Effect of metacarpophalangeal joint position on A1 pulley and flexor digitorum tendons in trigger digit

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Abstract

Background: The aim of this study was to evaluate and compare the cross-sectional area (CSA) of flexor digitorum tendons and the thickness of first annular (A1) pulleys between contralateral normal digits and trigger digits (TDs) at positions of finger flexion and extension using a noninvasive ultrasound system.

Methods: Seventeen affected fingers of 17 patients (6 men and 11 women) with TDs in one hand, and 17 contralateral normal digits without trigger finger symptoms were examined. The sonographic appearances of flexor digitorum tendons and A1 pulleys were observed at two positions of metacarpophalangeal (MCP) joint flexion: 0° and 60°.

Results: The findings of this study indicate that CSA of flexor digitorum tendons and A1 pulley thickness were significantly larger in both positions of 0° and 60° flexion of MCP joint compared with contralateral normal digits ($p < 0.01$). In TDs, there was a significantly thicker A1 pulley at 60° flexion of MCP joint than that at 0° flexion ($p < 0.01$), but no significant change on CSA of flexor tendons.

Conclusion: Our results suggested that TDs lead to the thicker A1 pulley and larger CSA of the flexor digitorum tendons. The mismatch in volume change between CSA of flexor digitorum tendons and A1 pulley thickness during MCP flexion may lead to the trigger phenomenon.

Keywords: Al pulley; Cross-sectional area; Flexor tendon; Trigger digit; Ultrasound

1. INTRODUCTION

Trigger digit (TD) is a frequent disorder of the hand accompanied by restricted movement of the affected digits at the metacarpophalangeal (MCP) joint.[1–4] The symptom of triggering results in locking, catching, and snapping of affected fingers at the proximal portion of first annular (A1) pulley when the finger moves from flexion to extension.[1–4] The mismatch of the A1 pulley-tendon sheath system might be the mechanism that causes triggering.[3] Although a variety of pathologic factors—idiopathic, intrinsic, and extrinsic—have been proposed,[5–7] the pathologic mechanism of triggering in TD is unclear. Therefore, evaluating the morphology of the A1 pulley-tendon sheath at various positions of finger flexion and extension would be helpful to understand the pathophysiology of TD.

In clinics, ultrasonography is easily accessible and is noninvasive; it also provides good visualization of the A1pulley-tendon sheath system in the fingers.[8–14] Clinical findings, eg, tenderness, triggering phenomena, and palpation on nodules at the level of the A1 pulley, and a history of TD are common criteria for diagnosing TD.[15] Recently, ultrasonography has been widely applied in TD to evaluate the changes of the A1 pulley and flexor tendon,[9,11,12] and therapeutic effects after a local injection,[16] by measuring the sonographic appearances of the A1 pulley and the flexor digitorum tendons. Ultrasonography is useful for investigating disease severity,[17] possible pathologic mechanisms,[18] and therapeutic effects.

According to the findings of sonography images, the increases of flexor tendon thickness and A1 pulley thickness in trigger fingers have been shown at the level of the MCP joint.[11,19–21] Other studies[11,19–21] indicate that the thickness and elasticity of the A1 pulley and flexor digitorum tendons are related to the severity of TD. The disparity of size of the A1 pulley-tendon sheath system might cause the triggering phenomenon because of the thickened A1 pulley or of the swollen flexor digitorum tendons.[18,22] However,

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the association between the ultrasound (US) appearances of the A1 pulley and flexor digitorum tendons remains unknown at positions of finger flexion and extension in TD. Evaluating the sonographic appearance of the A1 pulley-sheath system in TD at positions of finger flexion and extension might help us understand the pathophysiology of TD. Therefore, the aim of this study is to evaluate and compare the cross-sectional area (CSA) of flexor digitorum tendons and thickness of A1 pulley between contralateral normal digits and TDs at positions of finger flexion and extension, respectively, using the noninvasive ultrasonography. We hypothesize that there is a mismatch between the CSAs of the flexor tendons and the A1 pulley thickness during flexion of the MCP joint.

2. METHODS

2.1. Patients

The study protocol was approved by the National Cheng Kung University Institutional Review Board (IRB: A-ER-103-096). Written informed consent was obtained from all patients. Consecutive adult patients with idiopathic TD were enrolled from January 2015 to December 2015. Inclusion criteria were a clinical diagnosis of a single TD on physical examination, no other affected digit, or a history of TD in other fingers. Exclusion criteria were trigger thumb, TD with Froimson Grade 4,[2] bilateral TD, the affected finger with a history of trauma, surgery, local injection, dialysis, gouty arthritis, rheumatoid arthritis, or carpal tunnel syndrome. Finally, 17 affected fingers from 17 patients (6 men and 11 women; average age: 49.0 ± 7.2 years old) with TD in one hand (10 affected fingers in dominant hands) were examined in this study. Seventeen corresponding normal digits (without TD) on the contralateral side were controls. The TDs included four index fingers, nine middle fingers, and four ring fingers. The average duration of TD symptoms was 11.4 ± 7.4 months. The severity of TD was clinically graded in accordance with the Froimson classification. Four patients had Grade 2 and 13 had Grade 3 TD. Five patients (29%) had comorbid hypertension and five had comorbid diabetes mellitus (Table 1).

2.2. Ultrasound examination and measurements

The hand and forearm were positioned with the palmar surface facing upward (full supination) and the wrist joint in the neutral position. A US linear-array transducer (5-10 MHz, SonoSite, Inc., Bothell, WA, USA) set at 10 MHz was placed with minimal pressure at the level of the MCP joint. The US examination began with the longitudinal view. The MCP joint, the flexor digitorum tendons, and the volar plate were clearly identified. At the center of the MCP joint, the transducer was switched to the transverse view of the long axis of the flexor tendons (Fig. 1). The thickness of the A1 pulley was defined as the distance between the inferior and the superior of the A1 pulley, including the hyperechoic area.[14] The maximum thickness of the A1 pulley above the MCP joint was recorded and was usually near the peak of the curve of the metacarpal head. The location was not fixed because the position of the thickest part of the A1 pulley varied.[18] At the same section for maximum thickness of the A1 pulley, the CSAs of flexor digitorum tendons were manually traced in the transverse view using ImageJ software (National Institutes of Health, Bethesda, MD, USA) along the rim of the flexor digitorum tendons in the hyperechoic area. The thin layer between the hyperechoic A1 pulley and hyperechoic flexor digitorum tendons is the synovial fluid space, which shows the hypoechoic area.[18] The ultrasonographic appearances of the flexor digitorum tendons and the A1 pulley were observed at two positions of flexion of the MCP joint: 0° (Fig. A, B) and 60° (Fig. C, D). The finger was examined using a custom-made fixture to maintain 60° flexion of the MCP joint. TDs in the affected hand and normal digits in the contralateral hand were examined in the axial view by an orthopedic surgeon, who was blinded to patient information and who had >8 years of experience working with musculoskeletal ultrasonography. All the US measurements were repeated during the examination session and the results were averaged. Intraobserver agreement was 0.91.

Table 1

Demographics of patients (n = 17)

Variable	Value
Number of patients, n (male:female)	17 (6:11)
Age, years (mean ± SD)	49.0 ± 7.2
Affected digit in dominant hand, n	10
History of systemic disease and upper extremity, n	
Gout[a]	0 (0%)
Hemodialysis[a]	0 (0%)
Hypertension	5 (29%)
Diabetes mellitus	5 (29%)
Rheumatoid arthritis[a]	0 (0%)
Carpal tunnel syndrome[a]	0 (0%)
Involved digits, n	
Index finger	4 (24%)
Middle finger	9 (53%)
Ring finger	4 (24%)
Froimson grade, n	
Stage 2	4 (24%)
Stage 3	13 (76%)
Duration of symptoms, months, n	11.4 ± 7.4

[a]Exclusion criterion.

2.3. Statistical analysis

The Wilcoxon signed rank test was used to assess differences in ultrasonographic appearances because our sample was small. Significance was set at $p < 0.05$.

3. RESULTS

3.1. TDs vs normal digits

The CSA of flexor digitorum tendons at 0° flexion of the MCP joint was 0.24 ± 0.05 cm^2 in TDs and 0.19 ± 0.06 cm^2 in normal digits. The CSA at 60° flexion of the MCP joint was 0.25 ± 0.05 cm^2 in TDs and 0.19 ± 0.06 cm^2 in normal digits. The CSA of flexor tendons in TD was significantly larger than those in normal digits at 0° and at 60° flexion of the MCP joint ($p < 0.01$; Table 2). The A1 pulley thickness at 0° flexion of MCP joint was 0.78 ± 0.20 cm in TD and 0.21 ± 0.10 cm in normal digits, and that at 60° flexion of MCP joint was 0.91 ± 0.21 cm in TD and 0.22 ± 0.10 cm in normal digits. The A1 pulley in TD was also significantly thicker than that in normal digits at either 0° or 60° flexion of MCP joint ($p < 0.001$; Table 3).

3.2. The effect of MCP posture

There were no significant differences in the CSAs of flexor digitorum tendons between 0° and 60° flexion of the MCP joint in TD or normal digits (Table 2). However, in TDs, the A1 pulley was significantly ($p = 0.006$) thicker at 60° flexion than at 0° flexion. For the normal digits, there were no significant changes in A1 pulley thickness between 0° and 60° flexion (Table 3).

4. DISCUSSION

We evaluated the effect of finger positions on the ultrasonographic appearances of soft tissue at the level of the MCP joint in TDs and compared the differences between TDs and

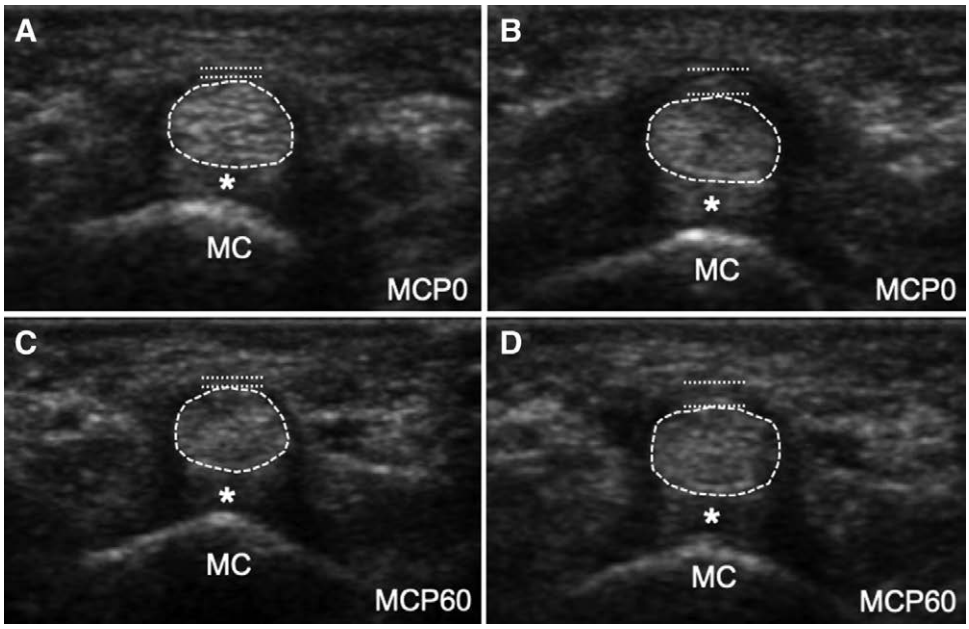

Fig. 1 The ultrasound examination began with the longitudinal view, and followed by the transverse view in the 0º flexion of the metacarpophalangeal (MCP) joint (MCP0, A and B). Then the examination was repeated in 60º flexion of MCP joint (MCP60, C and D) hold with a custom-made fixture. The difference of the A1 pulley thickness (dotted line) and the cross-sectional area of the flexor digitorum tendon (area circled by the dash line) between normal digits (A and C) and trigger digits (B and D) were clearly demonstrated. *: volar plate; MC, metacarpal head.

Table 2

Ultrasound measurements of the cross-sectional area of the flexor digitorum tendons

	MCP0	MCP60	*p*
Normal digits, cm^2	0.188 ± 0.057	0.189 ± 0.057	0.180
Trigger digits, cm^2	0.240 ± 0.048	0.253 ± 0.051	0.067
p	0.008[a]	0.003[a]	

[a]Significant difference between normal and trigger digits.
Values are presented as the mean ± SD.
MCP = metacarpophalangeal; MCP0 = 0º flexion of the MCP joint; MCP60 = 60º flexion of the MCP joint.

Table 3

Ultrasound measurements of the thickness of the A1 pulley

	MCP0	MCP60	*p*
Normal digits, cm	0.206 ± 0.103	0.218 ± 0.101	0.157
Trigger digits, cm	0.782 ± 0.198	0.900 ± 0.212	0.006
P	<0.001[a]	<0.001[a]	

[a]Significant difference between normal and trigger digits.
Values are presented as the mean ± SD.
MCP = metacarpophalangeal; MCP0º = 0º flexion of the MCP joint; MCP60º = 60˚ flexion of the MCP joint.

contralateral normal digits at the 60º and 0º MCP joint flexions. We found that TDs had significantly larger flexor digitorum tendon CSAs and significantly thicker A1 pulleys at the 0º and 60º flexion positions than did the contralateral healthy digits. Moreover, during flexion of the MCP joint to 60º, the thickness of the A1 pulley increased, but the CSAs of the flexor digitorum tendons did not in TD. The phenomenon was not observed in normal digits.

The repetitive forceful compression and friction within the soft tissue might be the cause to induce the thickened A1 pulley or swollen flexor digitorum tendons and to further constrict the proximal portion of flexor digitorum tendons gliding at the base of the trigger finger.[3] There is support for the hypothesis that TD is a type of tendinosis[23] or stenosis of the A1 pulley.[24] Ultrasonographic transverse examinations of TDs in the neutral position show thicker A1 pulleys and larger flexor digitorum tendon CSAs than in contralateral normal digits.[9,18,22,24] The larger CSAs might be ascribable to collagenase degeneration and synovial proliferation changes.[15]

The A1 pulley, a dense arciform connective tissue, is important for stabilizing flexor digitorum tendons by encircling them and closing the phalanges, which prevents bowstringing during finger flexion.[25,26] Even cadaver studies[14] have reported that the A1 pulley should be the hyperechoic soft tissue over the hypoechoic synovial fluid space, but there is no consensus about the ultrasound measurement of A1 pulley thickness, ie, the hypoechoic bundle over the rim of the flexor digitorum tendons[12,26] or over the hypoechoic synovial fluid space.[14,27] However, previous studies[6,12,18,20,22,24,28] are consistent in their claims that TDs have significantly thicker A1 pulleys than do contralateral normal digits. TDs, but not contralateral normal digits in our study, showed consistently significant increases in flexor digitorum tendon CSAs and A1 pulley thickness at both flexion positions. Thus, ultrasonography was used to confirm clinically diagnosed TD and to monitor its response to therapy.[16]

Triggering usually occurs in the proximal portion of the A1 pulley during finger extension.[1–4] The continuous and dynamic evaluation of A1 pulley thickness and the CSA of flexor digitorum tendons would be helpful for understanding the pathophysiology of TD, but is impossible in ultrasound evaluation. Therefore, some studies have evaluated the changes in A1 pulleys and flexor tendon CSAs in certain finger positions.[18,22] Chuang et al[18] reported that the CSAs of flexor digitorum tendons and the A1 pulleys in TDs are significantly thicker in three different finger postures than they are in the contralateral normal digits in the transverse view. Sato et al[22] claimed that the A1 pulleys in TDs are thicker and the flexor tendon CSAs are larger than

are those in contralateral normal digits in either the neutral or in the hook grip position. In the full flexion of interphalangeal joints (hook grip position), the anteroposterior CSA area of flexor tendons is significantly larger than in the neutral position. However, there is no significant difference in A1 pulley thickness between the two positions. We focused on the position change in the MCP joint. The great mismatch between flexor tendon CSAs and A1 pulley thickness during MCP flexion might be one mechanism for triggering. The thicker A1 pulley in TDs might generate more stress, friction, and blockage during finger flexion than during extension, which, in turn, might affect the larger CSAs of the flexor digitorum tendons, and the loss of smooth tendons might prevent them from easily gliding.

4.1. Limitations

This study has limitations. First, our sample is small, but the differences between our comparison groups are significant, which suggests that the sample is large enough to test our hypothesis. Second, we did not enroll patients with Froimson grade 4 TDs because a US examination cannot be correctly done in the fixed flexion position. Third, we focused only on the position of the MCP joint because other studies examined a variety of interphalangeal joints. More than 60° flexion of the MCP joint prevents correct placement of the US probe; therefore, we did not evaluate that position.

In conclusion, TDs led to thicker A1 pulleys and to larger flexor digitorum tendon CSAs than did the contralateral normal digits. During flexion of the MCP joint to 60°, the A1 pulley thickness increased, but the CSAs of the flexor digitorum tendons did not. The mismatch between flexor digitorum tendon CSAs and A1 pulley thickness during MCP flexion might be a triggering mechanism.

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