The diagnostic value of nerve ultrasound in an atypical LESION OF THE palmar cutaneous BRANCH OF THE MEDIAN nerve

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INTRODUCTION

The palmar cutaneous branch of the median nerve (PCBMN) arises in the distal forearm before the median nerve (MN) enters the carpal tunnel, then it crosses anterior to the flexor retinaculum, and gives sensory innervation to the anterior wrist capsule and the area over the thenar eminence. Distal to the carpal tunnel, the MN divides into three palmar divisions (PDMNs), which innervate the skin of the distal palm, the webspace, and the palmar side of the fingers up to the lateral half of the ring finger (Fig.1A). Injury to the PCBMN is the most common complication of open surgery for MN release in carpal tunnel syndrome. Apart from iatrogenic cases; reports of PCBMN damage are rare. We report a man who underwent PCBMN traumatic lesion after a cut to the wrist and showed an abnormally enlarged area of hypesthesia in the palm of the hand.

CASE REPORT

A 60-year-old man reported hypesthesia, paresthesia and pain in the palm of the left hand after a glass cut at the wrist. His previous clinical history was unremarkable. Examination showed an area of tactile and punctate hypesthesia in the left palm (Fig. 1B) with no motor signs. Tinel sign could be evoked at the left wrist. Nerve conduction study (NCS) documented that the left PCBMN sensory nerve action potential (SNAP) to orthodromic stimulation was absent (Fig. 1C). The SNAPs from the fingers innervated by left MN neither showed left-right asymmetries nor differed in comparison to fingers innervated by ulnar and radial nerves (Fig. 1C). The left thenar eminence compound muscle action potential and the needle electromyography in median-innervated muscles of the left hand were normal. Since the area of palm hypesthesia was larger than the cutaneous distribution of the PCBMN, the patient underwent high resolution MN ultrasound (US) with a Toshiba Xario 200 (Toshiba Medical Systems Europe, Zoetermeer, Holland) equipped with a high-frequency bandwidth (13 – 18 MHz) PLU-1204BT linear-array transducer. Nerve US documented enlarged left PCBMN cross sectional area indicating a neuroma (Fig. 2A), and coexisting fascicular lesion to the superficial side of the MN in the carpal tunnel, distal to the point where the PCBMN left the main nerve trunk (Fig. 2B). Repeated examination showed two distinct Tinel signs, one over the PCBMN neuroma, which evoked pain and paresthesia in the thenar eminence area, and another one over the MN superficial neuroma that evoked paresthesia in the distal lateral palm. Surgical exploration documented a fascicular MN lesion.

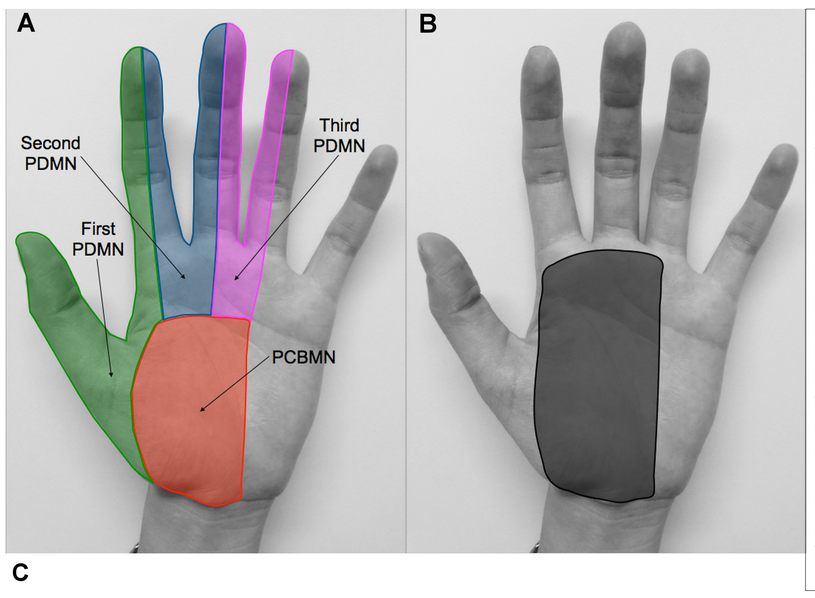
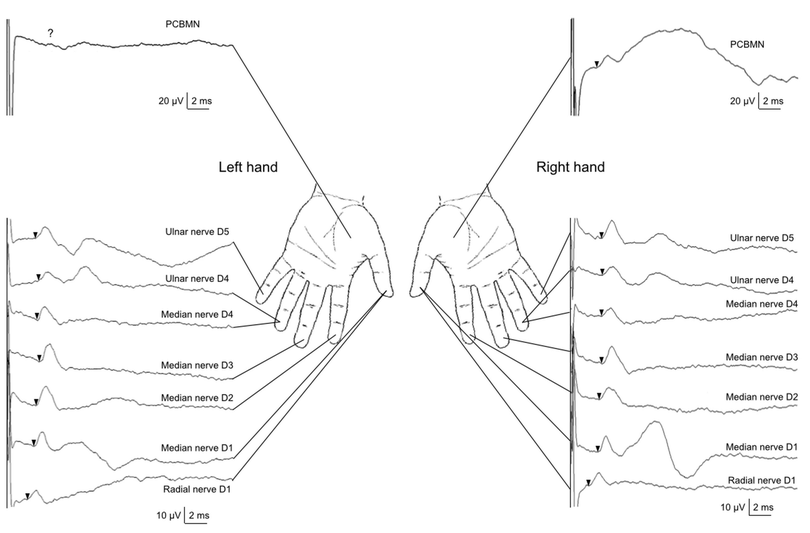


Figure 1. A. The cutaneous sensory distribution of the palmar cutaneous branch of the median nerve (PCBMN), and the first, second, and third palmar divisions of the median nerve (PDMNs). B. The area of tactile and punctate hypesthesia in the patient. The sensory deficit was larger than the distribution of the PCBMN. Please note that the hand was from a model and not that of the patient. C. Nerve conduction study results. The orthodromic sensory nerve action potential (SNAP) from the left palmar cutaneous branch of the median nerve (PCBMN) was absent (marked by ?), while that from the right PCBMN was within normal range. The antidromic SNAPs recorded from fingers innervated by the left median nerve through its palmar divisions were also within normal range, and we found neither left-to-right asymmetries nor difference in comparison to ulnar and radial antidromic SNAPs. Please note that the PCBMN SNAP was recorded orthodromically (i.e.: stimulation at the palm, recording at the wrist) while the other SNAPs were recorded antidromically (i.e.: mixed nerve stimulation at the wrist, recording at the fingers) to reduce artifacts. The filled triangles mark the SNAPs onset. D1 = thumb finger, D2 = index finger, D3 = median finger, D4 = ring finger, D5 = small finger.



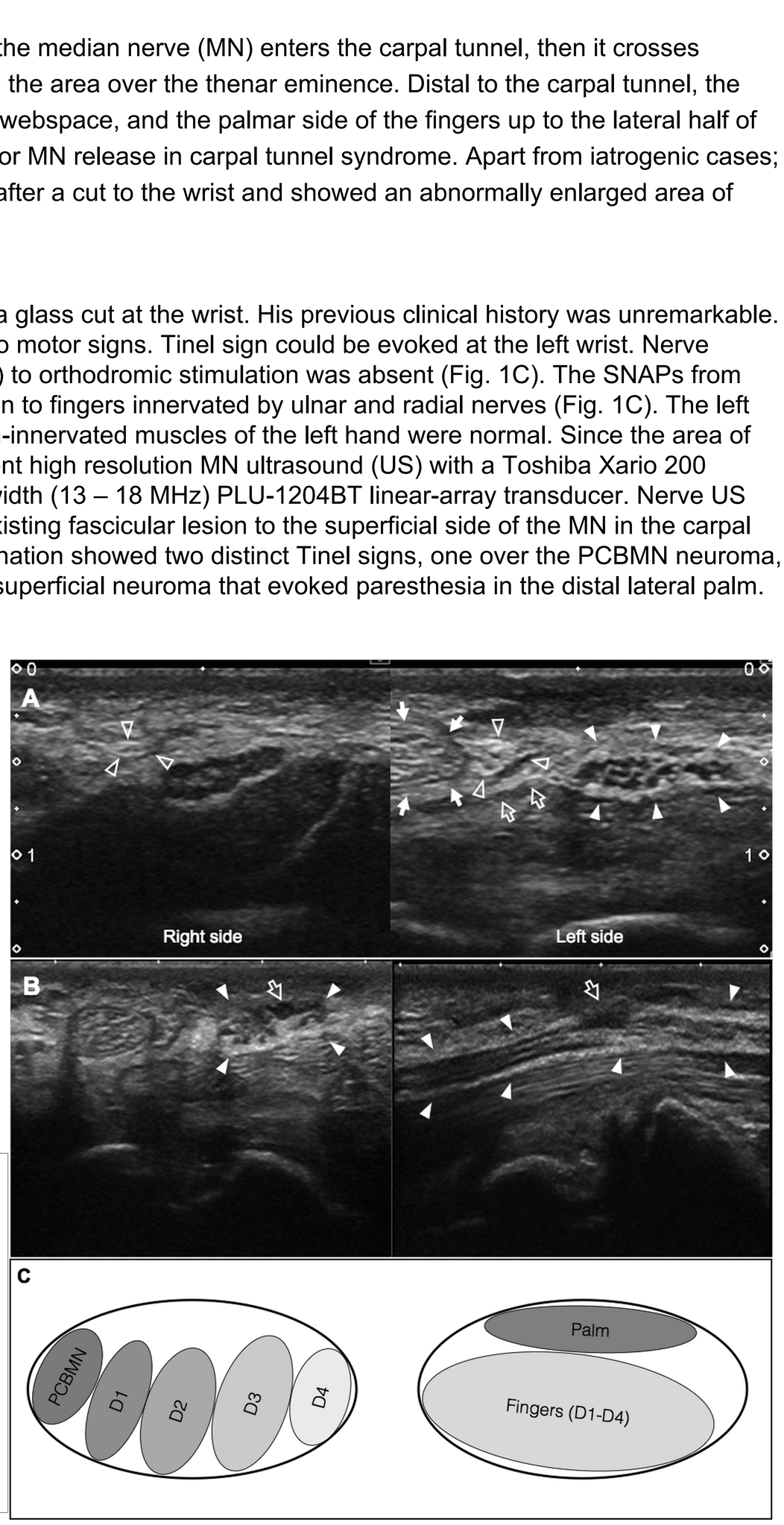


Figure 2. Nerve ultrasound findings. A. The cross sectional area (CSA) of the left palmar cutaneous branch of the median nerve (PCBMN, open arrowheads) was larger (3 mm^2) than that of the left PCBMNB (1 mm^2) and to normative data. Closed arrowheads indicate the median nerve (MN); open arrows indicate the flexor retinaculum; closed arrows indicate the flexor carpi radialis tendon. B. The axial scan of the left MN (closed arrowheads) in the carpal tunnel, distal to the point where the PCBMN left the main nerve trunk, showed a superficial fascicular hypoechoic enlargement (left panel, open arrow), which appeared as a lateral neuroma with epineurial loss-of-continuity on the longitudinal scan (right panel, open arrow). C. The somatotopical arrangement of MN fascicles. Left panel shows the lateral-to-medial distribution according to autopsy studies. Right panel indicates the possible proximal-to-distal fascicular arrangement, as suggested by our patient. D1 = thumb finger, D2 = index finger, D3 = median finger, D4 = ring finger.

DISCUSSION

We describe a patient with left PCBMN traumatic neuroma who presented an atypical enlarged distribution of sensory symptoms. This case may be of interests for two reasons. First, it supports the role of nerve US in addition to NCS in the diagnosis and management of peripheral nerve lesions. Second, it may offer some new insights into the somatotopy of MN fasciculi. In our patient, NCS documented left PCBMN damage, but thorough examination showed that the negative sensory signs (i.e: tactile and punctate hypesthesia) involved a cutaneous area, which was larger than the PCBMN distribution (Fig. 1A, B). Positive sensory symptoms (i.e.: pain and paresthesia) may sometimes spread to territories outside the damaged nerve, but this phenomenon is uncommon for negative signs. For this reason, we searched for additional nerve damage with US and documented a coexisting superficial lateral neuroma of the left MN in the carpal tunnel, distal to the PCBMN, and likely secondary to the same trauma that caused PCBMN damage. We speculate that a double crush was the reason why the patient showed the atypical enlarged sensory distribution. This hypothesis is supported by evidence of MN fascicular lesion at surgical exploration, and by the presence of two separate Tinel signs with different projection of pain and paresthesia, and corresponding to the two distinct nerve lesions. The presence of a PCBMN anatomical variant was ruled out by nerve US. Abnormal communication between the MN and other nerves may lead to unexpected sensory examination following nerve injury, and median to ulnar nerve communications have been reported, but they were unlikely to contribute to abnormal sensory findings in our patient, because hypesthesia did not spread to the ulnar territory, and US and surgical exploration showed coexistent damage to the PCBMN and the MN. The present case confirms that NCS and US may offer converging and complementary information in the diagnosis and management of peripheral nerve lesions. The MN fascicular anatomy was investigated by autopsy studies, which indicate a lateral-to-medial sensory somatotopy of MN fascicles at the wrist according to the innervated fingers (Fig. 2C). This arrangement has been confirmed in patients with traumatic fascicular damage of the MN. At variance, data on the distal-to-proximal somatotopy are lacking. Based on our patient, we suggest that fascicles innervating the distal palm are located more superficially than those for the fingers in the carpal tunnel. This view is in keeping with normal finger SNAPs. The superficial location of the sensory fibers for the distal palm is anatomically sound, as they are the first to leave the PDMNs after the carpal tunnel. Detailed knowledge of the fascicular nerve anatomy represents the basis for microsurgical repair and for the application of neural prostheses. Our preliminary data need further confirmation in a larger population of normal controls and patients.