

Case Report

Giant median nerve in bilateral carpal tunnel syndrome

Hosein Ahmadzadeh Chabok

Department of Surgery, Shamsoshomoos Hospital, Malekoshora Bahar 54, Shariati Square, Mashhad, Khorasan Razavi, Iran

Address for correspondence: Dr. Hosein Ahmadzadeh Chabok, Shamsoshomoos Hospital, Malekoshora Bahar 54, Shariati Square, Mashhad, Khorasan Razavi, Iran. E-mail: h.ahmadzadeh.c@gmail.com

ABSTRACT

We introduce a middle age healthy man with sequential bilateral carpal tunnel syndrome. At the surgery, we encountered a wide median nerve in both wrists. Although enlargement of median nerve in carpal tunnel has been well documented, 25 mm width of the nerve is a rare scene, underscoring that leaving the nerve under the unyielding pressure would lead to a fibrous atrophic median nerve.

KEY WORDS

Blood-nerve barrier; carpal tunnel syndrome; compressive neuropathy; median nerve; neural edema

INTRODUCTION

Carpal tunnel syndrome is the most common entrapment neuropathy involving the median nerve under the unyielding fibrous transverse carpal ligament. Acute or chronic elevation of carpal tunnel pressure may lead to carpal tunnel syndrome.^[1,2] Surgical release of carpal tunnel is an effective treatment and in substantial majority, surgery is necessary to relieve the symptoms efficiently.^[1,2]

CASE REPORT

An otherwise healthy 41-year-old patient presented to our orthopaedic clinic with wrist pain. He was secretary of an office with a heavy writing job and a recently sudden increase in his duty without a chance to reduce his heavy working load.

He had no important sign and symptom but a localised pain at the volar surface of the wrist of his right dominant hand. A ten days course of NSAID was prescribed. Three weeks later, he came back with more severe pain at the wrist and we advised him to immobilise the wrist with a volar wrist splint and another two weeks of NSAID therapy. One month later, he returned with a localised swelling on the volar aspect of the wrist, numbness and tingling on the palmar surface of the entire hand and a burning pain radiating proximally to the forearm. Electrodiagnostic evaluation revealed moderate to severe median nerve neuropathy at the wrist suggestive of carpal tunnel syndrome. Since the symptoms were not typical for carpal tunnel syndrome in order to exclude unusual etiologies, we suggested a wrist MRI which revealed median nerve swelling and notable edema in carpal tunnel. We planned open surgical release of the carpal tunnel with a classical incision at the wrist. At the surgery, we were surprised of a wide 25-mm swollen median nerve occupying nearly entire width of the carpal tunnel with tiny surface vessels [Figure 1]. Transverse carpal ligament was incised. To prevent recurrence, 2 mm of the ligament was excised. The patient experienced a painless post operation period and soon returned to work.

Access this article online

Quick Response Code:



Website:

www.ijps.org

DOI:

10.4103/0970-0358.113735

Eleven months later, he came back to our clinic with a painful fusiform swelling along the palmar aspect of the middle finger extending to the palm of the left hand. We advised him to give a rest to his hand and a 10 days course of NSAID therapy; with good response and subsidence of swelling. Three weeks later, he was suffering from a severe pain and mild swelling at the volar surface of the left wrist with tingling and numbness on the palmar surface of all digits. The pain was burning in nature and radiating up to the forearm. Having previous history in mind, after an electrodiagnostic evaluation suggestive of carpal tunnel syndrome, we planned open surgical release of transverse carpal tunnel ligament as soon as possible. We observed a sizable swollen median nerve about the same size as in the other hand occupying the entire width of the carpal tunnel [Figures 2 and 3]. After incising transverse carpal ligament, we excised 2-mm width of the ligament. Symptoms completely relieved after the surgery and he returned to previous job. Thirty-two months after the first surgery and nineteen months after the second surgery, the patient had no pain, no swelling and no neurological symptoms and objective signs of sensory deficits, weakness and/or thenar muscle atrophy.

DISCUSSION

High carpal tunnel pressure compromises median nerve electrophysiological functions leading to a package of signs and symptoms recognised as carpal tunnel syndrome.^[1,2] Postural factors including non-neutral wrist posture, forceful repetitive hand-work and vibration are some predisposing factors elevating carpal tunnel pressure but in majority, there is no identifiable cause.^[1,3,4] High carpal tunnel pressure as an external load initiates some internal responses leading to other various responses in the nerve (cascade model).

First, high pressure as a compression trauma increases permeability of epineurial vessels leading to epineurial edema. Epineurial blood vessels are more vulnerable to trauma but with exceeding pressure, breakdown of blood-nerve barrier in endoneurial microvessels leads to endoneurial edema and high endoneurial pressure. Blood-nerve barrier is a principal structure controlling endoneurial milieu. Since endoneurial space lacks lymphatics and for selective diffusion barrier formed by epineurial membrane, edema cannot be drained out. Edema reduces endoneurial microcirculation through occluding openings in perineurial membrane where



Figure 1: Sizable, swollen median nerve of right dominant hand. Median nerve is obviously edematous and occupies nearly all width of the carpal tunnel



Figure 2: Giant swollen median nerve with venous engorgement in left hand



Figure 3: Notice the size of edematous median nerve in proportion to palmaris longus tendon in left hand

the anastomosing vessels between epineurium and endoneurium pass obliquely (valve mechanism). Resultant ischemia induces other events.^[1-5]

Second, high pressure reduces epineurial venule flow. Increasing pressure compromises arteriolar flow and endoneurial capillary blood flow; consequently, ischemia occurs.^[3-5]

Cyclic Ischemia and reperfusion as an ischemic stress releases overwhelming free oxygen radicals, malonaldehyde bis (diethyl acetal). Continued oxidative stress and resultant free oxygen radicals lead to cellular injury in the nerve and synovial tissue.^[2,6,7] Cellular injury initiates the metabolism of arachidonic acid to cyclooxygenase products such as PGE2. This product as a potent vasodilator enhances vascular permeability and also sensitises nerve endings to mechanical and chemical stimuli, so that normal stimulus can be painful. Tissue levels of PGE2 and malonaldehyde bis (diethyl acetal) in patients with carpal tunnel syndrome are significantly higher than normal. Cellular damage contributes to production of cytokines (as IL6) that originate fibroblast proliferation and fibrosis in connective tissue container of the nerve. It has been demonstrated that tissue levels of IL6 in patients with carpal tunnel syndrome is highly elevated.^[6-8] Formation of fibrosis decreases excursion of nerve fibers and neural gliding, producing dynamic ischemia. These serial of events construct a cascade model of responses and a vicious circle. The end result of this sequence of events would be nerve fibers atrophy massive fibrous changes of soft tissue container of the nerve with permanent nerve injury.^[8,9]

Cross-section area of the median nerve in carpal tunnel in asymptomatic adults has been measured less than 10 mm² with Sonographic evaluation. Although enlargement of median nerve in carpal tunnel syndrome due to edema has been demonstrated by sonography in various studies, to our knowledge, this amount of enlargement (25 mm wide), as defined in our case, has never been reported.^[10]

This huge amount of edema leaving the nerve under the unyielding pressure may very soon develop to a nerve ischemia and subsequent necrosis leading to a fibrous atrophic median nerve. Prompt surgical release of carpal tunnel interrupts the process, alleviates the symptoms and promises good results.

ACKNOWLEDGEMENT

The author has received no funding and has no financial relationship to disclose.

REFERENCES

1. Aroori S, Spence RA. Carpal tunnel syndrome. *Ulster Med J* 2008;77:6-17.
2. Thatte MR, Mansukhani KA. Compressive neuropathy in the upper limb. *Indian J Plast Surg* 2011;44:283-97.
3. Viikari-Juntura E, Silverstein B. Role of physical load factors in carpal tunnel syndrome. *Scand J Work Environ Health* 1999;25:163-85.
4. Rempel D, Dahlin L, Lundborg G. Pathophysiology of nerve compression syndrome: Response of peripheral nerves to loading. *J Bone Joint Surg Am* 1999;81:1600-10.
5. Lundborg G, Myres R, Powel H. Nerve compression injury and increased endoneurial fluid pressure: A "miniature compartment syndrome". *J Neurol Neurosurg Psychiatry* 1983;46:1119-24.
6. Sud V, Freeland AE. Biochemistry of carpal tunnel syndrome. *Microsurgery* 2005;25:44-6.
7. Tucci MA, Barbieri RA, Freeland AE. Biochemical and histological analysis of the flexor tenosynovium in patients with carpal tunnel syndrome. *Biomed Sci Instrum* 1997;33:246-51.
8. Sommer C, Galbraith JA, Heckman HM, Myers RR. Pathology of experimental compression neuropathy producing hyperesthesia. *J Neuropathol Exp Neurol* 1993;52:223-33.
9. Mackinnon SE. Pathophysiology of nerve compression. *Hand Clin* 2002;18:231-41.
10. Abicalaf CA, de Barros N, Sernik RA, Pimentel BF, Braga-Baiak A, Braga L, *et al.* Ultrasound evaluation of patients with carpal tunnel syndrome before and after endoscopic release of the transverse carpal ligament. *Clin Radiol* 2007;62:891-4.

How to cite this article: Chabok HA. Giant median nerve in bilateral carpal tunnel syndrome. *Indian J Plast Surg* 2013;46:140-2.

Source of Support: Nil, **Conflict of Interest:** None declared.