Carpal tunnel syndrome

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Compression of the median nerve as it enters the palm produces a syndrome of numbness of the radial fingers of the hand, nocturnal hand discomfort and ultimately thenar muscle atrophy. It is usually a chronic condition, more common in females and adults 30–50 years of age.

It can have a sudden onset when trauma produces a sudden elevation of pressure in the carpal tunnel.

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History

Sir James Paget first described median nerve compression at the wrist in 1853. It wasn't until 1930 that surgical release of the transverse carpal ligament was described by Learmouth. Phalen made median nerve compressions at the wrist a well-known entity in the 1950s.

Anatomy

The carpal tunnel has the median nerve and nine flexor tendons passing through it. The scaphoid and trapezium form the medial side; the triquetum, pisiform and hamate are on the ulnar side. The lunate, capitate and proximal metacarpals make up the posterior wall, and the roof is formed by the transverse carpal ligament or flexor netinaculum.

The cross-sectional area (CSA) of the carpal tunnel can be determined by CT scan. CT studies have shown decreased CSA in normal women compared with normal men, in women with carpal tunnel syndrome (CTS) compared to normal women, and workers' dominant hands compared with normal hands, and in the 'available area' to the median nerve in patients with CTS.

Aetiology

The common feature in CTS is a disparity between the size of the carpal tunnel and the volume of its contents. Consequently there is an increase of intra-carpal canal pressure and resultant reduction of median nerve conduction.

Wrist flexion produces an increase in canal pressure, which is greatest when flexor tendons are also tensed. This position of the working hand is commonly reported by patients with work-related CTS.

Trauma may produce a reduction in size of the CT. Fractures of the wrist or carpal bones are associated with CTS.

It is more common that there is an increase in the canal contents, with a resultant increase of pressure. Bone or joint abnormalities such as osteophytes, bursae, or ganglia, may cause this.

Gout, pseudogout, amyloidosis and psoriasis all are associated with CTS. Twenty-five per cent of patients with rheumatoid arthritis have CTS.

There is also a strong association between diabetes and CTS.

Haemorrhage into the canal produces acute CTS. Intra-neural haemorrhage occurs in haemophilia. Benign and malignant tumours (e.g. haemangioma or lipoma) as well as anomalous muscles in the canal can all produce CTS.

Systemic disorders associated with CTS include amyloidosis, myxoedema and diabetes.

Pregnancy and menopause related hormonal changes and fluid retention are associated with increased frequency of CTS.

Work-related CTS is commonly associated with wrist position, frequency of repetition, and lifting requirements. Average lifting over 4kg and repetitive movement is associated with a 30-fold increase in symptoms.

Diagnosis

Most patients with CTS are adults, but children and adolescents are sometimes affected. The dominant hand is most commonly affected, and both hands in half of the cases. Approximately two-thirds of patients are female.

Presenting symptoms are usually numbness and tingling, starting in the middle finger then spreading to adjacent fingers and thumb.

Nocturnal pain often wakes the patient from sleep. There may be clumsiness and weakness. Excessive activity and elevation of the hand may aggravate symptoms.

Reduced response to light touch and vibration are the first sensory alterations noted. Thenar muscle atrophy is usually seen much later.

Percussion over the CT, producing paraesthesia (positive Tinel's sign) indicates axonal sprouting.

Flexion of the wrist for 30 to 60 seconds producing numbness and tingling (positive Phalen's test) is a strong indicator for CTS.

Thenar muscle atrophy is a sign of advanced median nerve compression.

Nerve conduction studies are helpful in diagnosing questionable, borderline or atypical cases.

Differential diagnosis

Compression of the median nerve at the elbow, thoracic outlet syndrome, cervical root compression, peripheral neuropathies and other compressive neuropathies need to be excluded.

Treatment

Conservative management

- Approximately 50% of patients with CTS can be managed with conservative treatment. Underlying causes need to be excluded and treated (diabetes, connective tissue diseases, hypothyroidism).

Modification of patients' work habits

- Attention must be paid to the patients' wrist position when working, and rest periods are important in repetitive work.

Wrist splints

- Nocturnal splinting of the wrist in a neutral position can be most helpful. This is the principal treatment during late pregnancy.

Anti-inflammatories

- Anti-inflammatory medication can be helpful. Injection of steroids into the carpal tunnel can be both therapeutic and diagnostic. Extreme care must be exercised – injection of steroids into the nerve can produce neurotoxicity with severe consequences.

Surgery

- Failure of conservative measures necessitates referral for surgery. So too thenar muscle atrophy indicates advanced nerve compression and need for surgical decompression. Carpal tunnel release is normally performed on an outpatient basis under tourniquet control with local, regional or general anaesthesia. Surgery for CTS can be expected to provide immediate pain relief and stop nocturnal wakening.
- Sensation and nerve conduction improves within two weeks.
- Sensory recovery may be incomplete in 25% of patients, particularly in the elderly or patients with long-standing CTS.
- Persistent weakness is sometimes seen, possibly relating to widening of the polunar arch.

Competing interests

None declared.

Statins and stroke

'...the SPARCL trial is likely to add to the gathering momentum favoring the promotion of ischemic stroke to a "coronary heart disease risk equivalent," the adoption of statin therapy into guidelines for treatment of ischemic stroke, the enforcement of statin therapy on discharge after a stroke as a "quality indicator," and the inclusion of statins in preprinted stroke orders to improve adherence by physicians. Those who might object to this collective-treatment approach to such a heterogeneous disease should be reminded of our abysmal performance as individual doctors taking care of individual patients. In one recent study, even among patients who were eligible for statin therapy according to the ATP II guidelines, only one third had discharge medications that included statins. This finding is especially egregious given the evidence that, as with acute myocardial infarction, hospitalization for stroke provides an excellent opportunity to initiate preventive therapy, and leads to rates of adherence higher than those observed when this therapy is initiated during follow-up. Although we can all agree with the calls for careful science, and although we await the various SPARCL substudies to help clarify some controversies, it does not take recursive subgroup analyses to show that the greatest current risk to patients with ischemic stroke vis-à-vis statins remains gross undertreatment.'

Kent DM. Stroke - An Equal Opportunity for the Initiation of Statin Therapy. N Eng J Med 2006; 355:613-615.