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A Case Report: Unilateral Sciatic Neuropathy Secondary to Drug-Induced Rhabdomyolysis

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INTRODUCTION

Peripheral neuropathy is an uncommon but clinically significant complication of rhabdomyolysis. Most reported cases involve bilateral sciatic neuropathy related to prolonged immobilization, compartment syndrome, or extensive muscle necrosis. In contrast, unilateral sciatic neuropathy after rhabdomyolysis is exceedingly rare, particularly in young patients without overt compartment syndrome. We report a case of severe unilateral sciatic neuropathy with persistent foot drop following drug-induced rhabdomyolysis, highlighting the diagnostic value of electrodiagnostic studies and clinical outcome.

CASE

An 18-year-old man with bipolar disorder was admitted after intentional overdose of psychotropic medications. He remained unconscious for a prolonged period and was diagnosed with rhabdomyolysis, with peak creatine kinase levels of approximately 51,000 U/L. After supportive treatment, mental status and laboratory findings improved, and he was discharged.
Following recovery, he developed severe burning pain in the right foot and posterior calf, accompanied by progressive ankle and toe weakness. Orthopedic evaluation and routine imaging were unremarkable, and he was referred for neurological assessment seven weeks after symptom onset. Neurological examination revealed marked weakness of right ankle dorsiflexion, plantar flexion, and eversion (MRC grade 3), absent great toe extension, mild knee weakness (MRC grade 4), and preserved hip strength. Atrophy of the right calf was noted compared with the contralateral side. Sensory loss involved the right foot and calf, sparing the medial calf. Distal deep tendon reflexes were reduced.
Nerve conduction studies showed absent motor responses in the right peroneal nerve, markedly reduced amplitudes and slowed conduction in the right tibial nerve, and absent sensory responses in the superficial peroneal and sural nerves. Needle electromyography demonstrated moderate to severe denervation and absent or markedly reduced motor unit potentials in sciatic-innervated muscles, with normal proximal and paraspinal muscles. These findings were consistent with a severe lesion of the right sciatic nerve.
Based on the temporal association with rhabdomyolysis, electrodiagnostic localization, and exclusion of radiculopathy or plexopathy, unilateral sciatic neuropathy secondary to prolonged immobilization was diagnosed. Despite rehabilitation and pain management, severe foot drop and muscle atrophy persisted.

Sensory NCS	Stimulation	Latency (ms)	Amplitude (μV)	
Rt. sural	Lat. malleolus	NR *	NR *	
Rt. sup. peroneal	Ant. Lat. Malleolus	NR *	NR *	
Lt. sural	Lat. malleolus	2.90	10.6	
Lt. sup. peroneal	Ant. Lat. malleolus	2.80	11.5	
Motor NCS	**Stimulation**	**Latency (ms)**	**Amplitude (mV)**	**Velocity (m/s)**
Rt. peroneal	Ankle	NR *	NR *	
	Fibular head	NR *	NR *	
	Popliteal fossa	NR *	NR *	
Rt. tibial	Ankle	5.5 *	3.6 *	
	Popliteal fossa	15.1 *	2.5 *	36.9 *
Lt. peroneal	Ankle	3.8	5.1	
	Fibular head	10.8	4.0	56.1
	Popliteal fossa	12.7	3.8	51.4
Lt. tibial	Ankle	3.5	23.3	
	Popliteal fossa	11.0	21.6	59.6

Figure 1) Nerve conduction study *Abnormal value.

Muscle	IA	Fib	PSW	IP
Rt. Peroneus longus	Normal	++	++	Partial
Rt. Med. Gastrocnemius	Normal	++	++	Partial
Rt. Ant. Tibialis	Normal	++	++	Partial
Rt. Extensor hallucis longus	Normal	++	++	None
Rt. Biceps femoris	Normal	++	++	Complete
Rt. Vastus Lateralis	Normal	-	-	Complete
Rt. Gluteus medius	Normal	-	-	Complete
Paraspinalis Rt. L3-S1	Normal	-	-	

Figure 2) Electromyography

CONCLUSION

This case demonstrates a rare but severe neurological complication of drug-induced rhabdomyolysis presenting as isolated unilateral sciatic neuropathy in a young patient. Even without overt compartment syndrome, secondary nerve injury may occur. When focal limb weakness or foot drop develops after rhabdomyolysis, early electrodiagnostic evaluation is essential for accurate localization, prognosis, and rehabilitation planning.