

ORIGINAL REPORT

THICKENING OF FINGER EXTENSOR TENDONS IN AFFECTED HANDS AMONG PATIENTS WITH STROKE: PREVALENCE AND SONOGRAPHIC FEATURES

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Objective: To evaluate the prevalence and sonographic characteristics of thickened finger extensor tendons in affected hands of patients with stroke.

Design: Observational survey.

Subjects: Thirty-six patients (12 women, 24 men) admitted for rehabilitation because of stroke. Control group: 30 right-handed healthy volunteers.

Methods: We used an 8–16 or a 6–12 MHz sonographic transducer to measure tendon thickness of the extensor digitorum communis of the middle (EDCiii) and ring (EDCiv) fingers at the wrist level.

Results: The thickness of EDCiii (1.79 mm, standard deviation (SD) 0.56) and EDCiv (2.31 mm, SD 0.63) tendons of affected hands were significantly greater than those of the contralateral hands (EDCiii: 1.31 mm (SD 0.23), EDCiv: 1.59 mm (SD 0.46), $p < 0.0001$). When the upper limits of side-to-side tendon thickness differences were set at mean value plus 3 times SD among healthy volunteers (EDCiii: 0.60 mm, EDCiv: 0.74 mm), 18 (50%) patients and no volunteers had tendon thickening. No clinical features other than hand weakness correlated with tendon thickening.

Conclusion: Thickening of finger extensor tendons of the affected hands is common among stroke patients. It should be considered in the differential diagnosis of swelling of the dorsal wrists and hands in stroke-affected upper limbs.

Key words: stroke, tendon, ultrasonography.

J Rehabil Med 2010; 00: 00–00

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Submitted November 21, 2009; accepted July 1, 2010

INTRODUCTION

Swelling of the affected hand is a common and important clinical problem among patients with stroke. The major differential diagnoses are oedema and complex regional pain syndrome or reflex sympathetic dystrophy (1, 2). To the best of our knowledge, the thickening of finger extensor tendons that causes swelling of the dorsal aspect of the stroke-affected wrist and hands had not been reported previously. In the rehabilitation ward, the authors have frequently noted painless swelling at

the dorsal aspect of the hemiplegic wrist in stroke patients. Preliminary soft tissue sonography (performed between 2002 and 2003) showed that the swelling could be ascribed to thickening of the finger extensor tendons of the fourth dorsal extensor compartment between the proximal carpal and mid-metacarpal levels. The affected tendons were up to 3 times as thick as the counterparts in the unaffected hands. These causal observations led us to undertake a more systematic examination of the possibility that thickening of finger extensor tendons may be prevalent in weakened or paralysed upper limbs in stroke patients. Considering that the major causes of finger extensor tendon thickening include rheumatoid arthritis and overuse (3–5), thickening of the extensor tendon in the affected hands in stroke patients is counterintuitive. The affected hands of patients with stroke are prone to disuse or immobilization, which is related to atrophy and decreased mechanical strength of the tendons and ligaments (6, 7). Oedema in the affected hands may make it difficult to detect tendon thickening; thus high-resolution ultrasonography (8) is helpful for clinicians to evaluate tendon morphology in the oedematous hands.

To shed new light on this largely unrecognized, but frequently observed, phenomenon of finger extensor tendon thickening in patients with stroke, we conducted the present study to establish the criteria for tendon thickening and to delineate its prevalence among stroke patients in a rehabilitation ward, and its sonographic and clinical characteristics.

METHODS

Subjects

This study involved a group of stroke patients who were admitted to the rehabilitation ward of the China Medical University Hospital for rehabilitation after acute stroke care between January 2004 and June 2006. Only patients without a history of previous stroke or other disorders of the central nervous system were included. Exclusion criteria were: those who had been previously afflicted by conditions that could influence the morphology or function of the upper limbs before the onset of stroke, including fracture, severe arthritis, gout and poliomyelitis. Recruitment of patients was interrupted 4 times because of problems in availability of the ultrasound instruments; a total of 36 patients (12 women and 24 men, mean age 58.4 years, standard deviation (SD) 16.7) were finally included in this study. The interval between stroke onset and the sonographic evaluation ranged from 18 to 173 (average 55.7 (SD 30.6)) days. Thirty healthy volunteers who were similar in mean age (56.2 years (SD 11.3)) and sex distribution (10 women and 20 men) to the patients were recruited as a control

group to help establish the criteria for tendon thickening. The study protocol was approved by the Institutional Review Board of the China Medical University Hospital. Informed consent was obtained from all subjects or their legal guardians.

Data collection

Clinical information on the patients, including age and sex, handedness, clinical diagnosis, motor status of the stroke-affected hands, the presence and severity of spasticity, and the presence of pain or oedema in the affected hand or wrist, was obtained through history-taking and review of the medical records.

Sonographic examination

We used an 8–16 MHz variable-frequency linear array transducer of Diasus system in 2004 and 2005, and a 6–12 MHz linear array transducer of LOGIQ 5 Pro in 2006 to examine the soft-tissue morphology of the dorsal aspect of the wrists and hands of the subjects. All sonographic examinations were performed by the same physician (the first author). Because of the complexity and anatomical variations of finger and wrist extensor tendons (8, 9), it is difficult to describe their morphology in detail using two-dimensional sonographic images. Our preliminary experience showed that the site of the most prominent tendon thickening among these patients was between the proximal carpal and mid-metacarpal levels, thus we measured the thickness of the extensor tendons of the middle and ring fingers at this site. Tendon thickness was measured as follows:

- *Positioning*: the wrist was placed in neutral position, supported by a soft mattress. All metacarpophalangeal and interphalangeal joints were placed in 0 degree flexion, taking care to avoid radial or ulnar deviation of the wrist and all fingers.
- *“Reference point” (RP)*: the transducer was positioned over the distal radio-ulnar joint. The most distal portion of the radius and the most proximal part of the dorsal radio-carpal ligament were visualized in the sonogram. A cross-sectional view of the fourth extensor compartment was then obtained. We designate this point as the RP.
- *“Measurement point” (MP)*: a ruler was placed next to the transducer and parallel to the third metacarpus. Using the ruler as a guide, we moved the probe 3 cm distally. This point was called the MP. We then fine-tuned the transducer placement and angle to obtain a cross-sectional image of the extensor tendons of the middle and ring fingers, and to optimize (maximize) the brightness of the tendon image so as to minimize the influence of anisotropy on measurement of tendon thickness. We used sufficient gel and the minimal pressure required to maintain good contact between the transducer and the skin.

The thickness of the tendons of the extensor digitorum communis of the third (EDCiii) and fourth (EDCiv) fingers at the MP was measured perpendicular to the width of the cross-sectional sonographic image of the tendons. The hands of all the patients and volunteers were examined using the method described above.

Statistical analysis

Student’s *t*-test was used to compare the mean thickness and side-to-side thickness differences and ratios of extensor tendons of the hands of all subjects. We used χ^2 and Fisher’s exact tests to examine the correlation between the presence of tendon thickness and other clinical variables. Microsoft Access 97, Excel 97 and SPSS for Windows 9.0 were used for data collection and statistical analysis. The level of significance was set at $p < 0.05$.

RESULTS

Tendon thickness comparisons and criteria for tendon thickening

Among the healthy volunteers, there was no significant side-to-side difference in thickness of the EDCiii and EDCiv at

Table I. Tendon thickness at the measure point

	Tendon thickness (mm)	
	EDCiii	EDCiv
	Mean (SD)	Mean (SD)
Volunteers ($n=30$)		
Right hand	1.29 (0.19)	1.53 (0.30)
Left hand	1.35 (0.25)	1.58 (0.38)
<i>p</i> -value	0.33	0.58
Patients with stroke ($n=36$)		
Stroke-affected hand	1.79 (0.56)	2.31 (0.63)
Non-affected hand	1.31 (0.23)	1.59 (0.46)
<i>p</i> -value	<0.0001	<0.0001

Student’s *t*-test. SD: standard deviation; EDCiii: extensor digitorum communis of the third finger; EDCiv: extensor digitorum communis of the fourth finger.

the MP (Table I). The average absolute values of side-to-side thickness differences of the EDCiii and EDCiv at the MP were 0.15 mm (SD 0.15) and 0.21 mm (SD 0.19), respectively. In contrast, the tendon thickness of the EDCiii and EDCiv of the stroke-affected hands (1.79 mm (SD 0.56) and 2.31 mm (SD 0.63), respectively) were significantly greater than their counterparts in the non-affected hands at the MP (EDCiii: 1.31 mm (SD 0.23), EDCiv: 1.59 mm (SD 0.46), Student’s *t*-test, $p < 0.0001$ for EDC iii and EDCiv). The tendon thickness ratios of the stroke-affected hand to contralateral hand in the patient group were significantly greater than the side-to-side tendon thickness ratios of the volunteers (Student’s *t*-test, $p < 0.0001$ for EDCiii and EDCiv, Table II). We defined the upper limit of “normal” side-to-side tendon thickness differences for both the EDCiii and EDCiv, measured at the MP, as the mean absolute value plus 3 times the SD of thickness differences of the relative tendons of the 30 healthy volunteers. The upper limits were 0.60 mm for the EDCiii and 0.74 mm for the EDCiv. According to the above criteria, none of the volunteers had any “thickened” tendons. Among the patients with stroke, 10 (28%) of the 36 EDCiii tendons and 17 (47%) EDCiv tendons of the affected hands were thickened, and 18 (50%) patients had at least one finger extensor tendon that was thickened at the MP. The thickened tendons were 1.4–3.2 times as thick as their counterparts in the contralateral hands. The width of tendons

Table II. Side-to-side tendon thickness ratios

	Range	Median	Mean (SD)
EDCiii			
Volunteers: right/left hand	0.67–1.27	1.00	0.97 (0.14)
Patients: stroke-affected/non-affected hand	0.7–3.2	1.33	1.38 (0.43)
			Student’s <i>t</i> -test, $p < 0.0001$
EDCiv			
Volunteers: right/left hand	0.75–1.50	1.00	0.99 (0.18)
Patients: stroke-affected/non-affected hand	0.8–2.7	1.46	1.53 (0.52)
			Student’s <i>t</i> -test, $p < 0.0001$

SD: standard deviation; EDCiii: extensor digitorum communis of the third finger; EDCiv: extensor digitorum communis of the fourth finger.

Table III. Tendon width of patients (n = 36), at the measure point

	Tendon width (mm)		p-value
	Stroke-affected hand	Non-affected hand	
EDCiii	Mean (SD) 4.55 (0.81)	Mean (SD) 4.39 (0.71)	0.60
EDCiv	Mean (SD) 4.70 (0.99)	Mean (SD) 4.57 (0.88)	0.44

Student's *t*-test. SD: standard deviation; EDCiii: extensor digitorum communis of the third finger; EDCiv: extensor digitorum communis of the fourth finger.

at the MP of the stroke-affected hands did not differ from that in the contralateral hands (Table III). Fig. 1 is a representative sonogram of thickened tendons.

Clinical and sonographic features

The mean interval between stroke onset and the sonographic evaluation did not differ between the patients with (58.9 days (SD 22.4)) or without (52.5 days (SD 37.6)) thickened tendon(s). There was a non-significant trend for patients with hand oedema to develop tendon thickening (Table IV, 8/12, 66%, $p=0.16$). Three of the patients reported pain in the stroke-affected hand (and shoulder) under the impression of shoulder–hand syndrome. These 3 patients had thickened finger extensor tendons. The other 15 patients with tendon thickening

Table IV. Tendon thickening and clinical variables

Variables	Number of patients		p-value
	With tendon thickening (n = 18)	No tendon thickening (n = 18)	
Sex			
Women	6	6	
Men	12	12	1.0*
Nature of stroke			
Haemorrhagic	11	7	
Ischaemic	7	11	0.18*
Handedness			
Right	17	16	
Left	1	2	0.38†
Brunnstrom stage of hand			
I	2	0	
II	9	6	
III	4	8	
IV	2	3	
V	1	1	0.39*
Upper limb spasticity			
Present	11	9	
Absent	7	9	0.50*
Stroke-affected hand oedema			
Present	8	4	
Absent	10	14	0.16*

* χ^2 test. †Fisher's exact test.

did not report pain in the hand. None of the 18 patients had tenderness over the thickened tendons. There was no significant correlation between the presence of tendon thickening and age, sex, stroke types (haemorrhagic or ischaemic), handedness, or motor status of the stroke-affected hand in terms of Brunnstrom stage and spasticity based on χ^2 and Fisher's exact tests (Table IV). While tendon thickening frequently developed in the finger extensor tendons of the fourth dorsal extensor compartment, we seldom observed this phenomenon in other finger or wrist extensor tendons. The location of the most prominent tendon thickening was between the proximal carpal and mid-metacarpal levels. Sonographically, the echogenicity of the thickened tendons was slightly decreased compared with normal tendons. The thickness of hypoechoic synovial tissue surrounding the thickened tendons was also increased, rendering a sonographic picture similar to that of a tenosynovitis. However, no obvious fluid accumulation was noted in the synovial sheath.

DISCUSSION

In the present study, thickening of finger extensor tendons of the affected hands is a common clinical observation among stroke patients in the rehabilitation ward, with a prevalence of 50%. This phenomenon has not been reported previously in the literature, possibly for the following reasons: (i) before soft-tissue sonography became widely accessible, physicians did not have a simple, non-invasive method to evaluate the cause of a swollen dorsal wrist; (ii) the thickened tendons are typically not painful nor tender, thus physicians were unlikely to arrange invasive or expensive examinations such as biopsy or magnetic

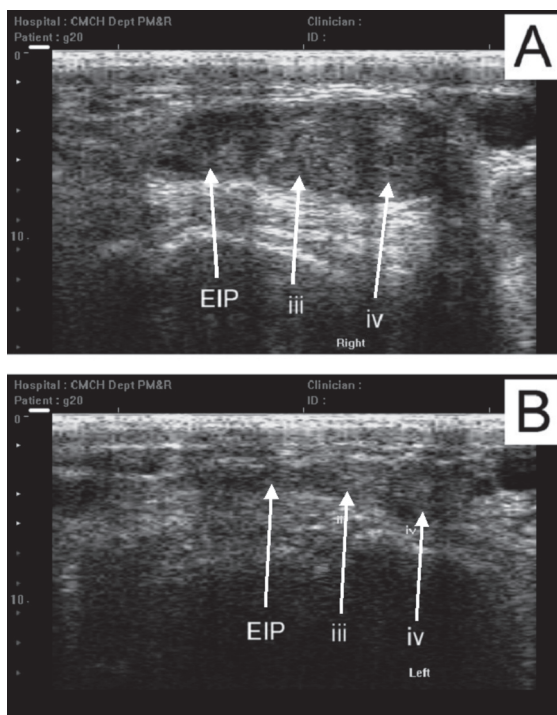


Fig. 1. Cross-sectional sonography at “measurement point” of the dorsal wrists of a 66-year-old woman (code g20) with left middle cerebral artery infarction, 103 days post-onset. (A) The stroke-affected right hand (Brunnstrom stage II). The extensor tendons of the fourth compartment were prominently thickened. (B) The left hand. EIP: extensor indicis proprius. EDCiii/ EDCiv: extensor digitorum communis of the middle/ ring fingers.

resonance imaging to investigate the cause of the swelling; and (iii) oedema of the stroke-affected hand could impede inspection and palpation and makes detection of thickened tendons difficult. Though a higher percentage of patients who had oedema of the stroke-affected hand developed tendon thickening (67%, 8/12) than those without an oedematous hand (42%, 10/24), the difference did not reach statistical significance, probably because of a relatively small sample size. Since our patients apparently developed tendon thickening regardless of the severity of motor involvement or the presence of spasticity, it is reasonable to suggest that a minor weakness of the hand is sufficient to induce thickening of finger extensor tendons. In fact, except for the stroke-induced hand weakness *per se*, we failed to find a risk factor “predicting” the development of tendon thickening. Among our patients who developed tendon thickening, the shortest onset-to-sonography interval was 30 days. Four patients underwent sonography within 30 (between 18 and 22) days post-stroke, and none of them had tendon thickening. It is possible that if we examined these patients for a longer period, some of them might be found to have tendon thickening. We did not use sonography to follow those patients who did not have tendon thickening at first evaluation; thus, the prevalence of finger extensor tendon thickening among our patients might in fact be underestimated. By following 3 of the patients for up to 2 years, we found that the degree of tendon thickening was most prominent between 3 and 6 months after stroke. Though the sonographic appearance of the thickened tendons and surrounding synovial tissue appeared similar to that of a tenosynovitis, there are features that help differentiate these two conditions: (i) most of our patients with tendon thickening were without pain or tenderness at the site of thickened tendons when sonography was taken; (ii) none of the patients in this study had a history of tendon overuse; and (iii) we never detected fluid within the synovial sheath as is seen in tenosynovitis (8).

We defined “tendon thickening” by the side-to-side difference and not simply the upper limit of tendon thickness (e.g. mean thickness + 3 times SD) for the following reasons: (i) there is great interpersonal variation in tendon thickness (some of the healthy volunteers had tendons more than twice as thick as others), which makes the “upper limit of tendon thickness” criteria insensitive to detect tendon thickening for patients with relatively thinner tendons; (ii) it is the difference in thickness between the two hands that gives the appearance of swelling of the dorsal aspect of the stroke-affected hands.

According to our criteria, none of the volunteers had any thickened tendons and 50% of patients had at least one thickened finger extensor tendon. The side-to-side thickness difference criteria that were used in the present study to define tendon thickening are both specific and sensitive.

The finger extensor muscles of the stroke-affected hands are usually weak or paralysed, and the resulting disuse or immobilization would be expected to cause tendon atrophy rather than thickening (6, 7). Thus, it is highly unlikely that “overuse” is the cause of tendon thickening among our patients. We propose 4 probable mechanisms to explain the thickening of finger extensor tendons, as follows.

- Wrist drop for extended period leads to increased intra-tendinous stress. The distal row of carpal bones and the bases of the metacarpal bones make an arch that bulges toward the dorsal side (9). When the wrist is kept in a flexed position, the extensor tendons of the fourth compartment could be seen as being taut at the top of a dome. Thus these fourth-compartment tendons receive greater longitudinal tensile stress and vertical compressive stress (perpendicular to the contact surface between the tendons and the carpal bones) than the tendons in the other compartments. An increased intra-tendinous stress could probably induce changes in the amount and quality of protein synthesis in the tendon, and the tendon morphology changes as a result (7, 10). We suggest that keeping the affected wrist in the neutral position would probably help prevent or improve the thickening of finger extensor tendons.
- Flexor muscle spasticity is increased in the stroke-affected upper limb. This persistent increase in flexor tone probably enhances the abnormal stress in the finger extensor tendons. However, thickening of finger extensor tendons was noted in 7 patients who did not have spasticity. Thus spasticity is obviously not necessary for the development of finger extensor tendon thickening.
- It is possible that minor injury to the finger extensor tendons could be induced by inappropriately executed range-of-motion exercise or stretching exercise. This is a less likely mechanism because flexor muscle tone is predominantly increased in the stroke-affected upper limbs. Accordingly, stretching of those extensor tendons is seldom emphasized in clinical practice.
- Minor tendon injury is induced by manual massage, which is commonly administered by caregivers for stroke patients.

A limitation of this study is that patients were examined only once, and thus the natural course of tendon thickening was not determined. This could be overcome by periodic sonographic examinations soon after stroke onset. Although a blinded research design is ideal to reduce observer’s bias, it is technically difficult to keep the sonographic examiner blinded to the affected hands. Manual contact with a patient’s hands in positioning and sonographic examination processes will lead the examiners to notice the abnormal muscle tone and/or weakness of the stroke-affected hand. The magnitude of difference in tendon thickness in this study based on objective and quantifiable data is likely to eliminate observer’s biases. Since the present study is directed at side-to-side difference in tendon thickness, the impact of anthropometric predisposition and activity profiles of the patients and volunteers is likely to be negligible. We only measured the thickness of the EDCiii and EDCiv at the MP, thus we did not present a detailed description of the dimensions of all the finger and wrist extensor tendons. Finally, our study does not provide histological features that underlie thickening of finger extensor tendons in the stroke-affected hands. Further studies are needed to address these shortcomings.

In conclusion, thickening of finger extensor tendons of the affected hands is a common clinical phenomenon among patients with stroke. This should be considered in the differential

diagnosis of swelling at the dorsal aspects of the wrists and hands of these patients. The upper limits of normal side-to-side tendon thickness differences could be set at 0.60 mm for the EDCiii and 0.74 mm for the EDCiv.

ACKNOWLEDGEMENTS

The authors would like to express their appreciation to Chung-Yi Hsu, MD, PhD and Tyng-Guey Wang, MD for their critical reviews.

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