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Review

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Entrapment neuropathies of the upper extremity

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ABSTRACT

Entrapment neuropathies are mononeuropathies caused by long term or recurrent compression of peripheral nerves as they pass through anatomically narrow areas. The diagnosis of entrapment neuropathies are based on a combination of anamnesis, physical examination and EMG findings. In this article, pathophysiology of entrapment neuropathies and some of the most common entrapment neuropathies seen in upper extremity will be discussed.

Keywords: Entrapment neuropathy, mononeuropathy, neuropathy, carpal tunnel syndrome upper extremity

INTRODUCTION

Entrapment neuropathies are mononeuropathies caused by long term or recurrent compression of peripheral nerves as they pass through anatomically narrow areas. Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy in clinical practice which is followed by cubital tunnel syndrome (CuTS).[1] Tarsal tunnel syndrome and radial neuropathy are other common conditions.[2,3] Syptoms may vary from pain, sensory deficit, paresthesia to muscle atrophy and permanent motor deficit depending on the degree of damage.[4] Early identification is crucial in determining the treatment approach and preventing damage progression.[2-4]

Commonly used synonymously in daily practice, entrapment neuropathy and compression neuropathy are different entities. Compression neuropathy refers to any damage to the nerve caused by acute or chronic compression. Entrapment neuropathy refers to damage caused by mild, long-term or recurrent compression. Some anatomical factors increase the risk of entrapment. These are localization and being adjacent to structures that may facilitate compression.[5,6]

Entrapment neuropathy is a very common condition in society. For example, the lifetime prevalence of carpal tunnel syndrome is 10% and this rate reaches 84% in patients with type 1 diabetes. It is twice as common in women than in men. Age distribution is bimodal, peaking between 25-30 years (related to occupation) and 40-60 years (because of hormonal factors). Pregnancy; various connective tissue diseases, (especially rheumatoid arthritis) endocrinological disorders; obesity or rapid weight loss; presence of polyneuropathy in the background; edema; presence of amyloidosis are some of the conditions that increase the incidence.[1,3,6-9]

In this article, pathophysiology of entrapment neuropathies and some of the most common entrapment neuropathies seen in upper extremity will be discussed. Shoulder girdle entrapment neuropathies are excluded due to the breadth of the topic.

PATHOPHYSIOLOGY

In entrapment neuropathies, there is a long-lasting or frequently recurring micro trauma of mild severity and nerve damage due to this trauma.

Intraneural transient ischemia is the first sign of entrapment. External compression of the nerve with a pressure above 20-30 mmHg is sufficient to disrupt venous circulation in the vasa nervorum. The pressure gradient must be normalized intermittently to ensure adequate blood flow to the area. Therefore, at this stage, paresthesia occurs which is triggered at night, in static position or in extreme positions and disappear with movement.[1]

Prolonged ischemia damages the vasculature followed by edema. The entrapped nerve may be visualized as enlarged and hyperintense on MRI.[7] Continuation of edema initiates both extraneural and intraneural fibrotic changes.[1]

The inevitable result of prolonged ischemia is focal demyelination which is characterized by segmental slowing of conduction velocities or, if damage developed acutely, conduction blocks.[8]

Structural changes occur in Ranvier nodes with demyelination. These changes lead to the generation of spontaneous ectopic action potentials. This is reflected in the clinical practice as a sudden electric-like pain, which may occur spontaneously or be triggered by Tinel test. Eventually, if the compression persists, permanent axonal damage occurs through Wallerian degeneration.[1,6,9,10]

Inflammation is known to play a role in nerve damage. Immune cells are activated at the damaged axonal site and release inflammatory cytokines. This increases damage to the vasculature and initiates a cycle of further inflammation and edema. Furthermore, mechanical compression and inflammation inhibit retrograde and anterograde transport in the surrounding axons. All metabolites that cannot be transported cluster in a restricted area and mediate the formation of new ion channels. This contributes to sensitization at the site of compression.[1,11-14]

CLINICAL FINDINGS

The diagnosis of entrapment neuropathies are based on a combination of anamnesis, physical examination and EMG findings. EMG is used not only to support the diagnosis but also to exclude alternative diagnoses; determine the exact location of the compression, predict the prognosis and to select the optimal treatment strategy.[6,15-18]

Typical EMG findings are focal slowing (prolonged latency, decreased conduction velocity). Since axonal damage does not occur immediately, there is no initial loss of amplitude and needle EMG shows no abnormal findings. Focal demyelination begins after 7-10 days of exposure, followed by remyelination on days 14-28. The disruption of the saltatory conduction via demyelinated areas and remyelinated fibers being thinner than normal prolong the time between stimulation and action potential generation.[1-4,19-24]

As the process goes on, first sensory and then motor amplitude decrease. Increased input activity, fibrillation, PSW (positive sharp waves) and large MUAPs (motor unit action potentials) can be observed on needle EMG. Amplitude loss is an important indicator of the severe damage.[4,6,10,15,26]

When evaluating EMG results, it is important to note whether there are any anastomoses between the nerves.[8,13,27,28] The most common one is the Martin Gruber between the median and ulnar nerves in the forearm. Here, some fibers of the median nerve or anterior interosseous nerve are carried over the ulnar nerve and innervate the intrinsic muscles of the hand.

There are three signs of this on EMG: initial positive deflection of the CMAP (Combined Muscle Action Potential) when the median nerve is stimulated at the wrist and the electrodes are placed over the abductor pollicis brevis; increased or negative median nerve conduction velocity (because some fibers do not pass through the carpel tunnel); larger CMAP amplitude on proximal than distal (because some of the distal fibers are carried through the ulnar nerve.[33,34]

Abnormalities that may compress the nerve can be scanned via MRI (magnetic resonance imaging). Findings indicating nerve damage can be revealed. Additionally, denervated muscles can be demonstrated.[15,19]

In USG, a normal nerve has a "honeycomb" appearance with groups of hypoechoic fascicles surrounded by hyperechoic perineurium. Also the nerve itself is surrounded by hyperechoic epineurium, which often blends indistinguishably into the surrounding tissue. In entrapment neuropathy, venous congestion causes widening of the cross-sectional area of the nerve, while ischemia causes flattening of the nerve and disruption of the normal fascicular structure. This widening may also be shown proximal to the entrapment due to impaired axonal transport.[18-20]

Increased vascularity due to inflammation and perineural fibrosis may be demonstrated on USG as echogenic halo appearance (thickening and prominence of the epineurium), intraneural echogenic fibrotic spots and pseudo neuroma formation. "Hourglass appearance" develops as a result of flattening of the nerve. USG also reveals hyper echogenicity in denervated muscle due to edema in the acute phase and increased heterogeneity, structural disruption and replacement of normal muscle tissue with fatty tissue due to atrophy in the chronic phase.[15,19]

MEDIAN NERVE

The median nerve is formed by the merger of fibers from the medial cord (C8-T1) and lateral cord (C5-C7) of the brachial plexus. It runs parallel to the brachial artery and ulnar nerve in the sulcus bicipitalis. It does not branch until the elbow level.[16]

When it reaches the antecubital fossa, it passes between the two heads of the pronotor teres (PT) after innervating the forearm flexor muscles palmaris longus (PL), flexor carpi radialis (FCR), PT, flexor digitorum superficialis (FDS). Here it crosses the ulnar artery anteriorly and gives the anterior interosseous nerve (AIN) branch. The AIN runs deep within the fibrous margin of the FDS. It innervates the flexor pollicis longus (FPL), the lateral side of the flexor digitorum profundus (FDP) and the pronotor quadratus.[16-19]

The median nerve extends to the wrist between the FDP and FDS. At the wrist, before passing through the carpal tunnel, it gives off its palmar cutaneous branch innervating the tenar eminence. Then it passes through the tunnel and innervates the thenar muscles and provides sensory innervation to the palmar surface of the first 3.5 fingers.[3,16,23]

The median nerve can be compressed in three places: elbow, forearm and carpal tunnel. In addition, a rare accessory fibrous band, the Struthers Ligament, lies proximal to the elbow between the supracondylar process of the humerus and

the medial epicondyle and can compress both the median nerve and the adjacent brachial artery. This condition is called Struthers syndrome.[3,16,19,21]

Carpal tunnel syndrome is the most common entrapment neuropathy in adults. The mechanism of its occurrence is not completely understood. However, it is suggested that excessive consecutive movements causing microtrauma in the wrist and the presence of osteoarthritis on the background of a nonspecific tenosynovitis facilitate the emergence of the syptoms. It is usually bilateral, predominantly in the dominant hand. Sensory complaints start first. Hypoesthesia and paresthesia are observed in the first three fingers and lateral half of the fourth finger. Thenar eminence is preserved. Patients wake up with numbness in the hand and swelling in the wrist. Complaints are aggravated by positions that require bending or holding the wrist in the air, such as reading the newspaper, driving a car or carrying a bag. After a while, patients experience a numbness that wakes them up at night. Shaking or rubbing the hand significantly relieves the symptoms; this is called the "flick phenomenon". In more advanced stages, motor deficits develop. Impairment in fine skills and weakness in grasping are seen. Thenar atrophy may be present.[2,3]

On examination, when the median nerve is percussed at the wrist level, there may be an electric-like sensation radiating to the fingers. This is called the 'Tinel' sign. Its sensitivity is 30-45% and specificity is around 65%. The Phalen Test is performed by instructing the patient to bend both hands at wrist level, press the backs of the hands together and wait 30-60 seconds. Its sensitivity is around 50-67% but its specificity is only 15-17%.[2]

When performing EMG, the affected extremity should be compared with the unaffected side and the results should be evaluated together with another nerve (mostly ulnar) in the same hand. Fifteen percent of patients may have normal nerve conduction study results even if they experience clinical symptoms. USG may be helpful to evaluate these individuals.

The classification of CTS according to AAEM (American Association of Electrodiagnostic Medicine) is as follows: >0.5 msec difference between median-ulnar nerve peak latencies recorded from the 4th finger is considered mild. Prolongation of the distal latency of the median motor nerve (>4.0 msec) in addition to mild stage symptoms is considered moderate. Decreased motor amplitude with low/absent sensory amplitude is considered severe CTS.[32]

Another areas where the median nerve can be entrapped are between the two heads of the PT, hypertrophic biceps aponeurosis and the proximal part of the FDS. Compression of the nerve between heads of PT is called Pronotor Teres syndrome (PTS), but compression in all other localizations have the same clinical presentation. PTS is usually seen in people who frequently perform pronation and tight grasping movements (carpenters, tennis players, housewives, waiters).[19]

PTS presents itself with pain in the forearm and sensory deficit in the first three fingers and the lateral half of the fourth finger, just like CTS. The main difference between the two is that in PTS, the sensation of the thenar region is also affected due to the involvement of the palmar cutaneous branch. Nocturnal findings are not observed in PTS.[5]

In PTS, complaints are triggered by resistant elbow pronation and flexion of the third finger. However, third finger flexion may also trigger CTS syptoms. Phalen's test is positive in 50% of cases. EMG is usually normal. The anterior interosseous nerve is often entrapped by the tendinous portion of the deep-seated head of the PT. Other less common sites are the proximal end of FDS, Gantzer muscle, accessory head of FDS or FDP. It usually develops after stretching exercises, peripartum period and thrombosis of radial or ulnar artery.[5,19]

Anterior interosseous nerve syndrome (AINS) presents itself with pure motor signs. The patient is unable to make a fist or button a shirt. When the patient is told to hold a piece of paper with the tips of two fingers, they do so by extending their fingers forward instead of curling the two fingers in an "O" shape. EMG shows signs of pure motor deficit. Denervation in the pronotor quadratus, FDP and FPL can be demonstrated via needle EMG.[19]

ULNAR NERVE

The ulnar nerve is the continuation of the medial cord (C8-T1) of brachial plexus after giving fibers to the median nerve. It runs parallel to the median nerve and brachial artery in sulcus bicipitalis for a short time. Shortly after piercing through medial intermuscular septum, it passes under the Struthers arch which connects the medial head of the triceps to the medial intermuscular septum.[19-21]

At the elbow level, it passes posterior to the medial epicondyle, surrounded by Osborne's ligament and the head of the flexor carpi ulnaris (FCU). Here these structures form the cubital tunnel. The medial border of the tunnel is marked by the medial epicondyle and the lateral border by the olecranon. After exiting the tunnel, the nerve innervates the FCU and the ulnar side of the FDP. Ulnar nerve travels between the two heads of the FCU in forearm. It gives dorsal and palmar cutaneous branches before reaching the wrist. At the wrist, it travels together with the ulnar artery in Guyon's canal.[20,21]

The palmar cutaneous branch travels through forearm on the palmar side. At the wrist level, it crosses over Guyon's canal and then innervates the ulnar side of the palm. From a few centimeters proximal to the palmar cutaneous branch, the dorsal cutaneous branch diverges and receives the sensory input of the dorsal ulnar side of the hand, the dorsal 5th finger and the dorsal ulnar half of the 4th finger. After leaving Guyon's canal, the ulnar nerve branches into two parts, the superficial sensory and branch the deep terminal branch. The superficial branch innervates the rest of the ulnar palmar side of the hand, the palmar side of the 5th finger and the palmar ulnar half of the 4th finger. The deep terminal branch is a pure motor branch which innervates almost all of the interstitial muscles of the hand.[21]

The most common sites of ulnar nerve compression are the cubital tunnel and Guyon's canal. Some other areas include Struthers' arc, medial intermuscular septum and between the two heads of the FCU.[19]

Cubital Tunnel Syndrome (CuTS) is the most common entrapment neuropathy of the ulnar nerve. Symptoms include sensory deficit in the medial forearm, hand, 4th and 5th fingers; tenderness in the medial elbow and pain radiating from the elbow to the forearm and hand. Complaints are aggravated at night during sleep or when the elbow is bent. In the chronic period, atrophy occurs in the first dorsal interosseous muscle, intrinsic muscles and hypothenar region. Loss of fine motor skills develop due to intrinsic muscle weakness. The thumb cannot be adducted due to paralysis of adductor pollicis. A claw hand appearance may be observed in which the 4th and 5th fingers are slightly abducted and the metacarpophalangeal joint of the thumb is hyperextended.[19,22]

Narrow cubital tunnel, cubitus valgus deformity, anteriorly located ulnar nerve are factors that increase the risk. The risk is also higher in long-distance drivers, prolonged cell phone users, smokers and those with a history of prolonged compression or trauma to the elbow.[19,22,23]

In examination, the elbow flexion test is performed by flexion o the elbow over 90 degrees, supinating the forearm and extending the wrist. It is considered positive if paresthesia occurs or present paresthesia increases within the first 60 seconds. Tinel's test may be positive when the medial side of the elbow is touched, but this test alone has low diagnostic value as it may be positive in one third of asymptomatic cases. The nerve compression test is performed by applying pressure to the elbow for 10 seconds. It is considered positive if numbness and paresthesia develop. In addition, if the patient is asked to hold a paper between the thumb and index finger, FPL activity becomes evident because the adductor pollicis is not working and the thumb flexes at the interphalangeal joint. This is called Froment's sign.[21]

EMG shows decreased ulnar nerve motor conduction velocity in the elbow segment. It is important to keep the elbow in 70-90° flexion during the measurement for optimal results. When the elbow is flexed, the arcuate ligament elongates and narrows the cubital canal as the ulnar nerve elongates and becomes vulnerable to entrapment. According to the AAEM, EMG findings of cubital tunnel syndrome are as fallows: a an ulnar nerve motor conduction velocity of less than 50 m/sec in the elbow segment with normal forearm and arm findings, an ulnar nerve motor conduction velocity that is .m/sec lower in the elbow than 20% in the CMAP amplitude over the elbow compared to bellow the elbow. Needle EMG shows fibrillation, PSWs and dilution in the participation of motor unit potentials indicating axon loss. These findings are most evident in the first dorsal interosseous muscle.[3,21,24]

Another region where the ulnar nerve is compressed is the wrist, which is more common in cyclists, golfers, basketball players, racquet sports players and in the presence of ulnar artery thrombosis or aneurysm (hypothenar hammer syndrome). Since the nerve divides into deep and superficial branches at the wrist level, findings in this region may be pure motor, pure sensory or mixed. There are no symptoms on the dorsum of the hand because the dorsal cutaneous branch is spared. Entrapment here can occur in four ways: At pisiform bone proximal to Guyon's canal, within the canal (deep motor branch), distal to the canal at the hook of the hamate bone (deep motor branch) and distal to the canal in the palmaris brevis muscle (superficial sensory branch). The compression within the canal is called Guyon's canal syndrome (GCS). In GCS, the sensory deficit involves only palmar side of the 4th and 5th fingers. The dorsum of hand, ulnar side of palm are spared. Furthermore, in CuTS the medial forearm is also affected, whereas in GCS the symptoms are limited to the hand. Apart from these differences, the findings are similar in both conditions. Patients with suspected GCS should be evaluated for hamate, pisiform fractures and vascular pathologies. Demonstration of latency prolongation in the wrist on EMG supports the diagnosis.[5,19,21]

RADIAL NERVE

The radial nerve is the continuation of the posterior cord originated from the C5-T1 roots. It runs posterior to the axillary artery. Then, with the brachial artery, it passes through the triangular space formed by the long head of the triceps muscle, the humerus bone and the teres major. It enters the spiral groove located posteriorly on the proximal part of the humerus. As it passes through the groove, it travels with deep brachial artery between the medial and long heads of the triceps muscle. During its course here, it gives two sensory branches, posterior antebrachial cutaneous nerve and the inferior lateral brachial cutaneous nerve. These two innervates the posterior forearm and the lower lateral part of the arm respectively. Radial nerve wraps around the humeral shaft as it moves from posterior to anterior within the groove. Here it pierces the lateral intermuscular septum and reaches the anterior compartment. It runs between brachial and brachioradial muscles in the cubital fossa. At the elbow level, after passing anterior to the lateral epicondyle of the humerus, it gives its superficial sensory branch and becomes the posterior interosseous nerve (PIN). The muscles it innervates before forming the PIN are the triceps, brachioradialis, anconeus and extensor carpi radialis longus (ECRL). The superficial sensory branch runs deep to the brachioradialis. It crosses the anatomical snuffbox distally and innervates the radial side of the dorsum of the hand and the dorsal surface of the first 3.5 fingers.[3,19,25-27]

Shortly after its formation, the PIN pierces the supinator muscle. Here, the thick proximal edge of the superficial head of the supinator is called the Frohse arc. After piercing the supinator, it moves between the deep and superficial heads of the muscle. Then it pierces the muscle again and enters the posterior compartment. After that it divides into two branches (medial and lateral) which innervates most of the forearm and hand extensor muscles.[3,19,25,27]

Radial nerve can be trapped at three sites: above the elbow, after becoming PIN and around the wrist. Above-elbow compression can occur in the axilla, spiral groove or rarely in the triangular interval. The chronic compression at the axilla level is called "crutch paralysis" while the acute form is called "saturday night paralysis". Crutch paralysis is caused by prolonged, inappropriate use of the crutch. In saturday night paralysis, the same compression develops acutely as a result of the patient falling asleep with their arm hanging down from the back of the chair. Drop hand Syndrome which the wrist and fingers cannot be extended. Sensory deficit occurs in all of the areas innervated by radial nerve. Elbow extension is impaired. The prognosis in these cases are generally good

as the nerve damage is usually at the level of segmental demyelination. In most cases, full recovery occurs within 2-3 months. In case of inadequate recovery, needle EMG examination at 12th week helps to evaluate prognosis.[3,19,25-28]

Compression on the spiral groove is usually caused by humeral shaft fractures. This group accounts for 70% of compression neuropathies of the radial nerve. Usually the injury is acute. The compression may originate from the fracture itself or, more frequently may be iatrogenic after correction surgery. Chronic entrapment may occur in intermuscular septum due to insufficient mobility after fracture and lateral head of the triceps after prolonged stretching exercise. Callus formation after fracture and fibrosis in the muscle due to chronic intramuscular injection may be possible causes for chronic entrapment Symptoms are similar to that of entrapment at the axilla level, but there are some differences. Here elbow extension is preserved since triceps is spared; but the forearm cannot be pronated or supinated while the elbow is in extension. In addition, while there is a sensory deficit in the distal forearm; sensation of the arm's posterior is preserved.[28,29]

Entrapment of PIN most commonly occurs when Froshe's arch thickens as a result of repetitive pronation supination movement. Less common sites of compression are the radial artery crossing the radial neck (Henry's leash), the distal end of the supinator, extensor carpi radialis brevis (ECRB) and cysts seen in rheumatoid arthritis. On rare occasions, the nerve may also be compressed after it divides into two branches. In PIN compression, there is typically weakness in finger extension. But wrist extension can be achieved by radially deflecting the wrist because ECRB and ECRL is spared. Atrophy develops at the forearm extensor muscles. There may or may not be mild pain in the lateral forearm. There is no sensory deficit.[19,30]

Here, the level of nerve compression can be understood by detecting the affected muscles. If ECU, extensor digitorum quadratus (EDQ) and extensor digitorum communis (EDC) are affected, it suggests that the medial branch is affected. If abductor pollicis longus, extensor pollicis brevis (EPB), extensor pollicis longus (EPL) and extensor indicis proprius (EIP) are affected, it suggests that the lateral branch is affected. If all of them are affected, it suggests that the PIS is affected at a level before it is divided into branches.[30]

The path of the radial nerve from the radial head to the distal end of the supinator muscle is called radial tunnel. Its borders are drawn by the supinator muscle, ECRL, ECRB and brachioradialis. Radial tunnel syndrome (RTS) is a condition characterized by isolated pain without muscle weakness or sensory deficit caused by recurrent mild compression of PIN within the tunnel. Tenderness over the radial nerve trace '5 cm'can be erased distal to the lateral epicondyle is the main symptom.

The pain worsens at night, may awaken the patient from sleep and may be aggravated by nerve-straining maneuvers such as elbow extension, forearm supination or wrist hyperextension. EMG findings are normal. MRI results are normal or shows nonspecific changes in most cases. The diagnosis is made by excluding alternative diagnoses.[31]

CONCLUSION

Entrapment neuropathies are conditions in which damage is often reversible with early recognition. Careful differential diagnosis and determination of treatment strategy before permanent deficits develop will reduce morbidity rates significantly.

ETHICAL DECLARATIONS

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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