



Nerve Ultrasound abnormalities mirrors the course of VZV sensory-motor radiculoplexopathy

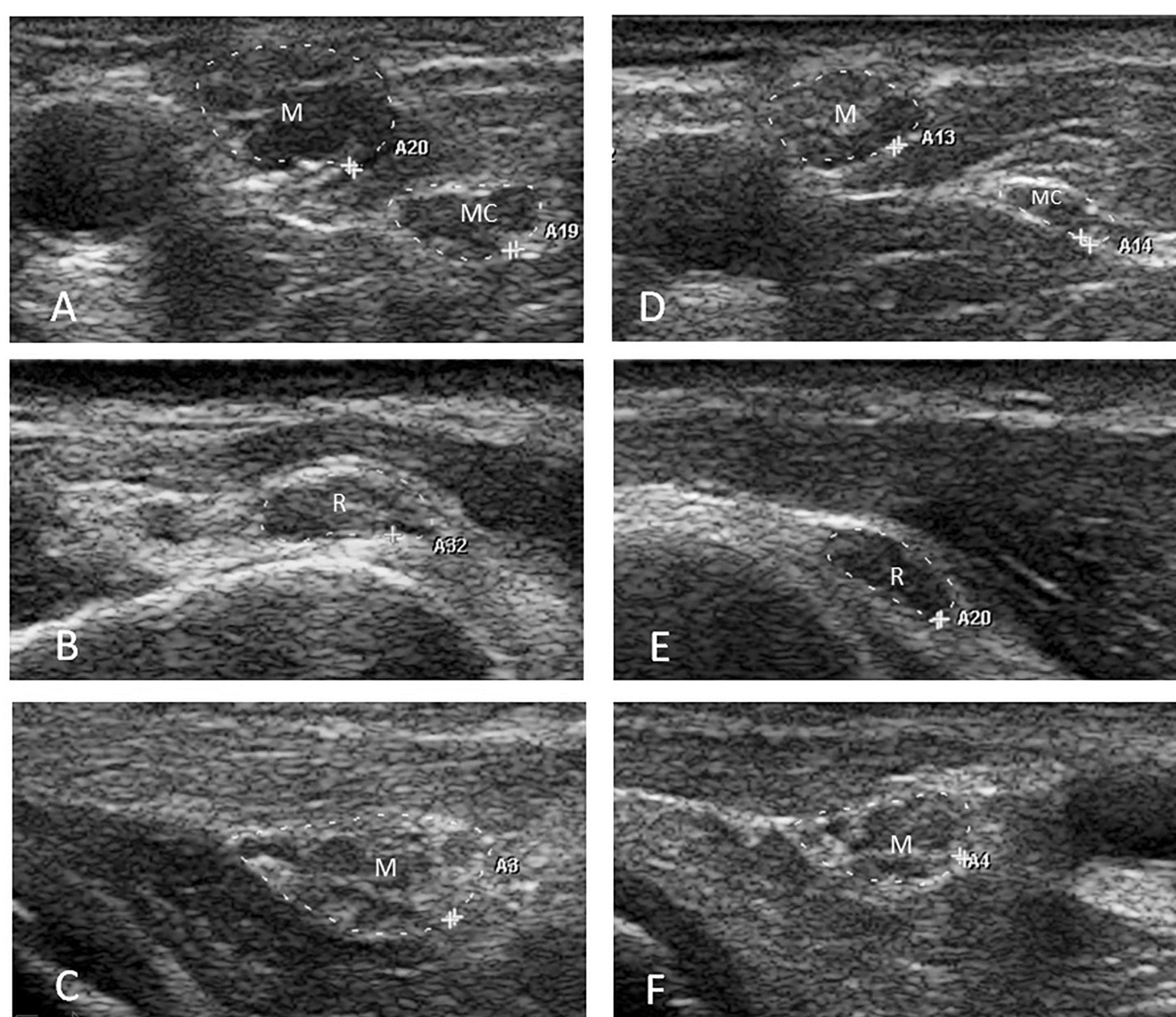
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Background: chronic lymphocytic leukemia (CLL) is the most common leukemia in adulthood. Neurological complications are mostly related to opportunistic infections, especially reactivation of varicella zoster virus (VZV). Motor radiculopathy in VZV infection is extremely rare, and thought to be secondary to spreading of inflammation from the dorsal root ganglia to the ventral roots. We describe a VZV brachial sensory-motor plexopathy in a CLL patients, in whom nerve Ultrasound (US) detected nerve abnormalities which mirrored the course of the disease.

Case report: a 77-year-old man, with a 20-yr history of CLL complained of neuropathic pain and vesicles in the left C5-C8 dermatomes. VZV was diagnosed and valaciclovir (2 g/day) started. In the subsequent days motor weakness occurred and rapidly progressed. When we first saw the patient 2 months later he had severe weakness in fingers extension (1/5 MRC), elbow flexion, index and digiti minimi abduction (2/5 MRC), elbow extension, shoulder abduction, wrist flexion and extension, thumb abduction, finger flexion (3/5 MRC). Vibratory sensation and reflexes at left upper limb were absent. Neurophysiology showed severe denervation and subacute neurogenic signs at left deltoid, biceps, brachioradialis, triceps, pectoralis major, serratus anterior, extensor and flexor carpi radialis, extensor digitorum communis, flexor digitorum superficialis, abductor pollicis brevis. Left radial, median and ulnar nerves sensory action potentials were absent. The pattern was consistent with brachial sensory-motor radiculoplexopathy.

Nerve US showed increased cross sectional area (CSA) of the median (16 mm² at the wrist, 20 mm² at elbow, 17 mm² at arm and axilla), musculocutaneous (9 mm²), ulnar (15 mm² at elbow, 17 mm² at arm), radial (9 mm² at omeral sulcus, 10 mm² at elbow), posterior interosseous (5 mm²), distal radial sensory (4 mm²) nerves (Fig 1 A-C). CSA of the cervical roots was increased (C5 7 mm², C6 11 mm², C7 13 mm²). The nerves at the right upper limb had CSA values within normal range. In the following 10 months the patient showed a slow progressive clinical improvement. Neurophysiology showed significant reinnervation in all muscles, and at ultrasound nerves regained normal CSA values (Fig 1 D-F).



Nerve ultrasound of the left arm at onset (A-C) and after ten months (D-F).

- A. Median nerve (CSA 17 mm²) and musculocutaneous nerve (CSA 9 mm²) at the proximal arm.
- B. Radial nerve at omeral sulcus: CSA 9 mm²
- C. Median nerve at elbow: CSA 20 mm²
- D Median nerve (CSA 9 mm²) and musculocutaneous nerve (CSA 4 mm²) at the proximal arm.
- E: Radial nerve at omeral sulcus: CSA 6 mm²
- F: Median nerve at elbow: CSA 10 mm²

Conclusions: In our patient with VZV sensory-motor plexopathy, US revealed nerves enlargement, likely secondary to nerve edema, in the acute phase, that progressively improved mirroring clinical resolution of the plexopathy. Whereas normalization or decrease of nerve CSA in response to therapy has already been described in immune-mediated neuropathies, this is the first case of VZV plexopathy in which enlarged nerves showed CSA reduction concurrently with clinical improvement.

Nerve US in post herpetic neuralgia has been previously reported in a patient with one year history of L5-S1 right ganglionopathy; sural and superficial peroneal nerves CSA were significantly smaller than those of the unaffected side. It is likely that in the chronic phase inflammation and edema decrease and axonal changes predominate accounting for the US pattern. In conclusion, the present case confirms the growing role of nerve US in the work-up of peripheral nerve diseases, adding a valuable contribution to neurophysiology and mirroring the clinical outcome.

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