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Rhabdomyolysis with Peripheral Neuropathy: A Case Series and Literature Review

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Case series

Patients: Female, 59 • Male, 52 • Female, 29
Final Diagnosis: Rhabdomyolysis
Symptoms: Limited movement ability
Medication: —
Clinical Procedure: —
Specialty: Neurology

Objective: Unusual clinical course





Background: Rhabdomyolysis is a syndrome characterized by muscle necrosis and secretion of intracellular muscle components into the blood circulation. Acute compartment syndrome is a potential complication of severe rhabdomyolysis.

Case Reports: We report 3 cases of compartment syndrome-related peripheral neuropathy in alcoholic individuals with rhabdomyolysis. All patients were confirmed to have peripheral neuropathy by electrophysiologic studies.

Conclusions: Patients with underlying metabolic abnormalities, such as those related to long-term alcoholism, should be aware that rhabdomyolysis is likely to cause neurological abnormalities.

MeSH Keywords: Compartment Syndromes • Peripheral Nervous System Diseases • Rhabdomyolysis

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/911602>

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Background

Rhabdomyolysis is a syndrome characterized by muscle necrosis and secretion of intracellular muscle components into the blood circulation. Additionally, creatinine kinase levels are markedly increased and myoglobinuria may occur. Acute compartment syndrome is a potential complication of severe rhabdomyolysis. Any condition that increases the intra-compartmental pressure, such as trauma, ischemia-reperfusion injury, coagulopathy, and prolonged limb compression, creates the risk for compartment syndrome development. Metabolic changes associated with long-term excessive alcohol consumption may also increase the risk of neuropathy in patients with rhabdomyolysis. We reviewed 3 cases of peripheral neuropathy accompanied by rhabdomyolysis in alcoholic individuals. We also investigated the occurrence of rhabdomyolysis caused by alcohol consumption and the increased risk of neuropathy in cases of rhabdomyolysis due to alcohol consumption.

Case report

Three patients had rhabdomyolysis, concomitant compartment syndrome, and peripheral neuropathy that occurred with long-term alcohol consumption. Objective neurological examinations such as electromyography (EMG) and nerve conduction tests (NCT) were performed for these cases.

Case 1

A 59-year-old alcoholic woman was admitted to our institution because of limited movement ability of her lower limbs. Nine days before being admitted, she was found asleep while sitting with her body in a twisted position on an electric pad. She had fallen asleep while drinking alcohol and denied using any medication. She had a normal appearance at 8:00 pm the previous night and was found the following morning at 7:00 am; therefore, her approximate duration of immobilization was 11 hours. She received normal saline hydration at the local clinic for 9 days; however, her movement restriction in the lower limbs did not improve. Therefore, she presented to our hospital. At the time of admission, her vital signs were as follows; blood pressure 120/80 mmHg,

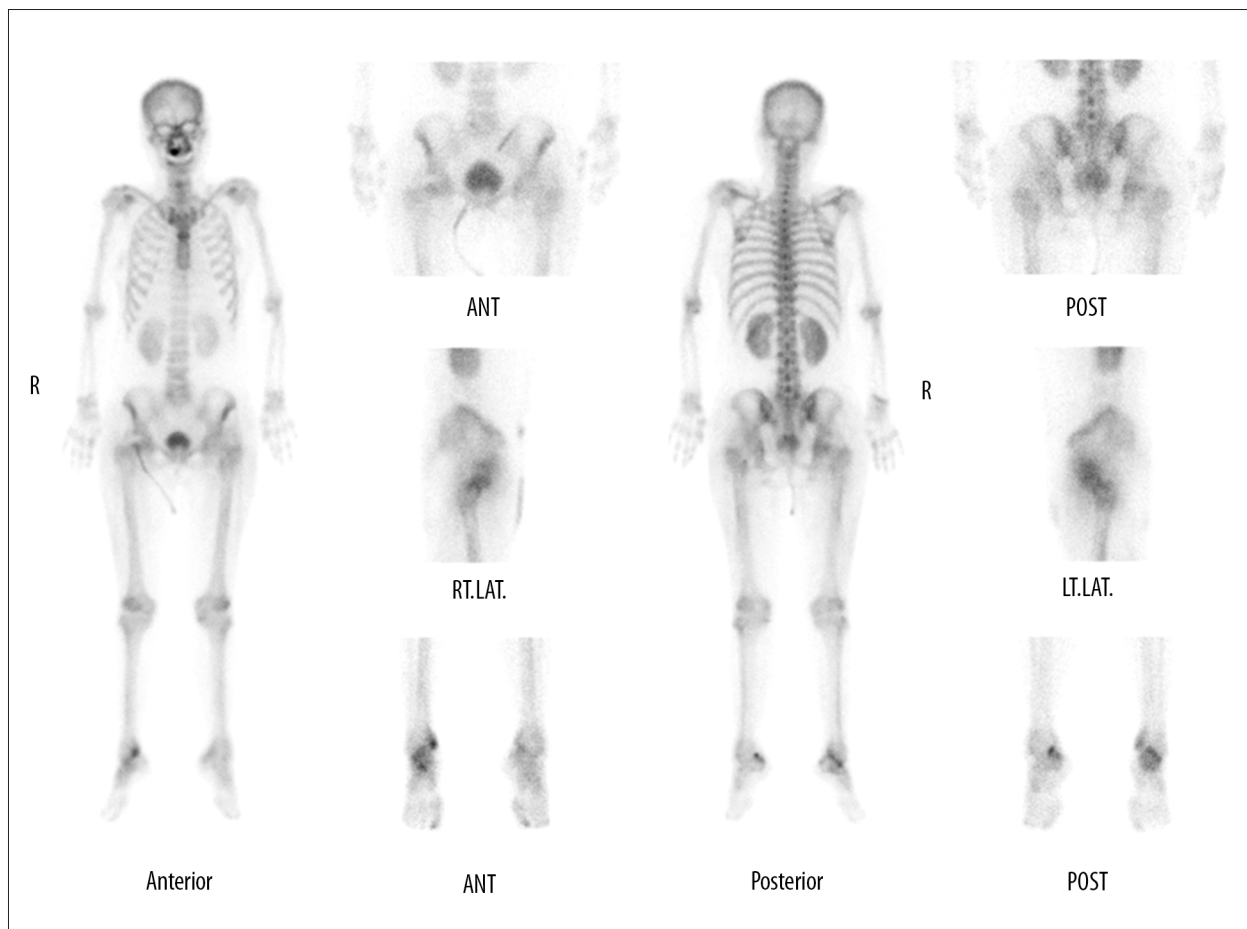


Figure 1. There was diffuse mild uptake along both gluteal muscles around the pelvic bone.

Table 1. Electrophysiologic abnormalities were observed in bilateral sciatic nerve lesions.

(A)

Nerve	DSL (m/s)	S amp (μV)	DML (ms)	M amp (mV)	NCV (m/s)	F-wave latency (ms)
Lt. median	3.1	19.5	3.9	12.6	54.9	26.1
Lt. ulnar	2.7	9.0	2.5	11.4	54.9	25.7
Lt. peroneal	NR	NR	NR	NR	–	NR
Lt. sural(S)/tibial(M)	NR	NR	NR	NR	–	NR
Rt. peroneal	NR	NR	NR	NR	–	NR
Rt. sural(S)/tibial(M)	3.7	0.7	3.9	5.0	40.8	50.4

DSL – distal sensory latency; S amp – sensory amplitude; DML – distal motor latency; M amp – motor amplitude; NCV – nerve conduction velocity; Lt – left; NR – no response; Rt – right.

(B)

Muscle	Insertional activity	Rest activity PSW/fibrillation	Recruitment	Volitional activity
Both VM	Normal	0	Normal	NMU
Both TA	Increased	2+	–	No MUAP
Both PL	Increased	2+	–	No MUAP
Both TP	Increased	2+	–	No MUAP
Both GMed	Normal	0	Normal	NMU
Both BFLH	Normal	0	Normal	NMU
Both BFSH	Increased	2+	Normal	NMU
Lt. GCM	Increased	2+	–	No MUAP
Rt. GCM	Increased	2+	Decreased	Decreased IP
Lumbar PSM	Normal	0	–	–

PSW – positive sharp wave; VM – vastus medialis; NMU – normal motor unit; TA – tibialis anterior; MUAP – motor unit action potential; PL – peroneus longus; TP – tibialis posterior; GMed – gluteus medius; BFLH – biceps femoris long head; BFSH – biceps femoris short head; Lt – left; GCM – gastrocnemius; Rt – right; IP – interference pattern; PSM – paraspinal muscle.

heart rate 74 beats/min, and body temperature 36.6°C. Her laboratory findings indicated a creatinine level of 1.47 mg/dL, lactate dehydrogenase (LDH) level of 1219 IU/L, creatine phosphokinase (CPK) level >10 000 U/L, and ethanol level of 0.9 mg/dL. Despite hydration and bed rest, her lower-limb movement was still restricted. Using a manual muscle test, her hip flexion and extension and knee flexion and extension had Medical Research Council (MRC) grade 4 power (active movement against gravity and resistance), bilateral ankle flexion had MRC grade 0 power, right ankle extension had MRC 0 grade power (complete paralysis), and left ankle extension had MRC 1 grade power (minimal contraction). She had decreased sensations to touch and pain on

the dorsum muscles of both feet. No ankle jerk or pathologic reflexes were observed. A whole-body bone scan was performed after administration of 25 mCi of Tc^{99m} hydroxy diphosphonate (HDP). There was diffuse mild uptake along both gluteal muscles around the pelvic bone (Figure 1). Electrophysiologic abnormalities suggesting bilateral sciatic nerve lesions were observed with NCT (Table 1A) and EMG (Table 1B). Therefore, she was diagnosed with bilateral sciatic neuropathy associated with rhabdomyolysis and underwent fluid therapy and rehabilitation. After 44 days, she was discharged without any sequelae. Her laboratory findings at discharge were creatinine 0.72 mg/dL, LDH 272 IU/L, and CPK 253 U/L.

Table 2. Electrophysiologic abnormalities were observed in Lt. Dorsal ulnar cutaneous nerve, Lt. Radial nerve, Lt. Superficial and deep peroneal nerve lesions.

(A)

Nerve	DSL (m/s)	S amp (μV)	DML (ms)	M amp (mV)	NCV (m/s)	F-wave latency (ms)
Lt. radial	2.8	6.4	NR	NR	NR	–
Lt. median	3.1	14.4	3.2	18.1	53.5	26.9
Lt. ulnar	2.9	24	2.3	13.3	51.6	30.6
Lt. dorsal ulnar	NR	NR	–	–	–	–
Rt. radial	2.7	18.8	–	–	–	–
Rt. median	3.1	19.8	3.4	23.5	55.1	26.6
Rt. ulnar	3.6	36	2.6	13.9	56.2	26.8
Rt. dorsal ulnar	2.4	11.4	–	–	–	–
Lt. peroneal	3.8	1.2	NR	NR	NR	NR
Lt. sural/tibial	3.6	4.1	3.3	24.1	41.7	48.1
Rt. peroneal	3.9	6.8	3.0	5.4	42.3	47.1
Rt. sural/tibial	3.4	7.4	3.7	18.8	40.6	50.4

DSL – distal sensory latency; S amp – sensory amplitude; DML – distal motor latency; M amp – motor amplitude; NCV – nerve conduction velocity; Lt – left; NR – no response; Rt – right.

(B)

Muscle	Insertional activity	Rest activity PSW/fibrillation	Recruitment	Volitional activity
Lt. brachioradialis	Increased	2+	–	No MUAP
Lt. EDC	Increased	2+	–	No MUAP
Lt. APB	Normal	0	Normal	NMU
Lt. FDI	Increased	1+	Normal	NMU
Lt. TA	Decreased	0	–	No MUAP
Lt. PL	Decreased	0	–	No MUAP
Lt. BF, short head	Normal	0	Normal	NMU

PSW – positive sharp wave; MUAP – motor unit potential; EDC – extensor digitorum communis; APB – abductor pollicis brevis; NMU – normal motor unit; FDI – first dorsal interosseous; TA – tibialis anterior; PL – peroneus longus; BF – biceps femoris.

Case 2

A 52-year-old alcoholic man was admitted to the Emergency Department of our institution. He drank alcohol and ingested sleeping pills (lorazepam 3 mg) the night before he presented to the Emergency Department. On the morning of admission, he could not move his left arm or both legs. Dents were found

in his left thigh, chest, and face. He had reduced sensation of the left ulnar distribution, which was accompanied by a tingling sensation. He could not move his left wrist extensor and finger extensor smoothly and could not lift his left thumb. Furthermore, he could not move his left ankle dorsiflexor smoothly. At the time of admission, his vital signs were as follows: blood pressure 130/75 mmHg, heart rate 68 beats/min,

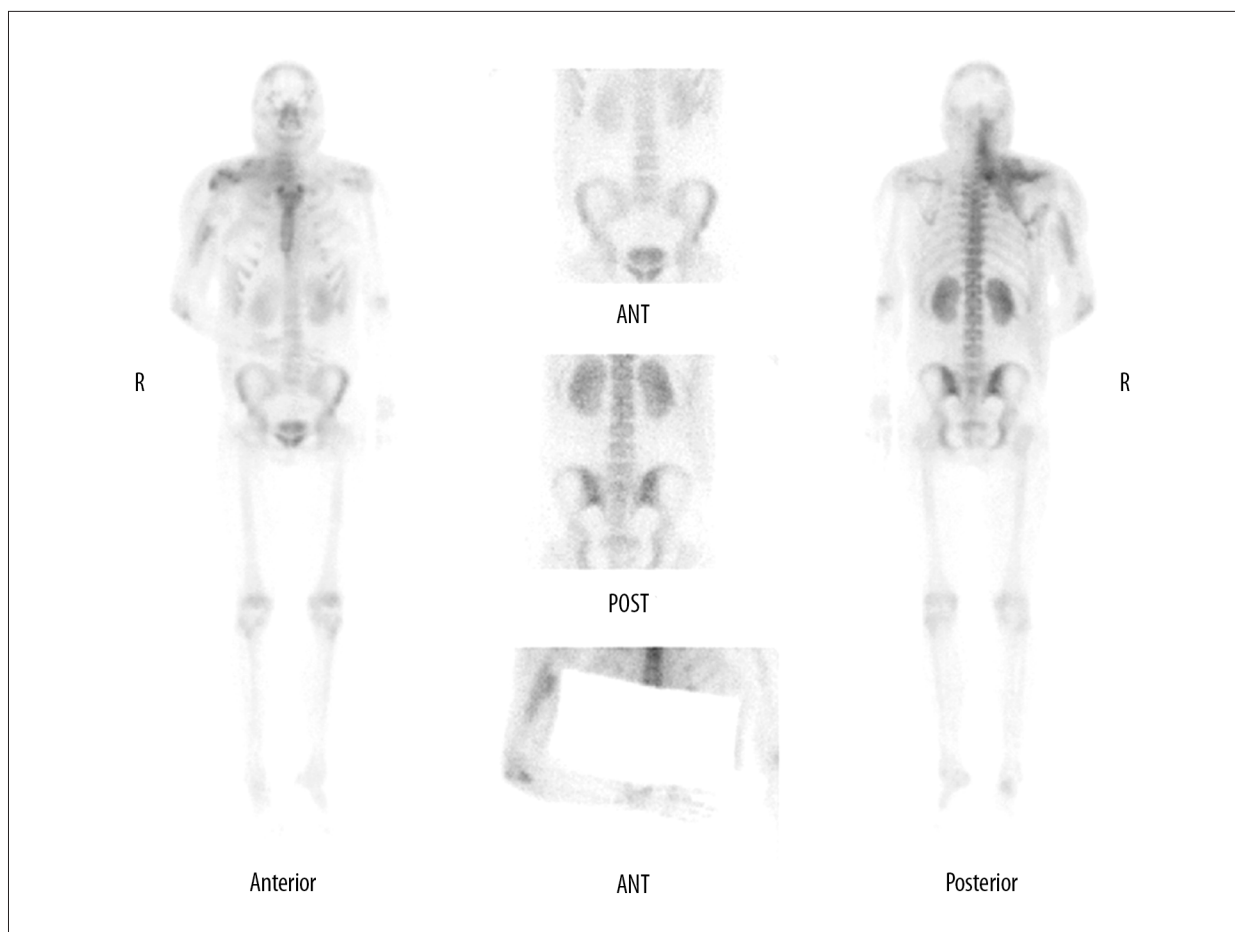


Figure 2. There was diffusely increased soft-tissue uptake at the right neck, shoulder, and upper arm, suggesting muscle injury.

and body temperature 36.8°C. Initial laboratory findings showed a creatinine level of 1.62 mg/dL, LDH level of 4030 IU/L, CPK level >10,000 U/L, and ethanol level of 1.1 mg/dL. During NCS (Table 2A) and EMG (Table 2B), electrophysiologic abnormalities were observed in the left dorsal ulnar cutaneous nerve, left radial nerve, and left superficial and deep peroneal nerves. He was diagnosed with complete injury of the left ulnar, radial, and common peroneal nerves associated with rhabdomyolysis and underwent fluid therapy and rehabilitation. He was discharged without any sequelae after 14 days. Laboratory findings at discharge were creatinine, 0.79 mg/dL, LDH 383 IU/L, and CPK 629 U/L.

Case 3

A 29-year-old alcoholic woman was admitted to the Emergency Department of our institution. She drank alcohol and ingested sleeping pills (zolpidem 20 mg) the night before admission. She reported dysesthesia of the right shoulder girdle area and posterior neck area. During the manual muscle test, right elbow flexion had MRC grade 4 power (active movement against gravity and resistance), right elbow extension had MRC

grade 3 power (weak contraction against gravity), right wrist extension had MRC grade 0 power (complete paralysis), and right finger extension had MRC grade 1 power (minimal contraction). At the time of admission, her vital signs were blood pressure 110/70 mmHg, heart rate 72 beats/min, and body temperature 36.7°C. Initial laboratory findings showed a creatinine level of 4.96 mg/dL, LDH level of 1229 IU/L, CPK level >10 000 U/L, and ethanol level of 1.0 mg/dL. A whole-body bone scan was performed after administration of 25 mCi of Tc^{99m} HDP. The study showed diffusely increased soft-tissue uptake at the right neck, shoulder, and upper arm, suggesting muscle injury (Figure 2). During NCS (Table 3A) and EMG (Table 3B), electrophysiologic abnormalities were observed in the right proximal radial nerve. She was diagnosed with incomplete axonal injury of the right proximal radial nerve associated with rhabdomyolysis and underwent fluid therapy and rehabilitation. After 37 days, she was discharged without any sequelae. Laboratory findings at discharge were creatinine 0.8 mg/dL, LDH 305 IU/L, and CPK 46 U/L.

Table 3. Electrophysiologic abnormalities were observed in right proximal radial nerve lesions.

(A)

Nerve	DSL (m/s)	S amp (μV)	DML (ms)	M amp (mV)	NCV (m/s)	F-wave latency (ms)
Lt. radial	2.6	29.1	1.95	4.6	58.8	–
Lt. median	2.5	31.0	2.9	8.5	62.5	23.5
Lt. ulnar	2.3	18.0	2.1	12.3	62.5	23.8
Rt. radial	3.0	3.4	2.5	1.1	54.4	–
Rt. median	2.7	19.4	2.4	9.5	57.7	24.6
Rt. ulnar	2.5	15.6	2.2	13.1	62.2	25.3

DSL – distal sensory latency; S amp – sensory amplitude; DML – distal motor latency; M amp – motor amplitude; NCV – nerve conduction velocity; Lt – left; Rt , right.

(B)

Muscle	Insertional activity	Rest activity PSW/fibrillation	Recruitment	Volitional activity
Rt. deltoid	Normal	0	Normal	NMU
Rt. biceps brachii	Normal	0	Normal	NMU
Rt. brachioradialis	Increased	1+	–	No MUAP
Rt. triceps	Increased	1+	–	No MUAP
Rt. EDC	Increased	2+	–	No MUAP
Rt. ECR	Increased	1+	–	No MUAP
Rt. FDI	Normal	0	Normal	NMU

PSW – positive sharp wave; Rt – right; NMU – normal motor unit; MUAP – motor unit potential; EDC – extensor digitorum communis; ECR – extensor carpi radialis; FDI – first dorsal interosseous.

Discussion

Rhabdomyolysis is a syndrome characterized by muscle necrosis and secretion of intracellular muscle components such as electrolytes, myoglobin, and creatinine kinase in the blood circulation. The most common clinical symptoms associated with rhabdomyolysis are trauma, immobilization, and overexertion. Its causes include cardiac surgery, prescription drug use (such as lipid-lowering drugs), alcohol use, and illegal drug use (such as cocaine use). Clinical manifestations of rhabdomyolysis may include muscle pain, decreased muscle strength, black urine, and acute renal failure. Acute compartment syndrome is a potential complication of severe rhabdomyolysis; however, compartment syndrome is not a rare finding in rhabdomyolysis [1,2]. Acute compartment syndrome can occur in any distinct anatomic compartment bound by unyielding fascial membranes.

The severity of rhabdomyolysis varies. This variability is not fully understood, but it can be related to physical fitness, morphological characteristics, and sex [3]. Risk factors for acute compartment syndrome include young age and male sex [4,5]. After achieving complete body growth, the size of the compartment is usually fixed; however, young people can have relatively large volumes of muscles. In contrast, elderly individuals may have smaller hypotrophic muscles, and their relatively higher blood pressure can provide tolerance to high tissue pressure [6].

Metabolic changes associated with long-term alcohol consumption may also increase the risk of neuropathy in patients with rhabdomyolysis. Alcohol-related rhabdomyolysis has been attributed to direct toxic effects on muscles and secondary metabolic changes associated with alcohol abuse. Repeated administration of ethanol can increase serum CPK activity and

produce ultrastructural changes in human skeletal muscles [7]. Furthermore, repeated administration of an alcohol-based diet to dogs resulted in subclinical myopathy, and ethanol consumption can result in hyperpolarization of the resting transmembrane potential of skeletal muscle fibers and a significant increase in Na-K-ATPase activity accompanied by increased sodium transport-dependent respiration [8]. With traumatic rhabdomyolysis, pressure causes necrosis of the muscles, and diffusion of calcium, sodium, and water into damaged cells occurs. Combined alcohol-induced direct neurologic abnormalities and crush injuries may also increase the risk of neuropathy in patients with rhabdomyolysis.

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Conclusions

We examined 3 cases of peripheral neuropathy in patients with rhabdomyolysis due to immobilization. Patients with underlying metabolic abnormalities caused by conditions such as long-term alcohol consumption should be aware that rhabdomyolysis is likely to cause neurological abnormalities. More careful observation is needed for these cases.

Conflicts of interest

None.