Carpal Tunnel Syndrome and Distal Radius Fractures

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KEYWORDS

• Acute carpal tunnel syndrome • Distal radius fractures • Nerve compression • Carpal tunnel release

KEY POINTS

- Three types of carpal tunnel syndrome occur after a distal radius fracture: acute, subacute or transient, and delayed.
- Acute carpal tunnel syndrome is likely caused by increases in carpal tunnel pressure and presents with progressive pain and median nerve dysfunction immediately after a distal radius fracture.
- Acute carpal tunnel syndrome should be treated with expeditious carpal tunnel release and fracture fixation.
- Subacute or transient carpal tunnel syndrome can frequently be treated with observation alone initially.
- Delayed carpal tunnel syndrome is typically due to alterations in carpal tunnel anatomy and requires etiology-specific treatment.

INTRODUCTION

Distal radius fractures (DRFs) are the most common fracture seen in the emergency department, with an incidence greater than 640,000 fractures per year.[1] Complications of DRF are many and include malunion, arthrosis, nonunion, tendon ruptures, complex regional pain syndrome, loss of motion at the wrist or fingers, compartment syndrome, and carpal tunnel syndrome (CTS).[2,3] It was not until 1933, when Abbott and Saunders[4] published a review of 9 cases, that the association of DRF and CTS was recognized as a more common phenomenon. CTS after DRF can be divided into 3 categories: acute, transient, and delayed.

Acute CTS, with an incidence of 5.4% to 8.6% after a DRF, is characterized by progressive pain and paresthesias in the median nerve distribution of the hand that develops over hours to days after a fracture. Its etiology is presumed likely due to elevated compartment pressure in the carpal tunnel.[3,5–8] In contrast, transient CTS, with an estimated incidence of 4%, has the least understood etiology of the 3 but is likely due to nerve contusion and/or stretch.[9] Unlike acute CTS, the symptoms of transient CTS can be present at the time of injury but classically do not progress, but rather gradually improve over days to weeks.[4,9,10] Lastly, delayed CTS, with an incidence of 0.5% to 22% after a DRF, can present months to years after an injury and is usually due to an alteration of the

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carpal tunnel anatomy after healing of the fracture.[3,8,11–14]

ACUTE CARPAL TUNNEL SYNDROME

Pathophysiology

Acute CTS after DRF is believed to be caused by a rapid increase in carpal tunnel pressure.[8,15,16] Increased carpal tunnel pressure can be due to traumatic wrist deformity with fracture displacement, hematoma formation, displaced volar fragments, generalized edema, local anesthetic injection, and wrist immobilization in excessive flexion or extension.[15–18] Gelberman and colleagues[16] evaluated carpal tunnel pressures in patients with DRFs and found that 45% of fractured wrists placed in 40° of flexion had carpal canal pressures greater than 40 mm Hg. Other factors that predispose patients to acute CTS include high-energy injuries, ipsilateral upper extremity trauma, women under the age of 48, multiple closed reduction attempts, DRF with greater than 35% fracture translation, fractures with significant comminution (AO type C), and radiocarpal dislocations.[6,7,18]

Diagnosis

It is essential that diagnosis of acute CTS after DRF be made in an expeditious fashion because a delay or missed diagnosis can lead to nerve injury and/or nerve dysfunction, such as complex regional pain syndrome.[19–21] Unrelenting pain and dysesthesias in the median nerve distribution of the hand are the hallmark symptoms of acute CTS. The sensory examination likely reveals altered 2-point discrimination and Semmes-Weinstein monofilament testing (monofilament testing being the most sensitive way to detect sensory threshold changes) in the median nerve distribution.[20] Other examination findings can include thenar motor weakness and a positive stretch test (pain at the volar wrist with passive extension of the fingers).[22]

Beyond examination findings, a diagnosis of acute CTS can also be made by measuring carpal tunnel compartment pressure. This can be done by inserting a wick catheter or the needle of an Stryker brand Intra-Compartmental (STIC) Pressure Monitor STIC device (Stryker, Kalamazoo, Michigan) 1 cm proximal to the wrist crease and just ulnar to the palmaris longus or in line with the ring finger ray if the palmaris longus is absent. The catheter is directed 45° distally and dorsally in a slightly radial direction until it sits just radial to the hook of the hamate[23] (**Fig. 1**). Mack and colleagues[23] suggest checking carpal tunnel pressure if the symptoms do not resolve within 2 hours of elevation and cast/dressing release. A pressure of greater than 30 mm Hg in the carpal tunnel can be considered diagnostic for acute CTS.

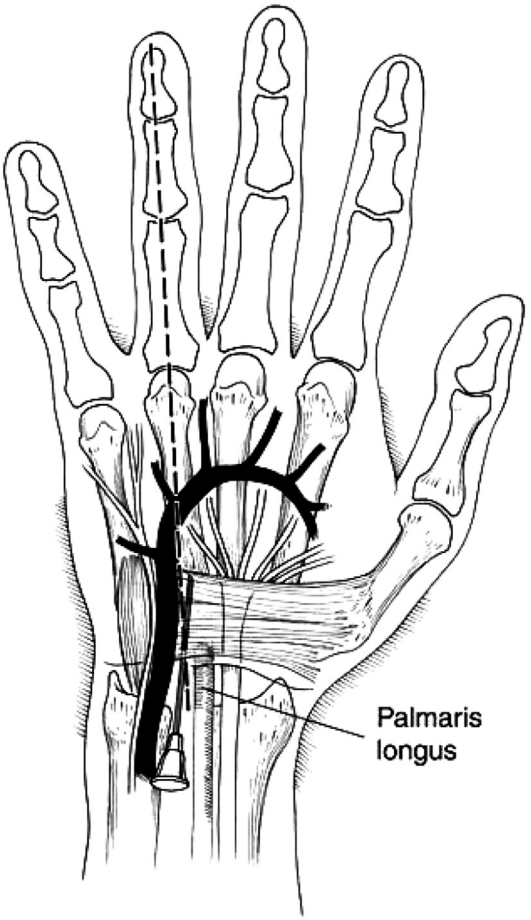

Fig. 1. Recommended catheter placement in the ulnar aspect of the carpal tunnel. (*From* Schnetzler KA. Acute carpal tunnel syndrome. J Am Acad Orthop Surg 2008;16[5]:280; with permission.)

Treatment

The treatment of acute CTS is immediate carpal tunnel release (CTR) with provisional or definitive fracture reduction and/or stabilization. In particular, any gross bony deformity or displaced bony fragments that impinge on the median nerve are corrected. Most investigators recommend immediate release of the carpal tunnel to achieve best outcomes.[3,14,24,25] Regardless of the method of fixation of the distal radius, the carpal tunnel can be released in the standard fashion, open or mini-open. Endoscopic CTR is not recommended in this setting due to the trauma and altered anatomy. If volar locked plating with a volar approach is planned for fracture fixation, it is preferable to use a separate dedicated incision for the CTR to

avoid inadvertent injury to the palmar cutaneous branch of the median nerve by connecting the 2 incisions.[26] Alternative techniques includes a hybrid flexor carpi radialis (FCR) approach.[26] Although there is no clear superior surgical technique for how best to release the carpal tunnel, it is clear that patients without signs of preoperative median nerve dysfunction after a DRF should not undergo a prophylactic CTR.[27,28]

For acute CTS, the preference of the senior author (P.T.) is to perform a standard open CTR, which is larger than a standard miniopen incision (miniopen incision measures 1.7–2.0 cm and starts 5 mm distal to the wrist flexion crease). The standard open CTR starts at the wrist flexion crease and is used to allow better inspection of the nerve within the carpal tunnel after the release (**Fig. 2**). A separate incision for the fracture approach and fixation is then longitudinally centered over the FCR tendon up to but not crossing the distal wrist flexion crease.

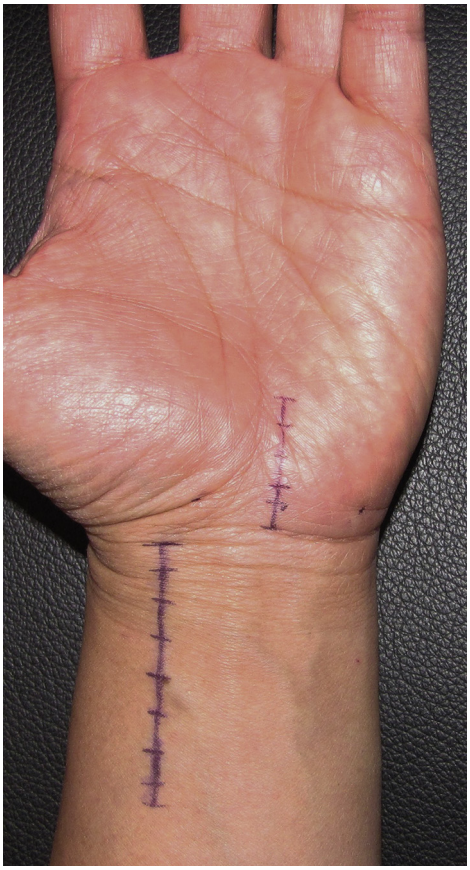

Fig. 2. Picture of standard open CTR incision with volar Henry incision. A dot is placed at the center of the distal pole of the scaphoid and well as the center of the pisiform. A dot is made at the midpoint between these first 2 marks. A dot is also placed at the wrist flexion crease in line with the radial border of the ring finger. The CTR incision is centered between the scaphoid/pisiform midpoint mark and the radial border of the ring finger mark, or the more ulnar of the 2 points. A more ulnar-based incision is preferred to avoid injury to a transligamentous motor branch with the assumption that if a transligamentous branch is present it will traverse the ligament more radially. The CTR incision starts at the wrist flexion crease and is approximately 3 cm long.

TRANSIENT CARPAL TUNNEL SYNDROME

Transient, or subacute, CTS after DRF is characterized by transient median nerve dysfunction that starts immediately after the injury and does not progress. In general, this type of CTS is most frequently caused by a median nerve stretch or contusion from initial fracture displacement or volarly displaced fragments but can also be related to edema or hematoma.[4,10] On examination these patients usually do not have any objective nerve deficits but they report subjective numbness in the median nerve distribution. In these patients, median nerve dysfunction generally resolve with observation, elevation, and adequate fracture reduction.[29] Although a majority of these patients' symptoms resolve with observation alone, in patients with volarly displaced fragments some investigators recommend early CTR with removal or reduction of displaced fragments to avoid continued median nerve compression.[10,30]

The senior author's preference is to perform a CTR during fracture repair for any patient with transient CTS who have any subjective numbness in the median nerve distribution. The senior author also performs a CTR in any patient with any preinjury symptoms of CTS. The reasoning for this approach is to address a potential secondary site of compression for the median nerve (carpal tunnel) if the nerve has been injured at the level of the distal radius, which is akin to a double crush phenomenon, and also to address suspected idiopathic CTS. Furthermore, the patient is already undergoing surgery and the concomitant CTR could obviate a second surgery. In terms of surgical technique, the senior author uses a standard miniopen CTR incision through a separate incision at the standard location at the base of the volar hand (**Fig. 3**).

DELAYED CARPAL TUNNEL SYNDROME

Delayed CTS, with an incidence of 0.5% to 22% after DRF, can present months to years after injury and is usually associated with the late effects of the altered anatomy in the carpal tunnel and not necessarily a critical increase in canal pressure. The etiology includes malunion, chronic tenosynovitis, volarly displaced fragments, cicatrix,

Fig. 3. Picture of miniopen CTR incision with volar Henry incision. The same landmarks are made as in Fig. 1. The miniopen CTR incision begins 5 mm to 8 mm distal to the wrist flexion crease and measure 1.7 cm to 2.0 cm in length.

enlarged volar callus, and prominent hardware.[3,11,12,14] The symptoms of delayed CTS after DRF are not well described in the literature but are generally assumed to be similar in presentation to idiopathic CTS.

The diagnosis of delayed CTS after a DRF follows the normal work-up for CTS once anatomic and radiographic abnormalities are assessed first. It is also possible that some patients with presumed delayed CTS may only have developed idiopathic CTS that is unrelated to their previous DRF, which would be supported if no anatomic abnormality related to the DRF is identified after radiographic evaluation of the wrist.[31]

Treatment of delayed CTS depends on the underlying cause but does not represent the emergency that is acute CTS. Moreover, up to 66% of cases of delayed CTS can avoid surgery with conservative measures (observation, splinting, and injections).[32] It is unclear, however, in the literature whether nerve conduction studies/electromyography should guide treatment in a similar manner as it does in idiopathic CTS. In other words, should conservative treatment be reserved for those with electrodiagnostically confirmed mild compression whereas surgery should be recommended only for moderate or severe compression? Also, should surgical treatment only consist of standard release of the transverse carpal ligament, or should surgery also address any correctable issues, such as flexor tenosynovitis, prominent volar callus, prominent hardware, or residual fracture malunion? Regardless, most commonly the issue is a thickened transverse carpal tunnel ligament and/or median nerve scarring. To address these most common etiologies, a standard CTR can resolve the thickened transverse carpal tunnel ligament and a median nerve neurolysis can free the nerve from any scarring at the level of the volar plate, assuming a locked volar plate was used previously. In a small study, Ho and colleagues[32] found

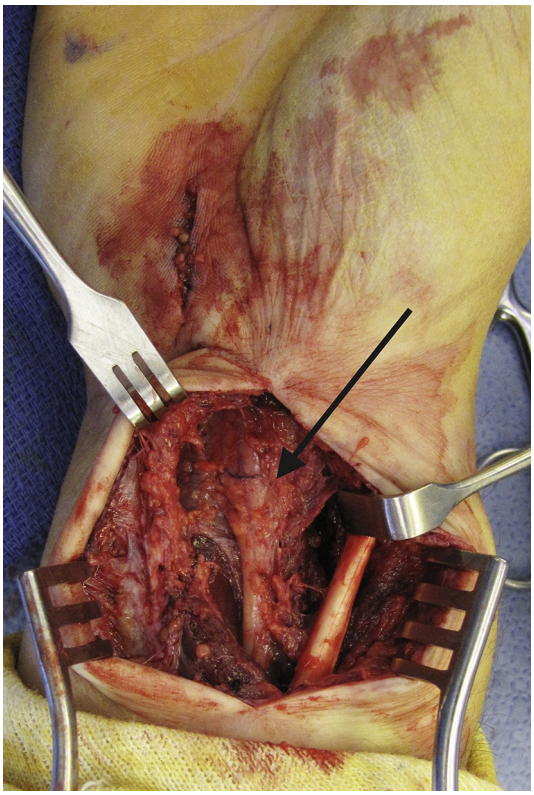

Fig. 4. Intraoperative photo of patient with delayed CTS after DRF. The initial treatment was reduction and repair of the distal radius with a locked volar plate without CTR (the patient had no median nerve symptoms at index surgery). The patient underwent a miniopen CTR and median nerve neurolysis at the distal radius level. Cicatrix formation was found encasing median nerve (*arrow*). The FCR tendon is on the right being retracted.

patients improved whether they underwent CTR alone or whether they underwent median nerve neurolysis alone. Arora and colleagues[33] noted improvement in patients with CTR and removal of hardware.

It seems that the most common cause would be cicatrix at the median nerve at the previous volar fracture repair site, so neurolysis of the nerve at the distal forearm should relieve this problem (**Fig. 4**). If this theory is correct, CTR should be unnecessary unless the carpal tunnel was released at the index procedure and this is now a site of cicatrix. If CTR was not performed at the index procedure and the problem area is the carpal tunnel, then the patient may have developed idiopathic CTS. Theoretically, nerve conduction studies/electromyography should be able to differentiate the problem area but due to the adjacent nature of the sites in question, the sensitivity of this study may be poor. Lastly, another possibility is the development of a double crush phenomenon where a patient's preexisting amount of carpal tunnel compression was not symptomatic until the nerve cicatrix formed at the distal radius level. Thus, for delayed CTS, to address any potential sites of median nerve compression, the senior author's preference is to perform a CTR (using an extended incision if CTR was done at the index procedure), median nerve neurolysis at the distal forearm with or without nerve conduit wrapping depending on the amount of scarring, and removal of the volar locked plate if present (**Fig. 5**). The CTR would be performed whether or not the patient had a CTR at the index procedure.

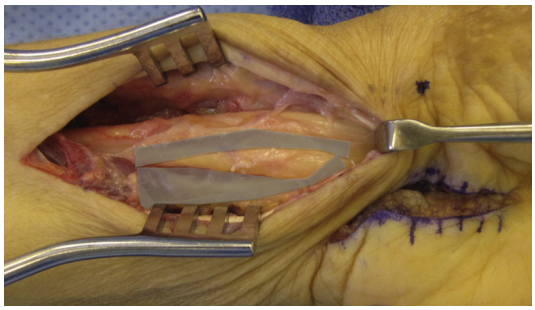

Fig. 5. Intraoperative photo of patient with delayed CTS after DRF. The initial treatment was reduction and repair of the distal radius with a locked volar plate and CTR for subacute CTS. Because this was a revision CTR setting, a longer incision was made distal and proximal to the previous incision. The proximal extent of the incision crosses the wrist flexion crease in an ulnar direction (to avoid injury to the palmar cutaneous nerve). Cicatrix was also found around the median nerve at the level of the plate. Median nerve neurolysis at the distal radius level was performed as well as removal of the volar plate. A nerve conduit (*gray shading*) was placed around the median nerve to try to prevent future scarring.

SUMMARY

CTS after DRF can be categorized as acute, transient, and delayed. Early and expeditious treatment of acute CTS is necessary, and failing to diagnose and treat it can lead to permanent median nerve dysfunction. Many patients with transient CTS after DRF do not require surgical release of the carpal tunnel. For patients with delayed CTS, all possible causes of nerve compression (fracture fragments, hardware, synovitis, and so forth) should be considered and subsequently addressed. Prophylactic CTR in the absence of signs and symptoms of CTS after DRF is not indicated.

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