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Objective: We present a case of an atypical progressive sensorimotor polyneuropathy with cranial nerve involvement, supported by neurophysiological assessments.

Materials: medical record

Method: A 62-year-old man was referred for a 4-month history of rapidly progressive gait disturbances and lower-limb dysesthesias. He had emigrated from Senegal a few months earlier and was employed as a factory worker. His medical history included type 2 diabetes mellitus and chronic kidney disease, but he had never reported neuropathic symptoms before, except for mild distal hypoesthesia and dysesthesia in the lower limbs. Neurological examination revealed a severe mixed sensorimotor polyneuropathy with distal predominance, impaired proprioception, and ataxic gait. Neurophysiological studies were performed, along with a diagnostic workup for autoimmune, infectious, and neoplastic causes. A detailed dietary history revealed recent consumption of large quantities of raw cassava in his native country.

Results: electrodiagnostic studies demonstrated a severe chronic axonal and demyelinating polyneuropathy with sensory predominance. Visual evoked potentials (VEPs) showed markedly delayed latencies, reduced amplitudes, and disorganized morphology bilaterally.

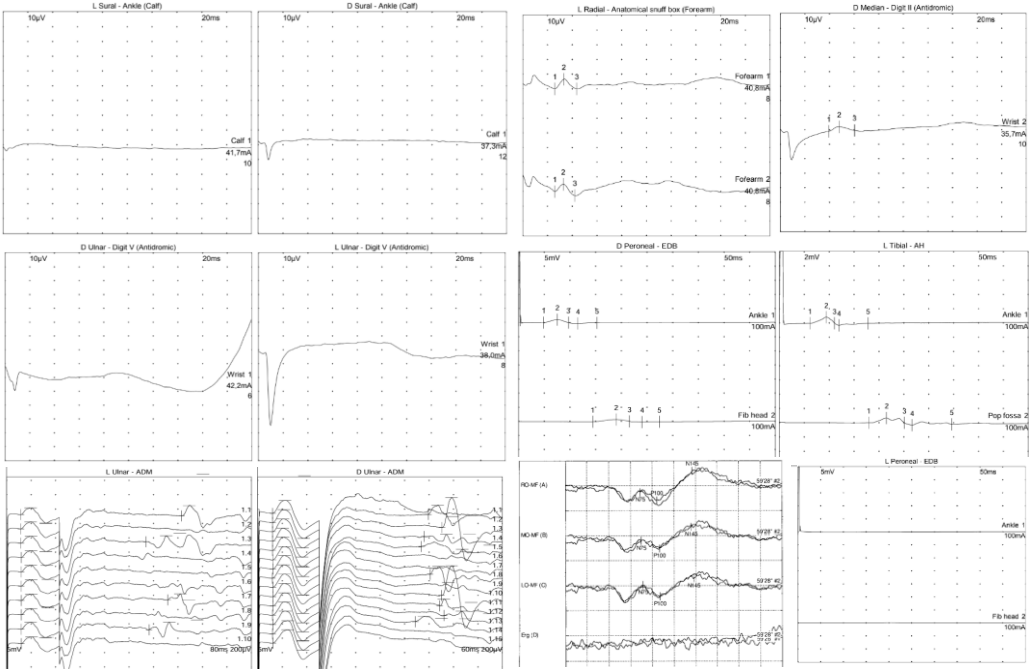


Fig. 1-6: Sensory action potential (SAP) recorded from limbs are absent except for right median nerve and left radial nerve, which has a very low amplitude and velocity conduction reduction (33 m/s; 38 m/s).

Figs. 7-9: Compound muscle action potentials (CMAPs) recorded from the right peroneal nerve (via extensor digitorum brevis) and from the left tibial nerve (via abductor hallucis) show low amplitude, disorganized morphology, and reduced conduction velocity (both 38 m/s). Right tibial nerve shows are similar (not shown here). CMAPs from left peroneal is absent. CMAPs recorded from upper limb (right median nerve, left and right ulnar nerve) are not shown here, but they are normal in amplitude, slightly low in latency and have velocity conduction reduced more distal than proximal (not shown here).

Figs. 10-11: F-waves recorded from the left ulnar nerve (abductor digiti minimi) show poor reproducibility and the presence of an A-wave on the left side. The right ulnar nerve shows disorganized waveform morphology and prolonged minimal latency (38.6 ms).

Fig. 12: Visual evoked potentials (VEPs) recorded from the right eye show atypical, disorganized morphology and prolonged latency. The left eye demonstrates similar findings (not shown). Brainstem auditory evoked potentials (BAEPs) are normal for waves I and II (not shown).

Discussion: The subacute worsening of neuropathy, without significant changes in glycated hemoglobin or body weight, together with electrophysiological findings consistent with a polyneuropathy showing marked sensory involvement, severe axonal and demyelinating features, and limited motor impairment, as well as optic nerve involvement, makes a purely diabetic etiology unlikely. Diabetic neuropathy typically presents as a chronic, distal, symmetric, sensorimotor polyneuropathy of predominantly axonal type. The clinical presentation in this case is more plausibly linked to the ingestion of cassava. The temporal relationship between symptom onset and cassava consumption supports this association. The condition related to chronic exposure to cyanogenic glycosides from inadequately processed cassava is known as Tropical Ataxic Neuropathy (TAN). It typically manifests with progressive sensory polyneuropathy (98%), sensory ataxia (84%), bilateral optic atrophy (48%), and bilateral sensorineural deafness (19%)¹. The relative contribution of axonal versus demyelinating pathology remains controversial and is still under investigation².

Conclusion: In patients presenting with severe or atypical neuropathy, alternative causes beyond diabetes should always be considered — including toxic and dietary exposures. This case underscores the crucial importance of a thorough environmental and dietary history in the diagnostic assessment of unexplained or atypical neuropathies, as it may reveal preventable or reversible causes. Moreover, it provides neurophysiological insight into a rare but significant neurological complication associated with chronic cassava ingestion.

References:

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