Ulnar Tunnel Syndrome

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Learning Objectives

- Clarify the anatomy of the ulnar nerve in the Guyon canal.
- List the zones of ulnar nerve compression in the wrist.
- Elucidate the pathophysiology of ulnar tunnel syndrome (UTS).
- Discuss the clinical presentation of UTS.
- Describe the management options for UTS.

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Ulnar neuropathy at or distal to the wrist, the so-called ulnar tunnel syndrome, is an uncommon but well-described condition. However, diagnosis of ulnar tunnel syndrome can be difficult. Paresthesias may be nonspecific or related to coexisting pathologies, such as carpal tunnel syndrome, cubital tunnel syndrome, thoracic outlet syndrome, C8–T1 radiculopathy, or peripheral neuropathy, which makes accurate diagnosis challenging. The advances in electrodiagnosis, ultrasonography, computed tomography, and magnetic resonance imaging have improved the diagnostic accuracy. This article offers an updated view of ulnar tunnel syndrome as well as its etiologies, diagnoses, and treatments. *(J Hand Surg Am. 2014;39(3):571–579. Copyright © 2014 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Ulnar tunnel, Guyon canal, pisohamate hiatus, ulnar neuropathy, ulnar nerve compression.

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ULNAR TUNNEL SYNDROME (UTS) is defined as a compressive neuropathy of the ulnar nerve at the level of the wrist, which was described in 1965 by DuPont et al,[1] concerning 4 patients with acquired ulnar neuritis. The ulnar tunnel proper, also known as *Guyon canal*, is 1 potential but not exclusive site of ulnar nerve compression. The eponym is derived from the description in 1861 by Guyon of a space at the base of the hypothenar eminence at which the ulnar nerve bifurcates and is vulnerable to compression from surrounding structures. Clinical presentation depends on the anatomical zone of compression and, therefore, may be purely motor, purely sensory, or both. In selected cases, nonoperative treatment such as activity modification may be helpful, but surgical exploration of the ulnar tunnel with decompression may be indicated.

ANATOMY

The ulnar nerve

The ulnar nerve emerges from the medial cord (C8−T1) of the brachial plexus and passes through the axilla into the anterior compartment of the arm, then pierces the intermuscular septum and travels in the posterior compartment medially. It then courses superficially and passes posterior to the medial epicondyle into the cubital tunnel. The nerve then continues in the forearm between the flexor carpi ulnaris (FCU) and the flexor digitorum profundus (FDP) and gives innervation to the FCU and the FDP of the ring and small fingers. Before its entrance into the ulnar tunnel, about 8.3 cm proximal to the pisiform, the ulnar nerve gives off the dorsal cutaneous branch that innervates the dorsoulnar side of the hand.[2]

The ulnar tunnel

This tunnel originates at the proximal edge of the palmar carpal ligament and extends distally to the fibrous arch of the hypothenar muscles at the level of the hook of the hamate. The ulnar nerve extends approximately 40 to 45 mm in its path through the tunnel. The boundaries of the tunnel vary along its entire course and the 4 walls are not distinct.[3,4] The roof of the tunnel consists of the palmar aponeurosis, palmaris brevis, and hypothenar fibroadipose tissue; the floor is composed of the FDP tendons, transverse carpal ligament, pisohamate ligament, pisometacarpal ligament, and opponens digiti minimi; the medial wall is made up of the FCU tendon, the pisiform, and the abductor digiti minimi; the lateral wall is formed by the extrinsic flexor tendons, the hook of the hamate, and the transverse carpal ligament.[4−6]

Within the canal lie the ulnar nerve, ulnar artery, concomitant veins, and connective fatty tissue.[1,3] The ulnar nerve lies slightly deep and ulnar to the ulnar artery (Fig. 1). During its course in the fibro-osseous tunnel, the ulnar nerve bifurcates into a superficial and a deep branch approximately 6 mm distal to the distal pole of the pisiform.[2,3] The superficial branch innervates the palmaris brevis and provides sensation to the hypothenar eminence, small finger, and ulnar aspect of the ring finger.

Pisohamate hiatus

The motor branch of the ulnar nerve exits the canal by coursing around the ulnar edge of the hook of hamate and runs radially between the abductor digiti minimi (ADM) and flexor brevis digit minimi and dorsal to the flexor tendons of the small finger.[3,4,7]

The flexor digiti minimi brevis muscle originates from the pisiform bone and the hook of the hamate. These origins are bridged by a stout, concave musculotendinous arch, first described by Hayes et al as pisohamate arch.[4−6,8] This arch may serve as a common origin of the hypothenar muscles (Fig. 2). The pisohamate arch forms the roof and the pisohamate ligament forms the floor of a narrow ovoid opening in the exit of the Guyon canal. Uriburu and coworkers[9] termed this opening the pisohamate hiatus, through which the deep branches of the ulnar nerve and artery leave the Guyon canal and enter the deep palmar space, which was also observed by McFarlane et al.[10] At the level of the pisohamate hiatus, the deep motor branch courses dorsally over the distal border of the pisohamate ligament and beneath the arch of the flexor brevis digiti minimi muscle. It then turns radially around the hook of hamate and passes underneath the flexor brevis digiti minimi and opponens digiti minimi muscles.

The branches innervating the abductor digiti minimi, flexor brevis digiti minimi, and opponens digiti minimi muscles arise from the deep branch of the ulnar nerve, but the branch to the ADM may arise either proximal or distal to the pisohamate arch.[8] In most cases, the branch to the ADM takes off proximal to the pisohamate arch and travels superficial to the arch; therefore, severe compression at the level of pisohamate hiatus causes weakness of the interosseous muscles but may spare the ADM.

ZONES OF COMPRESSION

Compression of the ulnar nerve at the wrist is not limited to the Guyon canal. In 1969, Shea and McClain[7] described 3 different types of ulnar nerve compression syndromes at the wrist based on the

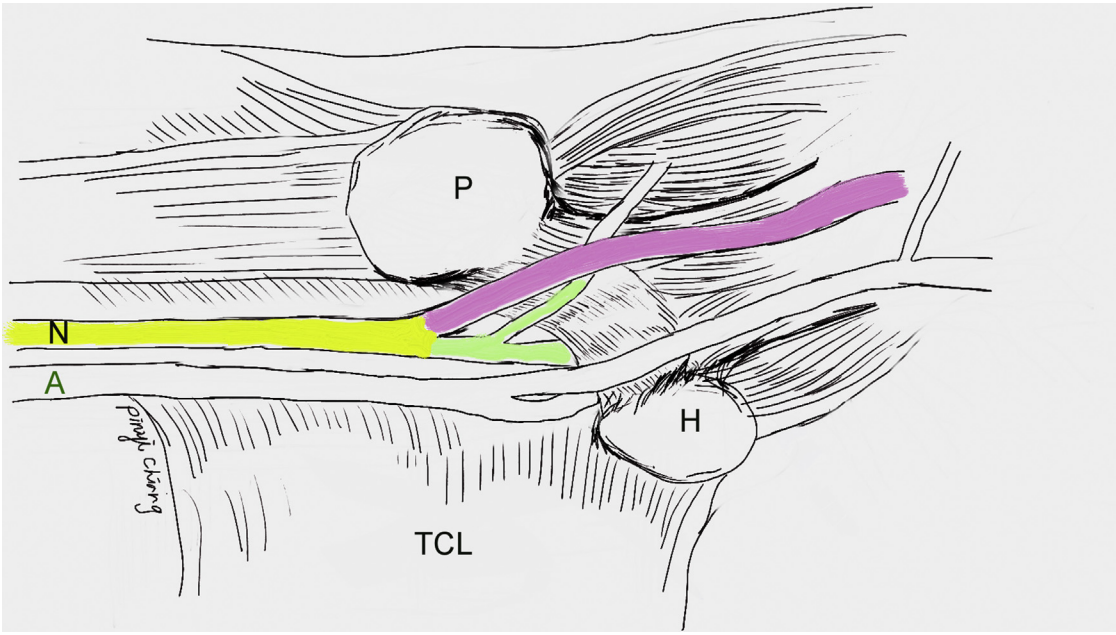

FIGURE 1: This diagram depicts the course of ulnar nerve at the right wrist, which is ulnar to the ulnar artery. The yellow color highlights the zone I, the green highlights the zone II, and the purple highlights the zone III. A, ulnar artery; N, ulnar nerve; P, pisiform; H, hamate; TCL, transverse carpal ligament.

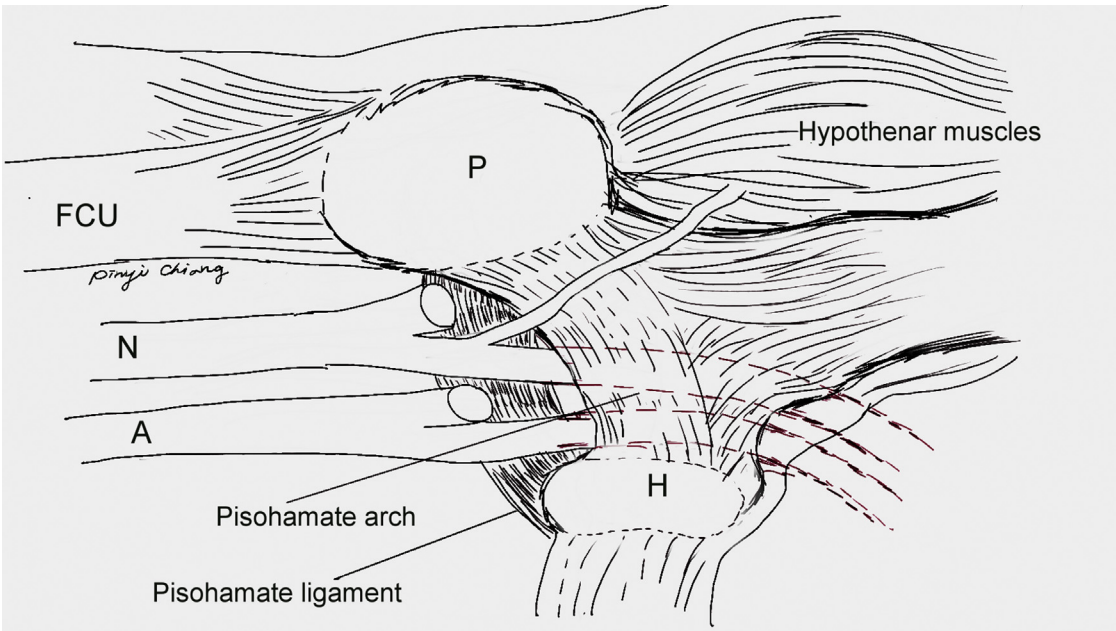

FIGURE 2: In this diagram, the superficial branch of the ulnar nerve was cut to show a better relationship of deep branch of ulnar nerve and the pisohamate arch. Note that the deep branch of ulnar nerve passes in between the pisohamate ligament and the pisohamate arch, and right after its takeoff from the ulnar nerve proper, there is a branch going to the hypothenar muscles. The pisohamate arch serves as a common origin of the hypothenar muscles, mainly the flexor brevis digiti mini. A, ulnar artery; N, ulnar nerve; P, pisiform; H, hamate; FCU, flexor carpi ulnaris.

anatomical course of the nerve. Later, Gross and Gelberman's cadaveric study[4] delineated the relationship between the symptoms and 3 anatomical zones of ulnar nerve compression at the wrist (Figs. 1 and 2).

Zone I

Zone I compression[4,7] refers to nerve compression proximal to or within the Guyon canal, before bifurcation of the ulnar nerve into the superficial and deep

branches. Zone I transmits both the sensory and the motor fascicles of the ulnar nerve, with the sensory fibers lie palmar to the motor fibers. Compression at this zone manifests as motor weakness of all the ulnar-innervated intrinsic muscles and sensory deficits over the hypothenar eminence and the small and ring digits.

Zone II

Zone II compression affects exclusively the motor branch distal to the bifurcation of the ulnar nerve. It manifests as motor weakness of the ulnar-innervated intrinsic muscles with intact sensation along the ulnar nerve distribution.

Compression at zone II may occur where the deep motor branch exits the Guyon canal at the level of the pisohamate hiatus after the branch to the abductor digiti minimi takeoff. Symptoms of compression at this zone thus manifest as weakness in interosseous muscles with possible sparing of the hypothenar muscles.[8–10]

Zone III

Zone III compression stems from compression of the superficial branch of the ulnar nerve; thus manifesting as sensory loss without hypothenar and interosseous weakness. Unlike the deep motor branch in zone II, which is purely motor, the superficial branch contains a few motor and many sensory fibers. It gives 2 motor branches to palmaris brevis right after its takeoff from the ulnar nerve proper, and then it becomes the proper digital nerve to the ulnar side of the small finger and the common digital nerve to the radial side of the small finger and the ulnar side of the ring finger. Because the palmaris brevis functions merely to wrinkle the skin on the ulnar aspect of the palm, its loss is seldom noticed by the patient and is difficult to detect clinically.

A lesion in zone III will produce sensory symptoms on the palmar aspect of the small finger and the palmar-ulnar side of the ring finger. Zone III lesions are most commonly caused by anomalous muscles or thrombosis of the ulnar artery.[4] Purely sensory symptoms can also be caused by a lesion in zone I that compresses only the palmar aspect of the nerve.

CAUSES OF ULNAR TUNNEL SYNDROME

The true incidence and prevalence of UTS are not clear. It is generally accepted that the incidence of UTS is much less than that of either carpal tunnel syndrome (CTS) or cubital tunnel syndrome. Numerous factors may cause UTS, and most publications are case reports describing various eiologies.[11–23]

Shea and McClain[7] reported that ganglia and occupational neuritis were the 2 leading causes of UTS. During the past decades, although reports on UTS secondary to occupational activities were scarce,[24] chronic repetitive trauma or compression over the hypothenar eminence has been implicated as a cause of UTS[25] and is not uncommon among long-distance cyclists.[26,27] Other causes include benign lesions, hook of hamate fractures, ulnar artery pathology or aberrancy, anomalous hypothenar muscles, and crystal deposition disease. Idiopathic cases have also been reported.[22]

Ganglion cysts and tumors

Ganglia are one of the most common cause of UTS,[28] as demonstrated by numerous reports.[1,7,18,22,29–31] In 1952, Brooks[32] described 3 patients who had intrinsic weakness and sensory deficits to the ulnar territory caused by ganglion cysts, and Seddon[33] also described a series of patients with ganglia compressing the deep branch of the ulnar nerve. In 1963, Brooks[34] further mentioned that symptoms may vary based on the location of the ganglia.

In 1968, Vanderpool et al[35] published 21 cases of ulnar nerve compression at the wrist, with 13 caused by ganglion cysts. Seven of the cases had ganglia located in zone I with motor and sensory deficits; 5 ganglia were in zone II distal to the takeoff of the branch to the hypothenar muscles and produced motor weakness to the interossei only.

Ganglia can be found in all zones of the ulnar tunnel and produce symptoms according to their anatomical location. Tumors can also produce similar symptoms. They can be found in all zones of the ulnar tunnel, and patients usually recover well after surgical removal.[36–39]

Anatomical abnormalities

Anomalous muscles are thought to be responsible for 16% of cases of UTS.[4,35,40–44] These muscles can compress the nerve in any of the 3 zones and produce symptoms accordingly.

Anomalous muscles either can be hypertrophic with normal anatomy or can have an unusual location. Pathoanatomical flexor digiti minimi,[45] ADM,[35,40,43,46] palmaris longus or brevis[47,48] have been described. A recently named hypothenar adductor muscle that was found deep to the palmaris brevis with insertion to the hypothenar fascia has also been reported.[42]

Other less common aberrant anatomies that can cause UTS include abnormally thickened ligaments,[4,41] an anomalous course of the ulnar nerve,[49] and carpal bone abnormalities.[35] Detailed 3-T magnetic resonance imaging (MRI) has been proposed to

be an excellent modality for evaluation of the aforementioned aberrant structures.[5]

Hypothenar hammer syndrome

Occupations that require the use of impact or vibratory tools (eg, jackhammers or other hand-held heavy equipment) with the wrist in extension, can lead to UTS.[50,51] Described by von Rosen in 1934,[52] hypothenar hammer syndrome results from direct trauma to the ulnar artery in its confined and vulnerable position between the hook of the hamate and the palmaris brevis and subcutaneous tissue. A direct blow causes the "hammering" of the artery against an anvil, the hook of the hamate. Repetitive trauma to the artery results in thrombosis and occlusion, followed by ischemia and related ulnar nerve symptoms.[1,53]

Cyclist's palsy

Repetitive and prolonged pressure to the hypothenar area by the handlebar in cyclists can also cause UTS. Depending on the anatomical location of nerve compression, the syndrome can be associated with pure motor deficits, pure sensory disturbances, or both.[26,27,54,55]

Fractures or dislocations

Fractures of the distal radius, hamate, trapezium, pisiform, and the base of the ring and small finger metacarpals can cause ulnar nerve palsy. In dorsally displaced distal radius fractures, there can be traction to the ulnar nerve. The swelling after the injury is another cause of compression to the nerve. However, direct damage to the nerve can happen, as with some fractures of the hook of the hamate.[56–62]

Carpal tunnel syndrome

One fifth to one third of patients with CTS have concurrent UTS.[63,64] By analyzing the relationship between the intensity of electrical stimuli and the size of motor response in CTS patients with negative ulnar nerve electrodiagnostic tests, Rossi et al[65] demonstrated that the pathological change of the ulnar nerve was contingent to the severity of CTS. In CTS, high pressure in the carpal tunnel may result in anatomical changes of the ulnar nerve with subsequent functional impairment. Carpal tunnel release (CTR) appears to reverse some symptoms,[66] resulting in a change of the shape and an increase in the volume of both the carpal and the ulnar tunnels.[67,68] These morphological changes correspond to a decrease in pressure in both anatomical spaces.[69] Ninety percent of these patients show improvement in their ulnar nerve symptoms after CTR. For this reason, operative release of the ulnar tunnel is infrequently indicated in these patients. Several studies disagree on the association between CTS and UTS.[69–74] The carpal and ulnar tunnels lie adjacent to each other, with the transverse carpal ligament constituting the roof of the carpal tunnel and the floor of the ulnar tunnel. Pressure within the carpal tunnel is transmitted to the ulnar tunnel, and vice versa. To completely decompress the motor branch of the ulnar nerve, the transverse carpal ligament needs to be sectioned distally beyond the hook of the hamate, as the motor branch runs on it. In Silver et al's series of 59 hands with CTS,[72] concurrent ulnar sensory deficiencies were found in 34% of cases. After CTR only, 94% of their patients had sensory improvements in the ulnar territory. Ablove et al[69] measured the pressure changes in the carpal and ulnar tunnels before and after endoscopic and open CTR. Following the release, pressure dropped significantly in both tunnels. They concluded that CTR alone could be used to treat patients with concurrent sensory deficit.

Other causes

Iatrogenic causes of UTS include distraction leading to neurapraxia or direct trauma of ulnar nerve during wrist arthroscopy has been reported.[75] Direct damage to the nerve during opponensplasty or other tendon transfers has been mentioned.[76,77] Pingree et al[78] reported a case of UTS following CTR owing to herniation of contents of the carpal canal.

Thermal burns may cause direct damage to the nerve, or the subsequent scar contracture may be responsible for UTS.[79] Rheumatoid arthritis and osteoarthritis may account for 3% of UTS because of associated edema, tenosynovitis, or bony deformities.[22,80,81] There is a report of edema secondary to an insect bite causing UTS.[82]

CLINICAL PICTURE

Common complaints

Patients with UTS usually complain of numbness and tingling in the small and ring fingers, weakness of grip strength, or ulnar-sided pain. Information on cervical and other peripheral joint pain (particularly elbow pain or trauma), the duration and progress of symptoms, aggravating and relieving scenarios should be clarified. If the patient can tell that the dorsal ulnar side of the hand feels normal, implying that the dorsal sensory branch is not involved, then a suspicion of ulnar tunnel syndrome should be raised.

History

The current history may include a recent injury, sports or occupations that include repetitive trauma or stress

to the hypothenar area, or long-term use of vibratory tool. Patients who play sports with a club or racquet can suffer from hamate fractures,[83,84] whereas long-distance cyclers may acquire UTS as a result of prolonged grasping of the bicycle's handlebars and direct pressure on the wrist.[26,27] Manual labor that requires constant hammering or repetitive traumatic activities may cause hypothenar hammer syndrome.[25]

The past history including rheumatoid arthritis, osteoarthritis, diabetes mellitus, or other disorders associated with peripheral neuropathy should be documented. Because UTS can be associated with CTS and usually resolves with release of transverse carpal ligament, the history should include details related to both CTS and UTS.

Physical examination

An initial step is observation for hypothenar or interossei wasting, clawing, the inability to cross fingers,[1,14,23,85] or any masses over the dorsal or volar wrist. Palpation may help in identifying the type and location of a lesion. Point tenderness over the hook of the hamate is a sign of hamulus fracture. Hardness and consistency of a lesion may provide clues about its nature.

Proximal sites of nerve entrapment should be ruled out, including cubital tunnel, thoracic outlet, and cervical radiculopathy. If possible, the examiner should try to localize the zone of compression within the wrist. CTS should also be ruled out because of the likelihood of concurrent presentation. Phalen's test and elicitation of Tinel's sign over the pisiform, the cubital tunnel, and carpal tunnel are common tests,[86] but their sensitivity and specificity are not clear. Grundberg[87] reported his 31 cases with zone 1 compression (involvement of both motor and sensory components of the ulnar nerve) and found that 92% of the patients had a positive Phalen test, whereas only 44% of the patients had a positive Tinel sign.

Objective sensory tests, including the Semmes-Weinstein monofilament test and 2-point discrimination, provide baseline information and severity of the disease. Paresthesias on the dorsoulnar side of the hand imply that the site of compression is proximal to the takeoff of the dorsal sensory branch of the ulnar nerve. In this scenario, the most likely site of compression is the cubital tunnel, and pinwheels can be used to define the region of paresthesia with better accuracy.

Objective tests that examine intrinsic and extrinsic motor function in both the median and the ulnar distributions, should include the first palmar and dorsal interossei, the lumbricals to the ring and small fingers, the hypothenar muscles (ADM, flexor digiti minimi, and opponens digiti minimi), and abductor pollicis brevis. Sometimes the hypothenar muscles are spared in cases in which the site of compression is distal to the takeoff of the hypothenar branch of the deep motor nerve. Comparison of grip and pinch strength to the opposite side should be recorded.

Vascular examination of the wrist is useful because UTS can be associated with ulnar artery pathology.[13,14,19,22] The vascular examination should include assessment of the radial and ulnar pulses as well as Allen's test to confirm the patency of the ulnar artery. If any pulsatile lesion is suspected, Doppler examination is quick and helpful in the clinic. Bruits or thrills over the ulnar artery may indicate pseudoaneurysmal dilation, and further vascular image study should be arranged.

DIAGNOSIS

Radiographic studies should include the standard posteroanterior and lateral views. If a hamulus fracture is suspected, a radiograph with carpal tunnel view or hook of hamate view or computed tomography (CT) is helpful. For localizing and characterizing soft tissue masses, ganglia, aberrant muscle, and vascular lesions, MRI provides useful information for preoperative planning.[14,18,88,89] The use of ultrasound in UTS has not been fully investigated, but Harvie et al[46] screened 58 asymptomatic volunteers (116 wrists) to determine the prevalence and morphology of anomalous muscles within the ulnar tunnel and found that 47% had aberrant ADM. But the definition of anomalous muscles was unclear. Ultrasound is noninvasive and cost-effective to help diagnosis,[20] and in select cases, it may aid in ganglion aspiration.[30] Arteriography is appropriate when ulnar artery pathology is suspected.

Electromyography and nerve conduction test are used as confirmatory studies in cases of positive physical findings to confirm the diagnosis and to localize the level of neuropathy. Data should include conduction velocity across the elbow as well as the wrist to rule out cubital tunnel syndrome and to serve as an internal control. Several studies suggested that electrodiagnostic yield can be improved by simultaneous study of motor conduction to the ADM and first dorsal interosseous (FDI), the so-called 2-channel test, which has emerged as a method to assess the segment of the ulnar motor nerve between the hypothenar branch and the FDI branch.[27,64,90] In zone I compression, the conduction velocity across the elbow is normal, but the latencies to the ADM and FDI are prolonged, and evoked sensory responses are diminished. In zone II compression, there

is normal conduction across the elbow and prolonged latency of the muscles distal to the lesion in zone II. According to the site of compression, the ADM may be spared, but the FDI is always involved. FDI and ADM conduction velocities are not impaired in zone III lesions and will show only diminished evoked sensory responses in the palmar distribution of the ulnar nerve.[91,92]

TREATMENT

Nonsurgical treatment

Nonsurgical intervention is indicated in mild cases without motor deficit. Usually, these cases are caused by repetitive trauma without an organic lesion such as fracture, ganglion, or thrombosis of the ulnar artery. Protective braces and nonsteroidal anti-inflammatory medications may help in addition to discontinuation of provocative activities. Bicyclists whose UTS stems from the position of the hands on the handlebars may improve simply by refraining from riding.[26,27,54]

Generally, activity modification has been shown to be effective when the cause of UTS is related to repetitive compression.[20,24,26] For UTS caused by ganglion cysts, successful treatment by aspiration alone has been reported[30]; however, surgical treatment is still preferred by most surgeons.

Surgical treatment

Surgery is indicated when there is an organic compressive lesion, a motor deficit, or failure of conservative treatment. Surgical exploration, removal of lesion, and decompression of the ulnar tunnel as well as the pisohamate hiatus is the standard surgical treatment. In cases with definite organic lesions, decompression of the ulnar tunnel should still involve exposure of the nerve from the distal forearm to the pisohamate hiatus. This approach is necessitated by the possibility of multiple sites of compression as well as the ambiguity presented by some zone I lesions. Cubital tunnel release is also indicated in the cases with motor deficit when their pathology cannot be clearly differentiated between UTS and cubital tunnel syndrome.

For patients with UTS related to acute fracture of the wrist, if dysesthesia does not improve within 24 to 36 hours after reduction, surgical exploration with decompression should be considered. In the event of UTS that presents after reduction or operative procedure but not in the beginning, immediate surgical exploration is recommended.

DISCUSSION

UTS is not as common as CTS or cubital tunnel syndrome, and as our understanding to its anatomy as well as improvement in diagnostic tools increases, the cause can be differentiated and localized more accurately. Organic lesions are the most common cause of compression. Thus, these etiologies should be kept in mind upon initial evaluation. Although conservative management can be successful in some cases, the mainstay of treatment for severe cases remains surgical. Reported outcome have yielded good results, although, at this time, large case series or comparative studies have not been published.

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JOURNAL CME QUESTIONS

Ulnar Tunnel Syndrome

Which of the following statements is most accurate regarding the anatomy of the pisohamate hiatus?

a. It is a passage for the superficial sensory branch of the ulnar nerve
b. The superficial arch of the hiatus gives origin to the thenar muscles
c. The pisohamate arcade forms the floor of the hiatus
d. The pisohamate ligament forms the roof of the hiatus
e. The abductor digiti minimi muscle branch travels superficial to the hiatus.

Which of the following is the most common cause for ulnar tunnel syndrome?

a. Lipoma
b. Thickened volar carpal ligament
c. Palmaris brevis muscle
d. Ganglion cyst
e. Iatrogenic from opposition Huber transfer

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