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ULNAR NEUROPATHY AT ELBOW

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Abstract:

Introduction: *In all disciplines and subspecialties, all surgeons who are treating complex elbow diseases must be aware of the principles in assessment and treatment of both acute and chronic ulnar neuropathies. Management of chronic compression neuropathy of the ulnar nerve is still considered a complex and a challenging aspect that faces physicians. Appraisal of the literature via systematic reviews and meta-analyses demonstrates that the best management for patients with ulnar neuropathy is still not clear. Understanding of the subtleties in clinical manifestations and thorough consideration of the timing and type of surgical interventions are essential to optimize results following management of ulnar neuropathy. In addition, treatment of acute ulnar nerve injuries has been recently altered following the introduction of advanced nerve transfer techniques.****Aim of work:*** *In this review, we will discuss Ulnar neuropathy at elbow****Methodology:*** *We did a systematic search for Ulnar neuropathy at elbow in the emergency department using PubMed search engine (http://www.ncbi.nlm.nih.gov/) and Google Scholar search engine (https://scholar.google.com). All relevant studies were retrieved and discussed. We only included full articles.****Conclusions:*** *Ulnar neuropathy is a commonly encountered condition, in both in the general population and in the athletes. Thorough clinical assessment and discerning assessment of electrodiagnostic tests are useful in detecting the progression of recovery with non-surgical and surgical modalities of treatment. The possible need for decompression at both the cubital tunnel and Guyon's canal should be well understood. Adherence to the principles of surgical decompression and the pearls which we stated above will help the surgeon in providing a reliable and durable outcome. Supplementation of decompression with supercharged end-to-side nerve transfer could potentially expedite motor recovery of the ulnar intrinsic muscles in the appropriately selected patient.*

Key words: *Neurology, Ulnar neuropathy, elbow, presentation, management.*

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INTRODUCTION:

In all disciplines and subspecialties, all surgeons who are treating complex elbow diseases must be aware of the principles in assessment and treatment of both acute and chronic ulnar neuropathies. Management of chronic compression neuropathy of the ulnar nerve is still considered a complex and a challenging aspect that faces physicians. Appraisal of the literature via systematic reviews and meta-analyses demonstrates that the best management for patients with ulnar neuropathy is still not clear [1]. Understanding of the subtleties in clinical manifestations and thorough consideration of the timing and type of surgical interventions are essential to optimize results following management of ulnar neuropathy. In addition, treatment of acute ulnar nerve injuries has been recently altered following the introduction of advanced nerve transfer techniques.

In this review, we will discuss the most recent evidence regarding Ulnar neuropathy at elbow

METHODOLOGY:

We did a systematic search for Ulnar neuropathy at elbow in the emergency department using PubMed search engine (http://www.ncbi.nlm.nih.gov/) and Google Scholar search engine (https://scholar.google.com). All relevant studies were retrieved and discussed. We only included full articles.

The terms used in the search were: Neurology, Ulnar neuropathy, elbow, presentation, management.

Clinical presentation

Patients who have compressive neuropathy of the ulnar nerve classically describe having numbness and tingling of the ulnar-sided digits of their hands, typically within the small finger and the ulnar part of the ring finger. Among the population, clinical manifestations often start intermittently and are usually worse at night, specifically when the elbow is flexed during sleeping. As the disease continues, paresthesias might happen more usually and during the daytime. The position of the elbow is very likely to contribute to the clinical manifestations' characteristics, as cadaveric studies have shown that the ulnar nerve at the elbow is vulnerable to both compression and traction during flexion [2].

Prominent bands of the Osborne ligament at the leading part of the flexor carpi ulnaris muscle can potentially increase ulnar nerve clinical manifestations while performing gripping activities, like driving. For throwing athletes, laborers, or others who perform high physical demands on their elbows, clinical manifestations might be occur following intense or prolonged periods of increasing activity. These patients might also manifest with concurrent bony and soft tissue conditions at their elbows, like medial epicondylitis, lateral epicondylitis, ligamentous instability, and/or stress fractures.

Cyclists and others who place long-term pressure on the hypothenar eminence are at higher risk for compressing the ulnar nerve at the Guyon's canal. therefore, detailed questions about specific provocative activities and positions, along with the timing and chronicity of clinical manifestations, is important to make clinical decision. Clumsiness and loss of dexterity suggest intrinsic muscle involvement. Cases where there is motor weakness with the absence of any sensory defects must have special attention, with concurrent cervical radiculopathy and/or myelopathy also receiving consideration. In rare cases, an upper motor neuron condition or a compressive mass might also be present. These different, however significant, causes might mimic cubital tunnel syndrome, making electrodiagnostic studies prudent in most cases.

Physical examination

Systematic evaluation of the muscles and sensory distributions which are innervated by the ulnar nerve is beneficial for determining the compression level of the ulnar nerve. Compression at the cubital tunnel from Guyon's canal might be distinguished with particular comparison of intrinsic versus extrinsic motor strength, as well as comparison of sensation in the volar versus dorsal ulnar nerve sensory distributions. The motor physical examination includes assessment of the flexor carpi ulnaris, ulnar-innervated flexor digitorum profundus, finger abduction, and finger adduction, whereas the sensory examination includes subjective responsiveness to sensation to light touch (Ten Test) [3] and two-point discrimination in the distributions of the dorsal cutaneous branch and volar main sensory branch of the ulnar nerve. In patients who have advanced ulnar neuropathy, more examination might show atrophy in the hypothenar eminence and in the first dorsal interosseous muscle bulk, as well as clawing of the ring and small fingers.

Eponymous findings like Froment's sign (overt flexion of the thumb interphalangeal joint with trying resisted pinch) and Wartenburg's sign (persistent abduction posture of the small finger because of the unopposed action of the radial-innervated extensor digiti minimi) might also be present. We also find a pseudo-Froment sign, where the interphalangeal joint of the involved thumb may might not have overt flexion but instead has a varying appearance when compared to the other side. This sign might specifically be noted in patients who have ligamentous laxity and hypermobile joints.

Provocative testing of the ulnar nerve typically includes percussion (Tinel's sign) and direct pressure which is applied over well-known compression points of the nerve. The highest sensitive (91 percent) provocative examination for ulnar nerve compression at the elbow is applying direct pressure over the ulnar nerve posterior to the medial epicondyle when the elbow is in the flexion position [4]. We use an alteration of the scratch collapse test [5] to detect distinct or concurrent points of the ulnar nerve compression. An area of nerve compression is detected by the presence of weakness in the resisted shoulder external rotation (with the arm being adducted at the side and the elbow being flexed 90°) after that site has been stimulated by the examiner's scratch. Additional points of compression could also be stimulated by the application of a topical anesthetic to the sites with a positive result. We have found this examination maneuver to be associated well with electrodiagnostic and intraoperative findings in patients who have ulnar neuropathy [6].

The determination of specific and/or concurrent points for ulnar nerve compression is specifically helpful through the evaluation of patients who have failed primary surgery. The hierarchy of the collapse points aids in detecting if the ulnar nerve is primarily irritated at the cubital tunnel or at Guyon's canal or at other sites of compression (like the arcade of Struther's in the mid-brachium). generally, the scratch collapse examination would be performed to detect correlated compression at Guyon's canal and the arcade of Struthers among patients who have failed primary cubital tunnel surgery. Compression at both of these areas is amenable to surgical treatment without necessarily revisiting the prior operated decompression over the elbow.

Stabilization of the ulnar nerve within its groove posterior to the medial epicondyle must also be evaluated. Hypermobility of the ulnar nerve was found to be present in seventy-three percent of participating healthy volunteers and might be also linked to higher nerve irritability rates [7]. Despite being not supported by uniform guidelines or strong, classical teaching indicates that individuals with clinically present subluxation of the ulnar nerve must undergo anterior transposition (instead than in situ decompression) to reduce the chances of continuous clinical manifestations, recurrent clinical manifestations, or postoperative deterioration of nerve subluxation. At the time of the surgical operation, any correlated subluxation of the triceps muscle should be assessed and managed, if found [8].

Classification and interpretation of electrodiagnostic studies

The pathology of chronic compression neuropathy spans a breakdown in the blood–nerve barrier, followed by sub-perineurial edema, localized (and then diffuse) demyelination, and later loss of the axons. Compressive ulnar neuropathy will present along a similar predictable spectrum of disease: dynamic ischemia, demyelination, and axonal loss. These stages could be differentiated through taking careful medical history from the patient physical examination, and interpretation of electrodiagnostic studies.

During the presence of dynamic ischemia, the compression of the ulnar nerve is the result of transient reductions in the perfusion of ulnar nerve. Clinical manifestations are largely position-dependent and improve when the blood flow to the nerve is recovered. Patients who show dynamic ischemia classically improve following nonoperative measures (like protective padding, night positioning in extension with a towel roll, and ergonomic adjustments) to prevent positions that cause manifestations. Electrodiagnostic studies (like nerve conduction studies and electromyography) are high likely to be negative, as conduction velocity has not yet reduced in the fastest-conducting nerve fibers. If manifestations warrant surgical intervention, the patient is highly likely to have near-immediate relief following performing surgery. As the condition process progresses, long-term ischemia might lead to demyelination of the
ulnar nerve. Conduction velocity, which represents the velocity of conduction along the fastest-conducting nerve fibers, is decreased. Clinical manifestations become more pronounced and less intermittent.

Relief of manifestations following surgical operation is usually expected within a period of three to four months following surgery as the remyelination occurs. Long-term or severe compression will cause

the development of axonal loss. Clinical manifestations are permanent, and alterations in two-point discrimination, motor weakness, and muscle atrophy are highly evident. Nerve conduction tests will show a reduction in the amplitude, which represents the overall reduction in the number of working nerve fibers. Electromyography will show abnormal activities during the insertional phase (suggesting muscle denervation), fibrillations during resting phase, and the presence of motor unit action potentials throughout the recruitment phase (suggesting attempts of reinnervation by either collateral sprouting or axonal reinnervation). Recovery following surgical operations is much more prolonged, as axonal regrowth usually occurs at a rate of one mm per day. Collateral sprouting from the adjacent, unaffected motor fibers to sprout collateral branches to the neighboring denervated muscle fibers might show the reinnervation process.

Nonoperative management

patients with dynamic ischemia are highly likely to respond to nonoperative therapies for ulnar nerve compression at the site of the cubital tunnel. A special protocol of eliminating elbow flexion at night and modifications of activities for three to six months may lead to significant improvements of the cubital tunnel syndrome in patients who have mild to moderate clinical manifestations [9].

Padua et al. showed clinical and electrodiagnostic improvement at one-year follow-up in estimated half of these patients receiving treatment with postural education [10]. Patients' education by the physician and surgeon regarding the positional nature of the neuropathy is important to establish the maximum efficacy of activity modifications and ergonomic adjustments. Protective soft padding over the medial site of the elbow could also reduce the irritability of the ulnar nerve. At night, a soft towel can be wrapped around the elbow to reduce the elbow flexion and is more likely to be tolerated by patients when compared to the use of rigid splints. A neutral wrist splint reduces stretch on the ulnar nerve in Guyon's canal. We maintain nonoperative therapies for patients who show mild to moderate clinical manifestations (which suggests the presence of dynamic ischemia) and motor nerve conduction velocity higher than forty m/second over the elbow. If patients do not improve following several months of non-operative therapy, we recommend the application of surgical interventions of the ulnar nerve. Patients who show significant axonal loss (as shown by the reduced amplitudes on nerve conduction studies) are not likely to response to conservative treatment and are considered candidates for surgical interventions.

Surgical management

Controversy is present regarding the proper surgical intervention to manage ulnar neuropathy sited at the elbow. The several techniques described to treat cubital tunnel syndrome represent options available to manage the ulnar nerve at the level of medial epicondyle. They can be usually categorized as in situ decompression (open, mini-incision, or endoscopic) or anterior transposition (which is accomplished via medial epicondylectomy, subcutaneous transposition, transmuscular transposition, or submuscular transposition). In situ decompression has shown an increase in popularity among practitioners in the US [11], despite the fact that recent reports have suggested significant rates of recurrence. Appraisal of the available evidence shows that the best management for cubital tunnel syndrome remains debatable.

In situ decompression of the ulnar nerve may potentially provide benefits in specific patients, but thorough counseling is generally advised before performing surgery to discuss the frequency of recurrent clinical manifestations, the frequency of reoperation, and the results following surgical operation if needed. The hypothetical advantages of in situ decompression can include more rapid recovery due to less extensive dissection and lower risks of wound-associated adverse events [12].

The hypothetical advantages of preserving intrinsic and extrinsic vascularity has been studied, as blood flow is kept for a diameter-to-length ratio of 1:63 within the ulnar nerve when only proximal intrinsic vascularity is normal [13]. moreover, a more recent trial demonstrated no significant difference in motor or sensory result regardless of the extrinsic vascular pedicle to the ulnar nerve was preserved. The frequency of persistent clinical manifestations and final anterior transposition has been demonstrated as low as seven percent at one-year follow-up. On the other hand, the same center has more recently published a less favorable revision rate of nineteen percent, with 77 percent of the revisions performed within only two years of the in-situ decompression.

Risk factors for requiring a revision surgery following an in situ decompression included the presence of a prior elbow fracture or dislocation and surgery performed for patients with mild clinical manifestations without the presence of motor weakness. When assessing the outcomes following

revision cubital tunnel surgery in a case–control examination, the patients who underwent revision surgery were more likely to have persistence of permanent clinical manifestations (53 percent of patients) and worse patient-reported results when compared to those who underwent primary surgical operation. The results of this research are specifically salient to those undergoing in situ decompression of the ulnar nerve, as 93 percent of these patients in the revision cohort had previously undergone an in-situ decompression. The recent guidelines regarding in situ decompression make us less optimistic regarding its general role in the management of cubital tunnel syndrome, specifically since the results following the revision surgery were found to be worse than performing an anterior transposition from the beginning.

Acute ulnar nerve injuries

Acute compression of the ulnar nerve because of an adjacent soft tissue swelling or fracture displacement might happen following any kind of elbow trauma [14], ligamentous reconstruction surgical operation of the elbow, or any fracture near Guyon's canal. Post-operative position of the elbow in flexion can also place the ulnar nerve at higher risk for compression.We recommend surgeons and physicians who manage individuals with elbow and wrist to identify the possibility for developing acute ulnar nerve compression and to manage this condition in a manner that is similar to the management during urgent release of the median nerve within the carpal tunnel in the setting of the presence of distal radius and forearm fractures. A good prognosis is usually expected if the acute compression is detected and managed in an expedient manner. The development of an Iatrogenic injury against the ulnar nerve might also happen during performing elbow surgical operation.

Neurotmetic injury to the ulnar nerve has also been reported during Tommy John medial ulnar collateral ligament reconstruction [15]. In cases where the injury happens at the cubital tunnel, we advise a multi-faceted protocol to improve the chances at functional recovery given the relatively poorer outcomes which are associated with this damage pattern. In addition to repairing the ulnar nerve and doing an anterior submuscular transposition, we also recommend performing a concurrent distal end-to-end (ETE) motor nerve transfer (pronator quadratus branch of anterior interosseous nerve transferred to deep motor branch of the ulnar nerve) to improve motor recovery, ETE sensory nerve transfers of the third web space branch of the median nerve to the ulnar sensory fascicles and the palmar cutaneous branch of the median nerve to the dorsal cutaneous branch of the ulnar nerve, and tenodesis of the flexor digitorum profundus (FDP) tendons to keep finger motion when still waiting for motor recovery [30]. Patients with high ulnar nerve lacerations and a Martin–Gruber interconnection are treated with SETS nerve transfer (rather than ETE) to maintain these axons. For damages which are distal to the cubital tunnel (within the forearm), we recommend performing a SETS nerve transfer protocol in addition to the FDP tenodesis which is used for proximal acute neurotmetic injuries. Recently, physicians have used short acellular nerve allografts (2.5 cm) in a side-to-side fashion from the median nerve (in the carpal tunnel) to the ulnar nerve (in Guyon's canal) with the outcome of bringing early sensation to the ulnar-innervated portion of the hand.

CONCLUSIONS:

Ulnar neuropathy is a commonly encountered condition , in both in the general population and in the athletes. Thorough clinical assessment and discerning assessment of electrodiagnostic tests are useful in detecting the progression of recovery with non-surgical and surgical modalities of treatment. The possible need for decompression at both the cubital tunnel and Guyon's canal should be well understood. Adherence to the principles of surgical decompression and the pearls which we stated above will help the surgeon in providing a reliable and durable outcome. Supplementation of decompression with supercharged end-to-side nerve transfer could potentially expedite motor recovery of the ulnar intrinsic muscles in the appropriately selected patient

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