

Sonographic Appearance of the Flexor Tendon, Volar Plate, and A1 Pulley With Respect to the Severity of Trigger Finger

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Purpose To evaluate trigger digits with sonography to determine morphological changes in the A1 pulley, flexor tendon, and volar plate in relation to the severity of triggering.

Methods We evaluated 67 trigger digits and graded them into 1 of 4 groups. We compared the groups according to severity and to contralateral fingers, which served as controls.

Results The thickness of the flexor tendons under the A1 pulley was proportional to the severity of triggering. The anteroposterior thickness of the flexor tendon increased significantly among the grades exhibiting triggering regardless of the affected digit. However, in digits other than the thumb, tendon thickness increased even in the absence of active triggering. Thickening tended to be greater with finger flexion. The A1 pulley exhibited the greatest thickness and the volar plate exhibited significant thickening in the group that exhibited continuous triggering that was easily reduced with active extension (grade III).

Conclusions The flexor tendon thickened significantly before patients experienced triggering except in the thumb. In the thumb, the flexor tendon and A1 pulley thickened significantly only after patients exhibited triggering. Thickening of the volar plate appears to have an important role in continuous triggering. Although most clinicians can easily determine the severity of a trigger digit by clinical examination, ultrasound might be helpful for objectively understanding the severity and response to treatment, by examining the thickness of the flexor tendon and A1 pulley. In particular, sonographic measurement of the A1 pulley might be useful in judging the progression of trigger finger severity. In cases where a Doppler signal is detected inside the A1 pulley, more conservative therapies might be worth considering before surgery. (*J Hand Surg* 2012;37A:2012–2020. Copyright © 2012 by the American Society for Surgery of the Hand. All rights reserved.)

Type of study/level of evidence Diagnostic III.

Key words A1 pulley, flexor tendon, trigger finger, ultrasonography, volar plate.

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THE PATHOPHYSIOLOGY OF trigger finger is not fully understood. Stenosing tenosynovitis at the level of the first annular (A1) pulley apparently creates a mismatch between the volume of the flexor tendon sheath and the tendon.¹

Ultrasound provides noninvasive visualization of the soft tissues. Normal flexor tendons have multiple parallel hyperechogenic lines regularly running inside the tendon, described as a fibrillar pattern.² Annular pulleys appear as predominantly hyperechogenic structures relative to the adjacent flexor tendons. However, pulleys

can appear hypoechogenic, similar to ligaments, because of anisotropy. Anisotropy is seen in tissues composed of linearly arranged compact bundles of fibers that act as reflectors of sound. The echogenicity of these tissues changes with the angle of incidence of the sound beam and can appear falsely hypoechogenic when the beam is not perpendicular to the interrogated structure. In a study using a transducer operated at the high frequency of 17 MHz, Boutry et al³ observed the A1, A2, and A4 pulleys as hyperechogenic structures prone to anisotropy. The volar plate of the metacarpophalangeal (MP) joint is fibrocartilage and can be clearly observed as an isoechogenic and homogeneous structure underlying the flexor tendon.

Guerini et al⁴ sonographically examined 33 fingers with painful triggering. They reported thickening and hypoechogenicity of the A1 pulley in all fingers, and saw hypervascularization of the A1 pulley on power Doppler imaging in 91% of the fingers. Thickening and hypervascularization of the A1 pulley was also observed to be a hallmark of trigger finger on ultrasound. That study suggested that thickening of the A1 pulley is a primary development in the pathogenesis of trigger finger. Sampson et al⁵ stated that primary thickening of the A1 pulley is an underlying factor in the blockage and restriction of tendon excursion, and the authors attributed these not only to A1 pulley changes, but also morphologically to tendon changes. Kim and Lee⁶ emphasized that some trigger fingers exhibit sonographic abnormalities of the tendon and its sheath, but without abnormalities in the A1 pulley.

The purposes of this study were to evaluate trigger finger and analyze its sonographic characteristics in relation to clinical severity by applying our clinical grading system.

MATERIALS AND METHODS

Over a 6-month period, we recruited consecutive patients clinically diagnosed with trigger finger in our clinic. Exclusion criteria included rheumatoid arthritis, dialysis treatment, local steroid injection within the past 6 months, and fingers with a history of local gouty or pyogenic disease or major hand trauma. We eliminated patients with small finger triggering because of difficulties in the sonographic evaluation. Plain radiographs were evaluated in all patients. We confirmed that none of the included patients had a history of trauma, tumor, ganglion, calcium deposit, or severe osteoarthritis. In total, a hand surgeon who had 12 years' experience in surgery and 1 year of experience in ultrasound studied 67 affected digits from 53 patients (17 men and 36 women, age 25–80 y; mean age, 61 ± 11 y) sono-

graphically at the level of the MP joint. Nine patients had hyperlipidemia and 5 had diabetes mellitus. All patients with diabetes had the disease well controlled with oral medication, exercise, and caloric restriction. We also analyzed 50 digits on the contralateral side that were judged to be asymptomatic. The examined digits included 26 thumbs and 4 index, 25 middle, and 12 ring fingers. The control group included 21 thumbs and 2 index, 18 middle, and 9 ring fingers. We graded each finger according to clinical findings, which resulted in 4 groups. Grade I represented a vague sense of tightness and tenderness around the MP joint, and patients did not exhibit triggering; grade II represented intermittent triggering; grade III represented continuous triggering with or without interphalangeal (IP) joint contracture and locking reduced with active extension; and grade IV represented continuous triggering with or without IP joint contracture. Grade IV patients required passive assistance to achieve maximal extension and could not completely flex actively.

Tissue measurements

We positioned the probe on the MP joint with minimum pressure and focused sonographic analysis on the A1 pulley, flexor tendon, and volar plate (Fig. 1). We positioned patients' hands in supination with the wrist joint in a neutral position. We observed these tissues in 2 IP joint positions on the sagittal plane and 1 IP joint position on the axial plane with the MP joint fixed in a neutral position (flexion degree of MP joint: thumb, 10° to 15°; other digits, 0°). We positioned the probe perpendicularly against the palmar surface of the examined digit and analyzed tissue measurements separately for the thumbs and other digits. In the index, middle, and ring fingers, we evaluated the flexor digitorum superficialis and flexor digitorum profundus as if there were a single tendon. This was because we were unable to distinguish these 2 tendons sonographically in a static position. We identified the flexor digitorum superficialis and flexor digitorum profundus tendons dynamically for differential motion in advance of the tissue measurements.

Sagittal plane study

While we fixed the probe so as not to change the view, we obtained 2 images: 1 with IP joint extension and the other with IP joint flexion in which the finger was flexed until the tip of the finger touched the probe (Fig. 2). Images were created on a display showing a side-by-side comparison. We made comparisons with each finger on each plane with

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