

Ulnar tunnel syndrome: pathoanatomy, clinical features and management

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Abstract

Ulnar tunnel syndrome is compression of the ulnar nerve at the level of the wrist within Guyon's canal. It is most commonly caused by a ganglion cyst but may also be secondary to fractures, inflammatory conditions, neoplasm, vascular anomalies, aberrant musculature or a combination of these.

Assessment should include a detailed history focusing on duration, site and progression of symptoms. The level of compression can be estimated clinically on examination by assessing motor and sensory changes in the hand.

Investigations are used to confirm diagnosis or to clarify the underlying cause. X-rays and computed tomography can be used to exclude fractures. Ultrasound is used to diagnose ganglion cysts and vascular anomalies, and can localise the level of compression. Nerve conduction studies can be used to support the diagnosis and look for proximal compression.

Mild symptoms can be managed non-operatively. Surgical exploration and decompression is the gold standard treatment for neuro-compressive causes with largely good outcomes.

Key words: Ulnar nerve; Compression; Guyon's canal; Hand surgery

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Introduction

Ulnar tunnel syndrome is the clinical picture of compression of the ulnar nerve at the level of the wrist within the canal of Guyon (Dupont et al, 1965). A number of causes have been described, commonly including congenital, traumatic, neoplastic, and inflammatory aetiologies. A lesion of one or more branches of the ulnar nerve within the relatively complex anatomical confines of the ulnar tunnel may give rise to several clinical presentations, which are clinically and anatomically distinct.

Guyon's canal, eponymously named in 1861 by prominent French urologist Jean Casimir Félix Guyon (Guyon, 2006), is a small, superficial, intra-aponeurotic space proximal to the hypothenar area of the hand, which houses the ulnar artery and nerve (Guyon, 2006; Loukas et al, 2007). Hunt (1908) reported several examples of the clinical effects of chronic compression of the ulnar nerve at the wrist, describing three patients with 'occupational neuritis of the deep palmar branch of the ulnar nerve', and later reported hypothenar involvement in five patients.

Since then, various studies have defined the local anatomy in great detail, and the understanding of the pathophysiology of the disease has yielded excellent treatments for this condition, with good results (Waugh and Pellegrini, 2007).

Pathoanatomy

The ulnar nerve, derived from C8, T1 nerve roots of the brachial plexus, lies between the two heads of the flexor carpi ulnaris as it enters the forearm and continues deep alongside the flexor digitorum profundus. In the forearm, it gives off several branches: muscular branches to the flexor carpi ulnaris and the ulnar half of the flexor digitorum profundus, the palmar cutaneous branch and the dorsal cutaneous branch. There is considerable variation in the pattern of motor innervation within the forearm, with between two and five muscular branches described. The main nerve trunk resurfaces at the level of the wrist where it enters Guyon's canal (Bachoura and Jacoby, 2012).

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Figure 1. Surface markings for Guyon's canal. Marked distally on the ulnar side are the hook of hamate, pisiform and the border of the flexor carpi ulnaris.

The entrance to Guyon's canal begins at the proximal edge of the palmar carpal ligament, and is triangular in structure (Bachoura and Jacoby, 2012). The tunnel ends at the fibrous arch of the hypothenar muscle at the level of the hook of the hamate (Figure 1), making it approximately 40–45 mm in length (Ombaba et al, 2010). While the boundaries of the canal vary along its length (Gross and Gelberman, 1985; Ombaba et al, 2010), they are generally considered to be as follows (Gross and Gelberman, 1985; Zeiss et al, 1992; Pierre-Jerome et al, 2011):

- Roof: the volar carpal ligament, palmaris brevis, and hypothenar connective tissue
- Floor: transverse carpal ligament, pisohamate ligament, pisometacarpal ligament, fibres of opponens digiti minimi
- Ulnar border: pisiform bone, abductor digiti minimi, insertional fibres of the tendon of flexor carpi ulnaris
- Radial border: hook of hamate, the transverse carpal ligament.

The relationship of Guyon's canal to the carpal tunnel is close, lying superficial and ulnar to it. Division of the transverse carpal ligament high on the ulnar side will easily guide a surgeon into the carpal tunnel and this close relationship may be why patients with carpal tunnel syndrome often complain of symptoms in all four fingers.

Contents

Guyon's canal contains the ulnar nerve, ulnar artery, associated venae comitantes, and connective fatty tissue (Ombaba et al, 2010; Bachoura and Jacoby, 2012). No flexor tendons pass through the canal although several reports have demonstrated anomalous muscles within the canal (Santoro et al, 2000; Paraskevas et al, 2012). The most common aberrant muscles include accessory abductor digiti minimi, variants of palmaris longus muscle and anomalous flexor digiti minimi, which, although generally asymptomatic, may cause entrapment of the ulnar nerve as they pass over it in Guyon's canal.

The ulnar nerve lies deep and medial to the ulnar artery at its entry to Guyon's canal, and subsequently bifurcates into superficial and deep branches approximately 6 mm (range 3.0–11.5 mm) distal to the pole of the pisiform (Ombaba et al, 2010) (Figure 2). The superficial branches provide motor innervation to the palmaris brevis, and sensory innervation to the hypothenar eminence, ulnar border of the hand, and medial one and a half digits (Ombaba et al, 2010). The deep motor branch courses around the hamate and runs between the abductor digiti minimi, flexor digiti minimi and opponens digiti minimi, supplying all three hypothenar muscles (Bachoura and Jacoby, 2012). It then runs laterally,

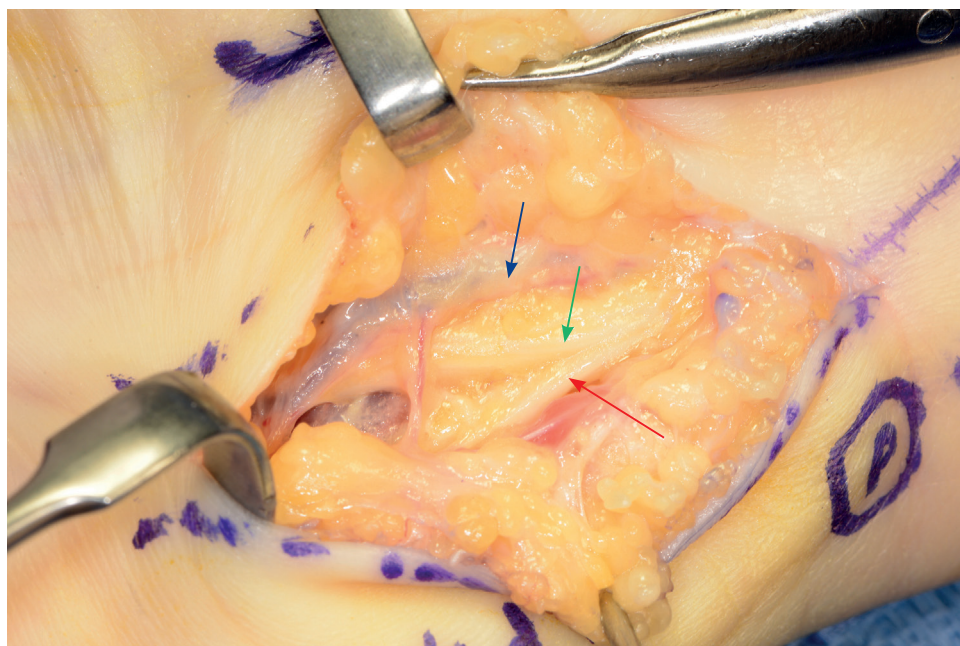


Figure 2. Normal structures within Guyon's canal. Visible are the ulnar artery (blue arrow), the superficial branch of the ulnar nerve (green arrow) and a branch of the superficial ulnar nerve supplying sensation to the ulnar border of the little finger (red arrow). The deep branch of the ulnar nerve has branched proximally, heading in a deep and dorsal direction, so is not visible.

deep to the hypothenar musculature, supplying the third and fourth lumbricals and dorsal interossei, terminating at the adductor pollicis.

Pathology

In 1969, Shea and McLain, and later Gross and Gelberman (1985) described three anatomical zones of the ulnar tunnel. Compression of the ulnar nerve in each of the zones produces a unique constellation of symptoms, and thorough clinical examination can help delineate the level of compression.

Zone 1 compression

This is also called type 1 ulnar tunnel syndrome. Compression of the main ulnar nerve trunk occurs within the ulnar tunnel, before the bifurcation of the ulnar nerve into the superficial and deep branches (Green, 2012). Compression will affect both sensory and motor fibres of the ulnar nerve and clinically manifests as motor weakness of the intrinsic muscles innervated by the ulnar nerve and sensory deficit over the hypothenar eminence and small and ring digits (Bachoura and Jacoby, 2012). Typically, ganglia or hook of hamate fractures cause such lesions (Bozkurt et al, 2005; Waugh and Pellegrini, 2007).

Zone 2 compression

Also called type 2 syndrome, this occurs at the distal end of the ulnar tunnel beyond the nerve bifurcation, radial to zone 3 and affecting the deep motor branch of the ulnar nerve. It includes only motor deficits, affecting the intrinsic muscles of the hand. The sensory branch is spared and thus there is no sensory loss in the distribution of the ulnar nerve. There is marked wasting of the interossei, weakness of the abductor pollicis and clawing of the ring and little fingers. Positive Froment's and Jeanne's signs are seen. Such lesions are most commonly caused by hook of hamate fractures (Bozkurt et al, 2005; Waugh and Pellegrini, 2007).

Zone 3 compression

Zone 3 compression occurs distal to the ulnar nerve motor and sensory bifurcation, ulnar to zone 2. It occurs secondary to compression of the superficial sensory branch of the ulnar nerve, which manifests as an isolated sensory loss (Bachoura and Jacoby, 2012). Such

lesions are most commonly caused by anomalous muscles (Bozkurt et al, 2005; Waugh and Pellegrini, 2007) or ulnar artery thrombosis and aneurysm.

Aetiology of ulnar nerve compression

One study in Finland (Hulkkonen et al, 2020) found the incidence rates for ulnar nerve neuropathies to be 26 and 36 per 100 000 person-years for women and men respectively in the secondary care setting. However, no study to date has looked at the incidence and prevalence of ulnar tunnel syndrome in the broader population including primary care. It is generally accepted that ulnar tunnel syndrome is less common than both carpal tunnel syndrome and cubital tunnel syndrome.

A range of pathologies has been described to cause ulnar tunnel syndrome, including traumatic (eg fractures of the hamate), inflammatory (eg rheumatoid arthritis), neoplastic (eg giant cell tumour of flexor carpi ulnaris), vascular (eg ulnar artery thrombosis) and anatomical variants (eg aberrant thenar or hypothenar musculature).

Ganglia are the most common cause of ulnar tunnel syndrome (Kuschner et al, 1988; Waugh and Pellegrini, 2007), comprising 80% of non-traumatic causes (Hatch, 2020). It has been observed that ganglia that occurred at different anatomical sites correlated with different symptoms (Brooks, 1963). Those arising proximal to the pisohamate ligament presented with both motor and sensory symptoms, whereas those arising distal to this ligament tend to spare sensation and motor function to the hypothenar eminence (Waugh and Pellegrini, 2007). More recently, other authors have described ganglia as a cause of ulnar nerve compression (Erkin et al, 2006; Inaparthi et al, 2008; Kwak et al, 2011).

Anomalous muscles also have significant mass effects on ulnar nerve function in the region of Guyon's canal. These may either be those that are normally found within the hand, or those that are in an unusual place, for example the flexor digiti minimi (Sälgeback, 1977), abductor digiti minimi (Harvie et al, 2004), or palmaris longus and brevis (Waugh and Pellegrini, 2007). One report (Gross and Gelberman, 1985) stated that anomalous muscles are responsible for 16% of cases of ulnar tunnel syndrome.

Clinical presentation

History

Initial history should revolve around the duration and progress of symptoms, as well as the presence of any concurrent joint pain, particularly cervical or elbow pain and trauma. A fall onto the ulnar side of the palm resulting in pain and bruising in the hypothenar area is suggestive of hamate fracture, which often presents late.

Patients with a history of club or racquet sports have a high incidence of hamate fractures, whereas long distance cyclists can undergo ulnar nerve compression as a result of prolonged use of handlebars (Bachoura and Jacoby, 2012), hence the alternative name for ulnar tunnel syndrome being 'handlebar palsy'. Repetitive traumatic activities, such as in manual labour using pneumatic drills, can damage the ulnar artery and produce a 'hypothenar hammer syndrome' (Marie et al, 2007).

Examination

Physical examination should first rule out more proximal sites of nerve entrapment, such as cubital tunnel syndrome or cervical causes, by examining the cervical spine, brachial plexus and the elbow. The ultimate aim is to try and localise the site of neural compression relative to the wrist, and more specifically whether the pathology is motor, sensory or mixed. Normal sensation on the dorsoulnar part of the hand with associated ulnar nerve symptoms suggests distal compression.

It is worth noting that patients recovering from ulnar nerve palsy at the elbow may present with similar signs distally with Tinel's sign no longer strongly positive at the elbow and an advancing Tinel's sign towards the wrist. In these cases, the history will differentiate the causes.

Inspection of patients with ulnar tunnel syndrome should rule out gross causes of compression such as ganglia and obvious fractures. Clawing of the ring and little fingers

may be present in lesions affecting the deep branch of the ulnar nerve, as a result of paralysis of the intrinsic muscles of the hand (adductor pollicis, deep head of flexor pollicis brevis, interossei and fourth and fifth lumbricals), but may also occur in a recovering high ulnar nerve palsy. Hypothenar atrophy, guttering between the metacarpals and loss of bulk in the first web space will also be seen if the motor branch is involved.

On palpation, point tenderness over the hook of hamate would imply a hamulus fracture, which should be high on the differential list of a patient presenting with ulnar tunnel syndrome (Sälgeback, 1977; Bachoura and Jacoby, 2012). Clinical presentation of hook of hamate fractures is often non-specific and requires a high index of clinical suspicion. The site of maximal pain does not always correlate with the area of fracture, and can be misleading.

Palpating any masses may help diagnose their composition. A systematic wrist examination may be warranted if there is pain and tenderness around the wrist joint itself.

In pure sensory deficits, motor examination will be unremarkable. Patients with motor compromise will have paralysis of the intrinsic muscles of the hand. This will result in weakened grasp as a result of alteration in rollup of their interphalangeal joints and/or a weak pinch caused by weakened thumb adduction and the index finger's ability to resist pinch with abduction.

Special tests

There is a variety of eponymous tests used to identify motor dysfunction caused by ulnar nerve compression (Table 1). These tests may be useful in aiding the diagnosis of ulnar tunnel syndrome, when used alongside other more specific tests for ulnar nerve pathology. Davidge et al (2015) described a 'hierarchical scratch-collapse test' to evaluate the site of ulnar nerve compression. The patient sits with flexed elbows and forearms in neutral and resists gentle internal rotation of the both arms by the examiner, who then scratches the skin of the patient over the suspected area of neural compression. A positive test results in loss of resistance and a 'collapse' of the affected arm into internal rotation. The hierarchical scratch-collapse test uses cold ethyl-chloride spray as an alternative skin stimulus, and the examiner uses the spray to sequentially evaluate all sites of ulnar nerve compression, from the elbow to the hypothenar eminence, until a positive scratch-collapse test response is elicited (Davidge et al, 2015).

Table 1. Clinical tests for examination of compression neuropathy within the ulnar tunnel

Muscle	Test
Adductor pollicis	Froment's sign: interphalangeal joint flexion compensates for a weak adductor pollicis muscle when grasping a flat object
	Jeanne's sign: hyperextension of the metacarpo-phalangeal joint of the thumb if there is a coexisting volar plate laxity of the metacarpo-phalangeal joint with a positive Froment's sign
	Bunnell's O sign: created with thumb to index pulp to pulp pinch
Interossei	First dorsal interosseous screening test: weak dorsal interossei against resistance
	Crossed finger test: inability to cross index and middle fingers
	Wartenberg's sign: the small finger lies abducted and cannot adduct and touch the ring finger
	Masse sign: flattened metacarpal arch as a result of interosseal atrophy
Lumbricals	Duchenne's sign: clawing of the ring and index fingers as a result of intact extrinsics and denervated intrinsics
	Roll up pattern of flexion: when making fist, distal inter-phalangeal joints flex fully first, then proximal inter-phalangeal joints pushing objects away as a result of loss of control from lumbricals

Investigations

Investigations in patients with ulnar tunnel syndrome are used predominantly for confirming diagnosis or to clarify the underlying cause.

Laboratory-based investigation

Blood tests have little significance in definitive diagnosis of ulnar tunnel syndrome but may be used to exclude other inflammatory or autoimmune wrist pathologies such as rheumatoid or septic arthritis.

Imaging-based investigations

Plain radiographs and computed tomography scans are useful in evaluating hook of hamate fractures. Owing to the overlap with other carpal bones, standard anteroposterior wrist radiographs can be of limited value (O'Grady and Hazle, 2012). Carpal tunnel, supinated oblique and lateral views with thumb abduction and wrist radial deviation can improve visualisation of the hook of the hamate.

Computed tomography is considered the reference standard for hook of hamate fractures and has a sensitivity close to 100% (Andresen et al, 1999).

Ultrasound scanning is useful in ulnar tunnel syndrome because of its non-invasiveness; it is cost effective (Ginanneschi et al, 2009). Ulnar nerve impingement will show ulnar nerve oedema at the wrist. Ultrasound scans can also be used to identify any aberrant muscles around Guyon's canal, exclude cubital tunnel syndrome, assess for ulnar artery thrombosis as well as identify ganglions or soft tissue tumours such as lipomas.

Arteriography may be appropriate where ulnar artery pathology is suspected if Doppler studies give insufficient information.

Magnetic resonance imaging is useful for evaluating and delineating soft tissue masses such as ganglion cysts, aberrant muscle and vascular lesions, and can be helpful in preoperative planning (Zahrawi, 1984; Subin et al, 1989; Ruocco et al, 1998; Chen et al, 2011).

Other investigations

Electromyography and nerve conduction studies may be considered after positive motor and sensory findings to confirm a diagnosis of nerve compression and to identify the level of compression.

Conduction velocity should be recorded both across the elbow and the wrist to exclude cubital tunnel syndrome. At the wrist, results are improved by using the two-channel test, with simultaneous study of motor conduction to both the abductor digiti minimi and first dorsal interossei muscles (Cowdery et al, 2002; Akuthota et al, 2005; Seror, 2013).

In zone 1 compression, latencies to the abductor digiti minimi and first dorsal interossei are prolonged, with diminished evoked sensory response (Chen and Tsai, 2014).

In zone 2 compression, there is prolonged latency of the muscles distal to the lesion in zone 2. First dorsal interossei will always be involved, but according to the site of the lesion, abductor digiti minimi may be spared, as its nerve supply may separate early from the motor branch.

In zone 3 compression, abductor digiti minimi and first dorsal interossei conduction velocities will be unaffected and will show only diminished sensory responses in the palmar distribution of the ulnar nerve (Campion, 1996; Cavallo et al, 1988). This may sometimes only be detected by inching techniques.

Management

Conservative

As with compression of other peripheral nerves, mild symptoms in ulnar tunnel syndrome or symptoms without an identified structural abnormality can be treated conservatively. However, there is little evidence regarding the effectiveness of conservative treatment (Bachoura and Jacoby, 2012). The mainstay of conservative treatment is activity modification, splinting and non-steroidal anti-inflammatory drugs. Activity modification is particularly effective when the symptoms are caused by repetitive trauma or compression (Patterson et al, 2003; Zambelis et al, 2005; Ginanneschi et al, 2009).

Ganglion aspiration alone has been successfully used to treat ulnar tunnel syndrome (Nakamichi and Tachibana, 2003), but there is limited evidence of the use of steroid injections into Guyon's canal for idiopathic ulnar tunnel syndrome.

Operative

If the cause of ulnar tunnel syndrome is neurocompressive, as a result of a mass or another structural lesion within the ulnar tunnel, or conservative treatment has failed, then the patient should be offered surgery. Surgical exploration and ulnar nerve decompression form the basis of treatment (Kaiser et al, 2012). Guyon's canal should be explored and the ulnar nerve and artery should be identified. The pisohamate hiatus must be decompressed and this is achieved by releasing the tendinous pisohamate arcade, which forms the floor of the hypothenar muscles (Kaiser et al, 2012). The principal branches of the ulnar nerve should then be identified and traced from proximal to the wrist to the bifurcation, and in particular identifying the deep motor branch, which often plunges posteriorly away from view, deep to the main trunk of the ulnar nerve within the canal. Soft tissue obstructing any branch of the ulnar nerve should be released, and any mass arising from muscle, connective tissue (eg giant cell tumour), nerve or artery should be excised under loupe magnification or the microscope as necessary.

Conclusions

While ulnar tunnel syndrome is not as common as some other neurocompressive syndromes in the upper extremity, such as carpal or cubital tunnel syndrome, recognition of the clinical signs and symptoms, and appropriate investigation, are instrumental in accurate diagnosis and prompt treatment. Understanding the anatomy of the ulnar nerve and its variations is vital in correlating symptoms with the site of compression and guiding investigation and subsequent management.

Organic mass lesions such as ganglia and giant cell tumours are the most common causes of compression and should be considered during initial assessment. However, an important alternative is hook of hamate fractures. They can be easily missed, and investigations are dictated by clinical history and examination findings, but can include plain radiographs, ultrasound, further imaging such as computed tomography and magnetic resonance imaging, and electromyography.

Although conservative management is successful in mild cases, surgical decompression of Guyon's canal remains the gold standard.

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Key points

- Ulnar tunnel syndrome is compression of the ulnar nerve at the level of the wrist, within Guyon's canal.
- Thorough clinical examination can help determine the level of the compression.
- Imaging, including X-rays, ultrasound and computed tomography, can be used to exclude alternative diagnoses.
- Mild symptoms can be managed non-operatively.
- Surgical exploration and decompression is the gold standard treatment for neuro-compressive causes.

Conflicts of interest

The authors declare no conflicts of interest.

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