

Localising Median Neuropathies: The Role of Different Investigations

A 50-year-old male with diabetic nephropathy and renal failure on maintenance haemodialysis through a left arterio-venous fistula was referred to the Neuroscience Clinic with pain, numbness and tingling over the lateral 3 fingers of the left hand of a few days duration. The pain increased when the fistula was used for haemodialysis. On clinical examination, the patient was unable to oppose the thumb and flex the thumb, Digit I and Digit II. He also had weakness of wrist flexion and forearm pronation, suggesting a proximal median neuropathy.

Electrodiagnostic studies (Table 1) indicated a mixed axonal and demyelinating sensori-motor polyneuropathy. Additionally, the median nerve study on the right was compatible with a median neuropathy at the wrist. Studies on the left, however, were consistent with a severe median neuropathy with absent sensory nerve action potential and markedly decreased compound muscle action potential. Distal latency to the flexor pollicis longus on proximal stimulation was prolonged indicating a left-sided proximal median neuropathy.

Which investigations can assist in localising the site of the left median nerve lesion? Which investigation is most useful?

- A. Needle electrode examination
- B. High resolution sonography
- C. Magnetic resonance imaging (MRI)
- D. Angiogram
- E. Serial nerve conduction studies

Discussion

The patient had a brachial artery haematoma compressing the proximal median nerve. High resolution sonography of the arm showed a lesion compressing the median nerve 13 cm proximal to the medial epicondyle (Figs. 1A and 1B). The nerve was moderately swollen with loss of fascicular architecture. Doppler ultrasound revealed a haematoma (1.26 cm x 0.75 cm x 1.3 cm) medial and superficial to the brachial artery which was compressing and displacing the median nerve. A loss in continuity of the mural architecture was also noted on sonographic studies suggesting a puncture of the brachial artery. The haematoma was drained and the patient underwent physiotherapy with complete recovery.

The identification of concomitant entrapment or compressive neuropathy in the presence of severe peripheral neuropathy can be challenging. Demonstration of a dual

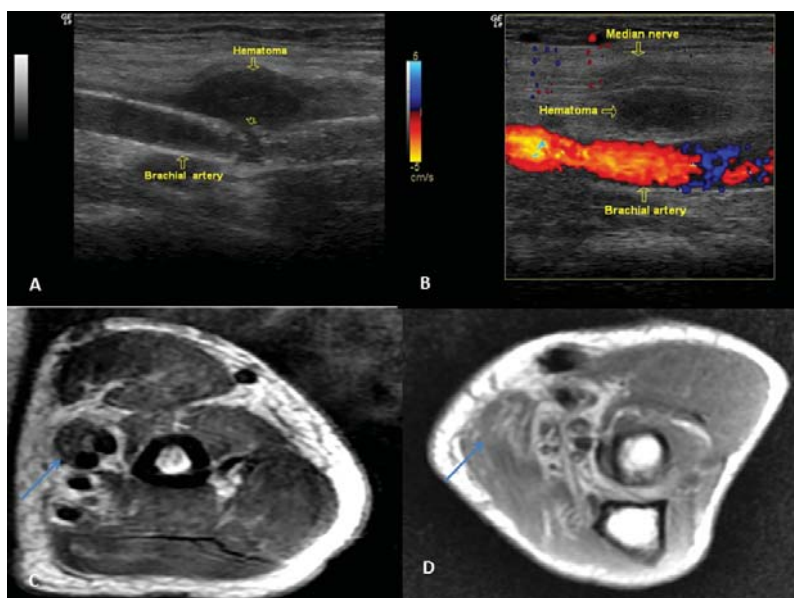


Fig. 1. Ultrasound in A and B show a haematoma medial and superficial to the brachial artery compressing the nerve as well as delineating the puncture of the median artery. In C, T1-weighted fat suppressed MRI shows a hypointense focal lesion in the median neurovascular bundle, which is approximately 13 cm proximal to the medial epicondyle of the humerus. In D, high signal intensity is seen in the pronator teres and flexor carpi radialis due to fatty infiltration and edema secondary to denervation.

Answer: B

pathology would necessitate the use of non-conventional electrodiagnostic studies or the need for imaging studies.

Electrodiagnostic studies in our patient were compatible with a severe diabetic peripheral neuropathy (Table 1). Additionally there was evidence of a carpal tunnel syndrome of the right. Confirmation of the clinical suspicion of a proximal median neuropathy on the left prompted us to examine the latency to the flexor pollicis longus which was prolonged when compared to the right. Needle electrode examination (NEE) can be useful in identifying the severity of nerve injury, and in differentiating a proximal from a distal median neuropathy. Needle findings can also help to ascertain the age of the injury (acute, subacute, or chronic) based on the extent of abnormal rest activity and the presence or absence of motor unit remodelling. In our case, NEE showed evidence of active denervation involving the left flexor pollicis longus, the flexor carpi radialis and the pronator teres consistent with an axonal loss lesion proximal to the elbow. Further localisation of median nerve pathology in the axilla to elbow segment is, however, limited due to absence of any muscular branches. Demonstration of a focal pathology in this segment would necessitate the use of imaging studies.

MRI scan of the left arm was able to localise the haematoma adjacent to the neurovascular bundle in the mid-arm, with associated signal changes within the median innervated muscles suggesting denervation edema. Further characterisation of the median nerve pathology (Figs. 1C and 1D), however, was not possible. High resolution sonography was able to demonstrate the nerve compression and the associated mural injury to the brachial artery.

Our case highlights the usefulness of peripheral nerve ultrasound in localising a proximal compressive median nerve pathology; especially so in the presence of concomitant diabetic peripheral neuropathy where nerve conduction studies were grossly abnormal. When a mononeuropathy is discovered ultrasound is well suited to look for entrapment or compressive pathologies. Peripheral nerve ultrasound is a painless, non-invasive useful adjunct to electrodiagnostic studies. It has several advantages: 1) it can be targeted directly to the area of concern, and 2) it can assess pulsatile flow and vascularity in real time. In cases where the underlying pathology is suspected to be vascular in nature, it may even be a superior modality compared to MRI scan by better delineating the vessels, looking for pseudoaneurysm and identifying an area of mural injury or “puncture”. However, ultrasound has its own set of limitations such as being operator dependent and not being able to depict muscle atrophy and edema to the extent of what MR images would do.¹

Table 1. Nerve Conduction Studies

	Right	Left
Median Nerve		
Sensory peak latency (ms)	5.3	Absent
Sensory amplitude (micV)	2.0	-
Sensory conduction velocity (m/s)	29.1	-
Motor distal latency (wrist) (ms) – Abductor pollicis brevis (APB)	5.4	7.3
Motor amplitude (wrist)(mV)	5.3	0.2
Motor latency (elbow) (ms)	11.15	16.95
Motor amplitude (elbow) (mV)	5.6	0.3
Motor conduction velocity (elbow-wrist) (m/s)	41.0	26.7
Motor distal latency (elbow) (ms) – Flexor pollicis longus (FPL)	4.2	6.45
Motor amplitude (elbow)(mV)	5.1	0.1
F wave latency (ms)	34.85	Absent
2L-INT (ms)	1.35	Median absent
Ulnar Nerve		
Sensory peak latency (ms)	Absent	Absent
Sensory amplitude (micV)	-	-
Sensory conduction velocity (m/s)	-	-
Motor distal at wrist (ms)	3.4	3.55
Motor amplitude at wrist (mV)	5.9	2.4
Motor latency (below elbow) (ms)	8.55	9.05
Motor amplitude (below elbow) (mv)	5.2	1.9
Motor conduction velocity (below elbow-wrist)(m/s)	37.9	37.8
Motor latency (above elbow) (ms)	9.6	12.4
Motor amplitude (above elbow) (m/s)	4.9	1.8
Motor conduction velocity (across elbow) (m/s)	49.5	39.4
Radial Nerve		
Sensory peak latency (ms)	2.25	2.05
Sensory amplitude (micV)	16.6	23.0
Sensory conduction velocity (m/s)	45.6	38.8
Med Antebrachial Cutaneous Nerve		
Peak latency (ms)	1.95	2.55
Amplitude (micV)	7.6	5.0
Conduction velocity (m/s)	50.3	43.6
Lateral Antebrachial Cutaneous Nerve		
Peak latency (ms)	2.6	Fistula
Amplitude (micV)	6.3	Fistula
Conduction velocity (m/s)	40.5	Fistula
Sural Nerve		
Peak latency (ms)	Absent	Amputated
Amplitude (micV)	Absent	Amputated
Conduction velocity (m/s)	Absent	Amputated

REFERENCES

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