

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 Saunders⁴ 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 colleagues¹⁶ 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 hamate²³ (Fig. 1). Mack and colleagues²³ suggest checking carpal tunnel pressure if the symptoms do not resolve within 2 hours of elevation

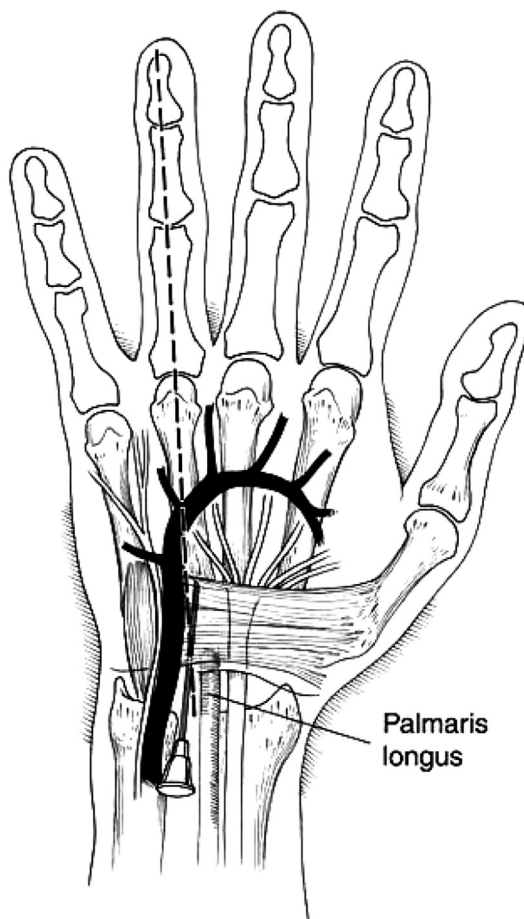


Fig. 1. Recommended catheter placement in the ulnar aspect of the carpal tunnel. (From Schnetzler KA. Acute carpal tunnel syndrome. *J Am Acad Orthop Surg* 2008;16[5]:280; with permission.)

and cast/dressing release. A pressure of greater than 30 mm Hg in the carpal tunnel can be considered diagnostic for acute CTS.

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

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