

Bilateral Lumbar Plexopathy and Rhabdomyolysis Complicating Carbon Monoxide Poisoning

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Introduction

Carbon monoxide (CO) is a colorless, odorless gas produced primarily as a result of incomplete combustion of any carbonaceous fossil fuel. The clinical symptoms of CO poisoning are variable including nonspecific viral illnesses, neuropsychiatric symptoms and delayed neurological syndrome. Previous reports of rhabdomyolysis due to CO poisoning are rare. Neuropathy is also rarely documented after CO poisoning. We present a case of CO poisoning with rhabdomyolysis that resulted in bilateral lumbar plexopathy. A case report: A 32-year-old man visited the rehabilitation department because of decreased muscle strength in his bilateral lower extremity during 40 days after the suicide event. From patient's history taking and medical record, we found out that he was affected by burning coal. He was a healthy employee prior to the incident and had no history of medical problem. He attempted suicide by burning coal in a closed space. He was admitted to internal medicine for 5 days and discharged at his will without permission. Initial laboratory data were as follows; creatine phosphokinase 24480 IU/L, CK-MB 45.5 IU/L, Troponin T 0.042 ng/ml, lactate dehydrogenase 985 U/L. These findings suggested that rhabdomyolysis had occurred due to CO poisoning. For treating rhabdomyolysis, only conservative treatment without surgery was done. The patient complained of numbness in the left medial knee area and lower extremity weakness after CO poisoning. Physical examination showed symmetrical knee jerk, diminished touch and pinprick sensation in the left medial knee. A manual muscle test identified weakness of bilateral hip flexor (MRC grade 2) and knee extensor (MRC grade 2). However, upper extremities and ankle strength were normal. We conducted nerve conduction study and electromyography 40 days after onset. There was no response in bilateral saphenous nerve stimulation in sensory study. In the motor study, bilateral femoral nerves showed no response. Except saphenous and femoral nerves, the other nerve conduction study in both lower extremities showed normal. In the electromyography, bilateral vastus medialis, iliopsoas showed fibrillation potential (FP) and positive sharp wave (PSW) at rest, and no motor unit action potential (MUAP) during voluntary contraction. Results showed bilateral lumbar plexopathy. (Figure 1.) 5 months after onset, we conducted follow-up nerve conduction study and electromyography. Nerve conduction study was almost the same compared to the previous study. In electromyography, bilateral iliopsoas showed FP and PSW at rest and partial to complete recruitment patterns and bilateral vastus medialis showed FP and PSW at rest and no MUAP during voluntary contraction. These findings suggested that regeneration change started from the proximal part. After taking a 6-month rehabilitation program for lower extremity weakness including therapeutic exercise and electrical stimulation therapy, the patient could walk a distance of 300-meters without gait aids.

motor				EMG		
Nerve	Latency(ms)	Amplitude	Velocity(m/s)	Muscle	Resting	On volition
Rt.Median	3.44/7.45	10.7/10.6	54.9	Paralumber	Silent	
Rt.Ulnar	2.86/6.46	12.6/12.5	58.4	Rt. Iliopsoas	P&F(2+)*	No motor unit potential*
Lt.Median	3.44/7.60	11.3/10.2	52.8	Rt. Rectus Femoris	P&F(2+)*	No motor unit potential*
Lt.Ulnar	3.18/6.56	11.0/10.1	62.1	Rt. Vastus medialis	P&F(2+)*	No motor unit potential*
Rt.Tibial	5.99/15.42	25.1/19.6	41.2	Rt. Peroneus longus	Silent	Normal motor unit potential Full recruitment
Rt.Peroneal	3.13/10.89	8.4/7.9	43.8	Rt. Tibialis anterior	Silent	Normal motor unit potential Full recruitment
Rt.Femoral	Not evoked*			Rt. Gastrocnemius	Silent	Normal motor unit potential Full recruitment
Lt.Tibial	5.83/14.38	25.4/18.1	43.3	Lt. Iliopsoas	P&F(2+)*	No motor unit potential*
Lt.Peroneal	3.54/10.83	9.0/8.8	46.6	Lt. Rectus Femoris	P&F(2+)*	No motor unit potential*
Lt.Femoral	Not evoked*			Lt. Vastus medialis	P&F(2+)*	No motor unit potential*
				Lt. Peroneus longus	Silent	Normal motor unit potential Full recruitment
				Lt. Tibialis anterior	Silent	Normal motor unit potential Full recruitment
				Lt. Gastrocnemius	Silent	Normal motor unit potential Full recruitment
sensory				FU EMG		
Nerve	Latency(ms)	Amplitude	Velocity(m/s)	Muscle	Resting	On volition
Rt.Median	2.55	51.1	50.9	Paralumber	Silent	
Rt.Ulnar	2.19	32.1	54.9	Rt. Iliopsoas	P&F(2+)*	Polyphasic motor unit potential* Reduced recruitment*
Lt.Median	2.5	56	52	Rt. Rectus Femoris	P&F(2+)*	No motor unit potential*
Lt.Ulnar	2.4	36.6	50.1	Rt. Vastus medialis	P&F(2+)*	No motor unit potential*
Rt.Sural	2.55	25.4	41.1	Lt. Iliopsoas	P&F(2+)*	Polyphasic motor unit potential* Reduced recruitment*
Rt.Peroneal	2.6	30.2	46.1	Lt. Rectus Femoris	P&F(2+)*	No motor unit potential*
Rt.Saphenous	Not evoked*			Lt. Vastus medialis	P&F(2+)*	No motor unit potential*
Lt.Sural	2.5	32.8	42	Lt. Iliopsoas	P&F(2+)*	Polyphasic motor unit potential* Reduced recruitment*
Lt.Peroneal	2.66	32.9	45.2	Lt. Rectus Femoris	P&F(2+)*	No motor unit potential*
Lt.Saphenous	Not evoked*			Lt. Vastus medialis	P&F(2+)*	No motor unit potential*

fig1. Initial and follow up nerve conduction study and electromyography