Acute Carpal Tunnel Syndrome

A Review of Current Literature

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KEYWORDS

• Acute carpal tunnel syndrome • Carpal tunnel syndrome • Review • Median nerve • Wrist trauma

KEY POINTS

- Acute carpal tunnel syndrome is a known complication of wrist and hand trauma including distal radius fractures and numerous atraumatic causes.
- Patient evaluation should differentiate ACTS, which is a progressive condition from normal sensation to loss of two-point discrimination, from neuropraxic injury, which is stable loss of sensation immediately after injury.
- Complete release of the transverse carpal ligament should be performed on emergent basis after diagnosis. Concomitant fractures and underlying medical conditions should be treated as indicated.

INTRODUCTION

Carpal tunnel syndrome (CTS) is the most common peripheral nerve compression. The incidence of CTS is 99 per 100,000 individuals and it is most common in patients older than 40.[1,2] Females also comprise between 65% and 75% of all cases.[3] It is often seen as a chronic progression of median nerve compression as the nerve passes beneath the transverse carpal ligament. Although elective carpal tunnel release (CTR) is performed in severe or refractory cases, conservative management and observation are used in milder cases. Acute CTS (ACTS) is a less common presentation and requires more urgent and aggressive management. Many conditions can lead to ACTS, but central to this diagnosis is a progressive worsening of median nerve function. This is an important distinction because neurapraxia and nerve contusion can present with a similar distribution of symptoms, but their severity remains stable and does not progress over time.

The onset of ACTS is often measured in minutes to hours, in contrast to chronic CTS. ACTS most commonly results following trauma; however, numerous other etiologies have been described at a significantly lower incidence. All causes of ACTS do share the same underlying pathology of an acute increase in pressure within the carpal tunnel. This results in compromise of the epineural blood flow and thus pain and dysesthesias in the distribution of the median nerve. Urgent surgical decompression of the median nerve is necessary to prevent further progression of symptoms.

ANATOMY

The carpal tunnel is an enclosed space bordered on three sides by the carpal bones and on the fourth by the flexor retinaculum. As a result, the volume of the carpal tunnel is relatively constant at around 5 mL,[4] with little room for expansion or swelling secondary to its inelastic borders. The tunnel itself is transversed by

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10 structures, nine tendons and the median nerve. The tendons include the four flexor digitorum superficialis tendons, the four flexor digitorum profundus tendons, and the tendon of the flexor pollicis longus. The dorsal floor of the carpal tunnel is abutted by the triquetrum, hamate, capitate, and the scaphoid. Radially, the scaphoid tubercle and the trapezium border the tunnel with the ulnar border being composed of the triquetrum, pisiform, and the hook of the hamate. Finally, the volar surface of the tunnel is composed of three structures that make up the flexor retinaculum. These include the deep forearm fascia, the transverse carpal ligament, and the distal aponeurosis that divides the thenar and hypothenar musculature. These include the deep forearm fascia, the transverse carpal ligament, and the distal aponeurosis that divides the thenar and hypo thenar musculature (Fig. 1).

The most proximal portion of the carpal tunnel begins at the volar wrist crease and then extends distally to a line running from the abducted border of the thumb to the hook of the hamate, Kaplan cardinal line. At Kaplan cardinal line, the average width of the tunnel is 25 mm.[5] The carpal tunnel is at its narrowest, around 20 mm, at the level of the hook of the hamate. At the proximal and distal portions of the tunnel, an opening exists; however, synovium at either end results in the properties of a closed compartment. When the pressure within the compartment rises above a threshold, blood flow decreases resulting in compromise to the median nerve and paresthesias in the nerve distribution.

The median nerve supplies sensation to the most radial 3.5 fingers, the thenar musculature, and the lumbricals of the index and middle fingers. The palmar cutaneous branch of the median nerve branches off just proximal to the wrist flexion crease between the pollicis longus and the flexor carpi radialis and runs superficial to the flexor retinaculum. This nerve divides into a lateral branch, which supplies sensation over the volar base of the thumb, and the medial branch, which supplies sensation to the radial side of the palm. This sensory branch is not affected by compression in the carpal tunnel, and thus its function can help to distinguish CTS from more proximal median neuropathy. The recurrent branch of the median nerve innervates the opponens pollicis, abductor pollicis brevis, and the superficial part of the flexor pollicis brevis. The branching of this nerve has substantial anatomic variability with 50% of the population having extraligamentous branching, meaning that the motor branches occur distal to the carpal tunnel. Up to 30% of the population experiences subligamentous branching, where the motor nerve originates within the carpal tunnel. Lastly, 20% of the population experiences transligamentous branching, where the nerve branches off within the carpal tunnel and then pierces the transverse carpal ligament on its course toward the thenar musculature.[6] Other terminal branches of the median nerve include the digital cutaneous branches, which supply sensation to the radial 3.5 digits on the palmar side and the dorsal tips of the 3 most radial digits. A small percentage of 1.2% to 23% of the population may also retain the gestational remnant of the median artery, which courses with the median nerve into the hand.[7,8]

PATHOPHYSIOLOGY

Many presentations of ACTS have been reported in the literature. Although ACTS itself is uncommon, its presentation is most often considered in the setting of trauma, such as distal radius fractures or perilunate injuries. Awareness of these possible causes should guide evaluation in emergency room settings; however, small case series and case reports describe innumerable other causes ranging from gout to parvovirus. This demonstrates the importance of a thorough nerve examination in all patients with any sign of progressive nerve symptoms. The underlying pathologic process that causes ACTS is the creation of mass effect from a space-occupying lesion in the carpal tunnel resulting in increased compartmental pressures. This rise in compartmental pressure creates a compartment syndrome that results in lack of epineurial perfusion and ultimately ischemia. The lack of perfusion causes local tissue edema, nerve conduction delays propagated by demyelination along the axon, and axonal transport dysfunction that inhibits recurrent nerve firing.[9–11] Short intervals of decreased perfusion are rapidly reversible; however, a longer duration of compression increases the latency period before recovery and also increases risk of permanent disability.

Previous animal and human studies show thinned nerves in the entrapped segment with swelling of the nerve proximal to that region. This is thought to be caused by accumulation of axoplasm, nerve edema, and chronic inflammatory fibrosis of the nerve.[12,13] Demyelination and remyelination of the affected segments leads to poorer nerve conduction and a loss of large myelinated axons leading to increased latency. The normal compartment pressure of the carpal tunnel is 2.5 mm Hg at rest, and this increases with wrist flexion or extension. The average