Carpal Tunnel Syndrome: A Review of the Recent Literature

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Abstract: Carpal Tunnel Syndrome (CTS) remains a puzzling and disabling condition present in 3.8% of the general population. CTS is the most well-known and frequent form of median nerve entrapment, and accounts for 90% of all entrapment neuropathies. This review aims to provide an overview of this common condition, with an emphasis on the pathophysiology involved in CTS. The clinical presentation and risk factors associated with CTS are discussed in this paper. Also, the various methods of diagnosis are explored; including nerve conduction studies, ultrasound, and magnetic resonance imaging.

Keywords: Carpal tunnel syndrome, median nerve, entrapment neuropathy, pathophysiology, diagnosis.

WHAT IS CARPAL TUNNEL SYNDROME?

First described by Paget in 1854 [1], Carpal Tunnel Syndrome (CTS) remains a puzzling and disabling condition commonly presented to Rheumatologists and Orthopaedic Hand clinicians. It is a compressive neuropathy, which is defined as a mononeuropathy or radiculopathy caused by mechanical distortion produced by a compressive force [2]. The American Academy of Orthopaedic Surgeons (AAOS) Clinical Guidelines on the Diagnosis of CTS defines it as a symptomatic compression neuropathy of the median nerve at the level of the wrist [3].

CTS is the most well-known and frequent form of median nerve entrapment [3-8], and accounts for 90% of all entrapment neuropathies [9]. An entrapment neuropathy is a chronic focal compressive neuropathy caused by a pressure increase inside non-flexible anatomical structures [10]. CTS is a neuropathy caused by entrapment of the median nerve at the level of the carpal tunnel, delimitated by the carpal bones and by the transverse carpal ligament [2]. Physiological evidence indicates increased pressure within the carpal tunnel, and therefore decreased function of the median nerve at that level [3].

Other forms of median nerve entrapment neuropathies include pronator syndrome and anterior interosseous nerve syndrome. Pronator syndrome is defined as compression of the median nerve in the forearm that results in sensory alteration in the median nerve distribution of the hand and the palmar cutaneous distribution of the thenar eminence [11, 12]. Anterior interosseous nerve syndrome [13] is characterised by complete or partial loss of motor function of the muscles innervated by the anterior interosseous nerve (AIN), a motor branch of the median nerve in the forearm [4].

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EPIDEMIOLOGY

CTS is the most frequent entrapment neuropathy [2], believed to be present in 3.8% of the general population [14]. 1 in every 5 subjects who complains of symptoms such as pain, numbness and a tingling sensation in the hands is expected to have CTS based on clinical examination and electrophysiological testing [4], idiopathic CTS being the most common diagnosis in patients with these symptoms [15].

Incidence rates of up to 276:100,000 per year have been reported [16], with a prevalence rate up to 9.2% in women and 6% in men [13]. More common in females than in males, its occurrence is commonly bilaterally with a peak age range of 40 to 60 years [17]; although it occurs in all age groups. The prevalence of CTS in the United Kingdome (UK) alone is 7-16% [9]); much higher than the 5% prevalence in the United States (US) [3].

In all western countries, an increase is reported in the number of work-related musculoskeletal disorders (WMSDs) caused by strain and repeated movements (biomechanical overload). In Europe, in 1998, over 60% of upper limb musculoskeletal disorders recognised as work-related were CTS cases cases [18,19]. Some industries such as fish processing have reported the prevalence of CTS in their workers to be as high as 73% [20].

This data may reflect the increasing level of sensitivity to this problem, which is translated into a higher number of reports, rather than reflecting an actual increase in the prevalence of CTS [2]. There certainly has been an increase in the number of CTS patients, but this could be due to the general increase in the life span of people and the increased number of diabetic patients [21]. Diabetic patients have a prevalence rate of 14% and 30% without and with diabetic neuropathy, respectively [22], whilst the prevalence of CTS during pregnancy has been reported to be around 2% [23].

SOCIOECONOMIC EFFECTS

Median number of days away from work due to CTS is amongst the highest in the UK at 27 days [24]. In the US in

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1995, 400,000-500,000 patients underwent surgical decompression, which equates to an economic cost of in excess of $2 billion [9], whilst UK surgical decompression rate is 43-74 per 100,000 [9]. This amounts to a heavy burden on the National Health Service to provide care for CTS patients in the form of clinicians' time, diagnosis, and conservative management and treatment options.

CLINICAL PRESENTATION

Primary features of CTS include pain in the hand, unpleasant tingling, pain or numbness in the distal distribution of the median nerve (thumb, index, middle finger and the radial side of the ring finger) [25]), and a reduction of the grip strength and function of the affected hand [26]. Symptoms tend to be worse at night, and clumsiness is reported during the day with activities requiring wrist flexion [27]). Patients often describe a phenomenon termed the "flick sign", in which shaking or flicking their wrists relieves symptoms [28].

Many patients report symptoms outside the distribution of the median nerve as well, which has been confirmed by a systematic study conducted by Stevens *et al.,* [29]. In 159 hands of patients with electrodiagnostically confirmed CTS, symptoms were most commonly reported in both the median and ulnar digits more frequently than the median digits alone. They also report location of symptoms in areas other than the digits. 21% of patients had forearm paraesthesias and pain; 13.8% reported elbow pain; 7.5% reported arm pain; 6.3% reported shoulder pain; and 0.6% reported neck pain [29]. Interestingly, trigger digit presentation accompanies idiopathic CTS in approximately 20% of patients [30].

A large multicentre study has confirmed that patients with mild to moderate CTS are more likely to report substantial symptoms and mild functional limitations, whereas patients with more severe disease may report less severe symptoms, but have more severe functional limitations of the hand [31]. This appears to be a contradiction, but in fact it relates to the fact that severe compromise of the median nerve may impair sensory functioning to the extent that the profound numbness minimizes the experience of tingling and pain. However, profound functional limitations will ensue as a result of such a level of numbness and motor impairment [32].

Patients suffering from CTS often report subjective feelings of swelling in their hands or wrists, but no apparent swelling can be observed. However, some clinicians find that this symptom has some diagnostic value attached to it [33]. In study of over 8000 patients with suspected CTS, symptoms on the radial part of the hand and nocturnal exacerbation of symptoms were most strongly predictive of positive NCS [34]. In a retrospective study of 1039 patients with a neurophysiological diagnosis of CTS, Nora *et al.,* [35] found that the most characteristic manifestation of the syndrome was parasthesia in the median nerve distribution, frequently extending to the whole hand. Pain was very common but less specific, and weakness was rare [35].

Phalen noted a volar wrist swelling in several patients - a visual and palpable swelling into the shape of a "hot dog". He studied this presentation in 82 hands with CTS and in 200 control hands, and found that it correlated well with Tinel and Phalen signs. He concluded that it is a useful diagnostic sign, because it depends on clinical observation rather than the patient's history [36].

CTS may be classified on the basis of symptoms and signs into three stages:

Stage 1: Patients have frequent awakenings during the night with a sensation of a swollen, numb hand. They report severe pain that irradiates from the wrist to the shoulder, and an annoying tingling in their hand and fingers (brachialgia paraesthetica nocturna). Hand shaking (the flick sign) relives the symptoms. During the morning, a sensation of hand stiffness usually persists.

Stage 2: The symptoms are present also during the day, mostly when the patient remains in the same position for a long time, or performs repeated movements with their hand and wrist. When motor deficit appears, the patient reports that objects often fall from his/her hands because they are unable to feel their fingers anymore.

Stage 3: This is the final stage in which atrophy (wasting) of the thenar eminence is evident, and the median nerve usually responds poorly to surgical decompression [4]. In this phase, sensory symptoms may diminish [6]. There is also aching in the thenar eminence, and with severe compression, weakness and atrophy of the abductor pollicis brevis and opponens pollicis [7, 8].

Some patients may present with atypical signs of CTS, such as "writer's cramp" or fatigue, pain in the shoulder only [37], cold sensitivity in the fingers (presumably reflecting the median nerve's supply of sympathetic fibres to part of the forearm and hand), forearm pain [38], or numbness in the third finger only [27]. Sometimes there may be no symptoms but patients present with visual thenar atrophy and denervation on nerve conduction studies [27].

In some instances, patients only have symptoms with rigorous activity, usually work-related, and present with minimal symptoms or objective findings when examined. This is termed "dynamic CTS" and patients usually benefit from conservative management, including alteration of work duties. Therefore, the importance of a well-defined history is particularly important in these cases [39].

ANATOMY

The carpal tunnel is composed of a bony canal, consisting of carpal bones, the roof of which is the fibrous but rigid transverse carpal ligament. The carpal tunnel contains the nine flexor tendons and the median [27], which enters the tunnel in the midline or slightly radial to it [32]. Atypical presentations could be explained by anatomical variations in the median nerve itself [32].

Sensory branches from the median nerve supply the 3 radial digits and the radial half fourth digit - hence why CTS symptoms are felt in these fingers. The palmar sensory cutaneous branch of the median nerve supplies the cutaneous skin of the palm, and arises, on average, 6 cm proximal to the transverse carpal ligament (TLC). Therefore, the palm is generally not affected in CTS [27].

RISK FACTORS ASSOCIATED WITH CTS

CTS remains an idiopathic syndrome, but there are certain risk factors that have been associated with this condition. The most significant of these are environmental

risk factors. Prolonged postures in extremes of wrist flexion or extension, repetitive use of the flexor muscles, and exposure to vibration are the primary exposures that have been reported [40-43].

Medical risk factors can be divided into four categories: (1) extrinsic factors that increase the volume within the tunnel (outside or inside the nerve); (2) intrinsic factors within the nerve that increase the volume within the tunnel; (3) extrinsic factors that alter the contour of the tunnel; and (4) neuropathic factors.

Extrinsic factors that can increase the volume within the tunnel include conditions that alter the fluid balance in the body. These include pregnancy, menopause, obesity, renal failure, hypothyroidism, the use of oral contraceptives and congestive heart failure [32].

Intrinsic factors within the nerve that increase the occupied volume inside the tunnel include tumours and tumour-like lesions. Extrinsic factors that can alter the contour of the tunnel could be the aftermath of fractures of the distal radius, directly or *via* posttraumatic arthritis [32].

Neuropathic factors, such as diabetes, alcoholism, vitamin toxicity or deficiency, and exposure to toxins, can play a role in eliciting CTS symptoms. This is because they affect the median nerve without necessarily increasing the interstitial pressure within the carpal tunnel [32]. In fact, diabetic patients have higher tendency to develop CTS due to lower threshold for nerve damage [4].

PATHOPHYSIOLOGY

The pathophysiology of CTS involves a combination of mechanical trauma, increased pressure and ischemic injury to the median nerve within the carpal tunnel [44].

Increased Pressure

There are many pressure related studies of the carpal tunnel in humans [45-47]. Normal pressure has been recorded to be in the range of 2-10 mm Hg [4]. There are dramatic changes of fluid pressure in the carpal tunnel with wrist position; extension increases the pressure 10-fold and wrist flexion increases it 8-fold [44]. Therefore, repetitive hand movements have been implicated as one of many risk factors for CTS. Experimental studies have suggested a dose-response curve - the greater the duration and amount of pressure, the more significant is the neural dysfunction [48].

Nerve Injury

An important step in injury to the median nerve is demyelination, which occurs when the nerve is repetitively subjected to mechanical forces [2]. Pressures much higher than systolic are necessary to produce focal demyelination [4]. Demyelination of the nerve develops in the compression site, and can then spread to the entire internodal segment, leaving the axons intact. A block of nervous transmission ensues (neuroapraxia). If the compression persists, bloodflow to the endoneural capillary system may be interrupted, leading to alterations in the blood-nerve barrier, and development of endoneural oedema. This starts a vicious cycle consisting of venous congestion, ischaemia and local metabolic alterations [2]. Axonal degeneration, macrophage attraction and activation, release of inflammatory cytokines, nitric oxide, and development of "chemical neuritis" are all consequences of this viscous cycle if it continues for a substantial amount of time [10].

Nerve Tethering

Nerve fibres have layers of connective tissue: the mesoneurium, epineurium, perineurium and endoneurium; which is the most intimate layer. The extensibility of these layers is critical to nerve gliding, which is necessary to accommodate joint motion; otherwise nerves are stretched and become injured [49].

The median nerve will move up to 9.6 mm with wrist flexion and slightly less with extension [50]. Chronic compression results in fibrosis, which inhibits nerve gliding, leading to injury and therefore scarring of the mesoneurium. This causes the nerve to adhere to the surrounding tissue, resulting in traction of the nerve during movement as the nerve attempts to glide from this fixed position [32]. This is the basis of the tethered median nerve stress test (TMNST), which is can be used to diagnose chronic low-grade CTS [51].

Ischemic Injury

Ischemic injury has been identified as an essential component in CTS due to Gelberman *et al.*'s observation that symptoms rapidly resolve after carpal tunnel release surgery [45]. Lundbrog *et al.,* demonstrated that limb ischaemia increases paraesthesias in carpal tunnel patients [52]. Ischemic injury in CTS has three stages: (1) increased intrafunicular pressure; (2) capillary damage with leakage and oedema, and (3) obstruction of arterial flow [44].

Breakdown in the Blood-Nerve-Barrier

The blood-nerve-barrier formed by the inner cells of the perineurium and the endothelial cells of endoneurial capillaries that accompany the median nerve through the carpal tunnel. These endoneurial microvessels are formed from nutrient branches that arise from the radial and ulnar arteries, proximal to the flexor retinaculum [32]. An increase in pressure within the tunnel can cause a breakdown of vasculature within this barrier, causing an accumulation of proteins and inflammatory cells [32]. This may induce a miniature closed compartment syndrome by increasing the permeability, contributing to increased endoneurial fluid pressure and development of an intra-fascicular oedema [53]. Patients with vascular problems or prolonged exposure to static loading are particularly prone to a breakdown in the blood-nerve-barrier [32].

Synovial Tissue

Abnormalities of the synovial tissue lining the tendons within the carpal tunnel have been implicated as a closely related factor to the development of idiopathic CTS. This has been confirmed by MRI, histological and biochemical studies [54,55]. Abnormalities include thickening of the synovial tissue, which may be caused by repetitive hand activity [47-56]. This increases the volume of tissue within the canal, leading to an increase in the fluid pressure within the carpal tunnel [47]. The most profound thickening of synovial tissue has been reported to be at the entrance and exit regions of the canal where the tendons slide over a fulcrum of the flexor retinaculum [47]. Strain and micro-

damage to the synovial tissue as well as the median nerve can occur due to the different degrees of excursion between the flexor tendons and the median nerve [55-57].

As a result, biochemical changes in the synovial tissue ensue. For example, repetitive exposure of the tendons to compression or tensile strength can increase the proteoglycan content in the tendon matrix. Hypertrophy of the tendon occurs, increasing the cross-sectional area, which then in turn increases the pressure within the carpal tunnel [58,59].

Inflammation

Tenosynovitis, inflammation of the synovial tissue of the flexor tendons, can also cause increased pressure in the carpal tunnel and result in CTS [60]. This has been confirmed by the presence of increased expression of prostaglandin E2 and vascular endothelial growth factor (VEGF) in synovial biopsy tissue from patients with symptomatic CTS [61]. In response to this injury, there is an increase in fibroblast density, collagen fibre size, vascular proliferation, and type III collagen in the synovial connective tissue [62]. Constrictive scar tissue is formed around the median nerve [63], which in turn can result in tethering of the nerve.

Involvement of Small Fibres

Most studies on compression and nerve function focus on the large myelinated nerves. However, the involvement of small fibres is very relevant and may help us understand the diversity of symptoms, such as the pain experienced by some patients in the distribution area of the median nerve [44]. This pain is caused by an abnormal diffusion of the Na+ channels into the damaged nociceptive fibres, which are small C-fibres, resulting in hyperexcitability and ectopic discharge induction. Inflammatory mediators, in particular TNFa, play an important role in the pain-related symptoms of CTS [10].

DIAGNOSIS

Two papers by the Quality Standards Subcommittee of the American Academy of Neurology [64] and American Association of Electrodiagnostic Medicine, American Academy of Neurology and American Academy of Physical Medicine and Rehabilitation define the guidelines for clinical and neurophysiologic diagnosis of CTS [65]. These papers stress the importance of a thorough case history, which must focus on the following [2]:

- symptom onset - which in the early stage is mainly nocturnal paraesthesias.
- provocative factors - such as hand positions and repeated movements.
- working activity - instrument use, vibrating tools.
- pain localisation and irradiation - in the cutaneous median nerve region with ascending, sometimes up to the shoulder, or descending irradiation.
- manoeuvres which alleviate symptoms - e.g. hand shaking, position changes.
- presence of predisposing factors - e.g. diabetes, adiposity, chronic polyarthritis, myxoedema, acromegaly, pregnancy.
- sports activity - e.g. baseball, body-building.

The two provocative test most commonly used in the clinical setting are Phalen's and Tinel's tests. In Phalen's test, the patient is asked to flex their wrist and keep it in that position for 60 seconds. A positive response is if it leads to pain or paraesthesia in the distribution of the median nerve [60]. The sensitivity of Phalen's test is in the range of 67% to 83%, whilst the specificity ranges between 40% and 98% [66-68].

Tinel's test is performed by tapping over the volar surface of the wrist. A positive response is if this causes paraesthesia in the fingers innervated by the median nerve: the thumb, index, middle finger and the radial side of the ring finger [6]. Tinel's test has a sensitivity in the range of 48% to 73%, whilst the specificity is 30% to 94% [66-68].

It is evident that there are significant variations in these values, which may be attributed to the fact that there are substantial inconsistencies in the method of examination and interpretation of the results [15]. Therefore, some researchers have questioned their diagnostic value [69]. This, coupled with the fact that both Phalen's and Tinel's tests have a low positive predictive value, supports the view that such provocative tests are insufficient and unreliable when used alone in the diagnosis of CTS. This emphasises the importance of considering them with a good clinical history and other appropriate methods of examination, such as nerve conduction studies (NCS) [70]. This view has recently been supported by a study conducted by EL Miedany *et al.* [71]. They found that both Phalen's and Tinel's tests were in fact more sensitive and specific for the diagnosis of tenosynovitis than for the diagnosis of CTS. Therefore, they concluded that there is a greater reliance on NCS as a diagnostic gold standard in the diagnosis of CTS [72], despite the fact that false positives and negatives are known to exist [34].

Although NCS can be regarded as a gold standard in the diagnosis of CTS, it is not a widely accepted and recognised phenomenon. This gives rise to problems in evaluating whether any individual test is accurate in diagnosing CTS, because there isn't an accepted "gold standard" against which other tests can be compared [34,73]. These tests include the Diagnostic CTS Scale [74], the Symptom Severity Scale (SSS) and Functional Scale (FS) [75], the Katz hand diagram [39], and the Hand elevation test [76].

Differential Diagnosis [2]

CTS must be differentiated from:

- Cervical radiculopathy (especially C6-C7)
- Brachial plexopathy (in particular of the upper trunk)
- Proximal median neuropathy (especially at the pronator teres level)
- Thoracic outlet syndrome
- CNS disorders (multiple sclerosis, small cerebral infarction)

DIAGNOSIS: NERVE CONDUCTION STUDIES

Nerve Conduction Studies (NCS) have been developed as a result of the discovery in 1956 that median nerve conduction times are slowed across the wrists of hands in CTS patients [77]. Prolonged motor and sensory latencies of the median nerve, and reduced sensory and motor conduction velocities are accepted as diagnostic criteria for

CTS [78]. Even so, some authors have recently reported that optimal diagnostic criteria still remain uncertain [79].

The aims of NCS [3]

(1) to confirm a focal damage to the median nerve inside the carpal tunnel

(2) to quantify the neurophysiological severity by using a scale

(3) to define the nerve pathophysiology: conduction block, demyelination or axonal degeneration

NCS is considered to be the gold standard in the diagnosis of CTS because it is an objective test that provides information on the physiological health of the median nerve across the carpal tunnel. The standard method of diagnosis is comparing the latency and amplitude of a median nerve segment across the carpal tunnel to another nerve segment that does not go through the carpal tunnel, such as the radial or unlar nerve. The nerve is stimulated by a transcutaneous pulse of electricity, which induces an action potential in the nerve. A recording electrode, placed either distally or proximally, detects the wave of depolarization as it passes by the surface electrode [44].

It is more accurate to compare the median nerve response to another nerve segment that does not travel through the carpal tunnel, as opposed to using 'normal' values for the amplitude and latency of individual nerves. This is because there are many factors that may influence the amplitude and latency of an individual nerve, giving a false positive or false negative result. Such factors include age, gender, finger diameter, concurrent systemic disease, obesity and temperature [80-82]. The use of a relative comparison of two nerve segments controls these factors. This is the most sensitive and accurate technique, with a sensitivity of 80-92% and specificity of 80-99% [44]. The study of motor conduction velocity and of distal motor latency (DML) in the median and ulnar nerves in the same hand may provide additional data [65].

However, false negative and false positives can still occur [83,84] possibly due to lack of a standardised diagnostic criteria, resulting in 16-34% of clinically defined CTS being missed with NCS [85]. Moreover, blanket referrals for NCS are an expensive and inefficient approach to the diagnosis of CTS [86]. Another important issue to consider is the fact that many studies have reported that NCS does not change the probability of diagnosing CTS, emphasising importance of clinical history and examination [72].

Nerve Conduction Analysis [3]

The electrophysiological classification [5], in agreement with the AAEM guidelines, follows the neurophysiological progression of CTS severity and includes the following classes:

Negative CTS: Normal findings on all tests (including comparative and segmental studies)

Minimal CTS: Abnormal findings only on comparative or segmental tests

Mild CTS: SCV slowed in the finger-wrist tract with normal DML

Moderate CTS: SCV slowed in the finger-wrist tract with increased DML

Severe CTS: Absence of sensory response in the finger-wrist tract with increased DML

Extreme CTS: Absence of thenar motor response

DIAGNOSIS: OTHER NEUROPHYSIOLOGIC EVALUATIONS

There are several types of clinical neurophysiologic evaluations of the median nerve across the wrist. These include vibrometry threshold testing, current perception testing, symptom questionnaire (hand diagrams), and other quantitative sensory testing (Semmes-Weinstein monofilament testing, tactile sensation and two-point discrimination. These techniques are not as sensitive as NCS because they have considerable subjective components [44].

DIAGNOSIS: ULTRASOUND

The use of ultrasound (US) has been implicated in the diagnosis of CTS because thickening of the median nerve, flattening of the nerve within the tunnel and bowing of the flexor retinaculum are all features diagnostic of CTS [87]. Several studies have concluded that cross sectional area is the most predictive measurement, but there is debate regarding the level within the tunnel that this measurement should be taken, and what constitutes abnormal values [88-90]. The cross-sectional area of the median nerve has been used in US to classify the severity of CTS as normal, mild, moderate and severe [91].

A recent prospective study comparing the diagnostic utility of US versus EDS found that the two techniques had almost equal sensitivities. Sensitivities for EDS and US were 67.1% and 64.7%, respectively. Interestingly, when EDS and US were used together the sensitivity increased to 76.5%, suggesting a role for US as a diagnostic adjunct to EDS. However, a significant flaw is that 23.5% of patients with clinically diagnosed CTS remained undetected [92].

DIAGNOSIS: MAGNETIC RESONANCE IMAGING

Magnetic Resonance Imaging (MRI) is excellent for picking up rare pathological causes of CTS such as ganglion, haemangioma or bony deformity - the presence of which may alter surgical intervention [93]. Furthermore, sagittal images are useful in showing the site accurately and allow the determination of the severity of nerve compression; with a sensitivity of 96%. However, specificity is extremely low at 33-38% [94].

Swelling of the median nerve and increased signal intensity on T2-weighted images [95, 96] indicating accumulation of the axonal transportation, myelin sheath degeneration or oedema [97] are the signs to look out for when diagnosing CTS.

MRI is able to predict those patients who would benefit from surgical intervention, because the length of the abnormal nerve signal on T2-weighted MRI and the median-ulnar sensory latency difference are good predictors of surgical outcome [98]. However, the results do not correlate well with patients' perceived severity of symptoms [98], mainly because MRI provides anatomical information as opposed to information on nerve impairment and function.

Nevertheless, MRI is preferred by patients. Jarvik *et al.,* reported that 76% of their patients found EDS to be unpleasant, whilst only 21% said the same about MRI [98]. On the other hand, it is an expensive procedure, and is therefore not routinely used. It is commonly used in determining the point of nerve entrapment after failure of

Carpal Tunnel Release (CTR), for differential diagnosis in the case of ambiguous symptoms and to confirm the presence of space-occupying lesions [15].

TREATMENT

The treatment of CTS falls under two categories: conservative and surgical. Conservative treatment is generally offered to patients suffering from mild to moderate symptoms of CTS [24]. Options of such treatment include oral and transvenous steroids, corticosteroids, vitamins B6 and B12 [99], nonsteroidal anti-inflammatory drug (NSAIDs), ultrasound, yoga, carpal bone mobilisation and the use of hand splints. O'Connor *et al.,* reported that patients experienced significant short term benefits with this method of treatment, but have concluded that their efficacy in the long term remains unclear. Other conservative treatment options such as magnet therapy, exercise or chiropractic treatment did not show any significant improvement in symptoms when compared to a placebo or control [100].

The use of steroid injections has been under significant scrutiny in research focusing on the conservative treatment of CTS [24]. A recent systematic review by Marshall *et al.,* [39]) reported that steroid injections given to patients with clinical CTS produced a greater clinical improvement in symptoms one month after the injection compared to a placebo. On the other hand, they were unable to show any significant symptom relief beyond one month [101].

Corticosteroid treatment is effective in reducing inflammation and oedema, but there are possible side effects that have to be considered when prescribing them to CTS patients. The main side effect is that it limits reduces collagen and proetoglycan synthesis, thus limiting tenocytes and hereby reducing the mechanical strength of the tendon. This leads to further degeneration [102-105].

Surgical treatment of CTS is in the form of a carpal tunnel release (CTR); a procedure in which the transverse carpal ligament (TCL) is cut to increase the space in the carpal tunnel and hence reduce the interstitial pressure. Approximately 70-90% of patients have good to excellent long-term outcomes following CTR [106].

CTR remains an interesting option for diabetic patients with CTS as well as peripheral neuropathy. In these patients, symptoms would not be expected to be totally relived by CTR since some of their symptoms reflect non-entrapment mechanisms [107].

CONCLUSION

CTS remains one of the most well-known and frequent form of median nerve entrapment, and accounts for 90% of all entrapment neuropathies. This review of the recent literature has provided an overview of this common condition, with an emphasis on the pathophysiology and its relevance to the various methods of diagnosis including nerve conduction studies, ultrasound, and magnetic resonance imaging.

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CONFLICT OF INTEREST

None declared.

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