Double crush syndrome of the median nerve in the arm: A critical review of the literature

Ignacio Rellán, Mariana Bendersky, Ezequiel E. Zaidenberg, Agustín G. Donndorff, Gerardo L. Gallucci, Pablo De Carli, Jorge G. Boretto

Hand and Upper Limb Surgery Department, Instituto de Ortopedia y Traumatología "Dr. Prof. Carlos E. Ottolenghi", Hospital Italiano de Buenos Aires (Buenos Aires, Argentina)

ABSTRACT

Double crush syndrome is the mechanical compression of a peripheral nerve at two different sites and is based on the hypothesis that a nerve that has been compressed at a distal site is especially susceptible to also be compressed, asymptomatically, at a more proximal site. While carpal tunnel release is a surgical procedure with predictable results, some patients do not improve as expected after surgery. If comorbidities such as diabetes, advanced cases presenting with muscle atrophy or incomplete decompressions are excluded from the analysis, many of these treatment failures could be explained by a second concomitant compression site, which is often underdiagnosed. The very existence of double crush syndrome is highly questioned, but also its incidence and pathophysiology. The objective of our paper is to perform a critical review of the literature available on double crush syndrome involving mainly the median nerve in the wrist and the elbow.

Keywords: Carpal tunnel; median nerve; pronator teres syndrome.

Doble compresión del nervio mediano en el brazo. Revisión crítica de la bibliografía

RESUMEN

La compresión mecánica de un nervio periférico en dos sitios diferentes a lo largo de su trayecto se define como síndrome de doble compresión. Esta enfermedad se basa en la teoría de la mayor susceptibilidad que tendría un nervio a nivel distal cuando este también se encuentra comprimido, en forma asintomática, a nivel proximal, debido a una alteración en el flujo axonal. Si bien la descompresión del túnel carpiano es una cirugía con resultados previsibles, hay pacientes operados por síndrome del túnel carpiano que no mejoran después de una cirugía, como cabría esperar. Si se excluye de este análisis a las comorbilidades, como diabetes, casos avanzados con atrofia muscular o descompresiones insuficientes, muchos de estos fracasos terapéuticos podrían estar fundamentados por el escaso diagnóstico de un segundo sitio de compresión concomitante. No obstante, existe gran controversia alrededor del síndrome de doble compresión que involucra no solo a su existencia, sino también a su incidencia y fisiopatología. El objetivo de esta publicación es presentar una revisión bibliográfica crítica del síndrome de doble compresión centrada en el compromiso del nervio mediano tanto en la muñeca como en el codo.

Palabras clave: Túnel carpiano; nervio mediano; síndrome del pronador redondo.

INTRODUCTION

Stemming from cervical and thoracic spinal nerve roots, the median nerve (MN) courses from the brachial plexus to the hand. During its trajectory, it may become compressed at several sites: the carpal tunnel (carpal tunnel syndrome [CTS]), the elbow (pronator teres syndrome [PTS]), the thoracic outlet (thoracic outlet syndrome [TOS]) or the cervical spine (cervical radiculopathy). Unlike CTS, where the anatomical site of compression never changes, the literature suggests that when the MN is compressed at the elbow, there are various anatomical structures involved, such as the deep fascia of the pronator teres (PT) muscle, hypertrophy at the site of the PT muscle attachment to the deep fascia, the arcade of the flexor digitorum superficialis muscle, or the bicipital aponeurosis.[1,2] The literature uses the term PTS broadly to refer to any compression of the MN at the elbow.

Received on January 4th, 2019. Accepted after evaluation on May 11th, 2019 • IGNACIO RELLÁN, MD • ignacio.rellan@hospitalitaliano.org.ar

How to cite this paper: Rellán I, Bendersky M, Zaidenberg EE, Donndorff AG, Gallucci GL, De Carli P, Boretto JG. Double crush syndrome of the median nerve in the arm: A critical review of the literature. *Rev Asoc Argent Ortop Traumatol* 2019;84(4):427-433. http://dx.doi.org/10.15417/issn.1852-7434.2019.84.4.937

In 1973, Upton and McComas coined the term *double crush syndrome* (DCS) to name the mechanical compression of a peripheral nerve at two different sites along its course. Their study was also the first to state the hypothesis that a proximal source of nerve compression will render the distant nerve segment more susceptible to a second site of compression.

Later, a Japanese research team conducted an experimental study on canine sciatic nerves. The animals were group as follows: Group 1 was applied a single compression clamp, while Group 2 was applied two compression clamps at two different sites. The study showed that mean motor nerve conduction velocity was higher in the double crush situation group. Histologic studies of the harvested nerves showed a reduction in the number of myelinated fibers and axonal degeneration distal to the compression site, which was more severe in the double crush situation group. Additionally, the loss of nerve function after a double lesion was greater than the sum of the deficits after each separate lesion. It was concluded that altering axonal transport could induce higher susceptibility in axons located further from the distal compression site.[3] The "sum of the deficits" concept was also studied by Dellon and Mackinnon,[4] using another animal model. Dellon and Mackinnon arrived at the conclusion that the existence of two sites of compression will result in a significantly higher nerve dysfunction than a single one.

However, these conclusions are still questioned within the scientific community.[5]

In addition to the controversy on the existence of the double crush syndrome, there is debate on its incidence[6-8] and even its underlying mechanisms. In a Delphi study,[9] a panel of experts concluded that four mechanisms were considered highly plausible: impaired axonal transport, neuroma-in-continuity, ion channel up/downregulation, and inflammation in the dorsal root ganglia. Although several animal studies revealed that axonal transport is impaired by nerve compression, no study has been able to establish that a simultaneous compression at two different sites entails a different pathophysiology.

There is also lack of consensus on whether the term *double crush syndrome* is the most accurate one to refer to the simultaneous compression of a peripheral nerve at two different sites.[10-12]

Our objective is to perform a critical review of the literature available on DCS involving mainly the MN entrapment on the wrist and the elbow.

APPLIED ANATOMY

The MN contains fibers from the 5 nerve roots of the brachial plexus: C5, C6, C7, C8, and T1. With the exception of an occasional branch to the PT muscle, the MN gives off no branches to the elbow. The MN passes below the bicipital aponeurosis and between the two heads of the PT muscle, and then goes deep below the fibrous arcade of the flexor digitorum superficialis muscle. The anterior interosseous nerve branches from the MN, 4-10 cm proximal to the wrist flexion crease, after which the MN gives off the palmar cutaneous branch.[13] The palmar cutaneous branch usually has its origin on the radial or volar aspect of the wrist and follows its way through the tendons of the radial flexor muscle of wrist and the long palmar muscle to innervate the skin of the thenar region. Then, the MN passes through the carpal tunnel. The roof of the carpal tunnel is formed by the flexor retinaculum which attaches to the following carpal bones: the scaphoid, the trapezium, the triquetrum, and the hamate. Nine other structures pass through the carpal tunnel: the flexor pollicis longus tendon, the four tendons of the flexor digitorum superficialis muscle, and the four tendons of the flexor digitorum profundus muscle. In its distal section, the MN gives off its recurrent motor branch, which innervates the abductor pollicis brevis muscle, the opponens pollicis muscle, and the superficial head of flexor pollicis brevis muscle. Although considerable variations in the anatomy have been reported, the most common motor branch patterns are as follows: extraligamentous (46-90%), subligamentous (31%), and transligamentous (23%). The MN then divides into three digital branches which innervate the thumb, the index and the middle fingers, and the radial side of the ring finger.

ELECTROMYOGRAPHY[14]

The electrophysiologic evaluation of a patient suspected of having CTS is directed toward the following:

1. Demonstrating conduction block of median nerve fibers across the carpal tunnel.
2. Excluding median neuropathy at the elbow, the brachial plexus or the cervical spine, especially at C6 and C7.
3. If a coexistent polyneuropathy is present, ensuring that any median slowing at the wrist is out of proportion to slowing expected from the polyneuropathy alone.

However, a patient with a single peripheral entrapment may present much higher nerve conduction values than a standard CTS patient, which may mislead the treating physician. The key to ruling out a concomitant neuropathy is to study the nerve conduction values of the ulnar nerve (UN) or the radial nerve and compare them to those of

the MN. If the nerve slowing is similar, the neuropathy diagnosis gains ground and peripheral entrapment should be ruled out, since polyneuropathies usually affect longer nerves. A second differential diagnosis (although much less common) is an autoimmune neuropathy affecting only the MN; however, by its very definition, the nerve conduction block in those cases has to take place at a random site, different from the common MN entrapment sites.

From a pathophysiological perspective, MN entrapment produces demyelination that may be associated with an axonal loss depending on the severity of the compression. On an EMG, a demyelinating lesion results in slowing of the motor and sensory latencies. However, in 10-25% of patients this study will be normal, so the CTS diagnosis will be missed. In these patients, it is vital to compare the MN with another nerve of the upper limb (usually, the UN) or with the MN of the other hand.

The most common median-versus-ulnar comparison tests are:

- Palm-to-wrist mixed nerve latencies.
- Median-versus-ulnar wrist-to-digit 4 sensory latencies.
- Median (second lumbrical)-versus-ulnar (interossei [INT]) distal motor latencies.

The physician must be extremely thorough with all the technical aspects of these tests, such as the identical distance and position between the stimulating electrode and the recording electrode, and the magnitude of the stimulus, so as to create an "ideal" setting where the only variable between the MN and the UN EMG recordings would be the passage of the MN through the carpal tunnel.

The diagnostic efficiency of EMG to assess the MN is close to 75% using routine motor and sensory studies alone, but it rises up to approximately 95% when using the previously mentioned median-versus-ulnar comparison tests.

The key muscle to evaluate in a CTS patient is the abductor pollicis brevis, because its exclusively innervated by the MN. This muscle presents normal results during the early stages and altered results during the intermediate and advanced stages where an axonal loss will result in denervation and reinnervation.

If the EMG recording of the abductor pollicis brevis is abnormal, the proximal muscles innervated by the MN should be examined as well as at least two muscles not innervated by the MN and innervated by the C8-T1 root (primary inferior trunk). Additionally, the muscles innervated by the C6-C7 root must be studied to rule out cervical radiculopathy. The PT and the radial flexor muscle of wrist become very handy in this approach, because both may be used as median-innervated muscles proximal to the carpal tunnel and as C6-C7 innervated muscles (since median-innervated distal muscles depend on C8-T1 roots).

What is the difference between the previously explained EMG approach for the diagnosis of CTS and the EMG for the diagnosis of PTS? The key lies in studying several median-innervated muscles proximal to the wrist (PT muscle, radial flexor muscle of wrist, flexor digitorum superficialis, index and middle finger flexor digitorum profundus muscle, quadrate pronator muscle or flexor pollicis longus). If any of these muscles provides abnormal results, the compression site should be proximal to the carpal tunnel. Patients with PTS present altered electromyographic patterns more commonly in the flexor pollicis longus and in the index and middle finger flexor digitorum profundus muscle, and less commonly in the flexor digitorum superficialis and, only in rare occasions, in the PT muscle itself (since the compression site is usually distal to its innervation).

If the EMG study of any proximal median-innervated muscles provide abnormal results, it is vital to evaluate other muscles innervated by the same myotome, but through different branches of the MN, to rule out a lesion located more proximally to the brachial plexus or the cervical nerve roots. At the very least, the test should evaluate a muscle innervated by C6-C7 and not by the MN (e. g. the triceps) and a muscle innervated by C8-T1 and also not innervated by the MN (e. g. the first dorsal interosseous). Also, C8 fibers and particularly T1 fibers that constitute the inferior trunk are commonly impaired in TOS. Sensory nerve conduction tests show altered conduction in the UN and the medial antebrachial cutaneous nerve, but normal conduction in the MN, since its sensory fibers come from the primary superior and middle trunk of the brachial plexus. Instead, in motor nerve conduction tests, thenar muscles innervated by the MN (such as the abductor pollicis brevis) are more affected than the hypothenar muscles on account of the great supply of T1 to these motor branches.

In 2007, the American Academy of Orthopedic Surgeons (AAOS) published a series of recommendations for the diagnosis of CTS.[15] Among them, recommendation 3.2 suggests that if a physician orders EMG tests for the diagnosis of CTS, the testing protocol should evaluate the following:

- Sensory nerve conduction to the MN with distal latency compared to the ulnar and radial nerve.
- Median motor nerve conduction in most patients.
- Needle EMG at the discretion of the physician.

The previous technical considerations and the AAOS recommendations allow us to state that there is no unique way to conduct an EMG, and clearly show why this is a highly operator-dependent test.

A CRITICAL REVIEW OF THE LITERATURE

CTS in its early stages is marked by paresthesia and pain in the median region at night-time. As the compression becomes chronic, symptoms may include thenar muscle atrophy as a consequence of the loss of strength and permanent paresthesia. Patients suffering from PTS usually experience pain on the anterior side of the forearm and have positive procedures that reproduce its symptoms, such as the external compression at PT level and resisted pronation, which indicates a dynamic compression of the MN in its course through both PT attachments. The resisted supination and flexion of the elbow, and the external compression on the bicipital aponeurosis may lead us to consider this structure as the origin of the compression.[16] Additionally, the resisted flexion of the middle finger indicates a possible compression of the MN in its course below the fibrous arcade proximal to this muscle. Two symptoms help to distinguish PTS from CTS. First, PTS-related paresthesia involves the thenar region innervated by the palmar cutaneous branch of the MN, which, as was previously described, originates proximal to the wrist. Second, PTS does not commonly produce characteristic nocturnal symptoms as CTS does during its early stages.[17,18]

There is an overall consensus on the role of complementary tests and especially of the EMG in diagnosing CTS; however, this is not the case for PTS. The poor diagnostic sensitivity of the EMG concerning PTS stems from the fact that although a nerve can be compressed and cause distal symptoms resulting from alterations on nerve conduction,[19] pressure levels are too weak to cause axonal lesions[20] and therefore they produce no visible changes in the EMG. In addition, electrophysiologic tests have also failed to provide a pathognomonic sign of DCS.[21]

Owing to its non-invasive and affordable nature, EMG is becoming increasingly popular to study peripheral nerve compressions, and provides a possible source of additional information concerning the potential origin of the compression.[22,23] However, its limitations are the scarce specific literature available and its operator-dependent nature. Notwithstanding all of the above, the tendency of using ultrasound as a diagnostic method seeks not to replace electrophysiologic tests, but to complement them.[24] The technological development of MRI has also enabled this procedure to be considered for studying peripheral nerve compressions. However, no abnormal findings can be detected before the advanced stages, and by that time the axonal degeneration is obvious.[25] This restricts its use to the study of the spine and tumor-related compressions.

While carpal tunnel release is a surgical procedure with predictable results, some patients do not improve as expected after surgery.[8,26-29] If comorbidities such as diabetes, advanced cases presenting with muscle atrophy or incomplete decompressions are excluded from the analysis, many of these treatment failures could be explained by a second concomitant compression site, which is often underdiagnosed (i.e. PTS). In a retrospective series of 39 patients who were operated on for PTS, Hartz *et al*.[29] reported that 3 of them had originally undergone surgery for CTS. In another similar study, Olehnik *et al*.[28] reported that half of their series of 36 patients had initially been treated for CTS. Mujadzic *et al*.[27] reported a series of 61 patients with concomitant CTS and PTS, who underwent simultaneous release of the MN at the wrist and the anterior side of the elbow. Postoperative results showed that 39 patients experienced complete relief, and 13 had partial relief, 5 of which had TOS, and 3 of which had cervical radiculopathy. It is worth noticing that in this study it was possible to intraoperatively confirm MN entrapment at the elbow in 55 of the 61 cases. The structures responsible for the compression were the fibrous band of the deep head of the PT (41 cases), the same band with agenesis of the deep head of the PT (12 cases), hypertrophy of the superficial head (also with agenesis of the deep head of the PT) (2 cases), the arcade of the flexor digitorum superficialis muscle (11 cases), the bicipital aponeurosis (7 cases), and Struthers's ligament (2 cases).

In another retrospective series, Luangjarmekorn *et al*.[30] studied the treatment outcome of patients with failed carpal tunnel release surgery who were suspected of having PTS. A group of 20 patients underwent a revision carpal tunnel release with PT release and was compared to another group (5 patients) that only underwent a revision carpal tunnel release. In the double decompression group, pain improved in 60% and paresthesia in 55%. The key surgical findings were deep head hypertrophy of the PT (90%) and compression of the bicipital aponeurosis (50%). The group that underwent revision carpal tunnel release alone did not improve. A noteworthy finding was a neoformation of the transverse carpal ligaments in 20 of the 25 participants. The study showed that MN release combined with revision carpal tunnel release in patients with proximal compression symptoms provided better outcomes than carpal tunnel release alone.

Hsiao *et al*.[31] published a retrospective series of 344 patients with CTS, 21 of which involved a double compression. Following the MN release procedure in both sites, 15 patients showed complete relief.

Although the term *DCS* has been around for almost half a century, its use has been recently questioned given that such definition may be considered to focus exclusively on mechanical compression, excluding other medical or pharmacological processes that might be partly responsible for this "increased susceptibility." As a result, the expression *multifocal neuropathy* was proposed,[10] which has also become a source of debate.[11]

CONCLUSIONS

Leaving aside any controversy, the concept of double crush syndrome is important, for it requires the surgeon to be aware of a global patient approach, not focusing only on the most common anatomical sites of compression. DCS is not just a nerve compression syndrome. DCS may be secondary to several metabolic and systemic diseases, such as diabetes, infectious diseases, hypothyroidism, vitamin deficiencies or alcoholism, which may alter nerve physiology and increase compression susceptibility in peripheral nerves.[32]

Patients who come to consult with hand paresthesia should be approached with an open mind and a global perspective which may allow for detecting cases featuring complex or multifactorial disorders, different from a standard case of CTS. A thorough medical history together with an "old-school" methodical, exhaustive and systematic physical examination would allow for reaching presumptive diagnoses that will guide the request for complementary studies. Although CTS diagnosis is based on clinical assessment, EMG has a role in testing patients with a presumptive diagnosis of DCS, ruling out other neuropathies and helping to confirm or to rule out other compression sites. As it is an operator-dependent test, we consider the communication between the surgeon and the EMG technician to be vital, for it enables for a better interpretation of the information an EMG may provide.

In cases where the second site of compression cannot be precisely determined, our approach is the same as that of other authors: to explain to the patient that, in the event that carpal tunnel release does not provide the expected improvement, it will be necessary to perform another surgery to release the second site of compression.

It is concluded that DCS does not only involve the mechanical compression of a peripheral nerve at two different sites. A second factor may in some cases be associated with an endocrine or pharmacological cause.

A full medical history, a comprehensive and systematic physical examination, and sensibly ordered complementary tests build up the foundations of our treatment approach to these patients.

Conflict of interest: Authors claim they do not have any conflict of interest.

M. Bendersky ORCID ID: http://orcid.org/0000-0002-8945-9920
E. E. Zaidenberg ORCID ID: http://orcid.org/0000-0002-1535-0586
A. G. Donndorff ORCID ID: http://orcid.org/0000-0002-6384-4820
G. L. Gallucci ORCID ID: http://orcid.org/0000-0002-0612-320X
P. De Carli ORCID ID: http://orcid.org/0000-0002-9474-8129
J. G. Boretto ORCID ID: http://orcid.org/0000-0001-7701-3852

REFERENCES

1. Mackinnon SE, Novak CB. Compression neuropathies. En: Wolfe SW, Hotchkiss RN, Pederson WC, Kozin SH, Cohen MS, eds. *Green's operative hand surgery*, 7th ed. Philadelphia, PA: Elsevier/Churchill Livingston; 2016:921-1172.
2. Zancolli ER, Zancolli EP, Perrotto CJ. New mini-invasive decompression for pronator teres syndrome. *J Hand Surg Am* 2012;37(8):1706-10. https://doi.org/10.1016/j.jhsa.2012.05.033
3. Nemoto K, Matsumoto N, Tazaki K, Horiuchi Y, Uchinishi K, Mori Y. An experimental study on the "double crush" hypothesis. *J Hand Surg Am* 1987;12(4):552-9. https://doi.org/10.1016/s0363-5023(87)80207-1
4. Dellon AL, Mackinnon SE. Chronic nerve compression model for the double crush hypothesis. *Ann Plast Surg* 1991;26(3):259-64. https://doi.org/10.1097/00000637-199103000-00008

5. Wilbourn AJ, Gilliatt RW. Double-crush syndrome: a critical analysis. *Neurology* 1997;49(1):21-9. https://doi.org/10.1212/wnl.49.1.21
6. Dorwart BB. Carpal tunnel syndrome: a review. *Semin Arthritis Rheum* 1984;14(2):134-40. https://doi.org/10.1016/0049-0172(84)90003-9
7. Bednarik J, Kadanka Z, Vohánka S. Median nerve mononeuropathy in spondylotic cervical myelopathy: double crush syndrome? *J Neurol* 1999;246(7):544-51. https://doi.org/10.1007/s004150050401
8. Baba H, Maezawa Y, Uchida K, Furusawa N, Wada M, Imura S, et al. Cervical myeloradiculopathy with entrapment neuropathy: a study based on the double-crush concept. *Spinal Cord* 1998;36(6):399-404. https://doi.org/10.1038/sj.sc.3100539
9. Schmid AB, Coppieters MW. The double crush syndrome revisited--a Delphi study to reveal current expert views on mechanisms underlying dual nerve disorders. *Man Ther* 2011;16(6):557-62. https://doi.org/10.1016/j.math.2011.05.005
10. Cohen BH, Gaspar MP, Daniels AH, Akelman E, Kane PM. Multifocal neuropathy: expanding the scope of double crush syndrome. *J Hand Surg Am* 2016;41(12):1171-5. https://doi.org/10.1016/j.jhsa.2016.09.009
11. Naam NH. First commentary on "Multifocal Neuropathy: Expanding the Scope of Double Crush Syndrome". *J Hand Surg Am* 2016;41(12):1176. https://doi.org/10.1016/j.jhsa.2016.09.010
12. Curtin C. Second commentary on "Multifocal Neuropathy: Expanding the Scope of Double Crush Syndrome". *J Hand Surg Am*. 2016;41(12):1177. https://doi.org/10.1016/j.jhsa.2016.09.011
13. Martin CH, Seiler JG, Lesesne JS. The cutaneous innervation of the palm: an anatomic study of the ulnar and median nerves. *J Hand Surg Am* 1996;21(4):634-8. https://doi.org/10.1016/S0363-5023(96)80017-7
14. Preston DC, Shapiro BE. Median neuropathy at the wrist. En: Preston DC, Shapiro BE. *Electromyography and neuromuscular disorders.* 3rd ed. Cleveland, OH: Elsevier; 2013.
15. American Academy of Orthopaedic Surgeons Work Group Panel. Clinical guidelines on diagnosis of carpal tunnel syndrome. 2007. Disponible en: www.aaos.org/research/guidelines/CTS_guideline.pdf
16. Hagert E. Clinical diagnosis and wide-awake surgical treatment of proximal median nerve entrapment at the elbow: a prospective study. *Hand (N Y)* 2013;8(1):41-6. https://doi.org/10.1007/s11552-012-9483-4
17. Rehak DC. Pronator syndrome. *Clin Sports Med* 2001;20(3):531-40. https://doi.org/10.1016/S0278-5919(05)70267-2
18. Rodner CM, Tinsley BA, O´Malley MP. Pronator syndrome and anterior interosseous nerve syndrome. *J Am Acad Orthop Surg* 2013;21(5):268-75. https://doi.org/10.5435/JAAOS-21-05-268
19. Rydevik B, Lundborg G. Permeability of intraneural microvessels and perineurium following acute, graded experimental nerve compression. *Scand J Plast Reconstr Surg* 1977;11(3):179-87. https://doi.org/10.3109/02844317709025516
20. Dahlin LB, Lundborg G. The neurone and its response to peripheral nerve compression. *J Hand Surg Br* 1990;15(1):5-10. https://doi.org/10.1016/0266-7681(90)90040-b
21. Lo SF, Chou LW, Meng NH, Chen FF, Juan TT, Ho WC, et al. Clinical characteristics and electrodiagnostic features in patients with carpal tunnel syndrome, double crush syndrome, and cervical radiculopathy. *Rheumatol Int* 2012;32(5):1257-63. https://doi.org/10.1007/s00296-010-1746-1
22. Akyuz M, Yalcin E, Selcuk B, Onder B, Ozçakar L. Electromyography and ultrasonography in the diagnosis of a rare double-crush ulnar nerve injury. *Arch Phys Med Rehabil* 2011;92(11):1914-6. https://doi.org/10.1016/j.apmr.2011.05.007
23. Liotta G, Granata G, Librante A, di Pasquale A, Caliandro P, Martinoli C, et al. Atypical double nerve lesion after humeral fracture: diagnosis by ultrasound. *Muscle Nerve* 2010;41(2):287-8. https://doi.org/10.1002/mus.21580
24. Seror P. Sonography and electrodiagnosis in carpal tunnel syndrome diagnosis, an analysis of the literature. *Eur J Radiol* 2008;67(1):146-52. https://doi.org/10.1016/j.ejrad.2007.06.017
25. Andreisek G, Crook DW, Burg D, Marincek B, Weishaupt D. Peripheral neuropathies of the median, radial, and ulnar nerves: MR imaging features. *RadioGraphics* 2006;26(5):1267-87. https://doi.org/10.1148/rg.265055712
26. Osterman AL. The double crush syndrome. *Orthop Clin North Am* 1988;19(1):147-55. PMID: 3275922
27. Mujadzic M, Papanicolaou G, Young H, Tsai TM. Simultaneous surgical release of ipsilateral pronator teres and carpal tunnel syndromes. *Plast Reconstr Surg* 2007;119(7):2141-7. https://doi.org/10.1097/01.prs.0000260703.56453.06

28. Olehnik WK, Manske PR, Szerzinski J. Median nerve compression in the proximal forearm. *J Hand Surg Am* 1994;19(1):121-6. https://doi.org/10.1016/0363-5023(94)90235-6

29. Hartz CR, Linscheid RL, Gramse RR, Daube JR. The pronator teres syndrome: compressive neuropathy of the median nerve. *J Bone Joint Surg Am* 1981;63(6):885-90. https://doi.org/10.2106/00004623-198163060-00004

30. Luangjarmekorn P, Tsai TM, Honsawek S, Kitidumrongsook P. Role of pronator release in revision carpal tunnel surgery. *SICOT J* 2016;2:9. https://doi.org/10.1051/sicotj/2016006

31. Hsiao C-W, Shih J-T, Hung S-T. Concurrent carpal tunnel syndrome and pronator syndrome: A retrospective study of 21 cases. *Orthop Traumatol Surg Res* 2017;103(1):101-103. https://doi.org/10.1016/j.otsr.2016.10.009

32. Morgan G, Wilbourn AJ. Cervical radiculopathy and coexisting distal entrapment neuropathies: double-crush syndromes? *Neurology* 1998;50(1):78-83. https://doi.org/10.1212/wnl.50.1.78