

Occurrence of Trigger Finger Following Carpal Tunnel Release

Abstract

Surgical treatment of carpal tunnel syndrome (CTS) was recently started in our department, and we noticed that the development of trigger finger (TF), with which neurosurgeons are generally unfamiliar, is not rare after such treatment. We summarized the clinical and pathogenetic aspects of TF and retrospectively analyzed the medical records of all 39 patients who underwent CTR in our department to investigate the occurrence of TF. In 39 patients with CTS, 46 surgical interventions were performed in our department. All surgical procedures were carried out by open release of the transverse carpal ligament under local anesthesia infiltration, but the distal forearm fascia was not released. The mean postoperative follow-up period was 21.1 ± 16.8 months. TF after CTR occurred in nine hands of eight patients (9 of 46 hands, 19.6%). The mean interval between CTR and TF onset was 5.3 ± 2.8 months. TF after surgical treatment of CTS is not rare; therefore, surgeons who treat CTS should understand the clinical features of TF and carefully assess affected patients, particularly at presentation and within 6 months postoperatively.

Keywords: *Carpal tunnel syndrome, surgery, trigger finger*

Introduction

Carpal tunnel syndrome (CTS) is estimated to occur in 1%–4% of the general population and accounts for about 90% of all nerve compression syndromes.^[1-3] Surgical treatment of CTS was recently started in our department, and to date, 46 surgical interventions have been performed in 39 patients with CTS.^[3] Our clinical experience with these cases has shown that the development of trigger finger (TF), also termed stenosing flexor tenosynovitis, is not rare after surgical treatment of CTS, indicating that surgeons who treat CTS should increase their awareness of TF. Both CTS and TF are common diseases that may occur in the same patient, and some authors have reported the concurrence rate between these two conditions; however, the clinical implications of this association have not been fully established.^[4-6] In the present manuscript, we (1) summarize the clinical and pathogenetic aspects of TF because neurosurgeons are generally considered unfamiliar with this disease, (2) retrospectively analyze our surgical cases, and (3) present a literature review of the occurrence of TF after surgical treatment of CTS.

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Summary of Clinical and Pathogenetic Aspects of Trigger Finger

Anatomy related to trigger finger

Finger flexion is finely coordinated by the flexor tendon, which is guided by a tendon sheath and complex pulley system. This pulley system is composed of five annular pulleys (A1 through A5) and three cruciate pulleys (C1 through C3) [Figure 1]. These structures maintain the position of the flexor tendon relative to the bones, and the power of flexion is increased by preventing bowstringing of the flexor tendon. The flexor tendon passes through the tendon sheath, allowing it to glide smoothly when the finger bends and straightens. The tendon passes through the pulleys as the finger moves. The pulley at the base of the finger is called the A1 pulley, which is most often involved in TF. In patients with TF, the A1 pulley is inflamed or thickened, making it difficult for the flexor tendon to glide smoothly as the finger bends.^[7-9]

Clinical symptoms of trigger finger

Patients with TF typically present with stiffness, swelling (mainly in the morning), pain, clicking, catching, and loss of motion of the affected finger. Progression of clinical symptoms is usually divided into Stages

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1–4. In the pretrigger phase (Stage 1), patients usually present with a slow onset of pain that occurs more frequently over time. The pain is aggravated by labor-intensive use of the hand. In the active phase (Stage 2), clicking of the symptomatic finger is recognized, although the patient is able to actively extend the finger. In the passive stage (Stage 3), mismatch between the tendon and flexor sheath diameter proceeds; as a result, the patient cannot extend (Stage 3a) or flex (Stage 3b) the finger without forcing it to straighten using the asymptomatic hand. In Stage 4, the finger remains in rigid flexion [Table 1a].^[10]

Epidemiology of and risk factors for trigger finger

TF most commonly occurs in the fifth-to-sixth decades of life, and the frequency is six times higher in women than men. The lifetime risk of onset of TF is 2%–3%, but it increases to 10% in patients with diabetes.^[7,11] Other risk factors for TF include hypothyroidism, osteoarthritis, rheumatoid arthritis, tuberculosis,^[7,12] and repetitive activities that can strain the hand, such as playing a musical instrument.^[7] The most commonly affected finger is the ring finger, followed by the thumb; however, TF can occur in the other finger as well.^[7,8,13]

Treatment options for trigger finger

Initial management of TF usually involves nonsurgical treatments such as immobilization of the affected finger, nonsteroidal anti-inflammatory medication, and corticosteroid injections into the area of the inflamed tendon.^[7-10] Surgical treatment is indicated when nonoperative treatment fails.

Occurrence of Trigger Finger after Carpal Tunnel Release in our Department

Surgical treatment of CTS was recently started in our department, and to date, 46 surgical interventions have



Figure 1: Anatomy of flexor tendon showing the intricate pulley system that guides finger flexion. The pulley most often affected in trigger finger and subsequently released during open release for trigger finger is the A1 pulley. A – Annular pulley; C – Cruciate pulley

been performed in 39 patients with CTS. The diagnosis was based on a standard history and physical examination and was confirmed by nerve conduction tests.^[3] CTR was carried out under local anesthesia, and a pneumatic tourniquet on the upper arm was used. A longitudinal palmar skin incision about 4 cm was made distal from the wrist crease. This provides a view of the transverse carpal ligament (TCL). The TCL was divided along the ulnar aspect of the median nerve to protect the median motor branches. Internal neurolysis or tenosynovectomy were not performed. When the median nerve was confirmed to be fully decompressed, the tourniquet was deflated and hemostasis was achieved. The skin incision was closed with monofilament nonabsorbable sutures. The medical records of all 39 patients who underwent CTR were examined to investigate the occurrence of TF.

Data are expressed as mean ± standard deviation. The Chi-square test or Fisher’s exact test was used for two-group comparisons of categorical variables. Student’s *t*-test was used to compare the mean ages of two groups. Variables were considered statistically significant at *P* < 0.05.

Of the 39 patients who underwent CTR, 19 were male and 20 were female. The affected side was the right in 16 (41%) patients, the left in 16 (41%), and bilateral in 7 (18%). The mean age of the patients was 70.2 ± 11.8 years. CTS was categorized into three stages based on the clinical symptoms [Table 1b]. Stage 1 comprised 12 hands, Stage 2 comprised 32 hands, and Stage 3 comprised 4 hands. The mean postoperative follow-up period was 21.1 ± 16.8 months.

Cases of TF were defined by the clinical history and/or presence of catching, clicking, or locking and tenderness

Table 1a: Classification of severity of trigger finger 29

| Stage | Characteristics |
|-------------------------------|---|
| 1. Pretriggerer | Pain and tenderness at the level of the A1 pulley; no palpable nodule or triggering |
| 2. Active | Tenderness, swelling, or tendon nodularity with occasional triggering or catching during active movements |
| 3. Passive | Manifestations of Stage 2 with frequent triggering or catching as well as locking of the digit |
| 4. Rigid in a flexion posture | Digit is flexed at the proximal interphalangeal joint |

Table 1b: Clinical stages of carpal tunnel syndrome 20

| Stage | Characteristics |
|-------|--|
| 1 | Sensory disturbance, but no muscle atrophy in the thenar eminence |
| 2 | Sensory disturbance and muscle atrophy in the thenar eminence, but no dysfunction of the opponens pollicis |
| 3 | Sensory disturbance, muscle atrophy in the thenar eminence, and dysfunction of the opponens pollicis |

over the A1 pulley. We found that TF after CTR occurred in nine hands of eight patients (9 of 46 hands, 19.6%). The mean age of the patients who developed TF was 66.7 ± 13.0 years. The mean interval between CTR and TF onset was 5.3 ± 2.8 months [Table 2].

The risk factors for TF after CTR were analyzed [Table 3]. However, we found no significant difference in the mean age of the patients with and without TF (66.7 ± 13.0 vs. 70.2 ± 11.8 years, respectively, $P = 0.36$). We also compared four age groups: >80 years, 79–70 years, 69–60 years, and <59 years. This comparison also showed no significant differences. A categorical comparison was performed by sex, laterality, clinical stage of CTS, and hemoglobin A1c (HbA1c) concentration ($\geq 6.5\%$ or $<6.5\%$). These analyses showed that female patients were significantly more likely to develop TF than male patients (odds ratio, 10.0; 95% confidence interval, 1.15–486). In addition, no significant differences were detected in the clinical stage of CTS, laterality, or HbA1c concentration.

Reports of Concomitant Occurrence of Trigger Finger and Carpal Tunnel Release

CTS and TF are both common diseases and may occur in the same patient.^[4-6] Several studies have investigated the concurrence rate between CTS and TF. In 2001, Garti *et al.*^[5] evaluated 62 consecutive patients with TF but no signs or symptoms of CTS. Nerve conduction studies of the median nerve in 39, of these 62 (63%) patients showed increased distal motor latency. In 2009, Rottgers *et al.*^[6] evaluated 108 consecutive patients presenting with CTS and/or TF. Of these 108 patients, 66 (61%) had evidence of concomitant CTS and TF. In 2009, Kumar and Chakrabarti^[4] reported that 43% of patients presenting with TF also had CTS.

As mentioned above, both conditions exist concomitantly at the time of presentation with a certain probability. Hands with either CTS or TF are conceivably affected by a pathologic condition of the connective tissues that results in the other pathologic condition. However, different histopathologic abnormalities of the connective tissues have been reported between CTS and TF. The main pathology in CTS is noninflammatory fibrosis with vascular hypertrophy and proliferation with wall thickening and obstruction in the subsynovial connective tissue.^[14] However, the main pathology in TF is irregular connective tissue with small collagen fibers and an abundant extracellular matrix containing chondroid matrix in the deep surface of the pulley.^[15] Therefore, CTS and TF are not the result of the same pathologic condition of the connective tissue, and other factors should also be considered to be involved in the mechanism of concomitance of these two conditions.

A limitation of our case analysis is that we did not sufficiently confirm whether TF existed before surgical treatment of CTS. The presence of TF should be carefully assessed at presentation or before surgical treatment of CTS because TF is highly likely to be missed when the symptoms of TF are mild.

Reports of Trigger Finger Occurrence after Carpal Tunnel Release

The development of TF in patients undergoing surgical treatment of CTS has been analyzed by various authors [Table 4].^[16-28] The reported incidence of TF after CTR is about 10%–20%, which is almost identical to the results of our study, and CTR has been attributed to acceleration of the development of TF in various studies.

The exact pathogenesis of TF after CTR is unknown. Some possible explanations include an edematous

Table 2: Summary of nine hands in eight patients who developed trigger finger after carpal tunnel release

| Patient number | Age/sex | Laterality | Clinical stage of CTS | TF | | | | HbA1c (%) |
|----------------|-----------|------------|-----------------------|---------------------------|----------------|------------------------|-----------------------------|-----------|
| | | | | Onset (after CTR), months | Clinical stage | Involved digit | Treatment | |
| 1 | 57/male | Left | 2 | 1.0 | 3 | Thumb | Steroid injection | 5.4 |
| 2 | 78/female | Right | 2 | 7.9 | 2 | Middle and ring finger | Immobilization, analgesics | 5.6 |
| 3 | 51/female | Right | 1 | 5.0 | 3 | Thumb | Steroid injection | 7.6 |
| | 51/female | Left | 1 | 11.4 | 3 | Thumb | Steroid injection | 7.6 |
| 4 | 52/female | Right | 1 | 4.3 | 2 | Middle finger | Immobilization, manalgesics | NA |
| 5 | 73/female | Left | 2 | 6.2 | 2 | Thumb | Immobilization, analgesics | 5.8 |
| | 74/female | Right | 1 | 4.1 | 2 | Thumb | Immobilization, analgesics | 5.8 |
| 6 | 78/female | Left | 2 | 4.9 | 2 | Middle and ring finger | Immobilization, analgesics | NA |
| 7 | 86/female | Right | 2 | 2.9 | 2 | Middle and ring finger | Immobilization, analgesics | 5.2 |

NA – Not available; CTS – Carpal tunnel syndrome; CTR – Carpal tunnel release; TF – Trigger finger; HbA1c – Hemoglobin A1c

Table 3: Univariate analysis of the predictors of trigger finger occurrence

| | TF | | OR | 95% CI | P |
|-----------------------|---------------|---------------|------|-----------|-------|
| | Present (n=9) | Absent (n=37) | | | |
| Sex | | | | | |
| Male | 1 | 21 | 1.0 | | |
| Female | 8 | 16 | 10.0 | 1.15–486 | <0.05 |
| Age group | | | | | |
| ≥80 | 1 | 10 | 1.0 | | |
| 79–70 | 4 | 15 | 2.3 | 0.21–144 | 0.63 |
| 69–60 | 0 | 7 | 0 | 0.00–61.2 | 1.0 |
| ≤59 | 4 | 5 | 7.2 | 0.53–430 | 0.13 |
| Laterality | | | | | |
| Right | 5 | 18 | 1.0 | | |
| Left | 4 | 19 | 0.76 | 0.13–4.18 | 1.0 |
| Clinical stage of CTS | | | | | |
| 1 | 4 | 8 | 1.0 | | |
| 2 | 5 | 27 | 0.4 | 0.06–2.39 | 0.227 |
| 3 | 0 | 4 | 0 | 0.00–4.81 | 0.56 |
| HbA1c | | | | | |
| ≥6.5 | 3 | 8 | 1.0 | | |
| <6.5 | 6 | 25 | 0.65 | 0.10–4.91 | 0.68 |

TF – Trigger finger; OR – Odd ratio; CI – Confidence interval; CTS – Carpal tunnel syndrome; HbA1c – Hemoglobin A1c

environment after surgery and an inflammatory process in the flexor tendons.^[22] In addition, the flexor tendons at the wrist are displaced anteriorly, which alters the tendon biomechanics at the A1 pulley. Karalezli *et al.*^[27] investigated the effect of TCL release on the entrance angle of the flexor tendons to the A1 pulleys in a cadaver study. They concluded that release of both the TCL and distal forearm fascia (FF) may be a predisposing factor for the development of TF by changing the entrance angle to the A1 pulley and consequently increasing the friction in this anatomic area, predisposing to triggering of the digit.^[27] Acar *et al.*^[16] demonstrated that patients undergoing TCL release and distal FF release had a higher risk of TF than those undergoing TCL release only. As mentioned above, we released the TCL but not the FF in all patients who underwent CTR. From the viewpoint of TF prevention, care should be taken to avoid releasing more FF than necessary.

Several independent risk factors for new-onset TF after CTR have been reported. In the present study, TF tended to be frequent in patients with diabetes mellitus, but this finding did not reach statistical significance. Although diabetes mellitus has been cited as a key predisposing condition for development of CTS and TF,^[28] controversy exists regarding whether diabetes is a risk factor for the

Table 4: Reports that analyzed the occurrence of trigger finger after carpal tunnel release

| Authors | Year | Study | Main results | Conclusion |
|---|------|--|--|--|
| Hombal and Owen ^[22] | 1970 | Retrospective analysis of 132 CTR cases | TF occurred in 29 hands (22%) within 1 year after CTR | Clinicians should be reminded of the inter-relationships between the various constrictive lesions of the hand and wrist. A prospective study on this topic is needed |
| Hayashi <i>et al.</i> ^[21] | 2005 | Prospective analysis of 164 CTR cases and 101 conservatively treated CTS cases | Logistic regression analysis in hands with severe CTS revealed that no factor analyzed was significant for the development of TF. When the analysis was restricted to those hands with mild or moderate symptoms, surgery was significant risk factor for TF | Surgery may accelerate the development of TF when CTS is mild to moderate |
| Harada <i>et al.</i> ^[20] | 2005 | Retrospective analysis of 875 CTR cases | Surgery for TF was required in 101 (11.5%) patients, most often after CTR, especially within 3 months. The next most common was at the same time as CTR | Trigger digit-associated CTS is associated with previous development of wide-ranging narrowing of the flexor tendon sheath |
| Goshtasby <i>et al.</i> ^[18] | 2010 | Retrospective analysis of 792 CTR cases | The incidence of new-onset TF after CTR was 6.3%. Osteoarthritis and undergoing an endoscopic procedure were the only two independent risk factors | Patients with osteoarthritis and endoscopic CTR should be informed that they have a greater risk of developing TF |
| King <i>et al.</i> ^[24] | 2013 | Retrospective analysis of 1185 CTR cases | The incidence of TF was 6.6% in the hand ipsilateral to CTR and 3.7% in the hand contralateral to CTR | Hands with recent CTR are more likely to develop TF |
| Kim <i>et al.</i> ^[23] | 2013 | Retrospective analysis of 633 CTR cases | TF or de Quervain's disease was observed in 85 of the 633 hands (13%) before surgery and developed in 67 hands (11%) after surgery | Cervical arthritis, basal joint arthritis, and TF commonly coexist with idiopathic CTS |

Contd...

Table 4: Contd...

| Authors | Year | Study | Main results | Conclusion |
|---|------|--|---|---|
| Grandizio <i>et al.</i> ^[19] | 2014 | Retrospective analysis of 1217 CTR cases | Of 1003 CTR cases without DM, 3% developed TF within 6 months of CTR and 4% within 1 year of CTR (compared with 8% and 10%, respectively, those with DM) | The presence of DM rather than its severity is an important factor for developing TF |
| Acar <i>et al.</i> ^[16] | 2015 | Prospective analysis of 159 CTR cases | The incidence of TF was statistically higher when the TCL and distal FF were released than when only the TCL was released (31.3% and 13.9%, respectively) | The risk of postoperative TF is higher especially when the TCL and distal FF are released |
| El-Hadidi ^[17] | 2015 | Retrospective analysis of 565 CTR cases | TF at presentation occurred in 13% patients. TF after CTR occurred in 10.44% | CTS and TF commonly occur together at presentation. Surgery may accelerate the development of TF, especially when the CTS is mild to moderate |
| Lin <i>et al.</i> ^[25] | 2015 | Retrospective comparison of 2605 CTR cases with 10,420 control cases | The overall risk of TF was 3.63-fold higher in the patients who underwent CTR. The time to development of TF was approximately 6 months postoperatively | CTR was significantly associated with the subsequent development of TF, especially within 6 months postoperatively |
| Zang <i>et al.</i> ^[26] | 2019 | Retrospective study of 1140 CTR cases | The incidence of TF was associated with 0.5 times lower odds during the year after CTR compared with the year before CTR | Patients can be counseled that CTR does not cause the new development of TF in the operative hand |

DM – Diabetes mellitus; CTS – Carpal tunnel syndrome; CTR – Carpal tunnel release; TF – Trigger finger; TCL – Transverse carpal ligament; FF – Forearm fascia

occurrence of TF after CSR.^[17-19] Seven of the eight patients with TF in our study were female. This result is convincing considering that a male:female ratio of about 1:6 has been reported among patients with TF both associated and unassociated with CTS. With respect to the clinical stage of our patients with CTS, patients with Stage 1 tended to more frequently develop TF, although this finding also did not reach statistical significance. El-Hadidi^[17] reported that surgery may exacerbate the onset of TF, especially when CTS is mild to moderate. He concluded that in severe cases of CTS, other factors such as hypertrophy of the flexor tenosynovium may mask the effect of surgery. Although all surgical procedures in our series were carried out by open release of the TCL, endoscopic procedures have been found to be a risk factor.^[17] The traumatic effect of the endoscopic procedure and hematomas in the carpal tunnel on the surrounding soft tissue may cause inflammation and swelling, consequently increasing the occurrence of TF.

In contrast, the authors of several reports have insisted that CTR does not accelerate the development of TF. In 2005, Harada *et al.*^[20] retrospectively analyzed 101 patients who required trigger digit release. They reported that trigger digit release was performed most often after CTR, especially within 3 months; however, the next most common was at the same time as CTR. Moreover, Zhang *et al.*^[26] recently demonstrated that although new-onset TF is frequent in the operative hand after CTR, the frequency is even higher

before surgery. They concluded that although CTR has some effect on postoperative TF, it does not increase the risk of newly developing TF in the operative hand.

The presence of TF should be carefully assessed at presentation or before surgical treatment of CTS, and patients should be informed that the symptoms of TF might appear or worsen after surgery, especially within 6 months.

Conclusion

Neurosurgeons are generally considered less familiar with TF. However, an understanding of the clinical features of TF is necessary for successful management of CTS. TF should be carefully assessed at presentation and within 6 months after surgical treatment of CTS.

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Conflicts of interest

There are no conflicts of interest.

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