Median Nerve Compression Syndromes. Literature Review and Update

Javier E. Sánchez Saba, Juan Francisco Civit, Paula Ramírez Vargas, Francisco Melibosky Ramos, Aldo Villavicencio Achurra, Javier Román Vees, Peter Cobb Craddock, Pablo Orellana Araya, Rene Jorquera Aguilera

Hand Surgery, Microsurgery and Upper Limb Arthroscopy Unit, Orthopedics and Traumatology Service, Clínica INDISA, Santiago, Chile. Orthopedics and Traumatology Service, Clínica Francesa, Dorrego, Mendoza, Argentina.

ABSTRACT
The median nerve is a nervous structure that begins to cross structures at the level of the elbow that might cause compression. The Struthers ligament, lacertus fibrosus, pronator teres, and flexor digitorum superficialis are among them. Finally, the transverse carpal ligament creates another compression site in the wrist. All these structures can develop pathological signs and symptoms of nerve entrapment, which favors nerve functional degradation. Our objective is to provide an update on these median nerve entrapment sites, as well as information on how to establish an accurate diagnosis and provide adequate treatment.

Keywords: Nerve compression; median nerve; pronator teres syndrome; carpal tunnel syndrome; surgical decompression.

Level of Evidence: IV

Síndromes compresivos del nervio mediano. Revisión y actualización de la bibliografía

RESUMEN
El nervio mediano descende por el brazo y, en el codo, comienza a atravesar estructuras que pueden generar compresión, como el ligamento de Struthers, el lacertus fibrosus, el pronador redondo, el flexor superficial de los dedos. Finalmente, en la muñeca, se encuentra otro sitio de compresión producido por el ligamento transverso del carpo. Todas estas estructuras pueden provocar signos y síntomas de atrapamiento nervioso y favorecer el deterioro funcional del nervio. Nuestro objetivo es dar a conocer una actualización sobre estos sitios de atrapamiento del nervio mediano, y cómo realizar un diagnóstico preciso e indicar un tratamiento adecuado.

Palabras clave: Compresión nerviosa; nervio mediano; síndrome del pronador redondo; síndrome del túnel carpiano; descompresión quirúrgica.

Nivel de Evidencia: IV

ANATOMY
The median nerve is constituted by the nerve roots of C5-C7 that form the lateral cord and of C8-T1 that generate the medial cord.¹ These roots coalesce forming the median nerve, which descends through the medial arm to the humeral artery and medial to the biceps and the brachii, without emitting collateral branches during this journey.²⁻³

In the elbow, it has been observed that the median nerve passes through a number of structures. Initially is the medial spur of the humerus, from where the Struthers ligament that extends to the medial epicondyle can originate. It then enters the cubital fossa in the depth of the bicipital aponeurosis and the pronator teres (PT) (both heads, humeral and ulnar). Running distally, it enters below the flexor digitorum superficialis (FDS). In this level, the motor branch or anterior interosseous nerve emerges, which runs in the forearm through the anterior region of the interosseous membrane and innervates the pronator quadratus, flexor pollicis longus, and flexor digitorum profundus muscles. The median nerve then continues its path in the forearm between the FDS and the flexor carpi radialis. ⁷ cm from the wrist, the palmar cutaneous branch of the median nerve emerges, which gives sensitivity to the thenar...
eminence. Finally, the nerve enters the carpal tunnel where the thenar motor branch emerges and ends up dividing to distal in the sensitive finger branches.1-4

**CARPAL TUNNEL SYNDROME**

Carpal tunnel syndrome is a compressive mononeuropathy of the median nerve in the wrist.5 From the pathophysiological point of view, it generates edema in the nerve, with demyelination and axonal damage.6,7 It is characterized by paresthesia or tingling in the sensitive region of the median nerve (radial edge of the hand), as well as pain that is usually exacerbated at night.5,6

**Diagnosis**

We describe a case associated with pain, numbness, hypotrophy of the thenar eminence, etc. Such symptomatology can be assessed using the CTS-6 questionnaire (6-item carpal tunnel syndrome symptoms scale), where a score of 5 indicates a low likelihood of having the syndrome, while a score of 12 or more indicates a high likelihood.8,9

A review by the American Academy of Orthopedic Surgeons identified certain features associated with the diagnosis of carpal tunnel syndrome, including the presence of thenar eminence atrophy, which is strongly related to the disease.9

Clinically, Phalen’s test and Tinel’s sign, and altered conduction nerve studies (taking into account that electromyography can give 10% false negatives) were markedly associated with this disease. It should be noted that these factors have no diagnostic value when considered separately.

On the other hand, there are also diagnostic tests that are moderately related to the diagnosis of the disease, such as the scratch collapse test, alterations in sensitivity through two-point discrimination or the monofilament test. In addition, there is a moderate association with other diseases, such as diabetes, repetitive manual activities, and vibrational activities.9

**Treatment**

Initial conservative treatment only provides benefits if wrist immobilizers are used to prevent flexion and corticosteroid infiltrations are administered, which achieve improvement in up to one third of patients.5

However, in the absence of a response, surgery is indicated. The objective is to decompress the tunnel by opening the transverse carpal ligament.10

Currently, both open and endoscopic techniques are the two surgical options.

In systematic reviews, it has been shown that there are no statistically significant differences between the techniques in terms of surgical time, improved grip strength, improved sensitivity at three months with the monofilament test or at one year with two-point discrimination.10,11

On the contrary, benefits of endoscopic treatment were found, with statistically significant differences in patient satisfaction, return to work and the absence of complications related to the skin wound (the open technique is associated with infections, pillar pain, and hypertrophic scarring).11 However, certain complications may occur, such as neuropraxia (not with permanent damage, in this sense, both are similar) and has the disadvantage of being more expensive.5,6,10,11

**Persistence, recurrence or new symptoms**

In the absence of symptom improvement, the possible scenarios are:6,12

- **Persistence**: symptoms are never relieved. In 58% of cases, it is attributed to incomplete release and in 37% to another compression site (such as the PT).12

- **Recurrence**: symptoms are relieved for a period >6 months and then the same symptoms reappear. In 88% of cases, it is usually associated with perineural adhesions, fibrosis and scarring. In these situations, revisions are indicated. It is recommended to improve the environment of the nerve including vein coverage, hypothenar fat pad flaps, etc.12

- **New symptoms after surgery**: up to 67% of the cases are related to iatrogenic complications. Revision with nerve repair or reconstruction is recommended.12
COMPRESSIONS OF THE MEDIAN NERVE AROUND THE ELBOW

While the incidence of elbow compressions is low, Hagert et al. indicate that this may be due to failure to diagnose this disease.13-16

From an anatomical point of view, compressions can be produced by:

1. The pronator teres. It is the most frequently documented site. The causes are usually thickened fibrous and tendon bands of the deep fascia (76% of reported cases).1,3,15,17,18

2. Bicipital aponeurosis. It is the second most reported site. Hagert et al. report that this is the main constricting structure. In their study, to corroborate proper decompression, they measure the strength of the flexor pollicis longus and the flexor profundus digitorum before release and after. It has been reported as a cause in up to 42% of cases.1,3,17,18

3. The FDS. It forms aponeurotic arches. It accounts for up to 36% of cases.1,3,17,18

4. Other less common causes, such as Struthers’ ligament or Gantzer muscle (accessory head of the flexor pollicis longus).1,7

Tang et al. pointed out that it is almost impossible to differentiate between compressions produced by the PT and the FDS; therefore they considered and treated both causes at the same time. The bicipital aponeurosis, which would be the other frequent etiology capable of generating such compression, is released by the closeness and ease provided by the 3.5 cm approach performed 6 cm from the elbow crease.19

Diagnosis

Currently, there is no consensus to establish diagnostic criteria for median nerve compression around the elbow.20 However, clinical symptoms and signs, and complementary studies are described.

Clinical signs and symptoms

Tinel’s sign on the forearm: only 50%.1,3,21

Compression test: usually generates pain: being deep structures, Tang et al. emphasize that it may be non-specific or absent.1,3,19,21 Compression should be performed 6 cm from the elbow crease and 4 cm lateral to the medial epicondyle.22

Pain in the forearm and paresthesia in the forearm and the thenar eminence: they are associated with this etiology, because the palmar cutaneous branch of the median nerve is compromised.1,2,4,5,17,20,23

Thenar eminence weakness: it has been linked to compressions in the elbow, while nocturnal symptoms as well as thenar atrophy are more common in carpal tunnel syndrome (distal compressions).4,20

Scratch collapse test: it has been proposed as a method to reveal compression sites. It is more accurate if, in addition, the test is carried out with ethyl chloride, as it achieves a similar sensitivity and specificity as other provocation diagnostic tests.6

Dynamic tests: they are considered positive if they produce paresthesia. The effectiveness (sensitivity and specificity) of diagnostic tests that evaluate the force against resistance to reproduce symptoms of neuropathies is about 90%.16 These include:

1. Pronation against resistance with the elbow at 45°: positive for the PT.
2. Flexion against resistance with the supinated forearm: positive for compressions where the constricting structure is the bicipital aponeurosis.
3. FDS flexion: third/middle finger against resistance for 1 minute: positive for FDS.1-3,22

Complementary studies

1. Radiography: this is useful for testing for a humeral spur.3

2. Electromyography: it has been positive in only 30% (low sensitivity).3,20 However, it is requested to evaluate secondary causes of compression (such as a cervical condition, which is the most common).3,6,16,18 Specifically in the forearm, it evaluates for positive fibrillations or waves, polyphasic or long-lasting, indicative of compression of the median nerve.20

3. Magnetic resonance imaging: it has low sensitivity to detect nerve lesions (5% according to Özdemir et al.).16,20,24 In acute and subacute stages, the presence of edema in the STIR sequence could indicate nerve compression, and appears earlier than nerve changes, which usually develop after at least three weeks. In chronic stages, it is associated with fatty degeneration of the muscles innervated by the median nerve.4,6,20,24,25
4. Ultrasound: it may show hypoechogenicity before the compression site, which is due to perineural edema. Özdemir et al. observed changes in up to 57% of cases, for example, alterations such as decreased median nerve cross-sectional area with maneuvers, such as supination. The median nerve cross-sectional area is normally 7-9.8 mm at the carpal tunnel level. In the forearm, ultrasound signs may not be evident, as the compressive bands are often too small to be visualized with this study. However, it is operator-dependent and should not be used in isolation to diagnose compressive neuropathy. Even with all limitations, Özdemir et al. note that ultrasound is the most sensitive study to diagnose median nerve compression in the forearm.

Treatment

Conservative treatment

It is the initial treatment to be instituted and should last at least three to six months. It is based on non-steroidal anti-inflammatory drugs, changes in activities to avoid exercises that require prolonged elbow flexion, forearm pronation and prolonged grip strength with the hands. In addition, kinesiotherapy is indicated for the stretching of the muscles of the forearm. It has also been proposed to administer corticosteroid injections, which achieve good therapeutic outcomes.

In some cases, conservative treatment may need to be prolonged for up to one year to achieve a favorable response. This occurs with compressions that generate only motor symptoms with the impossibility of performing the OK sign (Kiloh-Nevin sign) in which anterior interosseous nerve syndrome should be considered as a diagnosis. This may be part of Parsonage-Turner syndrome (a brachial plexus neuritis secondary to a viral infection that presents with omalgia, symptoms of irritative nerve disease, and is usually associated with anterior interosseous nerve syndrome). In these cases, conservative treatment should be prolonged for up to one year. In the presence of this syndrome, decompression is only recommended if there is associated severe neurological damage.

Surgical treatment

Surgery has historically been based on an anterior S-shaped approach to the elbow, which allowed all structures that might be involved to be explored and released.

Then, selective releases started to be performed through smaller incisions of the structures that were most often related to compression, and that achieved similar improvement rates of up to 70-90%.

Surgical options

1. Hagert proposes a 3 cm approach in the elbow crease, with the WALANT technique (Wide Awake Local Anesthesia No Tourniquet) to perform preoperative and intraoperative measurements of the force of the flexor pollicis longus and the flexor digitorum profundus. With their mini-invasive technique, they operated on 82 patients and the DASH score improved from 35 to 12.7 (a statistically significant value). In addition, pain and numbness measured with the visual analog scale improved to 1 after 6 months, and patient satisfaction in their series was 8.8. In a 13-patient series, Lee reported resolution of paresthesia and a >50% improvement in the DASH score.
5. Ultrasound-guided hydrodissection. It is performed under local anesthesia placed proximal to the compression site. Then, with the injection of 5 ml of 1% lidocaine, it is attempted to generate a 360° decompression in the compression zone determined by ultrasound. Following decompression, the site is immobilized for four weeks before the patient can resume normal activities. The limitation is the need to visualize the compression site by ultrasound (present in only 50% of cases. It is visible as fascicular edema, epineural thickening, and changes in nerve gauge). In some case series, symptoms are reported to have improved more than 75% in 70% of patients treated with this technique.26

FINAL CONSIDERATIONS

Compression of the median nerve can occur at different levels in the upper limb. The lack of an accurate and proper diagnosis can lead to the persistence of symptoms; therefore, we believe that a detailed and thorough physical examination can help reduce errors due to the lack of proper treatment. Finally, initial treatment is usually conservative. If this fails, surgical resolution techniques can be chosen, supported by reports of satisfactory outcomes in symptom improvement when treated with any of the multiple surgical options and techniques currently available.

J. F. Civit ORCID ID: https://orcid.org/0000-0001-5496-3613
P. Ramírez Vargas ORCID ID: https://orcid.org/0000-0002-1869-8156
F. Melibosky Ramos ORCID ID: https://orcid.org/0000-0003-3806-6707
A. Villavicencio Achurra ORCID ID: https://orcid.org/0000-0003-5482-494X
J. Román Vees ORCID ID: https://orcid.org/0000-0002-8917-1062
J. Román Veas ORCID ID: https://orcid.org/0000-0001-9765-6984
P. Orellana Araya ORCID ID: https://orcid.org/0000-0001-6506-6936
R. Jorquera Aguilera ORCID ID: https://orcid.org/0000-0003-3288-3787

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