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The thickness of the A2 pulley and the flexor tendon are related to the severity of trigger finger: results of a prospective study using high-resolution ultrasonography

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Abstract

We aimed to investigate the relationship between the pulley-tendon complexes and the severity of trigger finger. The thickness of the A1 and A2 pulleys, and the cross-sectional area of the flexor tendon under the pulleys, were prospectively assessed using 17 MHz high-resolution ultrasonography, in 20 patients with trigger finger (31 fingers). A control group comprised 15 asymptomatic fingers. The thickness of the A1 pulley and the proximal part of the A2 pulley, and the cross-sectional area of the flexor tendon under the A2 pulley, were significantly increased in the patient group. Clinical grade was significantly correlated with the thickness of the A1 pulley, the thickness of the proximal part of the A2 pulley, and the cross-sectional area of the flexor tendon under the proximal part of the A2 pulley. This study confirmed that the thickness of the A2 pulley and flexor tendon under the A2 pulley seems to be related to the severity of trigger finger.

Level of evidences: Level III

Keywords

Trigger finger, A2 pulley, cross-sectional area of the flexor tendon, high-resolution ultrasonography

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Introduction

The pathophysiology of trigger finger (stenosing tenosynovitis) has been studied extensively. Some authors have referred to trigger finger as 'stenosing tenovaginitis' based on the understanding that, histologically, the inflammatory changes are localized specifically to the tendon sheath (tendovaginal) (Burman, 1952; Fahey and Bollinger, 1954); however, others have reported that the histologic abnormalities of trigger digits are not related to inflammation (Drossos et al., 2009). In a recent histologic study, inflammatory changes were observed in the tenosynovium of the flexor tendon (Uchihashi et al., 2014), while results of a genomic study by Lundin et al. indicated that trigger finger might be a tendinosis, similar to Achilles tendinopathy (Lundin et al., 2014). Furthermore, a recent study revealed that volar migration of the flexor tendon after surgical release of carpal tunnel syndrome is related to the occurrence of trigger finger (Lee et al., 2014).

Although the exact pathophysiology of trigger finger is unclear, there seems to be no disagreement that trigger finger occurs as a result of a mismatch between the volume of the flexor tendon sheath and

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the tendon (Miyamoto et al., 2011; Ryzewicz and Wolf, 2006; Sato et al., 2012). Release of the A1 pulley is generally accepted as the surgical treatment for trigger finger (Makkouk et al., 2008; Ryzewicz and Wolf, 2006). However, in clinical practice, we have experienced more than a few cases of trigger fingers that have required simultaneous release of the proximal part of the A2 pulley, as well as the A1 pulley, for complete resolution of triggering. Al-Qattan also described similar experiences, and reported, in a study of 50 adult patients, that such cases have an incidence of 8% (Al-Qattan, 2007). A review of the literature indicated that, with the exception of a single recent report (Sato et al., 2014), no study has been conducted on the anatomical involvement of the A2 pulley in trigger finger.

The purpose of the current study was to investigate the anatomical relationship between elements of the pulley system (the A1 pulley, the A2 pulley, and the cross-sectional area of the flexor tendon under the pulleys) and the severity of trigger finger, using 17-MHz high-resolution ultrasonography (US).

Methods

Population

Local institutional review board approval in Hanyang University Hospital was obtained for the study. We performed a prospective study involving 58 patients, referred to the authors' institute with symptoms and signs of triggering digits, between May and November 2014.

Inclusion criteria were clinically diagnosed triggering, examined using US. Exclusion criteria were the presence of trigger thumb; a history of previous surgical release of trigger finger on the same digit; a history of rheumatoid arthritis, chronic renal failure requiring dialysis, or uncontrolled diabetes mellitus. A total of 32 patients with trigger thumbs, five patients with a history of surgical release of trigger finger on the same digit at other hospitals, and one patient with Behcet disease were excluded; the remaining 20 patients (7 male, 13 female; 31 fingers) were included in the final study. The mean age was 55.9 years (range 21–79). Two patients had bilateral trigger fingers involving the same digit; nine patients had triggering on two fingers simultaneously, on ipsilateral hands. A total of 20 cases occurred on the right hand, and 11 cases occurred on the left. Four cases involved the index finger, 18 cases involved the middle finger, nine cases involved the ring finger, and one case involved the little finger. The severity of each case was graded using the clinical grading system described by Froimson (1999); Grade I, pre-triggering pain, tenderness over the A1 pulley,

history of catching, but no demonstrable catching; Grade II, demonstrable catching, patient can actively extend; Grade III, demonstrable catching requiring passive extension or inability to actively flex; Grade IV, demonstrable catching with fixed flexion proximal interphalangeal (PIP) joint contracture. A total of 15 asymptomatic digits in the contralateral hand of the study subjects were also examined with US, and served as the control group.

Sample size calculation

Based on the study by Sato et al., the mean thickness of the proximal A2 pulley and its standard deviation were 0.435 mm and 0.24, respectively, in the trigger finger group, and 0.27 mm and 0.09, respectively, in the control group (Sato et al., 2014). With the statistical model of the independent student *t*-test, a significance level of 0.05, a power of 80%, and ratio of patient:control of 2:1, the required sample size was 27 trigger fingers and 13 control group fingers.

Ultrasonographic evaluation

With the examinee seated on a chair, we positioned their forearm on a table, supinated with the wrist in a neutral position. The metacarpophalangeal (MP) and PIP joints were fully extended on the table, and an adequate amount of ultrasound gel was dispensed onto the examinee's hand on the palmar side, in the area between the distal palmar crease and the PIP joints. Using sufficient ultrasound gel on this area is important, because the gap between the transducer and the palmar digital crease can interfere with the clarity of US images, particularly around the A2 pulley. The transducer was positioned perpendicular to the palm of the examinee's hand with minimal pressure. Sagittal and axial plane studies were performed in this standardized position.

In sagittal plane studies, we measured the thickness of the volar plate at the metacarpal (MC) head (Figure 1). In axial plane studies, we measured the thickness of the A1 pulley; the thickness of the proximal and distal parts of the A2 pulley; and the cross-sectional area of the flexor tendon. While sweeping the transducer from distal to proximal on the MP joint area, there is a point at which the volar plate emerges between the flexor tendon and the MC head. At this level, we measured the thickness of the A1 pulley and the cross-sectional area of the flexor tendon under the A1 pulley. Slightly tilting the transducer in a distal to proximal direction usually helped to identify the A1 pulley in this area. The pulley could be visualized as a hyperechoic (relative to the adjacent flexor tendon) linear structure surrounded by the hypoechoic space

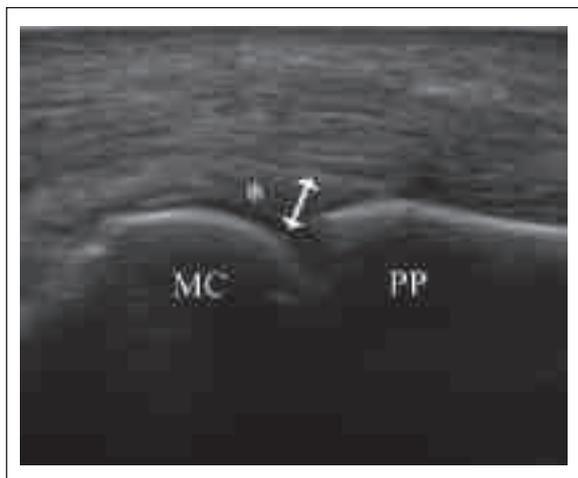


Figure 1. Sagittal plane image of MP joint. The thickness of volar plate was measured at its thickest portion (double-headed arrow).

*: volar plate; MC: metacarpal head; PP: proximal phalanx.

around the flexor tendon (Boutry et al., 2005; Sato et al., 2012). The hyperechoic linear structure is hardly identifiable in some cases; as such, we regarded the whole thickness of the hypoechoic space that contained a hyperechoic linear structure, as the thickness of the pulley (Sato et al., 2012). Inclining the transducer along the medial to lateral edge of the flexor tendon could aid identification of the hyperechoic linear pulley structure by eliminating anisotropy, and facilitating visualization of the pulley, coursing dorsally toward the MC bone (Figure 2(A)). After assessing the thickness of the A1 pulley, and the cross-sectional area of the flexor tendon under the A1 pulley, we directed the examinee to flex their finger to an approximately 45° angle at the MP joint, 60° angle at the PIP joint, and 45° angle at the distal interphalangeal joint, while the transducer was kept in the same position. Because of the contact between the finger and transducer, it was not possible for the finger to be fully flexed. In this slightly flexed finger position, the cross-sectional area of the flexor tendon was measured again.

We then swept the transducer distally, to identify the proximal part of the A2 pulley. The MC head and volar plate disappeared from view during this movement, and the flexor tendon could be observed running in close contact with the cortex of the proximal phalanx. The thickness of the A2 pulley, and the cross-sectional area of the flexor tendon under the proximal part of the A2 pulley were assessed with the finger in extension, and also in slight flexion (Figure 2(B) and (C)). Finally, the distal part of the A2 pulley was identified while sweeping the transducer more distally, and its thickness was measured. All US evaluations were

performed using 17 MHz high-resolution ultrasound (E-CUBE 15 with L8-17X transducer; ALPINION medical systems, Seoul, Korea) by an orthopaedic surgeon with 6 years' experience in US examination, who was blinded to the subjects' information. All images were stored and assessed on the PiViewSTAR measurement tool (Pi-view 5.08; Infinit, Seoul, Korea). All the variables were measured by another independent orthopaedic surgeon who was blinded to the prior rater's results and clinical information of the subject, to estimate measurement reliability.

In summary, the structures assessed with US in the current study comprised eight variables; the thickness of the A1 pulley; the thickness of the proximal part of the A2 pulley; the thickness of the distal part of the A2 pulley; the thickness of the volar plate located at the metacarpal head; the cross-sectional area of the flexor tendon under the A1 pulley with the finger in extension and in slight flexion; and the cross-sectional area of the flexor tendon under the proximal part of A2 pulley with the finger in extension and in slight flexion.

Each measured variable was compared between the two groups. Correlation analysis was performed between the clinical grade and pulley and tendon complex variables within patients group.

Statistical analysis

To determine measurement reliability, intraclass correlation coefficients (ICC) were calculated for all the measured variables. Kolmogorov–Smirnov tests were used to evaluate the normality of the data. To compare the measurements between the two groups with a continuous scale, the independent two-sample Student *t*-test or the Mann–Whitney *U*-test were used, according to the normality of the data. Dichotomous data were compared using the chi-square test or Fisher exact test. The cut-off thickness of the A1 and the A2 pulley that distinguished between the two groups was determined with the use of a receiver operating characteristics curve analysis; and the area under the curve, the sensitivity, and the specificity were estimated. To evaluate correlation between clinical grade and variables measured on US examination within patient groups, Pearson correlation coefficients or Spearman rho were estimated, according to the normality of data. The significance level for all statistical analyses was set at 0.05.

Results

The baseline demographic data of the subjects were listed in Table 1. The results of the analysis of measurement reliability are reported in Table 2. ICC for

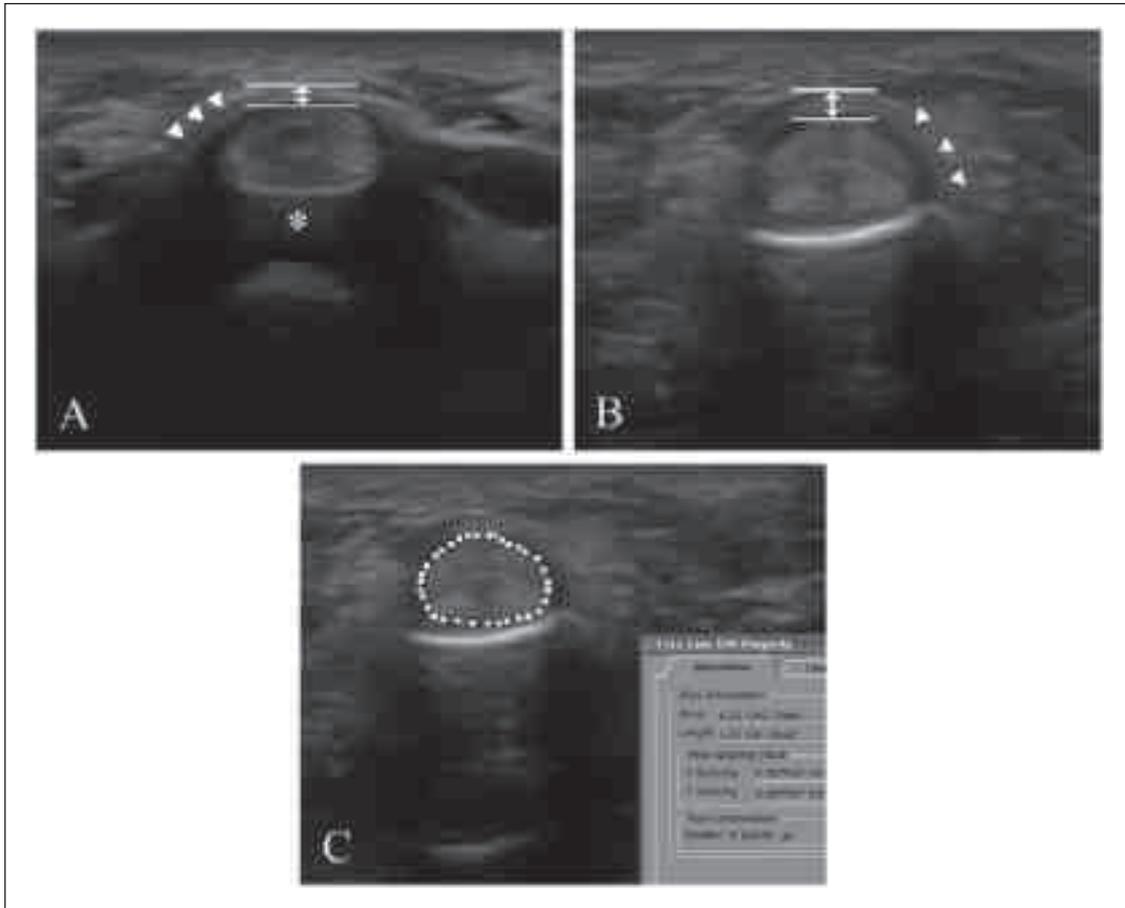


Figure 2. Axial plane image of the pulleys. Double-headed arrow indicating the thickness of the pulley. Arrow heads indicating the pulley coursing dorsally toward cortex of digital bone. (A) The A1 pulley. (B) The proximal part of the A2 pulley. (C) Measurement of the cross-sectional area of the flexor tendon under pulleys. The measurement was done using PiView-STAR measurement tool (Pi-view 5.08; Infinitt, Seoul, Korea).

Table 1. Baseline demographic data of patient.

Number of cases	31 (20 patients)
Sex (men: women)	7:13
Mean age (years, range)	55.9 (21 to 79)
Side (right:left)	20:11
Clinical grading (I:II:III:IV)	5:7:12:7
Involved finger (number)	
Index finger	4 (13%)
Middle finger	18 (58%)
Ring finger	8 (26%)
Little finger	1 (3.2%)
Clinical grading of each finger (mean, median)	
Index finger	3.0 (3)
Middle finger	2.5 (2.5)
Ring finger	2.88 (3)
Little finger	3

most of the measurements showed acceptable measurement reliability, ranging from 0.711 to 0.941

(Hirschmann et al., 2011; Landis and Koch, 1977), except the thickness of the distal part of the A2 pulley (ICC=0.669). Comparison of the results of the two groups is shown in Table 3. Compared with the control group, the patient group had a significantly thicker A1 pulley ($p < 0.001$), proximal A2 pulley ($p < 0.001$), and distal A2 pulley ($p = 0.034$). The cross-sectional area of the flexor tendon under the A2 pulley was greater in the patient group, compared with controls, regardless of the finger position (finger in extension, $p = 0.014$; finger in slight flexion, $p = 0.024$). The results of the receiver operating characteristics curve analysis performed to determine the cut-off thickness of the A1 pulley and the proximal part of the A2 pulley are depicted in Figure 3. The area under the curve for the thickness of the A1 pulley was 0.899 [95% confidence interval (CI), 0.810 to 0.988; $p < 0.001$], and the cut-off thickness was 1.15 mm. With this cut-off value, the sensitivity was 71%, and the specificity was 87%. The area under the curve for the thickness of the A2 pulley was 0.886 [95% CI,

Table 2. Interobserver measurement reliability of the variables.

Variable	ICC	95% CI	<i>p</i> value
A1 pulley thickness	0.741	0.327 to 0.900	0.003
A2 pulley thickness (proximal part)	0.711	0.250 to 0.889	0.006
A2 pulley thickness (distal part)	0.669	0.114 to 0.876	0.014
Volar plate at the A1 pulley	0.812	0.499 to 0.930	0.001
Cross-sectional area of the flexor tendon under the A1 pulley	0.814	0.518 to 0.929	<0.001
Cross-sectional area of the flexor tendon under the A1 pulley (finger in flexion)	0.787	0.430 to 0.920	0.001
Cross-sectional area of the flexor tendon under the proximal part of the A2 pulley	0.941	0.847 to 0.977	<0.001
Cross-sectional area of the flexor tendon under the proximal part of the A2 pulley (finger in flexion)	0.893	0.714 to 0.960	<0.001

ICC: intraclass correlation coefficients, CI: confidence interval.

Table 3. Comparison results of patients and control groups (Mean, standard deviation).

	Group I (patients)	Group II (control)	<i>p</i> value
<i>n</i>	31	15	—
Age (years)	56.1 (12.77)	58.2 (10.43)	0.786
Sex (M:F)	8:23	6:9	0.495
A1 pulley thickness (mm)	1.36 (0.26)	0.92 (0.20)	<0.001
Proximal A2 pulley thickness (mm)	1.30 (0.26)	0.88 (0.25)	<0.001
Distal A2 pulley thickness (mm)	0.94 (0.31)	0.77 (0.25)	0.030
Volar plate thickness at A1 pulley (mm)	17.4 (0.32)	17.3 (0.35)	0.643
Cross-sectional area of the flexor tendon under the A1 pulley (mm ²)	20.1 (3.76)	18.7 (3.28)	0.228
Cross-sectional area of the flexor tendon under the A1 pulley with finger in flexion (mm ²)	24.2 (4.97)	24.7 (4.01)	0.475
Cross-sectional area of the flexor tendon under the proximal part of the A2 pulley (mm ²)	22.7 (5.89)	19.0 (3.74)	0.014
Cross-sectional area of the flexor tendon under the proximal part of the A2 pulley with finger in flexion (mm ²)	23.8 (7.66)	18.9 (5.58)	0.024

Bold indicates statistically significant results ($p < 0.05$).

0.793–0.979; $p < 0.001$), and the cut-off thickness was 1.05 mm. With this cut-off value, the sensitivity was 81%, and the specificity was 80%.

The results of the correlation analyses are presented in Table 4. The thickness of the A1 pulley, the thickness of the proximal part of the A2 pulley, and the cross-sectional area of the flexor tendon under the proximal part of the A2 pulley with the finger in slight flexion, correlated positively with the clinical grade with statistical significance.

Discussion

We investigated the relationship between the thickness of the pulley-tendon complexes and the severity of trigger finger. A notable feature of the present study is that the A2 pulley and the flexor tendon under the A2 pulley area have been evaluated using 17 MHz high-resolution US. To the best of our knowledge, few

previous studies have described pathology of the A2 pulley in trigger finger; those that have tend to be case reports (Hirata et al., 1996; Ikeda and Osamura, 2010; Nagaoka et al., 2007). Only the study conducted by Sato et al. investigated the pathology of the A2 pulley in relation to the severity of trigger finger. In this study, the authors could not find significant correlation between the thickness of proximal part of the A2 pulley and clinical grading of trigger finger. Instead, they noted that the cases with PIP joint contracture had a thicker proximal A2 pulley, as well as a thicker A1 pulley, and a thicker antero-posterior diameter of the flexor tendon under the A1 pulley (Sato et al., 2014).

The current study is the second that has investigated the thickness of the A2 pulley in relation to trigger finger. The new finding revealed in this study is that the thickness of the proximal part of the A2 pulley, as well as the A1 pulley, and cross-sectional area

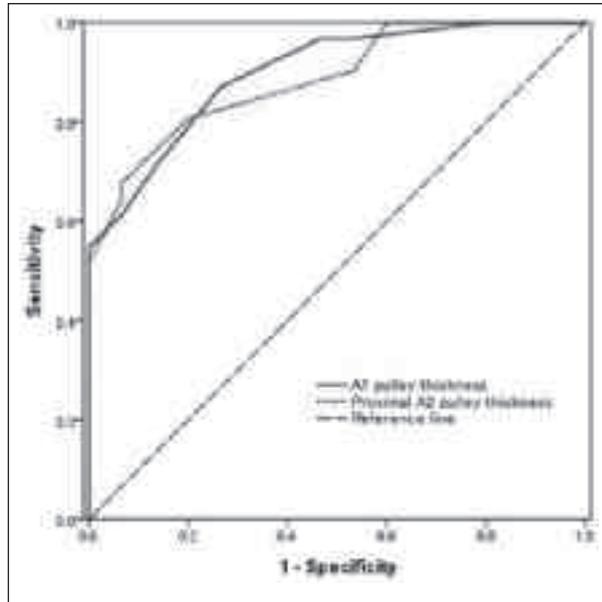


Figure 3. Result of receiver operating characteristics curve analysis. Area under curve of the thickness of the A1 pulley was 0.899; of the proximal part of the A2 pulley was 0.886.

of the flexor tendon at the proximal part of the A2 pulley (but not at the A1 pulley), are significantly correlated with the clinical grade of trigger finger. There is some conflict when these results are compared with previous research; Sato et al. (2012) and Sato et al. (2014) found that the diameter of the flexor tendon under the A1 pulley is correlated with clinical grade of trigger finger. A possible reason for this difference might be that, unlike previous researchers, we used the cross-sectional area of the flexor tendon rather than using just the diameter of the flexor tendon. It seems likely that the use of cross-sectional area of the tendon is more accurate than the use of its diameter in estimation of the tendon volume.

We hypothesized that, because the swollen flexor tendon is severely squeezed under the thickened and stiffened A1 pulley, the cross-sectional area of the flexor tendon under the A1 pulley may appear to be smaller than it actually is. As the trigger finger worsens, the inner gliding surface of the A1 pulley begins to wear, and is replaced by an invasive hyperplasia from the outer layer to the inner layer (Drossos et al., 2009). Miyamoto et al. (2011) also revealed that the stiffness of the A1 pulley is increased in trigger finger (Miyamoto et al., 2011), and Ferree et al. (2014) suggest that the tight A1 pulley might be related to the genes. As the A1 pulley thickens and the volume of the flexor tendon just distal to the A1 pulley is increasing, it also becomes more difficult for the tendon to slide into the A1 pulley during finger flexion; thus,

Table 4. Correlations between clinical grades of trigger fingers and tendon-pulley complexes.

	Correlation coefficients	<i>p</i> value
A1 pulley thickness	0.432	0.015
A2 pulley thickness (proximal part)	0.440	0.013
A2 pulley thickness (distal part)	0.261	0.156
Volar plate thickness at the A1 pulley	-0.155	0.406
Cross-sectional area of the flexor tendon at the A1 pulley	0.130	0.484
Cross-sectional area of the flexor tendon at the A1 pulley (finger in flexion)	0.132	0.478
Cross-sectional area of the flexor tendon at the proximal part of the A2 pulley	0.198	0.287
Cross-sectional area of the flexor tendon at the proximal part of the A2 pulley (finger in flexion)	0.463	0.009

Bold indicates statistically significant results ($p < 0.05$).

intermittent triggering could progress to continuous symptoms. The increased volume of the flexor tendon at the proximal part of the A2 pulley might cause wear to the inner surface of the A2 pulley, resulting in pathological changes to the A2 pulley. In the same context, Seradge and Kleinert described that one of their patients with previous A1 pulley release and subsequent flexion deformity of the PIP joint had fusiform enlargement of the flexor tendon at the level of the A2 pulley (Seradge and Kleinert, 1981). Ryzewicz and Wolf also noted that 'distal triggering' appeared to occur in patients with very long-standing disease (Ryzewicz and Wolf, 2006).

The results of the current study indicate that the development of continuous triggering (Froimson Grade II), progression to limitation of motion that needs passive extension (Grade III), and further progression to fixed flexion contracture of the PIP joint (Grade IV) are related, not only to the thickness of the A1 pulley, but also the thickness of the A2 pulley and the flexor tendon under the A2 pulley. Our finding seems to be clinically meaningful, because it implies that the prediction of distal triggering might be possible preoperatively with US examination. In the cases demonstrating a thickened A2 pulley, or increased volume of the flexor tendon under the A2 pulley, residual signs of triggering after A1 pulley release should be carefully evaluated during surgery by asking patients to fully flex the finger. It also could help the surgeon in preoperative counselling of

possible residual triggering after classic A1 pulley release, and in planning of the surgical procedure. It may be thought appropriate to make an oblique incision that can be extended distally to allow resection of the ulnar slip of the flexor digitorum superficialis, or partial release of the proximal part of the A2 pulley, although the latter runs the risk of inducing post-operative bow-stringing of the tendons. Further study to confirm the direct relationship between the increased volume of the A2 pulley-tendon complexes and intraoperative confirmation of distal triggering is warranted. In addition, decisions about further surgical procedures should be correlated with intraoperative findings.

The current study has some limitations. First, we used the same, asymptomatic digit in the contralateral hand of the patient as the control group. There might be subclinical changes in the pulley-tendon structures of other digits in the same patient. Future study, including age and sex matched controls without trigger finger, might be helpful in validating the results of the current study. Second, we assumed other digits of one single patient to represent statistically independent units of analysis. This might cause some bias in statistical analysis. However, many of the results in the current study demonstrated high statistical significance; we consider that this indicates they are probably meaningful, despite this possibility of bias. Third, we did not perform interobserver reliability testing on clinical grading. However, the senior professor responsible for the clinical grading has 26 years of experience in hand surgery, and he has very extensive experience of this condition. Therefore, we consider the risk of bias related to measurement accuracy in clinical grading to be minimal. Fourth, we did not consider the potential influence of sex, age, body mass index, hand dominance, and occupation of the subjects to the thickness of pulley-tendon complexes. In spite of this limitation, our result seems to be clinically relevant and theoretically sound, and could be a cornerstone for future study. Fifth, we did not arrange US examination by two independent observers at different times; we consider that the pulley-tendon structure may change with time and treatment. The method we used to test measurement reliability in the present study does avoid bias related to different observers measuring the distance or cross-sectional area on the image, although there still remains the possibility of bias related to individual differences in the performance skills of US examination. However, the rater who performed all the US examination has 6 years of experience in US examination, and we feel that strict adherence to the measurement protocol minimized the risk of bias.

Finally, the large transducer used for US examination interfered with flexion of the examined finger; the finger could not be fully flexed during the US exam, therefore the diameter of the flexor tendon under the A1 and A2 pulley with finger in full flexion could not be included in the current study. A smaller hockey stick-type transducer might enable assessment of the exact cross-sectional area of the flexor tendon with the finger in full flexion, and the performance of dynamic motion studies to describe the distal triggering at the A2 pulley area. However, an advantage of the 17 MHz high-resolution transducer used in this study is that it enabled us to achieve very clear images of the pulley systems.

In conclusion, the severity of trigger finger seems to be related to not only the thickness of the A1 pulley, but also the thickness of the A2 pulley and the cross-sectional area of the flexor tendon under the A2 pulley. Further studies that investigate the direct relationship between distal triggering and the thickening of the A2 pulley-tendon complexes might provide more clinically relevant information.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Local institutional review board approval in Hanyang University Hospital was obtained for the study.

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