley stretching increases the CSA of the A1 pulley luminal region. A1 pulley stretching has the potential to reduce the severity of trigger finger in patients facing surgery.

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Figure legends:

- Fig. 1. A1 pulley stretching is achieved by fully grasping a block
- Fig. 2. Mechanism of A1 pulley stretching
- Fig. 3. Ultrasound probe and finger position
- Fig. 4. Ultrasonographic image
- Fig. 5. Average rate of change in the cross-sectional area and height of the A1 pulley luminal region
- Fig. 6. Average rate of change in the cross-sectional area and height of the A1 pulley luminal region
- at 150 N traction, immediately after traction and 15 minutes after traction

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