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Letter to the Editor

Diagnosing and Treating Carpal Tunnel Syndrome in Primary Care Requires Identification of the Cause and Exclusion of all Differential Diagnoses

¹ Josef Finsterer, ² Carla A Scorza, ³ Fulvio Scorza

¹ Department of Neurology, Neurology & Neurophysiology Center, Vienna, Austria

^{2,3} Federal University of São Paulo (UNIFESP/EPM), São Paulo, Brazil

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Corresponding Author: Josef Finsterer

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We read with interest the article by Sze *et al.* on an overview of the clinical symptoms, causes, pathophysiology, diagnosis, treatment, and outcomes of carpal tunnel syndrome (CTS) for primary care physicians ^[1]. It concludes that family physicians play a crucial role in the early detection and treatment of CTS, that a thorough clinical examination is key to diagnosis, that the medical history should focus on nocturnal numbness, tingling in the median nerve area, and the “flick sign,” that the physical examination should include sensory and motor tests, observation for thenar atrophy and provocation tests, that differential diagnoses of CTS should be considered, that the diagnosis should be confirmed by nerve conduction studies (NCS), that NCS can quantify the severity and rule out differential diagnoses, and that moderate CTS may respond to conservative treatments, while severe or progressive cases may require surgical intervention ^[1]. The review is appealing, but some points need discussion.

The first point is that anatomical variations of the median nerve and its motor and sensory branches have not been sufficiently discussed ^[1]. In addition to the anastomoses between the median nerve and the ulnar nerve according to Martin-Gruber and Berretini ^[1], innervation of all fingers can occur either through the median nerve or the ulnar nerve (all ulnar hand, all median hand), the thenar branch can leave the median nerve within the carpal tunnel, the recurrent motor nerve (RMB) can run transligamentary, the RMB can leave the median nerve on its ulnar side, the RMB can run over the top of the transverse carpal ligament; There may be a double RMB, a high division of the median nerve with a persistent median artery (PMA) between the two parts, a high division of the median nerve with a thinner ulnar part, and a high division of the median nerve with a thicker ulnar part ^[2].

The second point is that the list of causes of CTS, as presented in Box 1 of the article, is also much longer than presented ^[1]. Additional causes include dislocation or subluxation of the carpal bone, oblique consolidation of a radius fracture, acromegaly, menopause, obesity, Dupuytren's contracture, heart failure, contraceptive use, chronic alcoholism, forceful gripping, repetitive flexion and extension of the wrist, vibration exposure, mucopolysaccharidosis types I, II, IV, and VI, direct fractures of the distal radius, tumors of the radius, leukemia, lymphomas, ganglion cysts, or PMA, exposure to toxins such as trichloroethylene, cellulose thinners, paints, varnishes, inks, dyes, and pesticides, and medications such as bisphosphonates, corticosteroids, warfarin, antiretrovirals, alendronic acid, gamma-hydroxybutyric acid, rofecoxib, tafamidis, and idursulfase ^[3]. The third point is that the list of differential diagnoses in Box 2 of Sze's article is much longer than presented. In addition to the differential diagnoses listed, CTS can only be diagnosed after thoracic outlet syndrome, pronator teres syndrome, Parsonage-Turner syndrome, Kiloh-Nevin syndrome, multiple sclerosis, overuse injuries, ischemic stroke, cervical myofascial pain, mononeuritis multiplex, radiation-induced plexopathy, and complex regional pain syndrome have been ruled out ^[4].

In summary, the diagnosis and treatment of CTS by the family physician requires comprehensive clinical, electrophysiological, and imaging examinations, as well as a thorough search for the underlying causes and the exclusion of all possible differential diagnoses.

Declarations

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